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Gadolinium-Enhanced Nerve Roots in Lumbar Disk Herniation

Girolamo Crisi,[1,2] Paolo Carpeggiani,[1] and Claudio Trevisan[1]

PURPOSE: To analyze contrast-enhanced features of intrathecal nerve roots in lumbar disk herniation in patients who have not had surgery and to evaluate the possible correlation with herniated disk, peridiscal enhancement, and symptomatology. **METHODS:** Twenty patients with a syndrome of lumbar herniated disk without surgery were studied with pre- and postcontrast MR imaging. **RESULTS:** In six patients (30%), ipsilateral intradural roots affected with large disk herniations showed consistent enhancement along their whole length, from the site of compression to the root-medullary junction. This enhancement persisted on delayed scanning. "Selective" enhancement differed from the minimal enhancement seen in normal intrathecal roots in 11 (55%) patients. Enhancement around herniated disks was a common finding (92% of patients) and did not correlate with selective or normal root enhancement ($P > .05$). No symptom differences were found among patients with and without selective enhancement. **CONCLUSION:** Selective intrathecal root enhancement seems to be the direct visualization of the radiculopathy resulting from disk herniation. It is probably a transient event more likely to be noted in the acute phase. Although there is no evidence of immediate clinical utility of this finding, gadolinium-enhanced MR gives insights into the basic pathophysiologic alterations involved in degenerative disk disease.

Index terms: Spine, intervertebral disks, herniation; Spine, magnetic resonance; Nerves, spinal; Subarachnoid space; Contrast media, intrathecal

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In an attempt to investigate, by means of contrast-enhanced magnetic resonance (MR), the interplay among disk disease, epidural tissue response, and involved nerve root, we incidentally observed some cases of unusual intradural root enhancement associated with large disk herniation on contrast-enhanced MR. Therefore, this study was undertaken to analyze the contrast-enhanced MR features of intrathecal nerve roots in lumbar disk herniation in patients who did not have surgery. Possible correlations with herniated disk, peridiscal enhancement, and symptomatology were looked for. A possible explanation for this occasional root enhancement in large disk herniations is proposed.

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Subjects and Methods

Twenty patients (Table 1), 11 men and nine women (24 to 67 years old; average age, 44.5 years), with symptoms of herniated disk without surgery were studied from June 1990 to March 1992. Examinations were performed on a 1.5-T superconductive magnet. Sagittal spin-echo images with section thickness of 3 mm, both short repetition time (TR)/echo time (TE) (600/20/2 [TR/TE/excitations]) and long TR/TE (2300/30–80/1), were acquired with a 256 × 256 or 256 × 192 matrix and a 26-cm field of view. Axial spin-echo images with 4-mm section thickness and short TR/TE (800/20/2), were obtained with a 256 × 192 matrix and 16-cm field of view. A total of 72 disk levels were examined by axial scans. Enhanced MR study, started immediately after administration of 0.1 ml/kg of gadopentetate dimeglumine, consisted of short TR/TE axial and sagittal spin-echo images, with the same parameters as in the unenhanced scans. In six patients, short TR/TE (600/25) coronal spin-echo images with 3-mm section thickness, 256 × 192 matrix, and 26-cm field of view, complemented postcontrast examination. In seven patients, delayed (40 to 60 min after the contrast injection) sagittal, axial, and coronal short TR/TE images were also obtained. Patient tolerance to the contrast injection was excellent.

For each patient, we have taken into consideration the following: level, size, and site of disk herniation; clinical

signs; and time interval between the onset of symptoms and MR study. Enhancement of nerve roots and peridiscal enhancement on postcontrast images were compared with precontrast scans at the same level. The size of disk herniation was classified as small, moderate, or large. The contrast uptake of nerve roots was defined as minimal, moderate, or intense. Peridiscal enhancement was evaluated as thin or thick. Eleven patients (12 disk herniations) were operated on and nine were treated conservatively. Three selected patients, two operated on and one treated conservatively, were re-examined after about 1 year (Table 1).

The resulting data were statistically analyzed using chi-square test with Yates correction for continuity and Student *t* test.

Results

All resulting data are summarized on Table 1.

Disk Herniation

Twenty-seven disk herniations were found in the 20 patients: five at L3-L4, eight at L4-L5, and 14 at L5-S1. Five patients had two disk herniations at different levels and one patient had three levels of disk herniation. Seven disk herniations were of small size, six were moderate, and 14 large.

Enhancement of Nerve Roots

Two types of nerve root enhancement were observed.

A minimal enhancement of intrathecal nerve roots was observed on immediate postcontrast scans in 11 patients (55%). It was shared by several roots at different points in the same cross-sectional area of the dural sac. This pattern of

TABLE 1: Summary of patients: root enhancement and associated MR and clinical findings

Patients				Disk Herniation				Root Enhancement			Peridiscal Enhancement		Symptoms	
No.	Name	Sex	Age	Level	Size	Site		Type	Intensity	Delayed	Thickness	Delayed	Side	Days
1	MC	F	33	L4-L5	+	PL	L	Normal	+	−	++	↑	L	90
				L5-S1[a]	+++	PL	R				++	↑		
2	MZ	F	63	L3-L4[a]	++	PL	R				++		R	90
3	DA	F	40	L5-S1[a]	++	PL	L	Normal	+		++		L	30
4	SN	F	67	L3-L4[a]	+++	PL	R	Normal	+	−	++	↑	R	120
								Selective	++	↑				
5	BP	M	34	L4-L5	+	PL	L	Normal	+	−	+	↑	R	180
				L5-S1	+	PM					+	=		
6	SL	F	50	L5-S1	+++	PL	L	Normal	+	−	+	↓	L	15
								Selective	++	=				
7	BA	M	38	L5-S1	+++	PL	R				++		R	30
8[b]	VL	M	45	L5-S1	+++	PL	L	Selective	+++		++		L	15
9	CE	F	40	L5-S1	+++	PL	R	Normal	+		++		R	60
10	FM	M	43	L5-S1	++	PL	L				++		L	90
11	RL	M	53	L3-L4	++	PL	R				++		R	60
				L4-L5	+++	PL	R				++			
12	ZL	F	37	L4-L5[a]	+++	PL	R				−		R	180
13	LP	M	44	L5-S1[a]	+++	PL	R	Normal	+		+		R	60
14	PG	M	48	L4-L5	++	PL	R	Normal	+		+		R	180
15	GV	F	46	L4-L5	+	PM					+			
				L5-S1	++	PL	R	Normal	+		++		L	30
16	CF	M	42	L5-S1[a]	+++	PL	L	Normal	+		+		L	30
17	RA	M	24	L3-L4	+	PM					+			
				L4-L5[a]	++	PM					+		B	180
				L5-S1[a]	+++	PM					+			
18	TR	M	59	L5-S1[a]	+++	PL	L	Selective	+++	=	++	=	L	60
								Normal	+	−				
19[b]	VR	M	38	L4-L5	+	PM					−			
				L5-S1[a]	+++	PL	L	Selective	+++	=	++	↓	L	15
20[b]	ZW	M	45	L3-L4[a]	+++	PL[c]	R	Selective						
						&	L	Bilateral	+++	=	++	↑	B	60

Note. F, Female; M, male. Size: +, small; ++, moderate; +++, large. Site: PL, posterolateral; PM, posteromedial. Intensity of root enhancement; +, minimal; ++, moderate; +++, intense. Delayed enhancement: ↑, increase; ↓, decrease; =, unchanged; −, not identified. Thickness of peridiscal enhancement: −, not identified; +, thin; ++, thick. R, Right; L, left; B, bilateral; *a*, patients operated on; *b*, patients re-examined after 1 year; *c*, hourglass shaped.

root enhancement appeared to be unrelated to the size or site of disk herniation (Fig. 1). In fact, it was found both in one patient with a small hernia and in patients with moderate (three cases) and large (five cases) disk herniations, and it was shared equally by roots on the same side and on the opposite side of disk herniation. This type of enhancement, which may be termed "normal" (see Discussion), was better appreciated on axial scan, when they were carefully compared with precontrast scans. In five patients, this enhancement disappeared on delayed scanning (40 to 60 min after the contrast injection).

