CASE REPORT

UNEVENTFUL UPPER CERVICAL MANIPULATION IN THE PRESENCE OF A DAMAGED VERTEBRAL ARTERY

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ABSTRACT

Objective: To discuss a case in which a patient with a previously injured vertebral artery underwent manipulation in the upper cervical spine without alteration of her symptom pattern. The literature concerning the relative safety of specific upper cervical manipulative techniques is reviewed.

Clinical Features: A 42-year-old woman had a 3-week history of unilateral suboccipital pain that she related to a sudden twisting of her head and neck that occurred while she was putting sheets of drywall on top of her car. Subsequent examination by a neurologist 2 weeks later was unremarkable, and a tension-type headache was diagnosed. Approximately 10 days later (3 weeks after injury), a single high-velocity upper-cervical manipulation (incorporating slight rotation and full lateral flexion) was performed with no change in her symptom pattern. Two weeks after that, the patient had development of a lateral medullary syndrome (also known as Wallenberg syndrome) after she briefly extended and rotated her upper cervical spine while painting a ceiling.

Intervention and Outcome: The patient was treated with anticoagulant therapy, and the lateral medullary infarct healed without incident. The spinocerebellar and subtle motor symptoms also resolved, but the ipsilateral suboccipital headache and the loss of temperature sensation associated with the spinothalamic tract lesion were still present 9 months later.

Conclusion: This case report demonstrates that vigorous manipulation of the upper cervical spine is possible without injuring an already damaged vertebral artery. It is suggested that the line of drive used during the single manipulation, almost pure lateral flexion with slight rotation, was responsible for the apparent innocuous response. Guidelines for the evaluation and management of vertebral artery dissection are reviewed. Because it is currently impossible to identify patients at risk of having a dissected vertebral artery with standard in-office examination procedures, rotational manipulation of the upper cervical spine should be abandoned by all practitioners, and schools should remove such techniques from their curriculums. (J Manipulative Physiol Ther 2002;25:472-83)

Key Indexing Terms: *Chiropractic Manipulation; Stroke; Vertebral Artery Dissection; Lateral Medullary Syndrome*

INTRODUCTION

Vertebral artery dissection (VAD) with subsequent brainstem infarct is without doubt the most feared complication associated with spinal manipulative therapy. Although occasionally fatal,[1] VAD may also be responsible for the development of a "locked-in syndrome" in which the fully conscious patient is able to listen and comprehend (because the auditory nerves and higher cortical centers are spared) but is unable to speak and is completely paralyzed except for vertical eye movements and convergence; hence, the description of this patient as a

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"corpse with living eyes." Terrett[2] notes that this syndrome occurs in approximately 5% of vertebral artery dissections, and it forces the patient, essentially a paralyzed mute, to communicate in code, incorporating an alternating series of upward and downward eye movements.

Even though research aimed at identifying the causes of VAD is still in its infancy, it seems likely that a small percent of the population is predisposed to dissection because of underlying arteriopathy that weakens the inner lining of the vertebral artery, allowing it to tear when exposed to mechanical stress. Although various researchers estimate the prevalence of VAD at anywhere from 1 in 400,000 to 1 in 14 million,[3-5] most researchers estimate prevalence at about 1 in 1.5 million.[6-8] In what is without doubt the most thorough study addressing the risks associated with spinal manipulative therapy, Klougart et al[7] reviewed all reported cases of VAD in Denmark between 1978 and 1988 and noted a 1 in 1.3 million risk of cerebrovascular accident with lower cervical manipulation and a 1 in 900,000 risk with upper cervical manipulation. It must be kept in mind, however, that even these numbers most likely significantly underestimate the potential for VAD, as advances in magnetic resonance angiography (MRA) may likely demonstrate that many patients with postmanipulative headaches or dizziness may actually have had an undiagnosed intimal tear or dissection that healed without subsequent neurologic sequelae.

In an attempt to identify patients prone to VAD, a variety of provocative tests have been developed that mechanically stress the vertebral artery. These tests, which are referred to by a variety of eponyms, including Maigne's, George's, Hautant's, Smith and Estridge's, and DeKleyn's and Wallenberg's, all incorporate a combination of cervical rotation and extension.[9-11] Theoretically, the individual who is predisposed to dissection will have brainstem ischemia caused by temporary occlusion of the vertebral artery in the test position. Symptoms of ischemia include dizziness, dysarthria, diplopia, tinnitus, vomiting, loss of consciousness, and hemiplegia.[12,13,16] Unfortunately, these tests have been proven repeatedly to be invalid.[9,11,14,15] The major drawback to all of the premanipulative testing procedures is their high rate of false-positives and false-negatives. False-positives are likely to occur because it is impossible to differentiate the dizziness associated with brainstem ischemia from the dizziness associated with stimulation of the upper cervical mechanoreceptors (which affect the vestibular nuclei). Conversely, false-negatives are likely to occur because the contralateral vertebral artery is able to fully compensate for a VAD by maintaining an adequate blood supply even when there is a complete obstruction of the ipsilateral vertebral artery.[17] As a result, Côtè et al[14] caution that labeling a patient as being prone to VAD because of a positive provocative test is "unethical and unacceptable" because of the potential for producing psychologic harm. Finally, Refshauge[13] points out that a negative provocative test in no way implies that a vertebral artery could tolerate the external forces applied during vigorous manipulation. It is because of these and other studies that so many investigators have come to the conclusion that no single premanipulative test will allow the practitioner to identify the patient prone to VAD a priori.[1,8,19] This presents an important challenge to all manual practitioners, that is, because it is difficult to identify the patient presenting with or potentially prone to VAD, what steps should be taken to minimize the risk of dissection?

