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# Otic or Mythic?

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## Diffusion Findings in Blood Clot: The Last Word?

Diffusion-weighted imaging (DWI) is most recognized for its diagnostic utility in stroke; however, recent attention has focused on other diseases that similarly exhibit restricted diffusion on DWI. DWI of blood clot is of particular interest because hemorrhage may complicate the appearance of stroke. Because the process of clotting involves transformation of a fluid to a semisolid, it is predictable that water diffusion would decrease in acute clot and hence be hyperintense on DWI. Thus, the *AJNR* articles by Atlas et al (1) and Maldjian et al (2) focusing on the diffusion characteristics of intracerebral hematomas are of interest in that they represented early, albeit incomplete, studies of the diffusion characteristics of blood clot as a function of time.

The earlier study, by Atlas et al, did not address the appearance of blood clot on diffusion-weighted images, restricting the focus to apparent diffusion coefficient (ADC) values. They reported significantly reduced ADCs compared with normal white matter in early hematomas (hyperacute, acute, and early subacute) but increased ADCs after cell lysis occurred—ie, in the late subacute phase (1). The finding of reduced ADC in clot was contested by Maldjian et al, who argued that automated ADC calculations may be underestimated when using vendor-supplied software because of thresholding effects at low signal intensity–to-noise (SNR) ratios. They argued that ADC was not restricted in phases of clot in which red cells are intact, concluding that those clots have essentially the same ADC as white matter when proper technique is employed. Although the mean ADC of the 12 hematomas in the study by Maldjian et al was not significantly different from white matter, two of the four hyperacute hematomas did have markedly decreased ADC, and late subacute clots were not studied at all. Thus, these two studies left unanswered questions regarding the DWI appearance and ADCs of intracranial blood clots. Hence, we sought to readdress the issue in a larger group.

Before we completed collecting our cases, however, we found a more recent and thoroughly performed study by Kang et al (3) whose results are so consistent with our own that we stopped the study early. The Kang et al study found that clots were bright on DWI in hyperacute and late subacute clots and that ADCs were reduced compared with normal brain tissue during all phases (hyperacute, acute, and early and late subacute). These data suggest that diffusion is restricted in clots before and after cell lysis, resulting in bright signal intensity on DWI unless T2 effects of intracellular unpaired electrons reduces the signal intensity (SI) (T2 dark-through effect [2]).

Our data mirror those of Kang et al. Twenty clots (hyperacute [$n = 3$], acute [$n = 7$], early subacute [$n = 5$], late subacute [$n = 5$]) were studied on T1-weighted images, T2-weighted images, DWI, and ADC maps, and the results were expressed as SI ratios (Fig). Hyperacute clot was markedly hyperintense on DWI in three of three cases; acute clots were markedly hypointense in seven of seven cases. In four of five early subacute clots, DWI SI was hypointense. In five of five late subacute clots, DWI was hyperintense. To address the concern of Maldjian et al regarding thresholding effects by using vendor-supplied software, we calculated ADCs by using 0% and 20% thresholds, and it made no significant difference in the appearance of ADC maps. In addition, for quantitative study (Fig), we recorded region of interest SIs and manually calculated ADCs by using the Stejskal-Tanner formula (4). We observed the marked hyperintensity of hyperacute clot to be associated with restricted diffusion (low ADC). The conspicuous hypointensity of acute and early subacute clots was also associated with low ADC, concurrent with marked T2 effects dominating SI. In late subacute clots, SI returned to a hyperintense appearance on DWI as T2 effects dissipated and restricted diffusion persisted.

In conclusion, our data concur with those of Kang et al in indicating that diffusion is reduced in hyperacute, acute, and subacute clots. Reduced ADC accounts for the marked hyperintensity on DWI scans in hyperacute and late subacute phases. Despite restricted diffusion, SI on DWI is not increased in the intervening acute and early subacute phases because of T2-induced hypointensity of clot, which dominates signal intensity on DWI (ie, "T2 dark-through").

