Third Edition

STROKE REHABILITATION

A Function-Based Approach

Glen Gillen

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chapter 1

Pathophysiology, Medical Management, and Acute Rehabilitation of Stroke Survivors

key terms

acute management
decubitus ulcer
early mobilization
hemorrhagic stroke
intensive care unit (ICU)
ischemic stroke
stroke diagnosis
stroke management
stroke prevention

chapter objectives

After completing this chapter, the reader will be able to accomplish the following:

1. Describe the pathophysiology of stroke.
2. Explain the diagnostic workup of stroke survivors.
3. Understand the medical management of various stroke syndromes.
4. Describe interventions to prevent the recurrence of stroke and its complications.
5. Understand normal and abnormal responses to acute stroke rehabilitation.
6. Be familiar with standardized assessments used during acute stroke rehabilitation.
7. Implement a comprehensive treatment that is safe for the acute and ICU settings.
8. Write appropriate goals for the acute and ICU settings.
9. Be able to prevent secondary complications such as skin breakdown and contracture after stroke.

Pathophysiology and Medical Management of Stroke

Matthew N. Bartels

PREVALENCE AND IMPACT OF STROKE

Stroke remains the third leading cause of mortality in the United States after cardiovascular disease and cancer, accounting for 10% to 12% of all deaths.[15,127] Globally, stroke is the second leading cause of mortality in developed nations with 4.5 million deaths every year.[109] An estimated 550,000 strokes occur each year, resulting in 150,000 deaths and more than 300,000 individuals with significant disability.[119] The United States has an estimated 3 million stroke survivors today, which is double the number of survivors 25 years ago.[54] The economic impact of stroke in 2007 was estimated at $62.7 billion, markedly increased from the estimate in 2001 of $30 billion, of which $17 billion

were direct medical costs and $13 billion were indirect costs from lost productivity.[119] Fortunately, modern medical interventions (mostly risk factor modifications) have decreased stroke mortality by approximately 7% per year in industrialized nations since 1970.[15] The advances continue, but with increased cost of care for more advanced treatments.

EPIDEMIOLOGY OF STROKE

Stroke is essentially a preventable disease with known, manageable risk factors.[16] The established risk factors for stroke include hypertension, cigarette smoking, obesity, elevated serum fibrinogen levels, diabetes, a sedentary lifestyle, and the use of contraceptives with high doses of estrogen.[101] The most important and easily treated of these risk factors is systolic hypertension. In the Multiple Risk Factor Intervention Trial, 40% of strokes were attributed to systolic blood pressures greater than 140 mm Hg.[130] Stroke incidence also increases exponentially with aging, with an increase in stroke from three in 100,000 individuals per year in the third and fourth decades of age to 300 in 100,000 individuals per year in the eighth and ninth decades of life.[16] Eighty-eight percent of stroke deaths occur among persons aged 65 years or older[15] Table 1-1 outlines modifiable and nonmodifiable risks.

Stroke prevention interventions have reduced mortality in industrialized nations primarily through treating hypertension in the elderly. Another cause of decreased mortality has been the establishment of dedicated stroke units that can prevent acute death and later development of life-threatening complications.

PATHOGENESIS AND PATHOLOGY OF STROKE

Definition and Description of Stroke Syndromes

Stroke. Stroke is essentially a disease of the cerebral vasculature in which a failure to supply oxygen to brain cells, which are the most susceptible to ischemic damage, leads to their death. The syndromes that lead to stroke compose two broad categories: ischemic and hemorrhagic stroke. Ischemic strokes account for approximately 80% of strokes, whereas hemorrhagic strokes account for the remaining 20%.[128]

Transient Ischemic Attack. Symptoms of a transient ischemic attack (TIA) include the focal deficits of an ischemic stroke within a clearly vascular distribution, but TIAs are reversible defects because no cerebral infarction ensues. The causes of TIAs can be thrombotic and embolic and could result from a cerebral vasospasm. By definition, the effects of TIAs must resolve in less than 24 hours. Since 35% of patients who have had a TIA will have a stroke within five years, they should have a complete evaluation for cerebrovascular disease and sources of embolism.[167] The treatment of TIAs depends on the source of the emboli or thrombi and can include anticoagulation therapy and/or surgery.

Ischemic Stroke

An ischemic stroke is the most common form of stroke with various causes. The one common endpoint among all the different subtypes of ischemic strokes is that injury results from tissue anoxia caused by an interruption of cerebral blood flow.

Table 1-1

Modifiable and Nonmodifiable Risks

TYPE OF RISK	RELATIVE RISK (PER 1000 PERSONS)
Modifiable risks	
Hypertension	4.0 to 5.0
Cardiac disease	2.0 to 4.0
Atrial fibrillation	5.6 to 17.6
Diabetes mellitus	1.5 to 3.0
Cigarette smoking	1.5 to 2.9
Alcohol abuse	1.0 to 4.0
Hyperlipidemia	1.0 to 2.0
Nonmodifiable risks	
Age	1 to 2/1000 at age 45– to 54–years-old to 20/1000 at age 75– to 84–years-old
Gender	1.2 to 2.1
Race (black or Hispanic)	2.0
Heredity	1.8 to 3.1

Embolic Stroke. Cerebral embolic strokes are the most common subtype of ischemic stroke. Embolic strokes usually are characterized by an abrupt onset, although they also can be associated with stuttering symptoms. Usually no heralding events occur, such as TIAs or previous small strokes evolving into larger strokes.[83] A warning with microemboli that cause smaller events are uncommon, and the usual clue to a possible embolic source is a completed stroke.[128] The source of approximately 40% of embolic strokes is unknown, even after the common sources have been evaluated extensively. Most embolic strokes of known cause occur after emboli that are cardiac in origin.[27] The second most common sources of emboli are atherothrombotic lesions that result in artery-to-artery embolisms. These lesions can be in the aorta, the carotid and vertebrobasilar systems, and, less frequently, smaller arteries.

Sources of Emboli

Cardiac Sources. Cardiac emboli can develop from numerous areas in the heart. Cardiac dysrhythmias, structural anomalies, and acute infarctions are the usual sources of emboli. The most common source of an embolism is the classical pattern of thrombosis in the left atrium of patients with atrial fibrillation. The usual mechanism of thrombus formation in atrial fibrillation is by clot formation in the left atrial appendage. This then breaks off and creates an embolus that can move through the arterial system. Patients older than 60 years are particularly prone to this type of embolization. Embolism is not limited to the brain, and infarction can occur in the kidneys, peripheral tissues, or any other location.

The most common cardiac structural cause of a cerebral embolism is due to a myocardial infarction.[83] In patients with left ventricular infarcts, particularly anterior wall and apical infarctions, the endocardial damage associated with a subendocardial or transmural infarction is an excellent nidus (a focal point where bacteria or other infectious agents thrive) for thrombus formation. The emboli most often develop during the first several weeks after the infarction, although the risk for developing them can persist for much longer.

Valvular heart disease also can result in thrombi, but they more frequently develop after valve replacement rather than result directly from the native valve. More commonly the native valvular heart disease causes the patient to be in atrial fibrillation and then to develop an embolus. Mechanical heart valves (e.g., St. Jude valves) are much more likely to cause emboli than porcine (tissue) valves, so patients with the mechanical type always continue to receive anticoagulation therapy.

Much less common sources of cardiac emboli are the vegetations resulting from bacterial endocarditis. These emboli cause small septic infarcts called mycotic aneurysms, which are at high risk of conversion to hemorrhagic infarcts. Other rare causes of cardiac emboli are atrial myxomas, which are tumors of the heart endocardium. In addition, embolic infarctions also may result from cardiac and thoracic surgery.[83]

Cardiac emboli usually (80% of the time) occlude the middle cerebral artery, 10% of cardiac emboli occlude the posterior cerebral artery, and the rest occlude the vertebral artery or its branches.[83] Anterior cerebral artery embolization from the heart is rare. The severity of the clinical syndrome is related to the size of the embolus. An embolus of 3 to 4 mm can cause a large stroke by occluding the larger brain arteries. Blood clots undergo lysis over a few days with the establishment of recanalization through the clot. Because clots naturally lyse, a stroke can convert from ischemic to hemorrhagic when reperfusion distal to the occlusion is present, because the blood vessels in the ischemic distribution may no longer be intact. This can lead to leakage from these damaged arteries, arterioles, and capillaries, leading to a phenomenon called hemorrhagic conversion. The possibility of hemorrhagic conversion contraindicates the use of anticoagulation therapy as initial treatment for large embolic strokes.

Vascular Sources. Strokes vascular in origin are far less common than cardiac strokes but are still one major type of embolic stroke. The sources of vascular emboli are usually atheromatous plaques in the walls of the aorta, carotid arteries, or smaller vessels in the cerebral circulation. Platelet activation and the formation of a fibrin clot can occur rapidly. The most common areas affected by the emboli of the vascular system are the same as those affected by cardiac sources of emboli. The most common areas for ulcerated plaques in the cerebral blood supply are the aorta and the proximal internal carotid artery. The plaques in the carotid artery can be visualized by Doppler sonography of the carotid artery system.[128]

Paradoxical Sources. Congenital atrial septal defects can create the opportunity for emboli to cross from the right-sided (venous) circulation to the left-sided (arterial) circulation, a rare source of cerebral emboli. A common source of paradoxical embolic material is deep venous thrombosis (DVT). The modern techniques of transesophageal echocardiography with a "bubble study" help identify patients at risk for this condition. One performs a bubble study by injecting a small bolus of air into the venous circulation while the echocardiographer observes the heart. If the air bolus, which is seen easily, has no portion cross over to the left-sided circulation, then no shunt is present. If the bubbles cross into the left-sided circulation, then a shunt is possible. One of the most common atrial shunting abnormalities is a patent foramen ovale. In young patients or patients who have had TIAs or strokes, the treatment of choice is surgical repair of the lesion.

Unknown Sources. Thrombi of unknown source often occur in patients with known hypercoagulability syndromes. These syndromes can result from acquired diseases (e.g., lupus anticoagulant and metastatic tumors) or inborn errors of the coagulation system (e.g., protein S and C deficiencies).

Surgery or medication therapies such as estrogen replacement can induce iatrogenic causes of hypercoagulable states. Even when the patient is known to be in a hypercoagulable state, the source of the emboli may remain unknown. In many patients the entire workup is unrevealing.

Thrombotic Stroke

A thrombotic stroke can result from a variety of causes, but most causes are related to the development of abnormalities in the arterial vessel wall. Atherosclerosis, arteritis, dissections, and external compression of the vessels are causes. In addition, some patients with hematological disorders develop thrombosis. The spectrum of disease includes stroke and TIA, and often the difference between a thrombotic and an embolic stroke may be difficult to determine. Thrombosis and embolism are often both present, especially in patients with atherosclerotic disease. The exact mechanism of infarction from thrombosis is still being debated, but atherosclerosis does play a significant role. Hypertension with associated microtrauma of the arterial intima is thought to play a role, as is hypercholesterolemia.[104,128] TIAs may result from the formation of microthrombi and their embolization. Large vessel thrombosis can also occur in extracranial vessels, such as the vertebral and carotid arteries, leading to devastating strokes.[117]

Pathophysiology. Atherosclerotic plaque formation is greatest at the branching points of major vessels and forms in areas of turbulent flow. Chronic hypertension is a common precursor, and damage to the intimal wall may be followed by lymphocyte infiltration. Foam cells then develop, and the first stage of atherosclerosis is formed. Calcification and narrowing with resultant turbulent flow follow. In this setting of turbulent flow, plaque ulceration can become a site for thrombus formation. If the thrombus forms and is degraded rapidly, a transient ischemic phenomenon can occur, which is the setting of a TIA. Classically, the symptoms of internal carotid disease include amaurosis fugax and monocular blindness. If the clot does not break up or lyse, a cerebral infarction can occur. The size and severity of the infarction depends on available collateral circulation and the size of the occluded vessel. In patients with extensive atherosclerotic disease, however, a limited amount of collateral circulation is available, and the sparing from collateral circulation may be limited.

Atherothrombotic Disease. The most common site for the development of atherosclerosis and the subsequent development of atherothrombosis that leads to TIAs and stroke in the anterior circulation is the origin of the carotid artery and in the posterior circulation is the top of the basilar artery. Other sites of atherosclerosis include the carotid siphon and the stems (bases) of the middle cerebral artery, anterior cerebral artery, and origin of the basilar artery.[51] The atheromatous plaques are sources of emboli that can cause distal symptoms in a TIA or stroke. These embolic events are similar events from other embolic sources. Table 1-2 lists common stroke syndromes, and Figs. 1-1 to 1-3 explain the anatomy of these strokes. Atherosclerotic disease is screened most readily by carotid Doppler ultrasonography and transcranial Doppler imaging. Magnetic

Table 1-2

Common Stroke Syndromes

ANATOMICAL DISTRIBUTION	STROKE SYNDROME
Common carotid artery	Often resembles middle cerebral artery (MCA) but can be asymptomatic if circle of Willis is competent
Internal carotid artery	Often resembles MCA but can be asymptomatic if circle of Willis is competent
Middle cerebral artery	

Main stem	Contralateral hemiplegia
	Contralateral hemianopia
	Contralateral hemianesthesia
	Head/eye turning toward the lesion
	Dysphagia
	Uninhibited neurogenic bladder
	Dominant hemisphere
	  Global aphasia
	  Apraxia
	Nondominant hemisphere
	  Aprosody and affective agnosia
	  Visuospatial deficit
	  Neglect syndrome

Table 1-2

Common Stroke Syndromes—cont'd

ANATOMICAL DISTRIBUTION	STROKE SYNDROME
Upper division	Contralateral hemiplegia; leg more spared Contralateral hemianopia Contralateral hemianesthesia Head/eye turning toward the lesion Dysphagia Uninhibited neurogenic bladder Dominant hemisphere Broca (motor) aphasia Apraxia Nondominant hemisphere Aprosody and affective agnosia Visuospatial deficit Neglect syndrome
Lower division	Contralateral hemianopia Dominant hemisphere Wernicke aphasia Nondominant hemisphere Affective agnosia
Anterior cerebral artery (ACA)	
Proximal (precommunal) segment (A1)	Can be asymptomatic if circle of Willis is competent, but if both ACAs arise from the same stem, then: Profound abulia (akinetic mutism) Bilateral pyramidal signs Paraplegia
Postcommunal segment (A2)	Contralateral hemiplegia; arm more spared Contralateral hemianesthesia Head/eye turning toward the lesion Grasp reflex, sucking reflex, gegenhalten Disconnection apraxia Abulia Gait apraxia Urinary incontinence
Anterior choroidal artery	Contralateral hemiplegia Hemianesthesia Homonymous hemianopsia
Posterior cerebral artery	
Proximal (precommunal) segment (P1)	Thalamic syndrome: Choreoathetosis Spontaneous pain and dysesthesias Sensory loss (all modalities) Intention tremor Mild hemiparesis Thalamoperforate syndrome: Crossed cerebellar ataxia Ipsilateral third nerve palsy Weber syndrome: Contralateral hemiplegia Ipsilateral third nerve palsy Contralateral hemiplegia Paralysis of vertical eye movement Contralateral action tremor

Continued

Table 1-2

Common Stroke Syndromes—cont'd

ANATOMICAL DISTRIBUTION	STROKE SYNDROME
Postcommunal segment (P2)	Homonymous hemianopsia Cortical blindness Visual agnosia Prosopagnosia Dyschromatopsia Alexia without agraphia Memory deficits Complex hallucinations
Vertebrobasilar syndromes	
Superior cerebellar artery	Ipsilateral cerebellar ataxia Nausea/vomiting Dysarthria Contralateral loss of pain and temperature sensation Partial deafness Horner syndrome Ipsilateral ataxic tremor
Anterior inferior cerebellar artery	Ipsilateral deafness Ipsilateral facial weakness Nausea/vomiting Vertigo Nystagmus Tinnitus Cerebellar ataxia Paresis of conjugate lateral gaze Contralateral loss of pain and temperature sensation
Medial basal midbrain (Weber syndrome)	Contralateral hemiplegia Ipsilateral third nerve palsy
Tegmentum of midbrain (Benedikt syndrome)	Ipsilateral third nerve palsy Contralateral loss of pain and temperature sensation Contralateral loss of joint position sensation Contralateral ataxia Contralateral chorea
Bilateral basal pons (locked-in syndrome)	Bilateral hemiplegia Bilateral cranial nerve palsy (upward gaze spared)
Lateral pons (Millard-Gubler syndrome)	Ipsilateral sixth nerve palsy Ipsilateral facial weakness Contralateral hemiplegia
Lateral medulla (Wallenberg syndrome)	Ipsilateral hemiataxia Ipsilateral loss of facial pain and sensation Contralateral loss of body pain and temperature sensation Nystagmus Ipsilateral Horner syndrome Dysphagia and dysphonia

resonance angiography (MRA) and carotid and cerebral angiography can further elucidate lesions, which can be treated surgically or medically.

Lacunar Syndrome. A lacunar stroke occurs in one of the perforating branches of the circle of Willis, the middle cerebral artery stem, or the vertebral or basilar arteries. The occlusion of these vessels results from the atherothrombotic or lipohyalinotic blockage of one of these arteries. The development of disease in these arteries correlates closely with the presence of chronic hypertension and diabetic microvascular disease.[107,128] These are small vessels, 100 to 300 μm in diameter, that branch off the main artery and penetrate into the deep gray or white matter of the cerebrum.[107] The resulting infarcts are from 2 mm to 3 cm in size and account for

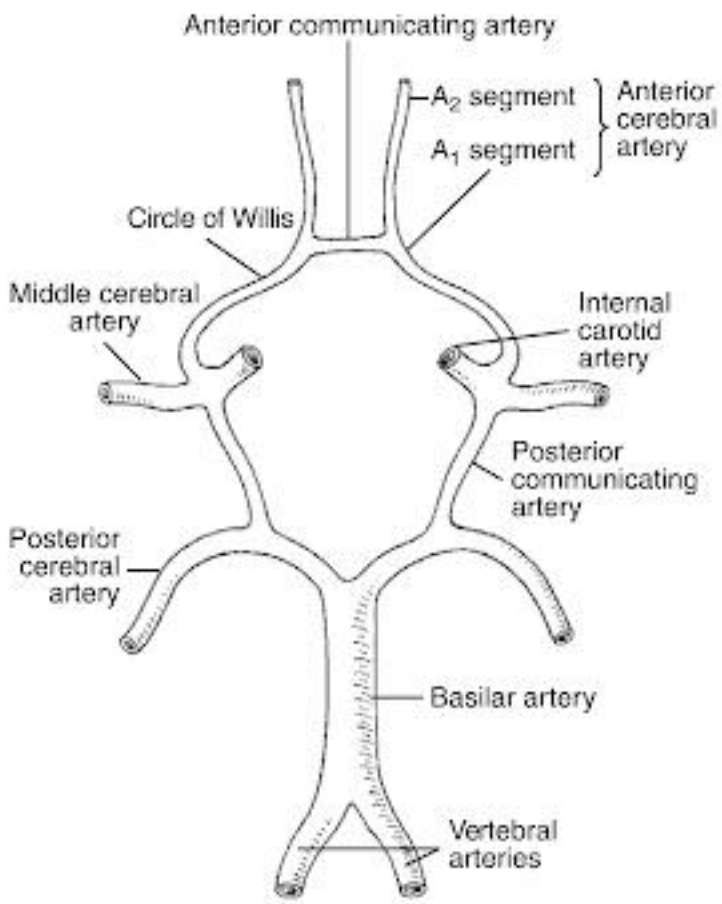

Figure 1-1 Circle of Willis and cerebral circulation.

roughly 20% of all strokes. These types of strokes usually evolve over a few hours and sometimes can be heralded by transient symptoms in lacunar TIAs. Lacunar strokes can cause recognizable syndromes (Table 1-3). The basic lacunar syndromes are (1) pure motor hemiparesis from an infarct in the posterior limb of the interior capsule or pons, (2) pure sensory stroke from an infarct in the ventrolateral thalamus, (3) ataxic hemiparesis from an infarct in the base of the pons or the genu of the internal capsule, and (4) pure motor hemiparesis with motor apraxia resulting from an infarct in the genu of the anterior limb of the internal capsule and the adjacent white matter in the corona radiata. Recovery from a lacunar stroke often can be dramatic, and in some individuals, near complete or complete resolution of deficits can occur in several weeks or months. In patients who have had multiple lacunar infarcts, a syndrome characterized by emotional instability, slow abulia (impairment in or loss of volition), and bilateral pyramidal signs known as pseudobulbar palsy will develop. This diagnosis is based on the symptoms and the use of computerized tomography (CT) or magnetic resonance imaging (MRI). MRI is especially useful in this situation for detecting small lesions in the deep brain structures or brainstem; the ability of CT to see lesions clearly in these areas is limited.[29]

Hemorrhagic Conversion. As a sequela of an embolic or ischemic infarction, a purely ischemic infarct may convert into a hemorrhagic lesion. Thrombi can migrate, lyse, and reperfuse into an ischemic area, leading to small hemorrhages (petechial hemorrhages) because the damaged capillaries and small blood vessels no longer maintain their integrity. These damaged areas then can coalesce (combine) and form a hemorrhage into ischemia.[83] These conversions are more common in large infarcts, such as an occluded middle cerebral artery, or in a large

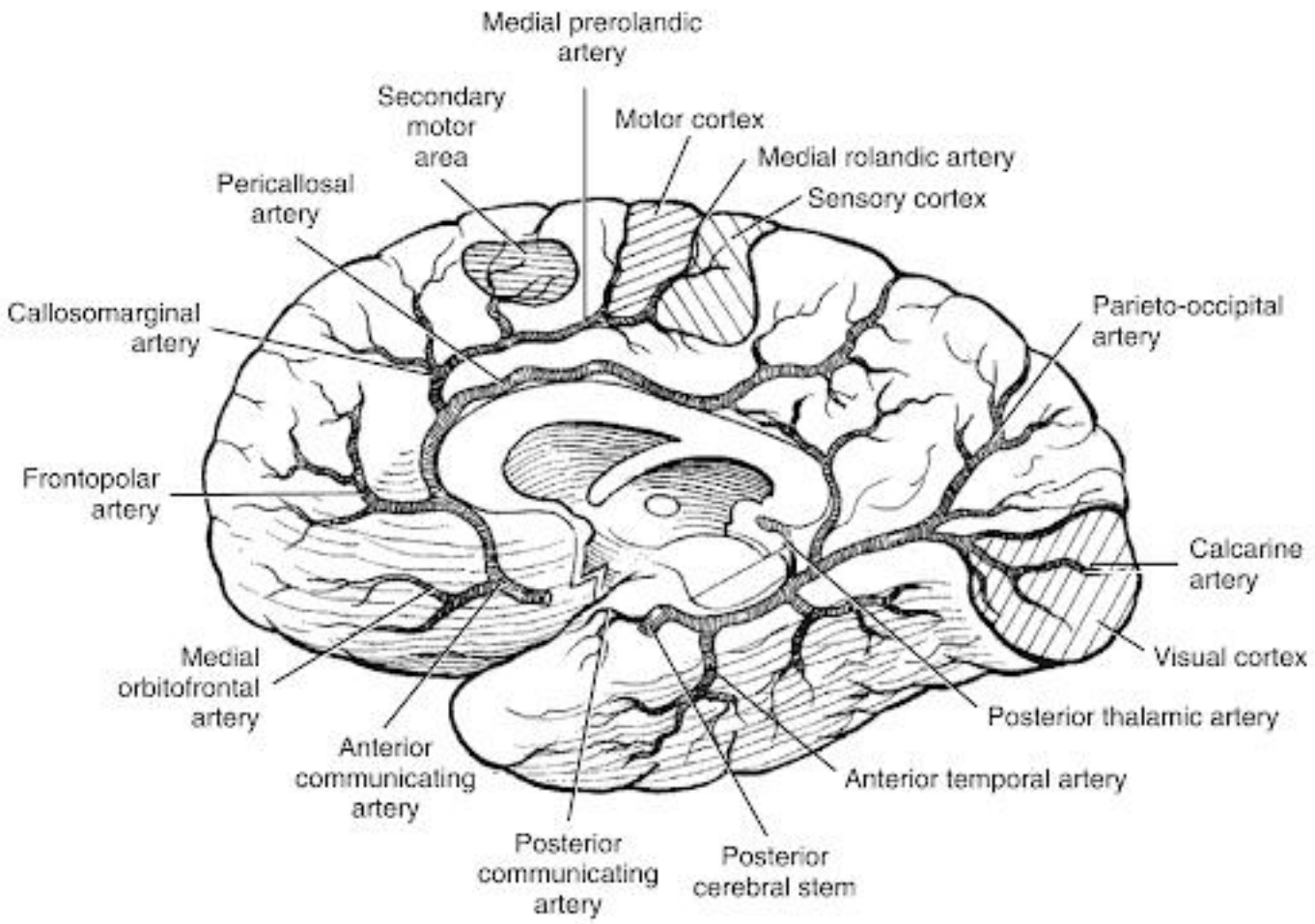

Figure 1-2 Medial view of brain with anterior and posterior cerebral artery circulation and areas of cortical function.

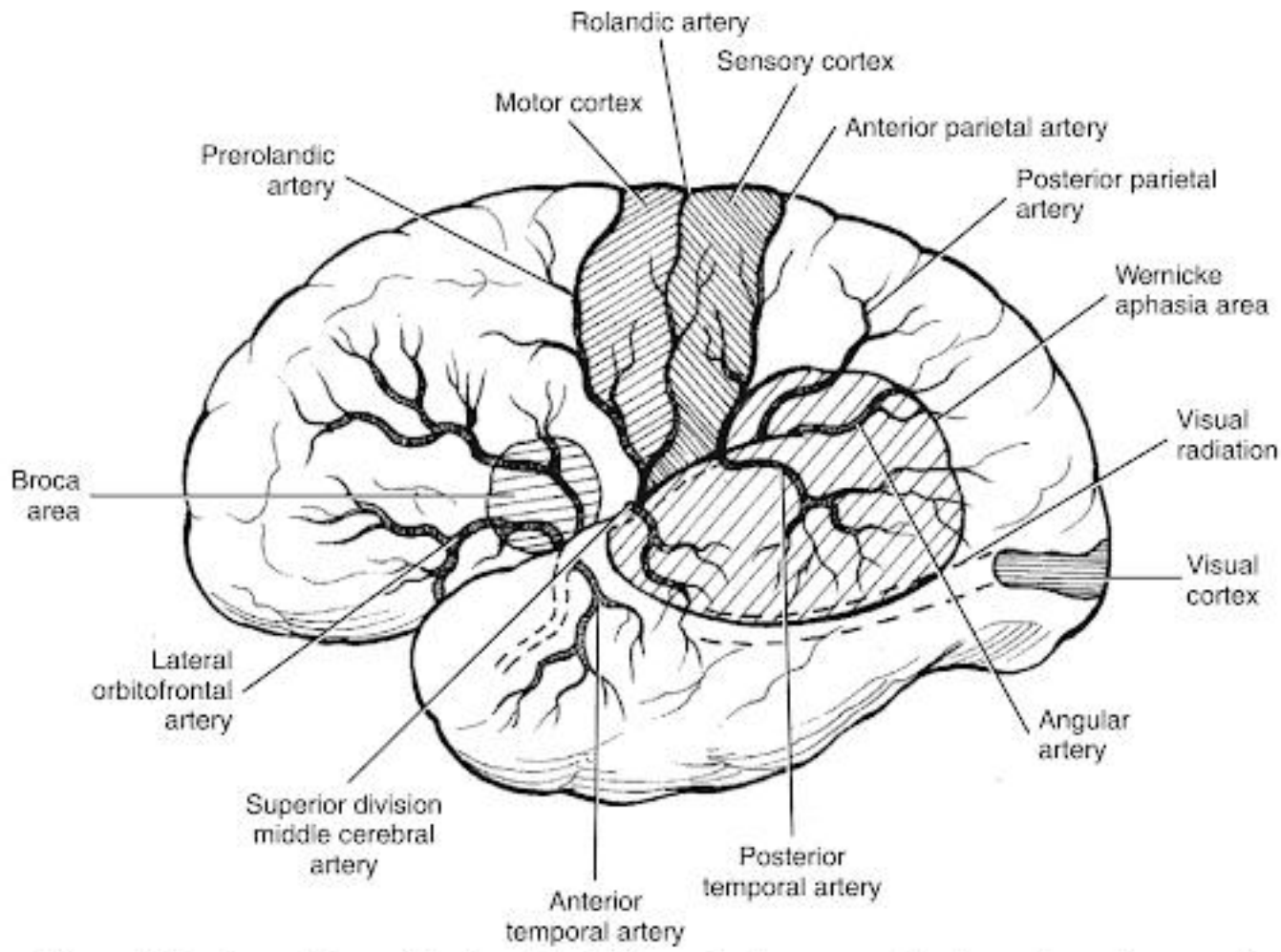

Figure 1-3 Lateral view of brain with middle cerebral artery and its branches and areas of cortical function.

Table 1-3

Lacunar Stroke Syndromes and Their Anatomical Sites

LACUNAR SYNDROME	ANATOMICAL SITES
Pure motor	Posterior limb of internal capsule Basis pontis Pyramids
Pure sensory	Ventrolateral thalamus Thalamocortical projections
Ataxic hemiparesis	Pons Genu of internal capsule Corona radiata Cerebellum
Motor hemiparesis with apraxia	Genu of the anterior limb of the internal capsule Corona radiata
Hemiballismus	Head of caudate Thalamus Subthalamic nucleus
Dysarthria/clumsy hand	Base of pons Genu of anterior limb of the internal capsule
Sensory/motor	Junction of the internal capsule and thalamus
Anarthric pseudobulbar	Bilateral internal capsule

infarction in the distribution of a lenticulostriate artery. In patients who have large infarcts with possibility of hemorrhage, anticoagulation therapy is not used because of the risk of hemorrhagic conversion. These types of hemorrhages have characteristics in common with hemorrhagic strokes.

Hemorrhagic Stroke

Hemorrhagic strokes have numerous causes. The four most common types are deep hypertensive intracerebral hemorrhages (ICHs), ruptured saccular aneurysms, bleeding from an arteriovenous malformation (AVM), and spontaneous lobar hemorrhages.[83]

Hypertensive Bleed. Hypertensive cerebral hemorrhages usually occur in four sites: the putamen and internal capsule, the pons, the thalamus, and the cerebellum. Usually these hemorrhages develop from small penetrating arteries in the deep brain that have had damage from hypertension. The pathological features of hypertension include lipohyalinosis (fat infiltration of pathologically degenerated tissue) and Charcot-Bouchard aneurysms.[50] The usual hypertensive ICH develops over the span of a few minutes but occasionally can take as long as 60 minutes. Unlike ischemic infarcts, hemorrhagic bleeds do not follow the anatomical distribution of blood vessels but dissect through tissue

planes spherically. This commonly leads to severe damage and complications, such as hydrocephalus and mass shift (movement of brain tissues to one side to accommodate the volume of the hemorrhage).[83,128] Within 48 hours of the hemorrhage, macrophages begin to phagocytize the hemorrhage at its outer margins. Patients with a cerebral hemorrhage often experience a rapid recovery within the first two to three months after the hemorrhage. ICHs usually occur while patients are awake and often while they are under emotional stress. Vomiting and headache are associated commonly with ICH and are unique features that differentiate ICHs from ischemic strokes. Table 1-4 outlines the four major hypertensive ICH syndromes.

Lobar Intracerebral Bleed. Lobar hemorrhages are ICHs that occur outside the basal ganglia and thalamus in the white matter of the cerebral cortex. These types of hemorrhages and hypertension are not correlated clearly; the most common underlying condition in patients with this type of ICH is the presence of AVMs.[83] Other associated conditions include bleeding diatheses, tumors (e.g., melanoma or glioma), aneurysms in the circle of Willis, and a large number of idiopathic cases.[49] Patients with lobar ICH initially have acute onset of symptoms, and most lobar ICHs are small enough to cause discrete clinical syndromes that may resemble focal ischemic events. Because lobar bleeds occur far from the thalamus and the brainstem, coma and stupor are much less common than they are in patients with hypertensive ICHs. Headaches are also common and can help differentiate lobar bleeds from ischemic strokes, which they can resemble so closely.[126] Detection of a hemorrhage on a CT scan or MRI is the best way to distinguish these two entities.

Saccular Aneurysm and Subarachnoid Bleed. A saccular aneurysm rupture is the most common cause of a subarachnoid hemorrhage (SAH).[150] Saccular aneurysms occur at the bifurcation (branching) points of the large arteries in the brain and are most commonly found in the anterior portion of the circle of Willis.[83] An estimated 0.5% to 1% of normal individuals harbor saccular aneurysms.[158] Despite the high number, bleeding from them is rare (6 to 16 per 100,000). Unlike other stroke syndromes, however, the incidence of SAH has not declined since 1970.[102] The rupture risk correlates best with the size of the aneurysm. Aneurysms smaller than 3 mm have little chance of hemorrhage, whereas aneurysms 10 mm or larger have the greatest chance of rupture.[95] SAH usually is characterized by acute, abrupt onset of a severe headache of atypical quality.[102] These headaches are often the most severe that patients have ever experienced.

Table 1-4

The Four Major Hypertension Intracerebral Hemorrhage Syndromes

TYPE	STRUCTURES INVOLVED	CLINICAL SYNDROME	COMMENTS
Putamenal	Internal capsule Basal ganglia	Contralateral hemiplegia Coma in large infarcts Eyes deviate away from lesion Can have stupor/coma with brainstem compression Decerebrate rigidity	Most common
Thalamic	Thalamus Internal capsule	Contralateral hemiplegia Prominent contralateral sensory deficit for all modalities Aphasia if dominant (left) thalamus involved Homonymous visual field defect Gaze palsies Horner syndrome Eyes deviate downward	
Pontine	Pons Brainstem Midbrain	Coma Quadriparesis Decerebrate rigidity Severe acute hypertension Death	Can lead to a locked-in syndrome
Cerebellar	Cerebellum	Nausea and vomiting Ataxia Vertigo/dizziness Occipital headache Gaze toward the lesion Occasional dysarthria and dysphagia	Nystagmus and limb ataxia are rare

A brief loss of consciousness, nausea and vomiting, focal neurological deficits, and a stiff neck at the onset of symptoms also may occur. The diagnosis is based on clinical suspicion, subarachnoid blood found on the CT scan, or blood found in the cerebrospinal fluid from a spinal tap. One determines the definitive location of the aneurysm by cerebral angiography.