This case history demonstrates the significance of this dilemma in that a patient had a 3-week history of unilateral headache that was eventually determined to be the result of a damaged vertebral artery. The literature regarding the relative safety of specific manipulative techniques is reviewed, and a brief discussion is included that outlines important factors to consider in the evaluation, diagnosis, and subsequent management of VAD.

Case Report

A 42-year-old woman had a unilateral left-sided suboccipital headache that had been present for 2 weeks. She related the headache to an incident in which she was helping to lift sheets of drywall on top of her car. The patient remembered "twisting" her neck as she was lifting the sheets of drywall and almost immediately having an "intense, deep pressure" that had persisted without change since then. Of particular significance, the patient repeatedly emphasized that this headache was unlike any headache she had ever experienced. The patient was a vegetarian, was not taking estrogen replacement therapy, had no history of allergies to medications, did not smoke, and was physically fit.

Her history was significant only for occasional tension-type headaches that had previously responded almost immediately to chiropractic manipulation of the upper cervical spine. The patient had received approximately 12 treatments in the last 2 years for the tension-type headaches, which were typically associated with poor posture at her workplace.

Neurologic examination was completely unremarkable; the pupils were equal, round, and reactive to light. Upper and lower extremity deep tendon reflexes were 2+ bilaterally, and vibration sense, point localization, and 2-point discrimination were within normal limits. Orthopedic testing revealed slight discomfort with cervical compression tests, and slight restrictions were noted in bilateral cervical rotation and flexion. Sensory examination with a Wartenberg pinwheel was nonremarkable, and motor examination revealed 5+ strength in the upper and lower extremities. There was no sign of intention tremor, and rapid alternating movements of the upper extremity were smooth and coordinated. The Babinski sign was plantar bilaterally, and there was no sign of ankle clonus. Examination of cranial nerves

II through XII was also nonremarkable; facial movements were symmetric, there was no sign of tongue or pallet deviation, and eye movements were intact in all planes. There was also no sign of nystagmus, and Horner's syndrome was absent. Of note, temperature sensation was tested by applying a cold metal ruler over the forearms, and there was no perceived difference between the 2 sides.

Despite the negative neurologic examination and previous excellent response to cervical manipulation for tension-type headaches in the past, the patient was not treated and was instead referred to a local hospital for additional testing. She was initially seen by a resident who performed a brief neurologic examination and requested a second opinion from her senior staff physician. This doctor, despite a negative neurologic examination, was also concerned about the unusual nature of the headache and therefore arranged for an appointment with a neurologist that day. Again, as with the other practitioners, examination by the neurologist was unremarkable, so he diagnosed a tension-type headache and prescribed barbiturates and rest.

These medications made little or no difference in the intensity of the headache. As a result, a trial of deep tissue massage, hold-relax stretches, and high-voltage galvanic electrotherapy in conjunction with the application of moist heat was attempted. It is interesting that the patient reported significant (albeit temporary) relief of the suboccipital pain only with high-voltage galvanic electrotherapy applied to the upper cervical spine. Because this finding reinforced the diagnosis of tension-type headache, a single high-velocity manipulation was performed (this manipulation occurred 1 week after her visit to the neurologist and 3 weeks after the initial development of the headache). The manipulation was a high-velocity, low-amplitude thrust with a PIP index contact on the left C2-3 articulation with full ipsilateral lateral flexion and slight contralateral rotation of the head ($<$45 degrees). A series of loud audible releases occurred, with cavitation of several upper cervical joints occurring simultaneously.

Unlike her previous response to manipulation for tension-type headaches, the patient had no change in her symptom pattern either immediately or over the following 48 hours. As a result, additional manipulations were not performed, and the patient continued to receive high-voltage galvanic electrotherapy applied with hot packs to the upper cervical spine. This treatment protocol continued to provide significant relief lasting anywhere from 6 hours to 24 hours before the headaches would return.

Approximately 2 weeks later, the patient attempted to paint the corner of a ceiling, during which time she maintained her head and neck in an extended position for a few moments. Shortly thereafter, the patient's headache significantly worsened, and she became mildly confused and dizzy. In spite of this, she continued to work, and within a few hours her voice became slightly hoarse, which she attributed to seasonal allergies. Later that evening, the patient was taking a hot shower and noted dysesthesias along the right side of her torso. Of particular importance, the patient stated that there was a precise mid-sagittal bisection running from her upper sternum to her pubic symphysis in which she was unable to adequately perceive the heat from hot water on the right side of this bisection line.

It is surprising that in spite of what seemed to be clear spinothalamic tract involvement, the patient was able to perceive cold stimulus applied to the upper extremities equally, and a complete neurologic workup was repeated and was again nonremarkable. Nonetheless, the patient was informed of the possibility of a cerebrovascular accident or tumor affecting the spinothalamic tract and was immediately referred back to the neurologist. The neurologist again performed a physical examination and, despite the mid-sagittal bisection and clear spinothalamic tract involvement, diagnosed stress-induced paresthesias and assured the patient "it's not a stroke or a tumor." In an attempt to lessen the patient's anxiety and because the patient had "no longitudinal encounters with medical doctors," the neurologist ordered magnetic resonance imaging to be performed the next morning and told her she would be "contacted in about 2 weeks with the results."

Approximately 2 hours after magnetic resonance imaging was performed, the patient was informed that there was a lateral medullary infarct (affecting the spinothalamic tract) with a clear obstruction of the upper vertebral artery blood flow. MRA was immediately ordered to delineate more clearly the damage to the vertebral artery. This study revealed a classic "string of pearls" pattern in the left vertebral artery with dissection extending proximally from just below the posterior inferior cerebellar artery (PICA). The patient was immediately admitted to the hospital, where she began heparin therapy for 7 days. Once her clotting times stabilized, she was released from the hospital, and she continued to take an anticoagulant for the next 6 months. The spinocerebellar symptoms quickly resolved, and a mild right-sided strength deficit that developed during her hospital stay also resolved quickly. The left suboccipital headache persisted and was often exacerbated by her habit of sleeping prone with her neck rotated and extended. The spinothalamic tract symptoms (ie, the right-sided decrease in temperature sensation) also remained unchanged, and the patient had to learn to test the temperature of hot items (such as oven racks and hot bath water for her children) with her left hand.

