



## Module 4. Ischemia in Carotid Territory Introduction and Key Clinical Examples

### Objectives for Module 4

#### Knowledge

- Describe two common TIAs (mini-strokes) that are produced by ischemia in carotid territory.
- Describe at least one mechanism that may explain transient monocular blindness.
- List 4 of the major signs and symptoms seen after occlusion of the MCA stem in the language-dominant and in the non-dominant hemisphere, and relate each to damage in a specific neuroanatomic structure.

#### Clinical Applications and Reasoning

- Explain how occlusion of the internal carotid artery in the neck (extracranial) can produce several different patterns of neurologic deficits (or in some cases, none at all).
- Compare and contrast the patterns of clinical findings seen after occlusion of the *superior* MCA division in the language-dominant (usually left) vs the non-dominant hemisphere.
- Compare and contrast the patterns of clinical findings seen after occlusion of the *inferior* MCA division in the language-dominant vs the non-dominant hemisphere.

#### Clinical Applications to Patient Education

- Develop points that you could use in explaining to a patient how he or she might personally experience a TIA produced by temporary occlusion of carotid branches.

Carotid territory includes the retina and much of the anterior and lateral cerebral hemisphere. Key clinical problems referable to ischemia in carotid territory can be produced by occlusion of either the *extracranial* internal carotid artery (ICA) or its major *intracranial branches*, including the ophthalmic artery, middle cerebral artery (MCA) or anterior cerebral artery (ACA).

### TIAs involving the carotid artery or its branches

#### Transient monocular blindness

##### Case Summary

The patient was sitting watching television when his vision suddenly went “blurry and cloudy” for about 3-4 minutes and then returned to its usual state over the next 10 minutes. He wasn’t convinced that anything had really happened, but then later on that evening he had another episode. This time he tried checking one eye at a time, and discovered that his right eye was fine, but that the vision in his left eye was “blurry.” He called 911 because he remembered seeing something about this kind of problem on a Brain Attack poster in his doctor’s waiting room.

**Expert Note:** This patient has experienced a typical episode of transient monocular blindness (*amaurosis fugax*). Many patients describe a blackout, a cloud or a “gray fog” that obscures vision in one eye, or in part of one eye. Occasionally it is likened to “a shade falling over the eye.” Usually the attacks are very brief, lasting about 1- 5 minutes, and are followed by full restoration of vision. Some patients have many repeated attacks; others have only one or a few.

Two general mechanisms are proposed to explain this type of TIA. (1) A tiny piece of thrombus formed on an ulcerated plaque at or near the carotid bifurcation, or a piece of the underlying plaque itself breaks off, enters the ophthalmic artery, and lodges in a retinal arteriole. Ophthalmoscopic observation of the retinal vessels during actual episodes of transient monocular blindness has shown bits of whitish material (small emboli) temporarily blocking the retinal arteries. (2) A low perfusion state resulting from ICA stenosis (and probably temporary formation of thrombus that further blocks the vessel) causes regions far from the site of stenosis (like the retina) to become briefly ischemic.

There can be a number of different reasons for a transient loss of vision in one eye, such as inflammation of the arteries that supply the eye. However, in older patients the cause is often carotid occlusive disease produced by significant atherosclerosis and plaque erosion in the extracranial ICA. Thus transient monocular blindness is a major warning sign of ischemic stroke.

### Transient hemispheric attacks

#### Case Summary

This 71-year-old woman noticed that her left arm suddenly went “limp.” This lasted about 20 minutes and then things were fine. She drove herself to the emergency room where they thought she might be having a heart attack. When cardiac testing showed no problems, she was sent home. The next day she noticed “numbness” in two fingers of her left hand that lasted a bit longer but resolved completely. The third day she again experienced numbness in the same two fingers and called her primary care physician, who arranged an urgent neurology consult. The patient was admitted to the hospital. Later that day she developed additional deficits involving her face, her enunciation of words (“I lisped”), and left arm strength. Auscultation of her neck revealed a right carotid bruit (noise from turbulent blood flow that can be caused by vessel narrowing). Right carotid stenosis, greater than 70%, was confirmed by ultrasound and cerebral arteriography. It was also found that she had developed significant external to internal carotid blood flow through anastomoses of these vessels around her right eye. She was treated surgically (a carotid endarterectomy), and is now taking antihypertensive medications, a statin agent, and a daily aspirin. Three years later, she has experienced no further neurologic problems.

