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Eye-popping fistulas: what's in a name?

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The Danger of Air Bags for Children in the Front Seat

In this issue of the *American Journal of Neuroradiology*, Marshall et al report the patterns of injury found in children suffering severe or fatal air bag trauma during low-velocity motor vehicle accidents (page 1599). Their results suggest that head trauma is the most important mechanism of injury in infants in rear-facing seats, and that craniofacial and cervical spine injuries are most significant in older children. These results make explicit the widely reported risk of air bag inflation to infants and small children. The authors have provided radiologists with valuable information on the patterns of trauma found in these children. This report will help guide radiologic evaluation and raise awareness of the type of injuries most likely to occur. The workup may be further tailored if details of the accident (eg, velocity of the vehicle, whether the child was restrained or unrestrained) are known.

This article reinforces the warning that children should ride in the back seat, properly restrained. It is striking that the major risk to children who are in the front seat of air bag–equipped cars is to those who are not properly restrained in front-facing car seats or lap-and-shoulder belts. Practical considerations largely limited this study to severe or fatal injuries; thus, it is impossible to know what proportion of low-velocity accidents with air bag inflation is represented by the severe injuries Marshall and colleagues report. Although 128 excess child deaths have been estimated as a result of widespread use of passenger-side air bags, the reliability of this figure is unclear. This report also does not address the potential value of full-power air bags for children who are not properly restrained but riding in the front seat, or whether, considering accidents at all velocities, restrained children in the front seat are safer with a deactivated or an active air bag. It is well known that children are at substantially lower risk for death and injury in automobile accidents than are adults (1).

As is often the case, the authors are chasing a moving target. The coming improvements in auto safety and air-bag design Marshall et al cite most likely will alter the pattern and severity of injuries found in the future. Depowering of air bags may save the lives of children who might otherwise have died as a result of air-bag injury. However, some of these children may have suffered less severe but still significant brain injury, which would not have been detected had full-power air bags been in use in the passenger seat. In other words, although autopsy records of patients who have died of air-bag injuries may yield some clues, milder head trauma produces few pathologic changes, and those that do occur are detected only as a result of a focused search for these lesions. Therefore, mild brain injury, to the extent that it may have occurred with full-power air bags in patients who died of trauma outside the CNS, may not have been detected to date. Of course, such mild injuries are also more likely with lower-powered air bags.

The nature and relative frequency of injuries, in addition to their severity, may be altered through the replacement of current air bags with depowered versions. Thus, it is possible that the predominance of CNS injuries in severe or fatal air-bag trauma may not persist when depowered or "smart" bags are widely available. The authors may need to repeat this study in a few years to update the radiologic community on the changing distribution of injuries found as air-bag technology evolves.

Side-impact air bags may pose a further risk to children in the front seat. These systems have not been in widespread use long enough to determine whether they contribute to the danger or provide any net protection for these children. Although one would hope that children will rarely ride in the front seat of vehicles equipped with passenger and side-impact air bags, future studies are certain to reveal the pattern of injuries associated with this safety innovation.

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Eye-Popping Fistulas: What's in a Name?

Graybeards in our ranks may muse reflectively upon the discourse set forth by Komiyama and colleagues in this issue of the *American Journal of Neuroradiology* (page 1641), in which they confirm a well-known but poorly documented occurrence: that major craniofacial trauma resulting in severe epistaxis causes angiographically demonstrable arteriovenous fistulas of the cavernous sinus. The fact is well known

among the arteriography teams who performed right brachial and carotid punctures to diagnose traumatic intracranial hematomas in the 1960s and 1970s, before the era of CT, and who, in the process, discovered a variety of vascular diseases, ranging from medusalike venous drainage of type A CCFs to faint contrast collections in the cavernous sinus region, collections whose precise source was difficult to determine on the unsubtracted or manually subtracted (as opposed to digitally subtracted) radiographs of the period. The advent of CT in the evaluation of acute trauma during the mid-1970s created a generation of neuroscientists with more limited experience in vascular evaluation in cases of hyperacute trauma. The small arteriovenous fistulas of the cavernous sinus (now known as indirect, dural, type B, C, or D CCFs) that were suggested in the pre-CT days were typically overshadowed by hematomas, which were frequently discovered and largely ignored, because, unlike their direct, type A, counterparts, seldom were symptomatic. The occurrence of dural fistulas in trauma series was poorly documented, so determining their precise frequency proved problematic. The article by Komiyama et al helps resolve that problem.

