Vertebrobasilar Ischemia After Neck Motion

Giovanni B. Frisoni, MD, and Gian Paolo Anzola, MD

Background. Vertebrobasilar ischemic strokes may occur after chiropractic manipulation of the cervical spine or, less often, after spontaneous and abrupt head movement.

Summary of Review. We describe three such cases of vertebrobasilar ischemic strokes and review 36 other reported cases.

Conclusions. We give evidence that 1) the population at risk cannot be identified a priori in the vast majority of cases; 2) symptoms may develop after many uneventful manipulations; 3) clinical syndromes consist of occipital lobe (5%), cerebellar (8%), locked-in (8%), Wallenberg's (28%), other brain stem (49%), and unclassifiable (2%); 4) mortality or very severe long-term impairment occurs in 28% of cases; 5) the development of transient neurological symptoms during previous manipulations, the presence of known or suspected ligament laxity, and, if known, the presence of a vertebral artery terminating in posterior inferior cerebellar artery should always contraindicate any chiropractic neck maneuver; and 6) the pathogenetic mechanism involves vertebral artery dissection at the atlantoaxial joint with intimal tear, intramural bleeding, or pseudoaneurysm that can lead to thrombosis or embolism. (Stroke 1991;22:1452-1460)

Cerebral and brain stem ischemia following rotational head movements is well known in the medical literature. It is mainly related to chiropractic manipulation, but may also occur after a simple fall, head hyperextension while engaged in archery or ceiling painting, or spontaneous head turning. Until now, 72 total cases of stroke following an abrupt change in head position have been reported; of these, 60 occurred after chiropractic manipulation. Vertebrobasilar distribution was involved in the great majority of the cases; only two patients with a carotid territory stroke and signs of carotid dissection after rotational insult to the neck have been described, although a trivial trauma often precedes spontaneous carotid dissection.

Controversy exists between medical and chiropractic literature regarding the possibility of identifying a population at risk. Chiropractors claim that stroke after neck motion occurs in patients with predisposing lesions, whereas medical physicians deny this. However, a detailed and systematic review of the subject is lacking; even the most recent paper on the subject focuses solely on Wallenberg's syndrome, thus limiting the general value of its conclusions.

The pathogenesis of ischemia is unclear. Some believe the cause is a decrease of blood flow in the vertebrobasilar distribution associated with head turning; others blame arterial spasm induced by stretching of the vertebral arteries in the transverse foramina of the vertebrae or arterial dissection, possibly associated with an arterial spasm.

We present here three chiropractic cases and review 36 cases reported in the literature to analyze the natural history of the disease, its pathogenesis, and the presence of risk factors.

Case Reports

Patient 1

A previously healthy 42-year-old man whose only risk factor for stroke was mild hypertension underwent a first chiropractic manipulation on January 27, 1989, for torticollis of some days' duration. Immediately after the manipulation, he had a sharp pain in the right side of his neck, followed by a brief syncope, paresthesias on the right side of his face, agitation, and inability to stand. Symptoms did not progress in the following hours. On admission, he was alert, but unable to stand, with torsional nystagmus on left lateral gaze, a right Horner's syndrome, decreased sensation to pain and temperature on the right side of his face, right facial paresis, marked dysmetria of the right limbs, paresis of the right musculature of the pharynx, deviation of the tongue to the right, and marked dysarthria. Glycerol therapy was instituted. Routine blood examination was normal.

A computed tomography (CT) scan of the brain performed the next day showed a small hypodense lesion in the right cerebellar hemisphere. A right
brachial angiography on February 24, 1989, showed a small right vertebral artery terminating into a normal posterior inferior cerebellar artery (PICA). A left brachial angiography on March 6, 1989, showed bilaterally doubled superior cerebellar arteries with mild atherosclerotic signs; the right posterior cerebral artery was not filled. Magnetic resonance imaging (MRI) on March 17, 1989, showed lesions in the lateral aspect of the right medulla (Figure 1) and in the right cerebellar hemisphere, both within the PICA distribution.

