To the Editor:

The editorial by Drs. Rosenblum and Kontos, which appears in STROKE (5:425-428 [July-Aug] 1974), articulated the need for continued research efforts directed at the cortical surface microvasculature; a plea I strongly support. In justifying this need they have dissected work that has been performed in this laboratory which differs from their findings and concepts. There are errors in their dissection.

First, they confuse "cannulation" measurements of pressure and "micropuncture" measurements. Precisely because micropuncture does not involve occlusion of flow it yields pressure measurements at the site of puncture rather than those prevailing at distant branch points. Our argument, which is cited (by page number) by these authors, points out this difference but is quoted by them as indicating that micropuncture occludes the vessel punctured. This is decidedly not true.

Second, puncture of a vessel can alter the local responsiveness of that vessel to vasoactive stimuli which may present as a localized diameter discontinuity. Localized constriction occurred at micropuncture sites after one and one-half to two hours in some of our preparations. However, such data were explicitly discarded from these preparations as indicated in our publications. Additionally, we have discussed (reference 9 of their bibliography) that the pressure at a point in the vascular network will be predominantly a function of upstream and downstream resistances and will be little influenced by local effects.

Finally, there may be great hazard in extrapolating from microvascular pressure measurements made in mesentery to those in the brain as they have done in developing their arguments. Such extrapolations between two circulations may be presumptuous. Our uneasiness regarding extrapolation was a primary motivation for making direct micropuncture measurements. It is evident from our results that our uneasiness was justified. Though many questions can be posed by analogy, real answers can be found only in the organ of interest, in this case the brain. In one respect, at least, I can strongly support the thesis presented by Drs. Rosenblum and Kontos: we do need to continue supporting the "renaissance of cerebral microcirculation." This means making further measurements in the cerebral microvasculature proper and includes the cortical surface vasculature.

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To the Editor:

Dr. Stromberg's letter agrees with our thesis that continued study of pial vessels is vital. We fear that such studies may be neglected because of erroneous interpretations of pressure gradient data. These interpretations assign a very minor role for the pial vasculature in the control of cerebral blood flow. Dr. Stromberg does not really confront this issue in his letter, but rather implies that such an interpretation may be correct.

With respect to the accuracy of the numbers gathered in micropuncture studies, Dr. Stromberg makes two points. He suggests that punctured vessels have normal reactivity if they fail to develop local constriction at the puncture site. We would prefer to see direct tests of reactivity, not only at the puncture site, but along the entire punctured segment. Absence of focal constriction is no guarantee of normal reactivity. He also points out that in discussing obstructed flow, he was referring to experiments with "cannulae" larger than his micropipettes. We regret this error in our summary of his work. However, both of these points were quite minor in relation to what he calls our "dissection" of work performed by him and his co-workers.

We carefully stated that the data were subject to reinterpretation even if all the pressure values were accepted as being correct. We then showed that in their analysis Stromberg and others placed some pial vessels in the post-pial vascular segment thereby underestimating the pial contribution to CVR. We also pointed out that in comparing the contribution of pial arterioles with that of "downstream" vessels, one must distinguish between each of the contributions made to the pressure gradient by parenchymal arterioles, capillaries and venules. Stromberg et al. failed to do this. When this is done, using the best data available for capillary and venous pressure, Stromberg's own pressure values suggest that the contribution to CVR made by the pial arterioles compares favorably with the contribution made by parenchymal arterioles. In short, even when we assume the accuracy of micropuncture values, we find no reason to conclude, as some have done, that pial arterioles contribute much less to CVR than do parenchymal arterioles.

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