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Abbreviations:

ADC = apparent diffusion coefficient
DSA = digital subtraction
angiography
FOV = field of view
ROI = region of interest
TOF = time of flight
2D = two-dimensional

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Correlation of Diffusion in Lumbar Intervertebral Disks with Occlusion of Lumbar Arteries: A Study in Adult Volunteers¹

PURPOSE: To evaluate the correlation of the diffusion values in lumbar intervertebral disks with lumbar artery status and the degree of disk degeneration.

MATERIALS AND METHODS: Sagittal T2-weighted images of the lumbar spine were obtained in 37 asymptomatic volunteers aged 22–68 years. The apparent diffusion coefficient (ADC) of 98 lumbar intervertebral disks was determined, and two-dimensional time-of-flight magnetic resonance angiography was performed on the corresponding 98 lumbar artery pairs (total arteries = 196). The degree of disk degeneration and the status of lumbar arteries were evaluated independently by two radiologists. ADC calculations were performed on the basis of the average signal intensities of the selected region of interest in lumbar disks. The association between ADC values of disks, the disk degeneration, and the status of lumbar arteries of the same level were analyzed with analysis of covariance, and pairwise analysis between groups (Scheffé post hoc multiple comparison) was performed with statistical software. *P* values less than .01 were considered significant.

RESULTS: The lumbar arterial status correlated strongly with the diffusion values of intervertebral disks, and the ADC values decreased with higher degrees of arterial narrowing. The correlation between disk degeneration and diffusion was not significant. Eight severely degenerated disks with normal lumbar artery status and diffusion values were found.

CONCLUSION: Impaired flow in lumbar arteries is significantly associated with decreased diffusion in lumbar disks and may play an important role in disk degeneration.

The lumbar intervertebral disk is the largest avascular tissue in the adult human body. Thus, its nutrition occurs by means of diffusion from blood vessels in the surrounding structures (1,2). The upper three segments in the lumbar spine are supplied by the four pairs of lumbar arteries. The fourth segment is partly supplied by the fourth lumbar artery pairs and partly by branches from the middle sacral artery, which originates just above the bifurcation of the aorta. The fifth lumbar segment receives its blood supply through the middle sacral artery and through the branches of iliolumbar arteries. There is anatomical variation in the blood supply of the lower part of the lumbar spine (3–5). Diffusion occurs mainly through the vertebral end plates and partly from the vessels surrounding the periphery of the annulus. Insufficient nutrition has been suggested to be the primary cause for the degenerative process of a disk (6–8). The abdominal aorta, and particularly the aortoiliac junction, has been shown to be one of the most common sites of arteriosclerosis (9), and atherosclerotic changes in the lumbar area are associated with disk degeneration (10) and low-back pain (11,12).

Magnetic resonance (MR) imaging is the most sensitive method to depict intervertebral disk degeneration (13–15). Degeneration of the disk is detected as a decreased signal intensity of the nucleus pulposus on T2-weighted images (13,16). Quantitative MR imaging studies of lumbar intervertebral disks have been established as a noninvasive method

to detect alterations in water and biochemical composition by using measurement of T1 and T2 (17–21) and also by using magnetization transfer MR imaging (22). At first, quantitative MR imaging was not generally suggested as a reliable or feasible method for in vivo studies (23). However, in a recent study, Antoniou et al (24) detected that quantitative MR analysis reflects not only the disk matrix composition but also its structural integrity. So far, in clinical practice, diffusion-weighted MR imaging has been mostly used for the evaluation of acute cerebral infarcts and diseases affecting white matter (25,26). Researchers have reported the use of diffusion studies of the spine for evaluation of vertebral bone marrow (27), of the spinal cord (28), and, recently, of intervertebral disks (29).

Angiography has been the reference standard in the evaluation of the aorta and its branches. With technical advances, MR angiographic methods have considerably improved. In some indications, MR angiography is already a useful alternative to conventional angiography (30).

The purpose of our study was to evaluate the correlation of the diffusion values in lumbar intervertebral disks with lumbar artery status and the degree of disk degeneration.

MATERIALS AND METHODS

Subjects

Thirty-seven asymptomatic volunteers (15 men, 22 women; mean age, 38 years; age range, 22–68 years) were recruited for MR examination. None of the subjects had low-back pain at the time of the study, nor had they any relevant history of back pain. All components of the study were approved by the local ethics committee, and informed consent was provided by the volunteers and nine patients (five men, four women; mean age, 58 years; age range, 50–68 years) who were diagnosed with arterial disease.

MR Imaging

The volunteers underwent imaging during the afternoon hours (1–3 PM). Imaging was performed with a 1.5-T MR unit (Signa Echospeed; GE Medical Systems, Milwaukee, Wis). A phased-array spine coil (CTL; GE Medical Systems) was applied for both anatomic imaging of the lumbar spine and MR angiography of lumbar arteries. Sagittal T2-weighted fast spin-echo MR (repetition time msec/echo time msec, 6,000/105) images of the lumbar spine were obtained for anatomic im-

aging. The diffusion measurements were carried out by using a non-phased-array general-purpose receive-only flexible coil (GPFLEX; GE Medical Systems) with two electronically summed surface coils. The coil was wrapped around the lumbar area of the subject, to generate a relatively uniform signal throughout the region of interest (ROI). The imaging area with sufficient signal covered by the coil was about 20 cm in the section-select direction. Only one to four disks could be studied successfully at a time (Fig 1). The diffusion imaging was performed after the anatomic imaging and MR angiography, about 20 minutes after the beginning of the MR imaging study.