The patient was discharged with good functional recovery and residual mild facial paresis and mild decreased pain and temperature sensation in the right face and left limbs.

**Patient 2**

A 39-year-old woman underwent a first chiropractic manipulation on February 18, 1987, for a moderate neck ache of some days' duration. During the manipulation, she experienced a sharp pain in the right side of her neck with severe vertigo and vomiting. Symptoms did not progress in the following hours. She was a moderate smoker and was hypertensive. On admission, the patient was unable to stand; she also had severe vertigo and vomited repeatedly, had a torsional nystagmus in all positions of gaze, a right-sided Horner's syndrome, decreased sensation to pain and temperature on the right side of the face and left limbs, marked dysmetria of the right limbs, and paresis of the right musculature of the pharynx. Routine blood examination was normal.

A brain CT scan performed on February 19, 1987, showed a small hypodense cortical and subcortical cerebellar lesion in the right hemisphere within the PICA distribution. A right brachial angiography on that same day showed a small right vertebral artery with multiple narrowings at C1 suggesting dissection; the extreme branches of the right PICA were not filled (Figure 2). A repeat right brachial angiography on March 24, 1987, showed normal vertebral artery canalization and PICA perfusion (Figure 3).

The patient was discharged with good functional recovery and residual mild tendency to fall to the right when walking with closed eyes, miosis of the right eye, mild decreased pain and temperature sensation in the right side of the face and left limbs, and mild incoordination of the left lower limb.

Magnetic resonance imaging performed on May 23, 1989, showed a lesion in the lateral aspect of the right medulla and small lesions in the right cerebellar hemisphere (Figure 4).

**Patient 3**

A 49-year-old woman underwent a third chiropractic manipulation on November 20, 1987. Less than 1 hour after the manipulation, she experienced vertigo, inability to stand, nausea, and repeated vomiting. Symptoms did not progress in the following hours.

On admission, the patient was alert and had frequent episodes of nausea with vomiting; she could stand, but gait was unsteady and staggering. She also had a horizontal nystagmus in left lateral gaze and mild dysmetria and incoordination of the right upper limb. A brain CT scan on November 24, 1987, showed a cerebellar hemorrhage involving the lower part of the upper vermis, mildly compressing the fourth ventricle. A cerebral angiography on December 8, 1987, showed an arteriovenous malformation, located posteriorly in the right cerebellar hemisphere and next to the midsagittal plane; the malformation was supplied by collaterals of the right PICA and
superior cerebellar artery (Figure 5). A plain film of the cervical spine showed mild intervertebral osteoarthritis.

The patient was discharged on December 11, 1987, with no residual neurological deficit. A few days later, she underwent successful surgery for ablation of the arteriovenous malformation.

Discussion

Table 1 summarizes details of reported cases.5–7,12–25 Patients in our series had a mean age of 39.3 ± 8.7 years; sexes were equally represented (51% females, 49% males). Symptoms were often (47%) accompanied by head or neck pain and homolateral to the side of the lesion.

The number of manipulations is known in 32 cases of our series; in 17 (53%) of these, symptoms developed during or after the first manipulation, while the remaining 15 patients were manipulated twice or more. Two patients had experienced neurological symptoms during a previous manipulation. A delay of 1 or more hours in the development of symptoms or their progression was present in 22 of the 39 cases (56%).

An explanation as to why symptoms may appear after repeated manipulations can be found in a recent study,26 suggesting that repeated cervical manipulations may cause small asymptomatic lesions in the arterial wall that can predispose to a final lesion. The patient described by Dunne et al12 underwent a number of uncomplicated manipulations 3 years before the fatal one. Pathological examination showed multiple recent dissecting aneurysms within both vertebral arteries and a small, old dissecting aneurysm in the left vertebral artery.