Conversely, six (30%) patients showed a second pattern of enhancement that we called "selective," because it exclusively concerned intradural nerve roots associated with disk herniations at the corresponding level. Selective intradural root enhancement was moderate in two cases and intense in four cases. Typically, intrathecal segments of nerve roots ipsilateral to a corresponding disk herniation were seen to enhance selectively, from the site of compression by the disk herniation to the conus medullaris (Fig. 2). The enhancement stopped sharply at the root-medullary junction (Fig. 3). Sagittal and coronal scans were more useful to demonstrate this "long-distance" pattern of enhancement. Five of these patients received delayed postcontrast scans (40 to 60 min after the contrast injection), showing the degree of the enhancement to be unchanged. In case 20, the disk herniation was hour-glass shaped, involving both sides of the spinal canal, and selective enhancement was shared by nerve roots bilaterally (Fig. 3). Co-existence of both types of enhancement, normal and selective, was

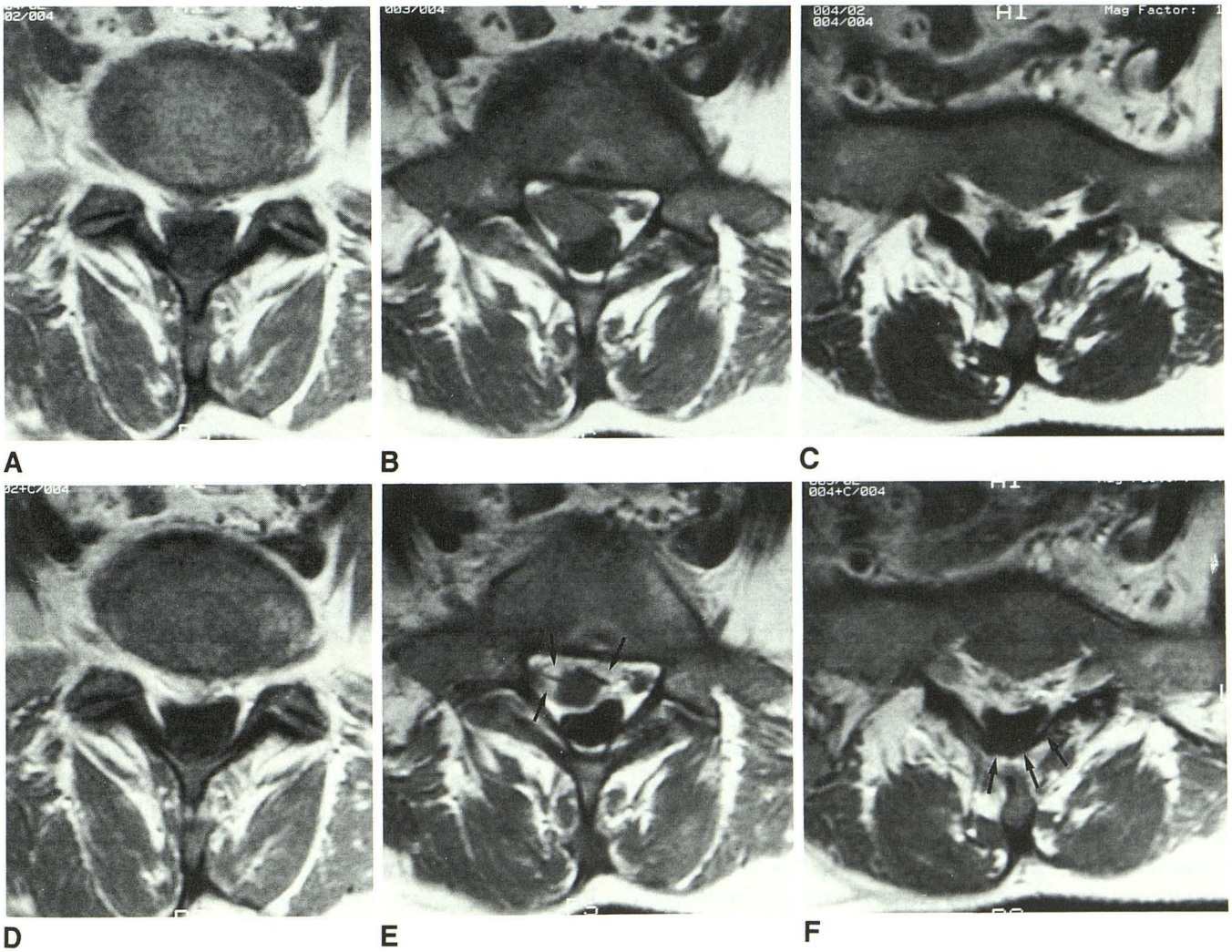


Fig. 1. *A–F*, Large L5-S1 disk herniation (case 9).

A–C, Axial precontrast T1-weighted scans above (*A*), below (*C*), and at disk herniation (*B*).

D–F, Axial postcontrast T1-weighted scans at the same level as *A–C*. Note thick peridiscal enhancement (*arrows*). A minimal normal enhancement is observed on comparing section C with identical postcontrast section F (*arrows*).

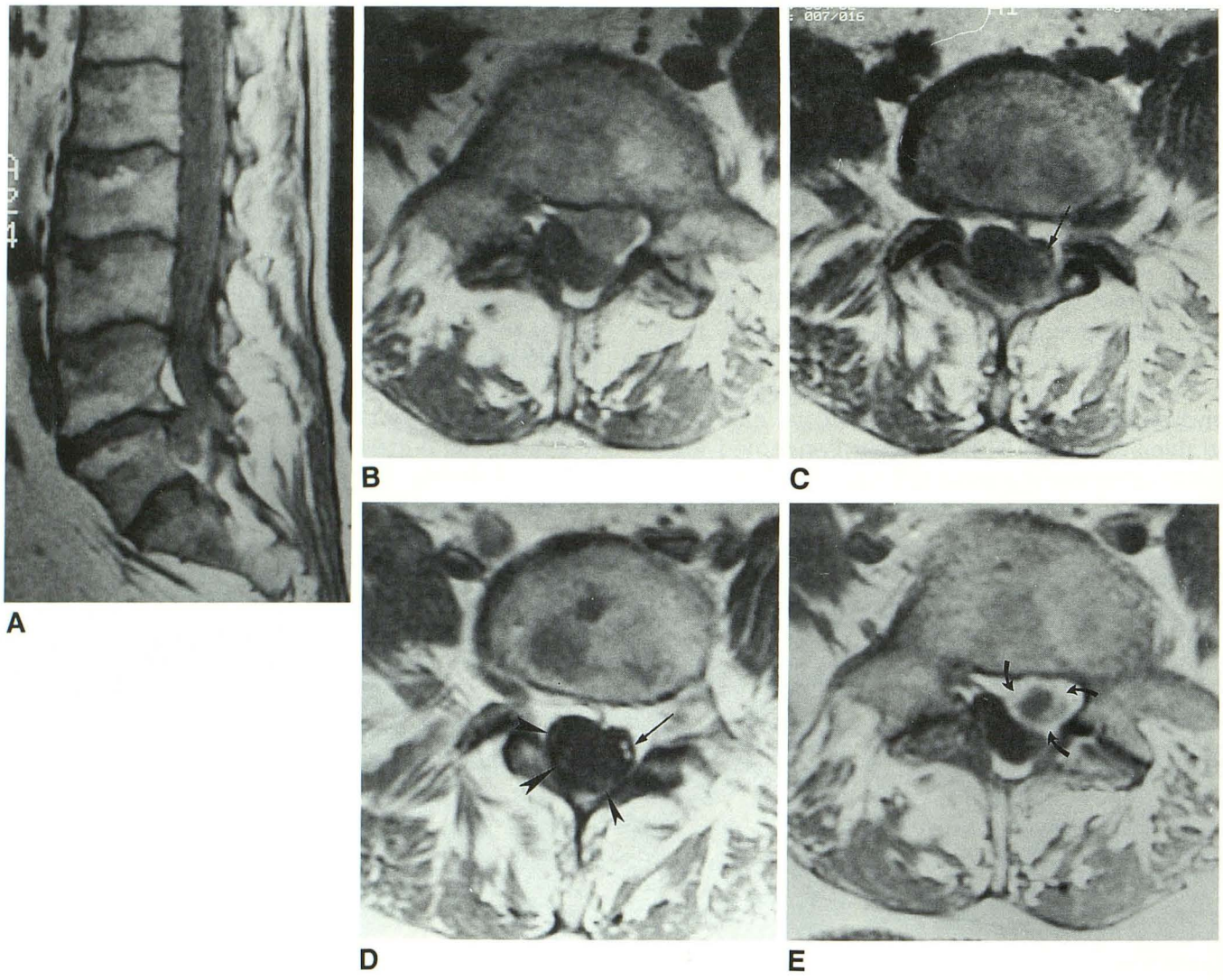


Fig. 2. *A–J*, Large L5-S1 disk herniation (case 18).