The diffusion-weighted images were obtained by using a spin-echo single-shot echo-planar imaging sequence (5,000/73 [effective]; 5-mm section thickness, 10-mm section gap, 40 × 20-cm field of view (FOV), 128 × 128 matrix, one signal acquired). This yielded spatial resolutions of about 3.1 and 1.6 mm in the readout and phase-encode directions, respectively. The imaging plane chosen in this series was transverse. Diffusion weighting was obtained by adding two diffusion-sensitizing gradients in both sides of the 180° refocusing pulse. The pulse duration was $\delta = 32$ msec, the pulse interval was $\Delta = 38.1$ msec, and the maximum gradient strength was $G_{\max} = 22$ mT/m. The gradient ramp time was 184 μ sec.

The time required to perform a diffusion study was about 10 minutes. The readout was in the vertical direction and the phase-encode in the horizontal direction. The effective echo time varied slightly from 70 to 75 msec and was set to the minimum allowable value. The diffusion-sensitizing gradients were applied sequentially in the x, y, and z directions (z was in the direction of the main magnetic field, x was in the horizontal direction, and y was in the vertical direction) by using diffusion-weighting factors (*b* values) of 250 and 500 mm²/sec. The *b* scale was also checked by using the apparent diffusion coefficient (ADC) value of 2.0×10^{-3} mm²/sec of water at a temperature of 23°C. Susceptibility artifacts were not detected on the images.

The L5-S1 disk was excluded from evaluations because the fifth lumbar segment receives blood supply through the middle sacral artery and through the branches of iliolumbar arteries, and the direction of these arteries could not be imaged in the two-dimensional (2D) time-of-flight (TOF) study at the same time with lumbar arteries. Also, the orientation of the presacral disk was markedly different from the

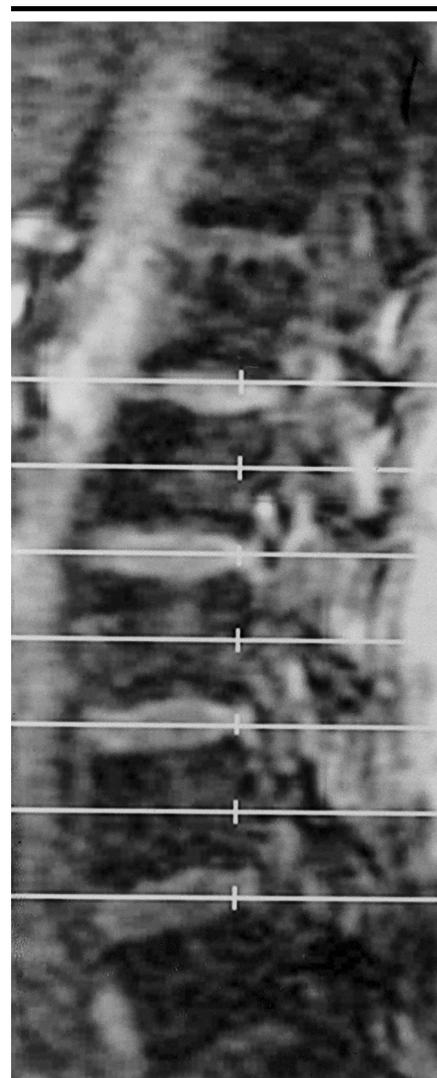


Figure 1. MR scout image (8.7/1.9; FOV, 36 × 36; section thickness, 3 mm; matrix, 256 × 128) shows diffusion weighting. Only L2-3 and L3-4 disks were parallel to the imaging plane and were acceptable.

orientation of the upper lumbar disks. Additionally, the measurements of ADC values were considered unreliable because of the orientation of the presacral disk.

Two-dimensional TOF MR angiography was used in the evaluation of the patency of lumbar arteries. MR images were obtained in the coronal direction to cover the area of the lumbar spine by using 29/5.1, flip angle of 60°, 256 × 512 matrix, a 30-cm FOV, and section thickness of 1.5 mm. A spatial presaturation pulse was placed posterior to the imaging sections to suppress the signal from the lumbar veins.

To test the validity of MR angiography (2D TOF) in evaluating the status of lumbar arteries, 72 lumbar arteries were eval-

