Letters to the Editor

Re: Socioeconomic Status and Ischemic Stroke
To the Editor:

Jakovljević et al1 find that persons with low socioeconomic status have considerable excess rates of morbidity and mortality from ischemic stroke in Finland.

Neurobiological features are suggested by the correlation of the rate of speech hesitation pauses of 1 second or more, 4.79 ± 2.48 per min, 1.50 ± 0.33 seconds (mean ± SD) with immobility in the face of stress, the state of the circulation (angina/ hypertension), and a 6-fold incidence of clinical coronary artery disease in 2 groups of normal, coronary-prone men followed prospectively for 10 years (P < 0.05). This hypothesis is supported by profound effects on angina through consciously focusing attention on breathing and intervening pauses, the association of the reduction of blood pressure with longer, less-recurrent pauses (about 2 seconds), reports that the microvascular response to the onset of neural activity is delayed consistently by about 3 seconds and is linked to increased coherence of electroencephalography gamma-band activity associated with the execution of more complex tasks, and increased blood flow may process glutamate during sudden increases in neuronal activation in order to maximize the power of intracortical processing within the gamma range of local field potentials.2

These findings give precise, objective methods with which to evaluate the effect of mental stress on the microvasculature and may be subject to editing or abridgment. Please submit letters in duplicate, typed double-spaced. Include a fax number for the corresponding author and the MCA and STA can be successfully compared. The segmental artery (STA) for comparison is probably the most difficult to emanate the biological system to various signals during an experiment. The various changes that have been observed, as pointed out by the authors themselves, were not the case to berry aneurysm of the distal branches of the MCA may certainly correspond to a misnomer; this meaningless denomination is traditionally used for subarachnoid aneurysms, and the one involved is likely to be subjacent. The extravascular space is therefore significantly different. The age of the lesion (the length of time that the aneurysm has been present and unruptured) and the age of rupture point to the exposure of the biological system to various signals during an undetermined amount of time. Stressing the role played by the extravascular space certainly points to the fact that the subjacent environment is significantly different from the subarachnoid in the generation of the aneurysm, its rupture, and the reaction to that rupture. The various changes that have been observed, as pointed out by the authors, cannot be assessed. Using the superficial temporal artery (STA) for comparison is probably the most difficult problem of the study. It certainly maintains the idea that the arterial system is homogenous enough that vessels as different as the MCA and STA can be successfully compared. The segmental vulnerability shows that this vessel has significant differences: phylogenetic, embryological, and hemodynamic. In addition, the STA does not develop aneurysms; these are characteristic of the external carotid biological evolution rather than that of the internal carotid branches. The hemodynamics and shear stresses in both systems are different, and the few aneurysms described in the STA are seen following trauma or MCA–STA anastomoses. This certainly emphasizes again the role played by the surrounding tissue and the surrounding vessel in the regulation and expression of the various genes.

The epidemiological references quoted certainly do not apply to children, even though they carry a genetic predisposition to the development of aneurysms (eg, polycystic kidney disease). The arterial immune reactions and their potential constitutional weakness (AIDS, familial candidosis) demonstrate the specificity of some targets and responses with regard to triggers. Finally, damaged repair systems in some hereditary diseases such as hereditary hemorrhagic telangiectasia type 1 with endoglin and transforming growth factor-β1 do not produce arterial aneurysms but rather different types of vascular alterations. One can envision the maturation over time of some genetic programs of modeling and remodeling of the brain vessels. The cell turnover and repair capacities are unlikely to be continuous and spread over an equal period throughout life. Postnatal

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Re: Molecular Anatomy of an Intracranial Aneurysm
To the Editor:

I read with great interest the recent Stroke article by David Peters and collaborators. This paper illustrates the passage of aneurysms as diseases of the lumen to the evidence that aneurysms are diseases of the arterial wall and sometimes even of the extravascular space. A few remarks need to be made concerning the case and its relevance in the approach to aneurysmal diseases.

The case occurs in a young child, where aneurysms are rare; it is seated on the distal middle cerebral artery (MCA), which is not a frequent location in general, even in children. It ruptures and gives rise to intracerebral hematoma and not subarachnoid hemorrhage. Such differences are significant if one wants to extrapolate such a case to subarachnoid aneurysmal rupture in adults.

The authors themselves mention that this was far from constituting an ideal model. I certainly agree with them, in particular because the diseases in children compared with adults are so different that the underlying genetic problems can probably be hardly compared.

In this particular case, we would certainly not describe the lesion as multiple aneurysms, since within a hematoma some of the pockets will necessarily correspond to a communication between the intravascular lumen and the hematoma cavity. Assimilating the case to berry aneurysm of the distal branches of the MCA may certainly correspond to a misnomer; this meaningless denomination is traditionally used for subarachnoid aneurysms, and the one involved is likely to be subjacent. The extravascular space is therefore significantly different. The age of the lesion (the length of time that the aneurysm has been present and unruptured) and the age of rupture point to the exposure of the biological system to various signals during an undetermined amount of time. Stressing the role played by the extravascular space certainly points to the fact that the subjacent environment is significantly different from the subarachnoid in the generation of the aneurysm, its rupture, and the reaction to that rupture.

