Post-Laminectomy and Post-Fusion Stenosis of the Lumbar Spine

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In previous communications[2, 3] the author has recorded his experience with lumbar spinal stenosis from 1964 to 1972. During the past 12 years there has been an increasing awareness of the importance of stenosis of the lumbar spinal canal.[1, 5–13, 15, 16, 18–21] In 1966, the author became aware of a type of severe compression of the cauda equina caused by massive hypertrophy of a laminal fusion encroaching upon the lumen of the spinal canal. We now present a series of 231 patients with postoperative spinal stenosis. The term "iatrogenic" is not appropriate, since the first operation may accentuate or aggravate a pre-existing stenosis. To qualify for post-laminectomy or post-fusion stenosis the patient must possess some degree of spinal stenosis with or without recurrent disk herniation after having had a previous lumbar fusion and/or laminectomy. However, in post-fusion stenosis, the iatrogenic feature must be considered primary.

Table 1 sets out the number of operations performed for disk herniations, for all types of spinal stenosis and for postoperative stenosis. It also sets down the types of postoperative stenosis for which operation was done, the associated pathology encountered in some cases, associated disk herniations and spinal fusion procedures undertaken in several instances (Table 1).

Table 2 gives the overall picture of all types of stenosis in this series and puts postoperative stenosis into perspective.

In the past attention has been focused on the ventral wall of the spinal canal, on the disk, and on the need to fuse the involved vertebral segments. Perhaps when Dandy postulated the "hypertrophied ligamentum flavum syndrome" as an explanation for sciatic pain, when the disk was normal,[4] he was describing a stenotic canal. We know now that the narrowing of the spinal canal normally involves not only thickening of the ligamentum flavum, but also thickening of the bony lamina, medial projection of the articular facets, posterior projection of disks or spurs, and pressure by scar tissue from previous surgery, with pressure on the cauda equina or nerve roots. As pointed out by Schlesinger and Tavaras[17] the developmentally small spinal canal can magnify the clinical symptoms of even small disk herniations. Therefore, it seems logical that enlargement of the neural canal by decompressive laminectomy and/or foraminotomy would serve to enhance the benefits of simple discectomy and/or nominal foraminotomy.

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TABLE 1.

Total number of disk operations, 9 years, 1966–1975					1,329
Total cases of spinal stenosis, all classes				552	
Total number of operations				570	
Total cases of post-laminectomy and post-fusion			231		
Total number of operations			246		
A. Post-laminectomy		125			
Post-fusion		106			
Solid fusion	57				
Pseudarthroses	49				
B. Associated pathology in this series of post-operative stenosis:					
1. Developmental or congenital			31		
2. Spondylotic with narrow posterolateral recesses (trefoil canal)			22		
3. Pseudospondylolisthesis			7		
4. Spondylolisthesis or spondylolysis			16		
C. Associated disk removal in 231 cases:					
Post-laminectomy			111		
Post spinal fusion			62		
Solid fusion		36			
Pseudarthroses		26			
Combination			7		
D. Spinal fusions associated with decompression of spinal stenosis (2d):					
1. Patients fused at time of decompression					60
2. Patients fused at later operation					12
3. Patients with previous spinal fusion					108
(a) No additional fusion done when decompressed				70	
(b) Re-fusion or extension of fusion at time of decompressive laminectomy				38	

With spinal canal narrowing, we have encountered a high incidence of foraminal nerve root compression within the intervertebral canal secondary to disk narrowing, encroachment upon the canal by bone hypertrophy, scar tissue, ligamentous thickening and intraluminal spurs. For the relief of nerve root entrapment, it is essential that these roots be adequately decompressed.

