

The Size of the Vertebral Canal and the Significance of Epidural Fat in Lumbar Spinal Stenosis

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Abstract

Measurements of mid sagittal diameter (MSD) and interpedicular diameter (IPD) in patients operated on for central lumbar spinal stenosis were compared to the control group. Both groups can be matched in terms of gender and age. We found that in the stenotic patients the MSD and the IPD were smaller than in the control group, all of the measurements except the IPD in male stenotic patients was statistically different. Sagittal and axial MR images of the stenotic patients were used to evaluate the status of the posterior epidural fat which was graded as normal, small, very small and absent. All the patients were surgically treated for lumbar stenosis, imaging studies and intraoperative finding were correlated. Reduction or absence of the posterior epidural fat (PEF) by the imaging studies were found to be related to the intraoperative findings and the duration of symptoms. PEF may be used as an intraoperative indicator for optimal surgical decompression.

Stenosis is a localized abnormal narrowing of a hollow tubular structure. In view of spinal stenosis, the shape and size of the canal (central or lateral) may have a significant effect on the production of symptoms and signs, which usually do not become symptomatic until late middle age. Canal diameter^(1,2) dural sac⁽³⁾ and reserve capacity⁽⁴⁾ have been measured to provide accurate diagnosis for lumbar canal stenosis. The sagittal and transverse diameter of the canal can be obtained easily and requires no invasive or expensive tests. Winston found that mean mid-sagittal diameter

(MSD) of lumbar vertebral canals were significantly smaller in patients operated on for lumbar disc herniation than in a control group⁽⁵⁾. To date, no comparison has been made between lumbar stenotic patients and normal subjects. Epidural adipose tissue has been reported to be the cause of spinal cord compression in obese patients and in patients receiving long term steroid treatment⁽⁶⁻⁹⁾. Absence or reduction of the posterior epidural fat (PEF) has been pointed out as an usual operative finding in lumbar spinal stenosis surgery⁽¹⁰⁾ and also is an indicator of a tight canal⁽¹¹⁾. Recently,

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absence or reduction of PEF has been mentioned and demonstrated by magnetic resonance imaging (MRI)⁽¹²⁾. Correlation between this imaging evidence and the surgical finding has not been reported in the literature. The purposes of this study were to compare the mean canal diameter (IPD, MSD) in operated lumbar spinal stenotic patients with the control group and to correlate the magnetic resonance images of absence or reduction of PEF with the surgical finding in lumbar Spinal stenosis.

MATERIAL AND METHOD

The study included two parts :

1. A comparison of the measurement of the mid sagittal diameter (MSD) and interpedicular diameter (IPD) in patients with spinal stenosis and in a control group.

2. An interpretation of the presence or absence or reduction of PEF on the T1 weighted MR images of 10 surgical patients which were confirmed at surgery.

MSD and IPD measurements

From January 1985 to January 1992, twenty three control patients and twenty three patients with lumbar stenosis were studied. Both groups consisted of 12 males and 11 females, the mean age was 55 years. The IPD and MSD of L1-L5 were measured with a vernier caliper by Eisenstein's method on standard AP and lateral lumbosacral plain roentgenogram⁽¹⁾. The IPD was measured between the inner most of the pedicles in A-P radiograph (Fig. 1). The MSD was measured as a distance from the middle of the back of the vertebral body perpendicular to the line AB which is the base of the opposing spinous process (Fig. 2). The inclusion criteria for the control were patients who presented with any other orthopaedic conditions except back pain. The inclusion criteria for the symptomatic stenosis were patients who had a clinical entity including back pain, intermittent claudication, sensory paresthesia and weakness in the lower extremities⁽¹²⁾. Measurement was done by one observer (Suntisathaporn N). AP and lateral view of the lumbosacral skeleton (Fig. 3A, 3B) were used for practice in order to be acquainted with the bony landmarks of the patients' X-rays (Fig. 1, 2). Two occasional random checkings for each level (both patients and controls) were carried out by the same observer for possible intraobserver errors. All patients had degenerative lumbar spinal

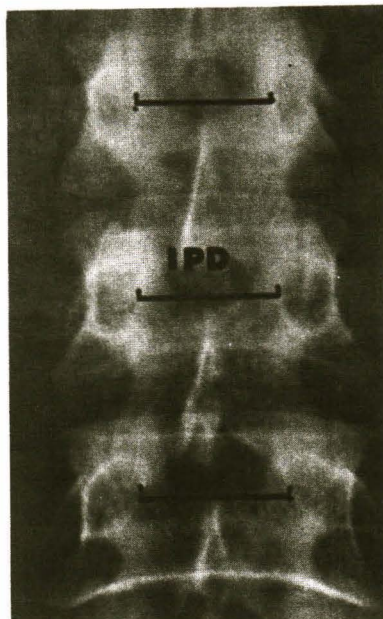


Fig. 1. The interpedicular diameter is a distance between the inner most pedicles.

