

MODULE 1: PATHOPHYSIOLOGY OF STROKE, NEUROANATOMY, AND STROKE SYNDROMES



Learning Objectives

Upon completion of this module, nurses will be able to define and/or describe:

- Types of stroke
- Etiology of stroke
- General brain anatomy
- Major blood vessels of cerebral circulation
- Common stroke syndromes
- Right-sided clinical deficits
- Left-sided clinical deficits

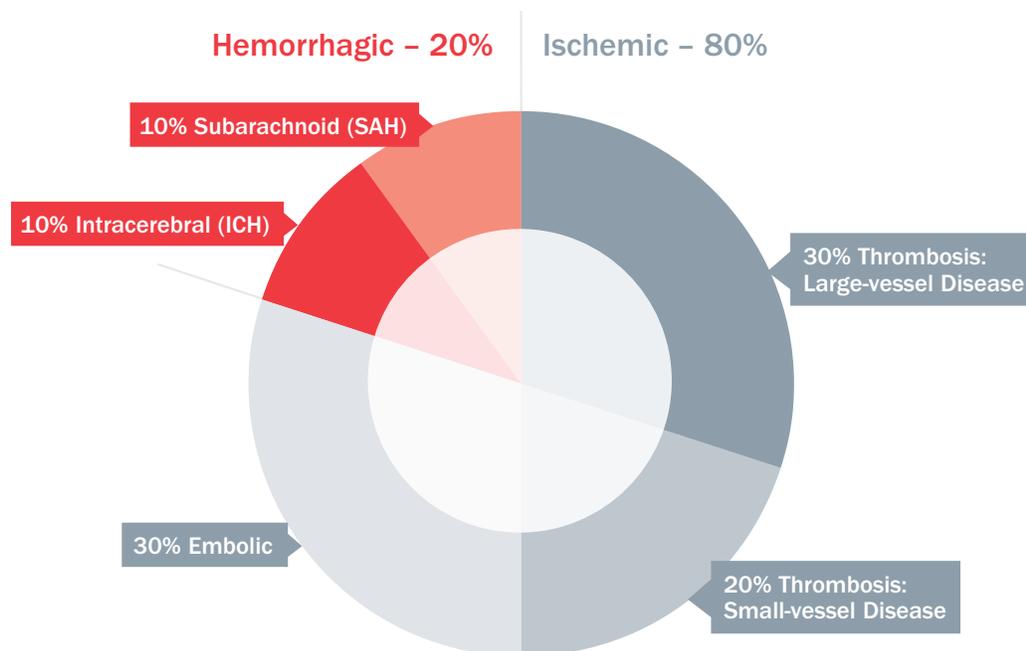
The following content is from the Acute Stroke Management Resource, Heart and Stroke Foundation of Ontario, Anatomy and Physiology workshop package. It has been edited and formatted for the Southwestern Ontario (SWO) Stroke Network's Acute Stroke Unit Orientation resource.

For more information on brain anatomy check out [Apex Innovations® \(http://swostroke.ca/certifications/\)](http://swostroke.ca/certifications/)

1.1 Classification and Etiology of Stroke: Pathophysiology and Anatomy

There are two types of strokes: ischemic and hemorrhagic.

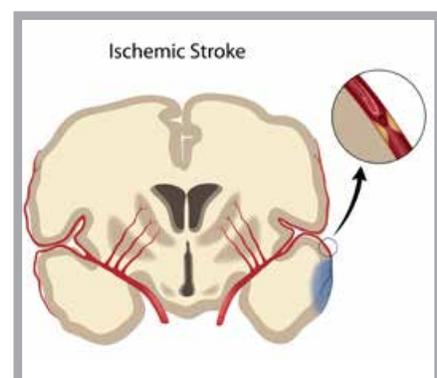
Breakdown of Stroke Subtypes



Ischemic Stroke

80% of all strokes are ischemic, meaning they are caused by the narrowing or blockage of an artery resulting in diminished blood flow.

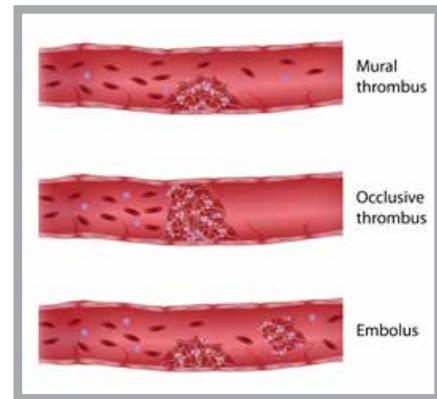
- Usually the result of a blood clot, either thrombotic or embolic in nature.
- May also occur because of progressive blood vessel occlusion, due to atherosclerosis, or because of local high pressure collapse of small blood vessels.



Clot stops blood supply to an area of the brain

Of the 80% of strokes that are ischemic:

- Approximately 50% are due to a **thrombosis**
 - 30% are related to **large-vessel disease**, especially of the carotid, middle cerebral, or basilar arteries.
 - 20% are related to small-vessel disease of the **deep penetrating arteries**, such as the lenticulostriate, basilar penetrating, and medullary arteries (these are known as **lacunar infarcts**).
- 30% are embolic



(See pie chart, “Breakdown of Stroke Subtypes”)

Etiology of Ischemic Stroke

The cause of ischemic stroke can be further classified as one of the following:

1. **Large vessel disease**, which may be classified as:
 - *Cardioembolism* – often a result of atrial fibrillation or left ventricular damage after myocardial infarction.
 - *Atherosclerosis* – causes a progressive narrowing of the blood vessel through deposit of plaque on the arterial wall.
2. **Small vessel disease**, known as *lacunar infarct*, is thought to be the result of occlusion of single, small perforating arteries, located deep in the subcortical areas of the brain. *Hypertension* is thought to be a major risk factor associated with lacunar infarcts.
3. **Cryptogenic strokes** are strokes with no identified cause or etiology. The classic risk factors for stroke are usually absent in cryptogenic stroke patients. However, *Ionita et al. (2005)* reported that echocardiography studies in patients with cryptogenic stroke showed an increased incidence of Patent Foramen Ovale (PFO) in up to 45% of cases. Cryptogenic strokes are more commonly found in people below age 45 (Ionita et al., 2005).