The incidence of 41 per cent with post-operative spinal stenosis in the series of 552 patients with all types of stenosis, appears high. Only 70 of 231 cases had their primary surgery at our hands. While even a canal of normal caliber can become stenotic by virtue of marked hypertrophy of the posterior bone graft, and even more important, by secondary changes at the first free joint above the fusion consisting of a combination of disk herniation, infolding of thickened ligamentum flavum and hypertrophy of articular processes with medial projection, it is the author's opinion that stenosis, especially after a laminectomy alone,

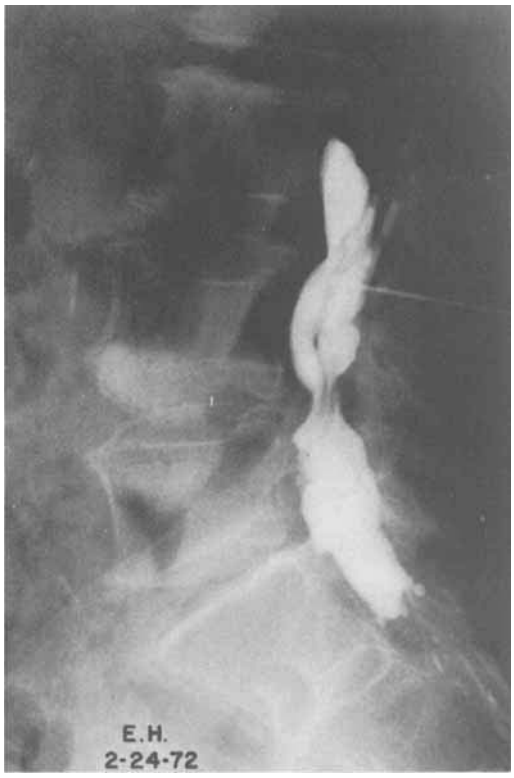


FIG. 1A. E.H., age 69; two previous laminectomies and foraminotomies, still presents severe posterior compression at L4–5, as well as disk herniation at that level.

develops in individuals rendered susceptible by possession of some of the anatomical features of various types of stenosis. These include: (1) Vertical placement of the laminae, especially at L5, producing a shingling effect; (2) Ventral projection and "beaking" of the superior edge of the spinous process; (3) Enlargement and medial projection of the facet joints with consequent narrowing of the transverse diameter of the neural arch; (4) Thickened ligamenta flava, even in relatively young people; (5) A trefoil-shaped spinal canal with narrow posterolateral recesses which predisposes to foraminal entrapment and magnifies the effect of laterally placed disk herniations; (6) A combination of any of the aforegoing with increased lumbar lordosis.

POST-LAMINECTOMY STENOSIS

It would appear that the 6 factors just mentioned are primarily responsible for the development of this type of stenosis. Two other factors must also be considered: (1) Subperiosteal stripping of the lamina has been noted to produce ligamentous thickening and bony thickening similar to the

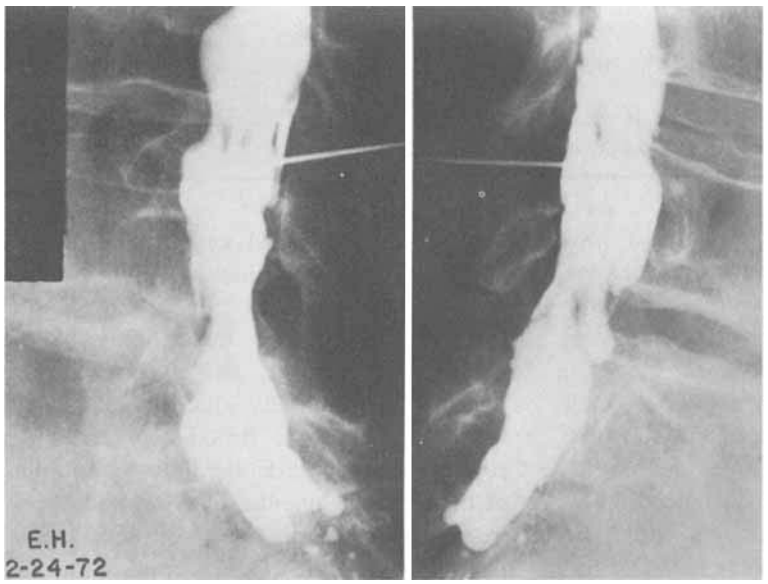


FIG. 1B. See legend for Figure 1A.