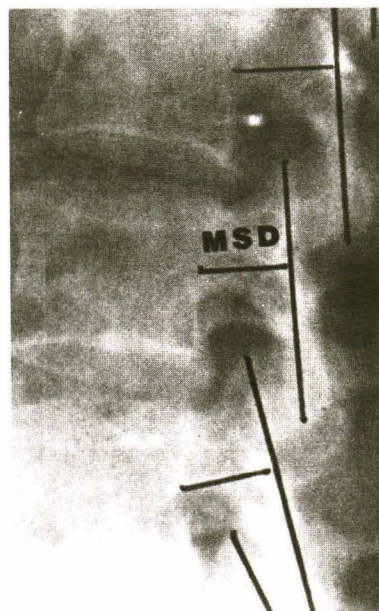


Fig. 2. The mid sagittal diameter is a distance from the middle of the posterior border of vertebral body perpendicular to a line that bisects tips of superior and inferior articular process.

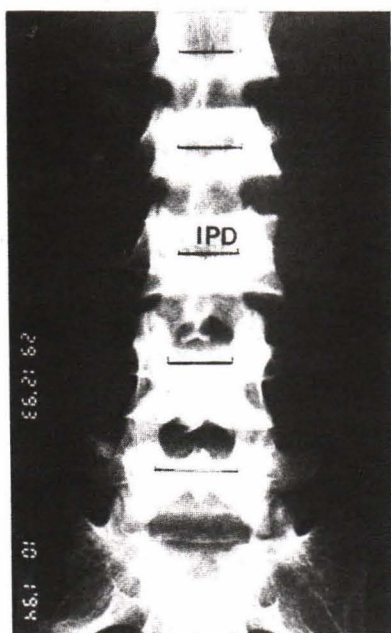


Fig. 3A. Shows IPD of the skeleton.

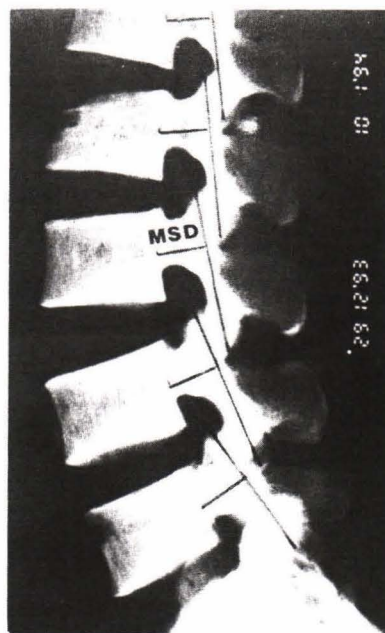


Fig. 3B. Shows MSD of the skeleton.

stenosis except one 18 year old male with developmental lumbar stenosis who presented with acute sciatica and severe intermittent claudication.

Interpretation of epidural fat

The lumbosacral MR images of 10 patients were evaluated for the presence, absence or reduction of PEF by one radiologist (Sriphojanart C). Normally the thecal sac at the disc level is surrounded by high signal intensity PEF on T1 weighted images. On axial T1 weighted images, PEF is identified behind the low-signal-intensity subarachnoid space. In normal subjects, the interface of the thecal sac and PEF on axial MR images has a posterior convexity, on sagittal images it is rather straight. Fatty tissues do not increase in signal on conventional T2 weighted images so only sagittal and axial T1 weighted images were studied. Axial cuts were made parallel to the planes of superior end-plates of the vertebral bodies and through the middle of the intervertebral disc. The thickness of the lower three lumbar PEF was measured with distance mode on the axial and mid sagittal view. All of the patients were surgically treated and intraoperatively confirmed for the absence or reduction of PEF. There were six

