THE UNIVERSITY *of* EDINBURGH

Spinal stenosis and disorders of the lumbar spine.

Richard W Porter

Submission for the degree of DSc

University of Edinburgh 1998

LIST OF CONTENTS

STATEMENT

This is a record of my publications on the subject "Spinal Stenosis and Disorders of the Lumbar Spine". It describes work which was carried out between 1978 and 1997. I was the originator of the hypotheses being investigated, and I was responsible for applying for grants, recruiting staff, and supervising the research. In more than half the publications I was the senior author. I generated all the hypotheses and personally supervised and led the research in all the studies. I took the leading role in writing about the work, though to a lesser degree in the writing of the papers [17] [18] [24] [55]. I was assisted in the research by many colleagues. Some of the research fellows were candidates for Doctoral theses under my direction [5] [13] [21] [35]. Papers [1] [2] [3] were submitted as part of my MD thesis in 1980. I have not submitted any of the other publications in whole or in part for a degree or diploma. All the papers except [4] were in refereed journals. Where reprints are unavailable they have been replaced with photocopies.

Signed Richard W Porter

ABSTRACT

This is a submission of 57 publications on the subject of "Spinal stenosis and disorders of the lumbar spine". It describes work personally carried out or personally supervised, between 1978 and 1997.

30 publications describe studies of spinal stenosis. The *development of the vertebral canal* is investigated from archaeological material, foetal collections and by studies of volunteers. Intrauterine factors are identified which influence the canal's size and shape. Epidemiological studies are presented which have measured the canal in large numbers of volunteer adults and children, and patients with low back pain. The *clinical significance* of the vertebral canal size in various back pain syndromes is identified and described.

The patho-physiological mechanism of *neurogenic claudication* is investigated, demonstrating by a series of clinical and laboratory studies, that multiple level stenosis in a developmentally small canal, causes venous congestion of the cauda equina. Laser Doppler studies show that as a result of

this venous congestion, there is probably a failure of arterial vasodilatation in response to exercise, responsible for leg symptoms when walking. Papers describe the natural history of stenosis syndromes, and how calcitonin was introduced and investigated as a method of conservative management of neurogenic claudication.

27 parallel publications describe investigations of *other lumbar spine disorders.* Biomechanical and ergonomic studies show that hard work can be good for the spine. In prolapse of the lumbar intervertebral disc, laboratory studies demonstrate the importance of pre-existing degeneration and the formation of a free fragment. The mechanism of clinical signs of disc protrusion, their repeatability and new signs are described. Spondylolisthesis is investigated. Studies examine back pain epidemiology, the differential diagnosis and classification of back pain, spinal surgery and failed surgery.

There is an appendix by title only, which records many of these studies in 7 text books and 19 contributory chapters.

SUMMARY OF THE PUBLISHED WORK

I first became interested in disorders of the lumbar spine whilst working as an orthopaedic surgeon in Doncaster, the centre of the South Yorkshire Coalfields. In 1978 I introduced the use of *ultrasound* as a non-radiation method of measuring the *vertebral canal* [1] [5] , and demonstrated the importance of the canal size in various back pain syndromes, receiving the first Volvo award for basic science [3]. I was the first to confirm what Verbiest had earlier suggested, that a small vertebral canal is a significant factor in patients with symptomatic disc protrusion [2] , and also demonstrated its importance in other back pain syndromes [3] [9] [10] . We showed that the canal size was important in patients attending general practice with back pain [7] , in the absenteeism of older coal miners [8] , and that it had prospective value in predicting severe back pain in a ten year follow up study of young adults [19] .

The stenotic syndrome of *neurogenic claudication* has been of particular interest [10] [16] and those clinical signs which support the diagnosis [14]. I was the first to use calcitonin treatment for neurogenic claudication [6], conducting a double blind cross-over trial [13]. Calcitonin is now widely recognised as a method of conservative management in neurogenic claudication [26] [51]. I was also the first to show that the pathophysiology of neurogenic claudication is usually related to multiple level stenosis [15]. I suggested this to Takahashi and we examined a two level stenosis in his animal model [18]. I further investigated this in our own animal study measuring blood flow with laser doppler, showing that a double level stenosis at low pressure causes venous congestion, and that with venous stasis there is probably failure of arterial vasodilatation which normally accompanies exercise. This study received a further Volvo award for basic science [23]. PET studies were used to help understand the mechanism of the leg pain in claudicating patients [24]. Our current understanding of neurogenic claudication has been presented in review articles [26] [30].

I have studied the *development of the vertebral canal* by examining many archaeological collections [4] [11] [21] [22] [25] . We have described the normal variations in adult life and have shown that the canal is surprisingly mature in infancy. Because there is no catch-up growth, I have suggested that the stenotic adult canal probably may result from adverse intra-uterine environmental factors [11] . As the conus rises in the second trimester, this is probably a crucial time for canal growth [27] . There appears to be a close link between neural and osseous development. The immune and neurological systems have a similar early growth pattern and they may be adversely affected at the same time. This would explain the association which we have observed between adult spinal stenosis and poor health and academic status [12] [20] . We have shown that a number of pre-natal factors, particularly social class and period of gestation, appear to affect the adult canal size [28] . From our understanding of the canal's growth, a new mechanism has been suggested for the occasional but clinically important trefoil shape at L5 [25] . These observations were summarised in the Henderson Trust lecture [29] . They provide new research opportunities to prevent spinal stenosis.

Although this research has focused on the vertebral canal, other parallel publications record studies about *other lumbar spine disorders*. These include biomechanical [42] [43] [55] and ergonomic studies of the spine [36]. I was early to demonstrate that rest could be detrimental in back pain, and that hard work was good for the back [36] [46]. I have taken an interest in the mechanism of the clinical signs of prolapsed disc [47] [51], their repeatability [37] [44] [54] and their significance [41] [43] [44] [57] and have also described new signs of disc protrusion [47] [51]. From laboratory studies we have evidence that the first symptoms of disc protrusion are probably preceded by long-standing symptomless pathological change with fragment formation, which has important medico-legal significance [49] [52]. I have investigated spondylolisthesis [31] [32] [39], back pain epidemiology [33] [35] [38] [53], the differential diagnosis and classification of back pain [40], spinal surgery and failed surgery [45] [56], and have reviewed the pharmacological management of back pain syndromes [50]. I reviewed the spine's physiology in "Upright man", the title of my inaugural lecture for the University of Aberdeen [48].

I wish to submit the following publications on "Spinal stenosis and disorders of the lumbar spine" for the degree of DSc.

Part one. PUBLICATIONS RELATED TO THE VERTEBRAL CANAL AND SPINAL STENOSIS

1) **Porter RW, Wicks M, Ottewell D.** Measurement of the spinal canal by diagnostic ultrasound. *J Bone Joint Surg* 60-B: 481-484, 1978.

2) **Porter RW, Hibbert CS, Wicks M.** The spinal canal in symptomatic lumbar disc lesions. *J Bone Joint Surg* 60-B: 485-487, 1978.

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5) **Hibbert CS, Delaygue C, McGlen B, Porter R W.** Measurement of the lumbar spinal canal by diagnostic ultrasound. *Br J Radiol* 54: 905-907, 1981.

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47) **Ismail AHMA, Porter RW.** Effect of straight leg raising on blood pressure. *Spine* 17: 1117-1118, 1992.

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53) **Varma KMK, Porter RW**. Sudden onset of back pain. *Eur Spine J* 4: 145-147, 1995.

54) **McLean IP, Gillan MGC, Ross JC, Aspden RM, Porter RW**. A comparison of methods for measuring trunk list. A simple plumbline is the best. *Spine* 21: 1667-1670, 1996.

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APPENDIX

(contributions recorded by title only).

TEXTBOOKS

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4) **Aspden RM and Porter RW** *Lumbar spine disorders: Current concepts.* World Scientific. Volume One 1995 and Volume Two 1996.

5) **Porter RW.** *Back injury and litigation*. Bios Scientific Publishers 1995.

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8) **Porter RW.** The value of myelography and ultrasound measurement of the spinal canal to the orthopaedic surgeon. In: "*Myelographic techniques with metrizamide*". Eds: Grainger and Lamb: 205-211, 1990.

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10) **Porter RW.** Development of the vertebral canal. In: "*The lumbar spine*". Eds: Weinstein JN and Wiesel SW: 589-593 1990.

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12) **Porter RW,** Spinal stenosis of the central and root canal. In: "*Lumbar spine and back pain*" Ed: Jayson MIV Fourth edition: 313-332, 1992.

13) **Porter RW.** The development of the vertebral canal. In:"*Lumbar spinal stenosis*". Eds: Andersson BJ and McNeill TW: 47-56, 1992.

14) **Porter RW.** (1) Conservative management of lumbar degenerative disease. (2) Soft tissue injuries of the lumbar spine. In: "*Surgery of the spine*". Eds: Findlay G and Owen R: 675- 694, 1992.

15) **Porter RW**. Central spinal stenosis. In:"*The degenerative lumbar spine*". Ed: Stromqvist: p 64-66, 1993.

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ACKNOWLEDGEMENTS

I wish to acknowledge the help of many research assistants and secretaries whilst I was a Consultant Orthopaedic Surgeon in Doncaster, then Professor of Orthopaedics in Aberdeen and subsequently Director of Education and Training at the Royal College of Surgeons of Edinburgh. I am grateful to the research assistants who have been co-authors in many publications. Without them this research would not have been possible.

I have been supported by grants from the National Coal Board, the Back Pain Association, the Arthritis and Rheumatism Council, The Colt Foundation, the Weldon Trust, the Scottish Office, the College of General Practitioners, Aberdeen Trust Funds and Wishbone Trust.

I have been thankful also for the abundant support of my wife Christine and four sons.

Reprints of publications

Part one - the vertebral canal and spinal stenosis.

publications 1-30

MEASUREMENT OF THE SPINAL CANAL BY DIAGNOSTIC ULTRASOUND

R. W. PORTER, MARGARET WICKS, DAVID OTTEWELL

From the Doncaster Royal Infirmary

A method is described of measuring the lumbar spinal canal by pulsed echo ultrasound. It is simple, safe and has a high degree of accuracy. The lumbar canal has been measured in over 800 subjects including 100 mining recruits and fifty nurses between the ages of fifteen and eighteen years. Ultrasound can demonstrate the degree and extent of bony stenosis. It may have value in preventive medicine, identifying the subject at risk.

The size of the spinal canal has attracted increasing interest since Scheslinger and Taveras (1953) and Verbiest (1954, 1955) described some of the effects of the narrow canal. The bony canal, however, is not easy to measure, especially in the clinically most significant midsagittal diameter. Rothman and Simeone (1975) state that it can only be measured accurately by direct measurement at operation. Jones and Thomson (1968) and Eisenstein (1977) have described methods of measuring the midsagittal diameter from a lateral radiograph, but in practice this is not always easy.

Myelography is probably the best method of demonstrating the constraint the canal places upon its contents, but its limitations have been well documented by Ehni (1969), Williams (1975), Jacobson (1976) and McIvor and Kirkaldy-Willis (1976). There can be significant reduction in the cross-sectional area of the canal from exaggeration of the trefoil shape, with an apparently adequate anteroposterior and lateral diameter. This oblique narrowing may not be recognised by myelography. Sheldon, Sersland and Leborgne (1977) have shown that computed transverse axial tomography will demonstrate this bony encroachment.

This paper presents a method of measuring an oblique sagittal diameter of the lumbar spinal canal by pulsed echo ultrasound. It is simple, safe and has a high degree of accuracy.

METHOD

An oblique diameter of the spinal canal was measured by pulsed ultrasound using a Nuclear Enterprise Ltd Diasonograph, a machine now widely used in obstetric diagnosis (Fig. 1). Olive oil was used as a coupling medium between the transducer and the skin. A 1.5 megahertz transducer was placed one centimetre from the midline of the lumbar spine, inclined at 15 degrees to the sagittal plane, and moved longitudinally at the same inclination from the first lumbar vertebra to the fifth (Fig. 2). With repeated movements and slight

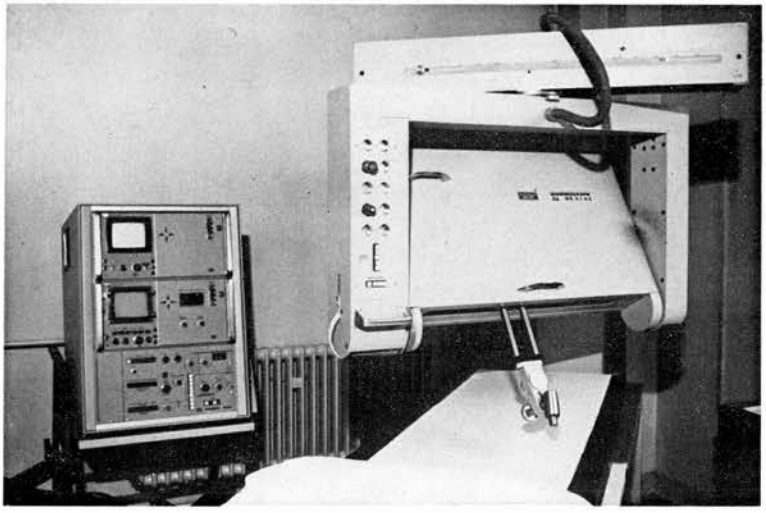

Fig. 1

Fig. 2

Figure 1—Nuclear Enterprise Ltd, Diasonograph. Figure 2—Transducer applied to the skin over the lumbar spine.

R. W. Porter, F.R.C.S., Consultant Orthopaedic Surgeon
Ms Margaret Wicks, Research Assistant
David Ottewell, B.Sc., Ph.D., M.Inst.P., Head, Medical Physics Department
} Doncaster Royal Infirmary, Doncaster DN2 5LT, England.

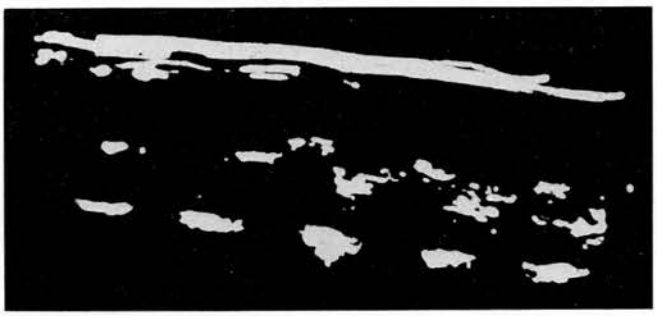

Fig. 3

Two-dimensional display showing echoes reflected from five lumbar vertebrae and laminae.

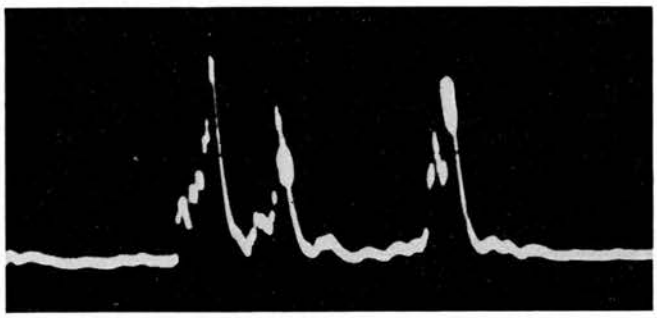

Fig. 4

A-scan display showing three major echoes from the posterior and anterior surfaces of the lamina, and from the posterior surface of the vertebral body.

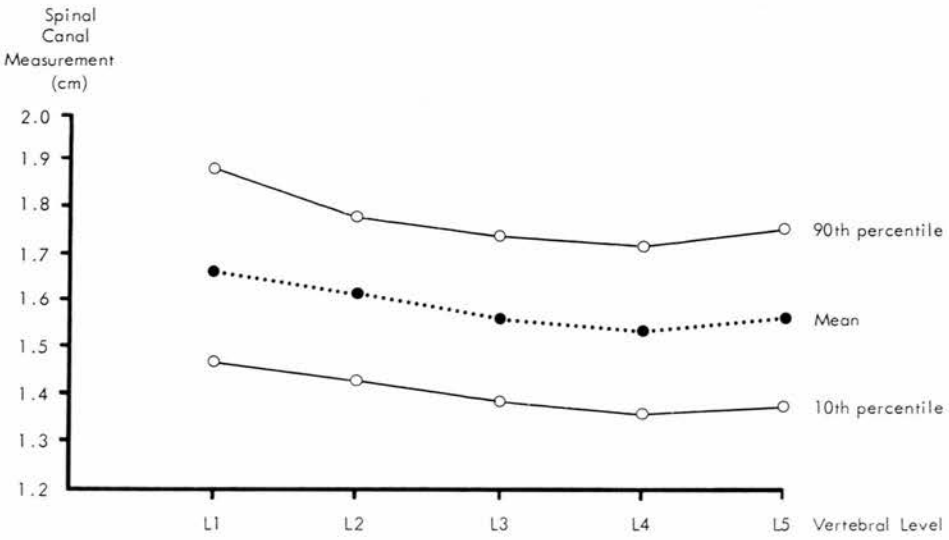

Fig. 5

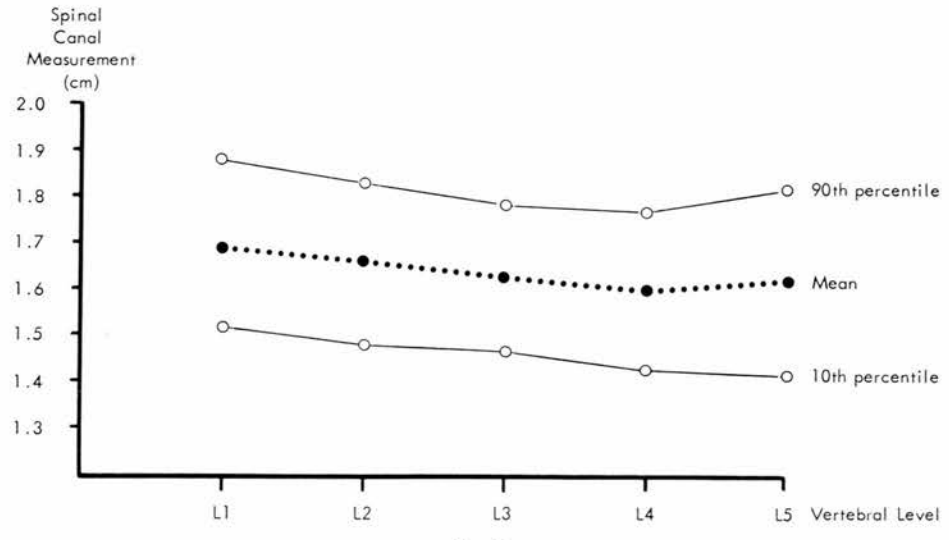

Fig. 6

Figure 5—Mean values and percentiles for 100 young asymptomatic males. Figure 6—Mean values and percentiles for fifty young asymptomatic females.

alteration of the transducer's lateral position, it was possible to obtain echoes reflected from the laminae and from the posterior surfaces of the vertebral bodies as shown in Figure 3. The Diasonograph permits a simultaneous A-scan display of echo amplitudes as a direct function of time, which itself is related to the depth of the reflecting surface below the skin. Three major echoes were demonstrated: from the posterior and the anterior surfaces of the lamina and from the posterior surface of the vertebral body at any one vertebral level (Fig. 4). Slight alteration of the position of the transducer made the amplitudes of these echoes as great as possible and spurious echoes were removed by electronic filtering. The time interval between the second and third echoes from the canal boundaries is related to the distance between the reflecting surfaces, and is measurable in millimetres on a digital read-out. This is facilitated by electronic calipers positioned at the apex of the second and third echoes on the A-scan, and simultaneously displayed on the B-scan identifying the vertebral level of the echoes. The identity of the reflecting surfaces of the three major echoes was confirmed by immersing cadaveric vertebrae in saline, ultrasound and direct measurements being identical.

RESULTS

The lumbar canal has been measured in over 800 subjects, including 100 male mining recruits between fifteen and eighteen years old, and fifty nurses of the same age. The degree of accuracy of measurement of the oblique sagittal diameter of the lumbar canal is shown by the fact that the inter-observer and intra-observer error is only 0.02 centimetre. Difficulty in measuring the canal occurs only in the very obese and, of course, after posterior spinal fusion.

The mean values, and the tenth and ninetieth percentiles for the 100 miners and fifty nurses are demonstrated diagrammatically in Figures 5 and 6. In the oblique sagittal diameter the lumbar canal is widest at the first lumbar level, narrowest at the fourth, and tends to widen again at the fifth level. The mean values of the canals of the young nurses are slightly wider than those of the young miners. Measurements from both the right and left sides of the lumbar spinal canal have been recorded from sixty patients with low backache with or without sciatica. The mean and standard deviation of the differences for each level are shown in Table I.

DISCUSSION

Ultrasonic energy transmitted into the tissues is partially reflected by the boundaries between different structures. This reflected energy is detected as an echo, and the pulsed emission enables the same transducer both to transmit the ultrasound and to receive the returning echoes. Consequently, echoes are received only when reflected from the surfaces at approximately 90 degrees to the axis of the beam. The reflected angle must lie within the solid angle defined by the width of the face of the transducer and the depth of the reflecting surface below the skin. However, the microscopic irregularity of the bony surface ensures that some echoes are received from the vertebral lamina and the posterior surface of the vertebral body, even though the surfaces macroscopically appear oblique to the direction of the incident beam of ultrasound.

Three major echoes on the A-scan are obtained over an extremely narrow band when the transducer is inclined at 15 degrees to the sagittal plane. This band corresponds to the acoustic "window" in the lamina, through which the sound can be transmitted and

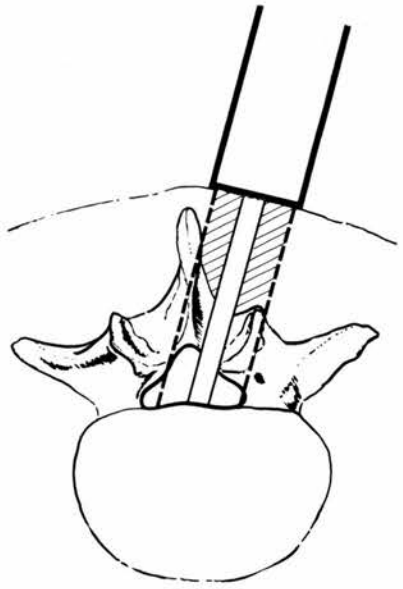

Fig. 7

Diagrammatical representation of part of a two-centimetre beam of ultrasound penetrating the lamina "window".

received. The echoes are lost if the transducer is moved either medially, because of the high absorption of sound by the bony spinous process, or laterally, from absorption by the facet joints and thickened lateral lamina. The "window" of thin bone is entirely covered by the two-centimetre diameter beam of ultrasound and is constant for each individual vertebra (Fig. 7). This probably explains the high degree of reproducibility of the results.

Table I. Differences between right and left oblique sagittal diameters for sixty subjects with backache, with or without sciatica

	Mean difference *(centimetres)*	**Standard deviation of difference**
L1	0.035	0.041
L2	0.042	0.045
L3	0.046	0.062
L4	0.073	0.113
L5	0.055	0.074

The mean oblique sagittal diameter measured by ultrasound is similar to the midsagittal diameter measurements reported by Eisenstein (1977) (Fig. 8). It is understandable that the oblique measurements are less than the midsagittal. The difference is most marked

at the fifth lumbar vertebra where the canal can be trefoil in shape. This will significantly affect our measurements because of the obliquity of the diameter recorded by ultrasound. The mean values for the female canal were found to be slightly greater than those for the male.

Ultrasound measurement of the spinal canal has several clinical implications. Bony stenosis responsible for symptoms of claudication can be identified in degree adequate. Transverse axial tomography will demonstrate this encroachment, but ultrasound offers the advantages of a non-invasive technique that permits accurate measurement. In addition, measurement at each lumbar level helps the surgeon decide the segmental extent of necessary decompression.

The simplicity, safety and accuracy of ultrasound measurement provides opportunity for preventive

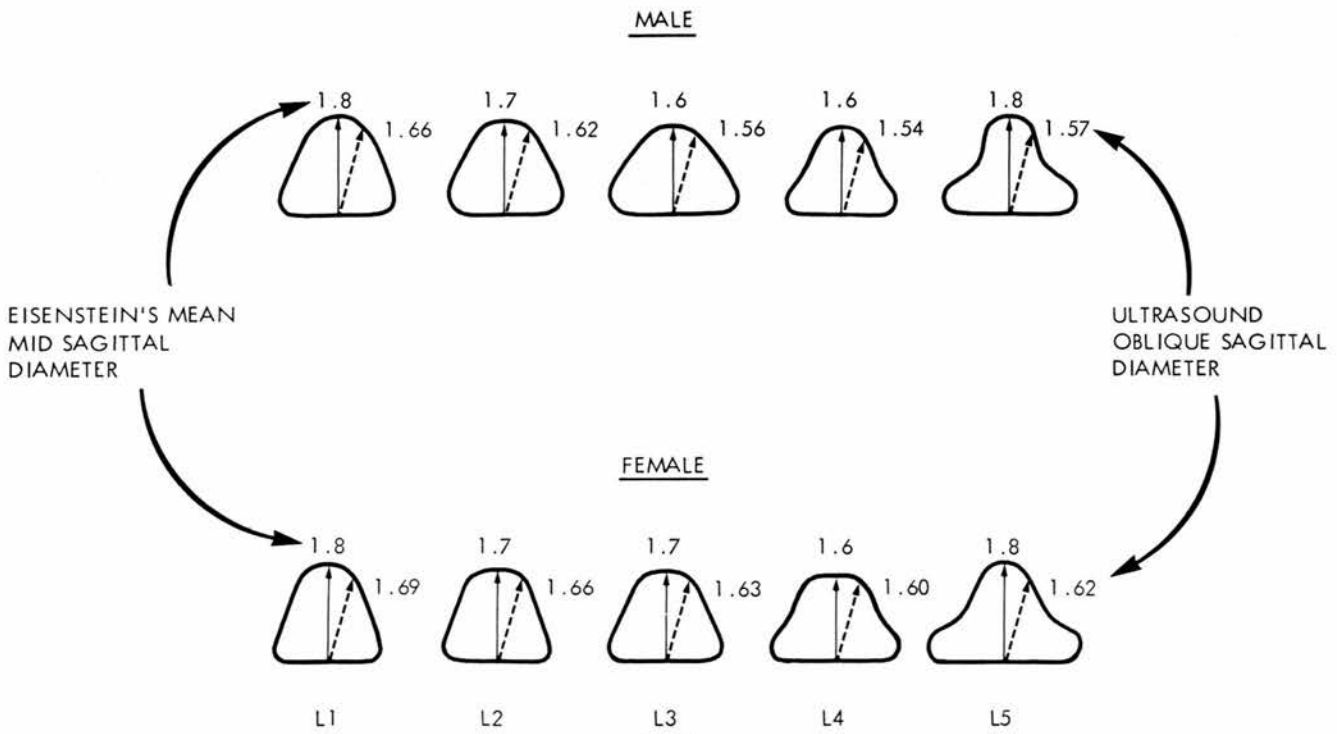

Fig. 8

Comparison between mean oblique sagittal diameters measured by ultrasound and Eisenstein's mean midsagittal diameters.

and extent. It is fortuitous that ultrasound measures an oblique diameter that is most affected in stenosis when laminar hypertrophy exaggerates the trefoil shape. This bony encroachment may not be detected by myelography when the midsagittal and coronal diameters are medicine if it can be shown that a narrow canal increases the risk of disabling symptoms from pathological changes in the disc and from degenerative changes. Young subjects at risk could be identified easily and advised against hazardous occupations and recreations.

We would like to thank the National Coal Board for their financial assistance, Mrs M. Platts for her secretarial help and Mrs C. Hibbert for her help in computing the data.

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THE SPINAL CANAL IN SYMPTOMATIC LUMBAR DISC LESIONS

R. W. PORTER, CATHERINE S. HIBBERT, MARGARET WICKS

From the Doncaster Royal Infirmary

The oblique sagittal diameter of the lumbar spinal canal was measured by diagnostic ultrasound in seventy-three patients with symptomatic disc lesions, and compared with measurements from 200 asymptomatic subjects. Results suggest that the available space in the spinal canal is highly significant in the symptomatology of disc lesions, and in the patient's response to treatment.

Following the description by Mixter and Barr (1934) of the syndrome of ruptured lumbar intervertebral disc, clinicians have shown more interest in the size of the lesion than in the available space in the spinal canal. It is increasingly recognised, however, that a disc protrusion or herniation can compromise an already narrow canal (Williams 1975; Choudhury and Taylor 1977; McCulloch 1977; Verbiest 1977). The relative importance of the size of the spinal canal in the symptomatology of the acute lumbar disc lesion has not been established because of difficulty in obtaining accurate measurement. Ultrasound measurement of the oblique sagittal diameter of the lumbar canal can now provide the opportunity to assess the significance of canal diameter in the presence of disc symptoms.

METHOD

The oblique sagittal diameter of the lumbar spinal canal was measured by ultrasound in patients with symptomatic disc lesions. They had to satisfy three or more of the criteria described by McCulloch (1977): unilateral leg pain in a typical sciatic root distribution, including discomfort below the knee; specific neurological symptoms incriminating a single nerve; limitation of straight leg raising by at least 50 per cent of normal; at least two neurological changes of muscle wasting, muscle weakness, sensory change, or hyporeflexia; and myelographic evidence of disc protrusion.

Measurements were obtained from seventy-three patients with a mean age of thirty-nine years (plus or minus eleven years). Forty-six were men and twenty-seven women. Twenty-eight patients settled with bed rest at home. Of the forty-five admitted for inpatient traction, twenty-one failed to improve and were treated surgically. The measurements were compared with those of 200 asymptomatic subjects, 100 mining recruits between fifteen and eighteen years old, and 100 nursing cadets of the same age.

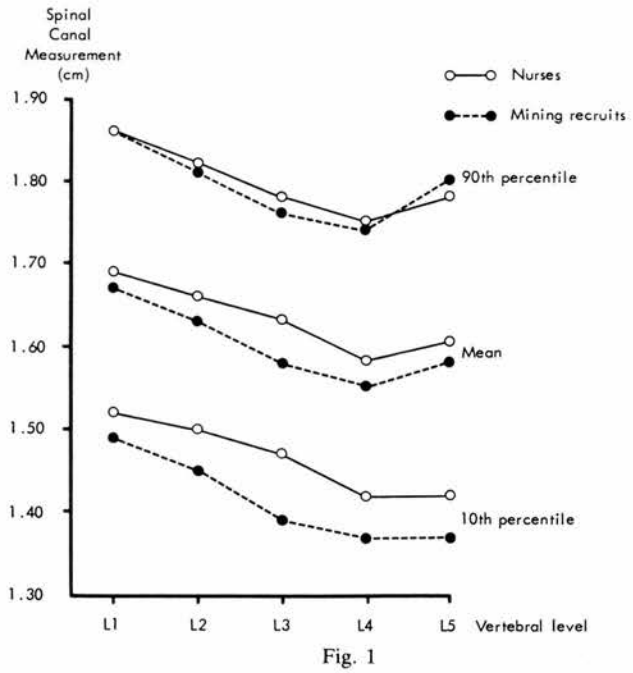

Fig. 1

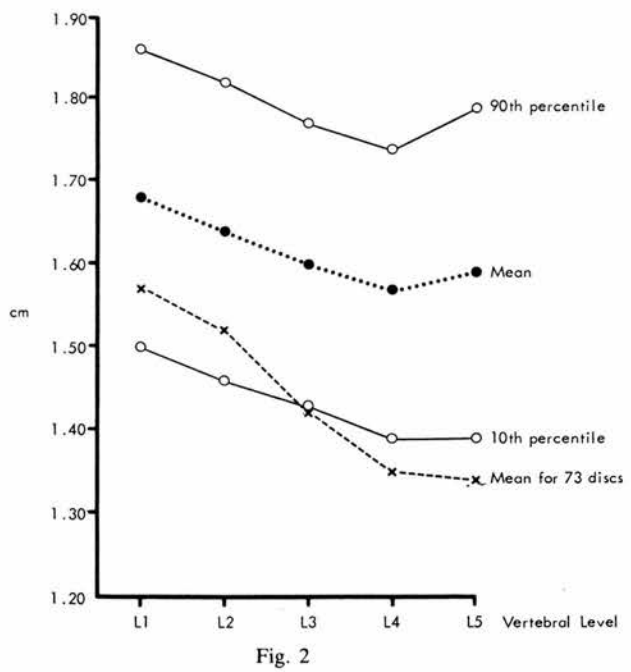

Fig. 2

Figure 1—Mean values and percentiles of oblique sagittal diameter from 100 miners and 100 nurses. Figure 2—Comparison between mean measurements from seventy-three patients with disc symptoms and 200 asymptomatic subjects.

R. W. Porter, F.R.C.S., Consultant Orthopaedic Surgeon
Mrs Catherine S. Hibbert, B.Sc., Research Assistant
Ms Margaret Wicks, Research Assistant
} Doncaster Royal Infirmary, Doncaster DN2 5LT, England.

RESULTS

The measurements of the spinal canals of the 200 asymptomatic subjects are shown diagrammatically in Figure 1, with mean values, and tenth and ninetieth percentiles for miners and nurses at each lumbar level.

The size of the canal at the fifth lumbar level was not related to the findings at operation. Nine patients with sequestrated discs had mean diameters of 1.28 centimetres compared with 1.27 centimetres for nine patients with disc protrusions (Table II).

Table I. Mean spinal canal measurements in centimetres

	200 asymptomatic subjects	73 symptomatic disc lesions	28 discs settled at home	24 discs settled with inpatient traction	21 discs treated surgically
L1	1.68	1.57	1.60	1.52	1.58
L2	1.64	1.52	1.56	1.47	1.52
L3	1.60	1.42	1.45	1.41	1.42
L4	1.57	1.35	1.38	1.32	1.33
L5	1.57	1.34	1.37	1.35	1.27

The mean values for the seventy-three patients with disc lesions are compared with the asymptomatic subjects in Figure 2, and the measurements at the fifth lumbar level are compared with the percentiles of the asymptomatic subjects in Figure 3. Fifty-five per cent were below the fifth percentile, and 68 per cent were below the tenth percentile.

There was a relationship between the size of the canal and the response to treatment. Twenty-eight patients whose symptoms settled at home had a mean diameter of 1.37 centimetres at the fifth lumbar level. This same mean diameter was 1.32 centimetres for forty-five patients requiring admission, and 1.27 centimetres for the twenty-one patients treated by operation (Table I).

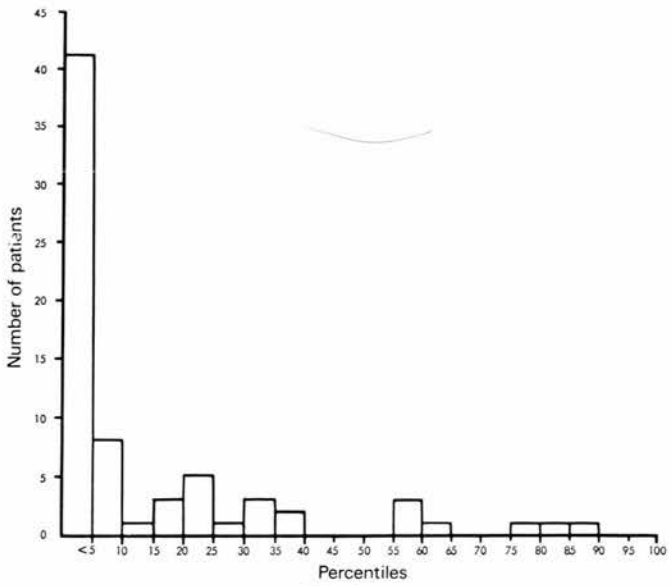

Fig. 3

Measurements from seventy-three patients with disc symptoms compared with percentiles of 200 asymptomatic subjects.

DISCUSSION

The available space in the spinal canal is highly significant in the symptomatology of disc lesions. The fact that 55 per cent of patients with disabling disc symptoms had canal measurements below the fifth percentile of asymptomatic subjects suggests that many patients with wider canals escape root involvement in the presence of disc prolapse. It is the patients with measurements less than 1.4 centimetres at the fourth and fifth lumbar levels who are at risk. The probability of failure to respond to treatment is also related to the size of the canal. The narrowest canals were recorded in the patients who required surgical treatment.

Table II. Mean spinal canal measurements in centimetres

	9 sequestrated discs	9 disc protrusions
L1	1.62	1.60
L2	1.55	1.54
L3	1.45	1.41
L4	1.41	1.27
L5	1.28	1.27

Lumbar nerve roots will be most vulnerable to compression in a narrow canal of trefoil shape. Eisenstein (1977) recorded the trefoil shape at the fifth lumbar level in 15 per cent of spines he examined, and concluded that this was a developmental variant rather than the result of degenerative change. The pattern of measurement throughout the lumbar spine suggests that many of the canals we have measured in patients with disc symptoms are narrow canals of trefoil shape (Fig. 4). The mean oblique measurement recorded by ultrasound decreased throughout the lumbar spine in

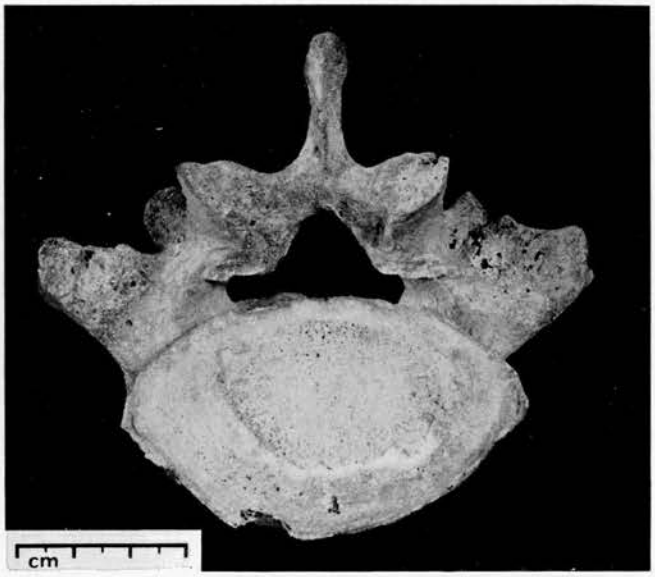

Fig. 4

Trefoil L5 spinal canal. Nerve roots would be vulnerable to compression in the presence of a disc lesion.

seventy-three patients with disc symptoms and was narrowest at the fifth level, whilst the mean measurements of 200 asymptomatic subjects were narrowest at the fourth level, widening again at the fifth. The trefoil shape most significantly affects the oblique sagittal diameter, and when this mean measurement is smaller at the fifth level than at the fourth, it probably indicates a high incidence of the trefoil shape.

No correlation was found between the size of the canal at the fifth level and examination of the disc at operation. We had anticipated that the canals would be wider in patients with herniations than in those with smaller disc protrusions, but this was not confirmed in this small series.

The high incidence of narrowing in the presence of disc symptoms is probably not generally recognised, and may account for some of the poor results of operation. It is difficult to appreciate the dimensions of the spinal canal when it is examined through the usual limited exposure. Naylor (1974) records a continuation of some symptoms in 62 per cent of patients after operations for disc prolapse, and Gurdjian *et al.* (1961) in 71 per cent. Verbiest (1977) explored and measured the canal at several levels with a "Stenosimeter". He reported surgical failures due to unrecognised stenosis in the presence of disc protrusions.

Measurement of the spinal canal before operation offers a more rational approach to surgical treatment. In the presence of a 1.1 centimetre canal and a small protrusion, it would be unreasonable to enucleate the disc through a fenestration and risk subsequent stenotic symptoms. Decompression would be a more reasonable procedure and might perhaps be necessary at more than one level. The larger sequestration in a wider canal can readily be treated by excision of the disc alone.

The risk of developing disabling symptoms from disc protrusion is inversely related to the size of the spinal canal. It is now possible in adolescence to identify subjects at risk and to offer vocational counselling and the benefits of ergonomics to this selected group. It would be economically relevant, and offer hope of reducing a major cause of morbidity in adults.

We are grateful to Mrs M. Platts and Mrs J. Reynolds for the typescript, and to the National Coal Board for its financial assistance.

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Reprinted from
SPINE • VOLUME 5 • NUMBER 2 • MARCH/APRIL 1980

1979 Volvo Award for Basic Science

3

Backache and the Lumbar Spinal Canal

R. W. PORTER, FRCS, FRCSE, C. HIBBERT, BSc, and P. WELLMAN, BSc

This paper records measurement of the lumbar spinal canal by diagnostic ultrasound in more than 700 subjects from early infancy until the age of 65 years. It demonstrates the range of canal size in a South Yorkshire population. The canal is relatively wide in children, reaches a maximum diameter in the late teens, and reduces slightly by late adult life. This does not appear to be related to occupation. Comparisons are made with more than 700 patients with symptoms of back pain, especially patients with disabling disc symptoms, root entrapment syndrome, and neurogenic claudication. The size of the central canal is particularly significant in patients who have neurogenic claudication and disc symptoms. It is less significant in root entrapment syndrome. [Key words: low-back pain, spinal canal size, ultrasound]

THE RELATION between back pain and the size of the spinal canal has not been established because of the difficulty of obtaining accurate measurement. The midsagittal diameter is difficult to measure by conventional radiographic methods, especially at the lower lumbar levels where the canal's posterior boundary is indistinct. Jones and Thompson[4] and Eisenstein[2] have demonstrated methods for its identification, but the methods are not always easy to apply. Verbiest[10] has measured the canal at operation with a "stenosimeter," and Rothman and Simeone[8] state that the canal can be measured accurately only by this direct measurement. Computed axial tomography will demonstrate bony encroachment.[3,9] It offers an opportunity to identify variations in the size and shape of the canal.

Pulsed echo ultrasound can provide a repeatable measurement of the spinal canal in the 15° oblique plane.[7] This paper describes the measurements obtained by ultrasound from a random sample of more than 700 subjects between infancy and the age of 65 years, and compares their results with more than 700 patients suffering from back pain.

METHOD

Measurements were obtained by ultrasound from two groups of young people between 15 and 21 years of age; the first were 188 male recruits to the coal mining industry, and the second, 102 female nurses. The canal was measured in 150 children from the second to the 18th year. These children had neither backache nor known pathologic conditions of the spine. Most had visited a clinic for treatment of upper limb fractures. Measurements were recorded from three groups of subjects between 50 and 65 years of age. Two hundred were underground miners who had received a financial incentive to attend for measurement. They represented half the underground labor force in that age group at one colliery. Eighty were male sedentary workers, mostly doctors, solicitors, or office workers. Fifty-two were female nurses. Comparisons were made between these groups and patients who had back pain.

Lower Lumbar Disc Lesion

One hundred fifty-four patients under the age of 45 years were considered to have symptoms associated

From Doncaster Royal Infirmary, Doncaster, England.

Presented at the Sixth Annual Meeting of the International Society for the Study of the Lumbar Spine, Göteborg, Sweden, May 29–June 2, 1979.
Submitted for publication June 18, 1979.
The authors express gratitude to the National Coal Board and the Back Pain Association for financial assistance, to Mr. G. Swann for photographic work, and to Mrs. J. Reynolds for secretarial assistance.
0362-2436/80/0324-0099$0100

with a lower lumbar disc lesion. They fulfilled two of the following three criteria: 1) unilateral leg pain in a nerve root distribution, extending below the knee, and worse in the leg than in the back; 2) straight leg raising less than 50°; and 3) two of four abnormal neurologic signs: diminished reflex, sensory loss, muscle weakness, muscle wasting.

Clinic Patients

Measurements were obtained from a consecutive series of patients attending an orthopaedic clinic, 165 under the age of 30 years, and 242 between 50 and 65 years old. They were unselected apart from the fact that they complained of pain in the lower back, the leg, or both.

Root Entrapment Syndrome

Seventy-eight patients over the age of 45 were considered to have symptoms of root entrapment. They had unilateral leg pain in a nerve root distribution from the buttock to the foot. Back pain was frequently absent or mild. The leg pain was severe and constant, often making the patient pace the floor at night. Unlike the pain of younger patients with disc symptoms, this pain was not relieved by recumbency. Abnormal signs were few. Back extension was frequently limited, and there was often slight lower lumbar tenderness. Straight leg raising was usually full or only slightly reduced, and neurologic signs were normal.

Neurogenic Claudication

The canal was measured in 138 patients who had classic symptoms of neurogenic claudication. Symptoms involved both legs in a generalized rather than in a root distribution and were aggravated by walking and relieved by rest. Abnormal signs were few, and the peripheral circulation was normal.

RESULTS

Canal measurement for the 15- to 21-year-old miner and nurses are recorded in Figure 1. The spinal canal in children is relatively large (Figure 2). In infancy the mean oblique diameter is 1.38 cm, which steadily increases to 1.58 cm at puberty (Figures 3 and 4). The measurements in the three older groups of miners, sedentary workers, and nurses between the ages of 50 and 65, are more than 1 mm less than the young adults, the sedentary workers having narrower spines than the miners (Figure 5).

There were 154 patients with disabling disc symptoms. Their average age was 37 years, and 66% were male (Table 1). Their mean measurements are compared with the tenth percentile for young people in Figure 6. Fifty-six percent are below the tenth percentile at L5 (Figure 7).

Measurements from the 78 patients who had root en-

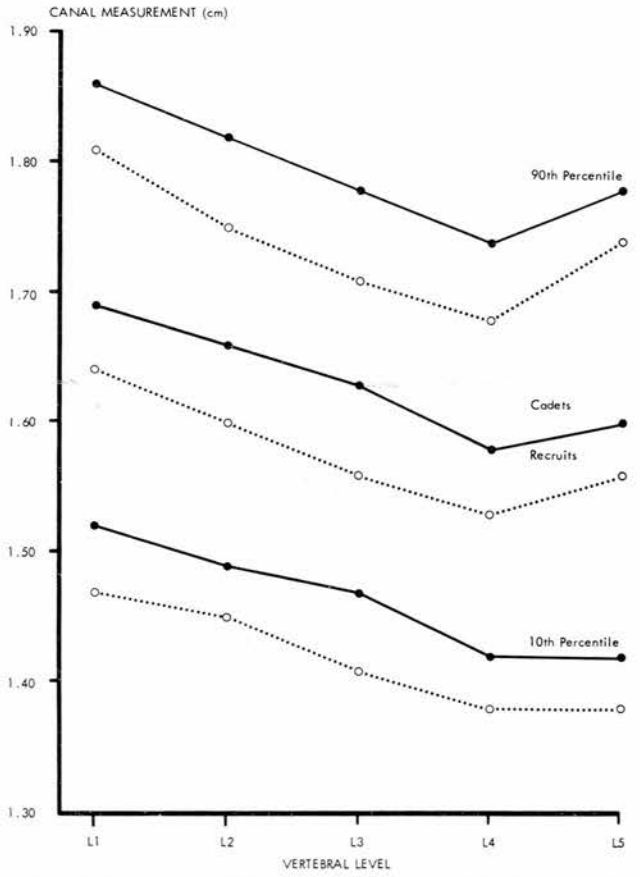


Fig 1. Fifteen-degree oblique sagittal diameter of the lumbar spinal canal, mean and percentiles, for 188 male mining recruits and 102 female nurses between 15 and 21 years of age.

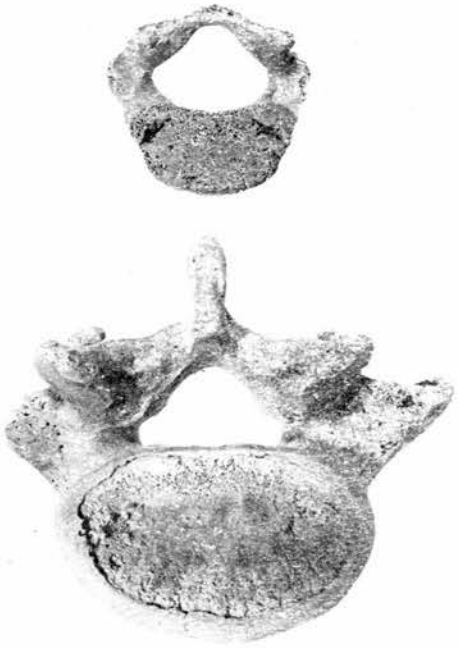


Fig 2. Photograph comparing canal size of the fifth lumbar vertebra of a 3-year-old child and that of an adult.

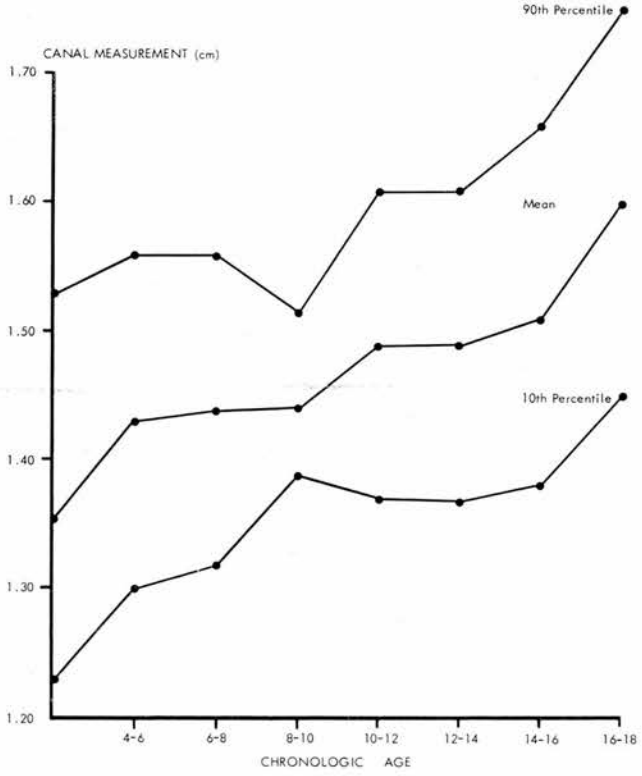


Fig 3. Mean value of canal diameter at L5 in 150 children.

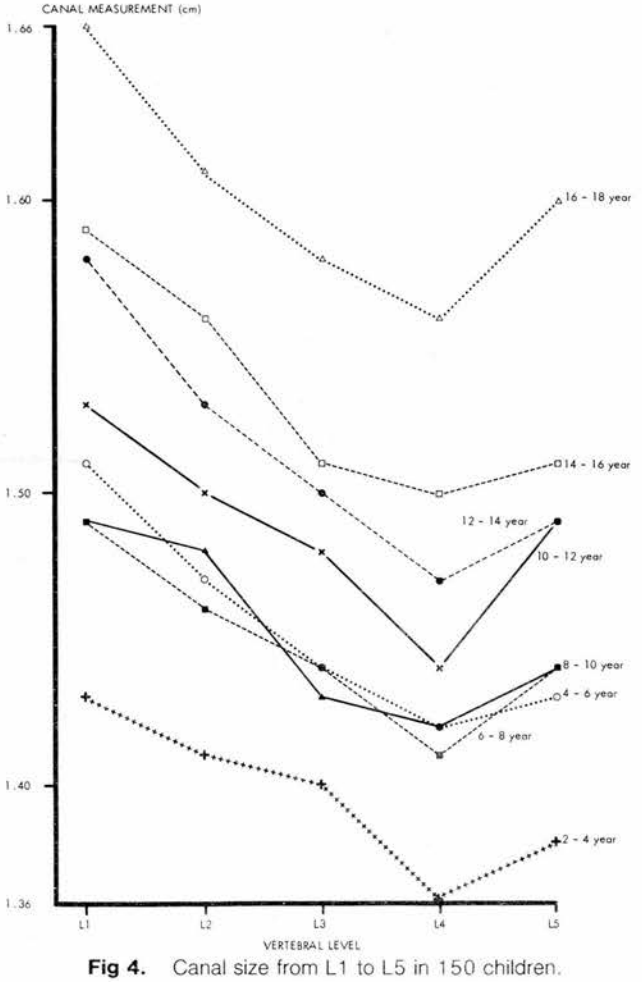


Fig 4. Canal size from L1 to L5 in 150 children.

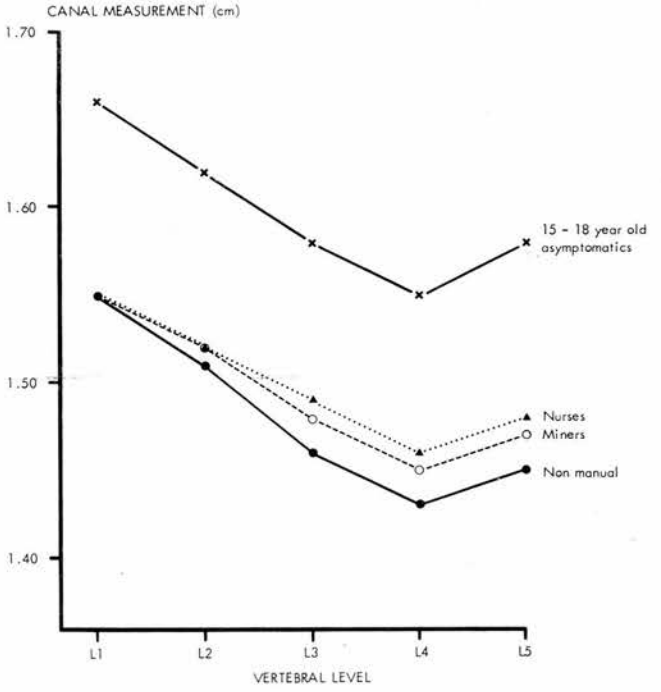


Fig 5. Mean canal size for three groups between the ages of 50 and 65 years (miners, male sedentary workers, and nurses), compared with 15- to 21-year-old subjects.

trapment syndrome and an average age of 56 years are compared with the mean and tenth percentile of the older subjects (Figures 8 and 9, Table 1). Only 30% are below the tenth percentile.

One hundred thirty-eight patients who had a history of neurogenic claudication had an average age of 49 years. Seventy percent had measurements below the tenth percentile for the 50- to 65-year-old subjects (Figures 10 and 11, Table 1).

Measurements from the two groups of clinic patients, unselected apart from the fact that they complained of back pain, show that 50% of those between 15 and 30 years old are below the tenth percentile, while 45% of those between 50 and 65 years old are below the tenth percentile for their own age groups (Table 1). The mean measurement for these two groups of patients is similar. Comparisons are made between the two clinic groups and the percentiles at L5 for randomly selected subjects of the same age (Figure 12).

DISCUSSION

We have previously recorded canal measurements for young nurses and miners. In this larger series the figures remain the same. The canal of women was found to be wider than that of men in the young adults and also in the 52 older nurses compared with the 280 men of the same age. The difference is very small but is statistically significant in the young adults ($P < 0.05$). A larger spinal canal would be more advantageous during pregnancy when the spine is subjected to altered mechanical forces and increased ligamentous laxity. The canal of

Table 1. Patients With Low-Back Pain Whose Spinal Canals Were Measured by Diagnostic Ultrasound

Pathologic condition	*Number of patients*	*Mean age (yr)*	*Male (%)*	*Below 10th percentile (%)*	*Age category*
Disc lesion	154	36.7	65.6	56	Young adults
Root entrapment	78	56.2	51.3	30	Older adults
Neurogenic claudication	138	48.8	73.2	70	Older adults
Clinic patients aged 15–30 yr	165	24.5	60.0	50	Young adults
Clinic patients aged 50–65 yr	242	56.0	59.3	45	Older adults

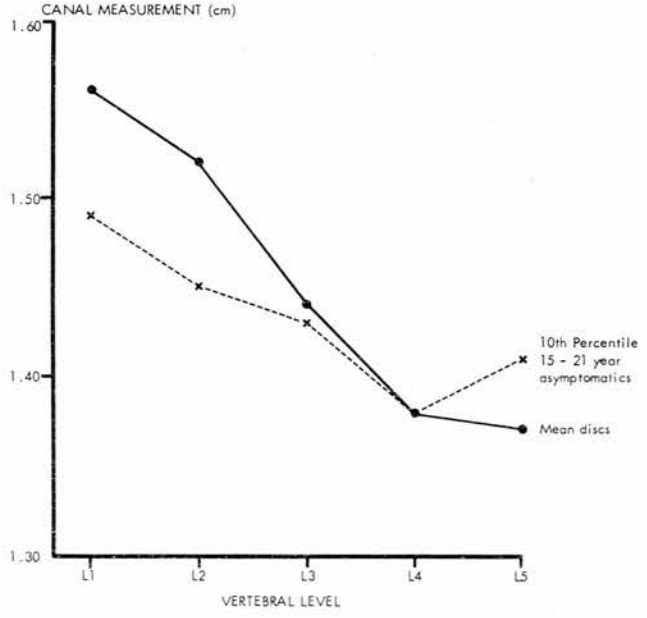


Fig 6. Mean measurements for 154 patients with disc symptoms, compared with tenth percentile of 15- to 21-year-old patients.

women may contain more extradural fat than that of men.

The canal is relatively large in infancy. Our measurements are probably artificially high in the very young children, because the thin neural arch increases the lamina "window" through which ultrasound enters the spinal canal. It may reflect the midsagittal rather than the 15° oblique diameter, because both laminae and the apex of the neural arch are included in the "window." A midline scan is possible in the infant, while the thickened spinous process absorbs the ultrasound in the older child and adult. The failure of measurements to increase in the first decade (Figure 3) is therefore probably more apparent than real.

The pattern of measurements from L1 to L5 remains the same throughout growth, with the canal decreasing in size from L1 to L4 and increasing again at L5 (Figure 4).

The relative position of the cord rising within the growing spine does not appear to affect the canal size.

Measurements from the miners, sedentary workers, and nurses over the age of 50 years shows that the 15° oblique diameter reduces with age. It is possible that the lamina "window" through which ultrasound enters the

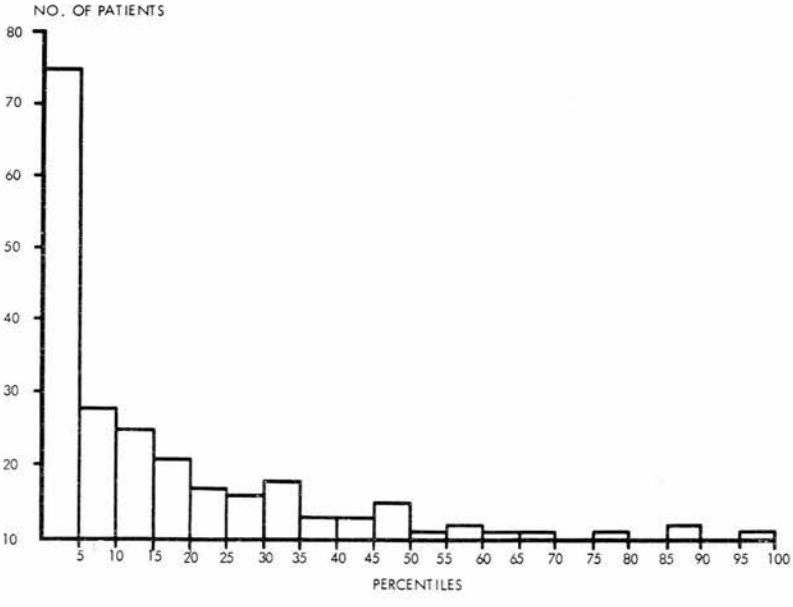


Fig 7. Histogram of canal size at L5 for 154 patients with disc symptoms, compared with percentiles for 15- to 21-year-old subjects.

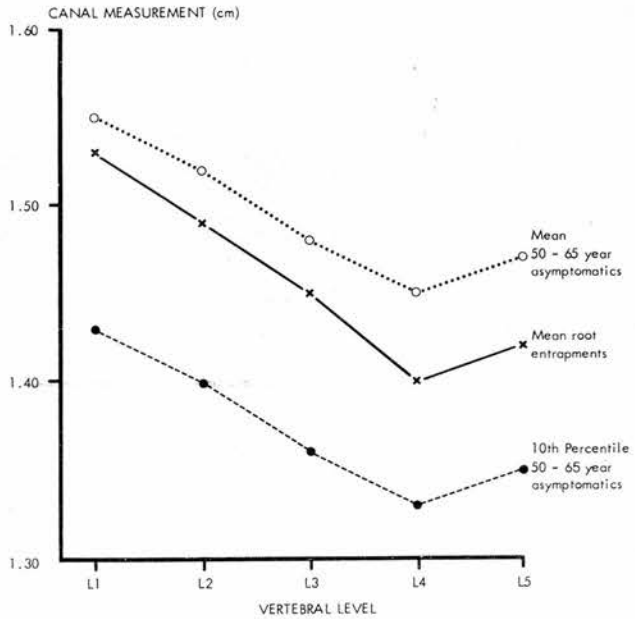


Fig 8. Mean measurements for 78 patients with root entrapment syndrome, compared with mean and tenth percentile of 50- to 65-year-old patients.

canal becomes smaller with age as a result of bony hypertrophy. The second and third echoes which are reflected through the window are certainly most clearly visualized in the young adult. Decrease in the size of the window, however, would not explain a reduction in canal measurement. If the window decreases, it is probably a uniform reduction around the periphery of the window which would not affect the measurement at all. Subperiosteal ossification at the attachment of the ligamentum flavum could explain this age-related narrowing.

The size of the canal does not appear to be related to occupation. Men who have worked for three and four decades underground, some of whom were involved in very heavy work in confined spaces in early adult life prior to mechanization of the coal industry, have canals slightly wider than men who have been in sedentary occupations. It is possible that the miners have selected themselves out of the industry and that those with troublesome narrow spines have taken other employment. If demanding work significantly affected the diameter of the spinal canal, however, it would probably have been demonstrated in this study.

The failure of occupation to affect the size of the canal suggests that the narrow canal should be considered to be developmental rather than degenerative. Degenerative change might produce localized encroachment, but would not affect the general shape and size of the canal.

Ultrasound measurements have shown that the majority of patients who have disabling disc symptoms have a narrow canal in addition to disc pathology. Fifty-six percent of 154 patients with disc symptoms were below the tenth percentile for the young people (Table 1). The figures are similar when considering all the patients between 15 and 30 years of age attending a hospital clinic with back pain, suggesting that many of these may have had a disc lesion compromising a narrow canal and yet have avoided serious root signs.

The pathologic condition responsible for root entrap-

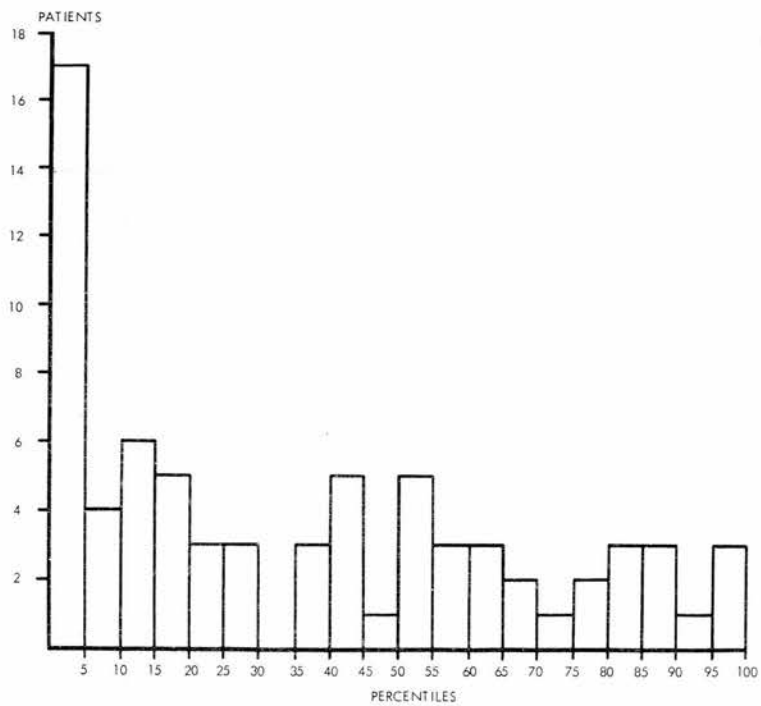


Fig 9. Histogram of canal size at L5 for 78 patients with root entrapment syndrome, compared with percentiles for 50- to 65-year-old patients.

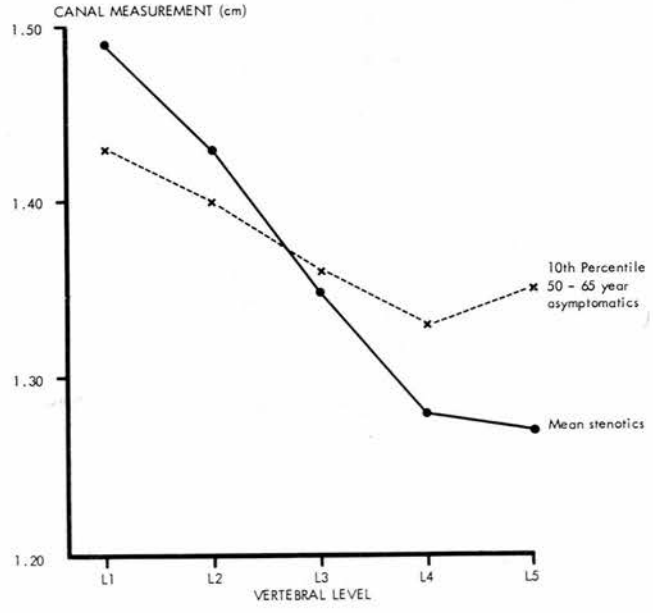


Fig 10. Mean measurements for 138 patients with neurogenic claudication, compared with tenth percentile for 50- to 65-year-old patients.

ment syndrome is probably within the root canal.[5] The central canal only is measured by ultrasound, but 30% of 78 patients diagnosed as having root entrapment had canals below the tenth percentile of the 50- to 65-year-old miners, nurses, and sedentary workers. A subject whose central canal measurement is below the tenth percentile is four times more likely than the rest of the population to have root entrapment symptoms. This increases to 11 times in the presence of a disc lesion. It is probable that a pathologic condition in the root canal is more likely to cause symptoms if the central canal is also narrow, though many patients with root entrapment do have adequate central canal measurement.

The relative importance of soft tissue and bony narrowing in the causation of neurogenic claudication has been in dispute. Measurements suggest that the size of the central canal is highly significant in the majority of patients who have claudication. Seventy percent are below the tenth percentile (Table 1). Not all subjects who have a narrow canal develop claudication, however. It is probable that soft-tissue thickening, bony encroachment, and "instability" are the added factors responsible for symptoms.

Almost half (46%) of the 138 patients who had claudication were miners. There is no evidence to suggest that the central canal is narrowed by heavy underground work, but additional factors such as previous disc pathology, vertebral bars associated with degenerative change, facet joint degeneration, thickened ligaments, and "instability" probably compromise the canal that is already narrow.

If the size of the central spinal canal is an important factor responsible for compression of the cauda equina, the shape of the canal must also be significant. The nerve root will be particularly vulnerable to compression when the canal is trefoil. There is a probability that the canal is trefoil at L5 if the ultrasound measurement at L5 is equal to or less than that at L4. It is significant that the mean values and percentiles are greater at L5 than at L4 in the children, young miners, and nurses and in the older subjects. This pattern is not observed in the patients who have disc lesion or stenosis. It suggests that the trefoil shape, in addition to the canal diameter, is symptomatically important.

Ultrasound measurements indicate that the available space in the central spinal canal is a factor, but not the only factor, in the causation of many types of disabling back pain. The canal size is highly significant in the presence of disc pathology and in a patient who has neurogenic claudication, and in most young patients who attend a hospital clinic with back pain. It is a relevant factor for many older patients attending hospital with back pain. It is less significant in patients suffering from root entrapment syndrome.

The development of many of the structural types of severe back pain is probably explained by the following diagram:

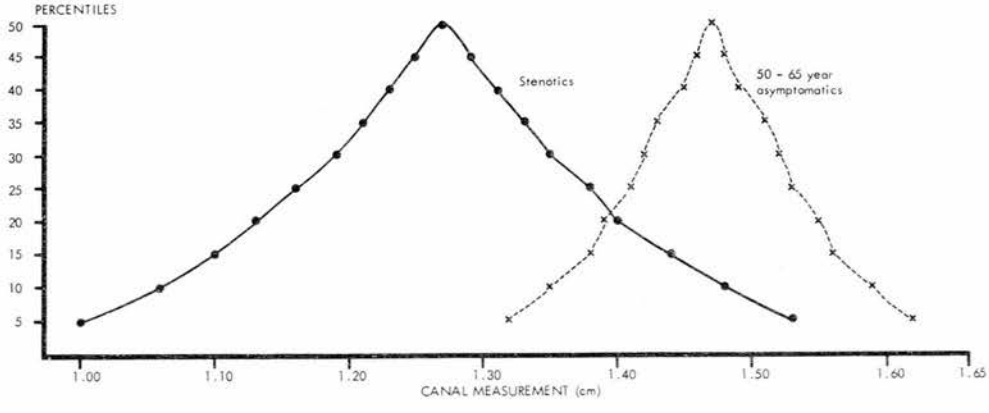


Fig 11. Comparison of canal measurement percentiles at L5 between 138 patients with neurogenic claudication and the 50- to 65-year-old subjects.

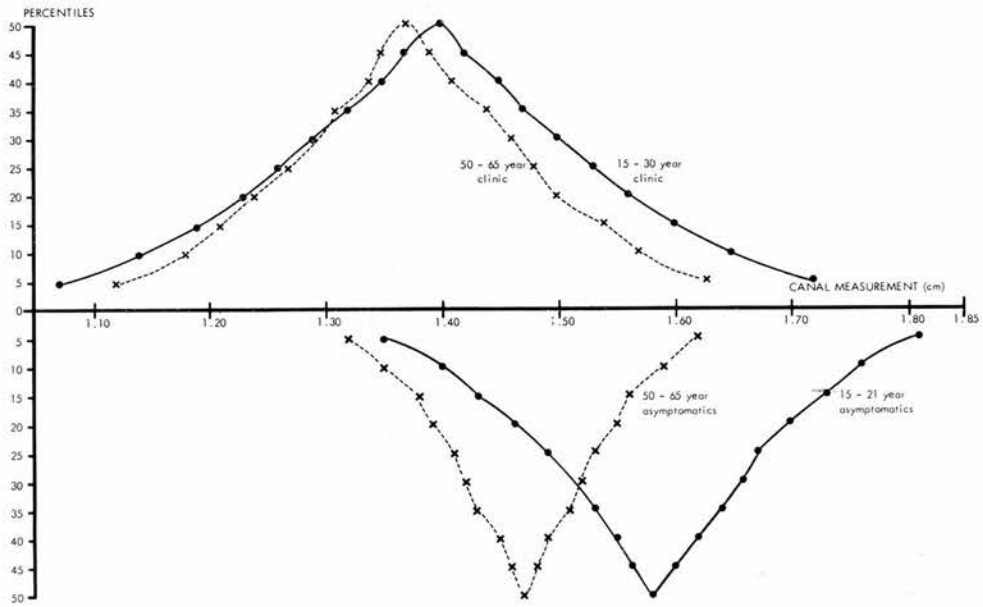
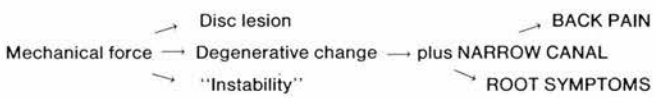


Fig 12. Comparisons of canal measurement percentiles at L5 between two clinic groups with back pain and randomly selected subjects of the same age.



Some of the mechanical forces responsible for pathologic conditions of the spine have been demonstrated by Nachemson and Elfstrom[6] and Davis and Stubbs.[1] They are theoretically preventable. Advice should be offered to that section of the population at greatest risk.

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Address reprint requests to

R. W. Porter, FRCS
Doncaster Royal Infirmary
Doncaster DN2 5LT
England

Accepted for publication June 18, 1979.

C122/80

THE SHAPE AND THE SIZE OF THE LUMBAR SPINAL CANAL

R. W. PORTER, FRCS, FRCSE, C. HIBBERT, BSc, P. WELLMAN, BSc, C. LANGTON, BSc and A. MILLINGTON, BTech
Doncaster Royal Infirmary, Doncaster

SYNOPSIS This study records the normal anatomy of the lumbar spinal canal and its variations within three skeletal populations, and compares the results with ultrasound measurements of the spinal canal from a group of South Yorkshire volunteers. By comparing the skeletal and ultrasound measurements, a method of suspecting the trefoil shape is suggested. If this is correct, many patients with disc symptoms and patients with neurogenic claudication have not only narrow but trefoil canals.

INTRODUCTION

1. The relationship between back pain and the available space for the cauda equina within the spinal canal has attracted increasing attention in recent years. (Refs. 1, 2, 3, 4, 5). We have used a photographic method to measure the dimensions of the central spinal canal in three skeletal populations, and have attempted to correlate the results with ultrasound canal measurements in vivo from nearly fifteen hundred subjects and patients from two pathological groups.

2. Canal Measurement from Skeletons

The lumbar vertebrae were examined from three populations.

a) A Romo-British population, fourth century A.D. from Poundbury, Dorset, loaned by the British Museum. It included vertebrae from 119 adult spines, 77 being complete, and also 22 children's spines.

b) A collection of spines from Eccles, belonging to the sub-Roman and Anglo-Saxon peoples of Kent, dating from the mid centuries of the first millenium A.D., loaned by Bradford University. There were vertebrae from 61 adult spines, 31 being complete and in addition, 5 children's spines.

c) A twentieth century collection loaned from Edinburgh University, mainly of Indian origin, including 60 adult spines, 57 being complete.

3. A photographic box was designed (Fig. 1) to produce a silhouette photograph of the spinal canal with an unmagnified image. The light source was a photographic flash gun. A pin hole was necessary for a sharp light source. The light was passed through a condenser to obtain parallel light. The vertebra being examined was positioned on a glass plate, the angle of which could be manually tilted through its mounting on a universal joint. The construction of the box permitted the examiner to observe the position of the vertebra by using an eye-piece and a mirror. The vertebra could be moved on the glass plate until the centre of the canal was superimposed on a point reflected from the base-plate through the canal and mirror. When in the correct position, the plate was tilted until the cranial and caudal edge of the posterior surfaces of the vertebral body were superimposed. It was considered that the maximum amount of light would then pass through the spinal canal. The hinged mirror was lifted to the back of the box, a film placed on the baseplate, and the flash gun operated by a foot switch. A silhouette photograph was obtained, (Fig. 2).

4. The parameters measured are shown in Fig.3, interpedicular, mid-sagittal and fifteen degree oblique sagittal diameters, the canal area, and an estimate of the degree of 'trefoilness' from the ratio of two transverse diameters.

5. The results of the measurements from the three populations are shown in Figs. 4, 5, 6, 7 and 8. The mean mid-sagittal diameter, the fifteen degree oblique diameter and the area decreased from L.1 to L.4, increasing again at L.5. The interpedicular diameter and the degree of 'trefoilness' increased from L.1 to L.5. The same general trends were identical in each population, the mean and standard deviation being similar. The Poundbury and Eccles collections showed a closer correlation than the Edinburgh spines. The two archaeological collections had a greater comparative homogeneity than the Edinburgh spines. It suggests the possibility of racial variations in the anatomy of the spinal canal which could be of significance when considering the epidemiology of back pain.

6. The trefoil canal places the neural elements at risk. The incidence of 'trefoilness', its identification in a patient, and factors which might be responsible for its development, are therefore of interest. The degree of 'trefoilness' at L.5 and its range is almost identical for the three skeletal populations, (Fig. 8). There is a correlation between trefoilness at L.5 and at L.4 but this is not shown at other vertebral levels, (Table 1).

7. Other anatomical features of the lumbar vertebrae were compared with the degree of 'trefoilness' to determine if the trefoil shape could be suspected at x-ray, and if factors responsible for its development could be identified. Right and left facet angle were recorded. The pedicle height and interfacet distance between the lateral aspect of the interior facet were expressed as a product. The vertebral angle at L.5 was measured as a ratio between the posterior and anterior vertical body depth. Osteophytes were measured at the maximum osteophyte projection on the superior and inferior margins of the vertebral bodies. These were correlated with the degree of 'trefoilness' (Table 2). There was a correlation of .50 ($P < .05$) with the vertebral angle at L.5. Many spines were markedly trefoil with a low vertebral angle, however, and many with an acute angle were not trefoil. Thus it cannot be assumed from an x-ray that a spine with an acute lumbo-sacral angle has a trefoil canal. The correlation does suggest, however, that a factor in the development of trefoilness is an acute lumbo-sacral angle. If a pliable triangular tube is gradually bent, it develops a trefoil shape, (Fig. 9).

8. Baddeley noted a relationship between the trefoil shape and a vertebra where the pedicle height was small and the facet joints close together (Ref. 6). In this series there was a correlation of .35 ($P < .05$), insufficient to be able to predict the canal shape from their features on x-ray.

9. There is a lack of information in the literature on the anatomy of the spinal canal in children. It is of interest that the trefoil shape was absent in the spines of 7 children under the age of 10 years and only minimal between 10 years and puberty (Fig.10). There may be a relationship between the development of 'trefoilness' and the development of the secondary curve of lumbar lordosis.

10. Canal Measurement in Vivo by Ultrasound

The fifteen degree oblique sagittal diameter of the lumbar spine was measured by ultrasound in 1479 subjects from a South Yorkshire population. This included 256 children, 301 young people between 16 and 18 years old, 462 adults between 20 and 50 years, and 460 between 50 and 65 years. The children's measurements are shown in Fig. 11, and the size of the adult spine related to age in Fig. 12. The results are slightly greater than those obtained from the skeletons. It is recognised however that the skeletal silhouette measurement is a minimum measurement, whilst the site of canal measurement by ultrasound is not necessarily the smallest measurement. The pattern of measurement by ultrasound, however, is the same as the skeletal fifteen degree sagittal measurement.

11. The Trefoil Canal

The pattern of measurement of the fifteen degree oblique sagittal diameter was examined for those skeletons with the most marked degree of 'trefoilness' (less than 57% or 15% of the series). The reduction in measurement from L.1 to L.4 was significantly more marked than the mean reduction, and in addition, L.5 tended to be equal or less than L.4 (Fig. 13). The trefoil shape will be reflected in the reduction of the oblique diameter provided that the mid sagittal diameter does not increase disproportionately at L.5. This suggests that the trefoil shape may be suspected by in vivo ultrasound measurements when there is a marked reduction in canal size from L.1 to L.4 and when L.5 is equal or less than L.4.

12. The pattern of measurement for two pathological groups of patients was examined and compared with the pattern for adult subjects. There were 151 patients with disc symptoms and 138 patients with neurogenic claudication. Their pattern of measurement (Fig. 14) shows a steep inclination from L.1 to L.4 and a further drop at L.5. It is probable that many of these patients have canals not only of small size but also of trefoil shape.

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ACKNOWLEDGEMENTS

We would like to thank Miss Rosemary Powers and the British Museum for the loan of the Poundbury collection, Dr. K. Manchester of Bradford University for permission to measure the Eccles skeletons, and Professor Romanes of Edinburgh University for the loan of the Edinburgh collection.
Mr. Garry Swann designed and constructed the photographic box and provided the figures.
We would like to thank Mrs. Jean Reynolds for her untiring secretarial assistance.

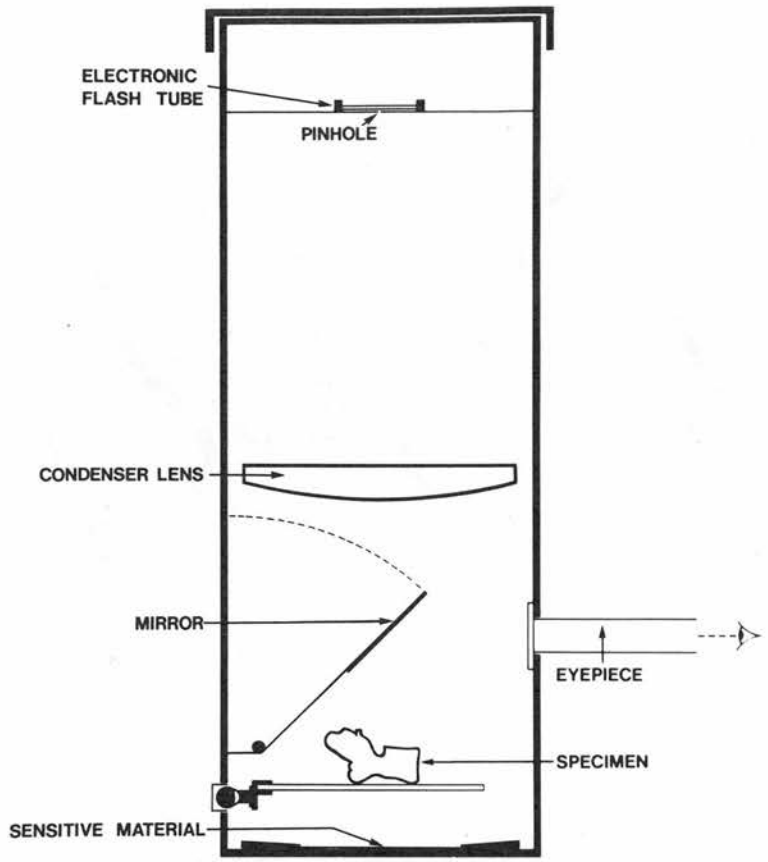

Fig. 1 Photographic Box

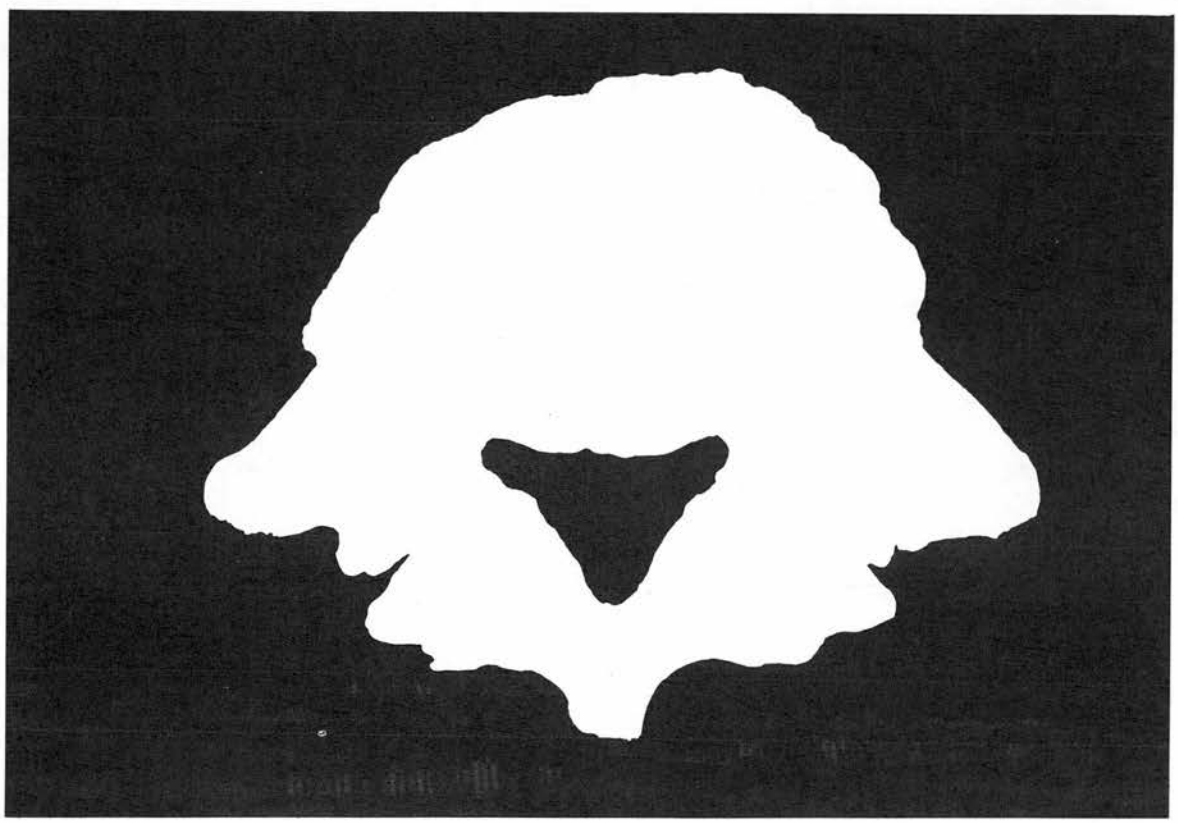

Fig. 2 Silhouette photograph of L.5 vertebra

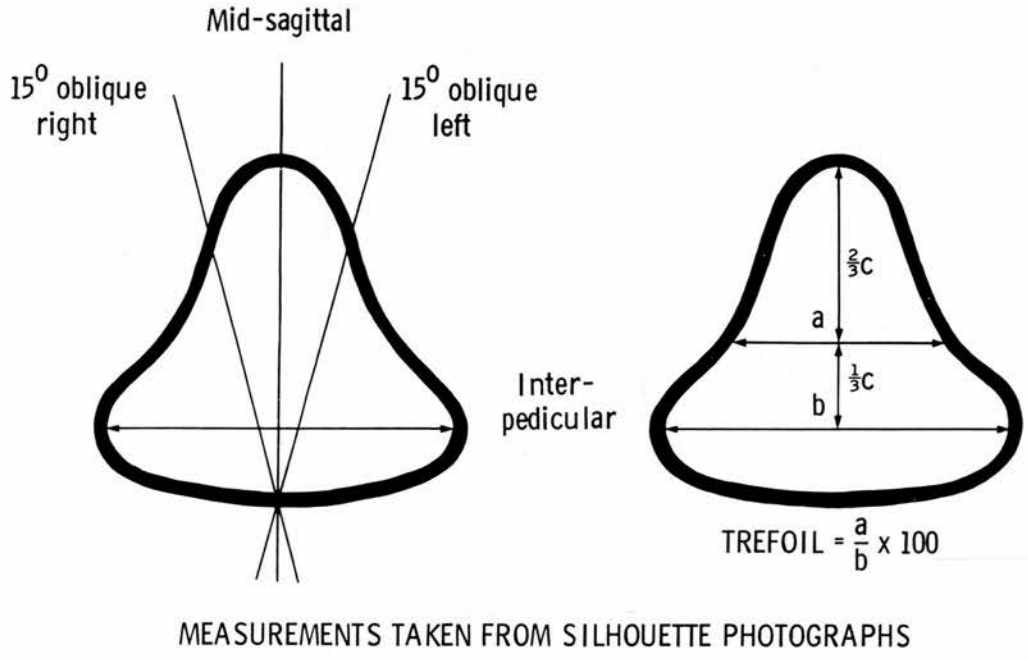

Fig. 3 Canal Parameters measured

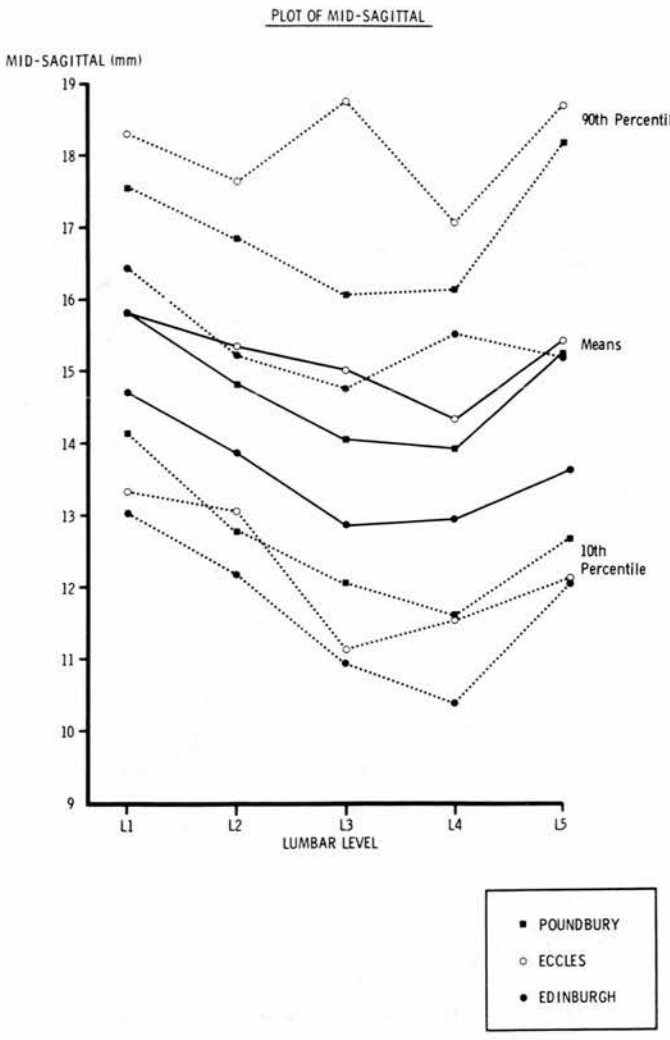

Fig. 4 Mid Sagittal Diameter of Canals, mean and percentiles

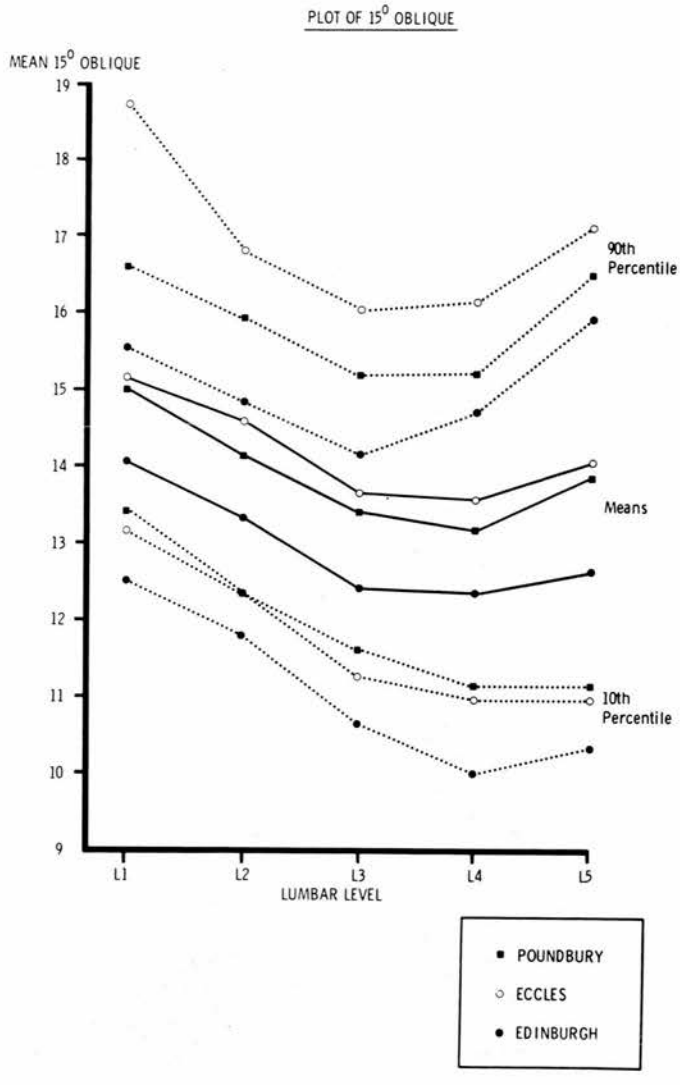

Fig. 5 15° oblique diameter of Canals, mean and percentiles

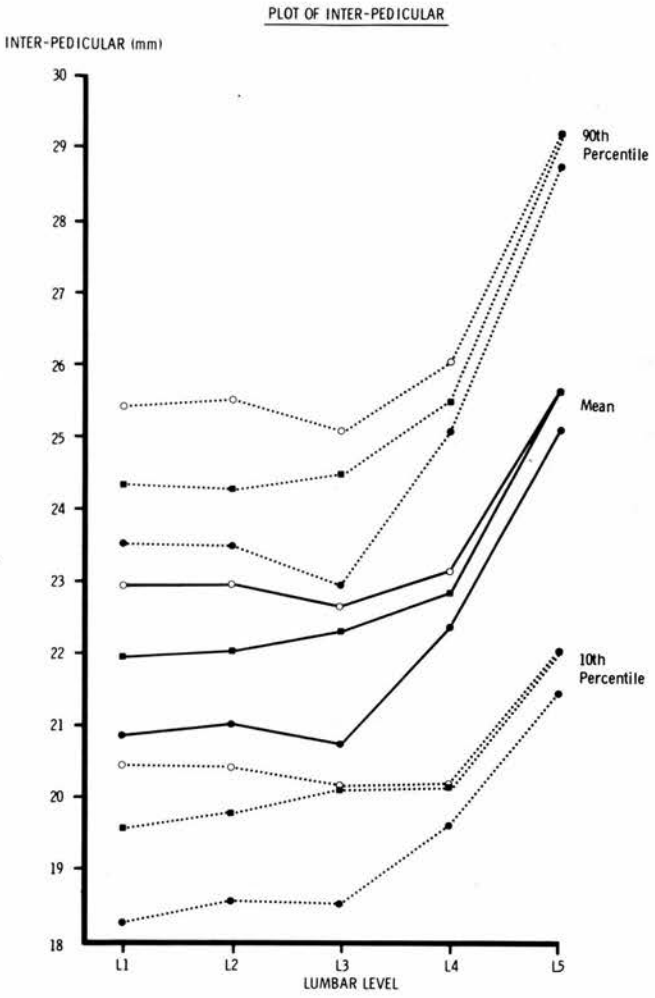

Fig. 6 Inter-pedicular diameter of canals, mean and percentiles

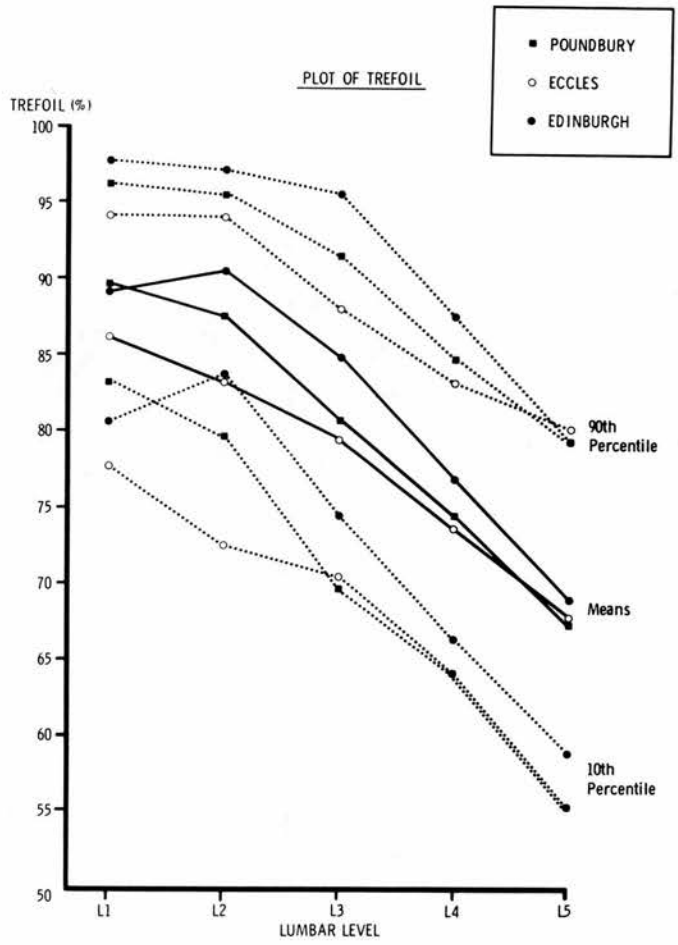

Fig. 7 Degree of "Trefoilness" of canals, mean and percentiles

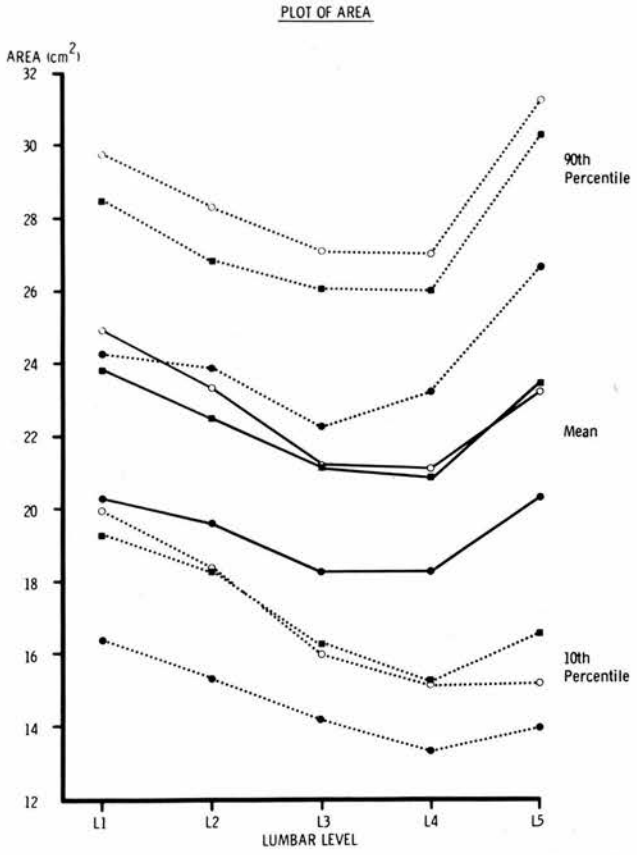

Fig. 8 Area of Canals, mean and percentiles

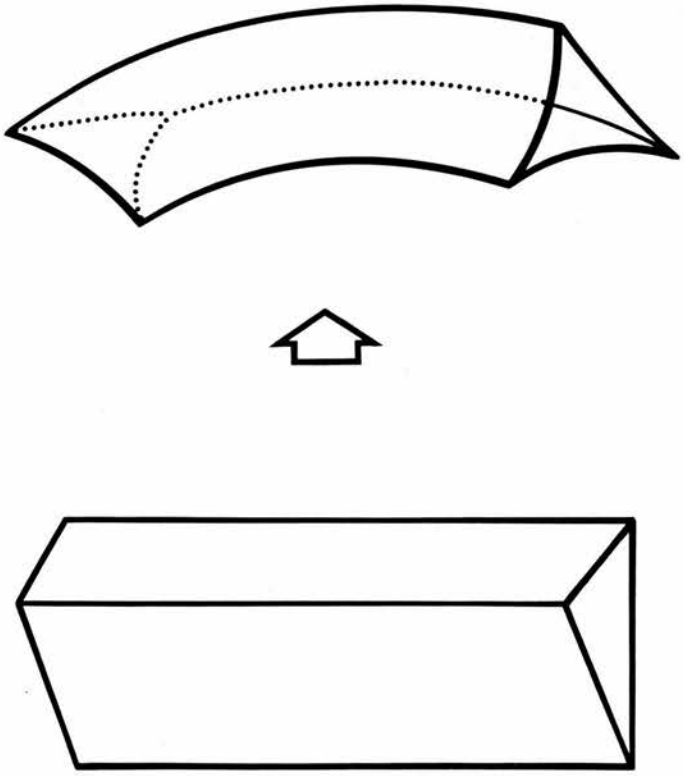

Fig. 9 Bending of triangular tube produces trefoil shape

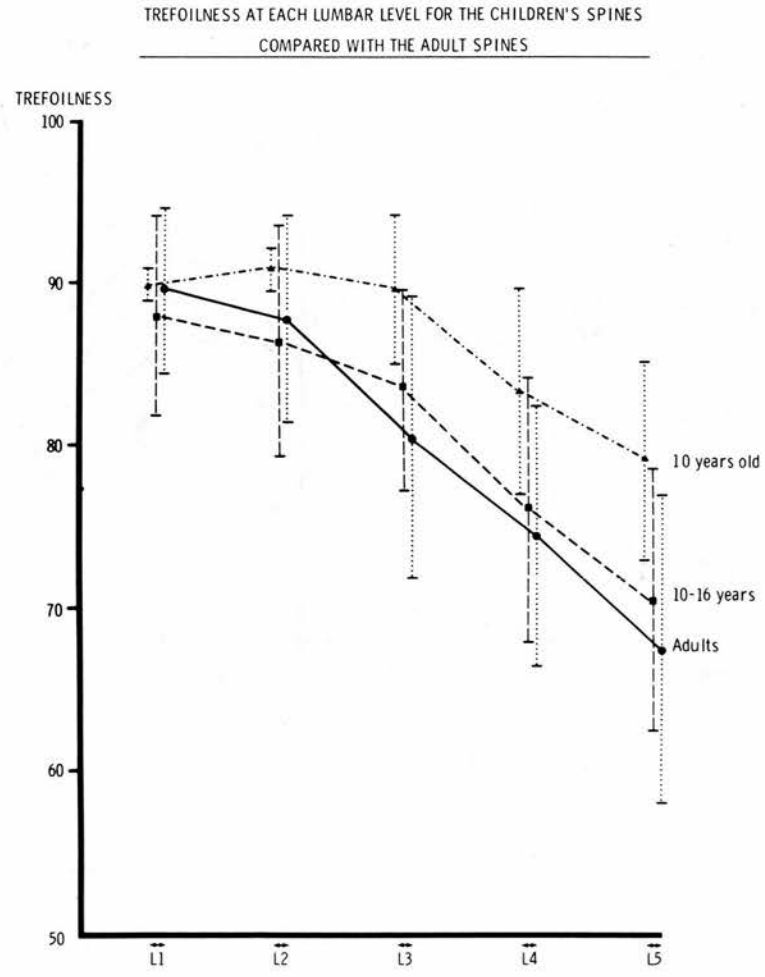

Fig. 10 Degree of "Trefoilness" of children's canals compared with Adults

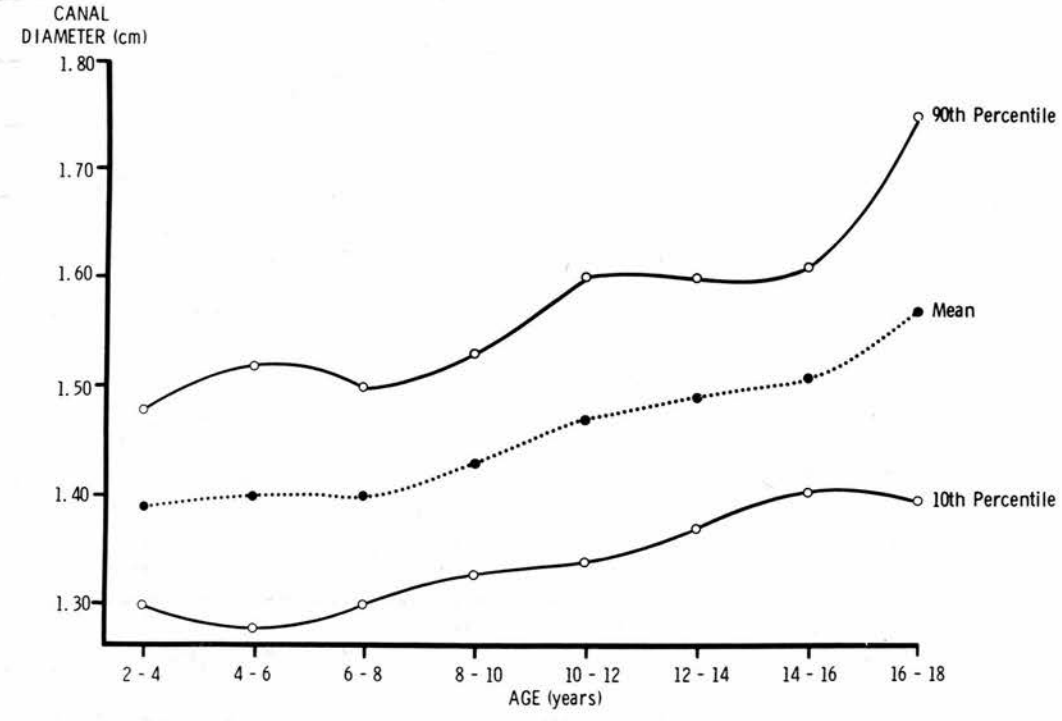

Fig. 11 Ultrasound measurements of children's canals, mean and percentiles, related to age

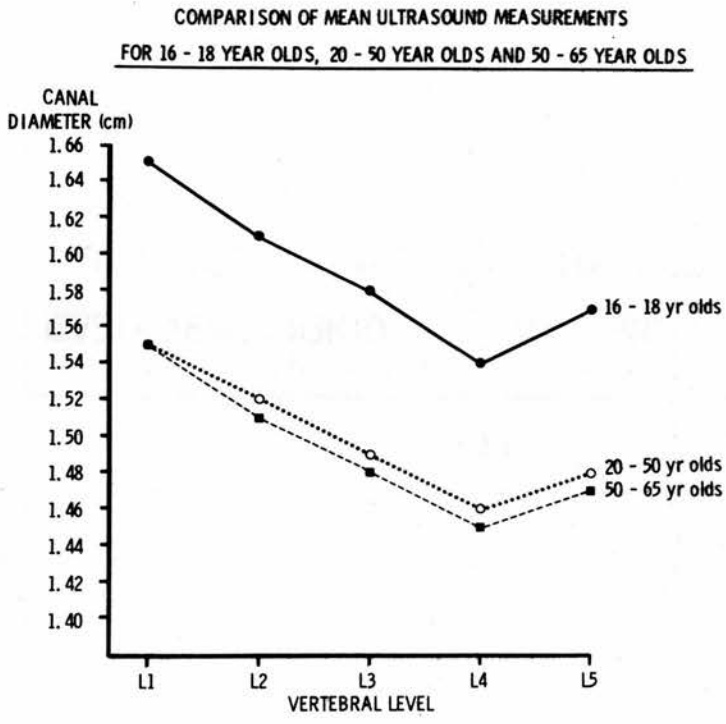

Fig. 12 Mean Ultrasound Measurements of Lumbar Canals in Adults related to age.