TABLE 2.

1a	Congenital-developmental, idiopathic	192
2a	Acquired stenosis, degenerative	
	(i) Central portion of spinal canal	88
	(ii) Peripheral portion of canal, lateral recesses and root tunnels	1
	(iii) Degenerative spondylolisthesis or pseudo-spondylolisthesis	10
2b	Any possible combination	68
2c	Spondylolisthetic or spondylolytic	28
2d	Postoperative	231
	(i) Post-laminectomy	125
	(ii) Post-fusion	106
	(iii) Post-chemonucleolysis	0
2e	Post-traumatic late changes	2
2f	Paget's disease and fluorosis	0

new bone formation which follows elevation of the periosteum of long bones. Several cases in this series presented a sheet of spontaneous new bone formation across the laminae after previous simple laminectomy. (2) Heavy scar tissue at the site of the previous subtotal laminectomy may create posterolateral pressure on the cauda equina. The pathology is represented by compression of the cauda equina by thickened ligamentum flavum and ventral projection of the spinous process, together with the diminished transverse diameter of the canal.

In cases of mild stenosis, we have often tried to enlarge the caliber of the canal by resecting the base of the spinous process and the thickened ligaments, but this usually provides inadequate decompression. We would, therefore, urge that Paine's recommendations of a complete central decompressive laminectomy be followed, even for the lesser degrees of post-laminectomy stenosis[14] (Figs. 1 and 2).

POST-FUSION STENOSIS

The most commonly encountered lesion is a severe concentric stenosis just above the fusion mass, with herniation of a disk, marked thickening and infolding of the ligamentum flavum both posteriorly and laterally, hypertrophy and medial projection of the articular processes and ventral projection of the upper margin of the fusion mass and spinous process. At this point the cauda equina is often reduced to a diameter smaller than a lead pencil. There is usually a lesser degree of diffuse narrowing under the fusion mass with annular accentuation at the level of each joint. The sacral canal is only narrowed at the superior margin of the first sacral lamina and spinous process.

Rarely, one encounters disk herniations under the fusion mass. Entrapment of nerve roots in the lateral recess and in the foramina is most common and must be decompressed, even if this involves sacrificing joint facets to free the nerve to the foramen. Mechanical instability thus produced is usually minor and fusion is not mandatory. In some cases a secondary fusion has been necessary (Table 1). A few of our patients develop slight spondylolisthesis or retrolisthesis. As stated in previous communications[2, 3] we question many of the classical indications for spinal fusion in association

