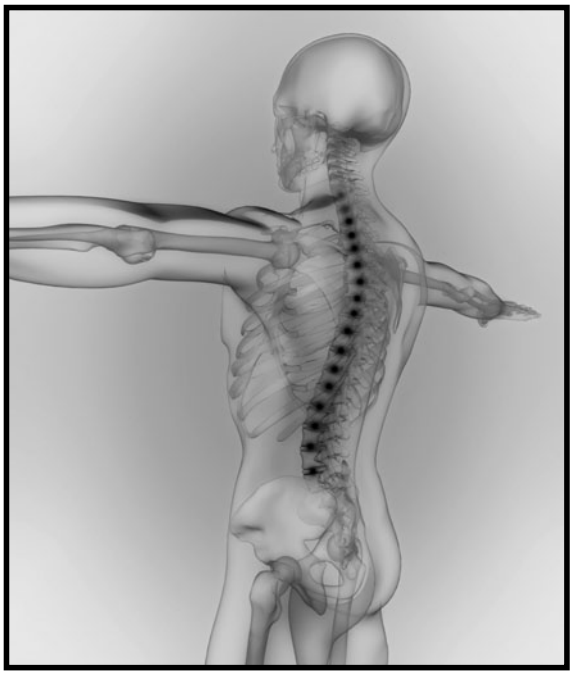
Orthopaedic Manual Therapy Diagnosis

SPINE AND TEMPOROMANDIBULAR JOINTS



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assessing eye movements are inadequate. It is probable that the proprioceptively induced positional nystagmus can be detected visually only when several upper cervical movement segments are blocked and proprioceptive input from the upper cervical joints to the vestibular nuclei is consequently much reduced.

MOTOR FUNCTION

Examination of motor function includes assessment of sensorimotor activities in patients with central neurologic disorders. They are asked to adopt postures and carry out movements that appear sequentially during normal motor development. The practitioner needs to have knowledge of central neurologic disorders to interpret motor symptoms.

Complaints relating to the movement apparatus, especially the vertebral column, require a different approach to interpretation. When assessing motor function in these cases, more emphasis is placed on the way in which the vertebral column behaves during basic activities, such as standing up, sitting down, bending, lifting, raising, carrying, pulling, pushing, throwing, and catching. This part of the examination, which is normally carried out during the patient inspection, can be repeated when the pain is reduced or has disappeared. It can then be regarded as therapy for functional recovery; this is also called sensorimotor training.

CIRCULATION

Aad van der El and Peter A. Huijbregts

To allow for a comprehensive examination, the practitioner needs to have good working knowledge of the entire vascular system that supplies the brain and spinal cord. However, there are particular blood vessels that have special relevance to examination of spinal movement.

Spinal Arteries

The direct blood supply to the spinal cord is through the posterior and anterior spinal arteries. (See **Figure 7–103**.) Both these arteries originate from the radicular arteries, which enter the spinal canal through the intervertebral foramen. Narrowing of the intervertebral foramen can cause radicular and pseudoradicular symptoms and can cause a disturbance in the perfusion of the spinal cord, which in extreme cases can lead to ischemia of the spinal cord.

Vertebral Arteries and Basilar Artery

The vertebral arteries coursing through the cervical region join together intracranially to form the basilar artery and are responsible for about 20% of the cervical and intracranial blood supply (Graziano et al., 2007; Kerry and Taylor, 2006). (See **Figure 7–104**.) Considering the course of the vertebral arteries through the cervical spine, they play an important role in manual diagnosis of this spinal region.

The vertebral artery normally courses through the intertransverse foramina of the cervical vertebrae. However, there are reports of atypical cases in which this is not the case, or only partly so. In the individual foramina, the vertebral artery has essentially fixed attachments, resulting in tension on it during movements around the vertical and sagittal axes. According to Penning (1978), the axis of flexion–extension of the cervical segments lies at the level of the vertebral artery, so movement around this axis does not lengthen it (**Figure 7–105**). It is subject to most tensile force during rotation of the cervical spine. The functional

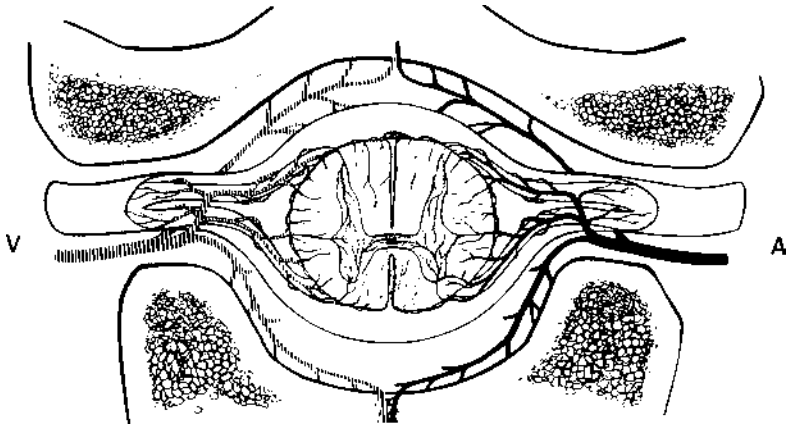

Figure 7–103 Vascular supply of the spinal cord.

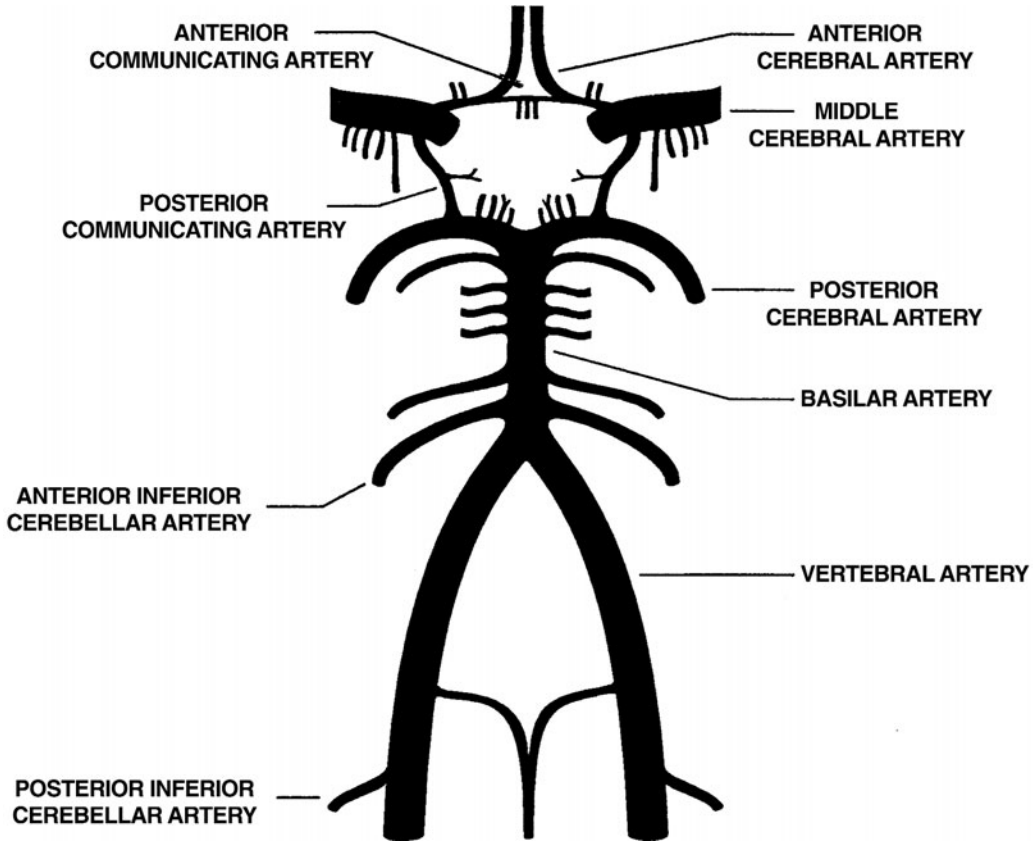

Figure 7–104 Vertebral and basilar arteries and their respective branches.

loop between C1 and C2 will be stretched when C1 is fully rotated in respect to C2 (**Figure 7–106**). The loop between C0 and C1, which is not stretched during movement, probably serves as a dampening mechanism for excessively strong pulsation (comparable to the carotid siphon, described by Dörfer and Spatz, 1935, in Penning, 1978).