**Expert Note:** This patient had not one but a series of TIAs initially involving the arm and hand area of the motor and perhaps somatosensory cortex, both supplied by carotid branches. TIAs in carotid artery territory typically produce varying combinations of:

- one-sided limb weakness or clumsiness
- one-sided numbness, paresthesias (abnormal sensations like burning) or loss of sensation
- Some problem with language (production or comprehension) – if L hemisphere involved
- Some problem articulating words, often described as ‘slurring’ words (dysarthria) Note that this is “lisping” as she called it is NOT aphasia, which is a problem with language.
- Partial or complete homonymous visual field defects (less commonly noticed by patients)

The patient’s well-established collateral circulation involving her ophthalmic artery suggests that carotid stenosis had been steadily progressing over a period of time. Surgical removal of accumulated plaque by carotid endarterectomy in patients with high-grade carotid stenosis (70-99%) has been shown to significantly decrease the risk of stroke. In individuals 70 years of age or younger, carotid artery angioplasty with stenting appears an acceptable alternative. Long-term data regarding the outcomes of stenting continue to be collected, but to date several studies suggest that clinical outcomes for endarterectomy and stenting are similar.

# Common Strokes involving the carotid artery or its branches

We will consider the following five Stroke Syndromes produced by occlusion of the internal carotid artery and its branches because they are the most common and therefore the most important to understand.

Blocked Artery or Branch	PATTERNS OF POSSIBLE DEFICITS
<b>Extracranial Internal Carotid</b>	Deficits depend on the extent of collateral supply, and how quickly occlusion occurred. As many as 30-40% of carotid occlusions near the bifurcation are clinically silent.
<b>MCA – main stem (M1)</b>	Contralateral hemiplegia and hemisensory loss Contralateral hemianopsia Global aphasia (L)* or denial, neglect, and disturbed spatial perception perhaps with emotional ‘flatness’ (R)* Eye deviation to side of lesion acutely (gaze preference)
<b>MCA – superior cortical division</b>	Contralateral hemiparesis (face and arm > leg; and somewhat less often a cortical hemisensory loss; Broca’s aphasia (L)* or neglect and/or disturbed visual-spatial perception (R)* Eye deviation to side of lesion acutely (gaze preference)
<b>MCA – inferior cortical division</b>	Wernicke’s aphasia (L) or denial, neglect and/or disturbed visuospatial perception (R)* Contralateral hemianopsia –mostly upper quadrants Contralateral somatosensory loss involving face, arm, hand possible; mild weakness of face/arm possible
<b>MCA – lenticulostriate branch</b>	“Pure motor” stroke often, but not necessarily, involving lower face, arm and leg equally but sparing sensation. Possibly dysarthria or ataxia as well.

\*Assuming left hemisphere dominance for language

## Extracranial Internal Carotid Artery

The mechanism of occlusion of the extracranial ICA is almost always formation of thrombus on an ulcerated atherosclerotic plaque located at or near the carotid bifurcation. Sometimes embolism from a cardiac source or carotid dissection is responsible, but this is less common. The clinical syndromes that result are variable – they can range from total infarction of the entire cortical and deep territories of the MCA and ACA (affecting most functions of one entire hemisphere) to a small cortical MCA territory infarction to no neurologic deficit whatsoever. **This incredible variability depends on what sources of collateral flow are available and how much blood the collaterals actually carried in the particular patient during the time immediately following the occlusion.**

**Question:** Can you think of several places where anastomotic flow might be established that could nourish carotid territory if the ICA were occluded slowly and progressively, allowing enough time for collaterals to increase their caliber and the amount of blood they carry?

4 possible answers:

- *Anastomoses via the circle of Willis:*
  - Enlarged posterior communicating artery connecting the vertebral-basilar to the carotid circulation
  - Enlarged anterior communicating artery connecting the carotid circulation on each side
- *External to internal carotid artery anastomoses, e.g. connections around the eye*
- *Cortical anastomoses between distal small cortical branches of MCA with ACA and PCA*

With all the variability in clinical presentation, you may be wondering how physicians come to suspect an ICA occlusion. The answer is often in the patient's the past and present medical history. Clues include repeated transient ischemic attacks always in carotid territory, a carotid bruit or direct evidence of stenosis from angiography or ultrasound studies, or reversed blood flow in ophthalmic artery branches, (detected by transcranial doppler).

**Question:** If there is a functional anastomosis between external and internal carotid artery enabling the external carotid to supply internal carotid territory, what direction would blood be flowing in the ophthalmic artery? (*Answer: inward*).

