

Magnetic resonance imaging in partial epilepsy: additional abnormalities shown with the fluid attenuated inversion recovery (FLAIR) pulse sequence

P S Bergin, D R Fish, S D Shorvon, A Oatridge, N M deSouza, G M Bydder

Abstract

Thirty six patients with a history of partial epilepsy had MRI of the brain performed with conventional T1 and T2 weighted pulse sequences as well as the fluid attenuated inversion recovery (FLAIR) sequence.

Abnormalities were found in 20 cases (56%), in whom there were 25 lesions or groups of lesions. Twenty four of these lesions were more conspicuous with the FLAIR sequence than with any of the conventional sequences. In 11 of these 20 cases, lesions thought to be of aetiological importance were only seen with the FLAIR sequence. In eight this was a solitary lesion. In the other three, an additional and apparently significant lesion (or lesions) was only seen with the FLAIR sequence when another lesion had been identified with both conventional and FLAIR sequences. The 11 additional lesions or groups of lesions were seen in the hippocampus, amygdala, cortex, or subcortical and periventricular regions. No lesion was found with any pulse sequence in 16 (44%) of the original group of 36 patients.

In the eight cases where a lesion was seen only with the FLAIR sequence, localisation was concordant with the electroclinical features. Two of the eight patients with solitary lesions seen only on the FLAIR sequence underwent surgery, after which there was pathological confirmation of the abnormality identified with imaging.

In one patient with a congenital cavernoma, the primary lesion was best seen with a contrast enhanced T1 weighted spin echo sequence.

In this selected series, the FLAIR sequence increased the yield of MRI examinations of the brain by 30%.

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Modern imaging techniques have had a profound impact on the management of patients with partial seizures. An underlying structural abnormality can be identified in most cases. The most common pathological substrates are

cortical dysgenesis, hippocampal sclerosis, vascular lesion, and tumours.¹

Magnetic resonance imaging employing T1 and T2 weighted multislice and volume techniques is generally used for imaging patients with partial epilepsy but there is a particular problem with T2 weighted spin echo images. With this type of sequence, the signal from the CSF is greater than that from the brain and the high signal expected from the common pathological lesions may be obscured by averaging effects between brain and CSF. To overcome this problem we have used the fluid attenuated inversion recovery (FLAIR) pulse sequence in which the CSF is nulled by the use of a preceding inversion pulse. A long echo time (TE) can then be used to provide very heavy T2 weighting.²

To assess the value of this approach in partial epilepsy, 36 patients in whom conventional CT or MR studies on one or more occasions had previously been negative were studied with conventional T1 and T2 weighted spin echo sequences and the FLAIR sequence.

Patients and methods

With the permission of the medical research ethics committees of the Royal Postgraduate Medical School and the National Hospital for Neurology and Neurosurgery, 36 patients (21 men, 15 women) (mean age 30.7 (range 18 to 57) years) with a history of partial seizures were studied. All patients were seen at the National Hospital for Neurology and Neurosurgery between August 1992 and July 1993.

Nearly all patients experienced frequent complex partial seizures. A clinical diagnosis of temporal lobe epilepsy had been made in 22 patients, although lateralisation was uncertain in five of these. Five patients had experienced prolonged febrile convulsions during infancy and 23 also had secondary generalised seizures. One patient had undergone a right temporal lobectomy two years previously without relief of his seizures. All patients had multiple EEGs with surface electrodes and the 10:20 system. Seizures were recorded in 15 patients (surface and sphenoidal electrodes).

All patients were examined with MRI using a Picker HPQ 1.0 Tesla system in both the coronal and transverse planes with conventional multislice T1 weighted (SE 720/20), T2 weighted (SE 2500/20 and 2500/80),

Institute of Neurology,
National Hospital for
Neurology and
Neurosurgery, Queen
Square, London
WC1N 3BG, UK
P S Bergin
D R Fish
S D Shorvon

The Robert Steiner
Magnetic Resonance
Unit, Royal
Postgraduate Medical
School, Hammersmith
Hospital, London
W12 0HS, UK
A Oatridge
N M deSouza
G M Bydder

Correspondence to:
Dr D R Fish.

