Atlando-Occipital Joint

TO THE EDITOR: In their recent paper (Scott EW, Haid RW Jr, Peace D: Type I fractures of the odontoid process: implications for atlanto-occipital instability. J Neurosurg 72:488-492, March, 1990), Scott, et al., stated that: “The atlando-occipital joint is essentially a shallow ‘ball in socket’ joint composed of two condyloid articulations with reciprocally curved ‘cuplike’ surfaces.” This notion seems to be unusual and different from the currently accepted classification of the joints. The so-called “ball-and-socket” joint refers to the ones in the human body with more or less spherical articular surfaces, such as the shoulder and hip, where the movements can be performed in all directions. The atlanto-occipital joint is a synovial joint, ellipsoid or condyloid in type. This classification has been accepted and described in almost all classical textbooks on anatomy.1-3

The atlando-occipital joints on both sides act as one, and the movements may occur around transverse and anteroposterior axes but not around the vertical axis, hence the joint is considered biaxial and not multiaxial as in the case of a typical “ball-and-socket” joint. The rotation of the head around the vertical axis occurs at the atlanto-axial joints. We would appreciate some clarification from the authors to support their statement.

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References

RESPONSE: We concur with Drs. Tran-Dinh and Torode that the atlando-occipital joint is indeed condyloid in nature having “an ovoid articular surface, or condyle, received into an elliptical cavity in such a manner as to permit flexion, extension, adduction, abduction, and circumduction but no axial rotation.” Our “ball-in-socket” description was not intended to “reclassify” the atlando-occipital joint nor to equate its function or range of motion with that found in the shoulder or hip. It was merely an attempt to help clinical surgeons visualize the morphological relationship of the round or ovoid occipital condyle to the concave “cuplike” C-1 articular surface.

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Anesthesia and Cerebral Coupling

TO THE EDITOR: I enjoyed the article by Robertson, et al. (Robertson CS, Narayan RK, Gokaslan ZL, et al: Cerebral arteriovenous oxygen difference as an estimate of cerebral blood flow in comatose patients. J Neurosurg 70:222-230, February, 1989). The possibility of estimating cerebral blood flow (CBF) by measuring arteriovenous differences in oxygen and lactate in patients while in the intensive care unit is of considerable interest. In the introduction, the authors stated that during anesthesia CBF and cerebral metabolic rate for oxygen (CMRO2) remain coupled and, if CMRO2 is decreased by anesthesia, CBF will also decrease.

This statement is not completely correct. During anesthesia with the majority of intravenous anesthetic agents, CBF and oxidative metabolism are likely coupled; thus, barbiturates decrease CBF and CMRO2 in a dose-dependent fashion, and ketamine increases both.2 Vice versa, all of the commonly used volatile anesthetic agents are known to cause dose-dependent global increases in CBF and to decrease overall CMRO2 in humans and animals.2 This phenomenon has been termed “uncoupling” of flow and metabolism and is an attribute of volatile anesthetic drugs. During anesthesia with volatile anesthetic agents, the CBF-CMRO2 relationship (coupling/uncoupling) appears to be related to depth of anesthesia. For instance, with isoflurane (a more recently introduced volatile agent) cerebral coupling is lost at anesthetic levels higher than one minimum alveolar concentration (MAC).1

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References

RESPONSE: Dr. Tommasino is correct in pointing out that not all anesthetic drugs have the same effect on cerebral blood flow (CBF) and cerebral metabolic rate for oxygen (CMRO2). The intent of our comments about anesthesia was to introduce the concept of the coupling of CMRO2 and CBF, not to discuss the effects of specific anesthetic agents. We would have been more precise to state that during barbiturate anesthesia CBF

RESPONSE: We concur with Drs. Tran-Dinh and Torode that the atlando-occipital joint is indeed condyloid in nature having “an ovoid articular surface, or condyle, received into an elliptical cavity in such a manner as to permit flexion, extension, adduction, abduction, and circumduction but no axial rotation.” Our “ball-in-socket” description was not intended to “reclassify” the atlando-occipital joint nor to equate its function or range of motion with that found in the shoulder or hip. It was merely an attempt to help clinical surgeons visualize the morphological relationship of the round or ovoid occipital condyle to the concave “cuplike” C-1 articular surface.

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and CMRO₂ remain coupled, and if CMRO₂ is decreased by anesthesia CBF will also decrease.

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Syringomyelia Extending to Basal Ganglia

TO THE EDITOR: I was interested in a conclusion reached by Okada, et al., in their report of a rare case (Okada S, Nakagawa Y, Hirakawa K: Syringomyelia extending to the basal ganglia. Case report. J Neurosurg 71:616-617, October, 1989). The authors considered that the cavity extension to the basal ganglia could have occurred at the fetal stage, mainly because of the lack of contralateral pyramidal signs despite the contiguity of the large cavity to the pyramidal tract. One has to recognize, however, that the only known mechanism for the development of a cephalad cavity (or cephalo-myelia), as for syringobulbia, would be through sudden upward surges of a fluid column during a Valsalva maneuver or muscular effort (the "slosh theory"), which could not have occurred in utero. The other theories for development of syringomyelia (hydrodynamic, craniospinal pressure gradient, and interstitial accumulation) cannot account for upward dissection of a syrinx. In addition, the patient had a Chiari I malformation, which is now considered to be a postnatal condition.

These observations make it unlikely that the reported abnormality occurred in utero. The lack of contralateral pyramidal signs could be explained by slow dissection along the white matter fibers.

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References

Acute Subdural Hematoma in Infants

TO THE EDITOR: While reading the recent article by Dr. Howard and his colleagues (Howard MA III, Gross AS, Dacey RG Jr, et al: Acute subdural hematomas: an age-dependent clinical entity. J Neurosurg 71:858-863, December, 1989), I noted again the racial difference in the occurrence of acute subdural hematoma. The authors described differences in the clinical characteristics of acute subdural hematoma in two age groups (those aged 18 to 40 years and those > 65 years); however, they did not include cases of infants. As I have reported previously, there is a distinct clinical entity in infants: namely, infantile acute subdural hematoma. This entity, which is caused exclusively by minor head trauma, is rare not only in the United States and European countries, but also in Hong Kong. For this reason, infantile acute subdural hematoma does not seem to be fully recognized outside Japan. In addition to the authors' two groups of patients with acute subdural hematoma, infantile acute subdural hematoma, which has a peak incidence at the age of 7 to 10 months, should be listed as an age-dependent clinical entity.

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References