Neurosurgical forum

Kernohan Notch

To the Editor: I read the article of Adler, et al. (Adler DE, Milhorat TH: The tentorial notch: anatomical variation, morphometric analysis, and classification in 100 human autopsy cases. J Neurosurg 96:1103–1112, June, 2002) with great interest and would like to make the following brief historical and physiological remarks to put the authors’ admirable work into proper perspective. I limit my comments to their statement that an “anatomical mechanism for the occurrence of a false localizing sign is understood, but its incidental occurrence has been difficult to explain. The dissimilarity in tentorial notch morphology and regional anatomy may help elucidate this phenomenon.”

Abstract

Object. Variations in the structure of the tentorial notch may influence the degree of brainstem distortion in transtentorial herniation, concussion, and acceleration–deceleration injuries. The authors examined the anatomical relationships of the mesencephalon, cerebellum, and oculomotor nerves to the dimensions of the tentorial aperture. On the basis of numerical data collected from this study, the authors have developed the first classification system of the tentorial notch and present new neuroanatomical observations pertaining to the subarachnoid third cranial nerve and the brainstem.

Methods. The mesencephalon was sectioned at the level of the tentorial edge in 100 human autopsy cases (specimens from 23 female and 77 male cadavers with a mean age at time of death of 42.5 years [range 18–80 years]). The following measurements were determined: 1) anterior notch width, the width of the tentorial notch in the axial plane through the posterior aspect of the dorsum sellae; 2) maximum notch width (MNW), the maximum width of the notch in the axial plane; 3) notch length (NL), the length of the tentorial notch from the superoposterior edge of the dorsum sellae; 4) anterior tentorial length, the shortest distance between the apex of the notch and the most anterior part of the confluence of the sinuses; 5) interpedunculooclival (IC) distance, the distance from the interpeduncular fossa to the superoposterior edge of the dorsum sellae; 6) apicotreccal (AT) distance, the distance from the tectum in the median plane to a perpendicular line dropped from the apex of the notch to the cerebellum; 7) cisternal third nerve distance, the distance covered by the cisternal portion of the third cranial nerve; and 8) inter–third nerve angle, the angle between the two third cranial nerves.

The quartile distribution technique was applied to all measurements. Mean values are presented as the means ± standard deviations. Quartile groups defined by NL (mean 57.7 ± 5.6 mm) were labeled long, short, and midrange, and those defined by MNW (mean 29.6 ± 3 mm) were labeled wide, narrow, and midrange. Combining these groups into a matrix formation resulted in the classification of the tentorial notch into the following eight types: 1) narrow (15% of specimens); 2) wide (12% of specimens); 3) short (8% of specimens); 4) long (15% of specimens); 5) typical (24% of specimens); 6) large (9% of specimens); 7) small (10% of specimens); and 8) mixed (7% of specimens). The IC distance (mean 20.4 ± 3.2 mm) was used to characterize brainstem position as prefixed (28% of specimens), postfixed (36% of specimens), or midposition (36% of specimens). The IC distance was correlated with the left and right cisternal third nerve distances (means 26.7 ± 2.9 mm and 26.1 ± 3.2 mm, respectively) and the inter–third nerve angle (mean 57.3 ± 7.3°).

The “false localizing sign” the authors are referring to is the ipsilateral paresis sometimes seen in lesions affecting one hemisphere, commonly (but erroneously; see later) attributed to “pressure cones” resulting in the crushing of the contralateral cerebral peduncle. This real localizing sign, which is diaschitic in nature (see later), was known by an eponym (the Ectors syndrome) before the simplistic explanation of Kernohan and Woltman became popular.1 The syndrome is one of the earliest recognized neurological entities, first described by an Egyptian (neuro) surgeon more than 4000 years ago (Case 8, Edwin Smith Papyrus).10 Neither the Egyptian author (who described it in a patient with closed head injury) nor Kernohan and Woltman1 mentioned the laterality of the paretic/apapractic side of their respective patients. Fortunately, Kernohan and Woltman did provide some photographs in their paper, all of which indicate that the lesion involved the left hemisphere. The credit for grasping the importance of this laterality aspect belongs to two other neurosurgeons, Peyser and Doron,11 who, when they reported a series of cases of ipsilateral paresis in space-occupying lesions affecting the left hemisphere, referred to it as an “amazing fact.” Several other examples from the past and present literature may be cited indicating the absence of any notching of the midbrain in cases of ipsilateral paresis/apraxia.12 The intellectual shock of unexpected encounters such as these have resulted in comical situations depicted in medical and lay literature, wherein the surgeon even convinces all the staff members that they have the films labeled wrong or have hung them in reverse on imaging or when looking at them before the emergency operation.

