Degenerative disc disease in the lumbar spine can lead to significant back pain and is a clinical problem that affects a large portion of our population. The management of LBP resulting from symptomatic lumbar disc degeneration ranges from conservative care to complex surgical treatments with mechanical device placement involving fusion or motion preservation of the spinal segment. The current clinical treatments are directed toward treating the myriad pathological conditions arising from the end results of disc degeneration rather than to prevention or reversal. With a better understanding of the pathophysiological characteristics of degenerative disorders, current treatment options can be directed toward prevention and repair rather than simply treating the symptoms.

The degenerative process is a complex interaction involving the loss of proteoglycans, disc matrix, and the development of endplate sclerosis, which is currently the topic of many research studies. Although the effects of disc degeneration are of great interest, appropriate animal models of disc degeneration are critical for the study of proposed interventions as well as to further delineate the degenerative process. The purpose of this study was to characterize a porcine model for disc degeneration confirmed on magnetic resonance (MR) imaging studies and histological analysis.

### Object
Appropriate animal models of disc degeneration are critical for the study of proposed interventions as well as to further delineate the degenerative process. The purpose of this study was to characterize a porcine model for disc degeneration confirmed on magnetic resonance imaging (MR) studies and histological analysis.

### Methods
Twelve miniature pigs were used (weight 48–65 kg) to study degeneration in the lumbar spine. Under fluoroscopic guidance, the disc was percutaneously punctured with a 3.2-mm-diameter trephine to a 5-mm depth into the annulus fibrosus. Control and experimental levels were randomized among 6 levels in the lumbar spine. The unlesioned spinal levels were used as controls and were compared with lesioned levels. Magnetic resonance imaging grading and disc height were serially recorded preoperatively, and at 5, 8, 19, 32, and 39 weeks postoperatively. The animals were killed in groups of 3 at 7, 18, 32, and 41 weeks postinjury, and the discs were examined histopathologically.

### Results
Consistent, sequential, and progressive degeneration of the annular injury was observed on MR imaging and histopathological studies from the time of injury to the final time point. The disc height and the disc height index also sequentially decreased from the time of the injury in a consistent manner. The uninjured control levels did not show any progressive degeneration and maintained their normal state.

### Conclusions
Based on MR imaging and histopathological findings, the authors demonstrated and characterized a reliable model of sequential disc degeneration in miniature pigs with percutaneous injury to the annulus fibrosus. In the early stages, as soon as 5 weeks after injury, significant disc degeneration was seen on MR imaging grading with decreases in disc height. This degeneration did not improve by the final time point of 39 weeks.

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### Key Words
- disc height index
- intervertebral disc degeneration
- magnetic resonance imaging grading
- miniature pig
Porcine IVD degeneration model
degeneration can be characterized in patients on radiography, they have not been characterized with MR imaging or histological studies, and the cause of disc degeneration remains unknown. The molecular effects, the disc matrix components, and the cellular changes in degenerative disc disease have also been documented, but again, the causes of these changes are still being studied.1-7,13,16,20 Intervening in this process and reversing or preventing disc degeneration would be the ultimate goal.

In studying the degenerative process, animal models are critical to allow us insight into the pathogenesis and the possible use of biological factors in the treatment of this condition. To develop effective biological treatments, experimental animal models with similarities to the pathophysiological process of disc degeneration in human patients are required so that researchers can assess the biological changes in IVDs and to evaluate the effects of regeneration materials.

A rabbit model of chronic, reproducible, progressive disc degeneration for the development of new therapeutic interventions has already been described.8-12,19-21,23-25 In this model, the anterolateral annulus fibrosus in the lumbar discs of adult rabbits was injured, resulting in slowly progressive and reproducible degenerative changes that can be assessed on MR imaging, radiography, and histological studies. These are similar to the changes seen in human IVD degeneration; however, rabbit discs are very small compared with those in humans. Larger animal models are needed to study the pathogenesis and treatment of disc disease that may be more relevant to the human disc. The purpose of the present study is to describe and characterize a porcine annular injury model of disc degeneration, characterized with MR imaging studies and histological analysis.

