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J F Bonneville

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LETTERS

Lumbar Neural Foramen Remodeling by Disk Herniations

The discussion of the case report by Dr. Castillo (1) of a neural foramen remodeling caused by a sequestered disk fragment prompts me to reply briefly. Dr. Castillo has written that bone remodeling or the neural foramen has not been previously reported in far lateral disk herniations. In an English-speaking paper (2) dealing with extraforaminal lumbar disk herniation (EFLDH), my coauthors and I have shown that 53% of our series of 47 EFLDH demonstrated a bony avulsion of the vertebral end plate facing the herniation, thus modifying the cortical bone lining the neural foramen. These bony changes correlated well with the usually responsible mechanism of EFLDH, ie, lateral bending and rotation of the trunk. This observation of the frequent avulsion of the bony site of attachment of Sharpey fibers in EFLDH, leading to a remodeling of the neural foramen was presented as an exhibit at the 26th Annual Meeting of the ASNR in Chicago, 1988 (3).

From a general point of view, non-American radiologic literature is sometimes not taken into account by American authors, thus leading to some frustration of non-American and especially European authors. I hope that the publication of my letter will attenuate this feeling.

J. F. Bonneville
Department of Neuroradiology
University-Hospital Jean Minjoz
25030 Besancon-France

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Reply

I have read with interest the letter by Dr J. F. Bonneville. He is correct in stating that I did not reference his paper in which he describes "scratch-like bony changes" at the level of extraforaminal disk herniations (1). However, my case report actually illustrates *EXPANSION* of a neural foramen in association with a sequestered disk fragment.

Mauricio Castillo
Department of Radiology
University of North Carolina School of Medicine
Chapel Hill, NC 27599

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Editor's note: I agree with Professor Bonneville that "non-American radiologic literature is sometimes not taken into account by American authors." However, in 2 years as an editor, it is my impression that non-North American authors just as frequently tend to overlook the North American literature. In any case, I do not believe the oversights (when they occur) are intentional but rather tend to reflect the difficulty in searching the enormous bibliographical databases that are expanding geometrically. The problem may lie in the "key words" used for the search, lack of access to particular journals, and, as in the case of the preceding correspondence, a true difference of opinion between authors as to the relevance of a prior publication to a particular manuscript.

However, I believe that authors who submit articles to this journal, both North American and non-North American, are persons of good will, and I suspect that oversights in citation are the result of the above listed reasons and are totally unrelated to the nationality of authors, although differences in language may occasionally be a barrier. If it is any comfort to Professor Bonneville, it is my observation that North American authors fail to properly cite their countrymen as frequently as they fail to cite non-North American authors.

I believe Dr Castillo has appropriately answered Professor Bonneville's letter. By the same token, I wish to thank Professor Bonneville for his letter which reiterates the fact that neuroradiology is an international discipline, and I am certain it will heighten the awareness of neuroradiologists everywhere to the need for a thorough search of the world's literature.

The Challenge of Carotid Occlusion

We read with interest the commentary by Eskridge (1) on the challenge of carotid occlusion published in the November/December issue of *AJNR*. He addresses the point of delayed hypoperfusion infarction due to inadequate collaterals and its potential prediction by performing HMPAO-SPECT studies during test occlusion. We perfectly agree with the pathophysiologic considerations. However, we would like to stress that HMPAO-SPECT uptake studies do not reflect cerebral blood flow (CBF) (2) as measured by standard ^{133}Xe-inhalation techniques. Asymmetric uptake gives qualitative information at best, but no quantitative data that is important to determine a preischemic level of CBF (3). HMPAO-SPECT studies cannot be easily repeated, so reactivity studies are cumbersome.

As measurements of CBF at rest give no information on the collaterals' ability to counteract an occlusion of a major cerebral vessel (4), functional stress tests to assess the cerebrovascular reserve capacity (CVRC) have been introduced that correlate well with changes in CBF (5) or O_2 extraction fraction (6). Clinical studies are a good parameter to predict hemodynamic hypoperfusion (7, 8).

This can be evaluated with transcranial Doppler (TCD) sonography, which can also be used to detect emboli in intracranial vessels. Therefore, TCD might play a key role in hemodynamic monitoring during endovascular procedures (9).

A. Piepgras, MD
P. Schmiedek, MD
V. Olteanu-Nerbe, MD
H.-J. Reulen, MD
Department of Neurosurgery
University of Munich
Munich, Germany

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Reply

I agree with Dr Piepgras that HMPAO-SPECT studies are not as quantitative as xenon CT. The drawback in the past with xenon CT has been the technical difficulty in performing the studies. There have been some technical difficulty in performing the studies. There have been some technical improvements in xenon CT of late and this should simplify the procedure and make it much more widely available.

We are currently making extensive use of transcranial Doppler in evaluating patients for temporary carotid occlusion. In particular we have been monitoring middle cerebral artery velocity changes and alterations in the pulsatility indices. We soon hope to correlate these numbers directly with cerebral blood flow as measured by xenon. You are also correct in stating transcranial Doppler is essential in any case where emboli are suspected. We have used this extensively and I am convinced that we have been able to prevent a number of strokes due to the fact that we rapidly instituted anticoagulation after the emboli were detected.

We are definitely in the preliminary stages of trying to determine which of these diagnostic tests will be the most useful as a predictor of cerebral dysfunction following carotid test occlusion.

Joseph M. Eskdridge
Assistant Professor, Radiology
University of Washington, Seattle, WA