Abrupt Change in Head Position and Cerebral Infarction

DAVID G. SHERMAN, M.D., ROBERT G. HART, M.D., AND J. DONALD EASTON, M.D.

SUMMARY Eight patients are described who developed infarctions in the vertebral-basilar artery distribution following chiropractic neck manipulation or spontaneous head turning. The angiographic and autopsy findings indicate that injury to the intima of the vertebral artery at the atlantoaxial joint forms a nidus for thrombus formation which may propagate or embolize to involve other vessels in the vertebral-basilar system and result in progressive brainstem infarction. The role of anticoagulation in these patients is discussed.

BRAIN OR SPINAL CORD infarction in the vertebral-basilar artery distribution has been noted in a variety of circumstances associated with head turning. Some infarctions have been associated with non-fusion of the odontoid with atlantoaxial subluxation, fracture or dislocation of the cervical spine, or cervical osteoarthritis. Others have occurred, without obvious structural spine abnormalities, following seemingly minor falls, hyperextension of the neck during athletic or work activities, spontaneous head turning or chiropractic manipulation.

The pathophysiology of stroke in these patients has been postulated, primarily on the basis of angiographic findings, to be injury of the vertebral artery in the region of the atlantoaxial joint which is the site of maximal movement with head turning. The intimal injury may be associated with thrombotic occlusion.

The clinical course in some of these patients has been one of delayed, stuttering or progressive brain infarction associated with high morbidity and mortality. Anticoagulation has been used in an attempt to prevent progressive infarction.

This report describes 8 patients who developed brainstem or cerebellar infarction in association with chiropractic neck manipulation or spontaneous head turning.

Patient 1

A 38-year-old woman turned her head while driving her automobile and experienced severe pain in the right occipital area with momentary complete visual loss. The headache persisted and 3 days later she developed vertigo, blurred vision, ataxia, dysarthria and dysphagia and she was hospitalized.

Physical examination showed an alert, dysarthric woman with right occipital headache. The right pupil was miotic and there was rotary and horizontal nystagmus with diplopia on rightward gaze. Right facial sensation was diminished and the right palate did not elevate. There was dysmetria of the right upper extremity, the deep tendon reflexes were brisk but symmetric and the plantar responses were flexor. There was no sensory loss nor was there any weakness of the extremities.

Skull and cervical spine x-rays were normal as was the cerebrospinal fluid (CSF). Cerebral angiograms showed marked narrowing of the right vertebral artery at the upper edge of the C1 vertebra. No dye flowed down the right vertebral artery with a left vertebral artery angiogram.

She developed progressive signs of brainstem dysfunction over the next several hours and was heparinized. Twelve hours later she suddenly became comatose and decerebrate. Periods of apnea necessitated intubation and assisted ventilation. She remained unresponsive. The next day she became hypotensive, apneic and flaccid without brainstem reflexes. She died on the fourth hospital day.

An autopsy showed hemorrhagic infarction of the ventral two-thirds of the pons and rostral medulla in addition to infarction of the cerebellum and the occipital paraventricular area. There was no subarachnoid hemorrhage.
Patient 3

A 44-year-old woman developed an occipital headache for which she consulted a chiropractor who relieved the headache somewhat with cervical manipulation. At 4:00 a.m. the following morning she was awakened with recurrent headache and found she was unable to swallow.

Physical examination showed ptosis of the left lid, reactive pupils of equal size, slight left facial weakness, an absent gag reflex on the left and pharyngeal and palatal weakness on the left. There was loss of pain and vibratory sensation on the right trunk and extremities and there was mild dysmetria on the left with finger-to-nose testing.

The CSF was normal. A left brachial angiogram demonstrated filling of a small vertebral and posterior inferior cerebellar artery but no visualization beyond. A right brachial angiogram showed normal right carotid and vertebral arteries.

Patient 4

A 39-year-old woman was having a chiropractic neck manipulation when she suddenly felt faint and noted blurred vision and oscillopsia. She returned home where she continued to note blurred vision and oscillopsia, difficulty with concentration and memory, and an unsteady gait. She had a right frontal and posterior cervical headache. When examined one week later, she was noted to have a normal neurological exam with the exception of a right superior homonymous quadrantanopsia. A CT scan revealed a lucency in the left occipital region and a left brachial angiogram demonstrated occlusion of the left posterior cerebral artery. The patient was not treated with anticoagulant and no further follow up was recorded.

Patient 5

Two weeks prior to hospitalization, a 35-year-old man fell from a horse without apparent injury and the following day underwent an uneventful dental procedure. The next day he developed a persistent left retro-orbital headache for which he subsequently received several chiropractic neck manipulations. Following cervical manipulation on the day of hospitalization, he suddenly developed nausea, vertigo, diplopia, and weakness and paresthesias on his right side. On admission to the hospital he was awake and alert with a systolic blood pressure of 85 mm Hg and a pulse rate of 84 per min. There was coarse, bilateral, horizontal nystagmus with lateral gaze. There was bilateral paresis of the lateral rectus muscles, more marked on the left. There was a prominent left facial paresis of the peripheral type, diminished hearing on the left, slight deviation of the tongue to the left and minimal clumsiness of the right arm and hand. No reflex, strength or sensation abnormalities were noted. Cerebral angiograms showed irregular narrowing of the left vertebral artery at the level of C4 with pseudoaneurysm formation (fig. 1A). A computed tomography (CT) scan was normal. CSF examination showed one red blood cell.

