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Flow voids in Time-of-Flight MR Angiography of Carotid Artery Stenosis? It Depends on the TE!

Nederkoorn et al (1) conclude that flow voids on nonenhanced 3D time-of-flight (TOF) MR angiography (MRA) images represent severe carotid artery stenosis. Although they present compelling evidence that this conclusion is correct for their particular MR imaging system with their particular imaging parameters, radiologists should be advised of the peril of generalizing these results to *any* MR imaging system, using *any* imaging parameters. Specifically, preliminary data from our neurovascular lab suggest that the presence of flow voids on 2D TOF MRA images, for a given degree of carotid artery narrowing, is critically dependent on choice of echo time (TE) for the TOF pulse sequence, specific MR imaging hardware, or both.

In a pilot study of patients who underwent both carotid duplex sonography and 2D TOF MRA for evaluation of suspected internal carotid artery stenosis, 20 were imaged on a newer LX unit (GE Medical Systems, Milwaukee, WI) by using a short-TE pulse sequence (TE $\approx$ 4.7 ms), and 24 were imaged on an older Signa unit (GE Medical Systems) by using a long-TE pulse sequence (TE $\approx$ 8.7 ms). Of the 20 imaged with the short-TE pulse sequence, TOF signal dropout was seen in one (100%) of one with hairline lumen, in three (50%) of six with peak systolic velocity (PSV) more than 400 cm/s, in four (50%) of eight with PSV between 200 and 400 cm/s, and in none (0.0%) of three with PSV less than 200 cm/s (two patients with PSV's of $\sim$ 370 and 540 cm/s had equivocal signal dropout). Of the 24 imaged with the long-TE pulse sequence, TOF signal dropout was seen in one (100%) of one with hairline lumen, in 10 (100%) of 10 with PSV more than 400 cm/s, in four (80%) of five with PSV between 200 and 400 cm/s, and in one (14.3%) of seven with a PSV less than 200 (one patient with PSV $\sim$ 300 cm/s had equivocal signal dropout). One patient was imaged twice, each imaging session a week apart without interval treatment, by using different TE values. The first images, which were obtained with a long TE of 8.6 ms, showed a flow void, whereas the follow-up images, which were obtained with a short TE of 4.7 ms, did not.

These findings are consistent with the fact that flow voids on TOF MRA images are caused by intravoxel dephasing and are thus less likely to occur with short than with long TEs. Additionally, the stronger gradients and more homogeneous magnetic fields present in newer MR units, which permit smaller voxel sizes, may also predispose to decreased intravoxel dephasing, and hence lower sensitivity for signal dropout from turbulent flow. Although, as Nederkoorn et al point out, 3D TOF MRA techniques "have higher spatial resolution, a greater signal-to-noise ratio, and lower sensitivity for voids because of the smaller voxels and shorter echo time(s)" as compared with those of 2D TOF MRA, the precise relationship between 3D TOF flow void detection thresholds and the specific MR imaging hardware and software used has yet to be determined. Until it has, we continue to advise a conservative approach to flow void interpretation on TOF MRA images. Indeed, radiologists ideally should calibrate TOF signal dropout for *their* particular MR units and pulse sequences with an external reference standard of stenosis, such as sonography or CTA, *before* image interpretation. This may be especially prudent in some clinical environments wherein surgeons consider patients with carotid artery flow voids to have "proved" severe

(>70%) luminal stenosis, and therefore to be candidates for carotid endarterectomy.

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Is Insufficient Use of Polymethylmethacrylate a Cause for Vertebroplasty Failure Necessitating Repeat Vertebroplasty?

I wish to compliment Gaughen et al on their continued valuable contributions to the literature as they critically evaluate their vertebroplasty patients. Sharing their experience with others is important to continue to make vertebroplasty an excellent treatment in selected patients.

In this article (1), the authors stress the point that they can perform a second vertebroplasty on previously treated vertebral levels as their major emphasis. They also mention that one possible cause for re-treatment resulted from inadequate cement deposition. Although they mention the fact of potential inadequate cement deposition, this feature in the article is not prominently mentioned or emphasized. In their article, there are several cases where polymethylmethacrylate (PMMA) is instilled with very small volumes (1-3.5 mL). These are extremely small amounts of PMMA, unless one is dealing with a severe *vertebra plana*. Because many of these patients have osteoporosis, if PMMA is placed in only a small part of the vertebral body, why should the remainder of the vertebral body not have a good chance to fracture later?

