Bervini et al. have provided us with an excellent retrospective review of Professor Morgan’s outstanding prospective database on cerebral arteriovenous malformations (AVMs) studying the issue of transdural arterial recruitment (TDAR) to brain AVMs. I should comment that this may be the largest and most comprehensive database on a single-surgeon series of cerebral AVMs. We have learned much about these lesions from different reviews of this database for the study of different aspects of cerebral AVMs. For this particular study Dr. Morgan’s team selected 769 patients who had no history of partial treatment for their AVM and who had a complete digital subtraction angiography (DSA) assessment. Of this group 6.6% of the patients had TDAR, and the comparison of these patients with the rest of the group forms the basis of this report.

Basically, after a well-performed multivariate analysis, the factors that the authors found to have a positive statistical correlation with the presence of TDAR were as follows: increased age, increased AVM size, a combination of anterior and posterior circulation arterial supply, and, interestingly, presentation with a neurological deficit without hemorrhage. The correlation with size is not surprising. Also, it is no surprise that increasing age correlates with TDAR; most of us believe that the development of TDAR is something that occurs secondarily rather than being a primary feature of cerebral AVMs. The authors comment on that, and offer the fact that none of the very young patients in their series had TDAR as confirmation of this belief. The correlation with the presence of supply both from the anterior and posterior circulation is also not surprising, and I suspect this is a function of size—all though it did come out as an independent factor in multifactorial analysis. To me the most surprising correlation is that of presentation with a neurological deficit without hemorrhage, and I will comment further on this.

The main reason I agreed to comment on this paper, other than to praise the quality of the work by this group, is the fact that I have had a somewhat different, or I should rather say complementary, impression about this issue. I should acknowledge, however, that my impressions are based on simple personal experience rather than a scientific review of my personal series of AVMs, which, although significant, is less extensive than that of Professor Morgan. The factors that through the years I have come to associate with the development of TDAR are these: a superficial location of the AVM, previous (in the past and not immediately preoperative) embolization, and a history of previous hemorrhage or previous surgery. That the AVM has to have a superficial location in contact with the dura mater to develop TDAR is obvious, and I am not sure why that was not one of the factors that Bervini and colleagues found to be of significance; it may simply be that they did not specifically look at this. In terms of embolization, it is a shame that Bervini and colleagues specifically excluded from analysis patients who had had previous partial embolization. It has been my contention that embolization with glue or Onyx leads to some sort of inflammatory reaction in AVMs that have a superficial location that in turn leads to the development of TDAR. I cannot prove this, but I am convinced that I see this problem much more commonly in patients who have had embolization in the past (not in the few days before surgery). In fact, I make a point of always being extremely careful opening the dura on these patients because of my certainty that they will have these difficult to deal with vascular connections between the AVM and the dura.

I have become convinced that those patients in my series who had an old hemorrhage were more likely to have TDAR. I always explained this fact to myself by thinking that something has to disrupt the normal leptomeningeal/CSF barrier between the superficial representation of the
AVM and the dura. This is also the explanation for the fact that in my experience those patients who have previously undergone operation are more likely to have TDAR, although my experience with patients who have had previous surgery for either partial removal of the AVM, feeder ligation, or any other reason is of course relatively limited. I do not have a good explanation for why in Dr. Morgan’s series a history of hemorrhage did not turn up as a significant factor for the development of TDAR. In fact, these authors specifically found that a history of neurological deficit without previous hemorrhage was a significant factor. Perhaps in their reply to this editorial they may comment on this issue.

Bervini et al. believe that the most likely explanation for the development of TDAR is that this is a secondary recruitment that results from increased shear stress in the primary arterial supply to the AVM. Their discussion in support of this theory is rather elegant, but I must confess that my simple mind is having some difficulty fully understanding it. They allude to elongation and tortuosity of the feeding arteries, which gradually brings them into contact with the dura and which then, through a process of angiogenesis and arteriogenesis, leads to the development of TDAR. I can easily visualize how these dilated arteries pulsating against the dura can produce a disruption of the normal leptomeningeal/CSF barrier and thus allow the development of TDAR. Beyond that, I admit, I cannot fully understand their proposed mechanism. Clearly, they do a very good job in their discussion in discarding other mechanisms such as one in which the TDAR is an intrinsic early feature of the malformation itself, or secondary ischemia as a result of steal into the malformation resulting in angiogenesis. I still think that inflammation brought about by embolization or disruption of the normal leptomeningeal barrier between the AVM and the dura by previous hemorrhage or surgery can be operative mechanisms in the development of TDAR.

Last, what is the importance of this phenomenon? I am convinced that the development of TDAR increases surgical difficulty, which Dr. Morgan and coauthors allude to. Interestingly, in their specific analysis of those patients from this series who had open surgery, they found no association between surgical complications and the presence of TDAR on multifactorial analysis. I suspect that this is largely due to the experience and skill of Dr. Morgan and the precautions that he takes—and describes very well in his article—when exposing cerebral AVMs with TDAR. If these precautions are not taken, catastrophe can occur from uncontrollable bleeding. I well remember a colleague’s case that occurred while I was in training. That patient exsanguinated from uncontrollable bleeding during the opening. To avoid this I have frequently had to use the maneuver described by Dr. Morgan of carefully opening the dura circumferentially around the AVM and leaving a piece of dura stuck to the lesion. I would repeat, however, that this phenomenon is not common in my experience, except for those cases that have had embolization in the past, surgery, or a hemorrhage.

This is an excellent article, and I congratulate the authors on a valuable contribution.

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References


Disclosures

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Response

Michael Kerin Morgan, MD

Department of Clinical Medicine, Macquarie University, Sydney, New South Wales, Australia

I am grateful for the chance to respond to the editorial review by Dr. Heros. The compliment paid to our work is very much appreciated, and I am most honored to have received this from such an outstanding surgeon and legend of cerebrovascular neurosurgery. I am not surprised that Dr. Heros found little that he did not already know in what we reported. Although what we have researched and demonstrated may be the first time that this has been reported in the literature, this work, and much of my previous work, confirmed insightful opinions, derived from thoughtful reflection on experience, from such outstanding cerebrovascular neurosurgeons as Drs. Sundt, Piepgras, Heros, Drake, and Spetzler. Having had the privilege to learn from and discuss their cases with these mentors and inspirational neurosurgeons helped me choose the selection of the variables that underpin my database established in the late 1980s. The database was used to challenge some of the doctrines espoused by these neurosurgeons who expressed their personal opinions. However, more often than not, our results confirmed that these doctrines divined from personal experience were insightfully brilliant.

There is no greater or more evident example of this than the Spetzler-Martin grading system. When it was published, the scale appeared to weight variables arbitrarily in a way that was not obvious (e.g., moving from a size < 3 cm to ≥ 3 cm was equivalent to the presence of either deep venous drainage or eloquent location in a small brain AVM [bAVM]). And what may not be appreciated by those who were not involved in neurosurgery in the 1980s was the fact that not all of the variables were immediately accepted as important (i.e., deep venous drainage).