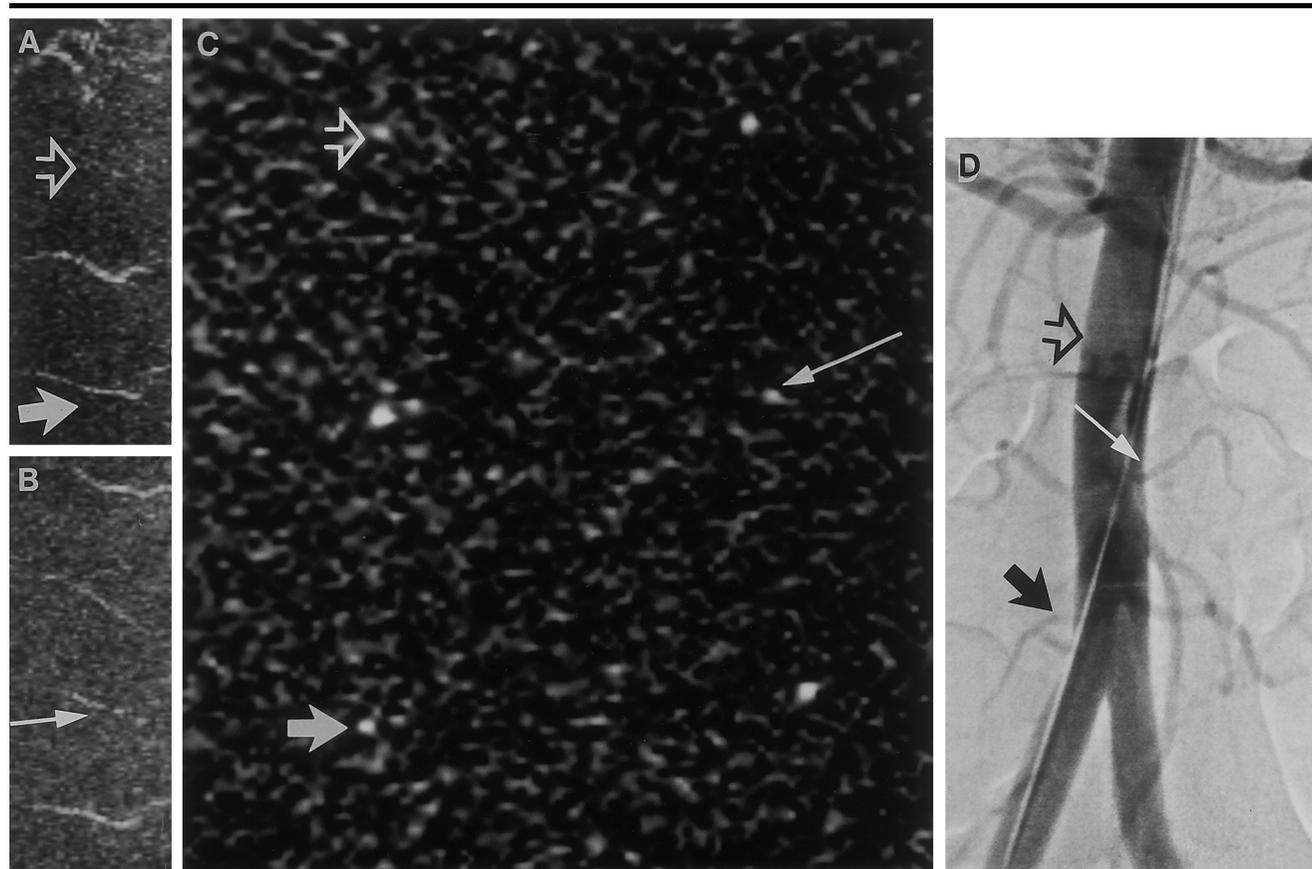


Figure 2. MR angiograms (2D TOF; 29/5; FOV, 24×18 ; section thickness, 1.5 mm; matrix, 256×512) and DSA image in 48-year-old volunteer. (A) Right and (B) left side maximum intensity projection MR angiograms. (C) Coronal MR angiogram. (D) DSA image. A–D show stenosis (narrowed lumbar arteries) in right L2 (open arrow in A, C, and D), right L4 (thick arrow in A, C, and D), and left L3 (thin arrow in B, C, and D) lumbar arteries.

uated separately with digital subtraction angiographic (DSA) aortography and MR angiography (2D TOF) in nine patients referred for angiography. The κ value between DSA and MR angiography in evaluating the status of lumbar arteries was 0.77. This indicates good agreement between MR angiography and DSA for the MR angiographic evaluation of the status of the lumbar arteries (Figs 2, 3). The disagreement between results of the two methods in analyzing lumbar arteries was found in only classes 0 and 1. In four cases, MR angiography showed slight stenosis (grade 1), while DSA showed normal findings (grade 0). In one case, the MR angiogram was graded normal (grade 0) when the DSA image showed grade 1 stenosis. These results indicate that MR angiographic findings may overestimate the DSA findings.

Image Analysis

The MR imaging findings in 98 intervertebral disks (L1-2 to L4-5) and the 2D

TOF MR angiographic findings in the corresponding 98 lumbar artery pairs (total of 196 arteries) were analyzed independently by two radiologists (M.K., L.K.) by using a workstation (Advantage Windows; GE Medical Systems). In the case of disagreement, a consensus was reached.

The intervertebral disks were classified according to three grades: grade 0, disks with high signal intensity or only slightly blurred intranuclear cleft, which represented normal disks; grade 1, disks with decreased signal intensity but normal height, which represented mild degeneration; and grade 2, disks with markedly decreased signal intensity and height loss, which represented severe degeneration. The signal intensities of intervertebral disks were compared with those of cerebrospinal fluid (Fig 4).

The lumbar arteries were evaluated as segmental pairs, which were classified according to the following grades: grade 0, normal vessels on both sides; grade 1, one segmental artery was narrowed; or

grade 2, both segmental arteries were narrowed or one or both arteries were occluded. The diameters of lumbar arteries were analyzed visually. The narrowing of the diameter of the vessels by 33% or more was recorded, and the contralateral vessel, if normal, or the next artery above was used as a reference standard. Both maximum intensity projection images (anteroposterior, oblique, and lateral views) and coronal source images were used to determine the stenosis of lumbar arteries (Figs 2, 3).

ADC values were determined by first calculating the average signal intensities from the selected ROIs; this was performed separately for image sets obtained with the b values 0, 250, and 500 mm^2/sec . A least squares fit was then applied to the resulting three-point attenuation curve. The ADC values, obtained from the slope of the fitted line, were determined in the three orthogonal directions.