When I first learned how to perform vertebroplasty, maximal filling of a vertebral body to prevent later collapse of the vertebra was stressed. Subsequently, published literature has stated that only a small amount of PMMA needs to be instilled into a vertebral body to gain effective treatment (2). I have attended a meeting where there have been comments stressing the point that only a minimal amount of PMMA needs to be injected to get satisfactory results. Anecdotally, people attending other meetings have told me that speakers stressed this same point, that only a small amount of PMMA needs to be injected. Subsequently, I realize that there are potentially two schools of thought with respect to vertebroplasty, one being that of the "minimalist" school, where only a small amount of PMMA is injected. The other is that of the "maximalist" school, where as much PMMA as is safe is injected into the vertebral body. I have been a supporter of this latter school since learning vertebroplasty because of my belief that if a vertebral body is fully or nearly filled with PMMA, it cannot collapse further. The criteria for adequate filling I have tried to achieve is filling of a vertebral body from superior to inferior endplate from one side of the vertebral body to at least the medial border of the opposite pedicle. One person with extensive percutaneous vertebroplasty experience (Dr. Gregory J. Lawler, Nashville, TN, personal communication, Sundance Vertebroplasty Conference, Sundance, UT, August 5-8, 1999)

subscribed to the "minimalist" school. He had had several cases where PMMA filled most of one side of a vertebral body and later the opposite side of the vertebral body without PMMA collapse. He then re-treated the area in that vertebral body without PMMA with relief of symptoms.

Since starting vertebroplasty, I have been involved with more than 900 vertebroplasties and have had two cases where there has been a need to inject the same vertebral body twice. One was a case where there was patchy distribution of PMMA throughout the vertebral body in a patient with multiple myeloma. He did well for 1 year. At the end of that year, he had two additional fractures, and an MR examination at that time showed a fracture cleft within the previously treated vertebral body. All three vertebral bodies were treated at that second treatment time, with elimination of the patient's presenting symptoms. That case was early in my experience. Today I recognize that patchy distribution of PMMA through the vertebral body may be faced with additional collapse in the vertebral body if there is not a solid column of PMMA extending from the superior to the inferior endplate. Another case that needed a second vertebroplasty at the same level was one where PMMA passed in the central part of the vertebral body and immediately started to flow through the superior and inferior endplates into the adjacent disks. I stopped the vertebroplasty at that point. The patient still had some residual pain afterward while making beds in a tourist lodge. She returned for a second vertebroplasty, and PMMA was placed both into the right and left sides of the same vertebral body to fill the vertebral body more fully, with subsequent relief of symptoms.

Injection of larger amounts of PMMA requires careful observation of well-opacified PMMA during vertebral body filling. In many cases, it may be necessary to stop PMMA injection temporarily to let PMMA thicken or harden, after which injection can resume. Needle adjustment by advancing or withdrawing the needle slightly may be valuable in selected cases. In other cases, bipedicular injection or placing a new needle into the same needle tract may be helpful (3). Potential leakage of PMMA during injection is a concern of everyone performing vertebroplasty; however, small amounts of leakage recognized early that do not pass into the spinal canal or impinge on exiting nerves are well tolerated (4). Using conscious sedation, keeping the patient awake enough to respond to pain rather than general anesthesia, also allows the patient to respond as soon as any symptom arises during injection. Midline pain of presenting type has been acceptable during injection, except when injecting metastases. With metastases, it is important to check PMMA placement carefully when any pain develops during injection before instilling more PMMA. With osteoporotic or metastatic diseased vertebrae, pain other than midline requires circumferential check of PMMA placement to be certain leakage out of the vertebral body is not taking place. Finally, as has been recently published (5), "blush venography" may be very helpful to plan injection strategy.

In summary, I compliment the authors on their honesty in bringing forth the possibility that vertebral bodies can be re-treated; however, I strongly recommend that persons performing vertebroplasty reconsider accepting installation of only a minimal amount of PMMA, because such vertebra can be associated with further fracture in the remaining portion of the vertebral body without PMMA. As the authors have demonstrated in their article, such untreated vertebrae can be the source of continued or recurrent pain.

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Reply:

We are delighted by Dr. Gilula's interest in our article regarding the therapeutic benefit of repeat vertebroplasty in previously treated levels. He raises many interesting points about the appropriate endpoint of a vertebroplasty procedure and implicates inadequate cement volume as an explanation for our failed cases.

What are the goals of percutaneous vertebroplasty? Most practitioners would agree that the primary goal of vertebroplasty is to reduce or alleviate the acute symptoms associated with painful osteoporotic vertebral compression fractures. A secondary and, as yet, unsubstantiated goal of vertebroplasty is to prevent further, long-term vertebral body collapse. The issue of how much cement deposition is appropriate should be viewed in the context of these two very different, but complementary, goals.

