# AJNR

## Reply

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# Letters

## Reporting Terminology for Lumbar Disk Herniations: Axial Segmentation of the Preneural Foraminal Portion of the Lumbar Nerve Roots

Through the past decades, many attempts have been made to standardize lumbar disk herniation–related terminology (1, 2). Two different, and not necessarily parallel, sets of characteristics of a herniated disk (HD) are taken into account in the reporting of cross-sectional images of the lumbar spine. First, is the absolute attributes of the HD (ie, morphology, location, volume, etc), and second is the relationship of the HD to its neighboring neural structures. The absolute attributes of a HD are only one set of factors that determine the pattern and degree of compromise of the neighboring neural structures. The morphology and cross-sectional areas of the involved lumbar vertebral and neural foraminae, along with the degree of the epidural fat, are the other determining factors.

Although standardized nomenclature has been proposed for the absolute attributes of a HD, descriptive terminology is used for the elaboration of the relationship of a HD to its neighboring neural structures. Terms such as "abuts," "impinges," "flattens," "indents," "compresses," "displaces," and "distorts" are useful in the description of the effects of a HD on the adjacent thecal sac (TS) and intracanalicular dural nerve root sleeves (DNRSs). The effects of a HD on specific intracanalicular nerve roots may at times also be ascertained by using high-resolution axial T2-weighted sequences of the lumbar spine. In particular, compromise (or maximal degree of compromise) of a lumbar nerve root (LNR) at a level just proximal to its respective neural foraminal exit could be pinpointed to a specific segment of the nerve.

In this context, a 3-tiered descriptive division of the preneural foraminal portion of a LNR, into the lateral thecal sac, junctional, and dural root sleeve segments (Figs 1 and 2) may be of value. This partitioning is particularly suitable at



Fig 1. Contiguous axial T2-weighted (3800/97.8) MR images through the L5–S1 level show the 3 segments of the preneural foraminal portion of the S1 nerve root. In this patient, individualization of all of the 3 segments is possible within a single zone.

*A,* Cranial-most image demonstrates the lateral thecal sac segment of the right S1 nerve root (*arrow*) in the right central zone.

*B,* Image shows the junctional zone segment of the right S1 nerve root (*long arrow*) in the right central zone. This segment, which is situated in the proximal portion of the dural root sleeve, is separated from the contents of the neighboring thecal sac by 2 adjacent layers of dura mater (*arrowhead*). The thickness of a single layer of dura mater is demarcated (*short arrow*) in the posterior portion of the thecal sac for comparison.

*C,* More caudally, image demonstrates the dural root sleeve of the right S1 nerve root (*arrow*) in the right central zone. Individualization of the contained dural root sleeve segment of the S1 nerve root is not possible in this patient.

Fig 2. Contiguous axial T2-weighted (3700/106.7) MR images through the L5–S1 level show the varying appearances (also refer to Fig 1*B*) of the proposed "junctional segment" of a lumbar nerve root.

*A,* Image demonstrates the junctional segment of the left S1 nerve root (*long arrow*) within the "pinched" portion (*arrowheads*) of the left ventrolateral angle of the thecal sac. The unrestricted contour of the right ventrolateral aspect of the thecal sac harbors the lateral thecal sac segment of the ipsilateral S1 nerve root (*short arrow*).

