
Doctor Jones replies:
We emphatically agree with the major point of Dr. Wolf's et al letter suggesting that selective bias may account for the differences in frequency of stroke in relationship to mitral valve prolapse in our two populations. This was one of the basic purposes of our report, i.e., to emphasize that the risk of stroke appears to be very small in the large general population of patients with well recognized mitral valve prolapse (MVP).

We acknowledge that MVP was "intensively sought out" in young stroke victims without evidence of carotid and other predisposing cardiac lesions. However, within this very small subset population, i.e., the young patient with stroke, the importance of MVP may not be "exaggerated" as well defined by Barnett et al.1 Because some of these patients may have multiple cerebral ischemic events, the identification of this source has obvious therapeutic implications.

The essential question for future studies is the identification of pathophysiologic mechanisms which uncommonly predispose a very few patients with MVP to the risk of stroke. One mechanism that does appear to be documented to date is that of subacute bacterial endocarditis such has been documented in 10% of those in one large series of patients with MVP and stroke.2 Other than for the appropriate prophylaxis for SBE we continue to advocate a conservative management of the asymptomatic MVP patient.

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References

Whole Blood Viscosity and Cerebral Blood Flow
To the Editor:
In a recent issue Grotta and coworkers reported on whole blood viscosity parameters and cerebral blood flow (CBF).3 Their results were further commented on in an editorial by Thomas.2 This report on blood viscosity and CBF deserved further comments.

Changes in hematocrit and blood viscosity may induce simultaneous changes in the blood's oxygen release capacity which has to be taken into account. Grotta and coworkers hardly discuss changes in oxygen release capacity in their paper, but seem to consider the effects as largely due to viscosity changes. Thomas on the other hand mentions that there is some debate on the relative importance of blood viscosity and blood oxygen carrying capacity for the flow changes.

First, it should be pointed out that the blood oxygen release capacity is not a simple function of its oxygen binding capacity but is also heavily influenced by shifts of the oxyhemoglobin dissociation curve. Thus, a shift to the left of the oxyhemoglobin dissociation curve results in a higher oxygen saturation at a given oxygen tension and less oxygen can be released in the tissue if its oxygen tension has to be maintained unchanged. A shift to the left of the oxyhemoglobin dissociation curve, thus results in a CBF increase.4 Changes in hematocrit result in blood viscosity, in oxygen release capacity and in CBF changes. The oxygen tension in jugular venous blood remains constant following acute decrease of the hematocrit in patients with normal or with high hematocrit.5 4 Here the oxyhemoglobin dissociation curve also remains constant. But, in patients with chronic changes in hematocrit it is known to shift; e.g. to the right in anaemia.

Grotta and coworkers performed multivariable analysis and demonstrated that the serum fibrinogen concentration in addition to the hematocrit seems of importance for the CBF. They interpreted this to changes in viscosity which very well may be the case. However, as the oxyhemoglobin dissociation curve is shifted to the right in anaemia, this would per se result in a slightly lower CBF. If such data had been available their multivariable analysis of fibrinogen concentration and hematocrit might well have yielded another result. Thus, caution must be exercised in the interpretation of these results.

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References

Doctor Thomas replies:
It is now generally agreed that under normal circumstances in patients with high hematocrit, the main reason for their having a low CBF is the higher oxygen carrying capacity of their blood. That the high viscosity, which accompanies high hematocrit, has any influence on CBF at all has been questioned. The paper from Grotta et al is valuable because it shows that factors, like fibrinogen, which raise blood viscosity, but apparently have no effect on oxygen carrying capacity or the oxygen dissociation curve, are associated with lower CBF. Furthermore, the position of the oxygen dissociation curve does not change after venesection.2

There may be differences between CBF measurements immediately after venesection and after a few days when the circulation is in a more stable state. In the acute phase, Henriksen et al showed a small rise in the oxygen delivery capacity (CBF x arterial oxygen content) which failed to reach significance in their 6 cases. They also showed a small rise in the cerebral metabolic rate for oxygen. Wade2 measured CBF several days after serial venesections in 20 cases and found that this rise in O2 carrying capacity (approximately 8%) was significant. He did not measure jugular venous PO2 on ethical grounds because of the higher risk of thrombotic complications in patients with low flow and high hematocrit so he was unable to confirm that the CMRO2 also increased.

Clearly, in patients with high hematocrit the brain is not frankly hypoxic even if CBF is low. However, the low flow may be potentially dangerous, especially if another factor tending to reduce flow or to enhance thrombus formation is introduced. An important point to reassure the cautious is that after venesection oxygen delivery seems to rise slightly, rather than fall. Whether venesection proves to be of value in preventing thrombotic complications remains to be shown.

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References

2. Wade JPH: Transport of oxygen to the brain in patients with high hematocrit before and after venesection. Brain (in press)

Doctors Grotta & Ackerman reply:

Dr. Paulson correctly points out that we did not consider changes in oxygen release capacity in our study. We designed our investigation to analyze the statistical relationships between hematocrit, fibrinogen, an index of hematocrit/fibrinogen interaction and CBF among human subjects.1 Dr. Paulson is concerned that the changes in CBF which we ascribe to alterations in viscosity may in fact be due to the effect of chronic changes in hematocrit, such as anemia, on the blood's oxygen release capacity. However, the hematocrits in our population sample were generally normal and clustered relatively well around the mean, with a percent coefficient of variation of 11% (mean ± S.D. = 39.3 ± 4.3; range 28–52).

As Dr. Paulson indicates, the oxyhemoglobin curve is shifted to the right in chronically anemic patients, which could result in a decrease in CBF. However, we found increased CBF in patients with a relatively decreased hematocrit; therefore, it is clear that a shift in the oxyhemoglobin dissociation curve was not the major factor affecting CBF in our sample. We agree with Dr. Paulson that if one is to study a different population, namely those with polycythemia, anemia, or therapeutic phlebotomy, one should consider changes in the oxyhemoglobin dissociation curve in addition to changes in viscosity.

Our data emphasize the importance of the effects of multiple determinants of viscosity on CBF. Our initial observations are supported by subsequent investigations in Houston, which show that while maintaining hematocrit and oxygenation constant, reducing fibrinogen will produce a significant increase in CBF.2 This additional work further indicates that CBF can be changed by manipulating viscosity factors alone, since the oxyhemoglobin dissociation curve should not be affected merely by reducing fibrinogen.

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References


Editor’s Note of Appreciation

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