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.

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'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.

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. . . 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.

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Negative symptoms of schizophrenia

SIR: I read with interest Phillips *et al* (*Journal*, August 1991, **159**, 226–231) who reported that negative symptoms (as measured by SANS, Scale for the Assessment of Negative Symptoms) were prominent among both acute and chronic schizophrenics in China, and that during the acute phase they responded significantly to neuroleptic treatment. They therefore recommended affirmatively that negative symptoms "were an integral part of all stages of schizophrenia", and "should be understood more clearly by clinicians who must include their systematic evaluation in routine clinical examinations and consider their management in standard treatment protocols for schizophrenic patients". We do not, however, see how such recommendations may be clinically useful.

Negative symptoms were poorly defined clinical entities (Kulhara & Chadda, 1987) until standard rating scales, which vary among themselves as to what they actually measure (de Leon *et al*, 1989), became available to make them measurable entities for researchers. Despite this, we find it clinically difficult to discern the significance of negative symptoms among acute schizophrenic in-patients. Some of the items on the SANS, e.g. poor eye contact, decreased movement, blocking, unsatisfactory grooming and decrease in sexual or recreational interest appear to be multifactorial constructs that can occur, in particular, as secondary reactions to a relapse of positive symptoms. Another SANS item, namely inattentiveness, has actually been correlated to positive symptoms (de Leon *et al*, 1989). Further, a negative correlation between negative and positive symptoms previously found by Andreason has not in fact been replicated by others (de Leon *et al*, 1989).

Aetiologically, if negative symptoms during acute schizophrenia are strongly related or even secondary to positive rather than the 'residual' negative symptoms that we commonly recognise, then their good response to neuroleptics becomes understandable. Indirect support for this is provided by the positive correlation sometimes found between negative and positive symptoms (de Leon *et al*, 1989), and by Kay *et al* (1986) who showed that the genealogical and predictive implications of negative symptoms were phase-specific, with those occurring during the acute phase indicating a good prognosis. Therefore, even if phenomenologically similar, negative symptoms during acute and chronic schizophrenia may be aetiologically, therapeutically and prognostically different entities. If this is so, it becomes arguable whether it is useful to lump them under the same term and conclude, as Phillips *et al* did, that negative symptoms respond to neuroleptics and therefore "the schizophrenic type is mutable". Instead, as the study had an impressively large sample of patients, it might be useful to correlate the two apparently different types of negative symptoms (i.e. phenomenotypes) with genetic or neurobiological factors (i.e. biotypes) in order to establish their validity in the complex schizophrenic syndrome.

It is important to stress that negative symptoms are non-specific, and may occur in neurosis, personality disorder and even healthy individuals. In particular, there is a great deal of resemblance between negative symptoms and the behavioural manifestations of depression, so that depressed patients can actually score higher than schizophrenics on the SANS (Kulhara & Chadda, 1987). Even developers of negative symptoms scales have astutely recognised that for acute young schizophrenics, depression rather than negative symptoms is being measured (de Leon *et al*, 1989). Phillips *et al*, however, made no attempt at all to assess depression which might have confounded the evaluation of negative symptoms, despite the fact that electroconvulsive therapy was administered to 1/6 of the patients.

Clincians have, over the decades, been confused by the ever-changing systems of classifying schizophrenic symptoms suggested by research experts. Today, they consider residual negative symptoms usually after the acute psychotic phase is over because of their empirical management and prognostic import. While negative symptoms, as measured during acute schizophrenia, may be of interest to researchers despite their questionable status, clinicians should perhaps not be burdened with this until better proof is available. In any case, Kraepelin in *Dementia Praecox and Paraphrenia* was probably not referring to them.

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