related to the continuing use of diazepam (Ashton, 1984). The severe exacerbation of these symptoms accompanied by depression and agitation three months before she was seen represented the onset of a depressive illness.

Total relief of panic, anxiety and phobic symptoms when benzodiazepine drugs are withdrawn after long usage can sometimes take as long as six months, and commonly takes two to three months. We thought that withdrawal from the benzodiazepines was responsible for the reduction in anxiety and in the agitated movement in the mouth which followed admission. Hynagogic jerks, which indicate a continuing effect on the brain, can persist for as long as two or three months after benzodiazepine drugs are stopped. There is thus clinical evidence which supports the view that there may be long-term effects on the brain which result from prolonged administration of benzodiazepine drugs. As to the mechanism of this, there are specific benzodiazepine binding sites in the brain and the benzodiazepine drugs act by enhancing the effects of gamma-aminobutyric acid. These changes could interfere directly with recovery from depressive illness, and they may also interfere with the efficacy of ECT.

This case study describes a dramatic change from being extremely ill to being extremely well. The only variables to have changed between the two administrations of the same treatment were the passage of time and the elimination of the effect of benzodiazepines on the brain. It is reasonable to make the hypothesis that the failure to respond to ECT at the first administration, not long after she had stopped the drugs, was because there were still substantial changes in her brain, and that despite the fact that she had adequate fits the drugs interfered with the psychic response. On the second occasion one might have expected her brain to have returned to normal. The alternative hypothesis, that the whole of this lady's improvement resulted from stopping the benzodiazepine drugs, is unlikely since the symptoms became progressively worse for many months after they were stopped. Finally, there is the possibility that this is one of those rare patients with depressive illness who fail to respond to ECT but recover when the treatment is repeated after a few months.

This case history indicates that the presence of benzodiazepine drugs in depressed patients may interfere with the ability of the brain to respond to bilateral ECT and to the need for these drugs to be discontinued in the treatment of depressed patients. We do not agree with Pettinati *et al* (1990) that this may be "clinically impractical for some patients"; it may be difficult if the doctor is not convinced that it is essential, but it can usually be achieved and without it a seriously ill patient may be deprived of the possibility of recovery.

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The Effect of Anticonvulsants on Cognitive Functioning Following a Probable Encephalitic Illness

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A 54-year-old man developed an acute encephalitic illness and subsequently suffered from marked memory difficulties. Considerable improvement with anticonvulsant therapy was demonstrated using a ward-based observation programme and serial neuropsychological assessments. The case report highlights the prominent role played by epileptic activity in determining the severity of the patient's amnesia.

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Post-encephalitic sequelae most commonly include a severe amnesia, secondary to the extensive brain damage which frequently occurs to the structures of the temporal lobe including hippocampus amygdala and uncus (Heirons *et al*, 1978). Care should be taken, however, to distinguish between the memory deficit arising from the encephalitis and that from other concomitant disorders. In particular, epileptic activity, whether clinical or subclinical (e.g. Binnie *et al*, 1987), may affect performance on tests of intelligence and memory.

Case report

T, a 54-year-old married salesman, developed fever with nausea and weakness, having no previous history of neurological or psychiatric illness. Mild jaundice was suspected but there was no history of passing highly coloured urine or pale stools. The question of food poisoning was raised, but *Salmonella shigella* and *Campylobacter* were not isolated in faecal specimens. Routine haematology was normal. Over the following two weeks the fever persisted; he developed anorexia, and lost 9.5 kg (21 lb). During this period he was episodically incoherent and confused, especially at night, when he would also experience visual hallucinations. Over the next week he gradually recovered and began to resume some of his daily activities but was not well enough to return to work.

Five weeks after the onset of the illness he became acutely disturbed. He was observed to be visually hallucinated and frightened, and collapsed to the floor, throwing his hands in the air. He was unconscious for a few minutes but there was no incontinence or self-injury. On regaining consciousness he was obviously confused and staggered about in an uncoordinated manner until an ambulance arrived to take him to hospital. A wide range of special investigations were all normal, including an examination of cerebrospinal fluid (CSF), a computerised tomography (CT) scan with contrast, two electroencephalograms (EEGs), a full virology screen, and thyroid function tests.

The most noticeable impairment suffered by the patient was that he was forgetful of day-to-day events. The problem was variable, however, with complete disorientation at times and fairly coherent orientation at others. He was frequently agitated, and occasionally assaulted other patients. Additionally, he had paranoid ideas, referring to poisoning of his food and drink by staff. He was started on oral chlorpromazine and later given a series of intravenous injections of Parentrovite (a Wernicke–Korsakoff syndrome had been suspected, even though there was no history of alcohol abuse). One such injection was followed by a tonic–clonic generalised seizure and clobazam (10 mg b.d.) was started.

Soon after this episode, during weekend leave, his wife reported that he was exhibiting childlike attitudes (e.g. asking whether he had to wash his hands before dinner), and had lost interest in sex. At times he was unable to find his way around their house. Further paranoid ideas were in evidence. He was transferred to the psychiatric ward, having been diagnosed as suffering from depression and an organic amnesic syndrome, and started on imipramine. There he remained confused, forgetful, and disorientated.

