**Background**

Middle cerebral artery stroke describes the sudden onset of focal neurologic deficit resulting from brain infarction or [ischemia](http://emedicine.medscape.com/article/1916852-overview) in the territory supplied by the middle cerebral artery (MCA).

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

Research has also focused on the correlation between specific neurologic deficits after [MCA stroke](http://emedicine.medscape.com/article/324386-overview) and differing outcomes and prognoses. Such efforts are important in ascertaining who may benefit from emergent antithrombotic therapies. Furthermore, these research efforts may later allow physiatrists to target rehabilitative efforts more effectively in appropriately selected patients who may derive benefit.

The medical management of acute stroke is not discussed in detail in this article. For more information on stroke, see [Hemorrhagic Stroke](http://emedicine.medscape.com/article/1916662-overview) and [Ischemic Stroke](http://emedicine.medscape.com/article/1916852-overview).

**Etiology of MCA Stroke**

The main causes of stroke include ischemia, cardioembolism, hypercoagulable states, hemorrhage, [hypertension](http://emedicine.medscape.com/article/261435-overview), and amyloid or arteriovenous malformation.

**Embolism**

Estimates suggest that 15-30% of all strokes are thought to be of embolic etiology. The remaining cases have either an undetermined or a combined etiology or else are caused by dissection. Most of the sources in the literature support [embolism](http://emedicine.medscape.com/article/300901-overview) as the primary etiology of middle cerebral artery (MCA) strokes.

Specifically, in a study by Moulin and coauthors, cardioembolism accounted for approximately 50% of total MCA strokes, 34% of deep MCA strokes, and 41% of cortical strokes.[[2]](javascript:showrefcontent('refrenceslayer');) This same study, with a relatively large cohort, suggests that cardioembolism may be greatly underdiagnosed and may play a more common role in posterior and anterior cerebral strokes than previously thought.

The study also revealed paroxysmal [atrial fibrillation](http://emedicine.medscape.com/article/151066-overview) in 65% of all stroke patients studied. This cardiac abnormality may be a common poststroke sequela, but its frequent occurrence certainly supports the need for cardiac monitoring and a high index of suspicion for cardioembolism in formulating a complete differential diagnosis.

Additionally, embolic strokes can occur through the atheromatous plaques of carotid disease. All of these data support widely accepted diagnostic studies, including carotid Doppler studies, transesophageal echocardiography studies, and telemetry to elucidate and treat pathology and prevent future embolic events.

The location of MCA stroke depends largely on the size of the embolic mass. Occlusion at the stem is rare and requires embolic matter of at least 3-5 mm. Emboli can arise because of intravascular, rigid foreign matter (eg, shotgun pellets, catheter tips, large thrombi combined with bacteria) or as a result of large calcific plaques formed through direct internal carotid trauma or puncture.

The etiology of occlusion of smaller and surface branches obviously is more diverse and most commonly involves cardiogenic emboli or material from an ipsilateral site of carotid atherosclerosis. Other sources of emboli include spontaneous dissection of a carotid artery, material from breast metastasis, a marantic embolus, fungal endocarditis, and paradoxical emboli due to a patent foramen ovale.

Embolization occurs with equal frequency in the right and the left MCA. Angiography reveals that these occlusions are usually found in the first 24 hours, but the vessels are generally patent within 48 hours. A persistent occlusion has a less favorable prognosis. The size of the infarct also depends on the collateral circulation, which is highly variable as a result of congenital vascular patterns and collateral vascular development secondary to long-standing atherosclerosis.

**Indirect ischemia**

Distal territories of the MCA are quite vulnerable to ischemia because of failure of [perfusion](http://emedicine.medscape.com/article/1162437-overview) due to arrhythmia and other causes of hypotension. Such compromise in circulation is especially significant in patients with carotid artery stenosis. The prevalence of strokes of this type is uncertain, but they are not thought to be uncommon, given the high correlation of carotid stenosis with distal territory stroke.