Discussion

Although the vertebral arteries may dissect spontaneously,[20] it is the traumatic dissections that are of most concern to manual practitioners. The first published case of traumatic VAD resulting in death was reported in 1872 in a letter to the editor that appeared in Lancet.[21] Since then, at

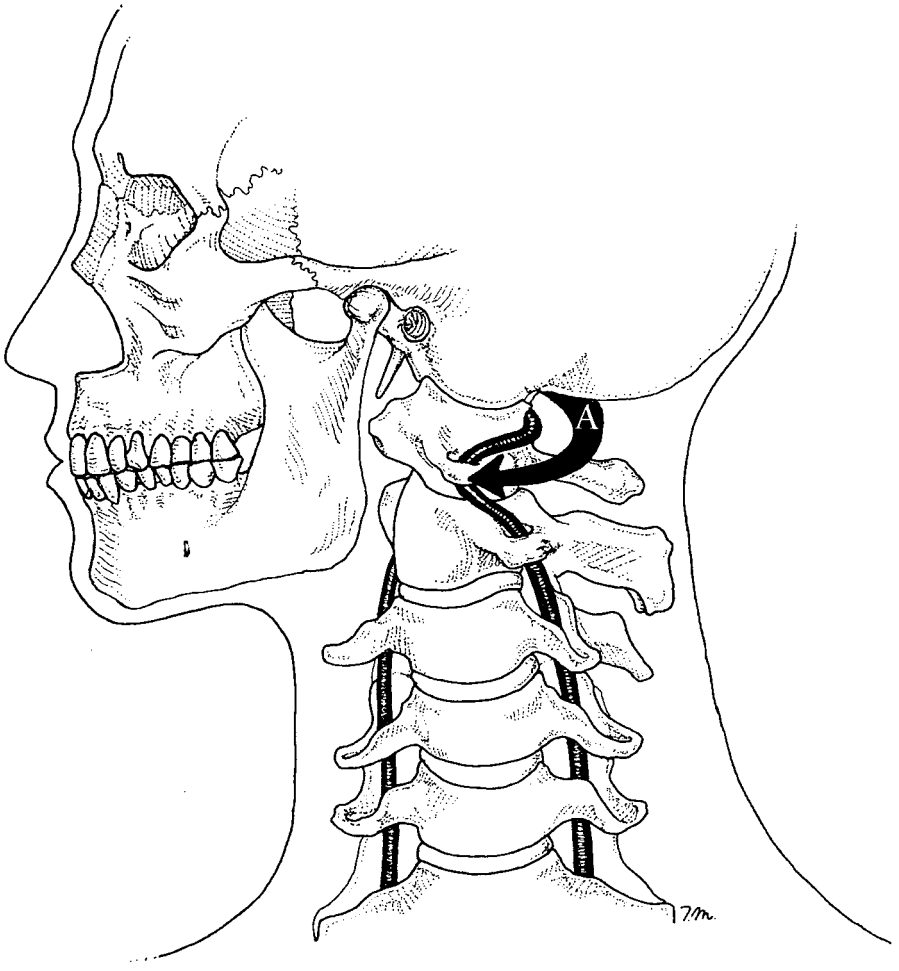

Fig 1. *Upper cervical rotation (A) stresses the vertebral artery as it passes through the transverse foramina of the atlas.*

least 138 cases of manipulation-related VADs have been published in the medical literature.[2]

The mechanism for vertebral artery dissection is relatively straightforward: because of the mobility of the upper cervical spine, the extracranial segments of the vertebral artery are especially vulnerable to a combination of shear and compressive forces, particularly at the atlanto-axial and atlanto-occipital articulations. This explains why dissection, which has been reported to occur in all 4 segments of the vertebral artery,[22] is most likely to occur between C1 and the occiput.[2,24] The typical explanation is that because the vertebral artery is relatively fixed in the transverse foramina of C-1 and C-2,[2] contralateral rotation of these vertebrae, which are capable of approximately 45 degrees of motion in each direction,[23] produces a significant degree of tensile and compressive strain where the artery pierces the transverse foramina of C-1: tensile strain where the artery is stretched and compressive strain on the concave side where the artery bends over bony landmarks (Fig 1).

In addition to undergoing strain with contralateral rotation, the vertebral artery may also undergo strain with extension of the head and neck; for example, because the vertebral artery is relatively fixed at the point of dural penetration[20] and where it wraps around the atlas,[25] atlanto-occipital extension is capable of placing significant tensile strain on the upper portion of the vertebral artery as the inferior aspect of the occipital condyle glides forward in the atlantial socket (Fig 2). Given the location of the dissection that occurred in this patient, this is the most likely mechanism for her injury. It must be emphasized that because rotation and extension of the upper cervical spine are coupled movements,[26] upper cervical rotation is also capable of producing significant degrees of suboccipital extension. For example, Mimura et al[26] performed a 3-dimensional motion analysis of the cervical spine in 20 men aged 25 to 31 years and noted that upper cervical rotation was coupled with an average of 14 degrees of extension between the occiput and C-2. This research suggests that upper cervical rotation may