The various changes that have been observed, as pointed out by the authors, cannot be assessed. Using the superficial temporal artery (STA) for comparison is probably the most difficult problem of the study. It certainly maintains the idea that the arterial system is homogenous enough that vessels as different as the MCA and STA can be successfully compared. The segmental vulnerability shows that this vessel has significant differences: phylogenetic, embryological, and hemodynamic. In addition, the STA does not develop aneurysms; these are characteristic of the external carotid biological evolution rather than that of the internal carotid branches. The hemodynamics and shear stresses in both systems are different, and the few aneurysms described in the STA are seen following trauma or MCA–STA anastomoses. This certainly emphasizes again the role played by the surrounding tissue and the surrounding vessels in the regulation and expression of the various genes.

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Finally, damaged repair systems in some hereditary diseases such as hereditary hemorrhagic telangiectasia type 1 with endoglin and transforming growth factor-β1 do not produce arterial aneurysms but rather different types of vascular alterations. One can envision the maturation over time of some genetic programs of modeling and remodeling of the brain vessels. The cell turnover and repair capacities are unlikely to be continuous and spread over an equal period throughout life. Postnatal...
maturation represents additional challenges that one should foresee in interpreting gene-expression disorders.

All these remarks are not meant to be restrictive for this extremely interesting paper but rather should stress the need to establish links between clinical observations and biological or genetic ones to ensure a rapid benefit in the treatment of patients.

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How Safe Is Carotid Angioplasty and Stenting in NASCET-Ineligible Patients?

To the Editor:

In a recent article, Malek et al1 reported 3.6% risk of stroke and death after percutaneous transluminal carotid angioplasty and stenting (PTCAS) in symptomatic patients ineligible for carotid endarterectomy (CEA) according to North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria. The authors achieved highly successful results in a subgroup of patients who were at high risk of surgical complications.

We conducted a retrospective, independent, third-party audit of medical records of patients undergoing PTCAS in a teaching hospital of University of Toronto. From 1996 to 1999, 55 PTCAS were performed in 50 patients with high-grade (>60%) internal carotid artery (ICA) stenosis. Mean age of patients was 68 ± 11 years, and there were twice as many males: 45% of patients had hypertension, 33% had diabetes, 88% had coronary artery disease, and 36% had contralateral ICA occlusion.

Thirty-three patients were symptomatic and 17 had asymptomatic ICA disease. According to NASCET criteria, 28 symptomatic patients were ineligible for CEA, leaving 5 patients eligible for carotid surgery. The major reason for NASCET ineligibility was unstable angina (n = 14), followed by age > 80 years (n = 5) and previous CEA (n = 5); 2 patients were unstable neurologically, 1 patient had cancer; and 1 patient had unstable congestive heart failure.

In the NASCET-ineligible cohort, there were 3 nonfatal periprocedural strokes (10.7%) and 2 deaths (7.1%), with a combined complication rate of stroke and death of 17.8%. Of 3 nonfatal strokes, 2 were major (Rankin scores of 4 and 5), and 1 patient had minor stroke (Rankin score 1). The cause of death in both cases was stroke: 1 patient developed stroke immediately after a procedure, and another patient had fatal stroke at day 6. In patients with periprocedural complications, the major reason for NASCET ineligibility was age > 80 years. Only a 77-year-old male patient with minor periprocedural stroke was rejected from CEA because of unstable angina. No complication was observed in 5 patients who were eligible for CEA.

We found an almost 5 times higher periprocedural risk of stroke and death associated with PTCAS in patients compatible to a cohort reported by Malek et al. In our population, the major cause of surgical rejection was unstable coronary artery disease (50%), whereas in the cohort reported by Malek et al, the major condition leading to exclusion from surgery was age > 80 years (21%). However, while this was the reason for excluding these patients from NASCET, most surgeons will operate on patients over this age if they are fit for surgery. In view of this, a 21% exclusion rate on the basis of age alone seems rather high. This may relate to the higher procedural complications observed in our series, but none of our patients died from myocardial infarction, and the only cause of death was stroke. It is also possible that our cohort had more severe ICA disease as measured by Sundt and European Carotid Surgery Trial (ESCT)/Rothwell scores of risks3,4; however, because of the retrospective nature of this study, we did not record plaque surface irregularity, which is a major component of those scores.

We believe that the higher complication rate in our series is not related to the technique of PTCAS. No complication was documented in a series of PTACS in patients who were eligible for CEA according to NASCET criteria. There were no major differences in the performance of PTCAS, or in postprocedural management of patients, including use of Schneider’s Wallstent, routine administration of heparin, and in postinterventional prescription of clopidogrel or ticlopidine in combination with aspirin. We were unable to demonstrate a “learning curve” in the performance of PTCAS, with less morbidity and mortality in recently performed procedures. The complications were equally distributed through 1996 to 1999.

Finally, we disagree with the author’s conclusion that PTCAS can be performed in high-risk patients rejected by surgeons with a complication rate compatible with published CEA series. In our series, the complication rate of 17.8% was twice that of the most severely affected patients with a Rothwell/ECST score of 5, and with a 5-year risk of stroke about 38%. Only a multicenter, randomized clinical trial, in which patients not eligible for CEA will be randomized into medical and PTCAS groups, may address the question of the superiority of PTACS compared with best medical treatment.

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