He was transferred to the Maudsley Hospital for an opinion concerning a possible hysterical basis for his memory difficulties, even though the referring consultant still considered them to be founded in brain damage. On admission he was only taking buspirone (5 mg t.d.s.), which had been commenced before transfer. The only significant findings on examination were myoclonic jerks of the right-sided limbs, with the legs being more frequently affected than the arms. These had commenced a few days before his transfer. On examination he was pleasant and co-operative, anxious at times and indifferent at others. No abnormal thoughts or perceptions were elicited.

Most remarkable were T's dense memory deficits for certain events, occasionally accompanied by confabulation. For example, when asked about his father on one occasion he gave a detailed account of how well he got on with him, although in fact he had been dead for 25 years. At a subsequent interview he was quite clear upon this issue. He provided an inaccurate account of what the family had done the previous Christmas and summer holiday, and also forgot matters that had been discussed only a few minutes earlier. On admission he could recall his own birthdate but not his wife's; neither could he remember his telephone number nor his two daughters' ages. He indicated that he had stopped working only two weeks earlier, and was unable to remember the previous hospital admissions or his physical illness. During the early days of his transfer he had great difficulty in finding his way around the ward. Noticeable, however, was the degree of insight retained by T into his impairment and the distress it caused him.

Although mostly calm and pleasant, he was sometimes irritable, agitated, and suspicious, believing for example that staff were plotting against him. When behaviourally disturbed, he was occasionally disinhibited, being over-familiar with both staff and other patients.

Extensive investigations were carried out to exclude potentially relevant haematological or metabolic disorders. All were normal. Syphilis serology was negative and lumbar puncture yielded normal results. Urinary and blood levels for heavy-metal poisoning and urine drug screens were negative.

However, serial EEGs initially showed abnormalities in the right anterior temporal region, with intermittent non-rhythmic slow activity and epileptiform activity in the form of sharp waves. Hyperventilation induced a simple partial seizure with a run of sharp and slow waves in the right centrotemporal region. During sleep the sharp waves became more distinct and widely distributed, appearing independently over the middle of both hemispheres and more prominently on the left than the right. Magnetic resonance imaging and CT scans were normal, but a single-photon emission computerised tomography scan showed hypoperfusion of the right anterior temporal region, and bilaterally in the temporooccipital regions.

In view of the marked EEG abnormalities, the frequency of the myoclonic jerks and the occurrence of two brief but clear-cut seizures on the ward, it was decided to embark on anticonvulsant treatment while monitoring his cognitive state, to see whether epileptic discharges might be interfering with memory functioning.

The patient was initially started on 75 mg phenytoin twice daily and gradually increased to 400 mg daily over five and a half weeks. During this time the myoclonic jerks decreased in frequency and his memory was observed to improve. Carbamazepine was gradually substituted for phenytoin, and was increased gradually from 50 mg daily to 400 mg twice daily so that four months after his transfer he was on carbamazepine alone. In view of his lability of mood and paranoid ideation he had been started on 800 mg sulpiride daily which was reduced to 200 mg *nocte* and maintained as a prophylactic against further psychotic episodes. His buspirone had been discontinued.

In order to evaluate the variability of T's memory performance, a ward-based memory and orientation assessment was devised. This involved four aspects:

(a) orientation (date, month, year)
(b) the number of trials taken to learn a seven-component name and address; a different name and address was given on each occasion
(c) the number of components of the name and address recalled after one hour
(d) whether T remembered to carry out a task after a delay of at least two and a half hours; scores reflected whether he remembered being given a task, what it was, and whether or not he executed it out without error, with higher scores reflecting better recall and performance.

This assessment was conducted by ward nursing staff throughout his admission.

Table 1 indicates T's scores on the four memory/orientation tasks during three periods: (a) with no anticonvulsant or neuroleptic medication; (b) with sulpiride and phenytoin; (c) with sulpiride and carbamazepine Unfortunately, it was not possible for measures to b obtained when taking sulpiride alone. Values of myocloni seizure frequency for periods (a) and (b) are likely to b underestimates, since T would spend time alone in his roor and seizure frequency was not formally recorded durin other activities such as occupational therapy.

Table 1
Mean (s.d.) scores and number of observations for tasks with different medication, together with mean observed seizure frequency for periods when tasks were administered

	Drug-free period	Phenytoin and sulpiride	Carbamazepine and sulpiride
Orientation (maximum score 3)	2.22 (0.83) ($n=9$)	2.36 (0.5) ($n=11$)	2.85 (0.39) ($n=7$)
No. of trials to learn name and address (minimum score 1)	2.5 (1.69) ($n=8$)	1.45 (1.21) ($n=11$)	1.42 (0.79) ($n=7$)
No. of components of name and address recalled after 1 hour (maximum score 7)	1.71 (1.38) ($n=7$)	4.09 (2.02) ($n=11$)	5.29 (1.49) ($n=7$)
Score on recall of task (maximum score 4)	0.875 (1.2) ($n=8$)	3.0 (0.95) ($n=12$)	3.28 (0.95) ($n=7$)
Mean no. of seizures observed by staff per day	2.67	0.58	0

It can be seen that T improved with the introduction o phenytoin and sulpiride, but achieved further improvemer with carbamazepine. Seizure frequency was reduced b phenytoin, but clinical epileptic activity was abolishe by carbamazepine.