The development of further delayed neurological deficits results from three major events: rerupture, hydrocephalus, and cerebral vasospasm. Rerupture occurs in 20% to 30% of cases within one month if treatment is not aggressive, and rebleeding has an associated mortality rate of up to 70%.[102] Hydrocephalus occurs in up to 20% of cases, and aggressive management often is required. Chronic hydrocephalus is also common and often requires permanent cerebrospinal fluid drainage (shunting). Vasospasm also is a common problem after SAHs, occurring in approximately 30% of cases.[102] The normal time course for vasospasm is an onset in three to five days, peak narrowing in five to 14 days, and resolution in two to four weeks. In half of cases, the vasospasm is severe enough to cause a cerebral infarction with resulting stroke or death. Even with modern management, 15% to 20% of patients who develop vasospasms still suffer strokes or die.[96] A permanent ischemic deficit develops in approximately 50% of patients with symptomatic vasospasms after SAHs.[69] Vasospasm therefore must be treated rapidly and as aggressively as possible to prevent permanent ischemic damage.

Arteriovenous Malformation. AVMs are found throughout the body and can occur in any part of the brain. They are usually congenital and consist of an abnormal tangle of blood vessels between the arterial and venous systems. They range from a few millimeters in size to large masses that can increase cardiac output because of the amount of their blood flow. The larger AVMs in the brain tend to be found in the posterior portions of the cerebral hemispheres.[50] AVMs occur more frequently in men, and if found in one family member, they have a tendency to be found in other members. AVMs are present from birth, but bleeding most often occurs in the second and third decades of life. Headaches and seizures are common symptoms, as is hemiplegia. Half of AVMs initially occur as ICHs. Although rebleeding in the first month is rare, rebleeding is common in larger lesions as more time passes. Contrast CT, MRA, and MRI are useful noninvasive tests, whereas cerebral angiography is the best test for delineating the nature of the lesion. The management of these lesions is accomplished best by a team approach, a combination of surgical treatment and interventional angiography for definitive management. Treatment of hydrocephalus and increased intracranial pressure is the same as treatment for SAH and ICH.

Posttraumatic Hemorrhagic Stroke. A traumatic brain injury commonly results in hemorrhagic damage to the brain in addition to ischemic and other injuries. The four major types of injury caused by traumatic brain injury include SAH and ICH, diffuse axonal injury, contusions, and anoxic injury from hypoperfusion (decreased flow in the vessels) and hypoxemia (decreased oxygen level). This combination of injuries leads to a constellation of findings that mixes the features of a number of individual ischemic and hemorrhagic injuries.

Other Causes of Stroke and Strokelike Syndromes

Arterial and Medical Disease. Numerous medical conditions can result in arterial system diseases and lead to thrombosis and thromboembolism. Some conditions may cause disease in the cerebral vasculature (Table 1-5).

Strokelike Syndromes. A number of conditions in addition to TIAs and cerebral infarctions can cause transient paralysis. These conditions generally resolve spontaneously with no long-term sequelae. The most common cause of transient hemiparesis is Todd paralysis, which develops postictally (after a seizure). Todd paralysis results from neurotransmitter depletion and neuronal fatigue in focal areas of the brain caused by the extremely high neuronal firing rate during a seizure.[37] Patients usually regain function within 24 hours. Another common cause of focal neurological deficits is migraine headaches. These headaches are actually thought to result from cerebral vasospasms, but an actual ischemic infarct rarely if ever occurs. The deficits resolve with the resolution of the migraine and are not permanent.

Cerebral Neoplasm. Obviously, cerebral neoplasms (whether primary or metastatic) can lead to focal neurological deficits that resemble a stroke. The treatment of the sequelae and the long-term management of the deficits are the same as they are in stroke patients. Treating the primary lesions is the focus of the acute care. Often the initial symptoms are seizures and ICHs.

STROKE DIAGNOSIS

The diagnosis of stroke and differentiation of stroke from strokelike syndromes is based on the clinical presentation and physical examination of the patient. The examiner needs to differentiate a true stroke from syndromes that can mimic a stroke, such as Todd paralysis, seizures, multiple sclerosis, tumors, and metabolic syndromes. Most often, the patient's symptoms in the emergency room include an acute onset of weakness or other neurological deficits. The patient history can help identify the risk factors for stroke and the nature of the lesion. The physical examination includes a general medical examination and a neurological examination. Only after a diagnosis of stroke

Table 1-5

Medical Conditions That Cause Arterial System Disease

CONDITION	FEATURES*	TREATMENT
Vasculitic/inflammatory		
Systemic lupus erythematosus	Most commonly associated vasculitis with stroke Vasculitic, thrombotic, and embolic events occur Greater than 50% recurrence rate Antiphospholipid antibody may play a role	Treat lupus Anticoagulation with warfarin
Binswanger disease	Rare condition Diffuse subcortical infarction Diffuse lipohyalinosis of small arteries	No clear treatment Anticoagulation
Scleroderma	Stroke in 6% of patients Antiphospholipid antibody may play a role	No clear treatment Anticoagulation
Periarteritis nodosa	Can cause a CNS vasculitis Can cause embolic stroke	Treat underlying condition
Temporal arteritis	Can cause a CNS vasculitis Can cause embolic stroke	Treat underlying condition
Wegener granulomatosis	Can cause a CNS necrotizing vasculitis Can cause thrombotic stroke	Treat underlying condition
Takayasu arteritis	Can cause embolic stroke	Treat underlying condition Anticoagulation
Isolated angiitis of the CNS	Rare primary CNS vasculitis Headache, multiinfarct dementia, lethargy	Treat underlying condition
Fibromuscular dysplasia	Mostly in young women Often asymptotic Can be associated with TIA and stroke	Anticoagulation Surgical dilation of the carotid arteries (if necessary)
Moyamoya disease	Vasooclusive disease of the large intracranial arteries Mainly in Asian population Cause of strokes in children and young adults	Role of anticoagulation controversial because of risk of hemorrhage Role of surgery controversial
Hypercoagulable state		
Antiphospholipid antibodies	Associated with recurrent thrombosis Embolic and thrombotic strokes occur	Anticoagulation with warfarin
Oral contraceptive agents	Relative risk increased 4 times over controls Thought to be caused by hypercoagulability	Stop oral contraceptives
Sickle cell disease	Microvascular occlusion caused by sickled cells Seen in 5% to 17% of patients with sickle cell disease	No good treatments exist
Polycythemia	Vascular occlusion caused by increased viscosity and hypercoagulability	Treat underlying cause (if known)
Inherited thrombotic tendencies	Include many familial clotting abnormalities	Treat abnormality (if possible) Anticoagulation

*CNS, Central nervous system; TIA, transient ischemic attack.

Continued

Table 1-5

Medical Conditions That Cause Arterial System Disease—cont'd

CONDITION	FEATURES*	TREATMENT
Others		
Venous thrombosis	Seen in meningitis, hypercoagulable states, and after trauma Increased intracranial pressure, headache, seizures Focal neurological signs, especially in legs more than arms Diagnosed with angiography	Anticoagulation May need surgical decompression
Arterial dissection	More common in children and young adults May present with TIA Often preceded by trauma, mild to severe	Surgical treatment as needed Anticoagulation after acute state

based on the clinical history and examination can a further diagnostic evaluation be performed. Modern technology has improved the tools available for the accurate diagnosis of stroke and includes an armamentarium of imaging studies to identify the exact nature of the lesions that may cause neurological deficits. Each imaging study available has benefits and limitations that are useful to know for assessing a patient who has had a stroke. The stroke evaluation also should include an evaluation for the cause of the stroke.

Cerebrovascular Imaging

The main tool used in stroke diagnostic evaluations is cerebral imaging, which historically included pneumoencephalography and other studies no longer performed. CT is probably the most common and the best known of the studies. MRI is now more common and has some advantages over CT, but availability and cost are still prohibitive in some areas. Positron emission tomography scans and single-photon emission CT scans are just being introduced and may have a role in stroke diagnosis.

Computerized Axial Tomography

CT is a readily available and useful technique that has become the standard for the evaluation of a patient experiencing an acute onset of stroke. The most important functions of CT scanning in an acute patient are ruling out other conditions (e.g., tumor or abscess) and helping identify whether evidence exists of hemorrhage into the infarction. In the acute phase of stroke, most CT scans are actually negative with no clear evidence of abnormalities. A negative immediate CT scan with an acute neurological deficit determined by physical examination actually can verify the impression of stroke because it rules out tumors, hemorrhages, and other brain lesions. The few changes seen in an acute stroke by CT are subtle and can include loss of distinction between gray and white matter and sulcal effacement. Acute bleeding, however, is visible on CT scanning and can be present in as many as 39% to 43% of patients.[29] By definition, hemorrhagic infarction occurs within 24 hours of infarction, and hemorrhagic transformation occurs after 24 hours of infarction. The cause of the hemorrhagic change is thought to result from reperfusion into areas of damaged capillary endothelium and is common in large infarcts with extensive injury. Hemorrhagic transformation occurs equally in all distributions of infarcts[113] and is not associated necessarily with hypertension or with older age.[27] Hemorrhagic transformation can be detected in the acute phase by CT; in this case, one should not use anticoagulants because they may increase in the severity of the cerebral hemorrhage.

In the subacute phase, the findings from CT clearly show the development of cerebral edema within three days, which then fades over the next two to three weeks; then a decrease in the signal intensity occurs over the infarction. This decrease corresponds with the change from the positive mass effect (swelling) of the acute phase to the negative mass effect (shrinkage) of the chronic phase. The infarct actually may be difficult to see again in two to three weeks but is clearly visible with the addition of contrast material. Long-term parenchymal enhancement develops, which is consistent with the scar formation that becomes the permanent CT finding. The loss of tissue volume (negative mass effect) and the permanent scar tissue are the characteristic features of a chronic infarct (Figs. 1-4 to 1-8).

Magnetic Resonance Imaging

MRI is now as commonly used in acute patients as CT, because cost and availability have improved. The MRI also has the advantage of allowing earlier detection of

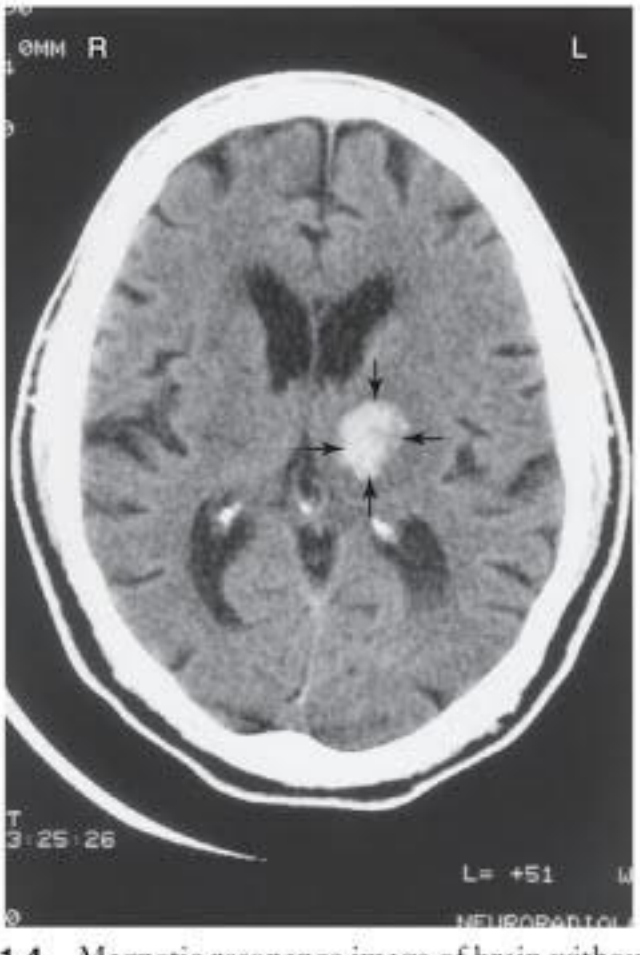

Figure 1-4 Magnetic resonance image of brain without gadolinium demonstrates an acute large left basal ganglia infarct. An acute infarct on the image appears white and is indicated by arrows.

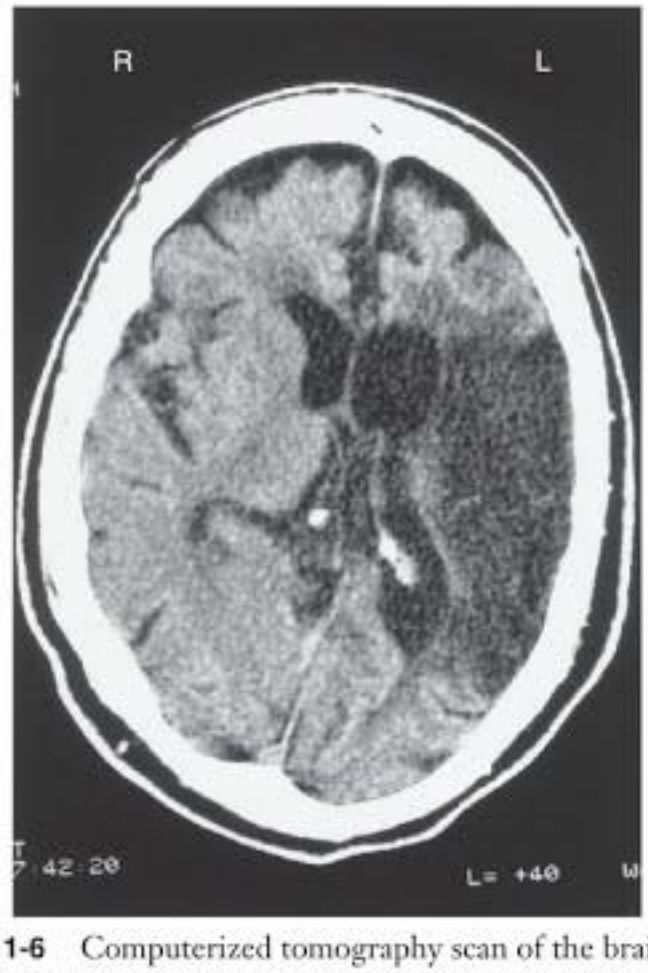

Figure 1-6 Computerized tomography scan of the brain without contrast demonstrates a large, previous, left middle cerebral artery distribution infarction. Loss of mass of brain tissue has occurred with dilated ventricles. Bleeding or acute infarction is not evident.

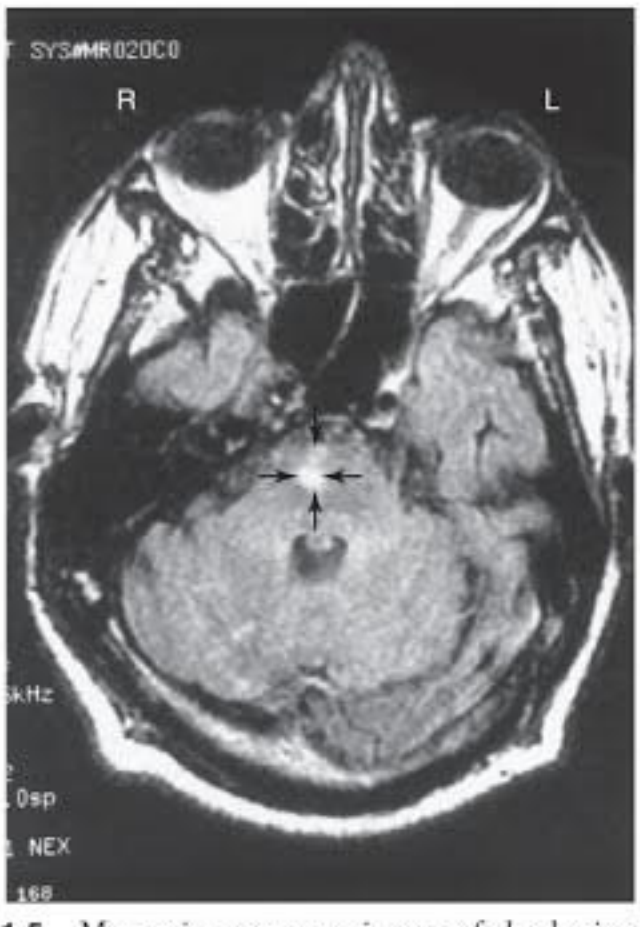

Figure 1-5 Magnetic resonance image of the brainstem and cerebellum without gadolinium demonstrates an acute right pontine infarct. The infarct appears white and is indicated by arrows.

infarcts and, as more acute interventions have become common, allows for better evaluation of the course of acute treatment. Newer techniques such as diffusion-weighted averaging have been used to help in the identification of early infarcts.[58, 141] MRI also can rule out other conditions and can screen for acute bleeding. In addition, MRI can be more sensitive for detecting cerebral infarctions in acute patients. Magnetic resonance images are created by mapping out the relaxation of protons after the imposition of a strong magnetic field. These images are then taken in two ways: T1- and T2-weighted images. In T1 images, fat and tissues with similar proton densities are enhanced (bright). In T2 images, water and tissues rich in water are enhanced. As in CT scans, sulcal effacement can be seen, but hyperintensity is also evident in affected areas on the T1-weighted images. Magnetic resonance images can show meningeal enhancement over the dura, which occurs in 35% of acute stroke cases.[44] MRI also can detect hemorrhage in much the same way as CT does.

The subacute changes of edema and mass effect can be seen with MRI, and use of contrast may be necessary to elucidate an infarct in the two- to three-week window. MRI has an advantage in determining a hemorrhage in a late stage because it can detect the degradation products

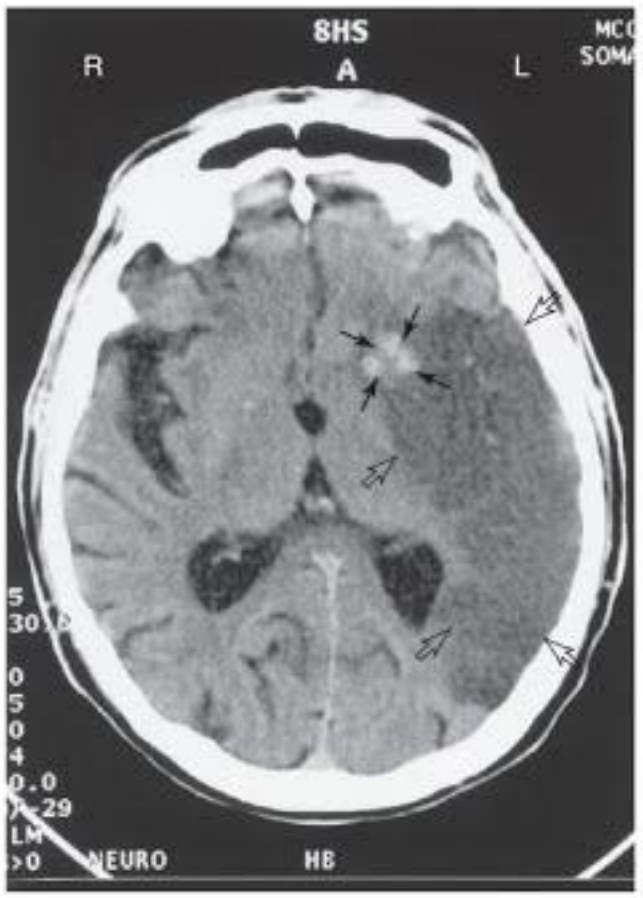

Figure 1-7 Computed tomography scan of the brain without contrast demonstrates a large subacute left middle cerebral artery distribution infarction, indicated by the hollow arrows. No loss of brain tissue mass has occurred compared with Fig. 1–6. Evidence of acute bleeding is in the basal ganglia on the left, which is white on the scan and is indicated with solid arrows.

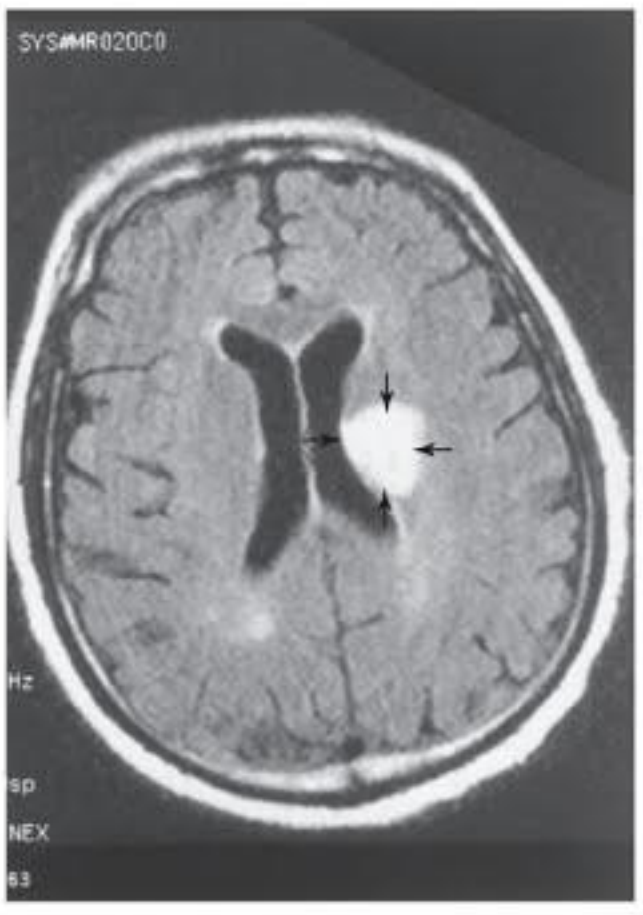

Figure 1-8 Computed tomography scan of the brain without contrast demonstrates a large, acute left thalamic hemorrhage. The acute bleeding in the thalamus on the left is white on the scan and is indicated with arrows.

of hemoglobin (hemosiderin deposits) and show hemorrhage areas well after CT can no longer detect a bleed. The changes on MRI in a chronic infarction are similar to those on a CT scan.

Positron Emission Tomography and Single-Photon Emission Computerized Tomography Scanning

Positron emission tomography and single-photon emission CT scanning are new techniques available only at selected centers. They have no clear role in the acute-stage evaluation of stroke.[2] In the subacute and chronic stages of stroke, these techniques help to distinguish between infarcted and noninfarcted tissue and can help delineate areas of dysfunctional but potentially salvageable brain tissue. These studies can also be used to try to assess brain function in the chronic setting. However, because of cost, limited availability, and an unclear definition of their use, they are essentially only research tools and do not have a role in the routine management of stroke patients.

WORKUP FOR CAUSE OF STROKE

The workup for the diagnosis of stroke is aimed at answering three main questions:

1. Is the stroke thrombotic or embolic?
2. Does an underlying cause require treatment?
3. Do any risk factors require modification?

Transcranial and Carotid Doppler

Transcranial and carotid Doppler studies allow for noninvasive visualization of the cerebral vessels. The advantages are that they provide useful therapeutic information on the state of the cerebral vessels and the blood flow to the brain. Approximately one third of patients who have had ischemic strokes that are cardiac in origin have significant cerebrovascular disease.[25] Patients with symptoms or evidence of posterior circulation disease are tested best with a transcranial Doppler study, including examination of the vertebrobasilar system. The cost is low compared with other tests such as MRA or cerebral angiography, which has significant associated morbidity and mortality. The evidence of carotid disease can help shape the patient's treatment plan and can encourage pursuit of definitive treatments such as carotid endarterectomy.

Magnetic Resonance Angiography

MRA is used to evaluate patients with stroke symptoms to detect any vascular abnormalities that may have caused the stroke or to look for alterations of cerebral blood flow that may have resulted from an embolic or thrombotic event. This is a very common noninvasive technique and is often done at the time of the MRI scan to assess the extent of cerebral injury; MRA is able to image vessels similarly to classical angiography.[160] The newer techniques of MRA have sensitivity for detection of 86% to

90%[111] for detection of severe stenosis, and the earlier issues of relatively low specificity of 64%[13,79] (due to over-detection by the earlier techniques) is now in the range of 89% to 96% for studies done with contrast enhanced MRA.[77] Despite these advantages, the spatial resolution is still less than traditional angiography, which may be an issue in cases where surgical management is planned. However, with constantly improving techniques and increased field strengths and parallel imaging, high resolution MRA may soon equal the resolution seen in CT angiography.[65]

Electrocardiography

Electrocardiography is used to evaluate patients with stroke symptoms to detect dysrhythmias (which may be a source of embolic material) or myocardial infarction or other acute cardiac events that may be related to an acute stroke.

Echocardiography

In patients with a history of cardiac disease and stroke, echocardiography usually is warranted. The types of cardiac disease that usually cause emboli and should be investigated with an echocardiograph include congestive heart failure, valvular heart disease, dysrhythmias, and a recent myocardial infarction. In some individuals, a patent foramen ovale (the fetal opening between the right and left sides of the heart) persists into adulthood and can be the source of a paradoxical embolus from the venous circulation that crosses from the right atrium into the left atrium. A transesophageal echocardiogram can then be useful in combination with a bubble study to assess for a right-to-left shunt. This specialized study also can visualize parts of the heart better in the search for emboli in areas such as the left atrial appendage when the standard transthoracic echocardiogram is inconclusive.

Blood Work

The standard acute evaluation of the stroke patient includes a complete screening set of blood analyses, including hematological studies, serum electrolyte levels (ionizing substances such as sodium and potassium), and renal (e.g., serum creatinine) and hepatic chemical analyses (liver function tests). The typical hematological evaluation has a complete blood count, platelet count, prothrombin time, and partial thromboplastin time. These studies help to rule out other causes of strokelike symptoms, to diagnose complications, and to allow for a baseline analysis before the initiation of therapies such as anticoagulation. The blood chemistry analyses allow metabolic abnormalities to be ruled out, as do the renal and hepatic chemistry analyses. The latter part of the stroke evaluation can involve numerous specialized tests chosen according to the clinical symptoms and development of the differential diagnosis as the evaluation progresses (Fig. 1-9). Table 1-6 provides a sample of some of these studies and their associated conditions.

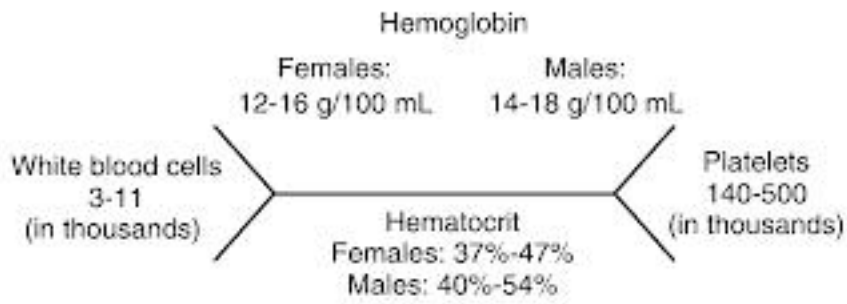

Figure 1-9 Complete blood count.

Table 1-6

Medical Studies Used to Clarify Diagnoses in Stroke Evaluation

SPECIALIZED STUDIES TO EVALUATE STROKE	ASSOCIATED CONDITIONS
Proteins S and C	Hypercoagulable state
Anticardiolipin antibodies (lupus anticoagulant)	Lupus erythematosus, hypercoagulable state
Erythrocyte sedimentation rate	Collagen vascular disease
Rheumatoid factor	Lupus erythematosus, collagen vascular disease
Antinuclear antibody	Lupus erythematosus, collagen vascular disease
Hemoglobin	Polycythemia
Sickle cell preparation	Sickle cell disease
Hemoglobin electrophoresis	Sickle cell disease
Blood and tissue cultures	Infectious emboli

MEDICAL STROKE MANAGEMENT

Principal Goals

As in the medical management of all patients, the care of stroke management requires good general patient care. All phases include caring for the conditions the patient may have and preventing medical complications and anticipating needs that will arise as the patient progresses through the acute phase into the convalescent, rehabilitative, and long-term maintenance phases after stroke. Care for acute patients is provided best in a specialized stroke unit that commonly deals with the issues and concerns unique to these patients.[2,102] Outcome studies have demonstrated the benefit of these units in the care of stroke patients.[91] Medical rehabilitation units also have been shown to be beneficial in the improvements of outcomes in the subacute and convalescent phases.

Acute Stroke Management

In management of acute stroke patients, basic medical needs have to be addressed and to include essentials such as airway protection, maintenance of adequate circulation, and the treatment of fractures or other injuries and conditions present at the time of admission. The neurological management of the acute stroke problems focus on identifying the cause of the stroke, preventing progression of the lesion, and treating acute neurological complications. Some specific approaches apply to treatment of each of the different types of stroke.

General Principles

The general principles of acute stroke management include attempting to stop progression of the lesion to limit deficits, reducing cerebral edema, decreasing the risk of hydrocephalus, treating seizures, and preventing complications such as DVT or aspiration that may lead to severe illness. (See the previous sections for a discussion of the studies used in acute patients to diagnose stroke.) Once the type of lesion has been defined, specific treatment can be instituted. Although numerous studies have been performed and are underway on the reduction of stroke mortality or disability,[136] no routine medical or surgical treatment has been shown to be effective. Currently, more aggressive methods such as angioplasty and thrombolysis are being studied, and the results of these trials are expected to lead to treatments that actually will improve the outcomes for individuals who have had strokes.

The basic principles in the approach to the treatment of acute stroke include an attempt to achieve improvement in cerebral perfusion by reestablishing blood flow, decreasing neuronal damage at the site of ischemia by modifying the pathophysiological process, and decreasing edema in the area of damaged tissue (which often can lead to secondary damage to nonischemic brain tissue). Many pharmacological and surgical treatments have been targeted toward at least one of these areas. Depending on the stroke mechanism, the agents and techniques of choice are used.

Ischemic Stroke

In patients who have had ischemic strokes, the restoration of blood flow and the control of neuronal damage at the area of ischemia are of the highest priority. In large strokes, edema can play a significant role, and mass shift can even lead to hydrocephalus. The pharmacological therapies are divided broadly into antithrombotic, thrombolytic, neuroprotective, and antiedema therapies. The surgical therapies include endarterectomy, extracranial-intracranial bypass, and balloon angioplasty.

Pharmacological Therapies

Antithrombotic Therapy (Antiplatelet and Anticoagulation). The principal rationale behind the use of antiplatelet and anticoagulation agents is that rapid recanalization and reperfusion of occluded vessels reduces the infarction area. The theoretical benefit also exists of preventing clot propagation and recurring vascular thrombosis. The risks associated with the use of these treatments includes hemorrhagic conversion, hemorrhage, and increased cerebral edema, all of which are associated with worse outcomes.[90] Current research has not established a clear advantage to the use of aspirin or heparin in acute stroke patients, but these agents still are commonly used in the hope that they may decrease injury from acute stroke. Aspirin, an irreversible antiplatelet agent, is administered when symptoms appear. Heparin is administered intravenously in a continuous infusion.[71] Both of these agents are started only after determination by CT or MRI that no hemorrhage is associated with the stroke. Ticlopidine, another antiplatelet agent, has been even less studied, and its role, if any, in acute stroke treatment is unclear. A recent metaanalysis of the trials of heparin and oral anticoagulation therapy in acute stroke treatment showed a marginal benefit from treatments with anticoagulation compared with no treatment at all.[135] Currently, numerous large, multicentric studies in the United States and Europe are examining the best approach to the antithrombotic treatment of stroke that should provide better guidance as their results become known in the next few years.

Thrombolytic Therapy. Thrombolytic therapy is attractive as a therapy for acute stroke, because it opens up occluded cerebral vessels and immediately restores blood flow to ischemic areas. However, a problem in using these agents in stroke treatment is that the treatment must start in six hours from onset of symptoms to be therapeutic. Most patients are symptomatic at a much later stage, and even if they have symptoms early enough, a rapid workup to rule out a cerebral bleed must be performed before initiation of therapy. The successful use of these agents—primarily urokinase, streptokinase, and tissue plasminogen activator—in the treatment of myocardial ischemia has aroused interest in similar use of these agents for acute stroke treatment. The mechanism of action of these agents is to cause fibrin breakdown in the clots that have been formed and thus to lead to lysis of the occlusions in the blood vessels. Reviews of thrombolytic therapy for stroke treatment have shown some reduction in mortality, but no definitive answer is available to date concerning efficacy.[163] Currently, streptokinase is out of favor because of increased mortality and morbidity from intracranial hemorrhage,[123,156] but tissue plasminogen activator, a more specific thrombolytic agent, has been able to achieve favorable results. The National Institute of Neurological Disorders and Stroke trial was the cornerstone trial in approval of treatment of acute ischemic stroke with thrombolytics.[3,6,103,157] The trial was a double-blind, placebo-controlled trial that revealed an improvement in early outcomes in 24 hours of treatment and demonstrated an increase in symptom-free survival

from 38% (placebo) to 50% (treatment) at three months. The strict use of a three-hour window from the onset of symptoms and the rigid blood pressure guidelines of the National Institute of Neurological Disorders and Stroke trial are probably contributors to the excellent outcomes; the exact treatment protocols are still being defined. On reexamination at one year, the treated patients continued to show a benefit, and this has encouraged the use of this agent in selected groups.[87] Other thrombolytic agents such as alteplase also have shown benefit and are being used routinely. The results are at the same level of effectiveness as tissue plasminogen activator.[5] Unfortunately, the three-hour window of efficacy limits the number of individuals who can receive benefit, and studies to expand the window of intervention to have hours or more have not shown clear benefits.[30,64] In the patient with stroke beyond three hours, the currently recommended interventions are mostly limited to the use of anticoagulants and antiplatelet agents to prevent further events.[103] Further active investigation continues to search for effective treatments in this large group of individuals with late presentation of stroke.

