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Does Filling the Crack Break More of the Back?

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My Legs Only Hurt When I Stand Up!

In this issue of the *AJNR,* Hiwatashi et al provide an important reminder of several fundamental medical concepts that have sometimes been forgotten by many radiologists. This study looks at a group of 200 symptomatic spinal stenosis patients examined with MR imaging by using routine supine imaging techniques. By using a device that applied an axial load equivalent to 50% of patient body weight, additional supine axial loaded images were obtained. Upright MR imaging techniques take a similar approach and offer the additional possibility of dynamic imaging. A subset of 20 patients whose images showed substantial change after axial loading was then analyzed by three neurosurgeons with regard to treatment recommendations. On the basis of axial loaded images, changes in surgical therapy would have been made for as many as 10 of these patients. No attempt was made to determine actual improvements in outcomes for these patients.

Most of our statistics for effective imaging tests have tended to look at single variables, such as herniated disk versus no disk herniation, whereas lumbar spinal stenosis is a multivariable disease that can challenge correct interpretation of images. The lumbar spine is a dynamic structure that permits flexibility within well-defined physiologic limits, and, under normal circumstances, no neural compression occurs. As spinal integrity deteriorates, abnormal motion or structural shifting occurs, which may only be evident in a certain position or mechanical loading situation. These more subtle abnormalities can cause patient symptoms. Failure to recognize these more dynamic structural abnormalities can lead to suboptimal surgical therapy in some patients. It is interesting that about 5% of the patients in this study had such a change in recommended surgical therapy. Although this number is not large, this distinction could be very important for that patient group.

Physicians understand that appropriate patient treatment is determined by a combination of patient history, physical examination, and diagnostic testing. As radiologists, we naturally focus on diagnostic testing, and we have great confidence in the quality of our work. Many of us consider routine MR as the only diagnostic test that is needed for evaluating the lumbar spine. As Hiwatashi et al show, for many but not all patients, this assumption is correct. Well-trained spinal surgeons are aware of the dynamic element of lumbar spinal disease, and many of their procedures are predicated on stabilizing symptomatic "instability." For them, diagnostic imaging is often done to confirm their clinical findings before surgery. If the imaging does not confirm their clinical opinion, additional testing may be needed. Some spine surgeons continue to use lumbar myelography as a problem-solving examination, particularly when they are concerned about dynamic changes in the spine or there is a significant discordance between the history, physical examination, and the routine MR imaging findings. Anyone who performs myelography has seen important structural findings in some patients that were "missed" on routine MR or CT images. Unfortunately many radiologists do not fully appreciate this point and treat myelography as a relatively unimportant and perhaps obsolete examination. The data in the article suggest that a further reduction in the need for myelography is possible with improved MR imaging strategies.

Because many spine surgeons perform imaging as a confirmatory test before surgery that they believe is indicated on the basis of history and physical examination, it can be rationally argued that for a small group of spinal stenosis patients it is more effective to perform CT myelography. This remains the best dynamic evaluation presently available at most medical centers, and the CT assessment of the lumbar spine is actually very effective, especially with multidetector CT and two-dimensional multiplanar reconstructions. If this is done as a presurgical study, additional imaging is almost never needed. In the previously instrumented patient, CT myelography is often the best study available. The same cannot be said for MR imaging.

With these concepts in mind, and with Hiwatashi's information, it can be suggested that current routine lumbar MR imaging techniques are less than fully adequate for a small group of patients. With further refinements and validation of MR axial loaded and dynamic lumbar studies, the need for lumbar myelography will be further diminished. In the meantime, lumbar myelography is still valuable in deciding on clinical management and surgical approaches for spinal stenosis.

CHRISTOPHER G. ULLRICH
Guest Editorialist
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Does Filling the Crack Break More of the Back?

The article in this issue of the AJNR by Lin et al represents an important addition to the growing literature on percutaneous vertebroplasty. The authors have studied the effect of vertebroplasty on the risk of subsequent fracture in a subpopulation of treated patients, specifically those in whom cement was noted to extravasate into the intervertebral disk space. Based on a sample of 14 incident fractures in 34 patients, the authors conclude that the presence of cement within the intervertebral disk increases the risk of subsequent fracture and, thus recommend that such extravasation should be avoided, if possible.