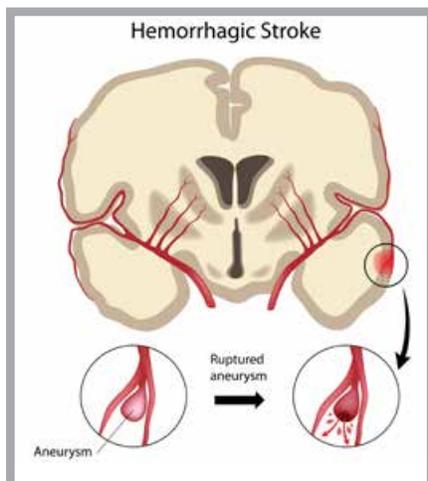
Hemorrhagic Stroke

20% of all strokes are hemorrhagic, meaning they are caused by **arterial rupture**. Hemorrhagic stroke can damage other brain tissue as a result of increased intracranial pressure and compression of brain tissue.

- 10% are due to **intracerebral hemorrhage (ICH)**, when a blood vessel in the brain bursts and spills into the surrounding brain tissue, damaging brain cells. Brain cells beyond the leak are damaged. Hypertension, trauma, vascular malformations, use of anticoagulants and other conditions can cause ICH.
- 10% are due to **subarachnoid hemorrhage (SAH)**, when an artery on or near the surface of the brain bursts and spills into the space between the surface of the brain and skull. This is often signalled by a sudden, severe headache and is commonly caused by an aneurysm.

Etiology of Intracerebral Hemorrhage (ICH)

ICH can be classified as either primary or secondary, based on the underlying cause of the bleeding. Primary ICH stems from the spontaneous rupture of small vessels and accounts for 78–88% of cases. Secondary ICH occurs in association with trauma, vascular abnormalities, tumours, or with impaired coagulation (Chakrabarty & Shivane, 2008). It can also occur in patients who initially had an infarction, but then developed subsequent bleeding from the surrounding fragile blood vessels (Cordingley, 2006).



Hemorrhage/blood leaks into brain tissue

1. Primary Hemorrhage

- Hypertension is responsible for approximately 75% of all cases of primary ICH.
- Cerebral amyloid angiopathy, a disease of small blood vessels in the brain with deposits of amyloid protein which may lead to stroke, brain hemorrhage or dementia, is also a common cause.
- Hemorrhage from the use of fibrinolytics and anticoagulants makes up approximately 10% of all ICH (Manno et al., 2005).
- Drug abuse may cause sudden and severe elevations in blood pressure resulting in ICH.

2. Secondary Hemorrhage

- Underlying vascular abnormalities such as aneurysm or arteriovenous malformation (AVM) are causes of secondary ICH and make up approximately 5% of all ICH.
- Hemorrhagic transformation, secondary bleeding from surrounding fragile blood vessels into the infarcted site (Cordingley, 2006), may be influenced by the size, location and cause of the stroke.
- The use of antithrombotics, including anticoagulants and thrombolytics increases the likelihood of hemorrhagic transformation.

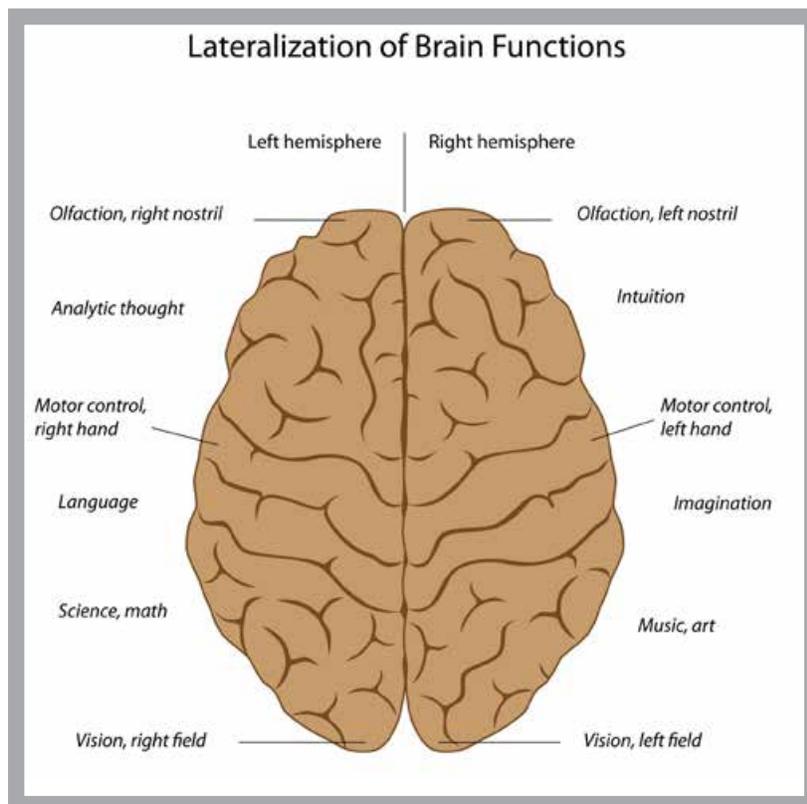
1.2 Brain Anatomy and Blood Supply

Cerebrum

The cerebrum is the **largest portion of the brain** and contains two hemispheres.

