EDITORIAL

And Moses was an hundred and twenty years old when he died: his eye was not dim, nor his natural force abated. (Deut 34:7; King James version)

Vascular Cognitive Impairment

DT Gilbert

Dementia, a syndrome of declining higher cognitive functioning, found in approximately 10 per cent of adults 65 years and older, and 50 per cent of adults older than 90 years. Alzheimer's disease (AD) accounts for 50 to 80 per cent of all dementing illnesses. Vascular dementia comprises 15 to 20 per cent of dementia and in some clinico-pathological series is the second commonest form of dementia, often coexisting with Alzheimer's disease. Other degenerative conditions such as Parkinson's disease with dementia (PDD), dementia with Lewy bodies (DLB) and frontotemporal dementia (FTD) account for 10–15% of dementia. Less than 10 per cent of dementias are caused by reversible conditions *eg* Vitamin B_{12} deficiency, hypothyroidism (1–3).

The criteria for the diagnosis of dementia, in general, and vascular dementia, in particular, continue to evolve. When Kraepelin wrote the 1910 edition of his influential textbook of psychiatry, he classified Alois Alzheimer's first patient (aged 51 years at diagnosis) as a presenile dementia and wrote that the main form of senile dementia was an "atherosclerotic dementia". With the 1950s and 60s came the suggestion that senile dementia be separated according to aetiology and clinical features into two groups – vascular and degenerative – with most patients with senile dementia recognized as actually having the "degenerative" changes of AD rather than vascular disease of the brain.

In a 1974 Lancet review article (4), Hachinski *et al* emphasized that "when vascular disease is responsible for dementia, it is through the occurrence of multiple small or large cerebral infarcts (multi-infarct dementia)". The term 'multi-infarct dementia' (MID) then became almost synonymous with dementia due to cerebrovascular disease, implying incorrectly that multiple brain infarcts are the only cause of vascular dementia. It is now recognized that vascular dementia can also result from single strategic infarcts, non-infarct ischaemic lesions affecting the white matter and basal ganglia, chronic hypoperfusion and haemorrhagic stroke, and includes autoimmune aetiologies such as systemic lupus erythematosus (5, 6).

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Numerous clinical criteria have been proposed for vascular dementia with differing emphases as to what is "vascular" or even what defines a dementia (7–9). Studies comparing these clinical criteria have demonstrated a low correlation. The term vascular cognitive impairment (VCI) was proposed as an attempt to better identify individuals with significant cognitive difficulties, but not necessarily memory dysfunction, arising from vascular causes (9–11). Vascular cognitive impairment includes three subtypes: vascular dementia, AD with a vascular component (mixed dementia) and impairment that does not meet dementia criteria (referred to as vascular cognitive impairment, no dementia or CIND).

The DSM-IV (12) and ICD-10 (13) criteria for dementia require short-term and long-term memory impairment as essential features. Cerebrovascular disease, however, can produce impairment in organizing, sequencing and abstracting – executive dysfunction – with little or only minimal effects on memory (14). The VCI concept in the evaluation of cognitive dysfunction highlights the fact that not all cognitive impairment is "dementia" with memory dysfunction. It allows for the identification of patients who are in the early stages of cognitive deterioration before memory disturbance affects functional abilities or those patients who have disturbances in other aspects of cognition apart from memory *eg* planning, abstraction, language and attention.

Dementia is an independent risk factor for mortality in patients with ischaemic stroke with some studies suggesting a three to fourfold increased risk of death relative to non-demented patients (15). In one study, vascular cognitive impairment without dementia was the most prevalent form of vascular cognitive impairment among those aged 65 to 84 years. Mortality and institutionalization were significantly higher for those with vascular cognitive impairment than those in persons who had no cognitive impairment (11, 16). In view of these associations, management decisions regarding vascular risk factors must include the impact on cognitive dysfunction. Early evidence *eg* suggests that treatment of hypertension in the elderly may be quite successful in reducing incident dementia (17, 18). Future research into the benefits of *eg* carotid endarterectomy, coronary bypass procedures or even congestive heart failure management must include morbidity statistics as they relate to VCI.

With an ageing population and rising prevalence of vascular disease in Caribbean countries, increasing numbers

of individuals are at risk of cognitive impairment. The economic and social burden associated with management of dementia is significant (14, 16). Early recognition of the cognitively impaired elderly would be advanced by understanding that (a) declining cognitive function in the elderly represents disease and not normal ageing (b) cognitive impairment is not only memory dysfunction (c) bedside "dementia" screening tests which emphasize memory dysfunction, when used alone, will underestimate the true extent of cognitive impairment and (d) vascular dementia and vascular cognitive impairment, the end result of multiple aetiological factors, represent a conceptual advance over "atherosclerotic dementia" or "multi-infarct dementia" but remain too poorly defined and must become linked to clear causal factors to change and improve intervention strategies in the management of cognitive dysfunction. Have we come full circle? Is Kraepelin's "atherosclerotic senile dementia" of 1910 a "vascular Alzheimer's disease" – a vascular dementia?

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