Materials and Methods

Experimental Animals

Twelve adult female miniature pigs (Micro-Yucatan; weight 48–65 kg, age 15–24 months) provided by Medtronic, Sofamor Danek were used in this study. The Animal Research Committee at the University of California at Los Angeles approved the surgical protocol prior to the performance of animal experiments. The animals were cared for according to the Medtronic Physiological Research Laboratories Standard Operating Procedures and the Guide for Care and Use of Laboratory Animals. The animals were kept in housing units that met the recommended weight–space specifications, and were provided with water and nutritionally balanced feed.

Surgical Procedure

Prior to intubation, animals were given atropine (2.8 mg) and telazol (165 mg) with acepromazine (1102 mg). Isoflurane was used to maintain the animal at an appropriate level of anesthesia. The animals were placed in the prone position, and 2 lesions were created to maintain the animal at an appropriate level of anesthesia. Isoflurane was used to maintain the animal at an appropriate level of anesthesia. The animals were then placed tail first in the right or left decubitus position in a 1.5-T MR unit (Siemens Symphony 2002B software). The animal was positioned on a spine array coil so that lumbosacral MR images could be obtained. Pulse oximetry and end tidal CO2 measurements were used to monitor the hemodynamic and anesthetic levels of the animals. Sagittal T1- and T2-weighted images were obtained using 4-mm slices and a 25% distance factor. A preseturation band was placed anterior to the spine to cancel out abdominal motion. Axial T1- and T2-weighted imaging of disc spaces was performed. Eight groups of 3-mm slices with a 27% distance factor were performed at each lumbar IVD space, starting at the first disc above the sacrum.

Disc Height Measurement

Processing of the MR images was performed using eFilm software; T2-weighted sagittal images were selected for the appropriate animal and time point. During the first of the triplicate measurements, the slice closest to the midline was selected based on the diameter of the spinal cord. Due to their orientations, some images required the use of 2 different slices at different points along the vertebral axis. During the second set of the triplicate measurements, investigators checked the results with the first set of measurements to ensure that the same image slices were used. The brightness and contrast were adjusted so that the endplates of the VBs were as defined as possible. The measurement lines were “roughed in” for all VBs in the image slice, and the image was magnified 400% (magnification × 7.4). Medial measurement lines were positioned along the midsections of the VBs. Anterior and posterior measurement lines were positioned parallel to the medial lines and ~0.5-cm apart. Pinpoint endpoints of the measurement lines were located over the endplates of the VBs, and additional measurement lines were drawn over the IVDs, placing the endpoints directly over those used for the VBs.

Displayed numbers were clicked and dragged to positions near their corresponding measurement lines. Screens were captured and pasted into a corresponding disc height spreadsheet. Note that eFilm software may shift existing measurement lines slightly; very small scrolling adjustments were required to maintain correct positioning of endpoints. The numbers from the screen captures were transcribed into the spreadsheet. The final disc height was determined with the software tool pointed directly to the disc height in the anterior, middle, and posterior portions of the IVD to be measured, and the total value was divided by 3. Disc height index calculations were performed as described by Masuda et al., by averaging the measurements obtained from the anterior, middle, and posterior portions of the IVD and dividing them by the average of adjacent VB heights.

Magnetic Resonance Imaging Degeneration Grading

Lumbar disc degeneration (Fig. 1) was evaluated with the Pfirrmann grading scheme.18 The discs were assigned 1 of 5 grades based on MR imaging findings. In Grade I, the structure of the disc is homogeneous with a bright, hyperintense white signal intensity and a normal disc height. In Grade II, the structure of the disc is inhomogeneous with a hyperintense white signal. The distinction between the nucleus and annulus is clear, and the disc height is normal, with or without horizontal gray bands. Grade III discs are inhomogeneous with an intermediate gray signal intensity. The distinction between the nucleus and annulus is unclear, and the disc
For each disc level of interest, the statistical comparisons were analyzed. Each decalcified sample was used to evaluate the morphological characteristics of cells in the samples, Masson trichrome was used to evaluate connective tissue, and toluidine blue to examine the extracellular matrix of the cartilage. Samples, Masson trichrome was used to evaluate connective tissue, and toluidine blue to examine the extracellular matrix of the cartilage. Histopathological Methods. Each decalcified sample was processed routinely, embedded in paraffin, cut in 4-μm slices using a microtome, mounted on glass slides, and stained for light microscopy evaluation. In some cases, the decalcified samples were difficult to cut with a microtome, and these samples were soaked in glycerin prior to embedding and sectioning. Hematoxylin and eosin was used to evaluate the morphological characteristics of cells in the samples, Masson trichrome was used to evaluate connective tissue, and toluidine blue to examine the extracellular matrix of the cartilage.

