To the Editor:

In their report on carotid endarterectomy in a community-based teaching hospital, Friedmann et al. suggest that the ‘‘combined mortality/major neurologic morbidity’’ risk of 3.2% was somehow a function of where the surgery was performed and by whom. There are, however, other plausible explanations.

Members of the study cohort may have been different from patients who undergo surgery at other hospitals. Friedmann et al. do not describe the neurological histories and baseline features of their patients in sufficient detail to allow readers to test this possibility. What symptoms were being diagnosed as transient ischemia? What angiographic findings were leading to surgery? What was the prevalence of cardiac comorbidity?

These are all clinical features that are necessary to explain to interpret the results of this study. Instead of this detail, Friedmann et al. state only the age and sex of their patients, the number of treatments for stroke by measuring a nonclinical variable, and they do not explicitly define their end points. We believe that the study design (i.e., retrospective versus prospective) and any missing information.

Research methods can have a profound effect on outcome, yet Friedmann et al. do not describe their methods. They do not describe the study design (i.e., retrospective versus prospective), the sources of data, or the mechanism for obtaining data, and they do not explicitly define their end points. We believe that this is a retrospective study. If such is the case, it is important to know who was the chart extractor, if the extractor was blinded to the study purpose, and what that extractor coded as a perioperative period? Any missing information.

Friedmann et al. have presented a study of short-term prognosis among patients selected for carotid surgery without adhering to scientific standards for such investigations.1-4

This study will be of value to patients at Baystate Medical Center who are deciding whether to accept the risk of an unproven therapy administered in their own institution. For investigators, surgeons, and patients in other institutions, however, this paper will be difficult to interpret. Lacking more information about the assembly of the cohort, the baseline features of members of the cohort, the surgery, and the research design, it is impossible to understand how the authors got the results they published or how these results compare with research at other institutions.

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References


Clinical Relevance of Stroke Models

To the Editor:

The insightful editorial by Molinari5 begins to shed light on a fundamental question in cerebrovascular research. As he stated, many therapeutic developments have shown great promise in the laboratory only to be found to have insignificant effects in clinical trials. Unfortunately, his conclusions and proposed solution to this problem are scientifically unsound in several ways.

Dr. Molinari proposes to solve the problem of clinical failure of treatments for stroke by measuring a nonclinical variable, namely, lesion size. It is indeed quite reasonable to assume that the preservation of tissue is better than its destruction. However, mere preservation of tissue, no matter how clear on imaging studies, cannot bring vision to amaurotic hemifields, make a paretic arm move, or restore speech to the aphasic. Measurement of comparative neuropathology in humans, although interesting in itself, is not directly relevant to the treatment of
Short-term results of carotid endarterectomy.

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