establish the prevalence of psychiatric disorders in an urban Aboriginal population in Perth. With the assistance and co-operation of the local Aboriginal Medical Service, a pilot study was recently completed. Following discussion with Medical and Aboriginal Welfare Officers, brief training sessions were undertaken to familiarise officers with the SRQ and the questions involved. All patients attending the service during one week were asked to complete the questions and were assisted by an Aboriginal Social Work student who kindly volunteered her services for the week.

Thirty patients declined to co-operate because they were either too sick, could not read, were under the influence of alcohol or felt the questions were too personal. There were 109 completed SRQ's comprising 71 females and 35 males. Taking advice from previous reports, those with a cut-off score of 10 or more positive responses to questions were regarded as being psychiatric cases. Using this criterion, 34 (31%) of cases were identified. We were particularly interested in psychotic symptoms and found that 36 patients gave one psychotic response, 22 gave two, 4 gave three and another 4 gave four psychotic responses.

The results were discussed with those who are familiar with the difficulties and stresses of Aboriginals attempting to assimilate into white society. The opinion was expressed that it is inappropriate of Western psychiatrists to regard all these responses as necessarily being pathological. Such phenomena as premonitionary dreams, telepathy, auditory hallucinations and awareness of ancestor spirits are all an intricate part of Aboriginal culture and belief systems.

The commonest psychotic response was an affirmative to the question, "Are you a much more important person than most people think?". The frequent "yes" answer to this question was thought to be an indication of what is perceived as resentment of the prevailing low opinion of Aboriginals held by white Australians. It is common knowledge that Aboriginal parents frequently reassure their children and adolescents that they are much more important than they think themselves to be.

In conclusion, we found that the SRQ was a suitable instrument for use in primary care situations but would advise caution in interpreting certain responses as indicating psychiatric pathology when they were in fact more likely to be a cultural response to anomie and demoralisation.

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Diagnostic Criteria for Dementia in DSM-III

DEAR SIR,

Jorm & Henderson (*Journal*, 1985, **147**, 394–399) argue that the disorder of dementia is continuous with normal senescence because the Mini-Mental State (Folstein *et al*, 1975) is distributed monotonically in community samples, neuropathological changes are found in both normal and demented people, and the disorder is usually described in terms of severity (pp. 394–395).

In the first of these arguments, Jorm & Henderson suggest that the failure to find a hump at the lower end of a distribution of cognitive test scores in the elderly population indicates the absence of a discrete disorder. Such evidence does not, however, rule out the possibility that the cognitive changes of dementia are qualitatively rather than quantitatively different from those of normal ageing because non-specific tests such as the Mini-Mental State may not measure the precise deficits which distinguish demented and elderly subjects. When more specialised tests are used, patients with dementia demonstrate serious specific impairments which suggest that they fail to use semantic information in memory encoding, and in this respect differ qualitatively from their peers (Weingartner *et al*, 1981). Findings such as those by Weingartner *et al* strongly suggest that quite different mechanisms (and hence causation) are involved in benign senescent forgetfulness and the memory deficits associated with Alzheimer type dementia.

In their second argument, Jorm & Henderson (1985) maintain that the neuropathological changes seen in Alzheimer patients 'merge' with those seen in normal elderly people, and such "merging" supports a dimensional relationship between Alzheimer's disease and normal ageing. Two issues need to be addressed here: first, whether pathological changes are similar in kind or degree in normal and demented groups, and secondly, what the significance of these neuropathological changes are for the continuity hypothesis. With respect to the first issue, contrary to Jorm & Henderson's position, some recent researchers (e.g., Keller, 1984) argue that the neuropathological changes in Alzheimer's disease differ in both location and in density from those observed in normal ageing (p. 41), suggesting a discontinuity, i.e. the superimposition of a disease process on senescence. Since intellectual performance appears to be relatively intact in brains which at postmortem show a certain degree of neuropathological change, a threshold effect is strongly indicated. Namely, once a certain level of neuropathology is reached, serious cognitive deficits appear. Presumably more research will clarify

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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