I have taken the time to argue for a cognitive neuropsychiatric methodology so that it may survive critics of Charlton's more general radicalism. Where I join with him on the barricades is the plea for a theoretically motivated psychological approach to psychiatric classification and in his belief that this will lead to a sensible and non-arbitrary classificatory system. Cognitive neuropsychiatry has first to demonstrate it can do this. It is early days and the results are mixed (Shallice *et al*, 1991; David & Lucas, 1993; David, 1994*b*). Cognitive probes of the sort envisaged by Charlton will need to be developed and tested before older methods are abandoned. Cognitive neuropsychiatry could turn out to be the saviour of psychiatric diagnosis, rather than its nemesis, leading it out of the current dark ages into a new era of rational enlightenment (Charlton, 1993).

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Method in Madness

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To both practitioners and outsiders, psychiatry is "a medical discipline long on disorders and short on explanations" (McHugh, 1995). Worse, the taxonomy of those disorders does not seem to be motivated in any principled fashion (Henderson, 1994); successive revisions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) are not always guided by the types of empirical evidence that would provoke classificatory change in other scientific domains. And worse still, there seems to be little consensus about what would constitute an explanation for any psychiatric phenomenon; it is difficult to seek the 'true' causes of disturbed cognition, affect, or praxis without having a moderately clear picture of what would count as an explanation (right or wrong!). In the absence of coherent modes of explanation (that is, theory), the collection and interpretation of data (whether clinical, epidemiological, or experimental) can only be piecemeal and capricious. That is, there are more constraints on how to support or disconfirm a theory than there are on how to revise a nosology.

As a pre-theoretical discipline, psychiatry is especially open to misdirection and the disastrous consequences that can follow from fad and fashion (McHugh, 1995). We can thus only applaud when

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Bruce Charlton supports the notion that normal information processing systems are the domain over which disorders of psychological function must be defined. How could it be otherwise? Does not the elucidation of a disorder of reasoning presuppose an account of how normal reasoning takes place? Does not the correct description of failures of reality-testing presuppose a theory of normal reality-monitoring (Johnson, 1991)? We were accordingly somewhat surprised when Charlton writes that "the concepts and language of cognitive psychology are unfamiliar to psychiatrists". Is this not like claiming that the concepts and language of normal human anatomy, physiology and biochemistry are unfamiliar to physicians?

It would be bizarre not to study disorders of, say, digestion against the background of the 'best theory' of the normal digestive system; it would be equally odd to study acquired disorders of language (the aphasias) without concern for the normal structure of language and the mechanisms whereby language is expressed and comprehended. A comparable point will hold with respect to phenomena that have traditionally fallen within the purview of the psychiatrist: the study of normal processes of belief-formation, their interaction with emotional state, and their (partial) determination by perceptual input must surely be the framework against which the causes, 'meanings' and consequences of delusion and hallucination are evaluated (Halligan *et al*, 1994). Likewise, the study of normal insight (Metcalfe & Shimamura, 1994), that is, the accuracy of one's judgements about the efficacy of one's own psychological functioning, must constrain the interpretation of 'lack of insight' as configured in (some) definitions of psychosis.

The failure of 'biological psychiatry' to which Charlton draws attention is indeed due to the misguided attempt to map directly between a syndrome and a neuronal (or biochemical) lesion. With respect to higher mental functions at least, the most striking aspect of 'traditional syndromes' is that they can invariably be fractionated. Thus, unless one really believes in an 'essence of schizophrenia', it would seem best to investigate how individual symptoms (e.g. the experience of thought insertion) can arise from an impairment to . . . ? Any one (or some combination) of the psychological mechanisms that have independent theoretical status in the explanation of normal cognition will fill in the blanks.

Examples where this type of model, derived from cognitive psychology and neuropsychology, has been explicitly used to explain psychiatric symptomatology include the delusional misidentification syndromes (Young *et al*, 1993), alien control (Frith & Done, 1989), thought echo (David, 1994), and aspects of autistic disorder (Baron-Cohen *et al*, 1994). As Persons (1986) reflected, there are clear advantages to "studying psychological phenomena rather than psychiatric diagnoses". It is true that patients will often present with clusters of symptoms, and thereby predispose the examiner to reify those clusters that occur frequently. But this reification may be one of the many illusions of reason to which the normal mind is subject (Piattelli-Palmarini, 1994). It certainly cannot be the case that statistical clustering in itself justifies the postulation of a disease-entity. And especially not so in psychiatry where the diagnostic labels often seem to suggest that the 'mad' are a different species. On the contrary, it may be wiser to assume that they are just like ourselves, only more so. And to let that insight guide the investigation of why they sometimes *seem* so different from us.

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