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Can You Get a Stiff Back from Lack of Spinal Stiffness?

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nous sedation and analgesia are required. The procedure is less invasive than open surgery, and does not entail the risks of significant blood loss that may be encountered with invasive therapy. Embolization, although extremely helpful in preoperative treatment of these lesions, cannot directly reach or destroy the hemangioma because of the presence of an intervening capillary bed separating feeding arteries from the hemangioma (2). The risks of embolization to the spinal arterial supply, as well as surrounding normal tissue, is well recognized and a dreaded, if fortunately infrequent, complication of embolization (2). Radiation therapy carries the risk of possible toxicity to the spinal cord. Direct injection of alcohol, on the other hand, destroys the hemangioma without these problems.

Goyal et al, however, omit an important caveat. The authors encountered two significant complications. The first was penetration of the pleural space in treatment of a thoracic hemangioma, with complicating empyema. A less acute problem was also noted in that a treated vertebral body underwent collapse subsequent to ethanol ablation; interestingly, Heiss encountered the same problem (4). This is likely attributable to the osteonecrosis that can occur with injection of alcohol. Although data is inadequate to be certain, it may be that larger hemangiomas or hemangiomas treated with larger volumes of alcohol may be more prone to developing this complication, as osteonecrosis may be more likely to develop in those situations.

All patients, except one, were being treated for progressive neurologic dysfunction, and all patients had transient worsening of neurologic status. We presume this is related to alcohol-induced inflammatory changes, including swelling of the lesion. Steroids were administered empirically to all patients with possibly reduced inflammatory response. For those considering this treatment for their patients, neurologic worsening will have to be an acceptable and accepted event that will usually get better—although with increasing experience there may be instances when a neurologic improvement will not occur. Therefore, treating patients who present for reasons other than neurologic compromise may be difficult to justify.

Regarding technique, the injection of alcohol need not be ‘‘a blind procedure’’ as the authors have indicated. Without significantly changing the absolute nature of the alcohol, we have made alcohol radioopaque by mixing it in metrizamide powder when using it for other vascular lesions. This might allow for a more precise volume administration and reduce the ‘‘subjective assessment’’ of the rate of opacification.

The precise role for percutaneous alcohol ablation remains unclear. Unlike embolization, this technique allows actual destruction of the hemangioma. This presumably would make the risk of recurrence after treatment less likely, although in the current series recurrent (or more likely residual) hemangioma was noted in one instance. Because it appears that collapse of the vertebral body may be a significant complication of this procedure, its use as the sole treatment for symptomatic hemangiomas is, at the present time, questionable. It may, however, play an important role as an adjunct to surgery, allowing devascularization of the hemangioma without entailing the risks of angiography.

Another rapidly expanding technique is that of percutaneous vertebroplasty where image-guided injection of methyl methacrylate bone cement directly into the hemangioma is performed (5). Experience with this technique is increasing rapidly as injection of cement does have the advantage of providing structural support at the site of the ablated lesion. Long-term follow-up with this technique also is presently lacking. Cement, of course, creates a permanent incompressible cast of the hemangioma, which may be a problem in patients who have extensive expansion of the hemangioma with ballooning of the vertebral body or involvement of surrounding soft tissues or epidural space.

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Most neuroradiologists have a keen interest in spinal tumors or spinal vascular malformations, but have little curiosity for back pain, except if they happen to suffer from it. Unless back pain is associated with a definite medullopathy or radiculopathy caused by spinal stenosis or disk herniation, traditional neurologists and neurosurgeons share this lack of interest, and tend to refer afflicted pa-

tients to rheumatologists, anesthesiologists, physiatrists, and orthopedic spine surgeons. These specialists usually interact with musculoskeletal radiologists experienced in diagnostic and therapeutic spine procedures who know that most lower back pain sufferers do not show evidence of a classical radiculopathy. Those patients actually complain of radiating pain to the lower extremities that cannot be explained by nerve-root compression or irritation, which is referred from the disk itself (1). During recent years, noninterventional neuroradiologists have received an increasing number of requests to perform diskograms, facet blocks, nerve-root sleeves, or epidural injections, not to mention chemonucleolysis and vertebroplasty. These fields are both rewarding and demanding. Becoming a key player as a pain management consultant requires an open mind and some effort to understand complex biomechanical concepts.