females and four males. The age ranged from 18 to 76 with the average being 56.6 years. The average duration of symptoms was 30.3 months, the shortest duration being 1 month and the longest duration 17 years. There were two spondylolisthesis, one developmental lumbar stenosis and seven degenerative lumbar stenosis. Twenty sets (sagittal and axial) of normal lumbar spine images were used for the evaluation of normal PEF (Fig. 4). There were 9 males and 11 females. The amount of PEF was graded in to absent, very small, small and normal. Total obliteration of the PEF was graded as absent (Fig. 5). Grading of the reduction of PEF may be difficult. The AP thickness of PEF above or below the stenosis level were used as a control limit to determine the reduction of fat. Small and very small were graded when there was a one half and three quarter loss of PEF respectively. Three cadaveric lumbar spines and Parkin's anatomic study of the lumbar epidural space were used for the evaluation of normal PEF (Fig. 6)⁽¹⁴⁾.

Operative findings :

Laminectomy was routinely performed by using thin chisel to make a rectangular window over the laminae above and below the compressed

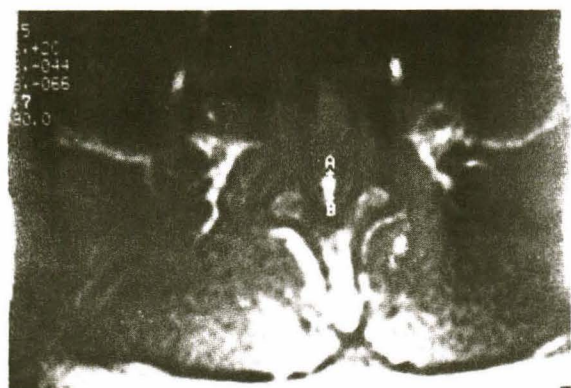
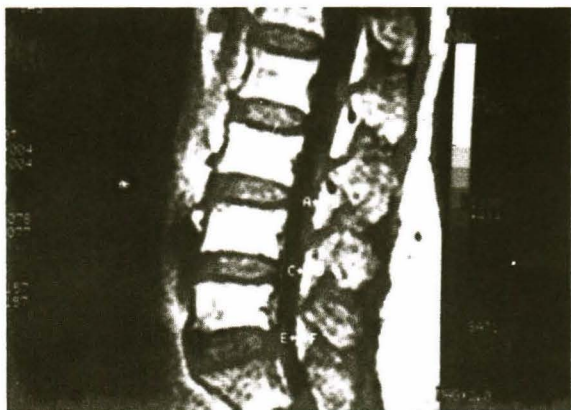


Fig. 4. The normal A-P limit of posterior epidural fat.

- A. Sagittal view. The A-P limit of L3-4, L4-5 and L5-S1 level are 4-7 mm, 4.7 mm and 5.1 mm respectively.
- B. Axial view of L4-5, The A-P limit of posterior epidural fat are 5.9 mm.



Fig. 5. Axial T1 weighted image through L4-5 disc, there is obliteration of the posterior epidural fat (arrow).



Fig. 6. Fresh cadaver is dissected. At the top view, normal triangular posterior epidural fat (arrow) is seen following total laminectomy of L3.

area. Hypertrophic ligamentum flavum was meticulously removed to expose the thecal sac (Fig. 7). The presence or absence or reduction of PEF was carefully recorded. Normally, the PEF presents as a clump of pyramidal shaped fatty tissue occupying the epidural space (between the dura mater and the bony and fibrous wall of the spinal canal). A prominent extension of this fat also follows the inferior and anterior surfaces of each lumbar nerve in the lateral recesses. Absence of PEF refers to complete loss of posterior epidural adipose tissue, small and very small refer to one-half and three quarter loss of posterior epidural fat respectively. Statistical analysis was performed with student's unpaired *t*-test, *p* values less than 0.05 were considered statistically significant.