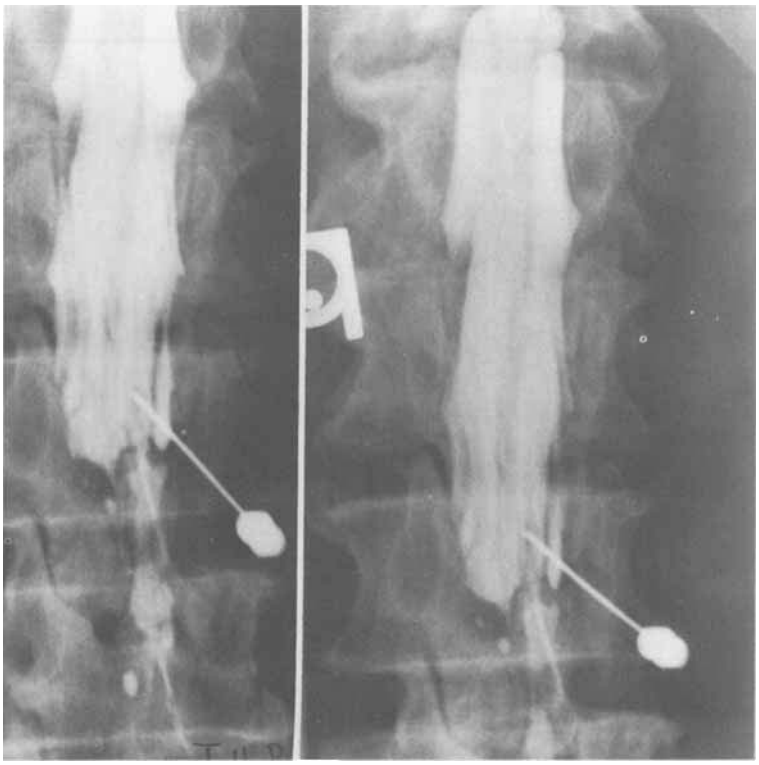
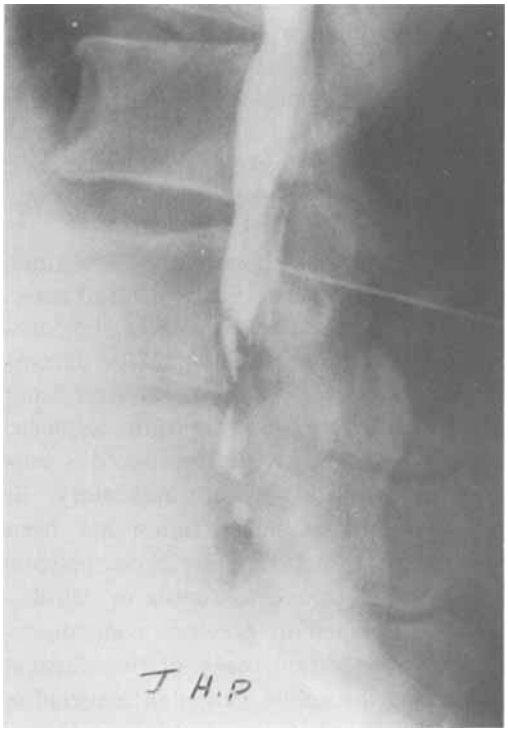


FIG. 2A. J.H.P., age 55; shows streaking of Pantopaque above level of complete block at L3. Previous laminectomy in 1962.



with lumbar disk herniation, but some patients do require this. Experience with post-fusion stenosis has led us to avoid laminal fusions. We now carry out fusion on the transverse processes wherever possible. There has been speculation as to whether pseudarthrosis of a laminal fusion would be more likely to produce spinal stenosis than a solid fusion mass. Review of our series indicated no significant statistical difference (Table 1-A). As previously stated, in 106 post-fusion cases, 57 followed solid fusion and the remaining 49 were cases in which some degree of motion of the fusion could be demonstrated at the time of surgery.

In our experience no significant mechanical weakness of the back develops in those patients where the articular facets have not been sacrificed, and where there is no pre-

FIG. 2B. See legend for Figure 2A.

existing spondylolisthesis. The sacrifice of entire facet joints probably does produce some mechanical deficiency, and in many cases is a deterrent for the more arduous occupations (Figs. 3 and 4).

DIAGNOSIS

As with other types of spinal stenosis, the symptoms of this syndrome may be protean and non-specific. Frequently such patients are dismissed as being neurotic. In this series 12 patients had longstanding motor weakness. Nine of these had foot drop. Three had weakness of the calf muscles. Of these 12, 8 improved significantly after decompression. The diagnosis must be established by myelography. This should be done as outlined by Paine elsewhere in this Symposium. In our series a significant percentage of the less advanced varieties of post-laminectomy fusions have been discovered at the time of re-exploration for nerve root entrapment and/or recurrent disk herniation.

