Temporal lobe arteriovenous malformations

ROBERTO C. HEROS, M.D.

Department of Neurosurgery, University of Miami, Florida

This issue of JNS includes a beautifully illustrated paper by Gabarrós Canals and colleagues summarizing the surgical experience of the senior author, Dr. Lawton, with 88 patients with temporal lobe arteriovenous malformations (AVMs) treated surgically over a 13-year period. This is quite a remarkable experience, which makes the “surgical pearls” included in that paper particularly useful. The article includes beautiful drawings and operative photographs that I’m sure readers would find not only attractive, but also very useful. Not surprisingly, considering the well-known surgical skill of Dr. Lawton, the results were excellent, with 87% of the patients having a good outcome at follow-up. There were four deaths, but two of them were due to the presenting hemorrhage and one was from an unrelated cause. Although the authors do not mention specifically the incidence of a partial field cut (superior quadrant anopia) from injury to the Meyer loop, I suspect that this occurred with some frequency because I know it has occurred in my own series of temporal lobe AVMs. I suspect that some of these instances were still included under “good results,” with which I agree fully, given that this type of field cut is rarely incapacitating and in fact many patients learn to ignore it with time. Interestingly, the only one of my patients that I remember for whom a superior quadrant anopia was a significant problem was a tennis player who would “lose the ball” while trying to serve!

In their article, Dr. Lawton and his colleagues focus their discussion on a classification of these lesions that depends on where the predominance of the AVM is located. Frankly, I am not sure that this or several other classifications that have been proposed are very useful clinically in that many of these AVMs overlap the different classifications; for example, the large lateral temporal AVMs frequently have a basal component fed by posterior cerebral artery (PCA) branches. The sylvian AVMs clearly can be purely within the sylvian fissure, but frequently involve either the temporal or the frontal opercula, or both. The intraventricular AVMs are rarely purely intraventricular (except those that are strictly confined to the choroid plexus) and most frequently involve other deep portions of the temporal lobe, and so on. Nevertheless, I agree that for descriptive purposes these classifications have a role. Therefore, I will use the authors’ classification to make some comments based on my own surgical experience with these AVMs, which will frequently be redundant, since the article in question is very comprehensive.

Lateral temporal AVMs should be exposed, as recommended by Dr. Lawton, with a pterional craniotomy for the more anterior lesions and a standard temporal craniotomy for the middle and posterior temporal lesions. For the anterior temporal lesions, when the AVM reaches the inferior surface of the temporal lobe, I prefer to use the combined pterional/anterior temporal approach that I have described mainly for the approach to basilar aneurysms. With this exposure, one has an excellent view not only of the anterior temporal pole, but also of the anteroinferior portion of the temporal lobe. As stated before, these lateral temporal AVMs, when large, frequently reach the basal aspect of the temporal lobe, and in those cases they acquire a significant PCA supply. I have found it particularly useful to embolize the PCA supply as thoroughly as possible, which when successful makes it unnecessary to retract the temporal lobe to control the PCA feeding vessels subtemporally. The middle cerebral artery (MCA) feeding vessels are readily accessible on the surface or in the sylvian fissure; it is not necessary to take the risk of embolizing these MCA feeders to which the surgeon has early, easy access.