RESULTS

IPD and MSD measurements (Table 1-3)

Among the twenty three patients with spinal stenosis, the average IPD and MSD were 25.21 and 13.88 millimeters respectively. The smallest were IPD-20.50/MSD-11.60 millimeters and the largest, IPD-32.70/MSD-16.70 millimeters (Table 1). For the control patients the average MSD was 15.67 millimeters and the average transverse diameter was 27.34 millimeters on radiographs (ranged from 20.25 mm to 35.85 mm for IPD and from 13.40-17.55 mm for MSD). The smallest IPD (20.20

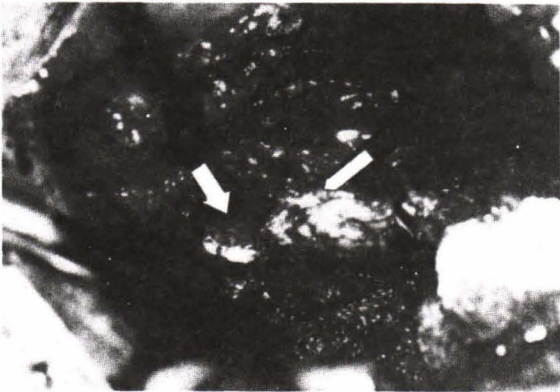


Fig. 7. A. Sagittal T1 weighted image of the L-S spine in a 68 year old male. There are disc protrusion at the L4-5 and L5-S1 level. The posterior epidural fat at L4-5 and L5-S1 disc level are absence. At surgery, the fat is replaced by the thickened ligamentum flavum (arrow).
B. View from above, following removal of the L4 lamina, the thickened ligamentum flavum is indicated by right arrow and the dural sac is indicated by left arrow.
C. Following excision of the thickened ligamentum flavum, the compressed segment (arrow) of dural sac is seen without any coverage of posterior epidural fat.

Table 1. The largest and smallest canal diameters in control and stenotic group.

	Largest (mm)		Smallest (mm)	
	IP	MS	IP	MS
Control	35.85(L ₅)	17.55(L ₅)	20.25(L ₁)	13.40(L _{1,2})
Stenosis	32.70(L ₅)	16.70(L ₂)	20.50(L ₁)	11.60(L ₅)

Table 2. Statistical differences of canal diameter between stenotic patients and controls (P<0.05-significal difference)
A. Shows the P value of the overall IPD and MSD.

			IP	MS
T-Test	LSS 23	Con 23	0.000	0.000
Tails - 2	LSS 12	Con 12	0.166	0.000
Type -3	LSS 11	Con 11	0.000	0.000

mm) and MSD (13.40 mm) of the control were found at the level of L, while the smallest IPD (20.50 mm) and MSD (11.60 mm) of the stenotic patients were found at L1 and L2 respectively. The result of the repeat study by random checking and the overall measurement was slightly different. The L5 vertebra of both groups had the largest IPD (stenosis 32.70 mm, control 35.35 mm). The largest MSD of the control and the patients were found at L5 and L2 respectively. The overall lumbar IPD and MSD in stenotic patients were smaller than those in the control with statistical significant difference (p<0.05). We then analysed the parameter of gender and found that 1). male patients had smaller canals than male controls but only the MSD was statistically significant 2). female patients had smaller canals (IPD and MSD) than female controls with significant difference (Table 2A). 3). Female stenotic patients had smaller canals than male stenotic patients, no statistical difference was found. The results were the same when each level was determined (Table 2B).

Table 2 B. Shows the P value of IPD and MSD in each level (L1-5)

			L1		L2		L3		L4		L5	
			IP	MS	IP	MS	IP	MS	IP	MS	IP	MS
T-Test	LSS 23	Con 23	0.003	0.000	0.004	0.000	0.000	0.000	0.001	0.000	0.000	0.000
Tails-2	LSS 12	Con 12	0.809	0.004	0.696	0.005	0.199	0.000	0.236	0.000	0.073	0.000
Type -3	LSS 11	Con 11	0.000	0.000	0.000	0.039	0.000	0.004	0.000	0.000	0.000	0.000

T-Test-student T test

Tail-2 - two tailed distribution

IP-interpedicular diameter

Type-3 - two-sample unequal variance (heteros cedastic)

MS-mid sagittal diameter

LSS-Lumbar spinal stenosis group (Total 23, male 12, female 11)

CON - control group

Table 3. The mean A-P limit of normal posterior epidural fat.

Disc level	L3-4		L4-5		L5-S1	
	Sag. (mm)	Axial (mm)	Sag. (mm)	Axial (mm)	Sag. (mm)	Axial (mm)
Male (9)	5.1	5.6	5.8	5.0	5.0	4.6
Female (11)	5.6	5.9	5.9	4.9	5.7	5.7
Mean (Total 20)	5.4	5.8	5.9	5.0	5.4	5.2

Interpretation of epidural fat on T1 weighted images

The average normal A-P thickness of posterior epidural fat at L3-4, L4-5 and L5-S1 in sagittal view were 5.4 mm, 5.9 mm and 5.4 mm, in the axial view the average limit was 5.8, 5.0 and 5.2 respectively (Table 3).

