the results in the literature for the latter procedure were no better than those reported by Taarnhøj and Gardner and Miklos, who merely “rubbed” the nerve root.

The recent paper by Bederson and Wilson is an important contribution. They found (arterial or venous) nerve root distortion in only 66% of people, which approximates to the figure for patients without trigeminal neuralgia reported by Hardy and Rhoton. Bederson and Wilson’s paper produces some evidence to support the traumatic effect of MVD: “Persistent partial hearing loss occurred in eight patients (3%), seven of whom had undergone MVD alone.” They comment that it was more likely to occur after MVD possibly due to the “increased cerebellar retraction necessary for adequate decompression.”

Fritz and others noted the occurrence of hearing impairment in two (9.5%) of 21 patients undergoing MVD for trigeminal neuralgia; this deficit was due to cochlear nerve damage from retraction. Thus, whatever else MVD for trigeminal neuralgia does or does not do, the cerebellar retraction necessary to perform this operation can cause eighth nerve root damage.

Finally, there is one extremely important epidemiological fact that has been ignored in this debate. Professor Levy (Zimbabwe), Professor Adeloye (Nigeria), and Professor de Villiers (South Africa) have all confirmed that (idiopathic) trigeminal neuralgia is hardly ever seen in Africans and yet hypertension is common. Professor de Villiers (South Africa) and others have noted the occurrence of hearing impairment in two (9.5%) of 21 patients undergoing MVD for trigeminal neuralgia; this deficit was due to cochlear nerve damage from retraction. Thus, whatever else MVD for trigeminal neuralgia does or does not do, the cerebellar retraction necessary to perform this operation can cause eighth nerve root damage.

We would like to draw attention to a methodological problem which has not been emphasized by previous authors. Recently, we have shown that cannulation of the lateral ventricle in patients with no acute brain damage resulted in release of CK-BB into the ventricular CSF in comparable amounts to those found in severe head injury. Moreover, the release of CK-BB was very variable. Especially in patients with less severe head injuries, the CK-BB release caused by cannulation is a major part of the total CK-BB activity. When the variability of this interference is taken into consideration, it is obvious that a possible strong relationship between the severity of injury and outcome, and CK-BB is easily blurred.

We suggest that future studies of biochemical markers in cases of central nervous system trauma are based on CSF samples obtained through very fine canulases or on CSF obtained from the subarachnoid space.

References

Response: Bach and Kruse question the prognostic value of cerebrospinal fluid (CSF) creatine kinase BB (CK-BB) assay after head injury and suggest that the assessment of initial brain damage using ventricular CK-BB could be overestimated by enzyme release related to ventricular cannulation. Indeed, a poor prediction of outcome based on CSF CK-BB determinations in head injured patients has been reported by some recent studies.
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authors. I completely agree with the fact that CSF enzyme release after brain trauma does not always represent an irreversible cell lesion but, at the present time, that ideal condition has not been achieved by any CNS markers. On the other hand, the patients' outcome can be influenced by different factors including neurological complications which are absolutely not related to initial brain damage. Moreover, as already stressed, knowledge of the temporal sequence of enzyme release and the timing of CSF sampling are essential to correctly quantifying the severity of a cell lesion. Thus, the extrapolated CK-BB activity calculated from different values measured at precise delays with regard to injury can be considered the most specific parameter in evaluating initial brain damage.

Samples of CSF withdrawn every 2 hours during the first 24 hours after trauma provide more information about CSF CK-BB decay than four or five samples taken within 60 hours as performed by Rabow, et al. Therefore, when considering diffuse brain lesions, extrapolation from a CSF CK-BB value measured at a given time to the value which should theoretically be measured immediately after injury is probably the best method for an accurate assessment of primary brain damage in outcome prediction.

As for the methodological problem, we addressed that question a few years ago by measuring CSF CK-BB after ventricular cannulation for intracranial pressure monitoring in nontraumatic hydrocephalic patients. In our patients, the mean CSF CK-BB activity was quite low (mean ± standard deviation: 26.4 ± 10.57 IU/liter) as compared with that measured in the same way after diffuse traumatic lesions. Therefore, in severe head injuries associated with great amounts of enzyme released, I do not believe that CK-BB levels in CSF obtained by cautious ventricular cannulation could actually interfere with quantification of traumatic brain damage.

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References