Module 5. Ischemia in Vertebral-basilar Territory
Introduction and Key Clinical Examples

Objectives for Module 5

Knowledge

- List 4 common symptoms that patients might experience during TIAs produced by ischemia in vertebral-basilar territory.
- List 5 important signs and symptoms that may be seen after infarction of the dorsolateral region of the rostral medulla, and relate each to damage to a specific neuroanatomic structure.
- Describe the problem with memory that is commonly produced by bilateral PCA occlusion.

Clinical Applications and Reasoning

- Explain how bilateral signs can be produced by occlusion of a single artery in the brainstem.
- Explain why crossed motor or sensory deficits are a signature of brainstem ischemia (e.g., the combination of ipsilateral cranial nerve signs but contralateral long tract signs).
- Compare and contrast the patterns of clinical findings seen after bilateral occlusion of all cortical PCA branches vs. occlusion of only those branches supplying the primary visual cortex.
- Explain the term 'macular sparing,' and suggest one explanation for why it sometimes occurs following PCA branch occlusion.

Clinical Applications to Patient Education

- Develop points that you could use in explaining to a patient how they might personally experience a TIA produced by temporary occlusion of vertebral-basilar branches.

Transient Ischemic Attacks involving vertebral-basilar branches

TIAs in the vertebral-basilar territory can produce a number of different symptoms, reflecting the many functions of the brainstem. Because the basilar artery is a single midline artery whose branches supply both sides of the brainstem, TIAs can produce bilateral as well as unilateral motor or sensory findings. In addition, because the basilar artery bifurcates to form both posterior cerebral arteries, temporary occlusion at this upper bifurcation can also produce ischemia in the thalamus or medial temporal and occipital lobes bilaterally.

The most common symptoms reported in vertebral-basilar TIAs include vertigo (this is a false sense of movement that may be described by a patient as 'dizziness'), nausea, and unsteady gait. Their high frequency most likely reflects the number of brainstem structures related to the vestibular system and cerebellum. However, episodes of 'dizziness' or nausea are not uniquely associated with TIAs (nor is every TIA producing 'dizziness' caused by blockage of vertebral-basilar branches). Accurate diagnosis and localization of vertebral-basilar territory TIAs usually depends on other symptoms in addition to 'dizziness' such as cranial nerve abnormalities.

Transient vertigo and dysarthria – a brainstem TIA

Case Summary

The patient suddenly developed vertigo ("the room was spinning") while getting up out of his chair, lost his balance, became nauseated and vomited on the living room floor. He was able to call out to for help, but when his wife came running in from the next room she noticed immediately that his speech was slurred and hard to understand. She called 911. EMTs arrived about 20 minutes later, and transported him to the nearest hospital. He began to improve in the ambulance. By the time they reached the ED he was speaking clearly again and could sit up. An electrocardiogram revealed atrial fibrillation. He was observed closely for the next 18 hours, and there were no further events. His neurologic exam showed no lasting deficits. The patient's atrial fibrillation was addressed by electrical cardioversion and antiarrhythmic medications, and anticoagulation therapy was initiated.

Expert Note: This patient had a TIA, most likely produced by a cardiac embolus that initially lodged in one vertebral artery and then broke up. The TIA lasted approximately an hour before the deficits began to clear. Like many patients with ischemia in vertebral-basilar territory, he experienced problems related to malfunction of the vestibular system. Vertigo can be caused by peripheral (labyrinthine or CNVIII) as well as CNS (brainstem) dysfunction, but the combination of vertigo *and* dysarthria often suggests a central lesion (brainstem or possibly cerebellar involvement).

Question: Why does atrial fibrillation increase the risk of TIA or stroke*? (Answer: ineffective contraction of the atrial muscle increases the likelihood that thrombus will form in the atrium – this is a potential source of emboli)*

Some symptoms that are commonly produced by vertebral-basilar TIAs include:

- Unilateral or bilateral** weakness, clumsiness or paralysis
- Limb ataxia, staggering or veering when attempting walking [cerebellar signs]
- Vertigo or 'dizziness' [especially in combination with other brainstem/cerebellar symptoms]
- Unilateral or bilateral** numbness or paresthesias (burning, tingling etc.) or loss of sensation
- Dysarthria
- Nystagmus (repetitive involuntary eye movements) or diplopia (double vision)
- Visual field loss or blindness (suggesting unilateral or bilateral damage to the visual cortex in the occipital lobe, which is supplied by the posterior cerebral arteries)

** Since branches of the basilar artery supplies both sides of the brainstem, its occlusion can affect motor or sensory long tracts bilaterally.