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Patients in whom the FLAIR sequence provided additional information

Patient No	Age/sex	Electroclinical features	FLAIR study
1 (fig 1)	45/M	Aura: gooseflesh. Postictal dysphasia; impaired verbal memory. Interictal EEG: left temporal delta; bitemporal spikes. Left temporal lobectomy: hippocampal sclerosis confirmed > 99% seizure reduction at one year after operation	Increased signal intensity in left hippocampus only seen with FLAIR sequence. Dilated left temporal horn seen with conventional and FLAIR imaging. Multiple deep white matter punctate lesions seen with both conventional and FLAIR scans. High signal in left hippocampus on FLAIR only
2	26/F	Complex partial seizures with manual automatisms (no aura). Interictal EEG: left temporal spikes	
3	30/F	Aura: urinary urgency and nausea. Seizure: oral and manual automatisms. Interictal EEG: non-specific, bitemporal abnormalities	Left hippocampus and amygdala high signal only seen with FLAIR
4 (fig 2)	36/F	Postictal dysphasia. Interictal EEG: left temporal spikes and slow waves. Lesionectomy confirmed focal developmental malformation at site of FLAIR abnormality	Left amygdala lesion seen only with FLAIR
5	40/F	Complex partial seizures with manual automatisms. Interictal EEG: bitemporal spikes	Increased signal in right amygdala apparent on FLAIR only
6	23/F	Complex partial seizures with oral-facial and manual automatisms. Interictal EEG: near continuous left temporal spikes and slow activity	Left amygdala lesion only seen with FLAIR
7	23/F	Aura: deja vu. Seizure: oral-facial automatisms. Able to converse during most attacks. Interictal EEG: right temporal sharp waves. Ictal EEG: discharge maximal right temporal region	Lesion in right amygdala only seen with FLAIR
8 (fig 3)	30/M	Aura: amaurosis, distorted vision. Interictal EEG: normal. Ictal EEG: left hemisphere discharge	Lesion in left occipital lobe only apparent on FLAIR
9 (fig 4)	18/F	Complex partial seizures with manual automatisms, dystonia right hand. Postictal dysphasia. Interictal EEG: left posterior temporal spikes	Left temporal lobe lesion on both conventional and FLAIR sequence. Left parietal lesion only seen on FLAIR
10	45/M	Aura: deja vu. Seizure: blank stare and automatisms. Interictal EEG: non-specific changes, maximal right temporal region	Additional lesions in left and right parietal regions seen only with FLAIR. Left temporal lesion seen with both sequences
11	26/M	Complex partial seizures with oral-facial and manual automatisms. Interictal EEG: right temporal sharp and slow wave activity continuously. Ictal EEG: bilateral or right hemisphere onset. Cavemous haemangioma resected from right temporal neocortex	Right anterior temporal lesion best seen with post enhancement. T1 weighted spin echo. Periventricular lesions only seen with FLAIR

Figure 1 Case 1: transverse SE 2500/80 (A), and FLAIR 6000/160/2100 (B) images in hippocampal sclerosis. The left temporal horn is shown in both (A) and (B) but the increased signal in the hippocampus is only seen in (B) (arrows). Histology showed sclerosis.

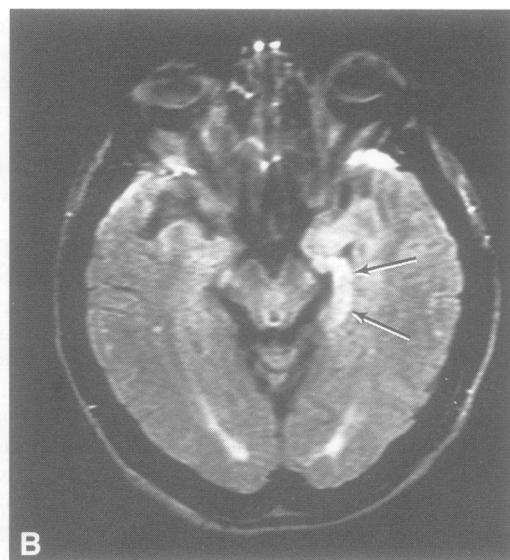


Figure 2 Case 4: transverse SE 2500/80 (A) and FLAIR 6000/160/2100 (B) images. The increased signal in the left amygdala is only seen in (B) (arrow). A focal developmental abnormality was found at histology after surgery.

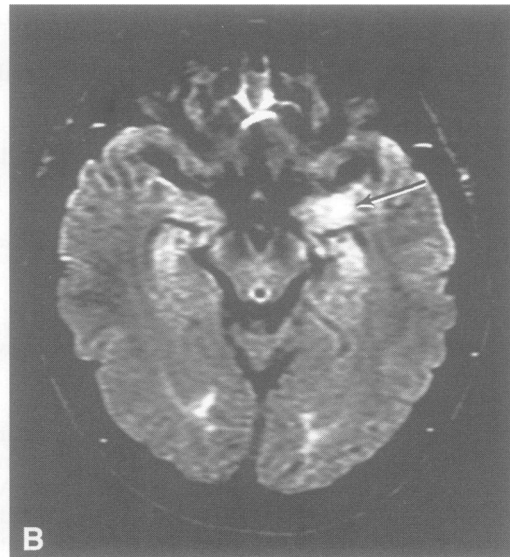


Figure 3 Case 8: transverse SE 2500/80 (A) and FLAIR 6000/160/2100 (B) images. The occipital lesion is only seen in (B) (arrows).