Other Treatments for Altering Cerebral Perfusion. A number of different treatments aimed at lowering blood viscosity or cerebral perfusion have been used, including hemodilution with agents such as dextran, albumin, and hetastarch. None of the 12 studies reviewed by Asplund demonstrated any clear benefit.[9] Similarly, studies of prostacyclins and several different types of cerebral vasodilators have also shown no clear evidence of increased survival rates or improvement in outcomes after treatment.[90] Research continues to be active in these areas, but so far none of these alternative treatments for increasing cerebral perfusion has yielded a favorable outcome.

Neuroprotective Agents. Neuroprotective agents are medications that can alter the course of metabolic events after the onset of ischemia and therefore have the potential to reduce stroke damage. No agent has shown clear benefits among this group of treatments. These agents include calcium channel blockers, naloxone, gangliosides, glutamate antagonists, and free-radical scavengers. Each of these agents has had promise in the theoretical or laboratory realm, but none has proved to be clinically efficacious.

The use of naloxone, a narcotic antagonist, is based on the in vitro observation that naloxone has neuroprotective effects. Unfortunately, the clinical trials to date have not demonstrated any benefit.[33] The therapeutic rationale of using calcium channel blockers is that they prevent injury to ischemic neurons by preventing calcium influx, which decreases metabolic activity in the neuron.[90] Initial hope was that the treatment results for SAH, in which nimodipine decreases secondary ischemia, would be similar for stroke. Unfortunately, the results of several studies have not shown any clear benefits from treatment with these agents,[108] and none of them currently are used routinely for stroke treatment.

In animal experiments, glutamate antagonists decrease the size of infarction area in stroke.[90] However, the few studies done in human beings have been inconclusive and have shown serious neuropsychiatric side effects.[33]

Gangliosides may reduce ischemic damage by counteracting toxic amino acids in ischemic tissue. Despite the many studies that have been performed, no clearly demonstrated benefits have resulted from use of these agents.[33]

The free-radical scavengers include 21-amino steroids (lazaroids), ascorbic acid (vitamin C), and tocopherol (vitamin E). They have not been well-evaluated, and some studies to establish their clinical use are being undertaken.[90] However, vitamin E has been demonstrated clinically to reduce the risk of heart disease, so secondarily its use may decrease the risk of stroke.

Agents for Cerebral Edema. Agents that reduce cerebral edema include corticosteroids, mannitol, glycerol, vinca alkaloids, and piracetam. All the studies done on persons receiving steroids[122] after an acute stroke demonstrated no clear benefits, and steroid use creates a risk of diabetes and DVT.[62] Use of the other agents also has no clear benefit in the treatment of acute stroke and are also not routinely used.

Cooling Therapy. An exciting new development in the treatment of acute stroke has been the initiation of cooling therapy on presentation with the induction of a medical coma to limit the extent of brain injury after stroke. In most patients who present with stroke, there is a natural tendency for the body temperature to be elevated between 4% and 25%, which is associated with increased injury and poorer outcomes.[18,35] Studies have shown that injury could be slowed with supercooling, and the technique has been used in surgery to help limit injury and to prolong safe surgical time in both neurosurgical and cardiothoracic procedures.[28,131,139] The pooled analysis of existing studies does not yet provide convincing evidence that death or long-term disability are significantly changed from the application of mechanical or pharmacological cooling, but the therapy is just starting to be used on a larger scale, and new research findings published in the next several years may show a benefit to routine cooling of acute stroke victims.

Surgical Therapies

Endarterectomy. A carotid endarterectomy is the surgical opening of the carotid arteries to remove plaque. This therapy has been shown to be useful in preventing recurrent strokes or development of stroke in individuals with TIAs, but it has not been used to treat acute stroke. In theory, the opening of the carotids could subject ischemic areas and their blood vessels to excessive pressure from

restored blood flow and lead to hemorrhage.[40] Concerns about using major anesthesia in a patient with a new stroke makes this surgery too risky to treat acute stroke.

Extracranial-Intracranial Bypass. Despite the initial attraction of bringing extracranial blood flow into the intracranial vessels through the use of bypass procedures, the large trial done in the 1980s demonstrated no improvement in patient outcomes, and the procedure has been largely abandoned.[47]

Balloon Angioplasty. Despite its efficacy in opening blocked coronary arteries in patients with heart disease and its successful treatment of acute myocardial infarction, the use of balloon angioplasty in acute stroke has not been studied. Clinical centers are actively investigating its possible uses.

Hemorrhagic Stroke

In patients who have had a hemorrhagic stroke, the size and location of the lesion determines the overall prognosis; supratentorial lesions greater than 5 cm have a poor prognosis, and brainstem lesions of 3 cm are usually fatal.[49] In these cases, the control of edema is important, and the techniques previously described can be used. In patients with SAH, the treatment regimen is usually more aggressive and focuses on several issues, which include the control of intracranial pressure, prevention of rebleeding, maintenance of cerebral perfusion, and control of vasospasm.

Prevention of Rebleeding. Before 1980, six weeks of bed rest were prescribed routinely for the care of patients with acute SAH to prevent rebleeding. In 1981 a study demonstrated that bed rest was inferior to surgical treatment, lowering of blood pressure, and carotid ligation.[158] Antihypertensive medications for the prevention of rebleeding are still controversial, and no consensus exists as to their use. Carotid ligation used to be popular, but more recent reevaluations of the benefits of the technique have not been as conclusive, and because of its surgical risks, direct repair of the aneurysm is a better choice. Antifibrinolytic agents have been studied and have been beneficial for low-risk patients in whom surgery must be delayed, but they seem to increase the risk of ischemic events. The placement of intraluminal coils, balloons, and polymers has shown some benefit in the short-term prevention of rebleeding, but the long-term efficacy is still unclear, and the techniques remain experimental.[102] Because the risk of rebleeding is also very high in post-SAH seizures, even though the incidence of seizure is low, the recommendation is that patients receive antiseizure medications for prophylaxis.

Control of Vasospasm. The treatment of vasospasm is important for the reasons previously outlined. The current treatments include the use of orally administered nimodipine, a calcium channel blocker shown to improve outcomes of patients who have had an SAH with vasospasm. The results of using other calcium channel antagonists are unclear. The use of hypertension/hypervolemia/hemodilution has been recommended by some studies. Creating more volume than normal results in hypertension. The stretch caused by the volume stimulates the smooth muscle pressure receptors that line the vessels. These receptors inhibit muscle action by a protective response, and the blood vessel dilates to accommodate the increased volume. Hypertension/hypervolemia/hemodilution is most effective in preventing vasospasm after surgically clipping the aneurysm. Significant cardiac and hemodynamic risks are associated with this therapy, so intensive care unit (ICU) monitoring is required.[102]

PREVENTION OF STROKE RECURRENCE

Ischemic Stroke

In general, the strategies to prevent recurrence of ischemic stroke can be divided into two areas: risk factor modification (which also applies to primary prevention) and secondary prevention to treat the underlying cause of stroke in individuals with a history of stroke. Following is a discussion of the secondary interventions that can be used to prevent recurrence of stroke.

Hypertension. Although the treatment of hypertension is an important primary preventive measure in the management of stroke, whether blood pressure reduction after stroke is beneficial has not been proved definitively. The transient rise in blood pressure after stroke usually settles without intervention.[164] Because of the uncertainty about whether overaggressive treatment of acute elevated blood pressure is harmful, definitive antihypertensive therapy probably should be delayed for two weeks.[90] At that time, one should follow the usual recommendations regarding adequate control of hypertension because some evidence indicates that it is beneficial. This seems especially appropriate in patients who have had a lacunar stroke because the development of multiple lacunae is related to uncontrolled blood pressure.

Antiplatelet Medications. In patients who have had a TIA or stroke, long-term use of aspirin has been shown to decrease the incidence of death, myocardial infarction, and recurrent events by up to 23%.[7] The doses of aspirin in numerous studies have ranged from 30 mg to 600 mg; all doses resulted in a 14% to 18% reduction in recurrent cerebral events, but gastrointestinal complications increased with the higher doses.[1,48,153] In general, a standard dosage of one regular adult aspirin (325 mg a day) is the usual treatment for recurrent ischemic stroke. Studies are underway that compare the efficacy of warfarin versus

aspirin in treating ischemic stroke; the results of these studies are not yet available. Ticlopidine is another antiplatelet medication effective in reducing the incidence of recurrent stroke.[81] Ticlopidine is most efficacious in women, patients who are not helped by aspirin therapy, and patients with vertebrobasilar symptoms, hypertension, diabetes, and no severe carotid disease.[62]

Anticoagulation. The incidence of recurrent stroke and TIA in patients with atrial fibrillation is approximately 7% per year. For patients who have atrial fibrillation with cardiac sources of emboli, warfarin is the clear treatment of choice; this is true for primary and secondary prevention. Although aspirin has some preventive effects, it is not as efficacious. In the presence of structural cardiac disease or atrial fibrillation, aspirin should be used only to treat patients in whom warfarin anticoagulation is contraindicated.[90]

The odds ratio for recurrence is approximately 0.36 in those treated with warfarin versus control and 0.84 for those treated with aspirin versus control.[45] However, problems exist with warfarin anticoagulation in the elderly. Cognitive and compliance difficulties can lead to an increase in complications. Unclear issues in anticoagulation use include when to start anticoagulants after stroke, the safety of anticoagulants in clinical practice, and the optimum anticoagulant blood level. Several studies are currently examining these questions.

Treatment of Dysrhythmias or Underlying Disease. Obviously, primary and secondary prevention should treat the underlying cause of the ischemic stroke. Prevention can include cardioversion to normal sinus rhythm and treatment with antidysrhythmic medications, and treatment of underlying medical conditions if they can be found. Unfortunately, only a small proportion of patients who have had TIAs and strokes can benefit from these specific treatments.

Carotid Endarterectomy. The surgical treatment of carotid artery stenosis has been shown to be beneficial in recent studies of stroke recurrence in patients with severely (greater than 70%) stenosed carotid arteries.[12,46] The data on the intermediate group of patients (stenosis from 30% to 70%) are being collected. For patients with high-grade stenosis, carotid endarterectomy reduces the range of stroke risk from 22% to 26% down to 8% to 12%.

Hemorrhagic Stroke

The mainstay of ICH prevention is controlling systolic and diastolic hypertension. No clear benefit exists for one group of treatment agents versus another as long as adequate hypertension control is maintained. In patients in whom the ICH follows vasculitis or the use of anticoagulants, the treatment for preventing recurrence includes treating the vasculitis or terminating anticoagulant use.[128]

The secondary prevention of recurrent stroke and SAH of AVMs and/or aneurysms includes surgical management of the lesions (the treatment of choice). Clipping or microsurgical dissection of the lesions is performed whenever possible and as soon as the patient is able safely to undergo the procedure.[102,149] In surgically unresectable lesions, alternatives include sclerotherapy, coating, trapping, and proximal arterial occlusion.[102]

PREVENTION OF COMPLICATIONS AND LONG-TERM SEQUELAE

General Principles

To prevent complications and long-term sequelae after a stroke, maximizing function, decreasing morbidity, and preventing rehospitalization from a complication are important. Prevention of these complications begins on the day the patient arrives at the hospital with symptoms of acute stroke. Many complications are associated with bed rest in general, but some are specific to stroke.

Musculoskeletal Complications

Contractures. Contractures are periarticular motion impairments that result from loss of elasticity in the periarticular tissues, which include muscles, tendons, and ligaments. Contractures can occur in any immobilized joint but are particularly prevalent in the paretic limbs after a stroke. In fact, only 10% of stroke patients recover limb strength and mobility rapidly enough to avoid developing contractures.[63] Shoulder pain, contractures, and muscle pain occur in 70% to 80% of patients who have had a hemiplegic stroke.[128] Chapter 10 addresses the management and related issues of the hemiplegic shoulder. Contractures also occur in other areas and begin to be problematic within a few days of onset or several days after the stroke when symptoms of immobility and spasticity may begin to develop. Usually contractures occur in a pattern of flexion, adduction, and internal rotation; muscles that span two joints are more susceptible to contracture formation.[66] To prevent shortening of the connective tissue in muscles and joints, an active range of motion (ROM) program must be initiated. Because certain muscles span two joints, joints must be positioned to allow full physiological stretch of the muscles involved. Once a contracture is present, the mainstay of treatment is gradual, prolonged stretch. The minimal treatment is a sustained stretch greater than 30 minutes.[84] Other treatments include splinting, deep-heating modalities,[23] and possible surgical release for long-standing, tight contractures[66] (see Chapter 13).

Osteoporosis. Bone is a metabolically active tissue normally in a state of equilibrium between active bone resorption

and deposition. The ratio of bone formation to bone resorption is influenced by the stressors to which the bone is subjected, a relationship known as Wolff law.[23] The lack of weight-bearing and normal stress on long bones on the hemiplegic side of a stroke patient leads to a predominance of bone resorption. This loss of bone mass can start as early as 30 hours after the beginning of immobility[155] and with bed rest can be as high as 25% to 45% in 30 to 36 weeks.[39] In patients who have had a stroke, osteoporosis is often worse, and the rate of hip fracture is far higher on the side of the hemiplegia.[67]

Osteoporosis prevention is accomplished best with measures that include active weight-bearing exercise and active muscle contraction. Medical therapies for individuals at risk for osteoporosis should be initiated. Therapies include bone-forming agents, calcium and vitamin D supplementation, hormone replacement, and other measures as needed. Box 1-1 shows some of the medical treatments available for osteoporosis.

Heterotopical Ossification. Heterotopical ossification is the deposition of calcium in the form of mature bone in the soft tissues. The condition is not particularly common after stroke but occurs with increased incidence after traumatic brain injury. The incidence ranges from 11% to 76% in various studies.[17] Spasticity is associated with the development of heterotopical ossification as are long-bone fractures and a prolonged coma. Symptoms of heterotopical ossification usually develop one to three months after injury with pain and limited ROM.[24] The diagnosis is based on clinical examination, elevated alkaline phosphatase levels in the serum, and a positive bone scan.

Treatment for heterotopical ossification includes active ROM; no studies indicate that the condition is caused or worsened by active ROM exercises.[17] Pharmacological treatment options include the use of etidronate disodium and nonsteroidal antiinflammatory drugs.[24] Other treatments include radiation therapy and, for refractory cases after the lesion has matured, surgical excision of the heterotopical ossification. Performance of ROM exercises after surgery is particularly important. Low-dose radiation or etidronate disodium can also be used to prevent recurrence.[34]

Box 1-1

Treatments for Osteoporosis

- Bone forming agents (etidronate and others)
- Estrogen replacement
- Calcitonin
- Calcium supplementation
- Vitamin D supplementation
- Fluoride supplementation
- Weight-bearing exercises

Falls. Falls are of particular concern in survivors of stroke. These patients are at increased risk of hip fracture because of developed osteoporosis, and the acuity of their balance, visual perceptions, and spatial perceptions is decreased. The increased risk of falls has been documented in several studies and is greater in patients who have had a right hemispheric stroke.[36,106,118] Fall prevention should emphasize balance and cognitive training, removing environmental hazards, and using adaptive devices. (These measures are reviewed in Chapters 8, 14, 15, 19, 27, and 28.)

Neurological Complications

Seizures. Seizures after strokes have been documented since the nineteenth century. The incidence of late-onset seizures (epilepsy) in the individuals who have had strokes ranges from 6% to 18%,[59,162] whereas the incidence of early seizures is approximately 10%, with reports ranging from 3% to 38%.[14,168] The risk for seizures is highest right after stroke; 57% of seizures occur in the first week, and 88% of all seizures after strokes occur in the first year.[14] Seizures are more common in patients who have had an SAH; 85% of these seizures are early seizures.[148] The timing of seizures that occur after stroke varies according to the mechanism of injury. The timing of seizures after thrombotic and embolic strokes appears about equal. Patients with SAH have more seizures soon after the stroke, whereas patients with ICH are more similar to patients with ischemic stroke and may have more late-onset seizures.[168]

The treatment and management of seizures associated with stroke are usually straightforward, and monotherapy often produces adequate results. If the patient only has acute-onset seizures in the setting of his or her stroke, the patient often does not require long-term antiseizure medication. A single, brief seizure or a nongeneralizing local seizure also can often be managed conservatively. If seizures do require treatment, a single agent usually suffices and is beneficial, because the drug interactions are fewer, and the compliance is better with monotherapy. Carbamazepine and phenytoin are the preferred agents for treating epilepsy after stroke. Management of the medication requires close follow-up to ensure that the desired outcome is achieved: an asymptomatic, seizure-free patient. Excessive medication can lead to a number of symptoms (Box 1-2). Inadequate control of the condition leads to additional seizures. For situations in which seizures become refractory to treatment, one must remember several factors.[168] Intercurrent illness or metabolic disarray that lowers the seizure threshold may make the seizures more frequent and difficult to treat. Patient compliance may be a problem, especially if the stroke created cognitive and behavioral deficits. Progressive lesions or new infarcts are also causes of increasing seizure frequency. Finally, a stroke that occurs in highly epileptogenic areas—such as the hippocampus,

Box 1-2

Signs of Excessive Antiseizure Medication

- Lethargy
- Drowsiness
- Depression
- Nystagmus
- Ataxia
- Irritability
- Distractibility
- Poor cognition
- Poor memory

the parietooccipital cortex surrounding the rolandic fissure, and calcarine cortex—may engender refractory epilepsy and require combination therapy. Table 1-7 lists the common seizure medications and their side effects.

Hydrocephalus. Hydrocephalus can occur acutely, especially in patients with SAH and ICH as discussed previously, or it can develop symptoms insidiously later. Hydrocephalus is usually heralded by the gradual onset of a triad of symptoms, including lethargy with decreased mental function, ataxia, and urinary incontinence. Once hydrocephalus is suspected, one should perform a CT scan promptly because the increasing size of the ventricles is readily visible. Once diagnosed, one should surgically place a ventricular shunt. The procedure is well-tolerated and can lead to resolution of all the symptoms of hydrocephalus if performed promptly. Patients with an occluded shunt have symptoms that mimic the initial symptoms of hydrocephalus.

Spasticity. Spasticity is defined as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks. Spasticity results from hyperexcitability of the stretch reflex (which is one component of the upper motor neuron syndrome).[89] In a normal recovery after a flaccid stroke, an initial period occurs with little resistance to passive motion of the muscles and joints. Approximately 48 hours after the stroke, tendon reflexes and muscle resistance to passive motion begin to return.[66] Spasticity is most pronounced in the flexor muscles and occurs throughout the hemiplegic side. The lower extremity later develops a component of extensor spasticity that can assist with function, whereas the upper extremity spasticity is usually in a flexor pattern.[10]

The management of spasticity includes encouraging voluntary movement, ROM exercises, and a functional rehabilitative approach.[66] The research data on the different neurorehabilitative treatment approaches do not define clearly which approach is most effective, so an individualized approach to treating each patient is the best course. Pharmacological treatments for spasticity are numerous, and they need to be tailored to each patient to find the best balance of side effects and efficacy. The most commonly used agents are baclofen, dantrolene sodium, and diazepam. These medications and a representative sample of the other medications used to treat patients who have had a stroke are presented in the table of medications and their side effects on the inside cover of the book. Other treatments for severe spasticity that are more invasive include phenol blocks and neurolysis, botulinum toxin (Botox) injections, and implantable baclofen pumps. Botox injections and baclofen pumps are still experimental approaches, and ongoing studies will elucidate their future roles (see Chapter 10).

Other Complications

Deconditioning. Physiological deconditioning in patients after a stroke results from the acute medical illness and the associated bed rest and immobility that may result. Table 1-8 lists some of the effects of deconditioning. All of these factors can alter the ability of the patient to recover. Therefore, to get the patient out of bed and to increase activity as early and aggressively as possible is important.

Psychological Complications. Stroke is a major life event and is associated with significant alterations in the individual's well-being and independence. Negative emotional reactions are common in patients following a stroke[152] and can have a significant effect on the patient's eventual outcome. After a stroke, patients may go through the four stages of bereavement described by Worden.[172] These include accepting the loss, experiencing the pain of the loss, adjusting to a new environment in which previous abilities are missing, and investing in new activities. Not all patients become depressed, and this lack of depression does not necessarily mean the patient is in denial.[173] Denial is a normal defense mechanism, and as long as it does not interfere with the rehabilitative process, it is not a concern.[152] The indifference reaction, a persistent denial reaction, is more common in patients who have had a right-sided stroke than a left-sided stroke.[53]

Another common consequence of stroke is emotional lability, which is rapidly shifting from one extreme emotion to another. Approximately 20% of patients have emotional lability six months after a stroke, and up to 10% have lability for one year. Emotional lability is more common in patients with pseudobulbar palsy and right hemispheric strokes, particularly if the patient is depressed.[74]

Anxiety is also common after stroke and is more frequent in patients with left hemispheric strokes[94] and cortical lesions.[144] Many sources of anxiety exist, including financial affairs, family issues, and a fear of dying or recurrent stroke. Reassurance and constant positive feedback during rehabilitation can help, and in severe cases, treatment with anxiolytics and psychological support may be needed.

Table 1-7

Medical Management of Seizures: Drug Therapy

MEDICATION	SIDE EFFECTS	PRINCIPAL USES
Phenytoin	Ataxia Incoordination Confusion Rash Gum hyperplasia Hirsutism Osteomalacia	Tonic-clonic (grand mal) Partial
Carbamazepine	Ataxia Dizziness Diplopia Vertigo Bone marrow suppression Hepatotoxicity	Tonic-clonic (grand mal) Partial
Phenobarbital	Sedation Ataxia Confusion Dizziness Depression Decreased libido Rash	Tonic-clonic (grand mal) Partial
Primidone	Same as phenobarbital	Tonic-clonic (grand mal) Partial
Valproic acid	Ataxia Sedation Tremor Bone marrow suppression Hepatotoxicity Weight gain Transient alopecia	Absence (petit mal) Atypical absence Myoclonic Tonic-clonic (grand mal)
Clonazepam	Ataxia Sedation Lethargy Anorexia	Absence (petit mal) Atypical absence Myoclonic
Ethosuximide	Ataxia Lethargy Rash Bone marrow suppression	Absence (petit mal)

Fortunately, outbursts and aggressive behavior are rare after a stroke, but when they occur, they are more common in patients with left-sided infarcts who are more aware of their deficits. The approach to management of these outbursts should not include restraints and threats but should be based on avoiding excessive frustration in the patient by removing emotional triggers and alternating easy and difficult tasks.[152]

Depression is common after stroke, developing in 20% to 50% of stroke survivors, with 30% being the most commonly accepted figure.[152] The depression can be a reaction to the stroke or a neuropsychological sequela of the stroke. The consequences of depression after stroke are numerous: hospital stays are longer,[42] cognitive impairment is greater,[125] and motivation decreases.[140] Depression is more common in patients with left cortical lesions[145] and lesions close to the frontal poles and is shorter in patients with subcortical and brainstem lesions. Depression after stroke often is treated best with antidepressant medications.[152] In patients who are unable to tolerate antidepressants, are unresponsive to therapy, or have active suicidal ideation, electroconvulsive therapy can be a last resort.[110] (See Chapter 2 for more information about the psychological effects of stroke.)

Table 1-8

Deconditioning Effects of Stroke

Musculoskeletal	Atrophy
	↓ Strength of tendons, ligaments, bones, and muscles
	Depression
	Anxiety
	Sleep disturbance
Cardiovascular	↓ Stroke volume
	↑ Heart rate
	↓ VO_2 max
	↑ Respiratory rate
	↓ Lean body mass
	↑ Body fat
	Orthostatic hypotension
Neurological/emotional	Sensory deprivation
	↓ Balance
	↓ Coordination
	Fatigue
Genitourinary	Diuresis
	Difficulty voiding
Endocrine	Impaired glucose tolerance
	Altered regulation of hormones
Body composition and metabolism	Nitrogen loss
	Calcium loss
	Potassium loss
	Phosphorus loss
	Sulfur loss

Urinary Tract Dysfunction. Urinary incontinence is common after stroke, affecting 51% to 60% of patients,[20] and can cause difficulties with rehabilitation, influence eventual discharge location, and place stress on caregivers.[43] One month and six months after stroke, 29% and 14% of patients, respectively, still have urinary incontinence.[11] The usual pathophysiology of incontinence is detrusor hyperreflexia, which is common in patients with cortical lesions. The incontinence assessment includes a thorough history of the urinary symptoms and can include urodynamic studies to help define the problem. Incontinence treatment includes timed voiding and use of pharmacological agents and intermittent catheterization. If these treatments do not work, incontinence may need to be treated by indwelling catheterization. This is performed on patients who cannot independently self-catheterize and do not have caretakers who can provide this care or on patients who have physical barriers such as urethral strictures that prevent regular catheterizations. Unfortunately, indwelling catheters have a high incidence of associated urinary tract infections. Male patients also may use external condom catheters, which can provide socially acceptable continence when the individual is traveling or physically active. Patients with continuous dribbling also benefit from condom catheters. The goal of all of these therapies is to maintain continence and prevent urinary tract infections and other complications such as skin breakdown from skin maceration.

Skin Breakdown and Decubitus Ulcers. Pressure ulcer formation is a serious health problem in debilitated and immobilized patients. After a stroke, patients are at particular risk for pressure ulcers because they have numerous factors contributing to skin breakdown. Abnormal sensation, contracture, malnutrition, immobility, and muscle and soft-tissue atrophy often develop and may be complicated by advanced age. Prevention of pressure ulcers, rather than treatment of developing ulcers, should be the focus of care. Preventive measures include frequent repositioning, keeping skin clean and dry, maintaining an adequate level of nutrition, and, especially in high-risk patients, using pressure-relief mattresses.[132] Once pressure ulcers have formed, in addition to strictly observing the preventive and pressure relieving measures previously noted, treatments include meticulous wound care with a variety of agents and possibly surgical reconstruction.

Dysphagia. Swallowing disorders are common after a stroke. Dysphagia is more common in the elderly, with an incidence of 25% to 45%.[59,61] Aspiration can lead to pneumonia, and a decreased eating ability can lead to dehydration and malnutrition. Chapter 24 covers the details of the pathology of aspiration and the methods of its treatment.

Aspiration. Aspiration causes chemical pneumonitis that can lead to a secondary bacterial infection. Because numerous anaerobic organisms are in the mouth, aspiration pneumonia can develop into an anaerobic abscess.[92] Such abscesses occur less frequently in edentulous individuals because they have less oral flora and can occur in up to a third of cases in hospitalized patients.[97] The treatment of choice is to reduce the risk of aspiration and to administer antibiotics. Examining a radiographic film for evidence of abscess cavities and the sputum for organisms can help one develop a specific medical treatment. Sputum culture growth often requires up to three or four days, so initial treatment is often empirical and should be the administration of a wide-spectrum antibiotic that is effective against hospital-acquired organisms (which are often resistant to certain antibiotics) and anaerobic bacteria.[92] The usual course of antibiotics is seven to 10 days, but cavitary pneumonia may require far longer treatment for eradication of the organism.[93] Determination of which specific antibacterial agents to use depends on the resistance patterns in the institution in which the aspiration takes place; the infectious disease team at that institution should make the decision about which antibiotics to use.

Deep Venous Thrombosis. DVT is a common problem after stroke and has an incidence of 23% to 75% depending on the severity of the stroke. Most of the morbidity and mortality associated with DVT results from venous thromboembolism (VTE). Pulmonary embolism after stroke has an incidence of 10% to 29% and a mortality rate of 10%.[19] The formation of DVT is caused by the triad of risk factors outlined by Virchow postulates: altered blood flow, damage to the blood vessel wall, and altered blood coagulability. Box 1-3 lists the common risk factors for DVT. Of the risk factors for DVT, stasis is one of the most important. After a stroke, DVT is 10 times more common in the paretic leg.[165] DVT usually begins in the calf, and although the emboli from calf thrombi are not dangerous, these thrombi propagate in about 20% of cases, and about 50% of the proximal deep venous thrombi embolize. About 20% of symptomatic pulmonary emboli are fatal.[134] After a stroke, ambulation in itself is not preventive in the subacute setting: pulmonary embolism occurred in 57% of ambulatory patients in the rehabilitation setting.[147] Lower extremity and pelvic DVT are the most common, but proximal upper extremity DVT also can occur, although it is rare. All of the diagnostic and management issues discussed in the section on VTE that follows applies to this condition as well.

The diagnosis of DVT in the clinical setting is unreliable,[19] and many patients with life-threatening embolism and thrombosis have no clinical symptoms of DVT. Other patients with swelling and tenderness may not have DVT at all and may have any of a number of other diagnoses. The differential diagnosis of lower extremity pain and swelling includes trauma, fracture, gout, cellulitis, and superficial phlebitis. The usual clinical signs of DVT include pain and tenderness, swelling, the presence of Homans sign (elicited by dorsiflexion of the ankle while the knee is flexed resulting in pain in the calf), superficial venous distention, a palpable cord, and fever. Some of these signs, such as Homans, are unreliable indicators. Homans sign is present in less than one third of patients with DVT and is present in half of patients without DVT.[73] Objective testing for DVT has venography as the gold standard, but this procedure is associated with significant risks, including anaphylaxis and causing DVT. More commonly used risk-free procedures are impedance plethysmography, which is a noninvasive test that measures volume changes in the leg with circumferential calf electrodes,[75] and Doppler ultrasound, which is also a noninvasive test that uses a handheld probe to detect blood flow in deep leg veins.[166] Doppler ultrasound and impedance plethysmography have similar sensitivities and specificities for DVT detection, but Doppler ultrasound is not as portable and has a higher cost than impedance plethysmography.[19]

Box 1-3

Risk Factors for DVT

- Immobilization
- Postoperative state
- Age >40 years
- Cardiac disease
- Limb trauma
- Coagulation disorders
- Obesity
- Advanced neoplasm
- Pregnancy

The clinical diagnosis of pulmonary embolism is also unreliable, and only 30% of patients with pulmonary embolism have clinical DVT, even though 70% have venographic evidence of DVT.[19] The symptoms of submassive pulmonary embolism overlap with the symptoms of many other pulmonary conditions, including tachypnea, tachycardia, rales, hemoptysis, pleuritic chest pain, pleural effusion, general malaise, bronchospasm, and fever. In patients with massive pulmonary embolism with greater than 60% of the pulmonary circulation obstructed, patients are critically ill and develop heart failure, circulatory collapse, hypotension, and coma and can die suddenly.[147] The gold standard for testing for pulmonary embolism is the pulmonary angiogram, but its use is associated with significant morbidity and mortality. The preferred noninvasive test is the ventilation/perfusion scan.[105]

The best approach to VTE is to prevent DVT. The National Institutes of Health Consensus Conference on the Prevention of Venous Thrombosis and Pulmonary Embolism recommends using low doses of subcutaneously administered heparin in all stroke patients with no hemorrhagic components.[121] In all other patients, external pneumatic calf compression is recommended. More recently, low-molecular-weight heparin has been introduced and actually may be more effective than standard heparin for DVT prophylaxis.[72] Low doses of warfarin for DVT prophylaxis in stroke patients has not been well-studied, but its use in other conditions has proved its effectiveness in DVT reduction. Dextran, aspirin, and static compression stockings are not effective for preventing DVT.[19] Physical treatments alone, such as ROM exercises, have not been studied. Ambulatory patients must be able to walk at least 50 feet to have a reduction in risk of DVT,[21] but as previously stated, the risk of pulmonary embolism in ambulatory patients is still significant.[147] The length of time prophylaxis should continue is still not definite, but evidence shows that continuing prophylaxis well into the subacute phase is warranted.[19]

The treatment of VTE (DVT and pulmonary embolism) is based on preventing pulmonary embolism, which

can be fatal. A patient who is identified with acute VTE is started on intravenous (IV) heparin as long as no contraindications to anticoagulation exist.[70] The effectiveness of the heparin is determined by monitoring the partial thromboplastin time, and the heparin is adjusted to a dose between 1.5 and 2.5 times control. In a patient with only DVT, warfarin can be started on the first day, and the heparin can be discontinued when the warfarin dose is therapeutic as measured by the increase in the prothrombin time or international normalized ratio. Targets are a prothrombin time of 1.25 to 1.5 times control or an international normalized ratio of 2 to 3.[19] In patients with pulmonary embolism, warfarin may be started a few days later, and after management of the acute stage, the patient keeps receiving it longer; patients with DVT receive warfarin for approximately three months, and patients with pulmonary embolism, for six months.[72] All patients who recently have been diagnosed with VTE are placed on bed rest initially and usually are allowed to become mobile two days after the partial thromboplastin time has become therapeutic.[76] The rehabilitation of patients with VTE who are beginning treatment should continue at the bed side, and, in the case of patients with lower extremity DVT, the rehabilitation program should include activity of daily living (ADL) training, upper extremity programs, communication work, and dysphagia treatments.

FUTURE TRENDS IN MEDICAL STROKE MANAGEMENT

Improved Primary Stroke Prevention

Because the treatments for stroke are so limited and the deficits that can result are so devastating, the primary prevention of stroke has to be the essential strategy to decrease morbidity and mortality from stroke. With a good understanding of the risk factors for stroke, risk factor modification can be targeted at groups and individuals who are at risk. Table 1-1 lists the preventable and nonpreventable risk factors for stroke. Fortunately, many of the risk factors are the same as those for myocardial infarction and vascular disease leading to death, so the modification of stroke risk factors also decreases the risk of cardiac-related morbidity and mortality. Due to greater awareness and risk factor modification and largely through the treatment of blood pressure, a decline of greater than 50% in the stroke mortality rate has occurred in the past 20 years.[169] Each of the modifiable risk factors are considered separately.

Hypertension

Diastolic and systolic hypertension are each independently and strongly implicated in causing stroke. Hypertension increases the risk of stroke in all age groups of men and women.[169] In fact, no threshold level of blood pressure exists below which the risk curve plateaus.[98] For every 7.5 mm Hg increase in diastolic pressure is a 46% increase in stroke incidence and a 29% increase in coronary heart disease (CHD). Reducing blood pressure in hypertensive patients has been shown to decrease the risk of stroke significantly, with an average reduction of 5.8 mm Hg leading to a reduction in stroke incidence of 42% but only a 14% reduction in CHD incidence.[32] Because these trials only spanned two to five years, the reduction in stroke incidence is a direct result of decreased blood pressure and not an alteration in atherogenesis (production of plaque in the arteries), which would take longer to develop.[169] Systolic blood pressure is also a factor; the treatment of isolated systolic hypertension (>160 mm Hg) has been shown to reduce the incidence of stroke by 36% and CHD by 27% over 4.5 years.[120] Treating all forms of hypertension in the older age groups is therefore essential because they are at increased risk for stroke, and most strokes occur in this age group. Screening for hypertension and aggressively treating systolic and diastolic hypertension should be the cornerstone of any primary prevention program for stroke.