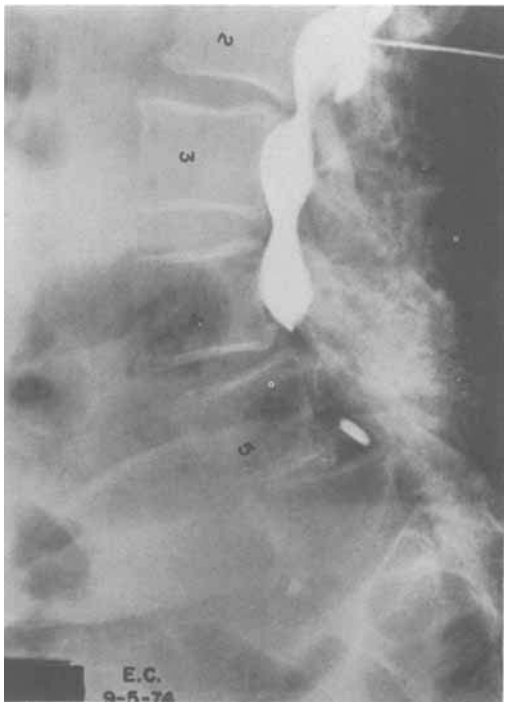


FIG. 3A. E.C., age 79; almost complete block at L4 under massive laminal fusion of 24 years duration.

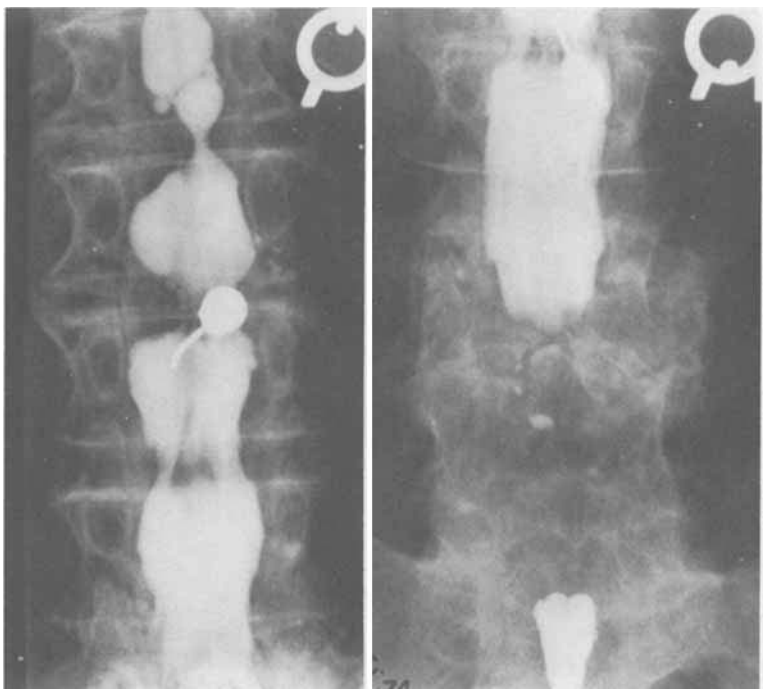


FIG. 3B. Spondylotic spinal stenosis proximal to that level at L2–3 and L1–2.

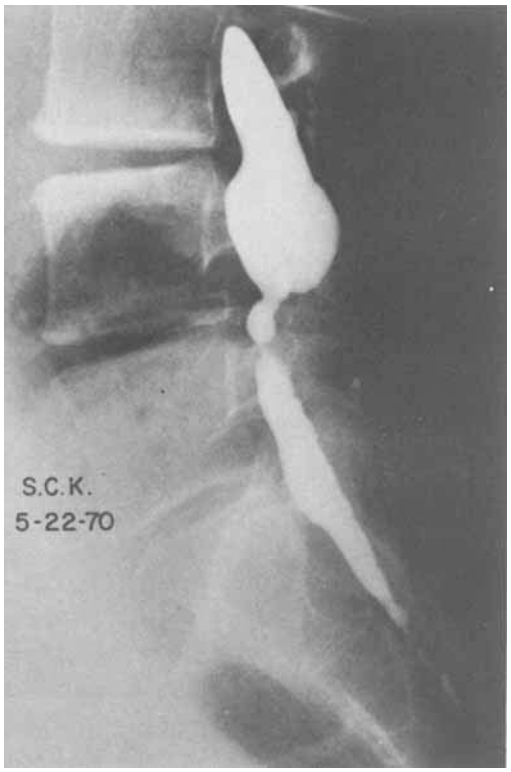


FIG. 4A. S.C.K., age 32; post multiple previous laminectomies and spinal fusions, showing marked narrowing of spinal canal under L5 and partial obstruction at L4–5.

