FOURTH EDITION

STROKE REHABILITATION

A Function-Based Approach

Glen Gillen

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To the hundreds of stroke survivors
that I have interacted with over the past 26 years.

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Preface

The fourth edition of *Stroke Rehabilitation: A Function-Based Approach* continues to strive to be the most up-to-date text on this topic, incorporating state-of-the-art and evidence-based tools and techniques to maximize function and quality of life for those living with stroke. This edition's contributors include expert clinicians, researchers, and scientists from across the globe. Contributors are experts in various disciplines, including neurology, occupational therapy, physiatry, physical therapy, psychology, and speech and language pathology.

The current text combines aspects of background medical information, a comprehensive review of standardized and nonstandardized evaluation procedures and assessments, and evidence-based interventions. It contains the most up-to-date research on stroke rehabilitation from a variety of rehabilitation settings and professions without losing its holistic perspective on the overall care of the people whose lives we as clinicians touch.

This text has overarching themes. First and foremost, clinicians are provided with specific suggestions to maintain a client-centered approach when working with stroke survivors. Furthermore, clinicians are challenged to use the most up-to-date treatment approaches (including both remediation and adaptation approaches) to decrease impairments; prevent secondary complications; improve the client's ability to perform meaningful activities; and, most important, decrease participation restrictions and improve quality of life.

Although this book is written primarily by occupational therapists, it is an appropriate reference for a variety of rehabilitation professionals, including physiatrists, physical therapists, speech and language pathologists, rehabilitation nurses, social workers, vocational counselors, and therapeutic recreation specialists. The immense value of an interdisciplinary team approach when working with the stroke survivor population cannot be overestimated. This text may also be beneficial to therapists who practice virtually alone in the community or as case managers because its research on the specific topic of stroke rehabilitation is comprehensive. The terms *patient* and *client* have been used interchangeably; it is recognized that stroke rehabilitation can take place in multiple settings.

Educators and students can use this text in the classroom setting. Key terms, chapter objectives, review questions, and case studies have been provided as learning tools. A text that can appeal to basic learners and specialists alike, this book is a good investment for any clinician who plans to work with neurologically impaired persons—specifically, adults who have had a stroke. This text spans the continuum of care—from acute to long-term management—in a variety of roles and settings.

This edition has been fully reorganized to place a clear focus on resuming everyday living skills as the outcome of therapy. **Part I: Foundations of Stroke Rehabilitation** includes five chapters that provide the necessary medical and therapeutic foundations that should be the basis of any intervention plan. The first chapter not only includes medical management but also a comprehensive approach to acute stroke rehabilitation because current practice dictates that rehabilitation services begin within 24 hours of stroke in many cases. Acute care evaluations and interventions are clearly delineated for those working in intensive care units, step-down units, and the acute hospital settings. Chapter 2, "Improving Participation and Quality of Life through Occupation," is included early in the text to remind clinicians that maximizing participation in life is the overarching goal of stroke rehabilitation. Chapters 3, "Task-Oriented Approach to Stroke Rehabilitation," and 4, "Activity-Based Intervention in Stroke Rehabilitation," provide readers with an overall view of current therapeutic approaches and should be understood before the chapters on specialized topics are read. Finally, Chapter 5, "Client Centeredness: A Survivor's Perspective" is also included early in the text to remind clinicians about the human experience of surviving a stroke.

Part 2: Maximizing Participation in Everyday Activities serves to provide clinicians with strategies to maximize involvement in specific aspects of daily living after a stroke, such as driving and community mobility, sexuality, leisure, instrumental activities of daily living, resumption of parenting roles after stroke, gait and mobility, return to work, and self-care. A new chapter on caregiving after stroke has been included in this section to remind clinicians that a team approach includes care partners.

Part 3: Maximizing Outcomes for Specific Problem Areas Following Stroke includes standardized evaluations and evidence based intervention for a variety of post-stroke challenges, including loss of motor control related to upper limb control, trunk control, and balance. Chapters related to robotic technology, orthotic interventions, vestibular rehabilitation, and edema control are included to promote a comprehensive overall approach to managing impaired motor control. Part 3 also includes chapters on other challenging impairments that interfere with daily living such as visual and visuospatial involvement, cognitive impairments, perceptual dysfunction, and speech and language deficits. Finally, this section includes detailed chapters related to dysphagia management, seating and wheeled mobility prescription, and home evaluation and modifications.

This fourth edition also includes a robust web-based platform. This platform contains two additional chapters,

"How Therapists Think: Exploring Clinicians' Reasoning When Working with Patients Who Have Cognitive and Perceptual Problems Following Stroke" and "A Survivor's Perspective II: Stroke." This platform also contains additional material to support Chapter 27, "Interventions for Cognitive-Perceptual Deficits: A Function-Based Approach." This material addresses the areas of decreased self-awareness and insight, apraxia, unilateral neglect, agnosias, attention deficits, memory impairment and executive dysfunction in depth including samples of standardized assessment and evidence based interventions. Instructor resources include an Instructor's Manual, PowerPoint Slides, an Image Collection, and a Test Bank. Additional student resources include chapter activities, references linked to MEDLINE, and a glossary of terms.