Each hemisphere controls the function of the contralateral side of the body. That is, the left hemisphere controls the right side of the body and the right hemisphere controls the left side of the body. The left hemisphere is the dominant hemisphere in 97% of the population.

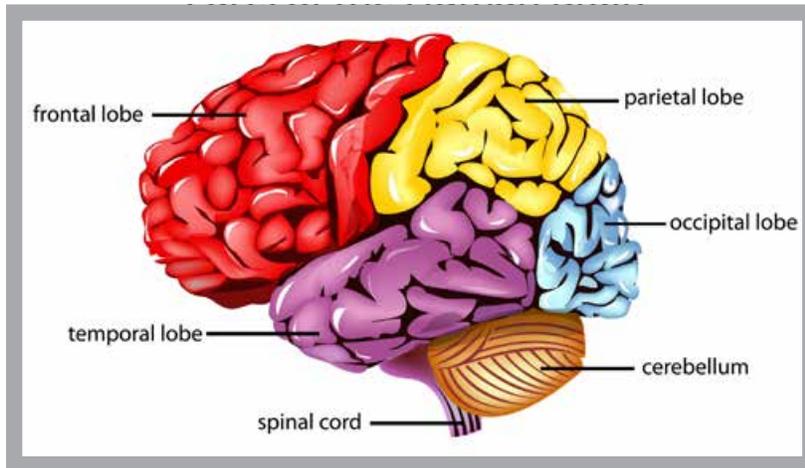
The two hemispheres are joined by the corpus callosum.



Cerebral Cortex

The cerebral cortex refers to the outer portion, or covering of the cerebrum. It is divided into four lobes:

- Frontal
- Parietal
- Temporal
- Occipital



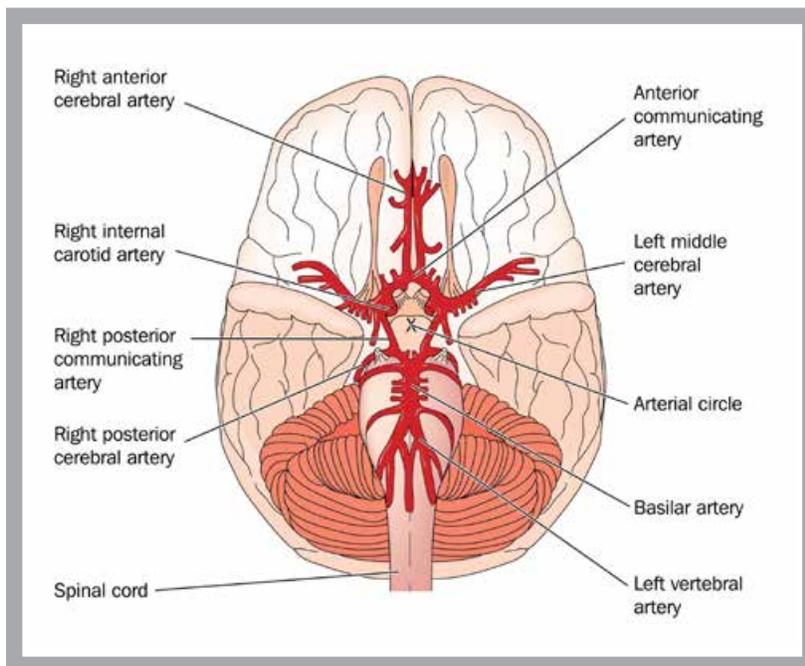
Two important structures are found in the frontal and parietal lobes:

- In the posterior portion of the frontal lobe, the **primary motor cortex** can be found. It is also referred to as the motor strip and is involved in the ability of the body to move various body parts. Damage to parts of the motor strip may result in symptoms such as paralysis of the face, arm or leg or difficulty speaking.
- In the anterior part of the parietal lobe is the **primary sensory cortex** or sensory strip. It corresponds directly to the body part locations of the motor strip and is involved in the ability to feel items or recognize textures.

Blood Supply to the Cerebral Cortex

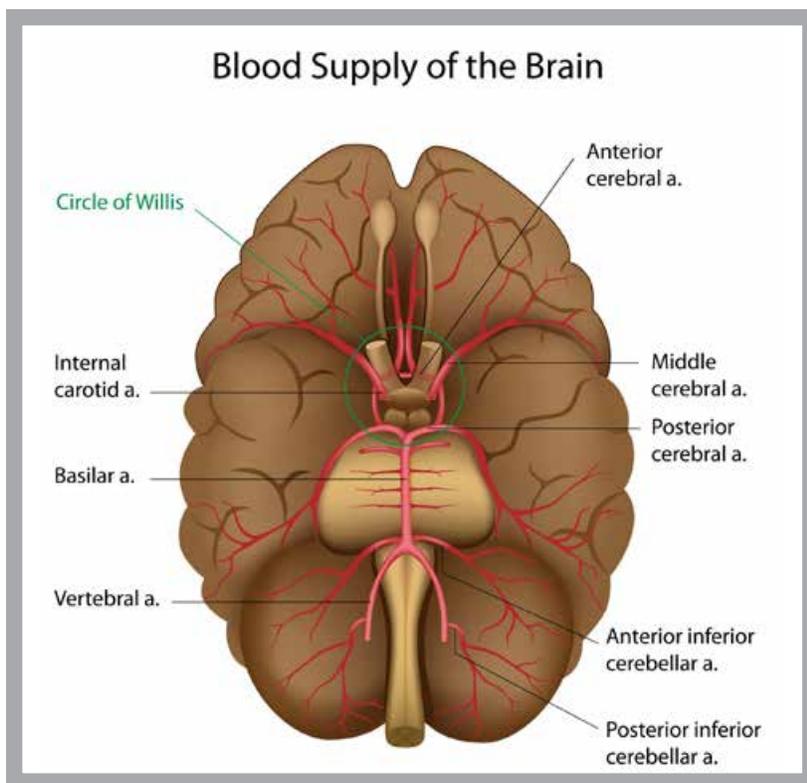
Arterial supply originates extracranially from carotid and vertebral arteries.

