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Middle cranial fossa not temporal fossa.

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LETTERS

Carotid Sinus Syndrome Secondary to Iatrogenic Dissection of the Carotid Artery

We read with interest the article by Eskridge et al regarding carotid sinus syndrome associated with embolization procedures (1). The authors bring attention to an uncommon disorder, which is rarely seen in an everyday clinical practice. We would like to share a recent experience we had with one patient who developed carotid sinus syndrome as a complication of diagnostic angiography.

A 54-year-old woman was referred to us for carotid angiography because of a high-grade stenosis of her left internal carotid artery, which was previously detected by Doppler sonography. Catheter angiogram showed a greater than 70% irregular narrowing in the proximal left internal carotid artery and occlusion of the left external carotid artery. A catheter was placed in the proximal right common carotid artery (catheter/wire never crossed the bifurcation), and then oblique and frontal projections (using 6 cc/sec for a total of 12 cc) were obtained and were normal. During the last run (lateral view), the patient complained of neck pain and developed bradycardia (44 beats/min) and hypotension (70/40). Radiographs revealed a dissection of the distal common carotid artery extending into the internal carotid artery causing a narrowing of approximately 80%. Atropine and increased intravenous fluids were given, and the symptoms were temporarily reversed. Because of persistent bradycardia, atropine was continued for the next 24 hours. The patient remained neurologically asymptomatic during and after the procedure. Four days later she was discharged and is pending left carotid endarterectomy.

Our case illustrates that carotid sinus syndrome may be iatrogenically induced during conventional diagnostic angiography. Patients with carotid sinus syndrome secondary to atherosclerosis usually have bilateral disease (2). When one significant carotid stenosis is already present, the creation of a contralateral dissection compromises the function of both carotid sinuses, giving rise to the full syndrome. Carotid sinus syndrome in patients with bilateral stenoses may compromise further an already tenuous cerebral blood flow. The immediate goal in these patients is to alleviate hypotension and bradycardia in order to avoid neurologic deficits. This can be achieved by administering vagal inhibiting drugs (such as atropine), which increase heart rate and intravenous fluids to increase blood volume. Symptoms may, however, persist for an indeterminate period of time while normalization of baroreception is achieved. The diagnosis of carotid body syndrome is confirmed if the symptoms are reproduced by massaging the involved carotid artery. However, we believe that in patients with acute carotid artery dissection, this maneuver may be dangerous and should be avoided.

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Reply

Drs Smith and Castillo describe another interesting complication involving the carotid sinus syndrome. The authors were wise to recognize this syndrome at an early stage and institute preventive therapy. Problems related to carotid sinus syndrome may be more common than we think, especially when diagnostic angiographic procedures are considered. I, in fact, accidentally induced bradycardia in a patient with an occluded right common carotid artery during diagnostic angiography. The common carotid had been previously occluded because of atherosclerotic disease and the mere injection of nonionic contrast into the occluded segment of right common carotid artery induced bradycardia down to 20 beats/min. The catheter was immediately withdrawn, and the heart rate returned to normal before the patient became symptomatic.

It is important for anyone performing diagnostic angiography to be aware of this possible complication. I commend Drs Smith and Castillo for their prompt recognition and treatment of this problem.

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Hemangiomas and Vascular Malformations of the Head and Neck: MR Characterization

We recently viewed with great interest the paper by Baker et al (1). Our initial reaction was one of delight that

the Mulliken and Glowacki Biological Classification of Vascular Anomalies had been presented in the *AJNR* and appeared to correlate with MR findings for vascular lesions of the head and neck. After reading the paper we were surprised at the authors' characterization of hemangiomas as "low-flow" vascular lesions, particularly in view of the literature cited and quoted.

Baker et al based their work on the Mulliken and Glowacki classification (clinical behavior and endothelial cell characteristics), as well as on published "vascular flow characteristics" (2, 3). The authors also cited prior publications addressing the imaging findings in vascular anomalies, including angiography, CT, and MR (2, 4, 5). The vascular lesions in this series were diagnosed according to pathology (five patients), angiography (nine patients), and/or "unequivocal clinical diagnosis" (15 patients). However, it was not specified which vascular lesion was diagnosed by what method.