Cigarette Smoking

The results of the Framingham Study and the Nurses' Health Study demonstrate that the cessation of cigarette smoking should lead to a prompt reduction in stroke mortality.[31,171] Risk of CHD decreases by 50% in one year and reaches the level of a nonsmoker's risk in five years. Smoking increases stroke risk by 40% in men and 60% in women (with no other risk factors being considered), and it seems to follow that smoking cessation leads to a reduction in stroke risk similar to the reduction in CHD incidence.

Cardiac Dysrhythmia and Myocardial Infarction

CHD, atrial fibrillation, and congestive heart failure lead to an increased incidence of stroke.[169] Preventing these conditions by modifying their associated risk factors leads to a reduction in incidence of stroke. In addition, treating patients who have established dysrhythmias and congestive heart failure with anticoagulants such as warfarin decreases the incidence of stroke (as explained previously).

Blood Lipids

The development of carotid artery atherosclerotic disease has been shown to be related to the levels of serum lipids.[133] However, to relate accelerated atherosclerosis clearly to an increase in the incidence of stroke has been difficult because other pathologies related to serum lipids have been observed. Levels of total serum cholesterol less than 160 mg/dL seem to be associated with ICH and SAH, whereas higher levels of serum cholesterol are associated with atherothrombosis. No relationship has been demonstrated between cholesterol and lacunar strokes.[169] This unusual relationship of low serum lipids and higher hemorrhagic infarct has been demonstrated in Japan and also recently in the United States in the group

of patients studied in the Multiple Risk Factor Intervention Trial.[78,124] Because of the ambiguity of these data, a clear statement of guidelines for the management of cholesterol to reduce incidence is difficult to make.

Diabetes

The rate of atherosclerosis development in coronary, femoral, and cerebral vessels is increased in diabetics. Stroke is increased 2.5 to 4 times in diabetics compared with nondiabetics.[86] In the Framingham Study, glucose intolerance (a blood sugar greater than 150 mg/mL) is only a significant, independent contributor to stroke in older women and is greater for women than men at any age.[80] Because of the associated risk of stroke, careful management of diabetes in addition to all other risk factors is prudent.

Oral Contraceptives

In female patients over the age of 35 who have other stroke risk factors, oral contraceptive use is associated with increased incidence of stroke.[142] The relative risk for oral contraceptive users is approximately five times greater if they are already in the high-risk group. With the use of lower estrogen formulation oral contraceptives, the risk has decreased substantially in recent years.[143] That the incidence of fatal SAH increased in oral contraceptive-using women with concomitant smoking is noteworthy; in the group over age 35 the incidence is four times higher.[52] Therefore, the recommendation is that women over the age of 35 avoid using oral contraceptives, and younger women who smoke should be advised of the increased risks associated with concurrent oral contraceptive use.

Alcohol

Heavy alcohol consumption is related to an increase in stroke and stroke deaths, whereas light to moderate alcohol consumption is associated with a reduced incidence of CHD.[38,85] Alcohol is clearly related to hemorrhagic stroke events, but the association with thromboembolic events is not definite. Regardless, patients at risk for stroke should avoid heavy alcohol consumption.

Physical Activity

Despite the clear benefits of physical activity in the reduction of CHD morbidity and mortality, no clear association exists between physical activity and the incidence of stroke.[114,115]

Public Education

The primary goal of primary and secondary prevention programs should be to educate individuals about risk factors and then to teach them the way to modify their risks. During routine visits, a physician should be able to identify at-risk patients through a combination of a history and physical. Routine blood pressure screening should be included in all evaluations, and patients who have hypertension should be treated. A stroke risk profile has been assembled from the Framingham Study data and can be used by physicians[170] (e.g., to help a physician decide which borderline hypertensive patients to treat). Education can start in the physician's office and be continued by all the other health professionals with whom the patient comes into contact. If the community at large is educated about the risk factors of stroke, those individuals who are at highest risk can seek out the attention they require. This model has been implemented and supported through research such as the Agency for Health Care Policy and Research Smoking Cessation Clinical Practice Guidelines.[116]

PART TWO: Introduction to Acute Stroke Rehabilitation

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The neuro-ICU may be the starting point of occupational therapy (OT) evaluation and treatment. Many patients are evaluated, by an occupational therapist, within 48 hours of a stroke. The ICU environment is often fast paced with the focus on monitoring the individual patient's medical status. The primary goals of any neuro-ICU are to stabilize the patient medically, progress the patient neurologically, and support the patient and family through this neurological crisis.[137] Medical testing and procedures take precedence over any OT treatment. Scheduling OT services may be difficult, treatments may be interrupted, and flexibility is necessary.

THE IMPORTANCE OF EARLY INTERVENTION

There are many common complications associated with a prolonged ICU stay, which include but are not limited to deconditioning, muscle weakness, contractures, skin impairments, depression, anxiety, and reduced quality of life.[60] Early OT, engaging in ADL and mobilization, can increase a patient's level of consciousness, enhance overall mental well-being, and foster functional independence.[129,146] Occupational therapists provide a variety of treatments in the ICU, including, but not limited to, evaluations, splinting, positioning, cognitive retraining, self-care, and functional mobility training.

TEAM APPROACH

There are many members of the neuro-ICU/acute care team, and the team may vary among settings. They include a primary team of physicians led by an attending neurologist specializing in critical care. Depending on each case,

there may be neurosurgeons also involved in patient care. At teaching hospitals, a team of residents may also make medical decisions regarding the patients. Along with the occupational therapist, the ancillary team consists of nursing, including the primary nurse and nurse practitioner, social workers, nutritionist, speech and language pathologist, and physical therapist (Table 1-9). An occupational therapist treating patients in this environment must foster these relationships to safely treat patients.

The relationship between the primary physician, nurse, and the occupational therapist is particular important. Daily communication with the physicians, residents, and primary nurse is necessary prior to initiating an evaluation or treatment session due to the fluctuating physical condition in the ICU phase of hospitalization.[4,137] Physicians, nursing, or the occupational therapist, using their own clinical judgment will determine if intervention should be delayed should a patient's neurological status deteriorate. Once the patient has been medically cleared for OT evaluation, a review of the patient's medical chart should be completed. The therapist can glean information relating to any precautions and complications that may interfere with the OT treatment (Box 1-4).

MONITORING THE ICU/ACUTE STROKE SURVIVOR

Any therapist treating in the ICU should not only be aware of the medical and nursing priorities in the ICU, but also of how to monitor the patient during OT treatment. The therapist needs to be competent in reading ICU monitors and handling ICU related drains and lines, so that appropriate parameters and precautions are adhered to during the treatment session. Common monitors, drains, lines, and clinical implications are listed later.

Table 1-9

Members of the ICU/Acute Team

MEMBER	ROLE
Attending physician	Leads the medical team is medical decision-making. May lead team rounds. Usually interacts with patient at least once a day.
Resident	At a teaching hospital, residents are responsible for the day to day, hour to hour care of patients. May be on the unit at all times to answer clinical questions regarding patients.
Nursing	Multiple responsibilities include but are not limited to: administering medications, ADL assist, education, positioning, and monitoring neurological status.
Nurse practitioner	In some facilities, nursing practitioners take the place of residents, writing orders and providing medical decision-making when needed.
Nutritionist	Usually the nutritionist evaluates the patient on a PRN (as needed) basis. Most patients in the ICU receive a nutrition consult when they are placed on tube feedings. The nutritionist, along with the physicians, will determine which type of tube feeding a patient should receive, along with the speed at which the feedings should be administered.
Social worker	In the ICU, the social workers are also usually a PRN service providing support to family members and beginning the discussion of discharge planning.
Speech and language pathologist	Speech and language pathologists can provide a twofold service in the ICU setting. They may provide therapy services in the form of language and communication evaluation and treatment. They may also provide bed side swallowing evaluations, along with the occupational therapist. See Chapters 20 and 24.
Physical therapist	The physical therapist provides bed side physical therapy services in the form of therapeutic exercise, mobility, and gait training if appropriate. Along with the occupational therapist, he or she also contributes to discharge planning. See Chapter 15.

Box 1-4

Initiating Treatment

1. Check to make sure occupational therapy orders are active. This should be done prior to each and every treatment session
2. Review the patient's medical record. The therapist should evaluate the medical record for potential reasons to hold a patient from therapy. Such reasons may be a change in mental status, development of a deep vein thrombosis or pulmonary embolism, or expansion of the stroke. Every facility has different standards for when therapy is to be held.
3. Review the patient's current status with the medical team. Using clinical reasoning the therapist will determine if the patient is appropriate for an OT session. The therapist should clear any treatment with the patient's nurse to determine if all medical information reviewed from the medical record is most current.
4. Begin evaluation and treatment with a gross assessment of mental status, strength, and vital signs. Great discrepancies from what is reported in the medical record should be reported to the nurse and treatment suspended. Proceed with therapy as indicated.

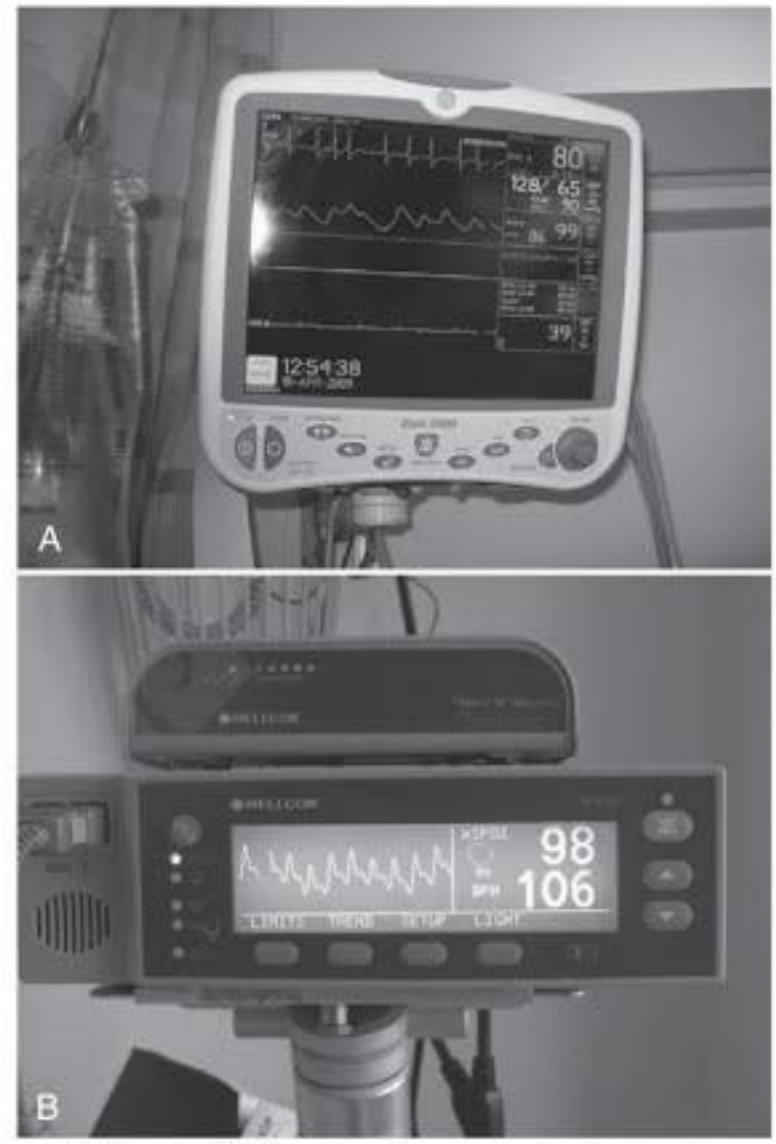

Figure 1-10 **A,** ICU monitoring system, indicating heart rate 80 beats per minute, blood pressure 128/65 (mean arterial pressure 90), oxygen saturation 99, respiratory rate 39. **B,** ICU monitoring system. This system monitors heart rate (106) and oxygen saturation (98%).

Basic ICU Monitor

Most ICU patients are connected to a monitor that allows constant display of all vital signs (Fig. 1-10). These include blood pressure, telemetry reading (which include heart rate and rhythm), respiratory rate, and oxygen saturation percentages. For normal versus abnormal vital sign responses to exercises, refer to Table 1-10. Blood pressure can be monitored either noninvasively (automated pressure cuff) or by invasive measures, such as an arterial line reading (also referred to as an A-line). A common insertion site for an A-line is either the radial or femoral artery (Fig. 1-11). With radial artery placement, passive ROM of the wrist should be avoided; with femoral artery placement, no hip ROM is allowed, resulting in bed rest.

Telemetry

Telemetry detects both the heart rate and rhythm and displays this reading on the monitor. Bed side telemetry is similar to an electrocardiogram (ECG). An ECG is read by placing 12 electrical leads to read heart rate and rhythm while the bed side telemetry uses either three or five leads. The primary nurse will set both heart rate and rhythm parameters on the monitor. Should the rate and rhythm become abnormal, an alarm will sound. Physical activity should be monitored accordingly.

Common Lines and Drains

Foley Catheter. A Foley catheter is indwelling and is used to drain urine from the bladder. The therapist should avoid clamping the catheter; doing so could result in a backup of urine in the bladder. The bag, which collects the urine, needs to be at a lower level than the patient's bladder for the urine to flow in the correct direction.

External Ventricular Drain. The external ventricular drain (EVD) is a small tube surgically inserted into the ventricles of the brain, which drains cerebral spinal fluid (CSF) (Fig. 1-12). The tube is connected to a device that measures the amount of this fluid. This procedure is used when the intracranial pressure is elevated, and the drain may be clamped for short periods of time by nursing only. Due to specific calibration, function of the drain, and accuracy in measurement the head of the bed must be elevated to a specific level. Unless the drain is clamped, the head of the bed may not be changed, and patients should not be mobilized.

Intracranial Pressure Monitoring Catheter. The intracranial pressure monitoring catheter (ICP) is a catheter passed through a burr hole and placed in the ventricles of the brain.

Table 1-10

Vital Sign Responses

VITAL SIGN	NORMAL RESPONSE TREATMENT	ABNORMAL RESPONSE TO TREATMENT	EXCEPTIONS TO THE RULES
Heart rate			
Normal heart rate 60 to 100 beats per minute Many patients may have a resting heart rate outside the normal value. Determine the patient's maximum heart rate (220−age)[146] prior to treatment to assess whether or not it is safe to proceed.	Slow and gradual increase in heart rate with activity up to 20 beats higher per minute.	Increase in heart rate greater than 20 beats per minute. A decrease in heart rate or a change in heart rhythm.	At times patients may not be able to tolerate an increase in heart rate that deviates from their baseline. At other times, with young, otherwise healthy, patients, the team may allow the therapist to work patients beyond an increase of 20 beats per minute. Some medications may cause a blunted heart rate response.
Blood pressure			
Normal blood pressure: systolic less than 120 mm Hg, diastolic less than 80 mm Hg. Again many patients may have a resting blood pressure above or below what is considered normal. Check the patient's chart to determine what the patient's blood pressure ratings have been over the past few vital sign cycles. Determine from there if it is safe to proceed.	Slow, gradual, and slight increase in systolic blood pressure with activity. No change or slight decrease in diastolic pressure.	Increase or decrease in systolic blood pressure greater than 20 points and a decrease of diastolic pressure greater than 10 points.[137]	Many times in the ICU, a patient's blood pressure is maintained high (i.e., 200/100) to profuse the brain. It is important to check with the team prior to holding therapy. However, as a general rule, if a patient's systolic blood pressure is greater than 200 and diastolic pressure is greater than 100, check with the team prior to treatment.
Oxygen saturation			
Normal range: 92% to 100% on room air or on supplemental O_2.	Slight drop or increase in O_2 saturation.	Drop in O_2 saturation below 92% (unless that is baseline).	In some cases, the team will allow the therapist to titrate the patient's O_2 needs to the activity by increasing O_2 via nasal cannula. It is important to remember that O_2 is considered a medication, and a written order from the MD is needed to change patient's O_2 consumption.

O_2, oxygen.

It is used with injuries such as hemorrhages, aneurysms, or head trauma that may lead to brain swelling and elevation of the intracranial pressure. This monitor measures any changes in intracranial pressure. The head of the bed is elevated to a set point (usually 30 to 45 degrees), as the intracranial pressure will increase when the head of the bed is lowered. Passive therapy, such as splinting or positioning, may be implemented with physician approval. Generally, ADL treatment and mobilization is held at this time.

Spinal Drain. A spinal drain is a catheter placed in the lumbar spine to drain CSF. It can be used for the treatment of CSF leak or to drain excess CSF fluid. The lumbar drain should be set to drain below the level of the leak. When the drain is open and is draining CSF, the spinal drain is set at a determined level next to the bed. At this time, when the drain is opened, patients are placed flat on their back to allow for drainage. Patients with this drain may get up and out of bed and may engage in ADL

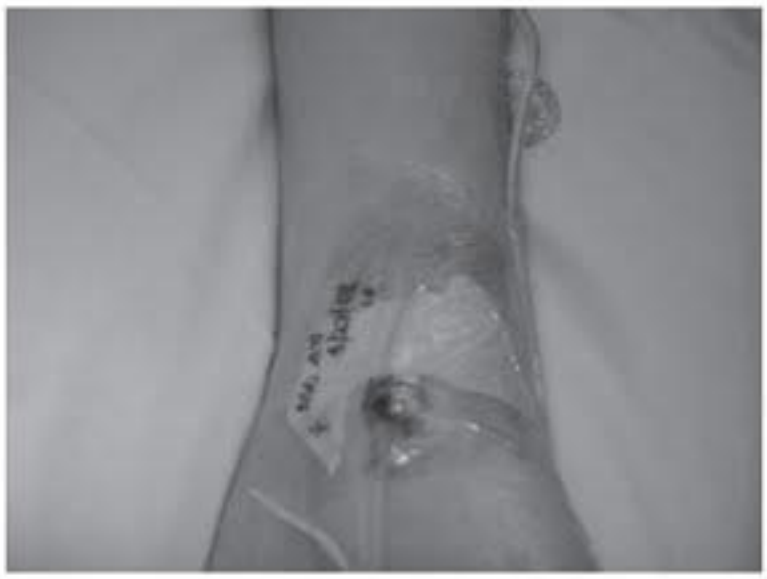

Figure 1-11 Arterial (A) line in the radial artery. (Photo courtesy of Millie Hepburn Smith.)

Figure 1-12 Exit site for an external ventricular drain on top of skull. (Photo courtesy of Millie Hepburn Smith.)

treatment only when the drain has been clamped by the nurse. While the drain is open to drain CSF, the patient must remain on bed rest.

Intravenous Line. IV lines are inserted into the peripheral veins and are generally used to administer IV fluids and medications. Because these lines are superficial, care should be taken not to place pressure from the positioning materials or splints directly over the area in order to avoid obstructed or dislodgment.

Feeding Tubes

In the event that a stroke patient is unable to swallow effectively or appears to be a high aspiration risk, alternate methods are used for nutrition intake.

Nasogastric Tube. A nasogastric tube (NGT) is placed through the nostril down the esophagus to the stomach for liquid feeds to pass. It is generally used as a short-term alternative for nutritional intake.

Percutaneous Endoscopic Gastrostomy. A percutaneous endoscopic gastrostomy is a tube inserted surgically with an endoscope through the mouth and into the stomach, exiting out through the stomach wall and dermis (Fig. 1-13).

Precautions for both feeding tubes include elevating the head of bed to 30 degrees or greater while administering the tubes to prevent aspiration. Depending upon the hospital guidelines, the therapist may be allowed to turn off the feeding prior to the therapy session, but it is recommended that the primary care nurse be consulted prior to doing so, for patient safety (see Chapter 24).

Ventilator

At times stroke can result in respiratory failure. When this is the case, patients often require a ventilator to assist them with or to perform the act of breathing for them (Figs. 1-14 and 1-15). When a ventilator is used, the patient also requires an artificial airway. In the first few days after acute stroke, a ventilator can be connected to the patient via an endotracheal tube. A breathing tube is then placed into the patient's mouth and positioned down into the patient's lung systems. If a patient is unable to be weaned from the ventilator, a tracheotomy will be performed. In this procedure, an opening is cut in the patient's trachea and a small endotracheal tube is placed in the opening, which is then attached to the vent via long tubing. Early mobilization of patients on ventilators is encouraged.[112] A recent randomized controlled trial[138] emphasized that early OT/physical therapy (PT) for those ventilated and critically ill is both beneficial and safe, resulting in better functional outcomes, decreased delirium, and more ventilator-free days.

Once the therapist is confident to handle the lines, leads, and monitors in the ICU, the patient's tolerance of the OT intervention should be monitored carefully. Vital signs should be observed during the entire treatment session and should be documented at the beginning, at mid-portion, and at end of treatment. In addition to vital signs, the therapist must also watch for changes in the patient's neurological status during treatment, which may include changes in

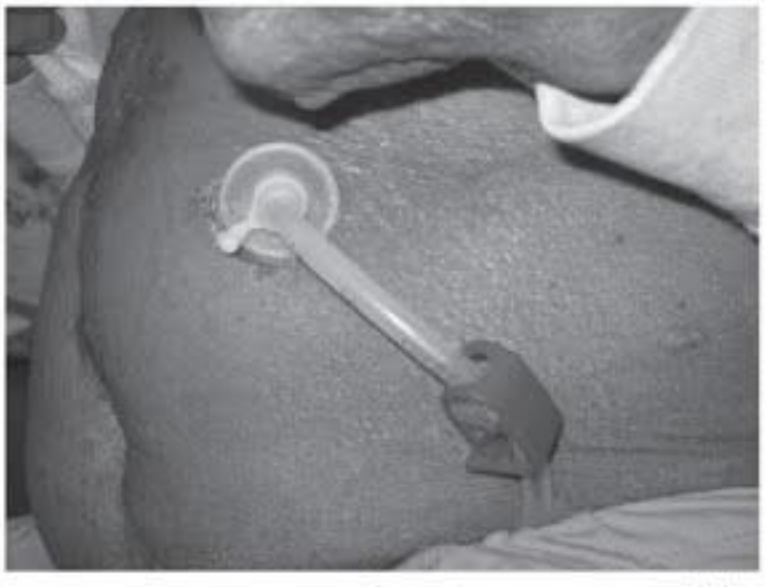

Figure 1-13 Percutaneous endoscopic gastrostomy in abdomen. (Photo courtesy of Millie Hepburn Smith.)

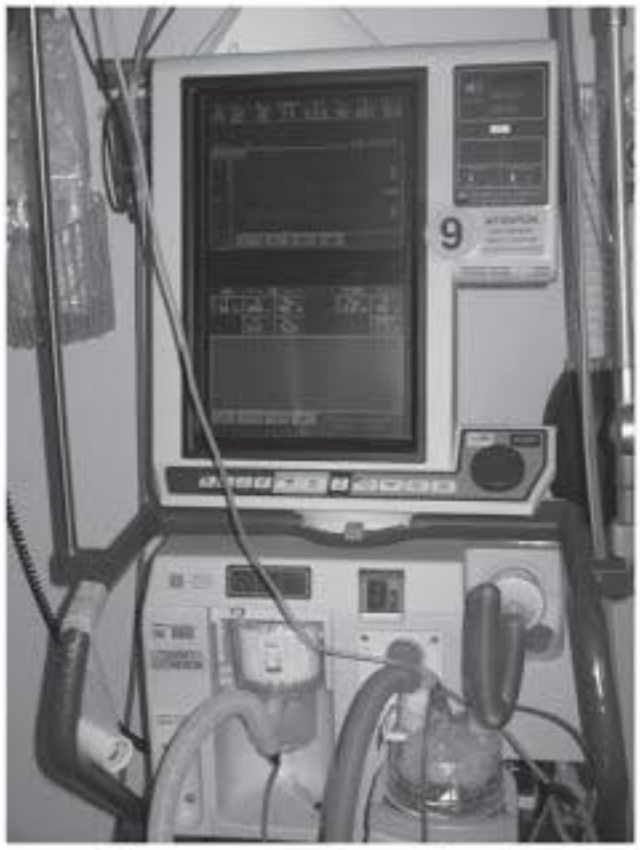

Figure 1-14 This is a commonly used ventilator in the ICU setting. The occupational therapist needs to be aware of the vent setting and alarms while working with the patient.

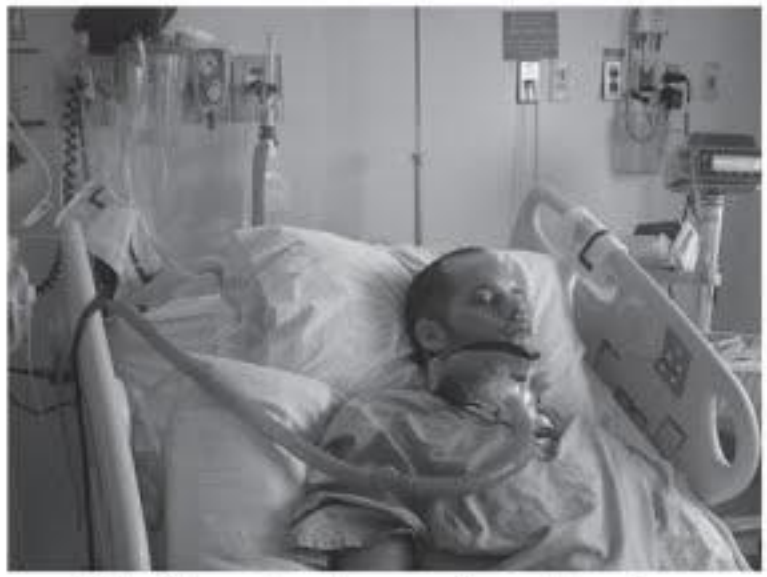

Figure 1-15 The patient is properly positioned on a trach collar and is currently being weaned from the ventilator.

decorticate or decerebrate posturing, tone, pupils, and/or in speech.[137] Patient subjective complaints must be considered. If any changes in the patient's status occur, terminate treatment and inform the medical team immediately.

ASSESSMENTS USED IN ACUTE STROKE REHABILITATION

There are a variety of standardized assessments available[82] to the occupational therapist in the hospital setting. In the acute/ICU setting, it is imperative for the occupational therapist to evaluate motor skills, cognitive function, and ADL. At times it may not be feasible for a patient to engage in ADL tasks secondary to medical status or sedation. Table 1-11 outlines some of the standardized assessments used during acute rehabilitation.

INTERVENTIONS FOR ACUTE STROKE REHABILITATION

The following sections will describe potential interventions for those in the ICU/acute stage of stroke rehabilitation.

Splinting

The primary goals at this early phase of splinting are to:

1. Correct any biomechanical malalignment and protect joint integrity.
2. Prevent shortening of soft tissues and development of contractures.
3. Maintain skin integrity.

Develop an appropriate wearing schedule to prevent learned nonuse behavior patterns. Splint-wearing at night may be more appropriate than day use, particularly if the patient has begun to initiate movement or attempts to incorporate the hand or upper extremity in functional activities. A wearing schedule should be practical to achieve compliance (Box 1-5; See Chapter 13).

Positioning

Because of the medical complexity of the ICU/acute stroke survivor, many of these patients spend most, if not all, of their time confined to bed. Therefore, positioning has because an integral part of OT treatment plan. The occupational therapist will work to develop a positioning schedule for each individual positioning. The occupational therapist must rely on other members of the interdisciplinary team, including nursing and physical therapists, and the patient's family members, if able, to carry out this portion of the treatment plan (Figs. 1-16 and 1-17).

Different members of the interdisciplinary team have different priorities when it relates to positioning. A primary goal of the team in regards to positioning is to prevent skin breakdown. The occupational therapist is encouraged to teach the team how to position the patient not only to prevent skin breakdown but also to reduce the risk of contractures and encourage joint alignment, and comfort. The occupational therapist should develop a turning schedule for each patient. Patients should alternately be positioned on the affected side, the nonaffected side, and supine. A clock drawn with specific positions can be used as a reminder for the nursing team. See Chapter 10.

When the patient is being positioned, the patient's lines and leads should be carefully observed for they provide vital medications and monitoring of each patient. Careful adjustments need to be made for head of the bed restrictions from feeding tubes or ICP/EVD. When a patient is being positioned with femoral arterial lines, care should be taken to avoid hip flexion, and the wrists of patients with radial A-lines should be maintained in a

Table 1-11

Standardized Assessments Used during Acute Rehabilitation

	ASSESSMENT	DESCRIPTION	SCALES/SCORES	LIMITATIONS
NIH Stroke Scale[37]	Standardized Prognostic Scale Total time to administer: 10 minutes	The NIHSS is a 15-item neurological examination for stroke patients used in many hospitals by physicians, nurses, and therapists. It evaluates levels of consciousness, language, neglect, visual fields, eye movement, motor strength, ataxia, dysarthria, and sensation.[22]	0 = No stroke 1–4 = Minor stroke 5–15 = Moderate stroke 15–20 = Moderate to severe stroke 21–42 = Severe stroke	No evaluation of functional tasks.
MINI FIM[61]	Standardized functional outcome measure. Total time to administer: greater than 30 minutes	Evaluation of functional tasks such as self-care, transfers, mobility, and cognition	Patient receives a score between 0–7 for each functional task. A score of 7 indicates independence while a score of 1 indicates total assist, and a score of 0 indicates the task has not taken place. The Mini FIM includes 7 items from the full 18 item FIM instrument.	Secondary to the medical complexity of ICU patients, many of the ADL or mobility sections may not be able to be completed.
Glasgow Coma Scale[154]	Standardized prognostic scale. Total time to administer: 10 minutes	This scale is used in numerous hospitals by both doctors and therapists. It evaluates best eye opening response, best verbal response, and best motor response.[21]	Each category is given a numeric response with 1 being no response. The responses are added together to create a final score. A score of less than 3 indicates vegetative state, 3–8 severe disability, 9–12 moderate disability, and 13–15 indicates mild injury.[154]	No evaluation of functional tasks.
Orpington Prognostic Scale[88]	Standardized prognostic scale. Total time to administer: 5 to 10 minutes	An evaluation of upper extremity motor function, proprioception, balance, and cognition	The numerical scores of each section are added together for the final score. Lower scores indicate less inpairment.	No evaluation of functional tasks. The cognitive evaluation is given verbally and therefore requires language and speech, eliminating patients with aphasia.
Barthel Index[100]	Standardized outcome measure. Total time to administer: greater than 30 minutes	Evaluation of functional tasks such as eating, grooming, bathing, bowel and bladder management, toilet use, dressing, mobility, transfers, and stairs.	Patient receives a score between 0–100, 0 indicating total dependence and 100 total independence with the evaluated activities.	Secondary to the medical complexity of ICU patients, many of the ADL or mobility sections may not be able to be completed (such as eating, toileting, and/or stairs).
JKF Coma Recovery Scale[56,57]	Standardized measure. Total time to administer: 15 minutes.	The scale consists of 23 items within six subscales, evaluating auditory, visual, motor, oral motor, communication, and arousal.	The lowest item on each scale represents reflexive activity, while the highest items represent higher level cognitive behaviors.	No evaluation of functional tasks

Box 1-5

Common Splints Used in Acute Stroke Rehabilitation

Resting hand splint	May be fabricated for the individual but also are available prefabricated.
Cone splint	May prevent long finger flexor tightness when used in conjunction with a wrist extension device and also maintain skin integrity (preventing skin maceration).
Adjustable inflatable hand splint	Contains an air bladder in the palmar surface, which can be adjusted to achieve the level of stretch placed on the long finger flexors. It may be an appropriate choice for the patient who has had more than one stroke and demonstrates increased muscle tone. This type of splint is prefabricated.
Blanket/towel roll	An alternative to a thermoplastic elbow extension or drop arm splint. It is rolled around the patient's arm to help prevent elbow flexion contractures. See Chapter 13 and Fig. 1–16.

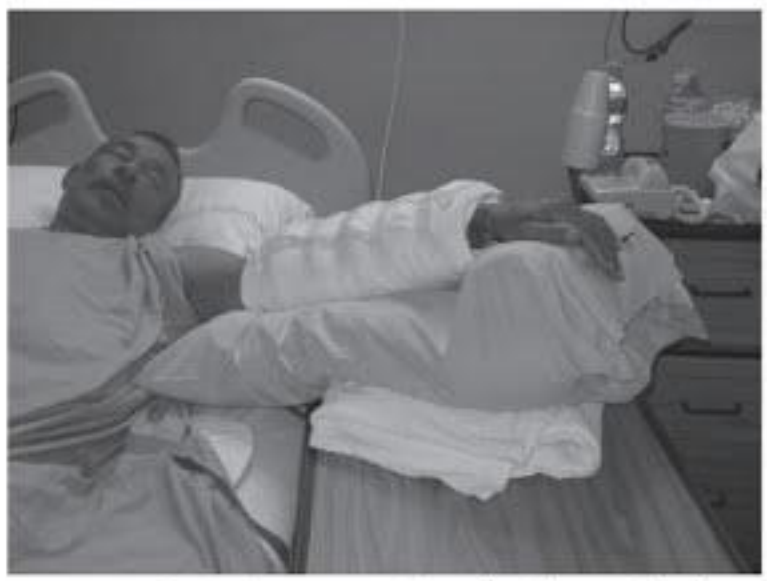

Figure 1-16 Patient's arm positioned with towel roll to increase elbow extension.

