to the hyperemias were accessible for reliable rCBF investigations, we found it justified to discuss therapeutic possibilities.

Our assumption of hyperemic borderzone as threatened areas with a potential to survive is derived from our own findings of impaired autoregulation, false autoregulation, and impaired CO₂-response in some of the hyperemic areas investigated. Experimental findings of histopathological changes in hyperemic brain tissue are available and quoted in the paper. Early surgical recanalisation of occluded arteries in stroke is known sometimes to produce hemorrhagic infarction and worsening the clinical outcome. Unlike other organs, the brain is placed within a closed skull. Focally increased blood volume and edema affect, therefore, the tissue pressure in the surrounding tissue much more in the brain than in other organs. Finally the type of hyperemia reported in our study seems to disappear within the first week after the stroke. It is not comparable to the hyperemias seen 2–4 weeks after the stroke which serves in the reabsorption process of necrotic material from the infarct.

Much work remains before the nature and clinical significance of cerebral hyperemia is clarified. The findings in our study indicate that acute cerebral infarction is not only a matter of cerebral ischemia. Hyperemia is quite as common and the entire infarct is sometimes even hyperemic without being hemorrhagic. We find it worthwhile to include the presence of focal hyperemia in therapeutic considerations.

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Important Points in Treatment of Progressing Stroke

To the Editor:

We found the editorial on “Treatment of progressing stroke” by Drs. Norris and Lassen provides an opportunity to emphasize again three of the many points made in our article “Treatment of Progressing Stroke.”

1. Over the decades it has become apparent that many physicians have a problem in dealing with the concept of the minute to minute uncertainty produced by the changing quality and quantity of neurologic deficit which is the hallmark of “progressing stroke” (stroke-in-evolution). Physicians who deal with acute illness in patients in intensive care units soon become familiar with the notion of frequent re-examination (every 30 minutes in our cerebrovascular intensive care unit) of the salient abnormality. Thus, a determination can be made as to whether the trouble is worsening, static or improving. For instance, a patient seen at 10 a.m. with mild left upper extremity weakness (history of onset at 9 a.m. that day), re-examined at 10:30 a.m. and found to have severe weakness of that extremity, would be classified as “progressing stroke,” but if the deficit had disappeared the categorization would be “transient ischemic attack.” The clinical stage designation may change several times for the same patient. This extreme variability in the natural history, when added to the diverse pathogenetic mechanisms causing the focal situation, admittedly does produce a complex set of variables with which the physician must grapple.

In our review, we wrote that “18 to 24 hours without progression is needed to be sure that further progression is unlikely,” for infarction in brain supplied by the carotid system, while in the vertebrobasilar system a longer period (up to 72 hours), should pass. This natural history is so variable that we included numerous details to illustrate those differences.

2. The use of anticoagulant, to prevent progression, is only recommended when there is accurate diagnosis and there is incomplete impairment of function. If the patient is hemiplegic and/or has a depressed mental status, anticoagulant therapy is used frequently. However, the truly progressing stroke, “transient ischemic attack,” is an entirely different entity. Those who work with animal models of experimental brain edema are working with and producing severe lesions. This is an entirely different population than patients with stroke coming to a clinical cerebrovascular service. In the latter situation, many patients have minimum focal brain damage when admitted — the objective is to keep focal brain damage from progressing to an extreme state as often represented by the experimental brain edema models.

A number of therapies are reviewed in our paper for treatment of progressing stroke — selection must be made depending on the mecha-

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nisms causing the focal ischemia.

We appreciate the chance Drs. Norris and Lassen have produced to write again about these important matters.

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Fletcher McDowell, M.D.

References
Important points in treatment of progressing stroke.

J W Norris and N A Lassen

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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/13/3/403.citation