Figure 1. Patient 5 — Left vertebral angiograms. A. Irregular narrowing of left vertebral artery at the level of C4 with pseudoaneurysm (arrow). B. One month later there is less vertebral artery narrowing and near complete obliteration of the pseudoaneurysm (arrow).
polymorphonuclear cells and one monocyte/mm³, and a protein of 78 milligrams percent. The patient required blood pressure maintenance with a dopamine drip for the first 36 hours and he was given heparin. His neurologic symptoms and signs improved and he was discharged on warfarin therapy and a rigid cervical collar. One month later a repeat brachial angiogram demonstrated less vertebral artery narrowing and a nearly complete obliteration of the pseudoaneurysm (fig. 1B).

Patient 6

A 24-year-old man consulted a chiropractor for a 3 day history of cervical pain. Immediately after a cervical manipulation involving rotation of the head to left, the patient noted lightheadedness, dimming of vision, hoarseness, numbness over the right face and body, and right arm and leg weakness with staggering to the right. He remained home for two days with an unsteady gait and on the day of hospitalization he developed hiccups and the numbness spread to affect the left side of the body.

The right pupil was 3 mm and the left 5 mm in diameter and both reacted to light. There was ptosis of the right lid and absent sweating over the right face and body. His gait was unsteady. There was diminished pain, touch, position, and vibratory sensation over the left body and right face. The deep tendon reflexes were normal and symmetrical and no pathologic reflexes were noted. Lumbar puncture showed normal CSF except for a protein of 76 mg%. Cerebral angiography demonstrated a small right vertebral artery with occlusion at the level of the C₁ vertebra. The patient was given heparin for 14 days during which his neurologic symptoms improved strikingly and he was discharged on aspirin 650 mg t.i.d. One month following discharge he had a persistent right Horner’s syndrome and a mild hemisensory deficit.

Patient 7

A 58-year-old man had a 3 year history of hypertension. Beginning 9 months prior to admission, he had experienced 6 to 10 episodes of heaviness of the left arm lasting from 1 to 12 hours and occurring over a 3 month period. Two days before admission to the hospital he was backing up his car. He turned his head to the right to look out the rear window and experienced bilateral visual loss for 5 seconds, bitemporal headache, nausea, vomiting and diplopia. He remained at home for the next 48 hours but presented himself for admission when his symptoms did not resolve. Physical examination showed a blood pressure of 140/84 mm Hg and a pulse of 84/min. There was inability to adduct the right eye past the midline with retained superior and inferior gaze, convergence, pupillary size and reactivity. There was minimal left arm and leg weakness with a slight increase in muscle tone and mild diminution of vibratory and pain sensation on the left side. His deep tendon reflexes were increased in the left upper and lower extremities but his plantar responses were flexor. Cerebral angiography demonstrated a small left vertebral artery. During the patient's hospitalization some neurologic improvement was noted and he was discharged on aspirin 650 mg twice daily, dipyridamole 50 mg twice daily and chlorthalidone 100 mg daily. Eleven months later he had returned to his work as a custodian but had experienced several episodes of vertigo, diplopia and unsteady gait. He admitted to poor compliance with his medications. He was encouraged to resume his medications as prescribed and he noted no further symptoms in the month that followed.

Patient 8

A 60-year-old woman had neck manipulations for several weeks because of neck stiffness. While starting to drive from the chiropractor’s office she suddenly slumped over the steering wheel. Ambulance personnel found her still slumped over the steering wheel with a carotid pulse at 140/min but no apparent respiration. Assisted ventilation was initiated but the patient became pulseless. Cardiopulmonary resuscitation was begun with a satisfactory response. She was hospitalized in the intensive care unit with atrial fibrillation and hypotension. She was comatose with no spontaneous respiration, dilated and non-reactive pupils, absent oculocephallic and caloric responses, and flaccidity of all 4 limbs. She was maintained on assisted ventilation and dopamine without neurologic improvement and she died on the fourth hospital day.

At autopsy the entire brain was semi-liquid, typical of the in vivo autolysis of brain death. Subarachnoid blood was present over the upper cervical spinal cord and in the basilar cisterns in the posterior fossa (see fig. 2A). No fracture or dislocation was present in the cervical spine on x-ray or at dissection. An area of hemorrhage surrounded the right vertebral artery and vein at the level of C₁ with microscopy showing antemortem clot within the vertebral vein. There was a 100 X 50 micra perforation of the right vertebral artery in this area surrounded by disruption of the media and internal elastic membrane (fig. 2B). Hemorrhage was present in the fibroareolar tissue between the artery and the vein.