In most, if not all, of the references cited (2–6), hemangiomas have been explicitly, or implicitly, categorized or characterized as "high-flow" vascular anomalies, particularly in the classic "proliferation" phase. Although Baker et al quote in detail the published clinical and histologic aspects of hemangiomas and other vascular anomalies, they have excluded hemangiomas in the proliferating phase. Furthermore the authors' illustrations of hemangiomas (Figs 1 and 2) are more consistent with venous malformations, or at the very least, hemangiomas in the involuting or involuted phase.

According to now widely published criteria (2–10), vascular cutaneous and muscular lesions are most accurately termed "vascular anomalies" by the Mulliken and Glowacki Biological Classification according to characteristic clinical, pathologic, and imaging findings. Vascular anomalies are further subclassified as either hemangiomas or vascular malformations. Hemangiomas are the most common tumor of infancy characterized early on by endothelial proliferation (proliferating phase) followed by diminishing endothelial turnover, then regression (involuting phase). The MR findings correlate with the pathologic and angiographic findings in both phases of this tumor's evolution.

In infants, hemangioma is a mass with high-flow arterial and venous components displayed as vascular flow signal voids on spin-echo sequences using presaturation, and as vascular high-intensity flow enhancement on gradient-echo sequences using gradient moment nulling (Fig 1) (5, 7, 9, 10). As hemangiomas involute between 1 and 8 years of age, their flow diminishes, thus late-involuting or involuted hemangiomas are low-flow lesions. Proliferating-phase hemangiomas are distinguished from high-flow vascular malformations (eg, arteriovenous malformations), in that the latter has no parenchymal component. Low-flow vascular malformations include venous malformations, lymphatic malformations, lymphaticonvenous malformations, and capillary malformations. These malformations do not manifest the high-flow vascular characteristics on MR as described above for proliferating hemangiomas and arteriovenous malformations. Combined high-flow/low-flow vascular anomalies also exist (eg, arteriocapillary malformations).

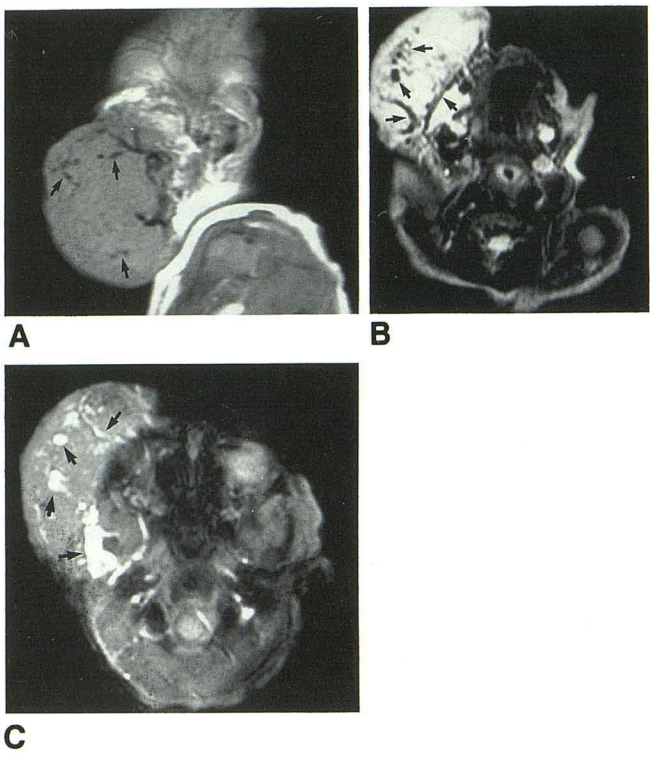

Fig. 1. Six-month-old boy with head and neck proliferating hemangioma. Sagittal T1 spin-echo MR (600/15/2 [repetition time/echo time/excitations]) with presaturation (*A*) demonstrates a large isointense/hypointense mass with arterial and venous flow signal voids (*arrows*). Axial T2 spin-echo MR (2000/90) with presaturation (*B*) demonstrates the hyperintense mass with vascular flow signal voids (*arrows*). Axial T2* gradient-echo MR (450/13, 30° flip angle) with gradient moment × (*C*) demonstrates the isointense mass with high-intensity vascular flow enhancement of the arterial and venous components (*arrows*) (from Wolpert and Barnes [10]).