It is my hope that this text will challenge practicing clinicians to consider their present approaches to stroke rehabilitation and serve as a foundation on which students can build their philosophies for intervention with the stroke population. It is my hope that this text will make a positive impact on improving the quality of life of those living with stroke.

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I am grateful to all of the professionals from my own community, across the country, and internationally for their contributions to this book. It literally takes a village! They accepted my challenge to put their knowledge and skill base into words. Their dedication to this project will inspire future generations of clinicians and researchers.

I continue to appreciate the dedication and persistence of the staff at Elsevier for supporting my work for almost 20 years, specifically Brandi Graham and Jolynn Gower.

Glen Gillen

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part

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Foundations of Stroke Rehabilitation

chapter 1

Pathophysiology, Medical Management, and Acute Rehabilitation of Stroke Survivors

Matthew N. Bartels; Catherine A. Duffy; Heather Edgar Beland

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 intensive care unit (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.

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 rates 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 age 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 elderly adults. Another cause of decreased mortality rates 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

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

comprise two broad categories: ischemic and hemorrhagic stroke. Ischemic strokes account for approximately 80% of strokes, and 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. Because 35% of patients who have had a TIA will have a stroke within 5 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, surgery, or both.

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.

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 is 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 myocardial infarction (MI).[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 hematologic 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 depend 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 Figures 1-1 to 1-3 explain the anatomy of

TABLE 1-2 Common Stroke Syndromes

Anatomic Distribution	Stroke Syndrome
Common carotid artery	Often resembles 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 (MCA)	
Main stem	Contralateral hemiplegia Contralateral hemianopia Contralateral hemianesthesia Head or eye turning toward the lesion Dysphagia Uninhibited neurogenic bladder Dominant hemisphere Global aphasia Apraxia Nondominant hemisphere Aprosody and affective agnosia Visuospatial deficit Neglect syndrome
Upper division	Contralateral hemiplegia; leg more spared Contralateral hemianopia Contralateral hemianesthesia Head or 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 or 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
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 or 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 or 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

these strokes. Atherosclerotic disease is screened most readily by carotid Doppler ultrasonography and transcranial Doppler imaging. Magnetic resonance angiography (MRA) and carotid and cerebral angiography can further elucidate lesions, which can be treated surgically or medically.

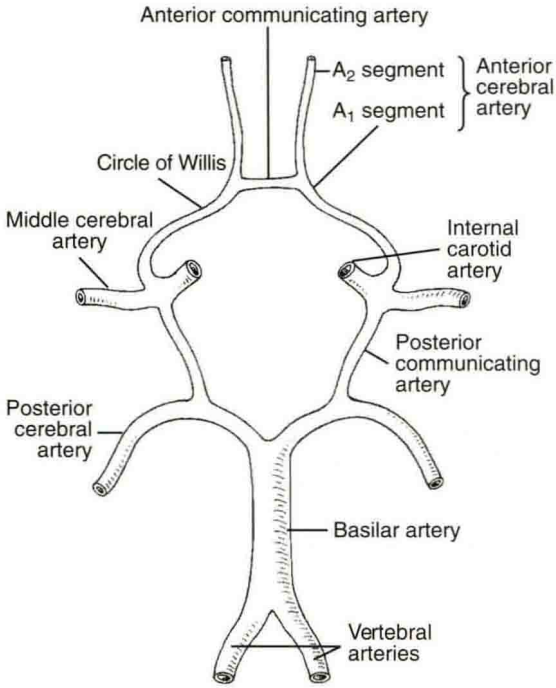

Figure 1-1 Circle of Willis and cerebral circulation.

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 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