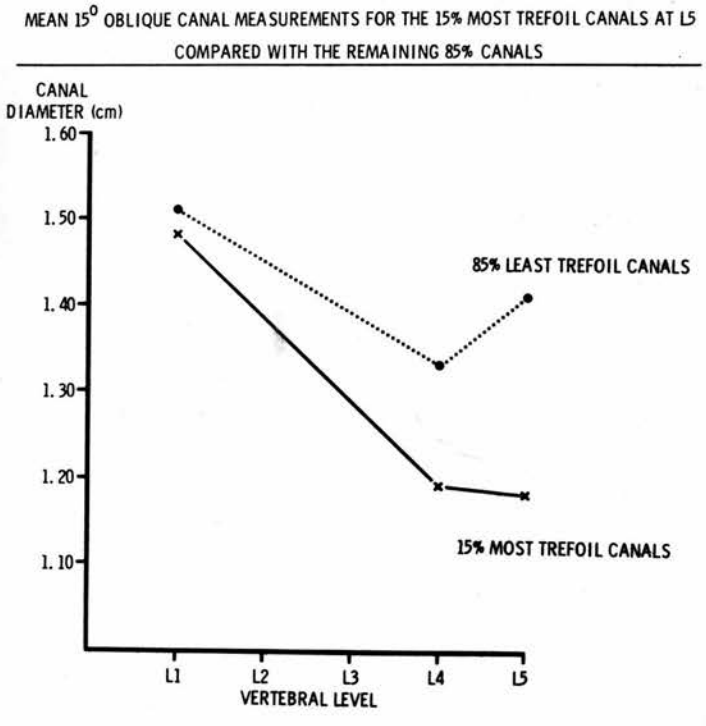

Fig. 13 Mean 15° oblique measurement of skeletal canals comparing the most trefoil canals with the least trefoil

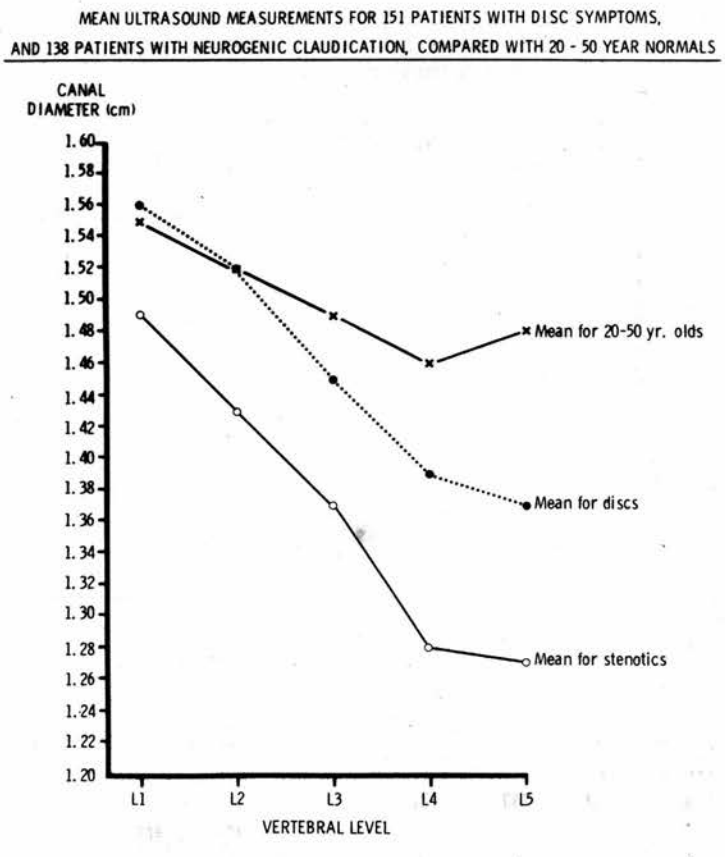

Fig. 14 Mean Canal Measurements by ultrasound for patients with disc lesion, neurogenic claudication, compared with asymptomatic subjects

Table 1 Correlation between "trefoilness" at L.5 and "trefoilness" at other lumbar levels

CORRELATION BETWEEN TREFOILNESS AT L5 AND TREFOILNESS AT OTHER LUMBAR LEVELS

	L1	L2	L3	L4
CORRELATION COEFFICIENT	-.147	.047	.070	.381

Table 2 Correlation between "trefoilness" and other vertebral measurements

CORRELATION COEFFICIENT BETWEEN:-

	FACET ANGLE	PEDICLE HEIGHT x FACETAL DISTANCE	WEDGING AT L5	INFERIOR DEGENERATIVE CHANGE	SUPERIOR DEGENERATIVE CHANGE
"TREFOILNESS"	0.04	0.35	0.50	0.02 ~~0.30~~	0.04

1981, *British Journal of Radiology*, *54*, 905–907 OCTOBER 1981

Technical notes

5

Measurement of the lumbar spinal canal by diagnostic ultrasound

By Catherine S. Hibbert, Carole Delaygue, Beatrice McGlen and R. W. Porter

Doncaster Royal Infirmary

(*Received March 1981 and in revised form May 1981*)

We have previously reported a method of measuring the spinal canal by ultrasound, and have commented on its clinical significance (Porter *et al.*, 1978). This paper describes in more detail the technique of measurement and its repeatability.

Method of Measurement

We have used an EMI Diasonograph to measure the spinal canal of over 6000 subjects. The fixed gantry has an advantage in the ease and accuracy of the technique. It is our practice to use a 1.5 MHz transducer, but measurements can be obtained with a 2.5 MHz transducer though with greater attenuation of sound.

The gantry is inclined 15° to the vertical plane, and a B-scan display of the five lumbar vertebrae obtained by scanning the lumbar region a finger's-breadth lateral to the spinous processes. Although Stockdale and Finlay (1980) state that "the identification of the images on the B-scan together with the location of the corresponding echoes on the A-scan is by no means an easy task", we have experienced little difficulty in obtaining a satisfactory B-scan. It may take one or two attempts before the transducer is correctly positioned lateral to the spinous processes, but this is usually achieved without difficulty, and a satisfactory B-scan produced (Fig. 1). The five lumbar vertebrae may not always be displayed together in the same plane if there is a lumbar scoliosis, but this is overcome by rotating the gantry.

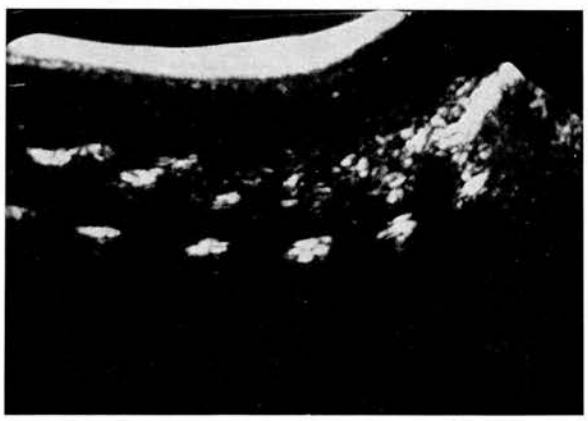

Fig. 1.
B-scan display of lumbar spinal canal.

Each vertebra is first identified on the B-scan before measurements are obtained from the A-scan. Selecting each vertabra in turn, the calipers are first positioned on the B-scan over the echoes believed to be reflected from the canal boundary. The anterior surface of the canal is easily recognized from the distinct echoes of the posterior surface of the vertebral body. It requires more care to position the caliper over the echo from the canal's posterior boundary. It is placed towards the cranial aspect of the laminar echoes. Having positioned the calipers as accurately as possible on the B-scan, actual measurement of the canal is made from the A-scan. In practice, only fine adjustment of the calipers is required for them to be at the apex of the A-scan echoes which are believed to be reflected from the canal's boundary. Large movement of the calipers should now be avoided without referring back to the B-scan to avoid incorrect selection of the A-scan echoes.

The echo from the canal's anterior margin is readily recognized on the A-scan, being the only echo of such large amplitude at this depth. The echo from the posterior canal boundary however is less distinct, being part of a composite echo pattern of reflections from the cranial edge of the lamina where the posterior and anterior surfaces converge (Fig. 2). An echo is sought on the downward slope of this echo complex (Fig. 3).

The transducer is moved slightly in order to maximize the amplitude of the echoes of interest on the A-scan, and when this is achieved, the calipers are adjusted finely to coincide with the apex of these two echoes from the canal boundary. A glance at the B-scan should show the calipers positioned at the boundary of the canal.

We appreciate that correct identification of the echo from the canal's posterior boundary is not easy, and that accuracy of measurement will depend on the ability to display and recognize this echo.

Repeatability

The intraobserver error was measured from 22 patients by the examiner first measuring the five

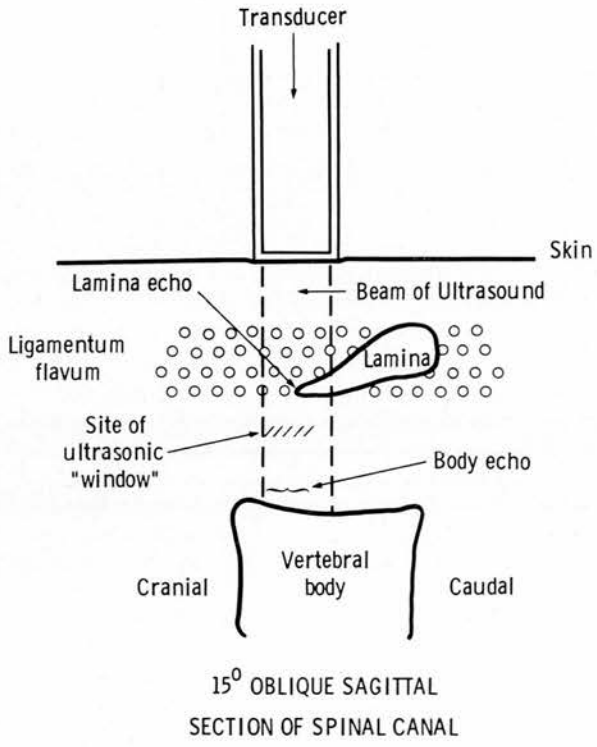

FIG. 2.

Probable origin of echoes from the spinal canal: 15° oblique sagittal section.

lumbar vertebrae as previously described. This was repeated but with another observer recording the measurements from a shielded digital display.

The interobserver error was measured from 100 patients. One examiner measured the five lumbar vertebrae and altered the digital display, and a second examiner who had been out of the room proceeded to measure the canal.

The interobserver error was also recorded between an experienced examiner and a non-medical trainee to determine how long it would take a person new both to the technique and to the concepts of ultrasound to develop a measure of repeatability comparable with that of an experienced examiner.

RESULTS

Table I records the mean and standard deviation of the absolute differences between the two measurements obtained by one examiner. The interobserver error calculated from the absolute difference of the two measurements obtained by two examiners is shown in Table II with a mean error of less than half a millimetre. Table III records the mean interobserver error between an experienced examiner and

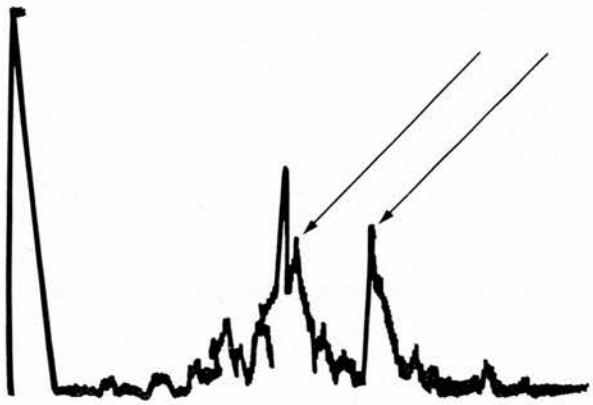

FIG. 3.

A-scan display showing the two echoes to be measured.

TABLE I

MEAN INTRAOBSERVER ERROR FOR 22 PATIENTS

	Mean (cm)	Standard deviation
L1	0.045	0.031
L2	0.044	0.030
L3	0.038	0.030
L4	0.040	0.021
L5	0.041	0.019

TABLE II

MEAN INTEROBSERVER ERROR FOR 100 PATIENTS

	Mean (cm)	Standard deviation
L1	0.038	0.028
L2	0.041	0.030
L3	0.036	0.030
L4	0.037	0.032
L5	0.046	0.037

TABLE III

MEAN INTEROBSERVER ERROR BETWEEN A TRAINEE AND AN EXPERIENCED EXAMINER

	Mean (cm)	Standard deviation
2nd and 3rd week: $n=34$		
L1	0.042	0.037
L2	0.041	0.032
L3	0.052	0.042
L4	0.048	0.040
L5	0.066	0.054
4th and 5th week: $n=36$		
L1	0.038	0.037
L2	0.043	0.044
L3	0.045	0.041
L4	0.050	0.040
L5	0.047	0.043

a trainee in the second and third weeks of training, and also in the fourth and fifth weeks, when a total of 70 patients had been examined.

DISCUSSION

We find a mean repeatability of less than half a millimetre encouraging when attempting to measure a space as complex as the lumbar spinal canal. It is difficult to say exactly what is being measured, but we believe it is some parameter of the central spinal canal in the 15° oblique sagittal plane at the cranial aspect of the lamina. The speed with which a non-medical examiner reached an acceptable degree of proficiency suggests that, with careful tuition, this is not a difficult technique to learn.

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Calcitonin Treatment for Neurogenic Claudication

6

R. W. PORTER, MD, FRCS, FRCSE and C. HIBBERT, PhD

Forty-one patients with a possible diagnosis of neurogenic claudication were treated with Calcitonin for four weeks. Eleven responded with considerable improvement in their walking distance. Ten agreed to enter a randomized double-blind cross-over trial, and eight made a correct assessment. It is concluded that Calcitonin is effective in relieving symptoms of neurogenic claudication for some patients. Five patients have received the drug for over one year with no serious side effects, and two have not relapsed after discontinuing the drug. Patients likely to respond will probably have symptoms affecting both legs equally pain extending below the upper calf, limiting walking to under a mile; an abnormal myelogram; and no more than one inappropriate sign. They are more likely to be men in late middle age who have been engaged in manual work. The beneficial effect of Calcitonin is probably the result of an arterial shunt mechanism, whereby a reduction in skeletal blood flow provides for a deprived cauda equina.
[Key words: calcitonin, neurogenic claudication]

THE SYMPTOMS of neurogenic claudication were first described by Verbiest in 1949.[12] All his patients were men with bilateral radicular pains, disturbance of sensation, and impairment of motor power in the legs when walking. Their symptoms and abnormal signs were relieved by rest. Myelography confirmed dural compression, which was also identified at operation. The subject has been widely reviewed by Kirkoldy-Willis et al[7]; Weinstein et al[16]; and Critchley.[1] There is no conservative method of management that has influenced the condition, and if symptoms are sufficiently severe, surgical decompression is recommended with a success rate in some hands of 85%.[10] Some patients, however, develop a recurrence of symptoms after surgery, requiring further operations.[5,11]

Douglas et al,[3] in 1981, recorded a marked clinical improvement in the paraparesis of eight patients with Paget's disease in the vertebrae when treated with Calcitonin, with a 90% favorable response in 19 additional cases. The response was frequently rapid, with the results described as good or better than those obtained by surgical decompression. Herzberg[6] and Ravichandran[9] have made similar observations.

This paper records the results of Calcitonin treatment for 41 patients with neurogenic claudication not associated with Paget's disease and the results of a randomized double-blind cross-over trial for ten patients who had responded dramatically to Calcitonin.

METHOD

Forty-one patients from 41 to 67 years of age with neurogenic claudication were treated with Calcitonin. They had bilateral leg symptoms extending from the thighs to below the knees, aggravated by walking, limiting the walking distance, and relieved by rest. A full clinical examination was carried out, noting especially the following inappropriate signs: discomfort in the lumbar region when vertical pressure was applied to the skull, discomfort when the pelvis was rotated on the lower limbs, pain when the skin over the lumbar spine was gently squeezed, widespread tenderness, straight leg raising limited to the same degree as hip flexion when the knees were flexed, and non-dermatomal sensory loss.[14] Paget's disease of the spine was excluded by radiography and scintigraphy, and the

From the Doncaster Royal Infirmary, Doncaster, England.
Submitted for publication December 10, 1982.
The authors would like to acknowledge the help of Dr. Brian Williams and Armour Pharmaceutical Limited in helping to design the trial, the National Coal Board and the Back Pain Association for financial assistance, Mr. Garry Swann for the illustrations, and Mrs. J. Reynolds for secretarial help.

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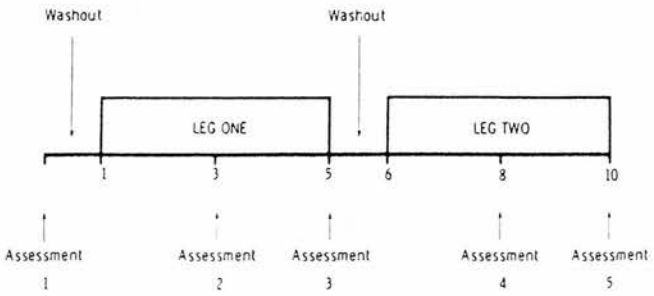

Fig 1. Plan of trial.

electrolytes were monitored. The peripheral circulation was recorded by doppler scan. Spinal stenosis was sought by Metrizamide radiculography and the canal measured by ultrasound. Prior to treatment, patients were asked to record their maximum walking distance, walking at the same time, pace, and site, three times daily for one week.

They were treated with 100 units of salmon Calcitonin (Calsynar) four times a week for four weeks, sometimes preceeded by Maxalon if nausea was a problem. During the fourth week of treatment, they were asked to complete the same walking record. Eleven patients improved their walking distances dramatically, from less than a mile (often considerably less) before treatment, to more than a mile at four weeks (often much more). Ten patients agreed to take part in a randomized double-blind cross-over trial. Patients were randomly allocated to receive four weeks' treatment with either Calsynar or matching placebo during weeks 2 to 5, then crossed over to receive the alternative treatment during weeks 7 to 10.

During the ten weeks of the trial, the patients were assessed by both authors on five occasions (Figure 1) using the Oswestry Disability Questionnaire.[4] Their walking ability was recorded on a walking chart, with 21 entries for the seven days prior to each assessment.

RESULTS

Eleven patients had a dramatic response to Calcitonin, increasing their walking distance from less than a mile before treatment to more than a mile after treatment (Table 1). Their present or past occupations are described. Ten were men, and nine were, or had been, miners. Except for one patient, the discomfort was felt equally in both legs, and all experienced symptoms below the level of the upper calves. Five had degenerative spondylolisthesis, and one had a lytic spondylolisthesis. None had a normal myelogram. Most patients had no inappropriate signs, though three had one inappropriate sign.

Thirty patients failed to respond to Calcitonin when the criteria of walking less than a mile before, and more than a mile after treatment, was applied (Table 2 and Table 3). Six patients had a sense of well-being and subjectively thought the drug was helpful, but because their walking distance was not improved, they were considered to be non-responders. Only three were women. Sixty-six per cent were, or had been, miners. Although the symptoms were bilateral, six stated that the pain was much worse in one leg. In eight, the discomfort did not extend distally to the upper calf. Six of the 14 myelograms in the unoperated group were normal. Seventeen non-responders exhibited two or more inappropriate signs.

No patient in the series had any evidence of Paget's disease from radiographs, scintigraphy, or serum alkaline phosphatase. No patient had an abnormal doppler scan, although some patients had been considered

Table 1. Responders to Calcitonin

Patient number	*Sex*	*Age*	*Occupation*	*Walking distance (yards)*		*Leg pain**	*Pain below mid calf*	*Vertebral displacement*	*Myelogram*	*Inappropriate signs*
				Before Calcitonin	*After Calcitonin*					
1	M	67	Miner	20	1760	B	Yes	Degenerative Spondylosis	None	0
2	F	67	Housewife	400	3520	B	Yes	Degenerative Spondylosis	None	1
3	M	66	Miner	1760	3520	B	Yes	No	+	0
4	M	67	Electrician	900	2000	B	Yes	Degenerative Spondylosis	Technically Impossible	0
5	M	60	Miner	400	3520	B	Yes	No	+	0
6	M	44	Miner	800	4320	B	Yes	No	+	1
7	M	52	Miner	400	7040	B	Yes	Degenerative Spondylosis	+	1
8	M	54	Miner	150	7040	B (L > R)	Yes	Lytic Spondylosis	+	0
9	M	57	Miner	50	5280	B	Yes	No	+	0
10 (Post op)	M	53	Miner	400	3520	B	Yes	Degenerative Spondylosis	+	0
11 (Post op)	M	56	Miner	860	1760	B	Yes	No	+	0

* B = bilateral; L = left; R = right.

Table 2. Non responders to Calcitonin

tient nber	Sex	Age	Occupation	Walking distance (yards) Before Calcitonin	Walking distance (yards) After calcitonin	Leg pain*	Pain below mid calf	Vertebral displacement	Myelogram	Inappropriate signs
12	M	51	Miner	615	580	B	No	No	–	0
13	M	51	Fitter at power station	100	100	B	No	No	–	0
14	M	55	Miner	200	100	B	Yes	Degenerative Spondylosis	Technically Impossible	0
15	M	61	Plumber	50	50	B	Yes	No	+	0
16	M	49	Miner	90	50	B (L >> R)	No	No	+	4
17	M	58	Miner	35	35	B (R > L)	Yes	Retro Spondylosis	None	2
18	M	66	British rail	80	180	B	Yes	Retro Spondylosis	None	0
19	M	59	Plant operator	60	53	B (L > R)	No	No	None	2
20	M	63	Miner	600	600	B (L >> R)	Yes	No	+	1
21	M	57	Miner	150	150	B (R > L)	Yes	No	–	5
22	F	65	Housewife	100	75	B (R > L)	Yes	Degenerative Spondylosis	+	3
23	F	61	Housewife	900	900	B (R >> L)	Yes	Retro Spondylosis	–	2
24	M	56	Miner	50	90	B (L > R)	No	No	+	1
25	M	50	Miner	200	400	B (L > R)	Yes	Lytic Spondylosis	+	0
26	M	56	Miner	150	800	B (L > R)	Yes	Degenerative Spondylosis	+	1
27	M	56	Crane driver	1760	1760	B (L > R)	No	Retro Spondylosis	–	2
28	M	58	Miner	400	400	B	Yes	Degenerative Spondylosis	+ (Complete Block)	0
29	M	41	Miner	100	100	B (L > R)	Yes	No	–	5

B = bilateral; L = left; R = right.

r treatment, and were excluded when there was me evidence of abnormal peripheral circulation.

The spinal canal was measured by ultrasound. The ean measurements of those who responded to Cal-tonin and those who did not is shown in Figure 2. ıeir mean measurements are not significantly dif-rent, but they are below the tenth percentile for symptomatic subjects.

Ten of the 11 patients who responded to Calcitonin treatment entered a randomized double-blind cross-over trial. With the aid of the walking chart and the disability questionnaire, the two authors and the patient decided at the third and the fifth assessment during which leg of the trial they had been treated with placebo and during which leg they were given the drug. Five patients received Calsynar in Leg One of

Table 3. Nonresponding Post Operative Patients

Patient number	Sex	Age	Occupation	Walking distance (yards) Before Calcitonin	Walking distance (yards) After Calcitonin	Leg pain*	Pain below mid calf	Vertebral displacement	Inappropriate signs
30	M	53	Miner	100	100	B (R >> L)	No	Retro Spondylosis	3
31	M	37	Painter/Decorator	25	25	B (R >> L)	Yes	No	5
32	M	55	Caretaker	150	150	B (L > R)	Yes	No	4
33	M	62	Miner	3520	4400	B	Yes	No	0
34	M	46	Builder	50	50	B	Yes	Retro Spondylosis	3
35	M	62	Miner	400	400	B	Yes	No	3
36	F	58	Housewife	1800	2560	B (R >> L)	Yes	No	2
37	M	52	Miner	1125	1025	B	No	No	2
38	M	56	Miner	1760	1760	B	Yes	No	3
39	M	54	Miner	325	200	B	Yes	No	1
40	M	52	Salesman	400	1200	B (L > R)	Yes	No	0
41	M	54	Miner	800	800	B	Yes	No	3

* B = bilateral; L = left; R = right.

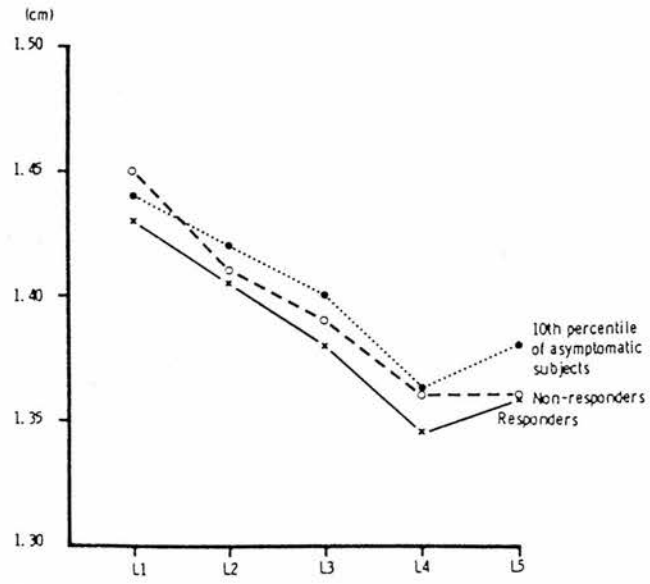

Fig 2. Ultrasound measurements of spinal canal for responders and nonresponders to Calcitonin.

the trial and placebo in Leg Two. Figure 3 shows the maximum walking distance of each patient before and after Calcitonin and the maximum walking distance during each two-week period of the trial. Patient number 2, the only woman in the trial, made the wrong assessment.

Five patients received placebo in Leg One of the trial, and Calsynar in Leg Two. The maximum walking distances before and after Calcitonin and during the trial are shown in Figure 4. Again one patient (Patient 9) made an incorrect assessment.

Calcitonin made it possible for five men to continue working underground. During the trial, one of these men was able to continue working, a second was off

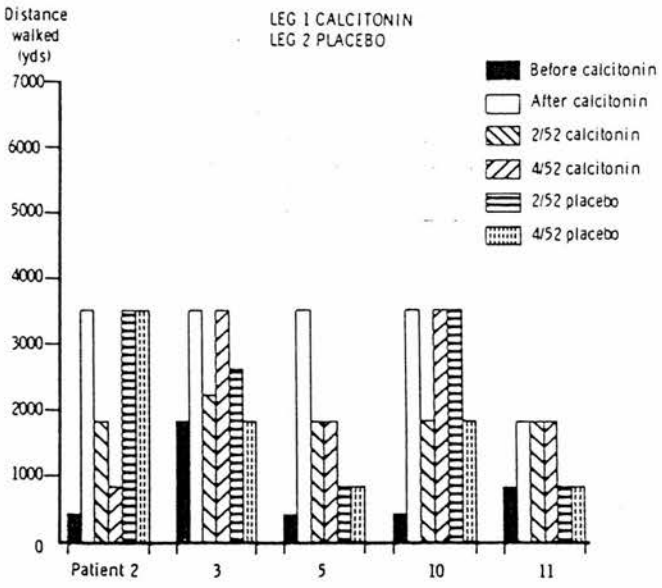

Fig 3. Maximum walking distance before and during trial.

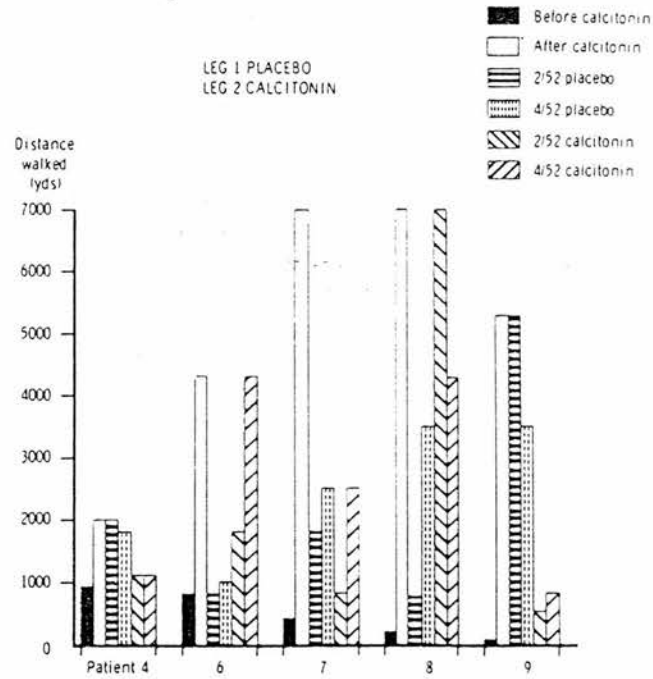

Fig 4. Maximum walking distance before and during trial.

work throughout the trial, and the other three had to have some time off work during the placebo leg of the trial, (Figure 5).

Two patients had to discontinue salmon Calcitonin because of its side effects. One developed urticaria twelve hours after the first injection. The second experienced flu-like symptoms with vomiting after the second injection, and we did not pursue treatment.

CASE REPORTS

Case 1. A 67-year-old retired miner presented in December, 1979, with weakness in his legs when walking. He first had back pain at the age of 44 years, had a plaster jacket, many periods off work, and left the colliery eight years later because of back pain.

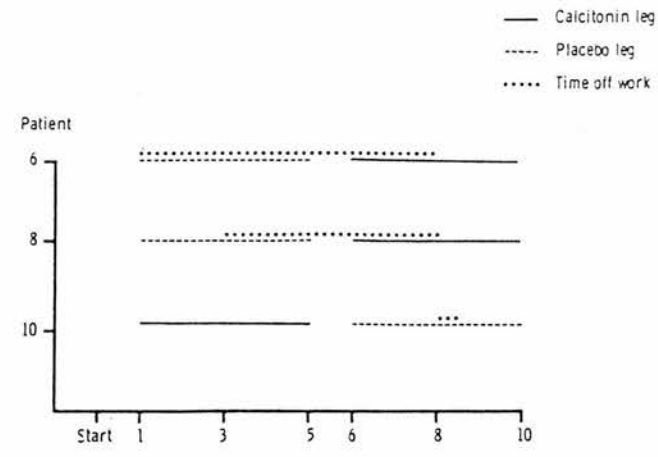

Fig 5. Absenteeism during trial.

Now he could walk only twenty yards before he had to stop and stoop forward. He had constant back pain, but his legs were the main problem, with cramps and restless legs at night. He rarely left the house, unless in a wheel chair. He could reach down to the mid tibias, but had no spinal extension. He was tender at L 3–5. SLR was 90/90. There were no abnormal neurologic signs. Doppler scan was normal. Radiographically, he had marked degenerative changes of all the lumbar spine, with normal bony texture. He was considered for a spinal decompression, which became unnecessary because after three injections of Calsynar, he wrote "I must express my gratitude for this miracle. . . . . I got a bus and walked around the town without experiencing any pain . . . . . it is the first time I have been able to do this for nine years." He maintained excellent symptomatic relief on two injections per week, sometimes being able to walk two miles, and visited his family in Australia a year later. He asked to stop the injections in April, 1982, and in October, 1982, can still walk steadily without discomfort in his legs. He was not included in the trial because he had stopped his treatment.

Case 2. A 67-year-old housewife had had back pain following a fall downstairs at 40 years of age. She continued with mild backache until, in 1974, her legs became tired when walking, and she experienced numbness and pins and needles from the buttocks and thighs, down to the feet, after walking 200 yards. She would sit or lean against a wall and, after a few minutes, walk again. When she presented in 1977, she could touch her ankles, but had only limited lumbar extension, was tender at L 4–5, SLR was 90/90, with no abnormal neurologic signs. Radiographs showed a degenerative spondylolisthesis at L 4–5 and degenerative change at L 3–4. She declined a myelogram, and it was suggested she lose weight, wear a corset, and improve her spinal and abdominal muscles. In June, 1982, she was asked to reattend, and agreed to have a course of Calsynar. Her best walking distance in the week prior to treatment was 400 yards, but after four weeks she could walk for two miles without having to stop, and she was aware that she did not have to stoop forward as she walked. She entered the controlled trial, felt well with the placebo and Calcitonin, and could not distinguish the two. Her assessment of the trial was wrong. Treatment has been stopped without relapse.

Case 3. In November, 1981, a 66-year-old man presented with discomfort like "toothache" in the thighs, calves, and ankles, limiting his walking distance to under one mile, even when he walked slowly. He had worked for 15 years on the coal face, and then worked as a refrigeration engineer until he was 60 years old, when constant pain in the back and problems with his legs caused him to retire. In 1979, he had an epidural injection for pain in a root distribution down to the left foot, but by 1981 the pain affected both legs, and this was more troublesome than his back. He had restless legs and cramping at night. He had no spinal extension, but could flex to touch his knees. He was tender at L 5, with SLR of 90/90 and no abnormal neurologic signs. A myelogram showed a narrow spinal canal with indentation of the column anteriorly by the four lower lumbar discs. He thought there was a little improvement after four weeks of Calsynar, and after eight weeks he could walk two miles. After nine months, he continues with three injections a week, and says he has only mild discomfort in his legs after walking two miles. His legs have ceased to trouble him at night. His assessment of the trial was right.

Case 4. A 67-year-old retired electrician presented in December, 1981, complaining of pins and needles from his feet to his thighs when he walked for 300–600 yards. He would stop because he was not sure where he was placing his feet and he described them as "feeling like rubber." His legs were restless at night. He had retired at the age of 61, with back pain and weak legs, but was now getting worse. He could reach to the lower tibias, had no spinal extension, was tender at L 3–5, SLR was 90/90, with no abnormal neurologic signs. A myelogram was abandoned because of failure to insert the needle. A week before he started Calsynar, he managed to walk 900 yards on one occasion. During the second week of injections, he was delighted to be walking 2000 yards in comfort, and stated that he could lie in bed with his legs extended for the first time in years. Nine months later, he has three injections a week and walks over a mile. His assessment of the trial was right.

Case 5. A 60-year-old retired miner was referred in July, 1981, with tightness in the legs when walking for half an hour. His thighs, calves, and heels became so uncomfortable that he would stop. He had worked on the coal face, with occasional back pain, until 55 years of age, but then discomfort in his legs when walking made him seek a light job. Two years later, he had to take early retirement. He could walk a mile slowly, but had to stop several times because of his legs. Sleep was disturbed with aching legs and cramping. After four weeks of injections, he could walk twice as far, could manage a mile without stopping, said his legs felt "freer," and had lost his night cramps. When he stopped the injections for two weeks, his legs felt tight, and his walking distance dropped to half a mile. He has now had injections for twelve months. His assessment of the trial was right.

Case 6. A 44-year-old miner, working at a light underground job, attended because he was having time off work with back pain and discomfort in his legs. His legs were painful and felt weak down to the ankles, making him stop after walking 300 yards. At best, he could walk up to half a mile slowly. He had restricted spinal extension, no abnormal neurologic signs, a normal doppler scan, and some radiologic degenerative change at L 2–3 and L 4–5. A myelogram showed disc protrusions at these levels. In September, 1981, he commenced treatment with 100 units of Calsynar four times a week. He said he felt "champion" after the first week, walking up to a mile and a half before stopping, and he was able to keep at work consistently, oiling machines underground. A year later, he still ob-

tains good results with two injections a week. His assessment of the trial was right.

Case 7. In July, 1980, a 52-year-old miner was referred by another surgeon for a spinal decompression. He first injured his back lifting at 41 years of age, and had nine months off work. His back caused him to come off work intermittently until, at the age of 47, he developed weak legs when walking and he had been off work for a further year. He could walk no more than 400 yards when tired legs, with discomfort down to the ankles, made him stop. His legs were "jumpy" at night. He could reach down to touch his knees, but could not stand erect. He was tender at L 3–5, had SLR of 70/70 with no abnormal neurologic signs. A radiograph showed degenerative changes at L 3–4, with slight degenerative spondylolisthesis, and myelography confirmed indentation of the metrizamide column at L 3–4 and L 4–5 from the laminas posteriorly. After four weeks of Calsynar, he could walk twice as far, and he started working again on the colliery surface. He stopped his injections after eight weeks, but within ten days, his leg symptoms recurred, and he came off work. In July, 1981, he was having three injections a week, and had taken an underground job on a transfer point, walking for 1½ miles on a rough surface underground, without having to stop. Now, 23 months after starting treatment, he has two injections a week. He is very impressed that he can work consistently at a job in the pit bottom, and he says his legs feel very good. His assessment of the trial was right.

Case 8. A 54-year-old mining deputy had developed a gradual onset of pain over the previous 12 years. He was having periods of five or six weeks off work because of pain in the thighs, calves, and feet when walking. Radiographs showed a lytic spondylolisthesis at L 5–S 1 with a 24% slip ratio and marked degenerative changes. The cauda equina was displaced on the myelogram at L 5–S 1, and there was bony encroachment at L 3–4. Prior to treatment with Calsynar, he could generally walk no more than 100 yards, but on one occasion he did record 650 yards. After four injections he was aware of improvement in walking, and after two months he could walk for 3½ miles without feeling he must stop. He maintains the ability to walk unlimited distances a year after starting treatment with three injections a week, and he now works consistently as an underground deputy. His assessment of the trial was right.

Case 9. A 57-year-old miner, who had recently left his job as a contractor on the coal face, presented in November, 1981, with pain in the lower back, thighs, and knees, limiting his walking distance to 50 yards. He weighed 210 pounds. He could reach to his knees, had some lumbar extension, SLR of 80/80, no abnormal neurologic signs, and a moderate degree of degenerative changes were present at L 2–5. After four weeks of Calcitonin treatment, he could walk two miles without stopping and was soon walking nine miles a day. He took a light underground job. After five months of injections, he had eight weeks without Calcitonin and his walking distance dropped to one quarter of a mile. Night cramps, which had settled with treatment, returned, and he began treatment again. His walking deteriorated in the first leg of the trial, when he received the placebo, and increased to 600 yards in the second leg when he received Calcitonin. His assessment of the trial was wrong.

Case 10. A mining deputy, aged 53 years, attended in February, 1981, concerned about his poor work record in the previous two years. He could walk only 200 to 300 yards before having to slow down and stop because of his legs. He said it felt like walking with "cricket pads on his legs," or like "being a deep sea diver." At the age of 33 years, he had lost almost a year of work, with back and right leg pain. At 40 years of age, he had a laminectomy to remove a disc, but was no better. In 1975, a myelogram showed bony compression at L 4–5, and he had a wide spinal decompression at L 3–5, but though he could return to work, his legs still felt heavy when walking. He could reach forward to the mid tibias, had a little lumbar extension, and an SLR of 80/80. Knee and ankle reflexes were equally reduced. There was no motor weakness or sensory loss. His maximum walking distance was 400 yards per week before starting treatment with Calsynar, but four weeks later he walked 1,000 yards underground without any discomfort. He described the response as "dramatic," and thought he could walk five miles, if necessary. Eight months later, he is having three injections a week, works consistently, and states that he can walk any distance. For the first time in ten years, he has walked up a half mile 1:5 gradient without having to stop, while before treatment he would stop ten times for a rest. His assessment of the trial was right.

Case 11. In June, 1982, a 56-year-old retired miner presented with aching in his legs when walking. Generally he could walk no more than 400 yards, but occasionally he could manage a mile. He injured his back in the colliery at the age of 43 years and had not worked since. In 1977, he could walk no more than 300 yards, when pain in the legs to the lower calves would make him stop. A myelogram demonstrated disc degeneration and encroachment at L 5–S 1, with extra-dural compression. He had a spinal decompression at L 3–5, and was initially pleased, walking half a mile in comfort. Six months after the operation, however, he deteriorated and could not walk so far, and had restless legs at night. His limitations persisted, but after four weeks of treatment with Calsynar he could walk over 1000 yards, and at six weeks he felt he had a "spring in his step," walking over a mile. After five months of treatment, he continues with three injections a week, now walking up to two miles. His assessment of the trial was right.

DISCUSSION

Calcitonin Treatment for Neurogenic Claudication

The symptoms of neurogenic claudication have been relieved successfully in 11 patients treated with Calcitonin. Their walking distances have improved considerably, and some have no discomfort at all when

alking. Restless legs and cramps no longer disturb eir sleep. Five men were in danger of losing their bs, but are now working underground in a colliery, ur of them walking considerable distances on rough ound for much of the shift. Most of these patients uld have been treated by surgery had they not re-onded to Calcitonin, and for two of them, this would ve been repeat surgery. There are obvious advan-ges in being able to treat neurogenic claudication by n-operative means, and for these 11 patients Calci-nin appears to have been effective. There have been serious side effects. Two patients have been able discontinue treatment without relapse, the re-ainder continuing on two or three injections a week. It is possible that a placebo response could account r a sense of subjective improvement, but it is prob-le that such a considerable increase in walking dis-nce has an organic basis. A randomized double-blind oss-over trial was designed to assess the placebo sponse in ten patients.

Two patients assessed the controlled trial incor-ctly. One was the only woman in the trial. She re-ived Calcitonin for five months, and was convinced its benefit, but could not identify the placebo. There e two possibilities. Either her original response to alcitonin was a placebo effect, giving her a remark-le increase in walking from two hundred yards to miles after four weeks treatment, or there was an perfection in the trial. A week's washout and four eeks of placebo may not have been sufficient to re-rse the benefits of Calcitonin. She has now stopped king the drug and continues in remission.

The second patient to make an incorrect assessment atient 9) had increased his walking distance with lcitonin from 50 yards to over two miles, but he eriorated during the placebo leg of the trial, drop-g his walking distance to three quarters of a mile. e failed to increase this distance in the Calcitonin leg the trial, but after two more weeks of Calcitonin he s walking two miles again. This could be a placebo sponse, but it is possible that it took more than four eeks for the Calcitonin to be effective.

The remaining eight patients in the trial made a cor-ct assessment, and it is difficult to attribute this en-rely to chance.

riteria for a Successful Response to Calcitonin

If Calcitonin is genuinely effective in relieving ymptoms of neurogenic claudication for some pa-nts, who is most likely to respond? A comparison tween the patients who responded and those who d not suggests that certain criteria should be present benefit is to be expected. The pain should affect both gs equally, extend distally beyond the upper calves, mit walking distance to less than a mile, there should e no more than one inappropriate sign, and the mye-ogram should be positive. No patient, in fact, responded to Calcitonin, whose pain was predominantly in one leg, whose pain did not extend below the upper calves, who could walk above a mile, who had more than one inappropriate sign, and who a normal myelogram. There are thus five absolute criteria which should be present before a patient is offered Calcitonin (Table 4), and four relative criteria. If we had restricted the drug to the 19 patients fulfilling these criteria, there would have been a successful response in 58% of those treated.

We were not able to find any consistent abnormal signs to support the diagnosis of neurogenic claudication. We were not convinced of any abnormal neurologic signs, either at rest, or a changing pattern after exercise. The diagnosis, therefore, probably depends on an accurate history, meeting strict criteria, and a positive myelogram. A CT scan and ultrasound measurement will confirm a narrow canal and support the diagnosis, but spinal stenosis is not synonymous with neurogenic claudication. It is possible that many of the patients who failed to response to Calcitonin did not have neurogenic claudication, although their walking distance was limited by pain in the legs. Neurogenic claudication is a condition with few abnormal objective signs, and unless strict criteria are applied, it is liable to be over-diagnosed.[15] Three spinal conditions can be confused with neurogenic claudication. It can be imitated by pain referred from the lumbo-sacral region to the thighs and upper calves. A mixed pathology of root pain and referred pain can also be confusing, but one would expect a dermatomal distribution of the root pain, and symptoms predominantly affecting one leg. Thirdly, a patient who exhibits several inappropriate signs probably has a considerable non-organic element associated with the symptoms, and not neurogenic claudication. This over-diagnosed condition is all too frequently confused with other spinal problems, but the symptomatology of those patients responding to Calcitonin may provide criteria whereby the diagnosis should probably be made.

Etiology of Neurogenic Claudication

We are still unaware of the pain source in neurogenic claudication, but an assessment of those patients

Table 4. Probable Criteria for a Successful Response to Calcitonin

Absolute criteria	*Relative criteria*
Pain equally in both legs when walking, relieved by rest	Male
	Over 40 years of age
Pain extending distal to upper calves	Heavy manual worker
No more than one inappropriate sign	Normal peripheral circulation
Walking limited to less than one mile	
Positive myelogram	

who responded to Calcitonin would suggest that there may be several factors.

Verbiest[13] associated its symptoms with a shallow central spinal canal and, until other syndromes were recognized in which the canal size is a significant factor, spinal stenosis was considered to be synonymous with neurogenic claudication. Restricted space in the central spinal canal is certainly related to the symptoms, but it is possible to have a shallow spinal canal with no symptoms at all. The patients in this series had developmentally narrow canals for many years before they experienced neurogenic claudication symptoms. Forward displacement, with an intact neural arch, in a man who already has developmental narrowing of the spinal canal, is probably an unhealthy combination. Five of the 11 patients who responded to Calcitonin in this series had degenerative spondylolisthesis.

A further factor in association with the narrow canal is probably the accumulated effect of mechanical stress, rather than one specific injury, to the lumbar spine. If the late effects of one incident, such as a disc injury, were associated with the symptoms, we would expect to find some sedentary workers in this series. Most of the men had spent their working life mining coal, and prior to the mechanization of the coal mines, they would have spent their days in a confined space using a pick and shovel.

The paucity of women with neurogenic claudication may reflect that they subject their spines to less abuse than men. There may, however, be other explanations for the low incidence in women, such as their canals being generally wider than men's,[8] or hormonal factors, or variation in the vascular anatomy of the pelvis.

The rapid improvement in walking experienced by those patients who responded to Calcitonin, as with the Paget's paraparesis recovery[3,6,9] can probably only be explained in relation to a vascular response. Domisse and Grober[2] described the abundant vascular system supplying the cauda equina "just adequate for its minimum needs." It is tempting to speculate that the reduced arterial blood flow to the skeleton, which accompanies Calcitonin therapy,[17,18] produces a shunt mechanism that satisfies the needs of a deprived cauda equina. Alternatively, an improved venous return may reduce the volume of Batson's extradural plexus of veins, increasing the oxygen supply to the rami, ganglia, and roots. In either case, vascular insufficiency is probably a factor in the aetiology of neurogenic claudication.

Calcitonin offers, for the first time, a useful, conservative method of treating neurogenic claudication. It also provides criteria whereby we can be more critical in diagnosis, and the response to treatment does suggest that there is an ischemic factor in the etiology of the condition. The mechanism of its action awaits hemodynamic and biochemical investigation.

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Address reprint requests to

R. W. Porter, MD
Consultant Orthopaedic Surgery
Doncaster Royal Infirmary
Doncaster, England

Accepted for publication January 21, 1983.

care shown in this study describes the type of approach that is necessary when caring for old people, and case finding might be a way of achieving this by combining prevention with care and cure. Furthermore, including a checklist of important points in the clinical notes might be an "aide memoire" to the doctor.

The practice health visitor is ideally the person to visit the patients who are not seen. A research project will be set up to test the benefits of this method of case finding.

I thank the doctors and receptionists at both the Rusholme Health Centre and the Westhoughton Group Practice for their help in carrying out this project. I also thank Mrs Brenda Firth and Mrs Joan McFarlane, the health visitors, for their enthusiasm, and Dr H W K Acheson for general advice.

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(*Accepted 27 October 1983*)

Value of ultrasonic measurement of spinal canal diameter in general practice

J N DRINKALL, R W PORTER, C S HIBBERT, C EVANS

Abstract

The diameter of the spinal canal was measured by ultrasonic scanning in 193 patients with back pain and 193 matched controls. The spinal canal was found to be significantly narrower in the patients compared with all 193 controls ($p < 0.01$) and compared with 132 of the controls with no history of back pain ($p < 0.001$), but measurement of canal size was found not to have any value in the prognosis or management of back pain in general practice.

Introduction

In 1980 Porter *et al* reported that in roughly half the patients attending hospital with symptoms of disc abnormalities ultrasonic measurement of the spinal canal yielded values below the 10th percentile for asymptomatic subjects.[1] We report here the results of a study of the diameter of the spinal canal in patients attending a general practice and the value of this measurement in the management and prognosis of back pain.

Doncaster Royal Infirmary, Doncaster DN2 5LT
J N DRINKALL, MRCS, LRCP, general practitioner
R W PORTER, MD, FRCS, consultant orthopaedic surgeon
C S HIBBERT, PHD, research worker
C EVANS, research worker

Correspondence to: Mr R W Porter.

Patients, methods, and results

A training practice of four principals, based in a south Yorkshire mining and manufacturing town and serving a population of 8000 patients, agreed to take part in a prospective study of its patients using the same diasonograph, technique, and technicians as in previously published hospital studies.[1 2] All patients attending the practice with back pain over 12 months were asked to undergo spinal scanning. A similar number of patients attending the surgery during the same period for some other condition were also asked to volunteer for the spinal measurement and were matched for age and sex. Any history of back pain was recorded.

Twelve months after presentation the patients were asked to complete a questionnaire on treatment, time off work, and their present experience of back pain. The general practitioners' records provided information about frequency of attendance for back pain and for other conditions, time off work, and the use of hospital orthopaedic services. The attendances of the control patients were also noted as were their records of any previous back pain.

During the 12 months of the study 207 patients attended the practice with back pain. Of these 193 attended for spinal scanning and were compared with 193 matched controls. One hundred and sixty five patients completed the questionnaire 12 months after

TABLE I—*Age, sex, and occupation of patients and controls*

	Mean (SD) age (years)	Men:women	Occupation						
			Heavy manual	Light manual	Semisedentary	Sedentary	Miner	Housewife	Unemployed
Patients with back pain (n = 193)	44·5 (14·8)	104:89	49	68	29	10	6	23	8
Controls (n = 193)	44·8 (15·2)	104:89	39	66	26	13	4	40	4

presentation. The mean (SD) age of the patients with back pain was 44·5 (14·8) years, and 104 (54%) were men (table I). The patients were fairly well matched with the controls for age, sex, and occupation. Of the patients with back pain, 147 (76%) had attended previously with back pain compared with 62 (32%) of the controls. Fifty nine (56%) of those who were employed had had time off work: two thirds were back at work within four weeks and only 16 still unable to work after eight weeks; three patients were still unable to work after a year. Sixteen (8%) of the patients with back pain were referred for spinal radiography. Eighteen (9%) were referred for orthopaedic investigation; in three this was their first referral for back pain. A further 32 had previously been referred to the hospital, as had 14 of the control group.

Table II shows a significant difference in the mean diameter of the spinal canal between the 193 patients and all 193 controls ($p<0·01$) and between the patients and the 132 controls who did not have a history of back pain ($p<0·001$). The patients and the controls who had a history of back pain had narrower canals than the patients who attended with their first attack of back pain, but the difference was not significant. There was no significant difference in canal size between those patients referred to hospital and those managed in the practice. Patients who lost time from work had narrower canals than those who kept at work, but the canal size was not related to the duration of time off work. There was no relation between canal size and either recurrence of back pain over 12 months or recovery.

TABLE II—*Mean (SD) diameters of spinal canals (cm) measured with ultrasound in patients and controls*

Lumbar level	Patients (n = 193)	All controls (n = 193)	Controls with no history of back pain (n = 132)
L1	1·48 (0·073)	1·51 (0·082)	1·52 (0·079)
L2	1·46 (0·072)	1·48 (0·084)	1·49 (0·077)
L3	1·43 (0·074)	1·46 (0·083)	1·47 (0·078)
L4	1·40 (0·075)	1·43 (0·083)	1·44 (0·078)
L5	1·41 (0·073)	1·43 (0·079)	1·44 (0·074)

Twenty (43%) of the patients attending with their first attack of pain had no further trouble over 12 months, but 19 (81%) of those with a history of back pain still had pain 12 months after their initial presentation. Unbeknown to the general practitioners, 21 patients admitted to attending an osteopath or physiotherapist and 18 of them reported recovery.

Discussion

Patients and doctors alike recognise the difficulty in diagnosing the cause of low back pain, and many patients, especially those attending with recurrent back pain or whose pain does not settle quickly, expect to be offered some investigative procedure. The diagnostic limitations of radiography are well recognised.[3] Investigation of the spine by ultrasonic scanning, if proved to be of value in the management or prognosis of back pain, could help general practitioners because it is a non-invasive technique of low cost.

The results of this study confirm that not only patients attending hospitals but also those attending general practices with back pain tend to have shallower spinal canals than those without pain. The morbidity of back pain, as measured by a history of back pain and the need to take time off work, also seems to be related to the size of the spinal canal. There was no relation, however, between canal size and incidence of referral to hospital or recovery over 12 months. In practical terms, therefore, although the canal size is of interest epidemiologically, it does not have any value in the management or prognosis of back pain in general practice.

Management of back pain is probably best helped by careful clinical examination and history taking; the best prognostic variable, as shown in this study and previously by Roland *et al*,[4] is a history of back pain.

We thank the doctors and staff of the Mount group practice, Doncaster, for their commitment to this study, Mrs J Reynolds for secretarial help, and the Trent Regional Hospital Board and the Back Pain Association for financial support.

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(*Accepted 2 November 1983*)

ONE HUNDRED YEARS AGO A recent trial in the Sheriff Court, at Glasgow, furnishes another illustration of the various channels through which disease of an infectious nature is spread amidst the community. The following are the facts of the case as brought out in evidence. The message-boy of a butcher had his fingers severely crushed in a mincing-machine, and for the injuries thus received the lad's father sought damages. With the legal liabilities of the case we have no concern, but the facts revealed in the course of the case have a wider importance than the court of justice where they were disclosed. It came out in evidence that the boy had been kept from school, because fever had broken out in his home. Such a procedure was a very proper one, but it lost much of its value, inasmuch as it seems the lad was allowed to associate as usual with his companions, and was also permitted to act as message-boy to this butcher, doing his errands and carrying parcels of food to his customers. Further, it was shown that from time to time he handled the food that was exposed for sale. This is scarcely a state of matters that can be called satisfactory, and must give rise to some uneasy reflections. To allow a boy from a fever-stricken house, with probably infection hanging about him, to manipulate food exposed for sale, and then carry it to the houses of the unsuspecting buyers, is a state of matters calculated to work untold mischief. As the judge remarked: "Heads of families and their medical men are often puzzled to know how it is in spite of every precaution infection gets into their houses. It is felt that in such a state of matters as here occurred a clue to the mystery might sometimes be found." Unfortunately it seems that for such disgraceful carelessness as was revealed in this case the law provides no remedy; but it is satisfactory that public attention has been drawn to the matter, for there can be no doubt that the fear of spreading infection which kept the boy from school should have had the same weight in preventing him from carrying parcels, and especially food. (*British Medical Journal* 1884;i:1265.)

The Relationship Between Spinal Canal Diameter and Back Pain in Coal Miners

Ultrasonic Measurement as a Screening Test?

E. B. Macdonald, M.B., Ch.B.; R. Porter, M.D.; C. Hibbert, Ph.D.; and J. Hart, S.R.N.

Back pain is the commonest cause of absence among coal miners. However, the degree of morbidity is variable. To determine whether the susceptible individual can be identified, ultrasound was used to measure the spinal canal diameters of 204 miners. The results show that those men with the longest histories and the longest times off work and those who had to leave the coalface or who left the industry — i.e., those with the greatest morbidity — had significantly narrower canals. Ultrasonic measurement of the spinal canal diameter is safe and noninvasive. Its use should be further evaluated as part of a preemployment screening procedure for back pain.

Musculoskeletal disorders are the largest cause of absenteeism in the coal mining industry, and the most significant of these is low back pain. It is responsible for up to 18% of sickness absence in some British coalfields. Approximately 70% of miners will have some time off work with back pain during their working life, but the fact that the remainder can work in the same arduous environment without significant back trouble, and that the degree of morbidity is variable, would suggest that there is an individual risk factor.

In the Western industrialized world, particularly the United States, considerable efforts have been made to screen potential employees for conditions that may predispose to back pain. Radiology has been used extensively though the consensus of opinion is that it is of little value[1,2] and ethically the implications of the radiation dosages involved are questionable. The work of Chaffin et al[3] has shown that if the individual's strength, as measured by dynamometry, is significantly in excess of that required to do the task, there is less risk of injury or back pain developing.[3] More recently Porter et al[4] have shown that the mean sagittal diameter of the spinal canal for patients hospitalized with back pain is less than that for the general population.[4] This diameter can be measured relatively easily by ultrasound, which is safe and noninvasive.[5,6] This article attempts to assess the relationship between the spinal canal diameter and back pain experienced by older miners.

Materials and Methods

A population of 373 coalface workers between the ages of 50 and 60 years, from a South Yorkshire, England, colliery, were invited to take part in this study and 204 attended. The spinal canal was measured by pulsed echo ultrasound. Each man was subsequently interviewed regarding his previous experience of back pain. He was asked the following questions: (1) Had he ever had low back pain? (2) What was his age at the first attack? (3) Was he ever off work with back pain? (4) What was his longest period off work with back pain? (5) Was back pain responsible for his leaving the coalface? and (6) Had pain affected his back only, or his back and legs also?

The colliery records for the previous three years were then examined to assess all causes of certified sickness absence among the 373 men, with special reference to absenteeism resulting from low back pain. Information obtained from the interviews and the sickness absence experience of the 204 attenders were then compared with their spinal canal measurements.

The study subjects were reviewed three years later. The results of their spinal canal measurements had not been revealed to them or their employers at any time. Further sickness absence and employment outcomes were re-

From the National Coal Board, Area Medical Centre, Markham Main Colliery (Dr. Macdonald, Principal Medical Officer and Ms. Hart, Nursing Officer) and the Doncaster Royal Infirmary (Dr. Porter, Consultant Orthopaedic Surgeon; Dr. Hibbert, Research Assistant).

This investigation was supported by the National Coal Board.

Address correspondence to Area Medical Centre, Markham Main Colliery, National Coal Board, Armthorpe, Doncaster, South Yorkshire, England DN3 2BW (Dr. Macdonald).

corded. Thus, retrospective and prospective studies were performed.

Results

Seventy-five percent of the 204 miners had experienced previous back pain. The mean measurements for those who recollected previous back pain and for those who reported no pain are shown in Fig. 1 and Table 1. The differences are significant at L.4 ($p < .01$) and at L.5 ($p < .05$).

Fig. 2 (Table 2) compares the mean measurements for the miners whose first attack occurred in the third, fourth, fifth, and sixth decades, those with narrower canals having the longer history of back pain. Fig. 3 (Table 3) compares the mean measurements with the maximum length of time off work due to back pain. Fourteen percent of the miners with back pain had required more than six months off work. Their canal measurements were significantly ($p < .001$) less than those of the remaining miners, who had had either no time off work or less than six months. Twenty-five men said they had had to leave the coalface because of back pain. Their mean measurements are shown in Fig. 4 (Table 4) and are compared with those of men still working on the face. Differences are significant at L.5

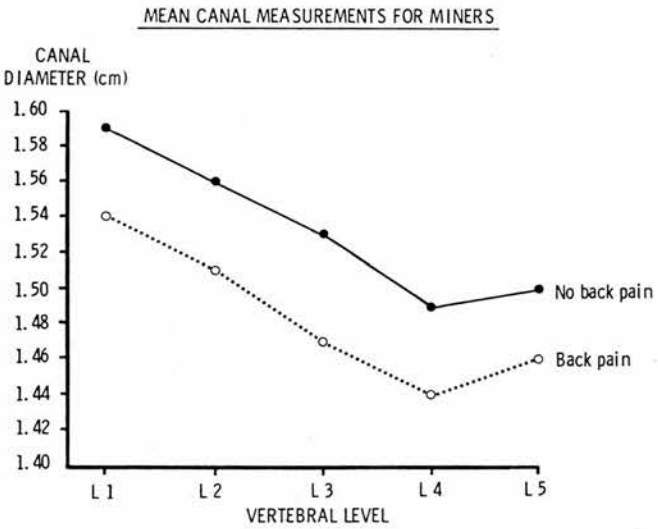

Fig. 1 — Mean canal diameter for miners with and without back pain.

Table 1 — Mean Canal Diameter for Miners With and Without Back Pain

	Miners Without Back Pain (n = 52)		Miners With Back Pain (n = 152)	
	Mean	SD	Mean	SD
L.1	1.59	0.10	1.54	0.09
L.2	1.56	0.10	1.51	0.09
L.3	1.53	0.10	1.47	0.09
L.4	1.49	0.10	1.44	0.09
L.5	1.50	0.11	1.46	0.09

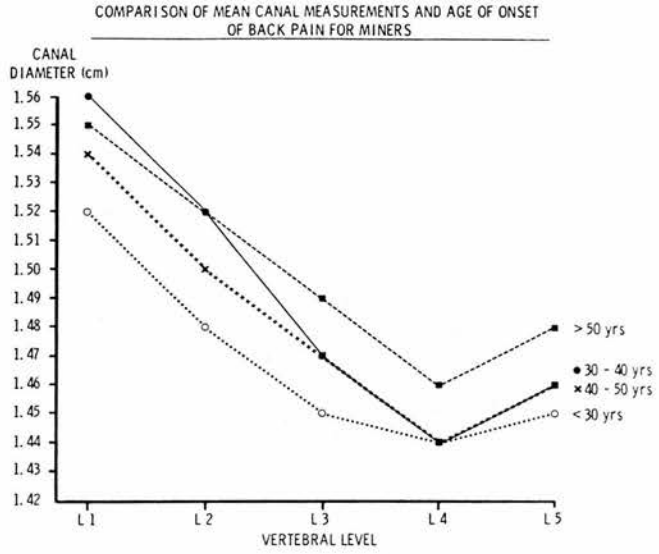

Fig. 2 — Comparison of mean canal measurements and age on onset of back pain for miners.

Table 2 — Comparison of Mean Canal Measurements and Age at Onset of Back Pain for Miners

	Age at Onset of Back Pain							
	< 30 yr (n = 38)		30-40 yr (n = 37)		40-50 yr (n = 57)		> 50 yr (n = 20)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
L.1	1.52	0.11	1.56	0.07	1.54	0.09	1.55	0.07
L.2	1.48	0.11	1.52	0.09	1.50	0.09	1.52	0.07
L.3	1.45	0.11	1.47	0.09	1.47	0.09	1.49	0.07
L.4	1.44	0.12	1.44	0.09	1.44	0.08	1.46	0.06
L.5	1.45	0.09	1.46	0.09	1.46	0.10	1.48	0.07

($p < .001$). Sixty-one men had had pain in both back and leg. Their measurements are compared with those of the miners who had had only back pain (Fig. 5 and Table 5) and at L.5 they are significantly narrower ($p < .01$).

The total numbers of days lost from work attributable to back pain in the three years prior to canal measurements were obtained from the colliery records of 191 men (15 records were unobtainable). The number of days lost for men in each percentile of canal size is shown in a histogram (Fig. 6). Of the days off work, 32.3% were lost by the 22 miners with the narrowest spines, those below the 10th percentile. This work loss was significantly greater than that of men above the 10th percentile ($p < .001$). In terms of a history of absenteeism due to back pain there was no significant difference between the men who participated in the study and those who did not, with 44% of the attenders requiring absenteeism from low back pain, compared with 45% of the nonattenders.

At follow-up three years later, 115 of the 191 men were found to have left the industry. A reduction in the labor force had occurred during that period and the majority of these men had left due to voluntary redundancy or through early retirement. Only one had retired on grounds of ill

Table 3 – Comparison of Mean Canal Measurements and Maximum Length of Time Off Work for Miners With Back Pain

	No Time Off Work (n = 16)		8-16 wk (n = 20)		16-26 wk (n = 17)		26-52 wk (n = 17)		> 52 wk (n = 4)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
L.1	1.55	0.08	1.53	0.07	1.56	0.07	1.51	0.08	1.45	0.05
L.2	1.50	0.07	1.50	0.09	1.52	0.07	1.47	0.08	1.44	0.04
L.3	1.49	0.07	1.46	0.08	1.46	0.09	1.43	0.09	1.41	0.03
L.4	1.47	0.06	1.43	0.06	1.43	0.09	1.39	0.09	1.36	0.04
L.5	1.50	0.08	1.45	0.08	1.46	0.09	1.39	0.10	1.37	0.06

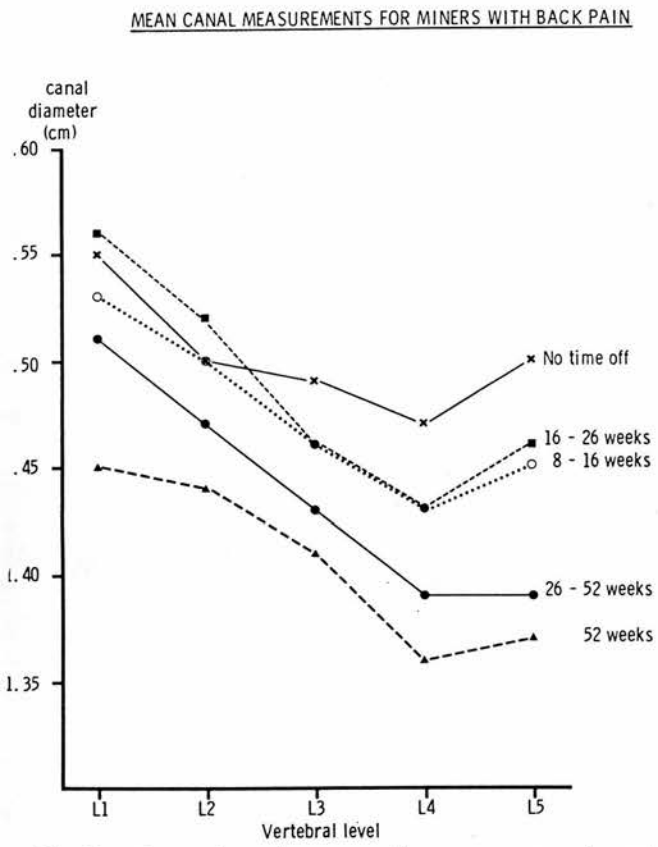

Fig. 3 – Comparison of mean canal measurements and maximum length of time off work for miners with back pain.

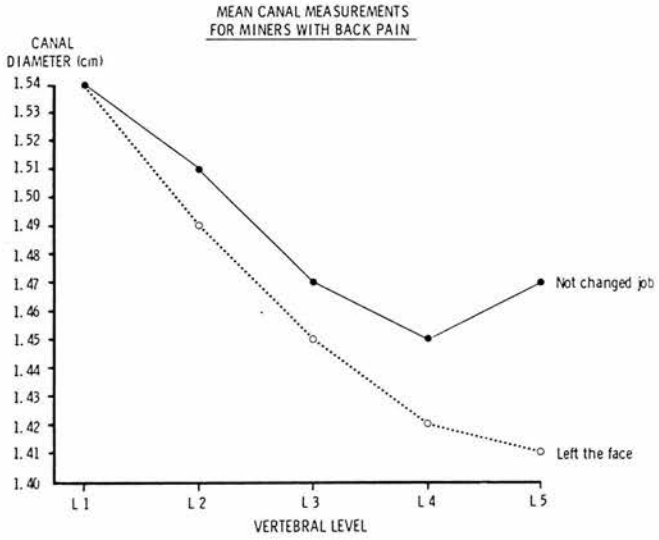

Fig. 4 – Mean canal measurements for miners who had to leave coalface with back pain and those who continued on face.

Table 4 – Mean Canal Measurements for Miners Who Had to Leave the Coalface With Back Pain and Those Who Continued on the Face

	Miners Who Have Left Face (n = 25)		Miners Who Have Not Left Face (n = 127)	
	Mean	SD	Mean	SD
L.1	1.54	0.11	1.54	0.09
L.2	1.49	0.11	1.51	0.09
L.3	1.45	0.11	1.47	0.09
L.4	1.42	0.13	1.45	0.08
L.5	1.41	0.09	1.47	0.09

health that was due to back pain. Discussions with management confirmed that the men offered redundancy were those who were known to have had, or to have, considerable health problems or disability. Thus, the redundancy scheme was used to encourage, by financial incentive, men with health problems to leave the industry. The redundancy payments were better then those for other forms of retirement, and men usually welcomed the opportunity to leave the industry under favorable terms.

Comparison of spinal canal diameters of the 115 men who left with those of the 76 men still employed shows that those who retired had narrower canals than those who remained (Fig. 7 and Table 6). The widest canals occurred in those at work with no history of back pain, and the narrowest in those who had retired with a history of back pain ($p < .01$).

In both the working and retired groups differences in mean spinal canal diameter between those with and those without back pain were statistically significant ($p < .05$). For the retired men, the number of days lost due to back

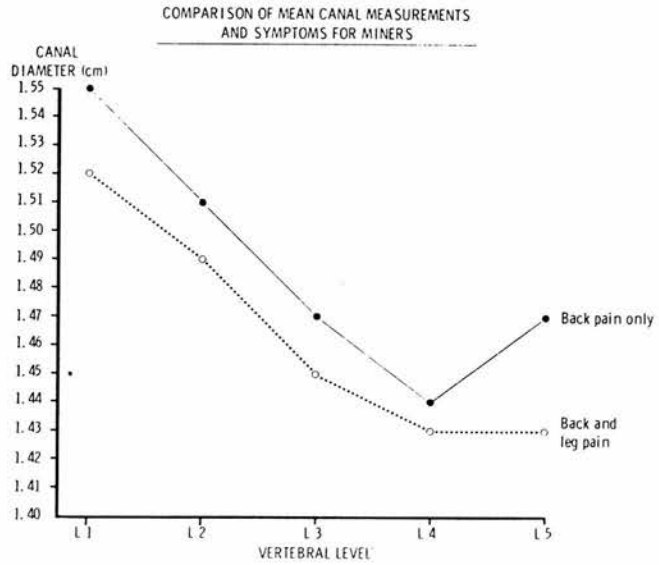

Fig. 5 – Comparison of mean canal measurements and symptoms for miners with back pain only and those with leg pain.

Table 5 – Comparison of Mean Canal Measurements and Symptoms for Miners With Back Pain Only and Those With Leg Pain

	Back and Leg Pain (n = 61)		Back Pain Only (n = 91)	
	Mean	SD	Mean	SD
L.1	1.52	0.09	1.55	0.09
L.2	1.49	0.09	1.52	0.09
L.3	1.45	0.10	1.48	0.08
L.4	1.43	0.11	1.45	0.07
L.5	1.43	0.09	1.48	0.08

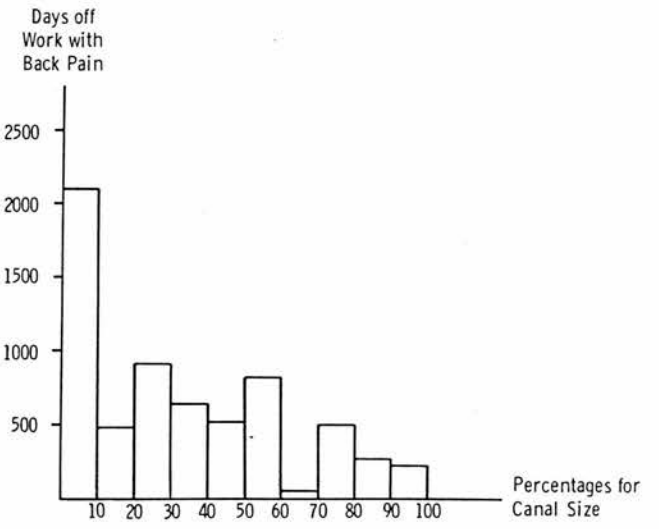

Fig. 6 – Histogram of days off work in previous three years for miners in each 10th percentile of canal size.

Table 6 – Comparison of Mean Canal Measurements for Those Men Who Remained and Those Who Retired With and Without Back Pain

	Men Still Working With No Back Pain (n = 30)		Men Still Working With Back Pain (n = 46)		Men Retired With No Back Pain (n = 63)		Men Retired With Back Pain (n = 52)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
L.1	1.60	0.09	1.54	0.09	1.55	0.11	1.54	0.10
L.2	1.56	0.10	1.51	0.08	1.52	0.11	1.50	0.10
L.3	1.53	0.09	1.48	0.08	1.49	0.11	1.46	0.10
L.4	1.49	0.14	1.45	0.08	1.46	0.11	1.43	0.09
L.5	1.51	0.11	1.46	0.09	1.48	0.11	1.44	0.09

pain in the three years prior to canal measurement is shown, for each percentile of canal size, in a histogram (Fig. 8). Again there is a significant difference in absenteeism due to back pain among those below and those above the 10th percentile ($p < .05$). Only 30 of the 191 men had had no absence due to back pain by the end of the six-year period.

Discussion

The magnitude of the back pain problem in those industries demanding heavy manual handling will be reduced by improving the environment to avoid accidents and by reducing the weight of the loads lifted and thus the associated spinal stress. It may also be reduced by educating employees to respect their spines. A third alternative, more attractive in industries where manual lifting is unavoidable because of an adverse environment, is to seek means of identifying those individuals at greatest risk and to deploy them to lighter work areas.

There are no doubt many factors that contribute to a subject's vulnerability to develop back pain. The resilience of the annulus fibrosus to injury, muscle strength to match the task,[3] pain tolerance and behavioral patterns may all be significant. The present study suggests that the size of the spinal canal is also a factor, and supports the observations of Verbiest[7] that many patients requiring spinal surgery have narrow canals. Symptoms associated with a disk lesion also tend to be more disabling if the patient has a narrow canal.[4]

Correlations between the canal size and information obtained from the miners about their previous back pain suggest that those men with the longest histories and, the longest times off work and those who had to leave the coal-face or who left the industry – in fact, those with the greatest morbidity – had significantly narrower canals. More convincing was the evidence obtained from the colliery records, where 32.3% of the days lost through back pain were contributed by the men with canal measurements in the narrowest 10th percentile of canal range. If these men had previously been deployed to other areas, and their place taken by men with a canal size above the 10th percentile, the economic advantage to the industry would have

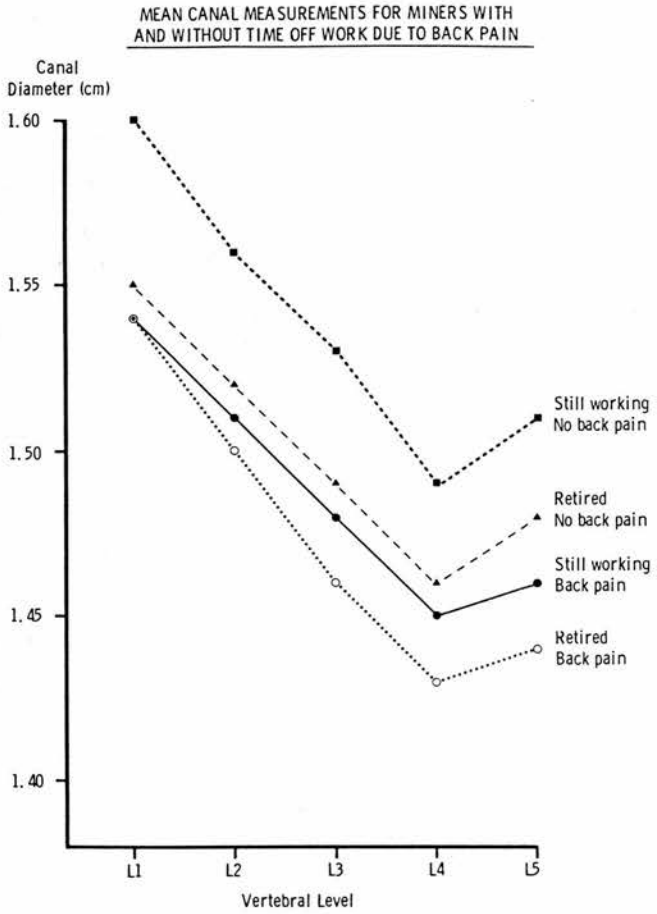

Fig. 7 — Comparison of mean canal measurements for those men who remained and those who retired, with and without back pain.

been considerable, and probably the grief and pain to the men and their families could have been prevented. It can be hypothesized that the cost of absence of a faceworker to the industry is $177,000 per year (Table 7). These figures do not take into account the considerable costs relating to compensation claims that occur in the mining industry. Replacing the bottom 10% by other men would theoretically have saved 1,618 working days for the 191 men over a three-year period, approximately eight man years.

We believe that the results of this study indicate that preemployment measurement of the spinal canal by ultrasound might identify the worker at risk of significant back pain. If so, the benefits in terms of avoiding morbidity for

Table 7 — Estimated Cost to the Industry of the Loss of a Coalface Worker for One Year*

In 1 yr	$
200 manshifts = 2,000 tons x $60/ton	114,000
Administrative costs — constant	4,500
Cost of training replacement faceworker	18,000
Approximate cost to National Coal Board in 1 yr	177,000

* One manshift = 9.5 tons x $60/ton = $570

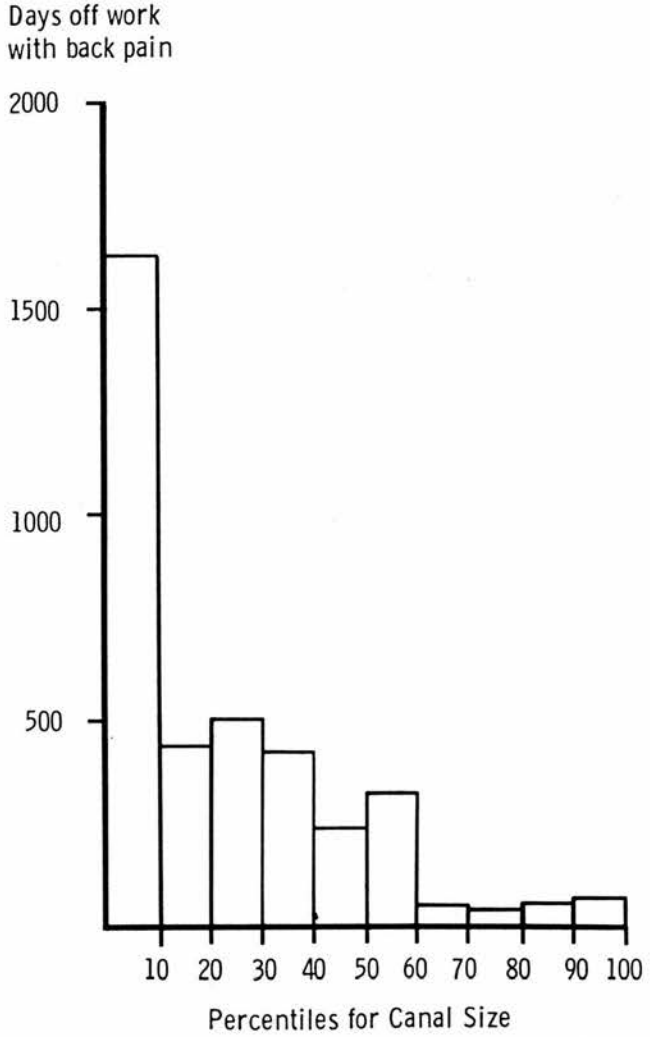

Fig. 8 — Histogram of days off work in previous three years for retired miners in each 10th percentile of canal size.

the miner or worker at risk and in terms of financial saving to an industry would be enormous.

Ultrasound provides a safe, noninvasive method of measurement with a mean repeatability of less than 0.5 mm.[5] It takes five minutes to perform, and is, we suggest, ideally suited to preemployment screening. We believe that the specificity and sensitivity of any preemployment screening procedure for back pain would be improved by the combined use of spinal canal measurement and other parameters. Strength testing and factors such as family history may well be important. However, further careful prospective appraisal of the significance of the spinal canal size in relation to back pain is required before recommending its use in the selection of potential workers.

Acknowledgment

G. Swann, Doncaster Royal Infirmary, did the photographic work.

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Editorial Commentary

Ultrasonography of the Spine

Spinal surgeons, as well as public health and occupational health professionals, have long been frustrated by the unpredictablity of back pain, since no statistical breakdown has been helpful in identifying the vulnerable group. Increasing interest and research in canal stenosis over the past decade, however, have brought canal size into focus as a key determinant of symptomatology. Thus, while major pathology, such as a massive disc herniation, in a capacious canal, may be asymptomatic, minor intrusions in a shallow canal can be totally disabling.

Although this concept presented an intriguing occupational health screening potential, no cost effective, non-invasive technology for mapping the lumbar canal has been available for general use. Intrathecal contrast studies were obviously unacceptable, as was the radiation exposure associated with computerized axial tomography (CAT scan). Ultimately, nuclear magnetic resonance (NMR) may offer a practical solution, but it will be many years until costs have scaled down to that point.

It had always been assumed that the high absorption of sound by bone would permit no meaningful ultrasound echoes from the spinal canal. About seven years ago, however, one of the authors of this paper, R. W. Porter of Doncaster, United Kingdom, was able to show that part of the vertebral lamina is thin enough to permit ultrasound to penetrate and be reflected back through the lamina, and he developed the technique for measurement of the oblique sagittal diameter of the lumbar spinal canal. He measured more then 900 subjects and noted difficulty in recording measurement only in the very obese and in patients who had had previous spinal fusions. He found the figures reproducible with an interobserver error of only 0.2 mm.

He subsequently evaluated 73 patients with symptomatic disc lesions and compared their canal size with 200 asymptomatic subjects. He found that the patients had intrinsically narrow lumbar spinal canals. Of the symptomatic patients, 55% were below the 5th percentile of a normal asymptomatic control group; 68% were below the 10th percentile for canal size. He also found that there was a correlation between the size of the canal and the patient's response to treatment, with the group requiring surgery having the smallest spinal canal measurements.

The paper presented by this same group from Doncaster is another exciting confirmation of the potential value of non-invasive mensuration of the spinal canal. The immense public health appeal for occupational low back screening is enormous. There is just one problem: no one else has been able to reproduce the model. In 1978 a group of us at the George Washington University Medical Center began a very ambitious project for the purpose of comparing spinal canal measurements mapped out by ultrasound with the dimensions obtained from computerized axial tomography. In spite of meticulous preparation and the best of equipment, our dedicated technologists could not obtain reproducible results. We finally gave up, and I know of no one else who has been any more successful than we were.

Unfortunately, until now, it seems to have only been another great idea with a single user.

Henry L. Feffer, M.D.
Professor of Orthopaedic Surgery
The George Washington University
Medical Center
2150 Pennsylvania Ave., N.W.
Washington, DC 20037

Avoiding Moral Dilemmas

Psychiatric consultations are a legitmate and necessary clinical activity in any general medical hospital or clinic. At times, however, psychiatrists are consulted when their fellow physicians face moral dilemmas related to patient care.

The temptation is great for the psychiatrist to assume the role of moral guide or moral decision maker. But the psychiatrist's mission has traditionally been one of helping patients arrive at decisions autonomously, of providing the opportunity to discuss and explore complex issues and feelings in a nonjudgmental setting. It would seem imprudent for the psychiatrist . . . to lend authority to another physician in influencing a patient's decisions (so as to conform to that physician's desires) when the patient is competent to choose among morally defensible options.

— From "Sounding Board: Psychiatric Consultation Masking Moral Dilemmas in Medicine" by M. Perl, M.B., B.S., and E. E. Shelp, Ph.D., in *The New England Journal of Medicine*, September 2, 1982.

The Natural History of Root Entrapment Syndrome

R.W. PORTER, C. HIBBERT, and C. EVANS

An attempt was made to recognize entrapment of the lumbar root within the root canal using four criteria; (1) severe, constant root pain to the lower leg, (2) pain unrelieved by bed rest, (3) minimal tension signs, and (4) patients over 40 years of age. Two hundred forty-nine patients fulfilled the criteria, representing 11% of patients attending a back pain clinic. Most had restricted spinal extension, but few had abnormal neurologic signs. Degenerative change was common, especially disc space reduction. Central canal size measured by ultrasound was normal, compatible with a variable past history of back pain. Patients with a long history of back pain numbered 80%, and 90.4% were managed by nonoperative means. Although 78% of these still had some root pain between 1 and 4 years after first attendance, most of them were not troubled sufficiently to have sought alternative help. [Key words: radiculopathy, entrapment, lumbar root canal, abnormal signs]

ENTRAPMENT OF THE LUMBAR NERVE ROOT laterally in the root canal, produces a clinical syndrome distinct from that of an acute disc lesion, although the pain may be in the same distribution. In root entrapment syndrome, the lesion affecting the lumbar root is believed to be in the root canal,[2,9] while the disc prolapse tends to affect the root in the central canal and in its lateral recess.

Root involvement from causes other than the acute disc lesion has been identified by electrical studies and confirmed surgically[6]; the benefits of surgical decompression of the root canal have been described by Crock[3], Venner[13], and Critchley[1]. Undoubtedly, however, some patients experience root symptoms from pathology at this site to a lesser degree and are prepared to accept their disability rather than seek a surgical remedy. Little has been recorded about the criteria of recognizing this syndrome, its incidence among back pain sufferers, nor its natural history. We have sought to identify patients with root canal pathology attending a first referral back pain clinic, and record management and progress over a period of 1–4 years.

PATIENTS AND METHODS

Sixty-one pieces of information from the history and examination and investigation of 2360 patients attending a first referral back pain clinic were stored by computer. Patients were considered to have "root entrapment syndrome"—the root probably being affected in the root canal from a chronic pathologic process—if they fulfilled the following criteria (identification was made possible by computer retrieval): (1) They had unilateral leg pain in a root distribution extending at lest to the lower calf, constant and severe. It radiated from the buttock to the posterior or outer thigh, calf, ankle, and sometimes to the foot. The pain in the foot was at times well localized to the big toe or outer foot, but was not infrequently diffuse pins and needles in the foot. The pain began proximally and extended distally in time. Pain was worse in the leg than the back. (2) In contrast to root pain of the acute disc prolapse, the pain was unrelieved by bed rest.[12] Instead, the patient would pace the floor at night and seek to keep changing position when in a chair. (3) Straight leg raising was equal or better than 70 degrees. Both Corck[3] and Leyshon et al[6] recorded minimal tension signs in patients with root canal pathology, even when symptoms were sufficiently disabling to warrant surgical decompression. (4) They were over 40 years of age. Pathologic change in the root canal is age related, and although placing into the criteria an arbitary age excluding the first four decades this may, in fact, on the one hand exclude some younger patients with root canal pathology subsequent to a juvenile disc lesion, or to a spondylolysis, and on the other hand include some older individuals with an acute disc prolapse later later in life yet whose tension signs are minimal.

These criteria were sought from the 2360 patients attending the back pain clinic, comparing the incidence with other back pain syndromes—the acute, symptomatic disc lesion using McCulloch's criteria,[8] neurogenic claudication as described by Verbiest,[14] and patients with black and referred pain who had no pain below the upper calf, straight leg raising equal or better than 90 degrees, and no abnormal neurologic signs.

Standard radiographs were examined, especially for disc space narrowing (Linden et al[7]). The central spinal canal was measured in all 2360 patients by ultrasound.[5,10] Contrast radiography was carried out only in those patients who were considered to require surgical treatment.

Subsequent prograss of patients fulfilling criteria for root entrapment syndrome was assessed by means of a postal questionnaire from those patients who first attended 1–4 years previously.

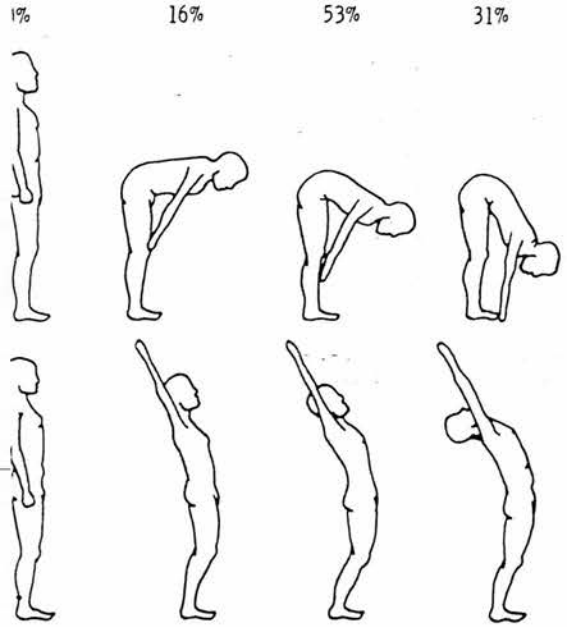
RESULTS

Root entrapment syndrome was recognized in 249 of the 2360 patients, representing 10.5% of the patients attending the back pain clinic. This contrasted with 8.7% of patients with a symptomatic disc lesion, 6.5% of patients with neurogenic claudication, and 18.2% of patients with back and referred pain. Those patients with root entrapment syndrome were 54% male, the average age being 51 years ± 7.6 years.

Root symptoms were severe, as required by the criteria, but abnormal signs were few. For instance, 84% could flex forward to touch below the knees (Figure 1), although 88% had some restriction of spinal extension, and 25% had no extension at all. The majority had no abnormal reurologic signs in the lower limbs (Table 1), that is normal reflexes, no muscle weakness or wasting, and no sensory loss. Over one-half had straight leg raising of 90 degrees (Table 2).

Submitted for publication October 20, 1981, and in revised form June 17, 1982.

The authors would like to thank the National Coal Board and the Back Pain Association for financial assistance as well as Dr. P. Butt of St. James Hospital, Leeds, for the CT scan. The authors also would like to thank Mr. Garry Swann of the Photographic Department at Doncaster Royal Infirmary, for the illustrations and Mrs. Jean Reynolds for her secretarial assistance.



. Percentage of patients able to flex to the knees, below the and to the floor, and extend legs one-third, two-thirds, and full

ny patients now with severe root pain had a long history of ıain. Eighty percent (80%) had previous back pain, the first e being on average 7.3 years before ± 6.5 years. Only 2% ad previous surgery.

liologic signs of degenerative changes were present in 80% of ıtients with root entrapment syndrome, mainly at L5–S1, isc space reduction being recognized in 56% (Table 3). The n of ultrasound measurements was the same as in the general ation though slightly less in value (Figure 2), in contrast to for patients with disc symptoms and those with neurogenic cation (Porter et al[11]).

active treatment was given for 81% of the patients. They were beginning to improve when first seen, or when the probable ɔsis was explained to them, they were prepared to await ry, and attempt to reduce the mechanical stress on the spine. e at the first consultation was reinforced by "Back School" ance in 22% of patients. Fourteen percent (14%) received an al injection, occasionally a second or third, generally se the symptoms were particularly severe. Twenty-four ts (9.6%) were treated by surgical decompression of the root This was considered necessary when pain was sufficiently ing to interfere with work and recreational activities and was ılly preceded by a lack of response to epidural injection. ulography failed to demonstrate a disc lesion, and was ılly normal, occasionally showing a thickened root. EMG s were not performed. The L5 and S1 roots both were ed at the L5–S1 level, by removal of the L5 spinous process minae, the L5 root canal being decompressed unilaterally be

1. Percentage of Patients with Abnormal Neurologic Signs

Abnormal	Reflexes	16%
	Sensation	18%
	Muscle power	5%
	Muscle bulk	3%

Table 2. Percentage of Patients with Restricted Straight Leg Raising

Straight Leg Raising	*Percentage*
100 +	6%
90	52%
80	16%
70	26%

excision of the inferior articular process of L5, and undercutting part of the superior facet of S1. If indicated from disc degeneration of L4–5 or from segmental instability on dynamic flexion and extension myelography, the L4 root canal also was decompressed.