*B,* Image at a slightly more caudal level shows the proximal-most portion of the left S1 dural nerve root sleeve (*arrowheads*), the medial wall of which is in close apposition to the ventrolateral wall of the neighboring thecal sac. This portion of the dural root sleeve is also regarded as housing the junctional segment of the S1 nerve root (*arrow*).
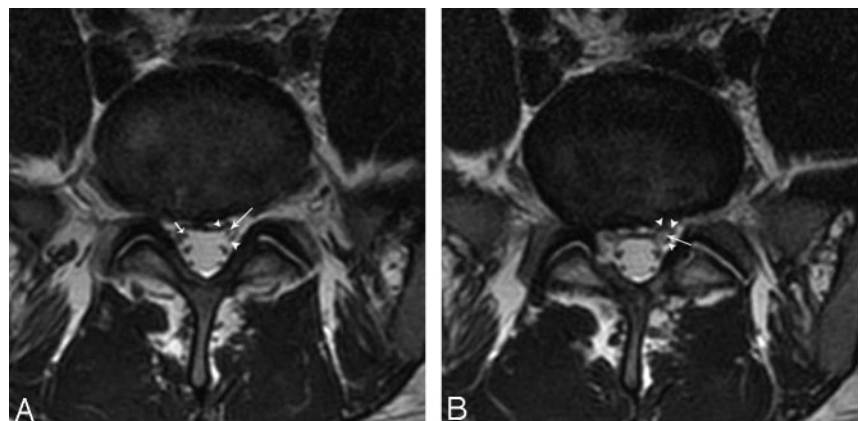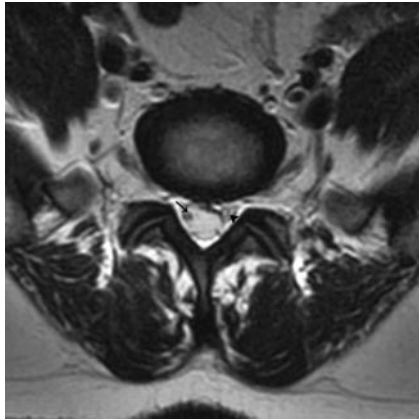
FIG 3. Axial T2-weighted (4000/98) MR image at the diskal-suprapedicular level of L5–S1 demonstrates the nonparallel segmentation of the S1 nerve roots. On the right, the lateral thecal sac segment (*arrow*), and on the left, the junctional segment (*arrowhead*), of the S1 nerve roots are visualized.

endorsed by the 2001 multidisciplinary task force (1). A particular "zone" may harbor different segments of a LNR (Fig 1), whereas a specific "level" may accommodate different segments of the opposing LNRs (Fig 3).

Awareness of the varying appearances of a LNR within the vertebral foramen may be of value in localizing the level—or maximal level—of neural structural compromise and correlating the imaging findings with the patient's symptoms. This is especially valuable in herniated disks that demonstrate a volume-neural structural compromise discordance (eg, a small-volume posteriorly HD may cause significant impingement of a neighboring ventrally located preneural foraminal LNR) (Fig 4), and may have a potential role in patient management.

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the lower lumbar levels that demonstrate a distinguishable lateral recess and parallels the compartmentalization of the preneural foraminal portion of dural coverings of the LNRs.

The intracanalicular portions of the LNRs demonstrate an intradural topography (the LNRs pierce the dura at the level of their respective neural foraminae) (3), either within the TS (Fig 1*A*) or in a DNRS (Fig 1*C*). These sleeves are pinched off from the ventrolateral angles of the TS. This TS-DNRS transition may be evident as "waisting" (Fig 2*A*) of the ventrolateral angles of the TS. More distally, the passage of the DNRS within the epidural fat (Fig 1*C*) is reminiscent of the intraorbital course of the optic nerve within the orbital fat.

These proposed segments do not necessarily comply with the localization system proposed by Wiltse et al (4) and later

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FIG 4. Contiguous axial T2-weighted (3800/97.8) MR images through the L5–S1 level with the S1 dural nerve root sleeves (individualization of the dural root sleeve segments of the S1 nerve roots within their respective dural sleeves is not possible in this particular patient).

*A,* Cranial-most image demonstrates the right (*long arrow*) and left (*short arrow*) S1 dural nerve root sleeves within the subarticular zones.

*B,* Image at a slightly more caudal level shows a mild central-subarticular zone disk protrusion that causes a relatively severe compression of the right S1 dural nerve root sleeve (*long arrow*). The left-sided intact complex is demarcated (*short arrow*) for comparison.

*C,* The S1 dural nerve root sleeves (*long and short arrows*) assume a symmetric appearance at a slightly more caudal level.

*Reply*

The multidisciplinary task force on lumbar disk nomenclature to which Dr. Khalatbari refers tried to devise a practical and simple classification of disk herniations, with the fewest categories, so that substantial interobserver agreement could be achieved (1). This is the main reason why the proposed system does not require observers to grade compromise of nerve roots by displaced disk material, an unreliable exercise in frequent situations (eg, suboptimal technical quality images, spinal stenosis, postoperative changes). In this system, the impact of a disk herniation on the thecal sac and individual nerve roots is suggested by assessing volume of displaced disk material with respect to available space. Thus, herniations are graded as mild, moderate, or severe, depending on extension of displaced disk material in the proximal, middle, or distal third of the available spinal lumen, in a specific anatomical zone, and at a specific anatomical level. In the example provided by Dr. Khalatbari (Fig 4), the right subarticular zone is completely obliterated by disk material at the suprapedicular level, and the term mild is inappropriate to classify such a herniation according to the system proposed by the multidisciplinary task force: this lesion qualifies as a severe herniation. This is an interesting case because it illustrates an important concept; that is, in the subarticular zone, displacement of a relatively small amount of disk material can cause severe nerve root compression. However, I can well understand the reluctance to use the term severe to describe such a localized herniation and, in retrospect, a numerical grading system (grades 1, 2, 3) would have been a better choice to describe the volume of herniated disk material. Radiologists who report on lumbar spine imaging studies according to the classification and definitions proposed by the task force are of course welcome to provide referring physicians all additional anatomical details that they feel may be clinically relevant, such as those discussed by Dr. Khalatbari. A grading system for nerve root compromise (0, normal; 1, contact; 2, deviation; 3, compression) has been recently proposed (2) and might be considered as a complement.