- **Internal carotid arteries (ICA)** supply anterior 2/3 of hemispheres. They come off the common carotid arteries. The anterior, middle, and posterior cerebral arteries are intra-cranial branches of the internal carotid arteries.
- **Vertebral and basilar arteries** supply posterior and medial regions of hemispheres. The vertebral arteries originate from the subclavian arteries.



Circle of Willis

The Circle of Willis is an important structure of cerebral circulation, and is located at the base of the brain. Its primary purpose is to provide multiple paths for oxygenated blood to supply the brain. The collateral routes available through Circle of Willis attempt to maintain circulation if a major vessel has become occluded.



The Circle of Willis is comprised of the following vessels:

- Anterior cerebral artery
 - Middle cerebral artery
 - Anterior communicating artery
 - Posterior cerebral artery
 - Posterior communicating artery
- } Anterior Circulation
} Posterior Circulation

The posterior and anterior communicating arteries are responsible for connecting the right and left side blood vessels so that circulation is seamless.

Major Cerebral Artery Regions

The **anterior cerebral artery (ACA)** originates from the internal carotid artery and supplies the anterior portion of the basal ganglia, the corpus callosum, the medial and superior portions of the frontal lobe, and the anterior part of the parietal lobe.

The key functional areas receiving blood supply from the ACA are:

- Primary motor cortex involving the leg and foot areas
- The centre for micturition found in the frontal lobe
- The motor planning centre found in the frontal lobe
- The anterior and middle portions of the corpus callosum



A patient who has a stroke involving the anterior cerebral artery may experience weakness in the leg and foot, difficulties with micturition, or difficulties with the ability to plan and carry out tasks such as dressing.

The **middle cerebral artery (MCA)** arises from the internal carotid artery, and is the largest of the major vessels. It supplies blood to over 2/3 of the cerebrum. The MCA has three branches and passes laterally under the frontal lobe and between the temporal and frontal lobes.

- The M1 branch of the MCA, also referred to as lenticulostriate arteries, is located in the deeper sections of the brain called the basal ganglia and most of the internal capsule. These lenticulostriate arteries are small vessels located deep in the brain and are also a common site for small vessel or lacunar strokes.
- The superior branch of the MCA supplies the lateral and inferior frontal lobe and anterior parts of the parietal lobe.
- The inferior branch of the MCA supplies the lateral temporal lobe, the posterior parietal lobe and the lateral occipital lobe.

The **posterior cerebral artery (PCA)** is responsible for the blood supply for midbrain, hypothalamus and thalamus, posterior medial parietal lobe, corpus callosum, inferior and medial temporal lobe and inferior occipital lobe.

The key functional areas receiving blood supply from the PCA are:

- Primary visual cortex in the occipital lobe.
- 3rd cranial nerve in the midbrain.
- Sensory control.
- Hypothalamus-body temperature control, hunger, thirst, hormone release (Antidiuretic hormone).
- Thalamus-relaying messages to cortex, level of arousal, awareness, pain.
- Communication between the hemispheres.



A patient who has had a stroke involving the posterior cerebral artery may present such symptoms as visual disturbances, problems recognizing objects, drooping eyelid, inability to move the eye in, up & out, down & out, difficulty maintaining body temperature, abnormal hormone responses, coma, or hyperesthesia.

Cerebellum

The cerebellum is located at the back of the brain, underlying the occipital and temporal lobes. Its major functions are **control of fine motor** movement, **coordination** of muscle groups and **maintaining balance** and equilibrium.

The cerebellum has its own major blood vessels, which originate from the vertebrobasilar vessels. These **3 cerebellar vessels** are:

- Superior cerebellar.
- Anterior inferior cerebellar.
- Posterior inferior cerebellar.

There are two syndromes often seen with cerebellar strokes:

1. **Lateral Pontine Syndrome:** involves the basilar and anterior inferior cerebellar arteries.

Symptoms: ipsilateral ataxia of arm and leg, contralateral weakness of upper and lower extremities, and contralateral hemisensory loss - pain and temperature.

2. **Lateral Medullary Syndrome (Wallenberg Syndrome):** involves distal and superior medullary artery branches of a vertebral artery and the posterior inferior cerebellar artery.

Symptoms: Ipsilateral sensory loss: face, pain and temperature; ipsilateral ataxia of arm and leg, gait ataxia, nystagmus, nausea and vomiting, vertigo, hoarseness, dysphagia, contralateral hemisensory loss – pain and temperature, Horner Syndrome (constricted pupil, partial ptosis, loss of hemifacial sweating), and hiccoughs.

Brain Stem

The brain stem connects the cerebrum with the spinal cord, and receives its blood supply from the posterior cerebral artery and the vertebral and basilar (vertebrobasilar) vessels.