**Atherosclerosis**

[Primary atherosclerosis](http://emedicine.medscape.com/article/1612610-overview) of the MCA and branches accounts for only 7-8% of symptomatic MCA disease. Furthermore, many of these cases probably represent recanalized embolism and not true atherosclerosis.

**Thrombosis**

Although thrombotic occlusion of small and large vessels is still widely accepted as the primary etiology of strokes in general, causing approximately 51% of all strokes in the anterior, middle, and posterior cerebral vasculature combined, it is a relatively rare cause of MCA strokes. Only about 2-7% of ischemic events in the MCA territory are due to thrombotic occlusion. The diagnosis can be excluded using repeat angiography, but this is of questionable utility.

**Other causes**

Amyloid angiopathy is a rare etiology for lobar cortical strokes in elderly patients. Dissection and stenosis of the MCA are rarely documented as causes of MCA stroke.

**Etiology in younger persons**

Hemorrhagic stroke is the most common etiology in younger persons (aged 18-45 y), with intracranial hemorrhage accounting for 41% and subarachnoid hemorrhage accounting for 17% of strokes in persons in the younger age group. The remaining 42% of strokes due to ischemia generally require a more exhaustive workup to elucidate an etiology. Consider carotid or vertebral dissection, collagen-vascular disease, and [coagulopathies](http://emedicine.medscape.com/article/955059-overview).

Studies reveal that dissection is an underrecognized cause of stroke in younger populations. Still, even with advances in diagnostic options, 20% of strokes in younger persons continue to be of unknown etiology.

## Clinical Presentation of MCA Stroke

### Basic physical findings

Patients with middle cerebral artery (MCA) stroke syndrome may have some basic physical findings, as follows.

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

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

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

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

### Specific neurologic sequelae

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

Motor deficits (hemiparesis and hemiplegia) may become apparent. Surprisingly, assigning clear-cut syndromes of weakness to specific territories of MCA infarct has posed a significant challenge. The prognosis of such motor deficit also has not completely been elucidated, with case reports of remarkable recovery from dense limb involvement.

Partial hemiparesis patterns have been mapped more readily to certain MCA territory infarcts. The National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) data bank project gathered pilot data from 488 patients with unilateral hemisphere strokes.[[8]](javascript:showrefcontent('refrenceslayer');) The following conclusions arose from the analysis of the project data:

* Equivalent weakness of the hip, foot, shoulder, and hand was the most common finding among the patients in the NINCDS project, accounting for 71.2% of cases.
* Hemiparesis with distal predominance describes another 23.5% of cases, with weakness of the lower face, lower legs, toes, fingers, and forearm and sparing of the forehead and proximal muscles of the upper and lower extremities. The resultant deficit is believed to be due to the large representation of the affected muscles in the homunculus.

Faciobrachial paresis describes weakness of the lower face, jaw, tongue, oropharynx, and ipsilateral upper extremity. The weakness of the upper extremity is often more pronounced in the distal musculature of the hand and forearm.[[9, 10]](javascript:showrefcontent('refrenceslayer');) These deficits result from ischemic insult of the insula and operculum.

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

Visual deficits are common. Hemianopia has long been known to accompany the syndrome following a large MCA infarct; yet, only the superior portion of the optic radiation is supplied by the MCA. The resultant hemianopia is probably due to a massive infarct with subsequent edema affecting adjacent structures. Quadrantanopsia can be attributed to a parietal infarct affecting the deep fibers of the upper optic radiation; however, this condition is rare.

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

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

Motor neglect with underuse of the side contralateral to the cerebral insult appears much like a hemiparesis. Special efforts must be made by the examiner to encourage the patient to demonstrate strength and dexterity.[[11]](javascript:showrefcontent('refrenceslayer');) Typically, the patient has delayed withdrawal to noxious stimuli, fails to place the affected hand in the lap when seated, and falls heavily to the affected side with no apparent effort to minimize impact.