Statistical Analysis

Statistical analyses included the Mann–Whitney U-test for non-parametric variables (MR grades, disc height, and DHI on lesion and control specimens). Comparison with preoperative and postinjury MR imaging grading, disc height, and DHI were analyzed using the paired Student t-test. Results were considered significant at probability values < 0.05. Statistical comparisons were analyzed with commercially available software (SPSS 12.0, SPSS Inc.).

Results

Radiological Results

The level of IVD lesion was SL-6 in 7 lesions (analogous to the human L1–2 intervertebral space), SL-5 in 3 (L2–3), SL-3 in 4 (L4–5), SL-2 in 3 (L5–6), SL-1 in 5 lesions (L6–S1). None of the animals received lesions at SL-4 (L3–4). A total of 22 lesions were created. There were 13 control IVD spaces, including 2 at SL-6, 2 at SL-5, 3 at SL-4, 2 at SL-3, 2 at SL-2, and 2 at SL-1. On MR imaging grading evaluation, results for the control levels were recorded as Grade I from the beginning of the examination until 39 weeks. In the lesioned spinal levels, the preoperative MR image was also Grade I. However, grades progressively worsened over 5 weeks, reaching a median score of 3.5 points (3.44 in the SL-6 group, 3.33 in the SL-5 group, 3.25 in the SL-3 group, 4.33 in the SL-2 group, and 3.2 in the SL-1 group). These values were significantly worse than those in the control group at as early as 5 weeks postinjury (p < 0.05). As seen in Fig. 2, preoperative MR results in all levels of the lumbar spine were categorized in Grade I, and follow-up data at all operated levels were quite comparable at Grades III and IV (p < 0.05). Grading of the final MR images was recorded to show mean grades of 3.5 at SL-6, 3 at SL-3, and 4 at SL-2 in the pigs that survived until the end of the study. The mean MR imaging grades assessed between 5 and 39 weeks did not change, indicating that the MR imaging grading score (denoting disc degeneration) could not improve spontaneously.

Changes in Disc Height

Preoperative mean disc heights on the control and lesion group were recorded as 0.41 cm and 0.43 cm, respectively. Mean disc heights at control levels at 5, 8, 19, 32, and 39 weeks after lesioning were recorded as 0.41, 0.41, 0.41, 0.42, and 0.42 cm for each time point, and the mean disc heights at lesion levels were 0.39, 0.38, 0.36, 0.36, and 0.38 cm (Table 1). In the follow-up periods, the disc height in the lesion group was found to have a lower value than in the control group (p < 0.05), except at 39 weeks (p = 0.12). For a more accurate analysis of the change in disc height, DHI was reevaluated in terms of the change in disc height. The preoperative DHI at the control and lesion level were individually noted to be 0.154 and 0.144, respectively, but the follow-up data differed at all of the follow-up readings in both groups, showing prominent decreases only in the lesion group (p < 0.05). On MR grading at 39 weeks, the lesioned levels for disc height and DHI were lower than those on the preinjury assessments and showed significant differences (p < 0.05; Fig. 3).

Gross Anatomical Results

At 7 weeks, the injured discs showed a loss of nucleus height is normal or slightly decreased. The structure of Grade IV discs is inhomogeneous with a hypointense dark gray signal intensity. The distinction between the nucleus and annulus is lost, and the disc height is normal or moderately decreased. In Grade V, the structure of the disc is inhomogeneous with a hypointense black signal intensity. The discs were graded by 4 spine surgeons (1 neurosurgeon and 3 orthopedic surgeons) blinded to the study.

Interobserver Reproducibility

All MR imaging grading, disc height, and DHI data were analyzed by 4 experienced surgeons blinded to the study. Interclass correlation coefficients were calculated to determine the interobserver reproducibility of this MR imaging data. The correlation was > 95%.

Pathological Analysis

Gross Anatomical Methods. For each disc level of interest, the VBs on either side of the IVD were bisected midbody using a reciprocating saw to create a “bone-IVD-bone” structure. Any bone tissue proliferations overlying the IVD were cut down to the surface of the disc. A sharp blade was then used to bisect the disc in the transverse plane.