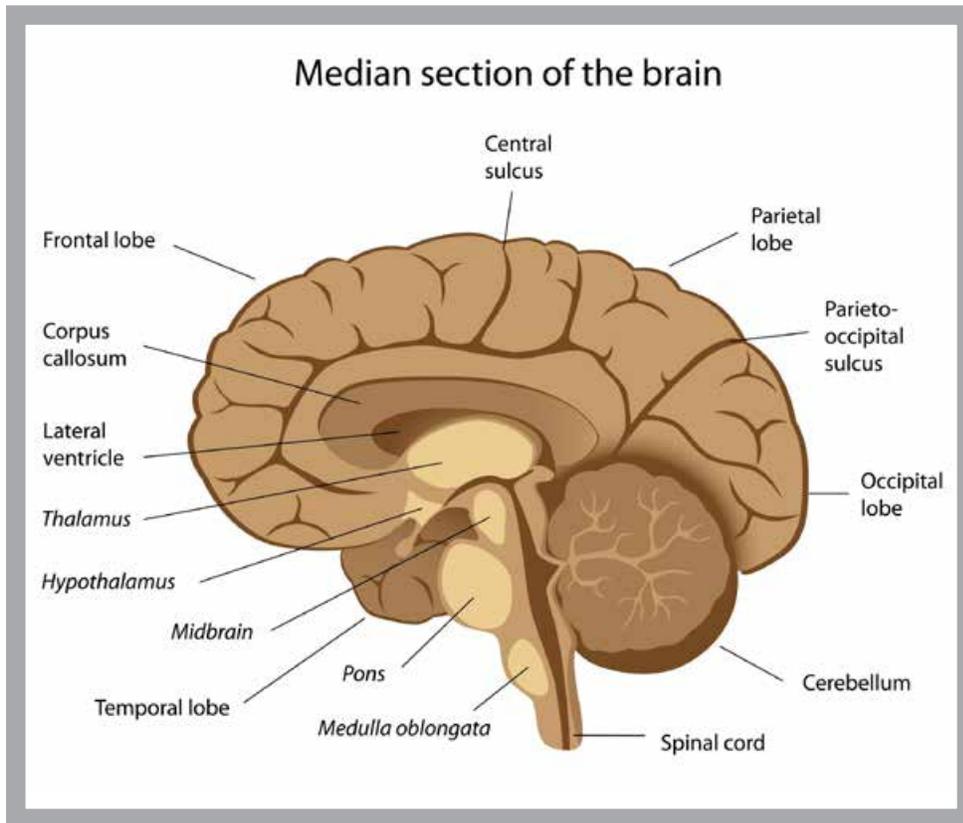
The brain stem is divided into 3 major sections:

- **Midbrain:** major functions include involvement in vision, hearing, eye movement and body movement.
- **Pons:** involved in motor control and sensory analysis, level of consciousness (LOC), and sleep.
- **Medulla:** responsible for maintaining vital body functions such as breathing and heart rate.

One of the major structures housed in the brain stem are the cranial nerves. There are **12 cranial nerves**; cranial nerves I and II originate in the frontal lobe and will not be discussed in this section.

Cranial nerves III to XII originate in the brain stem. Patients that experience a stroke in the brain stem will present with symptoms that involve cranial nerve functions such as difficulty with swallowing, eye movements, facial expression, and tongue movements.

The brain stem serves an important role as a pathway between the spinal cord and the brain. The afferent and efferent pathways run through the spinal cord and connect with brain centres for interpretation and response to stimuli.



The **reticular activating system (RAS)** originates in the brain stem and is responsible for wakefulness and attention. It is a very sensitive system that spans the brain and reacts to interruptions in its ability to work. An expanding stroke will interrupt the RAS's ability to keep the patient awake, resulting in the patient presenting with a decreased LOC.

Patients who experience a brain stem stroke may present with any of the following:

- Decreased LOC
- Ipsilateral lower motor neuron facial weakness or sensory loss
- Contralateral hemiparesis
- Pupillary changes
- Hiccoughs
- Vertigo
- Bilateral motor findings
- Diplopia, gaze palsies, intranuclear ophthalmoplegia
- Dysphagia
- Dysarthria
- Ataxia

Collateral Circulation

Collateral circulation is an important feature of the brain and very important for stroke patients, especially in cases where the patient may be a candidate for Endovascular Treatment (EVT) or thrombectomy. In these cases CTA (CT Angiogram) is often used to assess the patient's collateral circulation. See the modules on "Pre-Hospital and Emergency Management" and "Diagnostics and Assessments" for more information on EVT.

Not all blood vessels are capable of creating collateral circulation. For example, the lenticulostriate arteries are **terminal vessels** that do not connect with other vessels. Therefore, if vessels associated with the lenticulostriate arteries become occluded, they will become ischemic.

However, **some vessels can connect or anastomose with other vessels**, creating a redundancy that can permit collateral circulation when one vessel is blocked. These include:

- External and internal carotid arteries via branches of the ophthalmic artery.
- Major intracranial vessels via the Circle of Willis which connects the anterior and posterior circulation.
- Small cortical branches of the anterior cerebral, middle cerebral and posterior cerebral arteries, and the cerebellar arteries.

A stroke may go unnoticed if collateral circulation takes over the function of supplying blood to an area of the brain.

1.3 Stroke Syndromes

Ischemic Stroke: *Carotid and Vertebrobasilar Syndromes*

The carotid arteries, and their branches, the anterior and middle cerebral arteries, form **anterior circulation**. The vertebral, basilar, posterior cerebral arteries and their branches form **posterior circulation**. Clinical stroke syndromes depend on the area of the cerebral circulation disrupted.