Since inception of the technique, practitioners of percutaneous vertebroplasty have questioned whether the presence of a rigid, acrylic polymer within a vertebral body would increase the risk of fracture of neighboring vertebra. The answer to this question is highly relevant, since it would impact not only the overall safety of the procedure but also would address the wisdom of prophylactic vertebroplasty. Numerous biomechanical and clinical studies have addressed the question, yet no consensus has been reached. Several daunting challenges face those researchers attempting to answer the question, "Does vertebroplasty increase the risk of subsequent fracture?" Perhaps the most difficult challenge is identification of an appropriate control group. The development of subsequent fractures following vertebroplasty is a complicated issue made increasingly difficult by a lack of understanding of the natural history of osteoporotic vertebral collapse. It is well documented that patients presenting with a new osteoporotic compression fracture have a fivefold increased incidence of developing a new compression fracture within a year. In addition, certain vertebral bodies; eg, those located at the thoracolumbar junction, are more susceptible to fracture than others. What has not been described in affected, untreated individuals is the order in which osteoporotic vertebral bodies collapse and the degree of fracture susceptibility at individual levels. If one is to prove that vertebroplasty increases the risk of adjacent fractures, then the natural progression of the disease, including location of subsequent fractures, must first be identified.

Lin et al have circumvented the difficulty finding an appropriate control group by markedly narrowing the scope of their study to include only patients with or without endplate extravasation. While this focus limits the overall applicability of their findings, it does offer the ability to perform statistical comparisons between groups. If one accepts the authors conclusions that endplate extravasation increases the risk of subsequent fracture, then one might be tempted to alter one's practice to minimize or eliminate such extravasation, as suggested by the authors. This would include, as noted by the authors, such maneuvers as placing the needle away from the center of the vertebral body, altering the consistency of the cement, and diminishing the volume of cement injected.

We would readily accept a common sense approach to avoid large amounts of cement deposition into the disk; however, before one accepts the authors' recommendations to systematically avoid the endplate region of the vertebral body, a critical assessment of their data would be warranted. In our opinion, two specific issues merit further scrutiny. First, proof of association does not necessarily indicate causation. In other words, even though endplate extravasation may be associated with increased fracture risk, it is not yet proved that the extravasation caused the fractures. Second, it remains unclear whether avoidance of peri-endplate deposition of cement would limit the efficacy of vertebroplasty, and thus modification of technique should be done cautiously.

1) *Does the association between endplate extravasation and subsequent fracture prove that such extravasation caused the fractures?* Some doubt regarding this conclusion is raised by the lack of correlation between "small" and "large" amounts of extravasation in the authors' own study. Further, existing biomechanical data suggest that, rather than the disk space itself, it is the bowing of the vertebral endplate with loading that allows cushioning of an axial load. If one aims to fill a fracture line in the subendplate region, then perhaps increased fracture risk will prevail even in the absence of disk space extravasation. Alternatively, the biomechanical alteration of the involved disk space by the mere presence of an endplate fracture may predispose a patient to fracture of the opposing endplate. Additional doubt regarding the causative effect of disk space dysfunction may be raised by the fact that other disk space abnormalities, such as dessication and degenerative disk disease, have not been associated with increased fracture risk, to our knowledge. Finally, it remains possible that significant selection bias may explain the apparent association between endplate extravasation and fracture. For example, the presence of large, nonhealing subendplate fracture lines, which likely increase the incidence of disk space extravasation, may be a marker for a more "aggressive" osteoporosis with higher likelihood of additional fracture, irrespective of therapy.

2) *Would systematic avoidance of peri-endplate cement deposition diminish the efficacy of the procedure?* The first decade of widespread application of vertebroplasty has failed to give definitive insight into the procedure's mechanism of action. It remains possible that pain relief is afforded by filling of micro- and macro-fractures within the vertebra. If one accepts the authors' recommendation to avoid peri-endplate cement deposition, then the procedure may fail to relieve pain. There is no clinical data in the study under consideration to suggest whether pain relief was affected by cement deposition in and around the endplate. Two small clinical studies have addressed the question with diametrically opposed results. Substantially larger studies are needed to show this effect. Thus, when one identifies on preprocedural imaging a fracture line in a subendplate location, it seems coun-

terintuitive to purposefully avoid depositing cement in the fracture line, irrespective of the perceived risk of disk space extravasation.

In summary, the study by Lin et al is a welcome addition to a growing but still immature literature on percutaneous vertebroplasty. Their data are intriguing and point to the need for further study of the effect of vertebroplasty on the natural history of osteoporosis, specifically regarding risk of subsequent fracture. The study may however raise more questions than it answers, and systematic modification of vertebroplasty technique should await the availability of larger, prospective studies.

