coexistent respiratory disease. We were concerned that if patients were only able to hold their breath for a relatively short time, and if there was any delay in the rise of velocity during breath-holding in some patients, this might lead to an underestimation of cerebrovascular reactivity. However, as described in our article, when we compared results from subjects who were able to breath-hold for a longer time (>27 seconds) with those who could breath-hold only for a shorter time (<27 seconds), the correlation with the "gold standard" method of measuring cerebrovascular reactivity (that of Ringlestein et al) was the same for the two groups (Spearman's \( r = 0.65 \) and 0.77 respectively; NS).

Although the response to hyperventilation (hypocapnia) is frequently measured, recent evidence suggests that this does not significantly improve the detection of abnormal cerebrovascular reactivity. Ringlestein et al found that hypocapnia alone separated hemodynamically significant lesions as well as combined stimulation with hypercapnia and hypocapnia. We have reviewed our results. We analyzed measurements of reactivity in the middle cerebral artery above 50 angiographically determined carotid stenoses (ranging from 10% to 99%). A correlation was made between degree of carotid stenosis and cerebrovascular reactivity as measured by hypercapnia alone, hypocapnia (hyperventilation) alone, and by both stimulations combined. The correlation was only slightly higher when hypocapnia was also tested than when response to hypercapnia alone was measured (Spearman's \( r = 0.64 \) versus 0.60), whereas the correlation with hypocapnia alone was much less (\( r = 0.29 \)). Therefore, our results support the findings of Ringlestein that the response to hypercapnia is most important, and we would not routinely include the use of hyperventilation with our breath-holding test.

We see the main use of the breath-holding test as a simple screening test that can be performed quickly on the ward or on outpatients with a portable transcranial Doppler machine. However, as mentioned in our paper, it requires further validation before being used as a definitive test. Currently, we would investigate abnormal results further with a reactivity test using administered carbon dioxide. With reference to deciding in which patients cerebrovascular reactivity is pathologically reduced, our data and those from positron emission tomography suggest that in patients with carotid artery disease there is a graded range of abnormality from mild to severely impaired and that any absolute definition of mild or severe impairment is arbitrary. The important question is whether it is possible to predict stroke risk, and benefit from carotid surgery, by measuring cerebrovascular reactivity. The important recent study by Kleiser and Widder suggests that in carotid occlusion it is a useful predictive marker, but larger clinical studies are required to demonstrate the natural history of reduced reactivity and determine whether it is useful in selection of patients for vascular surgery. If the studies demonstrate that cerebrovascular reactivity is important, as we suspect, they will answer the question as to what level of reduction in reactivity is clinically relevant.

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References

Disagreement Over Terms
I think that the editorial by Julien Bogousslavsky,1 "The plurality of subcortical infarction," which appeared in the May issue, deserves some comments.

Lacune is only a pathological term, and I agree that it is essential to define the term and the pathogenesis of the lesion, but it is also relevant to assess the clinical correlations and neuroimaging features. The definition of lacunes rests on their size, which ranges from 6 mm to 15 mm and sometimes to 20 mm (giant lacunes). Instead of evaluating the area, it is better to evaluate the volume (multiplying the area by the thickness of the section[s] showing the lesion; volume between 1.8 and 4 ml). Lacunes with a diameter of less than 6 mm cannot be visualized.2

Moreover, in the CT-MRI era, it could be acceptable to denote as "lacunar infarctions" the small areas exhibiting low density on computed tomography or an altered signal intensity on magnetic resonance imaging and accept the term "lacunar syndrome" as defining a clinical picture reasonably attributable to the lesion.3 A more specific point concerns the clinical features (particularly "pure hemiparesis"4), we found in 150 of 594 patients with cerebrovascular disease. A proportional hemiparesis, involving face/arm/leg, was observed in 85 patients, of whom 44 (51.8%) suffered from a lacunar infarction. The distribution over one side of the body was not uniform in 95 patients; of these, lacunar lesions were found in 59 (62.1%) (\( \chi^2=1.56 \), Yates' correction; df=1; \( p > 0.2 \), not significant). Thus, I cannot state that a proportional hemiparesis is significantly associated with lacunar lesion, whereas no uniform distribution of hemiparesis is not.

Regarding the pathogenetic hypothesis, we found cardioembolic and large-artery obstructions in about 25% of the patients and hypertension as the most frequently related factor in 75% of the patients.5 In my opinion, the crucial point is the relation between the lesion and the clinical syndromes. In our pathological observations, the lacunes were asymptomatic in 68% of the cases; on the other hand, a lacunar syndrome shows a normal CT in 26% of the patients.6 A lacunar lesion shown on CT must be carefully evaluated to be considered the cause of the observed clinical features. Finally, I consider the label "subcortical infarctions" incorrect and misleading. The infarction lies in a given vascular territory and can occupy (but rarely does) the entire area supplied by a given artery or only a portion of it, sometimes borderzone territories. The label subcortical is too vague and inadequate to correctly qualify an ischemic lesion. In fact, to define an ischemic lesion we should specify the vascular territory involved and the damaged parenchymal structures.