The 24 surgically treated patients were assessed after 12 months. The root pain was completely relieved in three patients, was less severe in fifteen, and was no better in six.

Of the 249 patients with root entrapment syndrome, 90.4% were not treated surgically. The progress of these nonoperated patients first seen 1–4 years previously was assessed by a postal questionnaire, from 169 (75%) replies. The average time from first attendance was 2.2 years ± 0.9 years. Seventy-eight percent (78%) of these patients still had some root pain, and of those who had not been seen for 3 years, 76% still were not consistently free of leg pain below the knee. However, 90% of all the nonoperated patients said they were sufficiently satisfied in spite of discomfort, not to have sought medical, physiotherapy or osteopathic help elsewhere.

DISCUSSION

The disc has enjoyed an era of popularity over several decades; at one time root pain was almost synonymous with a diagnosis of disc lesion. It is now recognized that a lumbar root can be affected by other pathology and at a different site from the acute disc lesion. Though the distribution of pain is the same, its nature is different. It tends to be severe, constant, and unrelieved by bed rest. The restricted straight leg raising is not greatly affected in root entrapment syndrome, and there are often few abnormal neurologic signs. We are reluctant to rely upon the patient's subjective assessment, but diagnostic difficulty lies in this sparsity of abnormal objective signs. Many patients, however, do have distinct restriction of spinal extension, and straight leg raising may be slightly limited. Leyshon et al[6] have shown that, even when neurologic changes of the affected root are recognized by EMG, the abnormal signs are few. It is the striking contrast between the symptoms and the signs that characterizes this syndrome.

We have sought to identify patients with a root canal lesion from four criteria, believing that it is generally the cause of severe root pain, in the older patient, unrelieved by bed rest, and yet without gross limitation of straight leg raising. If these criteria are correct, a root canal lesion was responsible for 11% of patients attending a first referral back pain clinic.

Root canal pathology can occur from spondylolysis, from congenital facet hypertrophy at L5–S1, and from previous trauma to the apophyseal joint, but the high incidence of disc space resorption (Table 3) suggests that previous disc pathology is a major cause of this syndrome. Radiographs of the lumbar spine showed a greater incidence of disc space narrowing than would be expected. Linden et al,[7] when examining prospective employees, noted an increased incidence of disc space narrowing with age, affecting 11% of the population between 50 and 54 years of age. In this series, 56% of patients had some disc space narrowing, mostly at L5–S1. Disc resorption is associated with many types of pathologic change in the root canal[2] (C), the fifth lumbar root

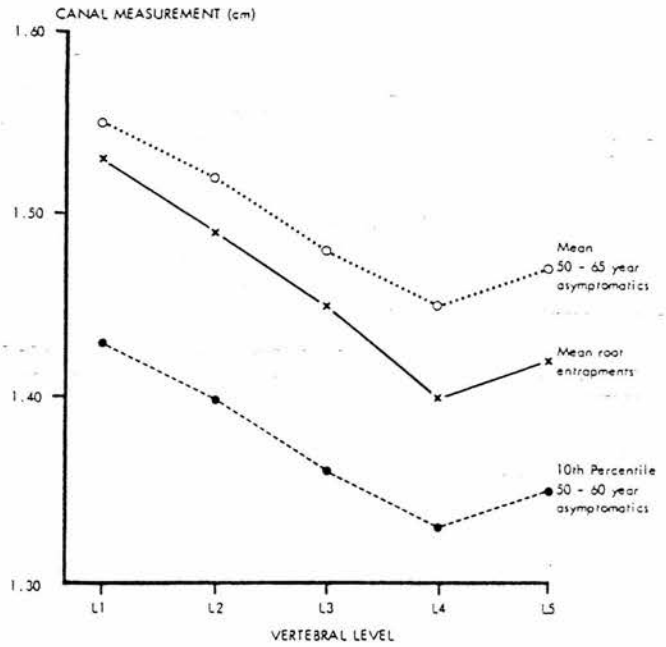


Fig 2. Mean ultrasound measurements of the canal for patients with root entrapment syndrome, compared with asymptomatic subjects.

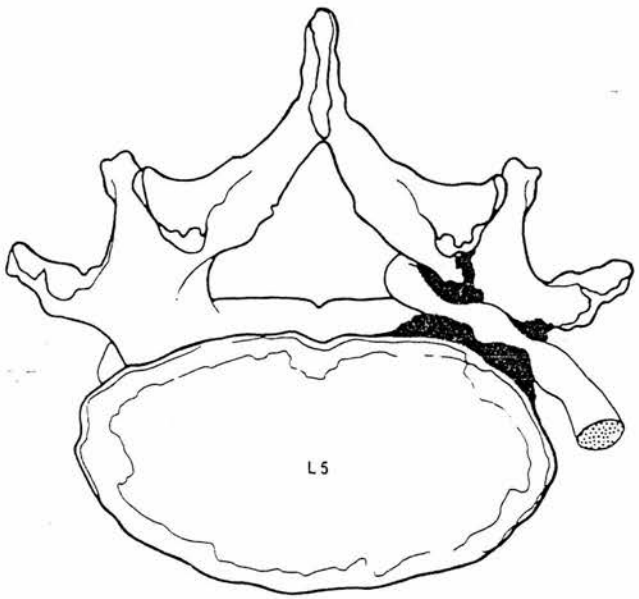


Fig 3. Diagram to show encroachment of degenerative changes into the root canal.

being at risk beneath the broad pedicle of L5 and the first sacral root behind the superior sacral facet. The disc lesion, many years before, can alter the normal anatomy of the root canal, especially if L5 settles onto S1 with reduction of the disc space. Bony degenerative changes encroach into the root canal from the apophyseal joint and from the posterior vertebral bar (Figure 3). The lesion is frequently too far lateral for demonstration by radiculogram. Ligamentous changes and soft tissue scarring from the previous disc lesion also reduce the available space for the nerve root. This affected dynamically by extension, when the superior facet approaches the posterior bar of the proximal vertebra, and by the abnormal segmental movement of a degenerative spondylolisthesis.

Bony encroachment of the root canal is well demonstrated by CT scan (Figure 4). It can be symptomless, but is clinically useful when it correlates with the symptoms and signs. A previous history of back pain, often many years before, was recorded in 80% of the patients. Now they presented with a new problem of root pain. Probably for many, their natural history of root entrapment began years before with a disc lesion, and the degenerative changes of bone and soft tissue so altered the root canal that the nerve root with restricted space or restricted excursion became vulnerable to trauma.

The normal pattern of ultrasound measurement suggests that the central canal of patients with this syndrome differs little from the general population (Figure 2). Ultrasound, of course, does not permit measurement of the root canal. Severe root pain from root canal pathology in middle life, is not incompatible with a relatively symptomless disc years before, if the central canal was sufficiently wide for the root to escape earlier damage. Many patients with root entrapment syndrome probably share the common etiology of a previous disc lesion, but the severity of the original symptoms will not depend unreasonably on the many factors affecting acute disc symptoms, least of all the canal size. There is no evidence, therefore, from ultrasound measurements that root canal symptoms are related to central canal size.

Once root symptoms have become sufficiently severe for the patient to seek a hospital opinion, it is likely to become a chronic problem. Only one in four will be free of symptoms a year later. Seventy-six (76%) of patients replying to the questionnaire admitted to some leg pain in a root distribution a year after their last hospital visit. There was evidence, however, that the pain was

Table 3. Percentage of Patients with Disc Space Narrowing and the Level Affected*

	L3-L4	L4-L5	L5-S1
Disc space narrowing alone		17%	30%
Degenerative spondylolysis	1%	5%	3%
Retro-spondylolysis	1%	1%	1%

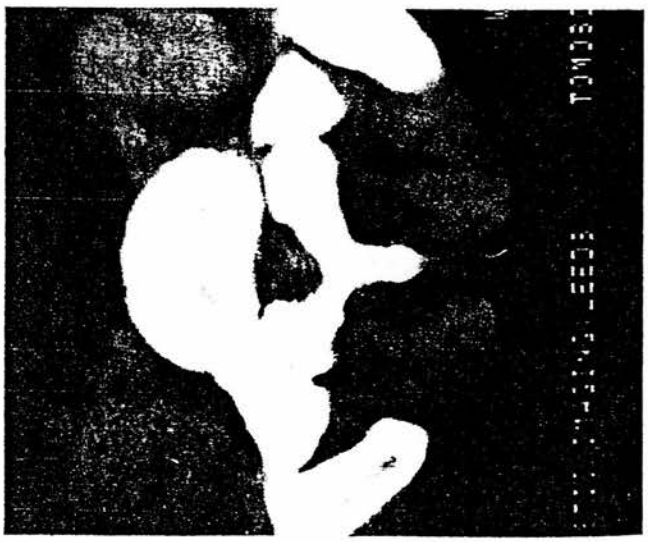


Fig 4. CT scan of bony encroachment of root canal at L5.

less severe, intermittent, and manageable. Patients presented with this syndrome less frequently in later life, with only 6% being over 65 years of age. It is probable that symptoms do settle with time, either completely or to manageable proportions.

The majority of patients were given no active treatment for the root pain because there is no evidence that any particular method of nonoperative treatment affects its natural history. Fourteen percent (14%) did receive one or more epidural injections, but their response to the questionnaire was not significantly different to those who had no treatment at all.

Root entrapment syndrome is a relatively common back pain syndrome to be encountered in hospital practice. It is probably preceded by a disc lesion, with or without major symptoms, but subsequent disc resorption over the years altering the root canal anatomy and making the root vulnerable to trauma. Once established, root entrapment syndrome tends to become a chronic problem, manageable for the majority, but requiring surgical decompression for the few.

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Address reprint requests to

R. W. Porter, MD
Consultant Orthopaedic Surgeon
Doncaster Health Authority
Doncaster Royal Infirmary
Doncaster, DN2 5LT
England

Accepted for publication November 1, 1982.

Spinal Stenosis

Richard W. Porter

THE VERTEBRAL canal is a complex anatomical space with considerable regional, racial, and individual variations. Spinal stenosis results from pathologic change so reducing the canal that limited space becomes a significant factor in the development of back and/or leg pain.

ANATOMY

The vertebral canal is arbitrarily divided into the central canal and the root canal (Fig 1). At the pedicular level, the central canal is bounded laterally by the two pedicles, anteriorly by the posterior surface of the vertebral body, and posteriorly by the cranial aspect of the laminae and the medial aspect of the superior apophyseal joints. Between each pedicular level, the central canal has the ligamentum flavum posteriorly, the vertebral body and disc anteriorly, and an artificial boundary laterally. It extends from one pedicular level to the next, and contains the cauda equina within the dural envelope.

The root canal is that space lateral to the central canal, in the intervertebral region between the pedicular levels. Anteriorly it is bounded by the posterior surface of the vertebral body above, the posterolateral aspect of the disc, and the posterior surface of the vertebral body below. Superiorly, it is bounded by the pedicle of the vertebra above, and inferiorly by the pedicle of the vertebra below. Its posterior relations are the lateral aspect of the lamina, and the superior articulation of the apophyseal joint of the vertebra below. Medially, it opens into the central canal, and laterally it ends at the intervertebral foramen.

The lateral recess is that lateral part of the central vertebral canal at the pedicular level anterior to the medial aspect of the superior apophyseal facet.[1] It is only trefoil-shaped canals that have a lateral recess, because dome-shaped canals have a continuous concave posterior surface to the canal with no lateral recess at all (Fig 2). The root canal has been loosely, but inaccurately, called the lateral recess.

In the sagittal plane, the central canal pursues a serpentine course (Fig 3). It is indented posteriorly by the cranial aspect of each lamina, and anteriorly by each intervertebral disc. In the coronal plane, the vertebral canal is narrowest at each pedicular level, widening into each root canal, and narrowing again at the next pedicular level.

The general dimensions of the vertebral canal tend to follow a constant pattern from the first to the fifth lumbar levels.[2] In the midsagittal plane at the pedicular level, it is generally widest at L1, reducing to L4, and widening again at L5. In the coronal plane, the interpedicular diameter measurements are fairly constant from L1 to L3, widening a little at L4, and then considerably at L5. The total cross-sectional area at the pedicular levels reduces from L1 to L4, and then increases at L5 to an area equal or even greater than at L1.

The range of midsagittal and interpedicular diameters is greatest at L5 with more variability in both size and shape of the canal. Eisenstein[3] described 14% of canals trefoil at L5. The trefoil configuration is a relative term caused by posterolateral identification of the neural arch. Two extremes of shape of L5 are shown in Figs 4 and 5, but in fact, no two canals are the same.

Significance of Anatomical Variations

An obvious argument against the vertebral canal being a significant factor in the pathogenesis of back pain is that although the canal does vary in size and shape from one individual to another, this variation is probably adequate for each subject. In other words, the neural contents may be greater with a large canal and less with a small canal. The lordotic curve and the range of movement for one individual may also be reflected in the canal's dimension and be satisfactory for that person. The neural contents undoubtedly influence the dimensions of the ver-

From the Department of Orthopaedic Surgery, Doncaster Royal Infirmary, Doncaster, England.

Address reprint requests to Richard W. Porter, MD, FRCS, FRCSE, Dept of Orthopaedic Surgery, Doncaster Royal Infirmary, Armthorpe Rd, Doncaster DN2 5LT, England.

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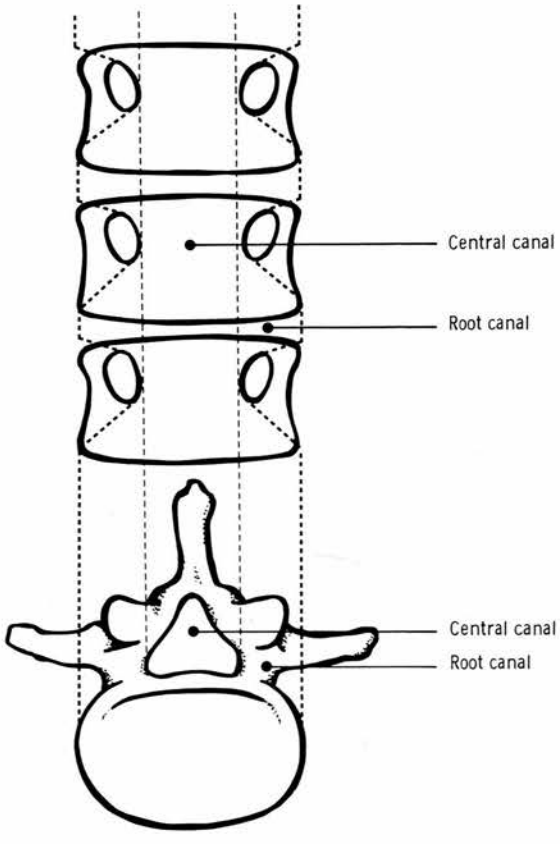
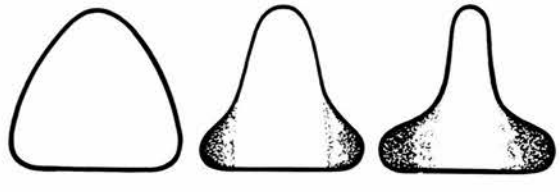


Fig 1. Diagram of the vertebral canal showing the central and root canals.

tebral canal to some degree,[4] much as the brain determines epigenetically the size of the skull in hydrocephalus and microcephalus.[5] In the spine, however, this is not the only factor. There must be other influences at work, both genetic and environmental, because clinical observations show great variation in the proportion of intradural to extradural space. We know from CT scans and surgical experience that many patients with back pain have a canal with a small midsagittal



L5: Central Canal

Fig 2. Diagram to show that dome-shaped canals do not have a lateral recess, that a mildly trefoiled canal has a shallow lateral recess, and that there is a deep lateral recess in markedly trefoil-shaped canals.

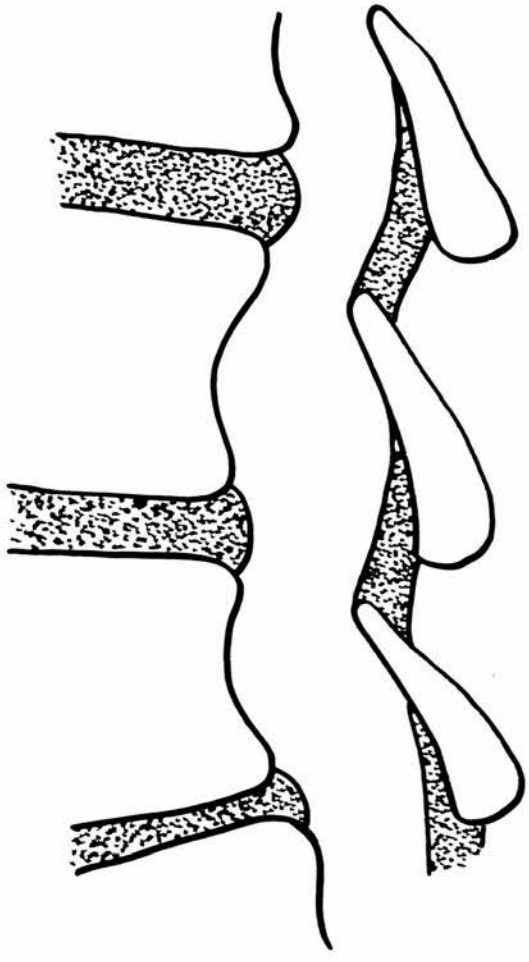


Fig 3. Diagram of vertebral canal showing the serpentine shape, being narrowest at the cranial end of the laminae.

Fig 4. Photograph of fifth lumbar vertebra showing a large dome-shaped central canal.

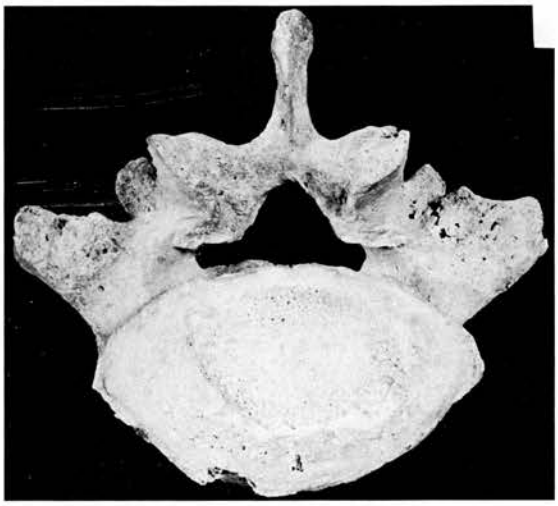


Fig 5. A fifth lumbar vertebra with a shallow trefoil-shaped central canal.

diameter, a trefoil shape, and a tightly packed cauda equina. In fact, the canal will often determine the pattern of symptoms from any particular pathology.

Sarpyener,[6] Schlesinger and Taveras,[7] Verbeist,[8] Epstein et al,[9] Ehni,[10] Salibi,[11] Porter et al,[12] Kornberg and Rechtine,[13] and many others have described how spinal pathology in the presence of a shallow vertebral canal can produce a variety of back pain syndromes. It is the sagittal diameter, rather than the interpedicular diameter, that is critical, the exception being in achondroplasia when a narrow interpedicular diameter can cause stenotic symptoms.[14,15] The argument that the canal size is insignificant because it reflects the contents and is adequate for that individual runs contrary to anatomical and clinic observations.

NEUROGENIC CLAUDICATION: CENTRAL CANAL STENOSIS

The term 'claudication of the spinal cord' was firs used by De Jerine[16] when describing three patients with claudication symptoms but normal peripheral pulses. Van Gelderen[17] reported a patient with symptoms of lumbar root compression that appeared on walking and was relieved by rest, which he thought was due to thickening of the ligamentum flavum. Bergmark[18] described 'intermittent spinal claudication,' attributing a neurospinal origin to the walking pains of two patients. It was Verbiest in 1954[8] who recognised that structural narrowing of the vertebral canal could compress the cauda equina and produce claudication symptoms, and since that time there have been numerous publications on the subject.[19–23]

Clinical Presentation

This intriguing syndrome usually affects men over 50 years of age who have been heavy manual workers. They complain of discomfort in the legs when walking, affecting both legs equally, usually in the thighs, calves, and feet. Describing the discomfort is difficult, but they describe the legs as feeling 'heavy' or 'tired,' as though it is difficult to drag one leg after the other. There is usually a threshold distance when the discomfort develops and a tolerance when they have to stop; the tolerance is about twice the threshold. The distance can vary during the day, from one day to the next, and even during one stretch of walking. The second period of walking can be longer than the first after a short rest. Often they find they gradually reduce the walking speed and stoop forwards until they finally stop—the stoop test[24] (Fig 6). They will lean forwards on a wall, or stoop forwards and tie up a shoe lace to save embarrassment, and after a few minutes the feeling in the legs recovers sufficiently for them to start walking again. The flexed position seems to relieve the discomfort and for that reason they may be able to walk better up a hill leaning forwards than down a hill leaning back. Extending the spine in the standing position can precipitate symptoms in the severely disabled patient. Patients say they can cycle for miles and climb a ladder and stairs, but not come down stairs easily. As the condition progresses, the walking distance reduces, sometimes to only 20 yards. It is probably not neurogenic claudication if a person can walk more than half a mile at a reasonable pace without having to stop. Nights are usually troublesome, with sleep being disturbed by restless legs, night cramps, and the need to often get up and walk about.

Back pain is a common, but not invariable, accompaniment. There is usually a long history of back pain, sometimes with previous surgery, and claudication symptoms are present for a number of years before help is sought.

Apart from the spinal posture, the examination is remarkable for its lack of gross abnormality. Patients may be able to flex well forward with

Fig 6. The typical stooped posture of a patient with neurogenic claudication. At the threshold of walking pain, patients stoop forwards—the 'stoop test'.

extended knees, though lumbar extension is usually absent. In fact, it may be difficult to even stand erect, with these patients adopting a 'simian stance',[25] with hips and knees slightly flexed. This can be corrected with an effort, but it quickly returns as they relax. If this posture is not present at rest, it tends to develop with walking, the patient gradually stooping further forward until he has to stop.

The stoop test makes use of this phenomenon in diagnosing claudication of neurogenic origin, the leg symptoms being relieved by stooping forwards at the point of walking tolerance, and returning by standing upright again. The lumbar spine is often tender over several segments, straight leg raising is generally full, the reflexes are normal, and the power and sensation are often normal.

It has been suggested that reexamination after exercise alters the neurologic examination but this is not our experience. The peripheral circulation can be normal, but not infrequently arterial disease will coexist. A treadmill enables us to establish an objective record of walking pain, noting the distance at which symptoms develop, the distribution of discomfort, the speed of walking, the changing posture, and the tolerance (Fig 7). The impression gained from the patient's history can be completely different from an objective assessment of walking. When measuring a response to treatment, a treadmill is invaluable.

A plain radiograph may raise the suspicion of a shallow vertebral canal and perhaps show a degenerative spondylolisthesis, present in half the men with neurogenic claudication. A myelogram is essential to confirm the diagnosis. It will show one or several segmental filling defects, or even a complete block (Figs 8 and 9). The lack of space in the central canal can make injection of the contrast medium very difficult, and the myelogram may have to be abandoned at the lower lumbar level.[10,26] When myelography is difficult, epidural venography will show the extent of stenosis.[27]

Fig 7. A treadmill is useful for objective evidence of walking tolerance and threshold, and in order to observe posture.

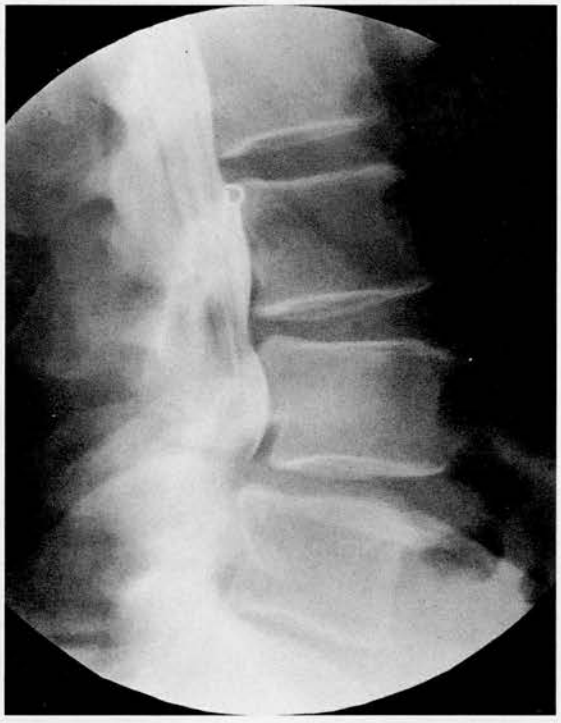


Fig 8. A radiculogram of a 63-year-old man with degenerative spondylolisthesis and symptoms of neurogenic claudication, showing a complete block of the metrizamide column at L4/5.

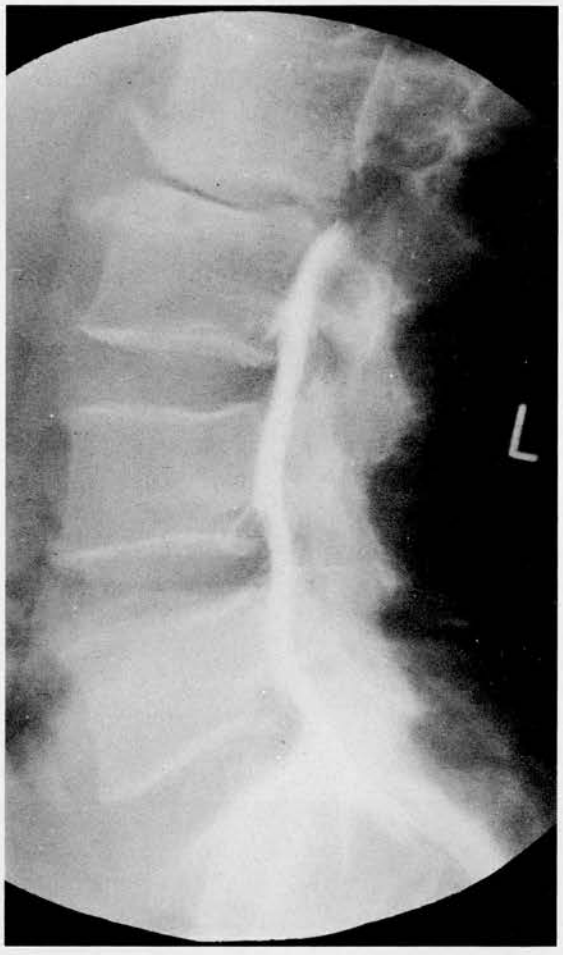


Fig 9. A lateral radiculogram of a 58-year-old man with disc degeneration at L2/3 and retrolisthesis occluding the metrizamide column. He has a narrow vertebral canal, but has only had neurogenic claudication for 4 years.

CT complements radiculography,[28] demonstrating the canal's midsagittal diameter, cross-sectional area, and shape. We must examine a longer segment of canal than the standard L3 to S1 for disc problems. This increases the radiation dosage, but it is outweighed by its advantages. Bony encroachment, thickened ligamentum flavum, and disc material may be evident. The extent of undercutting facetectomy can be predicted.

Ultrasound measurements confirm a reduced midsagittal diameter of the vertebral canal; 57% of our patients with neurogenic claudication had measurements below the tenth percentile and 42% below the fifth percentile. A narrow canal supports the diagnosis, but obviously narrow canals exist without neurogenic claudication symptoms. A wide canal is incompatible with the diagnosis.

Differential Diagnosis

Intermittent claudication, a phrase coined by Charcot in 1858 to describe ischaemic pain from peripheral vascular disease, is difficult to distinguish from neurogenic claudication by the history alone. The bicycle test of van Gelderen,[17] modified by Dyck and Doyle,[21] is helpful in differentiating between these two types of claudication (Figs 10 and 11). Impalpable peripheral pulses and femoral bruits will suggest periperal vascular disease. If clinical examination is difficult, a Doppler scan may be more objective, but it can take an arteriogram to be certain of the relative importance of the peripheral arterial circulation. Cerebral somatosensory-evoked potentials after walking may help to differentiate neurogenic from vascular intermittent claudication.[29] To confuse the issue, intermittent claudication and neurogenic claudication often coexist.[30]

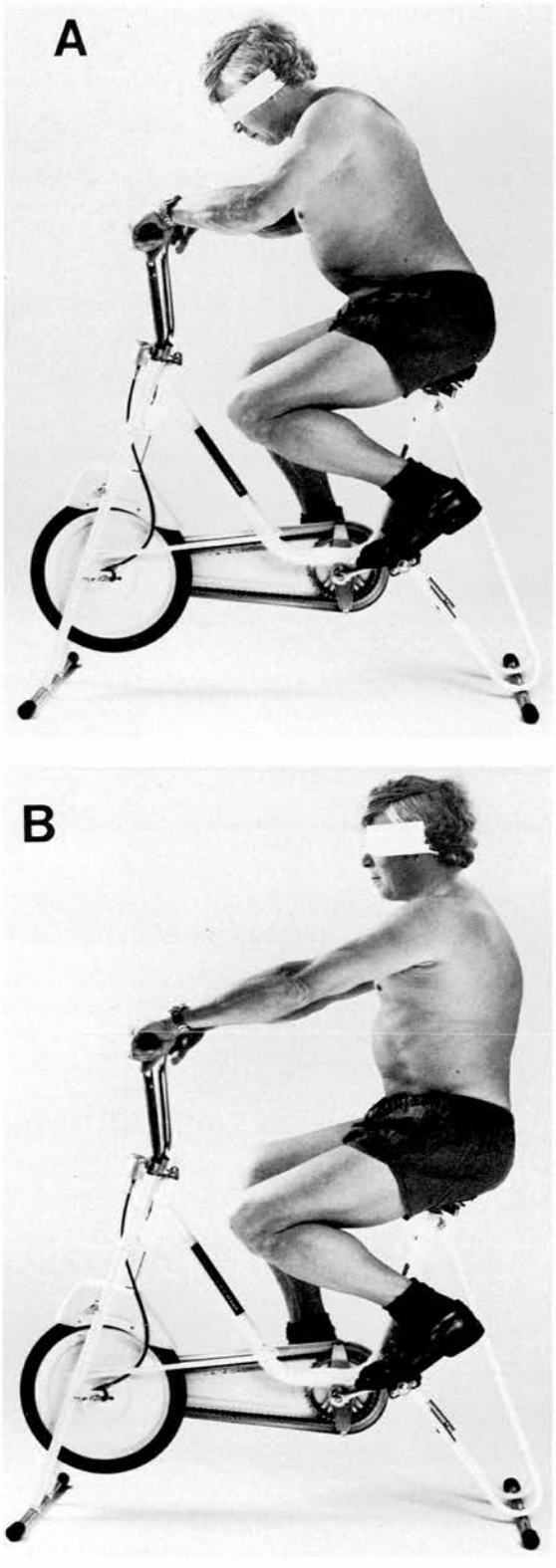


Fig 10. Van Gelderen's cycle test. (A) Cycling in the flexed posture is more comfortable than with (B) an extended spine (see also Fig 11).

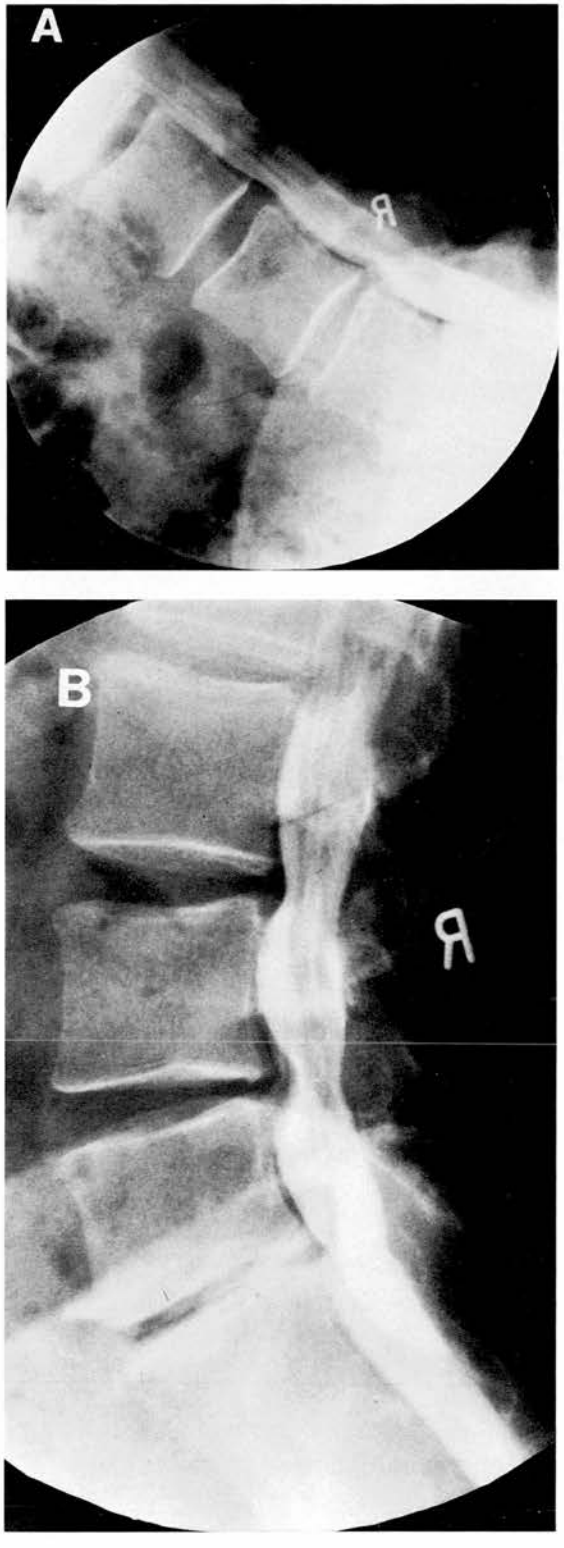


Fig 11. (A) A myelogram in flexion demonstrates an adequate metrizamide column but this is compromised by (B) anterior segmental indentations in extension.

Lamerton et al[31] described sciatic claudication, an insufficiency of the inferior gluteal artery producing claudication in a sciatic distribution from ischaemia of the sciatic nerve.

Referred pain from the lower lumbar region in the buttocks and thighs, even to the upper calves, can be aggravated by walking. Eighteen percent of Crock's patients with isolated lumbar disc resorption[32] had increasing leg pain or paraesthesia on walking distances up to 500 yards.

Some types of root pain and multiple root pathology are made worse by walking,[33] probably if segmental instability is a factor in producing root symptoms, and if venous engorgement contributes to restriction of space in the root canal.

Claudication pain is sometimes a symptom of distress. Abnormal pain behaviour patterns are common in patients who have had a long history of back pain, and not infrequently, a symptom inappropriate to the underlying organic problem in the spine is pain in the legs when walking. There are usually inappropriate signs also.[34] It is difficult to accurately assess the claudicating patient who also has a litigation problem.

There are other less common causes of claudication pain. Venous claudication can follow thrombosis before the colateral circulation takes over. Myxoedema claudication results from the limited potential of muscle to increase its metabolism with exercise. Rarely a localised deep arteriovenous fistula will present with aching and pain in the legs aggravated by exercise and standing.

Pathology

Verbiest[8] recognised that neurogenic claudication was associated with a shallow vertebral canal. In fact, the term spinal stenosis has unfortunately become synonymous with neurogenic claudication, when in fact, a shallow canal is only one factor in the pathology. Symptoms develop after middle life but there is no evidence that the vertebral canal becomes narrower with age. There can be a little encroachment into the canal from hypertrophy of the apophyseal joints and marginal osteophyte formation, but this is more into the root canal than the central canal. Also, posterior vertebral bar formation on the lower and upper posterior margins of the vertebral bodies can reduce the sagittal diameter to some degree. In general, however, the central canal retains the same cross-sectional diameter throughout life. An individual with spinal stenosis and neurogenic claudication has, therefore, had a narrow canal for many years before the development of leg symptoms,[11,35,36] and many patients with stenotic canals never have claudication pain. The canal is, therefore, only one factor in the pathology.

A second factor is degenerative disease of the lumbar spine associated with heavy manual work. The majority of patients with neurogenic claudication have been involved in heavy work. Few have been sedentary workers. It would seem that the generalised accumulative effects of the mechanical stress of labouring work plays a part in pathology rather than the localised degenerative process from one disc insult in the earlier life of a sedentary worker. The high male incidence of nine to one may be due to manual work, or indicate that hormonal factors are significant.

Vertebral displacement with an intact neural arch will critically narrow an already small canal (Fig 8). Degenerative spondylolisthesis effectively reduces the canal size at the level of displacement.[37,38] Although degenerative spondylolisthesis is more common in women, half of the men with neurogenic claudication in our series had a degenerative spondylolisthesis.[39] Women with degenerative displacement rarely develop claudication symptoms (Fig 12).

Neurogenic claudication must be very unusual in children, but Birkensfield and Kasdon[40] described it in two adolescent boys with congenital lumbar ridges producing ventral defects on myelography.

Symptoms are probably the result of inadequate oxygenation of the cauda equina, but the mechanism is at present purely speculative. There may be arterial ischaemia or venous engorgement, which just permits adequate nerve function at rest, but inadequate function during exercise. The fact that patients are generally over 50 years of age, when arteriosclerosis is becoming more common, is compatible with an ischaemic component to the pathology. Many claudicating patients have a stenosis at L3/4 level. This may have a neuroischaemic explanation. The proximal third of the cauda equina is an area at risk, being supplied by an astomosis of both proximal and distal radicular arteries. If the supply is just adequate for its needs,[41] then

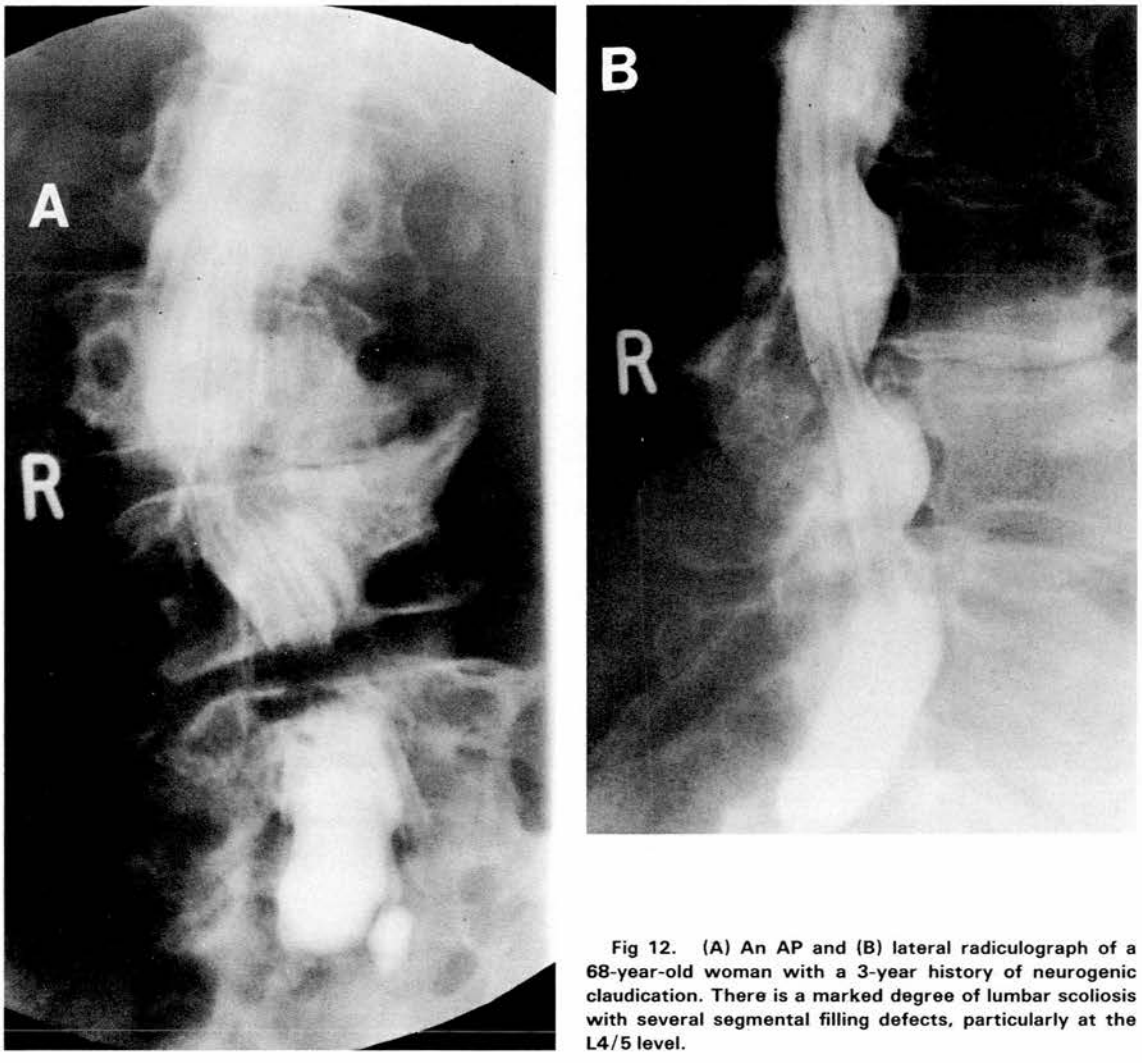


Fig 12. (A) An AP and (B) lateral radiculograph of a 68-year-old woman with a 3-year history of neurogenic claudication. There is a marked degree of lumbar scoliosis with several segmental filling defects, particularly at the L4/5 level.

deprivation could precipitate claudication symptoms at this level.

Fifty-two years ago, Reichert et al[42] described ischaemia of the spinal cord due to arteriosclerotic involvement of the lumbar arteries, giving weakness to the lower limbs on exertion. He noted similar temporary weakness in dogs by ligation of the lumbar artery. Cauda equina ischaemia may have a similar mechanism.

There is probably localised vasodilatation of the radicular arteries in response to exercise. Exercising the single limb of a mouse will produce vasodilatation of the ipsilateral region of the spinal cord.[43] In addition, the selective paralysis in poliomyelitis is probably related to the vasodilatation of the anterior horn in response to muscular activity in the preparalytic stage of the disease.[44] Should the vessels of the cauda equina likewise dilate with exercise, they will be vulnerable to ischaemia if space is at a premium. The ischaemia factor seems to be particularly relevant in some patients.

A vascular steal syndrome could explain the claudication symptoms of some patients with claudication and Paget's disease, when the vertebral canal may not appear significantly narrow.[45–47]

Iatrogenic neurogenic claudication can follow spinal surgery after a spinal fusion or discectomy. The patient is at risk whose disc is excised from a narrow canal, or whose developmentally narrow canal is fused. It was previously thought that bony ingrowth from the posterior fusion mass compromised the canal causing symptoms,

but it is more likely that symptoms arise at the segment proximal to the fusion. It can become unstable, and the narrow canal, segmental instability, and ischaemia from iatrogenic scarring combine to produce symptoms.

Claudication is related to the dynamic activity of walking. There are probably three processes caused by walking that precipitate symptoms in a cauda equina already deprived and vulnerable. Segmental rotation which accompanies walking, especially segmental instability, will reduce the available space in an already narrow canal. Secondly, the increased venous return from the exercising lower limbs will be accompanied by engorgement of the pelvic veins and of Batson's venous plexus, reducing the available space for the cauda equina. Thirdly, the arterial system of the cauda equina must respond to the increasing demands of exercise, and may do so inadequately when space is limited. Nutrients fail to reach the nerve roots, metabolites are not removed, and function is affected. The first process is probably the most critical because it is not the exercise of the lower limbs that produces the symptoms, but rather the torque and posture of the spine at the time of that exercise. A fourth mechanism may be responsible if there exists a lumbar artery shunt. In health, the intraosseous arterial branches of the lumbar arteries may vasoconstrict and shunt blood to the radicular branch during the activity of walking or running. If there is already incipient ischaemia of the nerve roots from restricted space in the vertebral canal, and if in addition the vertebral shunt should fail as a result of bony degenerative pathology, neurogenic claudication symptoms would develop in times of physiological stress.

As yet, we do not know how important is the cerebrospinal fluid in the normal function of the cauda equina. It is possible that this is the key to the mechanism of the symptoms of neurogenic claudication.[48] The cauda equina probably needs to be bathed in a free circulation of cerebrospinal fluid for its nutrition, for removal of metabolites, and for insultation. When the fluid is deficient because of reduced space in the canal, and especially when there is a closed sac of fluid distally, the circulation will be deficient. Magnaes[49] was able to record a high cerebrospinal fluid pressure in claudicating patients caudal to a stenosed segment and related to posture. One can imagine a claudicating patient stopping after a few hundred yards, leaning forwards on a wall for a minute, and as the fluid above the stenosis is permitted to exchange with the closed sac below, the discomfort rapidly clearing from the legs. It is interesting that neurogenic claudication does not usually occur from stenosis at L5/S1 alone. It is usually from segmental narrowing at the two or three more proximal segments that permits a closed sac of dura distally. If the sac is too large from stenosis at the thoracolumbar level, claudication symptoms are rare.

Management

If symptoms of claudication are sufficiently severe, they are generally relieved by surgical decompression.[50–52] Most patients are immediately impressed with the improved sensation in their legs and are soon walking long distances. Not a few relapse as a laminectomy membrane of fibrous tissue develops over the posterior dura. Their walking distance again becomes reduced. Verbiest recorded that 70 out of 74 of his patients with neurogenic claudication were relieved by decompression; this probably depends on careful patient selection and adequate decompression. Russin and Sheldon[53] and Lassale et al[54] likewise recorded excellent long-term follow-up results with decompression for stenotic symptoms but back pain is the most common persisting problem.[55] Advanced age is no contraindication to decompression: it will often improve the quality of life for the elderly.[35] Some results are less satisfactory and in these it is claimed that decompression was less than adequate, that epidural scarring spoilt the results, or that long-standing ischaemia of the nerve roots became irreversible. Most operative series have a hard core of failures.

Some patients with neurogenic claudication will respond to calcitonin.[39] It is a useful first line of treatment, when 100 units administered subcutaneously four times a week will often produce a response within 4 weeks. This nonoperative management is helpful in the hope of avoiding surgery in the patient unfit for anaesthetic and for the patient who has had a postoperative recurrence of claudication.

The mechanism of response to calcitonin, if not placebo, is probably vascular. It is too rapid to be due to remodelling of the bone. Canal

measurements using ultrasound have not shown any increase in diameter in 16 responding patients with neurogenic claudication, nor in three patients whose Paget's paraparesis recovered with calcitonin.[46] Likewise a response in postoperative patients who have relapsed after a previous decompression is not likely to be due to bony remodelling. It could be a placebo response. Calcitonin is also a powerful central analgesic drug. Calcitonin may, however, improve the radicular circulation by selectively reducing the intraosseous blood supply via the branches of the lumbar arteries. This drug does reduce skeletal blood flow,[56] and it could stimulate a shunt mechanism providing for a deprived cauda equina. Venous blood would likewise be reduced, draining from the vertebral bodies into Batson's extradural plexus, and the neural elements then enjoy more space.

ROOT ENTRAPMENT SYNDROME: LATERAL CANAL STENOSIS

The site of the lesion can be quite variable, but the root most commonly involved is the fifth lumbar, probably because of the frequency of degenerative change at L5/S1, and the length of the root canal at L5, inferior to the broad pedicle (Fig 13). The L4 and L3 roots are occasionally affected in their root canals. In the central canal, the L5 root can be involved from degenerative change at the L4/5 disc space (Figs 14 and 15)

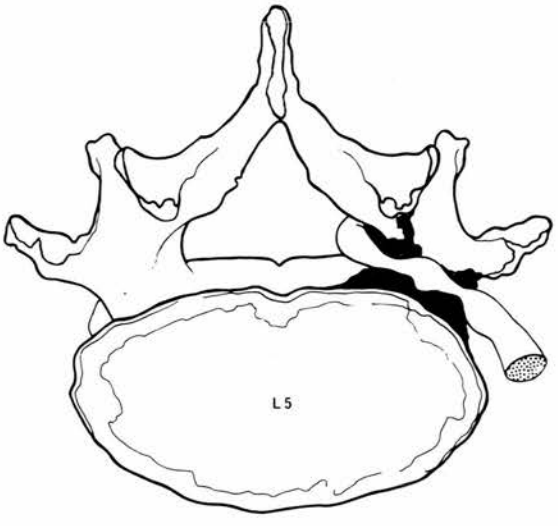


Fig 13. A diagram of the fifth lumbar root canal showing how degenerative change from the vertebral body and apophyseal joint can compromise the root.

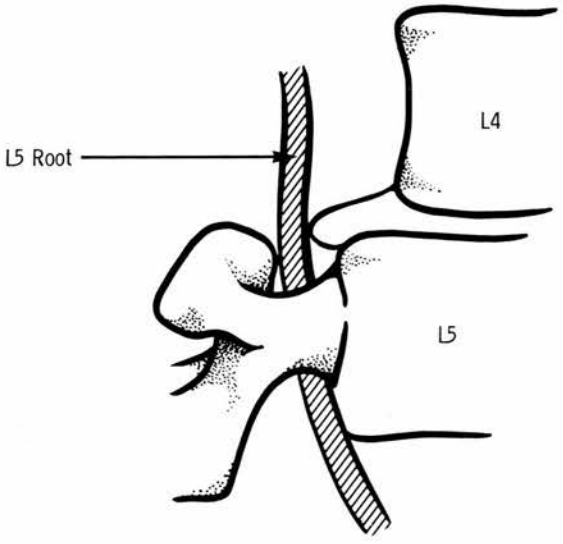


Fig 14. A diagram to show that the L5 root can be involved in the central canal at L4/5 level in the presence of degenerative spondylolisthesis.

and the S1 root anterior to the cranial lip of the upper sacral lamina.[51]

The original size of the root canal is variable and must be highly significant. The contents of the root canal also vary, the nerve root and ganglion measuring between 4 to 7 mm in diameter.[57] Discrepancy between the root canal and its contents makes the root vulnerable to bony and soft tissue encroachment. The root canal will be reduced by a posterior vertebral bar on the inferolateral border of the body, by osteophytes

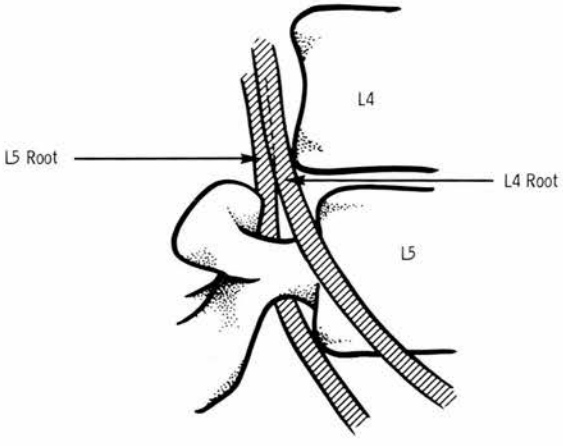


Fig 15. A diagram to show that a retrolisthesis, or rotational displacement, of L4 can involve the fourth lumbar root in the root canal, and also the fifth root in the lateral recess of the central canal.

from the margins of the apophyseal joints (Figs 16 and 17), or by a hypertrophied uncinate process. The overhanging medial lip of the superior facet is a common site for 'subarticular entrapment' with the root tightly stretched against the pedicle in the lateral recess. Bony encroachment may follow ossification of spinal ligaments. In spite of the gross degenerative change sometimes encountered in the lower lumbar spine, it is surprising how the nerve root tunnel is always preserved. It may be reduced but never occluded. If a CT scan should give the impression that the canal is nonexistent, this is but an artefact of the mathematical display.

Soft tissue involvement of the root canal adds to the bony encroachment. Organisation of an annulus after a disc protrusion, or fibrosis of extruded or sequestrated nucleus, reduces the available space for the root. The posterior longitudinal ligament can thicken, the ligamentum flavum infold,[58] the apophyseal joint capsule hypertrophy, and soft tissue of a lytic pars proliferate until space for the nerve root is at a premium (Fig 18). Venous engorgement in the root canal may critically affect the function of the nerve root.

Segmental movement of the spine adds a dynamic factor.[59] Extension and rotation further reduce available space, and are both limited and painful in this syndrome. It becomes particularly significant when there is posterior or rotation displacement of the vertebra.[60] Degenerative spondylolisthesis can produce the same symptoms. Several dynamic factors can be responsible for symptoms. Postural movement, especially extension, can compromise the root and precipitate symptoms. The activity of walking can produce root symptoms by both intervertebral segmental rotatory movement and by the epidural venous engorgement associated with exercise. The dynamic factors involved in normal root excursion in activities such as walking and bending can assume significance in a pathologic root.

Just as the tunnel is never occluded, the root is never trapped. There is some excursion, even if at operation the root gives the impression of being tight. The lumbar roots normally have an excursion of a few millimetres limited by proximal and distal attachments.[61] These attachments probably make the root vulnerable to traction symp-

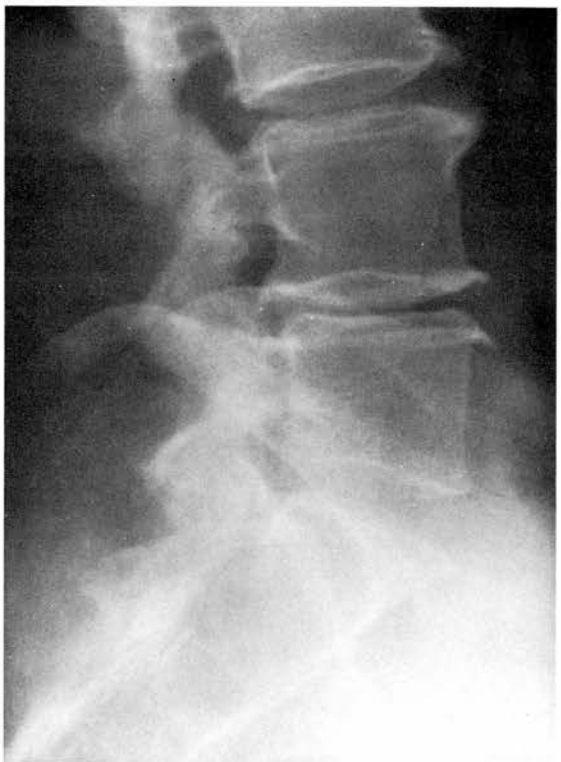


Fig 17. Lateral radiograph showing a reduction in cross-sectional area of the root canal in the presence of disc degeneration (reduction of disc space and traction spurs).

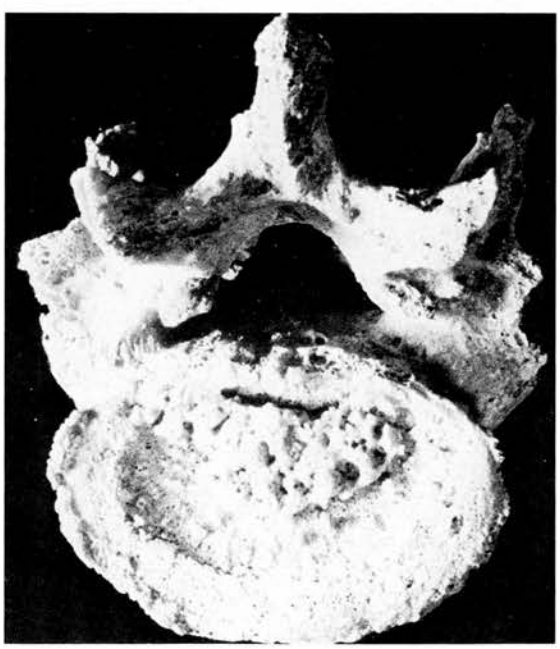


Fig 16. Photograph of fifth lumbar vertebra showing osteophyte encroaching into the root canal.

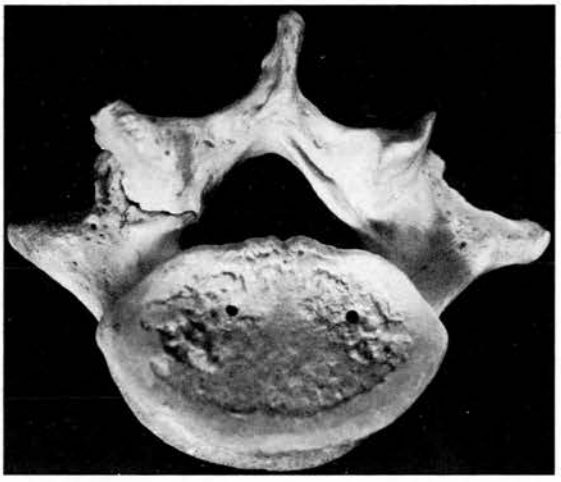


Fig 18. Specimen of the fifth lumbar vertebra with a unilateral spondylolysis demonstrating its proximity to the lumbar root. Osteophytes and soft tissue hypertrophy can reduce the space in the root canal.

toms in the presence of pathologic change. A mobile root in a restricted space will produce root irritation and ischaemia. Friction on a tethered root or anomalous root with limited excursion will have similar results.[62] The root then becomes considerably thicker, harder, and inelastic from perineural fibrosis.

Wiltse et al[63] described an unusual cause of root entrapment syndrome in which gross displacement of an isthmic spondylolisthesis compresses the L5 root between the fifth lumbar transverse process and the alar of the sacrum—'the far out syndrome.'

There are many pathologic changes that can cause lumbar root pain in the middle-aged and older patient. The most common site of these changes is in the root canal, related to the effects of disc pathology of a previous decade.

Clinical Presentation

Though the pain from root entrapment is in the same distribution as the sciatica from a disc lesion, from the buttock, thigh, and calf to the foot, its character is different. It is described as a severe pain often unremitting day and night. Whilst the pain from a disc is frequently relieved by lying down, this pain is so troublesome at night that the patient will walk about. Sitting is uncomfortable, driving far impossible, as though the whole length of the sciatic nerve is oversensitive. Unlike root pain of disc origin, it is unaffected by coughing and sneezing.

The periodicity of the pain is variable. One patient may experience constant severe pain, and presents at the consulting room after many sleepless nights. Another may have mild pain with episodes of severe pain in relation to posture, especially to sitting or standing for long. Another may say that walking is the main cause of pain. If only walking produces pain, one should suspect a vascular component, either venous engorgement of the root canal or arterial insufficiency of the root.

The past history is also variable. There has usually been previous disc pathology, but the previous disc symptoms may have been either classical, with root pain, or have produced only back pain. They may even have been entirely occult. The degree of original disc symptoms years before depends much on the size of the central vertebral canal. There may have been no symptoms at all from a disc protrusion into a wide dome-shaped central canal, but with disc space narrowing over the years, bony and soft tissue degeneration, and perhaps slight vertebral displacement, the root becomes compromised. The very first symptom of the silent lesion years before is now severe root pain from root canal entrapment.

The progress of the root pain, once it has developed, is unpredictable. It can develop and subside in weeks, months, or years, and patients may therefore present with long or short histories. It has no typical pattern, sometimes being severe and gradually resolving, and at other times getting steadily worse, requiring surgery.

The abnormal signs are generally few. Only one third of the patients of Getty et al with root entrapment had significant restriction of straight leg raising,[64] and this was similarly recorded by Macnab.[65] For many patients with little to find clinically, the diagnosis begs, therefore, a good history. Most patients have some radiologic evidence of degenerative change, reduction of L5/S1 disc space being the most consistent finding. Central canal measurements are little different from the general population.[66] There is a greater incidence of abnormal neurologic signs in those patients who have had previous surgery, and in those referred for surgery from other units

because of the intensity and duration of root symptoms.

In expert hands, electromyography can provide objective evidence of impaired root function and it will sometimes identify the root affected.[67] Other studies with electromyography have not confirmed the ability of this investigation to predict which nerve roots are responsible for symptoms,[68] probably because of the variable anatomy of root innervation. Electrodiagnostic methods can complement other investigations and help in the overall evaluation but probably have limited value, especially after previous laminectomy.[69]

Radiculography has a poor sensitivity for root entrapment syndrome,[70] but it will exclude other pathologies in the severely disabled patient. CT scan is essential if surgery is being seriously considered. Bony encroachment may, of course, not be the cause of the symptoms. They may arise from another level, but CT scan is a valuable adjunct to surgery (Fig 19).

The absence of fat in the root canal and in the lateral recess can be demonstrated by nuclear magnetic resonance, and will support the diagnosis of root entrapment syndrome.[72]

Management

Most patients presenting to an orthopaedic surgeon with root entrapment syndrome can be managed by a careful explanation of the cause of pain, and advice.[72] The natural history favours a slow, but steady, resolution of symptoms. For some patients, the pain is so intense it is not reasonable to offer advice only. It is difficult to obtain statistical evidence that an epidural injection is better than placebo,[73] but there is strong circumstantial evidence that this is perhaps the best indication for an epidural. For a few patients, surgical decompression of the root canal is essential. Careful patient selection is vital to obtain a good result. The diagnosis must be correct, the symptoms sufficiently severe and not resolving by conservative means. The diagnosis can be difficult and supplementary evidence from investigations is always welcome. Myelography is generally unhelpful, the lesion being too far lateral to be detected. Electromyograms may support the clinical diagnosis and a CT scan is imperative, especially if there has been previous surgery. The CT scan is most helpful in demonstrating the bony contours of the central and root canals, and the degree of encroachment by bone and ligamentum flavum. When facet degenerative change is a significant factor, the extent of undercutting of the superior facet can be predicted.

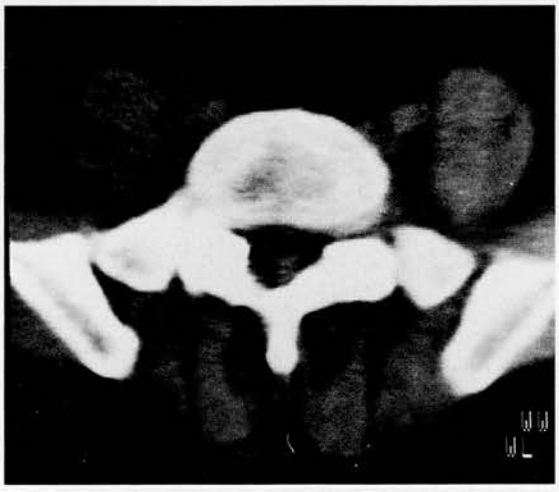


Fig 19. CT scan at L5 showing bony encroachment.

Decompression of the root requires adequate exposure of the root over the length at risk.[74] The confidence with which one views the investigations will determine the extent of surgical exploration. Some are happy to perform a limited decomposition, removing the window of lamina and part of the apophyseal joint, undercutting the lamina, and removing the ligamentum flavum.[64] These are obvious advantages in a more limited exposure, provided the decompression is adequate, but one must be confident that the root is not compromised proximally in the central canal. Most patients experience early postoperative relief of their leg pain but not a few have persisting symptoms of varying degree, perhaps the result of irreversible root pathology.

PREVENTION

Although the symptoms of central canal and root canal stenosis arise from a pathology that is multifactorial, there are two common denominators: (1) mechanical insult to the spine and subsequent degenerative change and (2) the size of the bony canal. Any hopes of prevention of spinal stenosis depends on influencing one or

both of these. A reduction of spinal injury will result from better understanding of these syndromes, and from epidemiologic and ergonomic studies. We may feel unable to influence the second, the size of the bony canal, but recent evidence suggests that the canal's growth is environmentally determined, and therefore, not entirely beyond our control.

Much of the growth of the sagittal diameter of the canal is accomplished in early life.[2] Clark et al[75] suggested that if early development is impaired, then the canal will remain permanently stunted, and catch-up growth will be impossible once the period of growth is over for that particular structure.[76–78] The adult with a small canal may carry spinal stenosis as a marker of some early insult that affected growth at a critical time. The hypothesis must be tested, but there is hope that maternal counselling may reduce the incidence of the developmentally small vertebral canal.

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Reprinted from SPINE, Vol. 12 • No. 9, November, 1987

11

The Vertebral Canal: I. Nutrition and Development, an Archaeological Study

R. W. PORTER, MD, FRCS, and D. PAVITT

The lumbar vertebral canal was measured in adult spines from two archaeological populations, and was compared with four physiological stress indicators, (cribra orbitalia, porotic hyperostosis, dental hypoplasia and Harris lines). The stature of 38 juvenile skeletons and their canal size were compared with those of the adults. By 4 years of age the midsagittal diameter and the area of the vertebral canal was fully mature and the mean interpedicular diameter 87% of adult size. There was a differential pattern of growth, with the proximal spine maturing first. The trefoil shape was not seen at L5 before puberty. Dental hypoplasia correlated with a small interpedicular diameter at L1, L2 and L3, and Harris lines with a small midsagittal diameter at L1, L3 and L5, a small area at L5 and a more trefoil canal at L4 and L5. There is evidence that adverse environmental factors are associated with the development of spinal stenosis. [Key words: vertebral canal development, physiologic stress, spinal stenosis, archaeology]

PREVIOUS STUDIES *in vitro* have suggested that the size of the lumbar vertebral canal may be affected by factors which impair early childhood development.[3] This may be masked by subsequent growth and an adequate adult stature, but the small canal is probably not capable of catch-up growth, and may remain as a marker of malnutrition.

This archaeological study was designed to compare the nutritional and general health status of an Anglo-Saxon population with four parameters of the lumbar vertebral canal, and to determine which, if any of them, was related to malnutrition. The canal sizes of Anglo-Saxon and Romano-British skeletons were compared with those of adult spines to determine the differential growth of the vertebral canal's diameters.

MATERIALS AND METHODS

The vertebral canal was measured from the lumbar spines of two archaeological populations. One was Anglo-Saxon from Raunds, Northamptonshire, and the other was Romano-British from Poundbury, Dorset.

Measurements were taken from silhouette photographs of the vertebral canal using a specially designed photographic box (Figure 1). The vertebra was placed on a fully mobile glass plate which was attached to the box by a universal joint and the sample was illuminated from above using a photographic flash-gun via a condenser lens, to obtain parallel rays of light. The vertebra was positioned using an eyepiece and a movable mirror to obtain a view from above, and to ensure that the largest possible amount of light passed through the vertebral canal. The mirror was then elevated, the flash-gun triggered, and a silhouette image of the vertebra was obtained on a piece of photosensitive paper (Figure 2).

Four measurements were recorded (Figure 3), the interpedicular and mid sagittal diameters, the canal area using a planimeter, and a measure of trefoilness. The latter was expressed as a ratio between interpedicular diameter and a transverse measurement taken 1/3 of the distance between the midpoint of the interpedicular diameter and the apex of the neural arch.[18] A vertebral canal is trefoil in shape if the ratio is less than 66.67%.

Four physiological stress indicators (cribra orbitalia, porotic hyperostosis, dental hypoplasia and Harris lines) had been recorded in the skeletons as a measure of nutritional and general health status.[22,9] The lumbar vertebral canal measurements were compared between those skeletons with and without these stress factors, and the stature also compared with dental hypoplasia.

The age of the subadult skeletons had previously been assessed. The four canal parameters of these spines was then compared with stature and with adult canal measurements.

RESULTS

The mean, tenth and 90th percentile measurements for midsagittal, interpedicular, area and trefoil shape are shown for the two populations in Table 1. The interpedicular diameters and the degree of trefoilness was the same in the two populations, but the area and the midsagittal diameters were greater in the Romano-British spines. Measurements were then taken from the canals of 155 juvenile vertebrae of 38 skeletons (Table 2).

The mean sagittal diameter for the juvenile vertebrae in each age group was not significantly different from the adult at L4 and L5, but it was significantly larger at L3 ($P < 0.05$) and at L2 and L1 ($P < 0.01$) (Table 3). The interpedicular diameter, however, was significantly smaller in the juveniles than in the adult vertebrae at the lower four lumbar levels. The canal area was not significantly different in the adult and juveniles at each level.

The juvenile vertebrae were significantly less trefoil than the adults at L5 and L4 and the adult canals were more "dome shaped" than the juveniles at L2 (Table 3).

Table 4 shows the relative size of the canal parameters of the juvenile spines as a percentage of the adult canal size. It is shown in percentiles in Figure 4. The ten vertebrae from the youngest spines below 4 years of age had mean midsagittal values 106% of the adult size, and interpedicular diameters 87% adult size.

From the Doncaster Health Authority, Doncaster Royal Infirmary, Doncaster, England.
Supported by British Coal.
Submitted for publication September 17, 1986, and revised April 1, 1987.
The authors would like to thank Dr. Keith Manchester and Miss Rosemary Powers for their helpful advice, and Bradford University and The Natural History Museum, London for the loan of the specimens.

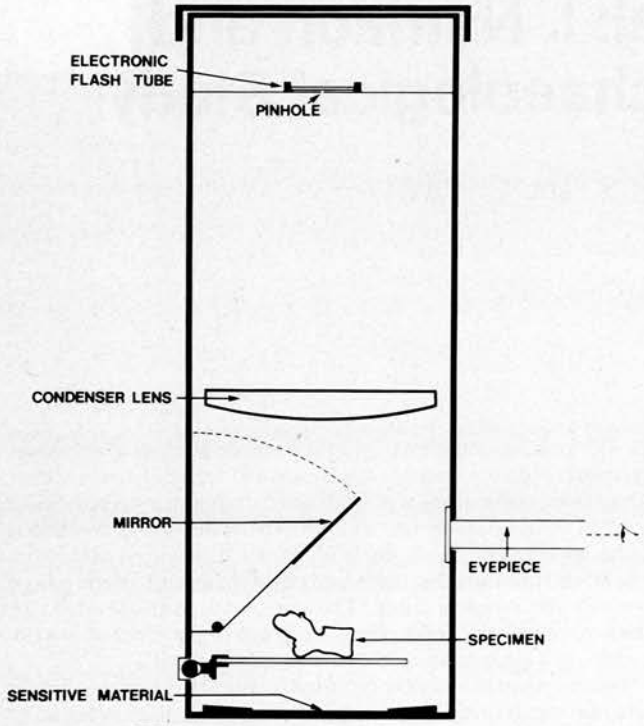

Fig 1. Diagram of a photographic box designed and constructed by Mr. G. Swann, to obtain an unmagnified photograph of the vertebral canal.

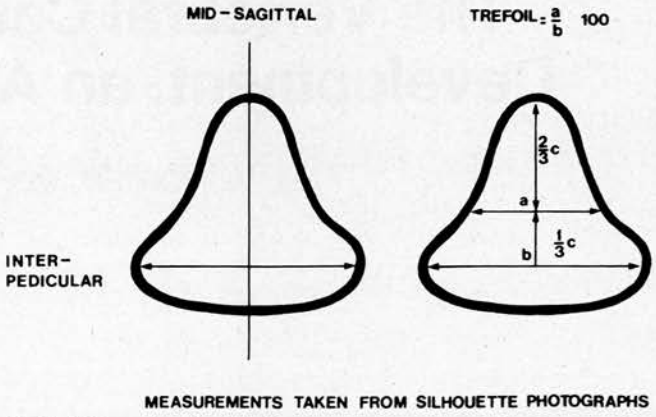

Fig 3. Diagram to show the four parameters measured of the vertebral canal.

Figures 5 and 6 show the midsagittal and interpedicular diameters of the juvenile spines at each age group and each vertebral level as a percentage of the mean adult size. The cranial vertebrae reached maturity first.

The differences between the juvenile and adult vertebral canal are illustrated in Figure 7. The midsagittal diameter is generally larger in the juvenile than the adult in the upper lumbar spine, and the interpedicular diameter smaller than the adult in the lower lumbar spine. The changing shape with maturity is responsible for the area remaining much the same.

Correlations between the four stress indicators and the canal parameters are shown in Table 5. Cribra orbitalia showed a positive correlation with a less trefoil shape at L5. Porotic hyperostosis correlated positively with a large interpedicular diameter at L1 and a large area at L1. Dental hypoplasia correlated with a small interpedicular diameter at L1, L2 and L3; it was unrelated to stature. Harris lines correlated with a small midsagittal diameter at L1, L3 and L5, a small area of L5, and a more trefoil canal at L4 and L5.

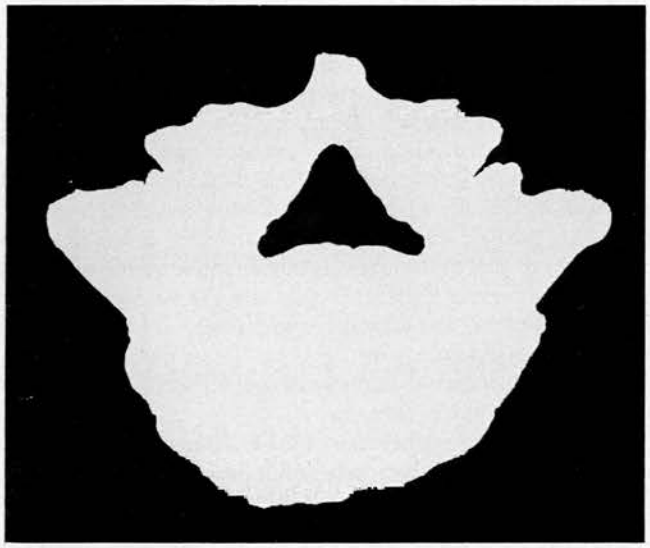

Fig 2. Silhouette photograph of a fifth lumbar vertebra.

DISCUSSION

The growth of the vertebral canal is of interest to clinicians treating back pain. It also has wider implications, because those factors that impair the canal's development may also affect other systems including the immune and central nervous systems.[4]

We know that the capacity of the vertebral canal in infants is relatively large when compared with the adult,[21,19,15] but little is known about the differential growth of the various parameters.

Radiological studies[12-14] have shown that the relatively large infants' interpedicular diameter increases further until puberty, and that it continues to grow in the lower lumbar spine after growth has ceased in the upper lumbar regions. Our own archaeological studies support this. Eighty-seven percent of the adult interpedicular diameter was achieved by 4 years of age and the remainder during childhood, with the upper lumbar spine reaching maturity before the more caudal segments.

The midsagittal diameters are less easily measured radiographically, but our archaeological measurements indicate that even in early infancy this diameter has reached adult size. In fact, in the upper lumbar spine, it is greater than the adult measurements. The slight reduction in size that takes place during childhood is due to the changing shape of the canal (Figure 7). At L5 the anterior border of the canal, which contributes to the canal's shape, is generally concave in infancy and convex in the adult.

The cross-sectional area of the canal is not materially different in the child and adult. In order for the midsagittal diameter to decrease in the proximal lumbar spine, and the interpedicular diameter to increase in the caudal segments while the cross-sectional area remains constant, the canal must change shape. This is confirmed by the trefoil measurements (Table 3), with L4 and L5 becoming more trefoil and L2 more dome shaped with maturity (Figure 7).

Table 1. Mean and Percentile Measurements of Four Canal Parameters from Two Adult Archaeological Populations

	Raunds			Poundbury		
	Mean	n = 10th p	n = 90th p	Mean	n = 10th p	n = 90th p
Midsagittal mm						
L1	15.23	13.30	17.14	15.85	14.143	17.56
L2	14.53	12.55	16.51	14.85	12.794	16.86
L3	13.64	11.58	15.70	14.08	12.07	16.09
L4	13.53	11.20	15.51	13.93	11.60	16.26
L5	14.63	11.11	18.15	15.23	12.68	18.29
Interpedicular mm						
L1	21.28	19.87	22.69	21.96	19.60	24.45
L2	22.34	18.94	25.74	22.00	19.79	24.30
L3	22.29	18.91	25.67	22.30	20.12	24.50
L4	22.61	20.22	25.00	22.84	20.16	25.50
L5	25.55	21.67	29.43	25.66	22.09	29.23
Trefoil						
L1	89.36	81.59	97.13	89.78	83.21	96.37
L2	87.69	79.14	96.24	87.69	79.84	95.54
L3	82.70	70.90	94.50	80.82	69.97	91.69
L4	74.41	64.62	84.13	74.54	64.11	84.97
L5	68.66	62.09	74.23	67.40	55.29	79.53
Area cm²						
L1	2.24	1.82	2.66	2.38	1.92	2.85
L2	2.16	1.69	2.64	2.25	1.82	2.68
L3	1.99	1.47	2.50	2.11	1.63	2.60
L4	1.90	1.25	2.55	2.08	1.57	2.60
L5	2.11	1.32	2.89	2.34	1.65	3.03

Table 2. The Numbers of Subadult Vertebrae in Both Populations

Estimated age (years)	*L1*	*L2*	*L3*	*L4*	*L5*
2–4	0	2	1	2	5
4–8	4	6	7	5	7
8–12	9	11	11	10	12
12–16	12	14	13	12	12

Table 3. Significance Between Parameters of 155 Juvenile and 839 Adult Vertebrae (Student *t* Test with Infinite Degrees of Freedom)

Level	*IP‡*	*MS‡*	*Tre‡*	*Area cm²*
L1	+1.019	−2.644†	−0.610	+0.193
L2	+2.384*	−3.142†	+2.104*	−0.652
L3	+2.342*	−1.966*	−1.192	−0.034
L4	+3.024†	−0.827	−2.295*	−0.206
L5	+3.214†	−0.571	−2.931†	−0.176

*Significant at the 0.05 level.
†Significant at the 0.01 level.
‡IP = interpedicular (mm); MS = midsagittal (mm); Tre = trefoil.

Table 4. The Relative Size of 155 Juvenile Mean Canal Parameters Expressed as a Percentage of Mean Adult Canal Size

Level	*L1*	*L2*	*L3*	*L4*	*L5*
IP	95.3	92.8	91.0	91.1	89.8
MS	103.3	107.2	102.0	100.8	99.4
Area	99.7	102.4	99.3	98.5	96.0

The four physiological stress factors that we examined produce physiological effects on the skeleton and teeth after the major part of the canal's growth has been completed. These factors could conceivably affect the terminal growth of the interpedicular diameter and the trefoil shape, but could not be expected to influence the midsagittal diameter or the area of the canal, which are fully developed by 4 years of age.

Cribra orbitalia[11] is a sieve-like appearance in and around orbits due to erythroid hyperplasia. Porotic hyperostotosis is a similar lesion in the supra orbital, frontal, parietal and occipital bones. They are related to hypochromic anaemia,[5] to dietary deficiency, parastic infestations such as hookworms, and host response to infection during the period of growth.[22] From 40 possible correlations between these physiological stress factors and the four canal parameters only three were positive: increased interpedicular diameter at L1, increased area at L1 and less trefoil canals at L5. The first phase of the physiological stress response is an increase metabolic rate, with stimulation of growth,[2] but as there were no other correlations with other parameters or at other levels, it suggests that vertebral canal development is largely independent of these stress indicators.

Dental hypoplasia[8] is related to the nutrition state throughout the development of dentition. There was significant reduction of the interpedicular diameters at L1 ($P < 0.001$) and at L2 and L3 ($P < 0.05$) in those skeletons exhibiting dental hypoplasia. This could be a direct relationship, with poor nutrition limiting postnatal growth and the final growth from infancy to puberty. It could alternatively be an indirect relationship; those mothers who were able only to provide a poor diet for their children would probably have had a poor diet themselves during pregnancy with possibly impaired fetal

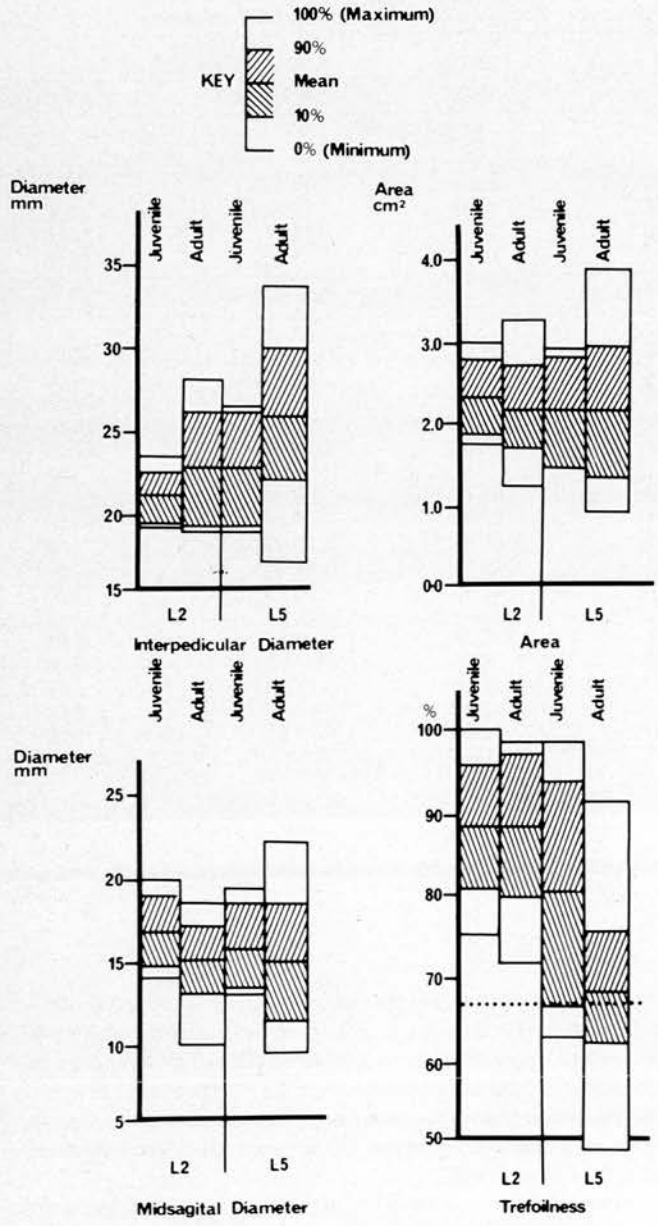

Fig 4. Mean and percentie measurements of 155 juvenile vertebrae compared with 839 adult vertebrae.

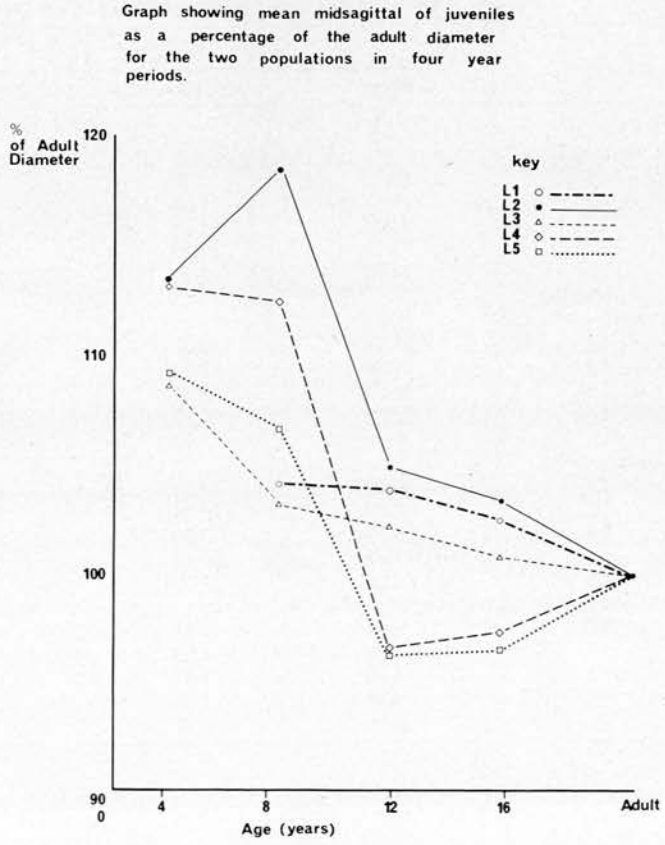

Fig 5. Graph of the mean midsagittal diameters of each age group, at the five lumbar levels expressed as a percentage of the adult canal size.

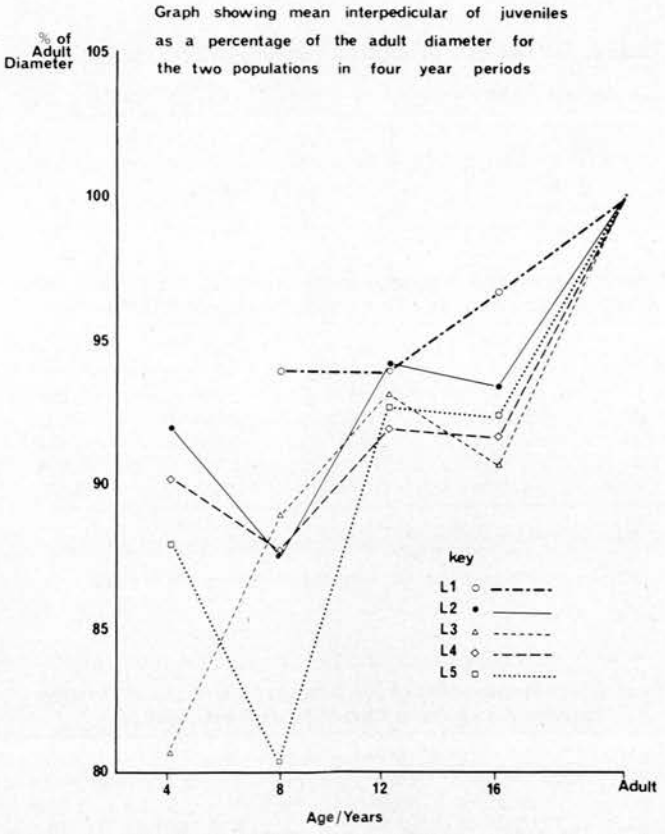

Fig 6. The mean interpedicular diameters of each age group as a percentage of the adult canal size.

canal development. In this case, however, one would have expected the midsagittal and the area also to be reduced in those spines with dental hypoplasia. The fetus is also singularly protected from the adverse effects of maternal malnutrition and the midsagittal diameter has probably completed its growth before birth. Postnatal and infantile malnutrition,[16] however, could still impair the growth of the canal's interpedicular diameter.

Previous studies have shown that individuals with large stature tend to have large, wide vertebral bodies, with wide pedicles,[20,4] and it seemed possible that the relationship between a small interpedicular diameter and dental hypoplasia was due to those individuals having a small stature. However, the adult stature in individuals with and without dental hypoplasia was not significantly different.

There was a highly significant correlation between the presence

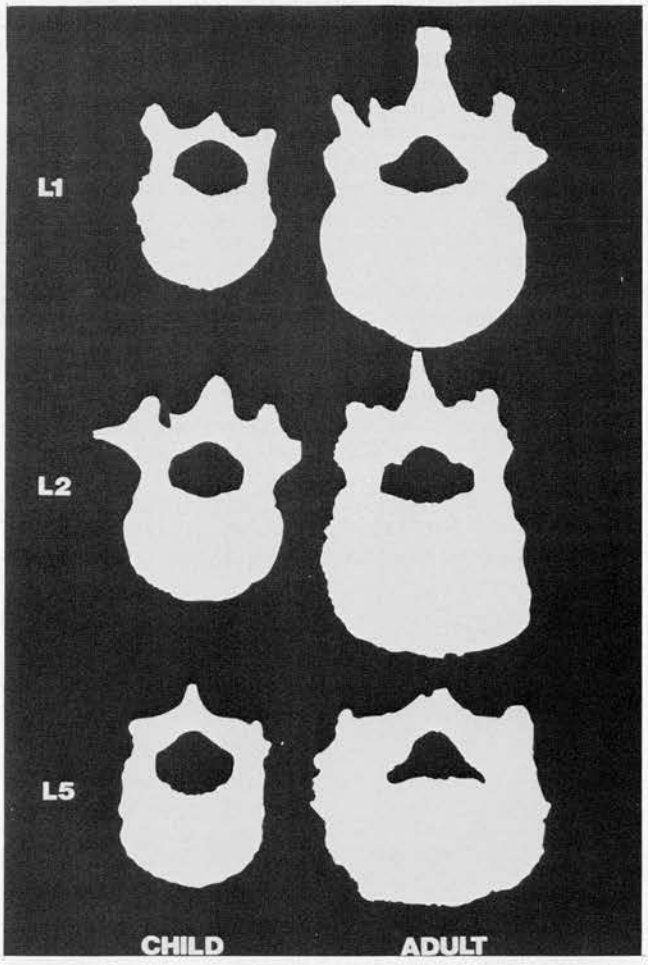

Fig 7. Representative silhouette photographs of juvenile and adult vertebral canals at L2, L4 and L5 to show the relative size and shape. At each level the midsagittal diameter of the juvenile canal is as large as the adult, the interpedicular diameter is a little smaller, and there is a difference in shape.

of Harris lines and a small midsagittal diameter at L1, L-3 and L-5 ($P < 0.001$, < 0.05, < 0.001), with a small area at L5 ($P < 0.001$), and a trefoil shape at L4 and L5. Harris lines[10] occurring predominantly in the long bones are formed when chrondrocyte maturation is halted by acute infection or by periods of dietary insufficiency.[6,17] Calcification increases to form a dense region of calcium salts, left behind as a fossil of periods of limited growth in earlier life. They may or may not resorb, and there is still debate about their significance.[4]

The midsagittal diameter and area of the canals will have been fully developed by the time many of these insults produced their Harris lines. The relationship is therefore indirect, and is compatible with the concept that factors which stunted the growth of the canal *in utero* could also have affected the development of the immune system, with resultant poor childhood health status.

Stenosis of the vertebral canal results not only from a small midsagittal diameter, but from the trefoil shape. This is an unhealthy combination if the canal is compromised by soft tissue encroachment at the disc-apophyseal joint level. The trefoil configuration occurs in about 15% of canals at L5[7] but we know little about its cause. It is generally associated with small pedicles[1] and a smaller midsagittal diameter than non-trefoil canals,[7,18] but the interpedicular diameter is unaffected.

The differential growth of the canal which has been demonstrated in this study now makes a hypothesis about trefoil development possible. If a subject has impairment of midsagittal development before 4 years of age, in the absence of any catch-up growth, that parameter will remain permanently small. In fact it may become smaller with the changing shape of the anterior boundary of the canal at L5. The interpedicular diameter, however, still has some growth potential until puberty, particularly at the lower lumbar levels. If there is no inhibition of interpedicular growth, then that canal will probably become trefoil in shape.

In spinal stenosis, the most significant clinical diameter is the midsagittal. If environmental factors affect its development, our search should now be directed to potential insults in early life, particularly in the interuterine period; an *in vivo* prospective study could be rewarding.

Table 5. Correlations between Four Physiological Stress Indicators and Vertebral Canal Parameters (Student *t* Test with Infinite Degrees of Freedom)

		Cribra corbitalis	*Porotic hyperostosis*	*Dental hypoplasia*	*Harris lines*
IP	L1	—	+2.147*	−3.319†	—
	L2	—	—	−2.463*	—
	L3	—	—	−2.385*	—
	L4	—	—	—	—
	L5	—	—	—	—
MS	L1	—	—	—	−3.494†
	L2	—	—	—	—
	L3	—	—	—	−2.138*
	L4	—	—	—	—
	L5	—	—	—	−3.709†
Area	L1	—	+2.056*	—	—
	L2	—	—	—	—
	L3	—	—	—	—
	L4	—	—	—	—
	L5	—	—	—	−4.199†
Trefoil	L4	—	—	—	−2.430*
	L5	+2.107*	—	—	−4.164†

*Significant at 5% level.
†Significant at 0.1% level.

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Address reprint requests to

R. W. Porter, MD, FRCS
Consultant Orthopaedic Surgeon
Doncaster Health Authority
Doncaster Royal Infirmary
Armthrope Road
Doncaster, DN2 5LT
England

Accepted for publication June 10, 1987.

Reprinted from SPINE, Vol. 12 • No. 9, November, 1987

The Vertebral Canal: II. Health and Academic Status, a Clinical Study

R. W. PORTER, MD, FRCS, J. N. DRINKALL, LRCP, MRC, D. E. PORTER, BSc, and L. THORP

This study was designed to test the concept that a shallow vertebral canal in the adult may act as a marker of impaired development in early life. If that same impaired development also affected the immune and central nervous systems, there could be a relationship between the vertebral canal size, health, and academic status. A retrospective analysis was made of the general practitioner records of 94 patients, some with very narrow canals and some with wide canals. There were significantly more episodes and total attendances with infections, more episodes of low-back pain, and more attendances with trauma in those with small canals. A prospective study compared the GCE examination results of 331 children whose canals had been measured by ultrasound 3 years previously. The children in the highest three deciles for canal size performed significantly better than those in the lowest decile ($P < 0.01$). There is evidence that a relation may exist between the sagittal diameter of the lumbar vertebral canal and health and academic status. [Key words: narrow spinal canal, impaired development, illness, academic achievement]

MUCH OF THE GROWTH of the sagittal diameter of the vertebral canal is accomplished in early life.[1,19] From archaeological evidence Clark et al[7] postulated that infantile malnutrition may impair development of the vertebral canal and leave its marker as adult stenosis, because once the normal period of growth is over for a particular structure, "catch-up" growth is impossible.[22-24] They suggested that the immune and central nervous systems, developing in early life may be similarly affected, resulting in poor health status and reduced life expectancy in the adult.[6] If the hypothesis is correct, one might expect the small adult vertebral canal, being a marker of deficient early development, also to be associated with a poor function of the immune and central nervous systems.

To test this concept, we have conducted two studies: first of the general practitioner records of 94 patients, one half with small and one half with large vertebral canals; and secondly a study of 331 13-year-old children from two schools in South Yorkshire, comparing the canal size with academic results in the national General Certificate of Education examinations.

From the Doncaster Health Authority, Doncaster Royal Infirmary, Doncaster, England.
Supported by British Coal.
Submitted for publication September 17, 1986.

MATERIALS AND METHODS

General Practice Study. The 15° oblique sagittal diameter of the lumbar vertebral canal had been measured by ultrasound in 386 patients from one general practice for a previous study.[8] Thirteen patients had left the practice. From the 373 records still available the 47 patients whose canals were the narrowest (below 1.41 cm at L1) were compared with the 47 widest (above 1.58 cm). There were 24 men and 23 women in each group, well matched for age (Table 1).

The original study had been designed to compare patients attending with low-back pain and the next age–sex-matched patient attending the practice without current back pain. Their distribution in this study is shown in Table 1.

Data were collected for each 10-year period of the available records for total attendances, and total episodes (for which there may have been several attendances). Separate data for each decade were collected for attendances and episodes of low-back pain, infections, trauma, allergy, psychosocial problems, gynecologic disorders, skin diseases, "other conditions" and hospital inpatient and outpatient visits. In addition the "Lloyd George" envelopes (EC6 and 7) and contents of the two groups were weighed. The significance of the differences between the means was tested by Student *t* test.

RESULTS

The weight of the 47 records of the patients with narrow canals was 22% heavier than for the 47 wide canals (3.834 kg compared with 3.1505 kg). The mean number of episodes and attendances per year was calculated from all the patients available records. The number of subjects whose records were available for each 10-year period was not significantly different for those with wide and narrow canals. Table 2 shows the mean attendances and episodes in each category. There were significantly more episodes of low-back pain in those patients with narrow canals ($P < 0.005$) and also more episodes and attendances with infections ($P < 0.1$ and < 0.05). The patients with narrow canals also attended with more trauma ($P < 0.1$). The episodes and attendances for allergic disorders were more frequent in those with wide canals ($P < 0.05$ and $P < 0.1$). There was no significant difference between the two groups when recording the other categories.

The mean number of total episodes and attendances for all conditions per year in each decade is shown in Figures 1 and 2. There was a significantly greater number of episodes for those patients with narrow canals in the first 10 years of life and also after 40 years of age. The mean attendances showed a similar trend, but it did not reach mathematical significance. There was a cross over period between 20 and 40 years of age, but the differences did not reach mathematical significance.

The episodes and attendances for infections during a similar period is shown in Figures 3 and 4. Those patients with the narrow

Table 1. Comparison between Patients with Narrow and Wide Canals

	Patients with narrow vertebral canals	*Patients with wide vertebral canals*
Number	47	47
Mean canal size (cm)	1.38 ± 0.03	1.64 ± 0.05
Men/women	24/23	24/23
Age	49.7 ± 13.8	47.9 ± 16.2
Selection in previous study patients/ controls	26/21	17/30

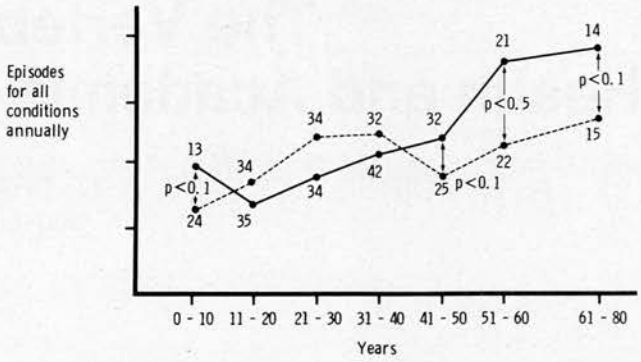

Fig 1. Graph showing the mean number of total illness episodes reported to the general practitioner for each 10-year period, comparing patients with narrow and wide vertebral canals (numbers indicate number of patient's records available for each decennium).

canals attended the surgery with significantly more episodes of infection in early and later life.

School Children Study. The vertebral canal measurements of 331 13-year-old children from two schools had been recorded by ultrasound in 1980. They were subsequently given an academic achievement score from the results of their General Certificate of Education (GCE) 0 level and CSE examination results in 1983 (Table 3) and this was compared with the size of the vertebral canal at L5. Seven children with canals below the tenth percentile and two above the 90th percentile left school before the examination and were excluded from the study.

RESULTS

At school A the mean General Certificate of Education score varied from 17.25 to 22.47 in the bottom seven deciles of canal size. In the top three deciles there was an increase in mean score up to 33.20 (Figure 5). The children at school B showed a similar trend, their mean score in the bottom seven deciles being 18.15 to 26.60, and in the ninth decile 32.7. Combined, the two schools demonstrate a general improvement in examination score from the tenth to the upper deciles (Figure 5, Table 4). Those below the tenth percentile for canal size performed significantly poorer for GCE examination than those above the 70th percentile ($P<0.01$). There was no association between an individual's canal size and examination score ($r = 0.091$), but grouping the children by deciles indicated a significant correlation between the mean examination score and canal decile ($r = 0.82$, $P<0.01$). Although children with wide canals tended to perform better academically than those with small canals (Figure 6) some children proved to be the exception, one of the best results being achieved by a child with a canal diameter of only 1.31 cm.