Clinical syndromes in our series were 5% occipital lobe; 8% cerebellar; 8% locked-in; 28% Wallenberg's; 49% other brain stem, sometimes with cerebellar but seldom bilateral signs; and 2% unclassifiable syndromes. In vivo morphological evidence of the lesion with brain MRI is available in seven cases; five were single lateral medullary lesions (two with small cerebellar lesions in the PICA distribution territory), one was a bilateral pontine lesion, and one was a cerebellar hemorrhage. It should be noted that there was a disproportionately high frequency of Wallenberg's syndrome diagnoses among patients who underwent MRI for medullary symptoms because all five patients with medullary symptoms and
MRI had a diagnosis of Wallenberg’s syndrome. The frequency of this syndrome is probably higher than old case reports would suggest.

Eight patients (20%) in our series died as a consequence of the cerebral (usually bilateral or pontine) lesion. Three patients had a locked-in syn-
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<thead>
<tr>
<th>Authors and reference</th>
<th>Sex</th>
<th>Age</th>
<th>Cause</th>
<th>Manipulations (n)</th>
<th>Delay</th>
<th>Pain</th>
<th>Acute onset</th>
<th>Progression</th>
<th>Clinical and/or pathological diagnosis</th>
<th>Radiology and vascular pathology</th>
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<tr>
<td>Okawara and Nibbelink*</td>
<td>M</td>
<td>43</td>
<td>CP</td>
<td></td>
<td>About 12-18 hours</td>
<td>Y</td>
<td>Y</td>
<td>?</td>
<td>R brainstem and cerebellar stroke</td>
<td>AGX: R VA obstruction at C1-C2; L VA marked stenosis at foramen magnum</td>
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<tr>
<td>Sherman et al*</td>
<td>F</td>
<td>38</td>
<td>SHT</td>
<td></td>
<td>Immediately after</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>R brainstem and cerebellar stroke*</td>
<td>AGX: R VA narrowing at C1</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>48</td>
<td>CM</td>
<td></td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Cerebellar stroke</td>
<td>AGX: small R VA and L VA; small L VA terminating in PICA</td>
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<tr>
<td></td>
<td>F</td>
<td>44</td>
<td>CM</td>
<td>1</td>
<td>About 10-20 hours</td>
<td>Y</td>
<td>?</td>
<td>?</td>
<td>L Wallenberg syndrome</td>
<td>AGX: L posterior cerebral artery occlusion</td>
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<tr>
<td></td>
<td>F</td>
<td>39</td>
<td>CM</td>
<td>?</td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>L occipital lobe stroke</td>
<td>AGX: L VA irregular narrowing at C1-C2 with pseudoaneurysm; no narrowing 1 month later</td>
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<tr>
<td></td>
<td>M</td>
<td>35</td>
<td>CM</td>
<td>M</td>
<td>About 5-10 hours</td>
<td>N</td>
<td>Y</td>
<td>?</td>
<td>L brainstem stroke</td>
<td>AGX: small R VA occluded at C1</td>
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<td></td>
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<td>CM</td>
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<td>Y</td>
<td>Y</td>
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</tr>
<tr>
<td></td>
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<td>58</td>
<td>SHT</td>
<td></td>
<td>Immediately after</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
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<td>...</td>
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<tr>
<td></td>
<td>F</td>
<td>60</td>
<td>CM</td>
<td>M</td>
<td>About 10-15 minutes</td>
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<td>?</td>
<td>Cerebral coma*</td>
<td>VP: R VA perforation at C1</td>
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<tr>
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<td>M</td>
<td>35</td>
<td>SSF</td>
<td></td>
