13. Veteran's Administration Cooperative Study Group on Anti-

Cervical Manipulation and Stroke

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SUMMARY Three patients are described who experienced vertebro-basilar distribution infarctions associated with neck manipulation. Two of the manipulations were chiropractic. Twenty-

INFARCTION of the brain or spinal cord has resulted from a variety of conditions producing injury to the vertebral arteries. These infarctions have been associated with non-fusion of the odontoid with atlantoaxial subluxation, 1-4 5-7 cervical osteoarthitis, 6,7 8 fracture or dislocation of the cervical spine, seemingly minor falls without cervical injury, 9 and hyperextension of the neck associated with athletic activities or work. 9,10

Chiropractic manipulations of the neck may also produce brain infarction. 17,18 Miller and Burton 17 emphasized that patients with cervical spondylolisthesis or vertebro-basilar insufficiency should not undergo chiropractic manipulation and that it should be terminated immediately in any patient who develops ischemic symptoms.

Many patients have progression of the ischemic symptoms over hours or days. Angiograms have demonstrated vertebral artery narrowing and the brain pathology has shown vertebro-basilar distribution infarction. For these reasons, immediate anticoagulation seems reasonable, and some authors consider it the treatment of choice. 18

This report describes three patients who developed brainstem and/or cerebellar infarction after neck manipulation. Two of them underwent chiropractic manipulation and one a spontaneous head turn. One patient was heparinized because the stroke was progressing and autopsy showed a large hemorrhagic infarction of the pons, medulla and cerebellum.

Case 1

A 38-year-old woman turned her head to look behind her while driving and experienced severe pain in the right occipital area with momentary total visual loss. The headache persisted and three days later she developed vertigo, blurred vision, ataxia, dysarthria and dysphagia.

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 but no sensory loss or weakness of the extremities. The deep tendon reflexes and the plantar responses were normal.

Skull and cervical spine x-rays were normal as was the cerebrospinal fluid. Cerebral angiograms showed marked narrowing of the right vertebral artery at the upper edge of the Cl vertebra (see fig. 1). No dye refluxed down the right vertebral artery with the left vertebral angiogram.

The patient was heparinized. Twelve hours later she became comatose and decerebrate. Periods of apnea necessitated intubation and assisted ventilation. 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 entire pons and rostral medulla. There was also softening of the caudal medulla, cerebellum, midbrain and occipital lobes. The vertebral column showed no evidence of injury and the basilar and vertebral arteries were patent.

Case 2

A 48 year old woman was undergoing cervical manipulation by a chiropractor when she suddenly developed left suboccipital pain, dizziness, slurred speech and incoordination of the arms and legs and she vomited.

Physical examination showed dysarthric speech and ataxia which was maximal on the right side. The deep tendon reflexes were brisk and symmetric and the plantar responses were flexor. No other abnormalities were noted.

The cerebrospinal fluid was normal. Cervical spine x-rays showed C4-C6 osteoarthritis. Cerebral angiography demonstrated normal vertebral and basilar arteries.

The patient was heparinized for four days. She remained neurologically stable so the heparin was discontinued. Six months later she had persistent dysarthria and incoordination of the limbs.

Case 3

A 44 year old woman had a one year history of intermittent chest and arm pains and a recent history of occipital headache. She consulted a chiropractor who manipulated her neck. Twelve hours later she was awakened with headache and inability to swallow.

Physical examination showed ptosis of the left eyelid, reactive pupils of equal size, left facial weakness, an absent gag reflex on the left and pharyngeal and palatal weakness on the left. There was impaired sensation on the right body and incoordination of the left upper extremity. The cerebrospinal fluid was normal.

A left retrograde brachial angiogram showed extreme narrowing of the vertebral artery between the origin of the posterior inferior cerebellar artery and the basilar artery which filled poorly (figs. 2 and 3). The right vertebral angiogram showed normal vertebral and basilar artery filling (fig. 4).

Four months later the patient had minimal neurological deficits.
Since 1947 twenty-two patients have been reported in the English literature who developed brainstem, cerebellar or spinal cord infarction associated with therapeutic cervical manipulations. There was no sex predominance and they ranged in age from 20 to 63 years. Twelve of them had the immediate onset of their symptoms at the time of the manipulation and experienced no progression. Seven patients had an immediate onset of their symptoms and then worsened beginning seven hours to four days later. Two patients had the onset of a fixed stroke one hour following the manipulation. The last patient had transient symptoms and then one day later had a fixed stroke. One patient recovered completely. Sixteen of the 22 had residual neurological deficits and the remaining five died from their stroke.

Of the seven patients with progressive strokes, two were given anticoagulants and survived. Five were not given anticoagulants and four died.

Twenty of the 22 patients had clinical, angiographic, craniotomy or autopsy findings of brainstem and/or cerebellar infarction. Occipital lobe infarction was noted in one patient and infarction of the thoracic spinal cord was found in another.