Common Strokes involving the vertebral-basilar circulation

Blocked Artery or Branch	PATTERNS OF POSSIBLE DEFICITS
One vertebral artery in the rostral medulla; in some cases, blockage of the PICA branch	Loss of pain sensation on the ipsilateral side of face, but contralateral trunk and limbs; hoarseness, impaired swallowing, and ipsilateral vocal cord paralysis; ipsilateral ataxia and Horner's syndrome; vertigo, nausea, and vomiting (complete Wallenberg's syndrome).
Penetrating paramedian basilar branch in the ventral pons	Contralateral hemiplegia; involvement of face depends on whether rostral or caudal pons (pure motor stroke); cerebellar ataxia if pontine nuclei / connections involved
Basilar occlusion affecting the ventral region of the <u>rostral</u> pons <u>bilaterally</u>	Bilateral complete paralysis rendering patient motionless and mute although alert, aware, and capable of perceiving sensory stimuli. Vertical eye movements and blink spared if midbrain is not involved (locked-in syndrome)
Penetrating PCA branch supplying the thalamus	Many possibilities. May produce loss of all somatic sensations in contralateral face and body; initially hemianesthesia --later may develop a thalamic pain syndrome with painful dysesthesias in affected (anesthetic) regions
Unilateral cortical branches of PCA supplying the occipital lobe	Contralateral homonymous hemianopsia with or without macular sparing depending on the extent and location of the lesion
Bilateral occlusion of all PCA cortical branches distal to the thalamic penetrators	Inability to form memories of new facts and events; cortical blindness, which may be accompanied in the acute stage by the patient denying any problem seeing

Occlusion of one vertebral artery (or PICA) supplying the lateral rostral medulla
Case Summary On recent examination, this patient had a hoarse voice, and often coughed when he attempted to swallow. His uvula deviated to the left when saying "aah", and the gag reflex was absent on the right. He had nystagmus that became particularly noticeable when he looked to the left. The patient's gait was unsteady, and the finger-to-nose and heel-to-shin tests demonstrated dysmetria of arm and leg movements on the right. He had lost pain and temperature sensation on the *right* side of the face, but on the *left* limbs and trunk. His right pupil was smaller than the left (right miosis) but was reactive to light, and his right lid drooped slightly (mild right ptosis).

Expert Note: The patient has infarcted the dorsolateral region of the rostral medulla on the right side. This is the most common ischemic brainstem stroke. The combination of signs and symptoms that he shows is often referred to as Wallenberg's syndrome. It used to be thought

that this syndrome was only produced by posterior inferior cerebellar artery occlusion; however more recent studies suggest that in many of these patients it is thrombosis (blockage) of one vertebral artery itself that is responsible.

The hoarse voice, difficulty in swallowing, leftward deviation of the uvula, and absent right gag reflex all suggest damage to axons of CNIX or CNX (or nucleus ambiguus) on the right. The nystagmus (together with the vertigo, severe nausea and vomiting that patients like this often experience) is produced by damage to the vestibular nuclei or vestibular connections with the cerebellum. Difficulty coordinating right limb movements suggests involvement of the inferior cerebellar peduncle or perhaps part of the cerebellum itself, both on the right side.

The loss of pain and temperature sensation on the *right side of the face* is likely caused by interruption of the descending trigeminal tract, which is uncrossed. The spinothalamic tract, which runs close to the descending tract crossed in the spinal cord, so it carries information about pain and temperature in the *left (opposite) side of the body*.
Clinical Reminder: Brainstem lesions typically produce cranial nerve signs ipsilateral to the lesion but long tract sensory deficits in the trunk and limbs contralateral to the lesion because the major somatosensory pathways for the body (spinothalamic tract and medial lemniscus) cross at or below the caudal medulla.

When a Horner's syndrome (ptosis, miosis, and less commonly facial anhidrosis) is produced by a lateral medullary lesion, the cause is interruption of descending fibers (from hypothalamus or reticular formation) traveling in lateral parts of the reticular formation that will synapse on the preganglionic sympathetic neurons of the intermediolateral column in the high thoracic spinal cord.
Clinical Pearl: In Horner's syndrome, the lesion is always on the same side as the symptoms.

Occlusion of a perforating branch of the basilar artery in the pons

Case Summary

On examination, this patient had a pure motor hemiplegia that involved the arm and leg on one side of the body. Reflexes in the paralyzed arm and leg were exaggerated, and the plantar response was extensor. There were no accompanying abnormal sensory or visual findings, and no cognitive deficits such as aphasia or neglect.