How much cement is enough to alleviate the pain associated with acute and subacute compression fractures? We have conducted a painstaking, retrospective review of our data base to determine whether clinical outcome correlates with cement volume. A portion of this work was presented in Vancouver at the 2002 ASNR Annual Meeting (1), in which we compared low-volume (<3 mL) with high-volume (>3 mL) procedures and showed no difference in post-procedural pain. Additional analysis of these data, with correction for degree of collapse and vertebral level, has failed to show any positive correlation between cement volume and pain relief (unpublished data).

How much cement is enough to prevent delayed vertebral body collapse? There is currently little to no literature available that directly addresses this issue. In vitro studies have suggested that injection of as little as 2 mL of cement results in the restoration of vertebral body strength (2). Although it is intuitively tempting to assume that filling a vertebral body with the maximal amount of cement possible will provide maximal prevention of further collapse, at the current time this remains an unproven assumption. In addition, the maximal filling of a vertebral body could potentially increase its stiffness to a point that it makes it more likely that adjacent untreated levels will fracture. We would point out that the article in question described re-treatment sessions in 2.5% of our cohort, which we consider an acceptable failure rate in light of vanishingly rare complications.

Where does this leave us? Without clear understanding of the mechanism of action of vertebroplasty, it remains impossible to determine a priori whether a small or large amount of cement will be needed. If vertebroplasty achieves pain relief by "sealing a fracture line," we should seek out these fractures and target small amounts of cement into them. There are some data to suggest that disk space leakage of PMMA is associated positively with complete pain relief during performance of activities of daily living (3). If vertebroplasty works by preventing additional collapse and increased cement volumes are shown to be beneficial with respect to this, degree of filling should be maximized. We anxiously await research aimed at answering these questions.

Until we know for certain that the benefits of maximal cement deposition outweigh the risks, we will remain firmly in neither of Dr. Gilula's "minimalist" nor "maximalist" schools

of practice, but rather in the “judicious” school, in which we try to “minimize” extravasation-related complications yet “maximize” good outcomes.

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Small Aneurysm Size Is a Risk Factor for Perforation during Coiling

With interest we read the meta-analysis of Cloft and Kallmes (1) on perforation during coiling of cerebral aneurysms. The discussion mentions that “data in the reports of perforation in the literature are incomplete regarding aneurysm location and size” and “size of all of the aneurysms that did not become perforated also must be known to evaluate for differences in risk that are dependent on the specific location and size (page 239).” In our article regarding risk factors for procedural perforation (2), we compared the sizes of the seven aneurysms that were perforated with 257 aneurysms that were not. A statistically significant relation between small aneurysm size and perforation was found. Although our study was cited, this important finding was apparently overlooked by the authors.

In the choice between surgery and coiling, we believe it is important to know that small aneurysm size indeed is a risk factor for perforation during coiling.

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Reply:

We appreciate the interest of Drs. Sluzewski and van Rooij in our report (1). The purpose of our meta-analysis was to quantitatively evaluate specific characteristics of perforations complicating aneurysm therapy. The technique of meta-analysis allows for the collection and pooling of data from similar studies to try to answer questions that none of the studies have sufficient sample size to answer alone (2). Our goal was to pool data from all of the available case series that reported aneurysm perforation during endovascular therapy to more precisely determine the risk of this complication. Other than the report by Sluzewski et al (3), the reports used in our meta-analysis did not report data that would allow us to evaluate size of aneurysm as a risk factor for perforation. Therefore, the relationship of aneurysm size to rate of perforation was not amenable to meta-analysis. We did state that small aneurysm size may be associated with an increased risk of perforation (1). Random or accidental displacements of endovascular devices by a few millimeters that are trivial in a large aneurysm might lead to catastrophic rupture in the more confined lumen of a small aneurysm.

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Erratum

Biography Charles M. Strother, 41st President of the ASNR. AJNR 24:1715–1716. Page 1716, third paragraph should read:

Always on the lookout for new ideas and news of approaching clinical problems, Charlie's sabbatical in 1988 took him to **Overlege Ulleval Sykehus** in Oslo, Norway, where he worked with a number of Norwegian colleagues - **Pyder Eldivik, Finn Lilleås, Raidar Dullerud, Johan Johansen, Søren Bakke, Per Nakstad** – all of whom remain Charlie's close friends and colleagues.