<td>Immediately after</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Brainstem stroke</td>
<td>AGX: R VA dissection at C1-C2</td>
</tr>
<tr>
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<td>M</td>
<td>43</td>
<td>CM</td>
<td>M</td>
<td>Immediately after</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Brainstem and cerebellar stroke*</td>
<td>AGX: C2 to foramen magnum bilateral dissections; VP: R VA and L VA multiple dissecting aneurysms; low R VA contusion</td>
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<tr>
<td>Frumkin and Baloh*</td>
<td>F</td>
<td>40</td>
<td>CM</td>
<td>4</td>
<td>Immediately after</td>
<td>Y</td>
<td>Y</td>
<td>?</td>
<td>R Wallenberg syndrome</td>
<td>AGX: R VA dissection at C1-C2; MRI: R lateral medullary lesion</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>33</td>
<td>CM</td>
<td>M</td>
<td>4 days</td>
<td>Y</td>
<td>N</td>
<td>?</td>
<td>R Wallenberg syndrome</td>
<td>AGX: C2 to C7 R VA dissection; MRI: R lateral medullary lesion</td>
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<tr>
<td></td>
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<td>CM</td>
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<td>Y</td>
<td>?</td>
<td>L Wallenberg syndrome</td>
<td>...</td>
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<tr>
<td></td>
<td>M</td>
<td>28</td>
<td>CM</td>
<td>1</td>
<td>Immediately after</td>
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<td>Y</td>
<td>Y</td>
<td>R Wallenberg syndrome</td>
<td>AGX: R VA dissection at C1-C2; MRI: R lateral medullary lesion</td>
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<tr>
<td>Green and Joynt*</td>
<td>F</td>
<td>31</td>
<td>CM</td>
<td>1</td>
<td>Immediately after</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>R brainstem stroke</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>55</td>
<td>CM</td>
<td>2†</td>
<td>1/2 hour</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>L Wallenberg syndrome</td>
<td>...</td>
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<tr>
<td>Smith and Estridge*</td>
<td>F</td>
<td>33</td>
<td>CM</td>
<td>2</td>
<td>During</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Cerebellar stroke*</td>
<td>VP: posterior fossa vasculature unmentioned</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>48</td>
<td>CM</td>
<td>1</td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>R Wallenberg syndrome</td>
<td>...</td>
</tr>
<tr>
<td>Pratt-Thomas and Berger*</td>
<td>M</td>
<td>32</td>
<td>CM</td>
<td>During</td>
<td>?</td>
<td>Y</td>
<td>Y</td>
<td>?</td>
<td>Bilateral brainstem stroke*</td>
<td>VP: basilar artery thrombus; vessels not indigated; VP: R VA and basilar artery thrombus; no gross abnormality of vessels</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>35</td>
<td>CM</td>
<td>M</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Y</td>
<td>Brainstem and cerebellar stroke*</td>
<td>...</td>
</tr>
<tr>
<td>Ford and Clark*</td>
<td>M</td>
<td>37</td>
<td>CM</td>
<td>1</td>
<td>At once</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>L Wallenberg syndrome*</td>
<td>VP: cerebellar hemorrhage 1 cm in diameter; no gross abnormality of vessels</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>41</td>
<td>CM</td>
<td>1</td>
<td>Almost at once</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>R brainstem stroke</td>
<td>...</td>
</tr>
<tr>
<td>Davidson et al*</td>
<td>F</td>
<td>42</td>
<td>CM</td>
<td>4</td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>?</td>
<td>R brainstem stroke</td>
<td>AGX: R VA pseudoaneurysm at C2 and stenosis below</td>
</tr>
<tr>
<td>Horn*</td>
<td>M</td>
<td>34</td>
<td>CM</td>
<td>1</td>
<td>During</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Locked-in syndrome</td>
<td>AGX: basilar artery occlusion; small R VA and L VA</td>
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TABLE 1. Continued