A–B, Precontrast sagittal and axial T1-weighted images show large left-extruded disk material migrated caudally along the posterior aspect of the body of S1, which is lumbarized.

C, Precontrast axial T1-weighted image above the level of disk herniation displays swelling of two ipsilateral roots (*arrows*).

D–E, Postcontrast axial T1-weighted scans at the same level as C (*D*) and B (*E*). Intense, selective enhancement of ipsilateral nerve roots (*arrows, D*). A minimal normal enhancement of remaining nerve roots is also observed (*arrowheads, D*). A thick rim of enhancement is seen around disk herniation (*curved arrows, E*).

seen in three patients (cases 4, 6, and 18). Additionally, selective enhancing nerve roots appeared to be swollen compared with unenhancing or minimally enhancing contralateral roots. Selective root enhancement was always associated with large disk herniations, as opposed to normal enhancement, which was seen in patients with small, moderate, and large hernias; however, these findings do not reach statistical significance ($P > .05$).

Three patients with the selective enhancement pattern were re-examined with MR after 1 year (cases 8, 19, and 20). Two (cases 19 and 20) patients had been operated on and the other one had been treated conservatively. On MR, case 19 showed usual postoperative changes with minimal epidural scar. The previously compressed left S1 root was thicker than the contralateral one, but root enhancement had disappeared. The patient was feeling well except for some paresthesia in the left S1 territory. Also, case 20 showed postoperative changes with no excessive epidural scarring. The hernia had been totally removed and root enhancement had disappeared. Conversely, a minimal normal enhancement of nerve roots was present (Fig. 3). The patient was feeling

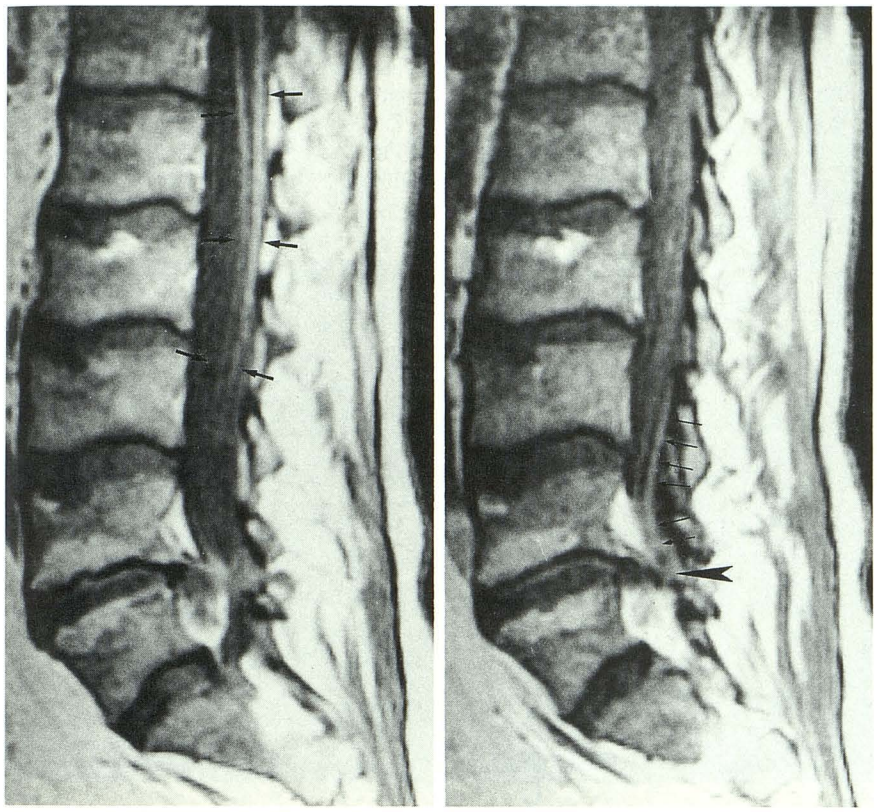


Fig. 2. *F–I*, Postcontrast sagittal (*F–G*) and coronal (*H–I*) T1-weighted images. The selective enhancement of intradural roots, affected with corresponding L5-S1 disk herniation (*arrowhead*), is seen extending from the site of compression (*arrows, G*) along their whole length (*arrows, F*). Coronal T1-weighted images confirm the long-distance selective enhancement (*black arrows, H–I*), up to the appearing conus tip (*white arrows, I*).

J, Delayed (60 min after contrast injection) axial scan corresponding to D shows persistence of selective enhancement (*arrow*). Normal enhancement is no longer evident.

well. The third patient (case 8) had become symptom-free and, on follow-up MR examination, both disk herniation and selective root enhancement were surprisingly no longer present (Fig. 4).

Peridiscal Enhancement

Thick peridiscal enhancement on immediate postcontrast scan was seen in 15 disk herniations, of which one (6.6%) was small, five (33.3%) were moderate, and nine (60.1%) were large. A thin peridiscal enhancement was seen in 10 disk herniations, of which five (50%) were small, one (10%) was moderate, and four (40%) were large. Two hernias, one small and the other large (cases 12 and 19), showed no peripheral enhancement. There is a tendency for large hernias to have a thicker peripheral enhancement, but this does not reach statistical significance ($P > .05$).

In seven patients (nine disk herniations), delayed (40 to 60 min after the contrast injection) postcontrast scan showed an increase in the enhancement in five disk herniations, a reduction in two, and no change in two.

A thick peridiscal enhancement was noted in six patients (54.5%) with the normal pattern of