Sagittal and axial T1 weighted MR images of ten patients were evaluated. Epidural fat was found to be absent in 4 patients, very small in 4 patients and small in 1 patient. Normal posterior epidural fat was found in one spondylolisthetic patient who presented with acute sciatica.

Operative finding

Intraoperative absence or reduction of PEF was observed in 10 patients. These findings correlated very well with the imaging studies. Normal PEF was observed intraoperatively in a patient who presented with acute symptoms of spondylolisthesis. One small PEF was found in a development lumbar stenosis patient who had an acute onset of intermittent claudication.

The amount of PEF was correlated with the duration of the symptoms. The absence of PEF was found in four patients who had symptoms for an average of 57 months (range from 24-84

months). The average duration of symptoms of very small (4 cases) and small (1 case) amount of fat were 10 months (6-12 months) and 5 months respectively. Duration of back pain of the spondylolisthesis patient who had normal PEF was 3 months.

DISCUSSION

Lumbar spinal stenosis refers to any narrowing of the spinal canal, nerve root canal resulting in highly variable signs and symptoms. The etiology of narrowing of the canal may be multifactorial including degeneration, trauma, tumor and iatrogenic conditions⁽¹⁵⁻¹⁸⁾. Even antero-posterior diameter of the dural sac and reserved capacity are more reliable and more accurate, statistic measurement of MSD has been used as a standard criterion for a pathological constricted spinal canal⁽¹⁸⁻²⁰⁾. From our study, the overall IPD and MSD of the surgical patients were significantly smaller than the control, the mean MSD (15.67 mm) of the control was similar to the study of Eisentein^(1,2). All of the measurements except the IPD of male patients was statistically significantly different. The degenerative process has no effect on the narrowing of the transverse diameter of the spinal canal and none have found the interpedicular distance to be of

clinical significance⁽²¹⁾. In the control, IPD and MSD were found to be smallest at the L1 level while L5 vertebra had the largest IPD and MSD. The smallest MSD of the stenotic patients was found at L5 which is the most common site for spinal stenosis.

Several series reported that the incidence occurs significantly more commonly in males than in females^(22,23). In an attempted meta-analysis the percentage of the male patients was found to be 55.84 per cent⁽²⁴⁾, but in some reports the rate appears to favor females over males^(25,26). Female sex was considered as one of the factors predisposing the patients to worse outcomes^(27,28). Though the mean of IPD and MSD in female stenotic patients were smaller than male patients, no statistical significance was found. Our population was too small to avoid type II (B) error as interpedicular and mid sagittal diameter of male spinal canals were found to be greater than in females with statistical significance in a total of 394 subjects⁽²⁹⁾.

Little has been written on the pathoanatomy of the stenotic spinal canal particularly on the pathologic changes of PEF^(14,30-32). Chronic degenerative changes of the circumferential structures may inevitably lead to various degrees of spinal stenosis and may render the additional reduction of the dimension of the spinal canal more critical⁽³³⁾. By experimental constriction of the intact cauda equina, Schonstrom found a distinct pressure increase among the nerve roots⁽³⁴⁾. With dynamic myelography, dilatation of extradural vessels was found to be the pathophysiologic mechanism of intermittent claudication in lumbar canal stenosis. This microcirculatory changes may lead to pressure increase in both the epidural and intrathecal space⁽³³⁾. In central canal stenosis, the pressure is usually gradually applied in a circumferential manner at a slow rate over a relatively long duration. This biomechanical deformation is likely to induce injury through differential effects on various tissue components, i.e., the nerve fibers, the blood vessels and the connective tissue⁽³⁵⁻³⁷⁾. The overall compression and local anatomic alteration in the spinal canal may be the important etiology of the absence or reduction of PEF. From our observation, pathologic process of the posterior spinal column is found to have more significantly effected on the reduction of PEF than the anterior degenerative changes.