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References
Response

It is a sign of health for an editorial to stimulate discussion, and I welcome the remarks by Loeb. I am happy to see that he seems to agree with my emphasis of the clinical implications of subcortical infarction. However, two astonishing comments about the size of lacunes make me wonder whether we are talking about the same thing: actually, several lacunes (and probably most of them in autopsy series) are smaller than 6 mm, and many lacunes with a ≤6-mm diameter can be visualized well by magnetic resonance imaging. Our reappraisal of the pure motor stroke syndrome has recently been published in full detail, and I advise Dr. Loeb to refer to it; in short, we showed that if motor weakness does not involve face, arm, and leg together, the probability of a small, deep infarct will drop. It would be interesting to know more about Dr. Loeb’s series, which seems to be as yet unpublished, as the comment is un referenced.

I do not understand exactly why Dr. Loeb judges the term subcortical infarction “incorrect and misleading” since it is purely descriptive and mainly emphasizes that the cortical pial arterial supply to the territory involved and the damaged parenchymal structures.

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References


Arrhythmias and Stroke Laterality

As a clinical cardiac electrophysiologist, I found the article by Lane et al both interesting and provocative. It brought to the fore many pathophysiological questions that merit further study.

The authors concluded that their “…data provide partial support for the hypothesis that the right and left hemispheres influence the nature and severity of cardiac arrhythmias in a differential manner.” I believe that this conclusion should be considered tenuous, or at least be further qualified, until the following issues are addressed.

1. Imprecision and temporal nonuniformity in timing of Holter monitoring relative to the acute event (strokes). Temporal changes in blood pressure, autonomic tone adrenergic state, etc. can significantly affect the cardiac electrophysiologic milieu and hence the propensity for arrhythmias. The authors merely stated that patients underwent Holter monitoring within 2 weeks of admission to the stroke unit.

2. Spontaneous variability of arrhythmias and duration of monitoring. It is well known that the complexity and frequency of arrhythmias can spontaneously vary strikingly from day to day. Hence, one cannot exclude the possibility that differences in arrhythmia type and frequency found by Lane et al were an artifact of the “limited” (i.e., 24 hours), temporally disparate recording periods. The importance of prolonged (e.g., 48-96 hours) continuous monitoring on all study patients beginning at a fairly uniform time relative to the acute stroke onset cannot be overemphasized if data are to be most meaningful.

3. Noncharacterization of ECGs and of supraventricular (SVT) and ventricular tachycardias (VT). Were SVT and VT nonsustained or sustained, and what were the rates? There are many potential mechanisms for SVT. Did SVT exhibit a wide (e.g., preexcitation or aberrancy) or narrow QRS complex? Evidence for “active” accessory pathway participation in SVT (i.e., identifiable anatomic basis) could alter interpretation of the relation between SVT and stroke laterality (although stroke could “trigger” SVT by various mechanisms).

Was VT polymorphic or monomorphic? This might reflect very different underlying mechanisms, possibly unrelated to stroke laterality. A prolonged corrected QT interval can be associated with serious ventricular arrhythmias.

The authors did not mention which, if any, patients had a prolonged QT, or whether its prevalence differed among patients with right versus left hemispheric strokes. Were pauses or sinus tachycardia observed during Holter monitoring?

4. Inaccurate characterization of cardiac structure. It is well known that the presence of structural heart disease can, in its own right, predispose patients to a variety of arrhythmias. The authors state that the presence of hypertension, coronary artery disease, and chronic obstructive pulmonary disease was determined by chart review. However, they did not provide information potentially critical to testing and interpretation of their hypothesis; e.g., was valvular heart disease present based on findings of physical examination and/or echocardiography? Was there evidence of ventricular hypertrophy, atrial enlargement, or other structural abnormality? How many of the 14 patients said to have “definite” coronary artery disease (their Table 1) actually had evidence of myocardial scarring, wall motion abnormalities, or depressed ventricular function? What was the prevalence of normal hearts, if present, similarly distributed among patients with right versus left hemispheric stroke?

5. Only 20% of patients admitted to the stroke unit underwent Holter monitoring. Would the findings have differed had the remaining 80% of patients with similar strokes undergone Holter monitoring?

6. Finally, while the authors’ hypothesis (i.e., association between right hemisphere strokes and SVT) is reasonable, some studies have observed supraventricular arrhythmias following left sympathetic stimulation in patients with normal hearts.

In summary, the study by Lane et al is an interesting attempt to further our understanding of neurocardiac interrelations. However, methodological limitations, including its retrospective nature, make it a study that raises more intriguing questions than it answers.

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The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/23/11/1681.citation

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