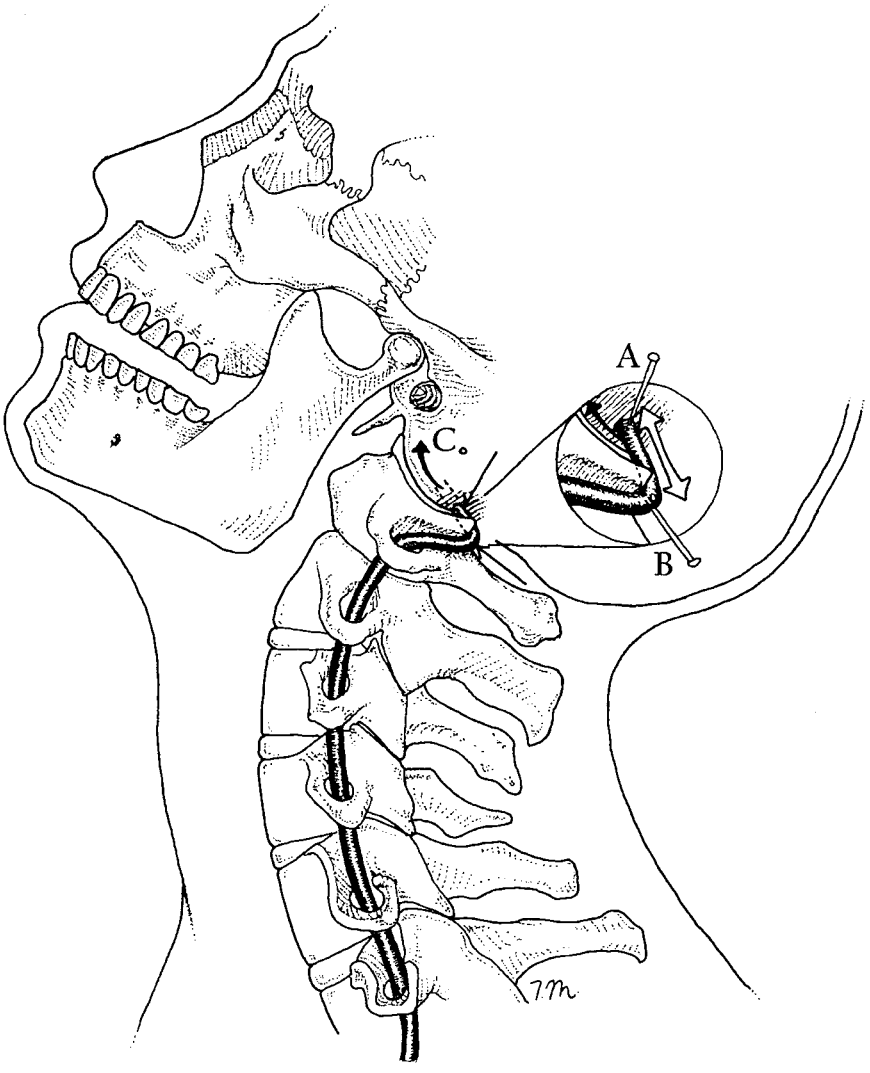

Fig 2. *Because the vertebral artery is relatively fixed where it pierces the dura (A) and where it wraps around the atlas (B), upper cervical extension (C) places significant tensile strain on the distal extracranial segment of the vertebral artery (white arrow).*

inadvertently induce large amounts of extension between the occiput and C-1 and could therefore place significant strain on the suboccipital portion of the vertebral artery.

Regardless of the mechanism, if the vertebral artery is sufficiently stressed, a tear may develop in the intima of the artery, thereby exposing the subsurface collagen to blood flow. If left alone, the damaged artery may heal without incident as platelets become adherent to the site of the tear, mix with fibrin, and form a "white thrombus" that retracts into the vessel wall and may or may not produce narrowing of the lumen.[27] However, if the damaged blood vessel is again stressed or if the initial injury to the intima is significant enough, blood, being forced under systolic pressure, may enter the artery wall, thereby splitting its layers. If this split occurs between the intima and tunica media, the result is often significant stenosis of the arterial lumen. Conversely, blood entering the wall through a split between the tunica media and adventitia will typically produce aneurysmal dilation of the artery.

If the dissection is of insufficient size to obstruct the lumen, blood will continue to make its way to the brainstem, and the only symptom, if present, might be the characteristic headache that is so often associated with this injury. This is particularly true when adequate collateral circulation from the contralateral vertebral artery is present. On the other hand, if the subintimal hematoma or subadventitial aneurysm is of sufficient size to obstruct blood flow, symptoms related to brainstem infarct eventually develop. Although arterial obstruction may be the result of emboli fragmenting off the hematoma and traveling distally to block the basilar or anterior spinal arteries, the most common scenario is obstruction of the PICA. Because the PICA supplies blood

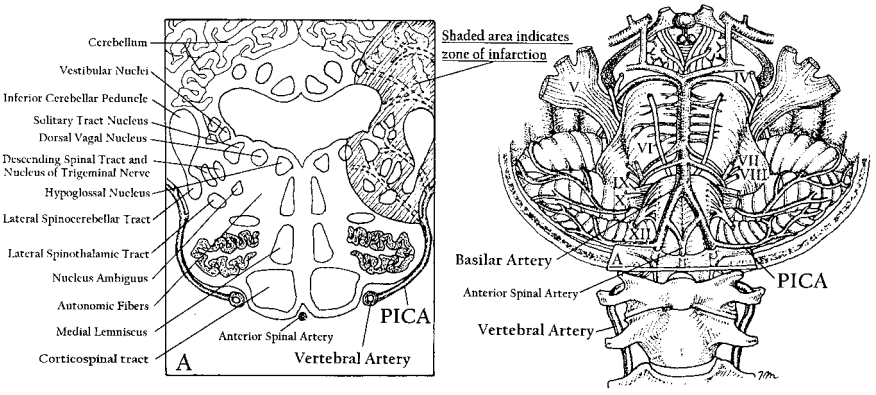

Fig 3. *The vertebral artery and its branches. The cross section (A) is taken through the brainstem at the level of the posterior-inferior cerebellar artery (PICA). Cross section partially modified from Caplan.*[32]

to the lateral aspect of the medulla (hence the term lateral medullary syndrome) and the cerebellum, a wide range of important tracts may be affected (Fig 3).