Severity and duration of symptoms before surgery have been reported to be the important predisposing factors related with poor surgical outcomes^(24,38). Herzog found that PEF may be an important component in the pathogenesis of thecal sac compression in patients with lumbar central canal stenosis secondary to facet arthrosis⁽²⁸⁾. He also found the reduction of PEF in some patients and suggested that the developmentally large facets may be the cause of this evidence. The inclusion criteria of Herzog's study was patients who presented with either back or leg pain and having central canal stenosis on their imaging studies, but duration and severity of the symptoms were not mentioned. The onset, rate and duration of compression have been demonstrated in experimental animals to have a significant effect on the degree of neural tissue changes⁽³⁹⁻⁴²⁾. Local injury due to mechanical compression, rather than circulatory insult has been shown to be the major factor for evoked spinal cord potential alterations in chronic cord compression⁽⁴²⁾. From our study, the absence or reduction of PEF was found in all patients who had longer symptoms, PEF was presented in patients who had acute onset of back symptoms. Magnitude and duration of compression that occur in the spinal canal may be related to various biomechanical and microvascular changes in the neural tissue as well as the epidural fat. The localized injury mechanism of disc herniation is more acute and the nerve root often asymmetrically compressed by the protruding disc, this may result in less pressure effect on the PEF⁽⁴³⁾.

Absence or reduction of the PEF are found to have a clinical significance and relate to the duration of the symptoms. The average A-P limit of normal posterior epidural fat in each level both sagittal and axial MR images of males was smaller than females as females tend to have a higher percentage of fat than males. The signal of PEF can be better seen in the sagittal than the axial images. More variables of fat thickness were measured from the sagittal views, this discrepancy may be due to the pyramidal shape of PEF and the technical deviation of the imaging plane. More studies are required to determine the A-P limit of PEF in normal and other spinal disorders.

Early and adequate decompression have been cited as an important factor influencing the satisfactory results, although the study of Dela-

marter did not support the contention that decompression of a cauda equina syndrome is an emergency⁽⁴⁴⁾. The optimal decompression that is most effective in relieving the symptoms of lumbar spinal stenosis is unknown⁽¹⁷⁾. More extensive laminectomy has been reported to be associated with a higher risk of instability⁽⁴⁵⁾ while a single level laminectomy is frequently associated with poor results⁽¹⁷⁾. Amount of PEF at the stenotic level should be determined on preoperative radiologic

images, absence of the PEF may be used as one of the indicators for decompression. Extreme care should be taken to protect the PEF as much as possible during spinal surgery. Failure to preserve this important structure may result in post laminectomy membrane or fibrosis. PEF may be used as an intraoperative indicator for optimal surgical decompression and we suggest that laminectomy should be extended to the level where epidural fat is present.

