dementia with Mini-Mental scores between 0 and 14/30 without amyloid deposits and with neuritic Braak stages I or II. These were found among 150 consecutive cases of the Vienna Longitudinal Study on Dementia,¹⁵ in which elderly patients underwent prospective clinical and psychometric examination at least every six months. The six patients, only three of whom had a history of previous stroke, are summarized in the Table. There were four males and two females aged at death between 67 and 93 years with duration of dementia between 3 and 7 years. Clinical diagnosis was probable AD in four, VD and Parkinson's disease in one, and VD in one. At autopsy, all brains except one showed severe atherosclerosis of the cerebral arteries, with hypertensive microangiopathy in four. All brains except one showed rather small left or bilateral infarcts in the medial thalamus. Severe lacunar state in basal ganglia or brainstem were seen in two. Additional old putaminal hemorrhage and left PCA infarction were seen in one each. None of these brains showed amyloid deposits/angiopathy or neuritic plaques; hence, all current neuropathologic AD criteria were negative. These data confirm the notion that VD unaccompanied by neuropathologic evidence of AD is rare. As demonstrated by our cohort, VD may often be related to small or medium-sized strategic infarcts mainly involving the thalamus without gross infarctions in cerebral hemispheres.¹⁶

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Reply from the Authors: We agree with Drs. Moroney and Desmond that our study was not carried out using a population-based sample. It was not, however, a referral sample focused on AD as they suggest. The neuropathologists at 10 medical centers were asked to submit "any" cases with progressive dementia clinically diagnosed as AD or multi-infarct dementia in which the autopsy revealed only cerebral infarction. CERAD criteria were not requisite for our study. Only one of our six cases, in fact, met CERAD criteria for the clinical diagnosis of probable AD.

As pointed out, the definition of progressive dementia may be subjective, but cases included had cognitive impairment sufficient to interfere with social and occupational function. Drs. Moroney and Desmond appropriately note that the neuropathology assessment was limited in several cases to one hemisphere. Although this approach may underestimate the extent and nature of vascular pathology, unilateral examination is adequate to assess AD changes. We agree that prospective clinicopathologic studies of patients with dementia and evidence of cerebrovascular disease are needed. Nonetheless, we believe that our study, despite inherent methodologic limitations acknowledged in our report, highlights the relative infrequency with which vascular dementia presents to neuropathologists at major medical centers. We were pleased to learn that Dr. Jellinger's extensive experience validates our findings.

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Correction

In the article "What are the obstacles for an accurate clinical diagnosis of Pick's disease? A clinicopathologic study" by Litvan et al. (*Neurology* 1997;49:62-69), N. Sastry's name was misspelled. We apologize for any inconvenience or confusion this may have caused.

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Correction

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