## Strokes in Middle Cerebral Artery Territory

**Infarctions in the distribution of the MCA are by far the most common strokes that are seen in clinical practice.** Unlike the situation with the *extracranial* internal carotid artery, there are limited ways to establish anastomotic blood flow that can “rescue” ischemic tissue if the MCA or its branches are blocked. **Question:** Can you think of one possibility? *Answer: anastomoses between cortical branches in the border zones between MCA, ACA and PCA.*

**Occlusion of the MCA stem** is most often caused by embolism from a cardiac source or artery-to-artery embolism from the extracranial ICA. In a complete occlusion, blood flow is blocked both to the 10-15 deep penetrating lenticulostriate branches, which supply much of the internal capsule and basal ganglia, and to the superior and inferior cortical MCA branches, which supply most of the lateral cerebral cortex. As a result, much of the hemisphere is infarcted. Partial occlusions of the MCA stem can block blood flow to only lenticulostriate or only cortical branches.

After near-complete MCA stem occlusion, brain swelling may cause downward herniation of the medial temporal lobes leading to brainstem compression that kills the patient. Those who survive the initial period will develop contralateral hemiplegia affecting the lower face, arm and (to a lesser extent) leg accompanied by exaggerated reflexes and an extensor plantar response (damage to frontal lobe motor cortex and its projections). *However acutely there may be flaccid paralysis with absent reflexes.* These patients also have contralateral hemianesthesia produced by damage to the anterior parietal lobe somatosensory cortex, and a complete contralateral homonymous hemianopsia resulting from damage to the visual radiations as they travel from the lateral geniculate nucleus to the primary visual cortex.

In addition, damage to frontal, parietal, and temporal lobes near the lateral (sylvian) fissure in the language-dominant hemisphere (almost always the left one), produces **global aphasia**. Although conscious, these patients understand nothing that is said, and are unable to utter more than a few stereotyped sounds or words.

Damage to the posterior parietal and temporal lobes (most likely parts of the frontal lobes as well) in the non-language, right hemisphere produces a **severe defect in attention and/or visuospatial conceptualization**. These patients may neglect the left side of their own body and of their world. They may deny that their left limbs are paralyzed, or even deny that the left limbs belong to them. They may shave only the right side of their face, or draw only the right side of a symmetrical object such as a clock. They may have major problems in putting on clothes correctly, an activity that requires understanding 3-D visuospatial relationships. They often appear emotionally blunted, dull, inattentive, apathetic, or confused.

## Occlusion of superior cortical branches in the left (language-dominant) hemisphere

### Case Summary

At examination two months after his stroke, this patient had right hemiparesis, milder in the leg than in the arm and the lower part of his face. The deep tendon reflexes were exaggerated on the right and the sign of Babinski was present (extensor plantar response – big toe up). Pinprick and two-point discrimination were mildly impaired on the right side of the face and arm, and joint position sense in his right fingers was much less accurate than in his left fingers. He could speak only a few words with great effort, and often answered questions using just a noun, leaving out grammatically correct connecting words like “the” and “of.” Writing was affected in the same way as speech. Additionally, he had difficulty repeating short phrases. However, he could carry out verbal commands, whether spoken or written, unless they were very long.

**Expert Note:** The patient’s right hemiparesis is produced by damage to either the left motor cortex or the corticobulbar and corticospinal axons that travel through the deep white matter of the hemisphere toward the internal capsule. The leg area of cortex should not be affected in an MCA branch occlusion since it is located on the medial aspect of the hemisphere (ACA territory). However the corticospinal axons for the leg must swing around the lateral ventricle as they travel to the internal capsule, and this puts them into superior branch MCA territory. Usually patients like this have much less weakness in the leg than in the arm and face. Explanations for the milder deficit in the leg are based on fact that leg motor cortex is still intact; however the exact cause is unclear. Right-sided sensory loss reflects milder damage to the left somatosensory cortex for face and arm, which again is located on the lateral aspect of the hemisphere.

This patient has Broca’s aphasia, which is an intellectual or cognitive deficit in assembling or expressing ideas with language, whether spoken or written. These same patients may also have difficulty enunciating words but that is because of weakness of muscles of facial expression in lower parts of the face and of the tongue (dysarthria). Much of the inferior frontal and parietal as well as superior temporal lobes and insular cortex near the lateral fissure is devoted to different aspects of language functions. Localization of specific aspects of language (speaking, reading etc.) is complex. A severe and lasting Broca’s aphasia like this patient has reflects damage to a large area centered in the left inferior frontal gyrus.