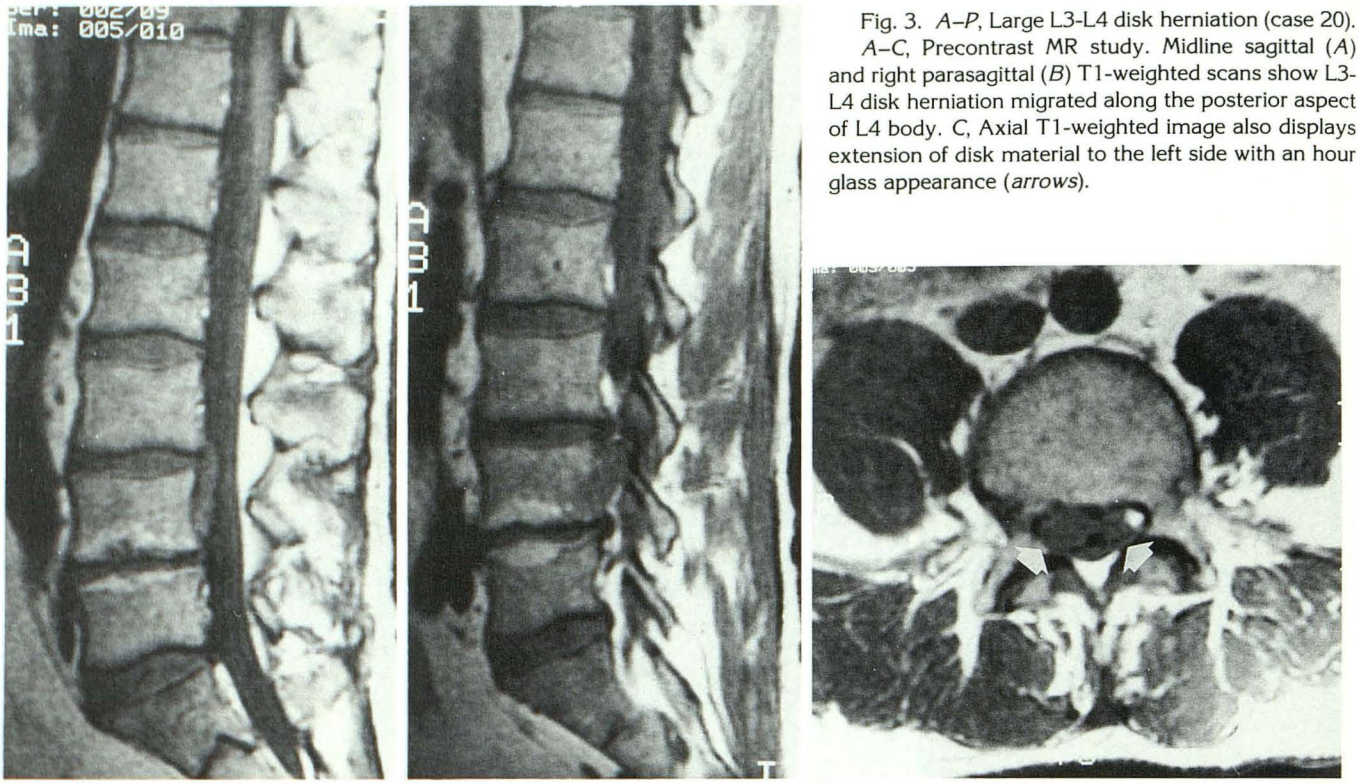


Fig. 3. *A–P*, Large L3-L4 disk herniation (case 20).
A–C, Precontrast MR study. Midline sagittal (*A*) and right parasagittal (*B*) T1-weighted scans show L3-L4 disk herniation migrated along the posterior aspect of L4 body. *C*, Axial T1-weighted image also displays extension of disk material to the left side with an hour glass appearance (*arrows*).

root enhancement and in five patients (83.3%) with selective root enhancement pattern. A thin peridiscal enhancement was shown in one patient (16.7%) with selective and in five patients (45.5%) with normal root enhancement, without statistical significance ($P > .05$).

Clinical Findings

At the time of MR examination, all the patients had symptoms and signs of radicular involvement corresponding to the site of disk herniation. In all but one of six patients with multiple disk herniation, clinical signs were related to the site of the largest hernia.

Timing of MR Examination

The time interval from the onset of symptoms and MR study was variable. MR studies were performed within 15 days in three patients, within 1 month in four, within 2 months in five, within 3 months in three, within 4 months in one, and within 6 months in four. The average interval between onset of symptoms and MR studies appears to be shorter in patients who showed selective root enhancement (42 days) compared with patients who did not show this type of enhancement (92 days). This did not reach statistical significance ($P > .05$).

Discussion

The large number of lumbosacral roots surrounding the filum terminale is known as the cauda equina. These roots arise directly from the conus medullaris without intermediate rootlets. The individual nerve bundles fan out from the cord as dorsal (afferent) and ventral (efferent) roots. They descend separately for a considerable distance within the lumbar dural sac before reaching their respective intervertebral foramina. Distal to the spinal ganglion, the dorsal and ventral roots unite and emerge as mixed spinal nerve (1).

The structure inside the nerve roots is similar to that in the nerve trunks (2). The endoneurium is the connective tissue in which nerve fiber fascicles are suspended. It remains unchanged from the peripheral nerve through the spinal root as far as the junction of the spinal cord, where there is a sharply defined transition from peripheral nerve to central nervous tissue (the Obersteiner-Redlich zone) (3).

Intrathecal spinal roots differ from peripheral nerves in their coverings (2). The details of the transition between the meninges and the coverings of peripheral nerves at the point where the roots exit from the dural sac have not been fully clarified. Nevertheless, it is well established (4) that the epineurium becomes continuous with the dura and the perineurium divides itself into two layers at the subarachnoid angle (Fig. 5A). The

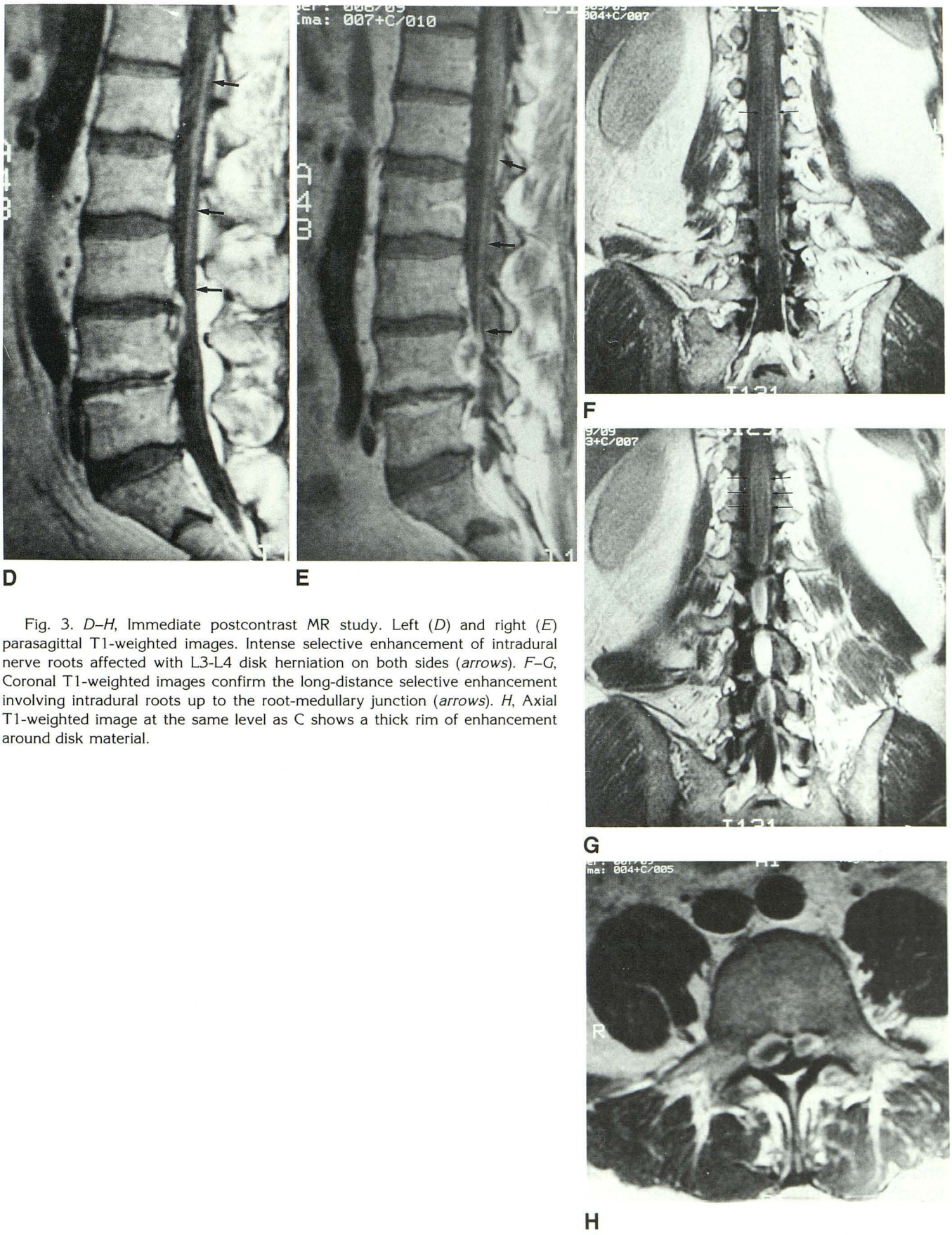


Fig. 3. *D–H*, Immediate postcontrast MR study. Left (*D*) and right (*E*) parasagittal T1-weighted images. Intense selective enhancement of intradural nerve roots affected with L3-L4 disk herniation on both sides (*arrows*). *F–G*, Coronal T1-weighted images confirm the long-distance selective enhancement involving intradural roots up to the root-medullary junction (*arrows*). *H*, Axial T1-weighted image at the same level as C shows a thick rim of enhancement around disk material.

