indeed represent the foretellers of an imminent lacunar stroke. The hypothesis should allow room for events such as these, which may be transient ischemic attacks in humans, why could it not be possible since emboli are reputed to account for a significant number of transient ischemic attacks in humans may at times be produced by microemboli, like those induced in normotensive rats by photochemical damage to the carotid artery? They have stopped short of suggesting that the more reasonable figure for adult practice. Obviously, more in vitro measurements through skulls should be made to establish this objectively.

Unlike Dr. Eden, we have never seen waveform improved by reduction of power. In all cases, there is an average waveform intensity loss of about 3.5 dB, or a 50% reduction in emitted power.

Since submission of that study, we have collected additional data on rates of failure at 1000 mW/cm\(^2\) estimated in situ intensity. For ages over 70 years, the rate of failure in white males is 7.5%, in white females 29%, in black males 33%, and in black females 73%; for ages 51–70 years the rate in white males is 1.6%, in white females 18.5%, in black males 10.4%, and in black females 52%. Too few younger subjects have been studied to make reporting of data from younger age groups meaningful.

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References

The Fallacy of the Lacune Hypothesis
To the Editor:

In a recent progress review, Milikan and Futrell have emphasized that lacunes or small strokes are not caused solely by a combination of hypertension and small vessel disease. They have listed four additional basic causes, including emboli, to account for the various lacunar syndromes. They have postulated that lacunes in humans may at times be produced by microemboli, like those induced in normotensive rats by photochemical damage to the carotid artery? They have stopped short of suggesting that the more durable clinical expression of a lacunar stroke might occasionally be preceded by identical, though transient, clinical manifestations. But since emboli are reputed to account for a significant number of transient ischemic attacks in humans, why could it not be possible that microemboli entering penetrating branches of the main cerebral arteries produce transient ischemic lacunar syndromes? The following personal observation of a cluster of transient pure motor hemiplegic attacks is submitted as an illustration of transient ischemic lacunar syndromes. A comprehensive lacune hypothesis should allow room for events such as these, which may indeed represent the foretellers of an imminent lacunar stroke.

A 65-year-old normotensive, nondiabetic, right-handed man had begun to recover from an initial bout of right-sided pure motor hemiplegia when first examined on October 19, 1990, in our emergency room 3.5 hours after its onset. Cardiac and fundoscopic examinations were normal. A loud left carotid bruit was heard. Within the next 2 hours, a computed tomography scan and an electroencephalogram were performed and both were normal. By then, the patient had entirely recovered and continuous intravenous heparin perfusion was started.

Over the next 3 days, six additional, shorter episodes of pure motor hemiplegia or hemiparesis, lasting 3–30 minutes, were observed. After adjustment of heparin rate delivery, no additional attack occurred. An electrocardiogram and a 24-hour Holter monitoring were normal. Angiography revealed an ulcerated stenotic plaque at the bifurcation of the left carotid artery. The patient underwent an uneventful left carotid endarterectomy on the tenth day of hospitalization; he has since fully recovered.

Because clinical documentation of cerebral emboli is not possible, equating this patient's episodic right-sided weakness with transient ischemic lacunar syndrome caused by microemboli from a left carotid plaque must remain speculative, though plausible. How often can such transient ischemic lacunar syndromes precede a more permanent lacunar stroke in the same arteriolar distribution? Perusal of the literature, including the retrospective study by Ueda et al\(^2\) in which ipsilateral lacunar infarcts were the most common finding observed on pathological examination of patients with earlier, unspecified types of transient ischemic attacks, offers no satisfactory answer. In the study by Sohn et al\(^4\) of the effect of capsular infarct size on clinical presentation of stroke in 74 patients, no instance of transient ischemic capsular syndrome is reported in any of their patients. In Fisher's clinicopathological study\(^5\) of 11 capsular infarcts, only one patient had reported what was considered to represent a premonitory transient ischemic attack—a single episode of weakness limited to the right hand for about 1 hour, 2 days before the occurrence of a more durable ipsilateral pure motor hemiplegia.

How common are transient ischemic lacunar syndromes? Does their natural history differ from that of other usual transient ischemic attacks? Among the six basic causes listed by Milikan and Futrell for lacunes, which is the one most commonly responsible for transient ischemic lacunar syndromes? How often are microemboli at play? Can microemboli in the same patient enter subcortical arteriolar branches at times and, at other times, larger cortical vessels? What is the optimal treatment of transient ischemic lacunar syndromes? These are some of the questions that reading Milikan and Futrell's challenging report brings to mind. Some need quick answers, particularly in caring for patients like ours who may present with transient ischemic lacunar syndromes that could foretell an impending lacunar stroke, producing catastrophic motor deficits despite its small size.

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References
Letters to the Editor


To the Editor:

It is provocative, but incorrect, that Millikan and Futrell regard the lacunar hypothesis as a fallacy. They may be right to some extent that indiscriminate use of the adjective “lacunar” can lead to errors, but a lacunar infarct, or “small deep infarct,” must be considered a separate stroke entity for the following reasons.

According to Bamford and Warlow, the validity of the lacunar hypothesis should be tested by answering two main questions. First, do lacunar infarcts usually cause specific lacunar stroke syndromes, and, second, are lacunar infarcts usually caused by a specific disorder of the deeply localized small perforating arteries? They emphasize the term “usually” to create general characteristics of this subgroup of cerebral infarction and to avoid a list of rarities. The first question has been answered in the affirmative by two different studies; only 6% of patients with a lacunar stroke syndrome (or should we say “a specific syndrome usually caused by a lacunar infarct”?) had lesions other than lacunar infarcts on computed tomographic scanning. In Fisher’s important study about the pathological features of lacunar infarcts in the internal capsule, appropriate obstructive lesions of small perforating vessels were usually found (in nine of the 11 patients). Therefore lacunar infarcts generally are thought to be caused by small vessel disease.

It is also confusing that Millikan and Futrell use evidence from an animal model to prove that emboli are a common cause of lacunar infarcts although there are several clinical studies proving that both atheromatous abnormalities of the extracranial arteries and cardiac sources of emboli were usually absent in patients with lacunar infarcts.

Recognizing different subgroups of cerebral infarction and accepting lacunar infarcts as one of these not only saves the patients with these lesions from unnecessary, but potentially harmful, procedures such as carotid angiography and carotid endarterectomy, but is also important for future clinical research, and seems to be nearer the truth than proclaiming that “a lacune is a stroke, just a small one.”

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