This brings us to the distinction between neural and behavioral handedness, best exemplified in crossed aphasia in which the laterality of the hand that showed paralysis and that showing the apraxia switched sides.2 The incidence of such an incongruity in the general public is estimated at not less than 15%.2,3,12 The chaos that such large numbers can cause in time-pegged laterality research (for example, those dealing with the interhemispheric transfer time) is readily apparent.

Indeed, the aforementioned scheme substantiates Liepmann’s clinical analysis as depicted in his drawings (Fig. 1) adopted from his 1908 and 1925 articles,13,19 and recently reviewed by Goldenberg.5 Thus, the contribution of the “incisura of the crus” to the symptomatology of such patients remains unspecified (if
We refer the reader to the references for a detailed review of the historical context and current understanding of cerebral dominance and hemispheric asymmetry. In summary, we are aware that the neuroanatomical basis of movement dictated by the cerebral cortices has been defined by functional magnetic resonance imaging and electroencephalography studies. Handedness, unidirectional transfer, and activation of the nondominant hemisphere as well as interhemispheric diaschisis, however, does not completely explain the occurrence of a hemiparesis in a patient with an ipsilateral mass lesion. The component of the uncrossed corticospinal tract (anterior corticospinal tract) and possible anatomical variation in this tract among individuals may be a more likely reason for what has historically been known as a “false localizing sign” or the Kernohan notch phenomenon. We feel that anatomical variation in the morphology of the tentorial incisura may also contribute to this rare and unusual neurological sign as very clearly demonstrated in the pictures in the article by Kernohan and Woltman.

Labeling the Kernohan notch phenomenon as a “real” localization sign may likely contribute to confusion among clinicians. The sign, although presumably explicable on the basis of neuroanatomical variation (cortical, incisural, and lesion location) remains the exception rather than the rule and therefore current nomenclature should remain in place.

References


Response: We thank Dr. Derakhshan for his historical perspective and anatomical insight. In summary, he suggests the following. 1) The Kernohan notch phenomenon represents a real localizing sign based on cerebral dominance. 2) Laterality or dominance determines the sidedness of the paralyzed or apractic side and not “pressure cones” or lesion location. 3) Interhemispheric diaschisis or hemispheric asymmetry (cerebral dominance) is the determining factor regarding ipsilateral weakness/apraxia rather than incisural anatomy.

The concept that the left hemisphere in the majority of individuals is responsible for movement on both sides of the body via the corpus callosum is not being disputed. We are aware that the neuroanatomical basis of movement dictated by the cerebral cortices has been defined by functional magnetic resonance imaging and electroencephalography studies. Handedness, unidirectional transfer, and activation of the nondominant hemisphere as well as interhemispheric diaschisis, however, does not completely explain the occurrence of a hemiparesis in a patient with an ipsilateral mass lesion. The component of the uncrossed corticospinal tract (anterior corticospinal tract) and possible anatomical variation in this tract among individuals may be a more likely reason for what has historically been known as a “false localizing sign” or the Kernohan notch phenomenon. We feel that anatomical variation in the morphology of the tentorial incisura may also contribute to this rare and unusual neurological sign as very clearly demonstrated in the pictures in the article by Kernohan and Woltman.

Labeling the Kernohan notch phenomenon as a “real” localization sign may likely contribute to confusion among clinicians. The sign, although presumably explicable on the basis of neuroanatomical variation (cortical, incisural, and lesion location) remains the exception rather than the rule and therefore current nomenclature should remain in place.

References