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of cases. We believe that, for aneurysms arising proximal to the anterior carotid siphon, therapeutic intervention should be reserved for patients with severe trigeminal pain, progressive ophthalmoplegia, progressive visual loss, or evidence of progressive aneurysmal enlargement in symptomatic patients.

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References

RESPONSE: Our data clearly demonstrate that developmental aneurysms arising from the cavernous carotid artery have an extremely low risk (1.4%) of developing a subarachnoid hemorrhage. We excluded aneurysms that arose from the distal aspect of the ascending internal carotid artery (ICA) because this segment of the carotid artery is not in the cavernous sinus. New work on this clinoidal segment of the ICA has confirmed this knowledge. Using careful neurosurgical dissection, Dr. Arthur Day has determined that the clinoidal portion of the ascending ICA is located outside the confines of the cavernous sinus (data presented at the 1993 annual meeting of the Neurosurgical Society of America, Boca Raton, Florida). The dural reflection from the inferior anterior clinoid extends just above the cavernous sinus to envelop the oculomotor nerve, and this thin membrane has been termed the “carotid oculomotor membrane.” Above this membrane, the ascending ICA is still not in the subarachnoid space but it is outside the cavernous sinus. It then penetrates the dura that extends from the anterior clinoid to surround the penetrating ICA to form a dural ring. Superior to this dura, the ICA is not within the cavernous sinus.

Clearly, an aneurysm with a neck that remains below the exit of the ICA from the cavernous sinus has more membrane restricting extension into the subarachnoid space. An aneurysm that arises above the cavernous sinus, even if it is still inferior to the dural ring, has less dura to restrict this extension to the subarachnoid space; these aneurysms are associated with subarachnoid hemorhage and visual loss but not with painful ophthalmoplegia. Thus, the neuroanatomy and the clinical distinction between these groups of aneurysms are closely correlated. It is not appropriate therefore to lump all aneurysms that involve the carotid artery in the cavernous sinus “area” together in terms of treatment consideration since their natural history differs.

We would like to thank Drs. Linskey and Sekhar for affording us the opportunity to restate and further clarify this important clinically applicable principle.

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The Myth of Eloquent Cortex, or What is Non-Eloquent Cortex?

TO THE EDITOR: In this so-called “Decade of the Brain,” when great efforts are invested in studying the intricacies and subtleties of the human cerebrum, it is somewhat curious that neurosurgeons have so eagerly adopted the term “eloquent cortex.” Indeed, it has become virtually impossible to read the Journal of Neurosurgery these days without encountering “eloquent cortex” or its elusive counterpart “non-eloquent cortex.” Does “eloquent” refer to that attribute of persons defined in the Oxford Dictionary as “possessing or exercising the power of fluent, forcible, and appropriate expression,” or does it mean “indispensable”? Are we to surmise that there are only two kinds of cortex, “eloquent” which has to be preserved, and “non-eloquent” which can be dispensed with? If so, where does non-eloquent cortex reside? Is parahippocampal gyrus “non-eloquent”? Are right frontal or right parietal cortices “non-eloquent”? If “eloquent” indicates the essential nature of this cortex, should the term then be extended to other areas of the central nervous system, hence “eloquent pons” and “eloquent spinal cord”?

Trying to trace the origin of this curious terminology proves difficult. It has frequently surfaced in the literature on arteriovenous malformation (AVM). Drake used this term rather extensively in his discussion of cerebral AVM’s. Yaşargil expressed distaste for such terminology when he stated, “With regard to location of AVMs, terms such as eloquent and non-eloquent, functional, critical and silent are best forgotten.” This terminology was also utilized by Spetzler and Martin in a proposed grading system for AVM’s, where the authors defined eloquent brain areas as “those that speak to readily identifiable neurological function and, if injured, result in a disabling neurologic deficit.” These authors considered the eloquent regions of the cerebral cortex to be merely the sensorimotor areas, language areas, and primary visual areas. Conspicuously missing from this privileged list are regions such as the right parietal cortex, where damage could result in a profoundly disabling syndrome.