Inaccuracies of roentgenographic interpretation are: (1) Interpreting longitudinal streaking of the dye under a thickened fusion mass as "arachnoiditis" (Fig. 2). (2) Symmetrical narrowing of the column at a joint—the "napkin ring defect," frequently referred to as "bilateral disk herniation," ignoring the posterior portion of the constriction (Fig. 5). (3) Small posterior indentations are overlooked because attention is directed to the anterior wall of the canal. (4) Postoperative irregularity of the column, interpreted as due to adhesions. (5) Complete or almost complete block, which is more often due to the stenotic syndrome than to arachnoid adhesions or to extruded fragments.

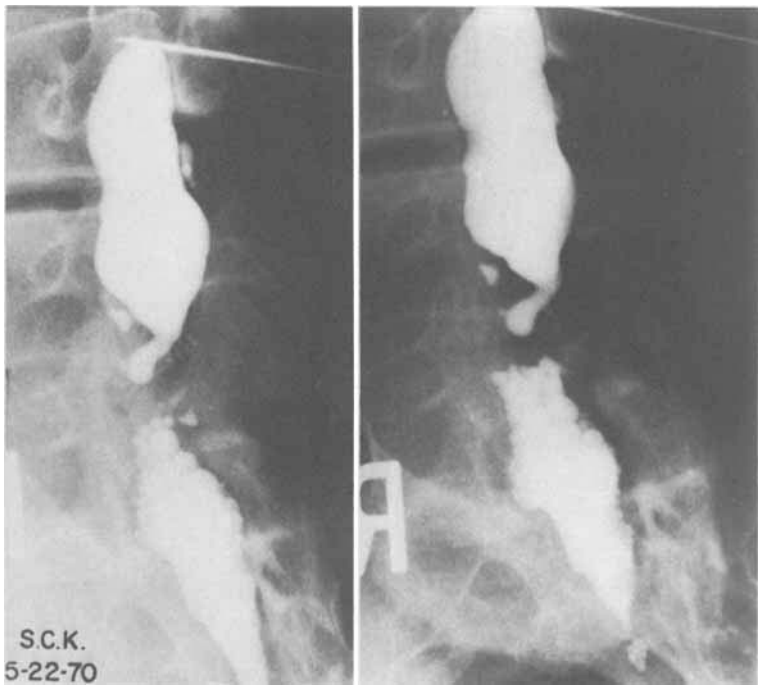


FIG. 4B. See legend for Figure 4A.

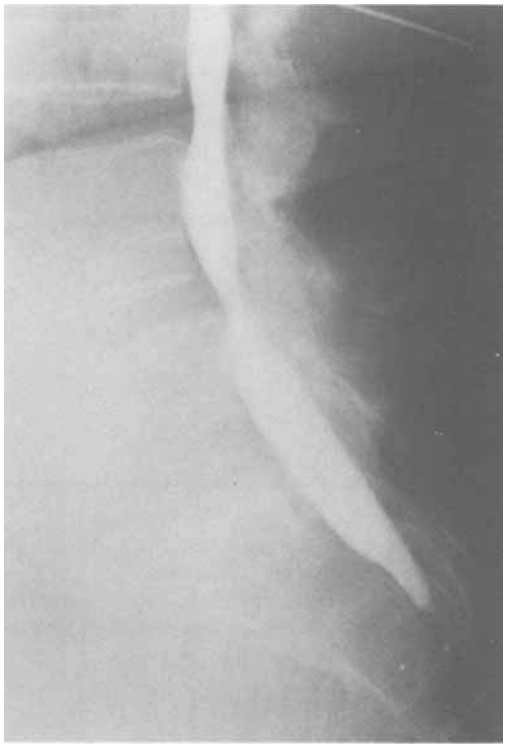


FIG. 5A. M.R.C.P., age 47; concentric "napkin-ring" defect at level of pseudospondylolisthesis at L4–5, previously explored by another surgeon.