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<tr>
<th>Authors and reference</th>
<th>Sex</th>
<th>Age</th>
<th>Cause</th>
<th>Manipulations (n)</th>
<th>Delay</th>
<th>Pain</th>
<th>Progression</th>
<th>Clinical and/or pathological diagnosis</th>
<th>Radiology and vascular pathology</th>
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<tr>
<td>Lyness and Wagman20</td>
<td>F</td>
<td>20</td>
<td>CM</td>
<td>1</td>
<td>A few moments</td>
<td>N</td>
<td>Y</td>
<td>Locked-in syndrome</td>
<td>AGX: basilar artery narrowing; R VA aneurysm at C1; R VA stenosis at dural entrance</td>
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<tr>
<td>Mehalic and Farhat21</td>
<td>M</td>
<td>40</td>
<td>CM</td>
<td>1</td>
<td>About 10–15 minutes</td>
<td>N</td>
<td>N</td>
<td>R brainstem and cerebellar stroke</td>
<td>AGX: hypoplastic R VA 2 mm in diameter with C1-C2 narrowing; L VA atherosclerotic narrowing near the basilar artery</td>
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<tr>
<td></td>
<td>M</td>
<td>30</td>
<td>CM</td>
<td>1</td>
<td>Immediately after</td>
<td>N</td>
<td>Y</td>
<td>Bilateral brainstem stroke</td>
<td>AGX: small L VA terminating in PICA; R VA and L VA segmental narrowing at C6-C7 and C4-C5; R VA narrowing at C1-C2</td>
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<tr>
<td>Miller and Burton22</td>
<td>F</td>
<td>52</td>
<td>CM</td>
<td>Many†</td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>Bilateral brainstem stroke</td>
<td>Rx: C5-C6 intervertebral narrowing and uncinate process hypertrophy</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>35</td>
<td>CM</td>
<td>1</td>
<td>During</td>
<td>Y</td>
<td>N</td>
<td>L occipital lobe stroke</td>
<td>AGX: L posterior cerebral artery occlusion; normal R VA and L VA</td>
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<tr>
<td>Mueller and Sahs23</td>
<td>F</td>
<td>43</td>
<td>CM</td>
<td>Many</td>
<td>During</td>
<td>N</td>
<td>Y</td>
<td>L brainstem stroke</td>
<td>Rx: C2 forward on C3 dislocation</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>38</td>
<td>CM</td>
<td>Many</td>
<td>A few hours</td>
<td>Y</td>
<td>?</td>
<td>R brainstem and cerebellar stroke</td>
<td>AGX: L VA step-off at C2; distal R VA irregularity. VP: hemorrhagic cerebellar infarction</td>
</tr>
<tr>
<td>Povlsen et al24</td>
<td>F</td>
<td>36</td>
<td>CM</td>
<td>2</td>
<td>During</td>
<td>N</td>
<td>Y</td>
<td>Locked-in syndrome</td>
<td>MRI: Large pointine lesion</td>
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<tr>
<td>Schmitt25</td>
<td>F</td>
<td>35</td>
<td>CM</td>
<td>3</td>
<td>10 minutes</td>
<td>Y</td>
<td>Y</td>
<td>Brainstem stroke*</td>
<td>VP: R VA and basilar artery dissection with intramural hemorrhage</td>
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<tr>
<td></td>
<td>M</td>
<td>51</td>
<td>CM</td>
<td>?</td>
<td>At the end of CM</td>
<td>N</td>
<td>?</td>
<td>L brainstem and cerebellar stroke</td>
<td>AGX: L VA occlusion on 2nd day; L VA complete filling on 8th day. VP: L VA parietal thrombus; L PICA occlusion; L lateral medullary and cerebellar infarction</td>
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<td>Present study</td>
<td>M</td>
<td>42</td>
<td>CM</td>
<td>1</td>
<td>Immediately after</td>
<td>Y</td>
<td>Y</td>
<td>R Wallenberg syndrome</td>
<td>AGX: small R VA terminating in PICA; L posterior cerebral artery provided by carotid artery MRI: R lateral medullary and cerebellar lesions</td>
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<tr>
<td></td>
<td>F</td>
<td>39</td>
<td>CM</td>
<td>1</td>
<td>During</td>
<td>Y</td>
<td>Y</td>
<td>R Wallenberg syndrome</td>
<td>AGX: small R VA with multiple stenoses at foramen magnum (dissection) MRI: R lateral medullary and cerebellar lesions</td>
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<tr>
<td></td>
<td>F</td>
<td>49</td>
<td>CM</td>
<td>3</td>
<td>&lt;1 hour</td>
<td>N</td>
<td>Y</td>
<td>Cerebellar stroke</td>
<td>AGX: cerebellar arteriovenous malformation provided by R PCA and R superior cerebellar artery CT: vermian cerebellar hemorrhage</td>
</tr>
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</table>

