clinical practices which can be easily monitored and evaluated.

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Delusional memory in schizophrenia

SIR: Buchanan (*Journal*, October 1991, **159**, 472–474) grapples manfully with the literature on delusional memory in schizophrenia but concludes that the overall significance of the phenomenon has yet to be clarified. To a large extent, the woeful lack of consensus between authors, highlighted by Dr Buchanan, as to what exactly constitutes delusional memory, is responsible for continued uncertainty about its diagnostic usefulness. As psychiatrists we have a tendency, when approaching phenomenological murky waters, to fall back on comparisons of our patients' experiences with those of the great early descriptive writers. In some ways this is constructive and may allow us to recognise and appreciate the significance of particular signs and symptoms, but where authoritative opinions on a particular phenomenon have not coincided, we are left in an uncomfortable situation. Such is the case with delusional memory. Before we can progress further in our exploration of this symptom and its diagnostic significance, we should perhaps stop thinking in antique terms, and start again from the simplest level by describing delusional memory experiences in terms of their basic components.

From the descriptions of what has been termed 'delusional memory' reviewed by Buchanan, we would suggest that three types of delusional memory are recognised by the following easy-to-understand steps in memory falsification.

Type 1. Simple memory falsification delusional memory. This corresponds to the PSE definition of 'experiences of past events which clearly did not occur but which the subject equally clearly remembers' (Wing *et al*, 1974). No delusional interpretation has been involved in the production of these memories, they are purely memories that have been fabricated

Type 2. True memory with delusionally attributed significance delusional memory. This has the two-memberedness of a delusional perception and is illustrated by the example of Kurt Schneider's patient who recalled that his fork when he was a child had a crown on it and that this signified he was of noble birth (Schneider, 1949).

Type 3. False memory with delusionally attributed significance delusional memory. This would be as for Type 2, except that, in the example given, the child's fork if still available would be found not to have had a crown on it. The distinction between Type 2 and Type 3 is generally based on the 'believability' of the events in the memory involved and not upon a hunt for an item of childhood cutlery!

Memory falsifications are not found exclusively in schizophrenia. Patients may confabulate to cover an amnestic disorder or just be telling lies. Nostalgia in healthy individuals involves a degree of memory falsification. Reduplicative paramnesia, like delusional memory, has come to mean many things, but as originally described was specifically a falsification of memory (Pick, 1903) and may indeed be confused with examples of delusional memory.

PICK, A. (1903) Clinical studies III. On reduplicative paramnesia. *Brain*, **26**, 260–267.

Schneider, K. (1949) The concept of delusion. In *Themes and Variations in European Psychiatry* (eds S. Hirsch & M. Shepherd). Bristol: Wright.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

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Biological basis of behaviour

SIR: Publicity in the media has recently been given to an article by Le Vay (1991) in which he showed that the size of a group of cells in the interstitial nuclei of the hypothalamus (INAH 3) was twice as large in heterosexual men as in homosexual men. The smaller size is similar to that found in women.

In 1986, Primrose showed that while only 15% (21/138) of adult males in a hospital for mental handicap had a lower than normal serum testosterone level, 73% of them (102/139) had an immature level of serum follicle stimulating hormone which is a hypothalamic-mediated pituitary hormone. In general, sexual expression in male mental handicap remains at an immature level, although testicular volume is usually of normal size. (In the series quoted, 20% or 74/377 had testes below normal size).

If such sexual behaviour patterns are biologically determined – and possibly from an early age – then this raises not only moral and legal issues for society, but also indicates the limits which can be expected from treatment of an individual, should this be considered necessary.

LE VAY, S. (1991) A difference in hypothalamic structure between heterosexual and homosexual men. *Science*, **253**, 1034–1037.

PRIMROSE, D. A. (1986) Sexual maldevelopment in male mental handicap. In *Science and Service in Mental Retardation* (ed. J. M. Berg), pp. 75–86. London and New York: Methuen.

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Enhancement of recovery from psychiatric illness by methylfolate

SIR: We were disappointed with Dr Procter's appraisal of the paper by Godfrey *et al* (1990) (*Journal*, August 1991, **159,** 271–272) which we felt was seriously flawed, but no criticism of the study was mentioned.

The study selected patients on the basis of borderline – low red cell folate (< 200 µg/l) although only 17 of 41 patients had actual folate deficiency (< 150 µg/l). Although the Hamilton Rating Scale for Depression was used as an outcome measure, there was no mention of baseline scores, threshold for inclusion, or the comparability of placebo-treated and methylfolate-treated groups. The overall clinical rating score, a crude four-point scale, was the major outcome measure, and no mention was made of the change in HRSD from baseline. A surprising finding that Beck scores correlated poorly with clinical outcome would suggest that the folate-treated patients were not as impressed by their folate supplementation as the researchers were.

Methylfolate was added to clinically determined 'standard' treatment, but the author's claim that depressed-folate and depressed-placebo patients received similar standard treatments is clearly not true. Lithium carbonate would not be considered by many to be a suitable first line out-patient antidepressant, meaning that six of 11 placebo-treated patients received an antidepressant (all tricyclics), while 11 of 13 folate patients did so (9 tricyclics, 2 monoamine oxidase inhibitor). Furthermore, there is no comment about dosages and whether these were similar.

By six-month follow-up, when the difference between the depressed groups was most significant, mean red cell folate of placebo patients was well in the normal range, but no comment was made as to how a normal value related to mental state.

We do not contest the view that the folate–mental disorder connection deserves further attention, but advise against overemphasising the importance of this study. We cannot agree with Drs Godfrey *et al* when they claim to have shown that replacement therapy with folate enhances the clinical response to standard psychotropic treatment or with their overstated view that the study 'provides the most compelling evidence yet' that folate and mental state are connected.

We are presently studying plasma methylfolate values in depressed patients receiving ECT, and preliminary results do not suggest a simple relationship with outcome. Twenty-seven patients with DSM–III major depression with melancholia or psychosis (mean HRSD score 33), who all received ECT, had lower plasma 5 methyltetrahydrofolate (5MeTHF) than 12 laboratory controls. Pre-ECT 5MeTHF values did not distinguish patients responding to ECT. Post-ECT folate levels were not significantly different between ECT responders and non-responders.

There are, of course, many mechanisms by which folate deficiency may influence depression (Abou-Saleh & Coppen, 1986). A metabolic connection rarely mentioned is that between folate and tetrahydrobiopterin (BH4), the essential cofactor for the formation of noradrenalin, 5-HT and dopamine. We have found evidence of impaired BH4 synthesis in severe depression which related to response to ECT (Anderson *et al*, unpublished). Tetrahydrofolate is required for BH4 synthesis, and a positive correlation has been found between plasma folate values and the urinary excretion of biopterin in euthymic patients attending a lithium clinic (Coppen *et al*, 1989). Further investigation of these associations is under way.

ABOU-SALEH, M. T. & COPPEN, A. (1986) The biology of folate in depression: implications for nutritional hypotheses of the psychoses. *Journal of Psychiatric Research*, **20,** 91–101.

COPPEN, A., SWADE, C., JONES, S. A., *et al* (1989) Depression and tetrahydrobiopterin: the folate connection. *Journal of Affective Disorders*, **16,** 103–107.

GODFREY, P. S. A., TOONE, B. K., CARNEY, M. W. P., *et al* (1990) Enhancement of recovery from psychiatric illness by methylfolate. *Lancet*, **336,** 392–395.

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