The ROI was positioned by one author (M.K.) in areas of the disk where signal

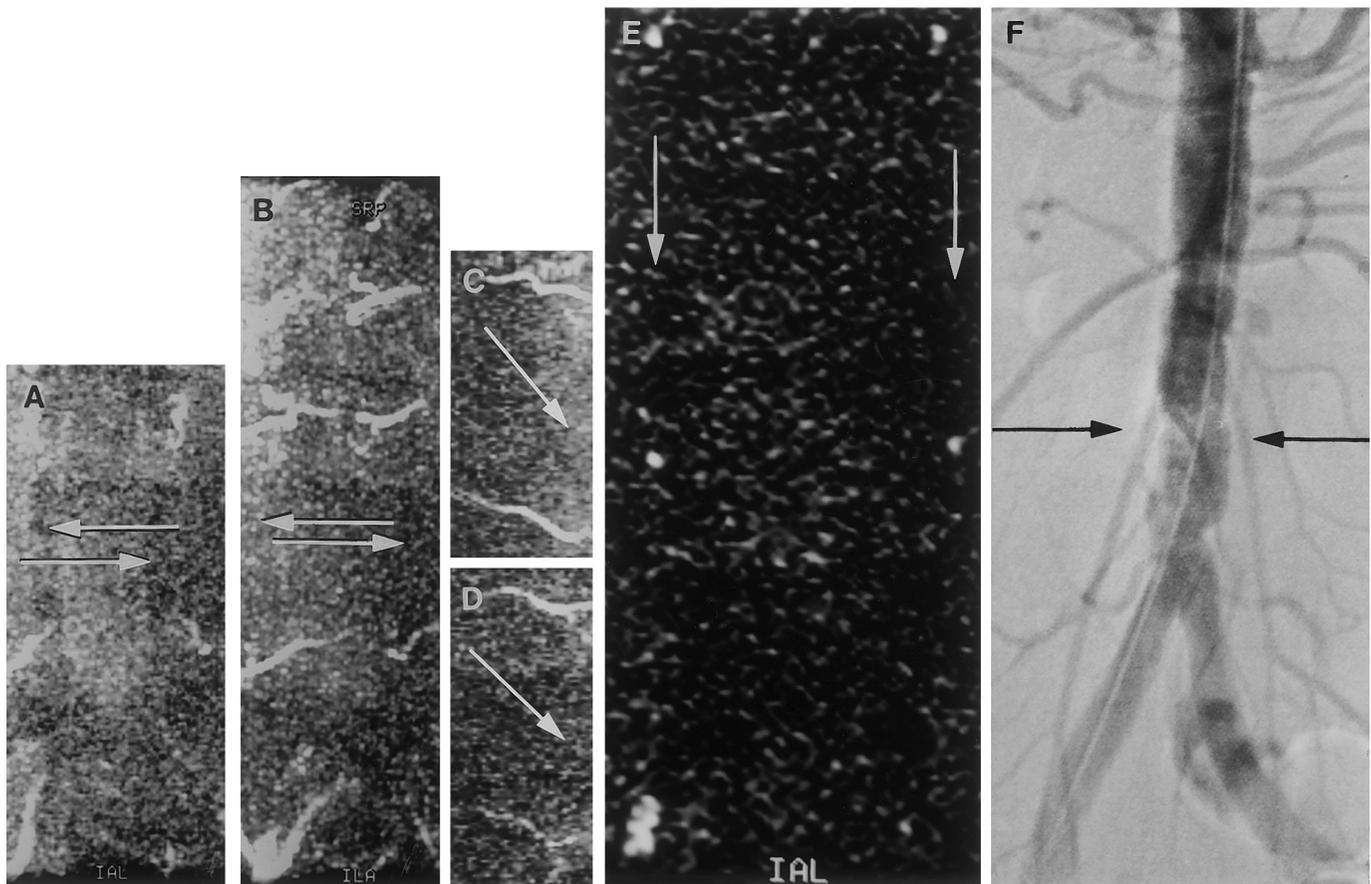


Figure 3. MR angiograms (2D TOF; 29/5; FOV, 24 × 18; section thickness, 1.5 mm; matrix, 256 × 512) and DSA image in 60-year-old volunteer. (A) Anteroposterior, (B) right oblique, and (C) right and (D) left side maximum intensity projection images show stenosis in left L4 lumbar artery. (E) Coronal MR angiogram and (F) DSA image show both L4 arteries are normal. A–F show stenosis (absent lumbar arteries) in both L3 arteries (arrows).

intensity was detected visually (Fig 5); the size of the ROI varied from 15 to 22 pixels, so the size of each ROI was approximately 1.50 cm². These areas of the disk mainly represented signal intensity of the nucleus. The signal intensity was highest on the images with no diffusion weighting; therefore, the ROI was placed on these images. These images were essentially ordinary T2-weighted images. The same pixels were then sampled on the diffusion-weighted images to calculate the ADC values. The ADC values were determined in the three orthogonal directions (ADC_x, ADC_y, and ADC_z).

Statistical Analysis

The association between ADC values of disks, the disk degeneration, and the status of lumbar arteries at the same level was analyzed by means of analysis of covariance, with age as covariate, and by using pairwise analysis (Scheffé post hoc multiple comparison test) between groups with statistical software (SPSS, version

9.0; SPSS, Chicago, Ill). *P* values less than .01 were considered significant.

RESULTS

Of the disks examined in this study, 59 were normal and 39 were degenerated. Twenty-five of the degenerated disks were graded as mildly degenerated (grade 1) and 14 were graded as severely degenerated (grade 2). The mean ADC values ($\times 10^{-3}$ mm²/sec) in normal disks in x, y, and z directions were 1.38, 1.52, 1.56; those in mildly degenerated disks were 1.05, 1.12, 1.21; and those in severely degenerated disks were 1.06, 1.10, 1.16, respectively. Forty-two lumbar artery pairs were classified as grade 0 (normal), 35 as grade 1, and 21 as grade 2. The mean ADC values in x, y, and z directions in disks with normal lumbar artery pairs were 1.49, 1.60, and 1.64; those in disks with grade 1 lumbar artery pairs were 1.35, 1.52, 1.57; and those in disks with

grade 2 lumbar artery pairs were 0.63, 0.66, 0.70, respectively.