Dr. Lawton found language mapping with the patient awake to be useful in some of these more posteriorly located AVMs in the dominant hemisphere. Clearly, preoperative definition of the critical speech areas by functional MRI studies or other means is useful in determining whether the AVM is operable, but I fail to see how intraoperative monitoring, once the dissection of the AVM starts, is very useful because once the surgeon begins to dissect the AVM he is usually committed to removing it—as opposed to tumor surgery, where the surgeon can stop the operation and leave tumor behind. Obviously AVMs do not lend themselves to this option. Perhaps Dr. Lawton has used mapping intraoperatively to decide whether to resect the AVM or whether to “back off” if it is found that the lesion clearly involves eloquent speech areas.
The AVMs predominantly located in the basal aspect of the temporal lobe against the tentorium of the middle cranial fossa are predominantly supplied by PCA feeders, and, again, I have found it extremely useful to perform as thorough an embolization of these feeding vessels as possible. This eliminates the need for early subtemporal retraction, which in at least one of my cases led to a catastrophic and difficult to control hemorrhage from accidental tearing of a large arterialized vein that went directly from the undersurface of the temporal lobe to a large venous channel in the tentorium. If the PCA supply is thoroughly embolized, the surgeon can work trans-temporally to control the MCA feeders that these lesions frequently have and to define the anterior, superior, and posterior limits of the AVM, leaving the lesion against the tentorium as the surgeon develops the medial aspect, which is the most difficult. After the lesion is completely dearterialized it can then be removed from the tentorium, because any draining veins in this area would already be “blue” and under low pressure. Dr. Lawton tells us that he found meningeal feeding vessels in approximately one-fourth of these basal AVMs. I found this interesting because in my experience, meningeal feeders with parenchymal AVMs occurred almost exclusively in cases in which there had been a previous hemorrhage or embolization. I suspect that the inflammatory reaction caused by both of these circumstances may be responsible for the acquisition of dural feeders.

The medial temporal AVMs are clearly the most difficult, particularly when large. For me it is interesting to reflect on the difference made by modern imaging in terms of being able to ascertain exactly where the AVM is located preoperatively. One of my earliest articles on AVMs of the medial temporal lobe in the days before MRI studies focused precisely on the issue of defining, on the basis of catheter angiography, whether the AVM was “operable” or whether extensions superomedial to the amygdala into the basal ganglia made the lesion either much more dangerous to resect or, frankly, “inoperable.” Obviously, nowadays the MRI findings define precisely the extension of the AVM, and this exquisite attention to the angiographic anatomy becomes less important in this regard. Like Dr. Lawton, I approach the more anterior medial temporal AVMs through a wide split of the sylvian fissure to control sequentially the early MCA feeders, the anterior choroidal artery (AChA) feeders, the occasional small feeding vessels from the posterior communicating artery, and, finally, the anterior temporal feeders from the PCA. Because these lesions almost always drain medially and posteriorly into the basal vein, it is sometimes possible to sacrifice arterialized veins that drain anteriorly into the sphenoparietal sinus and the cavernous sinus to be able to expose the medial surface of the temporal lobe. To this effect, I agree 100% with Dr. Lawton that lateral temporal retraction is much better tolerated than the superior retraction that is necessary with the subtemporal approach.

The more posteriorly located medial temporal AVMs have to be approached, as indicated by Dr. Lawton, trans-temporally. When I first wrote about these AVMs, I recommended entering the temporal horn through a cortical incision in the inferior temporal gyrus, as described by Dr. Lawton, and then simply taking the AChA after it entered the choroidal fissure, given that this artery is frequently one of the predominant feeders to these middle and posterior medial temporal AVMs. I thought then that the AChA could be sacrificed as it entered the choroidal fissure because all the medial branches to the basal ganglia had already been given by its cisternal segment. However, I learned subsequently from Dr. Rhoton that in fact there are branches given by the AChA that exit the temporal lobe and go to the optic tract and the basal ganglia after the artery has entered the choroidal fissure.

Although in my initial series of AVMs I had no problem from this maneuver, I recently encountered a case in which I clipped the AChA as it entered the choroidal fissure to feed a large medial temporal AVM, and the patient ended up with a most disabling stroke in the posterior limb of the internal capsule that I have to attribute to that maneuver; therefore, caution in this respect is desirable. If it is possible to take the branches of the AChA to the AVM and preserve the main trunk, an effort should be made to do so, although I must admit that this is frequently difficult. Dr. Lawton tells us that he excluded insular AVMs from this series of “temporal” AVMs, but I am sure he has encountered some patients in whom these medial temporal AVMs extend across the temporal stem into the insula. I have encountered a few of these lesions and they can be extremely difficult and frequently require a combined approach through a wide opening of the sylvian fissure and a more inferior transtemporal approach.