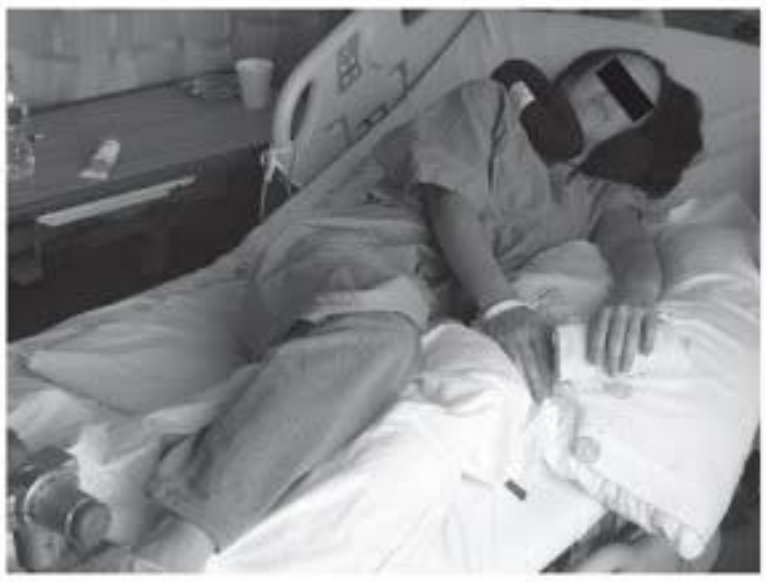

Figure 1-17 Side lying position, with patient positioned on the affected side. Pillow placed under affected upper extremity to maintain proper alignment of the head of the humerus.

neutral position. Foley and rectal tubes should be moved to the same side to which the patient is positioned.

While in the ICU, many patients require a ventilator to provide respiratory assistance. These patients can also be positioned side to side and supine. Care should be taken when moving ventilation tubes. There are many extra articular handles that allow for addition mobility of the patient on a ventilator. If these articular handles do not provide enough length to position a patient in the proper alignment, discuss with the respiratory therapist regarding switching the ventilator from side to side every other day or so.

Functional Activity Suggestions during the Acute Phase

Bed Mobility

Rolling to the Affected Side. Rolling to the affected side promotes early active trunk control and may increase awareness of the weaker side.

Rolling to the Unaffected Side. Rolling to the unaffected side promotes awareness and initial management of the weak upper extremity by teaching the patient to passively guide the arm across the trunk (Fig. 1-18).

Maintaining Side Lying. A rolled pillow placed at the midthoracic spine to the lumbar area may assist the patient in maintaining the side-lying position. A towel roll can be placed under the patient's waist to provide a stretch to the shortened trunk. A primary goal is to assure proper spine alignment, to avoid pressure build up over the bony prominences in the lower extremities (knees and ankles), and to position the scapula in protraction if the patient is positioned on the weakened side.

Bridging. Bridging strengthens the back and hip extensors. From a functional perspective, this movement

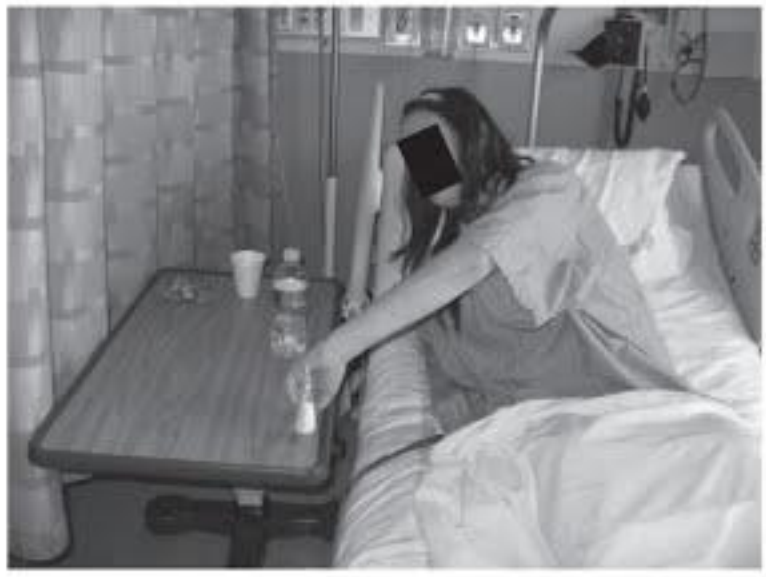

Figure 1-18 Bed level activities. Rolling to the unaffected side and engaging the affected arm in early reaching task and at the same time engaging affected trunk and lower extremity muscles.

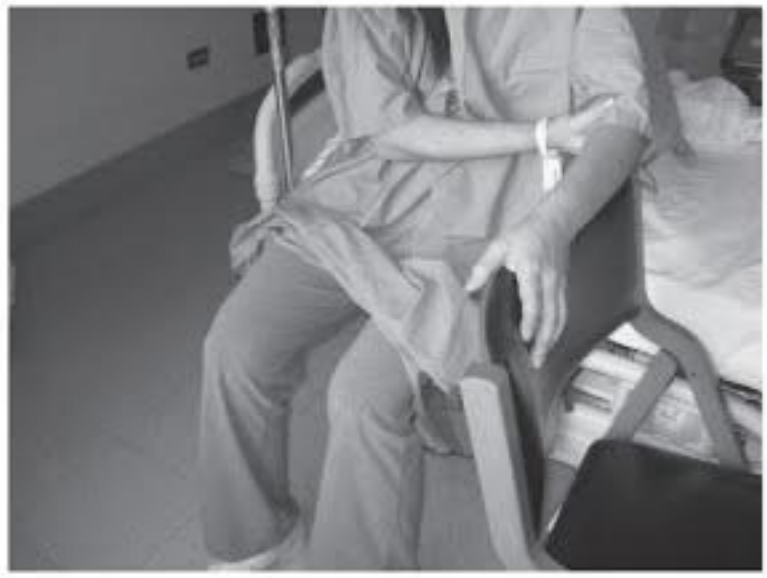

Figure 1-19 While the patient sits on the edge of the bed, a bed side chair is used to facilitate upper extremity weight-bearing activities.

aids in getting on and off the bed pan, can be used during lower body dressing, and also assists moving the lower body toward the side of the bed in anticipation of assuming a sitting position.

Side Lying to Sitting toward the Affected Side. Side lying to sitting toward the affected side promotes early stage weight-bearing on the weak upper extremity. The therapist needs to ensure that the shoulder is properly aligned, and the patient will usually require assistance with initiation of the movement.

Side Lying to Sitting toward the Unaffected Side. Therapists need to be mindful that the involved shoulder remains in a forward position during the motion of side lying to sitting toward the unaffected side.

Weight-Bearing for Function

Upper extremity weight-bearing activities may be done while the patient is side lying as mentioned previously, during bed mobility, or for stabilizing items. It can also be accomplished using the bed side table during meals or grooming tasks. The arm or back rest of a chair can be incorporated in the treatment plan for positioning and setup for weight-bearing (Figs. 1-19 and 1-20). The patient should be taught to push off with both upper extremities when moving from sit to stand. Weight-bearing as a postural support can reverse or prevent tissue shortening of the elbow, wrist, and finger flexors. It can also be used to strengthen the scapula musculature and the triceps. Arm extended weight-bearing can be done in front of the sink during grooming or be done in front of the bed side table while reaching for items nearby (Fig. 1-21).

For the lower extremity, bed level activities include: bridging, sitting at the edge of the bed with both feet on the floor, and early transfer training once patients are medically stable.

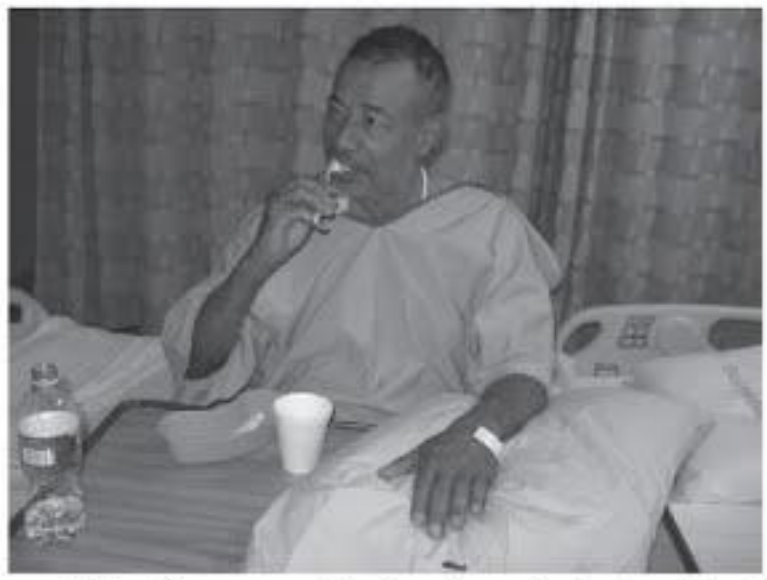

Figure 1-20 Forearm weight-bearing on bed side table while patient dangles off edge of bed.

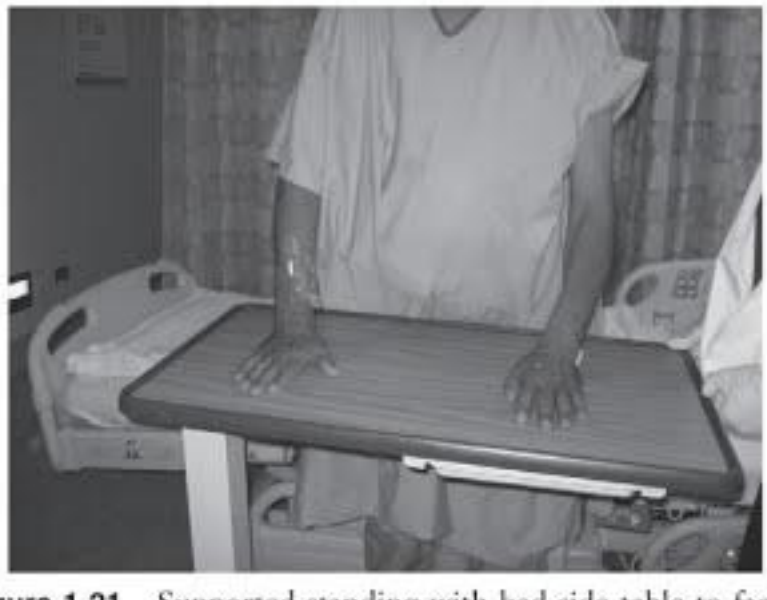

Figure 1-21 Supported standing with bed side table to facilitate upper extremity involvement in activity. Early upright ADL training can be initiated, and weight shifting through the lower extremities is encouraged.

Graded Sitting and Standing Activities

Supported Sitting in Bed. For the supported sitting in bed position, the head of the bed should gradually be raised in approximately 30- to 40-degree increments to avoid an orthostatic hypotensive response. As the patient tolerates the change in degrees of elevation, the therapist should continue to monitor vital signs. If there appears to be no change in the patient's blood pressure, the therapist should continue to elevate the head of the bed to approximately 80 degrees. Sitting at a slightly reclined position is less taxing on the patient's energy and requires less recruitment of the neck, trunk, and back musculature to maintain an upright position. At this point, the patient should be engaged in functional activities, such as feeding, light grooming, upper body bathing and dressing, and leisure activities.

Supported Sitting in a Chair. If the patient is well-supported and can endure sitting in a chair at the bed side, "sitting tolerance" or "out of bed tolerance" can be increased. Pillows may be useful at this early stage to support the lumbar spine and weaker upper extremity. When a therapist is placing a pillow under the upper extremity, he or she should make sure the shoulder alignment is in neutral. Adequate postural support may reduce pain and fatigue. Focus of treatment can include but is not limited to the patient performing self-care tasks, visual scanning activities, and weight-bearing through the upper and lower extremity.

Unsupported Sitting. Unsupported sitting may be done in the bed in a "tailor" (crossed legged) position, depending on the amount of ROM the patient has in the lower extremities. The head of the bed can be elevated, but should not touch the back of the patient. It is used as a safety catch should the patient lose his or her balance in a posterior direction. Pillows may be propped against the bed rails to protect the patient if he or she leans or falls laterally to the weaker side. While seated in this position, the patient can practice righting himself or herself or maintaining a midline position, and the patient should then be engaged in functional activities as tolerated.

Unsupported Sitting at the Edge of the Bed with Feet Dangling. In this position, the patient can be challenged with increased demands on alignment, trunk control, and forward and lateral weight shifts. Scooting to the edge of the bed can be introduced in anticipation of progressing to sit to stand. Postural control may be noticeably improved once the patient's feet contact the floor. The therapist should ensure equal weight-bearing on both lower extremities. See Chapter 7.

Sit to Stand: Pretransfer Phase. To prepare for the sit-to-stand pretransfer phase, therapists should ensure that all lines and IVs have enough length to eliminate pulling or tension. Increasing the surface height the patient rises from will require less work. This transition may require the assistance of more than one person to gain the patient's confidence and safety. The therapist should assure appropriate alignment of both lower extremities with feet placed firmly on the floor and then have the patient begin with several partial sit-to-stand trials. Assess how the weaker lower extremity reacts to weight-bearing, provide appropriate blocking or support to prevent collapse, and check vital signs while the patient is upright.

Supported Standing in Front of a Raised Bed. To initiate supported standing in front of a raised bed, the therapist should position the patient in a chair that faces the side of the bed. With appropriate assistance, the therapist should stand the patient and sit in a chair on the patient's weakened side to support the hip and knee extensors. In this standing position, the patient may practice early weight shifting through the lower extremities and bear weight on the upper extremities in either forearm or arm extended positions (see Fig. 1-21).

EDEMA MANAGEMENT

Evaluate the potential cause if edema is present. Discuss with nursing whether the swelling may be associated with the presence of a blood clot or an IV infiltrate. Check to see if the patient's limb is cool or warm to the touch, observe the skin color, and assess the firmness of the swelling (soft, fluidlike, or pitting).

In the ICU, the preferred method for treating edema is positional elevation, as compression garments or ace wraps may not be appropriate due to various IVs and line access needed by nursing. The extended limb should be positioned above the heart. Active or active assistive ROM should be encouraged and followed by manual massage (Fig. 1-22). See Chapter 12.

SHOULDER MANAGEMENT

Many patients may experience upper extremity edema, pain, humeral head subluxation, and/or impingement after a stroke. Many of the upper extremity interventions provided in the ICU/acute stage are prophylactic measures to prevent these problems.

To protect the shoulder against potential pain and subluxation, the team should be educated in proper rolling techniques and bed mobility, so they can avoid pulling on the extremity. The team should be instructed to roll the patient by placing the hands on the trunk rather than pulling on the extremity. Signage can be hung behind the patient's bed indicating the patient may have shoulder subluxation and informing the team to not pull on the patient's arm (Box 1-6).

Due to the medical complexity of the ICU/acute patient, most are not getting out of bed to the chair for prolonged periods or engaging in prolonged upright activities. While supine, out of bed in a chair, or dangling at

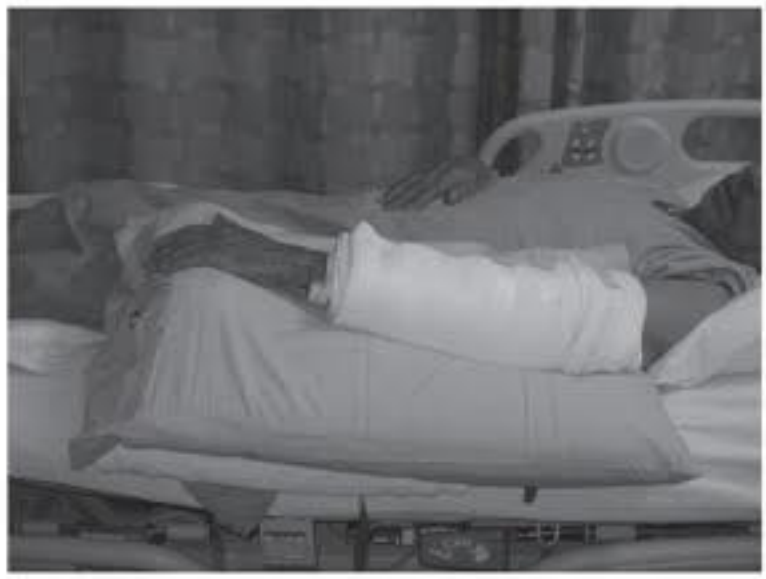

Figure 1-22 Patient's affected upper extremity positioned in towel roll and elevated on pillow to prevent and decrease edema.

Box 1-6

Patient with Right Shoulder Subluxation

Please do not pull on patient's arm. Please contact occupational therapy at 555–8724 with questions or concerns.

the bed side, support for a weak shoulder can be provided via proper positioning.

Supine

Provide support to the affected upper extremity with pillows and/or towels. The occupational therapist must use clinical judgment to determine proper positioning for each patient. However, as a general rule, the affected scapula should be protracted, the arm in a forward position, with the wrist neutral and fingers extended.[26]

Edge of Bed

The affected upper extremity is supported on the bed side table or on numerous pillows.

Out of Bed in a Chair

The affected upper extremity is supported on the bed side table, on numerous pillows, or on the arm support of the chair.

Most ICU/acute patients do not require supplemental shoulder supports such as sling, clavicle strap, and/or taping. These supports may be used once patients are performing ADL upright and are spending more time out of bed. See Chapter 10.

In addition to positioning, the occupational therapist will provide the ICU patient with passive and active ROM and will engage the affected upper extremity in functional tasks. The therapist should mind lines and leads while providing these services. When an A-line is present in the radial artery, wrist flexion/extension should be avoided.

INCREASING SPATIAL AWARENESS BY ARRANGING THE ENVIRONMENT

Although the ICU environment may be more restrictive than a rehabilitation setting, there are subtle yet important interventions that can be implemented to increase spatial awareness. Strategically place items of common use, such as the television remote control, on the involved side while providing cues to assist the patient in locating them. Strategically place food items on the meal tray during feeding to encourage scanning and locating desired items to eat. Verbal cues should be diminished as the patient's awareness increases. Reverse the position of the bed, if able, so that the patient's involved space is stimulated (e.g., facing the hallway instead of facing a blank wall). Position the bed side table and phone on the neglected or weaker side of the patient. Use brightly colored bands tied to the bed side rails on the involved side as cues to attend to this side. Hang pictures of family and friends on the involved side while providing cues for the patient to locate them.

EARLY COGNITIVE MANAGEMENT

Patients may spend numerous days to weeks in the ICU. A well-known phenomenon called ICU psychosis can develop within days of being admitted to the ICU.[55,99] ICU psychosis has been defined as a fluctuating state of consciousness characterized by fatigue, distraction, confusion, disorientation, restlessness, clouding of consciousness, incoherence, fear, anxiety, excitement, hallucinations, and delusions.[41] Many factors related to the ICU environment can contribute to the development of ICU psychosis. Some include psychosocial stress, sleep deprivation, sensory overload or underload, and immobilization.[41] Many patients are unable to differentiate between day and night secondary to lighting in most ICU.[41]

The occupational therapist can assist the primary nursing team in a variety of ways to help lessen the effects of ICU psychosis. Some measures that nursing may implement are providing tactile and verbal stimulation, involvement of the patient in his or her care, and supplying effective rest periods.[99] The occupational therapist can minimize environmental monotony and mobilize and engage the patient in familiar self-care tasks. When providing a patient with OT services, communication with patient via gentle touch and voices can help calm patients. Incorporating music and massage into OT treatments can also help reduce anxiety, fear, and depression.[99] See Box 1-7 for treatment ideas.

SKIN PROTECTION AND PREVENTION OF BREAKDOWN

Skin breakdown and development of pressure ulcers are common complications associated with an ICU/acute admission. After stroke, patients are at risk for developing

Box 1-7

Treatment Ideas to Manage ICU Psychosis

- Mobilize and engage in self-care.
- Engage patient in time appropriate tasks (if it is 8 AM complete oral care with window shades open and lights on).
- Use a calm gentle voice and touch when engaging patients.
- Decrease or increase sensory stimulation during OT treatment session depending on patient's needs.
- Educate patient's family in orientating patient not only to date and place but also to time of day.
- Keep clocks and calendars in view.

pressure ulcers due to prolonged bed rest and immobility. Other risk factors include poor circulation, poor nutrition, edema, low level of arousal, confusion, and incontinence.[8] Pressure management and skin protection should become a part of each treatment session. See Table 1-12 for a review of the stages of pressure ulcers.

Prevention of skin breakdown is a team responsibility. The occupational therapist has a unique set of skills to assist the team in protecting the patient's skin. The occupational therapist is often the first team member to mobilize patient and can observe the entire body for signs of skin breakdown. Areas of concern for the ICU patient include sacrum, occiput, heels, greater trochanter, and elbows. Therapist can suggest elbow and heel pads to protect these areas from pressure and friction. Heels can also be floated via positioning or multipodis boots (Fig. 1-23). The therapist can develop positioning devices to assist the nurse with elevating pressure on the occiput (Fig. 1-24) and the sacrum. The occupational therapist can also recommend specialized mattresses to best serve the patient's needs.

COMMUNICATION

For the patient unable to communicate verbally, whether due to mechanical ventilation or aphasia, alternative methods of communication will be necessary. Options may include use of a communication board. Single word choice or pictures that represent feelings or needs can be placed strategically on a small poster board. Examples may include Nurse, Doctor, Pain, Thirst, etc., to which the patient can then point. Alphabet boards are generally not used, as they require energy and time for the patient to "spell" words. For the aphasic patient, words might be eliminated altogether. Other alternatives may include signals for Yes/No questions, such as head nodding or thumbs up or down, and an eye blink system. Working in conjunction with the speech-language pathologist, the occupational therapist may assist with facilitating a communication system that is consistently used by other staff and family members (Box 1-8; see Chapter 20).

DYSPHAGIA SCREENING

Acute swallowing difficulties or dysphagia are often associated with stroke.[159] The risk of aspiration is high and often leads to pneumonia. Other medical complications associated with dysphagia include malnutrition and dehydration.

During the initial admission to the hospital, patients may be placed on "NPO" (nothing by mouth) precautions. Under these circumstances an NGT is usually inserted through the nose and down the esophagus to the stomach. If the patient is conscious, the occupational therapist may initiate a swallowing or dysphagia screening at the bed side.

Before beginning the assessment, the therapist should be aware of the patient's level of alertness, fatigue, and ability to follow commands, as these factors may significantly influence the ability to participate safely. An oral motor examination should precede administration of foods and liquids. The assessment should begin with the patient seated with the head of the bed elevated. If an oral suction device is available at the bed side, it should be turned on (Box 1-9; Fig. 1-25).

Based on the results of the bed side assessment, instrumental testing may be necessary to further evaluate the phases of swallowing that cannot be seen at a bed side oral motor examination. If the patient appears to have adequate oral and swallowing function and a physician's order has been obtained, a feeding trial may be initiated using graded food textures and liquids of various thickness (Box 1-10; see Chapter 24).

SELF-CARE TRAINING

Training in ADL is an integral part of OT treatment. It is important to engage the patient in self-care tasks as soon as they are medically stable.

Energy expenditure is often an issue for the low level patient, so grading the self-care task is as important as the choice of activity. The acute patient may also be limited by IVs, lines, and artificial ventilation. If the patient is having difficulty managing secretions, begin by teaching them how to use an oral suctioning device. Using an adapted call light to request assistance from nursing is also an appropriate goal.

For those with limited motor return, the upper extremity should at least be used as a stabilizer. ADL compensatory strategies can be initiated. If the patient demonstrates active movement, the upper extremity should be incorporated into the self-care task (see Chapter 28).

Table 1-12

Pressure Ulcer Stages

STAGE	DESCRIPTION	ADDITIONAL INFORMATION
Stage I	Intact skin with nonblanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.	The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Stage I may be difficult to detect in individuals with dark skin tones. May indicate "at risk" persons (a heralding sign of risk).
Stage II	Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.	Presents as a shiny or dry shallow ulcer without slough or bruising. This stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation. Bruising indicates suspected deep tissue injury.
Stage III	Full thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.	The depth of a stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep stage III pressure ulcers. Bone/tendon is not visible or directly palpable.
Stage IV	Full thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.	The depth of a stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule), making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.
Unstageable	Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed.	Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as "the body's natural (biological) cover" and should not be removed.

Courtesy of National Pressure Ulcer Advisory Panel

Figure 1-23 This technique is termed "floating the patient heels." It is used while supine in bed to maintain skin integrity and to prevent breakdown.

Figure 1-24 Cervical roll used to keep occiput off the bed to decrease pressure that may cause breakdown. The roll allows for head/neck rotation in both directions.

Box 1-8

Communication Keypoints

- Use a normal tone and volume of voice. Avoid shouting at the patient or talking to them in an infantile manner.
- Give the patient enough time to respond to the question.
- Try to stay on the same subject.
- Gesture whenever possible and provide tactile cues as appropriate.
- Speak slowly and directly to the patient's face.
- Simplify questions to Yes/No
- Try to reduce background noise to eliminate distraction. Close the door and turn off the radio or television.
- Only one person should communicate with the patient at one time.
- Be aware of signs of frustration by observing facial expressions.

Box 1-9

Oral Motor Screening

- Observe for the presence of facial asymmetry. Facial drooping or weakness is common in association with the weaker extremities. Foods can pocket in the cheek of the weakened side.
- Observe mouth and lip closure. Can the patient purse his or her lips? Have him or her attempt to blow air into his or her cheeks while keeping his or her lips pursed. Observe if air escapes through one side of the mouth.
- Request the patient to stick out his or her tongue. Does it drift or deviate to one side? Can he or she lick his or her lips and perform lateral movements with the tongue?
- Use a long stick swab to assess the patient's sensation both extra- and intra-orally.
- Use a tongue depressor to assess the patient's gag reflex. Is it present, absent, or delayed?
- Check the soft palate. Use a flashlight to ask the patient to open mouth and say the word "AH." Observe for soft palate elevation.
- Assess the patient's vocal quality. Is it gurgly or wet? Can the patient "clear" his or her voice? Secretions may pool or linger around the vocal cords. Is there hoarseness of the voice? If so, it may be due to inadequate closure of the vocal cords.
- Can the patient demonstrate a volitional cough? Assess the strength of the cough. Is it adequate to clear the airway?
- Is the patient managing his or her own secretions? Does he or she choke or cough on his or her own secretions? Observe whether the swallow is present or delayed.
- A standardized bed side swallowing assessment is recommended (Fig. 1-25).

The initial position may be with the head of the bed elevated. This position provides support of the head and trunk. Vital signs should be monitored throughout the activity. As patients progress, they might be positioned in sitting at the edge of the bed. Demands are greater as patients must maintain their balance while performing the task. Once a patient is able to tolerate sitting at the edge of the bed, the progression should lead to performing tasks seated in a chair. If the patient is able to stand for short periods, then appropriate self-care activities should be performed in standing, such as brushing teeth at the sink or combing hair. Chaining the tasks together will demand more tolerance. Self-care tasks can be graded from simple to complex (Box 1-11).

FAMILY TRAINING

The primary purpose of family training in the ICU/acute setting is to allow for the patient to engage in as many therapeutic activities as possible immediately following the neurological event. Family members should be empowered to assist their loved ones to achieve their therapy goals. Occupational therapists may spend as much time educating the family as they do treating the patients. When training family members, the therapist should demonstrate the tasks and then provide an opportunity for the family member to attempt the tasks. Positive feedback should be provided with corrections given as needed. Families should be provided with written instructions for any tasks they are asked to carry out. During one OT session, no more than three tasks should be given to the family members. This will ensure greater carryover of the tasks provided. The following are suggestions for a family training scheduled in the ICU/acute setting.

Occupational therapists must use their clinical reasoning when providing family training. Many ICU/acute care patients are too medically complex for the family to provide additional therapy services. Such patients may require constant monitoring during physical activity, while other patients may have lines and leads that require a nurse or therapist to handle.

After evaluation patients, family members should be instructed in the following.

- Safely moving noncomplex lines and leads. These may be noninvasive a blood pressure cuff, an O_2 monitor, an IV, and, in certain cases, A-lines.
- Positioning of affected extremities
- Splint wearing schedule, donning and doffing the splint, and performing skin checks
- ROM for elbow, wrist, and hand
- Setting up environment for patient during ADL tasks supine and interacting with patient on affected side (in the case of neglect or sensory loss)

As treatment progresses, the family can be further engaged in the treatment and trained in the following areas:

- Shoulder management: Families must be educated in positioning of the involved upper extremity in bed,

GUSS
(GUGGING SWALLOWING SCREEN)

Name: _____
Date: _____
Time: _____

1. Preliminary Investigation/Indirect Swallowing Test

	YES	NO
Vigilance *(The patient must be alert for at least 15 minutes)*	1 ☐	0 ☐
Cough and/or throat clearing *(voluntary cough)* *(Patient should cough or clear his or her throat twice)*	1 ☐	0 ☐
Saliva Swallow: • Swallowing successful	1 ☐	0 ☐
• Drooling	0 ☐	1 ☐
• Voice change (hoarse, gurgly, coated, weak)	0 ☐	1 ☐
SUM:	(5)	
	1–4 = Investigate further[1] 5 = Continue with part 2	

2. Direct Swallowing Test (Material: Aqua bi, flat teaspoon, food thickener, bread)

In the following order:	1→	2→	3→
	SEMISOLID*	**LIQUID****	**SOLID*****
DEGLUTITION:			
• Swallowing not possible	0 ☐	0 ☐	0 ☐
• Swallowing delayed (> 2 sec) (Solid textures > 10 sec)	1 ☐	1 ☐	1 ☐
• Swallowing successful	2 ☐	2 ☐	2 ☐
COUGH (involuntary): *(before, during, or after swallowing – until 3 minutes later)*			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
DROOLING:			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
VOICE CHANGE: *(listen to the voice before and after swallowing – Patient should speak "O")*			
• Yes	0 ☐	0 ☐	0 ☐
• No	1 ☐	1 ☐	1 ☐
SUM:	(5)	(5)	(5)
	1–4 = Investigate further[1] 5 = Continue liquid	1–4 = Investigate further[1] 5 = Continue solid	1–4 = Investigate further[1] 5 = Normal
SUM: (Indirect Swallowing Test AND Direct Swallowing Test)			____ (20)

*	First administer 1/3 up to a half teaspoon Aqua bi with food thickener (pudding-like consistency). If there are no symptoms apply 2-5 teaspoons. Assess after the 5th spoonful.
**	3, 5, 10, 20 ml Aqua bi – if there are no symptoms continue with 50 ml Aqua bi (Daniels et al., 2000; Gottlieb et al., 1996) Assess and stop the investigation when one of the criteria is observed.
***	Clinical; dry bread; FEES; dry bread which is dipped in colored liquid
1	Use functional investigations such as Videofluoroscopic Evaluation of Swallowing (VFES), Fiberoptic Endoscopic Evaluation of Swallowing (FEES)

Figure 1-25 The Gugging Swallowing Screen. (From Trapl M, Enderle P, Nowotny M, et al: *Stroke* 38 (11):2948–2952, 2007.)

GUSS
(Gugging Swallowing Screen)
Guss – EVALUATION

RESULTS		SEVERITY CODE	RECOMMENDATIONS
20	Semisolid/ liquid and solid texture successful	Slight/no dysphagia minimal risk of aspiration	• Normal diet • Regular liquids (First time under supervision of the SLT or a trained stroke nurse!)
15–19	Semisolid and liquid texture successful and solid unsuccessful	Slight dysphagia with a low risk of aspiration	• Dysphagia diet (pureed and soft food) • Liquids very slowly – one sip at a time • Functional swallowing assessments such as Fiberoptic Endoscopic Evaluation of Swallowing (FEES) or Videofluoroscopic Evaluation of Swallowing (VFES) • Refer to Speech and Language Therapist (SLT)
10–14	Semisolid swallow successful and liquids unsuccessful	Moderate dysphagia with a risk of aspiration	Dysphagia diet beginning with • Semisolid textures such as baby food and additional parenteral feeding. • All liquids must be thickened! • Pills must be crushed and mixed with thick liquid. • No liquid medication! • Further functional swallowing assessments (FEES, VFES) • Refer to Speech and Language Therapist (SLT) *Supplementation with nasogastric tube or parenteral*
0–9	Preliminary investigation unsuccessful or semisolid swallow unsuccessful	Severe dysphagia with a high risk of aspiration	• NPO (non per os = nothing by mouth) • Further functional swallowing assessment (FEES, VFES) • Refer to Speech and Language Therapist (SLT) *Supplementation with nasogastric tube or parenteral*

Figure 1-25, cont'd

Box 1-10

Symptoms of Potential Dysphagia

- Facial weakness
- Weak tongue movements
- Poor lip closure
- Drooling
- Coughing on secretions
- Poor or wet voice quality
- Residual food accumulation in mouth

during bed mobility, for transfers, during ADL activities, and while upright. Family members can be instructed to don and doff shoulder supports if needed.

- ADL training: Family members can be trained in setting up the environment using the bed side table, giving simple verbal cues, and providing physical cues to engage the affected upper extremity. If the patient is to go home directly from the acute care setting, family training of both compensatory and remedial techniques for ADL trainings should be initiated.

Box 1-11

Grading ADL during Acute Stroke Rehabilitation

SIMPLE	COMPLEX
Sitting with back supported	Sitting with back unsupported
Finger feeding	Feeding with utensils
Drinking from a cup	Pouring liquids and drinking with a straw
Brushing teeth with set-up	Brushing and cleaning dentures
Washing face with cloth	Washing face and upper body
Donning pullover shirt	Donning a button-down shirt
Donning shorts in bed with bridging	Donning pants while standing to pull up

CASE STUDY 1

Ischemic Stroke: Management of Acute Case and Complications with Workup—cont'd

time of her discharge, she is able to move short distances with a hemiwalker and needs assistance with dressing her lower extremities and setting up for her basic ADL.

G.H.'s one-year follow-up is notable for the continuing intractable painful spasticity in her left arm, so treatment with Botox is instituted and results in adequate pain relief. She remains stable until five years after her stroke when she suffers a fall with a subsequent hip fracture. Evaluation of bone density shows accelerated osteoporosis in the left hip. She needs left hip hemiarthroplasty but is unable to regain her previous level of function, despite aggressive therapy, and finally has to be admitted to a nursing home when discharged from the hospital.