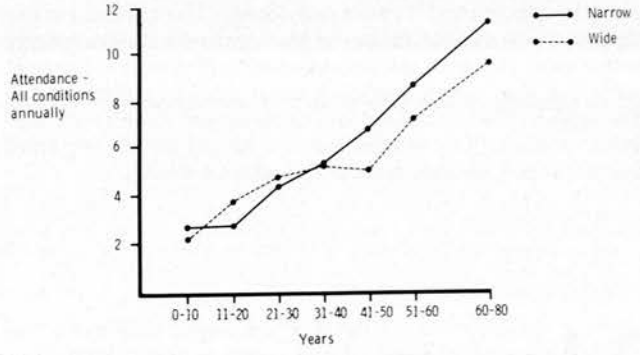

Fig 2. Graph showing mean number of total illness attendances for each 10-year period comparing patients with narrow and wide canals.

Table 2. Mean Number of Episodes and Attendances per Year for Patients with Narrow and with Wide Vertebral Canals

	Mean number of episodes per year			*Mean number of attendances per year*		
	Wide canals (n = 47)	*Narrow canals (n = 47)*	*Signficance (P)*	*Wide canals (n = 47)*	*Narrow canals (n = 47)*	*Significance (P)*
Total	2.18	2.40	ns*	5.01	5.64	ns
Low-back pain	0.107	0.205	<0.005	0.273	0.509	ns
Trauma	0.165	0.209	ns	0.277	0.402	<0.1
Psychosocial	0.243	0.266	ns	0.887	1.03	ns
Gynecologic	0.592	0.514	ns	1.01	1.21	ns
Infections (n = 23)	0.663	0.837	<0.1	1.18	1.58	<0.05
Allergies	0.140	0.033	<0.05	0.342	0.102	<0.1
Skin complaints	0.155	0.115	ns	0.315	0.195	ns
Others	0.432	0.479	ns	1.20	1.24	ns
Outpatient	0.290	0.348	ns			
Inpatient	0.0741	0.0818	ns			

*ns = not significant.

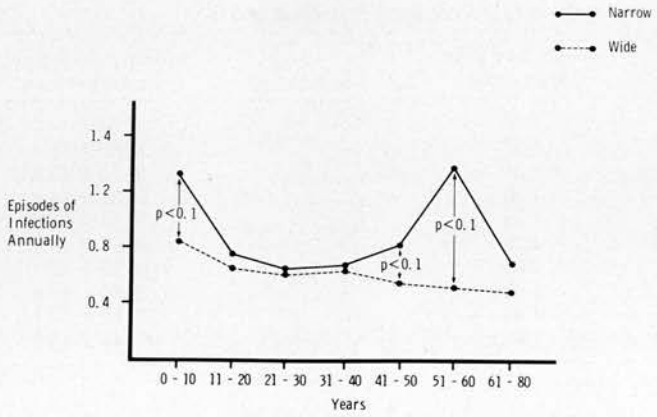

Fig 3. Graph showing mean number of episodes of infections for six 10-year periods comparing patients with narrow and wide vertebral canals.

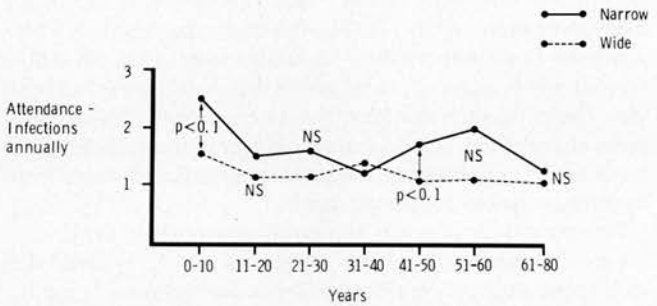

Fig 4. Graph showing mean number of attendances with infections for six 10-year periods, comparing patients with narrow and wide vertebral canals.

Table 3. Academic Achievement Score Given to Each Grade Passed in the General Certificate of Education (GCE) Examinations

O-level examination (grade)	*CSE examination (grade)*	*Score*
A	—	7
B	—	6
C	1	5
D	2	4
E	3	3
U	4	2
—	5	1

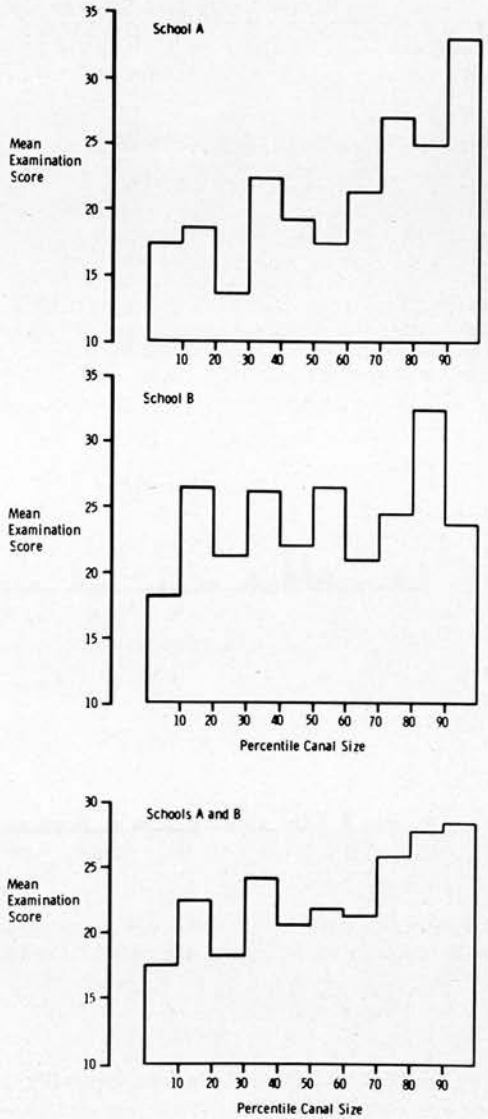

Fig 5. Histograms showing mean examination score in each of the ten percentiles of canal size in schools A and B, and the two schools combined. Best-fit linear equation: mean examinations score = 45 × mean canal sizes − 39.3.

DISCUSSION

The concept of an association between the size of the vertebral canal and both health and academic status is both novel and reasonable. Despite the limitations imposed by this study, it does support that such an association exists.

Measurement of the sagittal diameter of the lumbar vertebral canal by ultrasound has a significant degree of error[10,11] but its probably the safest method of measuring a volunteer population and is of value for epidemiologic studies.[14] We selected 94 subjects with the narrowest and widest canals from a population of 373 in one general practice, with some confidence that the canals size of these two groups were significantly different. A mean repeatability of 0.5 mm, however, means that in the school study the apparent canal size from ultrasound measurement may have placed a child in the wrong decile for true canal size. Nevertheless we would expect that in a large series the deciles would represent genuine differences within a population.

A GP record study is only a crude measure of health status, but this was a teaching practice with good documentation and an interest in research. It is accepted that entries represent patients' awareness of illness rather than a true measure of health, that patients with narrow canals are more likely to attend with low-back pain[8,12,19,26] and that patients with low-back pain are more prone to attend with other conditions than patients without back pain.[2] In fact, patients with small canals did attend particularly in later life with more episodes of low-back pain and infections than patients with larger canals.

The patients had been selected from those taking part in a previous study, one half with back pain, and one half controls. It was inevitable that in the narrow canal population there would be a bias toward the "back pain" patients and toward the "controls" in the wider canal group. In early life, however, before they could have experienced back pain those subjects with small canals attended

Table 4. Examination Score for Children in Percentiles of Canal Size Combining Schools A and B

Percentile of Canal Size	*Number*	*Mean canal size at L5 (cm)*	*Mean examination score and SD*
0–10	33	1.26	17.76 ± 14.0
10–20	34	1.32	22.65 ± 18.3
20–30	33	1.34	18.10 ± 13.8
30–40	34	1.36	24.31 ± 15.0
40–50	33	1.38	20.57 ± 16.2
50–60	34	1.39	21.93 ± 16.6
60–70	33	1.41	21.25 ± 14.1
70–80	34	1.43	25.86 ± 16.4
80–90	33	1.45	27.88 ± 14.1
90–100	34	1.49	28.16 ± 16.8

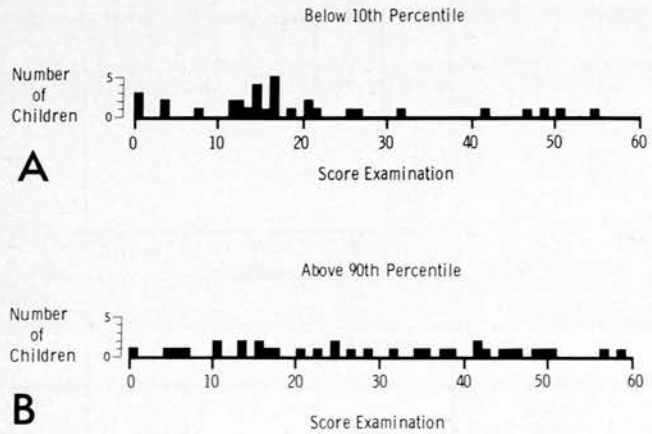

Fig 6. A, Examination scores for 33 children with canals below the tenth percentile. **B,** Scores for the 34 children with canals above the 90th percentile.

with more episodes of childhood illnesses, especially infections, than those with wide canals ($P<0.1$). Either the early illnesses were responsible for the development of the small canal or that canal was already developmentally small and was associated with poor infantile health status. Despite the limitations imposed by a relatively crude retrospective analysis of records, it has provided evidence that a relationship could exist between the canal size and health status that makes a prospective study imperative.

The children whose academic status was compared with canal size were from similar socioeconomic backgrounds in two mining villages, and they had shared the same educational system from 5 years of age. Both schools independently showed the same trends. There were exceptions, but pupils with larger canals tended to perform more successfully in GCE examinations.

There are several possible explanations. A poor socioeconomic environment may be responsible not only for inadequate canal development but for failure to stimulate the child intellectually. Alternatively, increased illnesses in those children with small canals could result in reduced school attendance and poor achievement. It is possible, however, that there is a genuine relation between the canal size and impaired neurologic development in early life. These three factors may combine.

If it is correct that the canal size is often related to academic achievement then it is unfortunate that many individuals with low-back pain and small canals will have academic qualifications which limit them to manual work.

Genetic factors may have some effect on the canal's sagittal growth; but epigenetic factors[15] and environmental factors are likely to be more significant. The interpedicular diameter, which continues to grow to puberty,[13] is related to height; a tall stature correlates with larger vertebral bodies and widely separated pedicles. The midsagittal diameter, however, is largely complete by 4 years of age[5,19] and is independent of other anthropometric measurements,[18] suggesting that this parameter will be influenced more by interuterine environmental factors.

Persons with small canals may not necessarily have demonstrable growth retardation at birth. In fact, there is no evidence that early growth retardation affects the incidence of chronic or specific diseases,[17] and the effect of small-for-date infants on later intelligence is contradictary.[4] Undoubtedly, very premature and stressed infants are immunologically compromised[25] and educationally impaired.[9,21]

Our search, however, should probably concentrate on the most rapid phase of growth from the eighth to the 16th weeks *in utero,* when the crown rump length of the fetus increases from about 5 to 15 cm.[23] Specific insults to the sensitive enzyme systems at this stage are likely to be more significant than maternal malnutrition from which the fetus is remarkably resistant.[16]

A study of the relation of such factors to the subsequent canal size could be rewarding, and their recognition have considerable medical, social, and economic significance.

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Address reprint requests to

R. W. Porter, MD, FRCS
Doncaster Health Authority
Doncaster Royal Infirmary
Armthrope Road
Doncaster, DN2 5LT
England

Accepted for publication March 23, 1987.

Reprinted from SPINE, Vol. 13 • No. 9, September, 1988

13

Neurogenic Claudication and Root Claudication Treated with Calcitonin

A Double-Blind Trial

R. W. PORTER, MD, FRCS, FRCSE, and C. G. MILLER, BSc

Forty-two patients with either neurogenic claudication or unilateral root claudication were analyzed in a double-blind comparison of salmon calcitonin (SCT) and placebo, receiving either 100 IU SCT or 1 ml saline four times a week for 8 weeks. Five of 20 SCT and one of 22 placebo patients were classified as responders. There was no statistically significant difference between the treatment groups in the proportion of responders. Seven of eighteen of the placebo group who later received salmon calcitonin improved their walking distance. The authors have not established that this is an organic response. [Key words: neurogenic claudication, root claudication, spinal stenosis, calcitonin]

PATIENTS WITH NEUROGENIC CLAUDICATION can respond to salmon calcitonin (SCT) with a considerable improvement in their walking distance.[4] We have observed a similar effect in patients with unilateral root claudication. We now report the results of a double-blind trial comparing SCT with placebo for patients with either neurogenic claudication or unilateral root claudication.

METHOD

Patients with defined criteria of either neurogenic claudication or root claudication (Table 1) were invited to enter the trial. Approximately one third of the patients were tertiary referrals. The spine was assessed by radiculogram, computed tomography (CT) scan, and ultrasound, and the peripheral arteries were shown to be patent by Doppler ultrasound. Claudication threshold and tolerance at constant walking speed was measured on a treadmill, and the degree of pain recorded on a visual analog scale. We obtained a verbal description of walking pain and of sleep disturbance at night.

Patients were randomly allocated to receive subcutaneous injections of either 100 IU SCT or 1 ml saline diluent four times a week for 8 weeks. At the end of this period, the code was broken and nonresponders in the placebo group were given a further 8-week course of open SCT (100 IU 4 times a week). Assessments of walking distance on a treadmill, pain, sleep, mobility, and analgesic requirements were made at weeks 0, 4, and 8 for all patients, and at weeks 12 and 16 for those patients who received open SCT therapy. Treatment success was defined as an improvement in walking distance by at least 100% and ability to walk more than 800 meters.

The statistical comparison of responders was by Fishers two-tailed exact probability test; the treadmill walking distance and visual analog pain scores by Mann-Whitney U-test between treatments, and Wilcoxon matched pairs signed ranks test within treatment change; and pain rating scores, sleep, and mobility by chi square tests, between and within treatments.

RESULTS

The main analysis was carried out on data from the first 8 weeks of the trial. The treatment groups were well-balanced with respect to history and severity of symptoms on entry to the study (Table 2). Forty-two patients entered the trial, but five SCT and six placebo patients did not complete the double-blind phase. All six of the placebo patients and three of those receiving SCT were withdrawn due to lack of efficacy. One SCT patient was withdrawn due to side effects (nausea and flu-like symptoms) and one patient receiving SCT had responded to treatment and the code was broken after 4 weeks (see Case Report). Side effects, mainly nausea, were reported by five SCT patients and one receiving placebo. These were severe enough in two patients receiving SCT to cause a withdrawal of treatment in one and a reduction in dose of the second.

CASE REPORT

A 56-year-old retired miner had a 5-year history of occasional low-back pain. For 12 months he had complained of pain in the left buttock, thigh, calf, and foot when walking, which limited his distance on a treadmill to 102 m. He had first been referred to a vascular surgeon, but his peripheral circulation tested by Doppler scan was normal.

His myelogram showed segmental narrowing of the dural sac at the L3–4 level (Figure 1), and a CT at this level demonstrated marked hypertrophy of the apophyseal joints, a shallow central

Table 1. Criteria for Treatment

Criteria for neurogenic claudication
Discomfort (tiredness, aching, heaviness, pain) in both legs when walking, relieved by rest.
Affecting both legs equally, above and below the knees.
Limiting walking distance to less than 500 m.
A positive radiculogram showing encroachment of the dural sac.
Criteria for root claudication
Discomfort (aching or pain) in one leg when walking, relieved by rest.
In a nerve root distribution, above and below the knee extending to the lower calf.
Limiting walking distance to less than 500 m.
CT Scan showing bony or soft tissue encroachment of the root canal.
Exclusion criteria
More than two inappropriate signs.
Major joint arthritis.
Paget's disease.

From the Doncaster Royal Infirmary, Doncaster, England.
Submitted for publication October 12, 1987, and revised April 6, 1988.
The authors wish to thank Rorer Health Care Ltd, for their assistance in this study, Mrs. L. Struthers, for the statistical analysis, and Miss E. Key, for typing the manuscript.

Table 2. Age, Sex, and Symptoms of Claudicating Patients Receiving Either SCT or Placebo

Patient details		*SCT*	*Placebo*
Age (yrs)	Mean	53.6	56.7
	SD	10.3	9.5
	n	20	22
	Range	38-74	38-72
Sex (nos. patients)	Males	18	17
	Females	2	5
	Total	20	22
Duration of pain			
Back pain (yrs)	Median	11	14
	Range	1-53	1-46
	n	19	22
Claudication pain (yrs)	Median	1.25	4.5
	Range	0.1-58	0.1-54
	n	20	22
Claudication Pain (no. patients)			
Bilateral claudication		9	13
Unilateral claudication		11	9
Total		20	22
Pain at night (nos. patients)			
None		10	7
Restless		3	4
Restless/walk about		2	0
Restless/walk about/cramp		1	1
Restless/cramp		1	3
Cramp/walk about		0	1
Cramp		2	3
Walk about		0	1
Other		0	1
Total		19	21
Onset of Claudication (nos. patients)			
Proximal		13	16
Distal		2	1
Proximal/distal		2	1
Total		17	18

canal, and anterior soft tissue encroachment, particularly of the left (Figure 2). His ultrasound canal measurements showed a very shallow canal well below the tenth percentile from L1 to L5, (1.43 cm, 1.39 cm, 1.33 cm, 1.28 cm, and 1.31 cm).

After the first week in the trial, having had four injections, he was having no side effects and he found he could walk unlimited distances without pain. When he attended at 4 weeks, he walked 908 meters on the treadmill without pain in the leg, and he said he was spending 8 hours a day on his feet free of symptoms. We broke the code, and he had been receiving SCT. After a year without therapy, he remains active and free of symptoms.

Five of 20 SCT and one of 22 placebo patients were classified as responders after the double-blind phase of the trial, with a weak trend in favor of SCT ($P = 0.14$, using Fisher's two-tailed exact probability test). There were no statistically significant differences between the proportion of patients withdrawn due to inefficacy or in the numbers of patients for whom side effects were reported during the first weeks of the study. There also were no significant differences in treadmill walking distances, analgesic usage, or in assessment of severity of pain, sleep, or mobility between the treatment groups (Table 3).

Eighteen patients from the placebo group went on to receive open SCT therapy. None of these responded to treatment according to the above definition, but seven of the original placebo group did improve their walking distance. Five patients experienced side effects in this phase of the trial, necessitating withdrawal of SCT

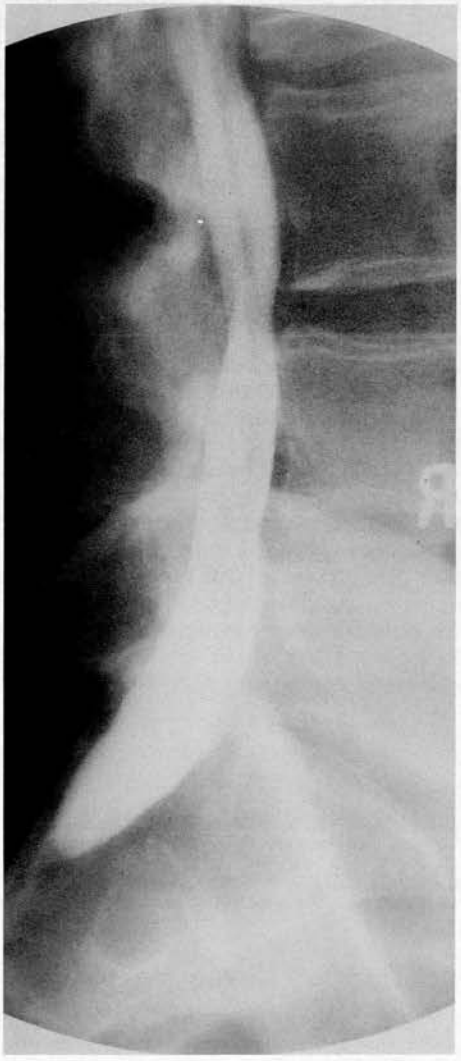

Fig 1. Lateral radiograph of myelogram of patient described in Case Report.

in one, and a 50% reduction in dose in a second. Ten of 20 SCT and three of 22 placebo patients subjectively assessed themselves as responders to the injections in spite of failing to increase their walking distance by 100%. Eight of 18 patients who had open therapy after placebo injections reported subjective improvement.

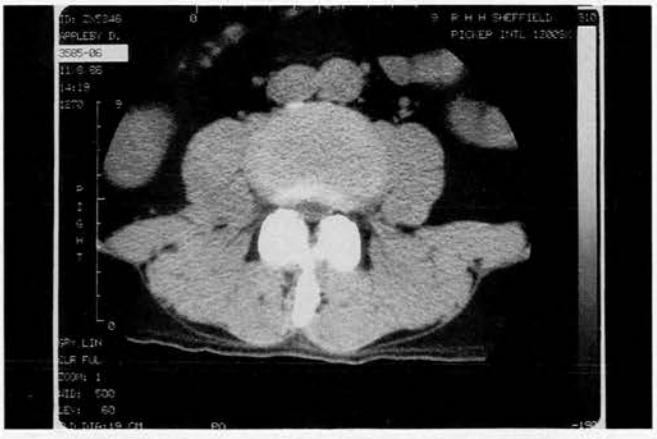

Fig 2. CT scan at L3-4 level of patient described in Case Report.

Table 3. Response of Patients Receiving SCT or Placebo

Treadmill walking distances		*Week 0*	*Week 4*	*Week 8*
Threshold distance (distance to onset of pain) (m)				
SCT	Median	50	48	85
	Range	0-303	13-570	12-600
	n	17	15	12
Placebo	Median	42	54	34
	Range	11-300	16-260	16-510
	n	15	17	12
Tolerance distance (distance when pain prevents further walking) (m)				
SCT	Median	243	214	150
	Range	22-1640	23-1660	22-1670
	n	20	18	15
Placebo	Median	91	103	97
	Range	20-756	31-515	17-1334
	n	20	17	13
Visual analog pain scores				
SCT	Median	52	46	37.5
	Range	19-91	11-88	0-76
	n	18	15	12
Placebo	Median	65	50	46
	Range	21-94	4-96	4-78
	n	19	18	15
Pain rating scale (nos. patients)				
SCT	Absent	0	2	2
	Mild	0	1	3
	Moderate	8	9	6
	Severe	8	3	3
	Intolerable	1	0	0
	Total	17	15	14
Placebo	Absent	0	0	0
	Mild	1	5	5
	Moderate	9	8	7
	Severe	9	7	4
	Intolerable	1	0	0
	Total	20	20	16
Sleep assessments (nos. patients)				
SCT				
Pain does not prevent sleep		11	15	11
Pain only allows fitful sleep		5	1	4
Pain prevents any sleep		1	0	0
Total		17	16	15
Placebo				
Pain does not prevent sleep		9	14	9
Pain only allows fitful sleep		10	6	6
Pain prevents any sleep		0	0	0
Total		19	20	15
Mobility since last assessment (nos. patients)				
SCT	Improved		7	6
	No change		11	8
	Decreased		0	1
	Total		18	15
Placebo	Improved		4	4
	No change		15	11
	Decreased		1	1
	Total		20	16

DISCUSSION

The beneficial effects of calcitonin on the paraparesis of patients with spinal Paget's disease has been noted previously.[2,3,5,7] Many patients, besides losing Paget's pain, also found that their walking distance improved suddenly and dramatically. We previously treated with SCT 11 patients who had neurogenic claudication but no Paget's disease, and observed the similar improvement.[4]

More recently, we have noticed an equally impressive response in some patients with unilateral root claudication.

We designed this double-blind study to confirm the previously observed and frequently dramatic response to SCT; however, the results did not show any statistically significant difference between the five of 20 patients responding to SCT and the one of 22 responding to placebo.

Our clinical impression, however, is that we are observing an organic phenomenon when patients with neurogenic claudication and unilateral root claudication improve their walking distance with SCT. Responding patients generally begin to improve after 2 or 3 weeks of injections, many do not relapse after cessation of treatment, and they at least are convinced of the drug's efficacy.

Calcitonin has a powerful central analgesic effect, with receptors in the hypothalamus. The drug is found in the cerebrospinal fluid of rats within 12 hours of injection.[6] If this was the mechanism of response, however, it is difficult to explain why only a proportion of patients are affected, why the response is delayed, and why many patients do not relapse.

We originally suggested that calcitonin's effect on the blood supply to the vertebral bodies would have a secondary response on the hemodynamics of the cauda equina, reducing venous engorgment, and improving the arterial supply. An alternative mechanism could be calcitonin's potent vasodilator effect on the extradural soft tissues. Its vasoactive properties have a persistant effect in the regulation of blood flow in both physiologic and pathologic conditions,[1] its pharmacological action extending locally to peripheral nerve endings.

If improved walking is indeed an organic effect of SCT, we may not understand its mechanism until we learn more of the altered neurophysiology that is responsible for claudication symptoms. The weak trial in favour of SCT would be understandable if there were a number of pathophysiologic processes responsible for the symptoms of neurogenic claudication, only one of which reacts positively to SCT treatment.

Although there is no doubt that some patients with neurogenic claudication and unilateral claudication will improve their walking distance with calcitonin, this trial found only a weak trend in favor of SCT. We could not establish that the response is an organic phenomenon. We recommend, however that salmon calcitonin be considered in the management of this disabling condition.

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Address reprint requests to

Mr. R. W. Porter, MD, FRCS
Consultant Orthopaedic Surgeon
Doncaster Royal Infirmary
Armthorpe Road
Doncaster, DN2 5LT
England

Accepted for publication May 19, 1988.

Reprinted from SPINE, Vol. 14 • No. 9, September, 1989

Walking and Cycling Tests in Neurogenic and Intermittent Claudication

GX DONG, MD,* and R. W. PORTER, MD, FRCS, FRCSE†

The walking and cycling tolerance of 19 patients with neurogenic claudication and 11 with intermittent claudication was assessed, first in the upright and then in 30° of flexion, repeating the tests on a second day. The flexed posture improved the walking and cycling distance, respectively, in 11 and six patients with neurogenic claudication. In two of the 11 patients, this posture significantly improved by more than 100% of both walking and cycling distances. In addition, one patient had claudication pain when he was walking, but could cycle unlimited distance. Only one of the patients with intermittent claudication could walk or cycle further in the flexed position. Although some patients with neurogenic claudication increase their walking distance by flexing forwards, we conclude that posture-related walking and cycling are insufficiently sensitive tests to distinguish between neurogenic and intermittent claudication. [Key words: neurogenic claudication, intermittent claudication, back pain, posture, diagnosis]

PATIENTS WITH NEUROGENIC CLAUDICATION from spinal stenosis or intermittent claudication from peripheral vascular disease both have discomfort in their legs that limits walking distance. It is not easy to distinguish between these two conditions from the history and clinical examination alone. It is necessary to have a myelogram or computed tomography (CT) scan to demonstrate the spinal stenosis, or ultrasound Doppler investigations and perhaps arteriography to identify peripheral vascular disease. It is generally believed that, because extension reduces the space within the vertebral canal, posture-related walking and cycling distances are helpful in the differential diagnosis.

We have investigated the relation between different postures in patients both walking and cycling, who have either neurogenic or intermittent claudication, to see if there are any consistent differences that would help in the clinical assessment of these patients.

MATERIALS AND METHOD

Our study included 19 patients with neurogenic claudication whose walking tolerance at 2 km/hour on a treadmill was less than 400 m. The diagnosis was confirmed by myelography or CT. Fifteen patients had multiple-level subtotal occlusion, and four had a complete block to the metrizamide flow. Doppler studies excluded coexistent peripheral vascular disease. These patients were compared with 11 patients who had intermittent claudication confirmed by arteriography.

They were asked if they had less symptoms walking uphill, downhill, or if there was no difference. They then were asked to walk on a treadmill in an upright posture at 2 km per hour recording the claudication tolerance, and after a 5-minute period of rest to relieve the effects of fatigue, they were asked to walk again on the treadmill, but leaning forward in 30° of flexion and the tolerance again was recorded. The patients then were asked to cycle in the upright posture, recording the speed and the tolerance, and after a 5-minute period of rest, they cycled again at the same speed in 30° of flexion, recording again the cycling tolerance. The patients were asked to return again within 7 days when the same walking and cycling tests were repeated. We used the 'Two-Sample Median' test for statistical analysis.

RESULTS

There was no significant difference in the age or the sex distribution of the 19 patients with neurogenic claudication and 11 patients with intermittent claudication (Table 1). More of the patients with neurogenic claudication had bilateral symptoms, and the past history was significantly longer in the patients with neurogenic claudication.

Only three (15.8%) of the patients with neurogenic claudication had less symptoms walking uphill than downhill. None of the patients with intermittent claudication preferred walking uphill, while eight (72.2%) had less symptoms going downhill (Table 2).

The walking tolerance in the two different postures on two separate days for 19 patients with neurogenic claudication is shown in Figure 1. Eleven of the 19 patients could consistently walk further in the flexed posture than in the neutral upright posture on both the first and the second day. For nine of these patients, the differences were less than 100%, but they were consistent, and for two of the patients there was a considerable increase, with over 100% improvement in the walking distance in the flexed posture on both days (Table 3). In Figure 2, nine of the 11 patients with intermittent claudication had no consistent improvement in walking distance by flexing forward, although two of the 11 did show slight improvement.

Figure 3 shows the cycling tolerance in the two different postures for the 19 patients with neurogenic claudication, with 11 patients failing to cycle any further by stooping forward. Five patients, however, had up to 100% improvement by leaning forward on both

Table 1. General History

	Neurogenic claudication	*Intermittent claudication*
Total	19	11
Male	18	9
Female	1	2
Mean age (yrs)	58.0(range, 40-69)	60.0(range, 36-73)
Bilateral symptoms	13	4
Mean history duration (mos)	116.0	32.5

From the *Department of Surgery, The Third Teaching Hospital, Beijing Medical University, Beijing, Peoples Republic of China, and the †Department of Orthopaedics, Doncaster Royal Infirmary, Doncaster, United Kingdom.

Submitted for publication February 18, 1988, and revised January 12, 1989.

The authors thank Mr. W. S. J. Mair, FRCS, of the Department of General Surgery, Doncaster Royal Infirmary, for his cooperation and for providing the patients who had intermittent claudication.

Table 2. The History of Patients' Symptoms Walking Uphill or Downhill

Less symptoms walking . . .	*Neurogenic claudication*	*Intermittent claudication*
Uphill	3	0
Downhill	6	8
No difference	10	3

Table 3. Number of Patients Whose Walking Tolerance Improved by Stooping Forwards on Both Days

Improvement in walking tolerance by stooping forward	*Neurogenic claudication*	*Intermittent claudication*
No improvement	8	9
Up to 100%	9	2
More than 100%	2	0

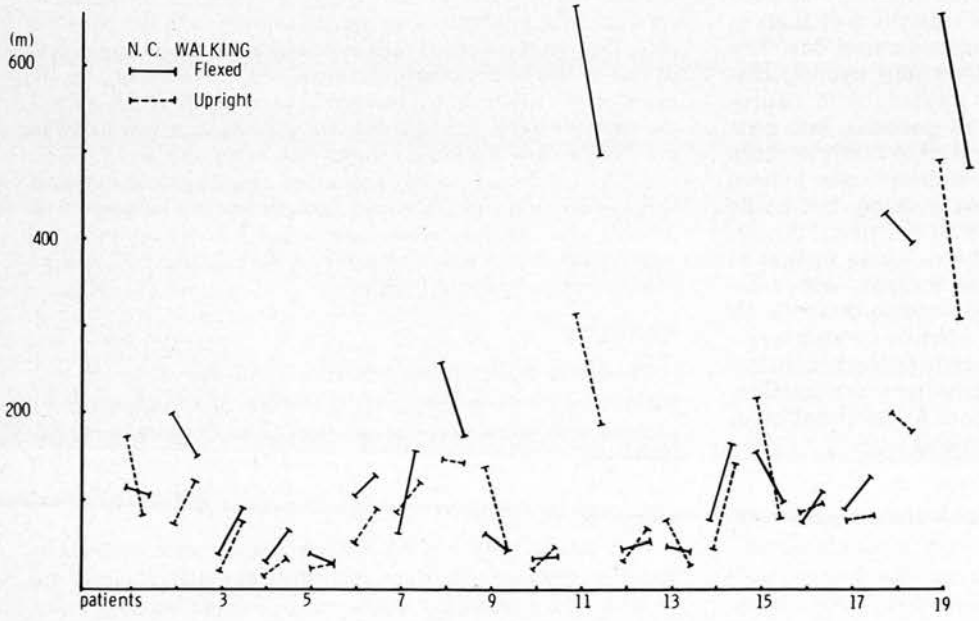

Fig 1. Diagram of the walking tolerance in the upright posture (dotted line) and leaning forward (continuous line) for 19 patients with neurogenic claudication (N.C.) on 2 separate days.

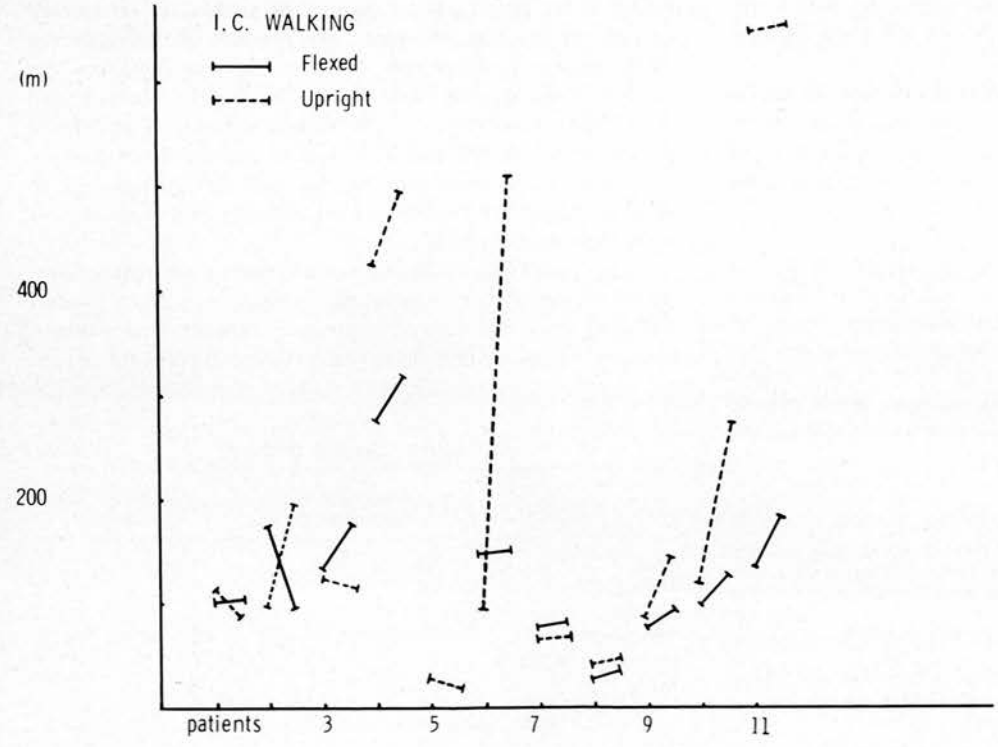

Fig 2. Diagram for the walking tolerance in the upright posture (dotted line) and leaning forward (continuous line) for 11 patients with intermittent claudication (I.C.) on 2 separate days.

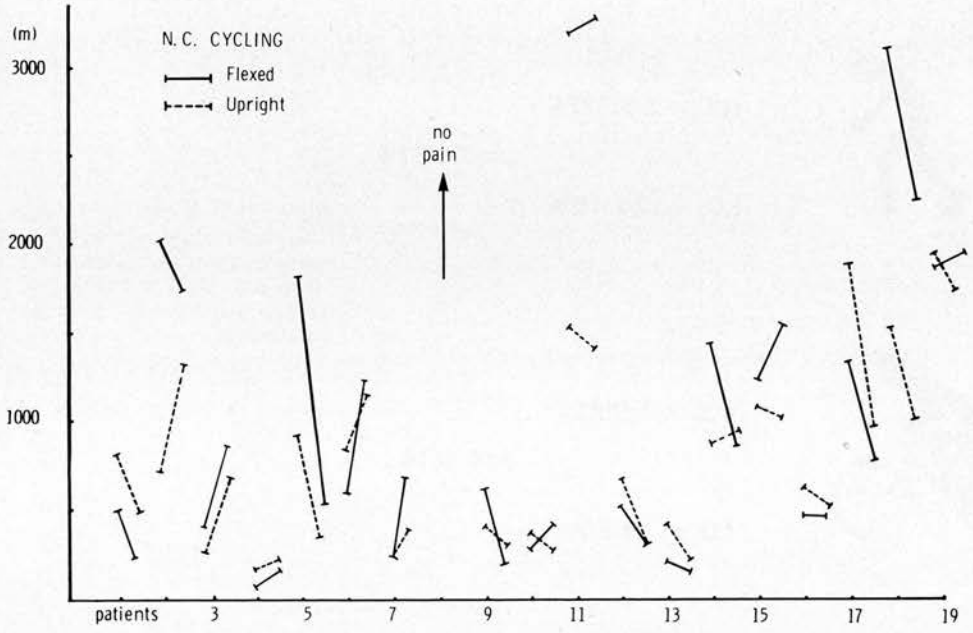

Fig 3. Diagram of the cycling tolerance in the upright posture (dotted line) and leaning forward (continuous line) for 19 patients with neurogenic claudication (N.C.) on 2 separate days.

days and two improved by more than 100%. One patient could cycle unlimited distance, although he had claudication pain when he was walking (Table 4). Figure 4 shows the cycling distance for 11 patients with intermittent claudication. Ten had no consistent improvement by stooping forward, although one did improve a little on both days when leaning forward.

The 19 patients with neurogenic claudication had a median walking tolerance change of +26.7% by flexing forward, but only +2.6% change in cycling tolerance (Table 5). By contrast, the 11 patients with intermittent claudication had a median walking tolerance change of −26.1% and cycling tolerance change of −12.5%. There was a significant difference in the walking and cycling tolerance when flexing forward between the two populations (P = 0.0014 and 0.034, respectively) (Figure 5).

The repeatability of the walking tolerance was less satisfactory on the 2 days for the patients with neurogenic claudication than for the patients with intermittent claudication, eight of the walking tests have more than 100% difference in walking tolerance. The walking distance for the patients with intermittent claudication was more consistent on the 2 days (Table 6).

DISCUSSION

A clinical test that distinguished neurogenic from intermittent claudication would be helpful, especially because spinal stenosis and peripheral vascular disease can coexist.[4] We have attempted to evaluate the diagnostic value of posture-related exercise.

Table 4. Number of Patients Whose Cycling Tolerance Improved by Stooping Forwards on Both Days

Improvement in cycling tolerance by stooping forward	*Neurogenic claudication*	*Intermittent claudication*
No improvement	11	10
Up to 100%	5	1
More than 100%	2	0
Unlimited distance	1	0

Patients with neurogenic claudication frequently complain of posture-related pain; they say that they are more comfortable walking in a stooped posture and that when they can walk no further they will bend forward, lean against a wall, or fasten a shoe lace until the symptoms settle. Often they are more comfortable walking uphill than downhill in lordosis. They may say that they can cycle long distance without symptoms in the legs,[1] described by van Gelderen as the cycle test.[3] Dyck and Doyle[2] modified this to distinguish between intermittent and neurogenic claudication by postural differences in the cycling distance. The same effects of postural activity are sometimes demonstrated by a patient with neurogenic claudication climbing a ladder without too much difficulty. It is assumed that these changing symptoms

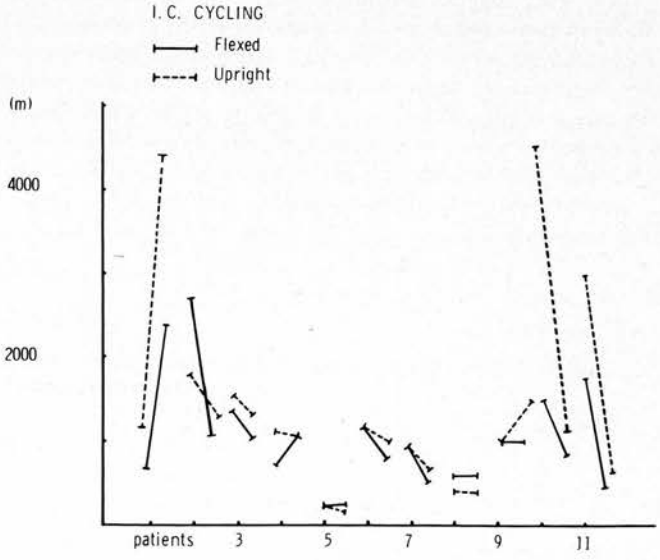

Fig 4. Diagram of the cycling tolerance in the upright posture (dotted line) and leaning forward (continuous line) for 11 patients with intermittent claudication (I.C.) on 2 separate days.

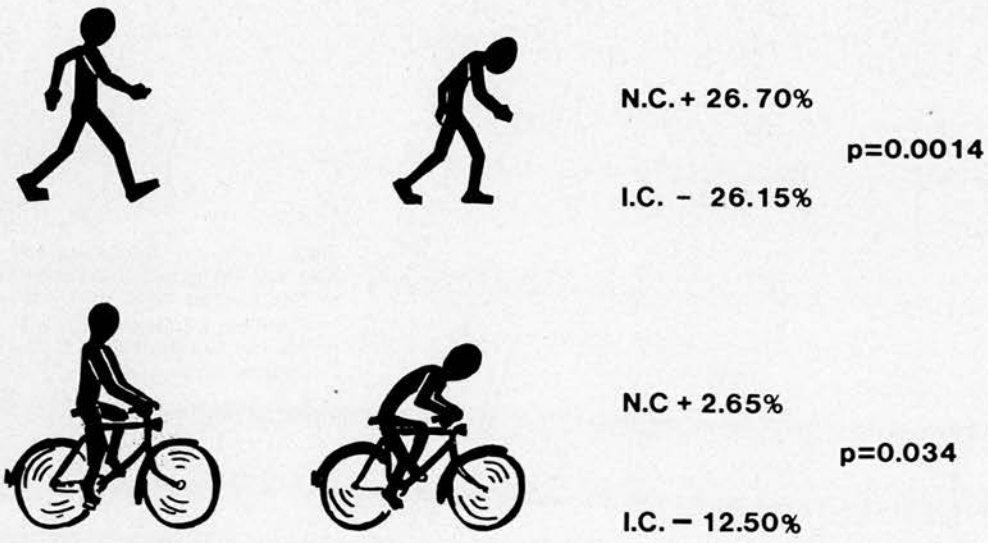

Fig 5. The median percentage differences in walking and cycling tolerance by flexing forward in 19 patients with neurogenic claudication and 11 with intermittent claudication.

are associated with dynamic changes in the cross-sectional area of vertebral canal (Figure 6). Posture-related symptoms are thought to be helpful in the differential diagnosis of vascular and neurogenic claudication,[4,5] but we found the walking history unreliable, with only three of the 19 patients with neurogenic claudication admitting to less symptoms walking uphill: eight of the 11 patients with intermittent claudication preferred walking downhill when there would be less oxygen requirement for the lower limb muscles. Similarly, we found that posture-related walking and cycling were of limited value as a diagnostic test. A failure to improve the walking or cycling distance by stooping forward could mean that a patient had either neurogenic or intermittent claudication. Slight improvement up to 100% is not likely to be diagnostic of either condition, but the results of the two patients who had a dramatic improvement in both walking and cycling tolerance by stooping forward did indicate that when this is occasionally observed, the patient is likely to have neurogenic claudication.

This study suggests that there are probably a number of different dynamic factors that are important in the pathogenesis of neurogenic claudication. In some patients who have spinal stenosis and degenerative change that has stabilized the spinal segments, bending forward will take place largely at the hips, and flexion of the lumbar spine will be more apparent than real. The posture then is not likely to affect the space within the vertebral canal, and stooping forward will not improve claudication distance. Other patients, however, may have some segmental instability in the sagittal plane, when lumbar extension is likely to aggravate the stenosis. For these patients, posture will influence walking distance to a variable degree.

The one patient who could not walk far because of claudication pain, but could cycle unlimited distance, had an isthmic spondylolisthesis with root claudication. Rotation is particularly significant in walking, but not in cycling, and it is probable that symptoms were related to rotational instability, a nerve root being affected by lumbar rotation in the transverse plane. There therefore may be a number of different pathologic mechanisms that in the presence of spinal stenosis can cause symptoms of neurogenic claudication. However, we found that posture-related walking and cycling are insensitive tests to distinguish between neurogenic and intermittent claudication.

Table 6. Repeatability of Walking Tolerance on Two Separate Days for Patients with Intermittent and Neurogenic Claudication

Difference between walking tolerance on 2 days	*Neurogenic claudication*	*Intermittent claudication*
<100%	30	20
>100%	8	2

Table 5. Comparison Between Walking and Cycling in the Upright and in the Flexed Posture, in Patients with Neurogenic Claudication and Intermittent Claudication

	Neurogenic claudication (n = 19)		*Intermittent claudication (n = 11)*		
	Median percent change by flexing forwards	*Semi I-Q range*	*Median percent change by flexing forwards*	*Semi I-Q range*	*Significance*
Walking tolerance	+26.70	26.65	−26.15	18.75	$P = 0.0014$
Cycling tolerance	+ 2.65	21.65	−12.50	20.82	$P = 0.034$

I-Q = Inter-Quartile range.

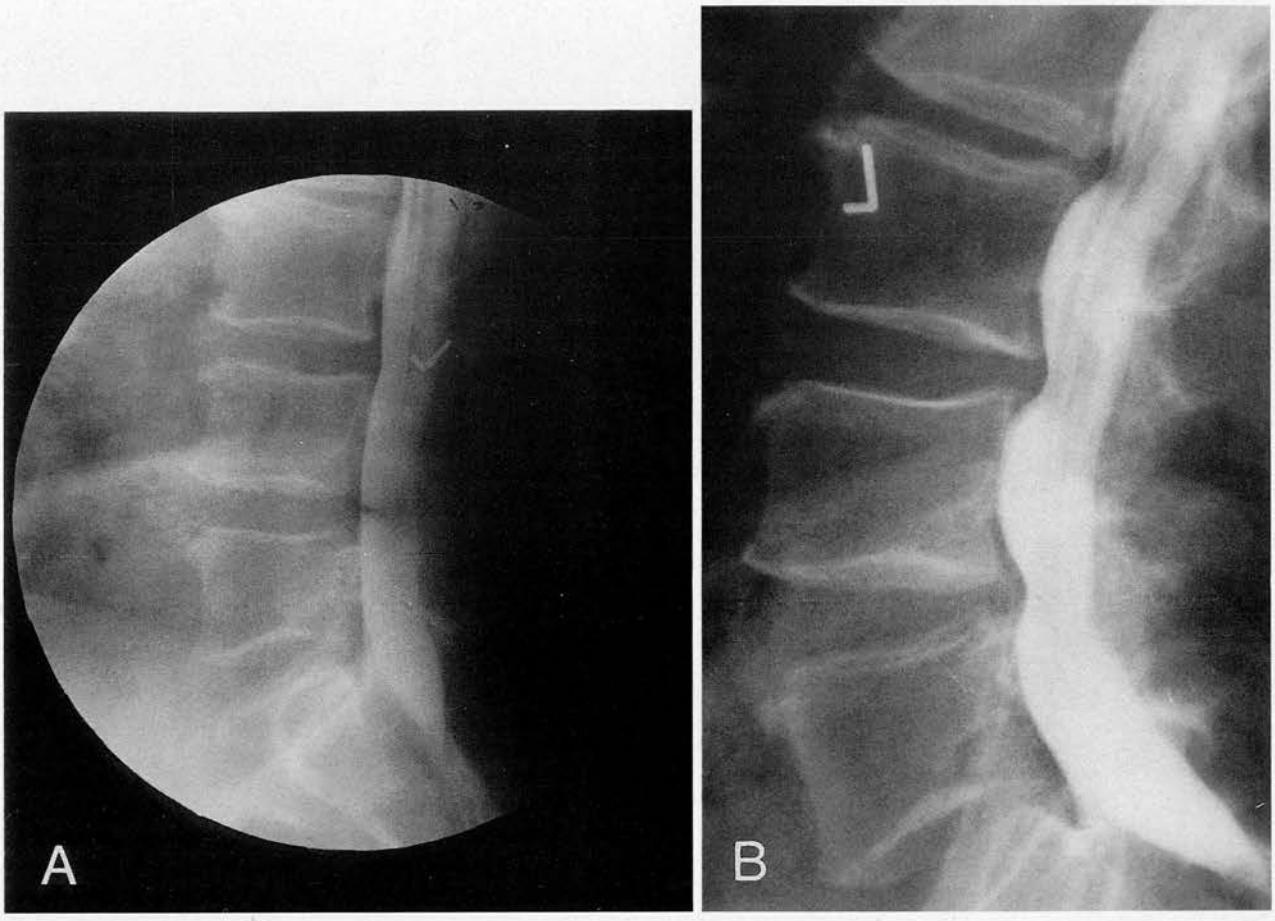

Fig 6. Lateral radiculogram of a patient with symptoms of neurogenic claudication in the flexed (**A**) and upright (**B**) posture.

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Address reprint requests to

R. W. Porter, MD, FRCS
Department of Orthopaedics
Doncaster Royal Infirmary
Armthorpe Road
Doncaster DN2 5LT
England

Accepted for publication March 30, 1989.

Reprinted from SPINE, Vol. 17 • No. 1, January, 1992

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Cauda Equina Dysfunction
The Significance of Two-Level Pathology

R. W. PORTER, MD, FRCS, FRCSE,* and D. WARD, FRCP†

The pathophysiology of neurogenic claudication is not well understood. It is generally believed that stenosis of the central vertebral canal is significant, but some believe that it is root canal stenosis or stenosis of the foramen that is important, not pathology in the central canal. There are, however, clinical anomalies incompatible with either view. In this article, 49 patients with neurogenic claudication were examined with myelography and computed tomography, recording the frequency of multilevel central canal stenosis and the presence of coexistent root canal stenosis. Of the 49 patients, 46 had either multilevel central canal stenosis or stenosis of both central and root canals. It was concluded that neurogenic claudication is generally associated with at least two levels of stenosis. A hypothesis of two-level venous compression, with venous pooling of one or several nerve roots, explains some of the pathophysiology of neurogenic claudication. This hypothesis is compatible with clinical and experimental observations, but it has yet to be confirmed. [Key words: neurogenic claudication, spinal stenosis, root canal stenosis, venous pooling]

IT IS GENERALLY ACCEPTED that central canal stenosis is an essential component to the pathogenesis of neurogenic claudication.[29] Many patients have multilevel central stenotic lesions,[9,10] and a one-level block is fairly rare in claudicating patients.[24] Some suggest, however, that stenosis in the root canal or at the foramen, not the central canal, is responsible for symptoms.[2,3,6,14,17] Our own observations suggest that in patients with neurogenic claudication, multilevel central stenosis is very common, but also that root canal stenosis frequently coexists with central stenosis. To assess the frequency of multilevel pathology in both central and root canals, we have examined a series of patients with unilateral and bilateral claudication symptoms.

METHOD

Forty-nine consecutive patients with neurogenic claudication attending a secondary or tertiary referral back pain clinic entered the study. They had pain in one or both legs above and below the knees which developed when walking, restricted walking distance to less than 500 m, and was relieved by rest. All patients were examined by myelography and computed tomography (CT) and had stenosis in the central and/or root canals. Those with two or more inappropriate signs[30] were excluded from the study.

Occlusion of the dura was recorded as "partial" when there was segmental narrowing of the dura without interruption of continuity of flow of the radiopaque material, "subtotal" when there was discontinuity of the radiopaque material but some flow beyond the stenosed segment, and "total" when there was no flow of radiopaque material beyond the block (Figures 1–2).

Computed tomography was performed at the lower three segments, and more proximally if the myelogram showed upper lumbar central stenosis. The sagittal and transverse diameters of the dural sac were measured at each disc level. The presence or absence of extradural fat was noted, as were the clarity of the nerve root image and hypertrophy of the ligamentum flavum.[32] The root canal diameter was measured at each segmental level; a root canal was considered to be stenotic if the diameter was 4 mm or less. The degree of degeneration of the apophyseal joints was recorded as "none," "mild," "moderate," or "gross," depending on the size of the facet osteophytes and the irregularity of the joint surfaces (Figure 3). No attempt could be made to evaluate measurement error or area of the dural sac on CT scans.

Lower limb circulation was examined by Doppler ultrasound and sometimes by arteriography; in addition, Doppler ultrasound studies were performed on 18 sex-matched patients of similar age who had no known spinal or vascular disease.

RESULTS

Forty-nine patients (36 male) were examined with neurogenic claudication. There were 30 patients with unilateral symptoms and 19 with bilateral symptoms. The mean age was 58.8 years ± 8.1 years. There were equal numbers of those with unilateral claudication who had pain in the right and left legs. Those with unilateral and those with bilateral symptoms had no significant differences in age, length of history of back pain, duration of claudication symptoms, and walking tolerance (Table 1). Twenty-four patients had some degree of vertebral displacement, degenerative spondylolisthesis being more common in the patients with bilateral symptoms and lumbar scoliosis more frequent in the patients with unilateral claudication (Table 2).

The results of myelography, CT measurements, and Doppler studies of the peripheral arteries are shown in Tables 3 and 4. L4–5 was the level most commonly affected by central canal stenosis, causing myelographic occlusion of the dural sac, but 39 (80%) had multilevel central canal occlusions (23%) with three levels and 5 (10%) with four levels affected. Seventy-six levels were partially occluded, 27 were subtotally occluded, and 7 had a total block of the radiopaque material. There were no significant differences in the number of levels affected and the degree of occlusions in the unilateral and bilaterally affected patients. The CT measurements of the dural sac showed reduction in size comparable to the myelographic occlusions. Computed tomographic measurements of the root canal showed stenosis of 4 mm or less in 37 (76%) of the patients, which was equally common in both unilateral and bilateral claudication. Of the 23 patients with unilateral claudication and root canal stenosis, 13 had the smallest diameter of root canal on the same side as the claudication, and in 10 the root canal was of equal size bilaterally; no patients had the greatest reduction on the contralateral side.

Of the 30 patients with unilateral claudication, 14 had a structural lumbar scoliosis. Five had claudication on the same side as the concavity of the curve, but five had symptoms contralateral to the concavity. Four more had a double lumbar curve, convex to one side in the midlumbar region and convex to the opposite side distally.

Ten patients had a one-level central occlusion on the myelogram. All were at the L4–5 level, and all but one had only a partial occlusion. The three patients with a single L4–5 occlusion and bilateral symptoms also

From the *Department of Orthopaedics, Aberdeen University Medical School, Aberdeen, Scotland, and the †Department of Radiology, Doncaster Royal Infirmary.

Accepted for publication September 3, 1991.

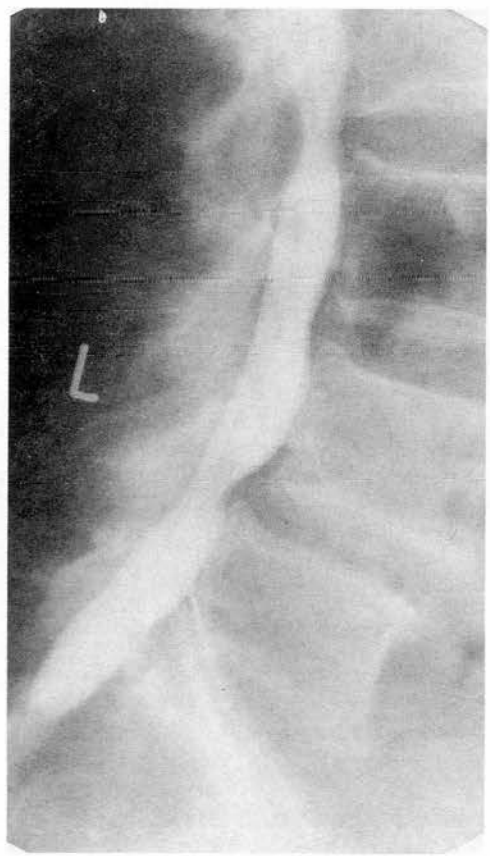

Fig 1. A two-level partial occlusion at L2–3 and a subtotal occlusion at L4–5 with degenerative spondylolisthesis at L4–5. The patient (Patient 5) is a 60-year-old man with a 13-year history of back pain and a 3-year history of bilateral neurogenic claudication limiting walking to 200 m.

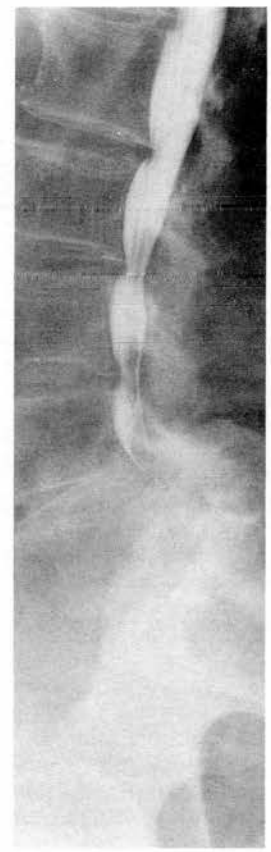

Fig 2. Lateral myelogram of a 72-year-old man (Patient 14) with a 3-year history of back pain and a 1½-year history of bilateral neurogenic claudication. The patient could walk only a few steps and was confined mainly to a wheelchair. Shown here are a total occlusion of the dural sac at L4–5, where there is a degenerative spondylolisthesis; subtotal occlusion at L3–4; and partial occlusions at L2–3 and L1–2.

had a bilateral root canal stenosis at L5–S1, two with gross facet joint degeneration at L5–S1. Of the seven patients with a one-level central stenosis at L4–5 and unilateral claudication, four also had root canal stenosis at L5–S1, two had a lumbar scoliosis with gross degeneration of the facet joints at L5–S1, and one had marked peripheral vascular disease.

Forty-six patients had an ultrasound Doppler examination of the peripheral arteries. Of the 46 patients, 11 had major disease in the aortoiliac or femoropopliteal segments and 7 had less marked disease in the posterior tibial or dorsalis pedis vessels; however, only 2 of the 18 control subjects had previously unrecognized mild arterial pathology on Doppler studies ($P < 0.05$).

DISCUSSION

These results suggest that multilevel stenosis is very common in claudicating patients. Ninety-four percent of patients had either 1) a two-level or multilevel central canal stenosis; or 2) a one-level central stenosis and associated root canal stenosis.

It would have been helpful to know the frequency of the multilevel pathology in a control population with no symptoms of neurogenic claudication, but this was precluded from the study for ethical reasons. We know from magnetic resonance studies, however, that symptomless spinal stenosis is not uncommon in the elderly population.[1,11,13,32] Furthermore, patients in this series presumably had multilevel stenosis for several years preceding the onset of their short history of claudication. Thus, it is not inevitable that multilevel stenosis will produce neurogenic claudication. Rather, our study suggests that neurogenic claudication is uncommon in the absence of multilevel stenosis.

What is the clinical significance of multilevel stenosis in neurogenic claudication, and does it help us understand the mechanism of the neurologic dysfunction? Can multilevel pathology explain some of the clinical anomalies in neurogenic claudication?

Attempts have been made previously to explain the pathogenesis of neurogenic claudication as a result of either central canal stenosis or root canal stenosis, but neither view satisfies the clinical anomalies of neurogenic claudication.[24] Why is neurogenic claudication uncommon in patients with a large central disc protrusion at L4–5, with a complete block of the cauda equina,[26,31] and why can a slow-growing tumor in the vertebral canal scallop the vertebral bodies and compress the cauda equina, but not produce claudication? Why does one patient with a complete block on myelography from central canal stenosis have bilateral claudication, and another patient with similar radiographic features have only unilateral symptoms? Unilateral symptoms could develop in the presence of a tight central canal stenosis as a result of unilateral subarticular entrapment in the lateral recess, but this generally produces root entrapment syndrome, with its constant, severe, unremitting root pain, not claudication pain. There is no satisfactory explanation as to why a single root trapped in a central canal stenosis will produce root entrapment pain in one case and unilateral claudication in another. If ischemia of the nerve root is responsible for neurogenic claudication, how can extensive segments of the abdominal aorta be replaced, with lumbar artery ligation and no subsequent claudication symptoms? These anomalies are difficult to explain in terms of a one-level central canal stenosis.

If, however, neurogenic claudication is the result of pathology in the root canal or at the foramen, why does central canal stenosis generally

Table 3. Results of Plain Radiographs, Myelography, Computed Tomographic (CT) Scans, and Doppler Studies in Patients with Bilateral Neurogenic Claudication

Patient No.	Plain Radiograph	Myelogram					CT*					CT†					CT‡				Doppler Ultrasound§
		L1-2	L2-3	L3-4	L4-5	L5-S1	L1-2	L2-3	L3-4	L4-5	L5-S1	L1-2	L2-3	L3-4	L4-5	L5-S1	L2-3	L3-4	L4-5	L5-S1	
1	No				P	P			10 11	7 7	7 8				4 4	4 4			M M	M M	Femoro popliteal disease bilateral
2	No				P				10 14	10 10	10 10			7 7	5 5	3 3			G G	G G	Aortoiliac femoro-popliteal disease bilateral
3	No				P	P				12 18	10 16				6 6	3 3			M M	M M	Normal
4	No			S	S	P			6 7	5 5	6 7			3 3	4 4	4 4		G G	G G	M M	Normal
5	D		P		S					7 7	8 9				8 6	8 8			G G	M M	Normal
6	LS			P	S									8 8	8 8	3 3		M M	M M	M M	Normal
7	D				S				9 8	8 5	7 7			4 5	4 4	4 4		N N	M M	N N	Normal
8	D			P	P				12 5	10 6	8 7			5 5	4 4	6 6		G G	G M	M M	Normal
9	No			P	P									5 6	4 4	6 7		M M	M M		Normal
10	No			P	S				6 6	6 7				7 7	6 5	5 6		G G	M M	M M	Normal
11	No				P				8 8	5 10				7 7	7 7	4 4		N N	M M	G G	Normal
12	D		P	S	T					7 6	10 10				7 7	6 6			G G	G G	Posterior tibial and dorsalis pedis disease bilateral
13	No				P	P				5 6	5 5				5 5	4 4			M M	G G	Normal
14	D	P	P	S	T				6 12	6 6	12 12			8 8	7 7	7 8		M M	G G	M M	Normal
15	D		S	T				8 10	6 6	8 8	8 10		4 4	6 6	4 4	6 6	G G	G G	G G	M M	Normal
16	No		P	S	T									6 6	4 4	6 6		M M	M M	G G	Femoro-popliteal disease bilateral
17	No			S					8 10	8 15	12 22			10 10	8 6	8 8		G G	G G	G G	Femoro-popliteal and posterior tibial disease bilateral
18	No	P	P	P	P				8 8	8 12	10 10			4 4	5 4	6 6		G G	G G	M M	Not done
19	D			P	P					8 10	10 12				5 5	4 5			M M	G G	Normal

D = degenerative spondylolisthesis; G = gross; LS = lumbar scoliosis; M = moderate; N = none; No = no displacement; P = partial occlusion; S = subtotal occlusion; T = total occlusion.
*Anteroposterior and transverse diameters of dural sac (in millimeters).
†Diameter of root canal right and left (in millimeters).
‡Facet arthritis right and left.
§Lower limb arteries.

Table 4. Results of Plain Radiographs, Myelography, Computed Tomographic (CT) Scans and Doppler Studies in Patients with Unilateral Claudication

Patient No.	Plain Radiograph	Myelogram					CT*				CT†				CT‡				Doppler Ultrasound§
		L1-2	L2-3	L3-4	L4-5	L5-S1	L2-3	L3-4	L4-5	L5-S1	L2-3	L3-4	L4-5	L5-S1	L2-3	L3-4	L4-5	L5-S1	
20	LS				P				15 14	12 12			7 8	6 6			M M	G G	Not done
21	LS	P	S	S	P		11 6	11 8	10 4	10 10	5 6	5 5	5 7	4 4	N N	M M	G M	M M	Normal
22	LS		S	T				10 5	10 10	16 16		5 5	6 6	4 4		G G	G G	M M	Normal
23	R				P			12 8	10 4	9 13		4 4	4 4	5 4		M M	M G	M G	Femoropopliteal disease (L)
24	No			P	P								4 4	6 6			M M		Normal
25	No				S	P			7 7	8 7			6 6	5 5			G G	G M	Normal
26	LS		P	S	T				7 7	9 8			4 5	4 4			G M	M M	Normal
27	LS	P	S	T				7 7	7 7	10 6		6 6	4 2	4 5		G G	G G	G G	Normal
28	LS				P							7 5	4 5	4 8			M M	N N	Normal
29	No				P	P							4 5	4 4					Aortoiliac femoropoliteal tibial disease (L)
30	LS			P	P				6 10	8 12		6 6	6 6	6 4		M M	M M	G G	Dorsalis pedis popliteal disease bilateral
31	D				P				5 7	8 10			4 4	3 5			G G	G G	Normal
32	No			P	T								4 4	4 5		M M	G G	M M	Normal
33	LS				P			10 10	5 5	10 10		7 4	6 6	5 5		G G	G G	G G	Posterior tibial and dorsalis pedis disease bilateral
34	No			P	P	P			10 10	10 10				4 6			M M	G G	Normal
35	LS			P	P	P		12 12	11 11	11 11		6 6	6 6	3 6		M M	G G	G G	Normal
36	D			P	S	P			6 6	6 6			4 4	5 5			G G	G G	Femoropopliteral (L) and posterior tibial disease bilateral
37	No				P	P		10 12	8 10	10 10		10 10	6 5	8 8		M M	G G		Doralis pedis and posterior tibial disease bilateral
38	No				P				10 10	16 17			8 8	4 4			M M	M M	Dorsalis pedis disease bilateral
39	No		P	S				10 14	12 18	12 12		5 5	3 4	4 4		G G	M M	M M	Normal
40	No				P				12 8	14 14			6 6	8 8			G G	M M	Femoropopliteral posterior tibial disease (R)
41	LS			P	S			5 10	5 10	14 12		7 7	6 6	6 6		G G	M M	M M	Dorsalis pedis disease (L)
42	LS	P	P	P	S			10 10	5 5	10 10		4 5	3 5	5 5		G G	G G	M M	Normal
43	No			P	S			10 4	5 5	12 12		6 6	5 5	7 7		M M	G G	N N	Normal
44	LS		S	P	P			6 6	10 10	12 14		6 6	5 5	4 4		G G	M M	G G	Normal
45	LS			S	P			7 7	6 6	7 7		5 3	4 6	4 6		G G	G G	G G	Aortoiliac femoropopliteral disease bilateral
46	No			P	P	P							4 5	4 4		M M	M M	G G	Aortoiliac femoropopliteal disease bilateral
47	No			P	P			10 18	10 14	12 10		8 5	5 6	4 6		G G	G G	G G	Dorsal pedis and posterial tibial disease bilateral
48	No		P	P	P	P		10 12	10 9	7 7		6 6	4 4	5 5		M M	M M	G G	Aortoiliac femoral disease (L) Dorsal pedis disease (R)
49	LS			P	P	P		5 5	5 5	6 6		5 5	5 4	5 5		G G	G G	G G	Not done

*Anterposterior and transverse diameters of dural sac (in millimeters).
†Diameter of root canal right and left (in millimeters).
‡Facet arthritis right and left.
§Lower limb arteries.
Note: See Table 3 for a list of abbreviations.

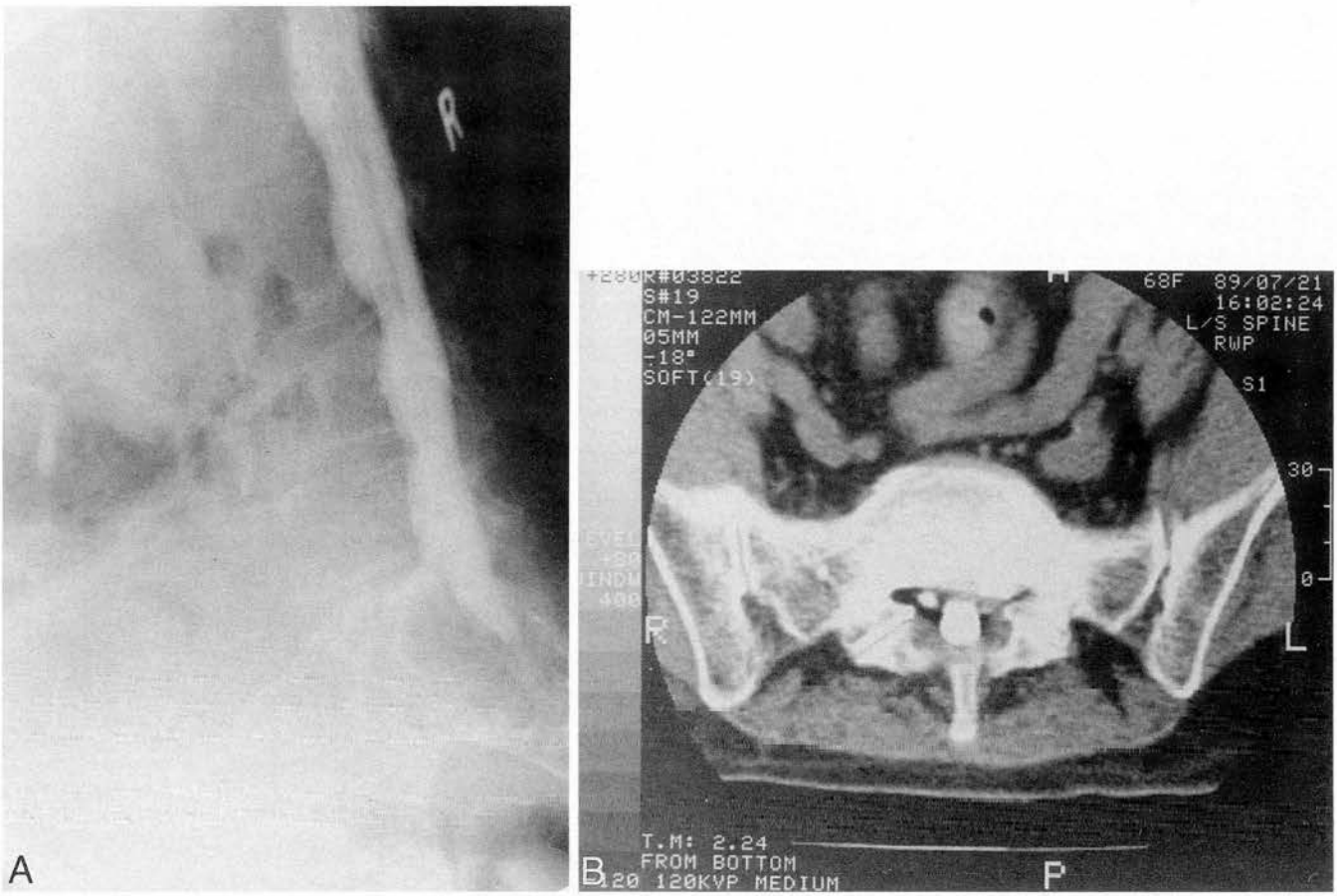

Fig 3. A, A two-level partial occlusion of the dural sac at L3–4 and L4–5 on the lateral myelogram of a 66-year-old man (Patient 47) who had a 5-year history of low-back pain and an 8-month history of left-sided neurogenic claudication above and below the knee. **B,** The CT at S1 shows gross degenerative change of the apophyseal joints, with an overhanging facet at S1 on the left.

coexist? And why would root canal stenosis produce root entrapment pain in one case and claudication pain in another? It is difficult to reconcile the symptom of neurogenic claudication to either central canal stenosis or root canal stenosis.

There is, however, a third possibility—that the symptoms of neurogenic claudication are the result of stenosis at two levels. Such a view is compatible with previously reported clinical observations of multilevel pathology in claudicating patients. It agrees with the observations of McGuire et al,[16] who reported two claudicating patients who had spinal tumors associated with pre-existing multilevel stenosis. It is also supported by our understanding of the circulatory anatomy of the cauda equina, and with experimental studies.[19,20]

The vascular anatomy of the cauda equina has been well documented.[4,6,21,22] The arterial supply is both proximal (from the spinal arteries) and distal (from the radicular arteries, which are branches of the lumbar arteries). Between these two supplies, there is a rich anastomosis in the proximal third of the cauda equina. Radicular veins drain centrifugally from each root to the foraminal veins.[4]

There is a proximal anastomosis around the conus but no drainage between intradural roots. Where the dural sleeve joins the nerve root, however, there is potential for both arterial and venous tributaries. Spencer et al[27] and Parke and Watanabe[23] described the variable extradural fixation and adhesions of the lumbar roots. There is a possibility of a minor segmental supply and drainage of the root between these dural attachments and the foramen, which could become important in the presence of stenosing pathology.

Because of its vascular anatomy even at low pressure, the nerve root will be at significantly greater ischemic risk from a two-level compres-

Table 1. Comparisons Between Patients with Unilateral and Bilateral Neurogenic Claudication

	Claudication	
	Unilateral	Bilateral
Number	30	19
Male	20	16
Age (yr)	58.8 ± 8.1	58.9 ± 8.3
History of back pain (yr)	14.8 ± 8.9	15.2 ± 8.3
History of claudication (yr)	1.8 ± 1.1	2.6 ± 2.5
Claudication tolerance (m)	139 ± 115	165 ± 124

Table 2. Vertebral Displacement in Patients with Unilateral and Bilateral Claudication

	Claudication	
	Unilateral (%)	Bilateral (%)
Vertebral displacement scoliosis	14 (47)	1 (5)
Degenerative spondylolisthesis	2 (6)	7 (37)
Retrolisthesis	1 (3)	0 (0)
No displacement	13 (43)	11 (58)

sion than from a one-level lesion. A one-level compression will affect only a small section of the nerve root at the site of the stenosis, with adequate arterial supply and venous drainage on both sides of the lesion. A two-level compression, however, at low venous compressive pressure, will affect a much larger segment of the nerve root. Two levels of central stenosis around the venous pressure will cause venous congestion and pooling of all the roots of the cauda equina between the blocks (Figure 4). A one-level central stenosis with a distal unilateral root canal stenosis will cause venous congestion of only one root (Figure 5). Capillary blood flow will decrease,[18] and although the nerve roots may receive some nutrition from the cerebrospinal fluid,[25] nerve root function will be impaired. Reflex vasodilatation from a buildup of metabolites will only increase the occlusive pressure.

The dynamic effects of walking will adversely affect a two-level lesion when there is already occlusion just below the critical level. Vasodilatation of the arterioles and capillaries of the cauda equina will cause an increase in the block pressure, venous stasis, and capillary stasis, leaving the oxygen needs of a large segment of the cauda equina unsatisfied.

A two-level hypothesis is supported by the myeloscopic studies of Ooi et al.[20] Their patients with neurogenic claudication did not show ischemia, but they did exhibit dilatation and venous engorgement of the cauda equina vessels when walking on a treadmill. This did not occur in the control group, which indicates that their finding was compatible with a two-level lesion. Similarly, the experimental studies of Olmarker et al[19] showed that a two-level porcine cauda equina compression at only 10 mm Hg produced much more pronounced effects on impulse propagation than a one-level compression at the same pressure. Furthermore, if symptoms are related to venous pooling rather than arterial ischemia, it is understandable that replacement of the abdominal aorta does not produce neurogenic claudication.

Several combinations of two-level lesions can cause different presentations of claudication. For instance, a two-level central canal stenosis, both around the venous pressure, could produce bilateral claudication symptoms when the pressure increases with walking (Figure 4). One level may have a higher block pressure than the other, but may produce symptoms only when the second level approaches venous pressure.

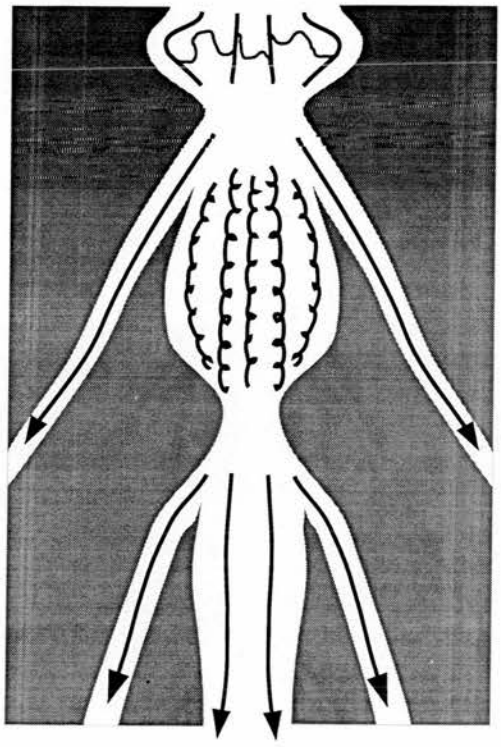

Fig 4. A two-level central canal stenosis near the venous pressure causes venous congestion and pooling in the nerve roots of the cauda equina, between the levels of stenosis.

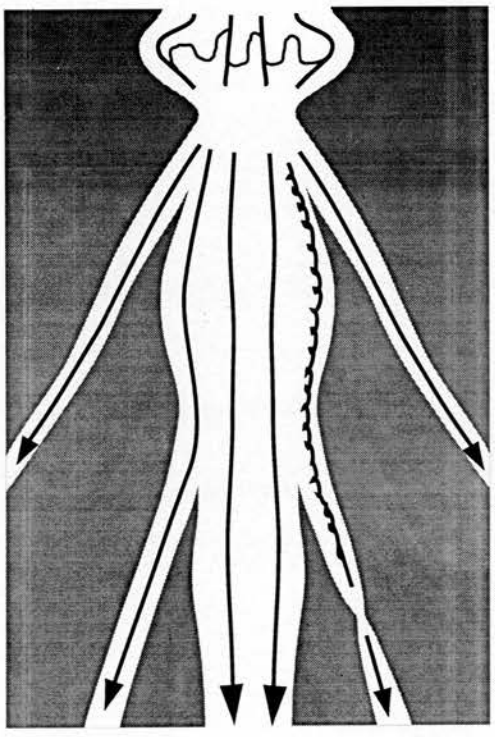

Fig 5. A one-level central canal stenosis combined with a one-level root canal stenosis near the venous pressure will produce venous congestion in one nerve root as well as pooling of venous blood.

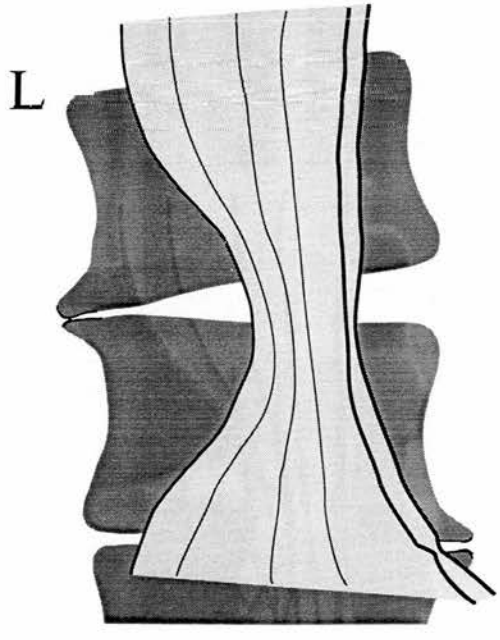

Fig 6. Diagram shows that a lumbar scoliosis concave to the left can cause central canal stenosis at the apex of the curve. One might initially expect left root claudication if the significant lesion were at the apex of the curve, but right L5 root canal stenosis can be part of a two-level lesion, with congestion of the L5 root and right-sided claudication.

Alternatively, a one-level central canal stenosis may also be associated with a single root canal stenosis, both around the venous pressure, and walking would then produce venous stasis of the single root as well as unilateral claudication (Figure 5). A one-level central canal stenosis associated with bilateral root canal stenosis would produce bilateral claudication.

If neurogenic claudication is the result of stenosis at two levels, then investigations should not seek the "significant level of block," but rather the "two significant levels of block." The root canal should be assessed as carefully as magnetic resonance imaging and CT scans will allow.

The concept of a two-level lesion has practical implications. It might be assumed that a total occlusion of the canal at L4–5 is the significant lesion. Decompression of the central canal at this level would fail to cure the patient if in fact the two significant lesions were a partial occlusion at L3–4 and root canal stenosis at L4–5.

If the pathology is related to fairly low block pressures around the venous pressure, it is understandable that conservative surgery is often adequate because decompression at one level will relieve symptoms from a two-level pathology. If low pressures are involved, however, symptoms can readily recur from epidural scarring.

Vertebral displacement with an intact neural arch was present in 50% of this series. There were some cases of degenerative spondylolisthesis with forward displacement and some cases of lumbar scoliosis with rotatory displacement. Forward displacement was generally associated with bilateral claudication and scoliosis with unilateral symptoms. The asymmetrical degenerative change in lumbar scoliosis may account for the variability of the side of symptoms, the claudication not necessarily being on the side of the concavity of the scoliosis (Figure 6). This is understandable with a two-level hypothesis, the proximal lesion being in the central canal at the apex of the scoliosis, and the distal lesion being at a lower level in the root canal on the convex side of the curve.

The combination of peripheral vascular disease and spinal stenosis can cause diagnostic difficulty. The two conditions are not easily distinguished by clinical tests,[7,8,28] and they commonly coexist.[12] In this series, significantly more patients with neurogenic claudication had pathology of the arteries of the lower limb, compared with 11% of the control subjects. It is possible that these patients represent a subgroup with arteriosclerosis, less able to adapt to ischemic changes in the nerve root.

There is probably more to neurogenic claudication than two-level stenosis. Its pathophysiology is undoubtedly complex, but a two-level stenosis at pressures near the venous pressure is a working hypothesis that could be highly significant.

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Address reprint requests to

R. W. Porter, MD, FRCS, FRCSE
Department of Orthopaedics
Aberdeen University Medical School
Polwarth Buildings
Foresterhill
Aberdeen AB9 2ZD, Scotland

Central spinal stenosis

Classification and pathogenesis

16

Richard W Porter

Department of Orthopedic Surgery, University Medical Buildings, Aberdeen AB9 2ZD, Foresterhill, Scotland
Tel +44-224 681818. Fax +44-224 685157.

Spinal stenosis is an abnormal narrowing of the bony and/or ligamentous structures of the vertebral canal. Only when it produces symptoms does it attract the clinician's attention, and then stenosis is only one factor amongst many which contribute to the syndrome. What are these syndromes, and what is the role of the small canal? What is the difference between stenosis of the central canal and stenosis of the root canal? And what is the etiology of the small canal?

The anatomy of the vertebral canal is bicompartmental. The first is the central canal—that space within the neural arch which extends to the next segmental level, with artificial boundaries at the proximal part of the root canal. The second compartment is the root canal—the space between the lateral part of the central canal at the intersegmental level extending laterally to the foramen. The shape of the central canal is variable. Most canals are dome shaped or bell shaped throughout the lumbar spine, but approximately 7% are trefoil at L4, and 15% trefoil at L5 (Porter 1980; Figures 1 and 2). The cross sectional area is also variable, and when a canal is trefoil, it also tends to be small in the sagittal diameter. A trefoil shaped canal has a deep lateral recess, with overhanging superior facets, whilst a dome shaped canal may have no lateral recess at all.

Classification

Congenital stenosis is assumed to be genetically determined. In achondroplasia, the canal is generally small in both sagittal and interpedicular diameters.

Developmental stenosis. The vertebral canal reaches maturity in the cross sectional area and in the sagittal diameter by four years of age (Figure 3). Thereafter the pedicular diameter widens, and the shape changes, but not the overall size. Environmental factors which impair growth before four years of age, particularly interuterine factors like toxins, drugs, alcohol, smoking, bacterial or viral infections, placental insufficiency, these can leave a permanently stunted canal, with no capacity for catch-up growth. The individual may become a six foot man but be hiding a small canal from some early growth deficiency. The whole canal from cervical to lumbar level is small.

Degenerative stenosis. Degenerative change of bone and/or soft tissues can affect isolated segments as a result of disc degeneration, or following a degenera-

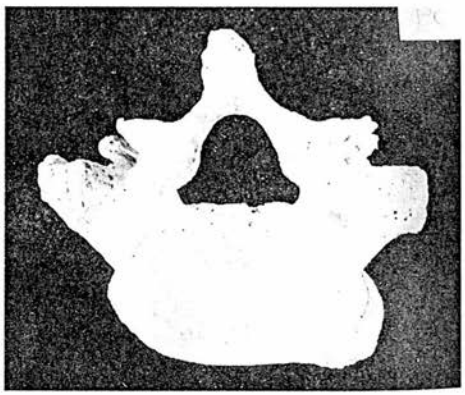

Figure 1. A fifth lumbar vertebra showing a large dome-shaped central vertebral canal

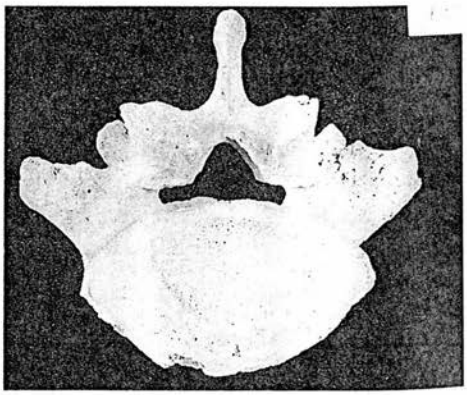

Figure 2. A fifth lumbar vertebra with a trefoil-shaped central canal, a small mid-sagittal diameter and deep lateral recesses.

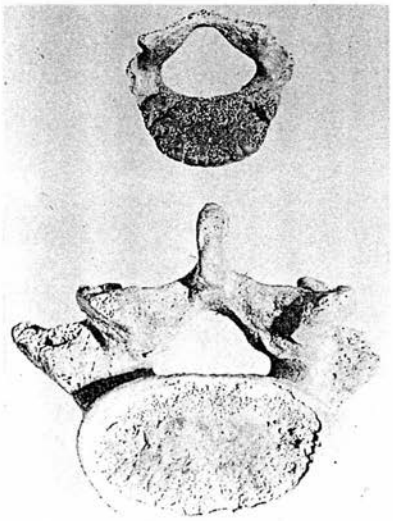

Figure 3. Two fifth lumbar vertebrae. The upper specimen is from a four year old child and the lower from an adult. The size of the vertebral body is different, but the cross sectional area of the vertebral canal is identical. The mid-sagittal diameter is slightly larger in the infant. In the adult the interpedicular diameter is wider.

tive spondylolisthesis. Alternatively many segments may be involved in systemic hyperostosis, or in degenerative lumbar scoliosis. Ossification of the posterior longitudinal ligament will stenose long segments of the spine.

Mixed stenosis is a combination of the above types, and it is this which usually occurs when stenosis produces symptoms.

Symptoms

Stenosis can be present with no symptoms at all. Boden and colleagues (1990) showed that 21 percent of asymptomatic subjects over 60 years of age had spinal stenosis. Stenosis is only one factor amongst many, but there are three syndromes where it is significant.

Neurogenic claudication. Patients experience pain or discomfort in one or both legs when they walk, but this is not present at rest. It is unusual before 50 years of age, and men are affected more often than women. They have little trouble at rest, but after a short distance their legs begin to feel uncomfortable, and at twice this distance they have to stop. They have multiple levels of stenosis, one always in the central canal and sometimes also in the root canal. There is coexistent peripheral vascular disease in about 30%.

Symptomatic disc protrusion. A protruding disc is not uncommonly symptomless. By 60 years of age, 36% of asymptomatic subjects have a herniated disc (Boden et al 1990). The presence of disabling symptoms depends to some degree on the available space for the nerve roots (Porter et al 1978). A small trefoil shaped canal places the root at risk from a postero-lateral bulge, but a far lateral disc can cause nerve involvement in the root canal even when the central canal is wide.

Root entrapment with degenerative changes. This can occur in the lateral recess of the central canal, though more commonly root entrapment is more distal in the root canal. The patient experiences quite severe pain in a root distribution, aggravated by extension, and often there are few abnormal neurological signs.

Pathogenesis

A single level central canal stenosis does not produce neurogenic claudication. If a nerve root is compressed by a bulging or sequestrated disc, this will produce a classical syndrome of root pain, worse on coughing, root tension signs, and perhaps a trunk list. But even with a massive and perhaps chronic protrusion, the patient does not have claudication symptoms. A slowly expanding tumor within the vertebral canal may produce bizarre symptoms, but not claudication. A single level degenerative process, with hypertrophied facet joints may cause a subtotal occlusion of the canal without claudication. Furthermore, experimentally the cauda equina of a dog can be occluded by 25% of its cross sectional area without causing any neurological deficit (Delmarter et al 1990).

A single level stenosis of the lateral recess or of the root canal or foramen, may cause thickening and inflammation of the nerve root with severe root entrapment pain, but not claudication.

Patients with neurogenic claudication generally have two or more levels of stenosis. There may be two levels of central canal stenosis or a single level of stenosis in the central canal, and a more distal root canal stenosis. The former tends to give bilateral claudication, and the latter claudication in one leg (Porter and Ward 1992).

A single level stenosis probably causes little neurological dysfunction, as the nerves are well supplied with oxygenated blood from a proximal and distal supply. The veins drain distally to the foramen, and proximally to the conus where they anastomose with other veins and drain distally along other roots to their respective foramina (Figure 4). A two level low pressure stenosis however will produce more profound effects. The arterioles will supply the uncompressed segment between the two blocks, but the venous return

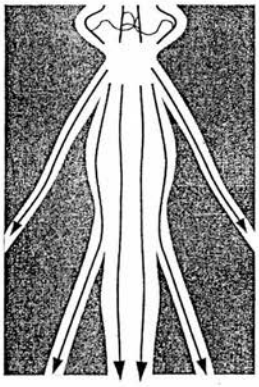

Figure 4. A single level central stenosis causes little vascular impairment.

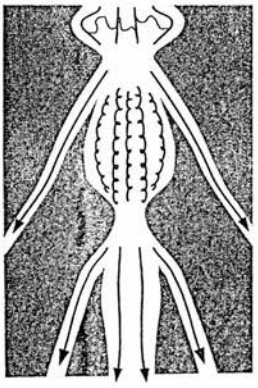

Figure 5. A double level central stenosis at low pressure, results in considerable blood pooling

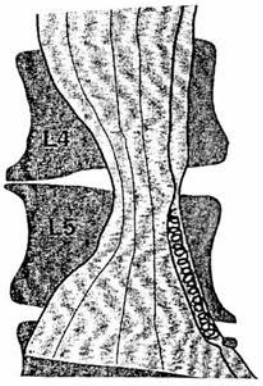

Figure 6. A single central stenosis and a more distal root stenosis causes pooling in the veins of a single root.

will be impaired, and a long segment of cauda equina will become congested. There will be a build up of metabolites, and reduced blood flow with impaired nutrition. In a two level central canal stenosis, all the cauda equina will be congested with bilateral symptoms (Figure 5). When there is a single level central stenosis with a more distal root canal stenosis, only a single root will be congested, with unilateral symptoms (Figure 6).

Forty percent of patients with bilateral claudication have a degenerative spondylolisthesis, with a second level of more proximal stenosis. In unilateral claudication, 50% have a degenerative lumbar scoliosis, with a central stenosis at the apex of the curve and an asymmetrical distal root canal stenosis.

Although spinal tumor does not cause claudication symptoms, McGuire and colleague (1987) reported patients with neurofibromata who had claudication symptoms. In addition to the spinal tumor, they had a previously asymptomatic spinal stenosis.

Exercise is associated with vasodilatation of the cauda equina. If the block pressure of one or both of stenosed segments is just below the venous pressure at rest, then as the cauda equina increases in volume with the dynamics of walking, the pressure can increase to above the venous pressure. This will result in blood pooling, and a reduced flow in the uncompressed segment between the blocks. The patient then experiences claudication symptoms and has to rest until there is a reduction in pressure at the stenosis.

This hypothesis agrees with the clinical and experimental observations of claudication. A single level compression of 10 mmHg in a porcine cauda equina model had little effect on function, whilst a two level compression of 10 mmHg caused marked reduction in blood flow by 64% (Takahashi et al 1992), and significantly reduced protein transport and nerve conduction (Olmarker et al 1992). A two level compression below arterial pressure is also supported by myeloscopy studies, which show not an ischemic, but a congested cauda equina in claudicating patients (Ooi et al 1990).

Although venous compression is the most acceptable explanation for the pathophysiology of neurogenic claudication, its frequent association with peripheral vascular disease has not been explained. It is possible that neurogenic claudication is not a homogeneous condition but a common expression of a number of different pathologies.

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Reprinted from SPINE
Vol. 18, No. 5 April 1993

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Measurement of Vertebral Foraminal Dimensions Using Three-Dimensional Computerized Tomography

G. A. Smith, BSc, R. M. Aspden, PhD, and R. W. Porter , MD, FRCS, FRCSE

The dimensions of lumbar intervertebral foramina were measured in cadaveric spines using three-dimensional computerized tomography. Six different image reconstruction protocols were used. The results were compared with measurements of the same foramina using calipers after dissection. All the three-dimensional computerized tomographic measurements underestimated the true foraminal dimensions. The best agreement, as well as the best images, were obtained using 4/3 slices and a reconstruction threshold of 300 Hounsfield Units. This method is not recommended for measurement of foraminal dimensions. However, there may be a use in assessment and planning surgical management. [Key words: intervertebral foramen, foraminal size, nerve root, entrapment, computerized tomography]

The purpose of this study was to assess the accuracy and reproducibility of measurements of the dimensions of the five pairs of lumbar intervertebral foramina from three-dimensional computerized tomographic (3DCT) images of isolated lumbar spines. Data were recorded using different slice thicknesses and images reconstructed with different thresholds to find the optimum protocol. Comparison was made with caliper measurements across the same points in the dissected spines.

The intervertebral foramina are small apertures situated between the pedicles of adjacent vertebrae. Each foramen forms the opening of the nerve root canal through which passes the nerve root and various blood vessels. We have taken the foramen to be the smallest aperture along the nerve root canal. Restriction of the dimensions of a foramen in the lumbar region due to soft or hard tissue intrusion is commonly associated with symptoms of nerve root compression.[13,18] The most common foraminal shape is oval[4,23] but this may become an inverted pear shape[11] or even auricular[21] especially in the presence of concomitant disc degeneration.[23] Foraminal heights are reported to be 11–19 mm[14] and 13–15 mm.[4] The only reported measurement of foraminal width appears to be that by Magnusson[14] who gives an average size of 7 mm from front to back. The nerve root occupies up to about 50% of the area of the foramen and, as it lies in the cranial portion of a lumbar foramen,[19] due to its oblique passage from the spinal cord, is likely to be adversely affected by restriction of the foraminal width or by a proximally directed osteophyte from the vertebral endplate.[7]

Observations using conventional planar radiography cannot make allowance for the changing orientation of the root canal from being almost horizontal in a parasagittal plane at upper lumbar levels to an anterocaudal direction at the sacrum. Computerized tomography (CT) is more useful for displaying the presence and location of many osseous and soft tissue changes associated with root entrapment.[2,7,20] In one study it demonstrated 75% of bony lesions found to be causing entrapment at surgery and was superior to myelography.[24] Principal drawbacks are its limited ability to image soft tissue and, as with planar radiography, the difficulty of matching the image plane to the plane of the foramen. Gantry tilt, criticized by Butt,[3] and multiplanar reformatting[7,10] have been used in attempts to obtain different planes of section and this latter is now widely used for assessment of root canal pathologies.[7] However these images are still two dimensional in nature, so a series of them must be produced to evaluate the length of a canal, and the plane chosen will generally still not contain the particular foramen selected.

Three-dimensional CT has been increasingly used for display of complex spinal injuries.[5,6,9,16,17,25,26] Spinal deformity, degenerative change, bony stenosis and nerve root entrapment have all been identified in this way[5,17] and Zinreich claimed that additional information was available from 3D images in more than half of patients examined.[25,26] However display artifacts have been reported that could cause confusion[12] and soft tissue changes such as disc herniation are not clearly shown.[17] No attempt has been made to take

From the Department of Orthopaedic Surgery, University of Aberdeen, Scotland.
Accepted October 5, 1992.

measurements from 3D CT images of the spine, although two studies testing the accuracy of surface rendered 3D CT images from other sites both give promising results. Measurements of distances between implanted stereotactic markers in cadaveric knee joints gave an accuracy of 0.68 mm when compared with direct measurements,[22] whereas those from dry skulls using recognized cranial landmarks gave an accuracy of 1.5 mm.[8] No test was made of the effects of altering other imaging parameters such as reconstruction thresholds or slice thickness.

■ Materials and Methods

Materials. Results described here were obtained from 77 foramina in 8 lumbar spines removed from human cadavers during routine postmortem examination. None had any history of spinal deformity, metastatic disease or back pain. Five were male and three female and their ages ranged from 58 to 77 years. The entire lumbar spine, the superior half of the sacrum and parts of the adjacent iliac bones were removed *en bloc* to preserve the normal anatomy of the foramina. Most of the spines were frozen immediately at -20 C until measured. However, two spines were scanned and dissected first to test whether freezing affected the measurements. The specimens were kept moist at all times during scanning and dissection. Five pairs of foramina from L1–2 to L5–S1 were studied from each spine, except two at L1–2, which were damaged when the spine was removed and another at L5–S1 which was partially obscured by a bony and cartilaginous growth.

Imaging. After removal of the spine from the cadaver air rapidly enters the subarachnoid space and can lie at a number of places close to the intervertebral foramina. Because air can considerably affect the CT images[15] it was displaced by injecting margarine into the nerve root canals and the spinal canal; margarine has a similar roentgen ray absorption (-70 Hu) to the soft fat normally present in the foramina (-50 to -100 Hu). To do this, the spine was brought to room temperature and held in an upright position. A Medicut intravenous cannula and a 20 mL syringe was used to inject warmed, semi-liquid margarine into each of the foramina in turn starting at L5 and working proximally. After injecting each pair of foramina a needle was inserted between the adjacent vertebrae at the midline and approximately 50 mL margarine injected intradurally to restore the normal dural outline.