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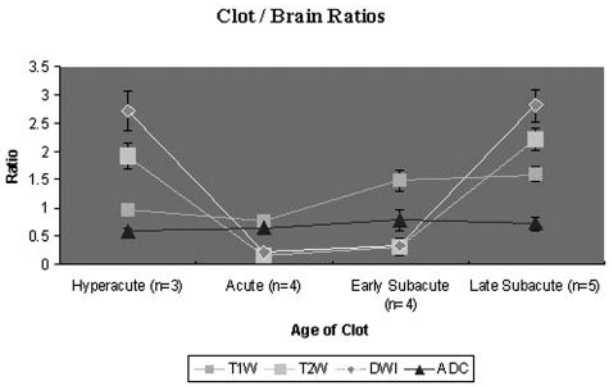
*Reply*

*Diffusion Findings in Blood Clot: The Last Word? Not!*

Shah and colleagues report that hematomas have slow diffusion in the hyperacute, acute, and early subacute stages, findings similar to those of Kang et al (1). The apparent diffusion coefficient (ADC) values of late, subacute hematomas are less clear in Shah et al's study, but the authors imply that it also was slower than those of normal white matter. We suspect that these results are classic examples of the artifact reported by Maldjian et al (2).

As previously described, standard vendor-supplied software does not produce valid signal intensity measurements in regions of extremely low signal (2). The attempts, first by Atlas et al (3), then by Kang et al (1), and now by Shah and colleagues, to report measurements from an area of signal void remain confounded by measurement procedures. The pitfall of susceptibility induced signal losses complicating ADC measurements in acute hematomas was even raised in an editorial published concurrently with the Atlas article (4). The purpose of our study was to provide a framework for computing ADC values in the setting of low-T2 signal (2). Neither Kang et al nor Shah and colleagues letter take this problem into account. Specifically, in acute and subacute hemorrhage, the T2*-weighted signal intensity can be close to that of background noise. This low signal intensity can result in acute hematomas appearing dark or black on diffusion-weighted images and ADC maps (T2 blackout). Obtaining an accurate diffusion measurement is problematic in this setting, because an individual pixel value may be dominated by the thermal and electronic noise of the imaging system. In the presence of background variations, it is even possible for a pixel at background intensity to show a higher magnitude of signal intensity on the diffusion-weighted image than on the baseline image, producing a spuriously negative ADC value (which is nonsensical and a violation of the second law of thermodynamics). Inclusion of such pixels in a region of interest (ROI) will lead to artifactually low mean ADC values. In our article, we provide a framework by using Expected Values to compute ADC values in this setting. We demonstrate that using vendor-supplied software that automat-




ically masks the background (intended to provide more visually pleasing results) is an inherently flawed method of computing ADC values in these cases. Even at low levels of background masking (2%), the resulting ADC maps show marked artificial decreases in values from masking out areas of low signal intensity (including these masked values in the ROI). Apparently, the Kang study (1) did not take this into account when generating ADC measurements. In fact, it is unclear how they were generated. Kang et al do not provide an ADC map for a visual assessment of their methodology.

In their study, Shah and colleagues state that there were no differences in ADC maps between 0 and 20% thresholds. It is difficult to know what this means. Systematically and selectively removing a large portion of data, those pixels with the lowest signal intensity would have to change the results. Absent noise, this exclusion would select against pixels with the fastest diffusion and produce artifactually low ADC values. If it did not, then something must be seriously wrong with the pulse sequence or the measurement. The authors report that calculating ADCs by using 0 and 20% thresholds made no difference in the appearance of ADC maps. It is not clear whether Shah and colleagues measured ADC values at both 0 and 20% thresholds and found no differences. If not, we do not know what to conclude from the statement that the maps appeared similar.

We have shown that at levels of background masking as low as 2%, significant numbers of dropped pixels are evident in ADC maps of acute hematomas (2). Did Shah and colleagues find no such differences? Acute hematomas demonstrate T2* signal intensity close to that of background noise. If T2 black hematomas had signal intensity >20% of the mean brain signal, they would not be T2-dark. With some vendor-supplied software, there may be masking present in the algorithm, even at the 0% nominal masking. It is also possible that there was no signal because of susceptibility effects, even at the 0% masking.