Typically, the anterior or carotid circulation stroke syndromes present with symptoms such as:

- Sensory and/or motor deficits
- Aphasia
- Cortical sensory loss
- Apraxia
- Neglect
- Visual field deficit
- Retinal ischemia

Strokes affecting the posterior circulation or vertebrobasilar system, present with symptoms such as:

- Diplopia
- Vertigo
- Coma at onset
- Crossed sensory loss
- Bilateral motor signs
- Isolated field deficit
- Pure motor and sensory deficit
- Dysarthria
- Dysphagia

Wake-up Strokes

When patients wake-up with stroke symptoms that were not present prior to falling asleep, it is referred to as a “wake-up stroke”. About 1 in 5 acute ischemic strokes are wake-up strokes. Often these patients are not eligible for acute reperfusion therapy as the true last seen normal time is unknown.

Rubin & Barrett, 2015.

Ischemic Stroke: *Lacunar Syndromes*

- Makes up 25% of all ischemic strokes.
- Presumed to be occlusion of single small perforating artery.
- Predominantly in the deep white matter, basal ganglia, and/or pons.
- Blood vessel: **lenticulostriate branches** of the anterior cerebral and middle cerebral arteries.

Lacunar infarction results from infarction of one of the lenticulostriate vessels, the penetrating branches of the Circle of Willis, the MCA stem, or vertebral or basilar arteries.

Patients who present with symptoms of a lacunar stroke, but who have not yet had diagnostic imaging performed may be described as suffering from lacunar stroke syndrome (LACS).

Type of Lacunar Syndrome	Patient Presentation
Pure motor hemiparesis Results from an infarct in the internal capsule or pons	Contralateral hemiparesis of face, arm and leg, dysarthria
Contralateral motor hemiparesis with motor aphasia Results from an infarct of the left frontal area with cortical involvement	Hemiparesis of face, arm and leg with inability to speak
Ataxic hemiparesis Results from an infarct in the pons	Paresis of the contralateral leg and side of the face, ataxia of the contralateral leg and arm
Dysarthria and clumsy hand syndrome Results from an infarct in the pons or internal capsule	Dysarthria, dysphagia, contralateral facial and tongue weakness, paresis and clumsiness of the contralateral arm and hand
Pure sensory stroke Results from an infarct in the thalamus	Contralateral sensory loss to all modalities that usually affect the face, upper and lower extremities, and may be painful

Kistler JP, Ropper AH, Martin JB. *Cerebrovascular diseases*. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, eds. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw Hill; 1994: 2233-2256. Fisher, CM. (1991). Lacunar Syndromes, 1, 311-320.

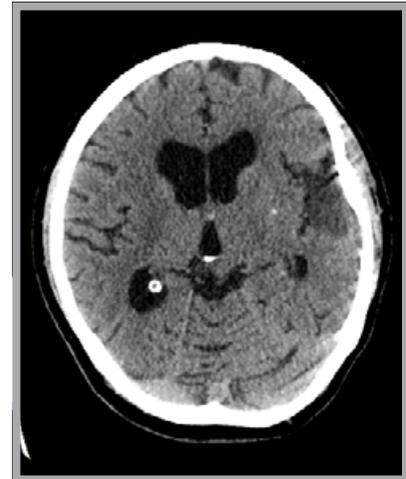
Ischemic Stroke: *Left (dominant) Hemisphere Stroke*

The MCA is the artery most often occluded in ischemic stroke.

The associated **neurological signs and symptoms** form a common pattern of stroke presentation involving the left, or dominant, hemisphere:

- Aphasia
- Right field deficit
- Left gaze preference
- Right upper motor neuron facial weakness
- Right hemiparesis
- Right hemisensory loss

*See Appendix A, *Common Effects of a Left Hemispheric Stroke*



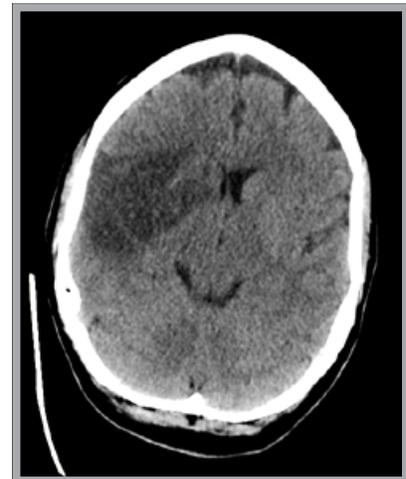
The early CT scans show an area of infarction in the territory of the left middle cerebral artery (MCA). Adapted from J. Mandzia (Neurologist), 2014. Reprinted with permission.

Ischemic Stroke: *Right (non-dominant) Hemisphere Stroke*

The associated **neurological signs and symptoms** form a common pattern of stroke presentation involving the right, or non-dominant, hemisphere:

- Left neglect, inattention
- Left field deficit
- Right gaze preference
- Left upper motor neuron facial weakness
- Left hemiparesis
- Left hemisensory loss, sensory extinction

*See Appendix B, *Common Effects of a Right Hemispheric Stroke*



The CT scans shows an area of infarction in the territory of the right middle cerebral artery (MCA), CT scan taken about 24 hrs post stroke. Adapted from J. Mandzia (Neurologist), 2014. Reprinted with permission.