In a review of symptoms associated with VAD, Sturzenegger[28] notes that the most common initial symptom is headache. The clinical significance of this cannot be understated, because the patient's description of the nature of the headache is often the only diagnostic clue signaling the presence of VAD. As noted by Swanson[18]: "a new headache of sudden onset always raises concern about an intracranial process, especially in middle aged or elderly patients." Other common symptoms associated with VAD and subsequent lateral medullary syndrome are described by Terrett[2] as the "5 Ds And 3 Ns": dizziness, dysphagia, dysarthria, diplopia, drop attacks, ataxia, nystagmus, nausea, and numbness. Hicks et al[29] note that ocular manifestations are particularly common, because they occur in approximately 86% of patients with VAD; for example, diplopia, blurred vision, conjugate gaze paralysis, nystagmus, sixth cranial nerve palsy, and loss of corneal reflex are frequently present. Less common symptoms include snout anesthesia, tachycardia, ipsilateral facial numbness, contralateral facial numbness, upside-down vision, hiccups, an inability to sneeze, and a runny nose.[30-32] A common yet subtle symptom associated with VAD is lateral pulsion.[32] This represents a gait abnormality in which the walking patient is unsteady and has a tendency to fall toward the ipsilateral side (this is particularly true when the patient is asked to change directions). Caplan[32] notes another interesting diagnostic clue in which the patient actively abducts his or her arms and is asked to suddenly stop the movement: when a lateral medullary infarct is present, the ipsilateral arm is unable to smoothly terminate movement and continues with a clumsy motion.

Although the vertebral arteries may be damaged with almost any motion of the head and neck,[22] most literature suggests that end-range rotation and extension are the most stressful.[2,34] This belief is supported anecdotally with a plethora of case histories in which VAD has been reported to occur after archery,[35] star gazing,[36] extension of the neck during radiography and a bleeding nose,[37] bike riding,[39] golf,[47] the act of resuscitation,[40] drinking in a bar[41] (also known as the bottoms-up syndrome), visits to a beauty parlor[42] (also known as beauty parlor syndrome), yoga,[43] sitting in a dentist's chair,[44] and sleeping prone with the head rotated, extended, or both.[45] (This last position is particularly troublesome with infants, because they possess poor collateral circulation and VAD has been implicated as a cause of sudden infant death syndrome.[46])

One of the biggest difficulties in identifying the motions most likely to produce VAD after spinal manipulative therapy is the almost universal failure of nearly all authors reporting on postmanipulative strokes to identify the type of manipulation responsible for producing the dissection. Although most writers reporting on postmanipulative stroke are quick to point out that the dissection resulted from chiropractic manipulation (even when the manipulation was performed by nonchiropractors[77]), few articles ever described the specific vertebra adjusted and the type of manipulative procedure. The exceptions to this are 2 articles by Klougart et al.[7,47] Not only did these authors attempt to identify the probability of VAD after manipulation, but they also went back to the treatment records to identify the segment adjusted (ie, upper vs lower cervical spine) and the line of drive used in the manipulation responsible for producing the stroke. In the 5 postmanipulative VADs reported, upper cervical rotation manipulation was the culprit in each case.[7] Another study that reported the type of manipulation responsible for producing VAD was referenced as a personal communication with Heyes et al by Haynes.[34] These researchers were able to identify the type of manipulation used in 6 out of 7 cases of postmanipulative VADs that presented to the Royal Perth Hospital between 1990 and 1994. Again, in all 6 cases, rotational manipulation was responsible for producing the injury.

The theory that rotation places the greatest stress on the vertebral artery was first suggested by Toole and Tucker[48] in 1960. With cadavers, these researchers forcefully per-

fused the vertebral arteries with water or "outdated blood" while maintaining the heads in different degrees of rotation, lateral flexion, or both. These authors noted that when the cadaveric necks were placed in contralateral lateral flexion, only 5.4% of the 37 arteries examined had significant reductions in flow, whereas 47% of the cadaveric vertebral arteries showed similar reductions when the necks were maintained in approximately 45 degrees of contralateral rotation.

To study the effect of head and neck rotation on vertebral artery blood flow in vivo, Faris et al[52] performed angiography (a potentially lethal procedure) on healthy male prison inmates and noted that 7.6% of the 79 arteries studied became occluded with contralateral rotation. Since then, numerous studies using advanced MRA and Doppler ultrasonography have demonstrated that contralateral rotation and extension may reduce blood flow in the vertebral arteries,[14,24,53-56] particularly when the neck is maintained in end-range rotation.[25] Of particular interest to the chiropractic profession, Arnetoli et al[55] and Weintraub and Khoury[53] used Doppler velocimeter and MRA, respectively, to demonstrate that occlusion of the vertebral artery may occur with contralateral neck rotation and that this finding constituted an independent risk factor for VAD.

Even though advances in diagnostic imaging in vertebral artery blood flow were allowing researchers to demonstrate that cervical rotation is capable of producing objective changes in blood flow, little research was being done to see whether lateral flexion produced similar changes. In 1996, Haynes[34] published an important article in which he insonated the vertebral arteries of 148 patients first with their necks in neutral, then 60 degrees or more of contralateral rotation, and finally with full contralateral lateral flexion. Of the 280 arteries evaluated in contralateral rotation, 14 (5%) had complete cessation of Doppler signal (with most having cessation of signal at the end-range of rotation), whereas none of the 187 vertebral arteries tested in lateral flexion had termination of signal. It is interesting that 6 of the vertebral arteries that were patent during lateral flexion had cessation of signals during contralateral rotation.