There was a significant correlation between the status of lumbar arteries and the ADC values of the lumbar disks. The differences between ADC values of lumbar arteries were statistically significant in all orthogonal directions (x, y, and z) at levels L1-2, L2-3, L3-4; for ADC in the x direction at L4-5 (*P* < .001); and for ADC in the y and z directions at the L4-5 level (*P* < .01). The results are presented in Table 1. When age was controlled for the association between ADC and lumbar arteries, the differences between the ADC values of lumbar arteries remained significant.

Although the ADC values for all orthogonal directions decreased with increasing degree of disk degeneration, there was no statistically significant difference between the ADC values of different degrees of disk degeneration (Table 2). The number of severely degenerated disks was small at L1-2 (*n* = 2) and L3-4

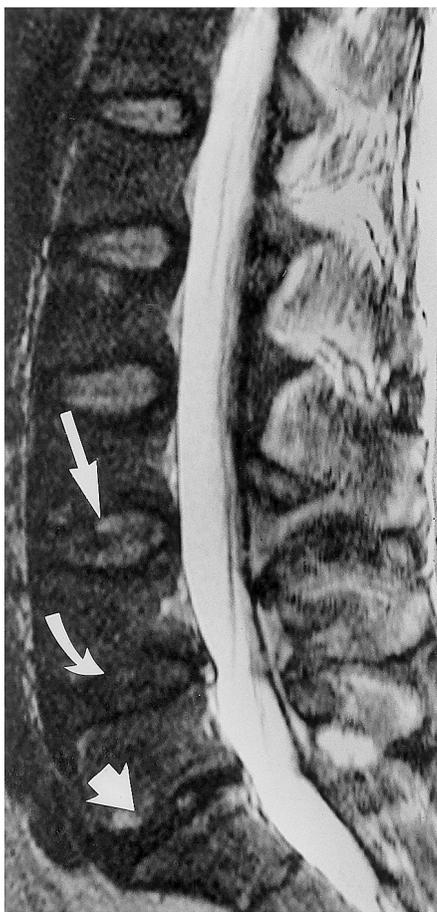


Figure 4. Grading of different levels of disk degeneration on T2-weighted MR image (6,000/105; FOV, 30 × 30; section thickness, 4 mm; matrix, 512 × 224). L1-2, L2-3, and L3-4 disks were graded 0; L3-4 disk with slightly blurred intranuclear cleft (thin arrow); L4-5 disk with decreased signal intensity was graded 1 (curved arrow); and L5-S1 disk with decreased signal intensity and height loss was graded 2 (thick arrow).

($n = 2$), and, therefore, statistical significance could be reliably assessed at only L2-3 and L4-5 levels.

Even though there was no correlation between disk degeneration and diffusion values, there was a trend with eight outliers: These eight severely degenerated disks had almost normal diffusion values for all directions (ADC_x , 1.36; ADC_y , 1.45; ADC_z , 1.54) and normal or grade 1 lumbar artery status (1.06, 1.10, 1.16, respectively) compared with all severely degenerated disks.

DISCUSSION

The results of this study showed that there was a strong correlation between diminished flow in lumbar arteries detected at



Figure 5. ROI measurements of ADC. The ROI is measured in a disk with high signal intensity on T2-weighted MR image (5,000/72.6; FOV, 40 × 20; section thickness, 5 mm; matrix, 128 × 128).

MR angiography and ADCs of lumbar intervertebral disks. Atherosclerotic changes in lumbar arteries have been shown to be associated with lumbar disk degeneration (10). Also, ADC values of intervertebral disks have been recently presented (29), but to our knowledge, our study is the first to show the clinical relevance of changes in ADC values. The strong correlation between lumbar artery occlusion and decrease in the ADC values may be due to diminished intradiskal osmotic pressure. However, the ADC values obtained with MR imaging may differ from the nutritional diffusion through the end plates. ADC values may also indicate the degradation and disintegration of the nucleus pulposus better and/or at an earlier time point than observational assessment of disk degeneration. Further studies are needed to better understand the molecular nature of these findings.

The diminished flow through stenosed or occluded lumbar arteries may create collateral vessels, which may also supply nutrients to the intervertebral disks (10). The image resolution of the 2D TOF MR angiographic method does not allow the detection of these possible collateral vessels. The role of collateral vessels in the intradiskal nutrition is, however, obscure (10). The ostia of the lumbar arteries were not well visualized in our TOF MR angiographic study, but any significant ostium stenosis can be detected as diminished flow on 2D TOF images. DSA has been the standard in the evaluation of blood vessels. Nevertheless, DSA is an invasive clinical test, and less invasive methods have replaced it in some indications. In our study, the correlation with normal DSA was good. There was moderate variation in findings in lumbar artery status between the disk levels. It was demon-

strated earlier that the correlation of atherosclerotic changes to disk degeneration was stronger in the upper levels of the lumbar spine than in the lower ones (10).

Surprisingly, eight intervertebral disks were severely degenerated, but the corresponding lumbar artery pairs and diffusion values were normal. This finding supports the previous understanding that pathogenesis of disk degeneration is multifactorial. In addition to nutritional determinants, environmental and genetic factors also have a major role in the initiation of disk degeneration (31,32).

