INTRODUCTION

Hippocampal sclerosis (HS) is found in approximately 65% of patients with refractory temporal lobe epilepsy undergoing temporal lobectomy. Prior to the development of magnetic resonance imaging (MRI) there was no reliable method to detect this lesion pre-operatively. The early literature on MRI in epilepsy regarding the detection of this abnormality was confusing and contradictory (Laster et al., 1985; McLachlan et al., 1985; Jabbari et al., 1986; Lesser et al., 1986; Sperling et al., 1986; Schörner et al., 1987; Heinz et al., 1988). Areas of high signal in T2-weighted images were frequently reported. These high signals were not always of diagnostic significance, in some cases being due to artefact or non-specific enlargement of the temporal horn. The early enthusiasm for the value of MRI in non-lesional cases of temporal lobe epilepsy waned in many centres, and it was widely regarded that it was not possible to detect HS by visual inspection of MRI scans (Sperling et al., 1987).

Recently, careful volumetric studies of the hippocampus showed unilateral hippocampal atrophy in a considerable proportion of cases (Ashtari et al., 1991; Cook et al., 1992; Jack et al., 1992) and in some studies, this has been shown to be correlated with the pathological finding of HS (Bronen et al., 1991; Cascino et al., 1991). Although the value of volumetric assessment has been widely endorsed, the possibility of accurate diagnosis of HS by visual inspection of MRI has received insufficient attention. This can be performed with great reliability, providing the scans are performed in the appropriate fashion, and are interpreted by an informed reader.
DIAGNOSIS OF HIPPOCAMPAL SCLEROSIS BY VISUAL INSPECTION

Using a 0.5 T magnet in Montreal we identified hippocampal sclerosis in spin echo images using visual criteria of a reduction in cross-sectional area of the hippocampus (analogous to current volumetric techniques), in association with a high signal seen on T2-weighted images (Berkovic et al., 1986a,b,c). In view of the shape and orientation of the hippocampus, we realised that the conventional orbitomeatal plane was inappropriate, and that examination of the hippocampus was improved by using an axial plane parallel to the base of the temporal lobe and coronal planes at 90° to this (Berkovic et al., 1986b). Moreover, for correct diagnosis the increased T2 signal must be localised to the hippocampus and proper interpretation is only possible by comparing the high T2 signal area to an anatomically defined abnormality on the T1-weighted scans (Berkovic et al., 1986c). The early Montreal experience showed that the side of the MRI abnormality correlated with the side of the electrical focus, and with the presence of pathological abnormality in the surgical specimens (Kuzniecky et al., 1987). However, adequate pathological material from the hippocampus itself was not obtained, so the view that one could recognise HS was not widely accepted, and indeed full publication of our observations was delayed (Berkovic et al., 1991).

Subsequently, in studies from Melbourne, we compared pre-operative “blind” MRI diagnoses, using a 0.3 T magnet, in 41 surgical cases with adequate hippocampal specimens of which 27 had HS and 14 were normal. The sensitivity for detection of HS was 93% and the specificity was 86% (Jackson et al., 1990). We have since studied a further 73 cases with hippocampal specimens and no major foreign tissue lesion, including 59 cases of pathologically proven hippocampal sclerosis. All 59 cases were recognised on pre-operative MRI by blinded observers. False-positive diagnosis of HS was made on five cases, all of which on review showed quite subtle abnormalities in hippocampal size or shape with little or no change in T2 signal. In four cases the lateralisation was correct. Of these, two showed microdysgenesis of the mesial structures on pathology. There was no definite abnormality in the other two, but in one of these cases the whole hemisphere was atrophic and the hippocampal volume loss presumably reflected this. In the fifth case, EEG and other data pointed to the temporal lobe contralateral to the subtle hippocampal abnormality. Temporal lobectomy was performed on the side of the EEG abnormality and the pathology was normal. The overall sensitivity and specificity for visual diagnosis of HS in the Melbourne series of 114 cases were 98% and 75% respectively. It should be emphasised, however, that of the seven false positives in the whole series, there was strong evidence that the seizures originated from the designated hippocampus in six, even though there was no pathological evidence of hippocampal sclerosis.

The high sensitivity and specificity of visual diagnosis of HS in the Melbourne series were based on two radiological signs using spin echo images; hippocampal atrophy on first echo images and increased hippocampal signal on second echo (more T2-weighted) images. More recently, Jackson et al. (1992), using inversion recovery sequences, described two more features of HS. The abnormal hippocampus has reduced signal intensity on T1-weighted images, and the normally visible internal layering of the hippocampus is absent, which equates with the pathological abnormality of destruction of the pyramidal cell layer. These four hippocampal MRI signs of HS (Table 1) each have a sensitivity of approximately 80% in properly performed and interpreted studies (Jackson et al., 1992).

In addition to these specific signs of HS, other abnormalities may be present in extra-hippocampal regions of the temporal lobe. In some cases the whole temporal lobe may be atrophied; the cortex may show thinning and loss of T1 signal and there may be dilatation of the temporal horn. Recently, abnormalities of the grey–white matter junction have been described in cases of HS (see Meiners et al., Chapter 14). The sensitivity and specificity of these extra-hippocampal features for diagnosis of HS are not known.