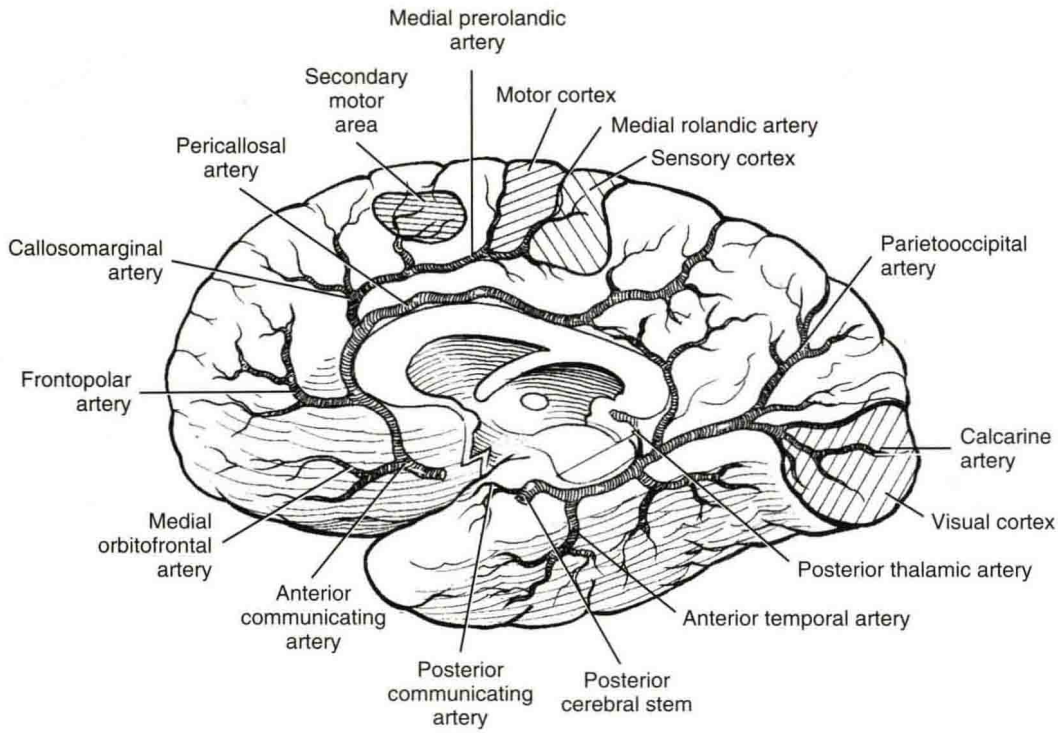

Figure 1-2 Medial view of brain with anterior and posterior cerebral artery circulation and areas of cortical function.

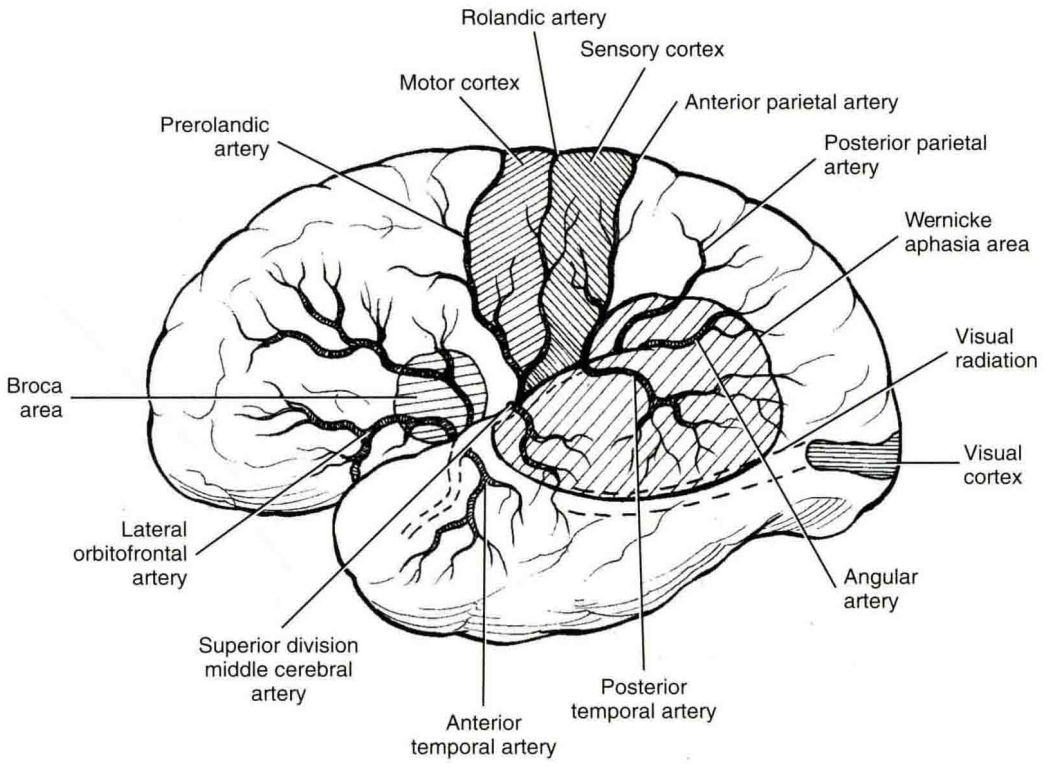

Figure 1-3 Lateral view of brain with middle cerebral artery and its branches and areas of cortical function.

TABLE 1-3 Lacunar Stroke Syndromes and Their Anatomic Sites

Lacunar Syndrome	Anatomic 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 or clumsy hand	Base of pons Genu of anterior limb of the internal capsule
Sensory-motor	Junction of the internal capsule and thalamus
Anarthria pseudobulbar	Bilateral internal capsule

signs known as pseudobulbar palsy will develop. This diagnosis is based on the symptoms and the use of computed 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 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, pons, thalamus, and cerebellum. Usually these hemorrhages develop from small penetrating arteries in