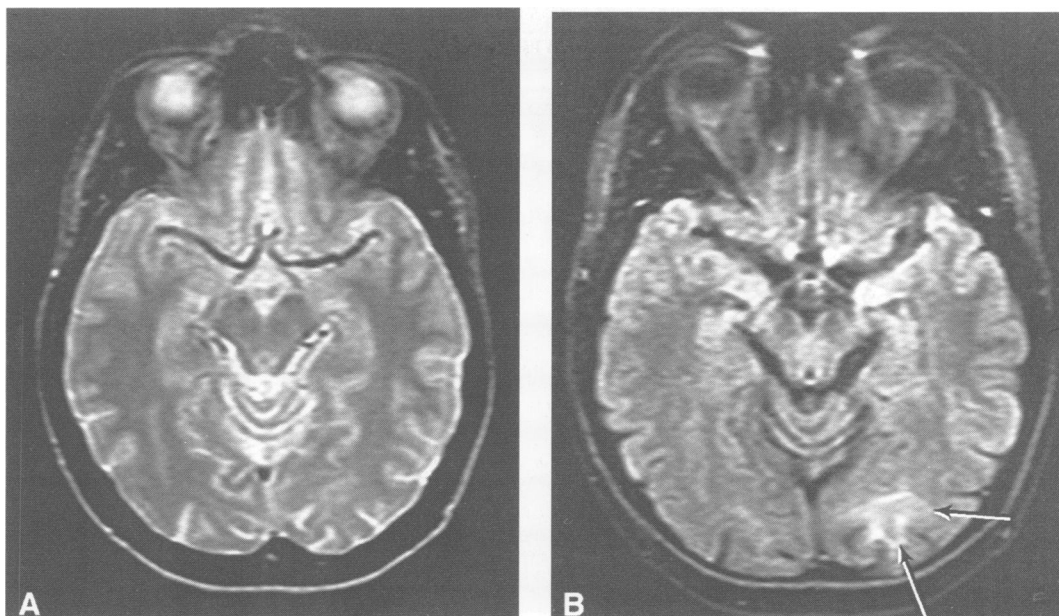
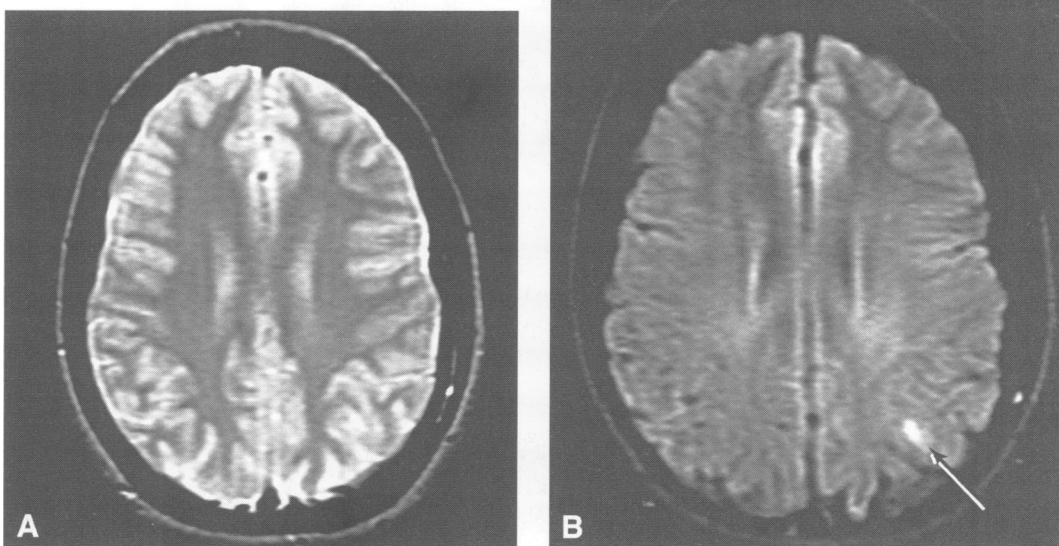


Figure 4 Case 9: transverse SE 2500/80 (A) and FLAIR 6000/160/2100 (B) images. A lesion is only seen in the left parietal region (B) (arrow).



and FLAIR (IR 6000/160/2100) scans. Intravenous gadopentetate dimeglumine (0.1 mmol/kg) was given in the 33 cases who did not have a history of allergy. The T1 weighted scans were repeated in both planes after this. A slice thickness of 6 mm and spatial resolution of 128×192 was used in each case. Two data acquisitions were used for all except the FLAIR sequence where only one acquisition was used.