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REFERENCES

1. Bolender NF, Schonstrom NR, Spengler DM. Role of computed tomography and myelography in the diagnosis of central spinal stenosis. *J Bone Joint Surg* 1985; 67A: 240-5.
2. Brodsky AE. Post-laminectomy and post-fusion stenosis of the lumbar spine. *Clin Orthop* 1976; 115: 130-9.
3. Cranshaw C, Kean DM, Mulholland RC, et al. The use of nuclear magnetic resonance in the diagnosis of lateral canal entrapment. *J Bone Joint Surg* 1984; 66B: 711-5.
4. Delamarter RB, Sherman JE, Carr JB. 1991 Volvo Award in experimental studies cauda equina syndrome : Neurologic recovery following immediate, early, or late decompression. *Spine* 1991; 16: 1022-9.
5. Dorwart RH, Vogler JB III, Helms CA. Spinal stenosis. *Radiol Clin North Am* 1983; 21: 301-25.
6. Eisenstein S. The morphometry and pathologic anatomy of the lumbar spine in South African Negroes and Caucasoids with specific reference to spinal stenosis. *J Bone Joint Surg* 1977; 59B: 173-80.
7. Eisenstein S. Lumbar vertebral canal morphometry for computerized tomography in spinal stenosis. *Spine* 1983; 8: 187-91.
8. Fessler RG, Johnson DL, Brown FD, Erickson RK, Reid SA, Krantzler L. Epidural lipomatosis in steroid treated patients. *Spine* 1992; 17: 183-8.
9. Ganz JC. Lumbar spinal stenosis : postoperative results in terms of preoperative posture-related pain. *J Neurosurg* 1990; 72: 71-4.
10. Garfin SR, Herkowitz HN, Mirkovic S, Booth R. Spinal stenosis, nonoperative and operative treatment. Chapter 25. The spine 3rd ed. Edited by RH Rothman, FA Simeone. Philadelphia, W.B. Saunders Co, 1992; 857-75.
11. Haid RW Jr, Kaufman HH, Schochet SS Jr, Marano GD. Epidural lipomatosis simulating an epidural abscess. Cases report and literature review. *Neuro Surg* 1987; 744-7.
12. Hall S, Bartleson J, Onfrio B, et al. Lumbar spinal stenosis. Clinical features, diagnostic procedures and results of surgical treatment in 68 patients. *Ann Intern Med* 1985; 103: 271-5.
13. Herkowitz HN, Kommos HE. Spinal stenosis, clinical evaluation and differential diagnosis. Chapter 25. The Spine 3rd ed. Edited by RH Rothman, FA Simeone. Philadelphia, W.B. Saunders Co, 1992; 827-30.
14. Herron L, Mangelsdorf C. Lumbar spinal stenosis: Results of surgical treatment. *J Spinal Disord* 1991; 4: 26-33.
15. Herzog RJ, Kaiser JA, Saal A, Saal JS. The importance of posterior epidural fat pad in lumbar central canal stenosis. *Spine* 1991; 16: S227-33.
16. Hogan QH. Lumbar epidural anatomy. A new look by cryomicrotome section. *Anesthesiology* 1991; 75: 767-75.
17. Johnsson KE, Redlund-Johnnell I, Uden A, Willner S. Preoperative and postoperative instability in lumbar spinal stenosis. *Spine* 1989; 14: 591-3.
18. Katz JN, Lipson SJ, Larson MG, McInnes JM, Fossel AK, Liang MH. The outcome of decompressive laminectomy for degenerative lumbar stenosis. *J Bone Joint Surg* 1991; 73A: 809-16.
19. Kirkaldy - Willis WH, Paine KWE, Cauchoir J, MC Ivor G WD. Lumbar spinal stenosis. *Clin Orthop* 1974; 99: 30-50.
20. Jarsen JL, Smith D. Vertebral body size in lumbar spinal stenosis. *Acta Radiol(Diagn)* (Stockn) 1980; 21: 785-8.
21. Olmarker K, Holm S, Rosenqvist AL, Rydevik B. Experimental nerve root compression. A mode of acute, graded compression of the porcine cauda

