whether the changes are in degree or kind, and will demonstrate their relationship to cognitive function.

As to the question of the significance of these neuropathological changes for the continuity hypothesis, logically, even if the changes observed differed in degree rather than kind in both states (senescence and dementia), this would not necessarily imply that the processes of ageing and dementia were continuous, since some separate biochemical factor may be responsible for accelerating 'natural' neuropathological change. Thus it may be unwise to immediately accept the 'merging' of neuropathological change as strong evidence for the continuity of ageing and dementia.

Finally, Jorm & Henderson (1985) argue that since Alzheimer's disease can be graded into mild, moderate and severe, support is given for the proposition that dementia is dimensional. This argument is weak, since it is quite possible to have a disease that can be graded in severity, without the necessary implication that the disease is continuous with a normal developmental process.

Although the continuity hypothesis is not the major focus of Jorm & Henderson's article, the issue is critical to an understanding of the decline of intellectual efficiency in old age, and without further research the acceptance of dementia "itself being a dimensional disorder" is unwarranted.

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Conversion, Paranoia and Brain Dysfunction

DEAR SIR,

I read with interest Shalev & Munitz's case of conversion paralysis with paranoia (*Journal*, February 1985, **148**, 198–203). In light of the positive response of both symptoms to haloperidol, is it possible that the paralysis may have in some way been mediated by dysfunction of the extrapyramidal system? And if so, what of the psychosis itself?

The basal ganglia are not infrequently implicated in neurologic disease states which affect complex aspects of emotion and behaviour and which may produce delusional and psychotic symptomatology, as recently reviewed in this journal by Cummings (1985) and reported elsewhere by others (Bowman & Lewis, 1980; Laplane *et al*, 1984). That the paralysis occurred in the right hand may relate to reported observations of paranoid-like syndromes occurring with organic left-hemisphere injury (Leftoff, 1983), as well as to findings of lateralised left-hemisphere neuropsychological deficits in psychiatric patients with paranoid symptomatology (Flor-Henry, 1979).

Reference to the brain in conceptualising somatoform disorders—indeed, psychiatric disorders in general—need not imply that we look for (or hypothesise) occult organic lesions in every case; rather, it is possible that any number of constitutional/developmental factors may bias the nervous systems of certain individuals to respond to stress with somatising symptomatological patterns. This type of stress-diathesis model is already utilised for some classes of psychopathology. Attention to the neuropsychological dimension of conversion and other somatoform disorders might help clarify their aetiology (Miller, 1984).

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