Sylvian AVMs can be purely within the sylvian fissure and in this case they can be removed completely, even in the dominant hemisphere, without a resulting speech deficit. I have found it interesting how opening the sylvian fissure actually becomes easier in these cases in which the lesion is located purely within the subarachnoid space of the sylvian fissure. Generally the loops of the AVM are surrounded by generous amounts of CSF, which facilitates their exposure. However, these lesions frequently involve either the frontal or the temporal operculum, and this makes them inoperable when located posteriorly in the dominant hemisphere. The MRI modality is extremely useful in determining whether the lesion is purely sylvian or whether it involves the opercular parenchyma of either the frontal or the temporal lobe, or both.

I agree with Dr. Lawton that intraventricular temporal AVMs are rather uncommon, but I remember at least one case that was entirely confined to the choroid plexus of the temporal horn. Obviously these AVMs are relatively straightforward to remove through a transcortical approach to the ventricle through the inferior temporal lobe as recommended by Dr. Lawton. Once the AChA is controlled anteriorly at the choroidal fissure, the AVM can be easily removed simply by removing the choroid plexus with the AVM in an anteroposterior direction.

I will finish by congratulating Dr. Lawton and his colleagues for an excellent article. It is true that many of us have written about these AVMs and that the techniques used by Dr. Lawton are not new, but it is very nice to see a comprehensive discussion in one article of all the nuances related to the surgery of these rather common AVMs.

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Editorial

Disclosure

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References


Response

MICHAEL T. LAWTON, M.D.
Department of Neurosurgery, University of California, San Francisco, California

The generous comments of this master neurosurgeon are appreciated. Few neurosurgeons reach the rarefied air of Dr. Heros’ AVM experience and even fewer can distill the wisdom with his clarity. I have always learned from his lectures, lessons, and publications on AVMs, and as his list of references demonstrates, there are many. I am relieved that many of his comments agree with our findings and that I will not have to respond to major criticisms.

First, I agree with Dr. Heros that AVMs do not respect the boundaries and labels in classification schemes. In fact, AVMs are an unruly lot with tremendous variability and overlap, extending across brain surfaces and lobes without regard for neat definitions. Of course I have seen examples of lateral temporal AVMs that wrap underneath to the basal surface and medial temporal AVMs that extend into the insula. Consequently, AVMs have defied these kinds of classifications. For most of my career I have viewed each one as unique, with a different mixture of feeding arteries, draining veins, nidus anatomy, and patient characteristics. However, I have come to believe that this perspective does not facilitate the learning process or strategic planning for AVM resection. Furthermore, I believe we need to rethink AVMs as a finite collection of recurring AVM subtypes that can be defined, characterized, and identified clinically again and again. Although overlap between AVM subtypes is undeniable, this perspective will help aspiring AVM neurosurgeons figure out AVM surgery and progress along their learning curve. The harmony between Dr. Heros’ and our observations on temporal AVMs speaks to the validity of this new perspective.

Second, I agree with Dr. Heros that mapping plays a limited role in temporal AVM surgery. It is true that one cannot partially resect an AVM like a tumor. However, some AVMs sit beneath the cortical surface and some tissue transgression may be required to expose the nidus. In these cases it helps to localize language function exactly. Language function is one of those sacred areas of eloquence, beyond others like somatosensory cortex or deep cerebellar nuclei, that demands extra protection in the dominant temporal lobe. Furthermore, the deep planes of an AVM adjacent to or beneath the Broca center may be diffuse or unfriendly, drawing the dissection into language areas and producing electrophysiological changes. In these cases, the goal of complete resection may be abandoned in favor of incomplete circumsedation and postoperative radiosurgery. Nonetheless, our use of speech mapping was limited to a handful of patients, and I find myself using it decreasingly as my surgical experience increases.

Dr. Heros’ pearl about the branches arising from the plexal segment of the AChA supplying the optic tract, basal ganglia, and internal capsule is important. So is his pearl about the inoperability of sylvian AVMs involving both temporal and frontal opercula in the dominant hemisphere. His enthusiasm for the nuances of AVM surgery is obvious in his editorial. From one AVM surgeon to another, I know this excitement to teach is born of some suffering in the operating room. These can be challenging operations with the capacity to inflict pain on the neurosurgeon and morbidity on the patient. Strategic planning and meticulous surgical execution are critical in battling this opponent and make the insights derived from experience so invaluable.

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