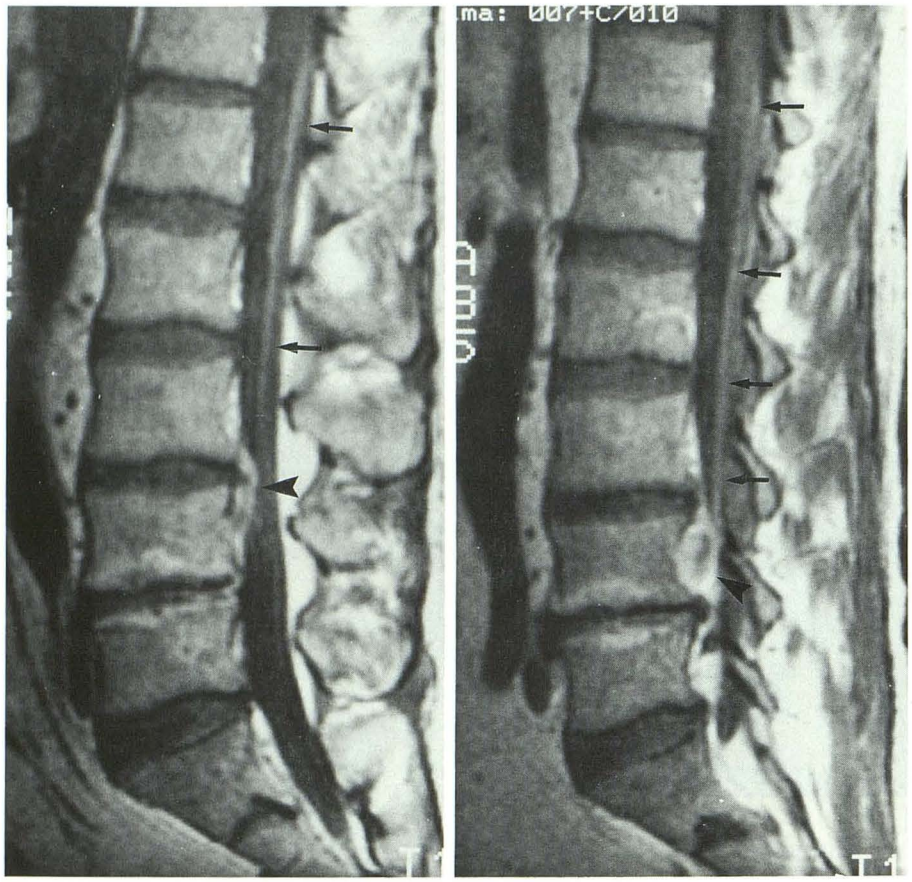


Fig. 3. *I–J*, Delayed (60 min) postcontrast MR study. Sagittal T1-weighted images corresponding to D (*I*) and E (*J*). Observe the persistence of intense selective root enhancement (*arrows*) and peridiscal enhancement (*arrowheads*). Disk material also exhibits a slight enhancement.

I J

outer layer passes between the dura and the arachnoid. The inner layer appears to continue over the roots. It is composed of flattened, compact, contiguous cells closely resembling those of the perineurium in the peripheral nerve. The perineurium in the peripheral nerve acts as a selective diffusion barrier to macromolecular substances and is part of the blood-nerve barrier. The permeability of the perinerium is markedly resistant to compression and to mediators of inflammatory response. This barrier, however, seems to be less efficient in peripheral ganglia and nerve roots than in peripheral nerves.

Intradural roots are supplied by their own radicular artery, which arises from the dorsal branches of the lumbar arteries (5). Each nerve root also has its own vein. Arteries penetrate the perineurium and reach the endoneurium. The endothelial cells of endoneurial vessels are linked to each other by tight junctions; this linkage gives them a barrier property (6). The blood-nerve barrier is similar to the blood-brain barrier but differs in a number of respects. It may be opened by mediators of inflammation such as histamine and serotonin. It is less selective and less effective to solutes of intermediate molecular weight. Experimentally, Cr-EDTA, a macromolecule very similar to Gd-DTPA (7), equilibrates in the interstitial fluid of the nerve within 6 to 8 hours, indicating considerably larger permeability than that of the brain (6). Finally, there is a considerable topographic variation with a greater permeability in the dorsal and ventral roots and even more in spinal ganglia where tight junctions are lacking.

These anatomophysiologic characteristics are the bases of reported normal features of nerves roots and ganglia on enhanced MR (8–10). Dorsal root ganglia show constant marked enhancement as they lack a blood-nerve barrier. Usually intrathecal nerve roots do not enhance (11), but possible minimal enhancement has been noted (8–10). This subtle enhancement, detectable only if pre- and post-contrast scans are compared side by side, probably accounts for the lower efficiency of the blood-nerve barrier in normal intradural nerve roots. Characteristically, this enhancement decreases in time after injection and is not seen on delayed postcontrast scans (10). The extradural nerve roots do enhance as they are ensheathed by the epineurium, which lacks a blood-nerve barrier (6). This pattern of enhancement corresponds to the minimal root enhancement observed in 11 (55%) patients of our series, which we called normal.