Autonomic dysfunction after MCA stroke often can be evidenced by contralateral edema of the hand and foot arising within hours of the infarct and lasting up to 2 weeks. This edema is in contrast to the dependent edema that develops subacutely in the distal aspect of a plegic extremity. Excessive sweating contralateral to the territory of an MCA stroke can be indicative of a larger lesion, affecting deep and superficial branches.

### Manifestations of left (dominant) hemisphere infarction

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

The specific manifestations of left hemisphere infarction largely fall under the heading of either [aphasia](http://emedicine.medscape.com/article/1135944-overview) (or dysphasia) or [apraxia](http://emedicine.medscape.com/article/1136037-overview) (dyspraxia).

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

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

Broca aphasia, also termed expressive or motor aphasia, describes the ability to comprehend written and spoken language, with nonfluent or impaired expression of either spoken or written language. The infarct responsible for Broca aphasia encompasses the insula and frontoparietal operculum.

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

Wernicke aphasia, also termed receptive or sensory aphasia, is caused most often by occlusion of the lower division of the MCA bifurcation or one of its branches. The infarct responsible for a classic Wernicke aphasia includes the dominant posterior temporal, inferior parietal, and lateral temporo-occipital regions.

Unlike patients with motor aphasia, patients with Wernicke aphasia vocalize smoothly and with expression, but they demonstrate paraphasias or speech with distorted phonetic structure, word substitution, and additional prefixes and suffixes. Their speech is fluent but is often missing key words and ideas and may be perseverative. Patients demonstrate pure-word deafness, with the inability to repeat words, along with alexia, the inability to recognize or comprehend written language.

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

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

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

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

refers to the inability to perform a previously learned task despite preserved strength, vision, and coordination. When referring to apraxia, Mohr states, “Motor engrams (programs) that guide skilled acts have either been lost or cannot be accessed. Generally, the ability is impaired rather than eliminated; thus, the term dyspraxia is more appropriate.

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

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

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

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

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

In the context of speech disturbances, the term dyspraxia is used to describe impaired cooperation of the oropharyngeal and respiratory elements necessary for speech. Individuals with this condition have a hesitant and somewhat telegraphic verbal response.

### Manifestations of right (nondominant) hemisphere infarction

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

The term extinction is used to describe inattention to one stimulus when 2 stimuli are presented simultaneously. Generally, the ignored stimulus is on the left side.

The term neglect is used to describe “a lack of responsivity to stimuli on one side of the body, in the absence of any sensory or motor deficit severe enough to account for the imperception.”In a stroke population studied by Battersby and coauthors, such unilateral neglect occurred in 29% of patients with right-sided brain damage versus 12% of patients with left-sided brain damage.

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

The term impersistence is used to describe an inability to persist in performing motor tasks; it is often accompanied by visuomotor and visuospatial deficits.[[20, 21]](javascript:showrefcontent('refrenceslayer');) This impairment places the patient at risk for an unfavorable rehabilitation outcome.

The term dressing apraxia applies to a condition in which the patient is unable to dress without assistance, despite having no apparent hemiplegia that would prevent the performance of this function. It is a much more common finding in cases of right-hemisphere infarcts and is attributable to difficulty distinguishing right from left and up from down.

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

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

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

The term constructional apraxia describes a difficulty in manipulating objects in space. This type of apraxia can be appreciated by having affected patients copy designs or build 3-dimensional models. This tendency is more common with right-sided lesions than with left-sided lesions, as is evident in a population of 67 patients with constructional apraxia studied by Piercy and colleagues.In this group, 25 had left-sided damage and 42 had damage to the right hemisphere.

The apraxia of patients with a dominant-hemisphere infarct often is described as decreased attention to detail. The apraxia with right-side damage is consistent with neglect, in which features to the left of midline are ignored.

The term allesthesia describes sensory referral. For example, a patient touched on the left side feels the touch on the right.