Delay, reported delay from last manipulation to symptom development; Pain, head or neck pain occurring with neurological symptoms; Progression, development of progressing neurological symptoms; M, male; F, female; CP, ceiling painting; Y, yes; N, no; ?, unreported or unknown; R, right; L, left; AGX, cerebral angiography; VA, vertebral artery; SHT, spontaneous head turning; CM, chiropractic manipulation; PICA, posterior inferior cerebellar artery; SSF, spontaneous sudden flexion of neck; VP, vascular pathology; MRI, magnetic resonance imaging; Rx, radiology; CT, brain computed tomography.

*Dead.
†Permanent neurological symptoms developed after both first and second manipulations.
‡Patient developed symptoms during manipulations previous to final one.
§Death caused by gastrointestinal ulcer bleeding.
drome that after 2.5–10 months was still severe. Overall, mortality or very severe long-term impairment occurs in 28% of the cases.

Pathogenetic mechanisms to explain the brain ischemia after neck motion consist of hemodynamic interruption of blood flow, arterial spasm, and anatomic damage to the vessels. Head rotation is often followed by an asymptomatic interruption of blood flow in one or both vertebral arteries at the level of the atlantoaxial joint. At this site the vessels, passing through the bony foramina of the atlas, perform an abrupt kinking before entering the suboccipital triangle. When the head is rotated, >50% of the total rotation occurs at the level of the atlantoaxial joint before any rotation can be detected in the lower segments. Thus, C1–C2 is the site where the vessels are most subjected to mechanical stretching and compression during head turning. Sometimes, head turning does seem to evoke transient ischemic symptoms, but only in patients with hypoplasia of a vertebral artery or a vertebral artery terminating in PICA territory. However, these conditions are not relevant in ischemia after neck motion (see below), and a transient rotational obstruction of normal vertebral arteries cannot possibly cause permanent ischemia.

Transient or intractable arterial spasm has been advocated in the past as a possible pathogenetic factor in ischemia after neck motion although no objective proof has ever been given of its real occurrence. Spasm is repeatedly cited even in the most recent literature as a possible pathogenetic mechanism, alone or with other lesions. Occurrence of arterial spasm is not likely for a number of reasons: 1) angiographic evidence of arterial spasm reportedly consists of single or multiple, local and concentric narrowing of the vessel lumen, which widely overlaps with evidence of dissection; 2) spasm alone as a consequence of angiography is seldom if ever responsible for arterial thrombosis and is short-lived; 3) no evidence of fast or drug-induced reversibility of the angiographic stenosis image is available; 4) pathological and angiographic evidence of dissection in ischemia after neck motion is available; and 5) no case with macroscopically and microscopically intact vessels is available. Thus, it seems likely that an angiographic image of stenosis represents a true anatomic lesion, not a functional one, such as dissection.

Damage to the vessels is often reported in verteobasilar ischemia after neck motion, but comprehensive evidence is not available. We detected direct or indirect pathological or radiological signs of dissection
in the vertebrobasilar vessels consisting of string sign, pseudoaneurysm, occlusion, stenosis, intramural hemorrhage, or perforation in 82% (23 of 28) of the collected cases. However, normal vessels in the remaining seven cases, suggesting arterial spasm or hemodynamic interruption of blood flow as pathogenic mechanisms, do not exclude dissection, because of delay from stroke to examination or paucity of data. In the 18 cases in which angiography has shown dissection in the vertebral or basilar arteries and in which the lesion site is reported, some damage at or around C1-C2 is evidenced in 16 cases (89%). These data strongly point to the atlantoaxial joint as the major site where an abrupt head movement can lead to arterial dissection.