Internal Carotid Arteries

The internal carotid artery branches off from the common carotid artery and runs cranially anterior to the transverse processes of C1–C3. It then enters the carotid canal in the petrous portion of the temporal bone (Clemente, 1985). Fixed to the anterior aspect of the vertebral body as well as in the carotid canal and traversing the sternocleidomastoid, longus capitis, stylohyoid, omohyoid, and digastric muscles it may experience tensile stress during cervical motions, especially contralateral rotation and extension-contralateral rotation, and the clinical tests for the vertebral arteries have also been proposed as tests for the internal carotid arteries (Haneline and Triano, 2005; Kerry and Taylor, 2006; Licht

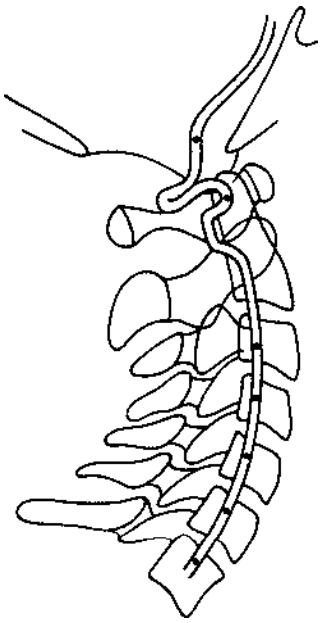

Figure 7–105 Localization of the flexion–extension axis in relation to the vertebral artery.

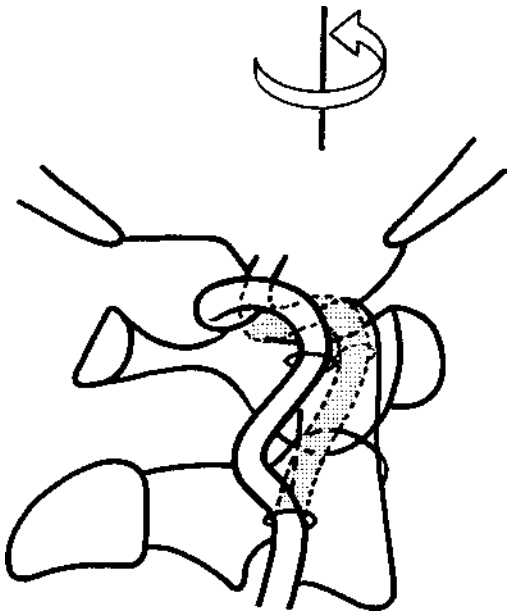

Figure 7–106 Elongation possibility of the upper cervical loop of the vertebral artery during rotation.

et al., 2002). Add to this the fact that 80% of the cerebral circulation is supplied by the internal carotid arteries and that, therefore, tests proposed in the past to examine the vertebrobasilar system could more reasonably be seen to test the ability of the internal carotid arteries to compensate for a physiologic and/or pathologic loss of posterior circulation, and the reemphasis in recent years from the posterior system to include also the anterior circulatory system and speak of cervical artery dysfunction becomes more understandable (Kerry and Taylor, 2006; Kerry et al., 2008). However, we need to still put this reemphasis in perspective by pointing out that Terrett (2000) in his review of 185 cases of manipulation-associated cervical artery injuries only found 5 cases (2.7%) that reported injuries to the internal carotid artery.

Structures Supplied by the Vertebrobasilar System

The area supplied by the vertebral arteries and the basilar artery, with their branches and anastosmoses with other arteries, include a cervical area and an intracranial area. (See **Figure 7–107**.)

The Cervical Supply Area

The branches of the vertebral artery that originate from it in the cervical region are the following:

- *Spinal arteries*. These divide into the anterior and the posterior radicular arteries that vascularize the anterior and posterior nerve root and the spinal ganglion. The anterior central artery and the anterior and posterior vertebral canal artery also branch from the vertebral artery.
- *The muscular, joint, and cutaneous rami*. These vascularize the intrinsic cervical musculature of the segments C2–C7, the interspinal ligament, the flaval ligament, the joint capsules of the intervertebral and uncovertebral joints, and the area of skin supplied by the dorsal ramus.
- *Ascending arteries of the axis*. These vascularize the body and dens of C2, the transverse ligament, and the cruciform atlantal ligament.

The branches of the vertebral artery that originate from it subforaminally (below the foramen magnum) are the anterior, posterior, and lateral spinal arteries. These arteries, which run longitudinally, with their transverse branches and anastosmotic connections with the radicular arteries, vascularize the cervical spinal cord. In most cases, the anterior spinal artery receives anastosmoses from the ventral radicular arteries only below C4. This means that the upper cervical spinal cord is more vulnerable with regard to its blood supply. In ischemia of the cervical cord, a number of clinical phenomena will occur that are consistent with a vertebrobasilar insufficiency (VBI) picture. The encephalic (or brain-related) symptoms indicative of other ischemic syndromes, however, are absent.

The Intracranial Supply Area

The vertebral arteries join together to form the basilar artery. Just before they join, the posterior inferior cerebellar artery branches off. This artery vascularizes the dorsolateral part of the medulla oblongata, a large part of the posterior lobe of the cerebellum, the cerebellar vermis, and a number of cerebellar nuclei.

The basilar artery with its branches vascularizes the medulla oblongata, the pons, the mesencephalon, and parts of the cerebellum. The reticular formation and the vestibular system with its nuclei also depend on the basilar artery and its branches for their blood supply. In fact, the labyrinthine arteries branch off early from the basilar artery or the anterior inferior cerebellar arteries to supply the vestibular nucleus and inner ears making these structures very susceptible to ischemia and explaining the symptom of dizziness as an early presentation of ischemic syndromes affecting the posterior circulation (Oostendorp, 1988).

The posterior cerebral arteries that originate from the basilar artery at the level of the clivus vascularize parts of