We acknowledge the authors' efforts to bring the Mulliken and Glowacki Biological Classification of Vascular Anomalies to the *AJNR*. We regret that this diagnostically and therapeutically relevant classification (2–8), with characteristic MR imaging findings (5, 7, 9, 10), has yet to be completely presented in the neuroradiology literature.

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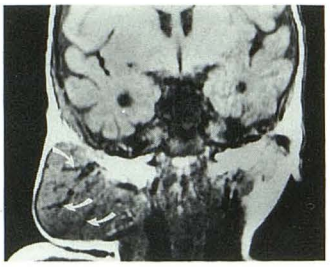

Fig. 2. Four-month-old child with a proliferating hemangioma. Coronal T1-weighted image (600/20) demonstrates an intermediate signal intensity mass containing vascular flow voids (*arrows*).

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Reply

We appreciate the interest Dr Barnes and colleagues have shown in our article on the MR findings in hemangiomas and vascular malformations of the head and neck (1). Regarding the semantic issue of the term *low-flow* lesion applied to hemangioma, we are referring to the degree of associated arteriovenous shunting, not vascularity. Although we are well aware that hemangiomas may demonstrate arteriovenous shunting, this is not a typical feature of hemangioma and, when observed, is more common in large proliferating hemangiomas, especially those within the liver (2). In fact, the angiographic study published by Burrows et al (3) reported that "direct arteriovenous shunting was not demonstrated angiographically" in their series of patients with hemangiomas. The figure enclosed by Dr Barnes and colleagues in their letter as well as those figures shown by Meyer et al (4) depict vascular flow voids within hemangiomas. The authors therefore use the term *high-flow* in their characterization of hemangiomas. The MR findings of signal voids are indicative only of vascularity, however, and the presence of arteriovenous shunting (high-flow) cannot be inferred by the mere depiction of vessels within a mass.

Nevertheless, we appreciate the emphasis Dr Barnes and colleagues have placed on the proliferative phase of hemangioma and the fact that vascular flow voids may be seen within solid tissue components of the hemangioma. Our small series of patients had hemangiomas that were either no longer growing or had begun to involute, accounting for the absence of vascular flow voids in our series. All patients subsequently demonstrated varying degrees of hemangioma involution over time, as documented by serial clinical examinations.

Since our initial article, we have had the opportunity to evaluate a small number of infants with hemangiomas in the proliferative phase, some of whom demonstrated vascular signal voids within solid mass components (Fig 2).

The Mulliken and Glowacki Biological Classification of Vascular Anomalies (5) is invaluable in the diagnosis and treatment of patients with vascular anomalies. We thank Dr Barnes and colleagues for their reiteration of this classification system and the corresponding MR findings.

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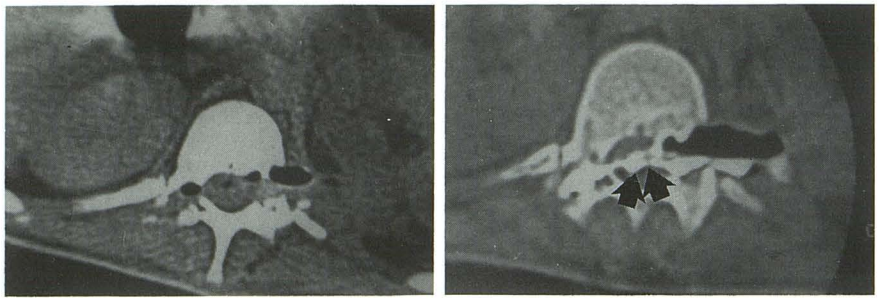
Epidural Pneumatosis: Not Necessarily Benign

We have read with interest the case reports by S. J. Willing (1), S. Balachandran et al (2), and T. Yoshimura et al (3), describing the condition of epidural pneumatosis. The authors have depicted the condition as a benign entity. We feel that this may not be necessarily so and describe one such case to illustrate our viewpoint.

