tently seen in the patients in which the hyperemias were accessible for reliable rCBF investigations, we found it justified to discuss therapeuti-
cal possibilities.

Our assumption of hyperemic borderzone as threatened areas with a
potential to survive is derived from our own findings of impaired auto-
regulation, false autoregulation, and impaired CO₂-response in some of
the hyperemic areas investigated. Experimental findings of histopatho-
logical changes in hyperemic brain tissue are available and quoted in
the paper. Early surgical recanalisation of occluded arteries in stroke
are 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, there­
fore, 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
more hyperemic without being hemorrhagic. We find it worthwhile to include
the presence of focal hyperemia in therapeutical 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.
Millikan and McDowell in Stroke 12, 397–409, 1981 both interesting
and controversial.

The opening sentence of the article implies that progressing strokes
are defined by their progression over minutes. When questioned closely,
patients with strokes of apparently “sudden” onset will often describe
an evolution of their symptoms over minutes. We feel that progressing
strokes are better defined as showing deterioration over hours though
the process is usually completed within one day. Deterioration over
days on the other hand is also seen in a large minority of acute stroke
patients.¹ The pathogenesis of these three evolutionary types of acute
stroke may be the determining factor in their differing temporal profiles.

The authors state that anticoagulants may be helpful in progressing
ischemic strokes but the implied progressing “thrombosis” may be a
theoretical concept which needs revising. Cerebral edema is the most
powerful adverse factor in the first few days of acute cerebral infarction
and serial CT scans in such patients in the acute and sub-acute periods
usually show no extension of the area of infarction.

Patients who progress or deteriorate in the acute stage usually reveal
a mass effect of brain swelling on CT, the suddenness simply representing
mechanical decomposition within the bony confines of the skull. Mod-
els of experimental brain edema suggest that more edema accumulates
outside than inside the infarction once the blood-brain barrier has been
breached. Attacking this problem may prove more fruitful than using
anticoagulant therapy for which therapeutic effectiveness has yet to be
demonstrated.

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lar Disease 3, Excerpta Medica, Amsterdam, p. 315, 1981

To the Editor:
The letter from Drs. Norris and Lassen provides an opportunity to
emphasize again three of the many points made in our article “Treat-
ment of Progressing Stroke.”¹

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 neurologi-
cal 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 “pro-
gressing 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 mecha-
nisms 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,”² for infarction in
brain supplied by the carotid system, while in “the vertebrobasilar
system a longer period (up to 72 hours), should pass.”³ 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 recom-
manded when there is accurate diagnosis and there is incomplete impair-
ment of function. If the patient is hemiplegic and/or has a depressed
level of consciousness, it can be assumed that maximum ischemia is
probably past and progression of focal ischemia is unlikely. In this
situation, it is not advisable to start heparin. In a number of centers,
where there is extensive experience with acute occlusive stroke, antico-
gulant therapy is used frequently.³,⁴

3. Cerebral edema which can follow cerebral infarction is the most
serious threat to life. In the same issue of “Stroke” in which our paper
appeared, Bounds et al.⁵ reported that transtentorial herniation was the
cause of death in 31% of 100 cases of acute cerebral infarction in the
brain supplied by the internal carotid artery studied at autopsy, and 60%
had some other cause of death. At that same cerebrovascular center the
mortality for acute occlusive disease stroke (carotid system) is 11%.
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 experi-
mental 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-
nisms causing the focal ischemia. We appreciate the chance Drs. Norris and Lassen have produced to write again about these important matters.

Clark Millikan, M.D.
Fletcher McDowell, M.D.

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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

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