The specimens were scanned using a standard lumbar spine protocol on a Siemens Somatom DRH CT scanner (Siemens AG, Karbouke, Germany). The tube current was 540 mA and the tube potential was reduced to 96 kV to slightly degrade the image quality to that found in CT imaging of a whole body, otherwise the absence of abdominal contents leads to considerably superior image quality. Images were stored in a 512 × 512 matrix at a zoom factor of 4.0. Each spine was scanned twice along its whole length; first using a slice thickness of 4 mm with a 3 mm spacing (4/3 images) and then with a slice thickness of 2 mm spaced at 1 mm intervals (2/1 images). Overlapping scans are recommended for the reconstruction programs. The gantry was not tilted. Three-dimensional images were reconstructed from the raw data using "3-D Display" software (© Siemens AG, 1987). Each foramen was reconstructed separately, to minimize time and data storage requirements, rather than forming a 3D reconstruction of the whole spine, and this was done six times: for each of the two slice thicknesses three images were made using thresholds of 150 Hounsfield units (Hu), the default bone threshold, 80 Hu, which should include some soft tissue structures which make up the foramen, and 300 Hu more clearly to delineate the cortical bone. The reconstructions were made as if looking at the root canal from the vertebral canal, as though the spine had been halved in the sagittal plane. These internal measurements gave a better representation of the foramina, which could not be seen so clearly from an external view, either using CT or directly, because of overlying structures. The reconstruction step was repeated on two specimens to test the repeatability of this process. The height and width were measured for each foramen.

The foraminal dimensions were determined using a light pen interactively on the 3D image stored in the computer memory. A point was marked on one margin and the pen moved over the opposite margin within the 3D matrix to find the minimum distance. The height of the foramen was measured between the superior and inferior pedicles and the width was bounded anteriorly by the vertebral body and posteriorly by the ligamentum flavum, though this was not always properly included in the image depending on the reconstruction threshold. The same criteria were used to determine the dimensions in the dissected specimen so that there should be direct comparability between these measurements. Each measurement was made ten times with reconstruction thresholds of 80 and 150 H, and subsequently using a 300 Hu threshold with 4 independent measurements. A limitation of the system is that it could only measure in steps of 1 mm. To test the repeatability of the reconstruction process, a second 3D CT image was made and the measuring procedure repeated for all the foramina in two spines.

There was no calibration certification with the software so to test the accuracy of the system a phantom was constructed. This consisted of 16 plastic tubes, of length 100 mm, internal diameter 10 mm, external diameter 20 mm, glued together in square array and placed in a water bath. The phantom was scanned both across and along the tubes using the same protocol as for the spines and a 2/1 slice thickness. Reconstructions were made from both data sets as though looking down the axis of the tubes.

Caliper Measurements. After scanning, the foramina were dissected and calipers used to measure the interior margins for comparison with CT measurements. Frozen specimens were halved in the sagittal plane using a band saw to enable an internal measurement to be made in the same way as for the imaging method. After thawing, the dural sac and epidural space were cleared and the nerve roots, ganglia and any fat removed by blunt dissection. Externally the psoas muscle was removed along with the lumbar plexus. A plastic plate was clamped over the intervertebral discs to prevent nuclear material bulging out and to maintain the normal disc height. Again, ten separate measurements were made of each dimension using a pair of internal calipers modified so as to be able to record distances as small as 3 mm.

Distances were recorded to 0.1 mm. The external dimensions of three pairs of proximal foramina were measured twice in two spines to ensure no changes occur during freezing and sectioning. Measurements were made immediately after removal from the cadaver and again after freezing and sectioning. The distal foramina could not be seen externally and were not used in this assessment.

Statistics. The caliper and CT measurements were compared using scatter diagrams and plots of difference against mean.[1] Correlation is not appropriate in this case as it simply determines whether two variables vary together in a linear fashion and measures the strength of that relationship. Thus, two variables can be very highly correlated but differ considerably in their actual values. The methods used here enable a comparison of the both the nature and the magnitude of the agreement between the CT measurements and those made using calipers. Where measurements were made twice, that is, before and after freezing and after repeated reconstructions with the same parameters, repeatability was assessed using a *t* test and values of the parameters are given where appropriate.

■ Results

Phantom

Two 3D reconstructions of the phantom were made from scans taken either perpendicular to or parallel with the bore of the tubes. Measurements from these images matched the phantom's dimensions to within the 1 mm measurement limitation of the equipment. No systematic or scaling errors were observed over the range of distances (5–80 mm) measured.

Caliper Measurements

Ten independent caliper measurements were made of each foraminal height and width and expressed as a mean and standard deviation. Figure 1 shows the average male and female foraminal heights and widths measured from these specimens. The apparently greater height of the female distal foramina is not statistically significant ($P > 0.05$). Comparison of the external measurements on two spines using a paired *t* test showed that freezing and sectioning the specimens had no significant effect on the dimensions ($n = 12$, $P > 0.05$).

Three-Dimensional Computerized Tomographic Images

The height and width of the 77 lumbar foramina were measured from a 3D CT image and expressed as mean and standard deviation. This was done for each combination of slice thickness and reconstruction threshold. These data are too numerous to tabulate and as the aim of the experiment is to compare these with caliper measurements they will be discussed in more detail later. The number of slices needed for each image varied between 9 and 11 for 4/3 images and 21 to 24 for 2/1 images, more being required at more distal levels. Figure 2 shows an L5-S1 foramen imaged using the protocols described and compared with a photograph. The ligamentum flavum and posterior margin

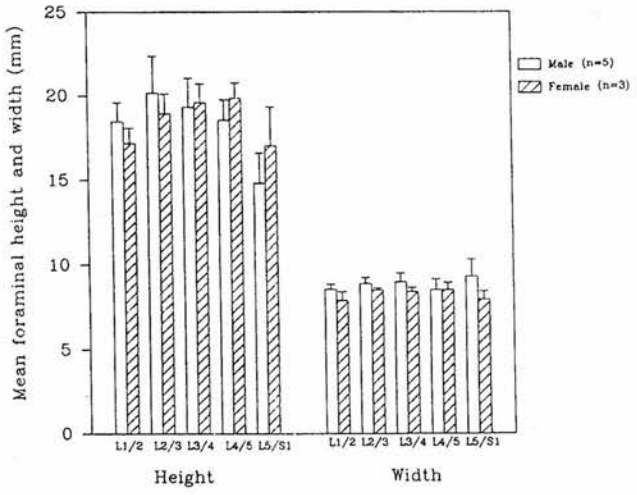

Figure 1. Heights and widths of the invertebral foramina measured using calipers from dissected cadaveric spines. The results are mean values and associated standard deviations from 5 men (n=10 at each level) and 3 women (n=6). The apparently greater height of distal female foramina is not statistically significant.

of the disc seen in the photograph are not visible in the CT images because of choice of threshold, even at 80 Hu. Step artifacts are more apparent with thicker slices. To determine whether a lower threshold would improve the soft tissue inclusion some images were reconstructed with thresholds as low as 30 Hu. Only the fat is excluded at this threshold and the nerve can clearly be seen in Figure 3. Unfortunately this considerably reduces the apparent dimensions of the foramen making imaging of small foramina impossible.

Comparison of CT with Calipers

Scatter plots of 3DCT against caliper measurements and the corresponding difference versus mean plots are shown in Figure 4 for the foraminal heights and Figure 5 for the widths for reconstructions at 80, 150, and 300 Hu. Measurements of foraminal height using 3D CT consistently underestimated the true value as measured using calipers. The width measurements under-estimated small foramina but appeared to overestimate larger ones as is shown by the difference in Figure 4 changing from negative to positive as foraminal width increases. The mean difference between the two measures and its standard deviation is shown in Table 1 for both height and width for all the protocols used. The foraminal width measurements made using 3D CT appeared to be larger than the true values because, even using the lowest reconstruction threshold, there was incomplete inclusion of soft tissue and essentially what was measured was the bony dimensions. This contrasts with direct width measurements which were made between the ligamentum flavum posteriorly and the vertebral body anteriorly; if caliper measurements were made to the bony margins

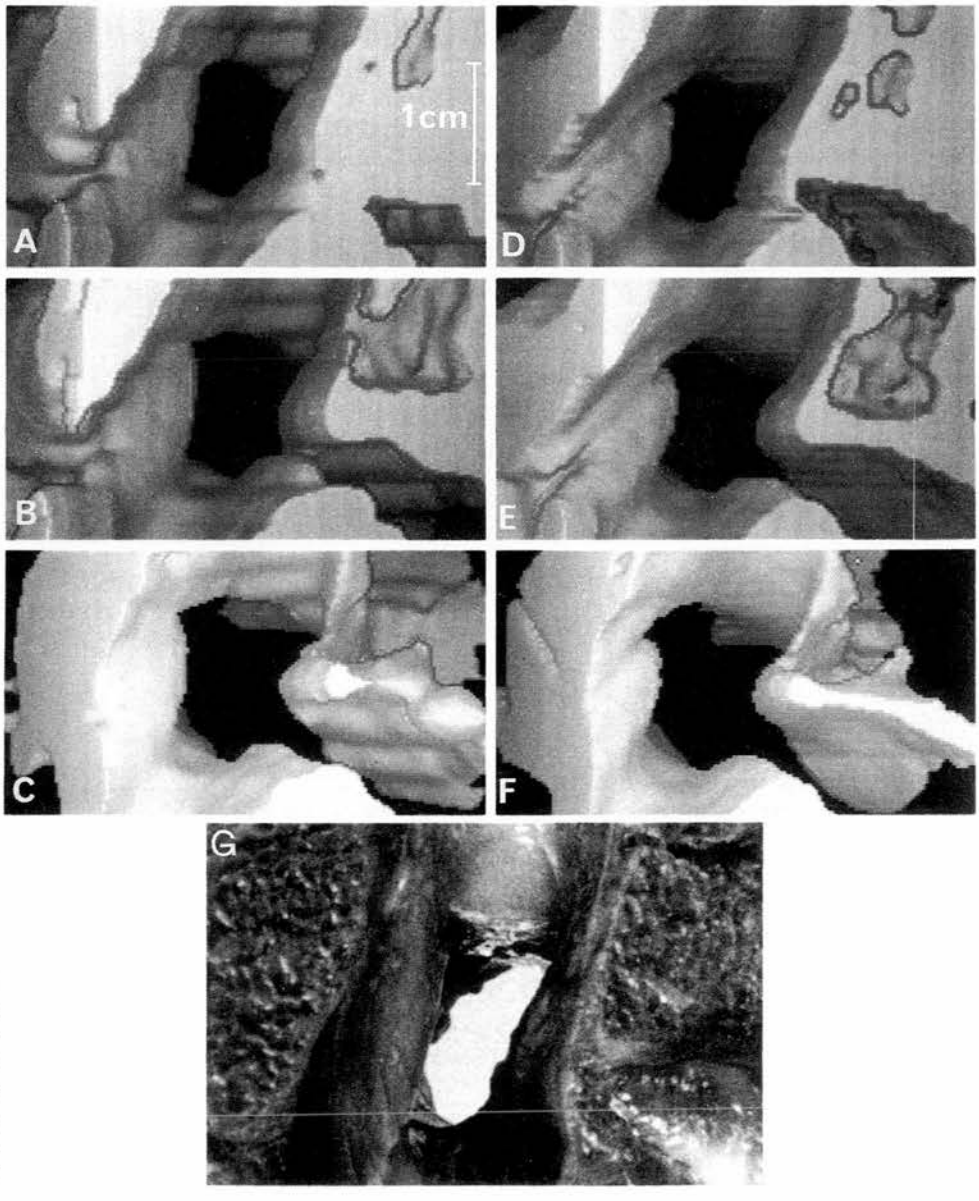

Figure 2. Internal views of an L5-S1 foramen seen as though looking from the vertebral canal and compared with a photograph of the same foramen after dissection. The parameters for each image are A, 4/3, 80 Hu; B, 4/3, 150 Hu; C, 4/3, 300 Hu; D, 2/1, 80 Hu; E, 2/1, 150 Hu; F, 2/1, 300 Hu.

then they were again larger than the 3D CT measurements. Repeated reconstruction and measurement of foraminal dimensions were significantly different ($P < 0.001$). However this difference was 0.1 mm and is therefore negligible when compared with the precision of an individual measurement which can only be made to ± 1 mm. The high significance of this difference arises from the large number, 120 pairs, of measurements made.

■ Discussion

Our results provide the first quantitative assessment of computerized tomography for the measurement of intervertebral foraminal dimensions. The experimental conditions were made to simulate as closely as possible those that might be found clinically. The spines were oriented in the same way as would be a person in the scanner and the accelerating voltage was reduced to degrade the image quality as though the spine were surrounded by abdominal contents. Unfortunately, although the quality of the images is extremely good, the results show that it is not a reliable method for measurement. Images reconstructed with a threshold of 300 Hu and a slice thickness of either 2/1 or 4/3 gave the most accurate representation of the intervertebral foramina. However the radiation dose given for 2/1 slices is high, twice that for 4/3 slices, and the images still are not accurate enough to justify the use of this protocol.

One of the major problems of the 3D imaging system we used is that the software creates images with voxel dimensions of 1 mm in each direction. This considerably affects the measurement of small dimensions such as the foraminal width where movement of

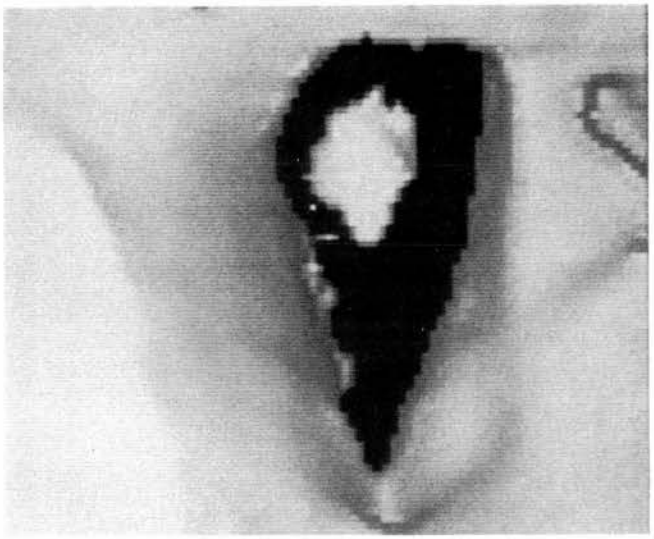

Figure 3. Internal view of an L1/2 foramen using a reconstruction threshold of 30 Hu. The nerve may be seen in the upper part of the foramen.

the light pen between adjacent voxels could make a difference of 20% in the measurement. The method consistently underestimated measurements made on the spines although the results from the phantom were very accurate. However, unlike the phantom, the tissues comprising the margins of the foramina are not uniform. The height of the foramen is defined by the bony pedicles whereas the width is defined mainly by soft tissues, posteriorly by the ligamentum flavum and antero-distally by the intervertebral disc. It was expected, therefore, that the 3D CT width measurements would overestimate the true width because of the difficulty of including soft tissue in a CT scan; ligamentum flavum would be missed and the posterior foraminal margin would be formed by the anterior surface of the apophyseal joint. For this reason a low reconstruction threshold of 80 Hu was included. However, in practice, this over-measurement serves to partially correct the general underestimate resulting in the width measurements appearing to be closer to the true values. A lower threshold served only to artificially narrow the foramina still further and gave images which were less easy to interpret.

If this underestimate was consistent then in principle a systematic correction could be applied to all measurements. Figure 4 shows that for height measurements this may be possible as there is an approximately Gaussian distribution of the individual differences about the mean difference. However the difference-mean plots of foraminal widths in Figure 5 show that the underestimate tends to increase with decreasing measured distance. More data are required to characterize this fully but it becomes doubtful whether such a systematic correction would be reliable.

The reason for the underestimated measurements is not clear. Partial volume effects, where a pixel takes a value proportional to the weighted mean of the absorptions, mean that a pixel is preferentially assigned a value appropriate to bone even though only a small proportion of it is occupied by bone and this would tend to narrow the foramina. This should affect the height more than the width as the orientation of the specimen in the scanner means that the width of a foramen lies in the plane of the slice, where the pixel size is 0.28 mm, whereas the height needs several slices to define it and these are of considerably lower resolution dictated by the slice thickness and spacing. Although there is a greater discrepancy in the heights than the widths the results are not much improved on changing from a 4/3 image to a 2/1 image. This effect might, however, be altered by other factors such as the way in which the software performs the interpolations and averaging required to form a three-dimensional image with uniform voxels from the raw data. Information on this was not available from the manufacturers.

Three-dimensional CT images reconstructed using this software consistently underestimate the true foraminal dimensions and cannot be recommended for quantitative measurement. However, 3D CT does have some positive advantages. It can provide clear qualitative images of the path of the nerve root canal, especially when 3D coronal or axial views are studied together, and enables good localization of bony lesions that may be a cause of root entrapment. An example of this is shown in Figure 6 where a growth, be-

Table 1. Overall Mean Difference Between 3D CT and Caliper Measurements for the Various Protocols Used

	4/3 Images			2/1 Images		
	80 Hu	150 Hu	300 Hu	80 Hu	150 Hu	300 Hu
Height (mm)	-5.0 ± 1.4	-4.0 ± 1.2	-2.4 ± 1.3	-3.6 ± 1.5	-3.1 ± 1.5	-2.4 ± 1.6
Width (mm)	+0.6 ± 1.3	+0.5 ± 1.5	+1.2 ± 0.4	+0.2 ± 1.3	+1.2 ± 1.4	+1.7 ± 1.4

Values are mean ± SD. The mean difference should be zero if only random errors are present (Figures 4 and 5).

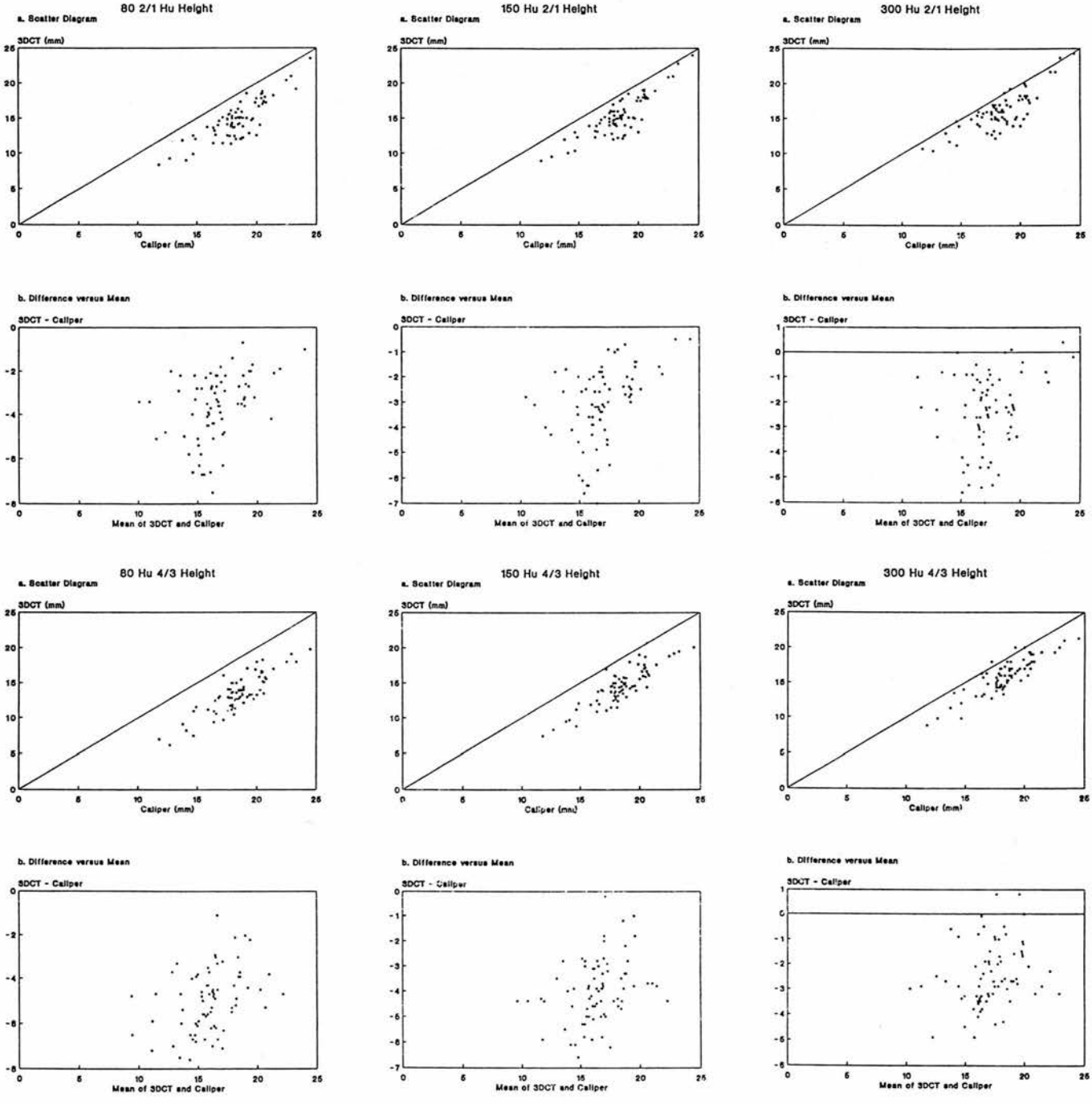

Figure 4. Scatter plots of 3DCT measurements versus caliper measurements and plots of the difference between these measurements versus their mean for all foraminal heights. The reconstruction parameters are indicated on each plot. Perfect agreement with only random measurement errors would cause the scatter plot to be a straight line with a slope of 1 and the difference versus mean plot to be Gaussianly distributed about zero.

lieved to be an osteochondroma, appears to be blocking the right L5–S1 foramen. However a coronal 3D reconstruction shows that the canal is clear if somewhat narrowed. We suggest that if CT is used for the assessment of root canal stenosis then a 3D reconstruction can quickly be made subsequent to the examination from standard contiguous 4/3 images. The radiation dose given to the patient need not be higher than for conventional CT scanning. Some detail is lost by using 4/3 slices and measurements are underestimated but the images are good enough to give a clear representation of the root canal. We found that a high threshold of 300 Hu for image reconstruction provided the most representative image of bony structures, soft tissues are not properly represented even at low thresholds and can confuse the image. This method, therefore, can be a useful adjunct to CT scanning. It takes very little more time and clearly

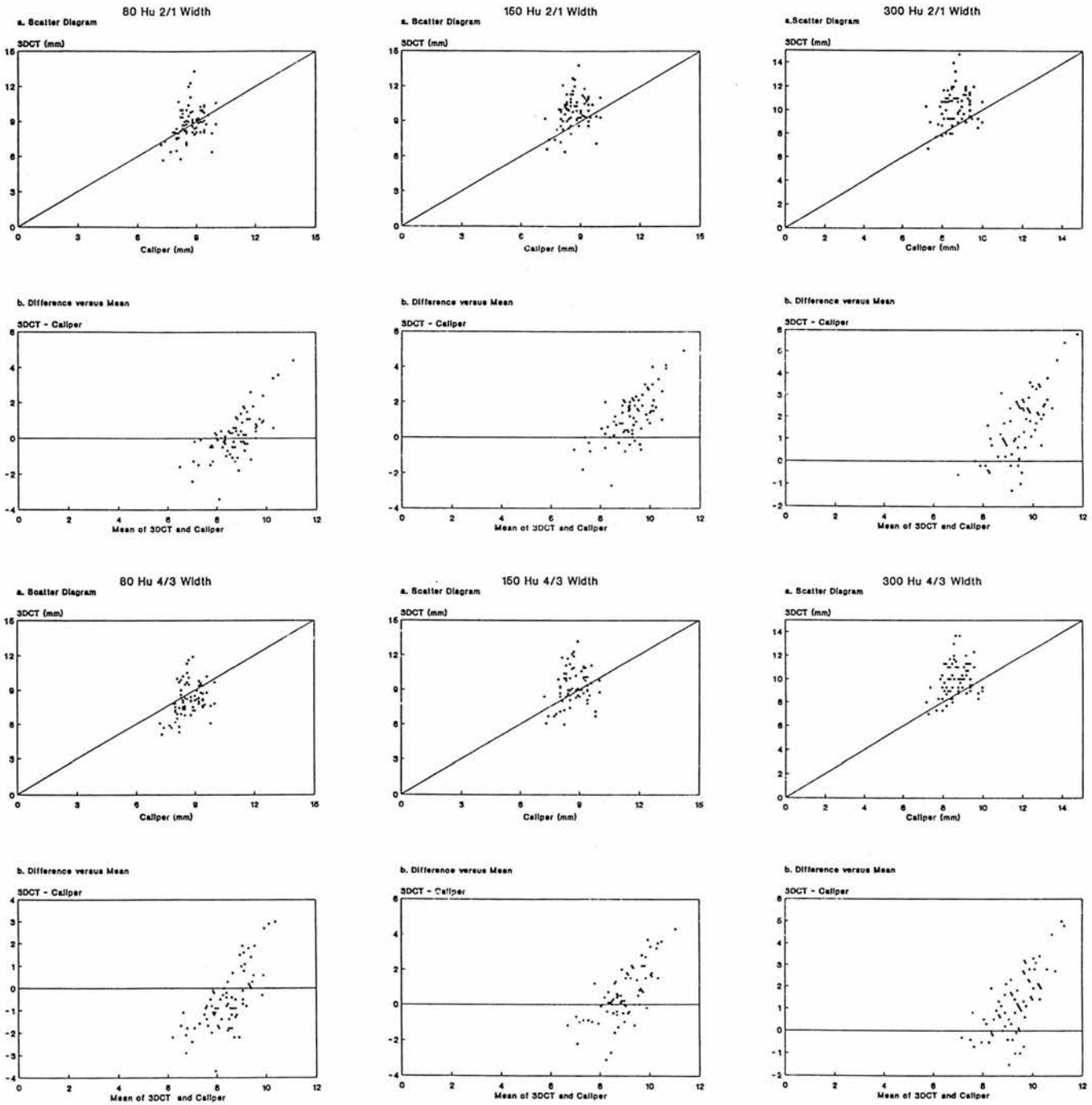

Figure 5. Scatter plots of 3DCT measurements versus caliper measurements and plots of the difference between these measurements versus their mean for all the foraminal widths. The reconstruction parameters are indicated on each plot.

shows the root canals and foramina in a way that is better than conventional CT.

Acknowledgment

We thank Dr A. F. MacDonald for use of the CT facilities in Aberdeen Royal Infirmary, staff of the Radiography Department for assistance with the scanning and M. Campbell for advice on the statistical analysis.

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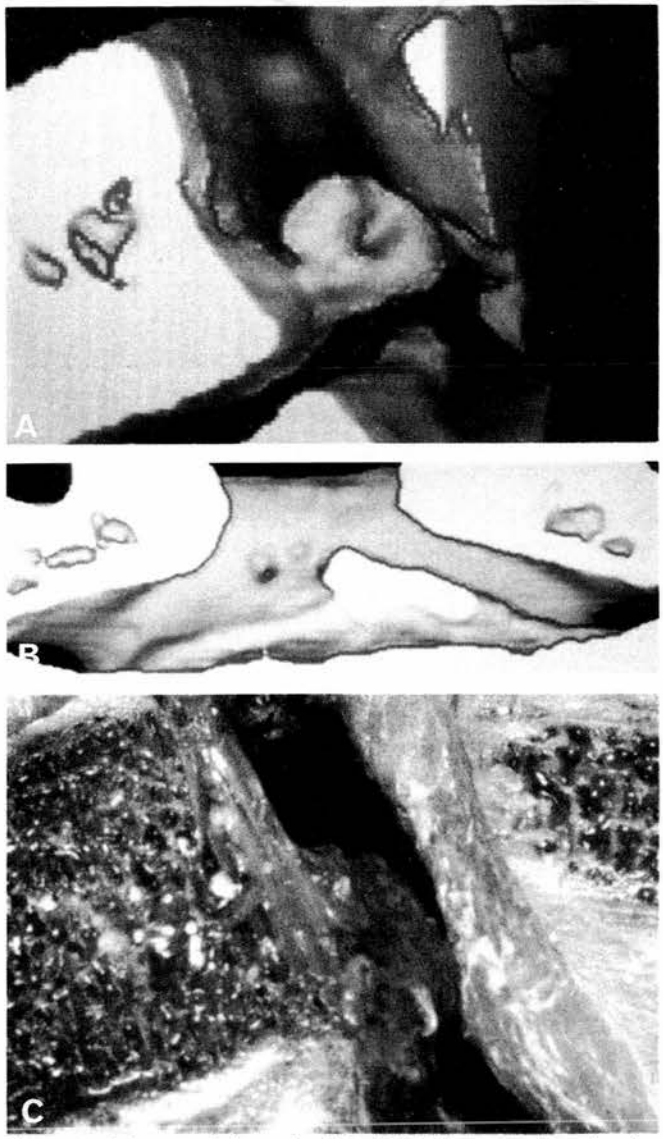

Figure 6. **A**, Internal view of a right L5-S1 foramen showing the presence of a bony growth apparently obstructing the canal. **B**, A coronal section shows that the canal is clear. **C**, Photographic appearance of the canal for comparison with **A**.

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Address reprint requests to

Dr. R. M. Aspden
Department of Orthopaedic Surgery
University Medical Buildings
Foresterhill, Aberdeen
AB9 2ZD, Scotland

Journal of Orthopaedic Research
11:104-109 The Journal of Bone and Joint Surgery, Inc., Boston

Double-Level Cauda Equina Compression: An Experimental Study with Continuous Monitoring of Intraneural Blood Flow in the Porcine Cauda Equina

‡Keisuke Takahashi,†Kjell Olmarker, *Sten Holm, §Richard W. Porter, and *†Björn Rydevik

**Department of Orthopaedics, Sahlgren Hospital, †Laboratory of Experimental Biology, Department of Anatomy, Gothenburg University, Gothenburg, Sweden, ‡Department of Orthopaedic Surgery, School of Medicine, Kanazawa University, Kanazawa, Ishikawa, Japan, and §Department of Orthopaedic Surgery, University of Aberdeen, Aberdeen, Scotland*

Summary: Compression of the spinal nerve roots may occur clinically at multiple levels at the same time: however, the basic pathophysiology of multi-level compression is largely unknown. In this study, the intraneural blood flow was analyzed continuously in the uncompressed segment between two compression balloons, with a pig used as an experimental model and a thermal diffusion method. At 10 mm Hg compression, there was a 64% reduction of total blood flow in the uncompressed segment compared with pre-compression values. Total ischemia occurred at pressures 10-20 mm Hg below the mean arterial blood pressure. After two-level compression at 200 mm Hg for 10 min, there was a gradual recovery of the intraneural blood flow towards the baseline. Recovery was less rapid and less complete after 2 h of compression. Double-level compression of the cauda equina can thus induce impairment of blood flow, not only at the compression sites, but also in the intermediate nerve segments located between two compression sites, even at very low pressures. These findings may have clinical importance in the understanding of the pathophysiology of multiple-level cauda equina compression.

Cauda equina compression is a common clinical condition. Nerve-root compression is often limited to only one location, but it can occur at double or multiple levels, particularly in patients with neurogenic claudication (16). Experiments have also shown that a two-level compression impairs nerve conduction more than a single-level compression at the same pressure (12); however, the basic mechanisms for this observed difference are poorly understood. It has been suggested that since there is no regional vascular supply to the nerve roots, double-level compression would induce blood-flow impairment in the nerve root segments located between the two compression sites, as well as at the compression sites. The aim of this study was to perform a continuous analysis of compression-induced changes in intraneural blood flow in the uncompressed segment of the cauda equina located between two compression sites.

Received December 13, 1991; accepted June 19, 1992.

Address correspondence and reprint requests to Dr. K. Olmarker at Laboratory of Experimental Biology, Department of Anatomy, Gothenburg University, Box 33 031, S-400 33 Gothenburg, Sweden.

MATERIALS AND METHODS

A total of 12 pigs, weighing 25-40 kg, were anesthetized with an intramuscular injection of 20 mg/kg body weight of Ketalar (ketamine, 50 mg/ml) (Parke-Davis, Morris Plains, NJ, U.S.A.), and an intravenous

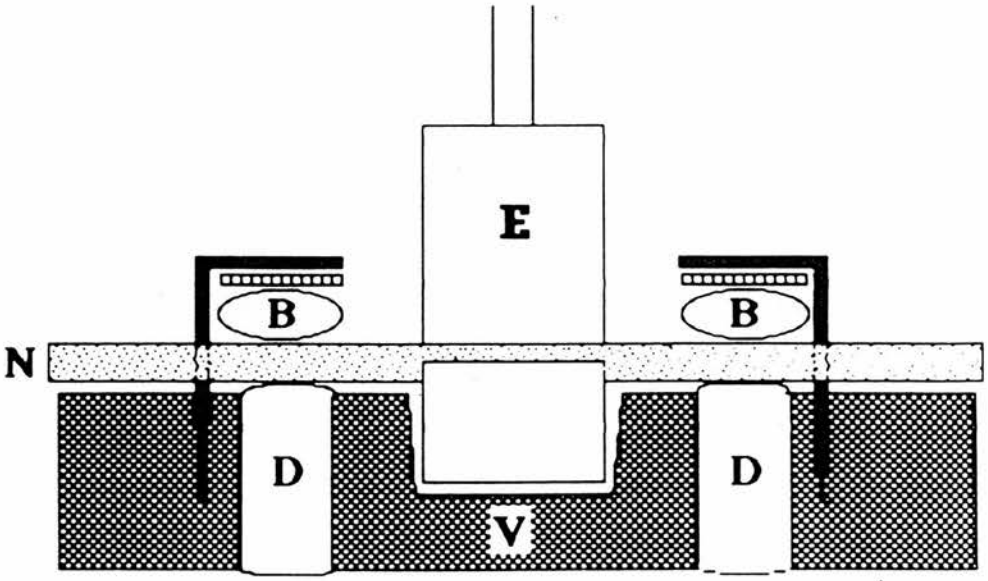

FIG. 1. Side-view of the experimental setup. The registering electrodes (E) are placed between the two compression balloons (B) in close contact with the cauda equina. The cauda equina is compressed by the balloons towards the adjacent intervertebral discs (D). To minimize elevation of the cauda equina, the posterior parts of the underlying vertebral body (V) have been removed. N = nerve.

injection of 4 mg/kg body weight of Hypnodil (methomidate chloride, 50 mg/ml) (AB Leo, Helsingborg, Sweden), and 0.1 mg/kg body weight of Stesnil (azaperone, 2 mg/ml) (Janssen Pharmaceutica, Beerse, Belgium). The pigs were tracheotomized, intubated, and ventilated on a respirator with room air. Anesthesia was maintained by additional intravenous injections of 2 mg/kg body weight of Hypnodil and 0.05 mg/kg body weight of Stresnil. The mean arterial blood pressure was continuously registered by a catheter in the thoracic aorta connected to a pressure transducer (P23; Gould Statham Instruments, Hato Rey, Puerto Rico) and a polygraph recorder (Grass 7B; Grass Instrument, Quincy, MA, U.S.A.).

Compression Model

The pigs were placed prone, and the cauda equina was exposed by a laminectomy of the three upper coccygeal vertebrae. Epidural fat and facet joints were also removed. One inflatable plastic balloon was placed over the cauda equina at the level of the CoI-CoII disc and another was placed at the level of the CoII-CoIII disc (10,11). The balloons, which were made of thin, pliable, polyethylene sheaths welded into cylinders which were sealed at one end and connected to a polyethylene tube at the other, were fixed to the vertebral body by L-shaped pins and Plexiglas plates (Fig. 1). Both balloons were connected to a graded compressed nitrogen system (Stille-Wemer, Stockholm, Sweden). When inflated, both balloons compressed the cauda equina towards the underlying discs at the same time and at the same compression pressure level. This compression model has been shown to have a high accuracy in pressure transmission from the balloon to the cauda equina (10,11).

Blood Flow Analyses

Intraneural blood flow was measured continuously with the thermal diffusion method described by Carter et al. (1), Koshu et al. (6), and Takahashi et al. (20). A special registration probe was developed for the pig cauda equina. Two gold plates, with a diameter of 1.0 mm and separated by 5 mm, were molded over the inner surface of the probe facing the cauda equina. The probe had a wire heater coiled around one of the two gold plates. A thermocouple (T-04-UE; Tokyo Wire, Tokyo, Japan) was attached to the center of the gold plate, and the difference in temperature between the two plates was continuously monitored. A thermo-gradient tissue blood flow monitor (BTG-42; Biomedical Science, Kanazawa, Japan) was used to measure the temperature difference between the gold plates as differences in thermal electromotive force. When the probe was attached closely to the cauda equina, this temperature difference was directly related to the total tissue

blood flow (4). The posterior part of the CoII vertebral body was removed to allow space for the registration electrode and to avoid elevation of the cauda equina (Fig. 1). In these experiments, CoII nerve roots were cut bilaterally and reflected proximally, since these roots were not compressed by both balloons. The blood flow was analyzed in the CoIII to CoVII nerve roots bilaterally. The humidity and local tissue temperature of the nerve roots were kept constant by continuous irrigation with saline solution at 37°C.

Experimental Procedures

Series I: Blood flow was recorded during incremental steps of compression and increased by 10 mm Hg at each step, until the segment of the cauda equina was completely ischemic. After each incremental increase of pressure, the blood flow was allowed to stabilize at a new level before the next increase.

Series II: When the analyses in series I were completed, the balloon inflation pressure was increased to 200 mm Hg and was maintained for 10 min (n = 6) or for 2 h (n = 6), in the same pigs as were used for series I. Recovery of the blood flow was studied for 10 min after release of the compression.

After each experiment, cardiac arrest was induced by an intravenous injection of 1 ml/kg of 15% potassium chloride solution. The temperature difference between the gold plates when there was no blood flow could thus be determined, and was used — together with the baseline values of blood flow before compression — for the calculations of relative changes in blood flow.

TABLE 1. *Relative reduction of intraneural blood flow, as compared with baseline, in the intermediate segment of the cauda equina at different compression pressure levels*

Compression pressure level (mm Hg)	Mean reduction of blood flow (±SD) (%)
10	64 ± 16
20	79 ± 15
30	86 ± 15
40	93 ± 10
50	96 ± 8
60	98 ± 4
70	99 ± 2
80	100 ± 1

N = 12 at all pressure levels.

RESULTS

The mean arterial blood pressure, as recorded by the aortic catheter, was found to be 72 mm Hg (SD = 6, n = 11). The systemic blood pressure was

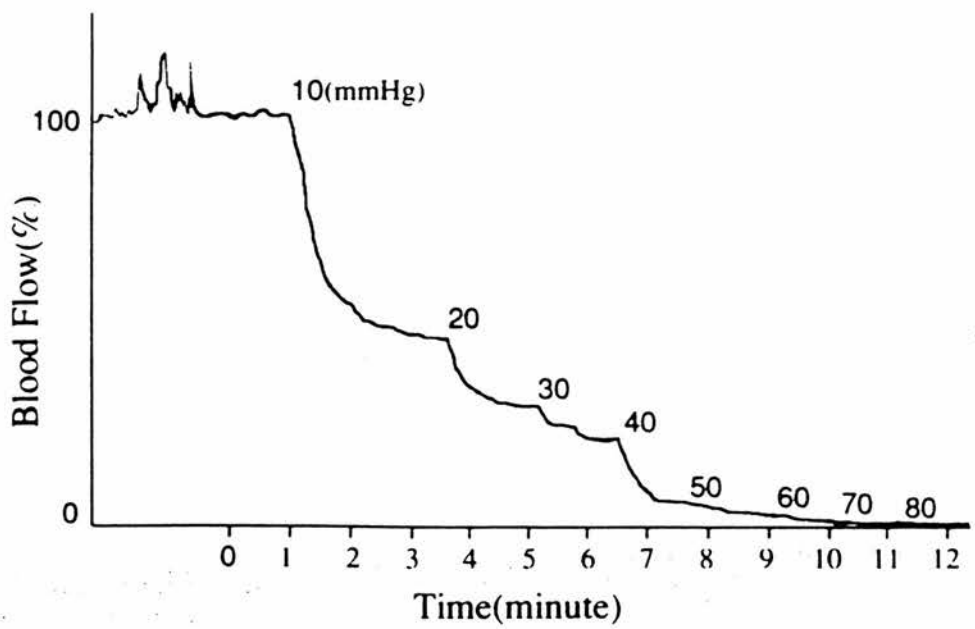

FIG. 2. Registration of compression-induced changes in the intraneural blood flow in the intermediate segment between two compression balloons in one animal. Blood flow impairment started immediately after the onset of each new pressure level, but there was a period of about 3 min during which blood flow gradually adapted to the applied pressure. At pressures exceeding 80 mm Hg, no further reduction of blood flow was observed in this experiment. This blood flow level was confirmed as "no blood flow" by calibration after cardiac arrest.

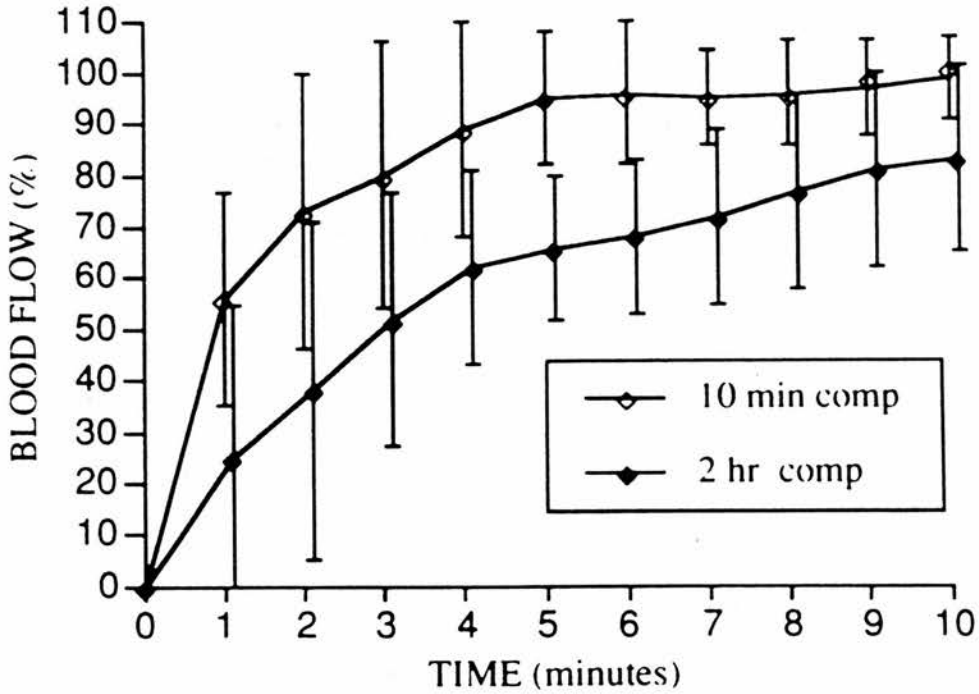

FIG. 3. Recovery of intraneural blood flow following decompression after compression at 200 mm Hg, once for 10 min and once for 2 h. In the 10 min compression series, the blood flow was almost completely restored within 3-5 min following release of pressure. In the 2 h compression series, the recovery of blood flow was slow and incomplete.

not changed by compression or release of compression of the cauda equina in any of the experiments.

Series I: The blood flow gradually decreased after onset of each new pressure level, and it stabilized at a new level within approximately 3 min (Fig. 2). Percentage reductions of blood flow levels, as compared with baseline values, for each applied pressure level are shown in Table 1. Even at 10 mm Hg compression, there was a 64% reduction of the intraneural blood flow in the uncompressed part of the cauda equina located between the two compression sites. With increasing pressure, there was increasing impairment, until at 50-60 mm Hg there was an almost complete stasis of blood flow. Ischemia was complete at 70 to 80 mm Hg.

Series II: The blood flow was rapidly restored after release of 200 mm Hg compression that was maintained for 10 min (Fig. 3). Recovery was less rapid when the cauda equina had been compressed at 200 mm Hg for 2 h, except for in two animals in which there was a relatively rapid restoration. No hyperemia was noted, except for in one animal in the 10 min compression series in which the blood flow level showed transient values that were higher than before compression. However, while the blood flow in the 10 min compression series was almost fully recovered within 10 min after decompression, the same was not true in the 2 h compression series.

DISCUSSION

The results of this study demonstrated that there is also a compression-induced impairment of the blood flow in the *uncompressed* nerve segments located between two compression sites in experimental nerve-root compression. This effect was seen even at pressures as low as 10 mm Hg, when there was a 64% reduction in intraneural blood flow. After compression at 200 mm Hg for 10 min, there was almost complete recovery of blood flow within 10 min, whereas recovery was less complete after compression at 200 mm Hg for 2 h. These findings indicate a relationship between the duration of compression and the time course of recovery of blood flow in nerve roots following decompression.

It was recently shown that nerve impulse propagation is significantly more impaired when the cauda equina is compressed at two levels than at one level, at the same compression pressure (12). However, the basic mechanisms for such a functional difference are not fully understood. It was suggested that the vascular anatomy of the spinal nerve roots

might, at least in part, explain this phenomenon (12).

The intrinsic vessels run within the nerve root tissue and are derived both from the spinal cord vessels and from peripheral vessels at the intervertebral foramen. There are thus both proximal and distal intrinsic vessels, which anastomose in the upper half of the nerve roots (13-15). By contrast, the peripheral nerves receive vessels from surrounding structures which approach the nerve trunks segmentally along their course (7). The intraneural blood flow has been shown to be relatively unaffected by the transecting nerve trunk in both the proximal and distal segments, which is an indication of the importance of the segmental vessels in the peripheral nerve vascular supply (7,18). Unlike peripheral nerves, however, the nerve roots do not achieve any regional blood supply by similar local branches (10,11,13-15). This implies that if blood flow is impaired at two locations, the blood flow in the segment between these two locations might be impaired as well. The results of the present study, in which compression at two levels induced a pronounced impairment of blood flow in the uncompressed nerve segment located between the compression sites, support this theory. In addition, preliminary data from a study of compression-induced impairment of the transport of nutrients to the nerve tissue indicate that the impairment is similar between the compression zones and the uncompressed intermediate segment (M. Cornefjord et al., unpublished data). These latter results also imply that the results of the present study might be regarded as an indirect measurement of the blood flow within the compressed segments as well.

Complete ischemia occurred at 70-80 mm Hg. Rydevik et al. (17) and Olmarker et al. (8) showed, through the use of a vital microscopic technique, that the pressure required to stop the flow in the arterioles in peripheral nerve and nerve roots, respectively, was close to the mean arterial pressure. These findings correspond well with the results of the present study, in which the mean arterial blood pressure was 72 mm Hg.

Surprisingly, a compression pressure of only 10 mm Hg induced a pronounced decrease in the blood flow of the intermediate segment (Fig. 2, Table 1). It is difficult to explain this reaction on the basis of arteriolar compression alone, but this pressure level previously had been found sufficient to induce venular congestion in the cauda equina (8). There have been several reports about the significance of venous congestion in compression-induced ischemia of the peripheral nerves and the nerve roots (2,5,8,19,21). Venous congestion induces an increase in venous resistance, and thereby also may reduce the blood flow in capillaries. At all compression pressure levels below 80 mm Hg, there may be some arteriolar blood flow. The presence of oxygenated arteriolar blood may result in leakage of substances such as toxic oxygen compounds, proteolytic enzymes, and long acting oxidants from the leukocytes (3). Such metabolic products may induce changes in the intraneural microenvironment and in such a way may also be related to changes in the normal function of the nerve tissue, as well as being involved in pain mechanisms.

Compression was found to induce a gradual impairment of intraneural blood flow for approximately 3 min. This slow onset of blood flow impairment might be related to the onset time for intermittent neuroischemic symptoms in spinal stenosis, as seen with postural changes of the spine and exercise. When compression was ended, there was a gradual recovery of the intraneural blood flow towards the baseline. The restoration time for intraneural blood flow seemed to be dependent on the length of time the nerve roots had been exposed to compression. The recovery of blood flow started immediately after decompression, and was almost recovered in 3-5 min. This gradual recovery might be considered to be one mechanism for the recovery from neurogenic intermittent claudication, and may thus be related, to give one example, to the delay between the moment when a person with such a condition stops walking and relief of symptoms.

Although this study was restricted to acute compression of the cauda equina, the results clearly indicate that double-level compression may induce a more widespread impairment of cauda equina blood flow than single-level compression. Therefore, there are reasons to believe that double-level nerve root compression may lead to more pronounced clinical symptoms than single-level compression. However, the gradual development of spinal stenosis may allow for some compensatory neuronal and vascular mechanisms.

CONCLUSIONS

In the present study, we demonstrated that the intraneural blood flow of the uncompressed parts of the cauda equina between two compression sites was impaired by acute compression and significantly reduced by even a low compression pressure. Compression was found to induce a gradual impairment of intraneural blood flow which stabilized at a new

level within approximately 3 min. The time to restoration for intraneural blood flow after decompression seemed to be dependent on the duration of compression.

Acknowledgment: This work was supported by grants from the Swedish Medical Research Council (8685, 9758), the Asker's Research Foundation, the Carin Trygger Memorial Foundation, the Hjalmar Svensson Research Foundation, the Salus Research Foundation, the Tore Nilssons Research Foundation, the Felix Neubergh Research Foundation, the Gothenburg Medical Society, and Gothenburg University.

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SPINE Volume 19, Number 2, pp 173–175

A Ten-Year Prospective Study of Vertebral Canal Size as a Predictor of Back Pain

19

R.W. Porter, MD, FRCS, FRCSE, and B. Bewley

This prospective study compared ultrasound measurement of the lumbar vertebral canal with subsequent back pain; 669 mining and nursing recruits had been measured before 1980, and 450 replied to a postal questionnaire in 1990. There was no significant difference in the prevalence of back pain or the need to be off work with back pain, in those whose canals were above and below the mean. However, going to bed with back pain was more frequent in those with small canals and approached significance ($P = 0.068$). Those with small canals were more likely to visit doctors and have treatment for back pain. Canal measurement is not a predictor for back pain, but as a risk factor for severe back pain in early working life, it approaches significance. [Key words: vertebral canal, back pain, ultrasound]

A small vertebral canal is a significant factor for many patients with back pain.[2,3,4,10,11,12,13] Many patients with symptomatic disc protrusion have a small canal,[8] and an inverse relationship exists between vertebral canal size and the morbidity of disc protrusion.[6] Patients who had back pain and were attending a general practice had significantly smaller canals than matched patients who had back pain and did not attend a practice.[1] Canal measurements of patients who attend a back pain clinic showed that 39% of them had canals below the tenth percentile for the general population.[9]

A small vertebral canal has clinical significance, but the prognostic importance of a small canal has not been established. We report a prospective study of back pain in nursing and mining recruits in whom vertebral canals were measured by ultrasound.

Method

Before 1980, a series of nursing and mining recruits had their vertebral canals measured by ultrasound,[7,5] and they agreed to enter a prospective study of back pain. A questionnaire was administered in 1990, designed to provide information about histories of back pain since the scan: if they had ever had back pain, its frequency, duration, whether they had to go to bed with back pain, whether they saw a doctor, were they admitted to a hospital, whether they had therapy, and whether it put them off work. After a pilot study and modification, the questionnaire was sent by mail, and a second questionnaire was sent to the nonresponders. The population was equally distributed about the mean. Subjects with canals above and below the mean (1.45 cm) at L5 were compared, and comparisons were also made among three equal groups with wide, average, and small canals. The numbers were tested using the chi-squared test and *t*-test.

From the Department of Orthopaedic Surgery, Aberdeen, Scotland.
Accepted for publication May 17, 1993.

Results

Before 1980, 669 nursing or mining recruits younger than 30 years had ultrasound measurements of the vertebral canal. In 1990, they were sent a questionnaire, which was returned by 450 subjects (67.3% of those scanned). There were 77 miners and 373 nurses; 72.5% said they had experienced previous back pain but the prevalence was not significantly different in those with canals above or below the mean (Table 1); nor were there significant differences when comparing those with wide, average, and narrow canals. 27.8% had taken time off work with back pain, and this was not different in those with wide or narrow canals (Table 2). However, 34.2% of the men with narrow canals had visited their doctors because of back pain compared with 23.1% with wide canals. Similarly, 29.1% of women with narrow canals has seen their doctors because of back pain compared with 22.3% of those with wide canals ($P = 0.068$) (Table 3); 31.1% of men with narrow canals and back pain had been in bed compared with 17.7% with wide canals. In women, the figures were 24.3% and 20.1%, respectively ($P = 0.131$) (Table 4); 21.1% of men with narrow canals had received medical treatment for back pain compared with 7.7% with wide canals, and the figures for women were 11.6% and 8.7% ($P = 0.107$) (Table 5). This trend was the same when comparing the populations with wide, average, and small canals, but did not reach significance.

Discussion

This epidemiologic study assessed the value of ultrasound measurement of the vertebral canal size, as a predictor of future back pain. It is limited by recording only the fifteen-degree sagittal diameter of the canal, but this is a diameter that is clinically relevant when other

Table 1. Answer to the Question "Have You Ever Had Pain in the Lower Back?" Which Was Accompanied with a Drawing of the Spine, Shaded Over the Lower Lumbar Region

	No %	Yes %	
Male			
Narrow (n = 38)	31.6	68.4	
Wide (n = 39)	30.8	69.2	
Total	31.2	68.8	
Female			
Narrow (n = 189)	29.1	70.9	
Wide (n = 184)	24.4	75.5	
Total	26.8	73.2	ns ($P = 0.343$)

pathology supervenes.[9] The study supports the view that a developmentally small vertebral canal in early adult life measured by ultrasound is not a significant risk factor for the subsequent experience of back pain and taking time off work for back pain is not necessary. However, for the more severe types of back pain, the canal size may be relevant. Those subjects with vertebral canals below the mean at L5 tended to visit their doctors for back pain more than those with wider canals, and this approached significance. Those with smaller canals were more likely to go to bed with back pain and seek medical treatment. Thus the vertebral canal size appears to be a small risk factor for more severe types of back pain.

Table 2. Answer to the Question "Have You Ever Been Off Work with Low-Back Pain?"

	No %	Yes %	
Male			
Narrow	65.8	34.2	
Wide	69.2	30.8	
Total	67.5	32.5	
Female			
Narrow	71.4	28.6	
Wide	75.0	25.0	
Total	73.2	26.8	ns ($P = 0.401$)

Table 3. Answer to the Question "Did You Ever Have to See Your Doctor About Low-Back Pain?"

	No %	Yes %	
Male			
Narrow	65.8	34.2	
Wide	76.9	23.1	
Total	71.4	28.6	
Female			
Narrow	70.9	29.1	
Wide	77.7	22.3	
Total	74.3	25.7	($P = 0.068$)

Table 4. Answer to the Question "Have You Ever Had to Go to Bed with Low-Back Pain?"

	No %	Yes %	
Male			
Narrow	68.9	31.1	
Wide	82.3	17.7	
Total	75.7	24.3	
Female			
Narrow	75.7	24.3	
Wide	79.9	20.1	
Total	77.7	22.3	($P = 0.131$)

Table 5. Answer to the Question "Have You Ever Had Physiotherapy or Manipulative Treatment for Low-Back Pain?"

	No %	Yes %	
Male			
Narrow	78.9	21.1	
Wide	92.3	7.7	
Total	85.7	14.3	
Female			
Narrow	88.4	11.6	
Wide	91.3	8.7	
Total	89.8	10.2	($P = 0.107$)

Back pain has many etiologies that are multifactorial, and although a small vertebral canal is probably important in some syndromes, it is not a sensitive predictor in the short term.

Acknowledgments

The authors thank Professor Kerridge for assistance with the statistical analysis, and Mrs. M. Russell for secretarial support.

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Address reprint requests to

R. W. Porter, MD
Department of Orthopaedic Surgery
Polwarth Building
Foresterhill
Aberdeen AB9 2ZD
Scotland

SPINE Volume 19, Number 8, pp 901–903

Spinal Stenosis and Health Status

20

R. W. Porter, MD, FRCS, FRCSE, and G. Oakshot, MB, ChB

Study Design. This study examined the hypothesis that a small adult vertebral canal is a marker of early impairment of growth, and that other sensitive systems may be impaired similarly, producing an adult relationship among a small canal, poor health, and academic ability.

Objectives. Comparisons of health and some aspects of academic ability were made between patients whose canals were above and those below the mean at L5.

Methods. Seventy-five patients were examined, 42 men and 33 women. They had comprehensive cardiovascular, respiratory, and digestive system health assessments. Their post-school qualifications were recorded, and they were assessed by the Mill Hill vocabulary test and the progressive matrices test.

Results. Twenty-two men and 21 women had canals above the mean, and 20 men and 12 women had canals below the mean. A significant difference did not exist in the age of those with wider and narrower canals. Cardiovascular symptoms and gastrointestinal symptoms were more common in men and women with narrower canals ($P = 0.04$ and 0.048), but there was no significant difference in respiratory symptoms. Those with wider canals had more post-school qualifications than those with smaller canals ($P = 0.04$), and in men, their performance in the Mill Hill vocabulary test and the progressive matrices test approached significance ($P = 0.08$ and 0.06).

Conclusions. The association between a smaller vertebral canal and impairment of health and certain intellectual abilities may result from an adverse environment that affects several growing systems early in life. If the small canal is a marker of a generalized developmental disturbance, it is, to some degree, preventable.
[Key words: vertebral canal, back pain, health, academic] **Spine 1994;19:901–903**

The vertebral canal reaches maturity early in life, the cross-sectional area and the sagittal diameter being of adult size by 4 years of age.[12] Because of no catch-up growth, impairment in early life might be expected to leave a small adult vertebral canal, masked by later appendicular growth.[14] Furthermore, other systems with similar early growth curve—the immune and neurologic systems—may be similarly impaired, with an adult relationship between a small vertebral canal and poor immune and neurologic function. Some patients with spinal stenosis are disadvantaged in health status[3,4,16] and academic ability.[16] The hypothesis that impaired vertebral canal growth is associated with impairment in other systems is examined in this study. Health and certain measures of intellectual ability and achievement are compared in patients with wider and narrower vertebral canals.

From the Department of Orthopaedic Surgery, Foresterhill, Aberdeen, Scotland.
Accepted for publication July 9, 1993.

Methods

The vertebral canal of each man and woman attending a hospital back pain clinic was scanned routinely by ultrasound to measure the 15° oblique sagittal diameter of the lumbar vertebral canal.[10,13] A consecutive group of 75 patients were invited to attend comprehensive health assessments, recording age, sex, height, weight, various anthropometric measures, a history of treatment for cardiovascular abnormality, blood pressure, a history of hospital treatment for respiratory disorder, pulmonary function measured by FEV and FVC, gastrointestinal disorders, dental examination, and audiometry.

Age at leaving school, participation in further education, and academic qualifications after leaving school were recorded. They completed the Mill Hill vocabulary scale,[17] which is a multiple choice of synonyms and a progressive matrices test, a multiple choice of perceptual reasoning that is uninfluenced by educational and cultural background. The score of these tests over the time to complete the tests produced a grade.[18]

Comparisons were made between two groups of patients identified as having "narrower" and "wider" canals, defined as patients whose canals were above and below the mean for the population (1.39 cm) at L5.

The numbers were tested using chi-squared tests, and the means were compared using *t* tests.

Results

Seventy-five patients were admitted to the study. There were 42 men (age 45.6 ± 9.89 years) and 33 women (age 42.3 ± 12.52 years) (Table 1). Twenty-two men and 21 women had wider canals above the mean at L5, and 20 men and 12 women had narrower canals below the mean. The mean for this population was 1.39 cm (range 1.08–1.72 cm), compared with the mean diameter for an asymptomatic population of 1.48 cm.[14] There was no significant difference among the ages of those with wider and narrower canals, (43.9 ± 10.7 years and 44.1 ± 11.7 years). There was no significant difference between the height and weight of those men and women above and below the mean for canal size ($P = 0.3$).

Cardiovascular symptoms were more frequent in the men and women with narrower canals than in those with larger canals ($P = 0.04$), and gastrointestinal

Table 1. Age, in Years, of Men and Women in the Study with Narrower (Below the Mean) and Wider (Above the Mean) Vertebral Canals

	Narrower (n = 32)	Wider (n = 43)
Female	42.42 ± 13.29 (n = 12)	41.70 ± 12.46 (n = 21)
Male	44.85 ± 9.12 (n = 20)	46.27 ± 10.72 (n = 22)

symptoms were also more common in those with narrower canals ($P = 0.048$). There was no significant difference in respiratory disorders ($P = 0.8$) (Table 2).

The mean age of leaving school was 15.1 years for those with narrower canals, and 15.3 years for those with wider canals, which was not significantly different ($P = 0.35$) (Table 3). Patients with wider canals had further education after leaving school than those with narrower canals, but this was not significant ($P = 0.16$). However more of those with wider canals had some post-school qualifications than those with narrower canals ($P = 0.04$). The Mill Hill vocabulary grade was higher, approaching significance, in those men with wider canals ($P = 0.08$) but not in women. The men with wider canals also had higher grades in the progressive matrices test ($P = 0.06$), but the women did not have higher grades.

We found no significant differences in blood pressure, dentition, or audiometry tests in those subjects above and below the mean canal size.

■ Discussion

Spinal pathology in the presence of a small canal can produce a variety of back pain syndromes.[7,8,19,20,21,22] Edwards and La Rocca[6] showed that 71% of their patients with back pain and degenerative change had canal diameters below the mean. Kornberg and Rechtine[11] demonstrated an inverse relationship between various canal parameters and the morbidity of disc protrusion. In a general practice study,[5] patients with back pain had significantly smaller lumbar canals than a randomized group of matched controls who did not have previous back pain. Thirty-nine percent of patients attending a back pain clinic had canals below the tenth percentile for the general population.[14] Thus a small vertebral canal has clinical significance for back pain.

Table 2. Cardiovascular Symptoms, Respiratory Diseases, and Gastrointestinal Disorders Compared in Those Patients with Narrower (Below the Mean) and Wider (Above the Mean) Vertebral Canals

	Narrower (n = 32)	Wider (n = 43)
Cardiovascular symptoms		
None	63%	81% Chi-squared test $P = 0.04$
Some	38%	17% sig at 5% level
Respiratory symptoms		
Not recorded	6%	10%
None	59%	60% Chi-squared test $P = 0.8$ n.s.
Some	34%	31%
Gastrointestinal symptoms		
Not recorded	0%	2%
None	47%	69% Chi-squared $P = 0.048$
Some	53%	29% sig at 5% level

Table 3. Age in Years When Leaving School, Further Education, Qualifications, Mill Hill Vocabulary Test Score, and Progressive Matrices Score, Compared in Those Patients with Narrower (Below the Mean) and Wider (Above the Mean) Vertebral Canals

	Narrower Canals	Wider Canals
Age when leaving school		
Male	15.00 ± 0.65	15.19 ± 1.17
Female	15.25 ± 1.24	15.45 ± 0.94
Total	15.09 ± 1.00	15.32 ± 1.06 *t*-test $P = 0.35$ ns
Further education		
Not recorded	3%	2%
None	47%	29% Chi-squared test $P = 0.1$ ns
Some	50%	69%
Qualification after school		
Now recorded	9%	5%
None	50%	29% Chi-squared test $P = 0.04$
Some	41%	67% (significant at 5% level)
Mill Hill vocabulary test		
Male	93.06 ± 11.66	98.48 ± 7.04 $P = 0.08$
Female	96.36 ± 7.58	94.80 ± 5.68 $P = 0.5$ ns
Total	94.36 ± 10.22	96.68 ± 6.60 $P = 0.2$
Progressive matrices test		
Male	115.00 ± 12.87	121.61 ± 7.43 $P = 0.06$
Female	111.91 ± 12.35	113.89 ± 17.84 $P = 0.7$ ns
Total	113.54 ± 12.54	117.65 ± 14.16 $P = 0.2778$

A relationship between a small vertebral canal and poor health and academic status may be indirect. It may not be the small canal, but back pain that is significant. For example, a subject involved in heavy manual work and prone to back pain may have poor health and low academic ability, not because of a small vertebral canal, but because of back pain and its socioeconomic consequences. It would not be the canal and its poor development that was related to deficiency in other systems, but back pain and its effects, which involved health and academic ability. To reduce this bias, we assessed the health and academic status of two groups of patients, some with wider and some with narrower canals, but both with back pain. All the patients had sufficient back pain to be referred to a specialist back pain clinic, although the severity and duration of back pain was not standardized.

Those with smaller canals were disadvantaged in their health and academic abilities. Those subjects with smaller vertebral canals had significantly more cardiovascular and gastrointestinal symptoms than those with wider canals. They also had fewer post-school qualifications. In addition, their poorer performance with the

vocabulary and the progressive matrices tests approached significance.

Subjects with few qualifications chose to do manual work and exposed themselves to environments that adversely affect their health. They might also have developed degenerative changes that reduced the size of the vertebral canal. However, it has not been shown that the vertebral canal reduces in size with age. Also no statistical difference existed in the ages of those with canals above or below the mean. In addition, the mean age of these subjects was only 44 years, early for degenerative changes to affect the canal. Some of the differences detected in health and academic status may be related to factors that affect the development of the vertebral canal.

The results of this study are compatible with the hypothesis that impaired early programming of canal growth leaves a small adult vertebral canal and that other sensitive developing systems can be affected similarly, producing an adult with a small canal and poor health and academic ability. The hypothesis has not been proven, but it is supported and deserves further study.

It is difficult to explain the association between the size of the vertebral canal and these variables unless the relationship begins in early life. The vertebral canal reaches maturity in infancy[12] and factors that affect its early growth probably affect other sensitive growing systems that share the same window of opportunity. The growth curve of the thymus, the central nervous system,[9] the cardiovascular system,[1] and the liver[2] are similar to that of the neuro-osseous system, and they can be affected similarly by an adverse early environment. The small canal may therefore be a marker of a generalized developmental disturbance in early life. If the hypothesis is correct, some types of back pain could be prevented by health care in early life, and there is more to spinal stenosis than a small canal.

Acknowledgments

The authors thank Professor Kerridge for statistical support and Mrs. M. Russell for secretarial help.

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Address reprint requests to

R. W. Porter, MD
Department of Orthopaedic Surgery
Polwarth Building
Foresterhill
Aberdeen AB9 2ZD, Scotland

SPINE Volume 19, Number 13, pp 1508–1511

Changes of the Lumbar Spinal Canal Proximal to Spina Bifida Occulta

21

An Archaeologic Study With Clinical Significance

Tibor Papp, MB, ChB, and R. W. Porter, MD, FRCS, FRCSE

Study Design. This archaeologic study, based on four populations, examines the incidence of spina bifida occulta in the lumbar spine and the size of the vertebral canal proximal to the lesion.

Objectives. To ascertain any significant change in the dimensions of the lumbar spinal canal of skeletons with spina bifida occulta. The incidence of the lesion also was compared in the separate genetic groups.

Methods. Central canals of 1760 lumbar vertebrae were examined. Silhouette, unmagnified pictures of the vertebral canals were measured by computerized image analysis.

Results. The mid-sagittal diameter at L4 and L5 and the cross-sectional area at L5 were found to be significantly larger proximal to the lesion compared with the unaffected spines. The overall incidence was 18%.

Conclusions. The capacity of the lumbar canal is greater proximal to spina bifida occulta. Therefore, delayed closure of the neural arch at a single segment has morphologic significance to the more proximal spine.
[Key words: canal size, spina bifida occulta] **Spine 1994;19:1508–1511**

Spina bifida occulta is a defect in the neural arch with intact skin, and in most cases, with no obvious neurologic abnormality. It is generally considered to be an innocuous vertebral anomaly with little clinical significance. We conducted an archaeologic study that examined the lumbar vertebral canal proximal to vertebrae with spina bifida and compared these spines with those of unaffected skeletons.

Materials and Methods

The lumbar vertebral canal was examined in four archaeologic populations:

1. A Romano-British population of the 4th century from Poundbury in Dorset.
2. An Anglo-Saxon population of the 6th to 7th centuries from Guildown, England.
3. An Anglo-Saxon population of the 10th century from Raunds, England.
4. An immigrant Huguenot French population of the 17th to 19th centuries from Spitalfield in London.

One-hundred-four lumbar spines were examined from the Poundbury population, 27 from Guildown, 77 from Raunds, and 144 from Spitalfield. The vertebral canal was photographed using a specially designed photographic box (Figure 1) to provide an unmagnified silhouette picture of the canal. The specimen was positioned to obtain the most proximal aspect of the central canal on a piece of photosensitive paper because this is the level where it is narrowest. Thus, in the presence of space-reducing pathology, this aspect has the greatest clinical importance.

The mid-sagittal diameter, interpedicular diameter, and the cross-sectional area were measured with great accuracy from these photographs by computerized image analysis. The vertebral canals proximal to the vertebrae with spina bifida occulta were measured and compared with parameters from unaffected skeletons. Dimensions of vertebral canals with spina bifida were not examined because the posterior endpoint of the mid-sagittal diameter could not be determined.

Student's *t* test and the Mann-Whitney rank sum test were performed for statistical analysis.

Results

Spina bifida occulta had occurred in 14% of the Poundbury, 37% of the Guildown, 12% of the Raunds, and

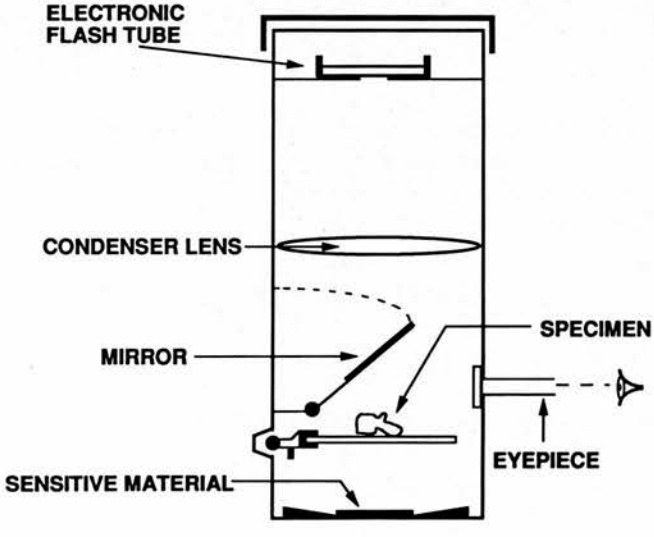

Figure 1. The photographic box that can produce the unmagnified image of the vertebrae.

From the Department of Orthopaedic Surgery, University of Aberdeen, Aberdeen, Scotland.
Accepted for publication: February 10, 1994.
Financial disclosure/device status categories: 1/7.

Table 1. Canal Dimensions of the Four Populations in Spines With and Without Spina Bifida

	Raunds			Guildown			Poundbury			Spitalfield		
With Spina Bifida												
Mid-sagittal Diameter (mm)												
	Mean	n	SD	Mean	n	SD	Mean	n	SD	Mean	n	SD
L1	15.7	5	1.34	15.1	8	1.53	15.3	12	1.72	15.3	10	1.69
L2	14.9	8	0.96	13.8	9	1.67	14.5	14	1.72	15.1	10	2.27
L3	14.8	8	1.28	14.1	9	1.82	14.0	14	1.74	13.8	10	1.97
L4	15.3	8	1.45	15.3	9	2.96	14.5	15	1.96	14.8	10	2.98
L5	16.5	9	2.51	15.8	10	1.93	16.3	15	2.34	16.4	10	2.15
Interpedicular Diameter (mm)												
L1	22.0	5	1.19	23.1	8	2.59	22.3	11	1.31	21.5	10	2.35
L2	22.4	8	1.47	22.7	9	1.94	22.0	14	1.53	21.2	10	3.22
L3	21.9	8	1.53	22.7	10	1.68	22.5	14	1.32	20.9	10	2.93
L4	22.3	9	1.64	23.6	10	2.12	22.9	15	1.59	21.3	10	3.37
L5	25.4	9	2.18	26.4	10	2.01	25.6	15	2.35	23.8	10	2.96
Area (mm^2)												
L1	228	5	17.0	263	9	29.2	237	11	35.2	227	10	39.01
L2	221	8	31.0	226	9	30.7	224	13	32.3	229	10	62.09
L3	216	8	16.0	241	9	30.6	236	14	86.1	201	10	45.34
L4	207	9	29.0	270	9	40.1	219	14	37.6	215	10	51.74
L5	238	8	50.0	270	9	40.1	256	15	59.8	262	10	56.76
Without Spina Bifida												
Mid-sagittal Diameter (mm)												
L1	15.1	55	1.35	14.5	14	1.80	16.0	92	1.30	14.9	101	1.57
L2	14.5	64	1.94	13.7	15	1.14	14.9	86	1.55	14.4	101	1.67
L3	13.8	68	2.06	14.0	17	1.60	14.1	91	1.56	13.9	101	1.59
L4	13.4	58	2.15	13.4	14	1.59	13.9	91	1.79	14.1	101	1.99
L5	14.2	45	2.45	14.4	16	2.43	15.1	86	2.13	15.2	101	2.82
Interpedicular Diameter (mm)												
L1	22.2	54	1.69	23.1	4	1.95	21.2	81	3.39	22.1	101	2.51
L2	22.4	62	1.74	23.2	15	1.25	22.1	88	1.78	21.4	101	2.46
L3	22.6	66	1.74	22.9	17	1.13	22.3	93	1.75	21.2	101	2.33
L4	22.7	58	1.98	23.3	14	1.54	22.8	95	2.17	21.1	101	2.94
L5	25.7	45	3.13	24.8	16	1.79	25.7	93	2.85	23.9	101	4.34
Area (mm^2)												
L1	221	57	30.0	247	14	32.7	238	87	35.7	208	101	31.90
L2	214	66	36.0	228	15	20.5	227	87	34.5	203	101	39.52
L3	199	70	38.0	241	17	38.9	214	94	36.5	193	101	37.69
L4	186	58	45.0	239	14	40.7	207	92	39.5	190	101	44.22
L5	207	45	58.0	239	16	61.5	232	37	53.1	215	101	58.75

11% of the Spitalfield populations, respectively. The Spitalfield collection also contained 42 well-preserved children's spines. The incidence among infants (1–5 years old) was found to be 10%; in juveniles (5–16 years old), the incidence was 44%. Of the vertebrae with spina bifida, 93% from Poundbury, 80% from Guildown, 89% from Raunds, and 100% from Spitalfield were at S1; the remainder were at L5.

The mid-sagittal diameter, the interpedicular diameter, and the cross-sectional area of the vertebral canal at each lumbar level in spines with and without spina bifida are recorded in Table 1.

In the Poundbury population, no significant difference was found in the size of canals between spines with the lesion and those without it.

In the Guildown series, the area and the interpedicular diameter was greater at L5 ($P < 0.05$) in the affected spines.

In the Raunds population, the mid-sagittal diameter was significantly wider at L4 and L5 in skeletons with spina bifida than in those without ($P < 0.01$ and $P < 0.05$, respectively).

In the Spitalfield population, in spines with spina bifida, the mid-sagittal diameter was larger at L5 ($P < 0.05$) and the cross-sectional area was greater at all levels, with increasing strength of significance from the L1 level downward (L1–L3, $P < 0.05$; L4, $P = 0.04$; L5, $P < 0.04$).

No significant difference was detected in dimensions at other lumbar levels.

Table 2. Combined Data of Canal Measurements

	With Spina Bifida			Without Spina Bifida		
Mid-sagittal Diameter (mm)	Mean	n	SEM	Mean	n	SEM
L1	15.3	35	0.25	15.1	262	0.63
L2	14.8	41	0.57	14.4	266	0.50
L3	14.2	41	0.43	13.9	277	0.13
L4	15.0	42	0.39	13.7	264	0.35 $P = 0.003$
L5	16.2	44	0.31	14.7	248	0.25 $P = 0.002$
Interpedicular Diameter (mm)						
L1	22.2	34	0.67	22.1	250	0.77
L2	22.1	41	0.65	22.2	266	0.74
L3	22.0	42	0.81	22.2	277	0.74
L4	22.5	44	0.97	22.5	268	0.90
L5	25.3	44	1.09	25.0	255	0.86
Area (mm^2)						
L1	239	35	16.80	228	259	17.40
L2	225	40	3.37	221	269	7.80
L3	223	40	18.48	214	282	19.34
L4	228	42	28.60	205	265	24.12
L5	256	42	13.60	227	199	13.88 $P = 0.023$

Pooled data from the four populations showed significantly greater mid-sagittal diameters in the affected spines at the fourth and the fifth lumbar vertebrae ($P = 0.003$ and $P = 0.002$, respectively) and a larger cross-sectional area at the fifth level ($P = 0.023$). Table 2 shows the combined data of canal parameters.

■ Discussion

This study shows that the vertebral canal proximal to spina bifida occulta tends to be larger than in the unaffected spines. This was previously suggested by Sand,[9] who examined a Norwegian archaeologic population. His results have been confirmed in the present, larger study. Therefore, spina bifida occulta is not a pathology of a single segment, but it bears morphologic significance to the more proximal vertebrae.

Sarpyener[10] found that in some cases of spina bifida, the laminae that normally meet posteriorly curved inward into the vertebral canal, causing its narrowing. However, in our large series, such a condition could not be found. He also described ring-like strictures of the lumbar canal cranially to spina bifida occulta. This was not observed in our populations.

Figure 2. Silhouette pictures of lumbar vertebrae. (Left) L5 vertebra from above spina bifida. Note the differences in the mid-sagittal diameters and cross-sectional areas.

Clinical studies report an incidence of spina bifida occulta ranging from 5% to 10%.[1,2,5,11] The two Anglo-Saxon populations in the present study had an incidence of 12% and 37%, respectively, demonstrating a large variation within similar genetic groups. In the Spitalfield population, the 44% incidence of juvenile spina bifida decreased to 11% in adulthood. This phenomenon may result from the delayed ossification of the cartilaginous connection that bridges the gap between the two ends of the laminae that in some children form a spinous process until adulthood. Therefore, the presence of spina bifida on a radiograph of a child does not predict the likelihood of persistence of the lesion in adulthood.

Folic acid deficiency in pregnancy can result in neural tube defects in the fetus,[6] but the relative importance of environmental and genetic factors in the causation of spina bifida occulta is unknown. Fredrickson and colleagues found that the overall incidence of spina bifida occulta in patients with pars interarticularis defect was 92% in childhood and persisted into adulthood in 70% of the patients.[4] We suggest that for the laminae to close and ossify, constant forces probably are needed, with fixed vectors in the laminae originating from the direction of the ossification centers. With the pars interarticularis defect, the base of the semi-arch is not fixed, resulting in a floating lamina with variable force vectors that do not induce ossification. As a consequence, a gap will remain in the neural arch.

Variations in the size of the canal are clinically significant. A small canal can compress the dura and the cauda equina or the nerve root in the lateral recess. Low

back pain syndromes are multi-factorial, and in those syndromes associated with root pain, space within the vertebral canal is a significant factor[3,7,8] (Figure 2). Reduced dimensions of the canal represent a disadvantage, especially in the presence of pathologies such as disc protrusion, enlargement of the ligamentum flavum, bony outgrowth encroaching into the vertebral canal, and segmental displacement. Patients with spina bifida occulta may have a low incidence of nerve root compression and cauda equina syndromes.

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Address reprint requests to

Tibor Papp, MB, ChB
Department of Orthopaedic Surgery
University of Aberdeen
Polwarth Building
Foresterhill
Aberdeen AB9 2ZD
Scotland

SPINE Volume 19, Number 24, pp 2770–2773

The Growth of the Lumbar Vertebral Canal

22

T. Papp, MBChB, R. W. Porter, MD, FRCS, FRCSE, and R. M. Aspden, PhD

Study Design. This study examines the growth and development of the lumbar spinal canal with emphasis on early life.

Objective. Changes in dimensions of the canal were investigated throughout life.

Summary of Background Data. Seven hundred and fifteen lumbar vertebrae were examined from the Spitalfield Collection of Skeletons at the Natural History Museum, London.

Methods. Unmagnified silhouette pictures were taken of the canals with a specially designed photographic box. Computerized image analysis provided the accurate measurements.

Results. Regarding the midsagittal diameter and the cross-sectional area, the cranial four lumbar vertebrae were already fully matured in infants. At L5 there was significant increase up to 4 years of age when the midsagittal diameter was even larger than in the adult. The interpedicular diameter significantly increased at L1 until 10 years of age, at the other levels until adulthood, as did the perimeter at L4 and L5 until 14 years of age. The shape of the canal was assessed by measuring the circularity, the 'trefoilness' and the situation of the centroid. The first measurement significantly decreased with age, the trefoilness increased until adulthood, and the centroid of the canal approached the vertebral body. In spines with spina bifida occulta, the lumbar canal was significantly larger proximal to the lesion than in the unaffected spines.

Conclusion. The lumbar spinal canal exhausts its growth potential by infancy as regards the midsagittal diameter and the cross-sectional area. Thus, in the case of delayed development, it is not capable of catch-up growth. [Key words: lumbar spinal canal, growth, shape, spina bifida] **Spine 1994;19:2770–2773**

Previous studies[8,11] from other archeologic populations have suggested that the lumbar vertebral canal reaches maturity in cross-section and midsagittal diameter at approximately 4 years of age. The midsagittal diameter at this age is larger than that of the adult's while the interpedicular diameter is still growing. The exact chronologic age of growth was however to be determined. This study was designed to establish the growth curve of the lumbar central spinal canal, which, due to the lack of spines to examine from very early period of life, has not been possible. In the present population, investigated in this study, there were many spines of children, with a unique opportunity to complete the assessment on the growth and development of the lumbar spinal canal.

From the Department of Orthopaedic Surgery, University of Aberdeen, Aberdeen, Scotland.
Device status category: 1.
Accepted for publication December 22, 1993.

Materials and Methods

One hundred and eighty five lumbar spines were examined from the Spitalfield Collection of skeletons at the Natural History Museum, London. This population was unique in that their exact age at death had been documented. The skeletons are those of French Hugenots who were born between 1646 and 1852 and died between 1729 and 1852. Of these 41 were infant (16 months to 5 years of age), 43 were juvenile (5 to 16 years of age), and 101 were adult (16 to 70 years of age). Two hundred and ten vertebrae were missing or damaged, leaving 715 lumbar vertebrae available for study. As the parts of the neural arch unite between 5–12 months and the arch ossifies to the vertebral body around the age of 12–16 months starting from the proximal lumbar levels,[6] the first already complete lumbar vertebrae were available at one year of age. The silhouette pictures were taken of the upper most aspect of the lumbar vertebral canals, since at this level the central spinal canal is the narrowest, therefore in case of any reduction in size it has the greatest clinical importance. The imaging was carried out with the help of a specially designed photographic box[9,11] which produced the unmagnified image of the canal on a piece of photosensitive paper. The measurements of the midsagittal diameter, the interpedicular diameter, the cross-sectional area, the perimeter, the circularity, the trefoilness and the centroid were established by video combined computerized image analysis. Following the calibration of the computer against a millimeter grid, the picture of the vertebra was positioned on a diascope underneath the lens of a video camera which transmitted the image of the canal to the computer. After designation of the chosen parameters, the exact values were calculated by the computer. This method provided the greatest accuracy in measuring the selected dimensions to the nearest 0.01 mm. Linear regression, Mann-Whitney rank–sum test, and t-test were performed for statistical analysis.

Results

Looking at the cross-sectional area at the cranial four lumbar levels no significant change was found throughout life; these vertebrae were already of adult size at 1 year of age. At the fifth level, the area increased ($P = 0.007$) until 6 years of age (Figure 1), with an average of 15.20 mm^2 per year rate between 1 and 6 years of age. Subsequently significant change was not detected.

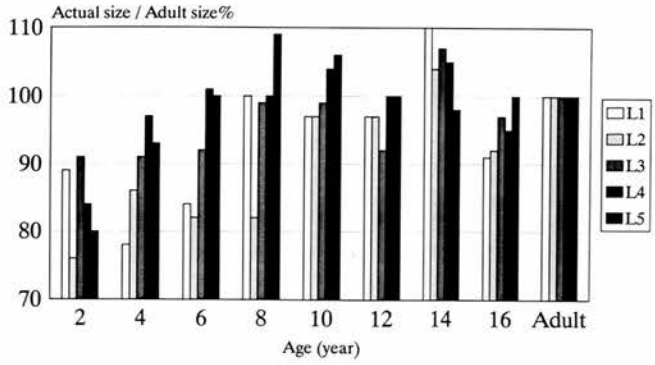

Figure 1. L1–L5 mean cross-sectional area. There was a significant increase only at L5 until 6 years of age ($P = 0.007$). At the other levels, there was no significant change throughout life.

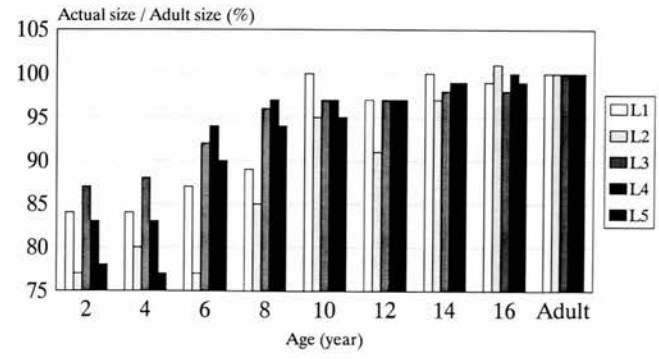

Figure 3. L1–L5 mean interpedicular diameter. At L1, it is of adult size by 10 years of age, whereas the rest of the vertebrae grow until adulthood ($P < 0.001$).

The smallest area was of an adult L5 vertebra with 116.03 mm^2.

The midsagittal diameter, which has the primary role in developmental stenosis[14] showed significant increase ($P = 0.021$) with 1.24 mm per year at L5 level until the age of 4 and was even larger ($P = 0.011$) in the 3–4-year age group than in the adult. It reduced to the adult size by the age of 6 (Figure 2). The reduction in size that takes place during childhood is due to the changing shape of the canal. The anterior border of the canal at the upper most, narrowest aspect, which contributes to the shape, is concave in infancy and becomes convex in the adult. The rest of the lumbar vertebral canals from 1 until 70 years of age did not exhibit any significant change in the midsagittal diameter with age, as Larsen and Smith found measuring radiographs of children from 3 until 14 years of age.[5] Although in advanced age the osteophytic outgrowths can be large and prominent on the anterolateral surface of many of the vertebral bodies, these osteophytes are rarely found on the posterior surface of any of the vertebral bodies which borders on the spinal canal. In those rare instances when bony outgrowths were observed in this location, these were tiny projections that did not significantly affect the diameter of the canal. None of the vertebrae was less than 11.05 mm in midsagittal diameter, which was of an adult L5 vertebra.

The interpedicular diameter of L1 vertebra was matured by 10 years of age, whereas the rest of the lumbar levels grew until adulthood ($P < 0.001$, Figure 3) by an average of 0.50 mm each year, as the transverse diameter of the vertebral bodies increases[13] distracting the pedicles. The lower limit was detected at 15.36 mm in a 3-year-old L1 vertebra.

The perimeter displayed increase at L4 ($P = 0.028$) and L5 ($P = 0.041$) until the age of 14; the growth rate was 1.34 mm per year (Figure 4). The minimum value was 45.33 mm measured in an adult L2 vertebra. Table 1 shows the mean measurements of the canal parameters. In order for the interpedicular diameter and the perimeter to increase, after the midsagittal diameter and the cross-sectional area have finished their growth, the canal must change shape.

The change in shape of the canal was assessed by the circularity, the 'trefoilness,' and the position of the centroid. The circularity shows how far a shape deviates from a circle. The value of circularity is calculated from the formula: $4 \times \text{pi} \times \text{area/perimeter}^2$. This parameter

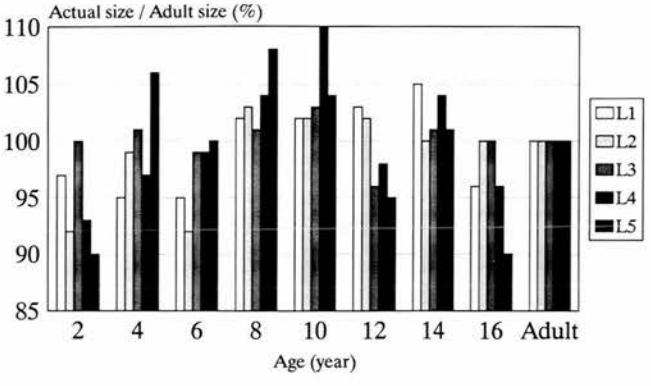

Figure 2. L1–L5 mean midsagittal diameter. At L5 there is a significant increase until 4 years of age ($P = 0.021$) when it has already reached adult size; at this age, the diameter is significantly larger than that of the adults' ($P = 0.011$). There was no significant change at the other levels from 1 until 70 years of age.

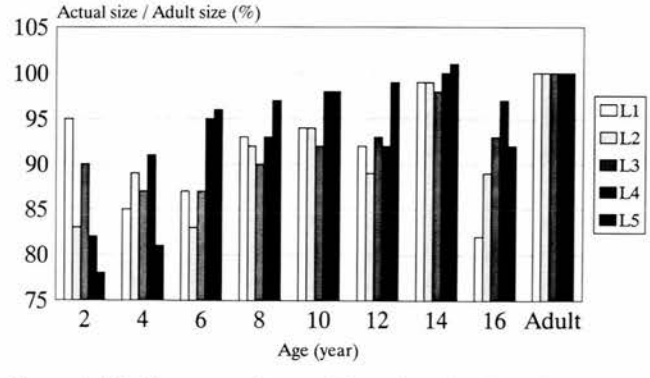

Figure 4. L1–L5 mean perimeter. There is a significant increase at L4 ($P = 0.028$) and L5 ($P = 0.041$) until 14 years of age while the other canals do not exhibit any significant change.

Table 1. Mean Canal Parameters (Midsagittal Diameter, Interpedicular Diameter, Perimeter, Cross-Sectional Area)

Age (yr)	mm	L1	L2	L3	L4	L5
	Midsag.	14.87	13.47	13.94	13.28	13.87
1-2	Interped.	18.16	17.25	17.72	17.66	19.43
	Perim.	58.82	51.76	54.76	50.90	54.68
	Area/mm²/	201.33	169.67	182.85	175.05	185.61
	Midsag.	14.67	14.46	14.14	14.99	16.50
3-4	Interped.	17.60	18.43	18.22	17.68	18.74
	Perim.	52.42	55.48	52.91	56.88	57.11
	Area/mm²/	177.28	190.55	184.45	197.70	214.26
	Midsag.	14.62	13.49	13.83	14.10	15.44
5-6	Interped.	18.35	17.26	18.92	20.19	22.77
	Perim.	53.76	52.80	52.74	58.98	67.43
	Area/mm²/	190.82	181.76	185.01	205.97	230.45
	Midsag.	15.74	15.05	14.13	14.43	16.18
7-8	Interped.	19.28	20.01	20.05	20.99	23.82
	Perim.	57.96	57.53	55.09	58.01	68.41
	Area/mm²/	239.35	218.94	199.76	204.49	251.23
	Midsag.	15.80	14.96	14.35	15.72	16.04
9-10	Interped.	21.14	21.37	20.90	22.29	24.53
	Perim.	58.01	58.41	55.99	61.99	68.72
	Area/mm²/	220.45	215.84	199.76	233.53	250.30
	Midsag.	15.90	14.94	13.44	13.96	14.99
11-12	Interped.	20.47	19.99	19.56	20.25	23.94
	Perim.	56.98	55.47	56.39	57.66	69.63
	Area/mm²/	219.74	215.73	185.32	203.75	225.25
	Midsag.	16.26	14.66	14.07	14.79	15.96
13-14	Interped.	22.55	21.59	21.93	22.54	25.65
	Perim.	61.54	62.58	62.58	62.66	71.02
	Area/mm²/	252.24	230.99	216.55	214.94	226.13
	Midsag.	14.77	14.08	13.87	13.68	14.45
15-16	Interped	20.16	20.22	20.28	20.38	23.85
	Perim.	54.04	55.69	56.54	60.50	64.93
	Area/mm²/	207.09	203.66	194.98	192.33	227.95
	Midsag.	15.40	14.62	13.96	14.23	15.31
Adult	Interped.	22.35	22.33	22.12	21.97	24.59
	Perim.	61.88	62.20	60.75	62.24	70.25
	Area/mm²/	227.99	222.26	201.87	203.77	231.07

displayed a significant decrease during the life span ($P < 0.001$). The dome-shaped lumbar canal, which features infancy and childhood, becomes triangular in adulthood. In some of the adult lumbar vertebrae, the shape of the canal begins to resemble a trefoil.

The trefoil shape manifested itself only in the fifth lumbar vertebrae with one exception at L4. The 'trefoilness' is a ratio between the interpedicular diameter and a transverse measurement taken at one third of the distance between the mid-point of the interpedicular diameter and the apex of the neural arch[9,11]. The shape was compared with an equilateral triangle, in which this value is 66.67%. Therefore the canal was considered trefoil if the ratio was less than this value. The ratio of 'trefoilness' exhibited a significant decrease ($P < 0.001$) until adulthood only. The trefoil-shaped canal was not detected before adulthood; the first one appeared at the age of 16. In trefoil canals, the midsagittal diameter was smaller ($P = 0.029$), in accordance with Eisenstein's findings[2] and the interpedicular diameter was larger ($P = 0.049$) than in the normal-shaped canals, although the t-test proved the significance to be weak as regards the interpedicular diameter.

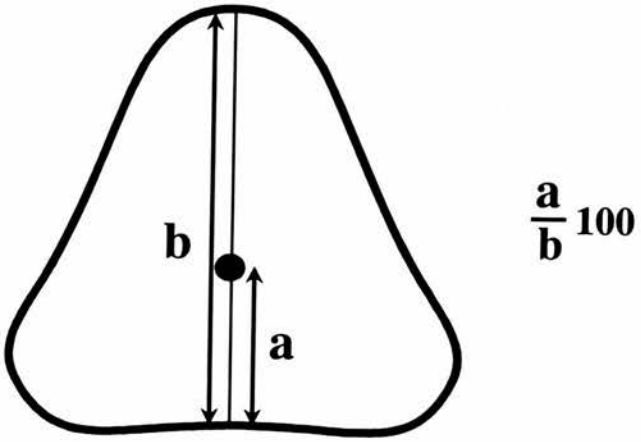

Figure 5. Measurement of the site of the centroid.

The centroid is a place on which a certain suspended object will be balanced. The site of the centroid in a symmetrical canal takes place on the sagittal plane and is expressed as the ratio between the midsagittal diameter and the distance between the anterior end-point of the midsagittal diameter and the centroid (Figure 5). This ratio also revealed a significant decrease ($P < 0.001$) until adulthood, in other words the centroid got closer to the posterior surface of the vertebral body. The posterolateral sides of the canal draw toward the sagittal plane, many times protruding inward resulting its concavity. These findings confirms the alterations of the shape of the lumbar central canal. Table 2 indicates the mean measurements of the shape.

Concerning the relevance of spina bifida,[12] the cross-sectional area of the lumbar canal was significantly large ($P = 0.041$) in the vertebrae above the lesion than in those spines without such defect.

■ Discussion

The results indicate that there is a differential pattern of growth with the cranial lumbar spine maturing first, as it was previously suggested.[11] As far as the cross-sectional area and the midsagittal diameter is concerned, the proximal four lumbar vertebrae have already fully matured by 1 year of age. These findings imply that what happens with the area and midsagittal diameter of the lumbar spinal canal is greatly determined before or

Table 2. Mean Measurements of the Canal Shape

Age (yr)	Circularity	Trefoilness(%)	Centroid(%)
1-10	0.75	92.23	57.56
11-20	0.65	82.55	55.37
21-30	0.63	72.88	56.31
31-40	0.62	72.01	56.62
41-50	0.63	71.96	52.50
51-60	0.58	70.21	51.90
61-70	0.54	67.98	52.11

very shortly after birth since by this time the canal has already exhausted its growing potentials. Though genetic factors probably influence the size of the canal[7], there can be important environmental factors that affect the growth of the canal very early in life[1]. Pre- and perinatal illnesses (maternal illness, toxins, infections, smoking, drugs, placental-insufficiency) may be significant to stunt the canal growth permanently. Subsequently an optimal environment may permit the long bones and the trunk to catch up with the suspended growth[15] resulting in an individual with tall stature however the lumbar spinal canal remains small as a consequence of early impaired development. Therefore this statement has important implications for the prevention of lumbar spinal stenosis. If we were able to perform preventative measurements in the established 'window' of growth and in case of delayed development ameliorate the relevant environmental factors, it would be possible to reduce the number of patients with narrow canal.

A small canal, which is trefoil in shape, is particularly troublesome if that canal is compromised by disc protrusion, degenerative change, or excessive motion. The cause of trefoilness is probably present very early in life although it is not manifest until adulthood. Considering that the midsagittal diameter matures early and does not significantly modify in adulthood, one may suggest that these individuals were born with anteroposteriorly flatter canals. Because the midsagittal diameter has no growth potential over the age of 4 and the interpedicular diameter is still growing until adulthood, the canal will probably become trefoil in shape.[10] Epstein and colleagues have found that the midsagittal diameter has a good correlation with the size of the root canal, hence vertebrae with narrow central canal have smaller foramina in diameter.[3] The radiologic finding of the trefoil shape is very often related to low back pain. Thereupon the mentioned individuals are prone to develop spinal stenosis symptoms as an adult in the presence of other space-reducing pathology.

Most of the clinicians and radiologists regard spina bifida occulta as an additional finding but do not attach to it any pathologic significance. As a conclusion of this and former studies,[12] defective closure of the neural arch is not a pathology of a single segment but has morphologic significance for the more proximal spine as well as resulting in a wider central canal. Little is known about the etiology of spina bifida although it has already been recognized that folic acid deficiency can result in neural tube defects.[4] There are likely opposing environmental factors of unknown nature that can cause either delayed closure with a more capacious canal or premature completion of the neural arch with a constitutionally small canal.

Acknowledgment

The authors thank Theya Molleson for giving us access to the Spitalfield Collection at the Natural History Museum, London.

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Address reprint requests to

Tibor Papp, MBChB
University of Aberdeen
Department of Orthopaedic Surgery
Polwarth Building
Foresterhill
Aberdeen AB9 2ZD, Scotland

SPINE Volume 20, Number 6, pp 660–664

Laser Doppler Study of Porcine Cauda Equina Blood Flow

The Effect of Electrical Stimulation of the Rootlets During Single and Double Site, Low Pressure Compression of the Cauda Equina

23

Andrew R. Baker, FRCS, Tanya A. Collins, BScHons, Richard W. Porter, MD, FRCS, FRCSE, and Cecil Kidd, PhD, FRSA, FIBiol

Study Design. This study involved a model of spinal claudication to assess the effect of single and double site compressions on blood flow in porcine cauda equina. Real-time monitoring of blood flow was achieved by using laser Doppler probes.