The danger of terminology of this kind is that it eventually replaces the neurological reality it sets out to simplify. The neurosurgeon who readily accepts it might
be tempted to believe that "non-eloquent" cortex could be removed without consequences. Indeed, a much deeper problem lies here. The criteria for neurosurgical success have for a long time been based on the brief bedside neurological examination. Patients who passed this examination could still be considered "neurologically intact" even if they were neuropsychologically devastated. The outcome, to personality and memory, of removal of seemingly "non-eloquent" cortex, such as right frontal regions or parahippocampal gyrus, is far from benign. Perhaps with improvement in standards of evaluation of neurosurgical outcome, no region of cortex could any longer be considered "non-eloquent." As a first step, this terminology should be excised from the neurosurgical literature.

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References

RESPONSE: I first used the word "eloquent" in remarks at the conclusion of a series of lectures by Dr. Wilder Penfield given to the general student body of the University of Western Ontario in the 1960's. Having been asked, as a young and only neurosurgeon in the large audience in the Great Hall to thank this great man, I gave some thought to my words. Previously at tea, after I had vented my frustrations at not being able to spend more time in the laboratory, he told me that if he had made any mark in neurosurgery, it was not with his earlier laboratory studies in anatomy and pathology but rather with his studies of brain functions in humans with epilepsy, by electrical stimulation of their exposed brains and then careful removal of epileptogenic brain tissue, even in or near the Rolandic or dominant Sylvian regions, while they were awake under local anesthesia. After speaking of his gratitude and in tribute to these patients who had allowed him to do this, he told me that with the wealth of clinical problems I faced I might not be able to pursue my neurophysiological interests and that perhaps I could use the operating room as he did, for after all there were no animal models for the cerebral aneurysms, arteriovenous malformations, and basal tumors of my concern. But I was to use whatever knowledge was available and, with reason, caution, and best judgment (what he called "a surgical conscience"), approach these uniquely human problems directly. Perhaps this is why he wrote later, "the opportunity to learn walks with any surgeon who enters the operating room with questions on his mind."

At any rate, in my thanks, I said how fortunate he had been in becoming deeply involved with "that most eloquent of all neurological disorders, epilepsy." Afterward, to my surprise, he walked to the back of the Hall where I was seated to thank me but particularly to mention his pleasure at my use of the term eloquent for epilepsy. I might have thought no more about the word except for this episode. Coming from such a man made it linger in my memory and come to mind when I was debating how far I might go with large arteriovenous malformations in Rolandic and Sylvian regions.

I do not believe I can or should retract or even perhaps ask for forgiveness for personalizing those areas of the human cerebral cortex that have such meaning for neurosurgeons and include much more than that around the major fissures.

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Myelography Still Useful

To The Editor: With reference to the article by Caputy and Luessenhop (Caputy AJ, Luessenhop AJ: Long-term evaluation of decompressive surgery for degenerative lumbar stenosis. J Neurosurg 77:669–676, November, 1992), I would like to indicate that I agree with their recommendation that myelography is essential for the full evaluation of patients with symptoms of degenerative lumbar stenosis. Myelography, and it alone, seems to give the best view from the surgeon's perspective of the amount of the lateral decompression needed from a rostral to a caudal direction.

In addition, it would seem that very careful attention should be paid, particularly in some patients, to the vascular system. We have recently treated three patients with vascular disease identified in the lower extremities; however, they were judged to have satisfactory blood flow in the lower extremities, which implicated more indirectly the back as the cause of their symptoms. In two of these patients the myelogram was normal, and subsequent testing by measurement of blood pressure in the lower extremities before and after treadmill exercise demonstrated that the blood pressure dropped significantly with the exercise, indicating significant arterial obstruction despite good blood pressure initially and good flow on Doppler ultrasound studies.

The third patient had fairly classical pseudoclaudivatio; the blood vessels were thought to be poor but good enough on the basis of Doppler ultrasound studies and blood pressure measurements in the legs not to explain his symptoms. The myelogram showed marked stenosis, which was corrected surgically, and his symptoms disappeared for about a month, only to recur. The patient underwent repeat myelography and the previous defects were not present. At that point, blood pressure measurements in the thigh, leg, and ankle, before and after exercise, demonstrated the real culprit, which was a vascular occlusive disease.

This information may be of interest to your readers, as the test to confirm severe vascular stenosis is not