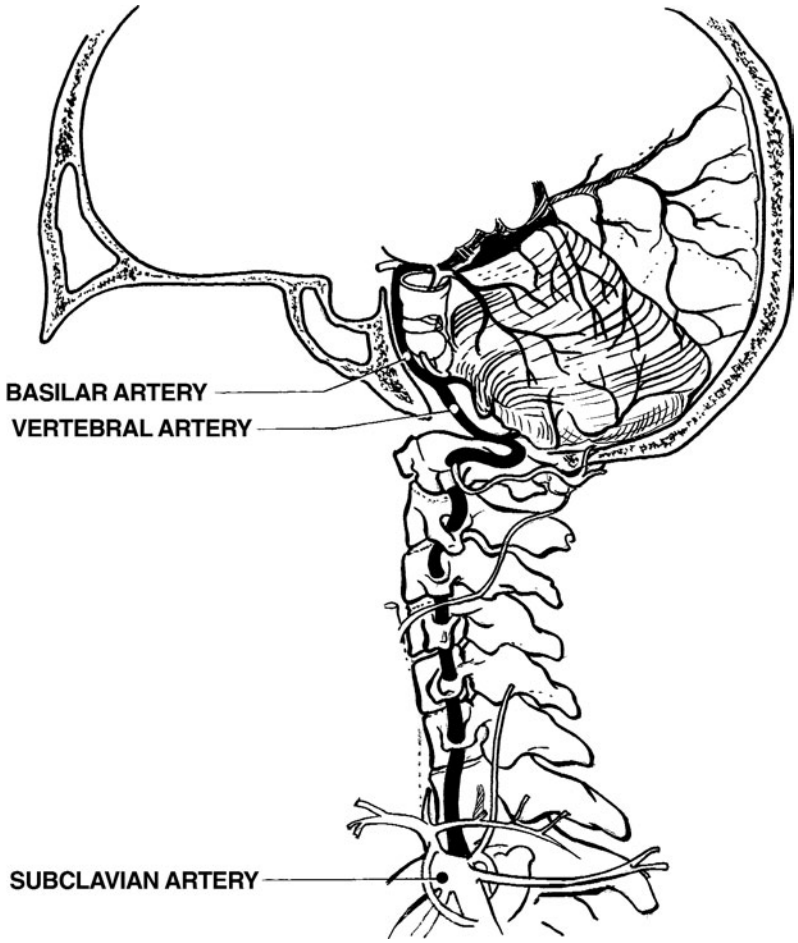

Figure 7–107 Extra- and intracranial areas supplied by the vertebral artery and basilar arteries and their branches.

the thalamus and hypothalamus, the occipital lobe, the basal part of the temporal lobe, the dorsal part of the capsula interna, and the lateral corpus geniculatum.

The posterior cerebral arteries and the posterior communicating arteries join together to form the circle of Willis.

Segments of the vertebral artery

As shown in **Figure 7–108** the segments of the vertebral artery are the following:

V_1: In 89% of cases, the *extravertebral part* runs from its origin from the subclavian artery to the transverse foramen of C6. It enters at C7 in 3%, at C5 in 6%, and at C4 in 1% of the population (Thiel, 1991). In this area, the vertebral artery runs ventrally of the transverse process of T1 and C7, and the first rib, and dorsally of the longus colli muscle and the anterior scalene muscle.

V_2: The *intravertebral part*, which in most cases runs anteromedially from C6 to C2 through the transverse foramina, is surrounded in between the foramina by ligament and muscle tissue. The uncovertebral joints form the anteromedial boundary. The cervical nerve roots and cervical spinal nerves lie dorsal to the vertebral artery.

V_3: The *atlantoaxial part* runs from C2 to C0. In this section, the vertebral artery forms a dorsolateral loop. From the transverse foramen of the atlas, which lies more laterally, the vertebral artery follows a medial path via the vertebral artery sulcus and behind the lateralis masses of the atlas. The ventral boundary is formed by the capsule of the atlanto-occipital joint and the dorsal boundary by the obliquus capitis superior muscle and the rectus capi-

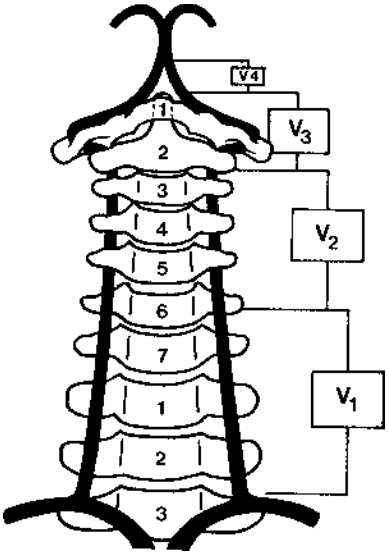

Figure 7–108 Segments of the vertebral artery.

tus posterior major muscle. In some patients, a bony bridge is present called the ponticulus posterior where the artery crosses over the posterior arch of the atlas: the added fixation is hypothesized to make the artery even more prone to tensile lesions at this level (Triano and Kawchuk, 2006).

V_4: The *subforaminal and intracranial part.* The subforaminal part of the vertebral artery passes through the posterior atlanto-occipital membrane, the dura mater, the pia mater, and the arachnoid, and continues its way intracranially.

Segments of the Internal Carotid Artery

As does the vertebral artery, the internal carotid artery also has four separate portions (Clemente, 1985): the cervical, petrous, cavernous, and cerebral portions.

The cervical portion is formed at the bifurcation of the common carotid into the external and internal carotid arteries at the upper border of the thyroid cartilage. The internal carotid artery is located here posterior and lateral to its external counterpart, overlapped by the sternocleidomastoid muscle and covered by the deep anterior cervical fascia, platysma, and the skin. Superiorly to this bifurcation the hypoglossal nerve (12th cranial nerve), the posterior belly of the digastric muscle, the stylohyoid muscle, and the occipital and posterior auricular branches of the external carotid artery traverse the internal carotid artery. Even more superior the artery is bordered by the glossopharyngeal nerve (9th cranial nerve), the pharyngeal branch of the vagus nerve (10th cranial nerve), the longus capitis muscle, the superior cervical sympathetic ganglion, and the superior laryngeal branch of the vagus nerve. At the skull base level, the artery borders the glossopharyngeal, vagus, accessory, and hypoglossal nerves (9th–12th cranial nerves). This close anatomic connection to the various cranial nerves will serve later to explain some of the nonischemic signs and symptoms of internal carotid artery dissection.

The petrous portion starts where the internal carotid artery enters the carotid canal in the petrous portion of the temporal bone. Relevant to the clinical presentation of internal carotid artery dissection is its close anatomic connection to the cochlea and tympanic cavity and also to the trigeminal ganglion (ganglion of the 5th cranial nerve).

The cavernous portion of the internal carotid artery is named after the close anatomic connection the artery has in this aspect of its course, with the layers of the dura forming the cavernous sinus. Most relevant to clinical presentation of a dissection is its close proximity to the abducens nerve (6th cranial nerve).

After perforating the dura mater the internal carotid artery passes between the optic and oculomotor nerves (2nd and 3rd cranial nerves) and then divides into its cerebral branches.

Structures Supplied by the Internal Carotid Artery

The cervical portion of the internal carotid artery gives off no branches (Clemente, 1985). The petrous portion mainly provides the caroticotympanic artery that supplies the tympanic membrane together with branches from the posterior auricular and maxillary arteries. The cavernous portion gives off the cavernous branches, hypophyseal branches, ganglionic branches, and the anterior meningeal branch, which provide the arterial supply to the hypophysis, the trigeminal ganglion, and the anterior cranial dura mater. The cerebral portion of the internal carotid artery provides the ophthalmic artery supplying the optic nerve, the orbital structures including the extraocular muscles, and the eyeball. After joining the circle of Willis by way of the posterior communicating arteries thereby establishing the major anastosmosis for a deficient posterior circulation, the cavernous portion of the internal carotid artery divides into the anterior and medial cerebral arteries that supply the major portions of both hemispheres (Clemente, 1985).

Movements of the Cervical Spine and Their Effect on Cervical Artery Perfusion

In 1927, De Kleyn and Nieuwenhuyse reported decreased and absent vertebral artery blood flow based on

cadaver perfusion studies in various head and neck positions. Based on these early perfusion studies and on the anatomic considerations with regard to the cervical arteries discussed earlier, the sustained extension–rotation and sustained rotation tests have been proposed and widely instructed and clinically used as tests to determine the presence of vertebrobasilar artery dysfunction.

Chrost (Gutmann and Biedermann, 1984) and Hillen and Fonville (1980) discussed the effects of physiologic movements on blood flow in the vertebral artery. In line with the work by De Kleyn and Nieuwenhuyse (1927), their work is an example of the earlier work on this topic that has dominated clinical reasoning within OMT for decades, with changes occurring in this area only recently. These authors concluded that there is a reduction in blood flow in the vertebral artery during physiologic movement of the cervical spine even in the absence of any anomaly or pathology. Rotation of the cervical spine seems to have a particularly marked effect in reducing blood flow in the contralateral vertebral artery.