## Occlusion of superior cortical branches in the right (non-dominant) hemisphere

### Case Summary

At examination 3 months after her stroke, this patient had left hemiparesis, much milder in the leg than in the arm and the lower part of her face, with exaggerated reflexes. Two-point discrimination and pinprick were mildly impaired on the left side of the face and arm. When the left arm or right arm was tested alone, she reported when it is touched accurately; however when both the left and right arm were tested *simultaneously*, she reported touch only on the right arm. It is not safe for her to drive, because although she sees fairly well in both left and right visual fields, she doesn’t pay sufficient attention to things going on in the left side of space.

**Expert Note:** Like the patient with this lesion in the left language-dominant hemisphere, the patient described here also has a contralateral hemiplegia and mild sensory loss involving primarily face, hand, and arm. However instead of severe impairment of language, she has a different specific cognitive disorder involving attention and visuospatial perceptions of her own body and of the outside world. In her case, which is quite mild, the problem causes her to ignore a stimulus delivered to the left side when stimuli are given bilaterally (extinction), although she responds to a stimulus given ONLY to the left side.

We really don't understand the details, but clinical evidence suggests that the right hemisphere plays an important role in attending to the *entire* visuospatial world while the left hemisphere has a more limited role and attends only to the right. Thus damage to the right hemisphere would be expected to particularly affect attention to and conceptualization of *left* personal and general space. The anatomic basis is unclear, but there is evidence that spatial conceptualization involves systems distributed in the frontal as well as parietal and temporal lobes. Parts of these systems are therefore nourished by the superior cortical branches of MCA, although other critical components are in inferior MCA territory.

**Additional Clinical Note:** Immediately after their strokes, patients with superior cortical branch lesions in either the left or right hemispheres may have their eyes (or their eyes and head) turned toward the side on which their brain is injured. This severe gaze palsy usually lasts for only a few days, even when other motor deficits remain.

**Question:** Do you know of some part of the gaze mechanism that involves the frontal lobes?  
**Answer:** *the frontal eye fields sometimes called the frontal lobe centers for contralateral gaze.* Each frontal eye field has a role in initiating voluntary eye movements to the opposite side. When it is damaged on one side the patient does not voluntarily move his eyes to the opposite side (and may show a gaze preference to the side of the lesion).

### Occlusion of inferior branches in the language-dominant hemisphere

#### Case Summary

This patient spoke fluently, and seemed fairly normal so long as conversation was confined to social pleasantries like responding to "How are you today?" However, as soon as conversation went beyond these stock phrases to matters of substance, spontaneous speech became progressively more and more devoid of meaning. The patient seemed totally unaware that he was talking nonsense. Verbal or written instructions were not understood unless they were reinforced with non-verbal visual cues. Repetition was impaired. The patient showed no reaction to any visual stimulus (including visual threat) given in the upper right visual fields. Other aspects of the neurologic exam were described as normal, including eye movements, somatic sensation (so far as it could be tested), and movements of the face, arm, and leg.

**Expert Note:** The patient's fluent, melodic spoken language with little meaningful content, combined with an inability to comprehend spoken language, is characteristic of Wernicke's aphasia. The stroke, which affects the posterior part of the superior temporal gyrus and neighboring cortex, has disrupted the circuitry essential for comprehending sounds as being part of language and assigning them meaning. These patients do not understand what is being said to them, and their language is empty and meaningless, filled with sound-alike words, words used incorrectly, or totally made-up, meaningless words. Reading, writing, and speech are all similarly impaired.

Part of the visual radiations representing the contralateral superior quadrants loop forward into the temporal lobe, where they lie lateral to the ventricle before turning and running posterior to the medial occipital lobe (anatomists name this Meyer's loop). These fibers may therefore be at special risk from an MCA inferior cortical branch occlusion. Detailed visual field testing in a patient who cannot understand even simple directions is difficult. However where the patient does and doesn't see can be roughly determined by whether or not the patient responds to threatening gestures in each of the visual quadrants ('visual threat') by closing his or her eyes or moving away from the stimulus.

The normal eye movements and normal motor and somatosensory components of the neurologic exam reinforce the idea that cortical regions supplied by the superior branch of MCA are intact.

