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"Hippocampal Malrotation": No Real Malrotation and Not Rare

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"Hippocampal Malrotation": No Real Malrotation and Not Rare

We read with great interest the article relating to hippocampal malrotation (HIMAL) by Gamss et al.¹ We did, however, find some of the terminology questionable. In particular, we thought that "incomplete hippocampal inversion" (IHI) would be a better descriptive term because the hippocampus is not "malrotated," but rather probably the inversion was never completed. The authors themselves acknowledge this possibility when they write that "in cases of hippocampal malrotation, hippocampal inversion fails to occur." Why then, we wonder, should this condition be called "malrotation"?

Various terms have been used for IHI, and the authors may not have checked for terms other than HIMAL in their literature search. There are earlier articles relating to hippocampal form in seizure-free populations, and this form variant is relatively common in these articles. Bronen and Cheung² described configurations other than oval in 12/58 hippocampi of healthy volunteers. We found IHI (a round or pyramidal hippocampus and a vertical collateral sulcus in each case) in 18%–19% of the subjects in populations of healthy volunteers and patients without epilepsy or obvious developmental brain anomalies.^{3,4} The differences in prevalence may depend on the criteria used in interpretation (ie, the inversion can be incomplete in a part of the hippocampus but complete in the rest). As was the case in the material of Gamss et al,¹ IHI was most often unilateral and left-sided. In our work, we have not found an association between age and the rounded appearance of the hippocampus, but the IHI prevalence seems to be the same as in adulthood from gestational week 25 onwards (D. Bajic, unpublished data, September 2009).

Comparison of the prevalence in 2 studies by different researchers is not an optimal method to investigate the relationships between IHI and seizures. In a recent blind study, we also found a statistically

significant difference between the populations without and with seizures (18% versus 30%, $P < .05$),⁴ but this difference was not as great as that presented by Games et al.¹ In our study, the IHI frequency was very high in some epileptic syndromes (ie, in cryptogenic generalized epilepsy), but there was no statistically significant difference between the patients having temporal lobe epilepsy and the control group when IHI was the only deviating finding in the temporal lobe. There was no correlation between electroencephalography and IHI laterality. Our conclusion was that there was no causality between temporal lobe epilepsy and IHI.

IHI, a common morphologic variant of the hippocampus, is not an etiologic factor in epilepsy but can be a sign of disturbed cerebral development that may affect other parts of the brain, leading to epilepsy.

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