Assessment of Cerebral Autoregulation Dynamics

To the Editor:

I want to bring to the attention of your readers that the paper by Aaslid et al.,1 recently published in Stroke, contains inappropriate comparisons between their data and earlier data published by my colleagues and me.2

Aaslid and colleagues measured blood-flow velocity with transcranial Doppler in the middle cerebral artery and in the straight sinus of human volunteers during acutely induced hypotension. From these measurements, they determined the onset of autoregulatory response in the cerebral circulation. They then compared their findings to earlier work my colleagues and I published in which we studied the effect of acute hypotension on the autoregulatory response of pial arterioles in anesthetized cats. Aaslid et al stated that the responses we obtained were much slower than those they found in humans, and they concluded that "the intact human cerebral circulation in the absence of pharmacological influences does not function as predicted from pial vessel observations in animals."1

The problem with these comparisons is that Aaslid et al compared the onset of the vasodilation in their experiments to the time delay required to reach an increase in vascular diameter 10% above baseline in our experiments, and not to the onset of vasodilation. The onset of the vasodilation in our experiments was of the order of 1–2 seconds; it was illustrated in individual experiments in Figures 8, 9, and 10 in our paper.2 Obviously, Aaslid et al compared apples with oranges.

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References


The following is in response:

To the Editor:

We appreciate the comment and added information supplied by Dr. Kontos on the comparison between autoregulatory response times evaluated by arteriolar diameter1 and Doppler measurements.2 It is indeed a very difficult task to compare results of different experimental designs, and the term "significant change" can have so many meanings that it can be almost meaningless. It is easy to unintentionally fall into the trap of comparing apples with oranges. Even though our main emphasis was put on half-maximal response times because they are more precisely defined than arbitrary significance levels, we also reported figures for "latency times," or time of onset defined as the time interval from acute hypotension until a distinct autoregulatory effect was seen in the Doppler tracings. We had hoped to resolve one of the concerns raised in an earlier editorial in Stroke3 in which our previous data on cerebral autoregulation dynamics in humans4 were compared with the cited experiments in anesthetized cats.1 This editorial stated that "It is difficult, therefore, to accept that active changes in vessel caliber in response to arterial hypotension occurred as rapidly as Aaslid et al assert." In his letter to the editor, Dr. Kontos adds first-hand interpretation of the data from pial vessel caliber recordings.1 This information resolves the concern quoted above: onset of active changes in cerebral arteriolar caliber does occur within 1 to 2 seconds after induction of acute hypotension in anesthetized cats. The same latency is also seen in Doppler recordings from humans.3,5 Even though the half-maximal response times to acute hypotension appear to be somewhat faster for the flow velocity in unmedicated humans than in the caliber of pial vessels of cats, the difference between the two species and methods has now been brought down to an acceptable level that could be explained by the effects of anesthesia and other details of experimental design and presentation.

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Confusion Over the Terminology of Subcortical Infarcts Visible on Computed Tomography Is Widespread

To the Editor:

The two cases reported by Cacciator and Rousso recently (Stroke 1991;22:1603–1605) emphasize again a widespread confusion in the terminology of subcortical infarcts visible on computed tomography (CT). In the recent stroke literature, it has repeatedly been postulated that lacunar infarctions occur due to brain embolism.1,2 As Fisher himself has pointed out,3 according to the "lacunar hypothesis," lacunar infarcts are small, deep cerebral lesions resulting from autotchonous occlusion of single small, penetrating cerebral arteries. He himself limited the maximum diameter of such infarcts to 1.5 cm. Another very typical feature of lacunar stroke syndrome, but did not show a lacunar infarct on CT. The lesion demonstrated in Fig. 1 of their article shows an infarct of the lentiform nucleus and the internal capsule, of the type referred to as "large lentiform nucleus infarctions"6,7 or "large striatocapsular infarcts."6,7 Such a lesion has clearly been demonstrated to occur as a result of multiple but simultaneous occlusions of the lenticulostriate arteries due to large-vessel disease,6,8,9 usually emboli to the proximal middle cerebral artery (MCA),10–12 whereas the superficial territory of the MCA grossly remains preserved due to retrograde leptomeningeal collateral flow.12
Assessment of cerebral autoregulation dynamics.
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