MARY E. JENSEN
Guest Editorialist
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Imaging NeuroAIDS

The neurologic complications of HIV infection remain a clinical challenge. In the early days of the epidemic, it became clear that the virus has a predilection for the CNS. Before the advent of antiretroviral therapy, a large proportion of HIV-infected individuals developed neurocognitive disorders of varying severity, including a profound dementia. With the introduction of antiretroviral therapy, and more recently with the clinical implementation of highly active antiretroviral therapy (HAART), the incidence of neurocognitive disorders has decreased dramatically. Although HAART has prolonged the life expectancy of HIV-infected individuals, it has raised the fear of an increasing prevalence of neurologic disorders in this population.

Neuroimaging has played a central role in the management of HIV/AIDS patients, particularly in the diagnosis of opportunistic infections and neoplasms that are seen in this population. Structural neuroimaging has had a limited role in the study of what has come to be known as "neuroAIDS." The neurocognitive abnormalities caused by HIV are thought to arise from injury and death of neurons; however, because the virus does not infect neurons themselves, neuronal injury is thought to arise by indirect mechanisms. The virus infects perivascular macrophages and microglia within the CNS, and it is believed that substances including cytokines and viral products generated by these cells ultimately result in neuronal injury. Early in the epidemic, severe atrophy and white matter abnormalities could be detected by use of imaging in patients with advanced neuroAIDS. In the era of HAART, by contrast, these findings are uncommon, except in populations with little or no access to health care.

Because few or no abnormalities are detectable by use of CT or MR imaging in the early stages of neuroAIDS, clinicians and investigators have used functional neuroimaging methods to measure objectively changes in the brain that are caused by the virus. Many methods have been employed successfully, including positron-emission tomography, single-photon emission tomography, blood oxygen level–dependent functional MR (fMR) imaging, dynamic contrast fMR imaging, MR spectroscopy, and standard diffusion MR imaging. In this issue of the *AJNR,* Ragin et al report that diffusion tensor (DT) imaging may be useful quantifying cumulative brain injury in neurocognitively impaired HIV-infected patients.

There would be many advantages to having a sensitive and reliable neuroimaging method suitable for study of early neuroAIDS. Despite the predilection of the virus for the brain, HIV encephalitis occurs in only one third of individuals who do not undergo therapy. This is thought to be due to both viral and host factors. Finding those who are susceptible to HIV-induced brain injury would be important to the administration of adjunctive therapies to prevent neuronal injury in this subset of patients. Such methods could also be important in elucidating the pathogenesis of neuroAIDS and developing suitable adjunctive therapy. The methods developed and used to date are not sufficiently sensitive to depict early changes in individual patients. The successful studies that have been performed to date have relied on cohorts of at least 10 patients. The great variability in progression of disease, not to mention the modification of its progression with HAART, makes studies of neuroAIDS exceedingly difficult to conduct.

Ragin et al demonstrate that DT imaging has joined the collection of functional neuroimaging techniques that may be useful in guiding the clinical management of neuroAIDS. They report that whole-brain fractional anisotropy (FA) was reduced in HIV-infected subjects and significantly associated with severity of dementia, as indicated by several widely used clinical and functional status measures. They also show that FA measures were more prognostic of dementia status than were apparent diffusion coefficient measures. They propose that FA measures the cumulative injury induced in the brain by HIV, that this methodology may provide insights into the biophysical basis of this injury, and that it may prove useful in understanding the pathogenesis of this disease. It is unclear whether DT imaging has the sensitivity for the early detection of this disease process.

The difference in FA that distinguished HIV-infected individuals from control subjects was quite small in Ragin et al's study. On the other hand, the dissemination of MR imaging systems with echo planar imaging capabilities has made DT imaging widely available. Also, there is little doubt that DT methodology will continue to evolve. It will be interesting to see how the evolution and application of DT imaging will affect our understanding of neuroAIDS and how it may help us in the management of patients with this disease.

R. GILBERTO GONZALEZ
Guest Editorialist
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The High-Field-Strength Curmudgeon

I undertook this assignment, to muse on the state of current 3T clinical imaging, with trepidation because, depending on the phase of the moon, I vacillate between wild unjustified, undocumented optimism of the technology and the opposite extreme of sounding like a fossilized curmudgeon incapable of appreciating technology (or, even worse, of not having the "vision thing"). Our 3T system (*head only*) was installed in June 2001 and runs half-time clinical, half-time research. Because this magnet replaced an aging (yet full-time) clinical system, considerable pressure exists to use it as efficiently as possible for a wide variety of clinical cases. This clinical caseload has not been viewed with excitement by the technologists, who by necessity know the foibles of the individual MR systems more thoroughly than do the neuroradiologists. Their enthusiasm for our 3T system is running slightly short that of spending a night at the Bates Motel.