Expert Note: This patient has a small-vessel ischemic stroke in the base (ventral) of the pons, caused by occlusion of a small paramedian penetrating branch of the basilar artery. The damage interrupts the corticospinal tract, which is located in the base of the pons, producing contralateral upper motor neuron paralysis of the arm and leg with exaggerated reflexes and an extensor plantar response. There is no cortical or thalamic damage so no cognitive or visual deficits are present, and the somatosensory system is also spared since these pathways run in the more dorsal parts of the pons. Recall that a small-vessel stroke in the internal capsule caused by occlusion of a lenticulostriate artery can also produce a pure motor stroke but there is usually weakness of the lower face as well as arm and leg. In pontine lesions, the face may be involved, or may be spared, as it was in this patient, because the corticobulbar fibers have separated from the corticospinal tract, and are located much farther dorsal. By contrast, the corticobulbar and corticospinal tracts run close to each other in the internal capsule, and both would likely be affected by a relatively small lesion.

If the pontine lesion is less severe, producing a milder motor problem (a hemiparesis instead of hemiplegia), there may also be ataxia or 'clumsiness' of the weakened limbs. Ataxia would not be seen if there is complete limb paralysis –- to detect the presence of cerebellar ataxia (incoordination of voluntary limb movements) it is essential that the patient be able to move the limb! The ataxia, a problem with cerebellar function, presumably reflects damage to corticopontine fibers, the pontine nuclei (griseum pontis) or their axons (pontocerebellar fibers) projecting to the contralateral cerebellum via the middle cerebellar peduncle.

Occlusion of the basilar artery in the rostral pons

Case Summary

When seen on work rounds, this patient showed no spontaneous movement of her face, tongue, neck, trunk or limbs. She was intubated to protect her airway. She was believed to be totally unresponsive until a medical student discovered that she could move her eyes vertically on command. Using a code of up for "yes" and down for "no," the student demonstrated that the patient knew what hospital she was in, how long she had been there, her diagnosis, the names of many of her doctors and nurses, and remembered many day-to-day events of her hospital stay. Although she had no other voluntary movements except vertical eye movements and the ability to lift her eyelids, she had preserved cognition, somatic sensation, vision, hearing, taste.

Expert Note: In this patient, occlusion of the basilar artery just distal to the superior cerebellar arteries has produced infarction of the base of the rostral pons *bilaterally*. Interruption of the corticobulbar and corticospinal tracts caused the complete paralysis of all voluntary movements except certain eye movements. The patient's neurologic deficit is referred to as the *locked-in* state.

Voluntary *horizontal* eye movements are impossible because they require intact projections from the frontal lobe (via the superior colliculi) to structures in the caudal pons. *Vertical* eye movements are spared since the entire pathway for voluntary vertical eye movements is contained in the midbrain (location of brainstem vertical gaze centers and the oculomotor and trochlear nuclei and nerves) and forebrain, all of which are intact because they are located rostral to the patient's lesion.

The reticular formation and other structures located in the midbrain continue to get adequate blood flow, accounting for the patient's being awake, alert, aware of herself and her surroundings, and capable of perceiving sensory stimuli. One possible explanation is that the size and arrangement of arteries in her Circle of Willis allowed enough blood to flow *from* the internal carotid arteries through the posterior communicating and *into* the posterior cerebral arteries to supply these areas.

Occlusion of a penetrating PCA branch to the thalamus

Case Summary

When seen in clinic, this patient could not feel any *gentle* stimulus over his entire right side including face, neck, body, and limbs. However, he was extremely apprehensive about sensory testing since any sufficiently intense right-sided stimulus (even shaking hands) produced an overwhelming wave of unbearable, surrealistic pain that lasted for several minutes. Cold stimulation was particularly effective in producing such pain, and he was terrified to even touch a glass containing cold water. Left-sided sensations were normal.

Expert Note: This patient has a small infarct in the posterior part of his left thalamus that involves the somatosensory nuclei (their names are VPL ventral posterolateral and VPM ventral posteromedial) where the spinal cord and brainstem somatosensory pathways terminate. Therefore all modalities of somatic sensation are impaired on his right side. His distressing over-response to cold stimuli is an example of *neuropathic pain* that reflects abnormal over-activity somewhere in the system for processing painful stimuli secondary to thalamic injury. Details of the mechanism of the *thalamic pain syndrome* are not known.

Spontaneous pain syndromes can result from lesions in other regions of the CNS as well, and by themselves do not localize the lesion to the thalamus. In this patient other neighboring parts of the thalamus and its fiber connections were apparently spared. The various thalamic nuclei provide critical inputs to specific regions of cortex, and lacunar strokes involving the thalamus may produce other deficits ranging from movement disorders to aphasias.