The hydration of the nucleus pulposus is important in determining its mechanical response, its nutritional transport, and its biologic properties (33). However, hydration is a dynamic variable and not a stable property of tissue. A normal diurnal variation occurs in intradiskal water content, with influx of water overnight and gradual reduction during the day. This produces a measurable change in hydration, height, and volume of the disk (34,35). Change can also be measured in signal intensity, although it is not visible at MR imaging (35,36) or quantitative MR imaging (37). With physical loads, the water content diminishes up to 5%–20% in the nucleus pulposus and inner annulus (33,34). However, the diurnal variation is significantly less pronounced in degenerative than in healthy disks (37). Some researchers suggest that diurnal variation has a clinical importance in spinal mechanics (38). The relationship between change in water content and swelling pressure depends on the composition of the disk rather than on age or degree of degeneration (33). Our volunteers underwent imaging during the afternoon, so the conditions were similar in this respect for all measurements.

TABLE 1
Association between ADC Values and Status of Lumbar Arteries in L1-2 to L4-5

Lumbar Artery Grade at 2D TOF MR Imaging*	L1-2 (n = 19)	L2-3 (n = 28)	L3-4 (n = 27)	L4-5 (n = 24)
ADC _x				
0	1.51 (0.11/0.03)	1.50 (0.11/0.03)	1.54 (0.10/0.02)	1.49 (0.08/0.03)
1	1.27 (0.05/0.02)	1.29 (0.04/0.01)	1.39 (0.10/0.03)	1.44 (0.12/0.04)
2	0.44 (0.21/0.15)	0.55 (0.32/0.13)	0.63 (0.39/0.15)	0.69 (0.46/0.16)
ADC _y				
0	1.64 (0.26/0.09)	1.55 (0.09/0.02)	1.67 (0.15/0.04)	1.66 (0.10/0.03) [†]
1	1.54 (0.28/0.10)	1.45 (0.18/0.05)	1.49 (0.16/0.05)	1.59 (0.18/0.06) [†]
2	0.16 (0.06/0.04)	0.54 (0.29/0.12)	0.55 (0.25/0.10)	0.90 (0.59/0.21) [†]
ADC _z				
0	1.64 (0.28/0.09)	1.61 (0.14/0.04)	1.77 (0.19/0.05)	1.80 (0.08/0.03) [†]
1	1.54 (0.18/0.06)	1.51 (0.29/0.09)	1.68 (0.23/0.08)	1.65 (0.29/0.09) [†]
2	0.16 (0.03/0.02)	0.49 (0.19/0.08)	0.71 (0.40/0.16)	0.92 (0.52/0.18) [†]

Note.—Values in parentheses are the SD/standard error. For all values except where otherwise indicated, the correlation between mean ADC values ($\times 10^{-3}$ mm²/sec) of the disks and the status of lumbar arteries (at 2D TOF MR imaging) was statistically significant (one-way analysis of covariance, $P < .001$).

* Grade 0 = normal vessels on both sides, grade 1 = one segmental artery was narrowed, grade 2 = both segmental arteries were narrowed or one or both arteries were occluded.

[†] $P < .01$.

TABLE 2
Association between ADC Values and Disk Degeneration in L1-2 to L4-5

Disc Degeneration Grade*	L1-2 (n = 19)	L2-3 (n = 28)	L3-4 (n = 27)	L4-5 (n = 24)
ADC _x				
0	1.35 (0.25/0.06)	1.35 (0.31/0.07) [†]	1.41 (0.35/0.09)	1.45 (0.13/0.04) [‡]
1	0.93 (0.90/0.60)	1.02 (0.60/0.27) [†]	1.22 (0.40/0.12)	0.90 (0.56/0.19) [‡]
2	1.30 (0.07/0.05)	0.98 (0.36/0.16) [†]	0.63 (0.73/0.52)	1.14 (0.52/0.23) [‡]
ADC _y				
0	1.53 (0.47/0.12)	1.45 (0.32/0.07) [§]	1.53 (0.37/0.09)	1.63 (0.16/0.04)
1	0.81 (0.86/0.81)	1.20 (0.47/0.21) [§]	1.21 (0.51/0.16)	1.15 (0.67/0.24)
2	1.40 (0.30/0.21)	0.87 (0.55/0.24) [§]	1.01 (0.93/0.66)	1.23 (0.50/0.23)
ADC _z				
0	1.36 (0.40/0.10)	1.49 (0.38/0.09) [#]	1.70 (0.46/0.11)	1.75 (0.19/0.05)**
1	1.02 (1.18/0.83)	1.19 (0.56/0.25) [#]	1.31 (0.41/0.13)	1.13 (0.64/0.22)**
2	1.57 (0.24/0.17)	0.92 (0.61/0.27) [#]	1.07 (0.90/0.64)	1.27 (0.46/0.21)**

Note.—Values in parentheses are the SD/standard error.

* Grade 0 = disks with high signal intensity or only slightly blurred intranuclear cleft, which represented normal disks; grade 1 = disks with decreased signal intensity but normal height, which represented mild degeneration; grade 2 = disks with markedly decreased signal intensity and height loss, which represented severe degeneration.

[†] The correlation between mean ADC values ($\times 10^{-3}$ mm²/sec) of the disks and disk degeneration (one-way analysis of covariance) was demonstrated by a P value of .085.

[‡] $P = .028$.

[§] $P = .024$.

^{||} $P = .072$.

[#] $P = .051$.

** $P = .016$.

The well-being of a disk depends on its cells (7). The cell density in the disk is controlled by nutritional factors (39). During the degenerative process, considerable alterations occur in the structure and biochemistry of the intervertebral disk. The effect of altered hydration on solute transport is complex (7), because a decrease in hydration means a lower ADC value, but, on the other hand, the height of the disk has decreased. Thus, the distances through which metabolites have to move is shorter (7). This may explain our findings that the ADC values were

not linearly decreased during the degeneration process. Findings suggest that decreased water content as such is not likely to markedly affect the balance between cellular requirements and metabolite transport, although the effect on the mechanical properties of the disk is obvious (7).

