low dose". Dr Dabholkar raised the question of whether catatonia in itself is an indication for ECT. We hypothesise that ECT (or antidepressant medication) will be effective in treating hysterical catatonia and other conversion disorders only in cases where the conversion symptom serves as the 'masked' expression of an underlying depression (cf. Fisch, 1987).

In this regard, it should be noted that psychogenic pain disorders have been successfully treated with antidepressant medication (Walsh, 1983), and that electrically or chemically induced seizures have been successfully used in the treatment of "bizarre psychogenic movements" (Edwards, 1968), and in the treatment of psychogenic amnesia (Daniel & Crovitz, 1986).

In the case reported by Dr Dabholkar, it is plausible that the conversion symptoms may have served as an alternative to a major depressive episode; in other cases (Daniel & Crovitz, 1986) the development of conversion symptoms may even have served as an alternative to suicide. Since ECT has known anti-depressive efficacy (Gregory *et al*, 1985), its utilisation or an adequate trial of antidepressant medication may eliminate conversion disorders which have a depressive aetiology.

WALTER F. DANIEL
RONALD A. YEO
JANE E. SMITH

Department of Psychology
University of New Mexico
Albuquerque, New Mexico 87131
USA

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Before Mrs Thatcher?

SIR: The Survey Psychiatric Assessment Schedule (SPAS, section 1), as described by Bond *et al* (1980), asks the subject who was the Prime Minister before the current Prime Minister, counting this as one item of twelve in the assessment of cognitive disorder. We wondered, given Mrs Thatcher's 9 years of office, whether this question is now appropriate.

To assess this, we randomly asked 50 members of hospital staff (age range 18–65) who the Prime Minister previous to Mrs Thatcher was. The results were that of the 50 people asked, only 24 answered correctly, i.e. 52% of a presumably cognitively unimpaired population were unable to answer this question.

We would therefore suggest that this item of information is no longer appropriate for use in psychometric assessment.

N. M. J. KENNEDY
D. GASPAR

Hollymoor Hospital
Tessall Lane,
Northfield
Birmingham B31 5EX

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Lipid-Lowering Drugs

SIR: There have been two large studies in which drugs have been used to alter the concentration of various lipoprotein components in blood. Cholestyramine, a non-absorbable sequestrate of bile acid, effectively lowers low-density lipoprotein (LDL); similarly gemfibrizol, a drug related to clofibrate, elevates high-density lipoprotein (HDL) and reduces LDL. It appears that there is a causal link between increased LDL and coronary heart disease, whereas raised HDL does not increase the incidence of coronary disease and may even have a protective effect.

In 1987 the Helsinki Heart Study (Frick *et al*, 1987), a prospective study of 4000 healthy men, showed that treatment with gemfibrizol produced a significant reduction of mortality from cardiac death compared with a placebo group. Similar results were observed with cholestyramine in the American Lipid Research Clinics Coronary Primary Prevention Trial (Lipid Research Clinical Program, 1984).

What is interesting is that in both studies the total death rates for the treated and untreated groups were not significantly different. This was accounted for by the fact that in the treatment groups in both studies there was an increased number of deaths caused by violence, accidents, suicide, or intracranial haemorrhage. In the Helsinki study 33% of the patients who died in the treatment group died from accidents, violence, or intracranial haemorrhage, as opposed to