the course of human societies, a particular social milieu throws up individuals who emerge as leaders at times conducive to change. The assumption that the influence of an individual on society is unidirectional is fallacious. A Germany fed on Nietzsche's philosophy and Wagner's music, unable to get over the World War I defeat, and reeling under intense unemployment and economic crises, could groom only a Hitler as its leader.

Cultural and economic factors that influence social processes do not always lend themselves to psychological explanations. Ryle makes the interesting distinction in his example: if a man returns from the market with his pigs unsold because the price was lower than he expected, the explanation is economic. However, if he returns with his pigs because he would not sell them at any price to a customer with a certain look in his eyes, the explanation might be psychological (Ryle, 1949).

I suspect that political decisions which have proved disastrous are explained away as resulting from individual psychological maladies. In the same issue of the *Journal*, Meyer Lindberg explains how German psychiatrists connived with the holocaust. To explain collective wrongdoing on the basis of individual pathology may be attractive as well as relieving, but it does not bring us any closer to the real explanations behind sociopolitical changes.

RYLE, G. (1949) *The Concept of Mind*. Middlesex: Penguin Books.

SWARAN P. SINGH

Department of Psychiatry
Queen's Medical Centre
Nottingham NG7 2UH

SIR: Your measured overview of the relationship between psychobiology and political institutions (*Journal*, July 1991, **159**, 19–32) correctly warns us against the overinclusive theories evolved by the psychohistorians and our easy tendency to pathologise political movements and leaders of whom we disapprove. The self-serving nature of the accusations (both Bush and Saddam recently calling each other 'insane') has done much to restrict the debate to the margins of both psychiatry and political theory: the psychiatrists now wary of retrospective diagnosis in the absence of its subject, the political scientists taking individuals as identical and interchangeable pieces in a social model which excludes biological variation.

The theoretical pitfalls in the area are great, and the mistakes bizarre. In a well publicised report, a group of American psychiatrists argued that Senator Barry Goldwater, then a candidate for the Presidency, was mentally unstable and were very nearly sued (Ballard, 1973). Similarly, the Press Council ruled that the *Sun* newspaper improperly published 'a psychiatrist's' opinion on the mental health of the Labour politician Tony Benn. On the whole, perhaps because psychopathology is a measure of 'difference', it is the innovators who tend to be perceived as insane, while the conservatives form the undifferentiated building blocks of the social theorist; Goldwater is (arguably) an exception.

Beyond the dangers of facile denigration and the political use of psychiatric categories, it does appear that one of the reasons for our refusal to venture very seriously into this area is the incommensurability of biological and social paradigms, the one articulating causal-linear models which presume events independent of our apperception of them, the other incorporating such characteristically human attributes as volition, identity, and memory.

It seems to me that one way of proceeding here is to avoid simplistic overall models of the interrelationship of biological difference with social difference, and to examine in certain limited cases how our two paradigms impinge on each other in a single instance. We can either start from biology – what social facts does thyrotoxicosis embody and represent? – or from sociology – what psychobiological characteristics seem to 'fit' a given institution? A useful idea may be Max Weber's notion of *charisma*. Originally used to characterise "the social recognition of certain extrasocially sanctioned qualities imputed to the person of the leader" (Weber, 1958), charisma has become a rather degraded notion, taking on, in the hands of psychohistorians and sociologists of religion, simply an assumption of perceived 'personality', the other members of the social group becoming merely passive and credulous vehicles (following Le Bon's or Freud's notions of 'the Crowd').

To tease out the biological–social dialectic, we have to look at the social reflections of the biological data (what are local notions of idiosyncrasy, madness or personality?) to get at the intervening (psychological) processes by which individuals dynamically form or reform social groups both through identification with those aspects of the sick innovator they shared but also through a structural opposition to those which they do not (Littlewood, 1991).

The notion of the innovator simply as a charismatic madman does not get us very far in examining how new political arrangements develop at certain times, while paradoxically it leaves the leader simply as one who shares and anticipates the personal dilemmas of his/her contemporaries and, solving

them for him/herself in eternal action in society, solves them for others (e.g. Erikson, 1958). Not only do we have to recognise the biological fact as such but simultaneously look at the local (and our) procedures which construct it as a biological fact.