Two months after transfer, a routine EEG indicate persistent sharp waves in the right anterior temporal regior in sleep, the sharp waves were more widely distributed, ove the right mid- and anterior temporal regions. At this time however, no left-sided epileptic activity was detected.

Validation of the nursing observations of improve memory in conjunction with decreased epileptic activit came from neuropsychological assessments. Table indicates the results of assessments conducted durin periods (a) and (b). The initial verbal-performance I(discrepancy mainly resulted from the disruption of moto behaviour by myoclonic jerks, which were particularl frequent when T was under pressure. After experiencin these jerks, T would often forget what he had been doing

Table 2
Neuropsychological assessment results comparing end o drug-free period to period on sulpiride and phenytoin

	Assessment during drug-free period	Assessment when on phenytoin and sulpiride
Verbal IQ[1]	103	107
Performance IQ[1]	94	116
Logical memory[2]		
immediate	11.25	12.25
delayed 1 hour	0	6.25
percentage recall	0	51
Rey–Osterreith figure percentage recall[3]	0	41
Benton visual retention test[4]		
correct	5	6
errors	9	4
Verbal paired associates[2] (maximum score 21)	9	14.5
Word recognition[5]		
scaled score	10	13
Face recognition[5]		
scaled score	8	7
National Adult Reading Test errors[6]	19	-

-, not assessed.
1. Wechsler (1986).
2. Wechsler & Stone (1945).
3. Rey (1959).
4. Benton (1974).
5. Warrington (1984).
6. Nelson (1982); predicts WAIS verbal IQ 112, performance IQ 11

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They occurred during verbal and non-verbal tasks, and appeared to contribute to the variability in his cognitive ability. By the second assessment the jerking had decreased considerably in frequency. His initial inability to recall having previously heard the logical memory passages or seen the Rey–Osterreith figure had resolved by the second session. Delayed recall was poor, however. His ability to reproduce material immediately after viewing it improved. New verbal learning and word recognition improved, although face recognition did not.

T's performance was also seen to improve considerably on a standardised battery testing everyday memory, the Rivermead Behavioural Memory Test (Wilson *et al*, 1985). This examines such abilities as remembering a name given to a photograph of a face; recognising pictures of objects and faces seen earlier; remembering a question to ask the examiner, and to ask the examiner to return a personal belonging hidden at the beginning of the session; and recalling a short story and route around the room. Two scores are obtained: the profile score, which takes into account the amount of detail recalled on each test; and a more stringent index of level of memory functioning, the screening score. Scores obtained on parallel forms during periods (a), (b) and (c) corroborate the observed improvement on the ward-based nursing assessments, with profile scores of 44.5, 56.5 and 59.5, and screening scores of 3, 6 and 9 respectively; this corresponds to 'moderate memory impairment' during periods (a) and (b) and 'mild memory impairment' during period (c). His ability to recognise and name members of staff had improved dramatically, and he was able to take walks alone near the hospital without getting lost.

Gradual re-integration into his home, although not back to his previous employment, was planned. His mental state was considerably improved compared with measures obtained shortly after admission, with a score of 0 compared with 10 on the 28-item General Health Questionnaire (Goldberg, 1972) a state anxiety score (State–Trait Anxiety Inventory; Spielberger, 1983) of 33 compared with 51, and a score on the Beck Depression Inventory (Beck *et al*, 1961) of 3 compared with 7. This was reflected in his changed behaviour on the ward, where he was calm and tolerant of others.

Discussion

The most likely diagnostic formulation is that T had experienced an acute encephalitic illness leading to multiple sequelae: generalised and simple partial epilepsy, dense amnesia, and behavioural changes. While his memory continued to show some impairment throughout admission, considerable improvement was witnessed once anticonvulsant medication had been instituted. Although this coincided with the resumption of neuroleptic medication, the abolition of observed seizure activity and improvement of the EEG by carbamazepine suggests that the anticonvulsants, in decreasing seizure activity, permitted T to process material sufficiently to be able to provide improved recall after a delay. Without withdrawing anticonvulsants we cannot affirm with certainty that this, rather than spontaneous recovery of his memory ability, has been the mechanism behind his improvement, but it was not felt ethically justifiable to try this manoeuvre.

The case indicates the importance of decreasing seizure activity before providing a final description of level of cognitive impairment, and of devising practical ward-based assessments which can be repeated frequently in order to monitor levels of functioning. It illustrates the potential contribution of epileptic activity to the memory deficit seen following an acute encephalitic illness.

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