In this issue of the *AJNR*, Haughton et al (page 1161) report the results of original experimentation involving 82 lumbar motion segments harvested from cadavers. These investigators have correlated the MR appearance of lumbar intervertebral disks with their stiffness. Motion segment stiffness is defined as the ratio of an applied load to the induced displacement or rotation. A loss of stiffness results in exaggerated movement of a spinal motion segment when torque is applied. In a strictly biomechanical perspective, loss of stiffness indicates spinal instability. Clinical criteria also have been proposed to define spinal instability, but these are more controversial. Trying to assess the validity of MR imaging for diagnosis of spinal instability is most relevant because the reliability of conventional lateral flexion and extension lumbar spine radiographs is quite poor. Using a classification of annular tears originating from the same institution, these investigators have found that the presence of annular tears on MR images of these specimens was significantly associated with a loss of motion segment stiffness. Moderate loss of stiffness was apparent in disks with transverse or concentric annular tears, and severe loss was documented in disks with radial annular tears.

This study is interesting in many aspects. In most cases, the diagnosis of a radial annular tear was made, not because the radial tear was directly observed, but because the involved disk revealed a high-intensity zone (HIZ) in the annulus fibrosus or a decreased central signal intensity with bulging of the annulus fibrosus. All suspected radial tears on MR images were confirmed by cryomicrotome pathologic examinations, which were reported in a complementary paper (2). This correlates very well with the findings of lumbar diskography in living individuals. In symptomatic patients, diskograms generally reveal radial annular tears in all disks with a definite loss of central signal intensity on T2-weighted images, and the majority of these tears appear responsible for contemporary symptoms. Surprisingly, these radial tears usually are quite localized. The MR images are misleading, because the loss of signal intensity usually involves the entire nucleus pulposus and inner annulus, and therefore suggests dehydration or some vague diffuse degenerative process. Most neuroradiologists who do not use diskography are reluctant to believe such disks may cause significant symptoms. It should be emphasized that radial annular tears do not represent features of the normal aging process. In this study, the age of cadavers ranged from 49 to 87 years (average age, 74 years) at time of death, and yet only 33% of the harvested motion segments demonstrated radial annular tears or advanced degeneration (defined here as a loss of more than 50% of the disk height or the association of large osteophytes). These findings are in line with those of Kieffer et al, who had performed lumbar diskograms in 106 cadavers, and had found radial annular tears in 37% of specimens from subjects over the age of 40 (3). Because they are present in only a minority of elderly disks, radial annular tears cannot be considered incidental findings of the normal aging process.

Transverse tears, however, defined as small horizontal tears at the junction of the outer annulus with the ring apophysis, usually are not associated with loss of central disk signal intensity, loss of disk height, or a bulging annulus. They are the rule rather than the exception in the older cadavers, and therefore they appear to represent a feature of the normal aging process. It is difficult to conceive they can account for a significant decrease in motion segment stiffness, as the results of this study suggest. Transverse tears were lumped with concentric tears in a group showing significant loss of stiffness in comparison to normal disks, although this loss was not as severe as the one observed in the radial tear group. The decision to proceed to such a grouping was probably dictated by sampling size requirements; unfortunately, the presentation of results does not allow one to assess the specific contribution of the transverse tear subgroup in the reduction of stiffness. I suspect that concentric tears had a much greater impact. The exact nature of concentric tears remains very controversial. The authors explain that they represent ''delamination'' between concentric lamellae of the annulus fibrosus, but Ahmed and Marchand, using a layer-by-layer peeling technique and microscopic examination of various cut surfaces of the annulus fibrosus, found no evidence of layer-to-layer connections or links between the concentric fibers of the annulus fibrosus (4). Until now, concentric tears have been thought to correspond to localized accumulation of mucoid material filling the potential spaces between the layers of the outer annulus. Is it really the case or do they actually represent bona fide localized annular tears that eventually can lead to the formation of a perceivable HIZ on MR images? And, if so, do radial tears simply result from the coalescence of such contiguous localized tears along a particular radius of the disk?