Histopathological Methods. Each decalcified sample was processed routinely, embedded in paraffin, cut in 4-μm slices using a microtome, mounted on glass slides, and stained for light microscopy evaluation. In some cases, the decalcified samples were difficult to cut with a microtome, and these samples were soaked in glycerin prior to embedding and sectioning. Hematoxylin and eosin was used to evaluate the morphological characteristics of cells in the samples, Masson trichrome was used to evaluate connective tissue, and toluidine blue to examine the extracellular matrix of the cartilage.

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pulposus mass and cavity formation with fibrillation. The tracts through which the injuries were created were visible through the annulus. At 18 weeks after lesioning, the injured discs showed a loss of normal nucleus pulposus, with 1 disc nucleus very dry with cavity formation and fibrillation, and the other disc nucleus retaining a small amount of glistening material and no evident fibrillation. Tracts were visible through the annuli of both discs. At 32 weeks, the injured discs showed a prominent tract, a dry nucleus pulposus, prominent fibrillation, and cavity formation along the tract from the periphery of the annulus and throughout the nucleus. At 41 weeks, the injured discs varied in appearance. One did not have a tract through the annulus, and showed dense connective tissue adjacent to some mildly disorganized peripheral lamellae, as well as bone present in the connective tissue at the left lateral aspect. The nucleus of this disc was asymmetrical (the left side was larger than the right), with glistening to drier areas present. The second injured disc had an extensive large tract present through the left lateral annulus with disruption of lining annular fibers. The nucleus was dry, had some cavity formation, and was discolored yellow (Fig. 4).

**Histopathological Results**

At 7 weeks, 25% of injured discs had inflammation within the annulus, and 75% of injured discs showed some degree of vertebral endplate bone loss. At 18–32 weeks, no inflammation was shown within discs, although 50% of injured discs showed vertebral endplate bone loss associated with extensive connective tissue proliferation. At 41 weeks, 50% of injured discs had mild endplate bone loss. In addition, there was extensive separation between the vertebral endplate bone and disc margin at many disc levels, with loss of intervening chondrocytes. In all of the lesioned discs, the nucleus pulposus was histologically abnormal; 9 of the 10 manipulated discs showed some degree of sepa-

**TABLE 1**

*Disc height and DHI values in injured and uninjured groups*  

<table>
<thead>
<tr>
<th>Time Interval (wks)</th>
<th>Injured Group</th>
<th>Uninjured Group</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>w/in 1 wk preinjury</td>
<td>0.43 ± 0.02, 0.144 ± 0.010</td>
<td>0.41 ± 0.02, 0.154 ± 0.036</td>
<td>0.16, 0.45</td>
</tr>
<tr>
<td>5</td>
<td>0.39 ± 0.01, 0.132 ± 0.007</td>
<td>0.41 ± 0.02, 0.140 ± 0.005</td>
<td>0.02, 0.00</td>
</tr>
<tr>
<td>8</td>
<td>0.38 ± 0.05, 0.134 ± 0.009</td>
<td>0.41 ± 0.02, 0.140 ± 0.005</td>
<td>0.03, 0.04</td>
</tr>
<tr>
<td>19</td>
<td>0.36 ± 0.02, 0.121 ± 0.012</td>
<td>0.41 ± 0.01, 0.139 ± 0.009</td>
<td>0.00, 0.00</td>
</tr>
<tr>
<td>32</td>
<td>0.36 ± 0.03, 0.121 ± 0.011</td>
<td>0.42 ± 0.03, 0.139 ± 0.009</td>
<td>0.01, 0.01</td>
</tr>
<tr>
<td>39</td>
<td>0.38 ± 0.03, 0.126 ± 0.012</td>
<td>0.42 ± 0.02, 0.143 ± 0.002</td>
<td>0.12, 0.01</td>
</tr>
</tbody>
</table>

*All values are given as mean ± standard deviation. Disc heights are given in centimeters. Abbreviation: DH = disc height.
ration at the bone/disc interface. Inflammation was rarely observed, and variable amounts of proliferating connective tissue (fibrocartilage or fibrocollagen) were occasionally present in the vertebral endplates, with associated mild to moderate bone loss. Granular acellular debris was present in the nuclei of some discs (Fig. 5).

