Guest Editorial

Occlusive hyperemia: a new way to think about an old problem

CHARLES B. WILSON, M.D., AND GRANT HESHIIMA, M.D.

Departments of Neurological Surgery and Radiology, School of Medicine, University of California, San Francisco, California

A. Al-Rodhan and his coworkers at the Mayo Clinic have introduced the new term "occlusive hyperemia" to explain on a hemodynamic basis the edema, either with or without hemorrhage, that may complicate excision of arteriovenous malformations (AVM's), usually larger AVM's with high flows. An identical occlusive hyperemia may follow preoperative embolization and, in our institution for one, postembolization occlusive hyperemia has occurred more often than postexcision occlusive hyperemia. In fact, we have encountered a handful of cases in which occlusive hyperemia (cerebral edema with associated hemorrhage) caused abrupt neurological deterioration after spontaneous thrombosis of a major draining vein, a condition that we have termed "venous overload." Venous overload is a common accompaniment of both symptomatic and asymptomatic AVM's, with the pathognomonic findings of venous outflow restriction and aberrant venous drainage patterns shown by angiography and findings of cerebral edema demonstrated on computerized tomography and magnetic resonance (MR) imaging. In our opinion, the evolution of a nidus aneurysm or angiographically obvious venous outflow restriction (or both) precedes rupture of many, if not most, AVM's that have spontaneous hemorrhage as their mode of presentation. Finally, precisely the same manifestations of occlusive hyperemia accompany dural arteriovenous fistulas (AVF's) when venous outflow becomes restricted and eventually occluded. Apart from ruptured aneurysms, interference with venous drainage represents the single factor responsible for almost all clinically significant complications of AVM's and AVF's preceding, during, and following treatment.

Restriction of venous outflow becomes the most critical determinant of what happens within and surrounding an AVM nidus. What are the exceptions? First, rupture of an AVM nidus (which, incidentally, is believed to occur nearer the venous than the arterial "end" of the nidus) can undoubtedly occur in the absence of any venous occlusive process. Second, pure postoperative hemorrhage from a retained portion of an AVM represents rebleeding, whether it is a matter of blowing out a clotted plug or of arterial overload as blood is diverted to the retained nidus. However, in the instance of a retained nitus coupled with venous restriction, the venous overload (occlusive hyperemia) becomes malignant! To eliminate or reduce the possibility of a retained nidus, we rely on intraoperative postexcision angiography and strongly recommend it.

Al-Rodhan, et al., do not speculate on the wide range of intervals, from less than 3 hours to 11 days, between the time the patient leaves the operating room and the onset of neurological deterioration. We suspect that early deterioration, possibly during the 1st day or so, reflects differing degrees of venous outflow restriction, and that later deterioration, certainly after several days, can be attributed to outright delayed thrombosis of one or more critical draining veins or even a dural venous sinus. Clearly, the matter of early versus late delay is not a black-and-white issue, but at least the possible mechanisms fit more or less into such a pattern.

Stagnation of arterial flow may be difficult to characterize in a background of postoperative edema, intraoperative occlusion of feeding arteries, and coexisting venous obstruction. We have the impression that stagnation of arterial flow is common in the immediate postoperative period and more likely to occur after resection of larger AVM's.

The person directing postoperative care may unwittingly promote venous thrombosis by "keeping the patient dry" as a well-accepted means of reducing postoperative reactive edema. Better postoperative management would be to assure an adequate blood volume, assiduously avoid hemocoagulation or even a hematocrit over 35%, and immediately after the operation commence a course of aspirin and low-dose heparin therapy. The known and unknown factors that cause spontaneous extracranial venous thrombosis after craniotomy must apply intracranially as well.

Al-Rodhan, et al., present two cases as examples, and both cases deserve comment. In Case 1 the respon-
sible neuroradiologist and the neurosurgeon carried out preoperative embolization in a patient with a previously ruptured aneurysm. The predictable hemodynamic changes after embolization did not lead to rupture of the aneurysm in this case, but very well could have. Even if the aneurysm had not ruptured previously, we would prefer to clip the aneurysm before undertaking embolization. One should never criticize success, but it is fair to commend the authors on good fortune. Admittedly, postembolization rupture of an associated aneurysm occurs infrequently, but it does happen and it is preventable. When the approach to the aneurysm can be made safely, which it cannot if access is blocked by large draining veins, we modify the craniotomy so that both clipping of the aneurysm and resection of the AVM can be performed through a single, usually large, craniotomy. Case 2 underscores the syndrome of venous overload: a pattern of venous outflow restriction with aberrant drainage, demonstrated in their Fig. 4, and progressive neurological impairment in the form of impaired speech and short-term memory. Often in such a patient the T2-weighted MR images show cerebral edema caused by the venous stagnation.

So where does this leave the theory of normal perfusion pressure breakthrough? Spetzler, et al., 2 never said that normal perfusion pressure breakthrough was either common or a universal explanation for intra- or postoperative edema and hemorrhage. They came up with the idea after observing two of our intraoperative disasters and thinking about possible mechanisms. Restoration of CO2 reactivity in the brain tissue around an excised AVM may not be the same as restoration of all autoregulatory function, and the slowed postexcision circulation time can be attributed to smaller vessel vasodilation as well as a reduced arterial gradient. Parthenetically, venous overload also slows cerebral circulation. We believe that normal perfusion pressure breakthrough exists, although the animal model supporting Spetzler's theory deviates from the reality of surgical interference with an intracerebral arteriovenous shunt. In some cases it is very likely that a combination of occlusive hyperemia and normal perfusion pressure breakthrough explains the complication produced by embolization and excision of AVM's.

What is the lesson that the reader should learn from this seminal communication from Rochester, Minnesota? Become a vein watcher, and begin now to respect the venous end of AVM's.

Morgan and his colleagues from Sydney have a closely related article in this issue. We take exception to their conclusion that one-half of all deaths in their series were caused by normal perfusion pressure breakthrough, for the reasons that have been given already. That one-half of the deaths were attributable to hemodynamic factors is undisputed; rather, it is the label and the cited underlying mechanism to which we object. No one will be surprised by their finding that complications are more likely to occur in patients who have preoperative neurological impairment and large AVM's. Morgan, et al., discuss complications of management and do it in an instructive manner.

The outlook for patients with AVM's has improved with the availability of MR imaging and superselective catheters for angiographic isolation and embolization of AVM components. We surgeons are now smarter because the literature contains critical information about the relevant cerebrovascular physiology of AVM's. Improved neuroanesthesia and more user-friendly surgical microscopes help, but the major contributor to better outcomes is a new breed of interventional neuroradiologists, some of whom are also neurosurgeons. Finally, radiosurgery is an exciting new modality for the treatment of selected AVM's. However, in using this relatively new and powerful nonsurgical form of therapy, the temptation to broaden indications could lead to overuse that in the extreme could be a sadly misdirected application of this sophisticated, technically precise therapeutic tool.

References


Address reprint requests to: Charles B. Wilson, M.D., University of California, Department of Neurological Surgery, c/o The Editorial Office, 1360 Ninth Avenue, Suite 210, San Francisco, California 94122.