Shah and colleagues state that their diffusion-weighted images were markedly hypointense (ie, black) for all the acute hematomas and hypointense for four of the five early subacute hematomas. That these hematomas were very dark on the diffusion-weighted images indicates that an accurate diffusion measurement cannot be obtained without accounting for the masking effect.

Atlas et al have observed that ADC is reduced on average in the acute phase of a hematoma, as compared with white matter, and has suggested that this is due to restricted diffusion (3). Although we would agree that restricted diffusion plays a role as a lower limit (2), we argue that variability in the measured ADC is dominated by the amount of extracellular fluid present, a quantity that we believe to be highly variable. In addition, the diffusion measurements made using the methods of Atlas et al, Shah et al, and Kang et al on T2-dark hematomas are all suspect. Atlas and colleagues further observe that lysis of the red blood cells will increase the ADC as compared with white matter; we have no reservations regarding this observation.

Shah and colleagues, on the other hand, state that the ADC is reduced in late subacute clots. From this observation, Shah et al conclude that "restricted diffusion persisted." "Slow diffusion" and "restriction" are not synonymous. Restriction is one of several potential causes of slow diffusion. The finding of slow diffusion by itself does not permit a conclusion that restriction is the mechanism. The finding of slow diffusion after cell lysis, in which there are no apparent barriers to produce restriction, would argue against restriction as a plausible mechanism.

Claims for hyperacute hematoma are also confusing. Atlas et al make no claim, whereas Kang et al and Shah et al report a mean ADC ratio of approximately 0.7. Given that the hyperacute hematoma most resembles fresh blood, its ADC would presumably also resemble that of plasma. This is additionally supported by a recent article by Wintermark et al (5) in which diffusion is increased in hyperacute hematomas, in contrast to the assertions of Shah and colleagues.

What is the last word on diffusion in blood clot? We don't know; but establishing the presence of restriction in hematomas would require a set of experiments that are rarely performed with clinical instruments. In order to determine the true diffusion signal characteristics of hematomas by use of MR imaging, it will likely be necessary to implement pulse sequences similar to that of the recently described propeller fast spin-echo technique (6), which are less prone to the susceptibility induced dephasing and distortions inherent in echo planar-based methods. Additionally, the relationship between blood susceptibility and ADC may be a multiexponential (7, 8) rather than a single exponential decay model assumed in the standard Stejskal-Tanner relationship (9). In fact, it is unlikely that a clear answer will be determined by using in-vivo human data, as the extracellular fluid fraction in intracranial hematomas may often be the dominant contributor to diffusion rates and can vary from patient to patient. It is clear, however, that the diffusion-weighted signal intensity for blood products is complicated, and measurements derived from areas of signal void and computed with incompletely documented algorithms will be of limited value.

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## The Magic Measurement

I read with dismay the article by Frisoni et al regarding the radial width of the temporal horn in Alzheimer disease (1). The authors claim that, armed with one CT scan of the brain and a

ruler, they can make a single measurement that will distinguish patients with early Alzheimer disease from age-matched control subjects with a sensitivity of 93% and specificity of 97%, up to the age of 90 years; this was better in fact than they could apparently achieve with MR imaging and far better than other measurements they tried, about which others have made similar extravagant claims. This, then, is another article with the "magic measurement," like the thickness of the substantia innominata, also in an article appearing in the same issue (2).

A moment's reflection surely makes clear the implausibility of a result like this. One suspects that the answer lies in the control subjects, selected because of no clinical or CT evidence of a neurodegenerative disease. It seems clear that subjects with CT findings of excessive atrophy were excluded as controls and that the results reflect only how efficient this exclusion process was. I, and many others, will take a lot of convincing by the authors that the situation is otherwise. Indeed, so many articles in related fields seem to make the same mistake that I think an editorial or commentary should be dedicated to it—not the sort of laudatory commentary as appear on page 33 of this issue (3), but a critical appraisal. The "past glory" is not all that glorious, and the "future promise" is most uncertain, if it is the truth about the real world we are seeking as opposed to the pursuit of producing nice studies that have value only as an art form.

J. Stevens

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*Reply*

Basically, Dr Stevens claims that the positive results of our study (1) were obtained by excluding unwelcome data (ie, control subjects with CT findings of excessive atrophy). We strongly disagree with Dr. Stevens' assertions.