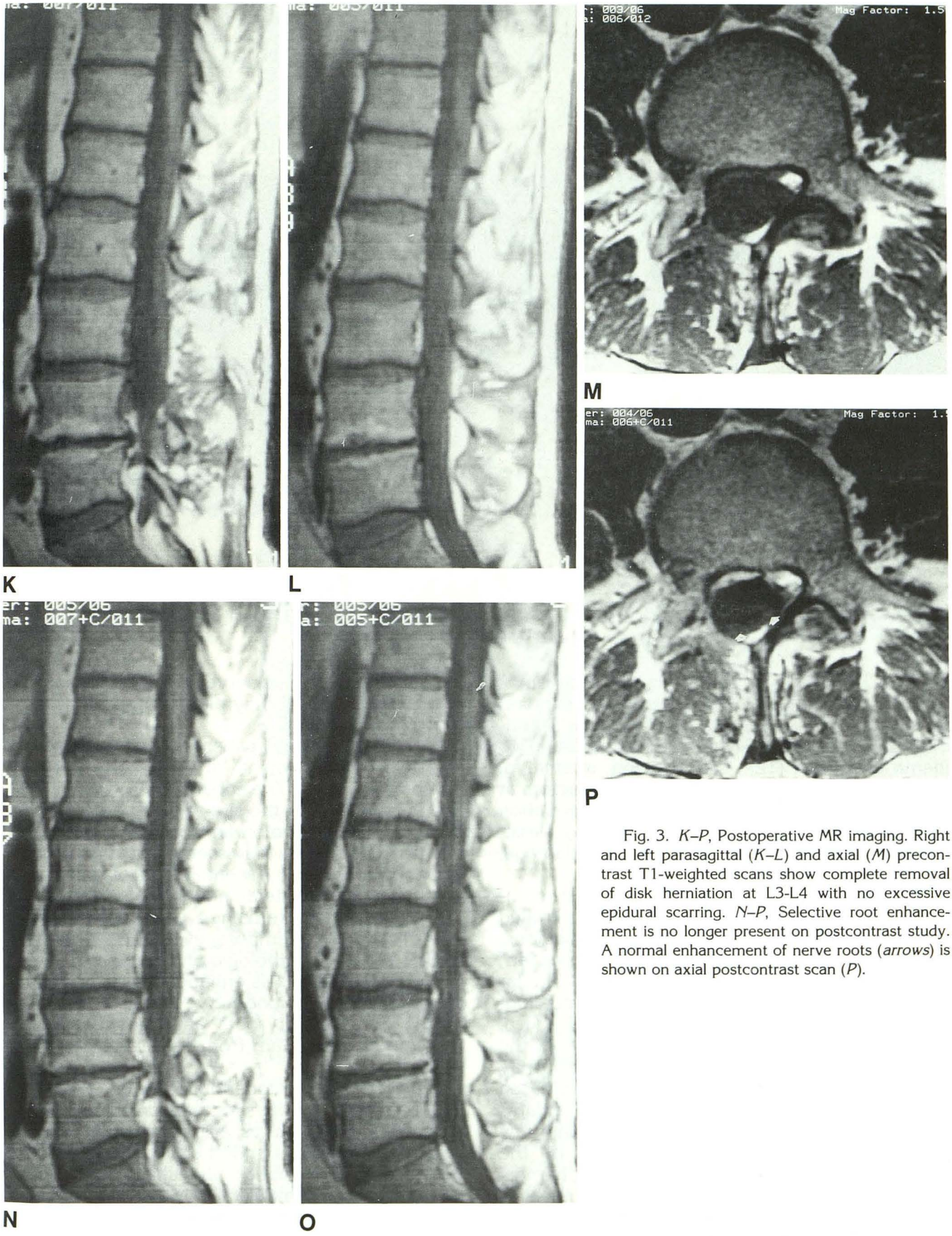


Fig. 3. *K–P*, Postoperative MR imaging. Right and left parasagittal (*K–L*) and axial (*M*) precontrast T1-weighted scans show complete removal of disk herniation at L3-L4 with no excessive epidural scarring. *N–P*, Selective root enhancement is no longer present on postcontrast study. A normal enhancement of nerve roots (*arrows*) is shown on axial postcontrast scan (*P*).

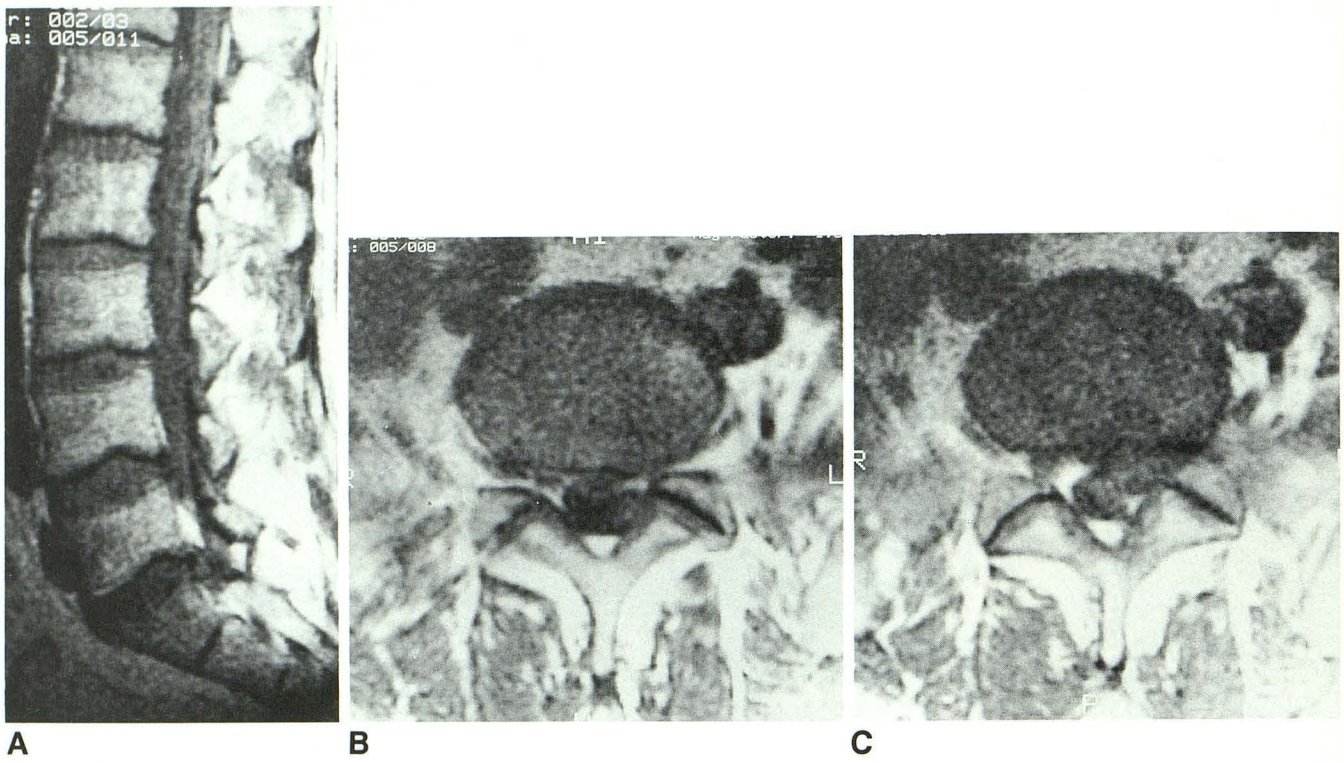


Fig. 4. *A–L*, Large L5-S1 disk herniation (case 8).

A–C, Precontrast T1-weighted images. Sagittal (*A*) and axial (*B–C*) scans display a large left-sided L5-S1 disk herniation extending to the corresponding neural foramen. Axial section B is cephalad to C.

Many pathologic conditions, including compression, ischemia, and inflammation, may induce a breakdown of the blood-nerve barrier leading to outflow of fluid and solutes and to edema formation (12). Compression, stretching, angulation, and friction are mechanical factors at the origin of radicular injury after disk herniation (13). Damage may be caused either by direct physical injury or indirectly by vascular damage, ischemia, or inflammation.

Our knowledge of the pathology of compressed roots involved in disk herniation is limited and based largely on operative observations, where roots are frequently described as "swollen" as a supposed consequence of edema. Similar swelling of the root may be observed on myelography (14) and on computed tomography (15). Occasionally, also on plain MR examination, the compressed nerve root and root sheath may appear to be enlarged and to have increased signal intensity, supposedly because of inflammation and edema (16). We could find only few reports describing the histopathology of the compressed root, showing demyelination, hemorrhages, and widening of endoneurial spaces reflecting edema (17, 18) and inflammatory reactions (19). Likewise, in neural tissue, blood vessel damage with blood-nerve barrier breakdown leads to edema and may play an important physiopathologic role (20).

In our series, six patients (30%) showed a selective enhancement of the corresponding compressed roots. The enhancement was intense and extended from the site of involvement along their whole length to origin from the conus medullaris. Additionally, they seemed to appear swollen compared with nonenhancing or minimally enhancing contralateral nerve roots. Moreover, the enhancement remained unchanged on delayed scans. This temporal evolution would be a further proof of pathologic enhancement (10). It is our opinion that this enhancement reflects the breakdown of the blood-nerve barrier with edema in the compressed roots and the consequent crossing of gadolinium from vessels to endoneurium.