Possible risk factors for vertebrobasilar ischemia after neck motion are vertebral artery size asymmetry, vertebral artery terminating in PICA, risk factors for vascular disease, atherosclerosis, osteoarthritis, and vertebral ligament laxity.

Angiographic defects of vertebral artery size are reported in 31% (eight of 23) of the collected cases (six unilateral and two bilateral). However, some degree of asymmetry can be detected in up to 90% of the general population.

The anatomic variant of a vertebral artery terminating in PICA has a reported frequency of <1% in the general population. In these cases, one vertebral artery continues full size into the basilar artery, and the other continues into the homolateral PICA. In our series, two patients (8%) of the 26 with pathological or angiographic examination had this anatomic variant. Obviously, occlusion of the anomalous vertebral artery at any level would cause ischemia and, in both cases, the patients had symptoms consistent with ischemia of the territories supplied by the vessel. The relatively higher frequency of the variant in our series, 8% versus <1% in the general population, suggests that this condition might be, although uncommonly, a predisposing factor.

Most reports do not provide information about risk factors for vascular disease and stroke (hypertension, smoking, diabetes, oral contraceptive use, migraine, transient ischemic attack) except in 11 cases, and the present study. Of these, one or two risk factors were present in six; signs of atherosclerosis were reported in three. The paucity of these data does not allow any conclusion, but young age and previous good health of almost all patients suggest that risk factors for vascular disease are probably not relevant.

Osteoarthritis was said to cause important compression of the vertebral arteries. In 20 patients with a plain cervical spine film, 18 did not show any osteoarthritic lesion, and the remaining two had no osteophytes in proximity of the vertebral transverse processes, the crucial site where one would expect compression to occur. The second patient of Mueller and Sahs had C2 forward on C3 vertebral dislocation, but it is not possible to state whether this lesion caused the vertebrobasilar ischemia or was simply another consequence of the forceful manipulation. In any case, osteoarthritis does not seem to play any pathogenic role in ischemic stroke after neck motion.

Vertebral ligament laxity could be a reasonable predisposing factor for vertebral artery damage caused by excessive stretching and compression during forceful neck manipulations. Pratt-Thomas and Berger reported that their patients had, at postmortem examination, an increased range of movement at the atlantoaxial joint, as judged by the pathologist. Vertebral ligament laxity has been recently proposed as a contributing condition in stretch syncope, a clinical syndrome with complete loss of consciousness during stretching with the neck hyperextended. Although an attractive predisposing factor for ischemia after neck motion, this condition is not supported by data in the previous literature.

In summary, we believe that pathogenic mechanisms for vertebrobasilar ischemia after neck motion can be summarized into the following steps: 1) damage to the tunica intima or tunica media of one or both vertebral arteries, which may be subclinical or progress to the further steps; 2) development of immediate symptoms when a manipulation causes dissection with lumen obliteration; and 3) development of progressive or delayed symptoms when a thrombus or a slowly progressive dissection forms, which may propagate to the basilar artery, occlude the PICA, or embolize into the posterior cerebral artery.

Finally, we emphasize that our three cases were seen in our neurological ward at a rate of one per year, an incidence similar to that reported in the latest paper on the subject. Because the population served by our institution is not over 400,000 people and underreporting is extremely likely, the disease is probably not at all exceptional.

From the above discussion a number of considerations arise: 1) patients at risk cannot be identified a priori; 2) the presence of a vertebral artery terminating in PICA can, although infrequently, be a contributing factor; however, its low frequency and need for detection by angiography make it useless in daily practice; 3) vertebral ligament laxity remains a mere speculation, but suggests avoiding manipulation of patients with known or suspected joint hypermobility (Ehlers-Danlos syndrome, Marfan disease, stretch syncope); 4) development of neurological symptoms during cervical manipulation should always strongly contraindicate any further chiropractic maneuver; and 5) the cervical manipulation risk–benefit ratio should always be carefully evaluated since possible complications are impossible to predict, can be very severe, and are probably more frequent than is usually accepted.

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G B Frisoni and G P Anzola

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