The scans were reviewed independently by two radiologists (NMdeS and GMB). Positive results were recognised only when both radiologists concurred.

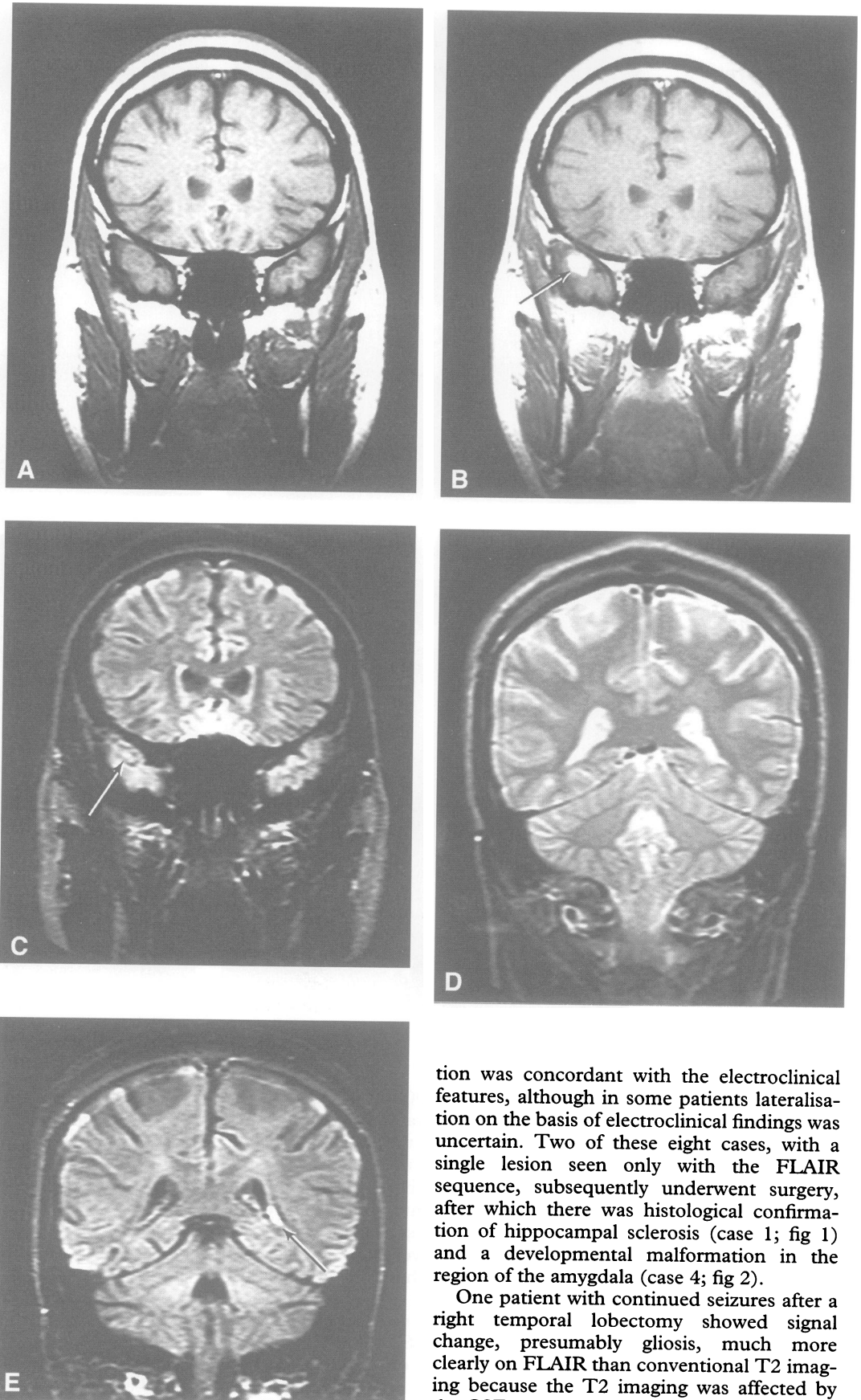
Results

Thirty six patients were examined and 20 (56%) were positive on one or more pulse sequences. In these 20 patients, a total of 25 lesions (or groups of similar lesions—namely, punctate or periventricular lesions) was seen.