### Occlusion of inferior branches in the non-dominant hemisphere

#### Case Summary

When seen in clinic, this patient had no apparent motor deficit: there was no weakness and no gait abnormality. Although movements of the arms and hands were fluid and skillful, he had to labor for several minutes to button a single shirt button because he seemed not to understand how the button related to the buttonhole. His repeated efforts to put on a pair of work gloves were equally unsuccessful, and he ended up with the left glove on his right hand and 3 fingers where his thumb should go. Through all these efforts his face remained blank, with no indication of either concern or frustration. He spoke in a monotone. Pinprick, temperature and touch sensation were normal over both sides of the face, arm and leg when they were tested separately. However when tactile stimuli were presented simultaneously on the left and right, only the right-sided stimuli was recognized. He could not see objects on his left with either eye (visual field testing demonstrated an incomplete left homonymous hemianopsia).

**Expert Note:** This patient demonstrates several aspects of left-sided neglect. In addition, he seems to have lost an understanding of 3-D spatial concepts in general, as exemplified by his inability to button his shirt or put on gloves correctly (this is sometimes called dressing apraxia). The precise localization of his lesion is not known, but often the posterior parts of the parietal and temporal lobes are involved. His face and speech indicate little emotion, but it is difficult to know whether he is unconcerned by his deficit or is unable to express emotion through facial expressions or intonation as a result of the stroke. The incomplete homonymous hemianopsia is produced by damage to some of the fibers in the visual radiations that run through this region. The primary motor and somatosensory cortex, which tend to lie in the territory of superior MCA branches, were ultimately spared in this patient, although in the acute phase of his stroke he showed some facial weakness because of brain swelling that affected the functioning of the primary motor cortex.

### Occlusion of a lenticulostriate branch

#### Case Summary

On his latest visit, this patient had severe paralysis of the lower part of the face, arm, and leg, all on the right. Tendon reflexes in the right arm and leg were exaggerated, and muscle tone was increased. The right plantar response was extensor (big toe up). He had marked dysarthria, but no other aspects of his neurologic exam were abnormal. Imaging studies at the time of his initial hospitalization showed left MCA stem stenosis, and an MRI confirmed a small infarct in MCA territory.

**Expert Note:** This patient had a pure motor stroke, without any impairment of sensation or any signs of cortical damage such as aphasia or neglect. The signs and symptoms can all be localized to the corticospinal and corticobulbar tracts on the left. Most commonly they are damaged either in the internal capsule or in the basal (ventral) pons. Imaging studies at the time of this patient's initial hospitalization showed severe narrowing of the left MCA stem near its origin. *MRI at 5 days showed a small infarct in the left internal capsule.*

The lenticulostriate branches of MCA are long, small diameter penetrating arteries that arise directly from the MCA stem and supply most of the internal capsule and basal ganglia. Imaging suggests that this patient may have experienced blockage of a lenticulostriate artery at its origin from the MCA stem. This produced a small infarct (or lacune) in the posterior limb of the left internal capsule, damaging the corticobulbar and corticospinal tracts on their way from the motor cortex to the brainstem and spinal cord. In another patient, a similar small infarct could be produced by collapse of a lenticulostriate artery occluding the lumen, likely caused by the lipohyalinotic wall thickening that was initiated by uncontrolled hypertension.

This small-vessel stroke produced major deficits because of its location, as in this patient who has significant motor deficits (a pure motor stroke). In other cases, a small-vessel (or lacunar) stroke may produce no abnormalities detectable on neurologic exam. The old real estate broker's adage, 'location, location, location,' is particularly applicable in this situation.

A pure motor stroke (with no abnormal sensory findings) can also be produced by damage in the base (ventral region) of the pons. Module 5 (Vertebral-basilar Territory) describes such a case.

## **Ischemia in the ACA-MCA Cortical Border Zone**

When blood flow in the carotid artery is suddenly reduced secondary to severe global hypotension or arterial dissection, structures in the cortical border zone that lies between distal branches of ACA and MCA can be at risk of ischemia. The ACA-MCA border zone is located in superior parts of the frontal lobe, which includes primary motor cortex concerned with the proximal limb girdle of the arm or leg, or both. However the exact position of the border zone varies between individuals, depending on factors such as whether there is long-standing carotid stenosis. One patient with border zone infarction suffered primarily paralysis of the contralateral hip sparing the rest of the leg. Another patient had a paralyzed shoulder and was initially worked up for a brachial plexus injury until other neurologic deficits made it clear that his problem was inside the CNS. The MCA-PCA border zone is also at risk if there is hypotension and poor distal perfusion.