Hillen and Fonville (1980) report that the reduced vascular resistance in the vertebral artery during rotation of the cervical spine is the greatest in the ipsilateral vertebral artery at the atlantoaxial segment. They believe that this is because of the relatively long distance from the transverse foramen of the atlas to the centrally located axis of rotation of the cervical spine, and to the compression at the level of the transverse foramen of the atlas (**Figure 7–109**).

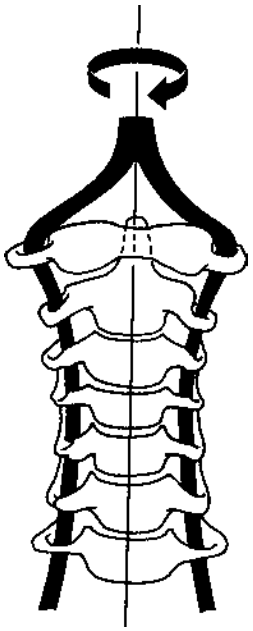

Figure 7–109 Distance of the vertebral artery to the axis of rotation of the cervical vertebrae.

Sidebending seems to have no effect on blood flow in the vertebral artery on the contralateral side, and only a limited influence (10%) ipsilaterally. Penning (1978) states that the position of the facet joints in the segments of C2–C7 during sidebending does not differ much from their position during rotation. He argues that the difference in osteokinematic trajectory between cervical rotation and sidebending must, therefore, be caused by the rotation of C1–C2 being added to the sidebending. This argument, and the relatively small reduction in blood flow during sidebending, supports the position of Hillen and Fonville (1980), namely, that during rotation in the atlantoaxial segment, the reduction in vascular resistance is greater on the ipsilateral side.

During flexion and extension of the cervical spine, there is no reduction in blood flow. This is also consistent with Penning's theory (Penning, 1978) (Figure 7–105). Although under normal circumstances extension does not lead to any reduction in blood flow in the vertebral artery, it seems capable of causing symptoms of functional vertebrobasilar insufficiency. This might be the result of a reflexogenic reaction to mechanical irritation of the smooth muscle tissue of the vertebral artery wall or mechanical stimulation of nociceptive units.

Chrost, referenced by Gutmann and Biedermann (1984), investigated the blood flow in both vertebral arteries during different movements of the cervical spine and produced a chart of the results (**Figure 7–110**). Expressed in percentages, these reductions are approximately as follows:

Flexion and Extension: 0%

Sidebending: Ipsilateral, 10%; contralateral, 0%

Rotation: Ipsilateral, 20%; contralateral, 75%

Flexion, rotation: Ipsilateral and contralateral, 55%

Extension, rotation: Ipsilateral, 50%; contralateral, 75%

Flexion, sidebending, contralateral rotation: Ipsilateral (with regard to the rotational component), 30%; contralateral, 95%

Although there is little or no difference in total blood flow between the two final movement combinations, the last one gives more information about the functioning of the less affected ipsilateral vertebral artery. The reduction of flow in the contralateral vertebral artery to 5% means that the body is almost completely dependent on the blood supply via the ipsilateral vertebral artery and the internal carotid arteries by way of the anastosmotic connection through the circle of Willis. It is not clear why Chrost (referenced by Gutmann and Biedermann, 1984) did not study the combinations of flexion with sidebending and ipsilateral rotation, or of extension with sidebending and ipsilateral and contralateral rotation. He also provides no explanation

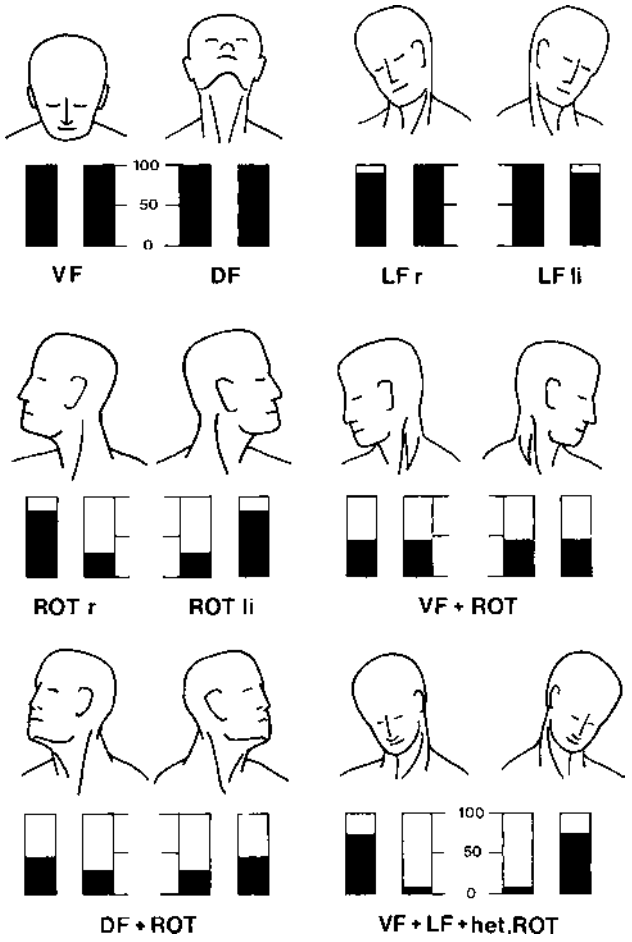

Figure 7–110 Percentage of perfusion of both vertebral arteries with uniplanar and multiplanar physiological motions of the cervical spine.

with regard to the fact that flexion and extension—unlike the combined movements—have no effect on blood flow in the vertebral arteries.

More recent studies have provided much less unequivocal findings than the older studies discussed earlier and have forced OMT clinicians to reevaluate the near-certainty with which in times past they approached the topic of cervical artery dysfunction based solely on history and physical examination data.

Extension–rotation as a clinical test—discussed in more detail in the section titled "Dynamic Coordination" earlier in this chapter—has been extensively studied with equivocal results. Some authors have reported significant decreases in blood flow (Rivett et al., 1999; Yi-Kai et al., 1999), whereas other studies have found no changes (Arnold et al., 2004; Licht et al., 2000). Case reports have noted false-negative results (Rivett et al., 1998; Westaway et al., 2003), and case series have reported 75–100% false-positive results (Arnold et al., 2004; Haynes, 2002). Research findings for the sustained cervical rotation test are equally equivocal with significant decreases (Arnold et al., 2004; Mitchell, 2003; Nakamura et al., 1998; Rivett et al., 1999; Yi-Kai et al., 1999) or no effect noted on vertebral artery blood flow or volume (Haynes et al., 2002; Licht et al., 1999). We discussed data on concurrent criterion-related validity and clinical implications in the section titled "Dynamic Coordination" earlier.

Earlier studies concentrated solely on the effect of physiologic movements on perfusion in the vertebral arteries, but more recent studies have also studied the effects on perfusion in the internal carotid artery. Refshauge (1994) noted an increase in right internal carotid artery blood flow velocity with sustained contralateral rotation in healthy volunteers. In contrast, Licht et al. (2002) found no change in peak flow or time-averaged mean flow velocity in the ICA during sustained extension–rotation test. Clinically relevant is that the patients in that study nonetheless experienced symptoms (vertigo, visual blurring, nausea, hemicranial paresthesiae) classically considered a positive response on this test. Rivett et al. (1999) reported an increase in internal carotid artery blood flow velocity with cervical extension and attributed this to narrowing in the internal carotid artery. In contrast to the other two studies, they noted a decrease in peak systolic and end diastolic blood flow velocity in both ICA during sustained rotation. Again relevant with regard to the clinical interpretation is the fact that these authors found no between-group differences for subjects that were positive or negative on this test.

