Concluding Remarks

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Clinical and pathological studies of lacunes returned to the fore in the modern neurological era after the pioneer papers of Fisher and, particularly, after the advent of neuroimaging techniques. Lacunes account for about 19% [1] or 21% [2] of a series of consecutive patients with stroke. Bamford and Warlow [3] in a review which appeared some months after this meeting took place, raised some relevant questions about lacunes. I believe that this study deserves some comment in this section instead of the usual summary of data presented at the meeting, especially with a view to future work on lacunes.

1. "The clinicopathological correlation of lacunes is far from having been satisfactorily assessed." "The assumption that small deep infarcts seen on CT scans represent lacunes requires further pathological confirmation" [3].

The correlation between clinical findings and the usually delayed pathological evaluation of the brain is fraught with obvious limitations. However, the problem in the field of lacunes seems to concern more with questions of terminology. In fact, a lacuna is a cavity, a pathological lesion which represents the evolution over time of an ischemic infarct, or rather of a small deep ischemic infarct or, if you wish, a lacunar infarct. If you examine the brain at the onset of clinical signs or symptoms, or in the few days after onset, the hypodense area revealed by the CT scan surely cannot be related to a lacuna but to an ischemic infarct of restricted size, which usually evolves into a cavity or, more rarely, into glial mesodermal scar tissue [4].

The second statement, concerning the CT-pathological correlation, is strictly related to the same conditions: the small deep hypodense area revealed by the CT scan is not a lacuna but a small infarcted area. The utmost care must be taken in assuming pathological or CT-clinical correlations since: (a) more than 20% of CT findings cannot be correlated with clinical features and they should be considered as asymptomatic lacunes, and (b) 26% of lacunar syndromes show normal CT findings. However, patients with a typical lacunar syndrome assessed shortly after the stroke can confidently be diagnosed as having small deep or lacunar infarcts, providing that the ancillary examinations exclude other less usual diseases. The use of MRI makes the diagnosis clearer because of the greater sensitivity of MRI to small deep infarctions [5].

2. Finally, lacunes and microinfarctions are found in other clinical pictures: reversible ischemic attacks and some other, less usual, diseases such as lacunar dementia andBinswanger’s disease. In our experience, about 11% of cases with transient neurological deficit are sustained by lacunes (although the Harvard Stroke Register would suggest that this figure should be increased to 23%), which is showing that lacunes are responsible for a considerable number of cases of transient neurological deficit. The role of lacunar infarcts in causing dementia is more debatable and conflicting views have been expressed in neuropathological studies [6]. The pathological lesions leading to subcortical arteriosclerotic encephalopathy are probably due to ischemia, while lacunes and larger infarctions may be an associated pathological feature.

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References