The possibilities of 3T imaging have captured the imagination of radiologists and the spreadsheets of the manufacturers. The ongoing debate of low versus mid versus high field strength (1.5T) is now elongated to 3T and beyond. What is the reality of current 3T clinical imaging? The operative word is "clinical," and few sites perform routine protocols, in routine time slots, at this higher field strength for purely clinical reasons. 3T is not presently at the level of mainstream "bread-and-butter" imaging. To undertake a 3T system in a strictly clinical environment begs for, if not trouble, then intense dissatisfaction and a lot of time talking to "Applications." A clinical system needs to provide excellent image quality quickly and with a minimum of fuss. The concept of good image quality does not refer only to a high-resolution, heavily T2-weighted image but rather the full gamut of T1-weighted spin-echo, balanced, T2-weighted, and more specialized sequences needed in a daily practice. For brain imaging, the ability to produce a high-resolution coronal T2-weighted image of the hippocampus does not validate the complete imaging package at 3 T. The 1.5T platforms have a long history with multiple manufacturers, and the broad expanse of optimized sequences available at 1.5T does not seamlessly transition to the 3T realm.

The hype and promise of 3T relates to the increased signal-to-noise ratio of the higher field strength. Magnetization increases as the square of the field strength, while noise increases linearly giving a potential doubling of signal to noise from 1.5 to 3.0T. This increased signal-to-noise capital can be traded off for higher resolution images at comparable imaging times to 1.5T, better quality perfusion/diffusion studies, or spent for quicker examination times. In reality, this doubling of signal-to-noise ratio has been illusive. Frayne et al did not find the expected doubling of signal-to-noise ratio at 3.0T but an increase on the order of 30–60% (1).

In daily practice, we have not been able to achieve a reduction in overall imaging time because of the constraints of the longer T1 relaxation at the high field and power deposition (2). Producing T2-weighted images is the easy part, and good-quality, high-resolution images are readily obtained at 3.0 T. A reversed T2-weighted image has been encouraged for use on 3T systems as a single encompassing sequence (3). I am loath to give up the multiplicity of sequences that has served us so well, for so long.

T1-weighted images are a much different story. The prolonged T1 at 3T has necessitated the use of either a gradient echo T1-weighted image or an inversion recovery sequence to obtain reasonable gray matter—white matter differentiation. The constraints of power deposition (increasing as the square of the field strength) at the higher field translate into fewer sections with less anatomic coverage. This requires an interleaved sequence, doubling T1-weighted image examination time. T1-weighted image quality is degraded by the increased amount of chemical shift artifact at the higher field. One can obviate this by replacing the conventional T1 with a 3D gradient echo T1-weighted image, but because this is not standard for us at the workhorse field strength of 1.5 T, why should it all of a sudden be acceptable at 3 T?

Limitations also arise in our head-only system because of coil design that distorts the periphery of the images and mandates precise head positioning to achieve optimum signal-to-noise ratio. The images are suboptimal for clinical situations, requiring a high degree of spatial uniformity, such as preoperative stereotactic localization studies, which we still perform on the 1.5T systems. Granted, these problems should be diminished with coil and magnet design improvements and are not such a significant issue on whole-body 3T systems. On the plus side, this inherent problem of the head-only system does relieve one of having to comment on upper cervical degenerative

disk disease on the sagittal images, because it is not visible!

Spectroscopy has not yielded a quantum improvement despite the potential of a 100% improvement going from 1.5 to 3.0T, with its improved signal-to-noise ratio and larger chemical shift. Improvements from 20–50% in signal-to-noise ratio can be seen at the higher field strength because of problems related to shortened T2, field inhomogeneities, and increased line width (4, 5).

Not to be a labeled a complete Luddite, immediate improvements can be seen at 3T with nearly any gradient echo technique, such as MR angiography (MRA) and 3D gradient echo sequences. Contrast-enhanced MRA has also been successful demonstrated at 3.0T (6). Susceptibility effects have been surprisingly limited, and diffusion image quality has been very good. Blood oxygenation level–dependent functional imaging studies are excellent, although it is difficult to sell a system to a community hospital on the basis of volume of fMRI studies. In the end, 3T is an evolutionary, not revolutionary, technology. As such, growing pains are to be expected and will be overcome in the future. Five years would be a reasonable time line for full maturation of this technology. In the shorter term, however, I would be cautious about a technology driven by manufacturers and academics when your patient referral base is at stake.

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Member, Editorial Board

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