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### Location, Location, Location: Angiography Discerns Early MR Imaging Vessel Signs Due to Proximal Arterial Occlusion and Distal Collateral Flow

The location or topography of hypoperfusion is likely a critical determinant of outcome in acute ischemic stroke. The spatial distribution of a perfusion defect is directly influenced by the site of vascular occlusion and corresponding collateral flow. Such vascular localization is readily apparent on angiography and may also be evident on MR imaging as early vessel signs (EVS) or intravascular signal intensity derangements, including fluid-attenuated inversion recovery (FLAIR) vascular hyperintensity (FVH), and gradient-echo susceptibility (GRE SVS). Although similar in appearance, proximal and distal EVS may represent different pathophysiologic substrates.

Schellinger et al (1) provide an important analysis on the diagnostic and prognostic value of such relatively subtle EVS in hyperacute stroke cases treated with intravenous thrombolysis. They explicitly note that their study is unique, because "no clinical study to date has taken thrombus composition as being reflected by GRE (and FLAIR) images and its role for rtPA [recombinant tissue plasminogen activator] response into account" (p. 623). Similar to prior reports of EVS, the authors combine proximal and distal EVS as a single entity. Proximal and distal EVS are both associated with perfusion abnormalities induced by proximal arterial occlusion or thrombosis, yet distal EVS may not be due to the MR imaging signal intensity characteristics of thrombus.

Distal EVS may be associated with retrograde collateral flow distal to thromboembolic occlusion of a proximal artery (Fig 1). Distal EVS are typically manifest in middle cerebral artery occlusion as serpiginous structures in the sylvian fissure extending distally through the convoluted architecture of the cerebral sulci. It remains highly unlikely and unproven that such intravascular signal intensity derangements are due to thrombus extending along the entire course of the middle cerebral artery. Contemporaneous angiography may reveal patency of these vascular segments filled by slow, retrograde collateral perfusion via leptomeningeal anastomoses (Fig 1). FVH likely represents exceedingly slow collateral flow induced by retrograde perfusion of an arterial tree with diminished intravascular pressure. Distal EVS on gradient echo, or hypointensity in distal segments, may be associated with intravascular deoxygenation of collateral blood due to precapillary oxygen loss (2). The appearance of such distal EVS on gradient echo is dissimilar to GRE SVS described in the report by Schellinger et al (Fig 1*A*). Intravascular gradient-echo hypointensity in distal segments rarely demonstrates the prominent, and frequently coexistent, blooming artifact apparent as GRE SVS due to proximal thrombosis.

The authors also explore the enticing hypothesis that clot burden evident on MR imaging as manifest by EVS may be associated with thrombolytic efficacy. In contrast to endovascular reperfusion techniques using mechanical clot disruption, intravenous thrombolysis is dependent on the extent of clot surface that is exposed to the lytic agent at only the proximal and distal ends of the clot. Clot location, not necessarily clot burden, may influence thrombolytic exposure to the distal clot surface via collaterals. Conversely, proximal diversion of blood flow to collateral routes may also diminish forward pressure on the clot, hindering recanalization of thromboembolic occlusion. Clot location and corresponding collateral routes may therefore be critical variables in assessing not just tissue perfusion, but also thrombolytic efficacy in acute stroke.

EVS on MR imaging may provide valuable information regarding collaterals, not just thrombosis. Conventional perfusion MR imaging and MR angiography may be used for detection of proximal arterial occlusion, yet selective techniques such as regional perfusion MR imaging may more reliably delineate collateral flow (3). Until such noninvasive techniques are refined, angiography will remain the gold standard for defining the location of proximal occlusion and corresponding collaterals.