**Discussion**

The treatment of degenerative disc disease has moved into a state of biological prevention or repair in order to combat the changes that are associated with significant clinical symptoms.\(^5,11,13\) Although animal models cannot elucidate the pain response or the effect of any biological intervention on low-back symptoms, the degenerative changes can be followed radiographically and histologically. Recent studies have reported the positive effect of growth factor infusion or stem cells injected into degenerative discs in small animal models.\(^1,4,10,13-16,21,24,27\) However, in spite of these encouraging data, these results cannot be directly applied to humans.

One of the reasons for this relates to the significant size difference between rabbit and human discs. The size of the needle used to create the annular injury is relatively massive for the rabbit disc, however, similar-sized needles have shown no effect on long-term results when used for injections into human disc segments. In addition, the size differential also shows greater variance when looking at the differences in the biology, area needed for nutrient diffusion, endplate size, and the actual biomechanical differences. Although the rabbit model is currently the standard for biological interventions for degenerative disc disease, there are other limitations that make larger animal models perhaps more attractive and possibly more similar to the human disc segment. A chronic, reproducible degeneration model in a larger pig may help to confirm the effect of newly developed growth factors or agents that may be initially tested in the smaller rabbit model. One limitation of all these models, including the porcine model described in the present study, is that they may not truly represent natural human disc degeneration.

The rabbit annular puncture disc degeneration model is an injury model with validated, subsequent progressive degeneration.\(^9,22\) Most importantly, this model has provided the ability to assess quantitatively the efficacy of biological therapies, including growth factor injections. The original proposed methods to stimulate degeneration in the rabbit is a 16–21-gauge needle punctured to a 5-mm depth into annulus fibrosus, which also provides a small injury extension into the nucleus pulposus.\(^9,22\) The model attempts to limit any extrusion of nucleus material through the injury portal. In humans, the first mechanism of disc degeneration appears to be a loss of hydration in the nucleus pulposus with protrusion of nucleus pulposus being a late response after degeneration has progressed.

The current porcine model, like the previous small animal models, attempts to limit the injury to the annulus fibrosus, which would hopefully allow the resulting nucleus degeneration to progress along a more natural pathway. Authors of previous studies have reported that this injury induces progressive failures in the inner annulus and nucleus, including loss of distinction between the annulus and nucleus, and then marked degeneration of the nucleus.\(^9,22\)

Our porcine model does differ in some ways from the earlier published studies. First, in this study, we inserted a guidewire into the disc and a trephine (3.2 mm) that passed over the wire was used to create a lesion. This trephine was punctured into the disc to a depth of 3 mm into the annulus fibrosus. Based on anatomical dissections, this 3-mm depth into the annulus fibrosus of the pigs did not reach the nucleus pulposus, limiting this to a purely annular injury model. Second, an open surgical approach through a large flank incision with open exposure of the disc and dissection of the paraspinal soft tissues has been used in prior studies.\(^9,10,15,16\) The model in the present study uses fluoroscopy to make this injury through a percutaneous stab injury without a large dissection and open surgery that would disrupt the paraspinal surrounding soft tissues. The disc degeneration was slower and perhaps a more natural progression over a longer period of time.

In prior studies in the rabbit model, signs of degeneration...
were reported as early as 3 weeks after annular injury, with definitive postinjury degeneration at 4–8 weeks.\(^9,22\) The porcine model begins to show degeneration after 5 weeks, with changes in the nuclear material with chondroid tissue appearing, severe clefting extending to the peripheral portion of the disc, sclerosis, and ossification of vertebral endplates.

The disc degeneration seen in the MR images as described in other studies was evaluated by taking quantitative measures of the nucleus pulposus from degenerative discs, unlesioned discs, and repaired discs, with grading according to the scales proposed by Thomson (Grades I–IV) or Pfirrmann et al. (Grades I–V).\(^10,18\) In the present study, we adapted the 5-point Pfirrmann grading system to a 4-point evaluation of IVD degeneration in pigs. During MR imaging evaluation, preexisting disc degeneration could be found in a few levels of some of the pigs and they were excluded from our study so that the mean preoperative MR imaging grade in all pigs was Grade I. The type of injury to annular fibrosus using 2–3-mm diameter trephine provoked Grade III to IV changes at just 5 weeks postinjury. A rating of Grade III indicated that the nucleus pulposus had changed from a white signal into a black signal, demonstrating decreased water content in nucleus pulposus. In

![Figure 4](image1.png)

**Fig. 4.** Photograph of gross findings at 41 weeks. *Left:* Normal disc with prominent border between nucleus pulposus and annulus fibrosus. *Right:* Lesioned disc with indistinguishable border between nucleus pulposus and annulus fibrosus. The amount of nucleus pulposus has decreased compared with normal disc. Scar formation on the left side of the annulus fibrosis for injury routes is shown.