Because the endoneurium lacks lymphatic channels, possible ways for reabsorption of edematous fluid and solutes are: 1) re-entering into vessel lumen across endothelial cells by pinocytosis or tubule formation, 2) leakage across perineurium, 3) proximodistal flow down the nerve trunk to nerve terminals where the perineurium is open-ended and fluid reaches the lymphatics,

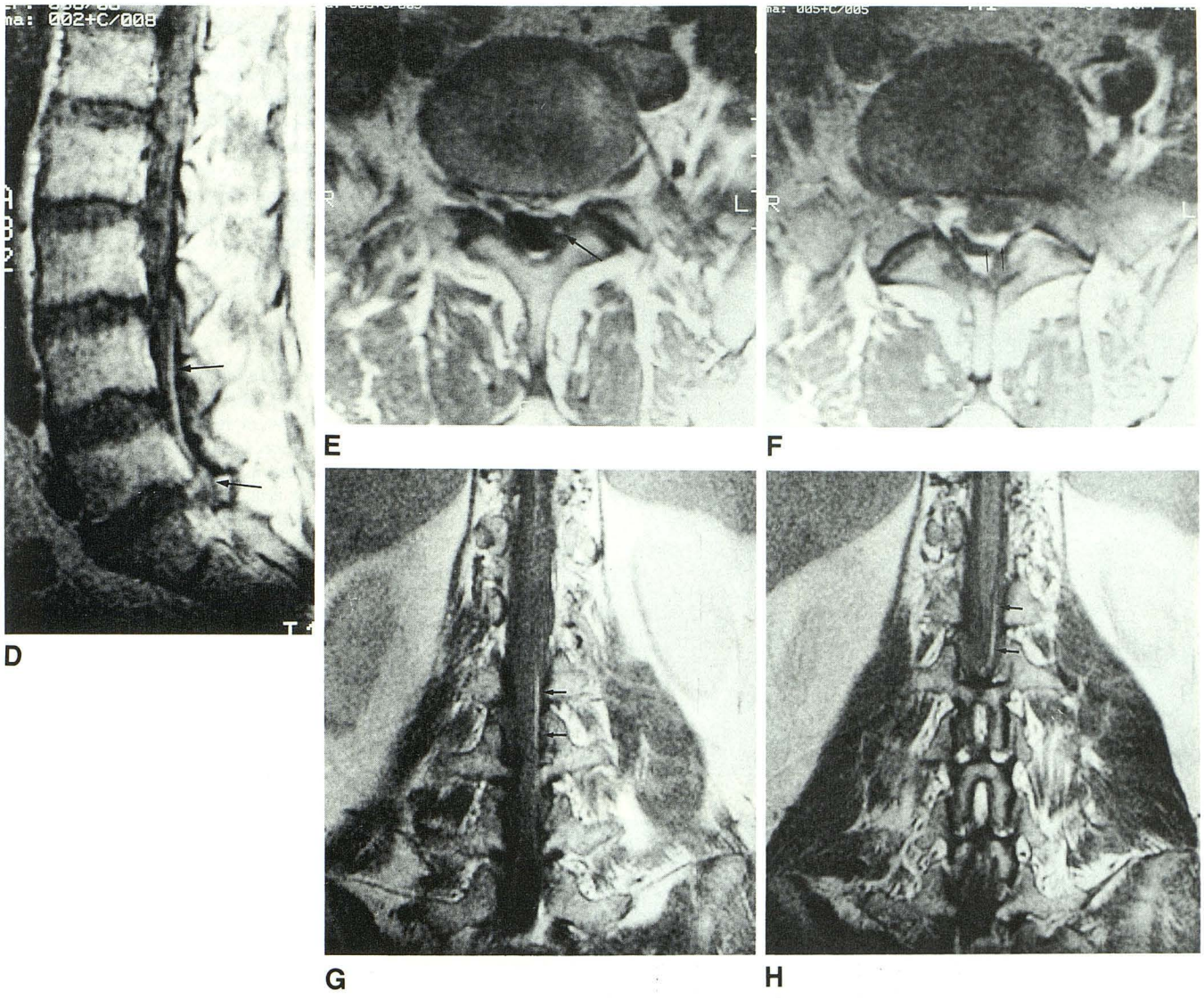


Fig. 4. *D–H*, Postcontrast study. Sagittal (*D*) and axial images (*E–F*) are at the same levels of precontrast study. The intradural left L5 root is seen to enhance intensely at the level of disk herniation and at some distance above disk space (*arrows, D–E*). A thick rim of enhancement is observed around the posterior border of disk material (*arrows, F*). Coronal images show the selective enhancement of L5 nerve root up to the conus medullaris.

and 4) centripetal flow up to nerve roots (12) (Fig. 5A). In our cases, the second mechanism is more likely than the third mechanism because of compression of the root by herniated disk (Fig. 5B). This would explain the diffusion of the gadolinium from the site of compression along the whole length of the intradural root. Edema itself promotes fluid flow along the nerve due to elevation of endoneurial pressure (12). After the injection of minute amounts of fluid into the endoneurial pool, the fluid spreads considerably over a very large distance. Experimentally, if dye is injected into the endoneurium, it spreads almost instantaneously up and down the nerve for a distance up to 6 cm, staining the entire endoneurial contents and remaining unchanged for many hours (21).

Enhancement depends upon the amount of extracellular space. At the region of attachment of spinal roots to the cord, there is a sharp transition from peripheral nervous tissue, in which the extracellular space (the endoneurium) is large, to central nervous tissue, in which the extracellular space is exceedingly small (3). This change is probably the reason the enhancement stops at the root-medullary junction.

Despite the number of investigations performed, many aspects of lumbar disk disease are still not fully understood. The mechanism by which a herniated disk causes radicular symp-

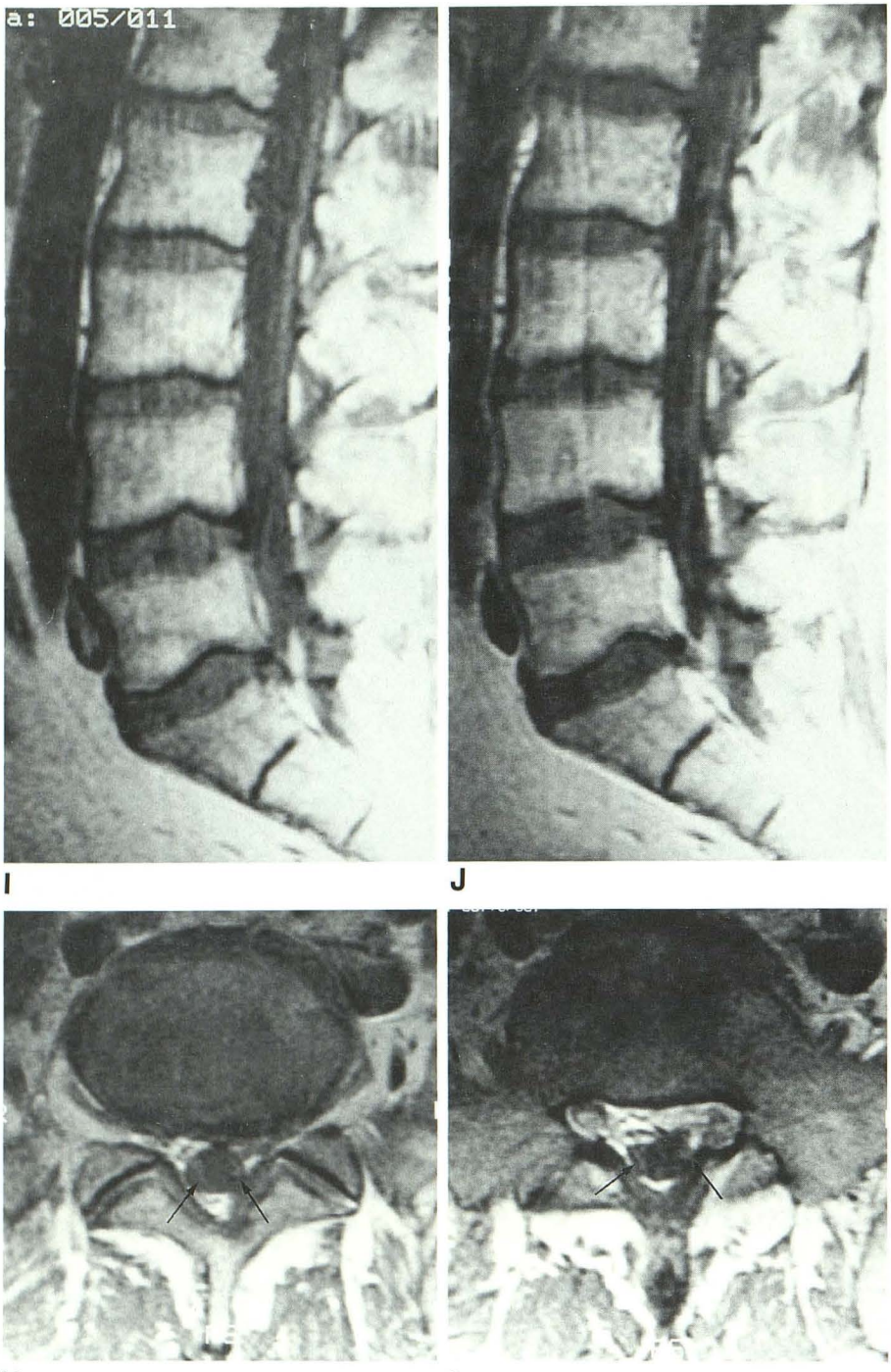
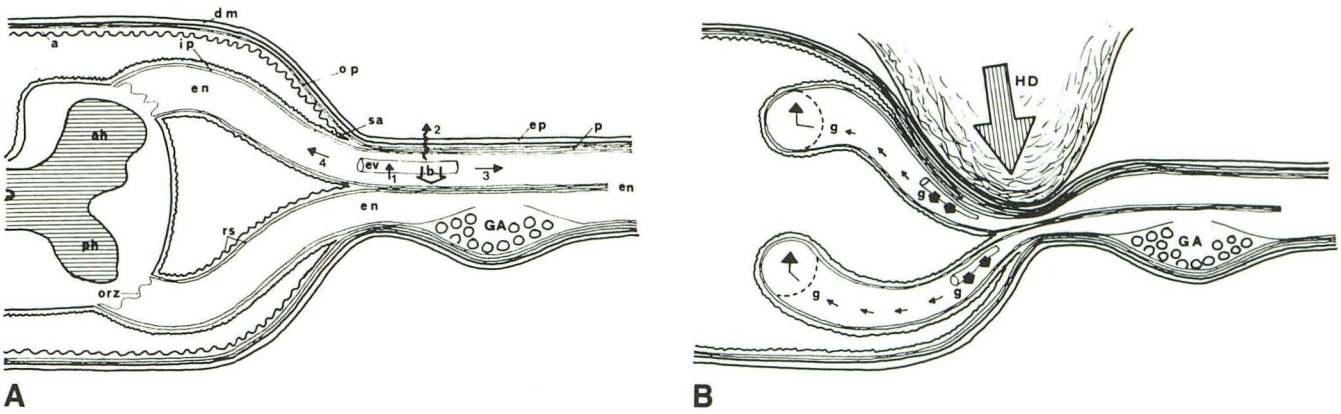