Our controls were persons with no cognitive symptoms who underwent CT mostly for headache or dizziness (83% of our 29 controls) and whose CT findings were normal. Thus, although we did not take measures of physical comorbidity, it is likely that these persons were in reasonably good physical health. Because age-associated temporal atrophy in physically healthy elderly persons is absent or minimal (2), a serene mind would not find it surprising that our controls had very little age-associated medial temporal lobe atrophy and that their atrophy measures separated them well from Alzheimer disease patients.

What might have been contended with more support is rather that these controls are not representative of the clinical world, where physicians are challenged with patients who do report cognitive symptoms. Indeed, our own is a phase I study of a diagnostic tool, aiming to answer the question, "Do test results in patients with the target disorder differ from those in normal people?" (3). Final evidence of clinical usefulness would require demonstration of high positive and negative predictive values (phase II), high sensitivity and specificity in clinically meaningful groups (phase III), and good test efficacy on ultimate health outcomes (phase IV). Radiologic, as well as nonradiologic, diagnostic tools are usually supported by phase I, seldom by phase II, and very rarely by phase III and IV studies (3).

Indeed, for our own diagnostic tool—as well as for most others—this evidence still needs to be provided (4). However, the high frequency of use of CT in the diagnosis of cognitive impairment and the often limited human and technological resources in diagnostic facilities make a feasible CT-based marker of Alzheimer disease a potentially significant incremental diagnostic value. This is not magic but simply good clinical practice.


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# Thromboembolic Events Associated with Guglielmi Detachable Coil Embolization of Asymptomatic Cerebral Aneurysms: Evaluation of 66 Consecutive Cases with Use of Diffusion-Weighted MR Imaging

We read with interest the article by Soeda et al (1) in January 2003 issue of the *AJNR*. The authors presented their experience with thromboembolic events detected by use of diffusion-weighted imaging that were associated with Guglielmi detachable coil (GDC) embolization. They concluded that thromboembolic events are relatively common in association with the balloon-assisted technique. We would like to take this opportunity to emphasize the following point.

In 1994, Moret et al (2) described the balloon-assisted technique for treatment of wide-necked or broad-based aneurysms with maximal sac diameter to neck size ratio of close to 1. The invention of the technique provided a new option in the treatment of wide-necked aneurysms and became the preferred method for their treatment. Soeda et al (1) found that diffusion-weighted abnormality was detected in 50% of small aneurysms with small necks, in 73% of small aneurysms with wide necks, 100% in large aneurysms, and 73% (22/30) in the procedure with balloon-assisted technique. They concluded that the occurrence of thromboembolic events depended on procedural complexity such as larger aneurysms ($P < .01$) and the use of balloon-assisted technique ($P <. 05$). Although we agree that larger aneurysms or those with poor morphology can cause more frequent thromboembolic events, we do not agree with authors' second conclusion that the use of the balloon-assisted technique more frequently causes thromboembolic events. We assume that authors should have used the balloon-assisted remodeling technique for small aneurysms with wide necks and in large aneurysms despite small aneurysms with small necks. (This information was not given in the study.) As a result, the

authors cannot conclude that the use of the balloon-assisted technique more frequently causes thromboembolic events. Small aneurysms with wide necks (73%) and larger, wide-necked aneurysms (100%) did not cause thromboembolic events more frequently than the use of the balloon-assisted technique (73%). Their conclusion might mistakenly discourage the use of the balloon-assisted technique, despite our experience (almost 50%) and Soeda et al's study (1) (49%) that the balloon-assisted technique should be used for treatment in a half of aneurysm cases accepted in the interventional neuroradiology suite.