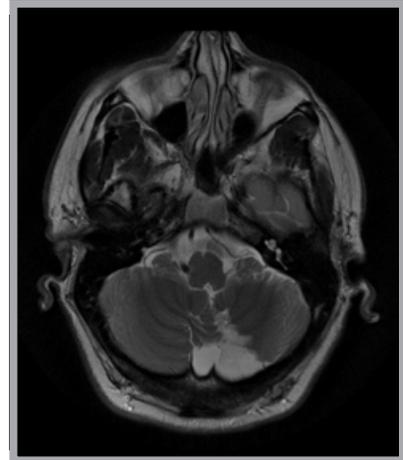
Ischemic Stroke: *Cerebellar Infarct*

The following associated **signs and symptoms** form a common pattern of stroke presentation involving the cerebellum:

- Headache, nausea/vomiting
- Vertigo, imbalance
- Normal tone, power, reflexes
- Inability to sit or stand
- Ataxia or loss of normal coordination

Late signs:

- Decreasing LOC
- Diplopia, gaze palsy
- Ipsilateral cranial nerve V, VII impairment

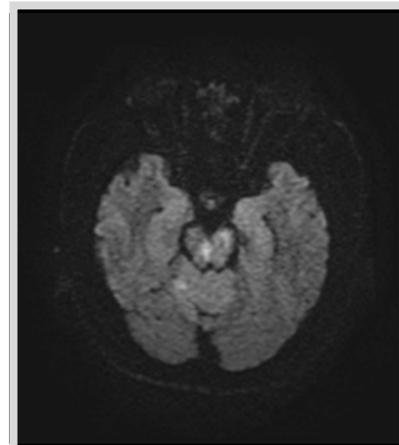


Reprinted by permission from Macmillan Publishers Limited: Remodeling of cortical motor representations after stroke: implications for recovery from brain damage (Nudo, R.J., et al), 1997.

Ischemic Stroke: *Brainstem Stroke*

The following associated **neurological signs and symptoms** form a common pattern of stroke presentation involving the brainstem:

- Decreased LOC
- Ipsilateral lower motor neuron
 - Ipsilateral lower motor neuron facial weakness or sensory loss **AND** contralateral hemiparesis.
- Pupillary changes
- Hiccoughs
- Vertigo
- Bilateral motor findings
- Diplopia, gaze palsies, intranuclear ophthalmoplegia
- Dysphagia
- Dysarthria
- Ataxia



The MR DWI sequence shows an Acute Brainstem -upper pontine stroke. Adapted from J. Mandzia (Neurologist), 2014. Reprinted with permission.

1.4 Neuroplasticity

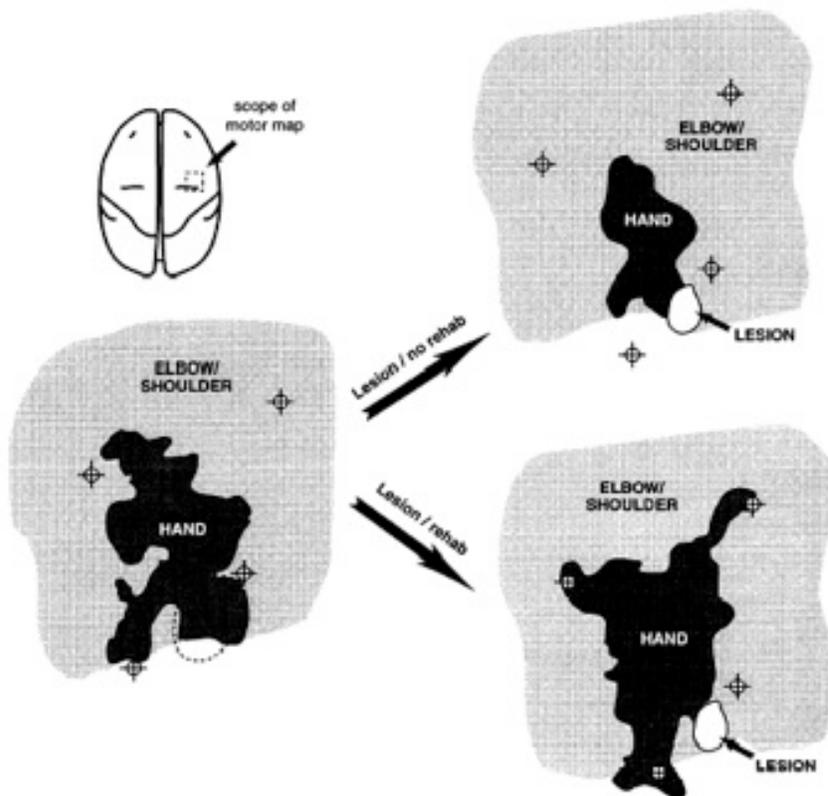
Neuroplasticity and Stroke Recovery

Early after stroke, a time-limited window of neuroplasticity opens and the greatest gains in recovery occur.

Mechanisms include:

1. **Rewiring:** *Where there's a wire, there's a way!* The brain will rewire functions through other existing pathways.
2. **Remapping:** *Location, location, location: neighbouring areas remap!* New circuits can form between related cortical regions. Surviving neurons at the border of an infarct undergo active remodeling after stroke and sow the seeds for recovery.

The diagram below shows that after stroke, the cortical territory devoted to the hand rapidly remaps to the neighboring cortex.



Reprinted by permission from Macmillan Publishers Limited: *Remodeling of cortical motor representations after stroke: implications for recovery from brain damage* (Nudo, R.J., et al), 1997.

This does not happen spontaneously. Plasticity is activity dependent. As in the diagram above, without activity and rehabilitation, the cortical representation for the hand shrinks. With activity and rehabilitation, the cortical representation remaps. The maps can be modified by experience.

Brain Cells

Early Recovery: Local CNS processes

- Resolution of ischemic penumbra – hours to weeks.
- Resolution of edema – weeks to 2 months.

Later Recovery: CNS reorganization

- Unmasking of ipsilateral and alternate pathways – immediate to months.
- Creation of new synapses – weeks to months.
- Neurotransmitter alterations – weeks to years.

The challenge for improving stroke recovery is to understand how to optimally engage and modify surviving neuronal networks.

Often, patients that have experienced a stroke exhibit continued functional recovery for many years following the initial injury.



Brain plasticity is ongoing ... recovery continues

- Repetition and practice are key
- Integrate into daily activities
- Keep moving!