Objectives. To ascertain the difference between single and double site compressions on blood flow and nerve conduction in the cauda equina. Also, to provide possible explanations for the mechanisms underlying the pathophysiology of neurogenic claudication.

Summary of Background Data. The model used was based on that of K. Olmarker. Double as compared with single site compression of cauda equina showed impaired nerve impulse propagation and decreased blood flow.

Method. Pigs weighing 22–27 kg were anesthetized and the cauda equina was exposed by dorsal laminectomy of the sacral and first three coccygeal vertebrae. Polyethylene balloons were placed over the rootlets at the first and third coccygeal segments, and stimulating electrodes were positioned on the rootlets proximally to evoke motor activity. Electromyographic activity was monitored from tail musculature. Laser Doppler probes monitored blood flow in the cauda equina both between and distal to the two compression sites.

Results. Single site, low level compression did not affect blood flow whereas double site compression decreased it profoundly. Proximal stimulation caused a marked increase in blood flow, which was not sustained during prolonged compression. Electromyographic activity diminished concomitantly during this compression.

Conclusions. Low pressure, double site compression significantly reduces cauda equina blood flow and prevents the sustained increase in blood flow required to maintain normal neurologic rootlet function. Local blood flow failure may therefore be responsible for claudication symptoms. [Key words: cauda equina, intermittent claudication, laminectomy, laser Doppler flowmetry, spinal cord compression, spinal stenosis]
Spine 1995;20:660–664

From the Departments of Biomedical Sciences and Orthopaedic Surgery, University of Aberdeen, Aberdeen, Scotland.
Supported by the Wishbone Trust, University of Aberdeen.
Recipient of the 1994 Volvo Award in Basic Science.
Presented at the 21st Annual Meeting of the International Society for the Study of the Lumbar Spine, June 21–25, 1994, Seattle, Washington.
Accepted for publication May 5, 1994.
Device status category: 1.

The association between structural narrowing of the vertebral canal and the symptoms of neurogenic claudication were first recognized by Verbiest.[8] However, despite significant interest in the mechanisms by which the symptoms of neurogenic claudication are produced, its pathophysiology is still largely ill-understood. Experimental and clinical studies suggest that a low pressure compression of the cauda equina at two sites may be important,[4,6] but it is difficult to understand why leg symptoms are generally evoked by exercise but are not present at rest, and how they can be relieved by rest. If the symptoms are caused solely by venous congestion of the cauda equina then it is important to determine the effects of exercising muscle on venous congestion.

In this study we attempted to understand more about the factors affecting blood flow in the cauda equina during exercise. An animal model was used to assess cauda equina blood flow during simulated exercise while a low pressure "block" is applied at two sites on the cauda equina.

Materials and Methods

Twenty-five pigs weighing 22–27 kg were studied. Anesthesia was induced with 30 mg/kg intravenous pentobarbitone sodium (Sagatal, Rhône Mérieux Ltd., Harlow, Essex, UK) and maintained by ventilating with halothane. The jugular vein was cannulated for infusion of more anesthetic agents and fluids; the carotid artery was cannulated for measurement of arterial blood pressure and the electrocardiogram was recorded. A temperature probe was inserted through the mouth and into the stomach; electrocardiographic electrodes were placed in standard positions. Continuous assessment of arterial blood pressure, heart rate, temperature, electrocardio-

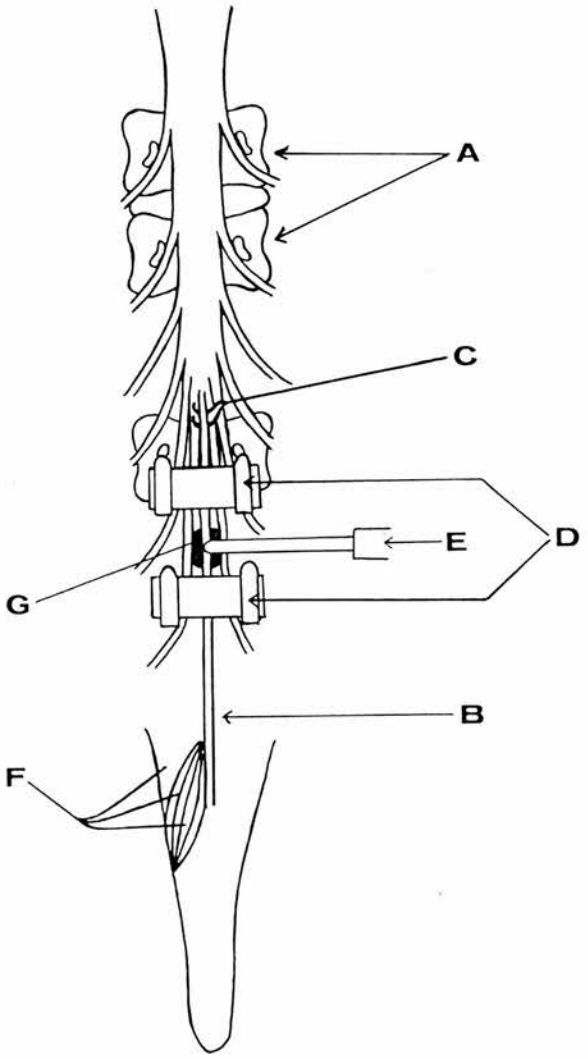

Figure 1. Schematic diagram of the model used to induce two site compression of the cauda equina with varying levels of pressure. **(A)** Vertebrae, **(B)** cauda equina, **(C)** stimulating electrode, **(D)** pins holding Plexiglas plates at coccygeal vertebrae 1 and 3, **(E)** laser Doppler probe, **(F)** electromyographic electrodes on tail muscles, **(G)** black vinyl discs.

gram, and expired carbon dioxide concentration was done and all variables were maintained within normal limits.

The pigs were placed in a prone position on a warmed operating table and a mid-line incision was made from just above the base of the tail to a point over the lowest segment of the lumbar spine. Dorsal laminectomy of the lowest sacral and upper three coccygeal vertebrae was performed to expose the cauda equina.[4] Welded polyethylene balloons were placed over the nerve roots and held in place by Plexiglas plates and stainless steel pins inserted into the pedicles of coccygeal vertebrae 1 and 3. The balloons were used to provide different grades of compression of the rootlets by varying inflation pressure with air: the pressure within the balloons was maintained using an adjustable screw clamp on a water manometer.

A laser Doppler probe (MBF 3, Moor Instruments, Millwey, Axminster, Devon, UK) was applied to the cauda equina between the balloons at coccygeal segment 2. Stability of the probe was critical. It was held in a stable position by a three-dimensional micromanipulator supported on the operating table by solid brass rods. A thin black vinyl disc, impermeable to laser light, was placed beneath the roots under each probe to ensure that measurements were limited to root blood flow alone.

The laser Doppler technique uses the change of frequency of near infrared light (Doppler effect) to measure the concentration and speed of red blood cells. From these two values flux can be calculated. Flux values refer to relative changes in the red cell movement, but not to absolute flow. Throughout, reference is made to changes in flux as representing changes in blood flow.

A bipolar electrical stimulating electrode was placed loosely under the nerve roots proximal to the first compression balloon to evoke contraction of tail muscles, which was assessed by electromyographic (EMG) responses. The electrode was held in position by a snake clamp (Polar Hydraulics, Verdict Gauge, Ltd., Crayford, Kent, UK). Electromyographic electrodes were positioned in the ipsilateral tail musculature (Figure 1). To prevent movement artifacts, the entire field of study was stabilized by bilateral supports that elevated the lower chest and trunk from the operating table. Pigs were allocated to one of five experimental groups, as follows.

Group 1: (Balloons positioned—no compression—no electrical stimulation). Five pigs were exposed to these essentially sham conditions. The balloons were positioned but not inflated. Laser Doppler flux measurements on rootlets between the balloons were averaged over a 6-minute period to establish a baseline value, and then recorded for a 60-second period every 3 minutes for 60 minutes.

Group 2: (Two sites of compression at different pressures—no electrical stimulation). Five pigs were exposed to a double site compression by inflating the two balloons to 1, 1.5, 2, and 3 kPa in random order. Flux was recorded as for group 1 for 45 minutes.

Group 3: (No compression—electrical stimulation). Five pigs were set up with the two balloons in position and without rootlet compression as for group 1. After a 30-minute period of flux recording under resting conditions, electrical stimulation of the rootlets began: flux was then recorded every 2 minutes for another 30 minutes. Electromyographic activity of the tail muscles was recorded during the same period. In two pigs movement of the tail was prevented to establish that the system was sufficiently stable to be confident that change in flux was unrelated to the induced tail movement.

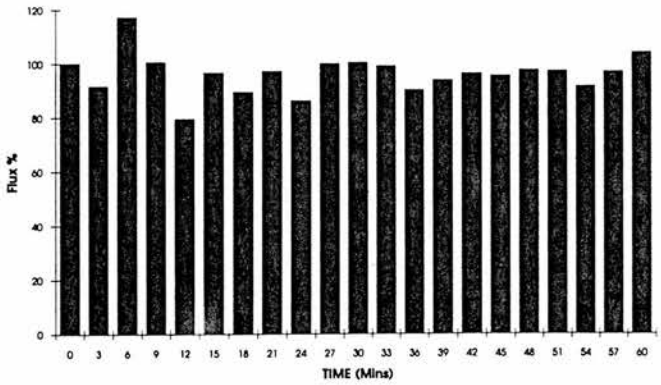

Figure 2. Blood flow in cauda equina during 60 minutes in sham conditions (Group 1): balloons positioned—no compression—no electrical stimulation.

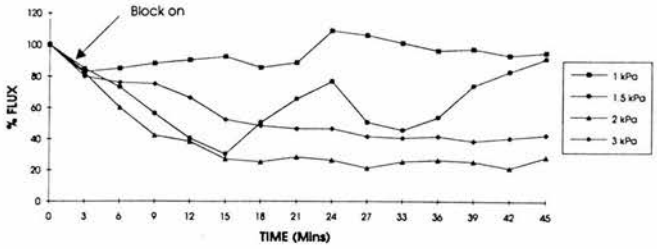

Figure 3. Blood flow during a period of 45 minutes, between two compression sites, at balloon pressures of 1, 1.5, 2, and 3 kPa (Group 2). There was a significant fall in flux at 1.5 kPa, which recovered during a 45-minute period. The reduction in flux at 2 kPa was not significantly different from that at 3 kPa, and neither recovered during a 45-minute period. (2 kPa was therefore selected as the appropriate level of pressure for subsequent studies).

Group 4: (Single site 2 kPa compression—electrical stimulation). Five pigs were monitored as for group 3, first with a 30-minute resting period, followed by a 20-minute period of electrical stimulation of the rootlets during a single site 2 kPa compression by the proximal balloon.

Group 5: (2 kPa compression at two levels—electrical stimulation). Five pigs were monitored as for group 3, initially for a 30-minute resting period, followed by another 30-minute period during which continuous electrical stimulation was applied to the rootlets, proximal to a double site compression of 2 kPa.

Results

Group 1: Under sham conditions (Balloons positioned—no compression—no electrical stimulation) flux values did not change significantly during the 60-minute period of recording. There was a small variation in the flux values around the mean (maximum 117%, minimum 79%), (Figure 2).

Group 2: In these animals (Double compression at varying pressures—no electrical stimulation) with a 1 kPa compression of the rootlets at two sites a small decline in flux levels of 15% was seen during the first 18 minutes; this then increased to 5% below the precompression level (Figure 3). With a 1.5 kPa compression

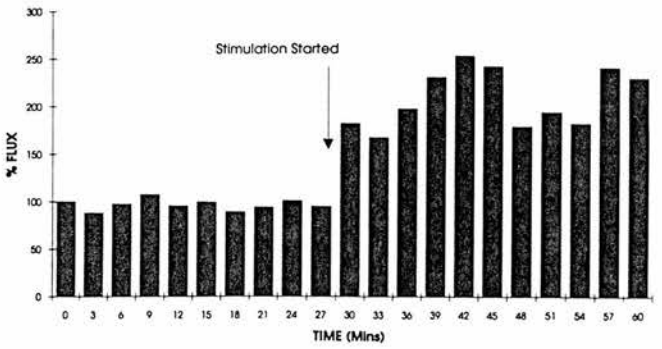

Figure 4. Blood flow during a 60-minute control period without compression (Group 3). Electrical stimulation was applied to the proximal rootlets after 30 minutes at rest. Flux rapidly increased by >100% and was maintained for another 30 minutes.

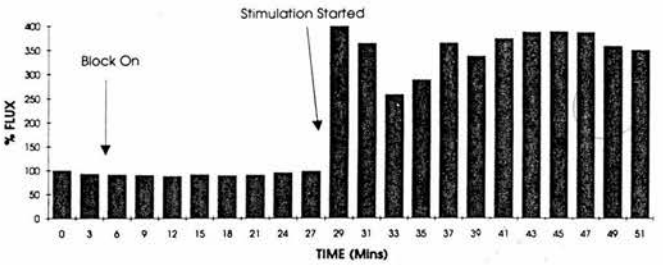

Figure 5. Blood flow measured during a single site 2 kPa compression period. The effects of proximal electrical stimulation were observed (Group 4). Flux rapidly increased by >100% and was maintained.

there was a steep decline in blood flow (70%) during the first 15 minutes (gradient −4.78 flux units per minute, r = 0.99). Flux then increased during the next 30 minutes to stabilize at 10% below initial values. With a 2 kPa compression a steep decline (the first 15 minutes) in flux occurred (73%, −4.90 gradient, r = 0.98). This then decreased to a minimum value of 21% of the initial flux (−0.02 gradient, r = 0.09). With 3 kPa, the flux decline during 15 minutes was smaller (48%, gradient of −2.69, r = 0.95). This then flattened to a minimum of 38%, (62% of the initial flux gradient −0.28, r = 0.79).

Group 3: (No compression—electrical stimulation). Initial mean resting flux was relatively constant (maximum 107%, minimum 88%) as seen with group 1 animals. When electrical stimulation was applied to the proximal rootlets there was a rapid increase in flux to a median of 197% (maximum 252%, minimum 167%), which was maintained during a 30-minute period of stimulation (Figure 4). An unpaired nonparametric *t* test (Mann Whitney) indicated that the increase was significant ($P < 0.0001$). There were strong contractions of the tail muscles and the EMG recordings increased in amplitude and duration in parallel with the increased flux during stimulation. Flux remained constant throughout the 30-minute period, as did the apparent magnitude of the tail muscle twitch.

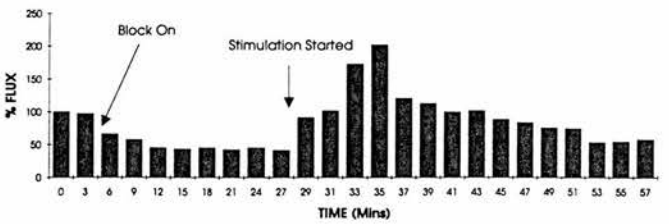

Figure 6. Blood flow measured between two sites at which 2 kPa compression was applied and the effects of proximal electrical stimulation were then monitored (Group 5). After a 30-minute control period flux fell to <50% of the resting values (note the early and later gradients). After stimulation of proximal rootlets it temporarily increased to 100% of the resting level during 6 minutes, then fell to 50% of the resting level during a 20-minute period. This was associated with a decrease in the electromyographic activity.

Group 4: (Single-level 2 kPa compression of the proximal balloon—electrical stimulation). Flux changes in this group were similar to those in group 3. There was a small fall in flux during the 30-minute rest period, to 88% of the resting flux values. After stimulation, flux increased rapidly to a median value 228% (maximum 401% minimum 165%) above the resting level that was maintained throughout the 20-minute stimulation period (Figure 5). The tail muscle EMG recording did not alter in amplitude or duration.

Group 5: (Double-level 2 kPa compression—electrical stimulation). Compression at two sites was followed by a marked decrease in blood flow (mean −29% resting) between the two compression sites during a 30-minute period. The initial gradient of fall in flux was −12.1 with the secondary gradient −0.62, similar to the decline seen in group 2 at 2 kPa. Immediately after electrical stimulation of the cauda equina, there was an increase in flux between the two compression sites of 54% (maximum 122%, minimum 24%) above resting flux (gradient 9.6) during a 4-minute period. However, the increase was not maintained. There was a steady fall in blood flow to 26% of the resting level thereafter (Figure 6). EMG recordings during stimulation initially showed strong activity, which was not maintained. EMG activity (the observed contractions) markedly decreased in parallel with the substantial reduction in flux. In all animals, continuous monitoring of mean arterial blood pressure indicated no significant differences among the groups, nor was there any change during the course of the trials.

■ Discussion

Previous studies of cauda equina blood flow in the segment between two sites of low pressure compression have shown that flow is reduced to 64% of the precompression level using a thermal diffusion method.[7] We used the same porcine model[3] but improved the technique in using a laser Doppler technique to measure blood flow. Provided the laser Doppler probes are carefully applied, the technique readily allows dynamic measurements of flux changes to be made accurately. Our studies confirm the previous work of Takahashi and colleagues,[7] in showing that low pressure compression at a single site induces no significant change in blood flow in the rootlets. However, with low pressure compression at two sites, blood flow rate falls significantly in the segment between the compression sites. In our experiments the fall was substantial, (75%) below resting flux values and was associated with a two-stage decline in flow: an early followed by a later component. This requires further investigation.

Patients with neurogenic claudication frequently have stenosis at least two sites, but it is difficult to explain the leg symptoms purely in terms of venous pooling between the sites. Why are symptoms not present in the legs at rest, and when symptoms develop, why are they relieved by a period of rest? If a build-up of metabolites between the two sites of compression were responsible for leg symptoms, one would expect at least some patients to have degrees of occlusion of blood flow high enough to cause symptoms at rest. On an experimental basis in the pig we therefore applied to the rootlets, a low level of compression at two sites in association with simulated exercise produced by contractions of the tail muscles. When the tail muscle was stimulated electrically in the absence of rootlet compression, blood flow increased rapidly to more than 200% above resting levels.

However, in animals in which a two-site low pressure compression had been applied and there had been a marked fall in flux as a result, we were surprised to observe that during muscle contraction blood flow in the rootlets, between the compression sites, increased substantially above (>50%) the resting levels. The change in flux (blood flow) was reduced compared to that seen in those pigs without a rootlet compression, and more importantly it was not sustained. During stimulation it progressively fell to a plateau still below resting (26%) and, this fall was associated with a parallel decrease in EMG activity and the associated tail muscle twitch magnitude.

It is likely that the increase in flux after proximal rootlet stimulation is the result of vasodilatation in the more distal parts of the cauda equina. In the presence of a two-site compression however, the response is short lived. It may be that in the chronic situation the failure of arterial vasodilatation in the cauda equina may well explain the symptoms of neurogenic claudication in some patients. The venous drainage of the roots of the cauda equina is centrifugal toward the foramen,[1] but in the presence of a distal compression this flow can reverse with a venous drainage toward the conus through anastomoses with the venous system of other roots. Blood in the "vasa radiculorum" and their accompanying radicular veins can flow in either direction,[5] because radicular veins normally drain proximally to the vasa carona or distally to the epidural veins. Therefore a two-site block at an appropriate pressure level is necessary to induce venous congestion of the roots. Although venous congestion from a two-site block will reduce blood flow at rest, it may be a failure of the sustained hyperemic response during exercise that is responsible for some of the symptoms of neurogenic claudication. Thus there may be several components to the pathophysiology of this condition. An arterial component would explain why patients are generally at an age common for arteriosclerosis, and why the symptoms of neurogenic claudication and peripheral arterial disease often coexist. The observation that the spinal cord is markedly affected by a single traumatic compression in the presence of underperfusion[2] suggests that nerve function is very sensitive to changes in local arterial pressure. A failure of arterial vasodilatation in the nerve

roots in response to exercise would explain why symptoms are not present at rest, why they develop after a short period of exercise, and why they are relieved by rest. It would also explain why, in spite of severe disability when walking, few patients with neurogenic claudication have serious neuropathy or "go off their legs."

Conclusion

We suggest that in neurogenic claudication, venous pooling between two sites of low pressure block is probably the basic underlying pathology. This in turn is responsible for an impairment of the vasodilatation response to exercise, resulting in impaired nerve function during exercise and symptoms of claudication when walking.

Acknowledgment

The authors thank the Wishbone Trust and the University of Aberdeen for their financial support of this research.

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Address reprint requests to

Andrew R. Baker, FRCS
Department of Orthopaedic Surgery
Medical School
University of Aberdeen
Aberdeen, AB9 2ZD
Scotland

Spine 20(4)1995
SPINE Volume 20, Number 4, pp 408–411

Measurement of Lower Limb Blood Flow in Patients With Neurogenic Claudication Using Positron Emission Tomography

24

Gary F. Keenan, FRCS (Glas),* George P. Ashcroft, FRCS, FRCS Orth,* Giles H. Roditi, MRAD, FRCR,† James D. Hutchison, PhD, FRCS,* Noel T. S. Evans, PhD,‡ Pal Mikecz, PhD,‡ Fiona Chaloner, PET Technician,‡ Maurice Dodd, Cyclotron Technician,‡ Christopher Leonard, PET Technician,‡ Richard W. Porter, MD,* Peter F. Sharp, PhD, FRSE,‡ and Frank W. Smith, MD, FFR, RCSI§

Study Design. Ten subjects (seven with neurogenic claudication and three control subjects) underwent examination of lower limb muscle blood flow before and after exercise using positron emission tomography.

Objectives. To investigate the hypothesis that lower limb muscle ischemia was the origin of symptoms in neurogenic claudication.

Background. Patients with neurogenic claudication secondary to spinal stenosis experience lower limb discomfort after exercise similar to that of ischemic claudication. However, they do not have clinical evidence of peripheral vascular disease. The authors postulated that the lower limb discomfort in patients with neurogenic claudication may arise from muscle ischemia due to inadequate dilatation of arterioles in response to exercise, this itself arising secondary to sympathetic dysfunction due to spinal stenosis.

Method. Using O^{15}-labeled water and positron emission tomography measured thigh and leg muscle blood flow response to exercise bilaterally in seven patients with unilateral neurogenic claudication and three control subjects were measured.

Results. The average values obtained for mid-thigh and mid-calf muscle perfusion at rest were 2.57 ml/min/100 g tissue (2.23–3.90) and 2.39 ml/min/100 g tissue (2.03–3.46), respectively. The average values obtained from mid-thigh and mid-calf perfusion after exercise were 4.41 ml/min/100 g tissue (2.8–6.0) and 4.87 ml/min/100 g (2.2–11.7). We found no difference in muscle perfusion between symptomatic and asymptomatic limbs in this group of patients.

Conclusion. These studies suggest that muscle ischemia is not the origin of symptoms in most patients with neurogenic claudication. [Key words: neurogenic claudication, pathophysiology, muscle blood flow, positron emission tomography] **Spine 1995;20:408–411**

From the Departments of *Orthopaedics, †Radiology, ‡Medical Physics, and §Nuclear Medicine, Aberdeen University Medical School, Aberdeen, Scotland.
Accepted for publication June 27, 1994.
Device status category: 1.

The pathogenesis of neurogenic claudication is incompletely understood. This condition typically produces discomfort in the thighs, calves, and feet after exercise, with symptoms lessened by rest and leaning forward. Neurogenic claudication frequently coexists with peripheral vascular insufficiency[4,6] and distinguishing between the two can be difficult. The nociceptive source in patients with symptoms of intermittent claudication (due to peripheral vascular disease) is thought to be within ischemic muscles whose supplying vessels are deficient. We postulated that the nociceptive source in patients with neurogenic claudication may also be within the limb muscles whose vessels do not dilate after exercise; not because of arterial pathology but because there is an efferent sympathetic nerve dysfunction at the spinal level that interferes with muscle arteriolar vasodilatation. The nociceptive source in both neurogenic and intermittent claudication being the same but the mechanism by which the symptoms are produced is different. The purpose of this study was to test this hypothesis using positron emission tomography (PET) to examine lower limb muscle perfusion in patients with unilateral claudication. If the hypothesis was accurate we would expect the symptomatic limb to be poorly perfused after exercise relative to the normal side.

Materials and Methods

The study was approved by the Grampian Area Regional Committee and all patients gave informed written consent before being enrolled into the study. Eight patients assessed at Orthopaedic clinics volunteered to undergo PET studies. Two young volunteers were also examined. Seven of the patients had symptoms of neurogenic claudication and radiologic evidence supporting this diagnosis, one of these had clinical signs of peripheral vascular disease. Another patient with back pain acted as an age-matched control subject. In addition to PET studies each patient underwent vascular assessment using

color flow Doppler ultrasound to exclude peripheral vascular disease as a cause for their symptoms.

Venous and arterial cannulas were each inserted into the dorsum of the hand and contralateral radial artery under local anesthesia. ^{15}O-labeled water was prepared by bombarding nitrogen with 15 MeV deuterons from the Aberdeen CS −30 Cyclotron using the N (d,n) ^{15}O reaction. The resulting ^{15}O-labeled oxygen was then combined with hydrogen using a palladium catalyst. The patients were examined using an E.G. and G Ortec ECAT II Tomograph (CTI West Inc, San Francisco, CA). Blood perfusion was examined in both lower limbs by transaxial images taken at mid-thigh and mid-calf levels. These levels were marked and the patient was asked to walk on a flat surface until symptoms developed. Once they occurred the patients were repositioned on the scanner and more images were taken at the same levels to assess changes in the blood flow within the muscle compartments. To obtain images at each level, approximately 1850 MBq of ^{15}O-labeled water in 4 ml of saline was injected via the intravenous cannula. Continuous arterial blood sampling monitored using a beta counter, gave decay data to produce an activity time course curve, allowing absolute quantification of blood flow. The attenuation corrected images were analyzed on a SUN SPARC workstation (Siemens, Erlangen, Germany) using software developed in Aberdeen.

Mean count values were obtained using a region of interest technique over the anterior and posterior compartments of the thigh and calf. Four regions were assessed in the thigh and five in the calf. The mean value was then taken to represent the muscle flow within the compartment. On a separate day, the patients underwent ankle brachial index and color flow Doppler ultrasonography (Hitachi EUB - 515) by a radiologist experienced with these techniques. The patients were exercised again in the gait laboratory on a treadmill and the distance walked before development of symptoms was recorded.

■ Results

Ten subjects were examined, seven patients with evidence of spinal stenosis and symptoms suggestive of neurogenic claudication, one age-matched control subject with back pain but no leg symptoms, and two young volunteers. The average muscle perfusion at rest estimated by PET was highly reproducible both within the study and in comparison to values obtained for muscle perfusion in a parallel study of tibial fracture patients.[1] Data from seven studies allowed absolute quantification of blood flow using arterial sampling to produce an activity decay curve. The average values obtained for mid-thigh and mid-calf muscle perfusion at rest were 2.57 ml/min/100 g tissue (2.23–3.90) and 2.39 ml/min/100 g tissue (2.03–3.46), respectively. The average values obtained for mid-thigh and mid-calf perfusion after exercise were 4.41 ml/min/100 g tissue (2.8–6) and 4.87 ml/min/100 g (2.2–11.7). Patient details and the results of the investigations are shown in Tables 1 and 2. Nonparametric tests (Mann-Whitney U) were used for statistical analysis of the blood flow data.

There was no consistent difference in muscle perfusion between the symptomatic and asymptomatic legs of patients with unilateral claudication in response to exercise. In patients 2, 3, and 8, a rise in muscle perfusion was not observed, probably because those patients had symptoms preventing exercise of sufficient intensity and duration to bring about a sustained increase in blood flow. Peripheral vascular disease was demonstrated in patient 1 using color flow Doppler ultrasound. In particular she had significant stenosis of the left femoral artery at the level of the adductor canal. This patient had reduced activity counts in the left leg at rest compared with the right ($P = 0.01$ for thigh, $P = 0.02$ for calf) mirroring the vascular insufficiency. The postexercise values did not show a significant difference. Patient 4 had a relatively smaller increase in perfusion in the symptomatic side. This was found to be statistically significant ($P = 0.02$), and would support our hypothesis, however, this observation was not repeated in the other patients.

■ Discussion

The syndrome of neurogenic claudication is characterized by heaviness or discomfort of thighs, calves and feet, precipitated by exercise and relieved by rest and forward flexion of the lumbar spine.[7] Central canal stenosis is considered an essential component to the pathogenesis of the condition.[9] More recently the significance of a two-level venous compression has been highlighted,[8] with pooling of venous blood leading to ischemia of a nerve root and generation of the symptoms. The two-level block may be produced by protrusions into the canal from degenerate segmental pathology with or without vertebral displacement, root canal stenosis from facet joint hypertrophy, rarely a spinal tumor, or any combination of these conditions.[5] However, it is well known that spinal stenosis may be demonstrated in patients without any symptoms.[2,5,6,10]

It is also recognized that patients with spinal stenosis (presumably with or without neurogenic claudication) may have peripheral vascular disease producing similar symptoms of intermittent claudication. However the discomfort of neurogenic and intermittent claudication are so similar it may be difficult to tell them apart without further investigation.[4] We have hypothesized that both symptoms arise from the same local effect, that is, detection of accumulated metabolites after exercise by nociceptors within the muscle. Unilateral neurogenic claudication is an ideal model to test this hypothesis. If the symptoms were due to muscle ischemia, we would expect less of an increase in muscle perfusion in the symptomatic compared with the asymptomatic limb. However, we have demonstrated this observation in only one patient.

Patient 1 had appearances of a significant vascular lesion at the left adductor hiatus (as suggested by a low ankle brachial index) based on a change in velocity and blunting of the normal triphasic wave form in the distal artery (popliteal) using color flow Doppler ultrasonog-

Table 1. Patient Details and Investigations

Patient 1 (73 yr woman)	Patient 2 (67 yr woman)	Patient 3 (71 yr man)	Patient 4 (58 yr man)	Patient 5 (77 yr woman)
Left calf pain on exercise rests and bends forward to relieve pain No abnormal neurology Femoral pulses normal DP/PT absent	Heaviness both legs, right worse than left, pain worse on walking No abnormal neurology Pulses normal	Long history of back pain 1 year history of left leg pain worse on walking No abnormal neurology Absent DP/PT bilaterally	Back pain, left calf pain brought on by exercise eased by rest and leaning Forward intact neurology Normal pulses	Back pain, heaviness in legs after walking but not cycling R > L Normal neurology Normal pulses
Walking distance 66 m @ 2 km/hr	Walking distance >500 m	Walking distance 30 m	Walking distance 85 m @ 2 km/hr	Walking distance 75 m @ 2 km/hr
MRI: disc prolapse (post) L2/3 and 3/4	MRI: spondylolisthesis L4/5, no evidence of intervertebral prolapse	Myelogram: Stenosis L2/3, left S1 nerve root compression	MRI: narrow spinal canal Degenerate discs at L3/4, L4/5 and L5/S1	MRI: retrolisthesis of L2 L1, spondylolisthesis L4/5 nerve roots narrowed
PET: no arterial data activity counts only, significant rise in counts postexercise, left side increase appeared less than right	PET: no arterial data counts only, no significant rise in counts demonstrated postexercise	PET: absolute blood perfusion showed resting level values postexercise 2.9 and 2.6 ml/min/100 g tissue	PET: absolute values, rest 2.3, 2.4; exercise R thigh 6.0, L thigh 4.3 (ml/min/100 g tissue)	PET: absolute values, rest 2.3, 2.5; exercise R thigh 6, L thigh 4.2 (ml/min/100 g tissue)
Ankle/brachial index 0.7	Ankle/brachial index 1.1	Ankle/brachial index 0.9	Ankle/brachial index 1.1	Ankle/brachial index 0.97
Color flow Doppler: significant stenosis at adductor hiatus left knee requiring treatment	Color flow Doppler: normal	Color flow Doppler: no significant peripheral vascular disease	Color flow Doppler: normal	Color flow Doppler: no significant vascular disease
Conclusion: radiologic evidence of two level block but symptoms of intermittent rather than neurogenic	Conclusion: insufficient exercise to produce a sustained increase in blood flow because of development of symptoms	Conclusion: insufficient exercise because of leg pain to produce rise in leg blood flow, discomfort at rest likely due to root pain	Conclusion: symptomatic leg showed less increase in blood flow with exercise normal vascular tree $P = .02$	Conclusion: no statistical difference between legs, greater average blood flow recorded in symptomatic limb

R = right; L = left; MRI = magnetic resonance imaging; PET = positron emission tomography.

Table 2. Patient Details and Investigations

Patient 6 (50 yr man)	Patient 7 (67 yr man)	Patient 8 (69 yr woman)	Patient 9 (30 yr man)	Patient 10 (28 yr man)
Back pain, 6 month history of left thigh discomfort on walking No abnormal neurology Normal pulses	Age-matched control had past history of sciatic type leg pain now back pain only, no leg pain Normal neurology Normal pulses	Back pain, right leg pain, paraesthesiae, worse on standing and walking absent AJ bilaterally blunting of pin prick sensation S1.	Young normal	Young normal repeatability study
Walking distance 550 m @ 2 km/hr	Walking distance 1 km @ 2 km/hr (no symptoms)	Walking distance 43 m @ 2 km/hr	N/A	N/A
MRI: degenerate discs L2, L3, L4	MRI: severe disease affecting all intervertebral discs T12 to L5/S1	CT: canal stenosis L2/3, L3/4	N/A	N/A
PET: absolute values, rest 2.4, 2.23; exercise R thigh 4.0, L thigh 5.9 Ankle/brachial index 1.2	PET: absolute values, rest 2.1, 2.25; exercise R calf 5.1, L calf 3.1	PET: counts only, no increase observed in response to the limited exercise performed Ankle/brachial index 1.1	PET: absolute values, rest 1.85, 1.42; exercise R thigh 3.5, L thigh 3.3	PET: absolute values, rest 3.93, 3.47; repeat 3.2, 3.0
Color flow Doppler: normal study	Patient died before vascular study	Color flow Doppler: normal	Normal	Normal
Conclusion: no significant difference observed between increases observed		Conclusion: despite radiological stenosis, symptoms are from S1 root pain inadequate exercise tolerance because of pain to induce increase in blood perfusion	Conclusion: rapid return of blood perfusion to normal values following cessation of exercise	Conclusion: along with repeat measurements taken on patients 4, 5 and 6 We demonstrated repeatability of the technique

R = right; L = left; MRI = magnetic resonance imaging; PET = positron emission tomography; CT = computed tomography.

raphy. It is believed that this lesion is responsible for her symptoms of left calf pain rather than neurogenic claudication. A very recent report[3] describes using PET to investigate blood flow in a group of vascular patients compared with normal controls. The resting muscle perfusion values obtained by these workers were consistent with ours.

■ Conclusion

The pathophysiology of neurogenic claudication is undoubtedly complex, particularly when other conditions co-exist. Our initial studies suggest that muscle ischemia is not the origin of symptoms in most patients with this condition.

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TREFOIL CONFIGURATION AND DEVELOPMENTAL STENOSIS OF THE LUMBAR VERTEBRAL CANAL

25

TIBOR PAPP, RICHARD W. PORTER, RICHARD M. ASPDEN

From the University of Aberdeen, Scotland

The midsagittal and interpedicular diameters and the trefoil shape of lumbar vertebrae of known age at death were measured in skeletons from a population aged between 1 and 70 years.

All the trefoil configurations were at L5 with the exception of one at L4. The overall prevalence was 25%, but this shape was not generally apparent until adulthood.

The midsagittal diameter in the trefoil canals was found to be significantly smaller than that in the unaffected canals. This did not change significantly after six years of age indicating that the cause of the trefoil configuration is probably present early in life. The trefoil shape was no more common in the spines of the elderly subjects.

Our findings indicate that the trefoil configuration of the lumbar vertebral canal has a developmental origin and is not a consequence of degenerative processes.

J Bone Joint Surg [Br] 1995;77-B:469-72.
Received 16 May 1994; Accepted after revision 5 August 1994

Verbiest (1954, 1955, 1975) first reported cases of central narrowing of the lumbar vertebral canal in adults in whom he drew attention to the hypertrophy of the laminae. He suggested that abnormalities in the diameters of the canal may be present at birth, but that progressive thickening of the laminae and articular processes in adulthood is the cause of compression of the neural elements. The trefoil shape of the vertebral canal occurs when the posterolateral sides are concave and protrude into the canal. This indention is symmetrical hence the trefoil or maple-leaf appearance (Armstrong 1952).

Some clinicians consider that facet osteophytes are chiefly responsible for the appearance of the troublesome trefoil shape of the lumbar canal (Verbiest 1954; Kirkaldy Willis et al 1974). Eisenstein (1977, 1980) suggested that developmental stenosis was associated with this configuration but he did not find any correlation with sagittal narrowness. Porter (1980) also supported the developmental hypothesis in finding no correlation between degenerative changes and the trefoil configuration. Epstein, Epstein and Lavine (1962, 1964) identified a relationship between small sagittal diameter and a narrow lateral recess.

The trefoil shape of the lumbar canal is commonly seen on CT or MRI in patients with low back pain and with radicular symptoms. Hence an understanding of its origin is important.

MATERIALS AND METHODS

We examined the lumbar spines of 185 skeletons from the Spitalfield Collection at the Natural History Museum, London. This collection originates from immigrant French Huguenots between the 17th and 19th centuries. Exact records of their births and deaths have been preserved. Their ages were between 1 and 70 years: 41 were infants (16 months to 5 years), 43 were juveniles (5 to 16 years) and 101 were adults (16 to 70 years). There were 210 lumbar vertebrae missing or damaged, leaving 715 available for investigation.

Silhouette, unmagnified photographs of the lumbar vertebral canals were taken using a specially designed photographic box as described by Porter and Pavitt (1987). The images obtained were of the most proximal aspects of the canals, since it is at this level that the central canal is physiologically narrowest and therefore most important clinically in regard to space-reducing lesions (Fig. 1).

The midsagittal diameter, interpedicular diameter and the shape were measured by computerised image analysis which gives an accuracy to the nearest 0.01 mm. The trefoil shape was expressed as a ratio of a transverse measurement taken at one-third of the distance from the midpoint of the interpedicular diameter to the apex of the neural arch, and the full interpedicular diameter. A vertebral canal is trefoil in shape if the ratio is less than 0.6667. The values through childhood and adult life were compared with the midsagittal and interpedicular diameters. The components of this ratio alter, however, as the length of the sagittal diameter changes. We therefore used another method to investigate a possible correlation between the midsagittal diameter and trefoil shape. One of the most relevant ele-

T. Papp, MB ChB, Honorary Research Fellow
R. W. Porter, MD, FRCS, FRCS Ed, Professor of Orthopaedic Surgery
R. M. Aspden, PhD, MRC Senior Fellow
Department of Orthopaedic Surgery, University of Aberdeen Medical School, Polwarth Building, Foresterhill, Aberdeen AB9 2ZD, UK.

Correspondence should be sent to Dr T. Papp.

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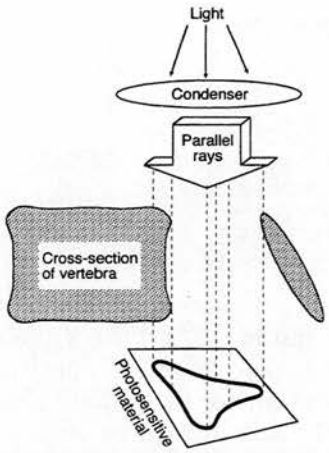

Fig. 1

Diagram showing method of obtaining unmagnified silhouette images of the canal.

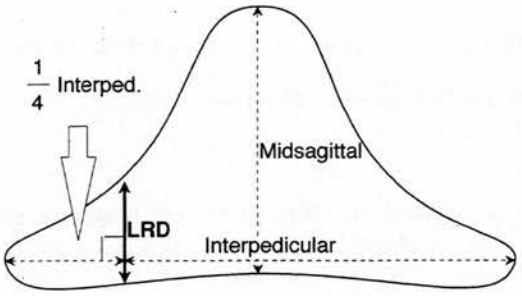

Fig. 2

Diagram showing the measurement of the lateral recess depth (LRD).

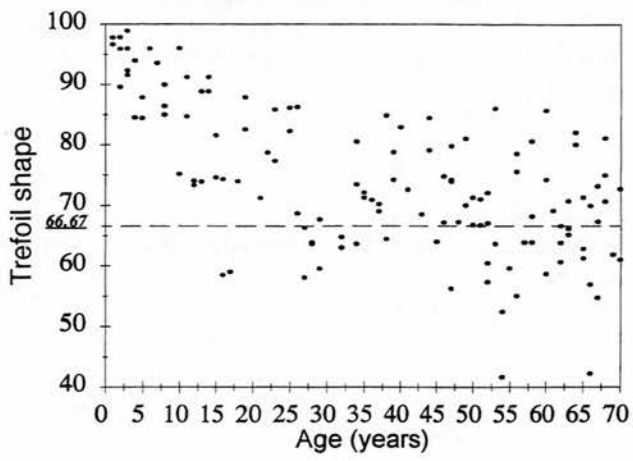

Fig. 3

The increase in trefoil shape was significant only until adulthood. Canals in which the ratio was less than 0.667 were regarded as trefoil-shaped.

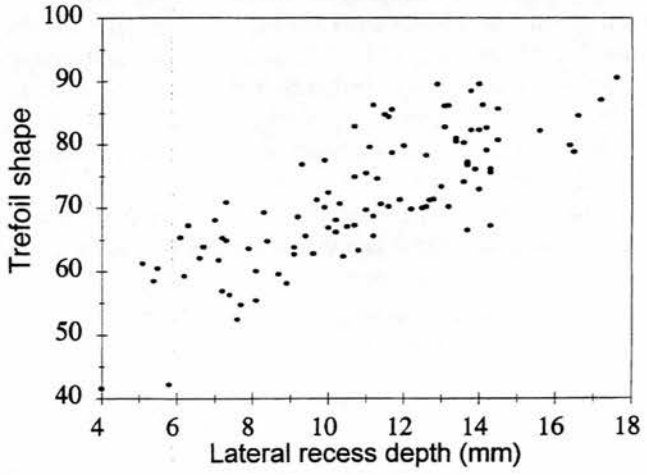

Fig. 4

Graph showing a significant linear correlation between trefoil shape and lateral recess depth.

ments of the trefoil shape is the formation of the lateral recesses. We measured the depth of the lateral recesses (LRD) at one-fourth of the interpedicular distance from the lateral end-point (Fig. 2). The LRD was then compared with the midsagittal diameter. Statistical analysis used linear regression, Student's *t*-test and the Mann-Whitney rank-sum test.

RESULTS

The trefoil configuration occurred only at the fifth lumbar vertebra except for one skeleton in which it was also present at L4 (0.7%). It did not occur in childhood; the youngest skeleton in which it was seen was 16 years of age. The overall prevalence was 25% and did not significantly change throughout adulthood. The trefoil shape was seen in 23% of 65 male and in 28% of 78 female skeletons. The occurrence was not significantly different between the sexes ($p = 0.157$).

The ratio tended to decrease in the first two decades of life ($p < 0.001$) as the shape of the canal approached that of a trefoil. Over 16 years of age no significant change was found in the measurements between the separate age groups (Fig. 3). In infancy an almost circular dome-shaped lumbar canal is usually seen. By adulthood it becomes triangular and in some of the vertebrae the sides become concave producing the trefoil shape. Table I shows the measured dimensions of trefoil and non-trefoil canals in different age groups.

In the adult spines the midsagittal diameter was significantly smaller in trefoil canals than in those of normal shape ($p < 0.03$). Since the measurement of trefoil shape is linked to the midsagittal diameter it compensated for the changes in AP dimension and underestimated the relation-

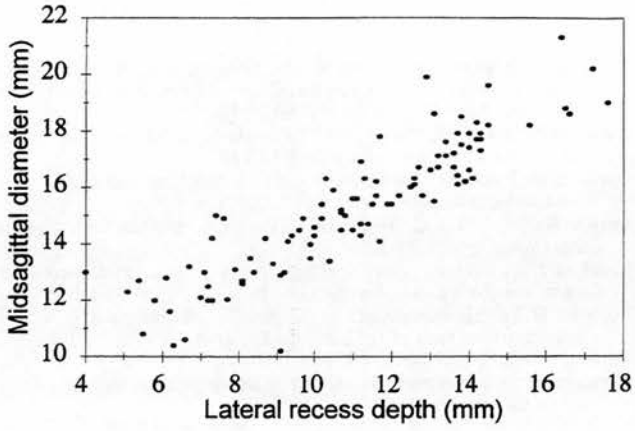

Fig. 5

Graph showing a highly significant correlation between the lateral recess depth and the midsagittal diameter.

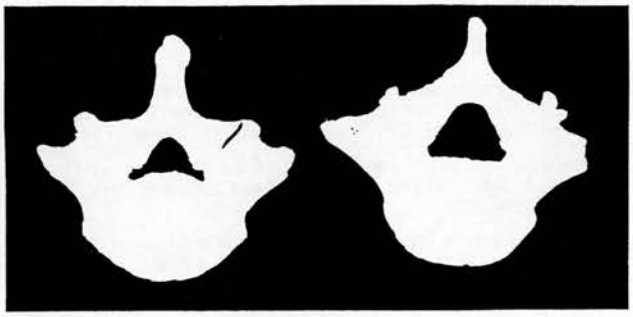

Fig. 6

The midsagittal diameter of the trefoil canal (left) is smaller than that of the normal canal (right).

ship between the midsagittal diameter and the trefoil shape. We found correlations of 0.49 between trefoil shape and LRD ($p < 0.001$; Fig. 4) and of 0.84 between LRD and midsagittal diameter which are highly significant ($p < 0.001$; Fig. 5). There was no significant difference in interpedicular diameter between the two groups ($p > 0.5$).

No significant relationship was found between the trefoil shape and degeneration of the articular facets or osteophytes encroaching into the vertebral canal, although in a few cases when these conditions occurred together with trefoil configuration, they produced additional narrowing of the canal.

DISCUSSION

The trefoil shape of the vertebral canal can have troublesome clinical consequences; when the cross-sectional area is reduced by osseous narrowing there is relatively less room for soft-tissue structures. Thus, in the presence of disc protrusion, segmental displacement or the dynamic activity of extension the neural contents may be compromised in a small canal. Both a small midsagittal diameter and a trefoil shape can have clinical significance, particularly in combination (Fig. 6). Our study has shown that a small LRD is an essential constituent of the trefoil configuration and that there is a significant relationship between trefoil shape and small midsagittal diameter. There was no correlation, however, between the interpedicular diameter and trefoil shape. The midsagittal diameter matures at L5 in infancy while the interpedicular diameter increases in size up to adulthood. Thus although the trefoil shape is not apparent until adulthood it is probably programmed in early life.

There was a significant increase in the occurrence of trefoil shape until adulthood in this cross-sectional population, but no significant difference was detected above and below 40 years of age in adults, suggesting that trefoil shape is largely developmental and that any contribution from degenerative change is relatively small.

The aetiology of trefoil shape and developmental stenosis is not known. It has been suggested that infant malnutrition may be responsible for sagittal narrowness of the lumbar canal (Clark, Panjabi and Wetzel 1985; Porter and Pavitt 1987). There may be independent factors involved which can only be resolved by longitudinal and genetic studies.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

Table I. Mean midsagittal (MS) and interpedicular (IP) diameters (mm) and trefoil shape (TS) (percentage) in trefoil and non-trefoil canals according to age for 143 L5 vertebrae

Age (yr)	Trefoil (n = 29)				Non-trefoil (n = 114)			
	MS	IP	TS (%)	Number	MS	IP	TS (%)	Number
1 to 10				0	15.74	18.97	*92*	27
11 to 20	14.73	24.61	*59*	3	14.84	22.57	*73*	14
21 to 30	12.09	24.17	*62*	4	15.87	23.96	*78*	15
31 to 40	15.01	24.22	*51*	2	15.73	22.84	*77*	15
41 to 50	14.93	25.30	*60*	5	16.18	24.93	*75*	13
51 to 60	15.02	26.10	*56*	7	14.95	23.20	*76*	15
61 to 70	14.09	24.91	*60*	8	15.19	24.28	*75*	15

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Reprinted from SPINE, Vol. 21, No. 17, September 1, 1996

Spinal Stenosis and Neurogenic Claudication

Richard W. Porter, MD, FRCS, FRCSE

SPINE Volume 21, Number 17, pp 2046–2052

Spinal Stenosis and Neurogenic Claudication

Richard W. Porter, MD, FRCS, FRCSE

Neurogenic claudication is diagnosed from a classical history and complementary spinal imaging. The abnormal signs may be few. It should be distinguished from intermittent claudication (peripheral vascular disease), referred pain from the back or root pain that is aggravated by walking, and psychological distress.

Pathologically, a developmentally small canal is usually affected by multiple levels of segmental degenerative change, with venous pooling in the cauda equina between two levels of low pressure stenosis. There is probably then a failure of arterial vasodilatation of the congested roots in response to exercise, with symptoms in the legs when walking.

Once established, symptoms tend neither to improve nor deteriorate. Conservative management is reasonable. Otherwise decompression at the most significant stenotic level is probably adequate to obtain a good surgical result. [Key words: claudication, decompression, spinal stenosis] **Spine 1996;21:2046–2052**

Verbiest in 1954[12] was the first to recognize that structural narrowing of the vertebral canal could compress the cauda equina and produce claudication symptoms. In the past four decades, we have developed a better understanding of its clinical presentation and its pathophysiology.

Clinical Presentation

History

Symptoms usually affect men aged more than 50 years. They complain of discomfort in the thighs, calves, and feet when walking. Bilateral symptoms occur with a male:female ratio of 8:1, whereas the ratio in unilateral claudication is 3:1.[10] Typically, symptoms are not present at rest, but after walking a short distance patients experience weakness, tiredness, or heaviness of the legs that gradually increases and causes them to stop. The walking tolerance (when the patient stops) is usually twice the threshold distance when they first feel discomfort.

From the Royal College of Surgeons, Edinburgh, Scotland.
Acknowledgment date: August 25, 1994.
First revision date: August 20, 1995.
Second revision date: January 2, 1996.
Acceptance date: May 15, 1996.
Device status category: 1.

It can vary during the day or from one day to the next or even during one period of walking. When walking, the patient stoops forward, gradually reduces walking speed, and sometimes will stoop forward until he or she finally stops—the stoop test. He or she may lean on a wall or stoop to tie a shoelace, and after a few minutes the legs recover and he or she start to walk again. Some find walking downhill particularly uncomfortable but cycling may be no problem and a useful discriminator between neurogenic and intermittent claudication (peripheral vascular disease). Most patients have a long history of previous back pain.

Examination

Apart from the spinal posture, the examination is remarkable for its lack of gross abnormality. The patient may be able to flex well forward with extended knees, although lumbar extension is usually absent. These patients may have difficulty standing erect, and they adopt a "Simian stance" with hips and knees slightly flexed. If this posture is not present at rest, it tends to develop with walking; the patient gradually stooping forward until he or she stops. The center of gravity gradually moves forward. It returns after each period of rest but takes longer to return to its initial position than it takes for symptoms to settle. The lumbar spine is often tender over several segments. Straight leg raising is generally full, and there are frequently no abnormal neurologic signs. Reexamination after exercise, however, may alter the results of the neurologic examination. The peripheral circulation can be normal, but not infrequently arterial disease will coexist.

Investigation

Assessment on a treadmill will establish an objective record of walking pain, with note of the speed of walking, the distance at which symptoms develop, the distribution of discomfort, the changing posture, and the walking tolerance. The impression from a patient's history can be completely different from an objective assessment of walking. When measuring a patient's response to treatment, a treadmill is invaluable.

A plain radiograph will raise the suspicion of a shallow vertebral canal and perhaps show degenerative spondylolisthesis, which is present in half the men with bilateral claudication.[7] Half of the patients with unilateral claudication have a structural lumbar scoliosis.

A computed tomography scan is helpful in recording the cross-sectional area of the central canal and stenosis of the root canal, but because most patients with neurogenic claudication have multiple level pathology, a magnetic resonance imaging scan is complementary. Some clinicians prefer myelography or computed tomography myelography to demonstrate the extent of the stenosis. It usually shows multiple level stenosis with redundant nerve roots.

It is advisable to have Doppler studies of the peripheral circulation and sometimes arteriography.

Differential Diagnosis

1. Intermittent claudication from peripheral vascular disease is difficult to distinguish from neurogenic claudication by the history alone. It is not affected by posture, but this is not a very sensitive discriminator between the two types of claudication. Cerebral somatosensory-evoked potentials after walking may help to differentiate neurogenic from vascular intermittent claudication. It is confusing when spinal stenosis and peripheral vascular disease often coexist.
2. Sciatic claudication is an insufficiency of the inferior gluteal artery, producing ischemia of the sciatic nerve and claudication in a sciatic distribution. It should be considered when there is no evidence of spinal stenosis and the peripheral circulation is good.
3. Referred pain from the lower lumbar region into the buttocks and thighs can mimic neurogenic claudication when the symptoms are aggravated by walking. However, referred pain will be present in activities other than walking, and despite symptoms, walking long distance, although painful, may not be impossible. An unstable isthmic spondylolisthesis may cause referred pain into the thighs when walking, but central canal stenosis is uncommon with pars defects because the vertebra displaces forward, leaving the floating lamina behind and widening the canal.
4. Some types of root pain and multiple root pathology are made worse by walking. In Crock's patients with isolated lumbar disc resorption, 11 18% had increasing leg pain or parethesia on walking distances up to 500 yards. Walking may aggravate a root entrapment problem, but these patients usually also have symptoms at rest.
5. Claudication pain is sometimes a symptom of distress. Abnormal behavior patterns are common in patients who have a long history of back pain, and pain in the legs when walking is not infrequently a symptom inappropriate to the underlying organic pathology in the spine. These patients usually exhibit inappropriate signs.
6. Litigation can sometimes so confuse the issue that it is not possible to decide how large the organic component is and whether the organic element of the leg pain is neurogenic claudication, root pathology, or referred pain.
7. There are other less common causes of claudication pain, including venous claudication after thrombosis, myxedema claudication with a limited potential of muscle to increase its metabolism with exercise, multiple sclerosis, and, rarely, deep arteriovenous fistula.

Pathology

1. Spinal stenosis has unfortunately become synonymous with neurogenic claudication, but a shallow canal is only one factor in the pathology. Spinal stenosis is sometimes entirely symptomless, 2 and stenosis is also a factor in other back pain syndromes, including disc protrusion and root entrapment from degenerative change. In addition, symptoms of claudication are unusual before the sixth decade of life, even though the vertebral canal will have been narrow for many years. Even in achondroplasia, with marked developmental stenosis, symptoms are not present in early life. The small canal is, therefore, but one factor in the pathology.
2. Degenerative soft tissue and bony pathology is invariably present in patients with neurogenic claudication. The ligamentum flavum is usually thickened or ossified. Some patients have diffuse idiopathic spinal hyperostosis (DISH) or Paget's disease.
3. Vertebral displacement with an intact neural arch will critically narrow an already small canal. Degenerative spondylolisthesis effectively reduces the canal size at the level of displacement. Although degenerative spondylolisthesis is more common in women, bilateral neurogenic claudication is more common in men, and approximately half the men with bilateral claudication have a degenerative spondylolisthesis.[7] The rotatory effect of a degenerative lumbar scoliosis can be an important factor in unilateral claudication.
4. The neuropathology is probably the result of inadequate oxygenation or accumulation of metabolites in the cauda equina. Nerve function is probably just adequate at rest but inadequate during exercise. The ischemic effect of compression on nerve function has been studied in animal models.[3,4] However, it is of interest that ablation of the lumbar arteries after aortic surgery is rarely followed by claudication symptoms.
5. Central stenosis at one level does not account for the symptoms. There are a number of clinical reasons why central stenosis alone does not explain the mechanism of claudication. First, a steadily progressing spinal tumor can completely block the central vertebral canal without producing claudication. Second, a large central disc protrusion can block the canal without claudication. Third, a single level stenosis from degenerative change at L3–L4 or L4–L5 may

almost occlude the dural sac and yet produce only back pain. Furthermore, imaging of asymptomatic subjects confirms that stenosis is common, and patients who present with claudication must have had symptomatic stenotic canals for many years. Again, it is surprising that in canine studies a single level experimental stenosis constricting the cauda equina by 25% did not cause neurologic deficit.[3]

6. Root canal stenosis does not account for the symptoms. A number of authors have thought that root canal stenosis or foraminal stenosis is responsible for claudication symptoms. However, isolated root canal stenosis may be asymptomatic or on other occasions responsible for the constant root pain of root entrapment but not claudication. If root canal pathology was important, why do patients with neurogenic claudication invariably have central canal stenosis?

7. One of the radiologic features of neurogenic claudication is the high frequency of multiple level stenosis in the central or root canals[7] (Figure 1). The venous anatomy of the roots of the cauda equina make them vulnerable to congestion at multiple levels. The veins of the roots (which do not anastomose between roots) generally drain distally to the foramen or, if this is occluded, proximally to the conus. A single low pressure block will affect only a small section of the root and probably not disturb conduction. However, in the presence of two low pressure blocks, there will be venous congestion in the intervening segment. The arterioles will continue to feed the segment at the higher arterial pressure, but impaired drainage will reduce the blood flow, the oxygen supply, and the nutrition, with a build up of metabolites in the uncompressed segment between the two blocks (Figure 2).

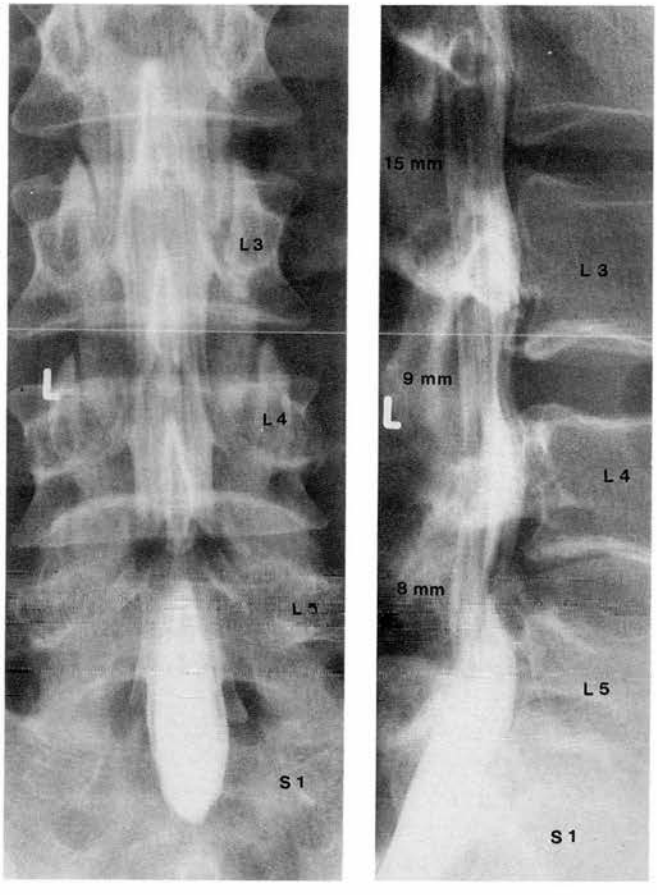

Figure 1. Myelogram of a patient with symptoms of bilateral neurogenic claudication. There is a partial occlusion at L4–L5 and to a lesser degree at L3–L4. The appearance of a "canal full of roots" suggests that the vertebral canal is developmentally small.

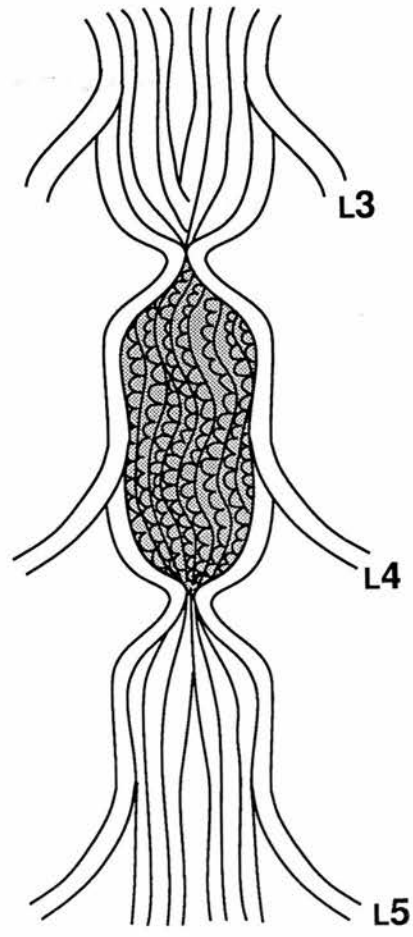

Figure 2. Diagram to show how all the roots of the cauda equina will be congested with venous blood between two levels of low-pressure occlusion of the central canal.

This hypothesis is compatible with experimental studies. A single level compression of 10 mm Hg in a porcine cauda equina model had little effect on the function, but a two-level compression of 10 mm Hg caused marked reduction of blood flow by 64%, 9 and there was significant reduction in protein transport and nerve conduction.[4] A two-level compression below arterial pressure is also supported by the myeloscopy studies, which show congested cauda equina in claudicating patients.[5]

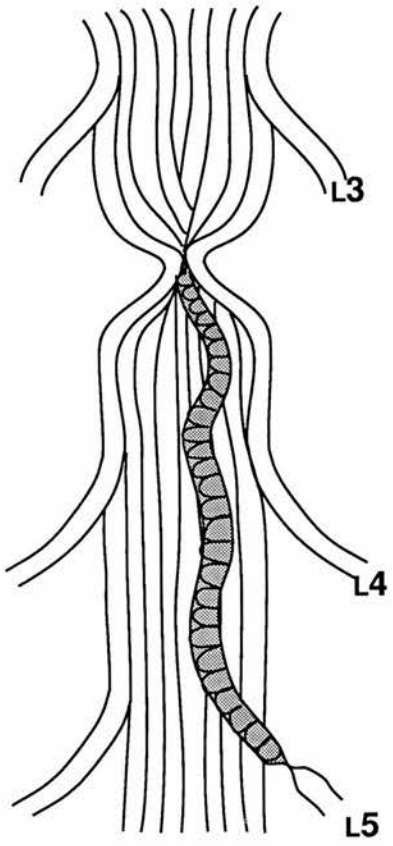

Figure 3. Diagram to show that a single level of central canal stenosis and a more distal level of root canal stenosis will cause congestion in a single nerve root.

A two-level stenosis hypothesis must include at least one level of central canal stenosis, but the second level of stenosis could either be in the central or in the root canal. Two levels of central stenosis will cause venous congestion in all the roots of the cauda equina between the two blocks. However, central stenosis at one level and bilateral root canal stenosis at the lower level will congest only two roots. Proximal central stenosis and distal unilateral root canal stenosis will produce single root congestion (Figure 3). This would explain why the symmetrical displacement in a degenerative spondylolisthesis is usually associated with bilateral and not unilateral claudication. The degenerative process is symmetrical, with central stenosis at one level and bilateral root canal stenosis at a more distal level. With degenerative lumbar scoliosis, however, the asymmetrical degenerative process is more likely to produce root claudication.

8. If venous pooling of the nerve roots of the cauda equina between two levels of low pressure stenosis is responsible for the symptoms of neurogenic claudication, one has to ask why are symptoms usually not present at rest but only when walking. One might argue that the block pressure at each level of stenosis will increase with the dynamic activity of walking. There will be local vasodilatation of the radicular arteries in response to exercise. Exercising the single limb of a mouse will produce vasodilatation in the ipsilateral region of the spinal cord. Blood flow in the nerve root is also increased with peripheral nerve stimulation. One might expect, therefore, that the arteries of the cauda equina will dilate with exercise, and if space is already at a premium, the stenosis block pressure will rise to a critical level.

Other features associated with walking will tend to increase the block pressure. Movement in the sagittal plane alters the epidural pressure at the site of stenosis, being above normal pressure even in flexion (15–18 mm Hg) and greatly above venous pressure in extension (80–100 mm Hg).[8] Segmental rotation associated with walking might also significantly affect the root canal where the degenerate capsule of the facet joint limits available space for the nerve root complex. In addition, the increased venous return from the exercising lower limbs will be accompanied by engorgement of the pelvic veins and Batson's venous plexus, reducing the available space for the cauda equina. Extra dural venous engorgement will then contribute to the block pressure.

There may be some patients with stenosis pressures at rest below the venous pressure, which rise above the venous pressure with exercise. However, one would expect that there will be some patients with multiple level stenosis who will have block pressures at rest above the venous pressure but not have leg symptoms at rest.

9. We have reported how there is considerable arterial vasodilatation of the porcine cauda equina to electrical stimulation and that this vasodilatation response is impaired in the presence of a two-level low pressure compression.[1] We have observed that in the response to stimulation and in the absence of any compression, there is an arterial vasodilatation producing 200% increase in the blood flow of the cauda equina, and this is maintained over 30 minutes (Figure 4, 5). However, in the presence of a double level low pressure compression, the increase in blood flow after stimulation is less pronounced and is maintained only for a few minutes (Figures 6, 7). The blood flow then rapidly falls to approximately 60% *below* the resting level. There is then a failure of nerve conduction.

If this is analogous to the chronic situation of multiple level spinal stenosis, it may explain the symptoms of neurogenic claudication.

A failed arterial response is compatible with the observation that patients with neurogenic claudication tend to be in the arteriosclerotic age group and that peripheral vascular disease and neurogenic claudication often coexist. Arteries that are already abnormal may be less labile, especially in the presence of venous pooling.

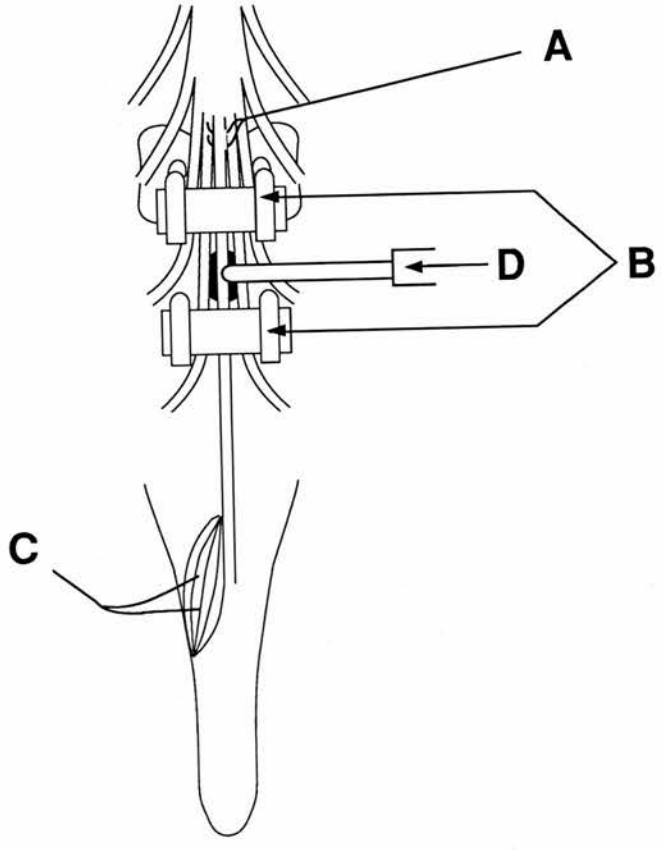

Figure 4. Diagram to show how the exposed porcine cauda equina is stimulated (**A**) proximal to a two-level low pressure occlusion (**B**), monitoring the tail muscle electromyographic activity (**C**) and the blood flow (**D**) in the cauda equina between the two levels of occlusion.

■ Management

Counseling

Patients with neurogenic claudication are either offered surgical decompression or are advised to live with their symptoms. If the disability is not too severe or if surgery is contraindicated, simple reduction of activities or alteration of lifestyle together with Back School instructions may enable the patients to live within their limitations. Once the syndrome is well established, however, conservative management rarely improves the quality of life.

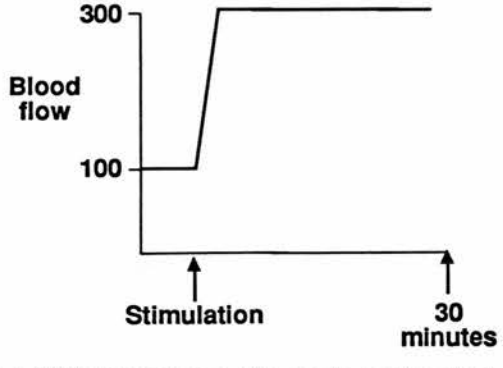

Figure 5. Without occlusion, the cauda equina blood flow increased by 200–300% when stimulated electrically. High flux and tail muscle electromyographic activity was maintained for 30 minutes.

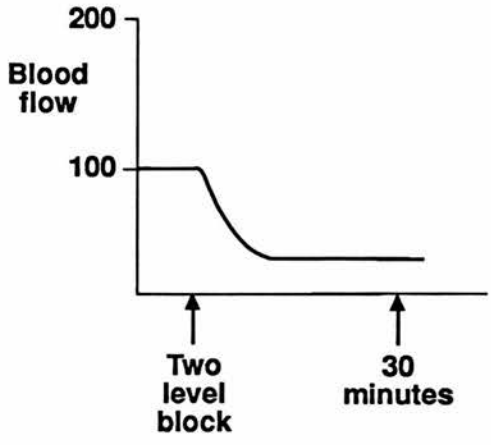

Figure 6. In the presence of a two-level compression of 10 mm Hg, blood flow fell to 35–40% resting level.

Calcitonin

There is circumstantial evidence that calcitonin is beneficial for approximately 40% of patients with neurogenic claudication. This drug can relieve the paraparesis of patients with spinal Paget's disease.

Many patients with Paget's disease and spinal stenosis, besides losing the Paget's pain, also find that with calcitonin their walking improves suddenly and dramatically. However, stenotic patients without Paget's disease can also dramatically improve their walking distance with a short course of calcitonin.[6] Calcitonin engenders a sense of well-being, and undoubtedly some patients experience a placebo response. Some become almost euphoric at their ability to walk unlimited distances again. The mechanism of a response is uncertain,

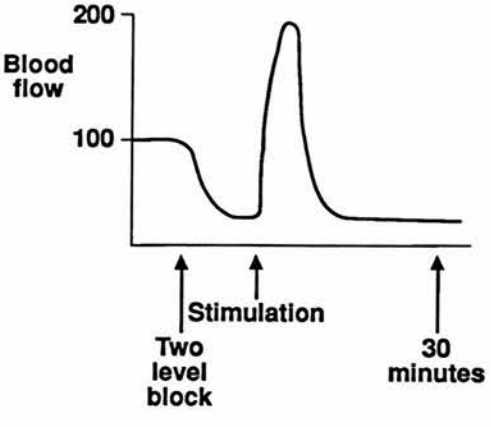

Figure 7. When a two-level compression was applied blood flow fell. The proximal cauda equina was stimulated with an increase in flux to 200% above resting level. This was only a short-lived response, with flux falling to below resting level, and with a failure of electromyographic activity in the tail muscles.

and a controlled randomized trial has not confirmed that the response is organic.

However, it is a useful first line of treatment, and responders avoid a surgical decompression.

Surgery

When recommending surgery, the clinician must adopt a different philosophy from that used for many other orthopedic problems. The patient with an arthritic hip, for example, is likely to have increasing disability, but neurogenic claudication is either present or absent. Once established, it tends not to progress, and when surgery is offered, it is for current disability and not to avoid future problems. No proof of severe deterioration is found in untreated patients, and observation for 2–3 years is an acceptable alternative to surgery. However, troublesome claudication symptoms seriously affecting lifestyle are generally relieved by surgical decompression. Most patients are immediately impressed with the improved sensation in their legs and are soon walking long distances. There may be some relapse after a laminectomy, when a membrane of fibrous tissue develops over the posterior dura, and the walking distance again becomes reduced. Some suggest that the results deteriorate with time, but others report good long-term results. Advanced age is no contraindication to the decompression, which will often improve the patient's quality of life. However, most operative series have a hard core of failures, and there are few patient characteristics that will predict outcome.[10]

The surgical decompression must be adequate. There are no clear guidelines to identify the significant stenotic levels. The clinical impression at the time of surgery is probably the best guide, although somatosensory-evoked potentials may have a place in determining the extent of necessary decompression. There is no longer a requirement to remove all the lamina. Retaining part of the lamina to maintain stability and reduce dead space is compatible with an adequate decompression.

If there is degenerative spondylolisthesis, it is essential not to unnecessarily increase the instability of that segment. Postoperative displacement is unusual even with wide decompression, provided there is already marked degenerative change, but one should be cautious if degeneration is minimal. The integrity of the apophysial joints should not be unduly disturbed, although the medial third of the joint can be removed and the facet undercut. It is necessary to form a decompression wide enough to ensure a completely free dura but not so wide as to produce either instability or such a shallow spinal gutter that a laminectomy membrane will soon compress the dura to a ribbon. Provided that there is not a degenerative spondylolisthesis, it is legitimate to sacrifice the major part of the apophysial joint on one side to obtain satisfactory decompression and not jeopardize stability. Decompression should be accompanied by a posterolateral spinal fusion when in a degenerative spondylolisthesis there is minimal degenerative change and usually when stenosis complicates structural lumbar scoliosis.

A double level hypothesis has surgical implications. If the symptoms result from a two-level stenosis, it is necessary to decompress only one of the levels to relieve the claudication. However, it is inadequate to decompress only the central canal of the most stenotic segment if root canal stenosis at this level is responsible for the distal stenosis, and a significant proximal central canal stenosis is still left untreated.

In summary, the symptoms of neurogenic claudication are probably associated with developmental spinal stenosis and secondary multiple level degenerative change. At low pressures, this will produce venous pooling of one or several roots of the cauda equina. In the arteriosclerotic age group, the arterioles of the nerve root fail to maintain a vasodilatation response to exercise when there is venous engorgement. This failed arterial response is associated with failure of nerve conduction producing tiredness, weakness, heaviness, and discomfort in the lower limbs when walking. As the patients stop, nerve function temporarily recovers to permit a further period of walking.

This hypothesis explains why patients with neurogenic claudication tend to reach a plateau of disability and then not deteriorate further. They can always walk a short distance. They rarely develop serious paraparesis or "go off their feet." For this reason, although surgery will often dramatically relieve the symptoms, a conservative approach is reasonable.

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Address reprint requests to

Richard W. Porter, MD
Director of Education and Training
Royal College of Surgeons
Nicolson Street
Edinburgh, EH8 90W
Scotland

SPINE Volume 21, Number 23, pp 2705–2708

Development of the Lumbar and Sacral Vertebral Canal *in Utero*

27

Traian R. S. Ursu, MD, Richard W. Porter, MD, FRCSEd, FRCS, and Visvan Navaratnam, PhD

Study Design. This study analyzed the development of the lumbosacral vertebral canal and dural sac in human fetus. A collection of fetuses and embryos was used to assess the development of different parameters of the spinal canal.

Objectives. The data were analyzed for the dynamics of development and also compared with mean adult spinal parameters.

Summary of Background Data. Transversely sectioned specimens and nonsectioned specimens free of abnormalities were selected from the Boyd Collection of human embryos and fetuses.

Methods. The sections were photographed alongside a micrometric scale, and the nonsectioned specimens were scanned by magnetic resonance imaging. The films were computer analyzed for spinal and dural parameters. The error of the measurements was assessed.

Results. The most rapid growth period of the spinal canal parameters is between 18–36 weeks' gestation. After 30 weeks of intrauterine life, the upper lumbar canal grows faster than the lower lumbar region. The distal end of the dural sac begins to rise from S5 after 14 weeks.

Conclusions. At the end of intrauterine growth, the interpedicular diameter of the spinal canal from L1 to L4 is 70% of the adult size, however, at L5, the canal is only 50% mature at birth. Therefore, if there is growth impairment in early infancy, the upper lumbar region is partially protected in contrast with the L5 level. [Key words: development, dural sac, extradural space, human fetuses, spinal cord, vertebral canal] **Spine 1996;21: 2705–2708**

A small lumbar vertebral canal is a significant risk factor in several back pain syndromes.[1,8,13,16] The canal's development is of clinical significance. It reaches maturity surprisingly early in life, the cross-sectional area at L3 and L4 being of adult size by 1 year of age.[2,3,9,11,12] However, the pattern of intrauterine growth is unknown. This report describes the early development of the lumbosacral vertebral canal.

From the Department of Orthopaedics, University of Aberdeen, Aberdeen, United Kingdom.
Supported by a grant from the National Back Pain Association.
Acknowledgment date: November 30, 1995.
Acceptance date: February 22, 1996.
Device status category: 1.

Materials and Methods

We examined the Boyd Collection of 67 human fetal specimens from the Department of Anatomy at Cambridge University. We identified 44 specimens of over 21 mm crown–rump length (over 8 weeks' gestational age[5]) that included the lumbar and sacral spines. They had been transected transversely. The slice thicknesses were 6–15 μm. Most had been stained with the Boyd method. In addition, five older specimens of 164, 174, 192, 196, and 300 mm in length were examined by magnetic resonance imaging (MRI).

The vertebral levels were identified from the position of the last thoracic vertebra and the 12th rib and also from the regional embryonic anatomy.[5,18] Because of the sequential sectioning, the more caudal levels could readily be identified. The section taken at the most cranial aspect of the pedicle was selected for each vertebral level. These were photographed on black and white film, superimposing a micrometer scale and using magnifications between 0.8 and 2.5.

The upper lumbar level slices were in general true transverse sections, with the discs having a symmetric appearance. More distally, the sections tended to become oblique because of the specimens' kyphotic shape. We used a method to correct errors of measurement. In the truly transverse sections, we identified a constant between the anteroposterior and lateral diameters of the vertebral bodies (0.887 ± 0.013). From this, we were able to calculate the angle of the section and used the correction factor for the various transverse parameters to be measured.

The images were analyzed by computer, measuring the vertebral canal area (A), perimeter (P), mid-sagittal diameter (AP), and interpedicular diameter (IP); and the dural sac area (DA), perimeter (DP), mid-sagittal diameter (DAP), and transverse diameter (DIP).

Transverse sections of the brain were available from 17 specimens to record the maximum fronto-occipital diameter.

The five older specimens in which the vertebral canal was examined by MRI were measured against a standard scale, recording the same canal and dural parameters.

The error of measurement was assessed by recording the parameters on 10 occasions in one embryo. The standard deviation was between 0.009 and 0.179 mm, and the standard error was between 0.003 and 0.060 mm.

Results

The data are plotted on three-dimensional graphs for each of the parameters. The vertebral canal area is shown in Figure 1A from specimens from 20 to 300 mm and in Figure 1B for specimens from 20 to 150 mm on a higher

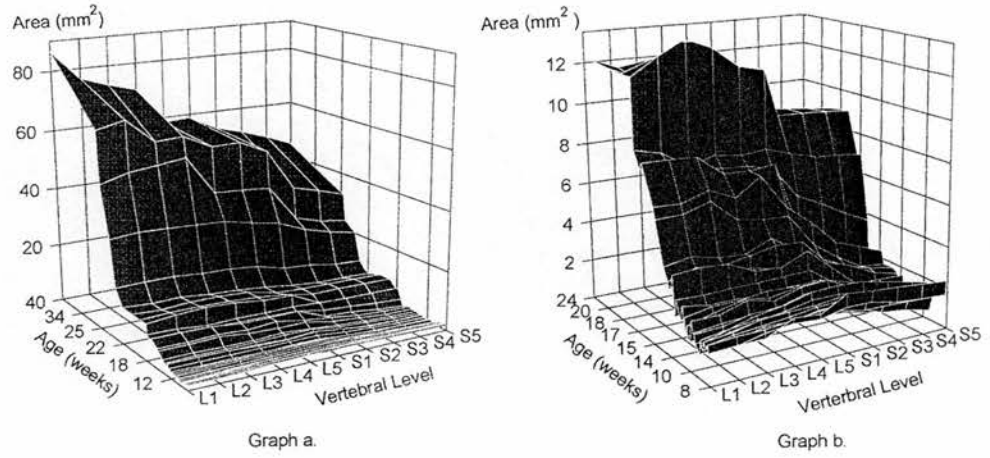

Figure 1. Development of the spinal canal area. **A,** From 8 weeks to birth. **B,** From 8 to 21 weeks.

scale. The size is converted to weeks of gestation for ease of reference.[5] Up to 14 weeks, the area of the canal is similar throughout the lumbar and sacral spine, but then there is differential growth, with the lumbar canal growing faster than the sacral canal. After 30 weeks, the upper lumbar canal grows faster than in the lower lumbar region. This pattern of growth is similar for the other parameters measured.

The mid-sagittal diameters at different fetal ages are recorded in Figure 2 for each lumbar level, showing that the period of maximum growth is between 18 and 36 weeks, with a greater differential between upper and lower lumbar regions after 32 weeks.

The cross-sectional area of the dural sac is shown in Figure 3 from 8 to 40 weeks and 8 to 20 weeks, respectively. The distal end of the dural sac begins to rise from S5 to the upper sacral region after 14 weeks. The lumbar dural sac grows faster than the upper sacral sac from 16 weeks, particularly at the upper lumbar levels.

To determine the size of the extradural space, the dural sac was subtracted from the size of the vertebral canal at each level (Figure 4). There is little extra dural space at L1 throughout intrauterine life, but there is a relative increase in this space distally, particularly after 24 weeks.

The mean diameters and areas of the vertebral canal at birth are compared with the adult measurements[10] (Figure 5). The mid-sagittal diameter at L1–L4 is 70% of adult size at birth. The upper lumbar canal matures earlier than L5.

The anteroposterior diameter of the brain showed a close correlation with the mid-sagittal diameter of the vertebral canal at L1 ($r = 0.99$) in the 17 specimens measured.

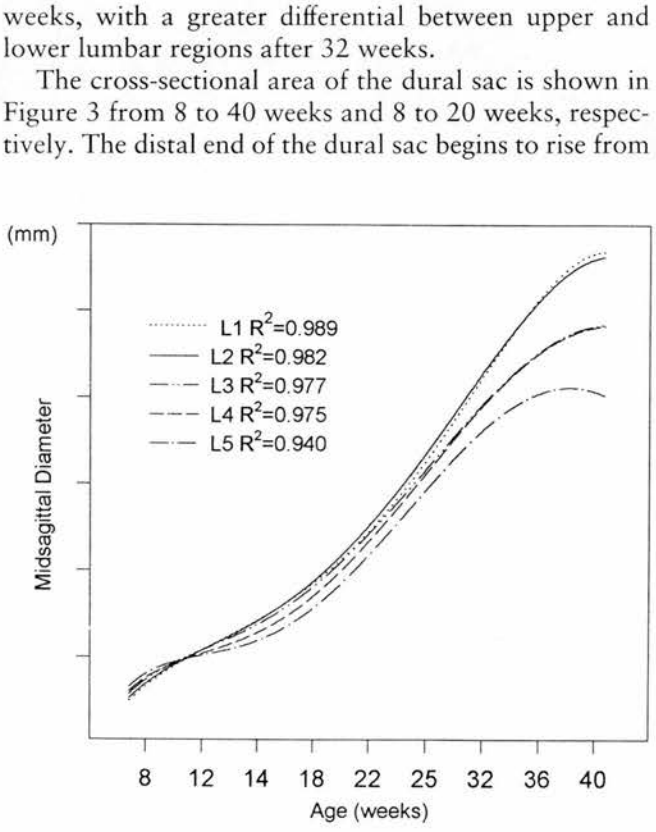

Figure 2. Development of the mid-sagittal diameter of the spinal canal in fetal specimens analyzed with regression slopes.

■ Discussion

The growth of the lumbar vertebral canal is of interest to clinicians because some of the more disabling back pain problems are related to developmental spinal stenosis. The vertebral canal reaches maturity early in life.[4,6,7,15–17] The canal at L3 and L4 approaches adult size by 1 year of age, although L5 continues to grow until 5 years of age.[11] Factors that impair the canal's growth may leave a permanently small canal, and these must presumably act very early in life. For this reason, the growth curves of the canal *in utero* are of importance.

The Boyd Collection of fetal specimens, carefully documented and well preserved, provides an unusual opportunity to assess vertebral canal growth. The relatively few older specimens were supplemented with five fetuses examined by MRI.

Previous data about the growth of the vertebral canal have been limited to archaeologic specimens, the best being from the Spittalfield Collection in the Natural His-

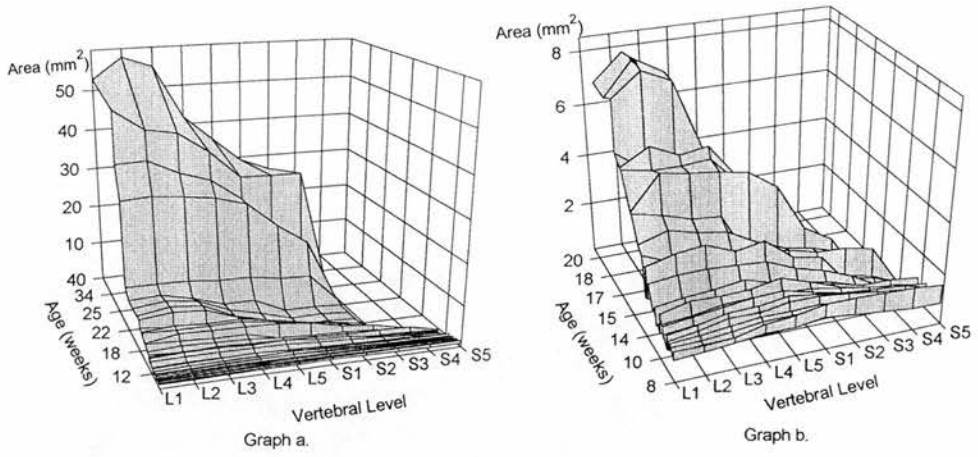

Figure 3. Development of the dural area. **A,** From 8 weeks to birth. **B,** From 8 to 20 weeks.

tory Museum (London), where the exact date of birth and death were known.[11,14] However, there were no specimens younger than 1 year of age. We have been able to show from the Boyd Collection that at the end of intrauterine growth, the interpedicular diameter of the canal at L1–L4 is 70% of the adult size, the remaining 30% being accomplished in the first year of life. At L5, however, the canal is only 50% mature at birth, the remaining growth taking place in the first 5 years of life. Thus, if the canal's intrauterine growth is impaired, it will affect mainly the canal at L1–L4, whereas L5 has more potential for catch-up growth. If, on the other hand, there is growth impairment in early infancy, the upper lumbar region is protected partially, having already accomplished most of its growth *in utero*.

This selective growth between L5 and the upper lumbar levels might have implications for syndromes like neurogenic claudication, where spinal stenosis frequently is in the mid-lumbar region. In contrast, in symptomatic lumbar disc protrusion, the lesion tends to be at L5–S1 or L4–L5, where a small canal at L5 significantly increases the risk of a protrusion becoming symptomatic.[8,11,13,14]

We have been able to show that the most rapid period of growth for all the lumbar levels is between 18 and 36 weeks. Factors that impair growth during this period may have permanent effects. Although programming for growth could occur earlier, it is in this period that fetal nutrition may have a significant[12] effect on canal size.

The development of the dural sac, its distal end rising from S5 to S2 and continuing to increase at L1 and L2 after 14 weeks, is probably related to the rising conus and will be the subject of additional communication.

The close correlation between the size of the canal at L1 and the anteroposterior diameter of the brain is relevant to previous work, which suggested that the verte-

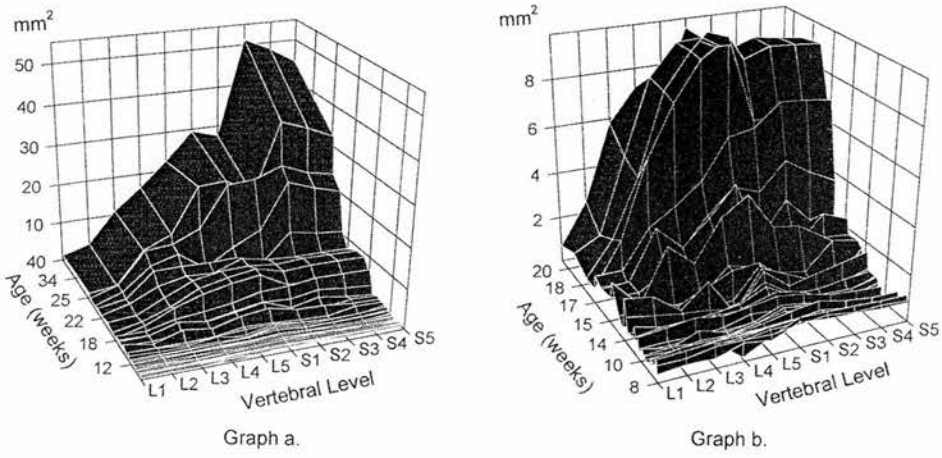

Figure 4. Extradural space calculated as the difference between the area of the vertebral canal and the area of the dural sac. **A,** From 8 weeks to birth. **B,** From 8 to 20 weeks.

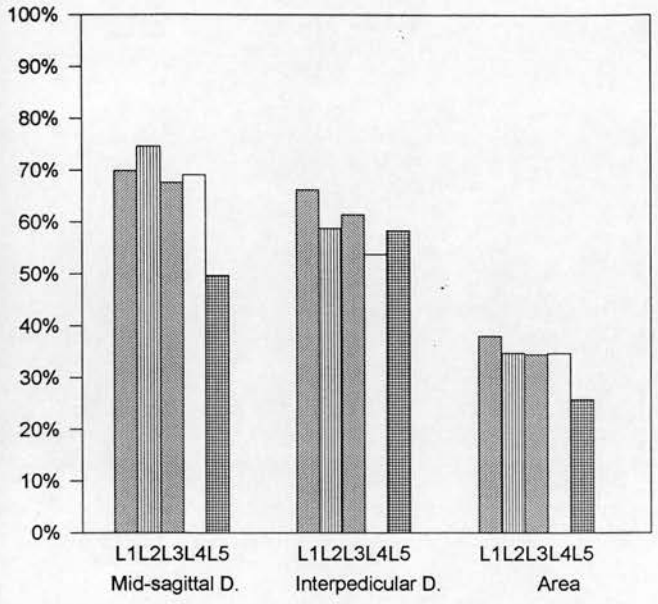

Figure 5. Vertebral canal parameters at birth compared with mean adult size vertebral canal parameters.

bral canal is a permanent marker of neural development.[3,12] Interest in the canal's growth therefore may have significance beyond the problem of back pain.

Acknowledgments

The authors thank Mrs. Montague (NBPA), Mr. Jessell (NBPA), Mr. Wooldridge (NBPA), Dr. Frank Smith (MRI Department, Aberdeen Royal Infirmary), and the staff from the photographic laboratory of the Department of Anatomy (Cambridge University).

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Address reprint requests to

Traian R. S. Ursu, MD
Department of Orthopaedics
Polwarth Building
Medical School
Foresterhill, Aberdeen AB9 2ZD
United Kingdom

SPINE Volume 22, Number 16, pp 1805–1810

Significant Antenatal Factors in the Development of Lumbar Spinal Stenosis

Tibor Papp, MBChB,* Richard W. Porter, MD, FRCS, FRCSE,* Catherine E. Craig,* Richard M. Aspden, PhD,* and Doris M. Campbell, MD, FRCOG†

28

Study Design. Adverse factors during pregnancy may permanently stunt the growth of the spinal canal. Subsequently, even in an optimal environment the canal cannot catch up in growth with the trunk and long bones because of its early maturation. The degree of retardation in canal size depends on the severity and timing of the adverse effect. The catch-up growth of the long bones mask the narrow canal, because the latter does not have growth potential, resulting in an adult of sufficient height and good proportions, but with a canal at risk for stenosis.

Objectives. To investigate the influence of the antenatal environment on the growth of the lumbar spinal canal.

Summary of Background Data. To date, little is known about the effects of an adverse environment on the growth of the spinal canal, and no data have been reported on antenatal influencing factors.

Methods. Lumbar magnetic resonance imaging scans from 58 patients were examined. Dimensions of the central spinal canals were measured by computerized image analysis and compared with the subjects' obstetric data from their mothers' pregnancies.

Results. The L3 canal was found to be the most sensitive to the influence of the examined factors. Gestational age was the most significant factor; if short, it resulted in small adult canal. Small placental weight, greater maternal age, primiparity, low socioeconomic class, and low birth weight were also found to be significant in affecting the growth of the canal.

Conclusions. An adverse antenatal environment does have a permanent, retarding effect on the growth of the lumbar spinal canal. [Key words: adverse environment, development, pregnancy, spinal canal, stenosis] **Spine 1997;22:1805–1810**

Little is known about factors affecting the growth and development of the lumbar spinal canal. Previous archaeologic studies indicated that the capacity of the canal is mainly determined before or very shortly after birth. The canal achieves its final, adult size in the proximal levels and more than 90% of its full size at the level of L5 by 1 year of age with regard to the cross-sectional area and mid-sagittal diameter.[11] These findings suggest that the period of growth preceding birth can play a significant role in determining the adult canal size. Varughese and Quartey[21] suggested that developmental stenosis is inherited or, at the least, genetic factors cannot be ignored. Postacchini et al[15] also found that the dimensions of the spinal canal may be regulated by genetic factors. This hypothesis seems to be consistent with morphologic studies showing that the mean diameters of the canal and the lower normal limits vary in different races as well as different populations of the same race.[7,16] The authors, however, imply that there can also be critical external factors that override regulatory channels of development and cause growth disruption of the vertebral canal. There are several adverse physiologic stressors known to slow or stop bodily growth, including malnutrition, infection, and psychogenic factors.[1] The degree of impairment depends on the type, timing, and severity of the adverse factor. Those features that are growing the fastest at the time of growth disruption are the most affected. It has been shown that "catch-up" growth depends primarily on the amount of growth potential in a particular feature before the time of growth interruption. Longitudinal studies have indicated that catch-up growth in head circumference cannot occur unless the environmental perturbation is ameliorated by 2 years of age; this is because the neural cranial size in infancy is already very near its adult size.[19] The trunk and the limbs, however, can recover from growth disturbance and recoup lost growth because they continue to grow until adulthood. The length of the trunk appears to be more sensitive to growth disruption than that of the limbs.[10] This suggests that disturbance caused by an adverse early environment is more pronounced in the spine than in the long bones.

Clark et al[4] investigated the effects of environmental factors on the growth of the spinal canal in childhood. The authors tested the hypothesis that infant malnutrition can produce abnormally small adult spinal canal diameters. They found that there was a significant, although slight, difference in anteroposterior canal size between groups consuming different diets. This dimension

From the Departments of *Orthopaedic Surgery and †Obstetrics and Gynaecology, University of Aberdeen, Aberdeen, United Kingdom.
Acknowledgment date: March 3, 1996.
First revision date: September 30, 1996.
Acceptance date: January 2, 1997.
Device status category: 1.

Table 1. The Combined Influence of the Examined Factors on Dimension of Different Lumbar Levels

	Lumbar Level											
	L3	L3	L3	L4	L5	L4	L5	L4	L3	L5	L4	L5
Dimensions	PM	CS-A	IP	PM	PM	MS	IP	CS-A	MS	CS-A	IP	MS
Correlation (r^2)	0.43	0.38	0.32	0.29	0.27	0.21	0.21	0.19	0.17	0.14	0.13	0.13

PM = perimeter; CS-A = cross-sectional area; IP = interpedicular diameter; MS = midsagittal diameter.

was smaller in the protein-deficient group. After also analyzing vertebral heights and tibial lengths, lumbar anteroposterior diameters appeared to be the best indicator of disrupted neonatal growth. It was suggested that this originated from the fact that much of the growth of the anteroposterior diameter is completed before birth. Structures with longer periods of postnatal growth (e.g., vertebral height, tibial length) could reverse the effects of early growth disruption. Consequently, these statural components can mask the presence of smaller canal diameters. The authors stated that canal size is a most powerful tool in the assessment of prenatal and neonatal growth disruption. It has also been shown that there is a significant correlation between adult serum levels of thymosin-α_1, which is a good predictor of immune maturity, and the anteroposterior diameter of the T7 vertebra.[5] The growth curves of the thymus and the central nervous system are similar to that for neuroosseous development, and they are likely to be similarly affected by an adverse early environment. In an immunologic study, Chandra[3] reported that in addition to genetic factors, environmental factors play an important role in influencing the developing human immune system *in utero*. Adverse factors that impair fetal growth also hinder immunologic maturation. Individuals who are small for gestational age show persistent immunologic impairment for several months, even years after birth. Because the growth schedules for vertebral canal size and the immune system are similar, this finding may have significance for determinations of the capacity of the lumbar central canal.

There have been few attempts to investigate the role of the antenatal environment in the development of a narrow canal. The objective of this study was to examine the correlation between the dimensions of the adult lumbar spinal canal and factors of the fetal environment before birth. It was hypothesized that an adverse factor acting on the fetus may result in a developmentally stenotic spinal canal in adulthood.

■ Materials and Methods

Horizontal magnetic resonance imaging sections of lumbar vertebrae were examined from 58 adult individuals, aged 18 to 43 years, who underwent scanning because of low back pain. The radiologic reports described disc protrusion in most cases (87%). The rest of the scans showed no abnormality. In none of the cases was bony spinal stenosis the diagnosis. Bias toward a diagnosis of stenotic canal might be considered because these patients were investigated for back pain.[13] To check this possibility, the dimensions of the lumbar canal were compared with those of the general population measured in a previous study. However, the measurements of the current population were not found to be significantly smaller. The mid-sagittal diameter, interpedicular diameter, cross-sectional area, and perimeter were measured by computerized image analysis to the nearest 0.1 mm. The measurements were taken at the pedicular level because only at this section can the osseous outlines of the canal be seen circumferentially on a magnetic resonance image. In most cases, only the L3–L5 levels were scanned for clinical reasons, and therefore, only the canals at these vertebrae were measured in this study.

Data regarding pregnancies were obtained from the Aberdeen Maternity and Neonatal Databank, which contains detailed information about pregnancies for the locally born population since 1950. The following antenatal factors were investigated: gestational age, placental weight, birth weight, parity, the number of the pregnancy, parental socioeconomic class, maternal height, maternal smoking habits, vaginal bleeding (both early, threatened abortion, and late, antepartum hemorrhage), hypertensive disease, anemia, and infection. Selection of the examined factors was determined by availability. Socioeconomic classes were classified according to the Registrars' General Classification using five groups: I–II, professionals; III, skilled nonmanual and manual workers; IV, partly skilled manual workers; V, nonskilled manual workers; and VI, others, such as armed forces. The standardized birth weight score (SBS), which allows comparison between subjects and the total population after adjusting birth weight for sex, parity, and gestational age, was used.[2] Infants were regarded as "small for gestational age" (SGA) if their SBS was under −2 standard deviations. Those who were between −2 and +2 standard deviations were "appropriate for gestational age" (AGA). The SBS of the examined population was found to be within ±1 standard deviation of that of the total.