The whole issue seems an interesting instance of current sociological concerns with the 'structuring' versus the 'structured', and one which reflexive developments in post-modernist theory have opened up again. It may well be that psychiatry will have some place in the debate, as predicted by the British psychiatrist W. H. R. Rivers in 1920.

BALLARD, R. (1963) An interview with Thomas Szasz. *Penthouse*, October, 69–74.
ERIKSON, E. (1958) *Young Man Luther*. New York: Norton.
LITTLEWOOD, R. (1991) *Pathology and Identity*. Cambridge: Cambridge University Press (in press).
RIVERS, W. H. R. (1920) *Instinct and the Unconscious*. Cambridge: Cambridge University Press.
WEBER, M. (1958) The sociology of charismatic authority. In *Essays* (ed. M. Weber). New York: Free Press.

ROLAND LITTLEWOOD

Department of Psychiatry
University College and Middlesex School of Medicine
Wolfson Building
Middlesex Hospital
London W1N 8AA

'Compulsive' water drinking in psychosis . . .

SIR: Crammer (*Journal*, July 1991, **159**, 83–89) outlines possible aetiological factors in polydipsia and water intoxication in psychotic patients. The association with psychotic symptoms suggests a common mechanism for both polydipsia and positive symptoms.

An alternative explanation in some cases could be that patients use water as 'self-medication'. As Crammer says, the majority of polydipsic patients experience no 'compulsion' to drink, but actively wish to do so. For example, Crammer suggests that drinking water may be anxiolytic, and would assuage a neuroleptic-induced dry mouth. Patients have also been described who enjoy the effects of intoxication (Cooney, 1989). (Indeed, the black market trade in anticholinergics, in some psychiatric hospitals, suggests a demand for cheap intoxicants!) A further possibility is that some of these patients have learned the trick of drinking to prevent subvocalisation, thus suppressing their auditory hallucinations (Forrer, 1960; Falloon & Talbot, 1981).

This suggests that we should not simply aim to contain or to control polydipsia. If there is reason to suppose that a patient's polydipsia might be intended as 'medication', we should consider interventions that could substitute for the patient's own attempts at treatment.

COONEY, J. A. (1989) Compulsive water drinkers. *British Journal of Psychiatry*, **155**, 266.
FALLOON, I. R. H. & TALBOT, R. (1981) Persistent auditory hallucinations: coping mechanisms and implications for management. *Psychological Medicine*, **11**, 329–339.
FORRER, G. R. (1960) Effect of oral activity on hallucinations. *Archives of General Psychiatry*, **2**, 100–103.

FRANCES KLEMPERER

York Clinic
Guy's Hospital
London SE1 9RT

. . . and mentally handicapped people

SIR: Bremner & Regan's article (*Journal*, February 1991, **158**, 244–250) on the prevalence of polydipsia in the mentally handicapped was an important addition to the literature. It has been our clinical experience that this very serious problem is relatively common in this population.

Drs Bremner & Regan conclude their article by suggesting that polydipsia be viewed as learned behaviour dependent upon a number of environmental factors. They emphasise the need to develop more effective interventions. We would like to call readers' attention to recent behavioural treatment interventions. McNally *et al* (1988) used a simple behaviour modification procedure to eliminate polydipsia in an autistic woman with severe mental retardation and a history of water intoxication. The intervention involved positive reinforcement of behavioural alternatives to drinking water and a mild punishment contingency. The procedure was effectively implemented by direct care staff with only basic behaviour-modification training. Polydipsia was eliminated in approximately six months, and gains were maintained after her discharge to a group home where she continued to reside polydipsia-free 18 months after placement (McNally & Calamari, 1989). More recently, Bowen *et al* (1990) have reported successful application of behavioural procedures to the treatment of polydipsia in a schizophrenic patient.

Although controlled studies are needed to evaluate further the efficacy of behavioural interventions for polydipsia, case reports encourage the use of these procedures either alone or as an adjunct to medical interventions.

BOWEN, L., GLYNN, S. M., MARSHALL, Jr, B. D., *et al* (1990) Successful behavioural treatment of polydipsia in a schizophrenic patient. *Journal of Behaviour Therapy and Experimental Psychiatry*, **21**, 53–61.