Building on these findings, Haynes[34] went on to theorize that there is a direct connection between decreased blood flow and stress on the vertebral artery. He claims that a significant reduction in blood flow during neck movement can be an indicator of the amount of mechanical stress being applied to the arterial wall. Specifically, he states "loss of Doppler signals during rotation may indicate that a large deformation of the arterial wall has occurred and could sound a warning of possible impending major stretch" and that "rotational neck manipulation could strain the arterial wall beyond its breaking point, resulting in intimal disruption, thrombus formation and possible stroke." This may be more likely to occur if there was superimposed arteriopathy such as fibromuscular dysplasia, which can weaken the arterial wall. This last remark is consistent with recent research demonstrating that as many as 20% of patients with cervical artery dissections (ie, carotid and vertebral) have a "clinically apparent but as yet unnamed connective tissue disorder."[20]

Certainly, this case history supports the theory by Haynes[34] and Terrett[2] that lateral flexion places less strain on the vertebral artery then rotation or extension in that this patient presented with a headache caused by a damaged vertebral artery, underwent vigorous manipulation in lateral flexion, and had no change in her symptom pattern whatsoever. Although it is possible that the manipulation did damage the vertebral artery but did not produce a stroke, this is not likely. In a thorough review of 185 cases of VAD after spinal manipulation reported in the English, French, German, Scandinavian, and Asian literature between 1934 and 1995, Terrett[2] found 138 cases in which the time between manipulation and onset of symptoms was reported. In 72% of these patients, symptoms were either immediate or within a few minutes. Another 17% had symptoms within the first 6 hours and 5% within the first 24 hours. Only 6% had symptoms develop after 24 hours.

Shortly after Terrett's[2] literature review was completed, Silber et al[57] reported on another 26 cases of VAD. The average time between the development of the headache (which signaled the beginning of the VAD) and subsequent neurologic sequelae was 14.5 hours. In a more recent review of postmanipulative VADs, Hufnagel et al[24] reported on 10 patients with VAD and carotid artery dissection after cervical manipulation. The authors of this study noted that 50% of these patients had symptoms immediately after manipulation, whereas the other 50% had symptoms within 2 days. In the 5 cases of VAD reported by Klougart et al,[7] symptoms were immediate in 4 patients and within 10 minutes in the fifth patient. One patient in this series was similar to the patient reported in this case history in that she had a long-term history of unilateral headache (over 3 months' duration) for which she received rotational manipulation at C1-C2. This upper cervical rotational manipulation resulted in an immediate lateral medullary syndrome (dizziness, nausea, and tingling in the fingers) that went on to produce infarct primarily affecting the spinothalamic tract, with a tendency for headaches even 1 year later (the average duration of headache after VAD is approximately 72 hours[57]).

In another case history bearing similarities to this case, Mas et al[58] reported on a 35-year-old woman with a 3-week history of unilateral neck pain that prompted her to seek chiropractic care. The patient received manipulation for the headache (the type of manipulation and line of drive were not reported), and the patient died as a result of rupture of the already damaged vertebral artery. Autopsy revealed a dissecting aneurysm in the third segment of the vertebral artery with evidence of a several-week-old dissection (which accounted for the initial neck pain) and a more recent dissection (from the manipulation) that precipitated the stroke by inducing bleeding within the initial dissection.

In the patient reported in this case history, the manipulation was performed 13 days after the development of the headache that signaled the initial injury to the intima of the vertebral artery. There was absolutely no change in the patient's symptom pattern both immediately after the manipulation and for another 10 days after manipulation. At that time she had almost immediate symptoms (intense headache, confusion, and vertigo) after cervical rotation and extension associated with a brief period of ceiling painting.

Another important possibility to consider is that the patient's initial headache may have been a tension-type headache and the incipient trauma responsible for producing the VAD may have occurred during the ceiling painting episode. Although this is possible, it is highly unlikely. As noted by Sturzennegger,[28] the headache associated with VAD starts suddenly, is of a sharp quality and severe intensity, and is "different than any previously experienced headache." In contrast, tension-type headaches are classically described as a "band of pressure" or "vise-like," build slowly over a period of hours, and tend to be more severe late in the day.[18] Swanson[18] states that "a sudden, severe headache that is maximal at onset and persists usually suggests an intracranial hemorrhage". The observation by Sturzenegger[28] that the headache associated with VAD is "unlike any other" is particularly significant as related to this case, because the patient repeatedly used those exact words to describe her headache. Looking back, it was because of this statement that she did not immediately undergo manipulation and was instead referred for neurologic evaluation. It is also significant that from the day of the initial injury, her headache could be reproduced immediately with the postures that stress the vertebral artery, for example sleeping prone with her neck rotated and extended, bike riding, and wearing a cervical collar that lifted her chin, thereby extending her neck. The fact that the vertebral artery is capable of producing head and neck pain was conclusively demonstrated by Nicholls et al.[60] These researchers inflated a balloon that had been inserted into the vertebral and basilar arteries of healthy subjects and noted that the subsequent stretch induced by the inflating balloon referred pain from the forehead and cheek to the occiput, posterior neck, and even upper trapezius muscle.

To my knowledge, this is the only case history reported in the indexed literature in which a patient with a damaged vertebral artery was able to tolerate the mechanical stress of upper cervical manipulation with no change in her symptom pattern but almost immediately had headaches and eventually had a VAD after another mechanical stress: rotation and extension of the head and neck. Rivett[59] recently reported on a similar case in which a 20- year-old man had a 6-week history of bilateral neck pain and frontal headaches after rapid neck movement while playing cricket. There were no symptoms indicative of VAD, and premanipulative testing procedures were negative. The patient underwent manipulation in lateral flexion at C3-4 on 3 separate occasions with no change in his symptom pattern. During the course of treatment, the patient, by chance, volunteered to participate in a study in which ultrasonography was used to evaluate the effect of various cervical spine positions on vertebral and carotid artery blood flow. Despite the patient's complete lack of symptoms in any of the test positions, ultrasonography revealed total occlusion of the left vertebral artery with full contralateral rotation and extension. This patient was fortunate to have received only lateral flexion manipulations during his course of treatment, because upper cervical rotational manipulation may have had disastrous consequences.