Discussion

At least 52 patients have now been described who developed brain or spinal cord infarction in conjunction with an abrupt change in head position. Their ages range from 7-63 years, with a mean of 37 years. Chiropractic or other neck manipulation preceded the ischemic symptoms in 41 of the 52 patients. Others were associated with minor falls or automobile accidents, ceiling painting, yoga or gymnastic exercises, archery practice, or, as in 2 of our cases, spontaneous head turning while driving an automobile. The clinical findings in these patients most commonly suggested infarction of the pons, medulla or cerebellum. Occipital lobe and cervical spinal cord infarction have also occurred. Head or
The clinical course in these patients indicates a tendency for delayed or progressive infarction. Of those patients for whom adequate information is available, 30% (13 of 43) had a delay of a few minutes to several days in the onset of their neurological symptoms and 35% (15 of 43) showed progression during their course. Of the total group, half (22 of 43) had either a delay in onset or progressive brain infarction. These observations suggest that a thrombus occurs at a site of vertebral artery injury, followed by progressive thrombosis or embolization of the thrombus.

Results of cerebral angiography were available in 24 patients, including 7 of ours. Narrowing or occlusion of the vertebral artery at the level of C1, was found in 18 of the angiograms, with an associated pseudoaneurysm in 5. One patient had a vertebral artery occlusion in the area of the posterior inferior cerebellar artery, 3 had filling defects in the basilar artery and 4 had a posterior cerebral artery occlusion.

Reported treatments generally consisted of supportive care and rehabilitation. Anticoagulation therapy was employed in 6 of the 15 patients in whom there was clinical evidence of progression and 1 patient (16.7%) died. There were 4 deaths in the remaining 9 patients (44.4%) with progressing stroke who were not anticoagulated.

Findings at craniotomy or autopsy have shown ischemic or hemorrhagic infarction of the cerebellum, brainstem or spinal cord with thrombosis in the basilar and vertebral arteries. The eighth patient in this series showed arterial wall destruction with perforation and perivascular hemorrhage.

The anatomic relationship between the vertebral artery and the cervical spine is important in understanding the association between neck movement and vertebral artery injury. The vertebral arteries pass superiorly through the transverse foramina of the atlas they turn sharply posteriorly, wind around the superior articular process, turn medially and penetrate the atlanto-occipital ligament, then ascend through the foramen magnum on the anterior surface of the spinal cord and medulla. The atlanto-axial joint is the major site of neck rotation movement, with 50% of the total rotation occurring at this joint before any rotation is noted in the remaining cervical spine. Blood flow through the vertebral arteries is reduced when the head is hyperextended and tilted or rotated. Angiography during head rotation has demonstrated vertebral artery compression at the level of C1 associated with the development of basilar-vertebral ischemic symptoms.

Clearing of the arterial occlusion and symptoms occurred when the head was returned to the neutral position. Angiographic demonstration of occlusion of one vertebral artery when the head is turned to the opposite side has also been shown in asymptomatic volunteers. These vertebral compressions are observed in the absence of cervical spondylosis or other identifiable structural disease of the neck. Others have noted that vertebrobasilar ischemic symptoms may result from vertebral artery compression in the lower cervical spine in association with spondylosis.

A congenitally small or absent vertebral artery may predispose a patient to ischemic symptoms with head turning when the major blood supply to the basilar artery arises from a single vertebral artery subject to intermittent compression with head movements.

In the majority of the patients described in this study the evidence suggests that a lesion of the vertebral artery at the atlanto-axial level is the cause of the patient's ischemic symptoms. We believe that rotation of the head stretches and compresses the vertebral artery at this most mobile cervical joint causing injury to the vessel, sometimes with an intimal tear and pseudoaneurysm formation. Ischemia may then occur as a result of vascular spasm or thrombus formation. Also, the injured vessel wall may become...
the site of delayed thrombus formation with propogation or embolization of the clot to the basilar or posterior cerebral arteries. This proposed pathophysiologic mechanism suggests that anticoagulants should be given early in hope of preventing progressive brainstem infarction. Untreated patients with clinical evidence of progressive infarction may have permanent morbidity or die. Since there is a risk of anticoagulant therapy causing hemorrhage into infarcted brain, only the analysis of additional patients will clarify the definitive role of this therapy.

Acknowledgment

The authors wish to acknowledge with gratitude the assistance of Drs. George Amromin and John Oro in the pathologic evaluation of Patient 8 and Sherry L. Lauf and Joan A. Bowe for preparation of the manuscript.

References

17. Miller RG, Burton R: Stroke following chiropractic manipulation of the spine. JAMA 229: 189-190, 1974
A abrupt change in head position and cerebral infarction.

D G Sherman, R G Hart and J D Easton

Stroke. 1981;12:2-6
doi: 10.1161/01.STR.12.1.2

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/12/1/2