- equina and an analysis of neural and vascular anatomy. *Spine* 1991; 16: 61-9.
22. Onel D, Sari H, Donmez C. Lumbar spinal stenosis. Clinical/radiologic therapeutic evaluation in 145 patients. Conservative treatment or surgical intervention. *Spine* 1993; 8: 291-7.
23. Ooi Y, Mita F, Saton Y. Myeloscopic study on lumbar spinal canal stenosis with special reference to intermittent claudication. *Spine* 1990; 15: 544-9.
24. Owen JH, Naito M, Bridwell KH, Oakley DM. Relationship between duration of spinal cord ischemia and postoperative neurologic deficits in animals. *Spine* 1990; 15: 618-22.
25. Ochoa J, Fowler TJ, Gilliatt RW. Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. *J Anat* 1972; 113: 433-55.
26. Paine KWE. Results of decompression for lumbar spinal stenosis. *Clin Orthop* 1987; 115: 96-100.
27. Park WW, Gammell K, Rothman RH. Arterial vascularization of the cauda equina. *J Bone Joint Surg* 1981; 63A: 53-62.
28. Parkin IG, Harrison GR. The topographical anatomy of the lumbar epidural space. *J Anat* 1985; 141: 211-7.
29. Pedowitz RA, Garfin SR, Hargens AR, et al. Effects of magnitude and duration of compression on spinal nerve root conduction. *Spine* 1992; 17: 194-9.
30. Porter RW, Hibbert CS, Wicks M. The spinal canal in symptomatic lumbar disc lesions. *J Bone Joint Surg* 1978; 60B: 485-7.
31. Ramsey HJ. Comparative morphology of fat in the epidural space. *Am J Anat* 1959; 105: 219-32.
32. Rauschnig W. Normal and pathologic anatomy of the lumbar root canals. *Spine* 1987; 12: 1008-19.
33. Rawlins BA, Digiaccinto GV. Epidural compression in the spinal canal caused by excessive adipose tissue. A case report. *Clin Orthop* 1992; 275: 140-3.
34. Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. *Spine* 1984; 9: 7-15.
35. Rydevik B, Pedowitz RA, Hargens AR, Swenson MR, Myers RR, Garfin SR. Effects of acute, graded compression on spinal nerve root function and structure. An experimental study of the pig cauda equina. *Spine* 1991; 16: 487-93.
36. Sato K, Mimatsu K, Saito H. Changes in the evoked spinal cord potentials associated with chronic experimental cord compression. *Spine* 1991; 16: 1283-9.
37. Savolaine ER, Pandya JB, Greenblatt SH, Conover SR. Anatomy of the human lumbar epidural space: New sights using CT-Epidurography. *Anesthesiol* 1988; 68: 217-20.
38. Schonstrom N, Bolender DM, Hansson TH. Pressure changes within the cauda equina following constriction of the dural sac. An in vitro experimental study. *Spine* 1984; 9: 604-7.
39. Shephard RH. Diagnosis and prognosis of cauda equina syndrome produced by protrusion of lumbar disk. *Br Med J* 1959; 2: 1434-9.
40. Spengler DM. Current concepts review. Degenerative stenosis of the lumbar spine. *J Bone Joint Surg* 1987; 69A: 305-8.
41. Turner JA, Ersek M, Herron L, Deyo R. Surgery for lumbar spinal stenosis. Attempted meta analysis of the literature. *Spine* 1992; 17: 1-8.
42. Vanharanta H, Korpi J, Heliovaara M, Troup JDG. Radiographic measurements of lumbar spinal canal size and their relation to back mobility. *Spine* 1985; 10: 461-6.
43. Verbiest H. Further experiences on the pathological influence of a developmental narrowness of the bony lumbar vertebral canal. *J Bone Joint Surg* 1955; 37B: 576-83.
44. Weisz GM, Lee P. Spinal canal stenosis. Concept of spinal reserve capacity: Radiologic measurements and clinical applications. *Clin Orthop* 1983; 179: 134-40.
45. Winston K, Rumbaugh C, Colucci V. The vertebral canal in lumbar disc disease. *Spine* 1984; 9: 414-7.
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ขนาดของโพรงกระดูกสันหลังสันเอวและความสำคัญของไขมันที่เอปิดูรา ในโรคโพรงกระดูกสันหลังแคบ

ธวัช ประสาทฤทธา, พ.บ.*, นพดล สันติสถาพร, พ.บ.*,
พงษ์ศักดิ์ วัฒนา, พ.บ.*, จิตติมา ศรีพจนารถ, พ.บ.**

คณะผู้รายงานได้วัด mid sagittal diameter (MSD) และ interpedicular diameter (IPD) ในผู้ป่วยที่ได้รับการผ่าตัดด้วยโรคโพรงกระดูกสันหลังแคบ โดยเปรียบเทียบกับผู้ป่วยกลุ่มคอนโทรล ผู้ป่วยทั้ง 2 กลุ่ม ไม่แตกต่างกันในเรื่องของเพศและอายุ จากการศึกษาพบว่าผู้ป่วยโรคโพรงกระดูกสันหลังแคบจะมี MSD และ IPD แคบกว่ากลุ่มคอนโทรลอย่างมีนัยสำคัญทางสถิติ

คณะผู้รายงานได้ทำการศึกษเปรียบเทียบขนาดของ posterior epidural fat (PEF) ที่แสดงในภาพ sagittal และ axial MR Images ของผู้ป่วยโรคโพรงกระดูกสันหลังแคบกับ PEF ที่ตรวจพบขณะผ่าตัด (intraoperative finding) โดยแบ่งเป็นขนาดปกติ (normal), ขนาดเล็ก (small), ขนาดเล็กมาก (very small) และขาดหายไป (absence) ผู้ป่วยทุกรายได้รับการผ่าตัดเพื่อรักษาภาวะโพรงกระดูกสันหลังแคบ จากการศึกษาพบว่าขนาดของ PEF ที่เล็กลงสัมพันธ์กับการตรวจพบขณะผ่าตัด และระยะเวลาของอาการกล่าวคือ ผู้ป่วยที่มีอาการของโพรงกระดูกแคบเป็นเวลานานจะพบ PEF มีขนาดเล็กลงกว่าผู้ป่วยที่มีอาการระยะสั้น PEF อาจจะใช้เป็นเครื่องตรวจสอบขณะผ่าตัดสำหรับการทำ decompression ของ canal stenosis

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