The case history reported in this article is interesting for several reasons. First and foremost is that it supports the theory by Haynes[34] and Terrett[2] that lateral flexion manipulation of the cervical spine produces significantly less strain on the vertebral artery than rotation or extension. Second, it emphasizes that the patient with a damaged vertebral artery is not always going to have obvious symptoms. Clinical evaluation before this patient's infarct revealed that not 1 of the 5 Ds And 3 Ns described by Terrett[2] was present. The only sign that the vertebral artery was damaged in this patient was her description of the nature of the headache. Silbert et al[57] studied the characteristics of head and neck pain with vertebral artery dissection and noted that head and posterior neck pain, which was always ipsilateral, was present 69% of the time and was the initial manifestation of VAD in one third of their patient population. The interval between development of the headache and neurologic manifestations was 14.5 hours, and the headaches, which were posterior in 83% of the patients, were described as steady in 56% and pulsating in 44%. The neck pain, which was always posterior, was present in 46% of the patients with VAD.

The final reason this case history was so interesting is that the patient's headache almost immediately resolved, albeit temporarily, exclusively with the application of electrotherapy to the suboccipital region (deep tissue massage, medications, and cold packs were all ineffective). Although this may simply represent an example of Melzacks and Wall's[51] gate theory of pain in which the large-diameter sensory nerves stimulated by the electric currents blocked entry into the spinal cord of the smaller diameter nerves supplying sensory information from the intima, it may also have occurred as a result of a reduction in tension of the tissues anchoring the vertebral artery at various points. Shimizu et al[61] report on a case in which a thickening of the atlanto-occipital membrane was responsible for fixing the vertebral artery in the vascular groove of the atlas, where it wraps behind the superior aspect of the lateral mass. The anchoring of the artery at this point produced a dynamic pinching of the vertebral artery with head rotation that resolved with surgical decompression of the atlanto-occipital membrane. Perhaps the electric currents produced a temporary soften-

ing of this membrane, thereby lessening its anchoring effect on the vertebral artery.

Following this line of thought, although rarely reported in the literature, it seems possible that soft tissue contracture in the intertransversarii, obliquus capitis superior and inferior, and rectus capitis posterior minor muscle may adversely affect tension in the vertebral artery by anchoring the artery in the vascular groove of the atlas. For example, Okawara and Nibblelink[62] demonstrated that the obliquus capitis inferior and intertransversarii muscles may compress the vertebral artery as it passes from the transverse foramen of C1-2. Also, the rectus capitis posterior minor muscle may play an important role, because researchers from the University of Maryland[63] recently discovered a fibrous bridge of tissue connecting this muscle to the spinal dura at the atlanto-occipital junction (which is near the pathway of the vertebral artery before its penetration into the skull). Akar et al[64] report on an unusual case in which dynamic angiography revealed that the vertebral artery could be compressed at the point of dural penetration and that surgical decompression relieved the associated symptoms. It seems possible that proximal stabilization of the vertebral artery caused by muscle tension may have increased tensile strains placed on the vertebral artery at a more distal site. In an interesting study that supports this line of reasoning, Dadstem and Skerhut[65] demonstrated that tension in the deep cervical fascia is capable of inducing rotation-related hypoperfusion of the vertebral artery with resultant transient ischemic attacks. It has also been well documented that tension in the anterior scalene and longus coli muscles is capable of producing dynamic obstruction of the proximal portion of the vertebral artery.

An important question facing the chiropractic profession is, because it is impossible to identify the patient prone to VAD in advance, should manipulation of the cervical spine be abandoned in favor of the more gentle mobilization techniques? DiFabio,[67] after reviewing the literature on VAD published between 1925 and 1997, states that even though the risk of VAD is small, the benefits of cervical manipulation do not outweigh the dangers and suggests that "a quick thrust to the neck should not be done by anyone." He goes on to claim that the risk of damaging the vertebral artery could be avoided with the use of mobilization (non-thrust passive movements) and supports this claim with the observation that physical therapists are responsible for less than 2% of VADs reported between 1925 and 1997.[66] Terrett[2] has a different view in that he feels "it is not the thrust that is the most dangerous component of the manipulation, but the extreme rotation." This is supported with a large number of case reports in which VAD occurred after non-thrust motions such as archery, yoga, ceiling painting, and star gazing. In fact, VAD after full head rotation associated with archery is so common in Japan that VAD is referred to as a "bow hunter's stroke."[68] The fact that physical therapists are associated with only 2% (although Terrett[2] found 4% in his review) has less to do with the inherent safety of mobilization and more to do with the number of patients exposed to chiropractic versus physical therapy neck treatments since 1925.

The statement by DeFabio[66] that mobilization is less likely to produce cervical spine injury (including VAD) than manipulation is unfounded. This fact was clearly demonstrated by Michaeli[69] as he reported on side effects after 228,050 manipulations and mobilizations performed by physical therapists in South Africa. Results of this study clearly demonstrate that mobilization techniques are no safer than manipulation. Even when adjustment was done for differences in the numbers of patients treated with manipulation versus mobilization (N of 48 vs 129), mobilization was more likely to cause post-treatment dizziness, severe headache, nausea, and brachialgia with neurologic deficit. Note that the only case of VAD in this large group was caused by mobilization.