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

### Complications

Spasticity (ie, velocity-dependent resistance to passive range of motion) is often seen as a result of stroke. This can lead to several complications, including loss of function, pain, skin breakdown, and heterotopic ossification.

Initially, the treatment of noxious stimulation, such as infection, skin breakdown, and pain, should be pursued. Further management generally involves a combination of therapy, bracing, and medication, sometimes combined with focal neuromuscular blockade with botulinum toxin or, more rarely, with neurolysis with phenol.

Surgical intervention, such as tenotomy, is generally reserved for the most severe cases in which function can be gained or severe pain relieved.

## Treatment of MCA Stroke

### Initial and postacute management of stroke

Most patients who have experienced a stroke have other comorbid diseases and are at an increased risk of an adverse cardiac event during the immediate poststroke phase.

Meticulous management of hypertension, diabetes, atrial fibrillation, congestive heart failure, and pulmonary diseases (eg, sleep apnea, chronic obstructive pulmonary disease) is essential in assuring maximal functional outcome from rehabilitation. Additionally, cardiac parameters and precautions must be monitored and used throughout the course of the patient’s acute and rehabilitative course of care.

### Control of serum glucose level

Managing hypoglycemia and hyperglycemia is important. Evidence supports the association of acute and significant hyperglycemia with poor outcomes, leading to conditions such as intracerebral hemorrhage and, ultimately, a dependent state.

Several points about glucose management in acute stroke have been noted. Hyperglycemia increases lactate production in the brain, and this facilitates the transition of hypoperfused, at-risk tissue into infarction. The American Stroke Association (ASA) guidelines recommend treating hyperglycemia with fluids and insulin to achieve a level of less than 300 mg/dL.

### Control of blood pressure

Hypertension is a major risk factor for cerebrovascular accident (CVA). However, acute reduction of blood pressure (BP) in the event of a stroke does not necessarily benefit the patient. The initial rise in BP after a stroke is believed to act as a neuroprotective response to increase blood flow to the brain.

If treatment is necessary, agents such as labetalol are recommended because they can be titrated easily and because they minimally affect the cerebral blood vessels. Oral agents such as nicardipine and captopril are also recommended.

Patients who are candidates for thrombolytic agents must be specifically managed, and the physician must follow guidelines for BP control.

### Normalization of body temperature

Fever appears to be related to stroke severity, infarct size, mortality, and outcome in persons with an acute CVA. For each 1°C increase in body temperature, the relative risk of poor outcome rose by 2.2.

A reasonable plan is to keep the patient normothermic with acetaminophen. However, a Cochrane review found no evidence from randomized trials to support routine use of pharmacologic or physical strategies to reduce temperature in patients with acute stroke.

### Thrombolysis

[Thrombolysis](http://emedicine.medscape.com/article/811234-overview) remains a controversial topic, and it is still undergoing much research to weigh the risks and benefits in acute stroke treatment.

The only therapy currently approved by the US Food and Drug Administration (FDA) for acute stroke is intravenous alteplase, a recombinant tissue-type plasminogen activator (tPA).This appears to improve functional outcome at 3 months if given within 3 hours of the onset of symptoms and the patient meets the rigorous criteria for treatment.

The eligibility criteria for treatment between 3 and 4.5 hours are similar to those employed for treatment prior to 3 hours, as established in the AHA/ASA’s 2007 guidelines,but with the exclusion criteria expanded to include any of the following patient characteristics:

* Age greater than 80 years
* Use of oral anticoagulants
* Baseline National Institutes of Health (NIH) Stroke Scale score >25
* A history of both stroke and diabetes

### Anticoagulation

Anticoagulants (heparin and low-molecular-weight heparin) are often used in the acute stroke inpatient setting to prevent recurrent stroke and to improve the outcome for neurologic function. They are also used at lower, prophylactic doses to prevent deep vein thrombosis (DVT) and subsequent thromboembolism.