CASE STUDY 2

Hemorrhagic Stroke: Management of Acute Case with Workup

C.C. is a 25-year-old man who works as a sales manager in a local retail store. While dismissing a store clerk whom he caught stealing from the store safe, he suddenly complains of a severe headache, sinks to the chair in his office, and slumps over to the right. Within a few minutes, he is unconscious, and the staff calls the ambulance. C.C. is admitted to the emergency room within 20 minutes, accompanied by the fired clerk who is proclaiming loudly that she has done nothing to him. In the emergency room, C.C. is in a deep coma, breathing deeply, and has dilated pupils and absent reflexes. He is intubated immediately for airway protection and is taken for an emergency CT scan. The study is not completed because C.C. has a seizure while in the CT scanner, but the partially completed study shows a great deal of blood in the ventricles. C.C. is diagnosed with a presumed SAH, and treatment is started. Hyperventilation and treatment with mannitol begin. An intracranial pressure monitor is inserted, and C.C. is given phenytoin and nimodipine. C.C. is managed closely in the ICU and after three days comes out of the coma. He remains intubated and has an MRI/MRA performed that shows a probable berry aneurysm on the anterior communicating artery.

A cerebral angiogram is performed, and a 2-cm aneurysm is clearly visible. C.C. has a good response to the treatment and is extubated on the sixth hospital day. His neurological examination reveals mild disorientation, dysarthria, and tetraparesis more pronounced on the right than the left.

The neurological and neurosurgical team, patient, and family have a discussion and decide that surgical clipping of the aneurysm is the best approach to treating the lesion. C.C. is scheduled for operative intervention the next day. However, in the middle of the night, he suddenly loses consciousness and stops breathing. He has a cardiac arrest but is resuscitated successfully. An emergency CT scan reveals a large recurrent hemorrhage that extends into the cerebral cortex and a herniated brainstem. Aggressive treatments are instituted, but despite all measures the herniation progresses, and C.C. lapses into an irreversible coma. One week later C.C. is declared brain dead, and according to his family's wishes, his organs are donated for transplantation.

REVIEW QUESTIONS

1. Which stroke risk factors are considered modifiable?
2. Which procedures are used to diagnose a stroke?
3. Which clinical signs indicate a patient is receiving excessive seizure medication?
4. What are the risk factors and recommended treatments for DVTs?
5. Other than neurological, what are the common complications that follow a stroke?

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To have a successful transition, there are stages in which the survivor and the family go through. One model suggests stages that include denial, which protect one from initial overwhelming emotion; grieving (as distinguished from depression), in which one mourns the loss of function; role transition, to include "care-receiver"; the development of optimal independence, which includes compensatory techniques and adjustment to a new body; rebuilding a social support system; and reintegration into the community via instrumental ADL.[11] It has also been suggested that there are three domains in recovery: physical, psychological, and social. Important to these domains is self-worth, which is related to participation and to quality of life. While stroke has great impact on cognition and physical function, it is also critical to address self-image and sense of being (psychological domain), and changes in relationships (social domain). As family members also change roles due to stroke, it is important to promote a positive self-concept and positive social support; both will have an impact on function.[119] The goal with each of these models is toward acceptance of any remaining disability and the return to a satisfying quality of life. See Chapter 3. Emotional reaction following stroke has significant implications for recovery. Feelings of helplessness or hopelessness affect survival rate,[63] apathy affects functional ability,[47] and depression and anxiety affect function and recovery.[2,14-16,45,50]

One's cultural background also may play a role in how one copes with illness, disability, and rehabilitation. As stated earlier, cultural values and attitudes may devalue any form of dependency. Consequently, a disability may add to feelings of alienation. From a cultural perspective, psychological conditions also may be viewed as a weakness of character. This further stigmatizes the individual and leads to the avoidance of acknowledging feelings and of being treated.[70]

Health professionals, without intending to do so, may become enablers of the loss of personal identity and dignity and contribute to a diminished self-esteem. When an individual is referred to in terms of a disabling condition (e.g., "a right hemi"), one's dignity and sense of personal worth are challenged. This adds to what may be emerging as a damaged sense of self within the context of social stigma. Many individuals go to great lengths to conceal their disabilities from others to avoid being identified as having had a stroke.[90] Although much has been written regarding the negative emotional reaction to stroke, the suggestion also has been made that for individuals whose lives ordinarily are characterized by crises, dealing with the consequences of stroke is not considered an extraordinary event but just another life change.[89] Although this challenges the general assumption that anyone who has experienced a stroke also will experience grief, loss, and distress,[96] considering the context of one's life in which stroke occurs is important.[66]

PERSONALITY CHANGE FOLLOWING STROKE

While it has been noted that a change in personality may follow a stroke, and this may be related to lesion location,[8] the change is characterized as any of the following types: aggressive, disinhibition, paranoid, labile, and apathetic.[33] Although some of the symptoms may appear to be consistent with the signs and symptoms of specific psychiatric conditions, they often emerge as negative emotions or behaviors that do not meet the criteria for particular diagnoses. These can range from euphoria to uncontrollable tears, from worry to agitation, from disinterest to hostility, or from paranoia and guarded behavior to excessive dependency. Despite the behavioral expression of these emotions, they tend not to reflect an underlying mood and may add to the embarrassment experienced by the patient.[8] These behavioral changes are particularly difficult for the caregiver to manage, and they do not respond to medication.[33]

Apathy is a common change that occurs, with some studies suggesting between 20% to 40% of stroke survivors display some apathetic behavior.[33] Although apathy can be a symptom of depression, it can also be a separate construct, occurs more frequently than depression, and affects rehabilitation and recovery.[43] By its very nature, the impact of apathy on energy and motivation clearly effects engagement in the recovery and rehabilitative process.

DEPRESSION

Among the most significant considerations in understanding the characteristics and consequences of stroke is the relationship of depression to onset, recovery, and rehabilitation of persons with stroke. Because of the neurophysiological changes and because of the reaction to the consequences of stroke, depression has major implications for the course of recovery. Despite the causes of depression, assessment and treatment of depression affects psychological, functional, and medical health.

The relationship between cerebrovascular disease and depression has long been studied. Depression is both a risk factor for stroke[33,55] and a major consequence of stroke.[126] For nearly three quarters of a century, the assumption held that depression following a stroke was related only to the functional and social consequences of the disability and not to the neurological damage of the stroke itself. Three decades ago, however, a study compared depression in individuals with stroke to individuals with orthopedic conditions, with both groups matched for functional ability. The significant increase of depression in the group with stroke led the researchers to believe that depression was related to something more than a reaction to functional inability.[35] More recent studies show that depression in stroke can occur at any time; during the

area to counter an inability of another area; rationalization, which provides reasons or excuses for not being able to accomplish tasks or goals; and diversion of feelings, in which unacceptable feelings are altered into socially appropriate behaviors.[31] How defenses are used also can give rise to how one is viewed by the treating therapist. The therapist may misinterpret behavior guided by maladaptive defense mechanisms and label the individual as a difficult patient.[78]

As the chronicity of the disability becomes apparent, the individual and one's social network must deal with the long-term effects of the stroke. Most immediate is the perceived change in oneself. Because role, lifestyle, and where one is in one's life cycle affect one's emotional reaction, trauma brings forth changes in what one can do and in how one sees oneself. Although time may enable one to develop the adaptive defenses necessary to deal with the anxiety surrounding illness, disability, and the unknown, one's psychological adaptation may be undermined if the symptoms are not alleviated. The resultant reaction to stress is often a universal loss of self-esteem followed by depression. Maladaptive uses of defenses may then ensue.[111]

Psychological adjustment to illness and disability also depends on personality constructs; consequently, individuals who have had strokes need to be understood from the perspective of their character traits, their cultural background, and the psychological consequences that are reactionary and physiologically based. Some evidence exists that personality characteristics play a role in the development of stroke, in the recovery from stroke, and in how one participates in treatment.

Almost a half a century ago, it was suggested that personality constructs characterize how one copes with illness and engages in treatment, and that health care professionals should understand and adapt their interactive styles based on the patient's character.[49] One approach to understanding personality is by using the classification system typical of those with personality disorders, such as the dependent and overdemanding personality, the controlling personality, or the dramatic personality. How individuals use those characteristics to cope with the stress and anxiety associated with illness can assist the therapist implement treatment.[78] For example, patients with compulsive personalities who ask for details and facts will benefit when the therapist provides adequate information to calm any anxiety, and when the therapist encourages the patient to take charge of certain aspects of treatment.[38] A second approach to understanding personality is based on coping styles used in stressful situations. This approach allows one to shape the rehabilitation process so that it reflects the patients' coping style.[19,96] A third approach is to identify whether an individual has certain emotional characteristics, characteristics which are thought to reflect positive rehabilitation outcomes: ability for reality testing, ability to self-reflect, and ability to acknowledge and grieve for loss.[9] Individuals who have sustained a significant physical illness or injury are struggling with emotional crises and revert to using characteristics from past situations.[38] Understanding personality and its role in coping is critical for rehabilitation, for different styles promote functional adjustment and improved quality of life.[27]

Culture is a major determinant of one's beliefs and attitudes, plays a major role in how one perceives illness and disability, and may influence how one interacts with health care providers. The meaning one ascribes to illness and how one behaves toward illness may be a function of personal and cultural health traditions. Assuming the sick role, which demands that one adjust to the role of patient and then relinquish that role to resume independence, may be determined culturally. For some, one's cultural background may promote motivation toward rehabilitation and recovery; for others, it might obstruct progress. Culture dictates how one interacts in any social organization (a clinic or hospital is a social organization); how and when one communicates; how one deals with personal space, particularly as others intrude on it; and how one considers future goals.[108] Cultural habits may influence how one expresses oneself and, if one is reserved, may be misperceived as one being unmotivated, guarded, or disrespectful.[70] Like personality traits, one's cultural habits may be expressed as a means to deal with stressful situations.

It may seem logical for an individual who has suffered a stroke to be open with health care providers with one's feelings, goals, and concerns. In patient-centered practice, health care professionals expect to rely on patients to inform and instruct them as they evaluate and plan treatment for optimal occupational performance. However, some cultures prefer the health care provider to assume somewhat of an authoritarian role,[57] others may express respect through the avoidance of eye contact yet expect the health care provider to be solicitous in recognition of social worthiness,[37] and others may appear mistrustful and uncommunicative.[70]

Having a disability that challenges one's independence is particularly difficult for those individuals for whom independence, control, and individuality are important values.[70] Indeed, these attributes eventually may motivate one in the rehabilitative process but initially make it more difficult to deal with a trauma that robs one of these values. In addition, culture often prescribes the roles one assumes in a social or family structure. For these individuals, coping with role change becomes even more challenging.

The psychological conditions so prevalent following stroke are particularly difficult for individuals to deal with if their cultural heritage is intolerant of psychological conditions. Although some cultural groups rely on verbal expression and take pride in expressing their feelings,

OCCUPATIONAL THERAPY PRACTICE

Throughout this chapter, reference has been made to the effect of psychological conditions and psychiatric disorders on recovery and rehabilitation. Personality traits[80] and levels of stress,[69,94] have been linked with mortality rates from stroke, as have severe forms of depression.[30] Personality traits related to self-esteem and coping style have been linked with ability to resume independence.[13,27] Participation in meaningful activities may be the best indicator of recovery.[5,19,22,28,46]

Depression and anxiety have perhaps the greatest impact on recovery and rehabilitation. Depression has been linked in general with recovery from stroke, with deficits in physical function,[30] and with deficits in impairment in daily living.[15,16,88] Even depressive symptoms without a clear diagnosis are linked to poorer functional status.[45] The presence of anxiety also reduces functional ability and diminishes social networks.[3]

Assessment and treatment of psychological conditions and psychiatric disorders is critical when working with individuals who have had a stroke and with their families. As reviewed elsewhere, studies have repeatedly demonstrated that medication is effective in the prevention[84] and treatment of these conditions[16] but should be coupled with psychological and social interventions.

In 2008, the American Occupational Therapy Association published its *Occupational Therapy Practice Framework*, 2nd edition.[103] Critical to the framework, which delineates the focus of practice and links evaluation and intervention with occupation, is the interdependency of performance in areas of occupation, skills, and patterns with context/environment, activity demands, and client factors. Key to the practice of occupational therapy is the understanding of how illness or disability affects occupation and how engagement in occupation depends on the interaction of physical, psychological, emotional, and social conditions.

When using the framework as a guide, one is compelled to evaluate all the patterns and skills necessary to engage in activity and occupation.[103] Ability to engage in everyday activities leads to participation in patient-selected contexts and results in satisfactory quality of life. Because quality of life is measured through physical, psychological, and social indicators,[63,120] the areas identified within this chapter require attention: personality traits; cultural attitudes and beliefs; psychological and cognitive consequences of stroke; emotional reactions to illness, disability, and recovery; and social context and support. This information has a direct bearing on the occupational profile developed, and it affects physical, psychological, and social functioning and the potential for independence.

The patient-centered focus of practice[103] is consistent with what should be the focus of evaluation and intervention. Patients measure success not by the therapist's standards but by their personal goals.[42] Indeed, the benchmarks that professionals use to determine functional ability is typically related to physical performance, whereas patients use quality of life measures.[10]

The Therapeutic Relationship

There is some evidence that psychosocial intervention may indeed prevent poststroke depression.[41] Given this, it is paramount to consider every interaction between the patient and therapist as a context for assessment and intervention.[96] The relationship that develops presents an ongoing opportunity to consider personal and social needs, to clarify and refine goals, and to address the ambient emotional conditions affecting progress. The relationship between therapist and patient may even predict positive functional outcome.[9] The therapeutic relationship begins the moment the patient and therapist interact. This may precede face-to-face contact, as each may have preconceived notions of what to expect. These notions may impede the therapeutic process if they lead to inaccurate or unrealistic assumptions, or they may facilitate the process if they promote the awareness of conditions and contexts that must be considered.

Fundamental to the relationship is respect, trust, concern for dignity, honesty, and the ability to be empathetic.[106] As the therapist and patient work to develop a collaboration that can result in optimal occupational performance, each needs to engage in the therapeutic process to provide meaning and value for the patient. Above all, this engagement is based on respecting the patient's individuality, making it possible for the patient to identify valued goals, and maintaining sensitivity for the fears, concerns, frustrations, and disappointments that emerge. A significant communicative tool in this relationship is empathy: the ability to convey an understanding of another's condition. Not to be confused with sympathy, pity, or identification, each of which can interfere with the therapeutic relationship,[20] empathy advances the helpful nature of the relationship. Conveying empathy, along with informing patients of the processes and rationale behind treatment, anticipating possible difficulties or obstacles, and soliciting social support from family or friends, improves cooperation and compliance in treatment.[107]

Evaluation

Evaluating the psychological conditions in an individual with stroke should be part of every therapist's assessment procedures. In addition to using specific measurement tools that target psychological and cognitive functions, the therapist should seek to answer a series of questions via interview of the patient and family and through observation. This process may be a challenge, particularly if speech, language, or visual spatial impairments are evident.

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are activities related to personal care (eating, grooming, and hygiene), getting around (mobility), and communicating. For a person to be self-reliant in any community, a level of competence is required that enables the accomplishment of tasks beyond those of basic self-care (which are referred to as physical self-maintenance). For this reason, M. Powell Lawton identified the use of the telephone, food preparation, housekeeping, laundry, shopping, money management, driving or use of transportation, and medication management as important daily activities and proposed the term *instrumental activities of daily living* (ADL) to describe them[35] (see Chapters 14, 21, 22, 23, and 28).

Often when persons are hospitalized, the focus is on achieving independence in self-care. Christiansen suggested that self-care tasks must be viewed as necessary from a societal point of view.[14] Although eating and hygiene tasks are essential for survival and health, dressing and grooming are important to social interaction and participation. Some expect persons to care for themselves. Sometimes therapists go too far in expecting an individual to perform self-care; some individuals prefer to spend their time in other occupations and accept the help of others to do basic self-care. Therapists are familiar with the use of personal attendants with persons following spinal cord injuries; persons who have had a stroke benefit from a personal attendant, so that they have choice in how they spend their time in occupations more important and meaningful to them.

A discussion of occupation cannot be complete without a discussion of self-efficacy and self-determination. Bandura used the term *self-efficacy* to describe the extent to which successes or failures influence expectations of future success or failure.[3] The experience of success in doing things (occupations) contributes to a positive sense of oneself as effective or competent. In contrast, a negative view of self and one's ability to influence events can lead to perceptions of helplessness. Gage and Polatajko observed that perceived self-efficacy has been shown to influence perseverance and well-being and that it can be modified through successful experiences.[21]

According to self-determination theory, intrinsic sources of motivation lead persons to encounter new challenges.[45] An important (and logical) part of this theory is the claim that settings in which persons experience success helps them feel good about themselves. This enables persons to face their daily challenges more readily and in the process to develop an understanding of who they are and their place in the world.

Following stroke, many individuals are not able to engage in their occupations as they have in the past. Therapy must create the environment for learning that fosters a person's view of self so that successful experiences can be experienced and sustained. Opportunities for success must be fostered as well, so that these persons are motivated to face their daily challenges.

Occupation is a concept that must be understood in terms of planning and describing the activities of an individual; it provides an important process that can and should be used in the rehabilitation program to improve a person's recovery. Box 3-1 highlights key statements that identify the importance of occupation; these can be translated directly into outcomes that practitioners can address today as they plan client-centered care.

CLIENT-CENTERED CARE

Clients who have had strokes need support to return to their lives as they lived them before the stroke. They require services that help them build endurance, increase movement and strength, increase awareness, obtain assistive devices such as wheelchairs and self-care tools, acquire accessible housing, and gain access to barrier-free workplaces and communities. These needs challenge rehabilitation professionals to extend their interventions beyond the clients' immediate impairments to focus on their long-term health needs by helping them develop healthy behaviors to improve their health and well-being and to minimize long-term health care costs associated with dysfunction.[7]

Rehabilitation traditionally has occurred in institutions and is a time-limited process aimed at helping a person with a stroke reach an optimum level of self-care function. This approach labels the recipient of service as a patient, has led the patient to understand that the therapist would

Box 3-1

The Importance of Occupation

- Occupation is the vehicle to acquire, maintain, or redevelop skills necessary to fulfill occupational roles and to provide satisfaction.[20]
- The lack of occupation leads to a breakdown in habits and physiological deterioration, which lead to loss of ability and competency to support daily life.[28]
- Individuals with cognitive loss who remain engaged in occupations retain higher levels of functional status and demonstrate fewer disturbing behaviors.[6]
- Engagement in individually motivating and ongoing occupations supplies sustenance for survival, safety, and enhanced health.[54]
- Meaningful occupations provide individuals with exercise to maintain homeostasis and to keep body parts and neuronal physiology and mental capacities functioning at peak efficiency and enable maintenance and development of satisfying and stimulating social relationships.[54]

Table 3-1

Summary of Tests and Availability

NAME OF TEST	REFERENCE	TIME TO ADMINISTER	SOURCE
Participation measures			
Activity Card Sort	5	30 minutes	American Occupational Therapy Association, AOTA Press
Canadian Occupational Performance Measure	13	approx 45 minutes	Law M, Baptiste S, Carswell A, et al: *Canadian occupational performance measure manual*, ed 3, Ottawa, 1998, CAOT Publications ACE.
Community Integration Questionnaire	55	10 minutes	Willer B, Rosenthal B, Kreutzer JS, et al: Assessment of community integration following rehabilitation for traumatic brain injury, *J Head Trauma Rehabil* 8(2):75–87, 1993.
Quality of life measures			
Stroke Adapted Sickness Impact Profile	51	15 minutes	van Straten A, de Haan RJ, Limburg M, et al: A stroke-adapted 30-item version of the Sickness Impact Profile to assess quality of life (SAS-SIP30), *Stroke* 28(11):2155–2161, 1997.
Stroke Impact Scale Version 3	19	30 minutes	User agreement and forms available at the following website: www2.kumc.edu/coa/SIS/Stroke-Impact-Scale.htm.
Reintegration to Normal Living	57	10 minutes	Wood-Dauphinee SL, Opzoomer MA, Williams JI, et al: Assessment of global function: the Reintegration to Normal Living Index, *Arch Phys Med Rehabil* 69(8):583–590, 1988.
Medical Outcomes Study Short-Form Health Survey (SF-36)	53	15 minutes	RAND Corporation, Santa Monica, California.
World Health Organization Quality of Life Scale (WHOQOL-BREF)	40	10 minutes	World Health Organization, 1993

Even in the absence of motor impairment, a cognitive deficit can greatly impair the ability of an individual to return to tasks done before the stroke.[23] Cognitive deficits incorporate areas of attention, orientation, perception, praxis, visuomotor organization, memory, executive function, problem solving, planning, reasoning, and judgment.[29] Tatemichi and colleagues showed that cognitive dysfunction was a significant predictor for dependent living after discharge and found that quality of life is related to sequential aspects of behavior.[48] Reading the newspaper, watching a movie, finding items on a grocery list, or knowing what to do if lost in the mall can be a challenge for some individuals following stroke.[26] Clients often report feeling overwhelmed with things that came automatically before the stroke. See Chapters 17, 18, and 19.

Impaired balance is cited in the literature as a key variable to independence in the community because of an increased risk of falls. For someone with impaired balance, a trip to the kitchen for a drink of water is a daunting task. Taking out the trash or resuming bowling may provoke enough fear to stop these activities. Addressing balance impairments in the hospital setting may not transfer to ability in the community, so testing of the individual's abilities outside of a sheltered rehabilitation clinic is essential. Decreased motor function and coordination contributes to poor participation in prior activities by limiting the ability to write, cut food, or resume playing tennis. See Chapters 8 and 9.

For individuals with limited mobility, home and community access is problematic. Difficulty with stairs or the inability to ambulate long distances limits the scope of activities for survivors of stroke. A home visit before discharge is recommended to resolve any immediate issues with inaccessibility, as individuals commonly receive equipment that does not fit in their homes. Obstacles including stairs, furniture, power

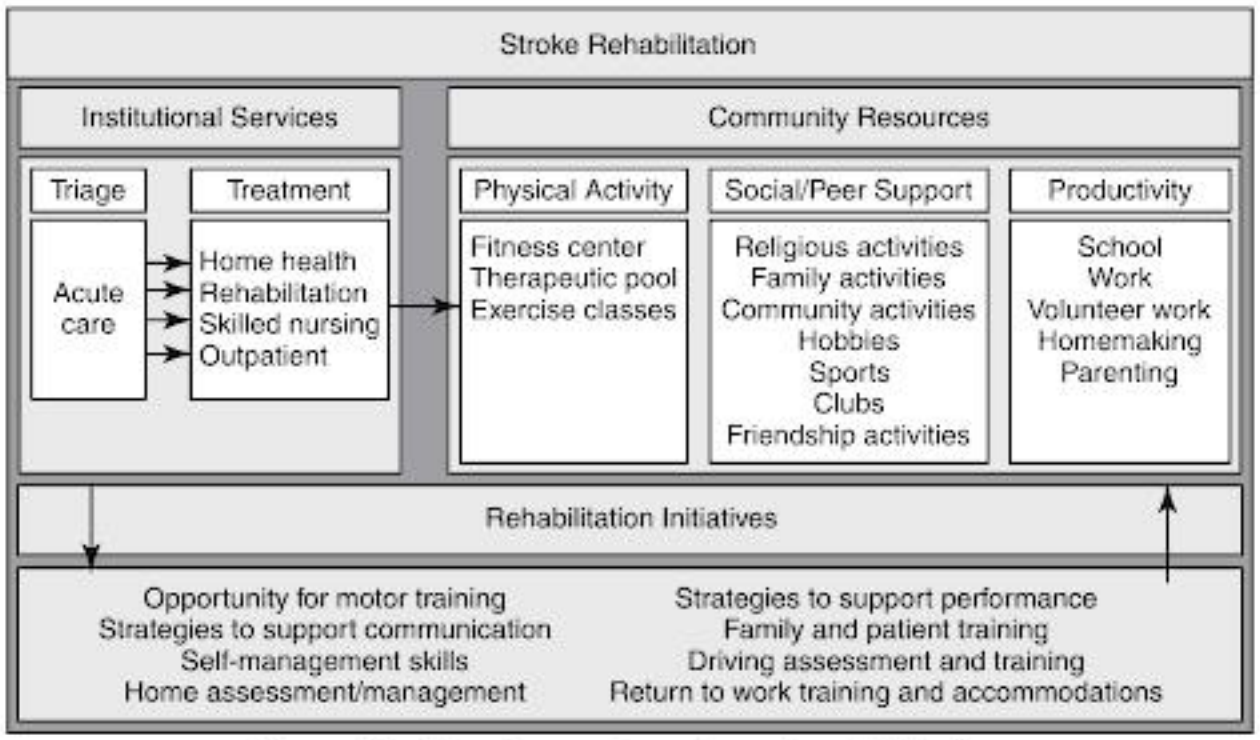

Figure 3-3 Continuum of care in stroke rehabilitation.

CASE STUDY

Improving Participation through Occupation—cont'd

promptly called 911. When paramedics reached her, the dysarthria was severe and she had complete left hemiplegia. She was oriented to her name and where she was but not to the date. In the emergency room, Rosemary was determined to have sustained a large right middle cerebral artery stroke. She was admitted immediately to the hospital and was referred to the stroke team for evaluation and treatment.

Rosemary's deficits included the following. She was unable to move her left arm or leg. She could roll in bed to her left side using the bed rail, but required maximum assistance to roll to the right. She was dependent with her transfers and basic ADL. She had a left visual inattention and decreased sensation on the left side of her body. She was sleepy and was unable to work with a therapist for more than 30 minutes at a time.

Over her first few days in the hospital, Rosemary began to improve. She was able to tolerate more time in therapy. She could support herself while sitting on the edge of the bed and began to play an active role in her ADL. Rosemary was able to move from her bed to a chair with 75% assistance from the nursing and therapy staff. She was tolerating sitting up in bed and a chair for extended periods throughout the day. The team met to determine the course of Rosemary's rehabilitation. At the team meeting, Rosemary's living alone in a two-story home located in the city was revealed. Multiple steps were required to enter. She had two bathrooms in the house; however, the bathroom with a shower was located on the second floor. She had no family locally. Her home was located within walking distance of the doctor and a large grocery store.

Rosemary was a violinist in a local quartet and taught violin on the side. She had few friends other than those in the group with whom she worked. In addition, she was driving (and using public transportation), cooking, shopping, and managing her finances independently before her stroke. Because of these responsibilities and her lack of support at discharge, the team decided Rosemary would benefit from inpatient rehabilitation.

On admission to inpatient rehabilitation, Rosemary was evaluated by nursing, physical therapy, occupational therapy, and speech therapy staff members. She required moderate to maximum assistance with basic ADL and transfers. She required 100% assistance to walk using a walker and an ankle/foot orthotic. She was able to move from her bed to a chair and back with 75% assistance. Her memory was good; however, she indicated that her attention was not, and she appeared easily distracted in the clinic. She was oriented to person, place, date, and situation. Her speech remained slurred, but her swallow was normal. Rosemary's endurance improved greatly. She continued to show subtle signs of a left visual inattention, and her left arm continued to be weak throughout. Manual muscle tests indicated strength at the shoulder and elbow was 3/5. Strength in the wrist and hand was 2/5. Sensation was normal to pin prick and temperature. She was diagnosed with depression and was treated medically. The only interests stated in her chart included playing and teaching violin and playing bridge.

virgil mathiowetz

chapter 4

Task-Oriented Approach to Stroke Rehabilitation

key terms

model of motor behavior
motor control
motor development
motor learning
task-oriented evaluation framework
task-oriented treatment strategies

chapter objectives

After completing this chapter, the reader will be able to accomplish the following:

1. Describe the motor behavior (i.e., motor control, motor learning, and motor development) theories and model that support the occupational therapy task-oriented approach to persons after stroke.
2. Describe the evaluation framework for the occupational therapy task-oriented approach and identify specific assessments that are consistent with the approach.
3. Describe general treatment principles for the occupational therapy task-oriented approach and their application to persons after stroke.
4. Given a case study of a person after stroke, describe occupational therapy task-oriented approach evaluation and treatment strategies that you would use.

This chapter provides a theoretical foundation for the occupational therapy (OT) task-oriented approach or a function-based approach for persons after stroke. Mathiowetz and Bass-Haugen[56] proposed this approach in 1994 based on the motor behavior/motor control, motor development, and motor learning-theories and research of that time. Motor behavior, OT theories, and research have evolved since then, so the OT task-oriented approach has evolved as well.[5,54] This chapter represents the most recent thinking regarding this approach.

The theoretical assumptions of the neurophysiological approaches, which include Rood sensorimotor approach,[71] Knott and Voss proprioceptive neuromuscular facilitation,[48] Brunnstrom movement therapy,[12] and Bobath neurodevelopmental treatment[8,9] were based on the empirical experience and research of their time. However, as the motor behavior theories changed in the 1980s and 1990s, the assumptions of the neurophysiological approaches were challenged,[34,77] and alternative approaches were proposed.[14,15,40,54-56] Recently the theoretical assumptions of

can affect occupational performance tasks and/or role performance. "In some cases, only one primary factor might determine occupational performance. In most cases, occupational performance tasks emerge from the interaction of many systems. The on-going interactions between all components of the model reflect its heterarchical nature."[54]

In addition, any occupational performance task affects the environment in which it occurs and the person acting. For example, if a patient with hemiplegia becomes independent in driving by using assistive technology and adaptive strategies, the patient's ability to drive would free family members from needing to provide transportation for appointments and social events. The patient would be able to resume the role of driver and the task of driving, which were likely meaningful to the patient's life. Thus the occupational performance task of driving affects persons and objects in the environment (i.e., assistive technology added to the car). The task also affects the person and the associated components. The ability to be less dependent on the family may affect the patient's self-esteem positively (i.e., psychosocial subsystem). The process of driving "provides the patient the opportunity to solve problems and to discover optimal strategies for performing tasks. This influences a client's cognitive and sensorimotor subsystems and the ability to perform other functional tasks."[54]

The specific components (subsystems) of the systems, which influence occupational performance tasks, may be framed in OT terminology.[1,2] Components of the cognitive (mental) system include orientation, attention, memory, problem-solving, sequencing, learning, and generalization ability. Components of the psychosocial system include a person's interests, coping skills, self-concept, interpersonal skills, self-expression, time management, emotional regulation, and self-control skills that could affect occupational performance tasks. Strength, endurance, range of motion, sensory functions and pain, perceptual function, and postural control are components associated with the sensorimotor system. The environment includes physical, socioeconomical, and cultural characteristics of the task itself and the broader environment. Components of the physical environment system include objects, tools, devices, furniture, plants, animals, and the natural and built environments, which could limit or enhance task performance. The social supports provided by the family, friends, caregivers, social groups, community, and financial resources are components of the socioeconomical system, which could influence choice in activities. Finally, components of the cultural system include customs, beliefs, activity patterns, behavioral standards, and societal expectations, which also could affect occupational performance tasks.

The inclusion of role performance in this systems model reflects an OT, not a motor behavior perspective. "Occupational therapists believe the roles that persons want and need to fulfill determine the occupational performance tasks and activities they need to do. Conversely, the tasks and activities persons are able to do determine what roles they are able to fulfill."[54] Box 4-1 summarizes the assumptions of the OT task-oriented approach.

EVALUATION FRAMEWORK USING THE OCCUPATIONAL THERAPY TASK-ORIENTED APPROACH

The therapist conducts the evaluation using a top-down approach as suggested by Latham.[49] Box 4-2 gives a framework for evaluation. Evaluation efforts focus initially on role performance and occupational performance tasks because they are the goals of motor behavior. A thorough understanding of the roles that a patient wants, needs, or is expected to perform and of the tasks needed to fulfill those roles enables therapists to plan meaningful and motivating treatment programs. After a patient has identified the most important role and occupational performance limitations, therapists use task analysis to identify which subsystem of the person or environment is limiting functional performance. This process may indicate the need for evaluation of selected subsystems of the person or environment.[25] The emphasis on role and occupational performance in the OT task-oriented approach is consistent with the idea that OT evaluation should be primarily at the participation and activities level rather than the impairment level, using World Health Organization[90] terminology. The therapist needs to use qualitative and quantitative measures during the evaluation process.[86] "Therefore, therapists use interviews, skilled observations,

Box 4-1

Assumptions of the Occupational Therapy Task-Oriented Approach Based on a Systems Model of Motor Behavior

- Personal and environmental systems, including the central nervous system, are heterarchically organized.
- Functional tasks help organize behavior.
- Occupational performance emerges from the interaction of persons and their environment.
- Experimentation with various strategies leads to optimal solutions to motor problems.
- Recovery is variable because patient factors and environmental contexts are unique.
- Behavioral changes reflect attempts to compensate and to achieve task performance.

Data from Mathiowetz V, Bass-Haugen J: Assessing abilities and capacities: motor behavior. In Radomski MV, Latham CAT, editors: *Occupational therapy for physical dysfunction*, ed 6, Baltimore, 2008, Lippincott Williams & Wilkins.

Third, patients rate their own performance and their satisfaction with their performance on the five most important problem areas. Therapists may use these performance and satisfaction ratings again as outcome measures, measuring change across time. If therapists are concerned that a patient cannot rate performance accurately because of a cognitive impairment or age, therapists may use direct observation of selected activities or a caregiver interview to verify the information. The information elicited by the COPM is unique to each patient and the individual's environment, which is an essential part of the OT task-oriented approach (Fig. 4-3).

Another recommended measure of occupational performance specific to ADL and IADL is the Assessment of Motor and Process Skills (AMPS).[24] The assessment is client-centered because the person chooses two or three ADL or IADL tasks to be performed, which ensures that the task or activity is familiar and relevant to the person being evaluated. The purpose of the AMPS is "to determine whether or not a person has the necessary motor and process skills to effortlessly, efficiently, safely and independently perform the ADL tasks needed for community living."[24] The AMPS is appropriate for persons from diverse backgrounds and with diverse needs and interests because it has been standardized internationally and cross-culturally. "A unique feature of the AMPS is that it can adjust, through Rasch analysis, for the difficulty of tasks performed and the severity of the rater who scores the client's performance. In addition, it allows a therapist to compare the performance of clients who performed one set of tasks on initial evaluation with the results of a re-evaluation on a different set of tasks."[54] The primary limitation of the AMPS is that it requires a five-day training workshop to learn how to administer the assessment in a reliable and valid way. Computer software to score the AMPS is provided as part of the workshop. Finally, the AMPS assists in the next step in the evaluation process, because it requires observation of patients performing occupational performance tasks (see Chapter 21).