In an attempt to put the risk/benefits ratio associated with cervical manipulation into perspective, Dabbs and Lauretti[6] compared the death rate after cervical manipulation with the death rate associated with use of nonsteroidal anti-inflammatories (NSAIDs) (which is the most common first-line medical treatment for musculoskeletal neck pain[70]). These authors found the estimated risk of death was 1 in 400,000 for cervical manipulation versus 1 in 40,000 with NSAID use. Keep in mind that the death rate associated with NSAIDs may actually be higher, as recent research demonstrates a 1 in 1,200 death rate with 2 months of daily use of NSAIDs.[71] Given these odds, it is unfortunate that so many neurologists caution their patients to avoid cervical manipulation because of the risks only to go on to prescribe anti-inflammatory medications, which are between 10 and 400 times more likely to produce serious complications or death.

Because manipulation has been repeatedly proven to be an effective form of treatment of cervicogenic and tension-type headaches,[72-75] it is suggested that rather than abandoning a proven safe and effective treatment in favor of riskier alternatives (eg, NSAIDs), manipulative techniques should be modified to lessen the risk of injury simply by avoiding end-range rotation and extension. Mobilization techniques should also be modified to avoid these movements, because certain techniques (particularly the ones described by McKenzie,[76] in which the patient is asked to maximally rotate and extend the neck for prolonged periods of time) are extremely stressful on the vertebral artery. Such techniques should be avoided, especially in patients with intense, unilateral upper cervical or occipital headaches that are "unlike any other headache." In these situations Terrett[77] suggests that these patients be treated with a trial of gentle manual techniques (such as soft tissue mobilization and massage) and, if symptoms decrease, it "is safe to proceed with spinal manipulative therapy." Unfortunately, this case history proves that this is not always the case,

because a patient with a damaged vertebral artery may have decreased symptoms after manual therapy that reduces tension in the suboccipital area.

Because premanipulative tests are useless and neurologic signs are rarely present in the early stages, Licht et al[33] suggest that patients with positive premanipulative tests be referred for ultrasonography of vertebral artery blood flow. These researchers performed a study in which 8 patients with positive premanipulative screening tests and normal duplex ultrasonography were treated with chiropractic manipulation of the cervical spine: 6 of the 8 patients became symptom-free, and the other 2 improved. Another 8 patients with similar findings refused treatment, and their symptoms persisted. This is just 1 of several studies demonstrating that ultrasonography may serve as an invaluable diagnostic tool that allows the practitioner to differentiate between the false-positives and false-negatives that so often occur with premanipulative screening tests.[11,13,34,55,56]

While the study by Licht et al[33] demonstrates that ultrasonography can identify patients with false-positive premanipulative tests, the case report of the cricket player described by Rivett et al[59] is particularly significant because it exemplifies the vital role ultrasonography can play in identifying false-negatives, because manipulation in these situations may have disastrous results. In a personal communication, Haynes[25] notes that the ultrasound instruments are easy to use, inexpensive (the typical unit costs approximately $750), and a complete examination of blood flow takes less than 2 minutes to perform. The directional capability of the Doppler units allows the practitioner to evaluate the direction of blood flow and may help identify cases where collateral circulation is compensating for VAD. Also, examinations with these hand-held devices possess excellent validity compared with the far more expensive duplex ultrasound scanners and also have very good inter-rater and intrarater reliability.[56] There is evidence that the Doppler facility of duplex scanners correlates well with MRA findings of VAD and may also play an important role in follow-up evaluation.[79]

Should the practitioner have the misfortune to perform a treatment that results in VAD, the first and foremost rule is to never remanipulate. The literature has too many examples of situations in which the vertebral artery was damaged with the first manipulation (which may have resolved without incident), only for the practitioner to perform a second manipulation (apparently in an attempt to "fix the problem") with disastrous neurologic results.[2] Terrett[77] describes a useful protocol should a patient have postmanipulative symptoms of vertigo, headache, or nausea. Because it is impossible to differentiate VAD from anxiety-induced hyperventilation (carbon dioxide is a cerebral vasodilator, so the hyperventilating patient often has strokelike symptoms), Terrett[77] suggests that the patient with postmanipulative symptoms should be observed and tested for signs of lateral medullary infarct. Should such symptoms develop, the patient should be transported to a local hospital, and the doctor should inform the emergency medical technicians of the possibility of VAD.

When a possible VAD is being evaluated, both magnetic resonance imaging and MRA are useful for identifying the site of lesion and degree of arterial damage, respectively.[38] Repeat follow-up MRA at 3-month intervals is suggested to evaluate the degree of arterial repair.[49] Schievink[20] notes that 90% of VADs recanalize within the first 3 months after dissection. Once the damaged artery has repaired itself, Jacobs et al[49] suggest switching from anticoagulant therapy (eg, heparin) to antiplatelet therapy (eg, aspirin). It must be emphasized that Coumadin and antiplatelet medications provide no protection from mechanical impingement of blood flow,[53] and the patient must be educated as to which head and neck positions should be avoided.

The long-term prognosis for patients with VAD is good,[50] with a 2% mortality rate during the first month and 1% per year for the next 10 years, with a decreased likelihood of repeat dissection if only 1 artery is involved.[20] Stahmer et al[78] note that 88% of patients with VAD achieve complete clinical recovery.

Conclusion

Dissection of the vertebral artery is a potentially life-threatening consequence of cervical manipulation. Because standard premanipulative screening procedures are unable to identify patients at risk of having VAD, it is suggested that all end-range cervical rotational manipulations/mobilizations be abandoned in favor of techniques incorporating lateral flexion. It is also suggested that practitioners consider incorporating diagnostic ultrasonography to identify the patient at risk for VAD. This case history supports the assumption that lateral flexion manipulation places relatively little stress on the vertebral artery.

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