METHOD

Surgery in the previously operated back requires the development of certain special skills and experience to avoid damage to nerve roots and to the cauda equina which are adherent to the walls of the canal. The use of sharp curettes to extend the margins of the previous laminectomy opening or to create such an opening, helps to develop planes of cleavage and prevent damage to underlying neural structures. The use of rongeurs or punches before good tissue planes have been developed beyond the scar, is to be condemned. Nerve roots bound down by scar tissue to previously operated upon disks and the lateral wall of the canal should be handled gently and freed with great care. It is helpful to decompress the nerve root distally, before attempting to retract it toward the midline to expose the disk. It is wise to decompress the stenotic

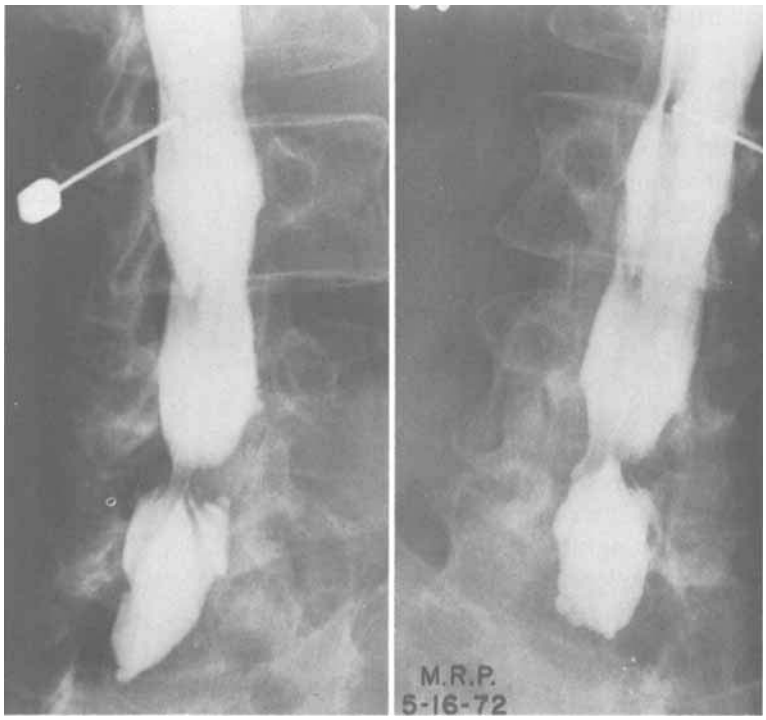


FIG. 5B. See legend for Figure 5A.

TABLE 3. Postoperative Spinal Stenosis (Results)

Average age: 46.9 years	
Average interval between operations: 21 months	
Average follow-up: 4.6 years	
Results:	
Excellent/good	158
Fair	39
Poor	24
Undecided	10
Total	231

canal before exploring the disk. It is important to note that often disk removal is unnecessary. The use of bi-polar coagulation is most helpful in controlling bleeding around the dura and nerve roots.

RESULTS

The success of surgery in our hands is outlined in Table 3. Other factors may contribute to the success or failure of this type of operation. Factors of secondary gain, psychoneurosis, arachnoiditis, permanent damage to nerve roots, either by virtue of previous surgery or by prolonged severe compression all influence the result. However, adequate decompression of stenosis of the spinal canal and nerve root canals in postoperative stenosis gives good or excellent results in most cases (Table 3).

SUMMARY

On the basis of a 9-year experience with 231 patients with post-laminectomy and post-spinal fusion stenosis of the lumbar spinal canal, we emphasize the importance of recognizing and adequately decompressing such lesions. In the majority of such patients pain relief has been achieved, but some of the failures can be attributed to technical reasons. Most failures are due to associated lesions and to functional factors. A special surgical technique required to decompress these lesions, and spinal fusion may be advisable. The indications for fusion however were few and require further investigation and evaluation. The significant failure rate points to the need for continuing research into the causes of low back pain and adequate management programs for the patient with the "multiple operated back" who still has disabling pain and drug dependence.

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