The location and not the mere presence of EVS on MR imaging may disclose distinct vascular correlates. In acute ischemic stroke, it is imperative to consider the location of proximal flow cessation, the location of thrombus, and the location of compensatory collateral flow.

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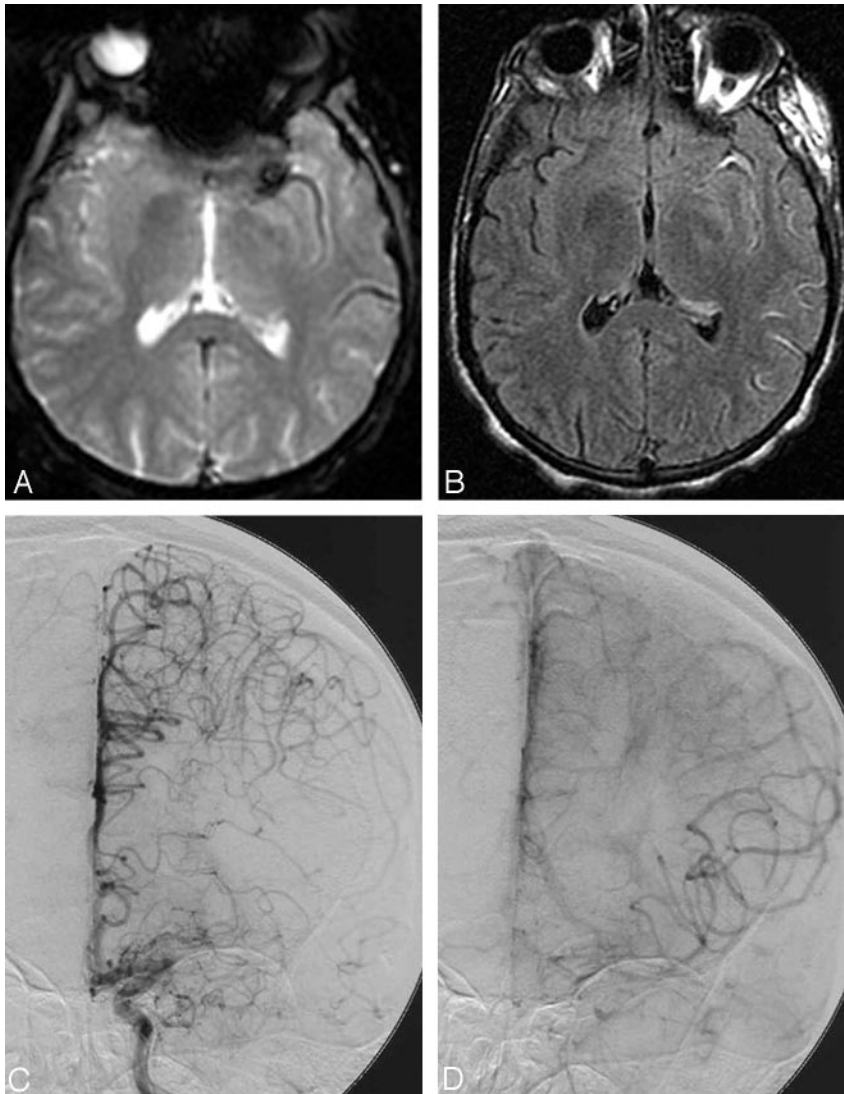*University of California*
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Fig 1. Contemporaneous MR imaging and angiography in acute left middle cerebral artery stroke. EVSs due to proximal thrombosis and distal collateral flow are evident on gradient-echo (*A*) and FLAIR (*B*). Angiography demonstrates proximal arterial occlusion (*C*) and slow, retrograde collateral flow (*D*).

## References

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*Reply*

We very much appreciated the letter of Dr. Liebeskind as well as the opportunity to reply to his comments. Dr. Liebeskind raises an important point with regard to location, thrombus extent, and early vessel signs (EVSs). His major concern is that not only the presence or location of EVSs, which indicate a thrombus, disclose a distinct vascular pathophysiologic correlate. EVS also represent areas with slow blood flow and therefore may indicate the location and extent of compensatory distal collateral flow (1).

First, the statement that the hyperintense fluid-attenuated inversion recovery sign (FLAIR HVS) and the gradient-echo susceptibility vessel sign (GRE SVS) may represent thrombus, as well as slow and/or retrograde flow (FLAIR HVS) of deoxygenated blood (GRE SVS), is entirely accurate and acknowledged in our manuscript in the second paragraph of the discussion (2, p. 622).