In summary, because balloon-assisted techniques are used for treatment of wide-necked large and small aneurysms alike and the frequency of thromboembolic events in associated with balloon-assisted technique (73%) was not greater than that of thromboembolic events in small aneurysm with wide neck (73%) and larger aneurysm with wide neck (100%), the authors cannot conclude that the infarcts related to the use of GDC embolization are more common sequelae with use of balloon-assisted technique

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### *Reply*

We thank Drs. Sait Albayram and Dogan Selcuk for their interest and comments regarding our article (1). Although they agreed that thromboembolic events occur more frequently in the Guglielmi detachable coil (GDC) embolizations of larger and wide-necked aneurysms, they doubt direct causal relationship between the use of balloon-assisted techniques and higher frequency of such events, assuming that we should have used this technique for larger and small aneurysms with wide necks. This is not the case. In fact, we used the balloon-assisted technique in 47% of small aneurysms with small neck, 50% of small aneurysms with wide neck, and 29% of large aneurysms. Of the small-necked aneurysms treated with balloon-assisted technique, the hyperintense lesions were detected in 70% of patients. Therefore, we concluded that the use of balloon-assisted techniques has a causal relationship to higher frequency of thromboembolic events, not the epiphenomenon of more frequent use of this technique for more complex aneurysms as they assumed.

Although the proportion of aneurysms treated with the balloon-assisted technique was not clearly reported, at most centers this technique was used after conventional treatment had failed. We used this technique for a high percentage of small-necked aneurysms. The reason for this high percentage in cases where we anticipated difficulties is because the microcatheter could not be introduced into the aneurysmal sac by conventional GDC techniques or because we feared that the coils would protrude into the parent artery. In such cases, we introduced the balloon into the parent vessel beforehand, obviating catheter exchanges.

In our recent retrospective study (2), most thromboembolic events related to the GDC embolization may be caused by placement of the guiding catheters as well as manipulation of microcatheters. This study supported previous results and suggested the risk of significant emboli will likely increase with increasing procedural complexity such as large aneurysm or use of balloon-assisted technique.

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## Otic or Mythic?

We read with great interest and anticipation the paper by Patel et al (1) describing the second angiographically demonstrated case of a persistent otic artery. From our early training, all neuroradiologists have studied the embryonic anastomoses between the developing carotid arterial system and the longitudinal neural axis, the future basilar artery. We all have a few examples of trigeminal arteries—some also have the odd hypoglossal or proatlantal arteries—but which of us has seen an unequivocal otic artery, about which we continue to teach our trainees? Searching the small literature on this elusive vessel, we see that most cases have actually been low-lying trigeminal arteries, while others have described what appear to be stapedial artery remnants (2). The poor quality of reproduction of images in some publications and the frequent presence of only a single angiographic projection make it difficult to be sure of the origin, course, and termination of the vessel, and therefore of its true nature. Does the otic artery really exist? Does this case provide the missing link?

The trigeminal, hypoglossal, and proatlantal arteries are surely segmental arteries related to the metameric embryonic structure of the diencephalon, rhombencephalon, and spinal cord and their related nerves. These three embryonic arteries follow a generally anteroposterior, slightly oblique, course, supplying blood to the developing basilar system. The otic structures clearly are not segmental and develop mainly from the otic placode. Thus, there seems no reason to expect a segmental communication at this level. Further, as Lasjaunias has pointed out (3), unlike the other three embryonic vessels, there is no evidence for the existence of an otic artery in lower animals. If there were an otic artery, it would necessarily have to follow a lateral course into the internal auditory meatus (Fig 1), a very different orientation from its fellow vessels.

Of course, anastomoses may occur between the internal auditory artery (branch of the anterior inferior cerebellar artery [AICA] and thus basilar artery) and the internal carotid artery, via trigeminal and stapedial remnants (3) and the "otic" artery shown in Newton and Potts' classic textbook (4) would fall into this category. Similarly, dangerous anastomoses are well recognized for example between external and internal carotid arteries (eg, via ophthalmic artery) and reflect overlapping vascular territories, rather than representing a single embryonic vessel in the sense of the trigeminal or hypoglossal artery.

