he was well enough to be discharged, and he went without UEP for two months, with only rare, soft, and indistinct hallucinatory 'mumbling'. Visual, but not auditory hallucinations gradually developed. A few weeks after readmission, right and left plugs were sequentially tried, but without effect. Auditory hallucinations did not recur until several months into this relapse as his general condition worsened. As the primary investigator had left the city, UEP was not again tried.

While not proving the efficacy of UEP this case strongly suggests it, and the recurrence of psychosis without auditory, but with visual hallucinations implies that UEP effects were modality-specific. Cross-modal 'compensatory' processes may have led to the emergence of visual hallucinations. This result with UEP opposite to the predicted side requires an alternative formulation to Green & Kotenko's (1980) model. One possibility is suggested by the reduced right hemisphere evoked response to left ear stimulation in schizophrenia (Connolly *et al*, 1985). By limiting auditory input to the left ear, a right ear plug might enforce better engagement of right hemisphere attention. There is also evidence of left hemisphere overactivation and dysfunction in schizophrenia (Gruzelier, 1983). By nearly eliminating right ear input, right UEP may help to normalize left hemisphere activation and function. The relevance of this to auditory hallucinations is suggested by the fact that plugging the ear contralateral to the affected hemisphere alleviates auditory illusions associated with temporal lobe epilepsy (Jacobs *et al*, 1973). UEP could interfere in several ways with processes underlying auditory hallucinations.

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Awake, Perchance Asleep?

SIR: It is generally agreed that patients suffering from depression show reduced Sleep Efficiency, decreased Slow Wave Sleep and latency to REM, and changes in the distribution of REM sleep. Some recent models of depressive illness have been based in major respects on these findings (Borbely & Wirz-Justice, 1982; Wehr & Wirz-Justice, 1981). However, recording of the EEG has been restricted to the nocturnal sleep period in the studies demonstrating these abnormalities of sleep. Studies in normal subjects show that the duration of preceding wakefulness has a profound influence on subsequent sleep (Borbely, 1982), and that daytime naps may result in nocturnal sleep which has two of the main features shown in depressed patients: decreased Slow Wave Sleep and REM Latency (Karacan *et al*, 1970). We are aware of only one study in which the EEG of depressed patients was recorded over a 24 hour period: the average duration of daytime sleep was 5.7 hours (Shimizu *et al*, 1979). Because patients in this study were selected on the basis of complaints of increased sleepiness and time in bed, the results may be biased, though to what extent cannot be determined.

Precise information about the occurrence of sleep over 24 hour periods in a random sample of patients would be required to define more specifically the EEG characteristics of sleep in depression. Some information can be obtained by behavioural observation. On the Treatment Evaluation Unit at the Kingston Psychiatric Hospital we obtained behavioural observations in 20 randomly selected patients suffering from major affective disorder (depression) according to DSM–III criteria (three bipolar, 17 unipolar; mean age 53.3 years). Over periods of at least five days of observation for each patient, all showed episodes of bed rest when seen at half-hourly intervals between 0800 and 2100 hrs. Bed rest was noted on 62% of the observation days, with a mean duration of 2.6 hours. Although in this sample we cannot rule out the possible sedating effects of psychotropic medication, this finding supports the notion that daytime sleep may occur frequently in depressed patients not selected for increased daytime sleepiness.

Behavioural observation may, however, underestimate the amount of sleep that occurs. We have now obtained a continuous EEG record of sleep activity over the last eight hours of a 48-hour sleep deprivation in a depressed patient. The record was made with a portable cassette monitoring system (Oxford Medilog). During the time of presumed wakefulness the patient accumulated 11.1 minutes of Stage 2 sleep. In most instances the episodes did not exceed 20 seconds in duration; the shortest was

as brief as four seconds and the longest was 48 seconds. In addition, longer episodes in the record showed slowing of the EEG, in the absence of eye movements, which were not scored as sleep because the wave forms did not meet the formal amplitude criterion. There were also many instances of sleep spindles against a background of eye movements and alpha frequency EEG. In spite of the patient's excellent co-operation in trying to keep awake, and constant observation by the nurses, neither they nor the patient were aware of the occurrence of sleep, though the latter had felt drowsy at times.

These observations indicate the need of continuous recording of EEG to verify presumed wakefulness in studies on the effect of sleep deprivation in depression. They also call into question previous attempts to explain the origin of the sleep disturbance typical of these patients, raising the possibility that it may be, at least in part, a consequence of the daytime discharge of non-REM sleep.

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Capgras Syndrome

SIR: I wish to report two cases of Capgras syndrome for time, a previously unreported phenomenon. Each patient also suffered from multiple other misidentification syndromes.

Case reports: (i) Ms A, a 33 year old white female with paranoid schizophrenia (DSM–III), had chronic delusions that multiple persons had been replaced by physically identical imposters who were either anonymous persecutors (Capgras syndrome), or specific other people (Fregoli syndrome). She also believed that certain places were identical duplicates of other locations known to her (reduplicative paramnesia). In addition she reduplicatively misidentified time, believing that 1984 was 1991. This occurred either episodically or simultaneously. At different interviews she might assert that it was 1984, or 1991, or both. It is important to note that she truly believed that two different periods of chronological time existed simultaneously—as opposed to being disoriented for an misnaming the true time in which she lived.

Her history and physical examination were negative for neurological abnormalities. Computerised tomography (CT) of the head, positron emission tomography of the brain, and evoked potentials were normal. Electroencephalography showed slow wave activity with lateralised predominance to each hemisphere at different times. Neuropsychological testing showed bilateral parietal and frontal lobe dysfunction on a pattern of diffuse right hemisphere dysfunction.

(ii) Mr B, a 31 year old white male with paranoid schizophrenia (DSM–III), had chronic delusions that duplicates of himself existed (syndrome of subjective doubles), that certain individuals were not only duplicates of others, but also looked like them (syndrome of intermetamorphosis), and that certain individuals were specific other people (Fregoli syndrome). He also had the longstanding belief that time was duplicated and that he existed in both the present and another time in which he was aged 82. When he thought he was in the future he believed that the entire world was also in the future, although he did not misidentify his surroundings. As with case (i) he had the delusion that two periods of chronological time simultaneously coexisted.

His past history was notable for three head injuries with loss of consciousness, one with coma requiring ventilation. His family history was positive for mental retardation and alcohol abuse. Physical examination and routine laboratory evaluation were unremarkable. EEG was within normal limits, and CT scan revealed bilateral frontal, temporal, and parietal lobe atrophy.

Classical misidentification syndromes include Capgras syndrome, Fregoli syndrome, syndrome of subjective doubles, syndrome of intermetamorphosis, and reduplicative paramnesia (Christodoulou, 1978; Joseph, 1986). It has also been suggested that psychotic syndromes of disorientation for place or time and reduplication of body parts are misidentification syndromes (Weinstein *et al*, 1954). The essential feature of these is a chronic psychotic misidentification of person, place, time, or other object, usually (but not always) with psychotic reduplication of that object. Weinstein *et al* (1954) discussed reduplication for time, but the case reports