of these patients was opiate positive. In the second group of patients, withdrawal symptoms could be observed neither during the continuous naloxone treatment nor after cessation of this drug therapy as soon as opiate excretion in urine was no longer measurable.

Our observations, which we will report in detail, seem to confirm the experimental data of Hendrie (1985), that withdrawal symptoms will be attenuated by high doses of naloxone. A short anaesthesia was administrated in these patients, because during the flooding phase of naloxone in the body, acute withdrawal symptoms had to be expected acutely after administration of a high dose of naloxone (Jasinsky *et al*, 1967). This seems to be why the naloxone treatment was acutely discontinued and not followed up in the study of Dr Vlissides *et al*. According to our hypothesis, the paradoxical attenuation of withdrawal symptoms may be observed only when the naloxone in the body reaches a certain level. Furthermore, from our study it can be concluded that naloxone treatment should be continued for as long as morphine alkaloids are present in the body.

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Temporal Lobe Epilepsy

SIR: We read with interest the article by Herzberg & Fenwick (*Journal*, July 1988, **153**, 50–55), and we would like to add our experience regarding this important topic. From a prospective sample of 65 temporal lobe epileptics (TLEs), derived from a variety of sources including a neurological clinic and private practice, 15 (23%) manifested aggression, according to the relevant outlines of Bear & Fedio (1977), as estimated by two psychiatrists by means of psychiatric interview and additional information from the patient's relatives.

The aggressive behaviour, as estimated by psychiatic interview as well as from the index of aggressiveness of the MMPI, was associated with early onset of seizures (and especially with onset before the age of 5). No relationship was found between aggression and EEG features or frequency of seizures. However, we did not find any relationship of the aggressive behaviour with the male sex, nor with the current age of the patients.

We cannot agree with the statement of Drs Herzberg & Fenwick that the existence of temporal lobe personality is dubious. We found that from the 65 TLEs, 16 (24.6%) who received a DSM–III diagnosis of organic brain syndrome (organic personality syndrome and/or organic delusional syndrome or organic affective syndrome) were also differentiated significantly from the rest of the TLEs and a control group of 36 non-TLEs in 9 out of 18 of Bear & Fedio's personality traits (Garyfallos *et al*, 1988). Those traits were religiosity, sexual alteration, aggression, viscosity, paranoia, circumstantiality, humourlessness, hypergraphia, and hypermoralism. We believe that a minority of TLEs manifest some personality traits. Not every patient who has traits has all of them, and perhaps the traits are not exactly the 18 reported by Bear & Fedio (1977). Furthermore, more recently Bear (1986) wrote that "not every patient with temporal lobe epilepsy develops interictal behaviour changes or changes to the same degree".

Concerning the aetiology of aggression in TLE, we agree that this cannot be attributed to the epilepsy itself, i.e. to the actual occurrence of seizures. However, we speculate that both the epileptic seizures and the aggression (but also other psychopathological and behavioural manifestations) are the result of the same cause, namely the limbic epileptic focus, or more correctly "the intermittent limbic spike focus" as Geschwind (1983) suggested. This does not necessarily mean that there is a structural brain damage detectable in the CT scan. The phenomenon of kindling, which was proposed as an experimental model of epilepsy, gives a possible explanation. Its action is not limited to the development of epileptic seizures, but also leads to interictal aggressive behaviour (Goddard, 1980) as well as to other emotional and behavioural alterations. It is possible that for the development of aggression through the kindling procedure, some indispensable conditions are needed. One of these can be its influence from very early age. According to our findings, the aggressive subjects were significantly more likely to experience onset of seizures before the age of 5. Furthermore, Geschwind (1983) reports that the influence of kindling is different in intrauterine life, in childhood

and in adult life. The authors' finding that the aggressive group was less educated and had poorer occupational records (we also found the latter) could probably be the result of their psychological and behavioural problems and not one of the causes.

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Capgras' Syndrome or Prosopagnosia?

SIR: Lipkin (*Journal*, July 1988, **153**, 117–118) reported a single-case study of Capgras' syndrome with "delusional" misidentification of spouse, in a 71-year-old woman. This was observed to be the initial presenting symptom of a dementia. Also, at no time did she misidentify other persons, and the onset of the disorder was considered to be sudden, as was its termination. A hallmark of Capgras' syndrome is that it is considered to represent *delusional* misidentification of familiar persons (Capgras & Reboul-Lachaux, 1923).

Prosopagnosia, a relatively rare neuropsychological disorder, presents in a very similar fashion. It was first described by Wilbrand (1892) in a 63-year-old woman as a result of bilateral occipital infarction. This disorder is also characterised by the failure to recognise *familiar* faces and is usually of acute onset (Damasio & Damasio, 1983). At autopsy, all had bilateral occipital lesions (Lhermitte *et al*, 1972), although controversy remains as to whether unilateral lesions of the occipital lobe will produce this disorder. Interestingly, Christolodou (1977) observed neuropsychological impairment in nine out of ten patients with Capgras' syndrome, and suggested non-dominant occipital pathology. This localisation is very similar to that reported in prosopagnosia.

It may be possible that Capgras' syndrome and prosopagnosia reflect a similar, or the same, underlying process. A distinction between the two is that Capgras' syndrome is considered to represent delusional misidentification of familiar others. This cannot necessarily be claimed in Dr Lipkin's case, as the presence of an organically-based dementia was established.

We would agree with Dr Lipkin's conclusion that organic pathology should be considered in cases of Capgras' syndrome. However, it may be possible that this entity can be defined even more specifically as a form of prosopagnosia, particularly in early stages. Concurrent with neurological and electrophysiological examination and brain imaging, specialist neuropsychological assessment (if available) may be of assistance in determining the presence of dementia, and neighbouring parieto-occipital signs.

The distinction between prosopagnosia and delusion of an imposter is an important one. When a labelled photograph of the misidentified individual is provided (Pritigano, 1981), prosopagnosics benefit from the label, while individuals with Capgras' syndrome would presumably continue to fail to recognise the individual. However, this strategy may be limited if individuals are in an advanced stage of dementia. The presence of such organically-based, as opposed to psychogenic, deficits certainly has relevance to future patient management.

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