Padgett (5) illustrates the otic artery arising below the level of the hyoid artery. Her reconstructions were based on sections of embryos, traced onto paper and then overlaid to give a three-dimensional effect. We are in awe of the ground-breaking nature of her classic work, and yet the sectioning of the embryos or the tracing process, could introduce artifacts and

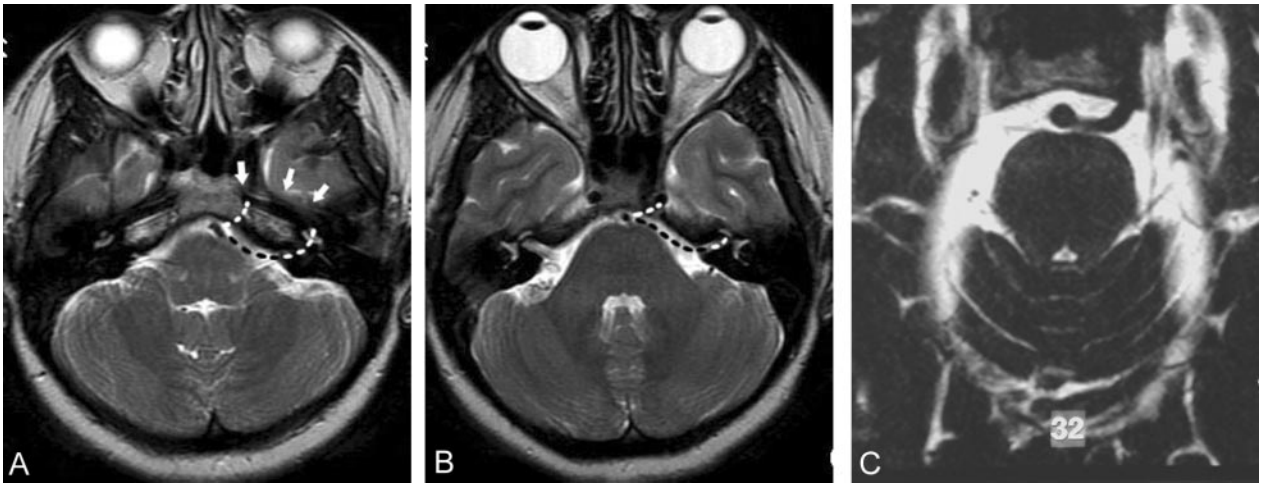


FIG 1. Axial T2-weighted MR imaging at level of petrous canal (*A, white arrows*) and at level of IAM (*B*). In both, dotted lines show posterolaterally the predicted course of an otic artery through the IAM to join the lateral portion of the petrous ICA. In *A,* the anteromedial dotted line shows the course of a low-lying trigeminal artery and, in *B,* the typical course of a trigeminal artery. *C,* Actual recent case of a trigeminal artery joining the cavernous ICA. The anomalous vessel is readily visible on standard cross-sectional images, and a true otic artery of the size shown by Patel et al would be equally visible on any standard MR imaging sequence.

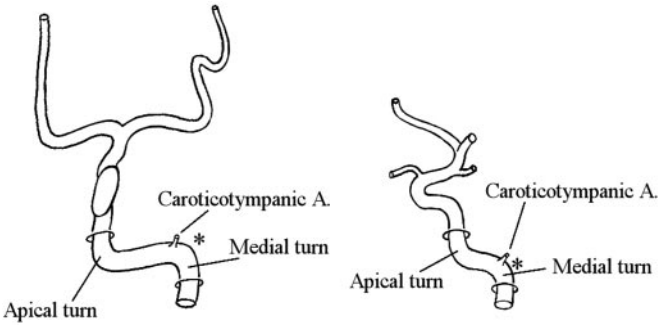


FIG 2. Anteroposterior and lateral drawings of the internal carotid artery. The rings denote the entry and exit from the petrous canal. Note the typical foreshortening of the petrous portion on the lateral view. Caroticotympanic artery (adult successor to the hyostapedial artery) arises close to the medial turn. Asterisk denotes predicted origin of a stapedial artery.

lead to misidentification of a vessel, especially one that she was expecting to see. Kelemen (6) stated that the hyostapedial (caroticotympanic) artery origin lies between the medial and apical turns of the petrous internal carotid artery and agrees with Padgett that the otic should arise proximal to that. Thus, the otic artery would arise in the adult from the lateral and proximal part of the petrous carotid (Fig 2). Reference to Figure 1 shows that it would therefore be in close proximity to the IAM, through which it must travel.