Twenty four of the 25 lesions were seen with greater conspicuity with the FLAIR sequence than with all the conventional sequences. Only one lesion, a congenital cavernoma, was better seen with a conventional sequence (a contrast enhanced T1 weighted spin echo) than with the FLAIR sequence. Sixteen patients were negative on both conventional and FLAIR imaging.

Of the 20 positive patients, eight had a single lesion that was seen only with the FLAIR sequence (cases 1–8, table) and thought to be of aetiological significance. These lesions were situated in the hippocampus (two cases; for example, fig 1), hippocampus and amygdala (one case), amygdala (four cases; for example, fig 2), and occipital lobe (one case; fig 3). In three cases (cases 9–11, table), an additional significant lesion or lesions (two cases) were only seen with the FLAIR sequence (for example, fig 4). These were situated in the parietal region (two cases), deep white matter,

Figure 5 Case 11: congenital cavernoma and tuberous sclerosis: Coronal SE 720/20 without (A) and with (B) intravenous contrast enhancement. Coronal FLAIR 6000/160/2100 (C) at the same level. Coronal SE 2500/80 (D) and FLAIR 6000/160/2100 (E) scans in a more posterior location. The congenital cavernoma is better seen in (B) than (C) (arrow). The periventricular lesion is only seen in (E) (arrow).



and periventricular regions. All lesions thought to be of aetiological importance were identified in both the transverse and coronal planes.

In all eight cases in which the lesion was seen only with the FLAIR sequence, the loca-

tion was concordant with the electroclinical features, although in some patients lateralisation on the basis of electroclinical findings was uncertain. Two of these eight cases, with a single lesion seen only with the FLAIR sequence, subsequently underwent surgery, after which there was histological confirmation of hippocampal sclerosis (case 1; fig 1) and a developmental malformation in the region of the amygdala (case 4; fig 2).

One patient with continued seizures after a right temporal lobectomy showed signal change, presumably gliosis, much more clearly on FLAIR than conventional T2 imaging because the T2 imaging was affected by the CSF signal from the resection.

One patient had a right temporal abnormality that was most evident after contrast enhancement (case 11, fig 5). This was confirmed as a congenital cavernoma (without evidence of previous bleeding) at histology. This was the only lesion that was better seen with the conventional sequences. Additional

periventricular lesions were seen in this case with the FLAIR sequence (fig 5) and this raised the possibility of coincidental tuberous sclerosis in this patient. Ocular and skin findings consistent with this diagnosis were subsequently seen.

Discussion

In this preliminary study of 36 selected patients with partial epilepsy, 11 additional abnormalities were identified with the FLAIR sequence. In eight patients only one abnormality was shown and this was only seen with the FLAIR sequence. The relevance of these lesions is clearly shown by the high degree of concordance with the electroclinical features, and pathological confirmation in those proceeding to surgery. Three patients with structural abnormalities on conventional and FLAIR sequences had additional lesions on the FLAIR sequence.

The detection of new findings with the FLAIR sequence may be important through providing the information necessary for surgery and also in showing previously unrecognised structural lesions that may be a contraindication to surgery, indicating the need for further neurophysiology, or accounting for some surgical failures.³

The basis for the improved performance of the FLAIR sequence is probably the suppression of CSF, the increase in T2 weighting, and the fact that the contrast between grey and white matter is reduced by the mild T1 weighting of the sequence introduced by the inversion time of 2100 ms. This reduces contrast between grey and white matter and provides a low contrast background against which the increased signal of the lesions can be seen. The behaviour of the lesions is consistent with the increase in T2 found in hippocampal sclerosis using the Carr-Purcell-Meiboom-Gill sequence, but the FLAIR sequence may be more sensitive and avoid the problems of misinterpretation due to the CSF signal.⁴

The poor conspicuity of the congenital cavernoma (fig 5) could have been due to flow of blood in the lesion. This may produce dephasing of the signal during the long echo time (160 ms) of the FLAIR sequence and lead to a reduction in signal. This would be consistent with the pronounced contrast enhancement seen at the shorter echo time of 20 ms with the T1 weighted spin echo sequence.

Performing FLAIR in both transverse and coronal planes doubled the scanning time in this study but did not increase the diagnostic yield over performing FLAIR in one plane. By the use of variants of the FLAIR sequence employing multiple data acquisitions with different phase encodings after each excitation (as in the rapid acquisition with relaxation enhancement (RARE)⁵) sequence it is possible to increase the speed of the sequence^{6,7} and this is likely to be of importance, making this approach more time efficient.

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