With all these studies, we have to of course acknowledge the chance of type II error (not finding a between-group difference where in reality one does exist) because of the small sample sizes used; for some studies, we must consider the effect of using asymptomatic subjects on external validity. In summary, research on the hemodynamic effect of the sustained rotation and the sustained extension rotation tests produces equivocal results, thereby calling into question our previously held assumptions with regard to the effect of physiologic movements of the cervical spine on cervical artery perfusion.

VERTEBROBASILAR INSUFFICIENCY

Both vertebral arteries and the internal carotid arteries form a functional unit. Under normal circumstances, reduction of the blood flow in one of the two vertebral arteries is completely compensated by an increase in blood flow in the other vertebral artery and the anterior system. If one

vertebral artery is not able to compensate the partial or complete loss of flow in the other one, this may reduce perfusion of the area supplied by the vertebrobasilar system, especially in the case where the anterior system is insufficiently capable of compensating. The use of provocative movement of the cervical spine has been suggested as a way of testing this compensatory mechanism. Disturbance of blood flow in the vertebrobasilar system is called vertebrobasilar insufficiency. Ausman et al. (1985) define the term as follows:

> Vertebrobasilar insufficiency is the term used to denote recurrent periods of relative ischemia in the area supplied by the vertebrobasilar arterial system, consequent upon a temporary reduction or blocking of blood flow in the vertebral artery and the basilar artery together with their branches.

Oostendorp (1988) distinguishes between vertebrobasilar insufficiency and functional vertebrobasilar insufficiency. The former can be a consequence of organic abnormalities, physiologic movements of the cervical spine combined with increased sympathetic activity in the cervicothoracic transitional area, and or a combination of the two. Distinctions can, therefore, be made between structural, functional, and combined vertebrobasilar insufficiency.

Structural Vertebrobasilar Insufficiency

Structural vertebrobasilar insufficiency is the form of vertebrobasilar insufficiency in which identifiable symptoms occur as a consequence of an organic abnormality of the vertebral arteries, or changes in its lumen as a result of structural abnormalities in the cervical spine or the musculature that lies directly ventral to the vertebral artery.

Functional Vertebrobasilar Insufficiency

Functional vertebrobasilar insufficiency is the form of vertebrobasilar insufficiency that occurs in the absence of structural abnormality and in which complaints and symptoms are provoked by movements of the cervical spine, especially rotation (Oostendorp, 1988).

Temporary reduction in blood flow in the vertebral arteries as a result of physiologic movements of the cervical spine, in which there is an absence of structural abnormalities, is hypothesized to be caused by increased sympathetic activity in the cervicothoracic transitional area.

Combined Vertebrobasilar Insufficiency

Combined vertebrobasilar insufficiency is the form of vertebrobasilar insufficiency in which the occurrence of identifiable complaints and symptoms is the result of the combined factors responsible for structural and functional vertebrobasilar insufficiency.

It is clear from these definitions that the identifiable symptoms of vertebrobasilar insufficiency are caused by different mechanisms, which may be present separately or in combination. The abnormalities and changes that can play a part in structural or combined vertebrobasilar insufficiency are described in the section titled "Structures That Can Compress the Vertebral Artery," which follows.

Movements of the cervical spine generally cause changes in the length of the vertebral artery. The extent to which the vertebral artery, which is anchored in the transverse foramina, can be stretched without damage to its walls depends on the amount of surplus length present and the number of elastic fibers in the tunica media of the artery. If the elongation capacity of the vertebral artery is exceeded, there is a danger that its walls may be damaged.

Vulnerable locations are the following:

- The transverse process of the axis
- The transverse process of the atlas
- The lateral mass of the atlas
- The place where the vertebral artery passes through the dura mater

Structures That Can Compress the Vertebral Artery

The *extravertebral part* of the vertebral artery (segment V_1) has as its ventral boundary the longus colli and anterior scalene muscles, and as its dorsal boundary the transverse processes T1 and C7 and the first rib. Three-dimensional combined movement of extension, ipsilateral lateral flexion and rotation of the cervical spine lengthens these muscles as well as the vertebral artery. If the muscles are hypertonic and shortened, they will press the vertebral artery against the first rib and the transverse processes of T1 and C7. If the resulting reduction in blood flow is not fully compensated, it may result in symptoms and complaints typical of vertebrobasilar insufficiency.

The *intravertebral part* of the vertebral artery (segment V_2) has as its anteromedial boundary the uncovertebral joints. If as a result of osteoarthritic changes of the uncovertebral joint there is lateral osteophyte growth in the direction of the transverse foramen, this may cause compression of the vertebral artery, especially during sidebending and rotation of the cervical spine (**Figure 7–111**). Processes such as spondylosis or spondylarthrosis can also cause temporary compression of the vertebral artery in this segment during movement of the cervical spine. However, a prolapsed disk is more likely to compress the dural sleeve and the nerve root than the vertebral artery.

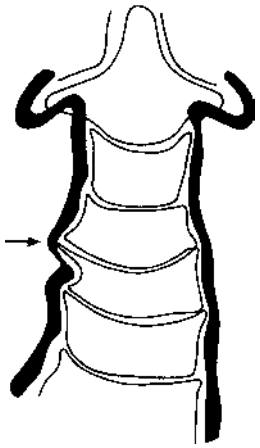

Figure 7–111 Compression of the vertebral artery as result of osteoarthritic changes of the uncovertebral joint.

In the *atlantoaxial part* of the vertebral artery (segment V_3), the changes in blood vessel resistance during rotation and extension of the cervical spine are the greatest in the contralateral vertebral artery. Normally, these changes in resistance do not cause problems; however, additional compressive factors such as congenital blood vessel abnormalities, or anomalies and abnormal positions of the segments of C1 and C2, can have a negative effect on blood flow. Other predisposing factors for vertebrobasilar insufficiency include congenital blood vessel abnormalities such as unilateral or bilateral hypoplasia or unilateral aplasia of the vertebral artery; anomalies of the circle of Willis; and anomalies, acquired structural changes, and positional deviations in the upper cervical spine including a ponticulus posterior (Triano and Kawchuk, 2006).

In the *intracranial part* of the vertebral artery (segment V_4), the only location where it is vulnerable from a biomechanical perspective is where it passes through the dura mater, the pia mater, and the arachnoid.

To study the commonly held assumption that the cervical artery seems most mechanically vulnerable in its V_3 segment, Kawchuk et al. (2008) studied two populations of patients with established cervical artery dissection. One group consisted of a 5-year retrospective cohort of 25 patients admitted to a hospital for vertebral artery dissection not related to major trauma or associated with cervical manipulation. The second group consisted of 26 of 64 cases on which Haldeman et al. (2002) reported in a retrospective review of medicolegal cases where manipulation was suggested as a causative factor for vertebral artery dissection. All cases included had diagnostic imaging or reports available to determine the level of the dissection. Kawchuk et al. (2008) noted that the V_3 segment was most affected with a prevalence ratio expressing prevalence in V_3 as compared to baseline prevalence in V_1 in the manipulation group of 8.46 (95% CI: 3.53–20.24). The prevalence ratio in the nonmanipulation group was 4.00 (95% CI: 1.43–11.15). The authors noted that there was a higher prevalence in the V_3 segment irrespective of exposure to cervical manipulation showing the inherent increased mechanical vulnerability of the V_3 segment.