![Figure 5](image2.png)

**Fig. 5.** Photomicrograms. *Left:* Typical intact, unmanipulated disc with normal nucleus pulposus (N), dorsal annulus fibrosis (d), and ventral annulus fibrosis (v) is demonstrated. Orientation: Cranial to left, dorsal at top. *Right:* Mildly disrupted disc profile showing condensed and fragmented abnormal nucleus pulposus with hypertrophy of fibrocartilage. Note thickening and hypertrophy of the fibrocartilage at the cranial and caudal bone/disc interface. H & E, original magnification × 10.
this disc degeneration model in pigs, injury to nucleus pulposus could be deduced only from annular injury without puncture of the nucleus. The degenerated disc did not exhibit improvement of grading on MR imaging throughout the 39 weeks’ observation period, with no evidence of spontaneous recovery to Grade I or II. Thus, injury induced by an annular trephine is a reliable method for initiating a progressive form of disc degeneration in pigs. Sobajima et al. reported that disc degeneration in intact control discs could be found in follow-up, possibly because of their proximity to and interaction with the adjacent degenerative disc. However, the noninjured discs in our animals did not show degenerative changes on follow-up MR images. Our porcine model does not induce degeneration in the noninjured discs from any adjacent-level phenomenon.

Disc height in the control levels did not change over the 39-week study period, but the disc height and DHI measured using computer software demonstrated significant degenerative changes at the injured levels over the course of the study. Based on our histological findings, the reasons for the decrease in disc height and dark signal changes at 5–19 weeks was acute inflammation, disc effacement, and vertebral endplate loss, all of which influenced the nucleus pulposus. However, the findings at 32–39 weeks showed a different response, perhaps a reparative phase with connective tissue formation around the injury site.

Although H & E staining can clearly demonstrate a variety of different tissue structures, specialized techniques may be needed to demonstrate certain structural features in the present model. Severe inflammation, disc effacement, and significant vertebral endplate bone loss associated with connective tissue proliferation were observed in animals at 7 and 18 weeks, but these changes were not present in animals at 32 and 41 weeks. The lack of inflammation in the later animal groups can be partially explained by the elapsed time, allowing acute trauma responses and reparative processes to resolve. However, the severe disc damage associated with the effacing connective tissue formation in some of the earlier groups (most notably the 18-week group) is not a reversible process. These findings can explain disc degeneration seen on MR imaging and decrease of disc height. Although the manipulated discs of the animal groups that were killed later in the study all had abnormal disc nuclei and variable proliferation of either disc tissue or vascular connective tissue, the disc profiles were relatively intact, indicating that the severe disc-destroying inflammatory and reparative processes did not take place in these animals. This also suggests either a significant difference due to variation in surgical manipulation between animal groups (such as improved imaging, lesioning, or injection techniques with practice as the procedures were performed), or again, a normal variation in an individual animal’s response to disc manipulation, made prominent by the low numbers of animals at each histopathological time point (2 animals).

Conclusions

In the present study, we characterized a porcine model of reliable disc degeneration after an annular injury. This model may allow the study of degenerative processes with a better understanding of its pathogenesis and may also allow for the study of interventions to prevent or reverse the process.

Disclosure

Jeffrey C. Wang, M.D., is a consultant for Medtronic, Sofamor Danek.

References

Porcine IVD degeneration model


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Address correspondence to: Jeffrey C. Wang, M.D., Chief, Orthopaedic Spine Service, Associate Professor of Orthopaedics and Neurosurgery and Biomedical Engineering, University of California, Los Angeles Comprehensive Spine Center, 1250 16th Street, Suite 745, Santa Monica, California 90404. email: JWang@mednet.ucla.edu.