As stated in the introduction, our study had 3 objectives, the first and foremost being diagnostic accuracy of early MR imaging vessel signs. For this, the mere presence as compared with MR angiography (MRA)/perfusion-weighted imaging (PWI) and the concordance of location of the occlusion with the most proximal end of the EVS were analyzed. If present (sensitivity of 65.9%), the proximal end of thrombus/occlusion on FLAIR was correlated significantly ($r = 0.66$; $P < .001$) with MRA/PWI. For this diagnostic objective it is not relevant whether the further distal extent of the vessel sign is thrombus or slow flow. We do acknowledge, however, the fact that GRE besides its low sensitivity (34.1%) was not at all correlated with MRA/PWI and is often seen more distally. Whether this represents the distal deoxygenated part of the thrombus or collateral flow of deoxygenated blood is not clear. In fact, this could imply that the sensitivity of the hypointense GRE sign for the clot is substantially <34.1%, because the hyperacute, still-

oxygenated clot is not seen within 3 hours. Therefore, if necessary, the FLAIR HVS rather than the GRE SVS should be used diagnostically.

Second, we aimed to assess the prognostic value of early MR imaging vessel signs for clinical outcome, recanalization, and intracranial hemorrhage. Here we did not take collateral status into account. We agree with Dr. Liebeskind that a sufficient collateral status likely is a predictor for a good outcome, if recanalization and reperfusion can be rapidly achieved. We believe, however, that there are also patients with thrombus extending from the proximal middle cerebral artery into distal MCA branches beyond the trifurcation. This has been seen on CT scans where the hyperattenuated MCA sign extends into the MCA branches in the Sylvian fissure (dot sign). We concur that, in acute ischemic stroke, it is imperative to consider the location of proximal flow cessation, the location of thrombus, and the location of compensatory collateral flow. We doubt, however, that early MR imaging vessel signs provide this information reliably and arterial spin-labeling techniques for the assessment of local perfusion are far from being routinely applicable in a timely fashion (5 sections, 20 minutes in healthy patients) (3).

Finally, we hypothesized that the MR imaging signal intensity characteristics of early MR imaging vessel signs may reflect the structure of the intraluminal thrombus in patients with a vessel occlusion and can predict response to recombinant tissue plasminogen activator. Here, the presence or absence of good collaterals could be a stronger predictive parameter—and therefore a confounding variable—we take into account. This issue would have been more important if we had detected that clot composition as supposedly reflected by MR imaging EVS is a positive predictor of therapeutic response. In that case a type 1 error might have occurred.

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## Are Cervical Nerve Root Blocks "Safe and Effective"?

Wagner (1) reported on the use of CT fluoroscopy to guide cervical nerve root blocks (CNRB). He detailed the technique and described the initial experience at his institution. He concluded that CT fluoroscopy is a safe and effective alternative during CNRB.

We want to emphasize the serious complications associated with CNRB. Five well-documented cases of catastrophic neurologic complications following CNRB have been reported recently in the literature (2–6). Other publications have alluded to several other unreported cases. Described injuries include brain stem and midbrain hemorrhage, massive cerebellar infarct, occipital cerebral edema, and 2 cases of anterior spinal artery syndrome. Four of the reported patients died as a result of the injuries, and 1 had permanent neurologic sequelae.

The exact mechanism of central nervous system injury following CNRB is not known. In the case reports citing complications following CNRB, a standard technique of radiologic guidance with radiocontrast material and either CT or fluoroscopy was used. A series of mechanisms have been proposed to explain the different neurologic injuries associated with this procedure. One explanation is that CNRB can compromise a radicular artery and jeopardize blood supply to the cervical spinal cord. Possible mechanisms include occlusion by particulate corticosteroids or spasm of the blood vessel. Direct injury of the vertebral artery, embolic microvascular occlusion by corticosteroids, and direct neurotoxicity of radiocontrast agents placed into vertebral intracranial circulation have been proposed to explain injury to more proximal central nervous system structures.

Cervical radicular pain is a common problem that is estimated to occur in 1 of 1000 people per year. Recently, CNRB has gained increased acceptance in the treatment of cervical radicular pain. However, use of CNRB for radicular pain has been advocated on the basis of only observational studies. Although good results have been claimed, this treatment has not been subjected to rigorous controlled studies. Furthermore, CNRB has not been proven to change the natural history of cervical radiculopathy or any other disorder associated with chronic spinal pain.