A 36-year-old man who sustained a gunshot injury presented with hypovolemic shock and paraplegia. He underwent splenectomy along with left nephrectomy. Three weeks later a plain CT scan of the dorsolumbar spine

Fig. 3. Plain CT showing intraspinal air at D_{12}.

Fig. 4. CT myelogram showing duroenteric fistula (*arrows*) with transection of the cord (*arrowheads*).



showed air in the epidural space at D_{12} (Fig 3). This was followed by CT myelogram, which revealed bilateral extraspinal extravasation of the intrathecal contrast, which was seen entering the small intestine on the left side, indicating a dural enteric fistula. Associated complete transection of the cord was also seen at the same level with the intrathecal contrast dividing the cord in two (*arrowheads,* Fig 4). The patient died 4 months later of intractable meningitis.

The presence of epidural air is generally considered benign, and the few cases described in the literature have been without any serious complications. We feel that the mere diagnosis of epidural pneumatosis may not be as important as is its cause, which can have serious implications on the final outcome and prognosis of the patient as is seen in this case.

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Reply

It is self-evident that penetrating spinal injuries may cause intraspinal air (pneumorrhachis). In my article I summarized the many possible causes of epidural air, including two previous references citing enteric fistulae. The point of this article was not to suggest that epidural air was always a benign occurrence, but to identify a specific group of patients who all had *blunt* trauma, no neurologic findings referable to the cord, and air in the epidural space; this concurrence of findings was labeled *benign epidural pneumatosis*, a term that had not been previously defined. The case presented by Kapur and Sandhu does not meet the diagnostic criteria for the condition of benign epidural pneumatosis and illustrates the usefulness of reserving this term for those in whom the specific diagnostic criteria cited in my article are met.

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More Association of Linear Sebaceous Nevus Syndrome and Unilateral Megalencephaly

We read with interest the paper "Association of Linear Sebaceous Nevus Syndrome and Unilateral Megalencephaly" (1). Cavenagh et al present in detail a representative case and give an excellent review of the literature starting with the first description by Feuerstein and Mims (2). The association between unilateral megalencephaly and linear sebaceous nevus syndrome appears to be well established. MR is superior in showing the cerebral abnormalities.

We would like to report our MR findings in three biopsy-proved cases of linear sebaceous nevus syndrome. The CT findings in one of them have already been published as case 1 in another paper from our institution (3). Our clinical and electroencephalographic findings were identical to those described by Cavenagh et al.

Our MR findings were similar to those shown by Cavenagh et al (Fig 5). However, there was a different degree of involvement of the cerebral hemisphere and there were no diploic abnormalities.

The gyral and white matter abnormalities were more pronounced in our cases, and we found bands of gray matter in the centrum semiovale in one case and periventricular heterotopia in another case (Fig 6).

Unilateral megalencephaly covers a wide spectrum of findings as recently reported by Barkovich and Chuang (4). The most common gyral abnormality, polymicrogyria, is thought to result from an insult in the late second trimester of pregnancy. Damage to the hemisphere can interfere with the neuronal migration and give rise to neuronal heterotopia.

In summary, our findings confirm the recent report by Cavenagh et al, but they illustrate the variable degree of affection of the abnormal hemisphere.