Fig. 4. *I–L*, One-year follow-up after conservative management. Sagittal precontrast (*I*) and postcontrast (*J*) T1-weighted images. L5-S1 disk herniation had disappeared with slight residual bulging disk. Selective enhancement of the corresponding L5 nerve root is no longer visible. *K–L*, Axial postcontrast images show normal appearance of the dural sac and normal enhancement of intrathecal nerve roots (*arrows*).

toms remains uncertain. It is known that disk herniation may be totally asymptomatic (22). However, nerve root compression can fully explain loss of nerve function, clinically shown as motor weakness, reflex changes, and sensory loss. The etiology of radicular pain is far more obscure and compression alone does not seem to be sufficient to cause it in every case (23).

Selective root enhancement stands out as the direct visualization of the radiculopathy resulting from disk herniation, as it probably reflects severe and diffuse edema of the nerve roots. This was found in only six (43%) of 14 patients with large hernias. Even if not all large hernias showed it, this seems to indicate that the extent of compression is an important factor in its etiology.

It has been proposed that inflammation and release of chemicals or metabolites associated with herniated disk could sensitize nerves to pain, playing an important role in the genesis of symptoms (23–25). Peridiscal enhancement corresponds to granulation tissue, which is the result



A B

Fig. 5. *A*, Diagram showing the relationships among root sheaths, peripheral nerve sheaths, and meningeal coverings of the cord. Perineurium (p) divides itself into two layers at the subarachnoid angle (sa). The outer layer (op) passes between the dura (dm) and the arachnoid (a). The inner layer (ip) appears to continue over the roots as a part of the root sheaths (rs). Epineurium (ep) becomes continuous with the dura. Endoneurium (en) remains unchanged from the peripheral nerve through the spinal root. At the Obersteiner-Redlich zone (orz), there is a defined sharp transition from peripheral nerve to central nervous tissue. Blood-nerve barrier breakdown (b) causes crossing of liquid and solutes from endoneurial vessels (ev) to endoneurium with edema formation. Possible pathways of reabsorption are (1) re-entering into the vessel lumen, (2) leakage across perineurium, (3) proximodistal flow down the nerve trunk, and (4) centripetal flow. Ganglium, GA; anterior horn, ah; posterior horn, ph.

B, Nerve root compression by herniated disk (HD) causes blood-nerve barrier to be disrupted and gadolinium (g) accumulation in the entire endoneurial space (see text).

of the nonspecific inflammatory response of the tissue to injury (11). Peridiscal enhancement was found around all but two herniated disks and it was usually thicker in large hernias. In the six patients with selective root enhancement, all large hernias had an enhancing peripheral ring, thick in five and thin in one. Our data, however, do not suggest a direct relationship between peridiscal enhancement and root enhancement. Peridiscal inflammation is a local phenomenon, closely confined to the extradural space and extension to the nerve root through meningeal sheath is unlikely. Furthermore, perineural permeability is strongly resistant to compression and to inflammatory mediators (5). This would indicate that breakdown of the blood-nerve barrier and edema formation in the endoneurium is independent of epidural inflammatory response.

All patients were symptomatic at the time of first examination. We could not precisely quantify symptoms, such as intensity of pain or grade of motor weakness, in each patient. Because of the wide overlap of pain and radicular signs, it is not possible to separate clinically patients with or without selective root enhancement. In other words, a specific clinical correlation is not evident.

Disk herniation and resulting nerve root injury are evolving processes with spontaneous healing or relapse and, not unusually, there is a discrepancy between clinical symptoms and pathologic and radiologic findings. Remission with conservative management is reported to occur in up to 70% of patients (26). The exact basis of this phenomenon is still open to dispute; it has generally been attributed to 1) decreased swelling of the affected nerve root, 2) adaptative root lengthening or shifting away from the protrusion (27), and 3) regression of the herniated nucleus pulposis (28). Selective root enhancement was seen in patients with a shorter average duration of radicular symptoms (42 days) than in other patients (92 days). It would seem that this MR finding may be related to, or more likely to be noted in the acute phase. One could argue that root enhancement is a common but transient event, vanishing as soon as adaptative arrangement of nerve root and/or slight retraction of herniated disk occurs (favored by flexed position that patients assume in conservative bed rest), thus reducing compression to some extent. Nevertheless, because most patients are still symptomatic, we have to assume that a decrease of root enhancement and associated edema is not necessary followed by symptom resolution.

Recurrent episodes after long or short symptom-free intervals are quite common. The detection of selective root enhancement in patients with long-term symptoms could be related to recurrence of the disk-induced radiculopathy.

The good result of surgery in patients 19 and 20 and the spontaneous healing in patient 8 indicate that the occurrence of root enhancement does not negatively influence the prognosis.

Our study is limited in many respects. The number of cases is modest and we could not precisely relate selective root enhancement with patients' symptomatology. Moreover, we could not follow its natural history step by step, relating it to the evolution of symptoms. How will this finding improve MR evaluation of patients with

lumbar-herniated disks? So far, whether or when the blood-nerve barrier of compressed roots breaks down seems to be of little importance. While there is no evidence of an immediate clinical utility of this finding, root enhancement further proves how gadolinium-enhanced MR may be of great interest in studying the natural history and basic pathophysiology of degenerative disk disease (29, 30). Nowadays, herniated disk identification by plain MR seems to be sufficient for the management of patients who have not been operated on previously and who have appropriate symptoms; routine administration of paramagnetic contrast is not needed.

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