

normal communication attitudes, and an internalised locus of control) that predict long-term success following stuttering therapy. However, we have serious reservations concerning the validity of their predictors.

Firstly, treatment success was assumed when clients were speaking with a frequency of stuttering as high as 2% of syllables uttered during a telephone call made in the clinic to a stranger 10–18 months “after treatment concluded”. It is generally accepted that stuttering treatment success can only be determined through multiple assessments over time in a variety of beyond-clinic conditions using evaluations of speech performance and speech quality (Bloodstein, 1987). Drs Andrews & Craig’s criteria do not begin to approach this standard. The inclusion of up to 2% syllables stuttered, for instance, is not only incongruous with a successful treatment criterion (individuals may even require treatment for this level of stuttering), but it is also absolutely incompatible with their claim that those who met this criterion were “fluent” (see Table II of their paper).

Secondly, the authors claim that a single within-clinic telephone call has been “shown to be a valid and reliable measure of treatment outcome (Andrews & Craig, 1982)”, yet their 1982 report makes no such claim at all. Such a claim would be quite difficult to justify, because the disorder is renowned for its variability across speaking situations (Bloodstein, 1987). The problem with the external validity of this task is magnified because their therapy actually trained this particular task in clinic conditions (see Andrews *et al.*, 1987) but did not evaluate it outside of those conditions.

Thirdly, Andrews and his colleagues have indicated elsewhere (see Andrews *et al.*, 1987) that many of their clients participate in self-help groups after formal treatment ceases in order to regularly practice their ‘fluency skills’. If these subjects also participated in these groups then, arguably, treatment actually continued through to the point of “outcome” evaluation. Since the number of subjects engaged in these regular speech practice programmes was not indicated, it is possible that the “successful group” simply contained more members of these groups.

Fourthly, recent evidence demonstrates that clients’ responses to the communication attitudes scale used in this study are largely based on the clients’ current speech behaviour rather than their communication attitudes (Ulliana & Ingham, 1984). This factor may also operate in the clients’ responses to the locus of control behaviour scale (Ingham, 1989). Consequently, it is entirely possible that the three treatment goals predicted “successful treatment”

outcome because they yielded more speech performance information and not, as Drs Andrews & Craig claim, data on attitudes or perceived source of control.

Finally, a number of authorities have argued that a clinically valid outcome evaluation not only requires adequate beyond-clinic sampling but also appropriate measurement tools (Bloodstein, 1987; Boberg, 1981). Because Drs Andrews & Craig’s therapy programme may ultimately produce speech that is unnatural sounding, it is now recognised that this type of therapy needs to incorporate evaluations for speech quality and is compromised if it does not (Bloodstein, 1987). For all of these reasons we believe that Drs Andrews & Craig’s predictors of successful outcome have dubious experimental and clinical validity.

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Conversion Disorders and ECT

SIR: Dabholkar (*Journal*, August 1988, **153**, 246–247) reported that a 20-year-old male patient developed hysterical catatonia on two separate occasions following the accidental death of his father, and after villagers threatened to burn his house down, respectively. On the first occasion, his hysterical catatonia (and other conversion symptoms) were successfully treated with ECT, oral haloperidol (up to 5 mg per day), and “benzodiazepines and antidepressants in low dosage”. Five months later (during which time the patient discontinued his medications), the patient re-developed hysterical catatonia, which was successfully treated with ECT and “oral diazepam in

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.

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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.

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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 sequester of bile acid, effectively lowers low-density lipoprotein (LDL); similarly gemfibrozil, 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 gemfibrozil 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