Philippe Demaerel

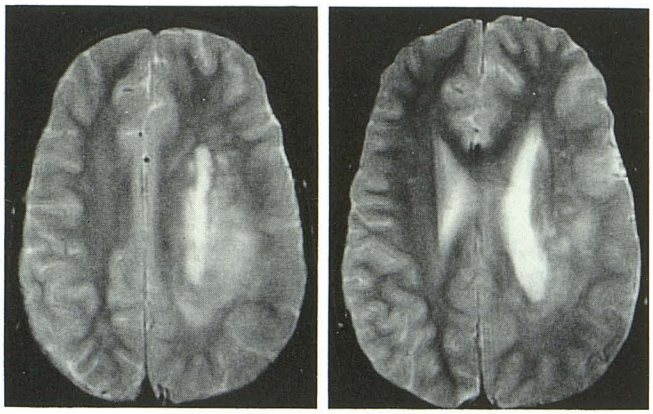

Fig. 5. Axial T2-weighted spin-echo sequence (2500/90). Broadening of the gyri and thickening of the cortex is seen. Note the extensive abnormal white matter signal.

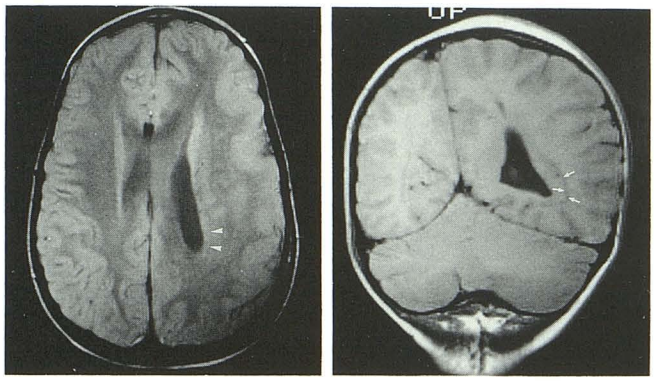

Fig. 6. Axial proton density- (2500/15) (*A*) and coronal T1-weighted (600/15) (*B*) spin-echo MR images. Periventricular heterotopia (*A, arrowheads)* and gray matter bands (*B, arrows*) in the centrum semivale are demonstrated.

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Reply

I thank Drs Demaerel, Wilms, and Casaer for their comments and additional examples of the association of unilateral megalencephaly with linear sebaceous nevus syndrome. We are interested to note the additional manifestations of neuronal migration abnormalities, including both band and focal periventricular heterotopias, that may accompany this disorder.

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Erroneous Placement of Side Indicators of Brain CT

I would like to report our experience with a 58-year-old woman with a large right cerebellar mass on CT performed at another center. She had no lateralizing symptoms or other neurologic abnormalities. Surgery revealed no tumor.

A repeat CT at our institution showed the mass to be in the left cerebellar hemisphere. The tumor, which proved to be a meningioma, was subsequently removed successfully. This human error was caused by pressing the "foot first" button instead of the "head first" button.

The suggested solution to assure correct sideness is to affix a polyethylene tubing to the head holder (1). This object, invisible on standard images, becomes evident with a wide window. Fortunately, in the majority of cases, discrepancies between clinical and radiologic findings alert the clinician before harmful actions are taken.

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Middle Cranial Fossa Not Temporal Fossa

I enjoyed the article in the recent issue of *AJNR*, "Adult Cerebellar Medulloblastoma: Imaging Features with Emphasis on MR Findings." I must however correct the caption for Figure 6 that draws attention to an incidental right temporal fossa arachnoid cyst. The figure shows the cyst in the right middle cranial fossa.

According to Stedman's Medical Dictionary (1) as well as the fourth edition of Anatomy by Gardner, Gray, and O'Rahilly (2), Grant's Atlas of Anatomy (3), and Dorland's Illustrated Medical Dictionary (4), the temporal fossa is the space on the side of the cranium bounded by the temporal

lines and terminating below at the level of the zygomatic arch. Please correct this mistake as I have seen it made often by my fellow radiologists as well as referring physicians. Certainly, I was surprised to find my second favorite bedtime companion (*AJNR*) make the same mistake.

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Editor's note: Dr Hatfield is correct. It is a common mistake to refer to the middle cranial fossa as the temporal fossa but one that has gradually crept into the literature because the temporal lobe sits in the middle cranial fossa. *AJNR* is appropriately embarrassed and thanks Dr Hatfield for bringing this to our attention.