Given the increased recognition of serious complications and the scarce evidence supporting the effectiveness of CNRB, it is imperative for clinicians to examine the safety and efficacy of this procedure and evaluate potentially safer forms of treatment. An objective assessment of the risks and benefits of CNRB needs to be performed before continued performance of this procedure can be justified. Although many practitioners consider the risk of spinal cord injury and stroke extremely small, the exact incidence and mechanism of injury during CNRB are not known. There is an urgent need to determine the incidence of severe complications, the circumstances associated with such complications, and assess the safest manner to perform CNRB. Only then will we be able to state with confidence that this is a safe and effective procedure.

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*Reply*

In response to Dr. Santiago-Palma's letter, I agree with most of the points that are made, and certainly the risks of this procedure should be known by anyone who performs them, as well as by their patients. The initial version of my article included a paragraph about the risks of cervical nerve root blocks (CNRBs) that touched on many of the same issues, but it was eventually deleted for diverging from the point of the technical note, which was meant to describe a new way of performing the procedure. Although I agree that there have been a number of cases involving serious complications during CNRBs, a closer review of these cases is necessary before making any assumptions about the safety, or lack thereof, of these procedures.

To begin with, many of the complications have been due to vertebral artery injections or injury, all of which, as far as I can tell, were performed with fluoroscopic guidance. Because CT-fluoroscopy (CTF) enables the radiologist to visualize the vertebral artery both before and throughout the examination, such complications can be avoided with the proper setup and technique. Therefore, although these cases should not be ignored when discussing CTF-guided CNRBs, their importance should be minimized. The second major complication that has been seen is the occurrence of cervical cord infarction, which has occurred in cases using CT, CTF, and fluoroscopic guidance. In these cases, it has been theorized that an injection of particulate steroid material into a dominant radicular artery coursing through the neural foramen is the causative factor, though direct proof of this occurrence is not described in the case reports. Certainly, the injection of contrast before steroid injection is important to ensure there is no vascular filling, and CTF is excellent for looking for such vascular enhancement. I also use a soluble steroid (betamethasone [Celestone], Schering-Plough Corp, Kenilworth, NJ) for all cervical injections and place the needle outside the neural foramen, rather than in it. This precise placement is made possible with the anatomic detail afforded by CTF to guide the needle tip and provide an additional layer of safety. I feel, however, that safety depends not so much on which imaging technique is chosen as on the skill and knowledge of the interventionalist.

It is interesting, however, that in a review of the cases in the literature, as well as medical-legal cases, a significant number of cord infarcts have occurred in cases where there has been contrast injection that demonstrated no vascular enhancement before steroid injection. This puzzling fact suggests that the culprit in at least some of these cases may be spasm of the radicular artery rather than direct injection into the vessel itself. If this is the case, there is really no way to avoid the rare instances when this may happen and in fact the presence of contrast material might act as a causative factor for vasospasm. Fortunately, these types of serious complications are rare when compared with the volume of cases in which these injections have been performed during the past half century. Although it would be interesting to find the exact incidence, the low numbers and the current state of the malpractice environment will likely prevent any meaningful number from being calculated.

In the end, Dr. Santiago-Palma's objection to my article seems to be my referring to CNRBs as "safe and effective." I agree that there have been complications and deaths (including at least 2 from unpreventable idiosyncratic drug reactions) that have occurred during these procedures and these risks should be discussed with the patient during the consent process. But ultimately, CNRBs are invasive procedures and as such have a risk of serious complications, including death, just like other invasive or surgical procedures. I stand by my statement that CNRBs are "safe and effective" procedures in well-trained hands. Not 100% safe or 100% effective, but in the world of spine intervention, what is?

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## Re: Angiographic Evidence of Aneurysm Neck Healing Following Endovascular Treatment with Bioactive Coils

We read with interest the paper by Gonzalez et al reporting their angiographic findings in 2 cases of intracranial aneurysms treated with bioactive coils (Matrix; Boston Scientific Corporation, San Jose, CA) in the April 2005 issue of the *AJNR* (1). The authors describe a well-defined radiolucent gap between the coil mass and the parent artery of the treated aneurysm, an angiographic finding they name the "white-collar sign." They speculate that this finding results from the formation of a thick connective tissue barrier at the aneurysm neck, on the basis of a histopathologic study performed on an animal model (2). Gonzalez et al seem to suggest that this new angiographic finding is specific to intracranial aneurysms embolized with bioactive coils.