These authors discovered nine traumatic type B, C, or D CCFs during a review of more than 300 reported symptomatic dural CCFs, several of which were type C meningeal AVFs draining to the cavernous sinus. Although not statistically significant, and subject to wide standard deviation, my own data would lead me to believe that traumatic symptomatic dural CCFs may be an underreported phenomenon. As stated in this article, other asymptomatic dural CCFs previously had been seen acutely but were not quantitated. Personally, I recall that several disappeared on follow-up arteriography, including one in a young woman who lost light perception within 24 hours of sustaining a contralateral type A CCF.

In a separate communication, Dr. Komiyama (1) described one patient in whom a delayed direct type A CCF developed that was identified after discharge. However, documentation of such occurrences is still insufficient to suggest that direct CCFs arise after severe craniofacial trauma.

In emphasizing that the four traumatic CCFs found in their series were type B (as documented by their selective ECA injections showing no AVF flow), Komiyama et al give some credence to the as-yet-unproved theory that some direct type A CCFs might begin as tears of the dural branches of the ICA, and, if torn close to their origin, could enlarge the branch origins by increasing the flow into an ostium several millimeters in diameter, creating a direct, type A, CCF. To the best of my knowledge, such a case has yet to be well documented elsewhere.

The authors report a prevalence of 50% of type B CCFs in this selected group of patients in whom arteriography was performed. Of those CCFs, 75% did not become symptomatic; however, partial ECA embolization may have played some part in diminishing potential ECA supply to the AVF and perhaps may have altered the natural history by promoting healing. Nevertheless, there is some comfort in recognizing that patients so afflicted and so treated will presumably close the AVF on their own without any further direct treatment. Nonetheless, a persistence rate of 25% may represent a potential problem, and the inclination is to perform additional diagnostic tests to ascertain the presence of fistula occlusion. Certainly, MR angiography might be considered in follow-up studies, considering the number of reports of successful delineation of direct and dural CCFs available. But while the sensitivity of MR angiography for detecting symptomatic CCFs may seem high (2, 3), the specificity remains unclear.

With the help of Serge Ouananou, I reviewed 75 3D-TOF MR angiographic studies performed with scanners of two different manufacturers and found up to a 36% rate of occurrence of venous signal in the cavernous sinus extending to the inferior petrosal sinus in the absence of signs or symptoms of a CCF. Such a venous signal, although never as conspicuous as an arterial signal, is reminiscent of flow created by a posteriorly draining fistula and was more common at 42/6.9 (TR/TE) (36%) than at 35/2.5 (4%) with the use of flow compensation, 1-mm section thickness, 20° flip angle, MOTSA technique, and routine evaluation of both reconstructed MIP projections and source images. Therefore, MR angiography to exclude CCF may lead to false-positive findings, and this potential pitfall should be recognized. The dilemma of choosing between the risk of a silent CCF and the risk of a false-positive examination, which must be clarified by arteriography, can usually be satisfactorily resolved by fully considering the clinical and imaging information particular to each case.

A true carotid cavernous (sinus) fistula is the type A (direct) variety that is typically caused by trauma, occasionally from spontaneous rupture of a vessel or aneurysm, as the disease process was originally understood by Cushing, named by Hamby in his epic monograph, and elaborated upon by others. Should we call the type B, C, D (dural, indirect) CCFs by those terms or describe them as DAVFs of the cavernous sinus? In all fairness to those who used the term CCF but did not realize they were dealing, in some instances, with what we now also term DAVFs of the cavernous sinus, I favor using the abbreviation CCF under all circumstances, but always combined with the appropriate descriptor: type A, B, C, or D (and perhaps even D1 or D2, depending on whether flow is unilateral or bilateral), or spontaneous/traumatic, direct/indirect, high-flow/low-flow, dural, or some combination thereof. Whereas the abbreviation DAVF of the cavernous sinus is appropriate, this particular DAVF conjures up such a specific clinical syndrome that continued use of CCF remains totally appropriate.