While evaluating occupational performance tasks, "therapists must observe both the outcome and the process (i.e., the preferred movement patterns, their stability or instability, the flexibility to use other patterns, efficiency of the patterns, and ability to learn new strategies) to understand the motor behaviors used to compensate and to achieve functional goals."[54] Determining the stability of the motor behavior is important to determine the feasibility of achieving behavioral change in treatment. "Behaviors that are very stable will require a great amount of time and effort to change. Behaviors that are unstable are in transition, the optimal time for eliciting behavioral change."[54] Thus, when behaviors are more stable, a compensatory approach may be most appropriate; when behaviors are unstable, a remediation approach may be more successful. Quantitative and qualitative measures are needed to evaluate the process of task performance.

The third step in the evaluation process involves task selection and analysis. The tasks selected for observation should be ones that patients have identified as important but difficult to do. Task analysis requires therapists to observe their patients performing one or more occupational performance tasks. In most cases, observation of performance happens as part of the second step described previously. Therapists use task/activity analysis to evaluate activity demands, context, patient factors, performance skills, and performance patterns to determine whether a match exists that enables persons to perform occupational tasks within a relevant environment. If the person is unable to perform the task, therapists attempt to determine which person or environment subsystems are interfering with occupational performance. "In dynamical systems theory, these are considered the critical control parameters or the variables that have the potential to shift behavior to a new level of task performance."[54] Each person has unique strengths, limitations, and environmental context after a stroke. Therefore, the critical control parameters that support or limit occupational performance tasks are also unique. An effective intervention strategy for one person after stroke may not be effective for the next person. Another concept of dynamical systems theory is that critical control parameters also change as persons and their environments change over time. Therefore, an intervention that worked well early in a patient's rehabilitation might not work well late in the rehabilitation process or vice versa.

The identification of critical control parameters is the most challenging part of the evaluation process. However, evidence in the research literature indicates that some variables or subsystems of the person and/or environment are potential critical control parameters for persons after stroke. Gresham and colleagues[36] reported that psychosocial and environmental factors were significant determinants of functional deficits in persons for the long term after stroke. In a review, Gresham and colleagues[35] reported that 11% to 68% of persons experience depression after stroke, with 10% to 27% meeting the criteria for major depression. In the cognitive area, Galski and colleagues[28] reported that for persons after stroke, "deficits in cognition, particularly higher-order cognitive abilities (e.g., abstract thinking, judgment, short-term verbal memory, comprehension, orientation) play an important role in determining length of stay and in predicting functional status at the end of hospital stay." In the sensorimotor area, weakness,[65] fatigue,[44] impaired motor function,[6] and visuospatial deficits[82] are associated with poorer functional outcomes. For example, Bernspang and colleagues[6] reported that motor function measured with the Fugl-Meyer Assessment[27] was correlated moderately ($r = 0.64$) with self-care ability.

Although blocked or repetitive practice of the same task normally is not recommended, such practice may be helpful or necessary when a patient is first learning the requirements of a new task.[74] However, therapists should shift to random and variable practice schedules as soon as possible to enhance motor learning. Random practice involves practicing more than one task within a session (i.e., avoiding repetitive practice of the same task). Variable practice involves experimenting with different tools for completing a task, with different location of the tools relative to the person, or with varied environments for performing a task. In addition, patients should practice tasks in their natural context whenever possible. Therefore, ADL tasks normally done in a patient's room should be practiced there rather than in the OT clinic. Even better would be patients practicing ADL tasks in their own homes.

When therapists are beginning to teach patients new tasks or new ways to perform previously learned tasks, they may need to provide some physical guidance and verbal feedback.[73] However, guidance and feedback should be tapered off quickly so that the person does not become dependent on them. For a therapist not to provide guidance and feedback when a patient is struggling to perform a task is difficult. However, providing physical guidance prevents patients from learning how to use their remaining resources to get the job done, and providing immediate and frequent feedback prevents patients from learning how to use their own feedback mechanisms to monitor and evaluate their own performance. If patients are unaware of a deficit (e.g., neglect to use involved extremity in a task), the use of a videotape of their performance can supplement their usual feedback mechanisms.[69] By the time a patient is approaching discharge, therapists should be providing minimal guidance or feedback. The therapist should remember that the goal of rehabilitation is to train the patient to be independent without the therapist's presence.

In a related issue, patients need to learn how to analyze tasks and to problem-solve on their own. If the therapist always analyzes tasks for patients and solves all their problems, the patients will not learn how to do those things themselves. In the limited therapy time available, preparing patients for all possible tasks, activities, and environments that they will confront after they are discharged is impossible. The therapist's role is to train patients how to do task analysis and problem-solving during the rehabilitation process, so that by the time they are discharged, they are capable of doing those things on their own. From early in rehabilitation, the therapist should involve patients in task analysis and guide them through the process. As occupational problems are addressed, the therapist should keep patients involved in trying to find solutions to problems. Therapists should encourage experimentation to find the optimal solution for that specific person. The therapist should remember that the same solution does not work for all patients (see Chapter 5).

Minimize Ineffective and Inefficient Movement Patterns

As described previously, during observation of a patient performing an occupational performance task, therapists attempt to identify what may be critical personal or environmental factors that are interfering with effective and efficient movement patterns. The following strategies are ways that therapists can intervene to reduce ineffective and inefficient movement.

Remediate a Client Factor (Impairment) if it is the Critical Control Parameter. When therapists identify person factors in the cognitive, psychosocial, or sensorimotor systems as possible critical control parameters, then they should attempt to remediate those factors, assuming that is possible. For example, Flinn[26] identified decreased strength as one critical control parameter that interfered with occupational performance tasks for a person after stroke. Thus, she attempted to remediate this sensorimotor variable through the use of exercise and increased use of the involved extremity for functional tasks. For this person, the use of exercise was meaningful because she saw a clear connection between her exercise program and her ability to use her involved arm and hand for everyday tasks. The therapist also encouraged her to use her involved extremity whenever possible in therapy and for various homework assignments.

In the case of G.W., decreased strength, impaired sensation, and neglect of the left upper extremity were identified as possible control parameters. Therefore, attempts to remediate these factors were warranted in this case. However, sometimes remediation of a potential control parameter is impossible because of the severity of the disease process or limited time available for therapy. In such cases, a more compensatory approach to treatment is indicated.

Adapt the Environment, Modify the Task, Use Assistive Technology, and/or Reduce the Effects of Gravity. For many patients, the quickest and most effective approach to improving occupational performance is to adapt the task and/or the environment. For example, Gillen[31] described a patient with severe limitations in self-care activities following multiple sclerosis and ataxia. Tremor, impaired postural control, paraparesis, and decreased endurance limited his occupational performance. The patient's priority was to gain access to the community and community resources. He did not have adequate motor control to operate a manual chair or to control a standard power chair. Therefore, a specialized power chair was prescribed that provided optimal head and trunk stability, allowed independent tilting, included

CASE STUDY

Occupational Therapy Task-Oriented Approach for a Stroke Survivor—cont'd

2. Increase independence in ADL and leisure tasks.
3. Begin planning for discharge to home and for possible roles for him on his son's farm.

The patient became aware through the evaluation process that he tended to neglect his left arm and hand and was motivated to improve its function. Thus, he was open to experimenting with using his left upper extremity to assist during functional tasks. He was taught one-handed dressing techniques with reminders to use his left arm and hand as much as possible. For example, G.W. was encouraged to raise his left arm as he slid his shirt on and to use his left hand to stabilize his shirt and pants while buttoning. Various options for tying his shoes were explored. He chose to use Kno-Bows because of the ease of using them compared with alternatives. A rocker knife was chosen to enable independent cutting of meat. The therapist communicated with his wife and nursing staff on what he was able to do relative to ADL tasks and what adapted equipment (e.g., bath chair) he needed to be independent. G.W. was independent in bathing himself when the bath chair was available to him. He expressed some concern about slipping and falling when he would get home. Plans were made to order the grab bars, bath chair, and nonskid bath mat.

In addition, various leisure activities including card playing were explored. He was able to pull cards toward himself with his left hand but was unable to pick them up or hold them. A cardholder was prescribed so that he could play cards immediately. Although he only had a mild interest in playing checkers, he found out that he could slide enlarged checkers with his left hand and was willing to work at this activity to improve his left arm and hand function.

During one session, his son and wife came to discuss his roles at home and on his son's farm. Both of them suggested that they could get help for the things that he could not do. Although G.W. agreed that there were some tasks he could no longer do or did not care to do, he still wanted to do some gardening and to help with some things on the farm. He did not want just to sit around and watch television. After brainstorming what roles and tasks might still be possible, the discussion shifted to adapted strategies and equipment that might be needed to make these tasks possible.

At the end of the first week, he was able to perform all ADL task with minimal supervision (i.e., reminders to use his left hand and to search his left visual space). He could now walk 30 feet with his cane and was practicing going up and down steps in physical therapy.

Week 2 Treatment Plan

1. Explore the possibility of driving and continued gardening.
2. Finalize plans for discharge to home, including ordering and installing adapted devices.
3. Finalize home program and follow-ups.

The patient was evaluated on some aspects of driving using a modified car. He was able to transfer in and out of the car with moderate supervision. He was discouraged that he was not able to push in the clutch with his left foot. He preferred driving a stick shift but could see that a car with an automatic transmission would be easier for him. He agreed to discuss getting a different car with his wife and son. Other adaptations that might make driving easier and safer were explored. The issue of neglect of his left visual field was discussed and evaluated using a driving simulator. He did have problems (i.e., simulated crashes) because of neglect. It was decided that additional practice with the simulator and other activities to improve his visual scanning were necessary before he could drive again.

G.W. continued to use various leisure and ADL activities to increase active use of his left arm and hand. Set-up of the activities was structured to require increased visual scanning as he did these activities.

Although G.W. continued to improve in his walking and stair-climbing ability, it was decided that a second handrail should be installed at both entrances to the home and in the basement and upstairs stairways. His son agreed to arrange for someone to do this. In addition, he agreed to install grab bars in the bathroom and in the hallway between the bathroom and bedroom. Sometimes, G.W. needed to use the bathroom at night.

Although he was improving in his performance on the driving simulator, he was told that he was not yet safe to drive. G.W. was referred to a regional driving center, which evaluates and trains persons with disabilities in safe driving. His wife or son would drive him until he could drive again.

A home program was developed with a variety of tasks and activities that required the use of his left arm and hand. He was now approaching the level of function that made him an appropriate candidate for CIMT. Unfortunately, access to this type of program was not feasible for G.W. because of distance and money. The therapist explained the concept of CIMT and developed a modified program that G.W. could do on his own. The modified program was adapted from a small study by Page and colleagues[67] and provided some evidence that an outpatient program of CIMT could be beneficial. Three outpatient follow-ups were scheduled to monitor and upgrade his home program.

joyce s. sabari

chapter 5

Activity-Based Intervention in Stroke Rehabilitation

key terms

function
capacity
performance
neural plasticity
learned nonuse
constraint induced movement therapy (cimt)
learning
training
practice
kinesiological linkages
generalized motor programs
cognitive strategies
strategies for community participation
declarative learning
procedural learning
implicit learning
explicit learning
generalization/transfer of learning
intrinsic feedback
extrinsic feedback
knowledge of performance (kp feedback)
knowledge of results (kr feedback)
practice conditions
repetitive practice
blocked practice
contextual interference
closed tasks
variable motionless tasks
open tasks
mechanical constraints to movement
self-monitoring skills
metacognition
task/activity analysis
postural set
postural adjustments
dissociation between body segments
activity synthesis
practice challenges
compensatory adaptations

chapter objectives

After completing this chapter, the reader will be able to accomplish the following:

1. Apply the principles of the International Classification of Function and the Occupational Therapy Practice Framework to occupational therapy intervention for stroke survivors.
2. Understand implications of neuroscience studies of plasticity and constraint induced movement therapy to activity based interventions in stroke rehabilitation.
3. Design effective practice opportunities for stroke survivors to recover motor, cognitive, and participation skills.
4. Understand the basis of interventions designed to enhance stroke survivors' potential to achieve maximal recovery.
5. Apply principles of activity analysis and synthesis when designing occupational therapy intervention for stroke survivors.

PRACTICE AND LEARNING

Goals of Training and Learning

Learning and training are two distinct phenomena, each with its own required style of practice. The goal of training is to memorize a prescribed solution to a selected task challenge, whereas the goal of learning is to develop one's own solution, which can be applied in a variety of situations. Based on each client's abilities and role demands, the occupational therapist determines whether the therapeutic goal will be to promote training or learning. In therapeutic training, practice entails repetitive performance of a designated sequence of behaviors. Task performance must occur in the actual setting in which the individual plans to perform the task, because there is no evidence that skills acquired through training can be successfully applied in different environmental contexts.[52,57]

Learning and training are both internal phenomena that cannot be observed directly. Therapists assume that training has occurred if performance of a specific task improves and persists over time. Therapists assume learning has occurred when a person is able to apply a new set of skills within a variety of situations.[46,51] Whenever possible, occupational therapy attempts to promote learning of motor and cognitive skills that will provide the individual with an infinite number of choices for task and role engagement. Practice for learning requires active engagement in tasks that require problem-solving and implementation of effective foundational strategies. Therefore, before providing practice opportunities to stroke survivors, occupational therapists must first prepare the clients with underlying motor, cognitive, and social foundational strategies.

Foundational Strategies for Task Performance

Kinesiological Linkages and Generalized Motor Programs

When the neuromuscular system is functioning optimally, a person can rely on automatic kinematic and kinetic linkages to serve as a foundation for functional movements. Although these linkages are described in a variety of ways,[5,46,51] motor control theorists and kinesiologists agree that they promote optimal mechanical interactions between muscles and body segments.

Often, stroke survivors have lost the automatic kinesiological linkages associated with efficient movement.[13,59] This may be a result of limited mobility of body segments, weakness of specific muscular components, or loss of the motor program that links muscles or joints during a given movement sequence. Several automatic kinematic linkages are commonly observed during optimal movement, but are unavailable to many stroke survivors:

- Pain-free shoulder abduction through the full range of motion relies on scapulohumeral rhythm, a kinematic linkage between the scapula and humerus[41] (Fig. 5-3).
- The deltoid and rotator cuff muscles are kinetically linked to ensure that the deltoid fibers produce the desired rotary force on the humerus. Without this linkage, an attempt to abduct the shoulder will instead result in a nonfunctional upward shrug of the shoulder[41] (Fig. 5-4).
- Glenohumeral external rotation is automatically linked with end-range humeral flexion and abduction.[41]
- Grasp patterns are automatically linked with wrist extension to allow for efficient use of extrinsic finger muscles.[41]
- Lumbopelvic rhythm provides for appropriate interactions between movements at the lumbar spine and adjoining pelvis. When rising to stand from a seated position, for example, forward trunk motion is most efficiently initiated at the hips and is accompanied by simultaneous pelvic anterior tilt.[41] See Chapter 14.

Kinesiological linkages can be conceptualized as generalized motor programs (GMPs).[31,35,46] These "prestructured sets of central commands" govern a particular class of actions. GMPs are designed to be modified in response to continuous changes in environmental and task parameters. Therefore, a unique pattern of activity, with core

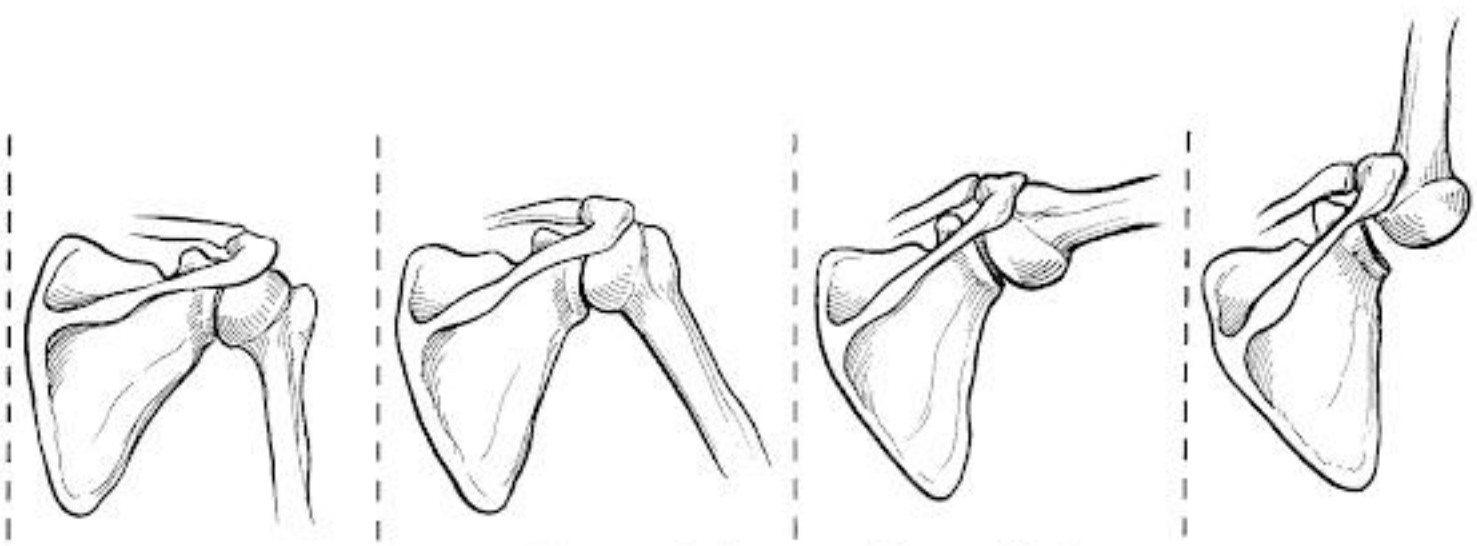

Figure 5-3 Kinematic linkage: scapulohumeral rhythm.

same lumbopelvic interactions previously practiced in different contexts. Research findings from studies with healthy participants provide support for the use of this approach for learning the invariant structure of a GMP.[18,33] In the terminology of motor learning science, these studies found that a constant or blocked practice schedule of the underlying GMP, using varied practice parameters, leads to enhanced transfer benefits.

Carr and Shepherd's program for optimizing motor function after stroke[12,13] uses five major techniques to assist patients with developing motor strategies: (1) verbal instruction, (2) visual demonstration, (3) manual guidance, (4) accurate and timely feedback, and (5) consistency of practice. In addition, patients develop skill in providing themselves with intrinsic feedback about the kinematics of their motor performance. Outcome studies[39] of individuals recovering from stroke provide support for this program's efficacy.[12,13]

Toglia[56,57] and Golisz[19] developed a systematic approach to promote generalization of cognitive strategies, in which the therapist grades treatment by changing certain characteristics of a task but leaving the underlying strategy the same. The following example illustrates a treatment sequence designed to facilitate learning and generalization of a strategy for categorizing information:

The initial task is the first activity performed by the patient, such as sorting a deck of playing cards into a red group (hearts and diamonds) and a black group (spades and clubs). Near transfer is an alternate form of the initial task. Using the previous example, the person might be instructed to sort the playing cards into four groups according to their suits or two groups of odd and even numbers.

Intermediate transfer has a moderate number of changes in task parameters but still has some similarities to the initial task. For example, the same person may be asked to create three categories for sorting a stack of photographs for eventual placement in a photo album.

Far transfer introduces an activity conceptually the same as but physically different from the initial task. Now the person may be asked to organize a collection of magazines into groups based on general interest areas (e.g., news, sports, fashion) for display in a clinic waiting room.

Very far transfer requires spontaneous use of the new strategy in daily functional activities. Before traveling to a neighborhood mall, the person may be asked to categorize items on a shopping list based on the type of store in which they can most likely be purchased.

This "multicontext approach" emphasizes the use of self-assessment and intrinsic KP feedback. Before attempting a new task, patients estimate their performance accuracy and efficiency and determine similarities and differences between the current task and previous activities. After completing a task, patients evaluate their performance and identify techniques that may be helpful in the future. The therapist's major roles are to structure the activity progression and guide patients in developing insights and strategies. See Chapter 19.

Practice Conditions

Several aspects of practice conditions have been studied under both laboratory and clinical conditions. Occupational therapists can use these findings to structure practice conditions in stroke rehabilitation programs. The key is to structure conditions during the acquisition phase that will produce optimal retention and transfer of the learned skills.

Practice Schedules. During blocked (or repetitive) practice, patients practice one task until they master it. This is followed by practice of a second task until it is also mastered. Random (or variable) practice requires patients to attempt multiple tasks or variations of a task before they have mastered any one of the tasks. In addition, the various trials are performed in a random order. Subjects who participate in variable practice perform better on transfer tests than subjects who participate in repetitive practice.[21] A study of stroke outpatients found that random practice was more effective than blocked practice for long-term retention of improvements in reach and manipulation skills.[22] An explanation is that variable practice facilitates generalization by preventing individuals from developing context-dependent inflexibility when using a newly learned skill.

Contextual Interference. Contextual interference refers to factors in the learning environment that increase the difficulty of initial learning.[7] Research studies consistently find that higher levels of contextual interference promote retention and generalization (transfer) of newly learned skills.[8,29,54] These findings are typically explained with the hypothesis that initial obstacles to skill acquisition prevent individuals from developing context-dependent inflexibility when using the learned skill in new situations.[7] Another explanation is that high contextual interference forces a person to use greater versatility in learning strategies in order to overcome the difficulty of initial practice during the acquisitional learning phase.[28] Limited KR feedback is one example of contextual interference that has already been discussed. Blocked and random practice schedules, described previously, are examples of low and high contextual interference, respectively. Although blocked practice may lead to quicker skill acquisition, random practice results in greater retention and generalization.[46]

Whole versus Part Practice. Therapists may intuitively believe that it will be easier for a client to learn small segments of a task than the task in its entirety. However, breaking a task into its component parts for teaching

STRUCTURING ACTIVITY DEMANDS TO PROVIDE EFFECTIVE PRACTICE OPPORTUNITIES

Activity-based intervention is a foundation of occupational therapy in stroke rehabilitation. During the evaluation process, an occupational therapist determines:

- Which activities are important to the stroke survivor as determined by the individual's roles, interests, and anticipated environment
- Which activities the stroke survivor can or cannot perform
- Which internal and external factors impede the survivor's ability to complete the identified activities

During treatment, occupational therapists use activities in two major ways.

1. Some activities may be designed to provide structured challenges to improve internal skills. For example, an occupational therapist may engage a stroke survivor in a modified card game. Depending on the skill-related goals for this individual, the occupational therapist may structure the activity so that it requires forward reach with a hemiparetic arm. Alternatively, the card game may require the person to place the cards along a wide horizontal surface while standing. This modification in activity parameters provides opportunities for learning balance strategies while shifting the center of gravity in a lateral direction.
2. Other activities are designed to provide practice of actual task performance in real-life situations. Examples include direct practice in performing a morning self-care routine or getting into and out of an automobile. Practice of individualized roles in real-life situations is critical, but typically unfeasible during therapy sessions. Therefore, therapists need to structure homework assignments for stroke survivors to practice at home and to discuss at the next therapy session.

Task Analysis

An occupational therapist assesses tasks of daily living in the environmental context in which the individual plans to perform each task. The therapist determines which skills are necessary for task performance and compares this analysis to the functional strengths and limitations exhibited by an individual stroke survivor. This task analysis enables the occupational therapist to plan an individualized treatment program that will improve relevant performance skills and enable the person to use compensatory strategies to overcome those limitations that show weak potential for significant improvement.

Analyzing an Activity's Requirements for Postural Set

The occupational therapist determines the optimal "postural set" for performing a selected motor task. To perform the simple act of standing up, individuals must posturally set themselves in several ways. Both feet must be positioned on the floor in an appropriate base of support; perpendicular angles are established at the ankle, knee, and hip joints; and the pelvis is tilted anteriorly to free the lumbar spine for forward movement.[13,51]

When standing, people automatically change the configuration of their bases of support in anticipation of the direction toward which they expect to shift their body weight. If they plan to shift forward, as is done when reaching ahead, they will establish an anterior-posterior base of support. If they plan to shift to the left or right, as is done when stepping laterally to position their bodies in front of a bathtub, they will establish a medial-lateral base of support. Persons with hemiplegia often assume postural support bases inappropriate for the upcoming activity. The occupational therapist facilitates future task performance by determining and then instructing the individual in choosing appropriate postural sets for specific activities. For example, assuming the most efficient postural set for standing in front of a toilet can determine whether a man will be able to safely urinate independently.

Just as appropriate postural sets are important precursors to efficient motor performance, preplanning is also instrumental in determining the success of cognitively or visually challenging tasks. Activity analysis includes a determination of preliminary cognitive strategies that will facilitate task performance. For example, a person with right hemisphere dysfunction may experience difficulty in spatially orienting a blouse or slacks for independent dressing. The individual may be unaware that, prior to the stroke, he used a quick and automatic process to visualize and orient the garments in relation to the body segments. The occupational therapist's skill in activity analysis enables this person to develop a "set up" strategy, such as lining up each garment before attempting to complete the additional steps of dressing.

Analyzing Activity Requirements for Weight Shift and Balance

Postural adjustments that serve as balance mechanisms during weight shift are often impaired after stroke.[13,43,51] Understanding a task's inherent balance challenges is critical for developing treatment goals and compensatory strategies. Success in shifting weight during activity performance can be facilitated greatly through appropriate postural sets. The importance of this class of prerequisite skills is important when bathing. If patients use a tub bench, they will need to posturally set themselves for a posterior weight shift from stand to sit onto the bench. Once sitting, they will need to rotate their pelvis and bring both legs into the tub. The next step will be to shift their weight laterally, while sitting, to position themselves on the tub bench. A forward weight shift will often be required to adjust the water, and significant challenges to

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Table 6-1

Evidence Table for the Neurodevelopmental Treatment/Bobath Approach

AUTHOR/ YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, AND PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOMES	RESULTS	COMMENTS
Langhammer et al, 2000	Compare outcome of Bobath therapy with motor learning program	Double blind RCT; 61 patients acute stage 1b; 6/10	Bobath therapy (N=28); 5 days a week for duration of hospital stay	MRP (N=33)	3 days post-admission, 3 weeks, and 3 months poststroke	1. MAS 2. SMES 3. BI 4. NHP 5. Length of stay, assistive device	Both groups improved at 3 months on MAS, SMES, and Barthel Index. MRP had better improvement on MAS and had shorter hospital stay.	Well-designed study; MRP better than Bobath approach
Langhammer et al, 2003	Evaluate effectiveness of Bobath and MRP 1 and 4 years after stroke	Double blind RCT; 61 patients acute stage 2b; 4/10	Bobath therapy (N=28); No intervention following initial therapy during acute stage	Motor Learning Program (N=33); No intervention following initial therapy during acute stage	1 and 4 year follow-up of patients from Langhammer et al, 2000	1. MAS, 2. SMES 3. BI 4. Nottingham Health Profile 5. Length of stay, assistive device, mortality 6. BBS	No difference in mortality rate; motor function decreased from year 1 to year 4 on MAS and SMES for both groups; independence in ADL decreased; QOL better at 1 and 4 years than at 3 months	Initial benefit of MRP not maintained in the long term, partly because therapy was discontinued.
Tang et al, 2005	Compare Bobath approach (NDT) with POWM therapy	RCT; 47 patients acute, subacute, or chronic stage; 2b; 6/10	Bobath therapy (N=22); 50 min. sessions 5× week; mat activity, sitting, standing, walking, stair climbing; focus on movement normalization	POWM therapy (N=25); using cognitive skills to focus attention and train memory	Pretreatment and posttreatment	1. Mini Mental State Exam; 2. STREAM	Both groups improved on STREAM; POWM better than Bobath on overall score, mobility, and lower extremity scores of STREAM	Well designed study; NDT not as good as active willed movement therapy, a form of task-oriented approach

Study Designs. The designs included in this review were seven RCT and two high-quality nonrandomized parallel design studies with a large number of subjects. All the studies were classified as either 1b or 2b on the Oxford levels of evidence scale.

Results of the Review. Of the nine trials examining the effect of Bobath approach, one compared Bobath to an orthopedic approach to stroke rehabilitation,[97] two studies compared Bobath with usual care including conventional PT and OT[24,25] and the other five compared Bobath approach with the task-oriented approach[41,42,67,86,91] or a variant of a task-oriented approach called the problem oriented willed movement therapy.[81]

The Bobath approach was marginally better than an orthopedic approach,[97] which is not the therapy of choice in stroke rehabilitation. When compared with conventional PT and OT, Bobath approach was no better for impairment or activity limitation outcomes.[24,25] When compared with a task-oriented approach, which represents a novel approach to stroke rehabilitation, Bobath approach was clearly less effective in four of the six studies.[41,67,81,86] There were two exceptions to this pattern: one study[42] found no differences in outcomes evaluated at one and four years after the initial therapy was administered, perhaps because patients did not receive therapy in the interim period. The other study[91] had methodological limitations that may explain the lack of differences. For instance, at baseline testing, there were differences across the two groups, the amount of time patients spent with the therapist was not the same, and finally the duration of therapy was much less compared with all other studies. Despite these two studies, the evidence overwhelmingly points to the lack of effectiveness of the Bobath approach when compared with a task-oriented approach.

Implications for Practice. Three recent systematic reviews have reported no evidence for the superiority of the Bobath approach.[34,48,62] The present review extends the results of the previous systematic reviews to demonstrate that the use of the Bobath approach needs to be reconsidered in stroke rehabilitation.

In the past few years, an attempt has been made among proponents of neurofacilitation approaches to integrate established techniques of NDT with the language of newly emerging knowledge in motor control and motor learning. This is readily seen in a recent text describing the theoretic basis of NDT.[30,34] Although this is typical during paradigm shifts, the amalgamation of old techniques with new theoretical knowledge is not useful either theoretically (since established Bobath techniques are not consistent within the new paradigm of motor control and learning) or for clinical practice (since numerous studies have demonstrated that there is indeed little evidence). The challenge for therapists is to design and evaluate techniques within the newly emerging paradigm of task-oriented training.

Functional Task-Oriented Training: The Second Paradigm Shift

The second paradigm shift in the treatment of neurological disorders began in the 1990s. Therapists began to regard neurotherapeutic approaches with less optimism. The dissatisfaction with the neurotherapeutic approaches is due, in part, to the fact that retraining normal movement patterns do not carry over into the performance of functional daily living skills, which is the ultimate goal of rehabilitation. In addition, there is a greater demand on therapists to use interventions that have demonstrated effectiveness. Evidence that demonstrates a lack of effectiveness of neurotherapeutic approaches, particularly the Bobath approach, has led to the development of novel training regimens based on what has been termed the *task-oriented approach.*[75]

Principles of the Functional Task-Oriented Approach. The task-oriented approach is based on a systems model of motor control and theories of motor learning. The approach attempts to understand the problems faced by the nervous system to control movements. This field of motor neuroscience represents a multidisciplinary approach to understanding motor control and learning from the perspectives of neurophysiology, biomechanics, and behavioral sciences. Within this framework, motor control is understood as an attempt by the nervous system to adapt movements to constraints imposed by the mechanics of the motor apparatus (including length, mass of limbs, and intersegmental dynamics of moving segments), constraints imposed by the environment (open or closed environment), and constraints imposed by the behavioral context. Studies on motor control often analyze movements at the biomechanical and behavior levels. See Chapters 4 and 5 for a detailed description.

Chapter 4 provides the reader with a more comprehensive description of the task-oriented approach. What follows is a brief description of some of the incipient principles of treatment, based on suggestions by Carr and Shepherd[10] and Gentile.[21] Within this framework, the responsibility of the therapist as a teacher of motor skills is to select contextually appropriate functional tasks, vary task parameters to ensure greater transfer of learning, structure practice schedules to encourage active participation of the patients, structure the environment so that all regulatory conditions of a given task are present, and provide feedback. To apply a task-oriented approach to treatment successfully, therapists need to become familiar with analyzing tasks and the processes underlying skill acquisition. The following two sections evaluate the literature on task-oriented approach to stroke rehabilitation.