Technical advancements have been considerable in both diffusion-weighted MR imaging and MR angiography. In diffusion-weighted imaging, very fast acquisition methods are used. As a fast imaging technique, the echo-planar MR imaging sequence, while minimizing motion arti-

facts, is disposed to susceptibility artifacts. However, susceptibility artifacts are almost the same in cortical bone, water, and soft tissues (40,41). Thus, although various tissue-bone interfaces are located close to the intervertebral disks, we do not suspect that they affect the ADC values; susceptibility artifacts were not detected on the images. Cerebrospinal fluid flow does not cause artifacts in the lumbar spine.

We measured ADC values in the transverse plane. We are aware that with sagittal imaging the possibility of a partial-

volume effect is smaller, but imaging in sagittal sections limits the ROI size more than transverse imaging does; therefore, transverse imaging planes were chosen for this study. To accurately quantify diffusion anisotropy, diffusion-tensor imaging, which would allow calculation of the diffusion ellipsoid, should be carried out. However, diffusion-tensor imaging was previously observed to be unreliable because of noise, motion, and magnetic field inhomogeneities (29). In our study, only the diffusion in three orthogonal directions was measured, with the z gradient being the direction of both the magnetic field and the vertical axis of the patient. Perfusion contributes to the ADC values (42–44), but this is expected to be negligible in disks because of their avascularity.

In previous diffusion imaging studies in the spine, steady-state (27), spin-echo (28), and echo-planar imaging sequences (29) have been used. We admit that various technical problems are associated with diffusion MR imaging of the spine, but we consider that it is worthwhile to study the future potential of this method; more sensitive methods for detecting early degenerative changes in intervertebral disks are needed. Even with its present limitations, diffusion MR imaging in the spine may become feasible in the near future when MR imagers with stronger gradients become available. The ADC values of normal disks in this study were in good agreement with the values presented in an earlier study of intervertebral disks (29).

In conclusion, findings in this study indicate that impaired flow in lumbar arteries is markedly associated with decreased diffusion in lumbar disks and may play an important role as a promoter of disk degeneration. Thus diffusion values may have a meaningful functional basis. However, the initiation of the disk degeneration process is not always related purely to the impairment of diffusion.

References

1. Urban JPG, Holm S, Maroudas A. Diffusion of small solutes into the intervertebral disc: as in vivo study. *Biorheology* 1978; 15:203–223.
2. Holm S, Maroudas A, Urban JP, Selstam G, Nachemson A. Nutrition of the intervertebral disc: solute transport and metabolism. *Connect Tissue Res* 1981; 8:101–119.
3. Crock HV, Yoshizawa H. The blood supply of the lumbar vertebral column. *Clin Orthop Rel Res* 1976; 115:6–21.
4. Ratcliffe JF. The anatomy of the fourth and fifth lumbar arteries in humans: an arteriographic study in one hundred live subjects. *J Anat* 1982; 135:735–761.