Occlusion of PCA cortical branches to the left occipital lobe

Case Summary

This patient had no other neurologic deficit except a complete right homonymous hemianopsia, which means that when his eyes were stationary he could not see objects on his right side with either eye. He was very aware of his visual loss, and compensated by moving his eyes in order to bring objects in his blind field into view. By this means, he was able to see objects on both sides of pictures and read across a whole page of written text without hesitation. Pupillary light reflexes were normal.

Expert Note: In this patient the medial aspect of the *left* occipital lobe is infarcted. The patient realizes that he can't see objects on his right, and moves his eyes to bring them into view. He does not neglect objects even if they are in his blind regions. By contrast, patients with a larger lesion in the *right* occipital lobe that involves neighboring parts of the right parietal and temporal lobes may lose the 'concept' of the left side of themselves or of their world.

Recall that central (macular) parts of the visual fields are mapped onto the more posterior parts of the medial occipital lobe. In different patients the macular region may be entirely in PCA territory, or it may be in the border zone between MCA and PCA cortical branches and therefore largely spared in a PCA infarct. In this patient the left macular region was apparently supplied only by the PCA, so it was entirely infarcted. Because PCA cortical branches do not supply the retina, the optic tract, or the midbrain, the pupillary light reflexes are normal.

Temporary blockage of most cortical branches of PCA bilaterally

Case Summary

This patient had heart valve surgery 6 years ago, and has a mechanical mitral valve. One day at work he abruptly collapsed, unconscious. By the time EMTs brought him to the hospital, he had regained consciousness. In the emergency department he was found to have "tunnel vision," i.e., he was blind except in the central parts of his visual fields. In addition, he could not remember new events or new facts for more than about 2-3 minutes and was therefore unable to learn any doctor's name or even to appreciate that he was in a hospital. By the following afternoon, the blindness and inability to memorize new information began to gradually clear up. Yesterday on rounds, his one remaining neurologic problem was total amnesia for a period of about 4 weeks prior to this incident and for the 24 hours that followed its onset.

Expert Note: It is likely that a piece of thrombus on the surface of the mechanical heart valve broke off. The resulting embolus probably passed readily through the large diameter basilar artery, but was arrested by the narrowing at its upper bifurcation. The patient's collapse and temporary unconsciousness were caused by the sudden interruption of input from rostral parts of the brainstem reticular formation to the cerebral cortex. Function was restored because the embolus obstructing the upper basilar bifurcation quickly broke up. However several of its pieces apparently entered both PCAs and briefly blocked cortical branches *bilaterally* before completely disintegrating. The nearly complete loss of vision reflects malfunction of much of the primary visual cortex produced by ischemia. Although these regions temporarily ceased to function correctly, their neurons did not die since the patient later regained normal visual fields. The patient's central vision was spared, suggesting that in his brain the macular representation must lie in a cortical region that never became ischemic.

Cortical branches of PCA to the inferior medial temporal lobes also supply much of the hippocampus/entorhinal area and associated white matter. These structures are important in declarative memory, as this patient illustrates. Recall that the patient was able to converse with his physicians throughout this episode, so he remembered how to use language. Furthermore, he was able to remember new people and factual information very briefly. However he was incapable of retaining or retrieving this new information after more than about three minutes elapsed. The patient soon recovered normal memory function, but experienced retrograde amnesia, a "hole in his memory," for events that occurred in the 4 weeks immediately prior to his stroke.

In this patient, brief ischemia involving the hippocampus/entorhinal area and its connections permanently disrupted the processing (sometimes referred to as "consolidation") of memories involving events just prior to the stroke. This suggests that conversion of a block of temporally-related memories from some transient to a more permanent form occurs slowly, over a period of weeks. In this patient the amnesia is the result of bilateral ischemic damage involving the medial temporal lobe.

What about the spinal cord – isn't it also nourished by vertebral branches?

Vascular disease in the spinal cord is uncommon. Unlike arteries in the brain, spinal arteries are not particularly susceptible to atherosclerosis or embolization. However, infarction of the spinal cord can occur after surgery that involves aortic repair, or in cases where a dissecting aneurysm of the aorta blocks radicular vessels. The collateral arteries that supply the spinal cord vary somewhat in size, and a catastrophic drop in blood pressure may result in ischemia in vulnerable segments (those at mid-thoracic levels are especially at risk). This is the spinal cord equivalent of a border zone infarct in the cerebral hemispheres.

When the ***anterior*** spinal artery is blocked, there is bilateral paralysis and a deficit in pain sensation below the level of the lesion. However discriminative touch, joint/limb position and vibratory sense are all spared since the posterior columns and neighboring white matter are supplied by the ***posterior*** spinal arteries.