Patients undergoing rehabilitation experience the greatest improvements during the first 3 months. However, patients who have experienced a stroke will exhibit continued functional recovery for many years following the initial injury.

(Duncan et al., 1994).

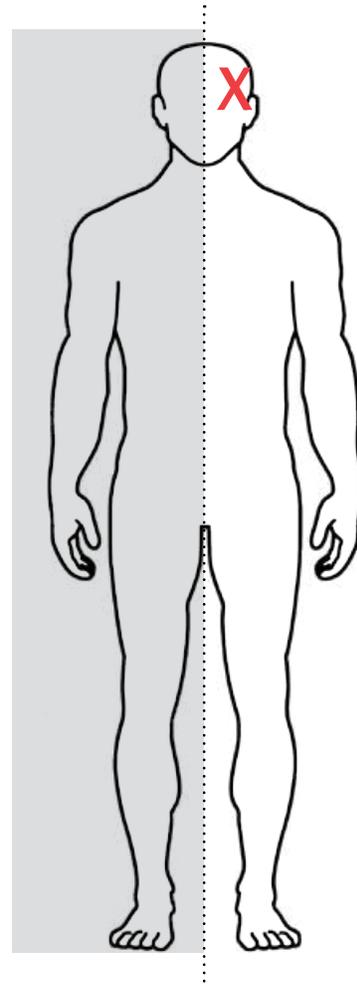


What helps brain recovery?

- Attention
- Concentration
- Interest
- Problem solving
- Repetition
- Trial and error
- Meaningful goals

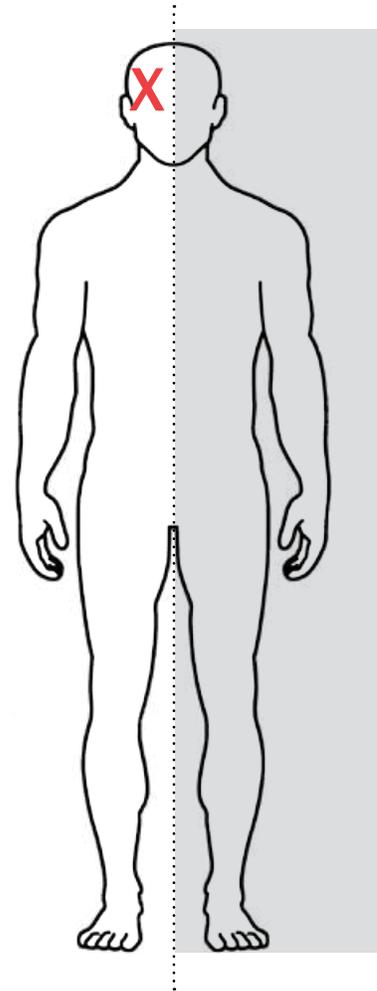
Appendix A: Common Effects of a Left Hemispheric Stroke

- Right visual field loss (homonymous hemianopsia).
- Dysphagia (swallowing difficulty).
- May develop aphasia (loss of language including spoken, written, reading and comprehension) but may also have dysarthria (difficulty producing speech).
- Right-sided weakness (hemiparesis) or paralysis (hemiplegia).
- Sensory impairment.
- Usually has normal perception, but not always.
- Judgment is intact with good insight into limitations.
- Short-term memory impairments (difficulty remembering new information) and apraxia (inability to carry out learned movement in the absence of weakness or paralysis).
- Often develop a slow and cautious behavioural style. They need frequent instructions and feedback to complete tasks.
- Better able to comprehend and express emotions



Appendix B: Common Effects of a Right Hemispheric Stroke

- Left visual field loss (homonymous hemianopsia).
- Dysphagia (swallowing difficulty).
- Usually retain language ability but may have difficulty producing speech (dysarthria).
- Left-sided weakness (hemiparesis) or paralysis (hemiplegia).
- Sensory impairment.
- Denial of paralysis, “forget” or “ignore” objects or people on their left side (neglect).
- Impaired ability to judge spatial relationships (misjudge distances and depth leading to falls, unable to guide hands to button a shirt, problems with directions such as up or down, no concept of time).
- Impaired ability to locate and identify body parts.
- Short-term memory impairments (difficulty remembering new information) and apraxia (inability to carry out learned movement in the absence of weakness or paralysis).
- Behavioural changes such as impaired judgment or insight into limitations, overestimate physical ability, impulsivity, inappropriateness and difficulty comprehending and expressing emotions.



References

The main source for this module was:

Acute Stroke Management Resource. (2007). Heart and Stroke Foundation of Ontario, Anatomy and Physiology of Acute Stroke power point presentation. Retrieved from http://www.heartandstroke.on.ca/site/c.pvI3leNWJwE/b.5346923/k.8D25/HCP__English.htm

That presentation listed the following sources for the content:

American Association of Neuroscience Nurses

<http://www.aann.org>

American Stroke Association

<http://www.strokeassociation.org>

Brain Attack Coalition

<http://www.stroke-site.org>

Canadian Hypertension Education Program

<http://www.hypertension.ca/chep>

Canadian Stroke Strategy

<http://www.canadianstrokestrategy.ca>

Heart and Stroke Foundation Prof Ed

<http://www.heartandstroke.ca/profed>

Heart and Stroke Foundation of Canada

<http://www.heartandstroke.ca>

Internet Stroke Centre

<http://www.strokecenter.org>

National Institute of Neurological Disorders and Stroke

<http://www.stroke.org/site/PageServer?pagename=HOME>

Scottish Intercollegiate Guidelines Network

<http://www.sign.ac.uk>

StrokeEngine

<http://strokengine.ca>

CNS Forum- Lundbeck Institute

<http://www.cnsforum.com>

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