To compare canal sizes and the effects of obstetric factors, statistical analyses were performed. As a first step, multiple linear regression was used to establish how much variance in a canal dimension can be explained by the collected independent variables. However, this type of analysis disregards the independent effects of each of the obstetric factors and examines only their combined influence. Thus, stepwise multiple regressions were performed to select factors that are significant and independent from the others. Finally, some of the variables were divided into groups according to well accepted, international "cut-off points," such as: 1) those of subjects born as "low-birthweight infants" (<2,500 g); and 2) those of subjects born with weights over 2,500 g. In these cases, Student's *t* test was performed, keeping in mind that this test disregards tendencies and may be influenced by other factors. A *t* test was also used in simple comparative cases, such as smoking (1, smoker;

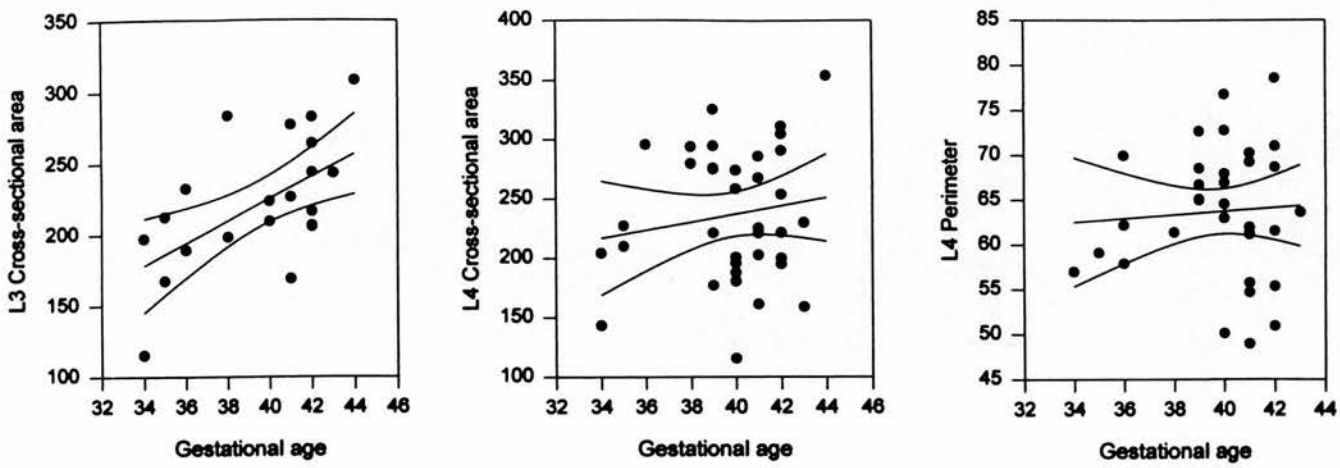

Figure 1. Spinal canal size was significantly smaller in adults born at short gestational ages. The time of gestation had an influence on canal size independent of all other antenatal factors.

2, nonsmoker). To analyze possible differences of subjects born in different socioeconomic classes, analysis of variance was performed.

■ Results

It appears that the examined factors have the greatest combined influence at the L3 level, followed by L4, then L5. The perimeter was found to be the most sensitive area to changes in environmental factors. A considerable 43% of the variance of the L3 perimeter can be explained by these factors (Table 1).

When examining the independent effects of the factors, the period of gestation was found to be the most significant. A short gestational period was correlated with smaller canal size. Separate from other effects, this variable alone accounted for 23% of the variance of the L3 cross-sectional area ($r^2 = 0.23$, $P < 0.05$). Gestation was also found to be significant for L4 cross-sectional area ($r^2 = 0.14$, $P < 0.05$) and L4 perimeter ($r^2 = 0.13$, $P < 0.05$; Figure 1). Older maternal age and low placental weight similarly affected the capacity of the canal, both accounting for 18% of the variance of the L5 perimeter ($r^2 = 0.18$, $P < 0.05$; Figure 2).

Other environmental variables did not enter the stepwise regression test at the 95% confidence level—that is, they did not have an independent effect on canal size, but only in combination with the other factors.

Preterm birth has been defined as birth after gestation of less than 37 weeks. The canal measurements of those born before 37 weeks of gestation were compared with those born at or over 37 weeks. The first group of adults had significantly smaller L3 ($P = 0.03$) and L4 ($P = 0.047$) cross-sectional areas and smaller L4 perimeters ($P = 0.017$).

One of the other definitions of prematurity for a newborn is having a birth weight below 2,500 g. After dividing the canal measurements into those of subjects born prematurely and those weighing over 2,500 g at birth, low-birthweight individuals were found to have canals with significantly smaller L4 interpedicular diameters than those with greater birth weights ($P = 0.039$). Taking the birth weight for gestational age into consideration, which is expressed by SBS, the canal measurements of SGA subjects were selected and compared with those of AGA ones. The statistical significance increased

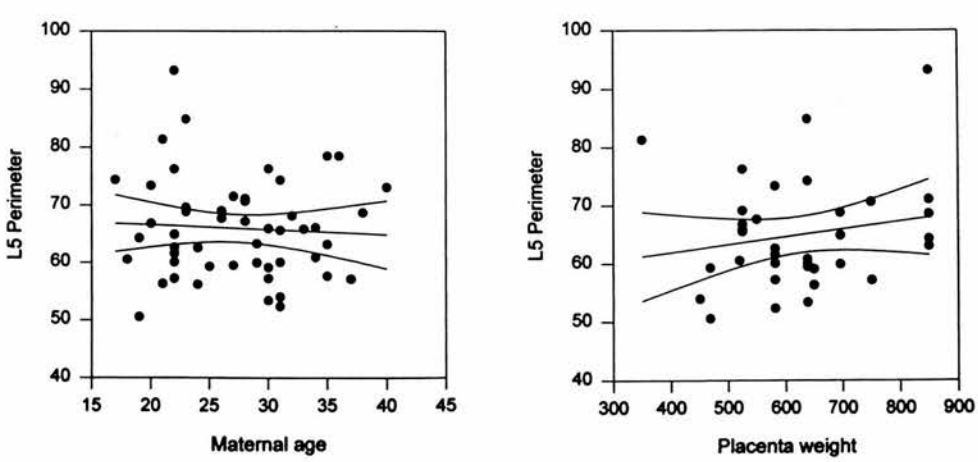

Figure 2. The effect of increased maternal age and decreased placental weight was less significant, but their correlation with canal size also showed a tendency. Those with older mothers and smaller placenta weight tended to have smaller canals.

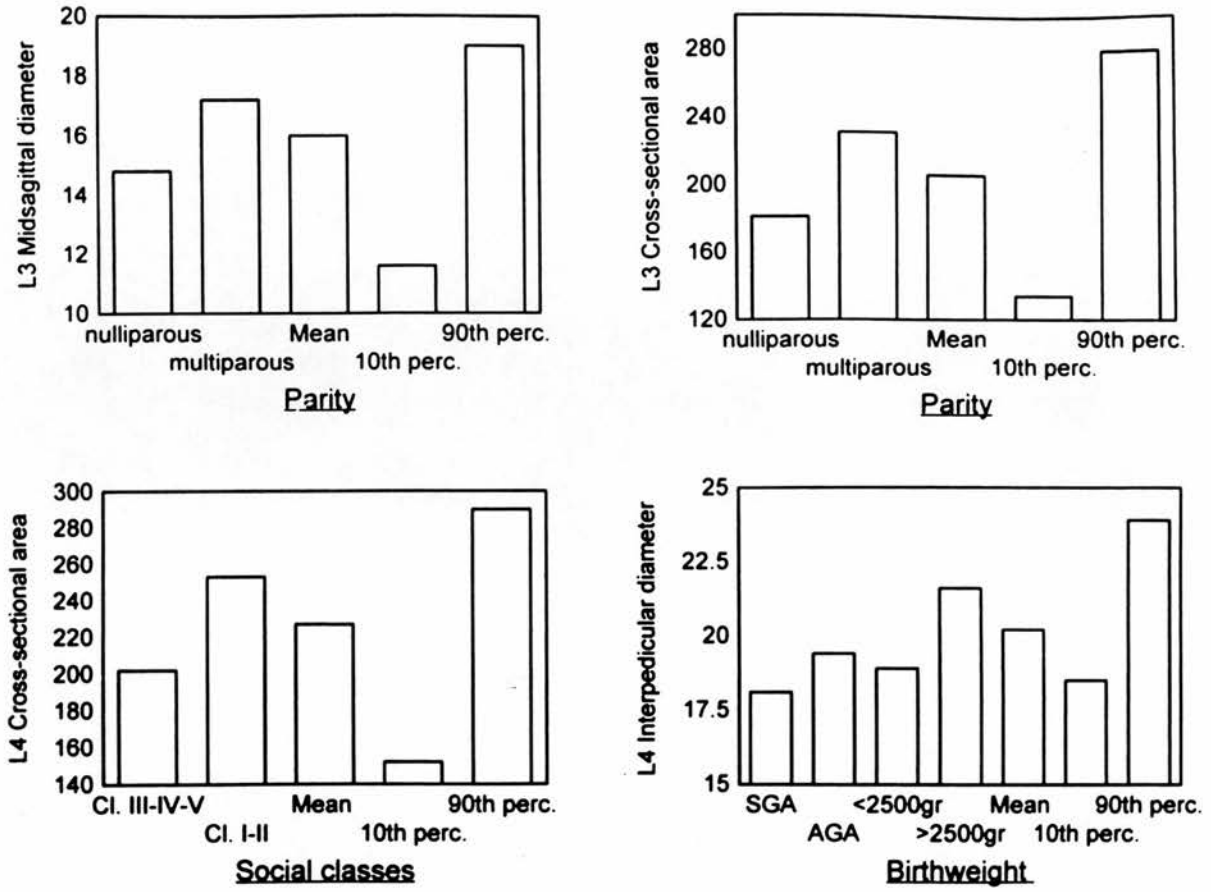

Figure 3. Parity, socioeconomic class, and birth weight were also found to be significant when they were divided into groups.

when those born as SGA infants in the low-birthweight group were compared with AGA infants ($P < 0.035$; Figure 3).

None of the socioeconomic classes showed an independent effect on canal size. However, when individuals were divided into two groups (Group 1, those born in classes I–II; Group 2, those born in classes III–VI), subjects from the lower socioeconomic classes had significantly smaller L4 cross-sectional areas ($P = 0.029$) than those from the professional classes (Figure 3).

Individuals born from nulliparous mothers (who had no other living children) had significantly smaller L3 mid-sagittal diameters ($P = 0.022$) and L3 cross-sectional areas ($P = 0.026$) than those born from primiparous or multiparous women (with one or more living children; Figure 3).

The remaining factors, maternal hypertension, vaginal bleeding, infection, height, anemia, number of the pregnancy, and maternal smoking habits, did not show any significant influence on the size of the lumbar canal.

■ Discussion

This study has shown that a number of intrauterine factors were associated with a small adult lumbar spinal canal. The most significant of these was the length of gestation, which, if short, adversely affected the growth of the canal. A preterm birth may be caused by many factors—maternal, placental, and fetal; unfortunately, however, the etiology can be identified in only a small number of early deliveries, and the problem is treatable only in a small proportion of these. In the current population, no significant correlation could be ascertained between the factors investigated and preterm birth. During the last 6 month of fetal life, and especially during the last trimester of pregnancy, emphasis is on fetal growth, on increase in size rather than increase in complexity. It has been shown that insofar as the nervous system is concerned, increase in cell number occurs predominantly before birth and in the first 6 months of neonatal life.[18] Preterm infants leave the optimal maternal environment during their period of rapid growth. Children who start life with a deficiency in brain growth may have permanent impairment in mental and physical performance.[9] If growth of the spinal neural elements and that of the spinal canal is linked, as is seen in the case of the brain and the neural cranium,[8] then the vertebral canal in premature infants will also have a significant growth disadvantage. The consequence is a small canal by the end of the process (Figure 4).

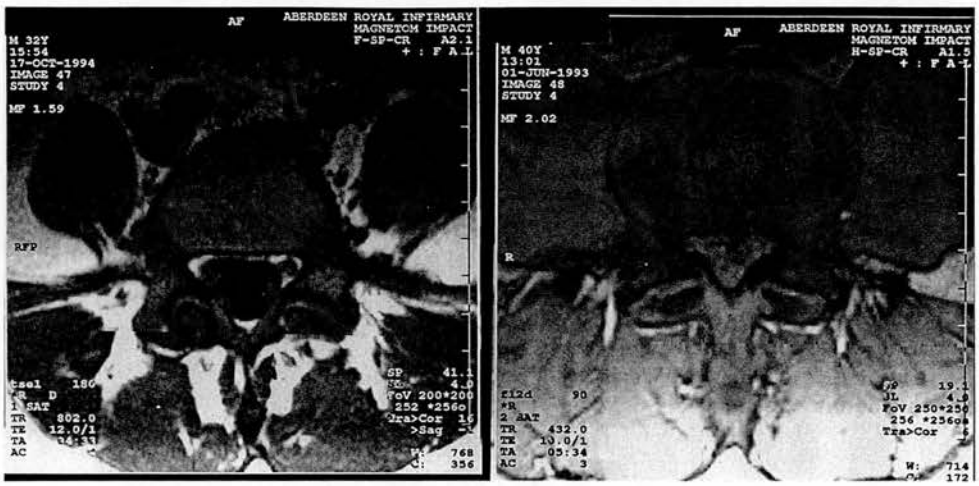

Figure 4 Vertebral canal of a subject who was born at term after 40 weeks of gestation **(left)**. Vertebral canal of a subject born prematurely, at 35 weeks' of gestation **(right)**; note the significantly smaller size of this canal.

Placental size is thought to be determined by the fetus and its need for nutrients and oxygen, but it is unlikely to be compromised unless considerable damage occurs. This study has shown an association between lumbar canal diameter and placental weight. Therefore, the lumbar canal may be more sensitive to changes in placental supply than to birth weight.

It is not clear what is the cause of the adverse influence associated with increased maternal age. A correlation between increasing maternal age and increased perinatal mortality has been reported.[17] A connection between general health status, the immune system, and the size of the canal has also been suggested.[14] Therefore, a negative effect of older maternal age on the development of the canal is conceivable.

Parity has also been shown to be significant in affecting canal size, with first-born subjects having significantly smaller canals than those from second or subsequent births. It has been shown that the birth weight of newborns increases from the first pregnancy to the second.[20] It is suggested that this is because the maternal system may adapt during while bearing the first child, and in subsequent pregnancies may be fully prepared, allowing enhanced fetal growth.

Bone development has been shown to be affected by adverse factors that cause low birth weight. In SGA infants, the total body bone mass measurements were only half the values measured in AGA infants.[12] It appears that the growth of the bones is severely hindered by factors that cause low birth weight. This may primarily or secondarily affect vertebral growth.

The influence of socioeconomic class on fetal growth has been well established.[6,22] Mothers of low socioeconomic status have a greater number of lower-birthweight infants than the professionals. Individuals starting life in the lower socioeconomic classes are at a significant disadvantage with regard to their general health. In addition, newborns encounter obstacles to adaptation to the extrauterine environment in a family of low socioeconomic status. It is therefore not surprising that the size of the lumbar vertebral canal, which achieves most of its final capacity by birth because of its early growth, is affected by parental socioeconomic status.

The results of this study show that the L3 vertebral canal was the area most affected by antenatal factors. It is of interest that the L3 and L4 levels are the most frequent sites for lumbar spinal stenosis. These levels are also the smallest ones in mid-sagittal diameter and cross-sectional area. Because the correlations with canal size were least significant at L5, and canal growth at this level does not fully mature until 6 years of age,[11] it is possible that an improved postnatal environment allows some catch-up growth at L5. However, at L3 and L4, which are fully grown by 1 year of age, the antenatal factors seem to be the most important. It is of clinical interest that disc protrusion occurs most frequently at L4–L5 and L5–S1. The development of disc symptoms depends partly on the size of the vertebral canal. For neurogenic claudication, however, the stenosis is most often at L3–L4 and L4–L5 levels.[13] One could postulate that the risk of both conditions might be increased by adverse intrauterine factors, but because there is a degree of catch-up growth at L5 in the first 6 years of life, postnatal development is also important, in addition to the epidemiology of symptomatic disc protrusion.

In conclusion, the *in utero* environment has a significant influence on the growth of the lumbar spinal canal. Early adverse factors may alter the genetically determined canal size, resulting in a developmentally narrow vertebral canal. Such a small canal may of course remain symptomless throughout life, but in the presence of other lesions, it can predispose to certain back pain syndromes. The results of this study suggest that the antenatal period is significant in the development of spinal stenosis, which provides potential for prevention.

Acknowledgments

The authors thank Marion Campbell, statistician, Health Services Research Unit, and Professor M. H. N.

Golden, FRCP, Department of Medicine and Therapeutics, University of Aberdeen, for their invaluable help.

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Address reprint requests to

Tibor Papp, MBChB
Department of Orthopaedics
University Medical School of Debrecen
98 Nagyerdie Krt.
4012 Debrecen
Hungary

J. R. Coll. Surg. Edinb., **43**, August 1998, 219—222

The Henderson Trust Lecture

29

The development of the vertebral canal and associated neurophysiological abnormalities*

The Henderson Trust Lecture was first delivered in 1924 on the subject of Phrenology. At that time it was thought that Phrenology might provide important answers to the study of brain function. Unfortunately, this was found to be a blind alley, and a relationship between the container and its contents was never established. However, this lecture today examines the same philosophy but in a different part of the neuroanatomy – the lumbar spine. We are assessing the relationship between the spinal contents and the vertebral canal container, and we may be surprised.

We shall first examine the anatomy of the lumbar vertebral canal and its normal variations. Secondly we shall consider how the size of the canal is related to certain neurological back–pain syndromes. Finally we shall examine the canal's development and how stenosis might be prevented.

The anatomy of the lumbar vertebral canal

(i) *The normal anatomy.* The vertebral canal is the bony container for the spinal cord, and below L2 for the cauda equina. It is arbitrarily divided into the central canal which contains the dura, and the root canals which extend laterally to the intervertebral foramen. The canal's perimeter is bony at the pedicular levels, but it is bounded by soft tissue at the level of the disc.

We have examined different archaeological populations, Roman and Anglo Saxon. Although the Roman canals were slightly larger than those of the Anglo Saxons, the relative change in size from L1 to L5 was similar in each population.[1] There was a gradual reduction in cross sectional area from L1 to L4 with an increase again at L5. The mid sagittal diameter followed the same pattern, but the interpedicular diameter gradually increased from L1 to L5. At the 5th lumbar level the 10th percentile of the mid sagittal diameter at L5 was 10 mm and at the 90th percentile 18 mm. Figure 1 shows an example of the variation in size and shape at L5.

(ii) *Trefoilness.* Of canals, 25% were trefoil in shape at L5, and this prevalence did not increase with age. The mid sagittal diameter in the trefoil canals was significantly smaller than in the unaffected canals.[2]

(iii) *Spina bifida.* The overall prevalence of spina bifida was 18%. The mid sagittal diameter at L4 and L5 and the cross sectional area at L5 proximal to spina bifida was found to be significantly larger than in unaffected spines.[3–7]

(iv) *Spondylolisthesis.* Isthmic spondylolisthesis occurs bilaterally in 5% of the population and unilaterally in 1%. We found that the mid sagittal diameters of the vertebra proximal to the defect was significantly larger than in unaffected spines.[1]

These three anomalies are clinically important when affected spines are compromized by other pathologies. It is not surprising therefore, that symptomatic disc protrusion is more common in patients with trefoil shaped vertebral canals, where space is at a premium, than in the general population, and it is less common in patients with spina bifida occulta and with isthmic spondylolisthesis where the canal is more spacious.

(v) *Comparative anatomy.* The size and shape of the vertebral canals of gorillas and chimpanzees is significantly different to that of the human lumbar spine. Whilst the area of the human lumbar canal reduces from L1 to L4 and increases at L5, in the gorilla and chimpanzee the area continues to reduce proximo-distally. The mid-sagittal diameters steadily reduce in size in the gorilla, and increase in the chimpanzee. The interpedicular diameter gradually reduces in both primates from proximal to distal levels. The mid-sagittal diameters in gorillas and chimpanzees is 50% greater than the interpedicular diameter, whilst it is 50% less in the human spine. These results should now make it possible to predict whether a particular set of lumbar vertebrae from antiquity belonged to an upright or a semi upright primate.

Pathology that can become symptomatic when neurological structures are compromised in a small vertebral canal

(i) *Back pain syndromes.* Many authors have shown that spinal stenosis is an important factor in a number of back pain syndromes.[4–7] We have measured the vertebral canal by ultrasound in several thousand patients attending a back pain clinic, and found a highly significant difference in the canal size when compared with asymptomatic volunteers.[8] More then half of the patients with root symptoms from disc protrusion have canal measurements in the bottom 10% of the population[9] (Figure 2). Seventy per cent of patients with neurogenic claudication and 30% of those with root entrapment syndrome from degenerative change in the root canal, have canals below the 10th percentile. The original canal size is highly relevant when the spine is affected by degenerative pathology. In neurogenic claudication, double level degenerative pathology appears to be highly significant when this is superimposed on a developmentally small canal.[10] This two level stenosis causes venous congestion of the cauda equina between the two segments. At rest this is not a problem, but with the activity of walking, the arterioles in the congested segment probably fail to vasodilate, causing nerve root dysfunction.[11]

(ii) *Absenteeism from work.* We measured the vertebral canal in 191 coal miners over 50 years of age, and then examined their work records for the previous 3 years. 32.3% days of work were lost by 22 miners with the smallest canals below the 10th percentile,

**Delivered at The Royal College of Surgeons of Edinburgh on 27 May 1997, by Professor R. W. Porter.*

Correspondence: R. W. Porter, The Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh.

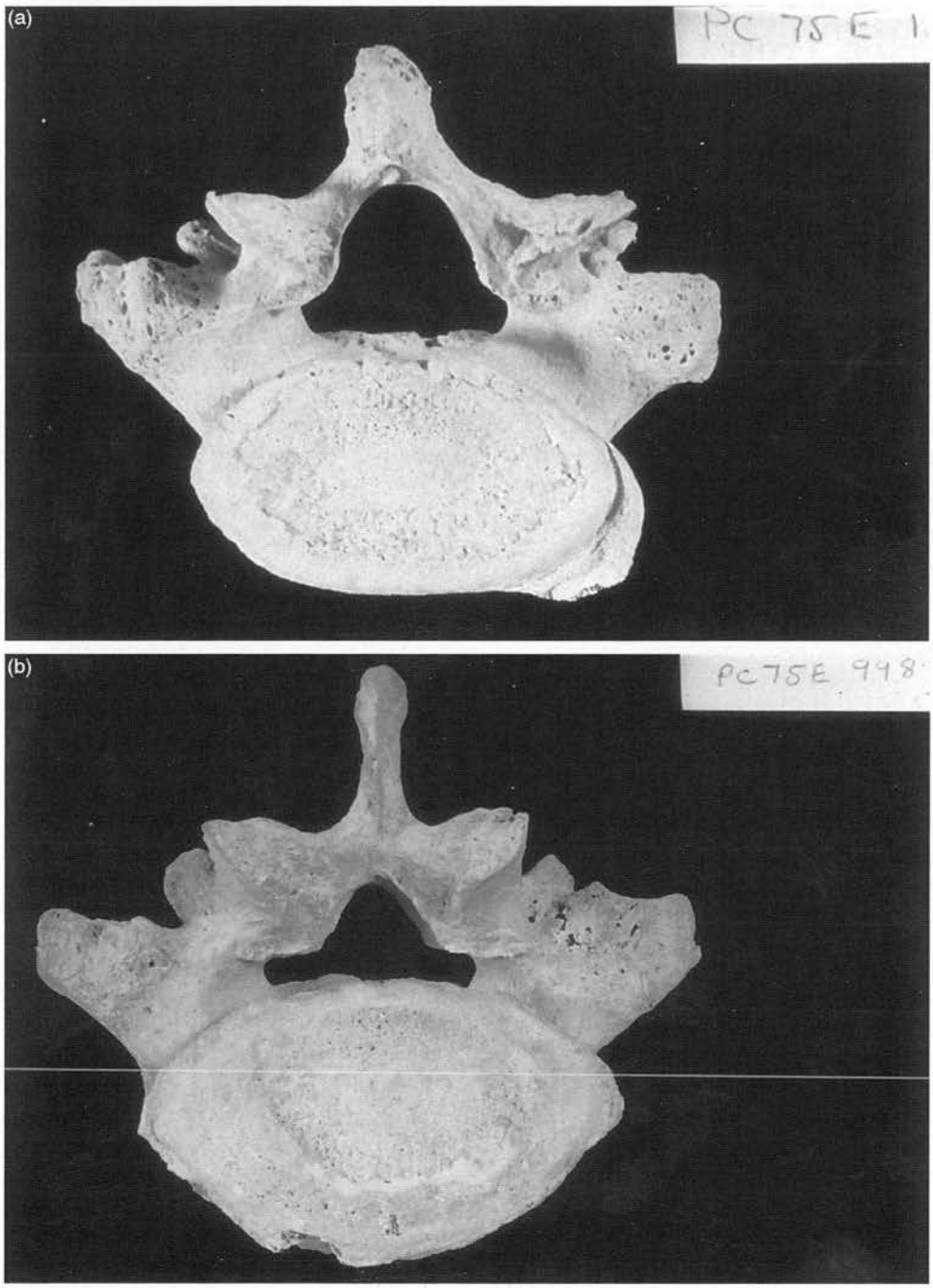

Figure 1 Compares two 5th lumbar vertebrae from the Poundbury Collection at the Natural History Museum in London. One vertebra has a large dome shaped canal whilst another has a shallow trefoil canal.

significantly greater than that of the men above the 90th percentile ($P < 0.001$).[12]

(iii) *Prediction of back pain.* We assessed the ability of ultrasound measurement of the vertebral canal to predict future back pain. 669 mining and nursing recruits were measured prior to 1980, and 450 replied to a back-pain postal questionnaire in 1990.[13] There was no significant difference in the experience of back pain reported by those with canals above the mean, when compared with those with smaller canals. However, going to bed with back pain was more frequent in those with smaller canals and this approached significance ($P = 0.068$). Those with smaller canals were also more likely to visit their doctor with back pain and seek manipulative therapy.

Development of the lumbar vertebral canal

(i) *Childhood growth.* When examining vertebrae from archaeological populations, we were surprised to find that the vertebral canals of infants were remarkably large (Figure 3). Similarly, ultra-

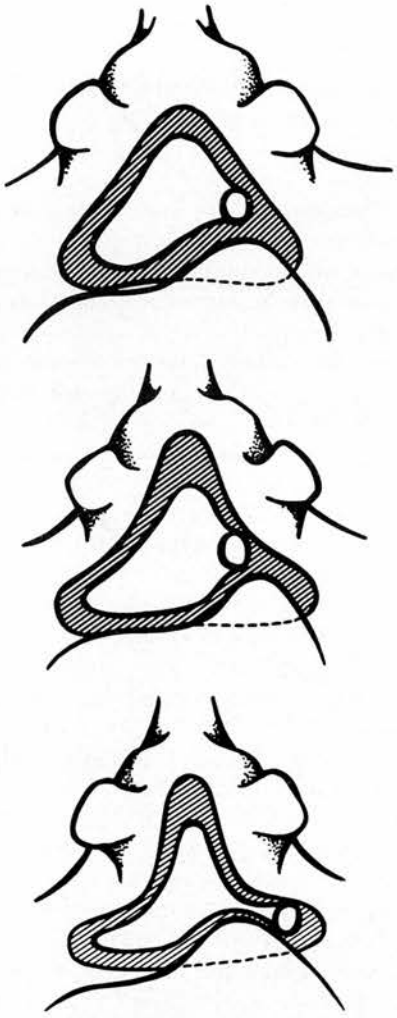

Figure 2 Illustrates how a nerve root in a large triangular canal can escape compression from a disc protrusion. A similar sized protrusion into a smaller canal can compromise the root.

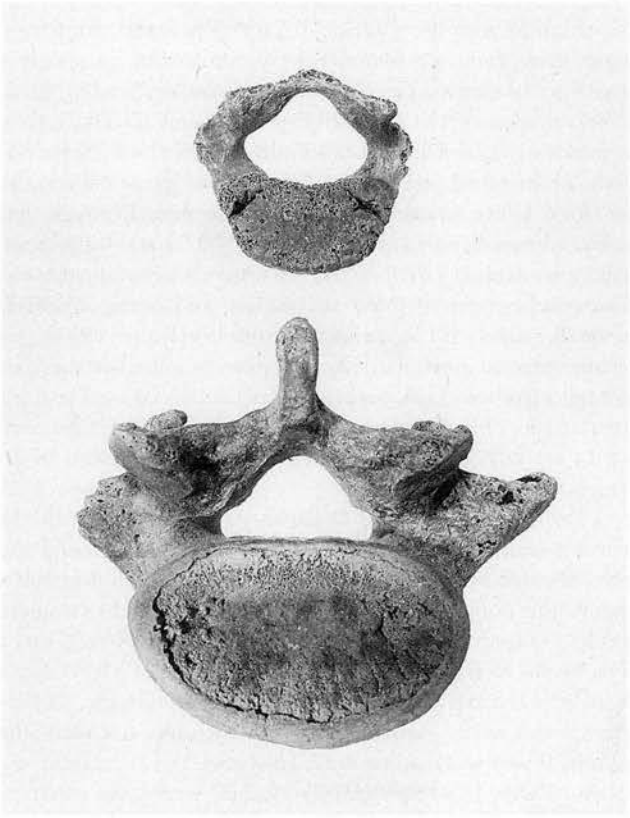

Figure 3 An infant and an adult's 5th lumbar vertebra. The vertebral canal is of comparable size.

sound measurements of children showed that their sagittal diameter was almost as large as in adults.[1]

We had the opportunity to examine 185 skeletons from the Spitalfield Collection at the Natural History Museum in London, skeletons from French Huguenots who were born between 1646 and 1852.[14] They were unique in that the exact date of birth and death was known, and there were 41 infants between 15 months and 5 years of age. We found that from L1 to L4 the cross sectional area and the mid sagittal diameters were fully mature by 1 year of age. The canal size reached maturity at L5 by 5 years of age. There was a gradual trend from a circular towards a trefoil shape at L5, but trefoilness was not present prior to adulthood.

(ii) *Malnutrition.* Clark[15] examined the vertebral canals and physiological stress indicators of two North American populations. He observed that the malnourished population had smaller canals than those with a better diet, and suggested that infant malnutrition might cause spinal stenosis.

It is probable that poor early growth leaves a small canal with no catch-up potential. The long bones may continue to grow concealing a small canal which has no second chance. If other systems with the same early growth curve – such as the immune and neurological systems – are similarly impaired, we would expect subjects with spinal stenosis to be disadvantaged in their health and neurological status.

(iii) *Other systems.* We conducted a comprehensive cardiovascular, respiratory and digestive system health assessment of 75 patients, comparing those with canals above the mean with those whose canals were below the mean.[16] Cardiovascular and gastrointestinal symptoms were more common in men and women with smaller canals ($P = 0.04$ and 0.048), but there was no significant difference in respiratory symptoms. We found that those with wider canals had more post school qualifications than those with smaller canals, and, in men, the difference in their performance in the Mill Hill Vocabulary test and the Progressive Matrices Test approached significance ($P = 0.08$ and 0.06).

We had previously found a relationship between the bone mineral content and the sagittal diameter of the lumbar canal in girls.[17] There is also evidence that the academic performance of children with wider canals at 16 years of age is better than that of their peers with smaller canals (Porter *et al.* 1987).

It is probable that developmental spinal stenosis is related to poor development of other systems very early in life. Specific insults to the sensitive foetal enzyme systems is more likely to be important than maternal malnutrition.

(iv) *Ante natal factors.* Professor Dougal Baird maintained excellent obstetric data in Aberdeen 50 years ago. We were able to identify and examine the obstetric records of the mothers of 50 patients who in the 1990s had a MRI scan for back pain, and

compare these with the adult canal size.[18] We found that the birth weight, gestational age, placental weight, maternal age, parity and parental social class had a combined influence accounting for 43% of the variance of the perimeter at L3,4 and 5. The area was independently affected by gestation at L3 (23% $P < 0.001$) and L4 (14%), by maternal age at L5 (18%) and by placental weight at L5 (18%). Those whose gestation was less than 37 weeks had a significantly smaller area at L3 and L4 ($P = 0.03$ and 0.047), and a smaller perimeter at L4 ($P = 0.017$). Those with a low birth weight, whose parents were of lower social class, and primiparous births had smaller canals at L3 and L4 ($P < 0.035$) (Papp 1997).

Thus, adverse antenatal factors appear to influence the size of the adult vertebral canal, particularly at L3 and L4, and to a lesser extent at L5. This is understandable when L3 and L4 have completed their growth at 1 year of age, whilst L5 continues to grow in infancy until 5 years of age.

(v) *Pre natal growth.* The Department of Anatomy at the University of Cambridge contains a large collection of foetal specimens – the Boyd Collection. We have been able to photograph and measure the transverse sections of 44 embryos and examine five complete older foetal specimens by MRI.[19] We observed that the conus began to rise from the lower sacral level at 10 weeks, and reached the level of L2 by 22 weeks of intrauterine life. The cross sectional area of the vertebral canal was the same size throughout the lumbar and sacral spine until 10 weeks, but then there was a relative increase in size proximally until 22 weeks, the distal canal being smaller after that time. The most rapid period of growth at all levels was from 18 to 36 weeks. The size of the canal closely matched the size of the dural sac above L2. There was a remarkable correlation between the size of the canal and the size of the brain ($P = 0.991$).

From this study it is possible to suggest that poor development before 22 weeks is likely to produce a generalized spinal stenosis. Impairment between 22 weeks and 1 year of age will affect L3 and L4 levels, and infantile growth disturbance up to 5 years of age will affect L5. The most sensitive period is probably between 18 and 36 weeks at L3 and L4, frequently the site of clinical problems. Deficiencies in spinal cord and canal growth are probably matched by deficiency in the growth of the brain.

SUMMARY

In this lecture I have attempted to demonstrate that the size of the lumbar vertebral canal has clinical importance. The canal develops very early in life, and impaired growth at this time affects other growing systems. The patient with spinal stenosis has more than a spinal disadvantage. Improved obstetric and childhood care has the potential not only to prevent some of the troublesome back problems, but also to influence the health and neurological status in adult life.

I hope that the first Henderson Trustees would have been encouraged by this lecture. It supports some of the philosophy that stimulated an interest in Phrenology. In the lumbar spine at least, the container – the vertebral canal – seems to have an important relationship to the function of its neurological contents.

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Paper accepted on 3 June 1997

Biomechanics of Spinal Stenosis

Richard William Porter

Spinal stenosis is usually developmental in origin and symptomless unless there is compromise of the neural elements from other pathology. Changes in posture, motion, and exercise can reduce space. In neurogenic claudication there is generally a two-level stenosis that is affected by biomechanical factors.

The Development of Spinal Stenosis

Spinal stenosis is an anatomical term used to describe a small vertebral canal. Its cause is usually developmental. There may be added degenerative change that further restricts the space for the cauda equina, but in the absence of developmental stenosis this does not usually compromise the nerve roots. There is normally adequate spare capacity in the extradural space in both the central and the root canals.

The Causes of Developmental Stenosis

The vertebral canal reaches maturity very early in life. By 1 year of age L1 to L4 has reached the size of the adult cross-sectional area, and L5 is mature by 5 years of age. The canal's most rapid growing period is between 18 and 36 weeks of intrauterine life.[1] Impaired nutrition at that time may permanently stunt the canal.

Biomechanical Factors That May Influence the Development of the Trefoil Shape

The shape of the canal is as important as its size, and about 15% of canals are trefoil at L5. Trefoilness is less common at L4 and rare at more proximal levels. The trefoil configuration and a small mid-sagittal diameter is an unpleasant combination, because the nerve roots can then be very tight in the lateral recess.

The shape of the lower lumbar canal begins to change from a round configuration to a triangular shape in childhood. In some spines this is more apparent than in others, until in a small proportion, the typical trefoil canal develops at puberty. The beginning of a change in shape from round to triangular coincides with the child's development of the secondary curve of lumbar lordosis, the assumption of the upright posture. If a pliable triangular tube is bent, it will buckle laterally and adopt a trefoil shape. It is possible that in predisposed individuals the assumption of a lumbar lordosis may influence the development of trefoilness.[2]

Dynamic Changes That Affect the Canal's Size

The vertebral canal is not a fixed bony tube. It is an anatomical space in a segmental structure the size of which is influenced by posture and motion. In addition, the extradural soft tissues within the canal are similarly affected by creep and the vascular engorgement that accompanies changing posture. Furthermore the size of the neural intradural contents change with exercise.

The Upright Posture

Axial compression of cadaveric spines[3] decreased the cross-sectional area by an average of 50 mm.[2] This has been confirmed in vivo by measuring the axially loaded spines during magnetic resonance imaging (MRI).[4] We have yet to discover how much of this space reduction is caused by buckling of the ligamentum flavum, and how much is the result of creep.

Spinal Flexion and Extension

Knutsson[5] used functional myelography in a stenotic patient in 1942, and showed that the continuity of a completely interrupted contrast column at L4/5 could be restored by flexion. Anatomical studies have confirmed that between flexion and extension, there is an average change in cross-section area of 40 mm.[2] This is the result of bulging of the soft tissues anteriorly and posterolateraly. The more a canal is stenotic, the greater will be the relative narrowing by changing posture. In the severe grades of stenosis, even the slightest degree of extension may compress the neural elements.[6]

From the Royal College of Surgeons of Edinburgh, Edinburgh, United Kingdom.

Address reprint requests to Richard William Porter, MD, FRCS, FRCSEd, Director of Education and Training, The Royal College of Surgeons of Edinburgh, Edinburgh EH8 9DW, United Kingdom.

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Posture and the Root Canal

The space for the nerve root may be restricted in the lateral recess of a trefoil-shaped canal. In flexion the root has a generous degree of freedom, but in extension there is a pincer action compressing the nerve between the superior articular process posteriorly, and the disc and inferior rim of the vertebral body anteriorly. More distally in the root canal, extension will similarly compress a tight root trapping it between the disc in front and the ligamentum flavum behind.

The Affect of Walking on the Stenotic Canal

Clinical studies suggest that the space in a stenotic canal is reduced further during the activity of walking. The epidural pressure measured by pressure transducer in stenotic patients increased by about 20 mmHg with each step.[7] The pressure was significantly less when a stenotic patient walked in flexion. However, there is not a marked increase in lumbar lordosis during gait[8] and therefore some of the explanation for increased pressure must be sought elsewhere.

There are two possibilities. First the activity of walking produces a segmental motion not only in one plane, but in all of the three planes of rotation. There is a combination of rotation, lateral bend, and sagittal motion all of which affect the space within a stenotic canal. Secondly, the contents of the canal increase in volume during the activity of walking, both from increased extradural venous pressure caused by an increased venous return, and also from vasodilatation of the vessels of the cauda equina.[9] Patients with spinal stenosis symptoms not only flex forward after the onset of claudication symptoms, but the center of gravity moves laterally with an increased sway.[10] This suggests that more space is created by movement in more than one plane.

The Clinical Significance of Spinal Stenosis

A small canal does not necessarily cause problems. It is often symptomless, and has been reported in 21% of asymptomatic subjects over 60 years of age.[11] However, stenosis is a factor in the symptomatology of a number of clinical conditions when the canal is compromised by other pathology. It can be important in three conditions. (1) Symptomatic disc protrusion, when a nerve root is compressed by a disc in a small canal. (2) Root entrapment syndrome, when in the presence of degenerative change, a nerve root can be compromised in the root canal. (3) Neurogenic claudication, when there is often multiple-level stenosis. This article examines how biomechanical factors influence the symptoms and signs in these three conditions in the presence of a stenotic canal.

Symptomatic Disc Protrusion

Pathology of the Protrusion. When a disc protrusion is symptomatic, it has been preceded by a long-standing degenerative pathological process. The biomechanics of this degeneration are not understood. In vivo, when an axial spinal load is applied to a healthy spine, the vertebral bodies fracture before the disc is damaged. Similarly in vitro, the bone fails before the healthy disc. Poor nutrition may be responsible for an unhealthy disc developing fissures in response to load, and with multiple fissures, fragments develop. The mechanics of the disc change considerably once a fragment forms, and with a fairly minimal load the fragment displaces posteriorly, causing the back of the annulus to bulge and sometimes to rupture.[12,13]

The Mechanics of Root Symptoms. Recent studies suggest that two components are responsible for the root pain in a symptomatic disc protrusion, compression and inflammation. Provided the canal is sufficiently large, patients with a protrusion may experience some back pain, but they are spared root symptoms because the root is not compressed. Patients having disc surgery can have root pressure over 100 mmHg.[14] This is reduced to zero after operation. The root pressure is not related to the degree of reduction in straight-leg raising, suggesting that this root tension sign is probably more related to inflammation than to pressure.

Trunk List. The trunk list sign is present in about half of the patients with disc protrusion. It is gravity induced but its biomechanics have still to be explained. It commonly occurs without root pain and its presence is therefore unrelated to the canal size.

Root Entrapment Syndrome From Degenerative Change

These patients with root entrapment syndrome have constant and severe root pain as a result of nerve root compression in the root canal. Unlike the patient with disc protrusion, they do not have abnormal root tension signs or a trunk list. The pain is present at rest. It is insidious in onset, and it frequently resolves over several weeks or months.[15]

Pathology of Root Compromise. In many patients there is a gradual increase in degenerative change

that slowly reduces the size of the root canal. It is a combination of bony change and soft tissue thickening (Fig 1), which involves the facet joint capsule, the posterior annulus, and the ligamentum flavum.[16]

Biomechanics of Root Entrapment. Many patients with root entrapment syndrome have some degree of vertebral displacement. If the root canal is already small, segmental displacement will reduce the available space further. This can be a particular problem in patients with degenerative spondylolisthesis. If the 5th lumbar root is already tight beneath the superior facet of L5, as the body of L4 displaces forward, the root becomes critically affected (Fig 2). There is frequently a degree of rotational displacement, which will then give asymmetrical symptoms. When lumbar scoliosis is associated with a stenotic canal, the root entrapment symptoms are particularly troublesome and progressive.

The Dynamics of Walking. The root entrapment pain can sometimes be made worse by walking.[17] These patients have constant pain at rest, but in addition, the motion of walking on a mobile spinal segment can subject a tight root to high, intermittent pressures until the patient has to stop. This syndrome is not to be confused with neurogenic claudication. Claudicant patients do not have pain at rest, but symptoms develop only when they have walked a distance.

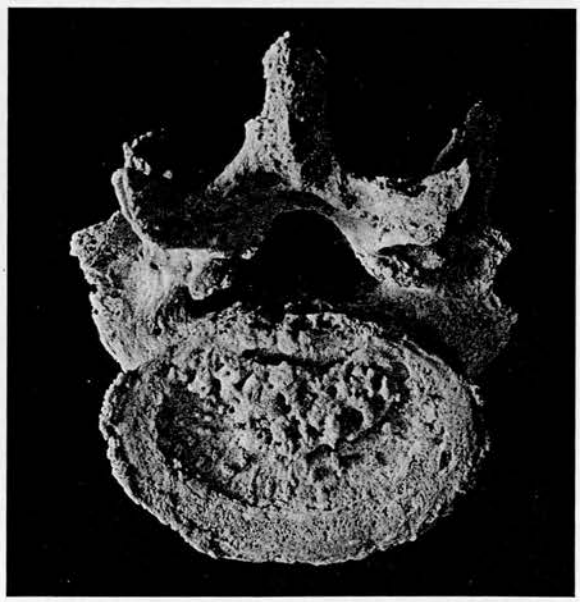

Figure 1. A degenerate fifth lumbar vertebra showing a large osteophyte in the left root canal. In combination with a thickened ligamentum flavum, this will significantly limit the space for the fifth lumbar root.

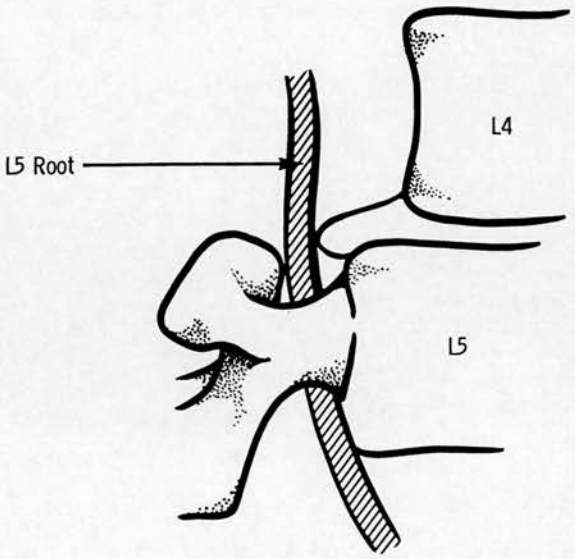

Figure 2. A diagram of a degenerative spondylolisthesis at L4/5, showing that the fifth lumbar root is compromised between the inferior facet and the thickened disc.

Neurogenic Claudication

The Symptoms of Neurogenic Claudication. Neurogenic claudication is a clinical condition causing discomfort, numbness, and pain or heaviness in one or both legs after the patient has walked a short distance. It is relieved by rest. There is no leg pain at rest. There is often a long history of back pain. Neurogenic claudication is sometimes called spinal stenosis, but stenosis is really an anatomical term. Stenotic spines can be symptomless. There is a biomechanical mechanism to explain why stenotic spines can produce symptoms with walking. This becomes apparent as we examine the abnormal signs.

Abnormal Signs and Characteristics of Patients With Neurogenic Claudication. Patients with neurogenic claudication tend to stoop as they walk, and at the limit of walking tolerance they stoop further and then rest[19] (the Stoop Test). Some patients can walk up a hill more comfortably than walking down a hill; and some can lean forwards and cycle for a long distance without leg symptoms[20] (the Cycle Test), although the Cycle Test is not a good discriminator between neurogenic claudication and intermittent claudication[21] (peripheral vascular disease). Patients with neurogenic claudication are generally over 50 years of age, with men affected more frequently than women. Most patients with neurogenic claudication have multiple-level spinal stenosis. Half the patients with bilateral claudication have a degenerative spon-

dylolisthesis, which usually affects men rather than women[22] (although degenerative spondylolisthesis is more common in women than men). Half of the patients with unilateral claudication have a degenerative lumbar scoliosis, and this combination is more common in women than in men.[23] Peripheral vascular disease and spinal stenosis often co-exist.[24] Calcitonin can relieve claudication symptoms in a proportion of patients.[25]

A Single Level of Spinal Stenosis. A single level of stenosis does not usually produce claudication symptoms. For example, a large disc protrusion may almost occlude the canal at one level and produce back and/or leg pain but not claudication. Similarly, a large spinal tumor in the lumbar canal may produce bizarre symptoms but not claudication. Compression of a nerve root in the central or root canal will cause root entrapment pain but not claudication. In canine studies a single-level, experimental stenosis constricting the cauda equina by 25% did not cause a neurological deficit.[26]

Multiple Levels of Stenosis. Most patients with neurogenic claudication have two or more levels of stenosis. There may be two levels of central stenosis, or one of central stenosis and one of a more distant root canal stenosis.[27] Animal studies have shown that two levels of cauda equina compression at just above venous pressure can produce major changes in nerve conduction, axon transport,[28] and blood flow.[29]

The Venous Anatomy of the Nerve Roots of the Cauda Equina. The venous anatomy of the cauda equina is highly specialized, with centrifugal venous drainage from the conus down the nerve roots to the foramen. By contrast, the arterial flow is centripetal. There is a physiological valve in the radicular veins at the level of the nerve root sleeve.[30] This prevents a back flow of venous blood from the veins in the extradural nerve root, protecting the cauda equina from high venous pressure.

If there is a single level of stenosis, the veins of the peripheral part of the nerve root drain to the intervertebral foramen, whereas the veins proximally drain back to the conus. These anastomose with other root veins and then drain distally to the foramen of the respective roots. There is no significant venous congestion. However, in the presence of two levels of stenosis (above the venous pressure) there will be venous engourgement of the root veins in the segment between the two stenoses to a pressure equal to the occlusion pressure (Fig 3). This might be as great as 100 mm Hg,[31] and it will probably affect nerve root function in a similar way to that shown in animal studies.

Although this hypothesis accounts for some of the features of neurogenic claudication, it is necessary to explain the absence of symptoms at rest, the age and sex characteristics, and the biomechanical affect of posture and of walking.

The Affect of Exercise on Claudication Symptoms. Electrical stimulation of the cauda equina in a porcine model is associated with electromyelography activity in the tail muscles and an increase in cauda equina blood flow to 300% of the resting level. This is maintained if the stimulation continues for more than 30 minutes. However, if a double level of occlusion is applied to the cauda equina, and the proximal region is then stimulated electrically, the increase in blood flow is less marked and of shorter duration (Fig 4).[29] This model suggests that in the presence of venous congestion, the arterial vasodilatation associated with exercise is inadequate for prolonged activity. It suggests that there is an arterial explanation for the claudication symptoms, and that with lower limb activity, arterial vasodilatation of the cauda equina fails and nerve conduction may be impaired. An arterial component of the pathology is compatible with these patients being in the arteriosclerotic age group, often having coexistent peripheral vascular disease, and sometimes responding to calcitonin, which is a potent arterial vasodilator.

The Affect of Posture on Claudication Symptoms. A stooping posture can help to relieve claudication

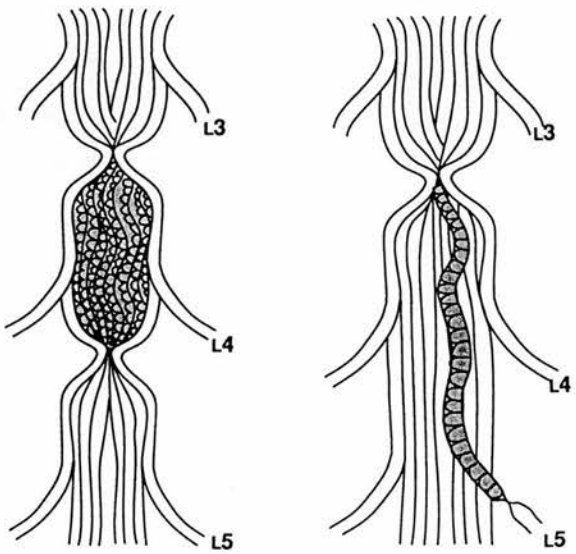

Figure 3. A diagrammatic representation of venous congestion of the whole of the cauda equina between two levels of central canal stenosis. This will affect the function of both legs. A central stenosis at one level and a more distal root canal stenosis will cause congestion of one root and unilateral symptoms.

symptoms in some patients. As they lean forward on a bicycle or when walking up a hill, they may have less discomfort. It has been shown that flexing the lumbar spine can increase the cross-sectional area of the central canal by reducing the posterior bulge of the annulus, and stretching a buckling ligamentum flavum. Similarly flexion will increase the cross-sectional area of the root canal.

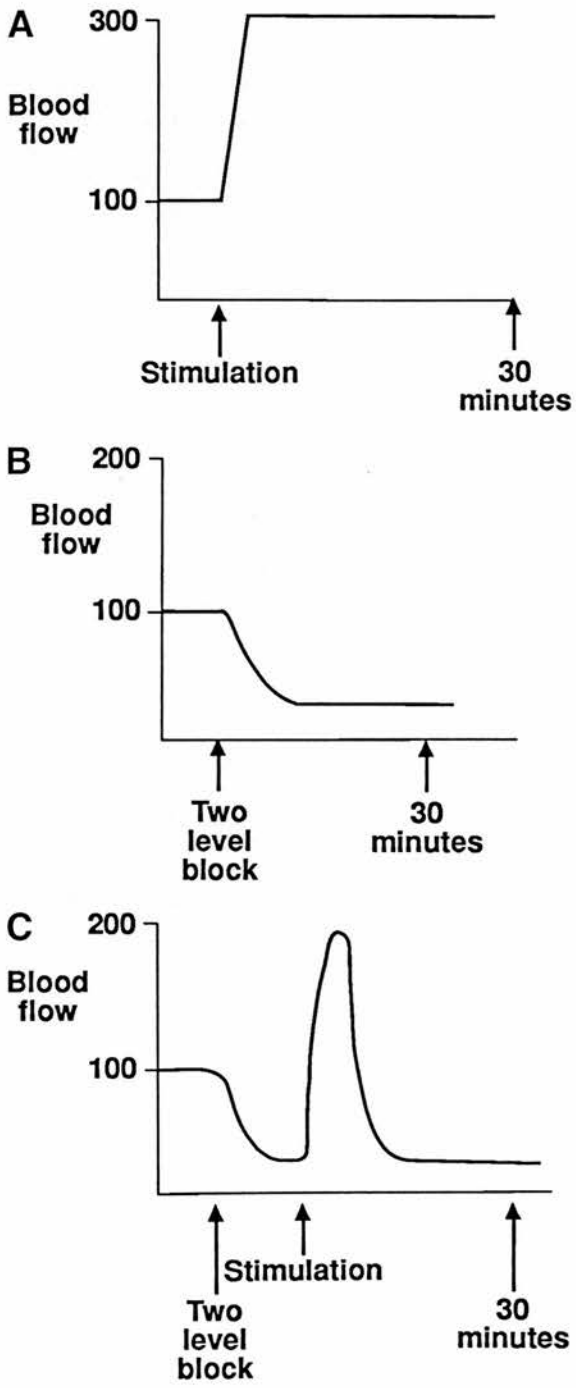

Figure 4. Blood flow in a porcine cauda equina. (A) After stimulation of the cauda equina proximally. (B) When producing a two-level block just above venous pressure. (C) Stimulation of the cauda equina in the presence of a two-level block.

It is probable that some patients have a block pressure at one of the stenotic levels just above venous pressure in extension, but just below venous pressure in flexion. These patients are able to walk in flexion without the roots being congested; but with extension and rotation, venous engorgement causes problems. However, patients with a very stiff ankylosed spine do not have sufficient segmental motion for symptoms to be influenced by posture.

Conclusion

Spinal stenosis does not usually cause symptoms unless there is an added pathology. The clinical syndromes are now clearly defined. An appreciation of the affect of load and motion on the stenotic spine and its contents should help us to understand more about the pathophysiology of these conditions and how best they can be managed.

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Reprints of publications

Part two - disorders of the lumbar spine

publications 31-57

UNILATERAL SPONDYLOLYSIS

R. W. PORTER, W. PARK

From the Royal Infirmary, Doncaster, and the Institute of Orthopaedics, Oswestry

Five vertebrae with unilateral spondylolysis are presented. The associated asymmetry of the posterior elements supports the concept of a localised form of growth deficiency. The defect is difficult to demonstrate radiologically, and is perhaps present more frequently than is recognised. It should be suspected clinically from asymmetry of the neural arch and from unilateral wedging of the vertebral body, and may be demonstrated by further radiographic examination. The clinical significance is uncertain, but one patient is presented in whom unilateral spondylolysis was associated with intermittent sciatic pain.

Detection of a unilateral defect in the pars interarticularis is extremely difficult. The purpose of this communication is to present the detailed morphological features associated with unilateral spondylolysis of the fifth lumbar vertebra in five skeletal specimens, and the case report of a patient with the same condition. The clinical significance and radiological implications are discussed.

MATERIAL AND METHODS

Four archaeological specimens of the fifth lumbar vertebra with a unilateral defect of the pars interarticularis were loaned from the British Museum. A fifth specimen was removed from a contemporary skeleton. Anthropomorphic measurements were made of the vertebral body, neural arch, apophysial joints and spinous processes. The specimens were photographed in sagittal, coronal and axial planes. Each specimen was then radiographed in the frontal, lateral and axial projections. Oblique radiographs and computerised tomograms were made of some specimens. The case report is presented separately.

FINDINGS

Vertebral morphology. The spondylolytic defect was clearly visible and easily identified on all the specimens and was on the right side in three. In each instance the pedicles and the superior apophysial joints were in normal symmetrical relationship with the vertebral body (Fig. 1). It was evident, however, that there was marked asymmetry of the neural arch, the inferior apophysial joints and the posterior elements (Fig. 2). The combination of these effects produced rotation of the spinous process away from the side of the lesion, the inferior apophysial joint on that side being placed more dorsally than the superior. When viewed from behind, the neural arch was rotated in an anticlockwise direction in the vertebrae with a right-sided defect and clockwise in the remainder. The combined deformity resulted in horizontal orientation of the lamina on the affected side.

Two of the vertebral bodies revealed quite marked asymmetrical posterior wedging. On the side of the defect there was a reduction in the vertical height at the posterior angle of two and four millimetres respectively.

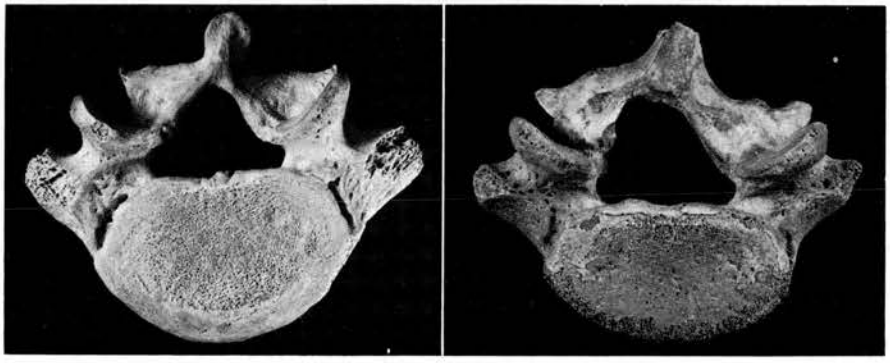
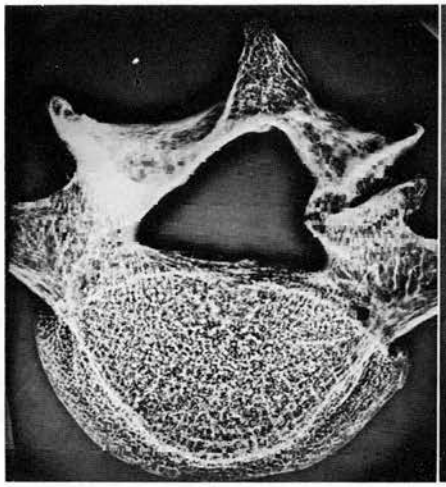


Fig. 1 Fig. 2

Figure 1—Photograph of specimen with unilateral spondylolysis showing symmetrical superior apophysial joints. Figure 2—Photograph of unilateral spondylolysis showing asymmetrically placed inferior apophysial joint with the spinous process deviated away from the defective side.

R. W. Porter, MD, FRCS, FRCS Ed, Consultant Orthopaedic Surgeon
Doncaster Royal Infirmary, Doncaster DN2 5LT, England.
W. Park, DMRD, FRCR, Director of Radiology
Institute of Orthopaedics, Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, Salop SY10 7AG, England.
Requests for reprints should be sent to Mr R. W. Porter.



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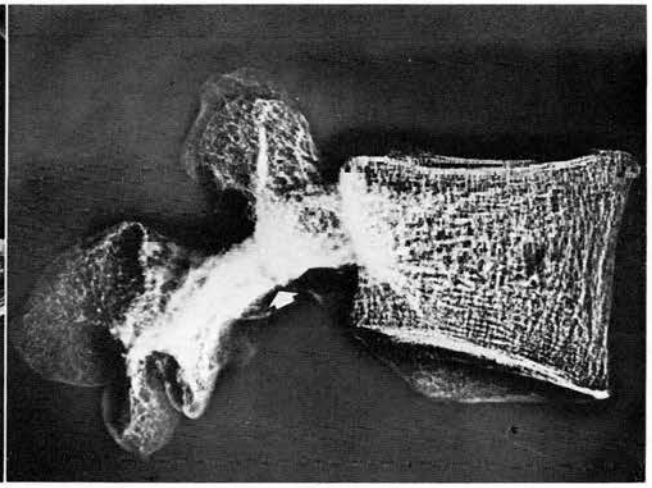


Fig. 4

Figure 3—Radiograph of unilateral spondylolysis. Figure 4—Radiograph of the same specimen showing marked asymmetrical wedging of the vertebral body. The spondylytic defect is marked by an arrow.

The pedicles and posterior elements on the unaffected side were examined for signs of overgrowth; in one specimen these were slightly enlarged when compared with a normal control.

Radiographic morphology. The radiographs revealed that the areas of asymmetry were accompanied not only by a reduction in size of the lamina, apophysial joint or transverse process, but also by thinner cortices and fewer trabeculae in these locations (Fig. 3). Posterior asymmetrical wedging of the vertebral body is shown in Figure 4 and this is accompanied by a tendency to horizontal orientation of the pars interarticularis and the inferior apophysial joint on the affected side.

The spondylolytic defects varied in width and this determined the facility with which they could be demonstrated radiologically. When the defect measured two millimetres or more (four specimens) the axial projection demonstrated this most clearly. A defect of one millimetre could not be demonstrated in this plane even using thin-section computerised tomography (Figs 5 to 7). An oblique technique with an inclined plane had to be developed to demonstrate this particular lesion: the standard 45-degree oblique projection was augmented by a 20-degree tilt of the x-ray tube towards the head (Fig. 8). In addition to the spondylolysis, one specimen had a spina bifida, resulting in a free-floating fragment of bone

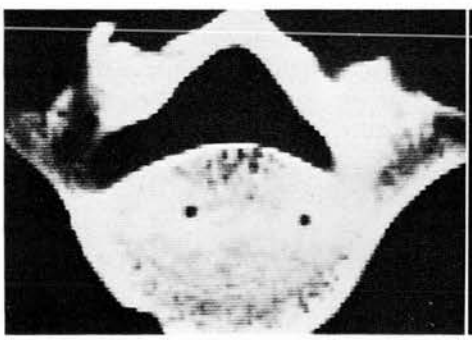
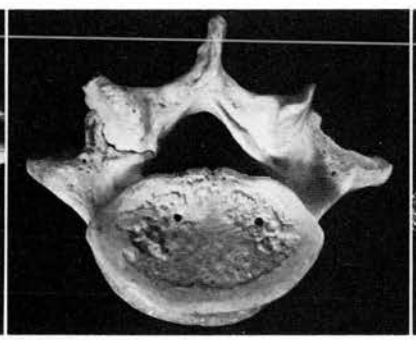


Fig. 5



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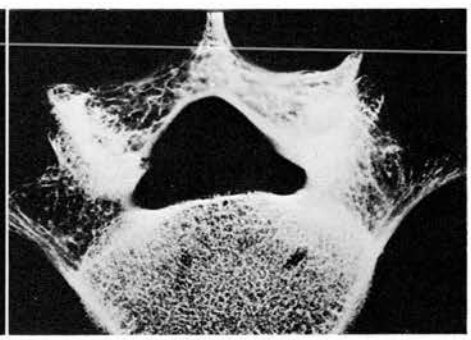

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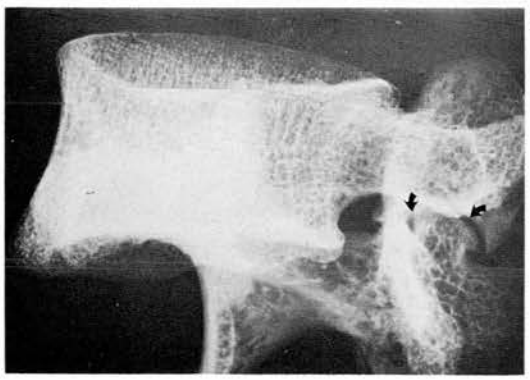


◀ Fig. 8

Figure 5—Computerised tomogram: this high-definition section does not resolve the detail of the defect. Figure 6—Axial photograph: the pars defect is clearly visible with some hypertrophic bone formation protruding into the spinal canal. Figure 7—Axial radiograph: only the most medial margin of the defect is visualised. Figure 8—Oblique projection with a 20-degree tilt of the x-ray tube: the defect is represented by an inverted V-line (arrows).

comprising the neural arch and the inferior apophysial joint (Fig. 9). In three specimens there were osteophytes round the defect (Fig. 3), evident on the axial radiographs and on the inclined oblique views as an indentation on the superior aspect of the nerve root canal. The surfaces of the defect had thin cortices with trabeculae extending uniformly up to the margin (Fig. 9). There was no evidence of bridging callus across the spondylolysis. The intact pars on the unaffected side showed marked condensation of cortical bone (Fig. 3).

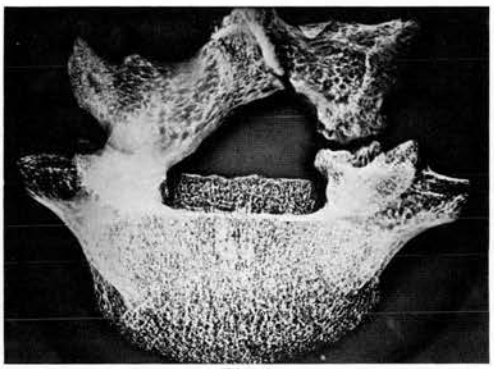


Fig. 9

Radiograph of unilateral spondylolysis with a free-floating fragment.

ILLUSTRATIVE CASE REPORT

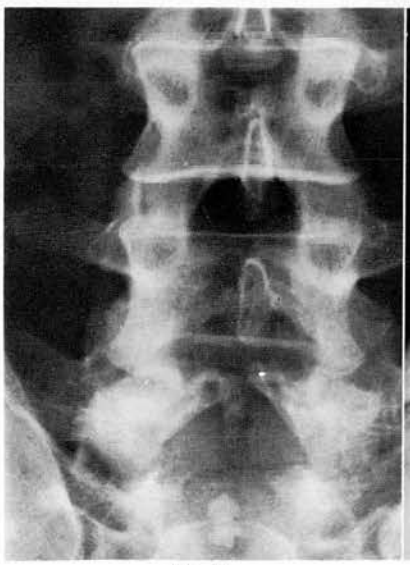
Three years before presentation to the hospital this 31-year-old farm worker slipped while lifting, and sudden discomfort resulted in the lower part of his back. He subsequently had repeated attacks of low backache with left-sided sciatica. He was admitted in January 1972 with an acute attack of pain after a sudden sneeze and difficulty in taking weight on the left leg. Forward flexion was limited and hyperextension was painful. His straight leg raising was limited to 50 degrees with the left leg but the right achieved 90 degrees, with pain and parasthesiae of L5–S1 distribution. Tendon and sphincter reflexes were normal. He was treated conservatively with traction and bed rest and after two weeks was discharged with no symptoms. Subsequently he suffered recurrent attacks of low backache culminating in a further admission in March 1976 with continuous pain, radiating down the right leg, making him unable to work. In contrast to the previous admission, he now had straight leg raising limited to 30 degrees on the right, causing severe referred pain. Examination revealed slight weakness of extension of the right great toe with decreased sensation on the outer side of the right thigh and calf and on the inner side of the foot.

Radiological examination revealed unilateral spondylolysis in the left pars interarticularis of L5 (Figs 10 to 13) with a minor degree of forward slipping of the vertebral body. Posterior wedging was present and associated with spina bifida occulta of L5. Myelography showed very minor forward subluxation of L5 on forward flexion. There was no evidence of disc protrusion nor of nerve root entrapment.

After bed rest the patient was discharged free of symptoms and has subsequently continued with intermittent mild symptoms but is still able to work as a farmer. His symptoms have not required further admission nor has operative treatment been considered.

DISCUSSION

It is estimated that bilateral spondylolisthesis occurs in approximately four per cent of the population. In contrast, unilateral spondylolysis is rather rare. It has previously been reported by Stewart (1953) and was present in one-sixth of the spondylolytic specimens of Roche and Rowe (1951). It is clear from examination of the dried specimens and from studying the clinical radiographs of one patient that unilateral spondylolysis



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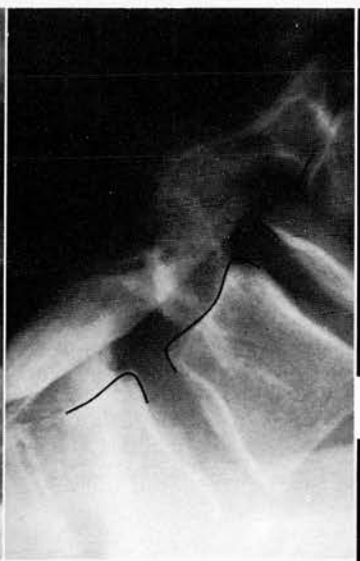

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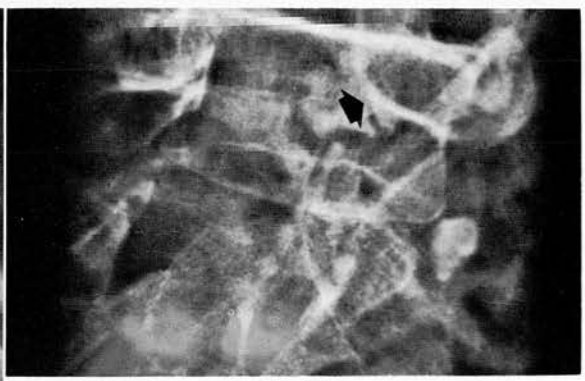

Fig. 12

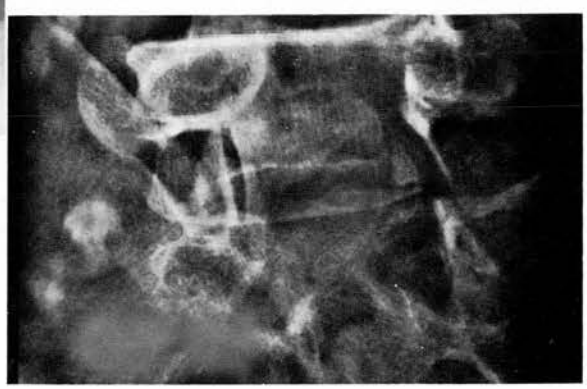

Fig. 13 ▶

Unilateral left spondylolysis. Figure 10—Frontal view showing spina bifida occulta. Figure 11—Lateral myelogram showing posterior wedging of the vertebra and in this flexed position, the forward displacement of L5 is clearly visible. Figure 12—An oblique radiograph with a 20-degree incline showing the unilateral defect on the left side. Figure 13—A 20-degree inclined plane on the unaffected side shows no abnormality.

may be extremely difficult to demonstrate radiologically by any single technique. Its presence may be suspected when there is asymmetrical posterolateral wedging of the vertebra, horizontal orientation of the lamina or hypoplasia of one inferior apophysial facet. However, even computerised tomography may not always reveal the defect. Radiographs taken at a 45-degree oblique angle with the tube inclined 20 degrees cranially may be of help and may give some additional information about hypertrophic bone indenting the nerve root canal. Because of these difficulties in visualisation it is possible that unilateral spondylolysis is more frequent than is generally acknowledged.

It is difficult to reconcile the findings in these examples of unilateral spondylolysis with the various theories on the aetiology of bilateral spondylolisthesis. Opinions vary on the cause of the lytic defect in the pars interarticularis. A classification of spondylolysis and spondylolisthesis has been proposed by Wiltse, Newman and Macnab (1976) but this refers to bilateral lesions. The major controversy centres on the dilemma of whether the defects are congenital or traumatic. A number of authors (Newman 1963; Farfan 1973; Troup 1976, 1977) have felt that the two are not mutually incompatible and that an individual may be congenitally predisposed to a stress fracture of the pars. Undoubtedly, fractures of the pars may be seen to heal (Devas 1963; Murray and Colwill 1968; Krenz and Troup 1973; Jackson, Wiltse and Cirincione 1976). In our specimens, however, there was no evidence of a healed fracture on the side opposite the defect, nor of attempted healing of the unilateral lysis. The very existence of unilateral spondylolysis must question the concept that spondylolysis is always the result of a stress fracture.

Observations in the present small series would suggest that this form of unilateral spondylolysis is associated with localised underdevelopment or hypoplasia of adjacent structures. This may involve the posterior angle of the vertebral body producing asymmetrical wedging, and the lamina and inferior facet joint producing asymmetry of the posterior arch. Since this is unilateral, it is probable that the effect occurred before skeletal maturity was complete and that the spondylolysis and hypoplasia are associated growth phenomena. Unfortunately, the findings do not indicate which is cause and which is effect. The skeletal specimens in this study are all mature and the abnormalities, therefore, likely to be permanent. This poses questions about the biomechanical implications and the clinical significance of the lesions. In the isolated vertebra, the most obvious deformity is asymmetry of the neural arch with deviation of the spinous process away from the side having the hypoplastic elements. On the other hand, in the articulated spine, the spinous processes may maintain a midline position. This could only occur with forward rotation of the vertebra on the affected side. Theoretically, this should produce the effect of a hemilisthesis, which in fact is evident in our patient.

Wedging of the displaced vertebral body in spondylolisthesis is well recognised. Wiltse (1962) and Sim (1973) have shown that vertebral wedging may progressively increase as the spondylolisthesis deformity advances in childhood. Blackburne and Velikas (1977) have shown that increasing displacement occurs mainly during the adolescent growth spurt. Further displacement is most unusual after cessation of growth even though the mechanical forces remain (Friberg 1939; Brocher 1958).

There is evidence that growth remodelling occurs in the posterior elements. Park *et al.* (1980) have studied the changes in grossly displaced adolescent spondylolisthesis. This revealed pronounced elongation of the pars interarticularis as a result of remodelling secondary to the altered stress loading. This was accompanied by underdevelopment of the posterior elements, notably of the inferior facet joints and intervertebral bodies.

The clinical implications of unilateral spondylolysis must remain speculative. Possible factors could include the effects of unilateral rotation, with torsional damage to the intervertebral disc and traction on the nerve root (Farfan *et al.* 1970) which could possibly be aggravated by hypertrophic bone projecting into the nerve root canal. A significant contributory factor is likely to be the presence of spina bifida which will produce a free-floating fragment and thereby contribute to segmental hypermobility.

In summary, this study reports the association of unilateral spondylolysis with localised hypoplasia of the vertebral body and neural arch. The abnormality and the findings in one patient suggest that this could be a contributory factor in low backache. The full clinical implications have yet to be fully documented. However, it should be specifically sought in patients with asymmetrical vertebral wedging and associated hypoplasia of the neural arch.

We would like to thank Miss Rosemary Powers of the British Museum for the loan of the four archaeological specimens; Mr Garry Swann of the Photographic Department at Doncaster Royal Infirmary and Mr David Jones of the Photographic Department at Oswestry for the illustrations; and Mrs Eileen Cook and Mrs Jean Reynolds for the secretarial assistance.

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Symptoms Associated with Lysis of the Pars Interarticularis

32

R.W. PORTER, MD, FRCS, FRCSE, and C.S. HIBBERT, BSc, PhD

Comparisons were made between the symptoms of 131 patients with lysis of the pars interarticularis and 2229 patients without lysis, attending a first referral back pain clinic. The canal diameter measured by ultrasound was compared in the two groups, and the slip ratio measured for those with olisthesis. There were significantly fewer patients who had symptoms associated with disc prolapse and lysis of the pars. It is suggested that an enlarged central spinal canal may protect the patient with a pars defect from disabling root problems in the presence of a disc lesion. The most common symptom in those attending with lytic defects was pain in the back and/or referred pain, occurring with nearly twice the frequency for other attenders at the clinic. The incidence of lysis (5.6%) in patients attending the clinic was probably no greater than its incidence in the general population. [Key words: spondylolysis, spinal canal diameter, disc herniation, symptoms]

LYSIS OF THE PARS INTERARTICULARIS is a condition that continues to interest clinicians treating back pain, because there is such an obvious anatomic anomaly that it is assumed to be responsible for the symptoms. It is, however, a common condition, with a variable incidence among selected groups and can be symptomless. Roche and Rowe[11] carried out the largest study of white skeletons and found on overall incidence of bilateral lysis in 4.2% of 2300 skeletons, there being a male to female ratio of almost three to one. Studies *in vivo* have shown a greater incidence in selected athletic groups, from 5–21%.[2,5,6,9] It is uncertain whether the high incidence in the athlete is the result of stress to the ars, or whether this career was chosen because the subject with sis is in some respects hypermobile. Studies of the gymnast's pine, however, in spite of the series being dissimilar in age, sex, nd nationality, clearly showed that a high proportion of subjects ith lysis have no symptoms at all, 33% in Bird's series,[2] 45% in ackson's,[6] and in Hitoshi's series,[5] 76% of subjects with lysis were ymptomless, there being no significant difference in back pain ncidence between those athletes with lysis and those without.

Semon and Spengler reached the same conclusion,[12] finding 27% of 506 college football players had back pain, but only 2.4% had pain and a spondylolisthesis. The lysis did not appear to have clinical significance in the short term.

In this study, we have recorded the incidence of lysis of the pars n patients attending a first referral back pain clinic and have ttempted to assess whether their pattern of symptoms differ from ther patients with back pain and no lysis.

From the Doncaster Royal Infirmary, Doncaster, England.
Submitted for publication June 20, 1983.
The authors would like to acknowledge the National Coal Board and the Back Pain Association for their financial assistance. They also would like to thank Mr. Garry Swann for his illustrations and Mrs. Jean Reynolds for her secretarial assistance.

PATIENTS AND METHODS

Sixty-one pieces of information were recorded from the history, examination, radiographs, and ultrasound measurements of 2360 patients attending a back pain clinic. This was stored by computer for subsequent retrieval. Two standard antero-posterior and two lateral radiographs were performed, and if spondylolysis or olisthesis was suspected from forward displacement, anterior sacral lipping or vertebral wedging on the lateral view, or from increased density of the lamina from forward tilting in the antero-posterior view, then radiographs in the standard 45° oblique projection were obtained. The central spinal canal was measured by ultrasound, and the slip ratio recorded when olisthesis was present.[3]

Four back pain syndromes were defined, and the incidence of patients fulfilling the criteria of these syndromes recorded for patients with and without lytic defects.

The Symptomatic Disc Lesion. Patients were required to satisfy three or more of the criteria described by McCulloch[8]: unilateral leg pain in a typical sciatic root distribution, including discomfort below the knee; specific neurologic symptoms incriminating a single nerve; limitation of straight leg raising by at least 50% of normal; at least two neurologic changes of muscle wasting, muscle weakness, sensory change, or hyporeflexia; and myelographic evidence of disc protrusion.

Root Entrapment Syndrome. The root is thought to be affected laterally in the root canal in association with degenerative change. They had unilateral pain in a sciatic root distribution below the knee at least to the lower calf, constant and severe; it was aggravated by sitting for long, and not relieved by lying down; straight leg raising was equal or better than 70°; they were over 40 years of age.

Back and/or Referred Pain. Pain in one or both buttocks, outer or posterior thigh, but not distal to the upper calf, relieved to some degree by bed rest; straight leg raising equal or better than 90°; no abnormal neurologic signs.

Neurogenic Claudication. Diffuse bilateral discomfort above and below the knees, poorly localized, limiting walking distance, improved by rest, with a normal peripheral circulation.

RESULTS

According to the classification of Wiltse, Newman, and Macnab,[15] 131 of the 2360 patients attending the first referral back pain clinic were found to have bilateral isthmic defects of the pars interarticularis, an incidence of 5.6%. This did not include patients with dysplastic or traumatic spondylolisthesis, and 86% of the defects occurred at L5 and 13% at L4.

The sex for patients with and without lysis was not significantly different, the male/female ratio being 63:37 and 60:40, respectively.

The incidence of the four back pain syndromes in patients with and without lysis is shown in Table 1. The most significant negative finding was that only two patients with lysis fulfilled the criteria for a symptomatic disc lesion. The most common syndrome recorded

Table 1. Incidence of Four Back Pain Syndromes in Patients With and Without Lysis

	Percentage of 131 patients with lysis of the pars	Percentage of 2229 patients without lysis of the pars	Significance
Symptomatic disc lesion	1.5	8.7	($P<0.01$)
Root entrapment syndrome	16.0	14.1	not significant
Back and/or referred pain	32.8	18.2	($P<0.001$)
Neurogenic claudication	4.6	6.5	not significant

in patients with lysis was back and/or referred pain, occurring with nearly twice the frequency of other attenders at the back pain clinic.

Ultra sound measurements of the central spinal canal were recorded in the patients with lysis, and the mean measurements with one standard error were compared with each of the four back pain syndromes and also compared with the mean values of 551 volunteer subjects (Figure 1). Admittedly, the number with neurogenic claudication and symptomatic disc lesions are few, but their mean values from L1–L5 contrast with the wider canals of volunteer subjects. However, the measurements of those patients with lysis and root entrapment syndrome and back and/or referred pain are not significantly different from the volunteers. Those patients with back and/or referred pain had a significantly greater slip ratio ($17 \pm 8\%$) than those with root entrapment syndrome, ($11 \pm 11\%$, $P<0.01$).

Three times as many women with lytic defects attributed the onset of their back pain to pregnancy when compared with the 913 women attending the back pain clinic without defects (Table 2).

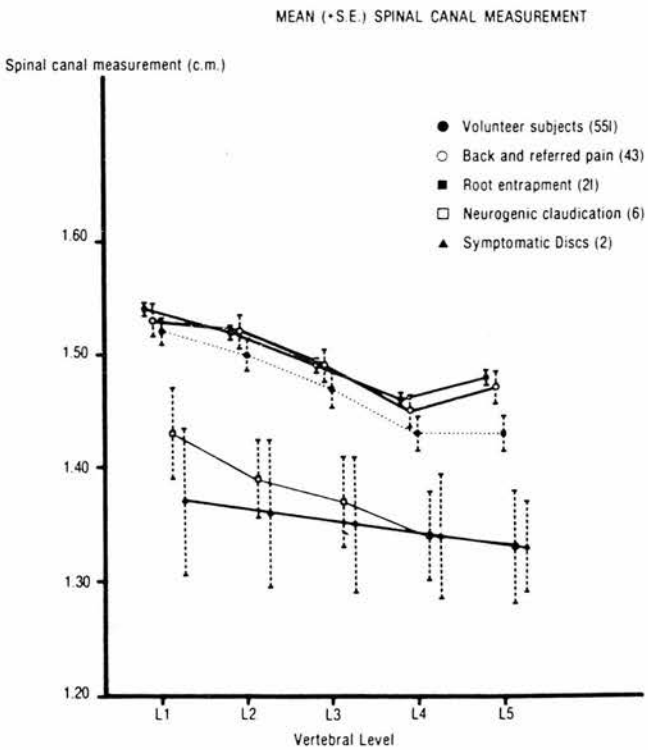

Fig 1. Mean canal measurements by ultrasound for patients with lytic defects and fulfilling criteria for the four back syndromes compared with mean measurements for 551 volunteer subjects.

Table 2. Comparison of Women With and Without Lytic Defects Related to Pregnancy

	Percentage who attributed onset of back pain to pregnancy
Women with lytic defects (49)	20.4%
Women without lytic defects (913)	6.6%

DISCUSSION

The incidence of 5.6% of bilateral lytic defects of the pars interarticularis recorded in patients attending a back pain clinic is probably no greater than the incidence of the defect in the general population. It could be suggested, within reason, that the demonstration of lysis is an incidental finding, unrelated to the cause of the back pain. Two facts, however, indicate that the defect is frequently significant as a factor in the development of symptoms. First, if the sex ratio of 3:1 recorded by Roche and Rowe[11] is correct for our population, then our ratio of 1.7:1 represents a higher proportion of women attending the back pain clinic than would be expected. The mechanical factors of pregnancy associated with joint laxity may explain why more women with lysis attributed the onset of their back pain to pregnancy and account for the greater proportion of women with lysis in the series than would be expected.

The second observation that would suggest that a lytic defect is not to be disregarded in assessing the pathogenesis of back pain, is the considerable difference in incidence of two of the back pain syndromes. Only two patients with lysis had sufficiently disabling root symptoms, tension signs and abnormal neurologic signs, to fulfill the criteria of a symptomatic disc lesion. It is not suggested that disc prolapse does not occur. Farfan[4] demonstrated that disc pathology not infrequently occurs at the level proximal to the lytic defect, as shown in the discogram of Figure 2. The infrequency of disc symptoms, however, may be associated with sagittal widening of the central canal as the lamina is left behind in the process of forward displacement. The canal then may be sufficiently deep for neurologic involvement to be avoided with a prolapse of a disc at L5–S1 or at L4–5. Alternatively, it may not be so much the size but the shape of the central canal that protects the patient with spondylolisthesis from severe disc symptoms. If the shape of the canal in unilateral spondylolysis[10] is similar in bilateral spondylolysis, then the trefoil-shaped canal with a deep lateral recess is unusual. There is confirmatory evidence from CT scanning, which demonstrates a large dome shaped canal in bilateral spondylolisthesis from attenuation and elongation of the pars (Figure 3). The patient with a lytic defect appears to be protected from the disabling symptoms of an acute disc prolapse. Negative findings tend to be neglected, but on reflection when a surgeon removes an extruded disc he rarely encounters the free-floating lamina of a spondylolisthesis.

In contrast to the infrequency of the symptomatic disc lesion, the patient with a lytic defect is more likely to attend with back and/or referred pain ($P<0.001$). It was the complaint of one-third of the patients presenting with lysis with full straight leg raising and no abnormal neurologic signs. The pain source is speculative, but the fact that bed rest generally relieves the pain, and that if the pain is sufficiently troublesome a fusion can be highly successful, it is not improbable that the pain source is associated with structures attempting to restrain unnatural segmental movement. This may be at the level of the defect or associated with disc degeneration at a more proximal level (Figure 4).

Fig 2. Discogram demonstrating pathological disc at L4–5 with a spondylolisthesis at L5–S1

The incidence of root entrapment was similar in patients with and without lysis. Degenerative changes at the site of the defect (Figure 5) could cause root symptoms. The average slip ratio for those patients with root entrapment syndrome was relatively small

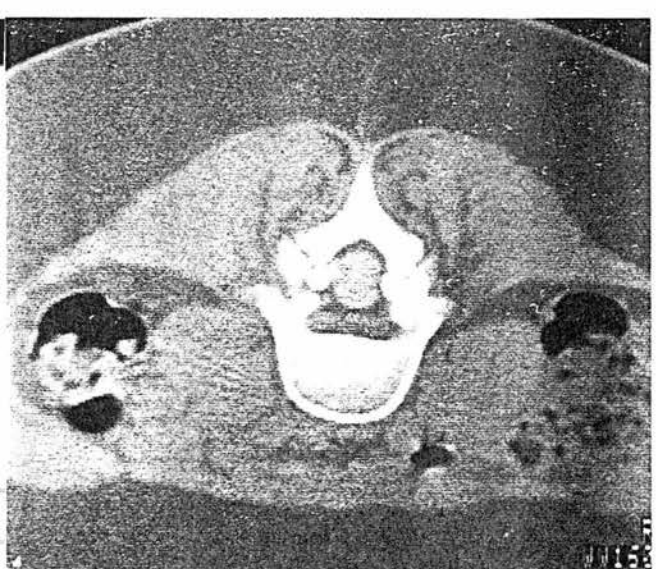

Fig 3. CT scan of L5 with bilateral defect showing dome-shaped spinal canal.

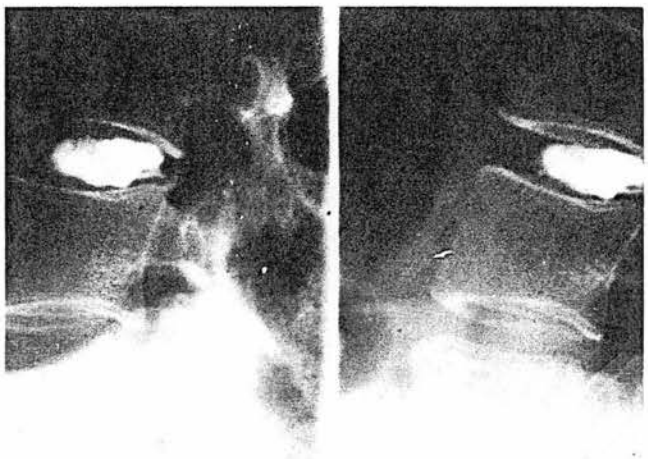

Fig 4. X-rays showing segmental movement at L4–5 with spondylolisthesis at L5–S1. Spine extended on the left, flexed on the right.

at 11%. It is possible that those patients with greater forward displacement and elongation of the pars avoided root involvement by the nerve and root displacing safely anterior to the defect. Wiltse et al[15] described impingement of the L5 root between the L5 transverse process and the ala of the sacrum when displacement approached 40%, but this was not encountered in this series.

In summary, it would appear that certain occupations, recreations, and pregnancy increase the risk of symptoms developing in a subject with a lytic defect of the pars, but in general a subject with lysis is no more likely to visit the hospital with back pain than the rest of the population. Indeed, should such individuals develop a disc prolapse, they appear to be protected from its acute disabling symptoms.

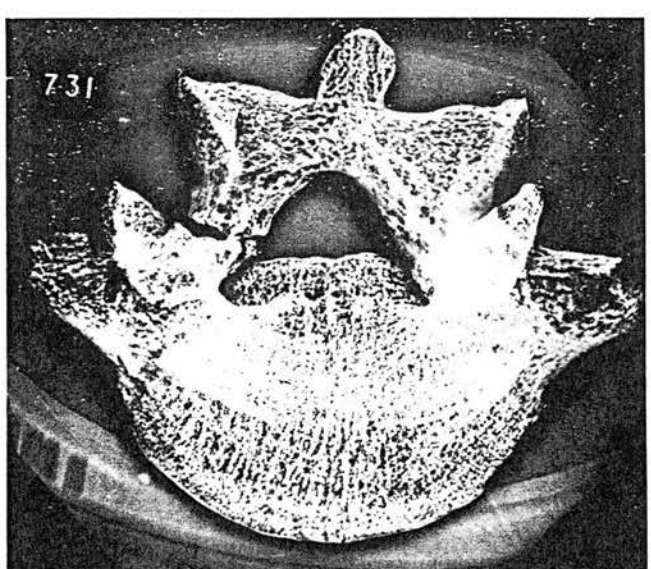

Fig 5. X-ray of specimen with unilateral spondylolysis at L5 showing degenerative change at the site of the defect.

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Address reprint requests to

R.W. Porter
Doncaster Health Authority
Doncaster Royal Infirmary
Arnthrope Road
Doncaster
DN 2 5LT
England

Accepted for publication June 20, 1983.

Clinical Biomechanics 1986; 1: 7–10 Printed in Great Britain

Back pain and neck pain in four general practices

Richard W. Porter, MD, FRCS
Catherine S. Hibbert, PhD
Doncaster Royal Infirmary, Doncaster, Yorkshire, UK

Summary

This study records the incidence with which married men and their wives attended four general practices with low back and neck pain.There was a higher attendance with low back pain, but not neck pain, for those men engaged in heavy manual work. Both men and women attending with back pain also attended more frequently with other conditions than those without back pain. A high proportion of the wives of the men who attended frequently were themselves low attenders.

There was a high correlation between the low back and neck pain attendance, especially amongst the men. There was a considerable variation in the methods of management of these conditions between the four practices.

Relevance

The inter-relationship between back and neck pain described here has both clinical and ergonomic implications but whether this is due to behavioural patterns, constitutional disability affecting spinal structures or other reasons, remains uncertain.

Key words: Epidemiology, Back pain, Neck pain, General practice

Introduction

An epidemiological study was carried out to determine the incidence with which married men and their wives attended four general practices with low back and neck pain. Factors were sought that might influence that attendance, and the relative use of consultant and radiographic services were compared in the four practices.

Method

The general practitioner records of the parents of all children born between September 1966 and August 1967 were examined from four general practices. Two of the practices were in mining villages within 10 miles of the town, and two were town practices.

The patient's age, occupation and number of years covered by the practice records were noted, together with the number of entries for all conditions, including neck pain and back pain. The need to be off work with these conditions and the use made of the hospital services was recorded.

Results

The mean age for the 393 men was 43·3 years, SD 4·5 years, and was not significantly different for the four practices (Table 1). There were approximately 100 men from each practice, with records available for a mean time of 21·2 years, SD 4·7 years.

A breakdown of the patients' occupations (Table 2) suggests that the two mining practices were comparable with a similar proportion of miners, and the two town practices had comparable numbers of light manual and sedentary workers.

Of the men from mining practice A 81 per cent had at some time consulted their practitioner with low back pain (Table 3), compared with 65 per cent in mining practice B. A higher proportion of those attending with back pain in the mining practices were certified unfit for work than in the town practices. There was a relationship between the incidence of attendance for back pain and the type of occupation (Table 4). The mean attendance per year (first and follow up attendances) for all conditions is shown in Table 5, with a more than twofold difference between mining practice A and town practice B.

Submitted: November 1984
Correspondence and reprint requests to: R W Porter MD, FRCS, Department of Orthopaedics, Doncaster Royal Infirmary, Doncaster DN2 5LT, England.

It is of interest that there was a significant difference between the non-back pain visits per year for those men with back pain and those without in all of the four practices (Tables 6 and 7), reaching considerable proportions in the two mining practices. There was a threefold difference in orthopaedic referral for the two town practices (Table 3).

The probability of a man attending with neck pain if he had had back pain was eight times greater than if he had not had back pain. Although the back pain attendance was related to the demands of occupation, the incidence of attendance with neck pain was much the same in each occupational group (Table 4).

We were not able to establish that all the events occurred during married life but the number of wives attending with back pain varied from 26 per cent to 63 per cent over a mean period of 24·3 years of records (Table 8), and again the use of radiological and orthopaedic services varied considerably.

The women with back pain in each practice behaved in a similar manner to the men, attending more frequently with other complaints than the women without back pain (Table 9). The wives of miners did not attend with back pain more frequently than the wives of other workers. The wives of those miners who had attended more frequently than the mean attendance rate, attended less themselves with back pain (34 per cent of 32 wives) than the wives of those men whose back pain caused them to attend less than the mean (54 per cent of 56 wives). This was only significant at the 0·08 level. There was a highly significant polarization of attendance rates between husband and spouse for all conditions irrespective of back pain, in mining practice A and town practice B, the wives of the low-attending men visiting the practices 1·5 times per year more often than the wives of the high-attending men (Table 10).

Table 1. Mean age and number of years covered by the practice records

	Number of men	*Mean age*		*Mean years covered by the practice records*	
		Husbands	Wives	Husbands	Wives
Mining practice A	96	43·2 (SD 6·6)	40·3 (SD 6·1)	21·8 (SD 6·1)	24·4 (SD 7·0)
Mining practice B	108	43·1 (SD 5·6)	40·4 (SD 5·5)	21·0 (SD 7·4)	24·0 (SD 5·0)
Town practice A	97	42·9 (SD 7·9)	40·5 (SD 4·8)	20·3 (SD 7·6)	23·9 (SD 8·8)
Town practice B	92	44·2 (SD 5·9)	40·8 (SD 5·7)	21·8 (SD 7·0)	24·3 (SD 5·9)

Table 2. Percentage distribution of occupations of the men in the practices

	Miners	*Heavy manual*	*Light manual*	*Semi-sedentary*	*Sedentary*	*Unemployed*
Mining practice A	41	8	22	8	10	10
Mining practice B	45	20	21	3	6	5
Town practice A	4	14	39	23	12	7
Town practice B	1	21	35	29	11	3

Table 3. Incidence of attendance for men with back pain and absenteeism in the practices.

	% attended with back pain	*% of those attending with back pain who were off work*	*% of men with back pain having spinal radiograph*	*% of men with back pain having orthopaedic referral*
Mining practice A	81	90	44	24
Mining practice B	65	83	23	21
Town practice A	62	65	33	13
Town practice B	50	46	37	35

Table 4. Incidence of neck and back pain attendance in occupational groups

	Number	*Percentage attending with:* Back pain	Neck pain and no back pain	*Neck pain and back pain*
Miners	93	82	2	22
Heavy manual	63	81	3	16
Light manual	114	56	2	18
Semi-sedentary	60	50	7	12
Sedentary	38	39	11	13

Table 5. Mean attendance per year of men with all conditions

	Mean attendance per year for all conditions	*Significance of difference*
Mining practice A	5·7 (SD 4·1)	
Mining practice B	5·5 (SD 4·9)	$p<0·001$
Town practice A	3·7 (SD 2·4)	$p<0·001$
Town practice B	2·3 (SD 2·1)	$p<0·001$

Table 6. Comparison between mean attendances for conditions other than back pain for men with and without back pain

	Men with *back pain* Mean attendance per year for conditions other than back pain	*Men* without *back pain* Mean attendance per year with other conditions
Mining practice A	5·57 (SD 3·98)	3·22 (SD 2·47)
Mining practice B	6·01 (SD 7·67)	3·65 (SD 2·71)
Town practice A	3·34 (SD 2·45)	2·22 (SD 1·87)
Town practice B	2·57 (SD 1·85)	1·74 (SD 2·08)

Table 7. Mean attendance per year for conditions other than back pain

	Men with *back pain*	*Men* without *back pain*
Miners ($n=93$)	4·99 (SD 1·26) $n=76$	2·26 (SD 1·8) $n=17$ sig. ($p<0·001$)
Heavy manual workers ($n=63$)	3·34 (SD 1·90) $n=51$	2·91 (SD 1·72) $n=12$ not sig. ($p=0·36$)

Table 8. Incidence of wives attending with back pain and use of hospital services

	Attendance of wives with back pain %	*% of wives with back pain referred for radiography*	*% of wives with back pain referred for orthopaedic opinion*
Mining practice A	54	40	26
Mining practice B	26	8	6
Town practice A	63	22	17
Town practice B	39	38	29

Table 9. Comparison between mean attendances for conditions other than back pain for women with and without back pain

	Women with back pain	*Women without back pain*
Mining practice A	4·06 (SD 2·00) $n=58$	2·84 (SD 1·39) $n=49$
Mining practice B	3·51 (SD 1·72) $n=36$	2·74 (SD 1·51) $n=100$
Town practice A	4·77 (SD 2·67) $n=69$	3·29 (SD 2·45) $n=41$
Town practice B	3·93 (SD 2·63) $n=42$	2·81 (SD 1·45) $n=67$

Table 10. Comparison of the incidence of attendance for all conditions for the wives of men who attend above and below the mean attendance rate

	Attendance rate for all conditions			
	For men *above* the mean attendance rate	For their wives	For men *below* the mean attendance rate	For their wives
Mining practice A	9·18 (3·57)	2·93 (1·84)	2·78 (1·06)	4·57 (2·01)
Mining practice B	6·79 (4·76)	2·96 (1·45)	3·98 (2·01)	3·43 (1·98)
Town practice A	4·68 (2·25)	3·35 (2·28)	2·01 (1·01)	4·26 (2·80)
Town practice B	3·40 (2·29)	2·65 (1·02)	1·34 (0·97)	4·04 (1·18)

Discussion

The pattern of general practitioner attendance does not quantify accurately the incidence of low back and neck pain in the community. A retrospective study has its limitations, and we are recording only a symptom, not a diagnosis, but attendance does reflect a measure of the problem[1]. The reasons for attendance will vary with the demands of the patient's occupation, probably with their expectations from their general practitioner, and with the doctor/practice management. Nevertheless, the variable attendance in different practices and in men of different occupations is of interest, in a problem which is common and of economic importance.