As Patel et al note, Lie (7) quotes three logical criteria for the putative otic artery. First, it should arise in the lateral portion of the petrous canal, close to the medial turn; in Patel et al, conversely, it arises from the medial portion of the petrous carotid, as the ICA turns up toward the cavernous sinus—ie, close to the apical turn (a well-recognized site of low origin of a trigeminal artery [Lie, p. 58]). Second, it should run through the IAM; this would be confirmed by MR imaging, but, although the authors state that an MR imaging was performed, unfortunately they do not show this. From their angiograms, it seems unlikely that the vessel traverses the IAM. Third, it should join the basilar artery at a caudal point. In the authors' case, conversely, it joins the midbasilar, clearly above the level of the AICA, a typical location for a trigeminal artery. Unfortunately, adding to the confusion, the model Lie used to illustrate the predicted course of the otic artery shows the vessel arising from the midportion of the petrous ICA; according to the adjoining text description it should arise more laterally, proximal to the caroticotympanic artery (hyostapedial remnant), and thus close to the medial turn.

For all these reasons, we believe that this case is actually, simply a low-lying trigeminal artery. The only other "convincing" case the authors refer to, by Reynolds et al (8), shows the anomalous vessel clearly, only in the anteroposterior plane. As in the current case, it arises from the medial part of the petrous portion of the ICA and does not appear to traverse the IAM. We believe this also to be a low trigeminal artery. Thus, we are still not convinced of the existence of the otic artery as an independent embryonic vessel. In view of the size of the anomalous artery in the current case, it must be clearly visible at MR imaging that the authors refer to in the report; we are intrigued as to whether it was visible entering the IAM, which would certainly support the authors' argument.

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## Persistent Otic Artery

I read with interest the case reported by Patel el al in the January 2003 issue of the *AJNR* (1). Lasjaunias and Berenstein (2) have noted that they have never seen convincing anatomic or angiographic evidence of a persistent otic artery and suggest that it might not exist. I share the skepticism of Lasjaunias and Berenstein, and I believe that the case reported by Patel et al is an example of a persistent trigeminal artery rather than a persistent otic artery.

Patel et al (1) note that, according to Lie (3), the persistent otic artery arises from the carotid artery within the carotid canal, emerges from the internal acoustic meatus, and joins the basilar artery at a caudal point. The case they report demonstrates none of these three features. The persistent embryonic anastamosis shown in Figure 1A arises distal to the horizontal petrous segment of the internal carotid artery, as the artery turns upward toward the cavernous sinus. The persistent otic artery should pass through the internal acoustic canal, yet Figure 2A demonstrates that the entirety of the persistent embryonic artery is medial to the internal auditory meatus, which is demarcated by the characteristic loop of the anterior inferior cerebellar artery (4). The persistent otic artery supposedly joins the basilar artery at a caudal point, yet Figure 2A demonstrates the artery joining the basilar artery near the junction of the middle and upper thirds. The origin, course, and termination of the persistent embryonic anastamosis described in the report therefore meets none of the criteria of a persistent otic artery but meets all of the criteria for a persistent trigeminal artery.

Finally, the authors state in their discussion that persistent trigeminal, hypoglossal, and proatlantal arteries have been associated with aneurysms distant from the persistent vessels (1). Such an association is dubious. The prevalence of aneurysms associated with persistent trigeminal artery is approximately 3%, which is similar to the prevalence of aneurysms in the general population (5).

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*Reply*

Drs. Bhattacharya et al and Cloft have raised the interesting possibility that the vessel we reported as a primitive otic artery (1) could represent, instead, a low-lying trigeminal artery. The angiographic features of our case match the angiographic appearance of the artery reported previously by Reynolds et al as a primitive otic artery (2). For that reason, we designated it "otic."

The excellent summary of the theoretical origins, courses, and terminations of primitive otic arteries by Drs. Bhattacharya et al provide an alternate method for characterizing a vessel as "otic." By their definitions, both our example and that of Reynolds et al could be designated primitive trigeminal arteries.

These differences in interpretation and criteria highlight the difficulty of agreeing on precise definitions for conditions, when the conditions are seen too rarely to know the full range of variation that should be accepted within each defined category.

Establishment of a data base for these variations might well help us to assemble sufficient numbers of actual cases to refine the present ambiguous classifications.

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