Letters to the Editor

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What Is a Lacune? Dogged déjà vu doggerel

To the Editor:

“Then you should say what you mean,” the March Hare went on. “I do,” Alice hastily replied; “at least—at least I mean what I say—that’s the same thing, you know.” “Not the same thing a bit!” said the Hatter. “Why, you might just as well say that ‘I see what I eat’ is the same thing as ‘I eat what I see’!”

Alice in Wonderland
—Lewis Carroll

Dr Wardlaw’s plaintive query documents 21st century survival of clinical science confusion driven by hopelessly specious terminology: “lacune,” “lacunar infarct,” “lacunar stroke,” “clinical stroke syndrome with typical symptoms and signs referable to a small subcortical or brain stem lesion,” “clinically evident lacunar infarct,” “clinical stroke syndrome of lacunar type where the underlying lesion is an infarct on brain-imaging,” “3 to 15 mm cerebrospinal fluid (CSF)-filled cavities in the basal ganglia or white matter, frequently observed coincidentally on imaging in older people, often not clearly associated with discrete neurological symptoms.” The sexy French L-word never did specify etiology or pathogenesis. Neither the arbitrary discrete neurological symptoms. 

The ancient term “stroke” specified sudden negative symptoms, often catastrophic; clinicopathologic correlation most often identified an anatomically pertinent brain hemorrhage or infarction. Before the advent of CT and MRI, Miller Fisher invented the lacunar concept to deal with his ultimate 65 to 70 stroke syndromes. Early on he further specified that the infarctions were the result of “lipohyalinosis:” arterial lesions caused by vascular hypertension. Thence his power “proposition that small artery disease tends to occur where it gives rise to symptoms [his descriptive variations] rather than being distributed by chance.” Most significant were his later pathological observations that 22 new “lacunar infarcts” included only 4 with lipohyalinosis. Four empty-vessel cases were presumed to represent lysed emboli, rather than spasm or other mechanism. The other fourteen cases included “stenosing atheroma with superimposed thrombus (5); stenosing atheroma (5); mural atheroma of the basilar artery (2); microaneurysm (1); and inconclusive (1).” By Authority of Inventor, Lacunar pathology is not consistent. It is certain that many “lacunes” are not associated with standardized syndromes or any symptoms at all. In 1993 a group of investigators (TOAST) set forth a protocol for stroke therapy with the worthy premise that “determining the cause of stroke does influence choices for management” because “the etiology of ischemic stroke affects prognosis, outcome, and management.” Their only tested drug, danaparoid, had no clinical value, but their stroke classification system survived to become the standard research protocol. Unfortunately, TOAST specification of cause lacks both replicable objective justification and pathological confirmation. Cause for each case is only the online majority vote of the clinician investigators. Statistical guess and attractive treatment are confusing values: for example, risk of atrial fibrillation/embolization inevitably trumps competitive infarction ideas. Regardless of putative etiology, the logical expectation in any population of surviving stroke patients is that most prevalent brain lesions are, indeed, small. An extensive stroke literature review found no significant differences among risk factor profiles between lacunar and nonlacunar infarcts. Hence the heuristic power of the lacunar label in stroke now matches the lost cause value of miasma vapors in the history of malaria infection.

Absent the lacunar prejudice regarding pathology or pathogenesis, every brain lesion may must be identified independently by site, volume, temporal course, clinical correlation, if any, and even histopathology.

Leukoaraiosis is the scholarly Greek title invented long ago by Drs Hachinski, Potter, and Merskey in order to deal critically with the new pathological entity, uniquely brought to discovery by clinical CT and MRI. [Wardlaw’s generic label, WML, is not a proper synonym.] They noted that the condition is “associated with cognitive impairment and to some extent, with vascular disease. Possible causes of white-matter changes and their relationships to Alzheimer disease are examined, and it is argued that a neutral term, exact enough to define white-matter changes, sufficient as a description or label, and demanding enough to require precise clinical and imaging descriptions is needed.” I believe that their invitation for proof of pathogenesis is still open, although popular repetition seems to justify the factoid presumption of obscure vasculopathy.

Wardlaw’s devastating critique of the presumptions, methodology, and conclusions of the LADIS study merits acceptance, with the footnote that none of the LADIS patients suffered the necessity of pathological evaluation.

William M. Landau, MD
Department of Neurology
Washington University School of Medicine
Saint Louis, Mo

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