Lowering of cholesterol to reduce the rate of IHD has been associated with increased deaths from suicide and accidents (Engelberg, 1992). Elliott (*Journal*, June 1993, **162,** 818–825) suggests that the overall result of diminished brain cholesterol would be reduced 5-HT (serotonin) activity by increased pre-synaptic 5-HT uptake or decreased post-synaptic transmission.

A lowering of total plasma cholesterol on recovery from depression might therefore inhibit the facilitation of 5-HT transmission generally considered to be the result of antidepressant treatment, if it were mirrored in the brain, but the cholesterol content of HDL may be more important for brain function. In our study there was no significant change in HDL on recovery (mean ± s.d.: 39.9 ± 10.3 v. 38.5 ± 10.1%).

The activity of the 5-HT pump is increased by low cholesterol, as is Na/K ATPase activity (Papahajopoulos *et al*, 1973). Red-cell Na/K ATPase is known to be low in depressed patients, and to increase on recovery (Naylor *et al*, 1980). Further studies of cholesterol levels in depression with concomitant observation of 5-HT receptor function, optimally by provocation tests, would be of value in clarifying the relationships of cholesterol to 5-HT activity, Na/K ATPase activity, and the risk of IHD in depressive illness.

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*F. COULTER

*Dykebar Hospital*
*Grahamston Road*
*Paisley PA2 7DE*

F. M. CORRIGAN

*Argyll and Bute Hospital*
*Argyll*

B. MOWAT
E. R. SKINNER

*Marischall College*
*Aberdeen*

*Correspondence

## Eye-movement desensitisation to overcome post-traumatic stress disorder

SIR: I read with considerable interest the article by Spector & Huthwaite (*Journal*, July 1993, **163,** 106–108) because over the past two years or so I have been attempting to use this procedure in my clinical work. Unfortunately, examples of post-traumatic stress disorder usually of a severe nature are all too prevalent in the area where I practise. In general I follow the procedure outlined by Shapiro (1989) but would tend to give more than the 20 saccadic movements in each set.

Initially I experienced some resistance from the patients, so now I would generally give them a short discussion on the association between post-traumatic stress disorder and rapid-eye movement sleep as outlined in the paper by Ross *et al* (1989). It is a time-consuming procedure both for the patient and the therapist, but in my opinion it can form a valuable part in the treatment of a potentially crippling condition.

ROSS, R. J., BALL, W. A., SULLIVAN, K. A., *et al* (1989) Sleep disturbance as the hallmark of post-traumatic stress disorder. *American Journal of Psychiatry*, **146,** 697–707.

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J. B. WALSH

*South Tyrone Hospital*
*Carland Road*
*Dungannon*
*County Tyrone BT71 4AU*

## How well are 'cured' anorexic nervosa patients?

SIR: Windauer *et al* (*Journal*, August 1993, **163,** 195–200) rightly stated that there was little support for my earlier contention (Hsu, 1988) that "for those who recovered from anorexia nervosa, normal weight bulimia nervosa (not major depression) is the most common diagnosis". The preliminary data from our own long-term follow-up study also indicated that eating disorder not otherwise specified (ED NOS) is the most common diagnosis, not bulimia nervosa (Hsu *et al*, 1992). From the data presented by Windauer *et al*, I would think that ED NOS is also the most common diagnosis among those who no longer meet criteria for anorexia nervosa.

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L. K. GEORGE HSU

*Eating Disorders Clinic*
*University of Pittsburgh*
*3811 O'Hara Street*
*Pittsburgh, PA 15213–2593*

**Psychiatric symptoms in cannabis users**

SIR: Thomas (*Journal*, August 1993, **163**, 141–149) reviewed the evidence for 'cannabis psychosis' being valid as a diagnostic entity. We have investigated this issue empirically in a consecutive series of psychotic admissions which has been described elsewhere (Jones *et al*, *Journal*, January 1993, **162**, 65–71). The Present State Examination (PSE) was used to assess the psychopathology of 23 acute psychotic admissions who were cannabis positive on urinary screening, and 46 matched drug-free psychotic controls. Cases and controls were indistinguishable in terms of the prevalence of PSE syndromes. Moreover, the groups were also similar with respect to DSM–III–R diagnoses, ethnicity and socio-economic class, differing only, as expected, in terms of their histories of substance use.

We also examined the morbid risk of psychiatric illness in first-degree relatives, using maternal interviews and RDC–FH criteria, and Weinberg's shorter method of age correction. Cannabis positive cases had a significantly higher familial morbid risk of schizophrenia than the controls (7.1% v. 0.7%; odds ratio = 10.2; 95% CI 1.12–234, $P = 0.02$), but similar rates for other conditions.

We agree with the author's conclusion that there is little evidence to support the concept of a 'cannabis psychosis'. Furthermore, we propose that psychosis occurring in the context of cannabis use may be particularly likely in those with a genetic predisposition to psychotic illness; cannabis use may trigger a 'functional' psychosis in those with a psychotic diathesis, rather than producing a specific 'cannabis psychosis' *de novo*.

PHILIP MCGUIRE
PETER JONES
ROBIN MURRAY

*Institute of Psychiatry*
*De Crespigny Park*
*Denmark Hill*
*London SE5 8AF*

**Fluoxetine – induced mania in a patient with post-stroke depression**

SIR: Although mania or hypomania are well recognised side-effects of fluoxetine in depressed patients with 'functional' unipolar depression (Settle & Settle, 1984) or bipolar disorder (Lebegue, 1987), cases of mania induced by fluoxetine in patients with 'organic' mood disorders have not been reported. The following case report describes a patient with post-stroke depression (PSD), who developed mania three weeks after starting low doses of fluoxetine.

*Case report*. A 63-year-old right-handed woman suffered a sudden left hemiparesis and dysarthria in association with an ischemic infarction that involved the right corona radiata. Two months after the stroke, the patient became moderately depressed. Clinical features of depression included sadness, anhedonia, social withdrawal, inappropriate guilt, recurrent thoughts of death, and initial insomnia. On the 17-item Hamilton Depression Rating Scale her score was 16 points. She had no personal or family history of bipolar disorder, mania or any other psychiatric disorder.

The patient was started on a regimen of fluoxetine (20 mg/day), and over the following days her depressive symptoms became less severe. Three weeks after starting fluoxetine, she became euphoric and markedly intrusive and had pressured speech, as well as flight of ideas and increased activity and libido. She also exhibited inflated self-esteem, impulsive spending, and diminished need for sleep. She met DSM–III–R criteria for organic affective disorder, manic type, and on the Beck Mania Scale her score was 19 points (definite mania). Fluoxetine was discontinued. Lithium carbonate (800 mg/day) was started, and the patient experienced a rapid recovery.

Nortriptyline, trazodone and electroconvulsive therapy have all been reported to be effective and safe for treating PSD without the induction of mania (Robinson & Starkstein, 1990). Less is known about the efficacy and side-effects of fluoxetine and other selective serotonin-reuptake inhibitors in the treatment of organic mood disorders, although Morris (1991) has recently reported recurrent orgasmic sexual experiences induced by fluoxetine in a patient with PSD.

Since post-stroke bipolar affective disorder has mainly been reported after subcortical right-hemisphere lesions (Starkstein *et al*, 1991), it is possible that in our case fluoxetine may have precipitated a latent bipolar disorder. Therefore, further studies are needed to provide information on fluoxetine's safety profile in patients with PSD and right-hemisphere damage.

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