We wish to add a personal observation of a similar finding in a 53-year-old female patient treated in 2003 for a wide-necked aneurysm of the left internal carotid artery (clinoid segment; Fig 1*A, -B*). The aneurysm was embolized with 9 bare platinum coils (Trufill; Cordis Neurovascular, Miami, FL). Immediate postembolization angiography demonstrated obliteration of the aneurysm without residual neck. Follow-up angiography 11 months later confirmed complete obliteration of the aneurysm and demonstrated a radiolucent band separating the coil mass and the lumen of the parent artery, similar to the "white-collar sign" described by Gonzalez et al (Fig 1*C, -D*).

Our observation shows that, though bare platinum coils produce a lesser degree of histologic reaction than bioactive coils, the documentation of a radiolucent band separating the coil pack from the parent artery is not specific to the latter and can be observed with bare platinum coils as well. Although it is tempting to speculate that such a finding represents a favorable prognostic factor for the long-term follow-up of aneurysms treated with coils, its exact implication remains to be evaluated.

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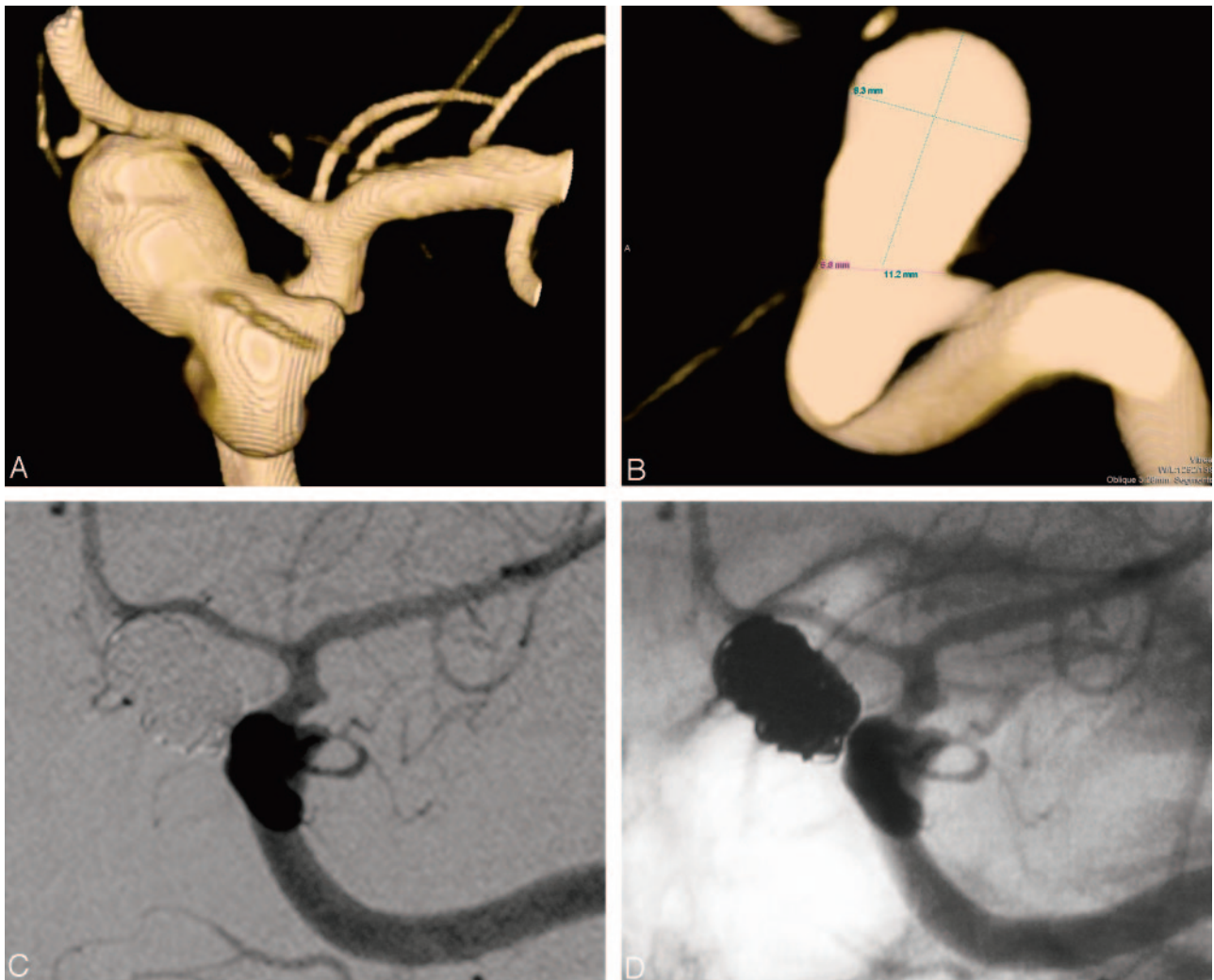*Johns Hopkins Hospital*
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FIG 1.    A 53-year-old female patient with an intracranial left internal carotid artery aneurysm.
*A,* Left internal carotid artery, 3D-digital subtraction angiography (DSA), oblique view, showing a paraophthalmic saccular aneurysm.
*B,* Left internal carotid artery, 3D-DSA, 2D reconstruction across the aneurysm sac and neck, showing the measurements of the aneurysm (height = 11.2 mm; length = 8.3 mm; neck = 5.8 mm).
*C,* Left internal carotid artery, DSA, subtracted oblique view of the follow-up angiogram obtained 11 months after treatment, confirming complete obliteration of the aneurysm sac.
*D,* Left internal carotid artery, DSA, unsubtracted oblique view (same view as *C*), showing a translucent separation between the coil mass and the parent artery.