Tonic Spasm of the Vertebral Artery

Another proposed important causal factor in vertebrobasilar insufficiency is vascular spasm (Gutmann, 1985). The pathophysiologic mechanism that causes vascular spasm is not fully understood, but it causes the smooth muscle fibers of the arterial wall to contract. The contraction may be of short or long duration. According to Lechtape-Grutter and Zülch (1971), a vascular spasm is a reversible contraction of the smooth muscle tissue of the arterial wall that may last minutes, hours, days, or weeks. This occurs in response to mechanical, and/or chemical, and/or neurogenic stimulation. Spasm of the vertebral artery, spreading to the basilar artery and its branches, causes temporary disturbances in perfusion of the area supplied by the vertebrobasilar arterial system. Gutmann (1985) distinguishes three kinds of factors that may cause vascular spasm. They are often present in combination.

Mechanical Stimulation

Rotation and movements that include rotation cause mechanical compression that may affect the blood flow in the vertebral artery. The mechanical effect on the vertebral artery, the nerve fibers of the perivascular plexus, and the vertebral nerve does not seem to be a direct cause of the vascular spasm; however, when mechanical stimulation causes local damage of the blood vessel wall causing the release of chemical substances, these might cause the vascular spasm indirectly.

Mechanically caused spasm of the vertebral artery without tissue damage has a short latency and generally lasts only a short time (seconds, minutes, hours); this means that the symptoms are often reversible. Mechanically caused spasm of the vertebral artery with tissue damage has a relatively longer latency and generally lasts longer (hours, days); the symptoms are becoming irreversible (Oostendorp, 1988).

Chemical Stimulation

When the wall of the blood vessel is damaged, substances such as prostaglandin E2 and serotonin are released. These substances increase the sensitivity to stimulation of the arterial wall and the free nerve endings of the perivascular

plexus of the vertebral artery (Gutmann, 1985). This increased sensitivity is a predisposing factor for the vascular spasm. It means that a mechanical stimulation that under normal circumstances would not cross the threshold of the free nerve endings will now result in depolarization of these nerves. The increase in discharge frequency of the unmyelinated fibers causes an increase in the release of substance P at the peripheral and central endings of the primary nociceptive neurons. This sensitizes the peripheral and central receptive fields of these neurons. This nociceptive activity can lead to a short-term or long-term increase in the activity of the sympathetic system (Oostendorp, 1988).

Neurogenic Stimulation

Stressors can increase tonic activity within the sympathetic system even in the absence of a peripheral source of nociceptive stimulation. When the activity of the sympathetic system is chronically raised by stress (discussed in Chapter 8) a minor mechanical stimulus can cause an angiospasm. The evidence that the raised activity in the sympathetic nervous system is a causal factor in angiospasms lies in the fact that injecting substances that block alpha-receptors can end these spasms. The reaction of norepinephrin is blocked through blocking alpha-receptors; this removes the sympathetic effect on the muscles in the wall of the blood vessel.

A long-lasting increase in the activity of the vasoconstricting subsystems of the sympathetic system leads to hyperreactivity of the smooth muscle cells of the vertebral artery and dysregulation of the blood flow to the vertebral artery and its branches via the vasa vasorum (Oostendorp, 1988). The resulting trophic disturbance may lead to an ischemic or vascular inflammatory reaction. This increases the sensitivity of the vessel wall and of the perivascular nerve fibers. An inflammatory reaction with chronic trophic disturbance of the vessel wall leads to fibrosis of the wall and fibrotic attachments to the surrounding tissues. This reduces the viscoelasticity of the blood vessel wall and the mobility of the vertebral artery during movements of the cervical spine. Both these factors increase the likelihood of damage to the arterial wall. Therefore, from a diagnostic perspective, it is important to determine the reactivity of the autonomic system in general and of the wall of the vertebral artery in particular before carrying out cervical provocation tests and assessing and interpreting the results of these tests.

CERVICAL MANIPULATION AND THE RISK OF STROKE

In the context of the OMT assessment of circulatory function of the cervical region as a precursor to potential manipulative intervention in this region, we cannot ignore the association made between cervical manipulation and stroke, more specifically, cervical artery dissection. Although overall population incidence at 2.6 per 100,000 is extremely low, cervical artery dissections account for approximately 20% of strokes in young versus 2.5% of strokes in older patients (Graziano et al., 2007). As Terrett (2000) noted: "The temporal relationship between young healthy patients without osseous or vascular disease who attend a spinal manipulative therapy practitioner and then suffer these rare strokes is so well documented as to be beyond reasonable doubt indicating a possible causal relationship."

There are two types of vertebral artery–related strokes (Terrett, 2000). In Wallenberg syndrome or dorsolateral medullary syndrome of Wallenberg occlusion of the posterior inferior cerebellar artery, frequently resulting from distal extension of a vertebral artery dissection, leads to destruction of the nuclei and pathways in the dorsolateral medulla oblongata. Another cause may be the occlusion of the parent vertebral artery, in which case the syndrome is called syndrome of Babinski–Nageotte. Ischemia of the inferior cerebellar peduncle leads to ipsilateral ataxia and hypotonia. Destruction of the descending spinal tract and the trigeminal nucleus causes a loss of pain and temperature sensation on the ipsilateral face in addition to loss of the ipsilateral corneal reflex. Destruction of the ascending lateral spinothalamic tract causes loss of pain and temperature sensation in the contralateral trunk, which together with the sensory loss in the ipsilateral face results in a pathognomonic presentation of alternating analgesia. Ischemia of the descending sympathetic tract causes Horner's syndrome; damage to the lower vestibular nuclei causes nystagmus, vertigo, nausea, and vomiting; and ischemia in the nucleus ambiguous of the glossopharyngeal nerve can cause hoarseness, dysphagia, or intractable hiccups.

Locked-in syndrome or cerebromedullospinal disconnection syndrome occurs as a result of occlusion of the midbasilar artery. This effectively transects the brain stem at the midpons level. Because the reticular formation and the ventral pons are unaffected, the patient retains consciousness but decerebrate rigidity develops as a result of the cerebrospinal tracts having been destroyed. The nuclei for the 5th through 12th cranial nerves are destroyed, but the 4th cranial nerve is spared leaving only eye convergence and upward gaze for the patient to communicate with his environment. Skin sensation remains grossly intact because the lateral spinothalamic tract is usually spared and the patient can still hear because the auditory nerves ascend in the brainstem lateral to the infarction area.

Evidence linking manipulation to stroke has included multiple narrative reviews of case reports found in the literature (Di Fabio, 1999; Ernst, 2002; Terrett, 2000; Triano

and Kawchuk, 2006). Hurwitz et al. (1996) acknowledged the likely high underreporting bias and noted an estimated risk adjusted for an only 10% reporting rate in the literature of 5–10 per 10 million for all complications, 6 in 10 million for serious complications, and 3 in 10 million for the risk of death.

Rothwell et al. (2001) compared 582 patients with vertebrobasilar accidents over the period 1993 to 1998 with age- and sex-matched controls from the provincial insurance database in Ontario, Canada. They also determined exposure to chiropractic using this same database. These authors found that subjects younger than 45 years were five times more likely (95% CI: 1.31–43.87) to have visited a chiropractor in the month preceding the stroke. This same age group was also five times (95% CI: 1.34–18.57) more likely to have had three or more chiropractic visits with a cervical diagnosis in the month prior to the stroke. No significant association was noted for subjects older than 45.

Cassidy et al. (2008) used a very similar study design comparing 818 patients with vertebrobasilar accidents to age- and sex-matched controls from a provincial insurance database and also found an odds ratio (OR) of 3.13 (95% CI: 0.52–1.32) for having visited a chiropractor in the month before the stroke in those younger than 45 years, whereas the OR was 0.83 (95% CI: 0.52–1.32) for those older than 45. However, these researchers also looked at visits to general medical practitioners preceding the stroke and found an OR of 3.57 (95% CI: 2.17–5.86) for those under 45 and 2.67 (95% CI: 2.25–3.17) for patients having visited their medical doctor in the month preceding the vertebrobasilar accident. These authors suggested that the similar association between chiropractic and medical visits might indicate that patients with an undiagnosed vertebral artery dissection seek clinical care for headache and neck pain before having a stroke.

