We used this methodology in Kenya (Dhadphale *et al*, 1983). The SRQ was locally validated and a 7/8 cut-off point was used. Our Department of Psychiatry has now adopted this methodology as a standard procedure for screening psychiatric morbidity in various settings; for example a traditional healer's clinic, during a follow-up study of post-natal women, and in infertility studies. By July 1987, five major epidemiological studies were planned and successfully completed by our postgraduate psychiatric students for their dissertations for the Masters degree in psychiatry.

Although we are generally happy and satisfied with this two-stage methodology, some of the shortcomings of the procedure are: (a) we find only 11 questions of clinical significance; (b) psychotic questions (21 and 23) are too vague and equivocal, especially in our local cultural setting; and (c) inclusion of the brief MAST is important, as both the SRQ and CIS do not appear to be very sensitive instruments for picking up alcohol-related psychiatric disorders. Hence, we have appended the brief MAST to our research protocol.

In a paper based on our extensive experience in East Africa (in preparation), we have discussed these and other parts more critically. We have also translated our research instruments in Kiswahili and other languages.

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Khat-Induced Paranoid Psychosis

SIR: Gough & Cookson (*Journal*, February 1988, **152,** 294) mentioned that in our description of the patient F. K. with khat-induced paranoid psychosis (*Journal*, February 1987, **150,** 247–249), the urine test was not in keeping with the diagnosis, because it was positive for morphine and dihydrocodeine but not for amphetamines.

We were not able to explain the presence of morphine and dihydrocodeine in the sample, as we mentioned in the original description. We also stated that the urine sample in question was taken nine days post-admission, at which stage breakdown products of khat, which might have registered a postitive test for amphetamine-like substances (depending on the specificity of the actual test used) would no longer have been present in the urine. In this case the diagnosis was made on clinical grounds and was confirmed by the patient bringing in the khat that she had been using, which was then identified by the Regional Poisons Laboratory.

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Down's Syndrome with Mania

SIR: Singh (*Journal*, March 1988, **152,** 436–437) responded to our previous case report (*Journal*, February 1987, **150,** 249–250) of DSM–III-diagnosed mania in a young adult with Down's syndrome with several points which we believe require further discussion.

Firstly, our report did not claim that the case was severe enough to require seclusion. However, it is our understanding that 'seclusion' has never been one of the diagnostic criteria for mania. That notwithstanding, the reported case meets the criteria for mania. However, we would hasten to add that developmental considerations *per se* might modify the clinical presentation of mania, and particularly the necessity for seclusion or other means of physical restraint.

Secondly, our discussion of Prange's hypothesis was not meant to suggest a heightened association of Down's syndrome with mania in the absence of clinical data. To the contrary, the intention of that discussion was to highlight the current lack of support for an association, in either direction, between any mental disorder and any physical disorder on the basis of current knowledge of neurochemistry.

Most importantly, Dr Singh presented the literature pertaining to catecholamines by writing that "post-mortem studies of the brains of patients with Down's syndrome clearly show the cell loss in the noradrenergic system of locus coeruleus and dorsal motor vagus, not only in the middle-aged, but also in younger patients." Careful review of the cited references reveals that only two Down's syndrome patients below the age of 48 had been studied: Yates *et al* (1983) found a decrease in hypothalamic but not caudate norepinephrine in one 27-year-old Down's syndrome patient, and Mann *et al* (1985) found decreased cell count in the locus coeruleus but not dorsal motor vagus in the brain of a 31-year-old patient with Down's syndrome. Thus, these are limited studies which do not lend themselves to the broad conclusions suggested by Singh.

Indeed, the study of affective disorders in patients with Down's syndrome may clarify relationships

between neurochemistry and pathogenesis of affective disorders. However, epidemiological data is necessary to confirm a different incidence of affective disorder in Down's syndrome. Age and developmentally-matched control subjects, and further neurochemical information regarding young adult Down's syndrome patients without dementia, are necessary before the two putative findings may be connected.

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Dangerous Delusions: The 'Hollywood Phenomenon'

SIR: We agree with Drs de Pauw & Szulecka that both the prevalence of delusional misidentification and its relationship to violence are underestimated (*Journal*, January 1988, **152**, 91–97). We report two cases of a variant of delusional misidentification of the environment. The delusion consists of the belief that the patient's environment has been changed to a film or theatre set peopled by actors and in which the patient has a role to play. We propose the term 'Hollywood phenomenon' for the delusion, which is a symptom rather than a syndrome. It can occur with a typical Capgras phenomenon (Enoch & Trethowan, 1979) (case ii), and may result in violence (case i) or verbal hostility and non co-operation (case ii).

Case reports: (i) Mr A, a 22-year-old single Australian man with a history of two admissions for bipolar affective disorder, left Australia in the early stages of a manic episode. On arrival in the UK his condition deteriorated, with elevated mood, decreased sleep, excess energy, and accelerated thoughts. He recognised that he was relapsing and consulted a GP, who arranged an urgent out-patient appointment. Before that appointment he became convinced he was "an actor and that everything that was going on was a film" in which he was the main player. He stole a car which he deliberately crashed because "it was a stunt car and I was a stunt man who was supposed to crash it . . . it was rigged so I wouldn't get hurt". He was arrested and later assaulted the police surgeon with what he erroneously believed was a bottle of "harmless sugar glass", causing severe injuries. Mr A. claimed that he, the surgeon, and the police were all play actors and that his actions would have "no real consequence". Remanded in prison for psychiatric reports, he was intermittently violent in response to similar misidentifications until he became euthymic following medication. He was transferred after conviction on a Hospital Order, and on admission had insight into his previous delusions.

(ii) Miss B. exhibited both a Capgras phenomenon and a 'Hollywood phenomenon'. She was a single retired midwife in late middle age, living alone. She had had several admissions with a diagnosis of depressive psychosis or schizophrenia. On this occasion she was depressed with early morning wakening, psychomotor retardation, appetite and weight loss, and felt hopeless and worthless. She believed relatives were impostors and was verbally aggressive towards them. She believed that the hospital was a film set peopled by actors, the admitting doctor a film director, and that the purpose of the interview was to obtain a script for the film. While she struggled and was verbally hostile at attempts to detain her, there was no serious violence. She recovered fully after ECT.

Both cases involved affective illness without organic impairment. However, we do not believe that the 'Hollywood phenomenon' is specific for affective disorders, and would be interested to hear of other examples. We believe that like the Capgras phenomenon itself, the 'Hollywood phenomenon' is not uncommon, but under-reported. It differs from the superficially similar transient experience in derealisation in that it has a real, not an 'as if' quality, is enduring, and has all the features of a delusion, including the tendency to be acted upon.

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Calcium Therapy for Neuroleptic-Induced Extrapyramidal Symptoms

SIR: Lichtigeld (1965) hypothesised that hypocalcemia disrupts nerve cell function in the basal ganglia, causing extrapyramidal symptoms (EPS). Schaaf & Payne (1966) reported neuroleptic-induced EPS in hypoparathyroidism only when these patients are hypocalcemic. We report two patients