British Journal of Psychiatry (1985), **146,** 211–216

Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all **references in Journal style.**

ATHEROMA, INFARCTION AND DEMENTIA: NEED FOR A NEW NAME?

DEAR SIR,

Wright & Whalley (1984) have commented that atherosclerosis of the cerebral vasculature is a variable feature in multi-infarct dementia and often appears to be insufficient to explain the observed degree of cerebral infarction and dementia.

We report 6 patients with "vascular" dementia (VD) diagnosed according to the Ischaemia Score (Hachinski *et al,* 1975) and 23 patients with cerebrovascular disease (CVD), 10 of whom had mild cognitive impairment not amounting to dementia. On computerised tomography, 3 of the 6 patients with VD had multiple lacunar infarction but no large, non-lacunar, hemispheric infarction. Thirteen of the 23 CVD patients had sizeable, non-lacunar hemispheric infarction but none had multiple lacunar infarction.

In terms of *quantity* of overall infarction, the CVD group could be deemed to be the more seriously afflicted but the cognitive impairment, when present, was non-specific and relatively mild. This would suggest that non-lacunar infarction by itself, especially when it is confined to one hemisphere, is not sufficient for dementia or even serious cognitive deficit. It also emphasises the important association of multiple lacunar infarction with cognitive dysfunction amounting to dementia. It has been shown (Ladurner *et al,* 1982) that bilateral infarction, especially in the thalamic region, distinguishes between demented and non-demented patients with CVD.

When the CVD group was considered in terms of carotid artery disease, bilateral disease on angiography was comparable in those with and without cognitive dysfunction, but patients with non-lacunar hemispheric infarction showed significantly greater cognitive impairment.

These results confirm that the *type* of infarction and its bilateral location are important in relation to dementia associated with CVD; that cognitive dysfunction is related to infarction rather than the extent of arterial disease; and support the contention that the relationship between dementia, overall infarction and arterial disease is a rather tenuous one.

The unsatisfactory nature of the nomenclature is also pinpointed. As dementia and even lesser forms of cognitive dysfunction seem related to cerebral lesions rather than arterial disease, terms like "arteriosclerotic" or "vascular" dementia would seem to be not quite appropriate. On the other hand, multiple lacunes are not always demonstrable. Therefore, there would appear to be a need for a new nomenclature and classification for this group of dementia, and one that would reflect its heterogeneous nature as well.

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LANGUAGE IN DEMENTIA

DEAR SIR,

Recent articles have used data on language disorders in senile dementia to hypothesise on the nature of the underlying deficit (Davis *et al, Journal,* April 1984, **144,** 383–386) or improve diagnostic acuity (Skelton-Robinson *et al, Journal,* August 1984, **145,** 168–171). Many specialised journals pursue these topics with little attention to some of the pitfalls accompanying word recognition tests. A

common problem involves definitions. For example, the act of crawling by a dystrophic person does not make crawling a pathological trait. The act is a necessary reversion to a simpler mode of locomotion. Likewise, memory failure in senile disorders is revealed by circumlocution but circumlocutory speech itself is not abnormal. In fact, circumlocution proves that only a name and not a memory is lost. These signs of dementia appear with the loss of higher, more complex functions, unmasking simpler, surviving neurolinguistic mechanisms. The memory tricks senile individuals employ are wrongly labelled pathological when the same tactics in normals are called mnemonic aids. Indeed, portmanteau words in name lists (e.g. horseshoe) are actually constructed by circumlocutory invention. Certain colour terms (e.g. orange) represent deliberate adjective/noun blurring to serve mnemonic convenience.

Important clues to normal brain function may be overlooked if the data on language disorders are wrongly interpreted. We may ask if the naming of orphans in alphabetical sequence in Charles Dickens' *Oliver Twist* is evidence of indifferent efficiency or compensation for a defective memory? What we regard as learning tricks may be partly imposed on the process of memorisation by the brain itself. The neuronal substrate for memory must be far older than the mammalian cortex and it should be expected to have properties that shape the storage of inflowing data. Serially arranged elements such as the days of the week, the colours in the solar spectrum, and number systems seem to be more strongly embedded in the memory than loosely linked items. This is probably because a lost item can be recovered from adjacent elements by some rules of continuity. It is probably safe to assume that sequencing is a primitive neurolinguistic device for grouping similar data in close synaptic proximity. If data are stored in a manner to facilitate recovery of the message with minimum loss, then we are following a process akin to the error-resistant coding used in computer science (Goode & Machol, 1957; Turner, 1968). The error-resistant Gray code is designed so that adjacent sequence items differ in the smallest possible manner. If each item is represented by a string of symbols (or neuronal pulses), and one symbol is lost during decoding, then the message error can still escape being total nonsense. A message string for the number "6" might be decoded after an error as "7", by use of a Gray code, and this may be allowable if we are trying to remember a birthday of a friend. If "6" prints out as "16" or "61" the number is no longer of value. The amino acid code for proteins and the nucleic acid code for the genetic material in the cell appear to be based on a molecular Gray code (Swanson, 1984). We may be missing similar insights on brain and memory functions by labelling inherent qualities as pathological traits rather than glimpses of normally functioning primitive mechanisms substituting for impaired higher language processes. The meaning of circumlocution, repetition, echolalia, etc. may need reexamination.

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REVERSIBLE DEMENTIA AND DEPRESSION

DEAR SIR,

Several recent letters have commented on our article 'Criteria for Diagnosing Reversible Dementia Caused by Depression: Validation by 2-year Follow-up.' We agree with Dr Colgan (*Journal,* August, 1984, **145,** 213–214), and stated in the paper, that a careful clinical examination is the best means for properly recognising such patients. As we noted, however, several studies have demonstrated that patients have been misdiagnosed as irreversibly demented rather than depressed in the past. We also agree with Dr Mahendra (*Journal,* August, 1984, **145,** 213) and noted in the paper that underlying brain dysfunction could be giving rise to the symptoms of mood disorder and cognitive disorder. We cited Post as having suggested this 15 years ago.

Doctors Spagnoli and Williams (*Journal,* September 1984, **144,** 339–340) question our use of the term "predictive value". This term is usually applied to assess screening tools and is affected by the prevalence of the disorders being studied. We used the term in a more constricted way to demonstrate the relationship between certain depressive symptoms and the reversible dementia syndrome of depression as compared to dementia. We wanted to emphasise that most symptoms used to identify depressive illness do identify persons with cognitive disorder who improve cognitively when the depression is treated. We did not