Table 6-2

Evidence Table for Task-Oriented Training—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Winstein et al, 2004	Evaluate effects of task-related training and strength training (ST) in acute and subacute stroke	Nonblinded RCT; 60 subjects with acute or subacute stroke (2 to 35 days); 1b; 6/10	Task-related training group (N=20) received SC plus repetitive practice of functional tasks; ST group (N=20) received SC plus resistive movements using theraband; Both groups received therapy 1 hour/day, 5 days/week for 4 weeks	SC group (N=20) included facilitation, neuromuscular electric stimulation, stretching, using an NDT approach and ADL training.	Pretreatment and posttreatment, 6 and 9 month follow-up	1. FM 2. FTHUE 3. FIM	Task-related training and ST groups had better FM and Isometric torque at posttreatment primarily in less severe patients; Isometric torque improvement was maintained at 9 months	Well-designed study; task-related training was better than ST in the long-term
Blennerhasset et al, 2004	Investigate whether additional practice of upper or lower limb task improves function in subacute stroke	Single blind RCT; 30 subjects with subacute stroke (11 to 49 days); 2b; 8/10	Upper limb group (N=15) received usual PT for 1 hour/day, 5 days/week, and additional circuit training involving practice of functional tasks; 1 hour/day, 5 days/week for 4 weeks	Mobility group (N=15) received usual PT for 1 hour/day, 5 days/week, and additional training on bikes and treadmill, and practice of sit-to-stand, obstacle course walking, standing balance; 1 hour/day, 5 days/week for 4 weeks	Pretreatment and posttreatment, 6 month follow-up	1. Six-minute walk test 2. TUG 3. Step Test 4. MAS 5. JTHFT	Upper limb group performed better on the MAS and JTHFT; lower limb group had better mobility scores on the TUG	Well-designed study shows the task specificity of training; small sample

Table 6-3

Evidence Table for Constraint-Induced Movement Therapy

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Dromerick et al, 2000	Examine if CIMT is more effective than conventional therapy in acute stroke	Single blind RCT; 20 subjects with acute stroke (<14 days); 2b; 5/10	OT treatment focused on ADL, functional upper limb training with affected arm; unaffected hand in padded mitten for 6 hours a day; 2 hours/day, 5 days/week for 2 weeks	Standard OT treatment including compensatory treatment for ADL, upper limb strength, ROM, and positioning; 2 hours/day, 5 days/week for 2 weeks	Pretreatment and post-treatment	1. ARAT 2. BI 3. FIM	ARAT scores were higher for CIMT group at discharge; no differences were seen for BI score; CIMT group had higher scores on the FIM UL dressing	Low dosage CIMT (2 hours) shows improvement only in impairment level measures in acute stroke; small sample
Page et al, 2002	Test efficacy of modified CIMT in subacute stroke	Single blind, multiple baseline RCT; 14 subjects with subacute stroke (1 to 6 months poststroke); 2b; 4/10	Modified CIMT group (N=7) treated in 30 min OT sessions for functional training of upper limb using shaping; 30 min PT sessions to improve balance and mobility; less affected limb restrained in hemisling for 5 hours of frequent arm use each day; 1 hour/day, 3 days/week for 10 weeks	Traditional therapy group (N=4) received OT and PT based on Proprioceptive Neuromuscular Facilitation; and compensatory training; Control group (N=6) received no therapy; 1 hour/day, 3 days/week for 10 weeks	Two pretreatment, post-treatment	1. FM 2. ARAT 3. MAL	Modified CIMT group improved more than traditional therapy and no therapy group on FM, ARAT, and MAL	Modified CIMT, based on distributed practice over 10 weeks, better than traditional therapy based on PNF or no therapy in subacute stage; small sample size

Table 6-3

Evidence Table for Constraint-Induced Movement Therapy—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Boake et al, 2007	Evaluate the effectiveness of CIMT on motor function of the upper limb in subacute stroke	RCT; 23 patients with subacute stroke (<14 days of stroke); 2b; 6/10	CIMT group practiced reaching, grasping, lifting, and placing objects with affected hand (N=10); shaping and approximation; unaffected limb in mitt for 90% of waking hours; 3 hours/day; 6 days/week for 2 weeks	Control group practiced daily living tasks with either hand to improve strength, muscle tone and ROM (N=13); 3 hours/day; 6 days/week for 2 weeks	Pre-treatment and post treatment 3- to 4-month follow-up	1. FM of motor recovery 2. Grooved Pegboard Test 3. MAL 4. Transcranial Magnetic Stimulation	Both groups improved on primary outcome (FM); no differences between groups at posttreatment or follow-up; CIMT group reported better outcome in quality of movement during ADL performance; no differences in motor threshold at posttreatment or follow-up	Same dosage did not highlight benefit of CIMT; small sample size
Wu et al, 2007a	Evaluate effect of mCIMT on motor control of upper limb and functional change in chronic stroke	Single blind RCT; 30 subjects with chronic stroke (12 to 36 months); 1b; 7/10	Modified CIMT (N=15) received OT treatment including practice of functional tasks using shaping, and normalization of tone; unaffected hand restrained in a mitt for 5 hours/day at time of frequent use; 2 hours/day, 5 days/week for 3 weeks	Traditional rehabilitation group (N=15) received OT using Bobath approach including balance, stretching, weight-bearing of affected limb; fine motor tasks and ADL skills using unaffected arm; 2 hours/day, 5 days/week for 3 weeks	Pretreatment and post-treatment	1. Kinematic analysis of arm movement 2. MAL 3. FIM	Modified CIMT group had lower movement time and displacement and higher percentage of movement time at peak velocity; modified CIMT had better arm use and quality of movement in MAL and better FIM scores	Well designed study; modified CIMT better than Bobath approach for improving arm kinematics and functional gains in chronic stroke; no follow-up assessment

Table 6-3

Evidence Table for Constraint-Induced Movement Therapy—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Lin et al, 2009	Compare modified CIT intervention with a dose-matched control intervention in chronic stroke	Single blind RCT; 32 subjects with subacute and chronic stroke (6 to 40 months post-stroke); 1b; 7/10	Modified CIMT group (N=16) received OT with functional training of upper limb using shaping, normalization of tone; unaffected hand in mitt for 5 hours/day; modified CIMT 2 hours/day, 5 days/week for 3 weeks	Control group (N=16) received OT focused on Bobath approach and functional task training and weight-bearing; unaffected hand in mitt for 5 hours/day; control group 2 hours/day, 5 days/week for 3 weeks	Pretreatment and post-treatment	1. FM 2. FIM 3. MAL 4. NEADL 5. SIS	Modified CIMT group performed better on FM, FIM (self-care and locomotion) and SIS (ADL, mobility, and hand function), on the mobility domain of the NEADL	Modified CIMT better than Bobath approach in improving motor function and quality of life in subacute and chronic stroke

Dromerick et al, 2009	Compare CIMT with traditional OT and examine if effect of CIMT is dose dependent in very early stroke	Single blind RCT; 52 subjects with stroke (<28 days of admission to inpatient rehabilitation); 1b; 7/10	Dose matched CIMT group (N=19) received 2 hours of shaping (5 days/week for 2 weeks) and wore a mitt for 6 hours/day; higher intensity CIMT group (N=16) received 3 hours (5 days/week for 2 weeks) of shaping and wore a mitt 90% of waking hours; both groups practiced functional tasks.	Traditional OT group (N=17) including compensatory techniques for ADL, ROM, strengthening; upper limb bilateral activities; 2 hours/day, 5 days/week for 2 weeks	Pretreatment and post-treatment, 3 month follow-up	1. ARAT 2. NIHSS 3. FIM 4. SIS	All three groups improved on the ARAT; no differences were seen between control and dose matched CIMT group; high intensity CIMT had lower gains on ARAT; no differences seen across groups on FIM score; SIS score was highest for the dose matched CIMT group at 90 days	Excellent study; CIMT was no more effective than control OT of same intensity; high intensity CIMT led to less improvement in upper limb function.

AAUT, Actual Amount of Arm Use Test; ADL, activities of daily living; AMPS, Assessment of Motor and Process Skills; ARAT, Action Research Arm Test; BI, Barthel Index; CIMT, Constraint Induced Movement Therapy; FIM, Functional Independence Measure; FM, Fugl-Myer Assessment; MAL, Motor Activity Log; MAS, Modified Ashworth Scale; NDT, neurodevelopmental therapy; NEADL, Nottingham Extended Activities of Daily Living Scale; NIHSS, National Institute of Health Stroke Scale; OT, occupational therapy; PET, positron emission tomography; PNF, Proprioceptive Neuromuscular Facilitation; PT, physical therapy; RCT, randomized controlled trial; RMA, Rivermead Motor Assessment; ROM, range of motion; SIS, Stroke Impact Scale; TMS, Transcranial Magnetic Stimulation; WMFT, Wolf Motor Function Test; UL, upper limb

group of patients most likely to recover from stroke based on spontaneous recovery.[32,36]

There was tremendous variability in the form of CIMT and the dosage of intervention. Seven of the nineteen trials tested the standard version of CIMT, which included six hours of practice in each session. The other three trials of standard CIMT included either two hours,[18] three hours,[9] or four hours of training in each session.[59] However, all the ten trials of standard CIMT provided massed practice over 10 sessions across two weeks. Nine trials tested a modified version of CIMT, in which practice was distributed over sessions ranging from 12 to 30. Modified CIMT trials have been designed to replicate therapeutic dosage similar to standard practice. However, as Table 6-3 demonstrates, there is tremendous variability in dosage, ranging from 30 minutes to six hours of practice per session.

Since one of the major principles of CIMT is constraint of the unaffected hand, all studies included some form of constraint using a mitt, sling, hemisling, or splint. There was a large variability across studies in terms of the hours of restraint (from five hours to 90% of waking hours).

Timing of Intervention. Four CIMT trials were conducted in the acute stage,[6,18,19,66] four trials were conducted in the subacute stage,[9,59,63,73] and six trials were conducted in the chronic stage.[46,65,80,85,101,104] Five trials included subjects both in the subacute and the chronic stage.[14,46a,103,105,106]

Outcome Measures. Most of the studies reviewed measured outcomes at the impairment and activity limitation level. Typical instruments used to measure impairment level measures were the Action Research Arm Test (which measures upper limb dexterity), the Fugl-Meyer Assessment (which measures the ability of the arm to move against the typical synergistic pattern), the Wolf Motor Function Test (which quantifies motor function after stroke), PET scan, and Transcranial Magnetic Stimulation. Activity limitation was measured by measures such as the Rehabilitation Activities Profile (based on the ICF and, which assesses disability and handicap), Motor Activity Log (which measures actual amount of use and quality of movement), Barthel index, and the Functional Independence Measure (which measures activity limitation). Participation restriction was measured by administration of the Stroke Impact Scale in a few studies.

Results of the Review. The results are equivocal at present. CIMT is clearly better than the Bobath approach either in the subacute or chronic stages. When compared with usual care or conventional functional OT and PT, CIMT is more beneficial in studies where the CIMT group received a higher dosage of therapeutic intervention. When compared with dose equivalent functional training, CIMT does not seem more effective. The one exception was the study by Taub and colleagues[85] who found CIMT to be more effective than general fitness. However, the results of this study have to interpreted with caution as there were differences between groups at baseline, and subjects were not randomized. In the acute stage, CIMT is no more effective than dose equivalent functional OT treatment. Higher dose CIMT was less beneficial as compared with low dose CIMT.[19] In the subacute stage, when CIMT is compared with dose equivalent functional training, both interventions result in similar improvement (see Table 6-3).

Clinical Implications. CIMT appears to be a beneficial approach, but future studies need to compare CIMT with dose equivalent functional task-oriented training, which is shown to be effective. Such a study may address the criticism that the improvements demonstrated are due to a nonspecific effect of increased intensity of treatment rather than to a specific effect of constrained-induced training. Most of the studies reported in Table 6-3 used a standard CIMT training protocol in which training was massed over a period of two weeks and was compared with a control group that received less intense conventional training or ineffective traditional approaches, such as the Bobath approach. Studies using a modified CIMT protocol, while demonstrating some benefits, had the limitation of small sample size or comparison with traditional therapy known to be less effective (Bobath approach).

According to Taub and Uswatte,[84] the improvements seen with CIMT could be a result of massing of practice. Given that similar positive results have been obtained by increasing the intensity of traditional therapy, van der Lee[90] argues that using traditional therapeutic procedures that often may be less frustrating to patients than CIMT may be just as effective.

Robot-Aided Motor Training for Upper Limb Function

Rationale and Principles. A recent addition to the arsenal of techniques for stroke rehabilitation is the use of robotic manipulators for providing training of arm movements. Robot manipulators have been used successfully in experimental paradigms that attempted to elucidate the mechanisms underlying normal motor control and learning[74] and also to clarify mechanisms underlying disorders of upper limb movements in patients with movement disorders.[76]

The rationale for using a robotic device in rehabilitation is to decrease the labor-intensive nature of therapy and to provide a device that could be used for quantitative evaluation and treatment.[40] Proponents of this approach contend that current therapeutic evaluations are usually subjective and that therapists spend much time on one-on-one interaction with patients. The idea is to have devices available at rehabilitation centers for use when the patient is not in therapy sessions. Given that patients spend a large percentage of time outside therapist interaction, an attempt at facilitating practice during this time

should be beneficial. Robot-assisted training attempts to provide intensive practice of repetitive and stereotyped movements. See Chapter 11 for a full discussion of this topic.

Outcome Studies. A review of studies testing the effectiveness of robot assisted training revealed ten RCT, as listed in Table 6-4. Typical training with this approach involves the patient making horizontal plane movements while grasping the handle of the robot manipulator. Target locations and patient movement are displayed on a computer screen in front of the patient. Typically, patients are trained to produce movements of the shoulder and elbow joints while the wrist and hand joints and the trunk are immobilized with restraints. The robot is typically programmed to either passively move the paretic limb or produce an assistive force during movements. The number of sessions (12 to 60) and the total training time (eight hours to 300 hours) varied tremendously across studies.

Timing of Intervention. Two studies tested patients in the acute stage,[52,68] four studied patients in the subacute stage,[20,28,50,93] and four studies tested patients at the chronic stage.[15,33,49,95]

Outcome Measures. Most of the studies reviewed measured outcome variables at the impairment and activity limitation levels. The exceptions were studies that tested outcomes only at the impairment level.[15,28] Typical instruments used to measure impairments included the Fugl-Meyer Assessment, Action Research Arm Test, Trunk Control Test, and kinematic analysis of arm movement. Instruments used to measure activity limitation were the Functional Independence Measure, Chedoke-McMaster Stroke Scale, and the Barthel index.

Results of the Review. The results of effectiveness of robot assisted training are fairly clear; when compared with robot exposure,[20,92] traditional therapy using the Bobath approach[49,50,52] or neuromuscular facilitation,[15,28] robot training is more effective in improving function. This result can be explained by the fact that subjects in the robot groups received more training of upper limb movements compared with the control groups. However, when robot assisted training is compared with dose equivalent functional training, robot assisted training offers no additional benefits.[33,68,95]

Clinical Implications. The results highlight that robot assisted training offers no advantage to functional training with a therapist. Its effectiveness is limited to studies where robot assisted training was compared with traditional approaches that have been shown to be ineffective (such as the Bobath approach). Given the expense and extensive training of personnel to use the robot device, and its limited effectiveness, it may be beneficial to think of testing robotic devices as an adjunct to therapy rather than as a primary method of therapy delivery. Before additional RCT are implemented, the rationale and experimental procedures need to be clarified. For instance, at present, robot training provides practice of pointing movements (movements of the shoulder and elbow) on the horizontal plane. In an effort to isolate movements to these two joints, the trunk and distal extremities are often stabilized by constraints producing rather unnatural conditions for practice of arm movements. Functional reaching movements involve coordinated movement of the trunk-arm complex and of the wrist-hand complex. Whether practice of isolated components of the shoulder-elbow complex would transfer to real-world situations is unclear, given the task-specific nature of transfer of training. The responsibility of therapists is to select appropriate, challenging functional tasks, vary task parameters, progress to more difficult tasks, and test for transfer. Given the complexity of therapeutic training, robot manipulators can perhaps serve best by providing quantitative evaluation of impairments rather than as a therapeutic tool.

Body Weight Support and Treadmill Training to Improve Gait

Rationale and Principles. Approximately half the individuals who suffer a stroke do not recover their ability to walk independently.[32] Given that independent walking is a necessary prerequisite to successful community reintegration, not surprisingly gait training has occupied an important role in therapeutic practice following stroke. Gait training following stroke involves practice of individual segments of walking, practice of walking over ground with assistance of therapists and/or assistive devices, or more recently, practice of walking on a treadmill with partial body weight support.

Experiments on animals have shown that the basic neural circuitry for producing the rhythmic alternating movements of the lower limb is at the spinal cord level. Locomotor training with weight support of the hindlimbs has been shown to improve gait to near normal levels in cats whose spinal cords have been transected at thoracic levels, thereby isolating lower cord segments from the rest of the central nervous system.[2] In fact, patients with spinal cord injury have been shown to improve after treadmill training with body weight support.[99] Apart from the limited early evidence of the benefit of treadmill training in patients with spinal cord injury, the rationale for this approach is that it removes some of the biomechanical and equilibrium constraints of weight-bearing and facilitates walking by activation of spinal locomotor circuits. See Chapter 15.

Outcome Studies. A review of studies testing the effectiveness of body weight support training revealed six RCT listed in Table 6-5. Typical training with this approach involves beginning gait training on a treadmill by

Text continued on p.152

Table 6-4

Evidence Table for Robot-Assisted Therapy

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Volpe et al, 2000	Test whether additional robotic training of the paretic limb enhances motor outcome in subacute stroke	RCT; 56 patients in sub-acute stage 2b; 7/10	Robot training involved pointing to a series of targets using motion at shoulder, elbow, or both joints; 1 hours, 5 days a week for 5 weeks	Robot exposure; 1 hour/week on robot	Beginning and end of training (5 weeks)	1. FM 2. MSS 3. Motor Power Score 4. FIM	Experimental group had better motor outcome related to shoulder and elbow movements and better FIM scores than control group	Robot training group had more training. Results show limited improvement in function. No follow-up
Lum et al, 2002	Compare robotic training with conventional PT (NDT) in chronic stroke	RCT; 27 subjects, chronic hemiparesis (>6 months), 2b, 6/10	Standard rehabilitation and robot-aided therapy that included pointing movements involving shoulder and elbow joints, 1 hour session/day for 24 days	Standard rehabilitation (Bobath approach) for 55 minutes and robot exposure for 5 minutes at each session; 1 hour session/day for 24 days	Beginning of training, one month, end of training (2 months) and 6 month follow-up	1. FM 2. BI 3. FIM (self-care and transfer sections) 4. Strength measured through force transducer 5. Reaching kinematics	Robot-assisted group had larger improvements on proximal FM at 1 and 2 months and higher FIM scores at 6 months	Small sample size, robot training better than Bobath approach at improving proximal movements
Fasoli et al, 2004	Examine effects of robotic training in subacute stroke	RCT retrospective, 56 patients with sub-acute stroke (>3 weeks); 2b; 6/10	Robot training group (N= 30) received passive or active assistive practice of planar arm movements involving the shoulder and elbow joints with the MIT-Manus; 1 hour/day, 5 days/week for 5 weeks	Robot exposure group (N=26) received training for 1 hour/week; subjects practiced planar arm movements without assistance	Beginning of training, during training and at discharge	1. FM 2. MSS 3. MRC test of motor power 4. FIM	Robotic group performed better on the FM, Motor Status Score, and MRC test of motor power; both groups improved scores on FIM; no differences were seen across groups	Control group did not receive dose equivalent practice; robotic therapy better than robotic exposure on impairment level measures

Daly et al, 2005	Compare effects of task-oriented plus robotic therapy in chronic stroke patients	RCT; 12 chronic stroke patients (>12 months) 2b; 5/10	In-motion robot training (1.5 hours/day) focused on shoulder and elbow movement accuracy and smoothness. Subjects also practiced functional upper limb tasks (3.5 hours/day). Training for 5 hours/day, 5 days/week for 12 weeks.	Functional neuromuscular stimulation (1.5 hours/day) involving wrist and finger activation. Subjects also practiced functional upper limb tasks (3.5 hours/day). Training for 5 hours/day, 5 days/week for 12 weeks.	Beginning of training, end of training (12 weeks) and 6 month follow-up	Baseline, end of treatment and 6 month follow up 1. AMAT functional ability 2. AMAT shoulder-elbow 3. AMAT wrist-hand 4. FM coordination scale 5. Target accuracy 6. Movement smoothness	The robot group showed improvement on the AMAT, the AMAT shoulder-elbow, FM, and movement accuracy and smoothness; the stimulation group improved on AMAT wrist-hand	Small sample size; addition of robot training to functional training is beneficial for improving performance at the impairment level.
Hesse et al, 2005	Compare computerized AT with ES in subacute stroke patients	RCT; 44 subacute stroke patients (4< 8 weeks); 1b; 7/10	Standard rehabilitation based on Bobath approach (45 minutes of PT, 30 minutes of OT), plus practice with arm trainer for pronation-supination and wrist flexion and extension movements; 20 min/day, 5 days/week for 6 weeks	Standard rehabilitation based on Bobath approach (45 minutes of PT, 30 minutes of OT), plus electrical stimulation of wrist extension movements; 20 min/day, 5 days/week for 6 weeks	Beginning of training, end of training (6 weeks) and 3-month follow-up	1. FM upper extremity 2. MRC Scale 3. MAS	AT group had higher BI score at baseline; FM, MRC scores improved more for AT group	Well-designed study; no functional outcomes measured; additional robot training improved motor performance

Continued

Table 6-4

Evidence Table for Robot-Assisted Therapy—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Lum et al, 2006	Compare unilateral and bilateral robotic training with conventional PT in subacute stroke	RCT; 30 subacute patients (1 to 5 months poststroke); 2b; 4/10	Three groups of robot training; unilateral, bilateral or combined group had 50 minutes of robot training; 1 hour/day for 4 weeks	Conventional therapy (Bobath approach); 1 hour/day for 4 weeks	Beginning of training, end of training (4 weeks) and 6 month follow-up	1. FM upper extremity 2. MSS 3. FIM 4. Motor Power exam	Baseline differences in MAS and MSS scores; combined robot group better scores on FM and MSS score at 4 weeks but not at 6 months; no improvement on FIM	Small sample size per group; baseline differences; no functional improvement
Kahn et al, 2006	Examine effects of active-assistive robot training in chronic stroke	RCT; 19 patients with chronic stroke (>1 year); 2b; 4/10	Active-assistive robot training including reaching to different directions; 24 sessions (45 min) over 8 weeks	Training of free reaching (unassisted) movements; 24 sessions (45 min) over 8 weeks	3 tests before training; 3 tests after training; 6 month follow-up	1. Pointing movement outcomes including stiffness, range, speed, smoothness and straightness 2. Chedoke McMaster score 3. Rancho Los Amigos Functional Test time to completion	No baseline differences between groups; both groups improved ROM speed; smoothness better for control group	Small sample size; no benefit of robot training over practice of reaching movements. No functional outcomes tested

Masiero et al, 2007	Examine effects of additional early robotic therapy on impairments and functional recovery	RCT; 35 patients with acute stroke (≤1 week); 1b; 6/10	Standard PT and OT (Bobath treatment); additional training with NeRebot, active assisted shoulder and elbow movements; 4 hours/week for 5 weeks	Standard PT and OT (Bobath treatment); additional robot (NeRebot) exposure; 1 hour/week for 5 weeks	Beginning of training, end of training (5 weeks); 3 and 8 month follow-up	1. MRC score 2. FM 3. FIM 4. Trunk control test 5. MAS	Robot training well tolerated; robot group performed better on FM, FIM, proximal MRC scores at the end of training; on follow up benefits sustained on FM, MRC deltoid and FIM	Robot group had higher baseline FIM score; control group had less exposure than experimental group; robot therapy may complement early rehabilitation
Rabadi et al, 2008	Determine the effect of activity based therapy using either an ergometer, robotic device or occupational therapy in acute stroke	RCT; 30 subjects with acute stroke (<4 weeks); 2b; 6/10	Robot group (N=10) used the MIT-Manus to practice passive and active assistive planar movements involving the shoulder and elbow joints; 1024 movements in 40 min session, 5 days/week for 12 days Ergometer group (N=10) used a bidirectional pedal for aerobic exercise of the upper limb; 2200 movements in 40 min session, 5 days/week for 12 days. Both groups received, in addition, standard rehabilitation for 3 hours/day.	Control group (N=10) received OT including ROM and active movements during functional activity; 640 movements in 40 min session, 5 days/week for 12 days, in addition to standard rehabilitation for 3 hours/day.	Beginning and end of training	1. FM 2. MSS 3. FIM total 4. FIM motor 5. FIM cognitive	All three groups improved on FM, MSS, and FIM scores. No differences were seen across groups. OT group had better scores on the FIM and FM compared with ergometer and robotic groups	Robotic therapy not better than functional OT training in acute stroke

Continued

Table 6-4

Evidence Table for Robot-Assisted Therapy—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Volpe et al, 2008	Compare intensive PT with robotic training in chronic stroke	RCT; 21 patients with chronic stroke (>6 months); 2b; 6/10	Robot group (N=11) used the MIT-Manus to practice passive and active assistive planar movements involving the shoulder and elbow joints; 1 hour/day, 3 days/week for 6 weeks	Therapy group (N=10) practiced active assistive and goal directed functional arm movements; treatment based on motor learning approach; 1 hour/day, 3 days/week for 6 weeks	Pretreatment and posttreatment, 3 month follow-up	1. FM Shoulder elbow 2. FM wrist hand 3. MAS 4. SIS 5. ARAT	Both groups improved over duration of treatment and maintained improvement at 3 months; no differences between groups	Robot training no better than task-oriented training in chronic stroke

AMAT, Arm Motor Ability Test; ARAT, Action Research Arm Test; AT, arm trainer; BI, Barthel index; ES, electrical stimulation; FIM, Functional Independence Measure; FM, Fugl-Myer Assessment; MAS, Modified Ashworth Scale; MIT, Massachusetts Institute of Technology; MRC, Medical Research Council score; MSS, Motor Status Score; NDT, neurodevelopmental treatment; OT, occupational therapy; PT, physical therapy; RCT, randomized controlled trial; RMA, Rivermead Motor Assessment; ROM, range of motion; SIS, Stroke Impact Scale;

Table 6-5

Evidence Table for Treadmill Training with Body Weight Support

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, AND PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Nilsson et al, 2001	Compare walking training over ground (based on a motor relearning approach) with treadmill training in subacute stroke stage	Double blind RCT; 73 patients at subacute stage (<8 weeks); 1b; 7/10	Treadmill training with BWS (N= 36); 30 minutes/day 5 days/week for 2 months	Walking training (N= 37); 30 minutes/day 5 days/week for 2 months	Pretreatment and post-treatment, 10 month follow-up	1. FIM 2. FM 3. FAC 4. Walking velocity (10 meters) 5. BBS	Both groups improved performance on the FIM, walking velocity, FAC, and balance. No differences were seen across groups.	Good study RCT 10-month follow-up; no benefit of BWS training
da Cunha et al, 2002	Compare BWS treadmill training and typical therapy with only typical therapy	RCT; 13 patients in subacute stage (<6 weeks) 20 minutes/day 5 days/week for 3 weeks 2b; 4/10	Supported treadmill training group (N=6) received regular PT which included stair climbing and walking on uneven surfaces and supported treadmill training; 3 hours/day of total therapy; 20 min/day treadmill training	Control group (N=7) received regular PT, OT, which included gait training and stair climbing; 3 hours/day of total therapy;	Pretreatment and post-treatment	1. FAC 2. Gait speed 3. Walking distance 4. Energy expenditure	Differences were seen in walking energy cost and walking distance. No differences were seen for other outcome measures.	Small sample size; no follow-up; BWS not more effective than regular therapy

Continued

Table 6-5

Evidence Table for Treadmill Training with Body Weight Support—cont'd

AUTHORS AND YEAR	AIMS AND RATIONALE	DESIGN, SUBJECTS, OXFORD RATING, AND PEDRO SCORE	INTERVENTION	COMPARISON INTERVENTION	ASSESSMENT	OUTCOME MEASURES	RESULTS	COMMENTS
Werner et al, 2002	Compare BWS treadmill training with electromechanical gait trainer in subacute stroke patients	RCT crossover trial; 30 patients in subacute stage (4 to 12 weeks); 2b; 7/10	BWS treadmill training (N=15); 15 to 20 min/day × 7 days/week × 6 weeks.	Electromechanical gait trainer (N=15); 15 to 20 min/day × 7 days/week × 6 weeks.	Pretreatment, 1, 2, 3, 4, 5 weeks of treatment, posttreatment, 6 month follow-up	Measured at baseline, 6 wks and 6 months. 1. FAC 2. Gait Velocity 3. RMA 4. MAS	Both groups improved in 6 weeks; subjects in electromechanical trainer group had better FAC scores at 6 weeks; and required use of 1 therapist assistance; treadmill group required 2 therapist assistance; no differences between groups at 6 months.	Good study. electromechanical trainer as good as BWS treadmill training and requires fewer therapists.
Barbeau and Visintin, 2003	Compare treadmill training plus BWS with treadmill training without BWS in subacute stroke	Single blind RCT; 100 chronic subacute patients (1 to 5 months); 2b; 4/10	BWS (with 2 therapists) treadmill training with 40% of body weight support; 20 min × 4 days/week × 6 weeks	Treadmill training without BWS; 20 min × 4 days/week × 6 weeks	Pretreatment and posttreatment, 3 month follow-up	Baseline, end of training (6 weeks) and 3 month follow-up 1. BBS 2. STREAM 3. Overground walking speed; 4. Endurance (walk distance)	Both groups improved over 6 weeks; greater improvement seen in BWS group for severely impaired patients on all outcomes.	Moderate quality study shows that BWS may be appropriate for severely impaired patients in chronic stage

Sullivan et al, 2007	Compare BWS with lower extremity strength training in chronic stroke	Single blind RCT; 80 chronic stroke patients (4 months to 5 years); 1b; 7/10	BWS with 30% to 40% weight; Group 1: BWS + UE 2. BWS + UE 3. BWS + Lower extremity progressive resistive exercise; 1 hour session, 4 × week, 6 weeks	Limb loaded cycling + UE; 1 hour session, 4 × week, 6 weeks	Pretreatment and post-treatment, 6 month follow-up	Baseline, after 12 and 24 treatment sessions; 6 month follow-up; 1. Overground self selected walking speed 2. Fast walking speed 3. 6-minute walk distance 4. FM 5. SIS; 6. SF-36; 7. Lower extremity peak torque	BWS treadmill groups improved on self-selected and fast walking speed; whereas limb loaded cycle group improved only on 6 min walk distance and flexor torque after 24 sessions and at 6 month follow-up	High quality study; shows that task-specific BWS training beneficial for chronic stroke patients compared with resistive cycling.
Yen et al, 2008	Examine effects of additional BWS training on motor performance and cortical excitability	RCT nonblinded; 14 chronic stroke patients (>6 months); 2b; 7/10	50 min PT session (stretching, strengthening, balance, overground walking) 2 to 5 sessions/week for 4 weeks + BWS (30 min session 3 days/week for 4 weeks) with 1 to 2 therapists	50 min PT session (stretching, strengthening, balance, overground walking) 2 to 5 sessions/week for 4 weeks	Pretreatment and post-treatment	Baseline and post-treatment; 1. BBS 2. Gait analysis (GAITRite); 3. Cortical area and motor threshold using TMS	PT + BWS group improved their BBS score, gait speed and step length and decreased motor threshold; control group improved in gait speed and cadence.	Study shows that additional training using BWS improves balance, gait and cortical excitability. Did not test additional PT in control group, so improvements may be a result of nonspecific additional training

BBS, Berg Balance Scale; BWS, body weight support; FAC, Functional Ambulation Classification; FIM, Functional Independence Measure; FM, Fugl-Meyer Assessment; MAS, Modified Ashworth Scale; OT, occupational therapy; PT, physical therapy; RCT, randomized controlled trial; RMA, Rivermead Mobility Assessment; SIS, Stroke Impact Scale; STREAM, Stroke rehabilitation assessment of movement; TMS, Transcranial Magnetic Stimulation; UE, upper extremity ergometry.

supporting the body in a harness. The initial support given was generally 40% of the body weight, which is gradually decreased as the patient improves. The length of training ranged from 12 to 42 sessions conducted across four to eight weeks. In two of the studies, body weight support treadmill training was coupled with gait training.

Timing of Intervention. Treadmill training was initiated in the subacute stage in four studies[3,13,61,98] and in the chronic stage in two studies.[79,107]

Outcome Measures. Most of the studies reviewed measured outcome variables at the impairment and activity limitation levels. Only one study measured outcomes at all three levels of the ICF.[79] Typical instruments used to assess impairment level measures were the Stroke Rehabilitation Assessment of Movement (which evaluates voluntary movement of the limbs and mobility), Berg Balance Scale (which evaluates balance during sitting and standing activities), walking speed, distance and endurance, the Fugl-Meyer Assessment (which evaluates locomotor function and control, sensory quality, and balance), and kinematic analysis of walking. Instruments used to measure activity limitation were the Functional Independence Measure, Functional Ambulation Classification (which quantifies amount of assistance needed in walking), and the Rivermead Motor Assessment. Instruments used to measure participation limitation were Stroke Impact Scale and SF-36.

Results of the Review. When compared with functional training of ambulation or training with an electromechanical trainer, body weight supported treadmill training was no more effective, all interventions producing similar, but positive, outcomes. When compared with control groups that did not have training of walking, body weight support was more effective in outcomes related to walking and balance. When body weight support was added to the PT intervention, it was more effective.[107] However, this benefit may be the result of additional training since the control group did not receive dose equivalent therapy. The clearest evidence for the benefit of body weight support treadmill training was seen for severely impaired patients.[3]

Clinical Implications. The review suggests that training of walking and balance may be task-specific, and body weight support treadmill training may not be more effective compared with functional training without body weight support. When examined in the context of the high cost associated with body weight support apparatus, and the number of therapists required to administer therapy, functional training may be more cost-effective and equally beneficial. The only indication for body weight support training may be in the case of severely impaired patients who may benefit from relearning the walking movement patterns without being encumbered with controlling their body weight and forward progression.

SUMMARY

A challenging yet exciting period for stroke rehabilitation is occurring as occupational and physical therapists are being asked to provide training based on sound scientific principles and with demonstrated effectiveness. The lack of support for traditional neurotherapeutic approaches, such as Bobath approach, recent advances in understanding of motor control and dyscontrol, and emerging technologies have facilitated a second paradigm shift toward a functional task-oriented approach. At present, the literature suggests that task-oriented training of the upper limb and functional walking training is the most effective method in stroke rehabilitation. The challenge for the next decade is to develop more creative, functional, task-oriented intervention techniques that will maximize the independent functioning of patients within their natural contextual settings[10] and to test these techniques in a systematical manner at different stages of the recovery process, in different practice settings, and at different intensities. Most likely, no one technique will offer a panacea for stroke rehabilitation given the varied nature of impairments and activity limitations.

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REVIEW QUESTIONS

1. What is evidence-based practice?
2. What are the principles of evidence-based practice?
3. What are the criteria for reviewing articles on treatment outcomes?
4. Describe the most common research designs used in outcome studies.
5. What are some of the basic principles of neurotherapeutic approaches?
6. Is there evidence to support the application of neurotherapeutic approaches?
7. What are some of the basic principles of the functional task-oriented approach?
8. Describe the evidence to support the task-oriented approach, CIMT, treadmill training and body weight support, and robot-assisted training.

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