Response

In their letter, Bladin and Burns repeat previously published arguments, without considering the particulars of our study. They have cited us incorrectly several times, suggesting that they did not read our paper accurately:

1. "An absolute precision of ±0.1 mm . . . was . . . assumed by the authors." In fact, we stated that "the calipers could be adjusted in 0.1-mm graduations."

2. The "Doppler angle, which the authors omit to report . . ." is actually listed in our Table 1.

3. "The unfortunate use of color duplex (rather than duplex-pulsed Doppler) . . ." We have accurately described the use of the pulsed-Doppler technique in measuring angle-corrected flow velocities. Color duplex sonography means pulsed-Doppler measurement of flow velocities in vessels that are simultaneously depicted in the B-mode scan and the superimposed color Doppler image.

4. As to "the differences in flow between the CCA and ICA+ECA . . . no data are given that will indicate the accuracy of an individual measurement . . ." Since we could not list every measurement in tables, we displayed the main data in scatterplots to make individual measurements evident to the reader. Furthermore, in Table 4 we did not only show correlations between our long-term reproducibility measurements but also the differences between them. And finally, we pointed out why we consider the measurements of ICA and ECA flow volumes more reliable than those of the CCA.

5. Duplex measurements of the ICA were not taken at the origin but at 1.0 to 1.5 cm away from the bifurcation. At this site (which corresponds to the "distal internal carotid artery site" in the paper of Ku et al1) no turbulences were detectable either in the color Doppler mode or in the pulsed Doppler spectrum, and the spectral broadening index was significantly lower (p<0.0001) than at the CCA recording site (cf Table 1 of our paper, statistic analysis unpublished). The "off axis" angle of carotid flow is of great importance at the origin of the ICA, especially when we consider maximum flow velocities or flow velocities at certain layers of flow. It is less important when time-averaged velocity (TAV), ie, the mean of all frequencies occurring above and below the baseline, is measured across the whole lumen. Given that the diameter of the vessel remains constant and a constant flow volume is transported along a vessel, TAV also has to remain constant. In this way, the TAV vector runs parallel to the walls of the vessel. Therefore, TAV must be measured with an angle correction along the walls of the vessel with the sample volume covering the entire luminal width at a site where the spectral broadening of the Doppler profile is normal. By measuring TAV in this way, we assume we have reduced—though not entirely excluded—the possible influence of non-axial flow components.

Our study is the first ever to report total CBF measurements by using color duplex sonography of the internal carotid and vertebral arteries. We think that we have provided some useful data for the clinical application of the method and future investigations, but we have also stressed that this is a preliminary study which has to be confirmed by intra- and interobserver reproducibility examinations. This study is under way and we will publish our data as soon as possible—whatever the results. There is a lack—and not abundance—in bedside methods of CBF estimation. We would be glad if the "direct methods" published 10 years ago could resolve all the technical problems. Must we wait yet another decade?

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References

Response
We thank Drs. Hart, Halperin, and Miller and Ms. Pearce for their interest in our article on the factors associated with the failure of aspirin treatment after stroke.

Unfortunately, the small sample size of patients with atrial fibrillation does not allow us to further stratify the patients to perform multivariate analysis (Cox proportional hazards model).

As for the second point, we indeed found that ischemic heart disease is a significant risk factor for aspirin failure, since even after Bonferroni correction the odds ratio for this risk factor remains statistically significant.

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Neurobiology of Passive Avoidance Impairment After Ischemia
Karasawa Y et al report that passive avoidance impairment after ischemia is related to damage of CA1 neurons of the hippocampus related to memory and habituation. The neurobiology is suggested by the release of dopamine preserved lateralized to the right hemisphere subserving passive avoidance. This hypothesis is supported by optimal response sequence at intermediate dopamine tone in a medial-frontal-striatal activation system and by operant conditioning of CA1 bursting at different concentrations of dopamine, showing a sharp peak at 1 mM and falling abruptly when this optimal concentration was either halved or doubled. The fact that delay-dependent speeding of reaction time, indicating motor readiness, is abolished by depletion of dopamine subserving mood prompts the evaluation of ischemia-induced behavioral changes by monitoring behavioral correlates of mood, ie, speech hesitance and switching pauses. This method is supported by the contribution of articularatory rehearsal to short-term memory and by the association of >2-second speech pauses with praxicarticulation repair and competitive and courtship activity. The findings suggest a causal relationship between impairment in passive avoidance and neuronal damage in the hippocampus, thus tending to confirm powerful isomorphisms between mind and body and the existence of deep and lawful mental structures governing human cognitive and emotional functioning reflecting properties of neuronal activity and firing.

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References

Experimental Stroke Treatment With High-Dose Methylprednisolone
We read with interest the recent article by de Courten-Myers et al. There are several parallels between this study and a study we recently published. Because ischemia is a primary pathophysiological mechanism in epidural brain compression, both studies involve transient regional cerebral ischemia. Both the steroids in the study by de Courten-Myers et al and the hypothermia in our study are considered to protect and resuscitate by affecting several sites along the ischemic cascade. In both studies treatment was initiated early, 30 minutes into the insult by de Courten-Myers et al and 15 minutes into the insult in our study. The results of the two studies are remarkably similar. In neither study did treatment decrease mortality, but in both studies treatment significantly decreased the cerebral infarct size ($P<0.05$ in the de Courten-Myers study and $P=0.07$ for infarct and $P=0.03$ for overt histological damage in our study). In the de Courten-Myers study treatment clearly improved cerebral blood flow during ischemia ($P<0.005$),
Aspirin in elderly atrial fibrillation patients.
R G Hart, L A Pearce, J L Halperin and V T Miller

Stroke. 1994;25:1525-1526
doi: 10.1161/01.STR.25.7.1525

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
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