Signs and Symptoms of Cervical Artery Dysfunction

Previously, we have discussed structural, functional, and mixed vertebrobasilar insufficiency. Within the context of structural insufficiency we have to address the possibility of vertebral artery dissection and subsequent thrombosis and embolization leading to both nonischemic and ischemic presentations. Despite the fact that clinical evidence indicates that cervical artery dysfunction predominantly affects the posterior circulation (Terrett, 2000), OMT clinicians also need to be familiar with the presentation of internal carotid artery dissection. This becomes all the more relevant considering the possibility that patients may seek out an OMT clinician with a stroke in progress mistakenly assuming that the initially mild symptoms may be helped with OMT intervention.

Clinicians are likely most familiar with the classic cardinal signs and symptoms (**Table 7–9**) of vertebrobasilar compromise. We should note that diagnostic utility of these symptoms has yet to be established. With regard to raising the clinical suspicion of cervical artery dissection, it is important to realize that ischemic symptoms are not the only symptoms that occur with cervical artery dissection. Nonischemic symptoms usually develop first and are likely the result of deformation of nerve endings in the tunica adventitia of the affected artery and direct compression on local somatic structures (Kerry and Taylor, 2006). In fact, these nonischemic symptoms occur hours to days and even a few weeks prior to the ischemic findings (Blunt and Galton, 1997). In the case of internal carotid artery dissection, this delay has even been reported to possibly be as much as years (Haneline and Lewkovich, 2004). Ischemic findings develop in 30–80% of all dissections. Up to 20% of patients progress to a full cerebrovascular accident (Blunt and Galton, 1997). Nonischemic symptoms are unique to the pathology of dissection but ischemic symptoms can, of course, be expected to be similar for all underlying causes of cervical artery dysfunction.

Although the classic cardinal signs and symptoms for vertebral artery compromise as discussed in Table 7–9 can be part of the presentation, additional symptoms have been described for cervical artery dysfunction. **Table 7–10** provides ischemic and nonischemic signs and symptoms associated with cervical artery dissection (Blunt and Galton, 1997; Haneline and Lewkovich, 2004; Kerry and Taylor, 2006). Relevant to the physical examination are the cranial nerve palsies that may occur with cervical artery dissection. In the section titled "Segments of the Internal Carotid Artery" earlier in the chapter, we discussed the close anatomic association of the internal carotid artery with multiple cranial nerves and their nuclei. It is easy to imagine how a dissecting artery with its increasing diameter may compress surrounding somatic structures including adjacent cranial nerve structures. Dissection of the internal

Table 7–9 Classic Cardinal Signs of Vertebrobasilar Compromise: Five Ds and Three Ns

Dizziness
Drop attacks
Diplopia (including amaurosis fugax and corneal reflux)
Dysarthria
Dysphagia (including hoarseness and hiccups)
Ataxia of gait
Nausea
Numbness (in ipsilateral face and/or contralateral body)
Nystagmus

Table 7–10 Nonischemic and Ischemic Signs and Symptoms of Cervical Artery Dysfunction

	Vertebrobasilar System	*Internal Carotid Artery*
Non-ischemic	Ipsilateral posterior neck pain Ipsilateral occipital headache Sudden onset and severe Described as stabbing, pulsating, aching, "thunderclap," sharp, or of an unusual character: "a headache unlike any experienced before" Very rarely C5–C6 nerve root impairment (due to local neural ischemia)	Ipsilateral upper and midcervical spine pain Ipsilateral frontal-temporal or periorbital headache Sudden onset, severe, and of an uncommon character Horner's syndrome Pulsatile tinnitus Cranial nerve palsies Ipsilateral carotid bruit Neck swelling Scalp tenderness Anhydrosis face
Ischemic	Five **D**s and three **N**s (see Table 7–9) Vomiting Loss of short-term memory Vagueness Hypotonia and limb weakness affecting arm or leg Anhydrosis: lack of facial sweating Hearing disturbances Malaise Perioral dysesthesia Photophobia Clumsiness Agitation Cranial nerve palsies Hindbrain stroke: Wallenberg or locked-in syndrome	Transient ischemic attack Middle cerebral artery distribution stroke Retinal infarction Amaurosis fugax: temporary blindness Localized patchy blurring of vision: scintillating scotomata Weakness of extraocular muscles Protrusion of the eye Swelling of the eye or conjunctiva

carotid artery mainly causes dysfunction of the 9th through 12th cranial nerves with the hypoglossal nerve initially affected and then the other three nerves; eventually all cranial nerves except the olfactory can be affected. Whereas cranial nerve dysfunction has a nonischemic etiology in internal carotid artery dissection, it is part of the ischemic presentation of a vertebral artery dissection. As noted earlier, ischemic signs and symptoms of cervical artery compromise can logically be expected to be similar irrespective of underlying pathology.

DIAGNOSIS

See the section titled "Examination Strategy" in Chapter 10, page 235 for more information.

History

When taking the history, the practitioner lists the patient's complaints and symptoms. Where cervical artery dysfunction is suspected, there are three complaints that are of primary diagnostic significance. These are the following:

- Headache
- Neck pain
- Vertigo

Terrett (2000) provided data on the presenting complaints of 137 chiropractic patients who subsequently had a manipulation-associated stroke:

- 47.4% noted neck pain and stiffness
- 19.7% noted neck pain, stiffness, and headache
- 16.8% had torticollis

Additional presenting complaints included low back pain (2.2%), abdominal complaints (2.2%), (kypho)scoliosis (1.5%), head cold (1.5%), upper thoracic pain (1.5%), upper limb numbness (0.7%), and hay fever (0.7%). Keeping in mind the distinct differences between indications proposed for chiropractic manipulation and OMT interven-

tion, we still have to note the nonspecific nature of these presenting complaints and, therefore, their limited value in identifying patients at risk for cervical artery dysfunction.

Whether cervical artery dysfunction is related to patients presenting with a stroke in progress or whether manipulation in fact causes these serious adverse events remains unclear, but it does seem plausible that in the case of a pathologically weakened artery mechanical forces such as those induced during OMT intervention may cause damage to the cervical arteries. In addition to the nonischemic and ischemic signs and symptoms of cervical artery dissection and other forms of insufficiency outlined previously, the OMT clinician also needs to identify possible risk factors predisposing the artery to dysfunction whether iatrogenic, caused by mechanical events other than manipulation, or spontaneous. **Table 7–11** provides risk factors as identified in the literature.

With regard to the role of iatrogenic causes and direct vessel trauma, we discussed the increased odds ratio of cervical artery dysfunction and cervical manipulation earlier but should note that association, of course, does not imply causation. Although no specific data are available relevant to OMT clinical practice, Beaudry and Spence (2003) attributed 70 of 80 traumatically induced cases of vertebrobasilar ischemia to motor vehicle accidents. From a differential diagnostic perspective it should be noted that many patients after whiplash trauma note dizziness and meet criteria for inner ear pathology (Grimm, 2002; Oostendorp et al., 1999; Wrisley et al., 2000).

Although age of 30 to 45 years and female gender have been proposed as risk factors, Terrett (2000) indicated that the overall distribution of patients with regard to gender and age attending for chiropractic care closely matches the gender and age distribution of those with serious adverse events, thereby somewhat discounting these proposed risk factors. Kawchuk et al. (2008) also found no association for age and gender and the incidence of cervical artery dissection.