Cervical spine x-rays were obtained in 14 of the 22 patients and were normal in nine. One patient had a dislocation of the C2 vertebra anteriodly on C3 and four had cervical osteoarthritis.

Seven of the 22 patients had cerebral angiography. Six of them showed obstruction, narrowing or pseudoaneurysmal formation in the atlantoaxial region. One patient had normal vertebral and basilar arteries with a left posterior cerebral artery occlusion.

Several factors may contribute to vertebral artery compromise in the cervical region. Congenital asymmetry of the vessels occurs frequently. Compression of the vertebral arteries by cervical osteoarthritis may be accentuated by hyperextension of the neck and cause symptoms of vertebro-basilar ischemia. The most common sites of compression from osteophyte formation are the C5-6 and C4-5 regions. Many of the patients reviewed here were young and without osteoarthritis. Also, their vertebral artery lesions were not in the lower cervical spine.

The atlantoaxial joint is the site of major movement with rotation of the head. Toole and Tucker demonstrated in cadavers that vertebral circulation was frequently reduced when the head was hyperextended and tilted to the opposite side. Since the angiographic abnormalities following cervical manipulation have been found in the atlantoaxial region, it has been speculated that trauma to the vertebral artery at this site may compress or stretch the vessel with resultant spasm or tearing of the intima and thrombus formation.

Cervical manipulation, prolonged hyperextension and rotation of the neck or, as in our first case, spontaneous head turning may result in infarction in the distribution of the
vertebral artery. The apparent pathophysiologic basis of the infarction and the tendency for the stroke to progress favor the use of heparin in these patients. Hemorrhagic infarction is a risk. Further experience may clarify the role of antiocoagulation in this situation.

References

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Recurrent Stenosis at Site of Carotid Endarterectomy

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SUMMARY Five patients studied by the authors and 28 mentioned in the literature indicate that recurrent stenosis occurs in no less than 0.6% of patients after carotid endarterectomy. The pathology of the recurrent stenosis was stated in only 10 cases indicating atheroarteriosclerotic disease in various stages of development in 7 and a fibrous intimal hyperplasia in 3. Correlation between risk factors for the development of atherosclerosis and the pathology of the recurrent disease was poor. Six patients developed recurrent disease despite postoperative prophylactic oral anticoagulation. Surgical technique appeared to have contributed to re-stenosis in 8 patients (1) by failure to remove the distal tongue of plaque or (2) narrowing of the lumen by the arteriotomy suture or (3) damage by a vascular clamp. In 18 symptomatic patients, 44% had symptoms by 3 years, 67% by 5 years, and 83% by 7 years after operation. The 8 patients with possible errors in surgical technique did not develop symptoms earlier than the other patients. Seventeen symptomatic patients had surgical correction of the re-stenosis (endarterectomy 9, vein patch 6, arterial homograft 1, not detailed 1). The incidence of recurrent stenosis after carotid endarterectomy is low and usually the operation provides a patent artery for life.

A PREVIOUS REPORT by the present authors detailed the histological fate of the human carotid endarterectomy site revealed by specimens obtained at postmortem examination from 1 hour to 11 years after endarterectomy. Three of those patients had recurrent stenosis at the original endarterectomy site and the 2 symptomatic patients had repeat endarterectomy. The observation of 2 additional cases of recurrent stenosis and the paucity of references in the literature to this phenomenon led to this assessment of the incidence and contributing factors.

To remove the distal tongue of plaque or (2) narrowing of the lumen by the arteriotomy suture or (3) damage by a vascular clamp. In 18 symptomatic patients, 44% had symptoms by 3 years, 67% by 5 years, and 83% by 7 years after operation. The 8 patients with possible errors in surgical technique did not develop symptoms earlier than the other patients. Seventeen symptomatic patients had surgical correction of the re-stenosis (endarterectomy 9, vein patch 6, arterial homograft 1, not detailed 1). The incidence of recurrent stenosis after carotid endarterectomy is low and usually the operation provides a patent artery for life.

Methods

Two patients with asymptomatic recurrent stenosis died 5 years and 11 years after operation. Two patients with symptomatic recurrent stenoses 16 months and 11 years after first endarterectomy died 51 days and 32 days respectively after repeat endarterectomy. The carotid arteries were removed at postmortem examination, fixed in formalin, decalcified if necessary, and the endarterectomy site sectioned transversely at several levels or serially. Tissue sections were stained for light microscopy with hematoxylin and eosin, Gomori connective tissue stain and Weigert's or Verhoeff's elastic tissue stains.

One patient with a symptomatic recurrent stenosis 6 years after first operation had it removed uneventfully by repeat endarterectomy.

Surgical specimens from all patients were examined to determine plaque morphology and thickness of intima and media which had been removed.
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