In 1995, Parkinson (4) wrote that the venous space is neither cavernous nor a sinus but rather the venous plexus of the lateral compartment of the cavernous sinus, a contention that only further clouds the terminology issue. To make a push for more specific

terminology might be as futile as Sisyphus' eternal attempt to roll a boulder up a hill only to see it tumble down again. In the case of CCFs, perhaps with next month's Journal.

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A New Clinical Application of MR Spectroscopy in Hepatic Encephalopathy

One of the first disease processes shown to have an abnormal proton MR spectrum was hepatic encephalopathy (HE), in a study in which Ross et al found decreased *myo*-inositol (mI) (1). Subsequent studies of HE further documented the changes in mI as well as increases in glutamine/glutamate (Glx) and decreases in choline (Cho). However, the clinical role of MR spectroscopy in this condition, as well as others, has only slowly evolved for a number of reasons, technical as well as clinical. Initially, proton MR spectroscopy was limited to single-voxel methods and long-TE techniques; the former factor limiting spatial sampling, the latter factor limiting spectral analysis. Single-voxel techniques have now been supplemented by chemical-shift imaging techniques, which better allow the study of localized and heterogeneous lesions such as ischemia and tumors. Short-TE techniques, necessary for mI and Glx detection, are now widely available and increase the ability to study brain metabolism in more detail.

With technological advances, it is the search for useful applications that is now being pursued. In this issue of *The American Journal of Neuroradiology,* Haseler et al (page 1681) push MR spectroscopy nearer to being a necessary clinical tool. The utility of a medical technique may derive from a variety of attributes. The technique could provide new insight into the pathophysiology of a disease, improve diagnosis, or, as proposed in the article by these authors, better direct therapy. The use of imaging techniques to better direct or monitor therapy is becoming increasingly important in the clinical arena. For example, imaging findings as indicators of treatment outcome have recently played a critical role in the evaluation, and eventual FDA approval, of multiple sclerosis drugs (2). Haseler et al show that lactulose treatment of HE causes MR spectroscopic changes to return to normal. Specifically, a week of lactulose therapy increased mI and decreased Glx, coincident with improvement of clinical HE grade. Good-quality spectra clearly illustrate the differences between control subjects and HE patients before and after treatment. All articles on spectroscopy should include plots of primary data of this type. Importantly, the results suggest that MR spectroscopy may provide more sensitive markers of therapeutic response, at least in part because of the statistical advantage of using continuous variables instead of categorical clinical grades. The concordance of the MR spectroscopic and clinical response to therapy also suggests that the spectral changes, while incompletely understood, are not insignificant correlates of HE but rather reflections of metabolism related to cerebral dysfunction.

While these MR spectroscopic results are encouraging, more work is needed to solidify this clinical application. The use of spectral peak ratios, rather than absolute metabolic levels, remains suboptimal. Numerous publications have demonstrated the feasibility of metabolite quantitation with the use of proton MR spectroscopy, and these techniques should be applied in clinical studies. The sample size in the present study was small, only eight subjects actually completed the week of lactulose therapy. In addition, the possibility of sample bias exists, as only subjects with HE grade II/III were included in the treatment group. This restriction is appropriate for a pilot study, but MR spectroscopic findings of therapeutic response in a larger group of patients, including those with HE grades I through IV, are needed. A comparison of MR spectroscopic values with other HE-related variables, such as ammonia, is also needed. And longer follow-up studies are essential to determine the persistence of the MR spectroscopic mI and Glx findings and to ascertain whether this therapy can reverse the more subtle but longer lasting Cho changes.

If the MR spectroscopic responses to therapy presented in the present article are indeed corroborated by larger studies, then not only may the technique be