
The following is in response:

To the Editor:

We thank Dr. Marta Moreno and colleagues for their comments on the importance of fluid and nutritional support in stroke. In our study, which involved small numbers, it would not have been valid to undertake small subgroup analysis. Furthermore, a primary aim was to study patients managed in the usual way in our hospital as a prelude to interventional protocols.

More specifically, no patient was enterally fed, the United Kingdom differing from many other countries in this respect. Thus all patients had been fasting at the time of blood sampling. We did record fluid balance, though not fluid type, and this has been related to changes in plasma osmolality and arginine vasopressin. On the basis of this work, we have misgivings about standardized fluid regimens after stroke.

We consider that there is considerable scope for the study of the management of patients in the acute stages of stroke, with fluid and nutritional support being of major importance.

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Reference

Occipitoatlantal Instability: A Hemodynamic Cause of Vertebralbasilar Ischemia After Neck Motion

To the Editor:

We read with interest the recent article by Frisoni and Anzola concerning vertebralbasilar ischemic strokes after neck motion. The authors concluded that the pathogenetic mechanism for head movement and stroke involves vertebral artery dissection at the atlantoaxial joint with intimal tear, intramural bleeding, or pseudoaneurysm that can lead to thrombosis or embolism. Based on 39 chiropractic cases reviewed, other pathogenetic mechanisms such as hemodynamic interruption of blood flow are not considered relevant. On the other hand, among possible risk factors for vertebralbasilar ischemia after neck motion, occipital dysplasia is not mentioned. We documented hemodynamic interruption of blood flow of the vertebral arteries in a patient with occipital dysplasia.

A 37-year-old woman had occipitoatlantal instability due to hypoplasia of the occipital condyles. For the last 2 years she had suffered from repeated episodes of neck pain, vertigo, nausea, vomiting, diplopia, dysarthria, left hemiparesis, and left hemifacial paresthesia; these episodes were regularly precipitated by extension of the neck. Lateral radiographs of the cervical spine showed C5-6 fusion and posterior subluxation of the occipital condyles during neck extension. Anteroposterior computed tomography of the craniovertebral region revealed short dysplastic occipital condyles. Bilateral brachial angiography showed no abnormalities on rotation or flexion of the neck; however, on moderate cervical extension accompanied clinically by a brief sensation of nausea and a convergence spasm, the column of contrast material in both vertebral arteries was interrupted at the C2 level. Immobilization of the neck in a Minnerva cast resulted in complete relief of symptoms.

Findings in our patient indicate that occipitoatlantal instability is another and potentially curable cause for vertebralbasilar ischemia after neck motion. Appropriate neuroradiological examination of craniovertebral junction appears to be called for before performing chiropractic manipulation.

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References

The following is in response: To the Editor:

We appreciate the argument of Drs. Bercliano and Coria on the possibility that vertebralbasilar ischemia may result from hemodynamic interruption of blood flow in vertebral arteries.

On the other hand, we do not feel that such a mechanism can be postulated in the reviewed cases of the literature or in the personal cases presented in the paper, since none of the patients showed evidence of a malformation of the atlantooccipital joint.

Nonetheless, we agree with the caveat of Bercliano and Coria that an x-ray examination of the cervical spine and the skull base should be performed before any cervical manipulation is entertained. This is especially so in cases such as that reported by Bercliano and Coria, in which the patient’s history clearly points to a tight relation between head movements and brain stem dysfunction.

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Correction

The legend for Figure 1 in the letter to the editor from Dr. Jong Sung Kim and Dr. Kwang Deog Jo published in the February issue entitled “Pure Lemniscal Sensory Deficit Caused by Pontine Hemorrhage” (*Stroke* 1992;23:300) should read as follows:

Figure 1. T1-weighted magnetic resonance imaging (repetition time 600 msec, echo time 20 msec) showing a high signal intensity in right pontine tegmentum of 67-year-old woman.

In addition, the third line should read, “...only case with pontine hemorrhage that we know...”

Nephrotic Syndrome, Accelerated Atherosclerosis, and Stroke

To the Editor:

The association of stroke with nephrotic syndrome or the association of other arterial thrombotic complications, particularly coronary artery thrombosis, is an infrequent event. On the other hand, the pathogenetic mechanism of these arterial thromboses has not been conclusively determined. We report on an example of nephrotic syndrome with accelerated atherosclerosis and stroke.

A 30-year-old man was admitted due to a sudden onset of weakness, nausea and vomiting, followed a few hours later by speechlessness and inability to follow commands. Nephrotic syn-
Occipitoatlantal instability: a hemodynamic cause of vertebrobasilar ischemia after neck motion.
J Berciano and F Coria

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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/23/6/921.1.citation