A study like this allows one to raise other questions which, I hope, will stimulate the authors to en-

gage in other similar fundamental studies. In the absence of annular tears, what characterizes the normal disk's aging process? Does motion segment stiffness increase or decrease as we get older? Is nature compensating for a loss of stiffness caused by ''age-related'' tears (ie, transverse and concentric) by producing osteophytes limited to the anterior and lateral aspect of the adjacent vertebral bodies, because they can be found in all skeletons of individuals over 40 (5)? Interestingly, disks with severe collapse and large osteophytes were shown to have increased stiffness with respect to disks with radial tears. After a radial tear has seriously compromised stability, the progressive replacement of the residual nucleus and annulus by collagenous fibrous tissue probably represents another mechanism nature uses to restore some of the lost stiffness.

With this study, Haughton et al have clearly demonstrated that a radial annular tear causes severe loss of motion segment stiffness and, therefore, significant biomechanical spine instability. The exact relationship between instability and pain, of course, remains to be established. As the authors suggest, exaggerated motion caused by instability may result in greater stress in adjacent innervated connective tissue, and may also cause greater risk of nerve-root compression and irritation in the foramina. I might add that, when severe pain occurs, a ''stiff back'' caused by muscle spasm may well be another mechanism nature has found to restore spinal stability temporarily.

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Unpublished Papers Perish

At the 1999 meeting of the American Society of Neuroradiology (ASNR), 293 papers, excluding posters and case reports, were presented. This represented 55% of submitted abstracts that were evaluated by the Program Committee. One may assume that the majority of such papers, having undergone this type of review, would find their way into peer-reviewed, indexed journals. It is, however, likely that such will not be the case. The data presented by Marx et al (page 1173), regarding papers presented in 1993 at the annual meetings of the ASNR and RSNA, suggest that only approximately 100 will find their way into leading journals in medical imaging such as the *American Journal of Neuroradiology, Radiology,* or the *American Journal of Roentgenology.* This is a surprisingly low number, and deserves further comment and analysis.

With only one third of presented papers making their way into peer-reviewed journals, the question is, what was the fate of the other two thirds? Did the authors simply not take the steps necessary to put their papers into publishable form (ie, never submitted for publication), or was the work submitted for publication, but subsequently rejected? Marx et al were not able to obtain the data necessary to separate these two possibilities. Nonetheless, if the former were the situation, one could hypothesize that either the authors never got around to formalizing their work into a full-length paper, or, upon deeper inspection, they simply did not have the appropriate data to construct a valid scientific article. If the latter were the situation, this has implications for the quality of submissions and the data submitted to a program committee. When evaluating submitted abstracts for a meeting, members of a program committee often only can determine if the ideas presented are new, potentially significant, interesting, and plausible, and using these criteria, such submissions are often accepted for presentation. But frequently, because of insufficient data, incomplete description of methods, absent images, or other pertinent information, the true scientific validity of the abstract is difficult to judge. Later, after the manuscript is submitted for potential publication, a deeper analysis is possible; then the paper may falter when a detailed analysis by journal reviewers and editors is undertaken. The paper then may never be published and perishes, suffering an academic death. With this in mind, one quickly can come to accept the reason many journals, including the *AJNR,* discourage citation of abstracts from various meetings. These may never have met the rigors of standard peer review and referencing. Such presentations could propagate invalid and erroneous conclusions.

To address this problem, a program committee could require a greatly expanded ASNR abstract to enable better evaluation of the scientific value of a submitted investigation. A glance back through the years (1984 to the present) of the ''Proceedings'' of the ASNR shows a remarkable improvement in this regard. Nevertheless, additional information