Arteriopathies predisposing the artery to dysfunction as a result of pathologic weakening of the vessel wall include Marfan syndrome, Ehlers–Danlos syndrome, fibromuscular dysplasia, cystic medial necrosis, osteogenesis imperfecta, and autosomal dominant polycystic kidney disease. Alpha-1-antitrypsin deficiency initially showed highly elevated odds ratios, but this association currently finds little support in the literature. In all, these risk factors can at best be suspected based on physical examination but would seem relevant if noted in the medical history. Research evidence for these arteriopathies in the etiology of cervical artery dysfunction, however, is limited.

Cardiovascular risk factors proposed include hypertension, tobacco use, hypercholesterolemia, hyperlipidemia, diabetes, and atherosclerosis. Most research into this area has compared patients with cervical artery dysfunction to patients with ischemic strokes. Perhaps as a result of this underlying difference in pathophysiology most cardiovascular risk factors actually show an odds ratio below 1, indicating a "protective" function. Of course, this is likely because of the methodology of the research. Hypertension with an odds ratio of 1.94 (95% CI: 1.01–3.70) was the only significant risk factor. Although seemingly plausible, the evidence for atherosclerosis as a risk factor is based solely on cadaver studies and the finding that blood flow is proportional to the fourth power of vessel diameter (Mitchell, 2002).

There is a noted seasonal variation in the incidence of cervical artery dissection with significantly more cases occurring in winter as compared to other seasons (Paciaroni et al., 2006). Indeed, one study showed an adjusted odds ratio of 3.1 (95% CI: 1.1–9.2) for an acute infection in the 4 weeks preceding a cervical artery incident (Guillon et al., 2003).

Rubinstein et al. (2005), in a systematic review noted additional clinically relevant risk factors for migraine (OR = 3.6; 95% CI: 1.5–8.6); recent infection (OR = 1.6; 95% CI: 0.67–3.80), and trivial trauma including cervical manipulation (OR = 3.8; 95% CI: 1.3–11). Triano and Kawchuk (2006) reported an OR of 1.6 (95% CI: 0.67–3.80) for

Table 7–11 Proposed Risk Factors of Cervical Artery Dysfunction

Atherosclerosis
Hypertension
Hypercholesterolemia
Hyperlipidemia
Hyperhomocysteinemia
Diabetes mellitus
Genetic clotting disorders
Infections
Smoking
Free radicals
Upper cervical instability
Migraine
Direct vessel trauma
Iatrogenic causes
Endothelial inflammatory disease (eg, temporal arteriitis)
Arteriopathies
Age 30–45 years
Female gender
Thyroid disease
Oral contraceptive use

coughing, sneezing, or vomiting; vascular risk factors and a current smoking habit had odds ratios of 0.14 (95% CI: 0.34–0.65) and 0.49 (95% CI: 0.18–1.05). Although earlier research implicated oral contraceptive use as a risk factor, Haneline and Lewkovich (2004) indicated that currently no consensus exists on relevance of this proposed risk factor.

Physical Examination

When the history has been taken and the data analyzed, the aims of the subsequent examination are formulated. At this stage, the following questions must be answered:

- Is examination by a manual therapist indicated or contraindicated?
- Is imaging needed to rule out possible contraindications?
- If examination by a manual therapist is indicated, what tests should be carried out?
- In what order should they be carried out?

If the history does reveal any central neurologic symptoms that would suggest structural vertebrobasilar insufficiency, targeted neurologic and other tests are carried out that do not put the patient at risk. In this context, we should think of cranial nerve examination, especially considering the place cranial nerve palsies have in the nonischemic presentation of internal carotid dissection and the ischemic presentation of vertebral artery dissection. Observation of the miosis (inability to dilate a pupil), ptosis (droopy upper eye lid), enophthalmus (deeper seated eye), and anhydrosis (decreased sweating ipsilateral head and shoulders) consistent with Horner's syndrome also should serve as a red flag in this regard. Auscultation for a carotid bruit may be indicated: Magyar et al. (2002) noted 56% sensitivity and 91% specificity for detection of a 70–99% carotid stenosis when compared to Doppler duplex ultrasound. In the absence of findings indicative of relevant pathology, the OMT clinician can continue with a careful progressive examination noting the very limited diagnostic utility of and potential dangers associated with the cervical extension and extension–rotation tests in the section titled "Dynamic Coordination." Any findings indicative of the nonischemic and ischemic presentation of cervical artery dysfunction require immediate referral for medical management.

For differential diagnosis of the headache complaint that may be part of the presentation of cervical artery dysfunction, it will be helpful to be familiar with the location and pain referral patterns of myofascial trigger points as described elsewhere in this text. Myofascial trigger points have been implicated in the etiology of tension-type headaches. Although less likely to be confused with cervical artery–related thunderclap headache, the clinician should be familiar with the International Headache Society diagnostic criteria of cervicogenic headache as well (Olesen, 2004). The headache most likely to be confused with thunderclap headache due to its intensity, location, and associated neurologic symptoms is migraine headache. The following clinical prediction rule for the diagnosis of migraine may be helpful in the differential diagnosis (Detsky et al., 2006). The rule consists of the following five questions:

1. Is it a pulsating headache?
2. Does it last between 4 and 72 hours without medication?

Table 7–12 Differential Diagnostic Characteristics for Cervicogenic Dizziness, Benign Paroxysmal Positional Vertigo (BPPV), and Cervical Artery Dysfunction

	Dizziness Type	*Nystagmus and Dizziness Characteristics*	*Associated Signs and Symptoms*
Cervicogenic dizziness	Positioning-type	No latency period Brief duration Fatigable with repeated motion	Nystagmus Neck pain Suboccipital headaches Cervical motion abnormality on examination
BPPV	Positioning-type	Short latency: 1–5 seconds Brief duration: $<$ 30 seconds Fatigable with repeated motion	Nystagmus
Cervical artery dysfunction	Positional-type	Long latency: 55 $\pm$ 18 seconds (Oostendorp, 1988) Increasing symptoms and signs with maintained head position Not fatigable with repeated motion	Ischemic and (depending on etiology) possibly nonischemic signs and symptoms as described in Table 7–10

3. Is it unilateral?
4. Is there nausea?
5. Is the headache disabling (with disabling headaches defined as headaches that disrupt a patient's daily activities)?

When the patient answers yes to four or more of these five questions, the positive likelihood ratio (LR) for a diagnosis of migraine headache is 24 (95% CI: 1.5–388). With a yes answer to three questions, the positive LR is 3.5 (95% CI: 1.3–9.2). For a yes answer to one or two of these criteria, the positive LR is 0.41 (95% CI: 0.32–0.52). The mnemonic POUNDing (Pulsating, Duration of 4–72 hours, Unilateral, Nausea, Disabling) may be helpful for clinicians when using this clinical prediction rule.

For differential diagnosis of a main complaint of dizziness the spin-stop test may be helpful for implicating the vestibular system provided that tests for more serious pathology have all been negative. A Hallpike–Dix maneuver can also be helpful to implicate benign paroxysmal positional vertigo (BPPV): positional nystagmus has been shown to identify patients with posterior semicircular canal BPPV with 78% sensitivity, and specificity as high as 88% has been reported. The sinusoidal rotation test (discussed earlier in the section titled "Static and Dynamic Coordination") can be used to help in the differential diagnosis of cervicogenic dizziness. **Table 7–12** provides details helpful for the differential diagnosis of the more common forms of dizziness. Vidal and Huijbregts (2005) have provided an extensive template for the diagnostic process in patients with a main complaint of dizziness.

If cervicogenic dizziness is implicated, the next stages in the examination are functional examination of the cervical spine and examination for possible changes in particular organs and tissues. The results of these examinations will show whether the patient is likely to have any of the following conditions:

- A structural or a functional vertebrobasilar insufficiency/cervical artery dysfunction
- A vestibular disturbance caused by a local problem
- Functional disturbances of the spine
- Tonically raised sympathetic activity