5. Louis R. *Surgery of the spine*. New York, NY: Springer-Verlag, 1983; 284–285.
6. Nachemson A, Lewin T, Maroudas A, Freeman MAR. In vitro diffusion of dye through the end plates and annulus fibrosus of human lumbar intervertebral discs. *Acta Orthop Scand* 1970; 41:589–607.
7. Maroudas A. Nutrition and metabolism of the intervertebral disc. In: Ghosh P, ed. *The biology of the intervertebral disc*. Vol 2. Boca Raton, Fla: CRC, 1988; 1–37.
8. Buckwalter JA. Aging and degeneration of the human intervertebral disc. *Spine* 1995; 20:1307–1314.
9. Hallisey MJ, Meranze SG. The abnormal abdominal aorta: arteriosclerosis and other diseases. In: Baum S, ed. *Abram's angiography: vascular and interventional radiology*. 4th ed. Boston: Little, Brown, 1997; 1052–1065.
10. Kauppila L, Penttilä A, Karhunen PJ, Lulu K, Hannikainen P. Lumbar disc degeneration and atherosclerosis of the abdominal aorta. *Spine* 1994; 8:923–929.
11. Kauppila L, Tallroth K. Postmortem angiographic findings for arteries supplying the lumbar spine: their relationship to low-back symptoms. *J Spin Dis* 1993; 6:124–129.
12. Kurunlahti M, Tervonen O, Vanharanta H, Ilkko E, Suramo I. Association of atherosclerosis with low back pain and disc degeneration. *Spine* 1999; 24:2080–2084.
13. Modic MT, Pavlicek W, Weinstein MA, et al. Magnetic resonance imaging of intervertebral disc disease: clinical and pulse sequence considerations. *Radiology* 1984; 152:103–111.
14. Gibson MJ, Buckley J, Mawhinney R, Mulholland RC, Worthington BS. Magnetic resonance imaging and discography in the diagnosis of disc degeneration: a comparative study of 50 discs. *J Bone Joint Surg Br* 1986; 68:369–373.
15. Schneiderman G, Flannigan B, Kingston S, Thomas J, Dillin WH, Watkins RG. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. *Spine* 1987; 12:276–281.
16. Tertti M, Paajanen H, Laato M, Aho H, Komu M, Kormanen M. Disc degeneration in magnetic resonance imaging: a comparative biochemical, histologic and radiologic study in cadaver spine. *Spine* 1991; 16:629–634.
17. Jenkins JPR, Hickey DS, Zhu XP, Machin M, Isherwood I. MR imaging of the intervertebral disc: a quantitative study. *Br J Radiol* 1985; 58:705–709.
18. Hickey D, Aspden M, Hukins D, Jenkins J, Isherwood I. Analysis of magnetic resonance images from normal and degenerate lumbar intervertebral discs. *Spine* 1986; 11:702–708.
19. Bobest M, Furo I, Tompa K, Pocsik I, Kuncz A. 1H nuclear magnetic resonance study of intervertebral discs: a preliminary report. *Spine* 1986; 11:709–711.
20. Chatani K, Kusaka Y, Mifune T, Nishikawa H. Topographic differences of 1H-NMR relaxation times (T1, T2) in the normal intervertebral disc and its relationship to water content. *Spine* 1993; 18:2271–2275.
21. Boos N, Wallin A, Schmucker TH, Aebi M, Boesch CH. Quantitative MR imaging of lumbar intervertebral discs and vertebral bodies: methodology, reproducibility and preliminary results. *Magn Reson Imaging* 1994; 12:577–587.
22. Paajanen H, Komu M, Lehto I, Laato M, Haapasalo H. Magnetization transfer imaging of lumbar disc degeneration: correlation of relaxation parameters with biochemistry. *Spine* 1994; 19:2833–2837.
23. Boos N, Boesch C. Quantitative magnetic resonance imaging of the lumbar spine: potential for investigations of water content and biochemical composition. *Spine* 1995; 20:2358–2366.
24. Antoniou JG, Pike GB, Steffen T, et al. Quantitative magnetic resonance imaging in the assessment of degenerative disc disease. *Magn Reson Med* 1998; 40:900–907.
25. Doran M, Hajnal JV, Van Bruggen N, King MD, Young IR, Bydder GM. Normal and abnormal white matter tracts shown by MR imaging using directional diffusion weighted sequences. *J Comput Assist Tomogr* 1990; 14:865–873.
26. Larsson HBW, Thomsen C, Frederiksen J, Stubgaard M, Henriksen O. In vivo magnetic resonance diffusion measurement in the brain of patients with multiple sclerosis. *Magn Reson Imaging* 1992; 10:7–12.
27. Baur A, Stäbler A, Bruning R, et al. Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic compression fractures. *Radiology* 1998; 207:349–356.
28. Clark CA, Werring DJ, Miller DH. Diffusion imaging of the spinal cord in vivo: estimation of the principal diffusivities and application to multiple sclerosis. *Magn Reson Med* 2000; 43:133–138.
29. Kerttula L, Jauhiainen JPT, Tervonen O, Suramo IJ, Koivula A, Oikarinen JT. Apparent diffusion coefficient in thoracolumbar intervertebral discs of healthy young volunteers. *J Magn Reson Imaging* 2000; 12:255–260.
30. Schoenberg SO, Prince MR, Knopp MV, Allenberg JR. Renal MR angiography. *Magn Reson Imaging Clin N Am* 1998; 6:351–370.
31. Battie MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K. Determinants of lumbar disc degeneration: a study relating lifetime exposures and magnetic resonance imaging findings in identical twins. *Spine* 1995; 20:2601–2612.
32. Annunen S, Paasilta P, Lohiniva J, et al. An allele of COL9A2 associated with intervertebral disc disease. *Science* 1999; 16:409–412.
33. Urban JPG, McMullin JF. Swelling pressure of the lumbar intervertebral discs: influence of age, spinal level, composition, and degeneration. *Spine* 1988; 13:179–187.
34. Adams M, Hutton WC. The effect of posture on fluid content of lumbar intervertebral disc. *Spine* 1983; 8:665–671.
35. Paajanen H, Lehto I, Alanen A, Erkintalo M, Komu M. Diurnal fluid changes of lumbar discs measured indirectly by magnetic resonance imaging. *J Orthop Res* 1994; 12:509–514.
36. Silcox DH, Horton WC, Wilwerstein AM. MRI of lumbar intervertebral discs: diurnal variations in signal intensities. *Spine* 1995; 20:807–811.
37. Boos N, Wallin A, Gbedegbegnon T, Aebi M, Boesch C. Quantitative MR imaging of

- lumbar intervertebral disks and vertebral bodies: influence of diurnal water content variations. *Radiology* 1993; 188:351-354.
38. Adams MA, Dolan P, Hutton WC, Porter RW. Diurnal changes in spinal mechanics and their clinical significance. *J Bone Joint Surg Br* 1990; 72:266-270.
 39. Stairmand JW, Holm S, Urban JP. Factors influencing oxygen concentration gradients in the intervertebral disc: a theoretical analysis. *Spine* 1991; 16:444-449.
 40. Sumanaweera TS, Glover GH, Binford TO, Adler JR. MR susceptibility misregistration correction. *IEEE Trans Med Imaging* 1993; 12:251-259.
 41. Schenk JF. The role of magnetic susceptibility in magnetic resonance imaging: MRI magnetic compatibility of the first and second kinds. *Med Phys* 1996; 23: 815-845.
 42. Turner R, Le Bihan D, Maier J, Vavrek R, Hedges LK, Pekar J. Echo-planar imaging of intravoxel incoherent motion. *Radiology* 1990; 177:407-414.
 43. Le Bihan D, Breton E, Lallemand D, Aubin ML, Vignaud J, Laval-Jeantet M. Separation of diffusion and perfusion in intravoxel incoherent motion MR imaging. *Radiology* 1988; 168:497-505.
 44. Yamada I, Aung W, Himeno Y, Nakagawa T, Shibuya H. Diffusion coefficients in abdominal organs and hepatic lesions: evaluation with intravoxel incoherent motion echo-planar MR imaging. *Radiology* 1999; 210:617-623.