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## Reply to Letter Regarding Interpretation of Results of ACTIVE Study

We appreciated the answer of Moret and Vinuela (*AJNR* 2005;26:2163) to our letter regarding the questionable interpretation of results of the ACTIVE Study on Matrix coils (1, 2). In the second paragraph of their answer, the authors agree on a fatal early rebleeding rate of 7% instead of the reported 3% after treatment with Matrix coils of ruptured intracranial aneurysms. Surprisingly, in the following sections the authors persist in reporting fatal early rebleeding rates as low as 2%.

In the evaluation of fatalities after any invasive medical treatment, it is not unusual to find, in hindsight, explanations for these disastrous events. For example, the indication for the particular treatment may have been wrong or the material was not optimized at the time. Also, in some cases the operator may have made a wrong decision or an overt mistake during the procedure. However, these explanations do *not* alter the fact that these fatalities occurred, and the frequency of these events should be clearly reported on accordingly, especially in a prospectively designed study such as the ACTIVE Study.

Therefore, we think it is not valid to report a fatal early rebleeding rate lower than 7% because "Two patients in the ACTIVE Study presented with dissecting aneurysms and should not have been included because the treatment strategy was not adapted for such aneurysms. *Thus, the rebleed rate should be 2%*" (emphasis added)*,* and because "At the time of the study, no other tools except Matrix coils were used to accomplish the treatment, which was a major limitation (this is not an excuse; it is a bias of the study and it was obviously a

mistake).” This sharply contradicts the conclusion of their study as stated in the *Matrix Newsletter*: “the results of 1st treatment with 1st Generation Matrix Detachable Coils are favorable”.

Furthermore, Moret and Vinuela imply that our statement “Apparently the Matrix coils allow residual filling of the aneurymal sac over an unknown period of time and during this period the patient is not protected against a rebleeding” is based on a visual appreciation of the packing density after a Matrix coil treatment. This is a misunderstanding. We did not raise this issue *at all*, nor was it mentioned in the *Matrix Newsletter*. We have merely cited the authors themselves on “residual filling of the aneurysmal sac immediately after embolization with Matrix coils” and “progressive thrombosis at 12 months follow up angiography” in about half of the cases. We emphasize that we do not question their study results, only their interpretation.

We sincerely hope that their statement “How many suspicious, negative, or destructive comments were made during the first 6 or 7 years of coiling aneurysms” does not refer to our criticism. We are deeply concerned about the devastating consequences of early rebleeding after coiling of ruptured intracranial aneurysms and have reported on this issue recently (3). In this study, we judged a fatal early rebleeding rate of 1.4% (95% CI: 0.57–3.09%) in a consecutive series of 431 patients after treatment with bare platinum coils already to be a major concern. In view of these findings, a 7% fatal early rebleeding rate after treatment with Matrix coils as found in the ACTIVE study is unacceptably high.

In conclusion, we think that the interpretation and conclusions of the ACTIVE Study remain questionable in the *Matrix Newsletter*. At the least, concern on the very high fatal early rebleeding rate should have been expressed, if not a serious warning. Because Matrix coils are widely used, we think it is mandatory to publish the results of the ACTIVE Study in a peer-reviewed journal.

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