Both the occupation of the patient, and the management of the practice appear to influence the attendance of men with back pain. The patient's occupation certainly affected attendance, there being a gradually reducing incidence of attendance from miners and heavy manual workers to the semi-sedentary and sedentary workers. However, men from both mining practices, well matched for occupation, had a considerably different incidence of attendance, 81 per cent compared with 65 per cent. It could be argued that the 1·67 m seams in the colliery of the former village were more stressful to the back than the lower 1·2 m seams in the second village where a man must work on his knees. However, the incidence of their wives attending with back pain was also different, 54 per cent compared with 26 per cent, and thus either the management of the practice or the ethos of the village was probably significant.

The ability to manage patients with back pain within the practice varied between the two groups. The radiological referral rate differed almost twofold between the two mining practices, and the difference between the orthopaedic referral rate in the town practices was even more striking. A comparative audit on practice management could change referral habits, protect some patients from unnecessary investigations and save hospital resources.

As a group, both men and women with back pain attendances also attended more frequently for other conditions than patients without back pain. There is no evidence that patients attending a general practice with acute back pain have a neurotic personality[2] but Becker and Karch[3] noted this high attendance with other conditions in North American women. We observed it in all four practices, and especially in the miners with back pain. The 76 miners with back pain attended with other conditions more than twice as often as the 17 miners without back pain. It suggests that some men are able to cope less effectively with the unattractive working conditions of the mine than others. It may be possible to identify such individuals prior to entry into the industry to their advantage and to that of their employers. They may be individuals who tend to somatize adverse circumstances[4] and experience higher pain ratings for externally produced pain of different origins than non-back pain sufferers[5]. Imprinting of abnormal illness behaviour will have been present from early life[6,7] and it could be recognizable from a careful history, a psychological profile and perhaps a record of school absenteeism.

The reason for both men and women with back pain attending more frequently with neck pain than non-back pain sufferers, could also relate to a behavioural pattern. Alternatively, they may have a constitutional disability affecting the intervertebral disc, or a tendency to develop degenerative change or similar morphology of the spinal canal.

The similar incidence of attendance with neck pain in each occupational group suggests that occupational factors are less obvious than for back pain.

The wives of those miners attending most frequently with back pain attended least themselves. They may have experienced back pain, but failed to attend with this complaint. Attendance rates for all conditions also tended to polarize between husband and wife. One partner may be influenced by the behaviour of the spouse, or by the failure of the spouse to respond to treatment. Contrasting patterns of illness behaviour may have been present prior to marriage, but whatever the cause, it awaits an explanation.

This is essentially a pilot study, but it does raise interesting questions about illness behaviour and the inter-relationship between back and neck pain. These can only be resolved by prospective investigation.

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Back Pain and Trunk List

R. W. PORTER, MD, FRCS, FRCSE and C. G. MILLER, BSc

association of trunk list with back pain was recorded in 0 patients, 5.6% of those attending a back pain clinic. vice as many patients listed to the left as to the right. A al of 49 patients fulfilled the criterion of a symptomatic mbar disc lesion, and 20 required surgical excision of the sc. The side of the list was not related to the side of the iatica nor to the topographic position of the disc in rela- n to the nerve root. There is some evidence that the side the list may be related to hand or leg dominance. [Key rds: lumbar disc herniation, trunk list, location of lesion]

THE ASSOCIATION OF trunk list and back pain is well recognized but poorly understood. In 1881 Remak[10] described alternating lumbar scoliosis, and in 1933 Capener[2] re- rded three more patients with "alternating sciatic scoliosis." anco[1] (1968) stated that the list was often associated with a disc otrusion, that the center of gravity often shifted from the side of lesion, but not invariably, and that it could alternate. Patzold et (1975) suggested that the list was related to the topographic sition of the protrusion, the root easing away from the disc, and s was supported by Kramner[5] (1981) and Scott[11] (1983). Grieve[3] 83) suggested that the "wind swept spine" was largely mediated psoas spasm.

Some patients have a list that is obvious when standing, whereas ers have a list only when flexing forward. Some have a recurrent that disappears as the symptoms settle, and others have a list t still persists after the back pain has resolved.

n an attempt to understand more of the mechanism of trunk list d back pain, we examined 100 consecutive patients who had a list ociated with back and/or leg pain, excluding those who had a list y when flexing forward, those with a short leg, and those with a uctural lumbar scoliosis.

ETHODS

One hundred consecutive patients who had both a trunk list and k pain were selected from a group of 1,776 patients attending a k pain clinic. The list was present when standing and was abol- ed by lying down (Figure 1). Not included were patients who had asurable leg shortening or a structural lumbar scoliosis, or who ed only when flexing forward.

The side of the trunk list was compared with the following clini- measurements: the presence of leg pain, its laterality and distribution; hyporeflexia, sensory loss, motor weakness, muscle wasting; straight leg raising; hand dominance; leg dominance (whether the patient crossed the right leg over the left or vice versa); failure of conservative management requiring surgical disc excision; the segmental level of the disc excision; and the topographic position of the disc protrusion, noted at surgery, in relation to the root.

RESULTS

A gravity-induced list was recorded in 100 of 1,776 patients (5.6%) attending a back pain clinic. The mean age of the patients was 38.0 years (± 10.1 yrs). Sixty-two percent were men. A total of 66 patients listed to the left, 31 to the right, and three had an alternating list, varying the side at will (Figure 2).

The majority of patients complained of leg pain as well as pain in the back. Sixteen had back pain only, 13 had pain in the thigh but not below the knee, and 71 had pain in a root distribution below the knee. There was no correlation between the side of the list and the side of the sciatica (Table 1).

Forty-nine patients fulfilled three or more of the criteria described by McCulloch[6] (1977) for symptomatic lower-lumbar disc lesion: unilateral leg pain in a typical sciatic root distribution below the knee; specific neurologic symptoms incriminating a single nerve; limited straight leg raising by at least 50% of normal; at least two neurologic changes of muscle wasting, muscle weakness, sensory change of hyporeflexia; and radiculographic evidence of disc protrusion.

Twenty patients required surgical excision of a disc. Twelve patients underwent excision at L5–S1, six at L4–5 and two at L3–4 (Figure 3). In these patients the side of the list was not related to the side of the disc protrusion (Figure 4), nor was the direction of the list related to the topographic position of the disc and the root (Figure 5). In five patients the direction of the list caused the root to lean toward a lateral disc protrusion.

The only four left-handed patients in this series all listed to the right. Patients who were most comfortable crossing their left leg over the right listed equally to the left and right, whereas those who crossed the right leg over the left tended to list to the left (Table 2).

DISCUSSION

An interesting feature about the trunk list demonstrated by many patients with back pain is that it is related to gravity. In contrast to patients with a structural lumbar scoliosis and patients with deformity and infection or osteoid osteoma (Kirwan et al.[4] 1984), the list of the patients in the current series was abolished by lying down. Listing patients did not have scoliosis when radiographs were taken in the supine position. Similarly, the list was abolished when a

rom the Doncaster Royal Infirmary, Doncaster, England.
upported by the National Coal Board.
ubmitted for publication December 28, 1984 and revised May 13, 1985.
he authors thank Mr. G. Swann, Doncaster Royal Infirmary, for the

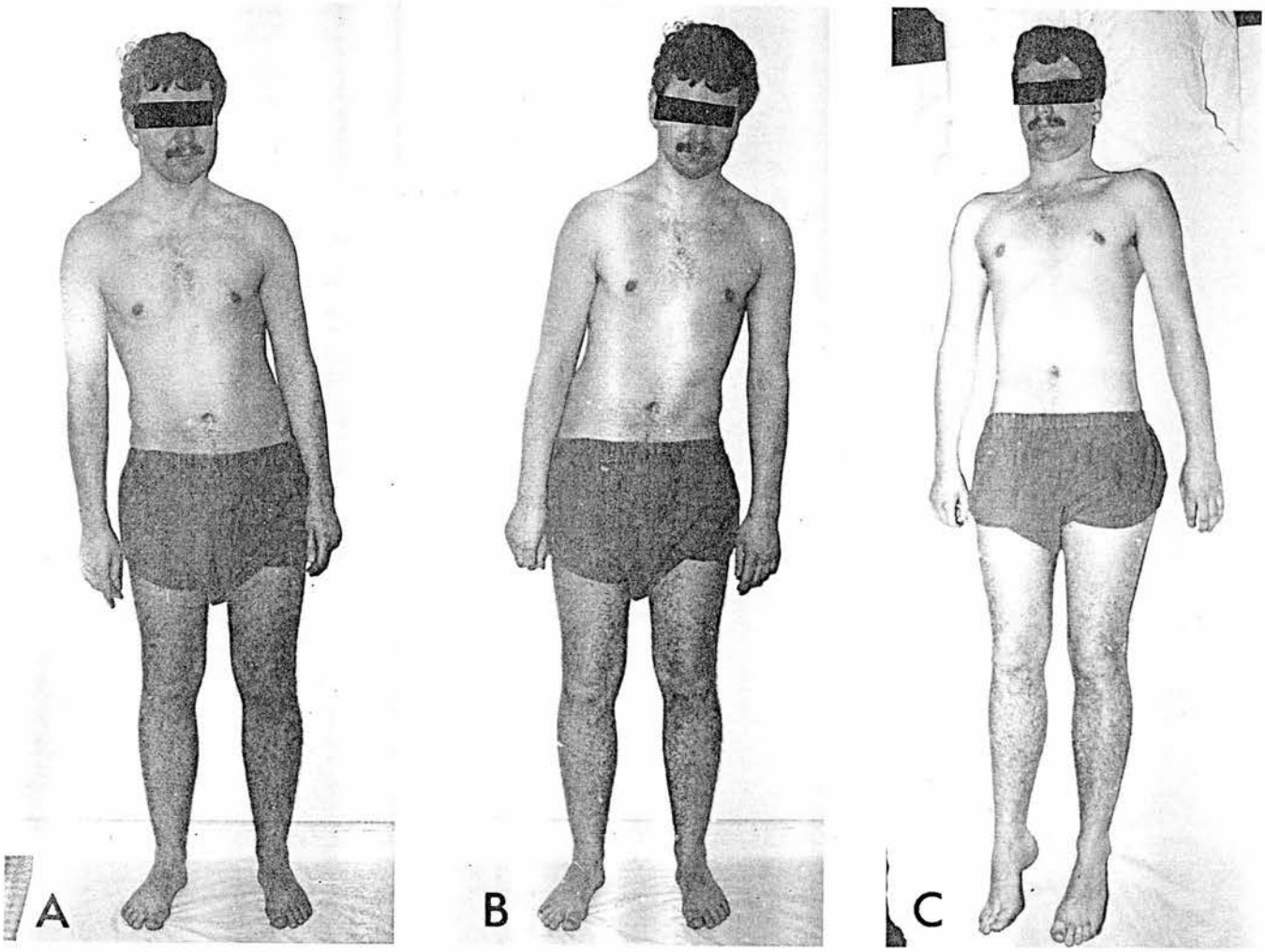

Fig 1. Patient with an alternating list, listing first to the **A,** right and then to the **B,** left at will. **C,** He could correct the list only by lying down.

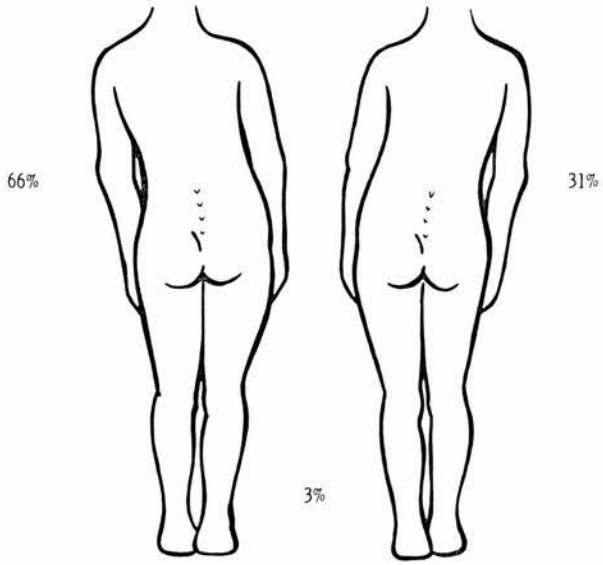

Fig 2. A total of 66% of patients listed to the left, 31%, to the right, and 3% had an alternating list.

It is difficult for the patient voluntarily to correct the list when standing. Even when the ipsilateral leg is raised from the floor and the patient stands on the contralateral leg alone, elevating the pelvis and contracting the psoas (Figure 7), the center of gravity moves to the midline, but the scoliosis remains (Figure 8). The mechanism by which gravity induces this list is obscure.

Table 1. Relationships Between the Side of the Trunk List and the Side of the Sciatic Pain in 67 Patients Who Had Unilateral Leg Pain Below the Knee.

	Number of patients listing to the left	*Number of patients listing to the right*
Left sciatic pain	18	10
Right sciatic pain	26	13

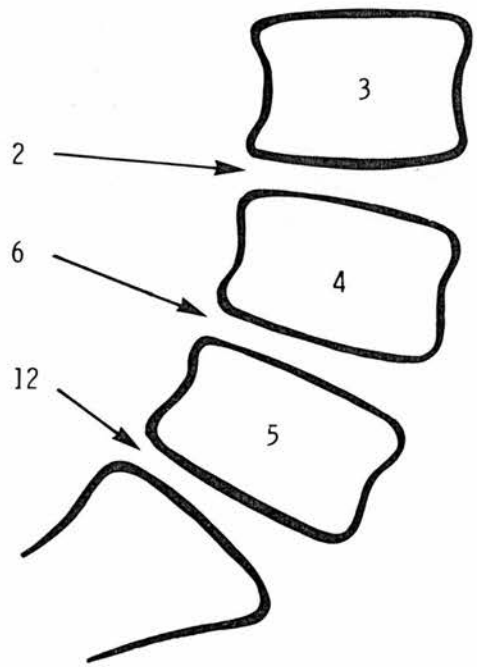

Fig 3. The vertebral level of disc excision in 20 patients who had a list.

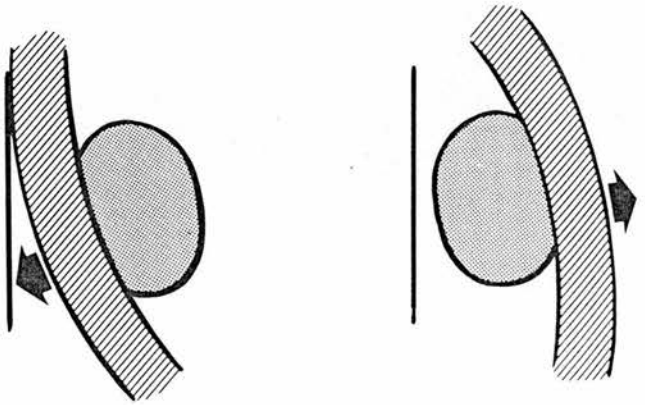

Fig 5. Diagram, to show how one might expect the root to ease away from the disc protrusion by the direction of the list, but this was not invariably observed.

Table 2. Relationships Between the Side of the Trunk List and Hand Dominance and Crossing Right Leg over the Left or *Vice Versa* in 47 Patients

	Number of patients listing to the left	*Number of patients listing to the right*
Right handed	34	9
Left handed	0	4
Comfortable crossing right leg over left leg	23	4
Comfortable crossing left leg over right leg	5	5
No preference	6	4

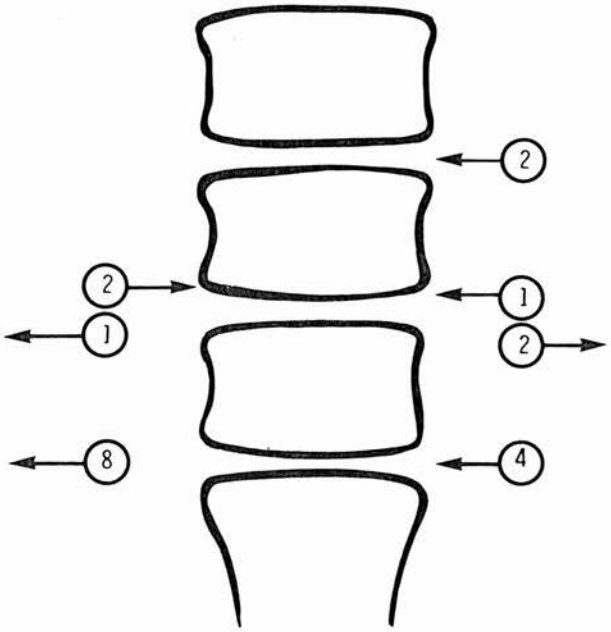

Fig 4. The relationship between the direction of list and the vertebral level and side of the disc herniation in 20 patients requiring surgery. Only four patients listed to the right, two with right-sided L4-5 discs and two with left-sided L4-5 discs.

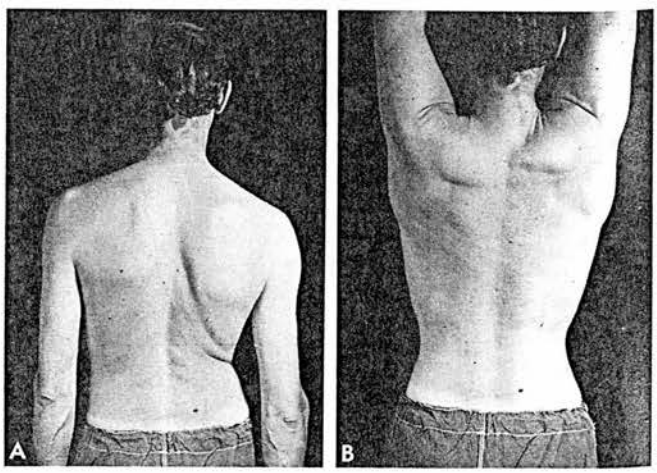

Fig 6. A, Patient unable to correct a list to the left when standing. **B,** List is abolished when patient hangs from a bar.

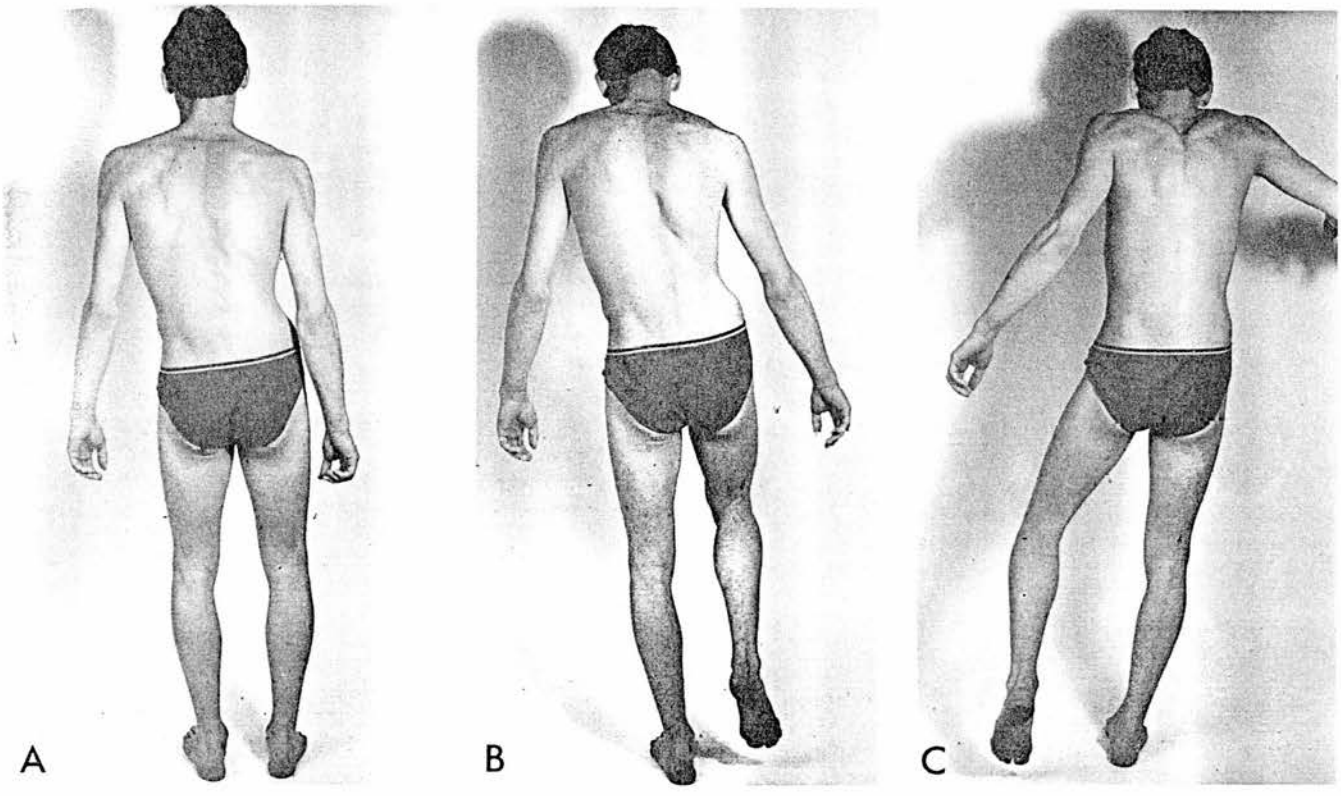

Fig 7. A, A patient lists to the left. **B,** The list remains when patient stands on the left leg. **C,** When standing on the right leg, he can apparently correct the list. The center of gravity moves over the right leg.

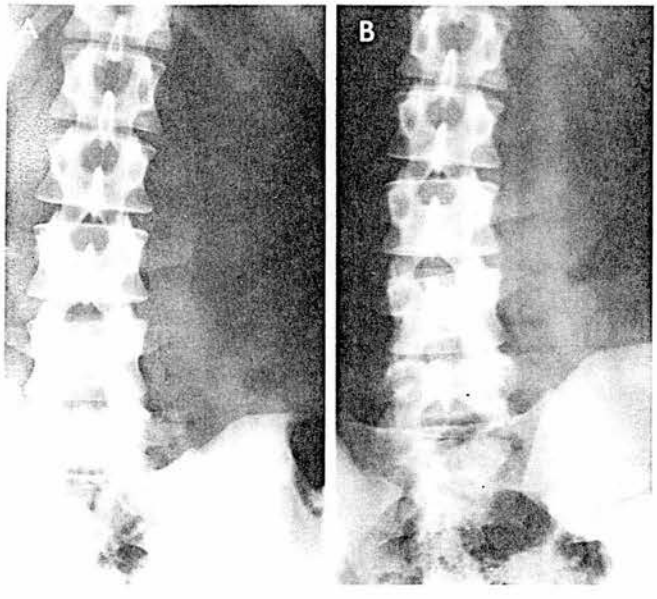

Fig 8. A, Radiograph of patient listing to the left when standing. The center of gravity is over the left sacroiliac joint. **B,** A radiograph of the patient standing on the right leg, when the list appears to be corrected clinically, shows that although the psoas is contracted, the left pelvis raised, the center of gravity shifted, the scoliosis remains.

There is little doubt, however, that a gravity-induced list is generally associated with a disc protrusion. Almost one-half of our patients fulfilled the strict criterion of a symptomatic disc lesion, and many more had symptoms suggestive of a disc lesion. The patients' sex and age distribution was similar to that of other patients with disc protrusion. We have yet to explain why only a portion of patients with disc herniation list, why it is not invariably associated with root symptoms, why the list can persist when symptoms have settled, why some patients list only as they flex forward, and why one list may correct rapidly after surgery and another may be delayed.

Patients who have a list and symptoms of back pain alone may have disc protrusion that affects nerve-sensitive structures yet spares the root. Their limited disability prohibits extensive investigation.

None of our listing patients had an isthmic spondylolisthesis, perhaps because disc symptoms are rare in patients with this pathology.[9]

A list is associated with a poor prognosis for conservative management. Twenty (40%) of our 49 patients fulfilling McCulloch's criteria subsequently required surgical excision of the disc compared with a surgical incidence of 5% for nonlisting patients fulfilling these criteria.

It has been suggested previously that the direction of the list was related to the side of the sciatica and the position of the disc in

relation to the root, but we found no evidence for this. We suspected that the strong lumbosacral ligament would prevent a list with disc lesions at L5–S1, but in fact most of our surgical excisions for listing patients were carried out at the lumbosacral level.

Twice as many patients list to the left as to the right, although the disc is no respecter of laterality. We must, therefore, seek factors other than the site of disc protrusion to explain the side of list. Observations on hand and leg dominance suggest that this may be significant in determining the direction list.

The mechanism of this common sign is obscure, but it does deserve more of our attention as we attempt to understand both the pathogenesis of disc symptoms and the mechanism whereby list and symptoms are sometimes corrected by manipulation (McKenzie,[7] 1981).

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Address reprint requests to

R.W. Porter, FRCS
Consultant Orthopaedic Surgeon
Doncaster Health Authority
Doncaster Royal Infirmary
Armthrope Road, Doncaster
DN 2 5LT

Accepted for publication August 5, 1985.

Clinical Biomechanics 1986; **1**: 216–221 Printed in Great Britain

Review Papers

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Back pain; the risk factors and its prediction in work people

J L Dales*
E B MacDonald‡, R W Porter§
*Department of Community Medicine, Guy's Hospital School of Medicine, UK
‡Medical Department, IBM (UK) Ltd, Portsmouth, UK
§Orthopaedic Department, Doncaster Royal Infirmary, UK

Introduction

Low back pain is a problem of considerable magnitude in industry[1]. It occurs in about 80% of the population and in the majority during their working life[2]. The epidemiology of back pain in and between various industries has been well studied and many of the high risk occupations are known[3–14]. The prevalence of back pain differs only marginally between 'heavy' and 'light' occupations[15,16]. Thus Hult found that the prevalence of low back symptoms in Sweden was 64% in 'heavy' occupations and 53% in 'light' occupations, but the work-handicap was twice as great in the heavy occupations[17,18]. In a survey of low back pain among office workers and manual workers in the Volvo company, there was little difference in the incidence of low back pain, but the manual workers had a significantly longer period of disability during both initial and subsequent episodes of low back pain[19]. In a comparative study of nurses and teachers in Dundee, little overall difference in prevalence was found but nurses complained earlier of low back pain and it was considered work related, while that which developed in teachers was more age-related and non-occupational[20].

Industries in which there is a high work-handicap from back pain include iron and steel mining, forestry, dock work, nursing and driving[3,4,5,7,8,11,12,13,17].

The precise diagnosis and underlying pathophysiology of back pain is often difficult to establish[21]. Most back pain symptoms, though, are acute rather than chronic, with four out of five workers with a low back injury returning to their jobs within about 3 weeks[3,22,23]. A Lancet editorial[24] has suggested that the 'advantages of the treatments have been marginal at best', and in fact treatment is largely irrelevant when most will recover spontaneously[25]. However, the value of different treatment strategies for the individual sufferer is not denied. The magnitude of the problem, the economic cost, and our ineffectual remedies make the quest for prevention imperative for the employer. This is normally attempted using a 'blunderbus approach': i.e. the combined tactics of pre-employment screening, training of workers in manual handling and application of ergonomic principles to task design[26,27]. Common sense suggests the value of training, but it has not shown proven benefit[28–31] and our experience is that the manual worker will not respect his spine until it produces pain. It is then, albeit too late, that advice on safe handling is heeded. Although our ultimate aim should be to eliminate, by correct design, the risk to the back in all occupations, this too is a counsel of perfection and there are many industries, such as mining, where it is very difficult to make all tasks ergonomically appropriate. The purpose of this review is to consider the first option: pre-employment examination. We describe some individual factors which are thought to be important in the development of back pain, how they can be measured and how they may be used to identify susceptible individuals.

Correspondence and reprint requests to: R W Porter MD, FRCS, Department of Orthopaedics, Doncaster Royal Infirmary, Doncaster DN2 5LT, UK.

Prediction of back pain

There is no single test and as yet no established procedure which has been used to identify potential back pain sufferers at the pre-employment stage, perhaps because back pain is a symptom of many different pathologies, each of which has a multifactorial aetiology[21]. However, the considerable advances in recent years have increased our knowledge of the relevant risk factors which leads us to suggest that techniques may become available to begin to predict those individuals who are most at risk from back pain in heavy occupations.

History

It has been shown that an important indicator of future back pain is a history of previous back pain[14,32,33].

Those returning to work after an absence of 5–6 weeks, after absence caused by falls, those with persisting back pain, especially affecting the legs, and those with restricted straight leg raising, weakness of hip and abdominal flexors, have a poor prognosis[34]. Unfortunately, prediction at this time is really one step too late and only has short term reliability.

Our own family history studies of 50 patients after discectomy, with 50 matched subjects who have never experienced back pain, and whose vertebral canals were below the 30th percentile when measured by ultrasound suggest that there is a familial association in patients with disc protrusion[35]. Forty-six of 192 first degree relatives of the discectomy patients had 'significant low back pain' compared with 19 of 155 first degree relatives of the control subjects ($\chi^2=6{\cdot}39$, $p<0{\cdot}02$). We were unable to identify any difference in the HLA antigens of the two groups.

Family history and perhaps genetic or biochemical indices may prove relevant as fundamental research continues[36].

Anthropometry

In a study of military recruits who subsequently developed lumbar disc lesions, Hrubec and Nashbold[37] showed that the relative risk of a lumbar disc lesion was 2·3 ($p<0{\cdot}001$) in men over 182 cm in height and was only 0·4 ($p<0{\cdot}001$) amongst the shortest. Tauber[38] found that tall men in an American steelworks accounted for a disproportionately large part of sickness caused by back pain.

Merriam et al.[39] found that back pain patients were taller than a control group, and were able to show that the difference was explained by their greater mean 'pelvic height'. There is good circumstantial evidence that tall men are at greater risk of back injury.

Lawrence[10] has reported that in the mining industry tall miners are at disproportionately greater risk. The combined evidence of intra-discal pressure increasing by 50% when the neck is flexed forwards[40], the association between back injury and frequent bending, and the study of Ridd[41] who showed that a 90% restriction in headroom reduced maximum lifting capacity by 50% suggests that posture is probably the important factor. It is particularly relevant in those occupations where there is restricted headroom or much bending. We observed a greater general practice attendance with back pain in a mining village where the seam was 1·67 m compared with a second village with a 1·2 m seam[42]—men crawl in a lower seam and suffer less spinal stress than from stooping in a higher seam. Weight and body build, however, have not been shown to have any consistent association with back pain experience[43–47].

Measurement of spinal flexibility and posture have not been shown previously to have any predictive value[48–52], but recent developments in the techniques for their assessment, such as biplanar radiography, have renewed interest in these areas[53,54,55].

Strength

Isometric strength relative to job demands has been shown to be a risk factor for both spells and severity rate of back pain absence[56,57]. However, no ready made strength test exists which has met with general approval and ideally each job component would require its own test[58]. Many different tests have been described[47,59,60]. Isometric muscle strength tests measure maximal force, which can be maintained for just a few seconds, yet for continuous work only about 20% of this maximum strength can be sustained[61]. Karwowski[62,63] has described preliminary work on a reconciliation between the 'biomechanical' and 'physiological' strength capabilities which is potentially interesting.

Lifting capacity should probably be assessed by dynamic techniques[64]. Kroemer[65] discussed the difficulties of dynamic strength assessment and proposed a new dynamic psycho-physical test called 'Liftest'. Subjects lift a weight from floor to overhead reach height within a fixed frame. He found good repeatability with this test (average coefficient of variation 3·5% compared with up to 13% in isometric strength tests). We found a coefficient of variation for the maximal overhead lifts to be 2·6%, confirming the relative accuracy of the technique[66].

The Liftest procedure is at present being assessed as an effective predictor of lifting capability on the job. Other tests, both simple and sophisticated, have been developed[47,55,67,68]. Kamon[69] presented a dynamic procedure for assessing lifting capacity by pulling, in a lifting maneouvre, a rope wrapped round a pulley preset to run isokinetically. Isokinetic dynamometers such as the Cybex are in general research use for the assessment of the strength of muscle groups in the trunk[70,71,72], but the procedures involved require elaborate test rigs, expensive equipment and are more appropriate to the research laboratory than the occupational health department. If successful, however, the more simple tests could be readily used in industry.

Simple measures of fitness deserve further evaluation following the work of Cady et al.[73]. They found that on follow up of 1652 firefighters, the men in the best physiological condition had suffered fewer spells of back pain absence. Whether general fitness has any predictive value or indeed relevance to the average industrial employee is still not known.

There is some evidence that fitness and strength protects an individual from disc rupture. Our data comparing the proportion of male attendances at hospital with criteria suggesting disc protrusion showed a significantly lower proportion of miners than men from other occupations amongst all back pain clinic attenders ($p<0{\cdot}01$). This was even more marked when examining men admitted with this diagnosis, and very few miners required discectomy (see Table). It suggests that the heavy manual work of mining in some way protects the annulus from serious failure and posterior protrusion, or conversely, light work confers a risk of

Table. Back pain in male adults and miners in a hospital catchment area over 3 years

Sample	*Number*	*% Miners*
Time Averaged Mean of the adult, male population, employed and in the hospital catchment area	~80 000	~21
Male back pain clinic attenders	1422	31
Men with criterion of disc protrusion	250	20
Men admitted with symptoms of disc protrusion	70	14
Men undergoing surgery for disc protrusion	33	9

annular disruption. It supports Chaffin's work that an individual whose strength is not adequate for the task has a spine at risk certainly of disc protrusion, and men should not suddenly be expected to perform heavy manual tasks who have previously been in sedentary work[74].

Vertebral canal

A small vertebral canal, or narrowing of the canal is implicated in some cases of back pain, particularly spinal stenosis[75,76,77]. The vertebral canal can be measured by ultrasound, which is well suited for screening purposes, being safe, non-invasive and relatively quick to perform[78]. The repeatability of the lumbar vertebral canal measurement by this technique has been estimated between ±0·3 and 1·0 mm[79,80] compared with a range in the A–P diameter of about 11–22 mm[81]. The technique, however, requires considerable operator training[82,83] and access to a diasonograph scanner. Our studies of the relationship between the size of the lumbar vertebral canal and sickness absence due to back pain showed that the 10% of 50-year-old miners with the narrowest canals accounted for 32·5% of sickness absence due to back pain over a 3 year period[84]. From the data in the study it can be hypothesized that substitution of the men in the narrowest decile by men with the back pain experience of those above would have reduced the overall morbidity due to back pain by approximately 25%. Measurement of the vetebral canal has not yet been used in screening but prospective studies designed to assess the importance of this technique are proceeding.

The CT scanner is capable of clearly displaying both the bony canal and soft tissues[85] but the radiological dose and great expense have largely prohibited its use, even in preventative research.

Observations by Kornberg and Rechtine[86] have shown that the mid-sagittal diameter and cross-sectional area of the vertebral canal were considerably smaller in symptomatic, and surgically treated, subjects compared with asymptomatic individuals.

The nuclear magnetic resonance (NMR) scanner, being non-ionizing, is potentially a valuable imaging device for the spine[87], but at present is prohibitively expensive.

Radiological abnormalities

In the past, radiology of the spine has been used extensively, particularly in the USA, as part of a pre-employment screening procedure[88,89], but critical review of the value of radiographs of the spine led the NIOSH and American Conference of Radiologists to conclude in 1973 that it was, 'of little assistance in predicting future trauma or disability from on-the-job stress'[90,91]. Montgomery[92] concluded, after reviewing many of the large studies performed, that there was no evidence that congenital or developmental abnormality predisposed to an increased incidence of low back injury. The lack of specificity[93], and the radiation dosages involved[94] lead us to suggest that there is no place for radiology of the spine in healthy individuals as part of pre-employment screening.

The demonstration of isthmic spondylolysis or spondylolisthesis is sometimes viewed as a serious risk factor, but this is not our experience. We observed 131 patients with lysis of the pars interarticularis in 2360 patients attending a first referral hospital back pain clinic[95], an incidence no greater than the incidence in the general population[96]. The lysis appears to widen the vertebral canal and protect the patient from cauda equina symptoms, though as a population they are more prone to symptoms of back and referred pain. The demonstration of a lytic pars without symptoms should not affect a man's employment.

Personality and mental illness

Much attention has been focused on psychological and psychiatric illness in back pain sufferers. Indeed from hospital or general practitioner surveys, increased anxiety, neurosis, depression and heightened somatic awareness have been found in back pain populations, but this has not been the case in industrial studies[97–101]. No psychological characteristics have yet been demonstrated which are associated with increased tendency to future back complaints[102,103]. Main[104] has summarized some of the problems of psychometric testing and considered it notoriously unreliable and difficult to apply.

Attempts have been made to identify individuals with recent onset of symptoms who will tend to chronicity[105–109]. There is potential, through prompt treatment and rehabilitation, for reducing disability and work handicap if good predictive indices can be established. However, we do not think there is yet support for psychological screening at the pre-employment stage.

Conclusion

There is no single test which can adequately define an individual's susceptibility to back pain. Strength and vertebral canal diameter measurement have been assessed independently of each other and have been shown

to be related to morbidity. They have not been considered together and it is possible that relevant strength testing in conjunction with vertebral diameter measurement may improve our ability to predict the individual at risk and thus reduce morbidity by appropriate job placement.

Many involved in research into back pain have suggested that the solution must be the correct design of the working environment rather than by identifying individuals at risk[110], but while the problem remains so vast, we must explore the potential of prediction. We suggest that suitable pre-employment screening tests should include a measurement of the lumbar vertebral canal diameter, strength and a consideration of other factors such as height or pelvic height, past history, and possibly family history. The relative importance of each of these requires further investigation and prospective studies are urgently required.

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Clinical Biomechanics 1987; **2**: 196–198 Printed in Great Britain

Does hard work prevent disc protrusion?

Richard W Porter, MD, FRCS

Doncaster Royal Infirmary, Doncaster, UK

Summary

This study compares the prevalence of coal miners attending hospital with three defined back pain syndromes, with the number of miners in the working population. Of the men who attended hospital with back pain there were more miners than would be expected (2·78% of the miners compared with 1·99% of the non-miners). 0·32% of the miners had criteria of disc protrusion compared with 0·4% of non-miners; significantly more had syndromes associated with degenerative change. Relatively few men requiring disc excision were miners, whilst there were many who had decompressive surgery. This is compatible with the concept that heavy manual work strengthens the spine, restraining encroachment of a disc protrusion into the vertebral canal.

Relevance

These findings suggest a need to identify and encourage activities in early life which may develop annular and ligamentous strength. Furthermore, unfit workers should not be deployed to areas of heavy work and we should re-examine advice about light work after the first disc protrusions.

Key words: Disc protrusion, Work, Epidemiology, Surgery, Degenerative change, Spine, Back pain, Miners

Introduction

There is conflicting evidence about the relationship between heavy manual work and the incidence of back pain. Most studies suggest that back pain is related to heavy load handling but Kelsey[7] found a high incidence of disc herniation amongst sedentary workers.

In an attempt to determine whether heavy manual work reduces the risk of symptoms from disc herniation, this paper examines the prevalence of coal miners presenting with various back pain syndromes, comparing this with the percentage of miners in the population.

Method

Men attending a back pain clinic over a 3-year period were asked if they had been underground coal miners from leaving school, or had worked underground for at least 5 years. The percentage of these miners fulfilling criteria for three defined back pain syndromes was then recorded.

1. Lower lumbar disc lesion (PID). Three or more of the criteria described by McCulloch[8]: unilateral leg pain in a typical sciatic root distribution below the knee; specific neurological symptoms incriminating a single nerve; limited straight leg raising by at least 50% of normal; at least two neurological changes of muscle wasting, muscle weakness, sensory change or hyporeflexia; and radiculographic evidence of disc protrusion.

2. Root entrapment syndrome (RE)—lateral canal stenosis. Pain in the leg below the knee incriminating a single root, severe and constant, unrelieved by rest, spinal extension less than a third of normal range, straight leg raising better than 70 degrees, over 40 years of age and radiological evidence of degenerative change[9,10].

3. Neurogenic claudication (NC). Discomfort in both legs above and below the knees when walking with less than 500 metres tolerance, relieved by rest, and a positive myelogram or CT scan showing central canal stenosis.

The percentage of miners of the total men admitted for bed rest, whose symptomatic disc lesion had not settled at home was recorded, and also the percentage of miners of the men requiring surgical disc excision, and surgical decompression for central or root canal stenosis[11–13].

Submitted: 24 April 1987. *In revised form*: 3 July 1987.
Correspondence and reprint requests to: Mr R W Porter, Department of Orthopaedics, Doncaster Royal Infirmary, Doncaster DN2 5LT, UK.

Table 1. Number of miners and non-miners in the Doncaster population, attending the back pain clinic, with three defined back pain syndromes and 'other causes', expressed as a percentage of miners and non-miners in the population

	Doncaster workers	*Back pain clinic*	*PID*	*R.E.*	*N.C.*	*Other*
Miners	16,000	445 (2·78%)	52 (0·32%)	93 (0·58%)	35 (0·22%)	265 (1·65%)
Non-miners	49,100	977 (1·99%)	198 (0·40%)	152 (0·31%)	21 (0·04%)	606 (1·23%)
		χ^2=33·4	χ^2=14·9	χ^2=6·9	χ^2=26·4	
		P<0·001	P<0·001	P<0·001	P<0·001	n.s.

Chi square tests between miners and non-miners performed on contingency tables for each diagnosis v all other patients)

Results

The collieries in the catchment area of Doncaster Royal Infirmary employed 16,000 miners in the period under review. In the same catchment area there were 65,100 working men between 20 and 60 years of age, 24·6% of whom were miners.

Four hundred and forty-five miners attended the back pain clinic over a 3-year period (2·78% of the miners in the population) compared with 977 non-miners (1·99% of the non-miners in the population). Only 0·32% of the miners in the population attended with criteria of symptomatic disc protrusion, compared with 0·40% of non-miners. The Chi-square (χ^2) test showed significant differences $P < 0·001$. Proportionately more miners however attended with root entrapment syndrome and neurogenic claudication than did other men in the population (Table 1).

There were 869 men with 'other causes' of back pain, including some with more than one of the three defined syndromes, comprising 1·65% of the mining population, and 1·23% of the non-miners.

One hundred and three men were admitted with symptomatic disc protrusion; 70 settled with bed rest, and 33 required surgery (Table 2). The proportion of the mining population having symptoms which settled with bed rest in hospital was 0·06%, compared with 0·12% of the non-mining population, whilst 0·02% of the mining population required discectomy compared with 0·06% of the non-miners. There were, however, many miners requiring decompressive surgery for central or root stenosis.

Table 2. Numbers of miners and non-miners in who required hospital bed rest and discectomy, expressed as a percentage of miners and non-miners in the population

	Admitted for bed rest and symptoms settled without surgery	*Required surgery*
Miners	10 (0·06%)	3 (0·02%)
Non-miners	60 (0·12%)	30 (0·06%)
	χ^2=0·55 P=n.s.	

Figure 1. A damaged underground roadway in a coal mine, caused by shifting strata. It will be repaired manually.

Discussion

Mechanisation of coal mines has largely relegated the pick and shovel to a previous era, but the fluid nature of underground strata and its effects on the roadways, the need to transport equipment to and from the coal face and the unpredictable failure of machinery in confined spaces, still subjects the miner's spine to large mechanical forces (Figure 1).

It is not surprising that musculoskeletal disorders are the largest cause of absenteeism in the coal mining industry. The most significant of these is low back pain[14], which is responsible for up to 18% of sickness absence in some British coalfields[15].

Understandably, more miners attend the back pain clinic than would be expected from their numbers in the population. Either it is more difficult to work underground with a painful back than in other environments, or heavy manual work increases the risk of acute or chronic back pain.

The risk would appear to be selective, however, when considering the three defined back pain syndromes. The miner is at no greater risk of presenting with the symptoms of disc protrusion. He is, however, more likely to complain of those syndromes associated with degenerative change—lateral canal and central canal stenosis (RE and NC)—and to require decompressive surgery for these degenerative conditions.

The incidence of miners attending hospital with disc symptoms was less than the percentage of miners in the

population, and the proportion of miners amongst those patients admitted with disc symptoms was significantly less ($P < 0{\cdot}001$). Few of these, however, failed to respond to conservative treatment. In fact, it was the dearth of miners requiring surgical disc excision that prompted this study.

Although the miner subjects his spine to greater loads than many others in the population, he appears to be protected from the disabling effects of disc protrusion. There is no evidence that his genetic stock is different to other workers. It is possible that the low incidence of disabling disc symptoms is the result of self-selection; men with vulnerable discs having left the industry early. But then it is difficult to explain why the same self-selection did not reduce the incidence of other back pain syndromes. There are two alternative explanations. Either a miner has less annular damage because of strong muscles and efficient protective reflexes, or he is equally prone to disruption of a disc, but is able to contain it more effectively. Heavy manual work in early life may strengthen the spinal ligaments, and possibly the peripheral annulus, thus restraining the disc nucleus from encroaching into the vertebral canal. A similar protrusion in a sedentary worker could extrude into the vertebral canal unprotected by a weak peripheral annulus.

If this concept is correct, we should identify and encourage activities which in early life develop annular and ligamentous strength. Unfit workers should not be deployed to areas of heavy load-handling. We may also need to modify advice about light work after the first disc symptoms and instead encourage a graduated return to work and recommend activities which strengthen the spine.

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BRIEFLY NOTED

Measurement of Lumbar Sagittal Mobility
A Comparison of Methods

PAMELA J. SALISBURY and
RICHARD W. PORTER MD, FRCS, FRCSE

MEASUREMENT OF LUMBAR sagittal mobility is useful in the clinical assessment of back pain syndromes, in epidemiology, and for measuring response to treatment, but there is some uncertainty about the relative merits of different methods used in clinical practice.[9,10]

Radiographic techniques first introduced in 1933[17] include biplanar radiography and computer-aided x-ray analysis.[15,5,14,11] Measurement of spinal motion by ultrasound was proposed by Hammond,[6] but it has not yet been assessed. Ultrasound also has the ability to identify bony landmarks prior to measurement by external techniques and, thus, reduce a potential source of error.

External measurements are less expensive, quicker, and are relevant to clinical practice. In this study we determine the reproducibility of four of these methods and assess an ultrasound technique.

MATERIALS AND METHODS

Five methods and instruments for measuring spinal movement were assessed, the kyphometer, goniometer, flexicurve, tape measure, and measurements of diasonograph ultrasound echoes. The first three use methods based on a mathematical theory of angles outlined by Loebl[7] and are expressed in degrees.

The spinous processes of the S2, T12, and L4 vertebrae were located by palpation and the skin was marked. These were compared with subsequent identification of the vertebrae by ultrasound.

Each instrument was used to assess the angle between the bony landmarks in maximum flexion and extension. In flexion the subjects sat on the edge of a chair, and with legs apart they bent over with the trunk, and arms were dropped fully between the knees (Figure 1). In extension the prone subject raised the head and shoulders until the upper arms were perpendicular to the couch (Figure 2)

Measurements were taken between 1,100hh and 1,500hh to standardize the diurnal effects of disc hydration.

Kyphometer. Model AZB CH-4802. The kyphometer had a protractor scale in degrees at the apex of two long arms (Figure 1). The base of each arm was placed over the spinous processes of S2, and T12. The angle produced on the scale was the angle between these vertebrae.

Goniometer. The goniometer (Figure 2) evolved from the spondylometer of Fox and Van Breemer,[4] to that of Dunham,[2] and then to the "hydro-goniometer" of Loebl. It was calibrated in degrees. A freely movable lead-weighted pointer had its movement dampened by low-viscosity paraffin. This behaved as a plumb line so that it quickly settled vertically. The friction nut to the base enabled the dial to be set to zero when positioned on S2. The goniometer was then moved to T12 where the angle between the vertebrae was recorded directly from the dial.

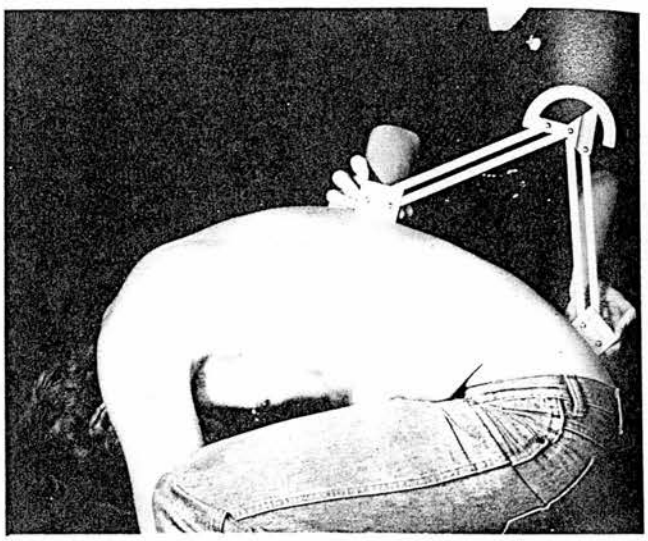

Fig 1. Kyphometer measuring maximum flexion between T12 and S2.

Flexicurve. A draftsman's flexible curve, modified with a permanent mark 20 mm from one end, and a movable brass slide was molded to the midline of the back[1] (Figure 3). The mark was placed over S2 and the brass slide moved until it was over T12. The contour was then drawn on a sheet of paper, the marks of T12 and S2 were recorded, and tangents to the curves at these points were drawn and the resulting angle measured.

Tape Measure. The tape measure recorded skin distraction.[8,3,12] Three marks were made with the subject sitting in an upright position, a mark over L4, one 100 mm above and one 50 mm below L4. After the subject had

Fig 2. Goniometer measuring extension at T12.

Supported by British Coal.
Submitted for publication September 16, 1986.
The authors thank Kim Burton for his helpful advice.

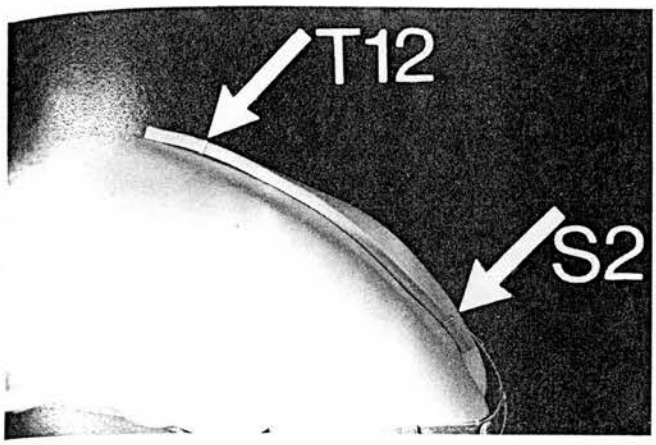

Fig 3. Flexicurve applied to the flexed lumbar spine.

reached maximum flexion, the distance between the upper and lower marks was measured. The increase was taken as a measure of flexion.

Diasonograph Ultrasound. Polaroid® photographs were taken of ultrasound scans of the lumbrosacral spine in flexion and extension and the angle was measured between the tangent at T12 and the sacral laminae (Figure 4).

A statistical contrast between the instruments was achieved by one-way analysis of variance. Significance values for differences between the instruments were computed by Student's *t* test. The five methods were compared by regression analysis.

RESULTS

Flexion and extension of the lumbar spine was recorded by the four external methods in 17 asymptomatic volunteer subjects between 20 and 35 years of age. Nine volunteers had flexion and extension ultrasound measurements. The vertebral levels were compared by ultrasound and by direct palpation on 34 occasions, and only once was the surface marking unsuccessful.

Flexion

The mean range of three measurements of the kyphometer, goniometer, and the flexicurve on two separate days, are shown in Tables 1 and 2, and the mean range of the six results on both days in Table 3.

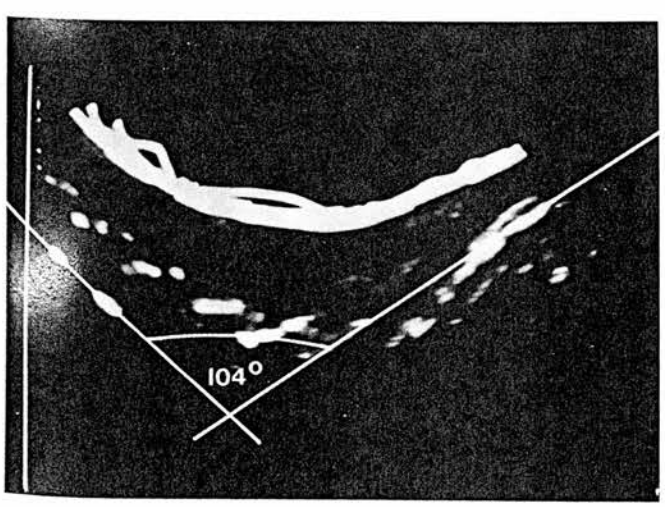

Fig 4. Diasonograph echoes of extended lumbar spine showing the angle between the tangents of the sacral lamina and the body of T12.

Table 1. Mean Range of Flexion for Day-1 Measurements

Instrument	*Mean range*	*SD*
Kyphometer	2.62	1.84
Goniometer	3.94	3.46
Flexicurve	2.75	1.72

No significance was found between these results.

There were no significant differences between the mean range of the instruments except that on the second day the flexicurve had a greater range than the kyphometer ($P < 0.1$) and the goniometer ($P < 0.02$) (Table 2).

The differences of the mean of the three measurements on the first and the second day was not significantly different in each instrument (Table 4).

Correlations of measurements of flexion between the five methods are shown in Table 5. The best being between the kyphometer, goniometer and flexicurve ($r = 0.94-0.99$), and ultrasound ($r = 0.78-0.79$). The tape measure did not show any useful correlations.

Extension

Measurements in extension were recorded as for flexion except that the kyphometer was not calibrated for the high degree of extension achieved by ten subjects. The mean ranges of the three measurements for the kyphometer, goniometer, and flexicurve in extension on two separate days are shown in Tables 6 and 7. The means of six measurements on the two days are shown in Table 8. The flexicurve had a significantly greater range than the kyphometer and goniometer on the second day and on both days together.

Table 2. Mean Range of Flexion for Day-2 Measurements

Instrument	*Mean range*	*SD*
Kyphometer	2.47	2.32
Goniometer	1.91	1.33
Flexicurve	4.23	3.17

The flexicurve had a greater range than the kyphometer ($P < .01$) and the goniometer ($P < 0.02$).

Table 3. Mean Range of Flexion Over the 2 Days

Instrument	*Mean range*	*SD*
Kyphometer	5.53	3.02
Goniometer	6.09	4.01
Flexicurve	6.03	2.36

No significance was found between these results.

Table 4. Mean Difference of Flexion Between Day 1 and Day 2

Instrument	*Mean range*	*SD*
Kyphometer	2.95	2.96
Goniometer	3.80	2.95
Flexicurve	3.15	2.00

No significance was found between these results.

Table 5. Flexion Correlations

x	(v)y	N	r
Group 1			
Kyphometer	Goniometer	17	0.99
Kyphometer	Flexicurve	17	0.93
Goniometer	Flexicurve	17	0.94
Group 2			
Kyphometer	Tape measure	17	−0.23
Goniometer	Tape measure	17	−0.04
Flexicurve	Tape measure	17	0.04
Group 3			
Ultrasound	Kyphometer	9	0.79
Ultrasound	Goniometer	9	0.78
Ultrasound	Flexicurve	9	0.79
Ultrasound	Tape measure	9	−0.20

$y = Mx + C$.
N = number of pairs of results.
r = correlation.

There were no significant differences between the means of the six extension readings from both days (Table 9).

Correlations of the means of extension results of the four methods are shown in Table 10. The flexicurve and ultrasound showed the poorest correlation (r = 0.69).

DISCUSSION

Lumbar sagittal motion can be measured either by direct methods such as radiography and ultrasound or by indirect external methods. They are subject to different errors, but are only rarely compared.[13,1]

The repeatability of external methods depends first on accurate recognition of bony landmarks. In this study we compared the vertebral levels identified by ultrasonography and by palpation, and on only 3% of occasions did a nonmedical examiner fail to locate accurately the correct spinous process. It is unlikely, therefore, that there is significant error in the external methods from inaccurate surface marking.

A second source of error is an inability to achieve repeatable flexion and extension. Troup[16] noted that spinal range was affected by the position of the hip joints, and we, therefore, recorded maximum flexion and extension with hips in a defined position described by Burton.[1] Speed of measurement and subject fatigue affects the repeatability. The results from ultrasonography when patients were required to maintain their positions for about 5 minutes in order to obtain the echoes, had less satisfactory correlations than the external angular methods (Tables 5 and 10). This error would presumably be greater in patients whose pain limits the range of movement.

Of the four external methods, the skin distraction method had the poorest correlation with other techniques. The tape measure has been shown to record limited movement in patients with back pain when compared with normal volunteers,[12] but Reynolds[13] found it less reproducible than the goniometer, which was our experience. This is probably because surface distraction is small compared with angular movement. In addition the tape measure does not readily measure extension.

The three methods that measured angular movement, the goniometer, kyphometer, and flexicurve, correlated well together and had similar degrees of repeatability. The drawing of flexicurve tangents introduces a secondary error, thus accounting for slightly poorer repeatibility than the other two methods. Though the flexicurve was least satisfactory, it was considerably less expensive than the other two instruments. We considered that the goniometer was the easiest to use and it produced results most rapidly, whereas the kyphometer was heavy with broad flat feet requiring careful positioning on the spine.

Because of simplicity and high repeatibility, the goniometer is, in our opinion, still the best instrument to measure sagittal mobility of the lumbar spine.

Table 6. Mean Range of Extension for Day-1 Measurements

Instrument	Mean range	SD
Kyphometer	3.06	2.22
Goniometer	3.00	1.44
Flexicurve	4.24	2.81

No significance was found between these results.

Table 7. Mean Range of Extension for Day-2 Measurements

Instrument	Mean range	SD
Kyphometer	3.14	1.80
Goniometer	2.85	1.34
Flexicurve	5.92	2.95

The flexicurve had a significantly greater range than both the kyphometer ($P < 0.01$) and the goniometer ($P < 0.001$).

Table 8. Mean Range of Extension Over the 2 Days

Instrument	Mean range	SD
Kyphometer	6.35	2.85
Goniometer	6.12	2.70
Flexicurve	9.35	2.96

The flexicurve had a significantly greater range than both the kyphometer ($P < 0.001$) and the goniometer ($P < 0.001$).

Table 9. Mean Difference of Extension Between Day 1 and Day 2

Instrument	Mean difference	SD
Kyphometer	3.16	2.24
Goniometer	3.10	1.98
Flexicurve	4.18	3.58

No significance was found between these results.

Table 10. Extension Correlations

x	y	N	r
Group 1			
Goniometer	Flexicurve	17	0.90
Flexicurve	Kyphometer	7	0.71
Goniometer	Kyphometer	7	0.92
Group 2			
Ultrasound	Kyphometer	5	0.83
Ultrasound	Goniometer	8	0.78
Ultrasound	Flexicurve	8	0.69

$y = Mx + C$ as in flexion.

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Address reprint requests to

Mr. Richard W. Porter, MD, FRCS
Consultant Orthopaedic Surgeon
Orthopaedic Department
Doncaster Royal Infirmary
Armthorpe Road
Donacaster DN2 5LT
England

Accepted for publication October 10, 1986.

Central Retinal Artery Occlusion in Association with Osteogenesis Imperfecta

C. F. BRADISH, MA, FRCS, and M. FLOWERS, MB, BS

CENTRAL RETINAL ARTERY OCCLUSION (CRAO) may develop after surgery, possible etiologic factors being hypotension and extrinsic pressure on the eyeball. We report a case in which CRAO developed in a girl with osteogenesis imperfecta after a posterior spinal fusion performed under hypotensive anesthesia and propose that patients with osteogenesis imperfecta are particularly susceptible to this complication.

Case Report

The patient, a 12-year-old white girl with osteogenesis imperfecta in association with blue sclerae (Sillence Type I) underwent a two-stage spinal fusion for her rapidly progressive scoliosis. The first stage, which consisted of an anterior spinal fusion between T10 and L4, proceeded uneventfully. At the second stage, 2 weeks later, a posterior spinal fusion was performed between T9 and L5. An internal fixation device was used. For this procedure the patient was placed in the prone position on a Montreal mattress with her head rotated to the right and supported on sandbags, the left eye being lowermost. This operation was performed under hypotensive anesthesia, the patient's systolic blood pressure being maintained at between 50 and 80 mm Hg (preoperative systolic pressure was 100 mm Hg). The operation lasted 3 hours and the total blood loss was 1,100 ml (patient's weight: 21.25 kg). Spinal cord monitoring had been used throughout the procedure with no abnormality of the trace.

Within 15 hours after the second operation the patient was aware of impaired vision in the left eye, affecting the whole visual field. Her visual acuity on the left was less than counting fingers, and although the acuity fluctuated, within 2 days she just had perception of light in that eye. Throughout this time she had a dilated left pupil and a left afferent pupillary defect. The left disc was edematous and pale and became chalky white within a few days with extreme attenuation of the retinal vessels. There were never any retinal hemorrhages. The visual acuity in the right eye was 6/9 with normal color vision.

A diagnosis of central retinal artery occlusion had been made at the onset of symptoms and treatment commenced with intravenous bolus acetazolamide and external ocular massage but without sustained improvement.

Subsequent investigations included orbital computerized tomography (CT) scanning that revealed no bony or soft tissue abnormality within the orbital margins or optic canal of the left eye. Visual evoked potentials to pattern reversal were absent from the left eye and the patient had an absent electroretinogram from that eye; responses from the right eye were normal. Chest x-ray, electrocardiogram, hemoglobin and electrophoresis, and erythrocyte sedimentation rate studies were normal.

DISCUSSION

This is the first time CRAO has been recorded as an operative complication in association with osteogenesis imperfecta and it is probable that it was due to the combination of hypotensive anesthesia and extrinsic pressure on the already susceptible eye of the osteogenic patient.

Givner and Jaffe[3] were the first to report CRAO after surgery, and they felt that it was due to a combination of hypotension and extrinsic pressure on the eyeball. They noted that in three of their four cases the nasal bridges were low. Hypotension alone is unlikely to be responsible, since severe blood loss is more likely to cause bilateral visual disturbances; and retinal ischemia after exsanguination is a rare event considering the common occurrence of massive hemorrhage, for example in war wounded.[1]

The reported incidence of retinal artery thrombosis following administration of hypotensive anesthesia is low. Little[4] reported

From the Royal National Orthopaedic Hospital, London, England.
Submitted for publication October 6, 1986.
The authors thank Dr. G. D. Schott, MD, FRCP, Consultant Neurologist, for his invaluable help and advice, and Mr. M. A. Edgar, FRCS, for permission to publish his case.

Journal of Orthopaedic Rheumatology **1**, 173–178 (1988)

Familial aspects of disc protrusion

R. W. PORTER and G. H. L. OAKSHOTT
Doncaster Royal Infirmary, Armthorpe Road, Doncaster, South Yorks DN2 5LT, UK

The probands consisted of 50 patients who had had discectomy and 50 matched controls who had had no previous history of back pain, to assess the familial aspects of disc protrusion. The probands had 31% and 14% of first-degree relatives respectively who had 'significant' low back pain, ($P < 0.01$), the discectomy relatives describing significantly more disc criteria. More controls were of blood group A and more patients of blood group B than would be expected.

Keywords: disc protrusion, back pain, family history

Introduction

Studies in comparative anatomy suggest that there is a genetic component to disc pathology. This is evidenced by daschund and beagle dogs and by 'pintail' mice who have a high frequency of disc protrusion and degeneration (Hansen, 1952; Berry, 1961). In men, certain spinal pathologies such as ankylosing spondylitis have a genetic association (Brewerton *et al.*, 1973; Schlosstein *et al.*, 1973), and there is circumstantial evidence that this may be true of disc protrusion (Jackson, 1948). Jayson and Barks (1973) observed similar annular fissures in adjacent post mortem discs of patients who had died from unassociated conditions and suggested that these were constitutionally determined. Likewise, it is fairly common to excise a disc from a patient whose relatives have also undergone similar procedures.

We have assessed the genetic and familial components of disc protrusion by comparing patients who had disc surgery with matched volunteers who had no previous experience of back pain.

Method

The probands consisted of 50 patients who had had surgical excision of a lumbar disc and 50 control subjects who had no previous experience of low back pain. The two groups were matched for sex and vertebral canal size, using ultrasound measurement. They were interviewed about their present and past health. A photographic record was taken of the iris and an ophthalmic surgeon examined the cornea. The range of movement of the 2nd metacarpophalangeal joint of the non-dominant hand was measured with the 'Leeds hyperextensiometer' (Jobbins *et al.*, 1979). The leukocyte system of iso antigens was assessed by tissue typing, and the A B O blood groups were compared.

The subjects and controls were asked about 'significant back pain' in their living first-degree relatives, (parents, siblings and children). Back pain was considered 'significant' if it caused them to consult a doctor, or put them to bed. These relatives were then approached by postal questionnaire for further information about their back pain (Table I).

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Table I. Back pain questionnaire (disc criteria, numbers 2 to 6)

1. Have you ever had back pain that caused you to see a doctor or put you to bed?
2. Did that pain cause you to twist to one side?
3. Did the pain extend into the leg below the knee?
4. Did coughing or laughing or sneezing make the pain worse?
5. When you had the pain and were lying down, did your doctor have difficulty lifting your heel more than a few inches off the bed?
6. Did you ever have a myelogram which showed you had a damaged disc?

Table II. Mean canal size of patients, controls and the general population

	Mean canal size (cm)				
Group	*L1*	*L2*	*L3*	*L4*	*L5*
Fifty discectomy patients	1.44	1.42	1.39	1.36	1.36
Fifty control patients	1.42	1.40	1.38	1.35	1.35
10th percentile the general population	1.44	1.43	1.40	1.36	1.38

Table III. Occupations of patients and controls

Occupation	*Disc patient*	*Control*
Miner	4	4
Heavy manual	12	6
Light manual	9	12
Semi-sedentary	15	16
Sedentary	10	12

Table IV. Present and past history of other diseases

Disease	*Disc patient*	*Control*
Lower limb arthritis	17	17
Dyspepsia	13	10
Heart disease	9	10
Asthma	1	3
Diabetes	1	0
Thyroid	2	2
Skin disease	13	10
Migraine	18	12
Allergies	12	11

Table V. Iris colour and appearance of the cornea of patients and controls (%)

	Disc patient	*Control*
Iris colour		
Blue	42	40
Brown	22	21
Green	10	9
Blue/grey	16	20
Blue/green	10	10
Corneal appearance		
Normal	84	82
Stahli-Hudson lines	20	28
Arcus senilis	12	30
Scar	10	16
Keratic precipitates	8	6
Endothelial changes	4	10
Degenerative changes	0	0

Results

There were 33 males and 17 females in each group. The mean age was 43.8 years ± 8.7 for the discectomy patients, and 50.9 years ± 7.3 for the controls. The mean vertebral canal size at L5 was 1.36 cm ± 0.07 in the patients, and 1.35 cm ± 0.04 in the control group (Table II). The occupation of the two groups is shown in Table III; there were a few more sedentary workers in the control group.

The prevalance of past and present illnesses is shown in Table IV. In this small series there was no significant difference in the disease pattern of the two groups.

The colour of the iris was not significantly different in the two groups, nor was there any difference in physiological or pathological changes in the cornea (Table V).

The range of the 2nd metacarpophalangeal joint was similar in the two groups, but it was significantly less in the male patients than in the male controls ($P < 0.01$; Table VI).

The HLA antigens were similar in the two groups, HLA-B27 occurring in three patients and three controls. There were more subjects with blood group A in the non-back controls than would be expected from the general population, and there were more discectomy patients with blood group B (Table VII).

There were 137 first-degree relatives of the discectomy patients, and 132 first-degree relatives of the controls. Totals of 31% and 14% of these respectively had 'significant low back pain' ($P < 0.01$). There was a 62% response to the questionnaire about this pain, summarized in Table VIII. Nine relatives of discectomy patients described two or more positive disc criteria, compared with none of the relatives of the controls. Five relatives of the discectomy patients, but none of the control relatives, had discs revealed previously by myelography.

Table VI. Mean range of the left second metacarpophalangeal joint in patients and controls (degrees)

	Disc patient	*Control*	*Significance*
Male	21.9 ± 9.6	28.0 ± 16.1	$P < 0.01$
Female	46.7 ± 15.9	41.6 ± 18.7	ns

ns = Not significant.

Table VII. Distribution of ABO blood groups (%)

	Disc patient	*Control*	*General population*
A	40	53	42
B	15	9	9
AB	4	0	3
O	41	38	46

Table VIII. Comparison of number of disc criteria (2–5 in Table I) in relatives of patients and controls, who had a history of 'significant' back pain

No. of disc criteria	*Relative of disc patients (n = 25)*	*Relative of controls (n = 13)*
One	4	5
Two	5	0
Three	3	0
Four	1	0
Positive myelogram	5	0

Discussion

The strength of the annulus (and disruption of the intervertebral disc) depends on many factors. It is related to age, with symptoms presenting in the 4th decade (Sprangfort, 1972) and sex, where men present for treatment more often than women. Occupation is important, with heavy manual workers apparently protected from the symptoms of disc protrusion (Porter, 1987). The familial and genetic component, however, has received little attention.

We considered it essential to match the vertebral canal size of the controls with that of the discectomy patients. Most patients who present with disc symptoms have a small vertebral canal (Rothman and Simone, 1972; Porter *et al.*, 1978; Kornberg and Rechtine, 1985); in this series the mean sagittal diameter of the 50 discectomy patients was below the 10th percentile for the general population (Table II). Patients with wider canals probably do develop disc protrusion, but escape disabling symptoms. Controls were therefore chosen with similarly small canals, lest they should have had a disc protrusion with symptoms masked by a wide canal.

There was a slight disparity of age between patients and controls (43.8 years compared with 50.9 years) because controls were sought who were old enough to have experienced disc symptoms if these were likely to develop. It could only produce an adverse bias to the study, with the children and siblings of the controls being older than those of the discectomy patients, and therefore having had longer to develop disc symptoms.

A comparison of the medical history failed to show any association between disc disease and other disorders. Neither was the examination of the eye productive. There are racial differences in back pain (Bremner and Lawrence, 1968), but in this series the pigment, at least in the iris, was not related to symptomatic disc protrusion. The collagen of the cornea bears some similarity to that of the intervertebral disc, but there was no obvious difference in the physiological and degenerative changes of the cornea between the two groups.

The reduced range of the metacarpophalangeal joint movement in the male discectomy patients was all the more significant because of their younger mean age than the controls. Joint mobility relates to the collagen of soft tissues, important in disc disease.

The high frequency of blood group A in the non-back pain controls and the increased frequency of blood group B in the patients suggest that there may be a genetic link between blood group and the strength of the annulus. If this were to be established in a larger study, blood group may prove to be a genetic marker and a predictor of back pain. The study is too small to show any association between HLA antigens and disruption of the annulus.

We expected patients and controls to be aware of any 'significant' back pain in their parents, siblings and children. This was reported in more than twice as many first-degree relatives of the patients as the controls. The characteristics of this pain was amplified by a postal questionnaire. Two or more disc criteria were reported by 14 of 25 relatives of the discectomy patients, but not by any of the relatives of the controls. Five discectomy relatives, but no relatives of the controls, had had disc protrusion revealed by myelography.

This study shows a familial association with protrusion of the lumbar intervertebral disc. We have not been able to identify a genetic marker, but if the strength of the annulus is related to blood group this would support the association being genetic.

Acknowledgements

We thank Mr S. P. Desai and Mr G. Swan for their assistance and British Coal for sponsoring this project.

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Accepted for publication 8 February 1988

Sept 1986. Published in: Pratt DJ, Johnson GR, eds. Biomechanics and orthotic management of the foot — 1. Derby: Orthotic and Disability Research Centre, 1988

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Vertebral displacement in spondylolisthesis

R W Porter MD, FRCS
C Hibbert PhD

Doncaster Royal Infirmary, Doncaster, UK

Summary

The causes of sagittal displacement in 162 patients with isthmic spondylolisthesis and 81 with degenerative spondylolisthesis have been assessed. The slip ratio was compared with joint mobility, the presence of spina bifida, the lumbosacral angle, a self-assessment of previous athletic ability at school and childhood injuries. There was a correlation between the slip ratio and the lumbosacral angle in isthmic spondylolisthesis (= 0·39, $p < 0·01$); but this may be secondary. Metacarpophalangeal joint hyperextension correlated with slip ratio ($r = 0·26$, $p < 0·05$). There was no significant difference in slip ratio between the patients with and without spina bifida. Those patients with a childhood injury before 11 years of age had greater displacement than those with a later injury, but this did not reach statistical significance. Many patients with degenerative spondylolisthesis rated their athletic ability at school as 'poor'.

Relevance

This study demonstrates that displacement in isthmic and degenerative spondylolisthesis is multifactorial. Environmental factors in childhood may be significant.

Key words: Spondylolisthesis, slip ratio, spina bifida, childhood injury

Introduction

The cause of sagittal displacement in spondylolisthesis is at present unknown. One patient with isthmic spondylolisthesis may have a 40% slip and another with the same pars defect have no displacement at all. In degenerative spondylolisthesis, in which the vertebra displaces forwards with an intact neural arch, the ratio rarely exceeds 15%[1,2]. We have attempted to identify factors which may be responsible for the variable sagittal displacement. The degree of displacement in degenerative spondylolisthesis has considerable clinical relevance, with increasing risk of neurological compromise[3,4]. Why is there such a variable degree of displacement, and what factors are responsible? We have attempted to identify these, by examining populations with the two types of spondylolisthesis.

Methods

One hundred and sixty-two patients with isthmic spondylolisthesis and 81 with degenerative spondylolisthesis were classified according to Wiltse, Newman and MacNab[5] using standard and 45° oblique radiographs. Patients with dysplastic, pathological and traumatic spondylolisthesis were excluded from the study. The slip ratio was measured[6,7] (Figure 1), spina bifida noted, and the lumbosacral angle recorded from standing lateral radiographs measuring between $L_{3/4}$ and L_5/S_1 disc spaces[8].

The degree of joint mobility was assessed in 95 of the patients with isthmic spondylolisthesis and in 48 with degenerative spondylolisthesis who attended for assessment, and they were compared with 109 patients without spondylolisthesis attending a back pain clinic and also with 59 volunteer subjects without back pain who were accompanying their relatives. The patients with back pain, the volunteers and the patients with isthmic spondylolisthesis were matched for age and sex.

Cervical, thoracic and lumbar spinal flexion wa measured in these four groups by goniometer[9], which i the most repeatable direct method of measuring sagitta spinal motion[10], and with a tape measure[11], whilst move ments of five peripheral joints were assessed by th Carter Wilkinson score[12]. The range of movement of th second right metacarpophalangeal joint was recorde with a hyperextensiometer (Figure 2)[13]. The degree o pes planus was measured from a photograph as the pa tient stood on a glass plate (Figures 3 and 4), recordin the foot contact area with a planimeter.

Received: 21 April 1988
Accepted: 27 August 1988
Correspondence and reprint requests to: RW Porter, MD, FRCS, Department Orthopaedics, Doncaster Royal Infirmary, Armthorpe Road, Doncaster, DN2 5LT, UK

0268–0033/89/010058–06 $03.00

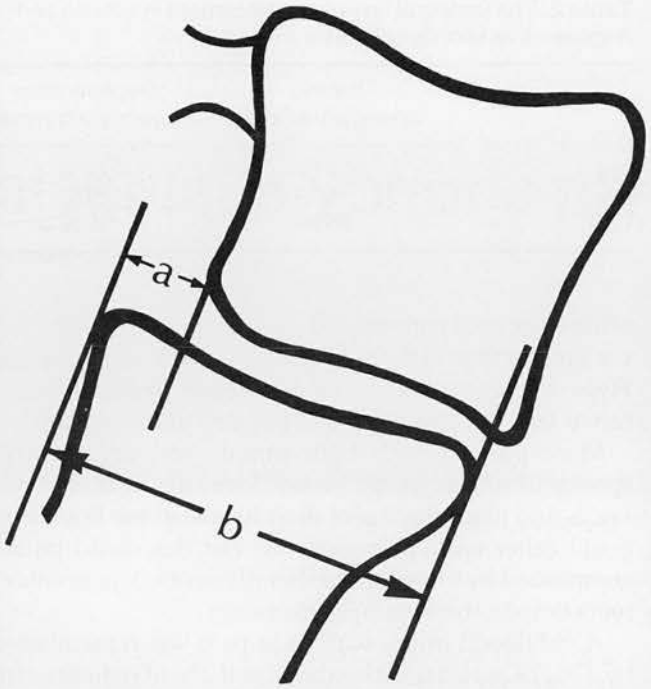

Figure 1. Diagram to show method of measurement of the slip ratio.

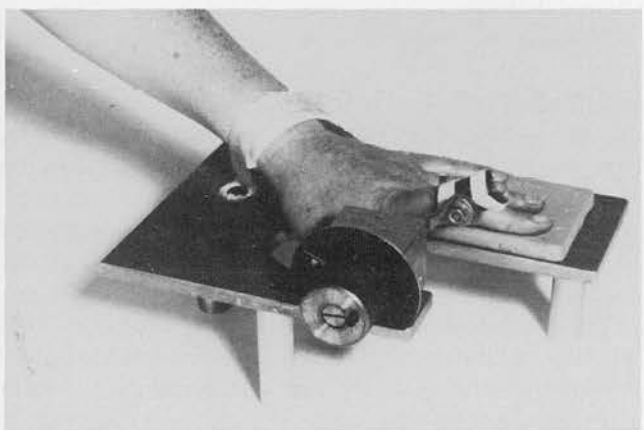

Figure 2. The Leeds Hyperextensiometer measuring the degree of hyperextension of the second right metacarpophalangeal joint.

Patients with spondylolisthesis and the volunteer subjects assessed their previous athletic ability at school: they were asked about childhood injuries and their method of birth (normal delivery, breech, forceps or caesarean section).

The slip ratio was compared with the lumbosacral angle, the presence of spina bifida, the tests for joint mobility, and the childhood history, in patients with isthmic and degenerative spondylolisthesis. These two groups were also compared with the other back pain patients and with the volunteers.

Student's *t*-test was used to estimate the statistical significance of differences between means.

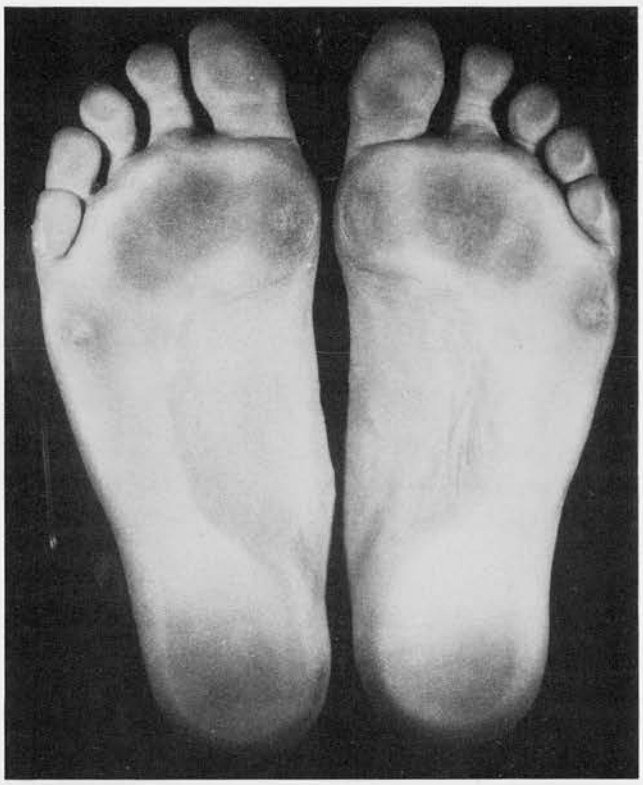

Figure 3. Degree of pes planus. The foot contact area photographed as the patient stood on a glass plate. The balanced foot contact area was measured with a planimeter, and expressed as a percentage of the area of the sole of the foot.

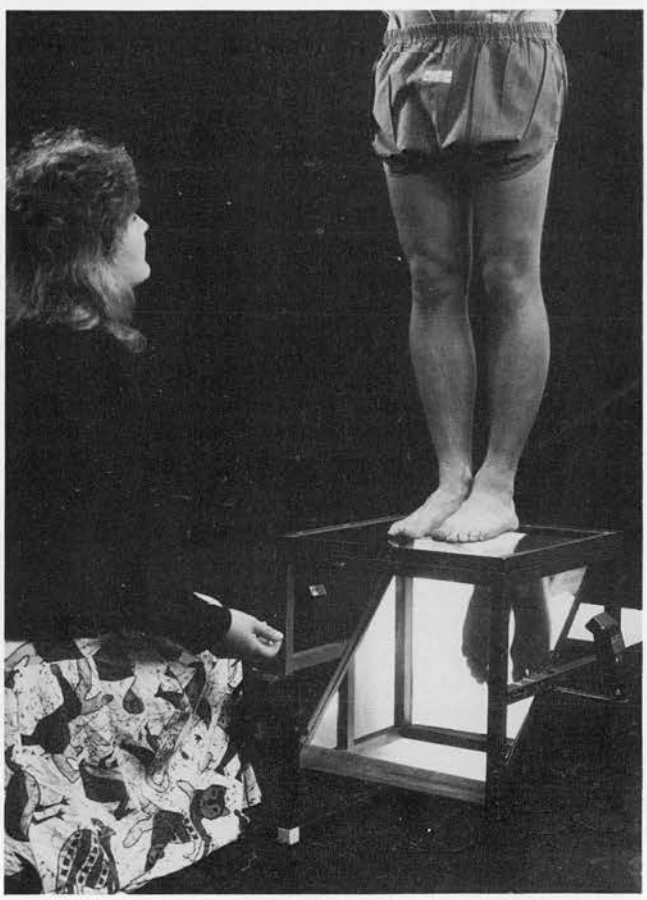

Figure 4. Method of photographing the sole of the foot as the patient stands on a glass plate (designed by Mr G Swann).

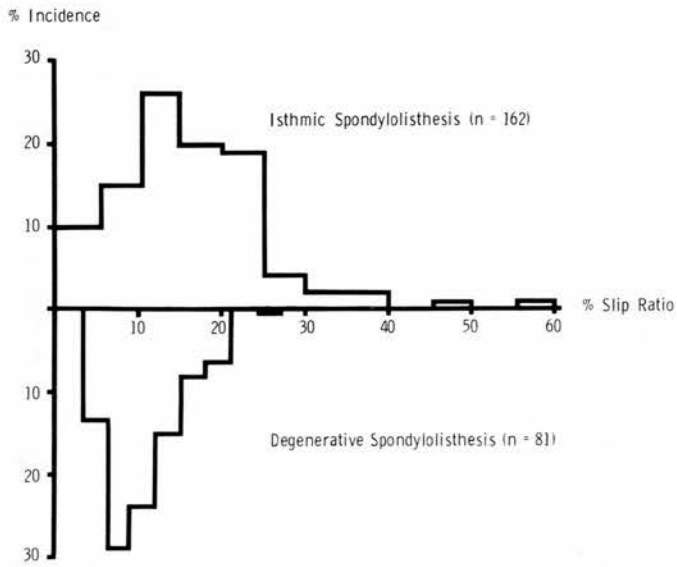

Figure 5. Histogram of the slip ratio of 162 patients with isthmic spondylolisthesis and 81 patients with degenerative spondylolisthesis.

Results

The mean slip ratio (Figure 5) for isthmic spondylolisthesis was 17% (s.d. 9%) and for degenerative spondylolisthesis 11% (s.d. 5%). The mean lumbosacral angle was significantly greater for the patients with isthmic spondylolisthesis (37·3°; s.d. 8·3) than for those with degenerative spondylolisthesis (30·8°; s.d. 7·1), and for the patients with back pain but no spondylolisthesis (30·3; s.d. 6·4), $p < 0{\cdot}01$. There was a correlation coefficient between the slip ratio and the lumbosacral angle of 0·39 ($p < 0{\cdot}01$) in those patients with isthmic spondylolisthesis.

Thirty-two per cent of those with a pars defect had a spina bifida occulta, but their mean slip ratio of 20% was not significantly different from the slip ratio of the 16% for patients without spina bifida. Only 8% of those with degenerative spondylolisthesis had a spina bifida.

The age distribution and sex of the four groups is shown in Table 1. Seventy-eight per cent of patients with degenerative spondylolisthesis were women, and they were two decades older than those with isthmic spondylolisthesis. Eighty-five per cent of the pars defects occurred at L_5/S_1 and 76% of the degenerative spondylolisthesis at $L_{4/5}$ (Table 2).

Table 3 records the spinal and peripheral joint mobility. Spinal flexion measured by goniometer was significantly greater in both types of spondylolisthesis than in other back pain patients at T_1 and S_2 ($p < 0{\cdot}01$) but was not greater than in the volunteer non-back pain subjects. Hyperextensiometer readings were not significantly different for the three groups of patients and volunteers.

Table 2. The vertebral level of displacement in isthmic and degenerative spondylolisthesis

	Isthmic spondylolisthesis	*Degenerative spondylolisthesis*
$L_{3/4}$	2%	12%
$L_{4/5}$	15%	76%
L_5/S_1	85%	12%

More patients with both isthmic and degenerative spondylolisthesis could stoop forwards with straight knees and place the flat of their hands on the floor than could other back pain patients, but this could be accomplished by a similar number of non-back pain volunteers despite their younger mean age.

A childhood injury with back pain was remembered by 17% of patients with isthmic and 7% of patients with degenerative spondylolisthesis, but none of the back pain patients without spondylolisthesis could recollect a significant back injury in childhood (Table 4). Patients with degenerate spondylolisthesis assessed their athletic ability at school less favourably than other patients. All the patients with spondylolisthesis had been born by vaginal delivery.

The slip ratio was not significantly different in those patients with and without a spina bifida (Table 5). It was not apparently related to the patient's self-assessment of athletic ability at school. Eight patients who remembered a childhood injury before 11 years of age had a mean slip ratio of 20% (s.d. 13), whilst 12 who had an injury after 11 years of age had a lesser mean slip ratio of 14% (s.d. 5). This did not reach mathematical significance, however.

Vertebral displacement did not correlate with spinal mobility but it did correlate with the degree of hyperextension of the second metacarpophalangeal joint ($r = 0{\cdot}26, p < 0{\cdot}05$).

Discussion

The causes of vertebral displacement in spondylolisthesis are undoubtedly complex, particularly when these are conditions of multifactorial aetiology. We have attempted to investigate four possible factors: the presence of spina bifida and the associated weakness of

Table 1. Age (years) and sex distribution of patients with isthmic and degenerative spondylolisthesis, patients attending a back pain clinic without spondylolisthesis and volunteer subjects [Mean (s.d.)]

	Isthmic spondylolisthesis (n = 162)	*Degenerative spondylolisthesis (n = 81)*	*Back pain but no spondylolisthesis (n = 109)*	*Volunteers (n = 63)*
Age	43 y (15)	61 y (12)	40 y (13)	43 y (16)
Sex	58% male	22% male	57% male	48% male

Table 3. Spinal and peripheral joint mobility measured in patients with spondylolisthesis and in patients attending a back pain clinic without spondylolisthesis and in volunteer subjects [Mean (s.d.)]

	Isthmic spondylolisthesis	*Degenerative spondylolisthesis*	*Back pain patients without spondylolisthesis*	*Volunteer subjects*
Spinal flexion goniometer (°)				
Cervical T_1	98·5 (18·1)	98·0 (16·7)	90·1 (16·9)	105·6 (12·6)
Thoracic T_{12}	37·7 (16·7)	30·4 (15·4)	30·3 (15·0)	38·2 (11·5)
Lumbar S_2	11·7 (12·6)	14·5 (11·0)	6·2 (12·0)	16·3 (11·3)
Spinal flexion tape measure (cm)				
T_1–T_{12}	4·8 (1·5)	4·4 (1·5)	4·7 (1·2)	4·9 (1·6)
T_{12}–S_2	6·9 (1·7)	6·9 (1·3)	6·8 (2·0)	7·3 (1·2)
Percentage of patients able to flex forwards with:				
palms flat on floor	18%	21%	9%	23%
finger tips to floor	35%	36%	22%	62%
unable to touch floor	47%	43%	69%	15%
Mean fraction of foot contact area				
Right	0·58 (0·07)	0·59 (0·07)	0·59 (0·08)	—
Left	0·60 (0·07)	0·61 (0·07)	0·64 (0·07)	—
Mean Carter/Wilkinson score	1·03 (1·73)	0·58 (0·99)	0·75 (1·55)	1·02 (1·55)
Mean angle of hyperextensiometer	55·5 (17·8)	52·9 (14·4)	52·0 (16·0)	57·1 (14·4)

Table 4. Childhood history of back injury and self-rated athletic ability

Athletic ability	*Isthmic spondylolisthesis*	*Degenerative spondylolisthesis*	*Volunteers*
Very good	7%	2%	0
Good	36%	14%	22%
Average	32%	23%	52%
Poor	25%	61%	26%
Very poor	0	0	0

Childhood injury	*Isthmic spondylolisthesis*	*Degenerative spondylolisthesis*	*Volunteers*
Injury with back pain	17%	7%	0
Potential back injury without back pain	9%	5%	2%
No injury	74%	88%	98%

Table 5. Comparison of the slip ratio Carter Wilkinson score and hyperextensiometer in patients with isthmic spondylolisthesis with and without spina bifida [Mean (s.d.)]

	Isthmic lesion with spina bifida	*Isthmic lesion without spina bifida*	
Mean slip ratio	0·2 (0·11)	0·16 (0·07)	n.s.
Carter Wilkinson score	0·5 (0·84)	1·1 (2·0)	n.s.
Hyperextensiometer	52·7 (16·1)	60·0 (16·7)	n.s.

posterior restraining structures; the lumbosacral angle and its effect of shear[14]; the relationship with generalized joint laxity; and the effect of spinal injury in early life.

The high incidence of spina bifida in patients with isthmic spondylolisthesis[6,15] and the gross displacement frequently found in dysplastic spondylolisthesis[16], where hypoplastic posterior vertebral elements are also associated with anomalies of the attachments of multifidus and the lumbar fascia, suggests that the lack of restraining forces may permit forward displacement. Although we found a slightly increased slip ratio in the patients with spina bifida compared to those without, it did not reach significance, and spina bifida would not seem an important factor in initiating displacement.

Patients with isthmic spondylolisthesis generally have a large lumbosacral angle[17]. Blackburne and Velikas[6] showed a correlation between the slip ratio and the lumbosacral angle, which we also confirmed in this series ($r = 0{\cdot}39$, $p < 0{\cdot}01$). It does not follow that the steep angle of inclination is an initiating factor in relation to the degree of displacement. Wiltse and Jackson[18] noted that displacement increases more rapidly if spondylolisthesis is detected at the start of a growth spurt. Displacement is probably a growth phenomenon[19], the wedging of the body of L_5 and the increased lumbosacral angle resulting from a growth change with slow displacement. However, once established, this increasing angle will produce increasing shear forces, tending to accelerate the displacement.

The elasticity of the spinal ligamentous structures may be a factor in displacement. Many patients with a spondylolisthesis seem to be highly flexible. They can frequently bend forwards to place the hands flat on the floor[20]. This is not because the lumbar spine is hypermobile, but rather because the straight leg raising is so good, often well above 90°.

Bird et al. 1980[21], questioning gymnasts with spondylolisthesis, reported that a high proportion had flat feet and probably hypermobile joints. We did not record any increase in the foot contact area of the patients with spondylolisthesis, nor were we able to establish a correlation between spinal mobility and the slip ratio. This was not surprising in patients whose spinal movements were limited by back pain. We do not know whether there is a relationship between mobility and displacement in asymptomatic subjects with spondylolisthesis. The Leeds hyperextensiometer is the most useful index of metacarpal joint mobility, and hyperextension did correlate with the slip ratio ($r = 0{\cdot}26$, $p < 0{\cdot}05$), suggesting that joint mobility may be one factor affecting displacement.

Childhood injury was recollected more frequently in patients with isthmic spondylolisthesis than in other patients. Those with injury before 11 years of age had greater displacement than those with later injury, but in this small group it did not reach mathematical significance. Unacceptable bending moments may cause an isthmic defect of the pars to become unstable in infancy, or an intact pars to fracture. If this remains non-united and unstable, and if the soft tissues are unable to restrain shear, there will be a long period of growth available for those forces to elongate the pars with remodelling[18,22], and considerable displacement. A similar injury later in childhood would have less time to cause displacement by growth. The defect that remains stable may produce no displacement provided the soft tissues maintain the stability.

Although there was no single factor that correlated well with vertebral displacement, we have identified factors that can explain the variable displacement. Childhood injury produces an unstable segment, and subsequent displacement is related to both the period of remaining growth, and to one or more deficient restraining structures.

One might expect other factors to be responsible for displacement in degenerative spondylolisthesis. The slip ratio is rarely more than 15%; it does not develop before the 5th decade, it is more common in women, and affects a different vertebral level.

Newman[16] suggested tht microfracture and remodelling of the facets may permit the forces of shear to initiate displacement. This is compatible with the self-rated poor athletic ability of many of our patients, who may have had poor mineralization of bone with a risk of menopausal microfracture of the subchondral facets. We did not observe, however, that patients with degenerative spondylolisthesis had a greater lumbosacral angle than the rest of the population. Neither did they have increased spinal or peripheral joint mobility, or a high incidence of spina bifida. However, poorly developed spinal ligaments in those who had poor athletic ability at school may not have been able to restrain shear in later life. Degenerative spondylolisthesis may therefore occur in a subject with a sagittally orientated apophyseal joint at $L_{4/5}$[23]. Here there is disruption of the intervertebral disc[24], poor restraining ligaments, and a vulnerability to microfracture of menopausal osteoporotic bones.

The causes of displacement are complex, but we have suggested a model from the known facts. If this is correct, then both isthmic and degenerative spondylolisthesis are the result not only of constitutional factors, but also of preventable environmental events in childhood.

Acknowledgements

We are indebted to Miss E Key for her secretarial assistance and Mr G Swann for the illustrations.

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Brief Report: Thesis Summary

Evaluation of a technique for measuring lumbar lordosis in the clinical assessment of occupational low back pain

Stephen Simpson MB, MFOM

Post Office, Colchester, UK

This preliminary study was designed to evaluate a possible technique for the clinical measurement of spinal mobility and range of movement using a flexible ruler (flexicurve).

The study population consisted of all manual grade British Steel Corporation employees (8000 men) based in Scotland. The subject group comprised men who were experiencing low back pain which radiated below the pelvis, and who had not been able to attend work for a period in excess of 4 weeks. Subjects who had undergone spinal surgery were excluded. The pilot study lasted for 9 months, and during this time 31 subjects satisfied the protocol definitions.

The control group consisted of men who were randomly selected whilst attending a clinic for reasons other than low back pain. They had never been away from work for more than 4 weeks with back pain, and were not currently suffering back pain.

Employees were not further sub-divided according to their individual jobs. The steel industry in the UK has undergone a major transformation in recent years with the loss of thousands of jobs. There are no 'light jobs', and all require a reasonable degree of mobility.

Spinal profile and range of movement measurements were obtained by using a draughtsman's flexicurve, which was marked at the centre, and at points 10 cm either side of the central mark; the length between the two extreme points was the measuring range.

Each subject was invited to attend for medical consultation, which comprised a full medical history and orthodox spinal examination. They were asked to stand in a comfortable position, looking straight ahead, with feet together and arms hanging loosely by the side. No support was allowed.

The lumbar lordosis was examined and the flexicurve was applied to the lumbar spine in the mid-line so that the central mark overlay the apex of the lumbar curve. The flexicurve was then moulded to the spinal contour and the curve between the two extreme points traced onto graph paper. This was called the standing profile. The subject was then asked to bend backwards as far as he could without incurring pain. A further profile was then taken and this was called the extension profile. This was also transferred to graph paper. The tracings were studied and various measurements and angles were calculated.

It was found that the average standing and extension profiles were different in the two groups; subjects with low back pain had flat backs and restricted extension movements. These readings were not associated with age. Tall individuals were more likely to be low back pain sufferers.

These results suggested that the degree of lumbar lordosis was an important and previously under-rated clinical sign. As controversy surrounds the merits of many treatments for low back pain, any addition to the range of objective measuring devices is of value.

Dissertation submitted in candidature for Membership of the Faculty of Occupational Medicine

Correspondence and reprint requests to: Dr Stephen Simpson, Area Medical and Safety Adviser, Post Office, Charles House, 24 St Peters Street, Colchester CO1 1EP, UK

0268–0033/89/010064–01 $03.00

Mechanical Disorders of the Lumbar Spine

Richard W. Porter

The lumbar spine is an impressive mechanical system but it can fail from injury or abuse. This paper reviews four recognisable back pain syndromes, which result from mechanical failure, symptomatic disc protrusion, root entrapment syndrome, segmental instability and neurogenic claudication — their clinical presentation, and what is understood about their multifactoral aetiology.

Key words: joint instability; low back pain; lumbar vertebrae; spinal canal; spinal nerve roots; spine.

(Annals of Medicine 21: 361—366, 1989)

Introduction

The lumbar spine is a well designed mechanical system, with an impressive relationship between form and function, but it can fail from injury or abuse. Failure can produce several recognisable syndromes of back pain, each with a multifactoral aetiology. The clinician attempts to identify these syndromes, and directs management of the patient accordingly. The epidemiologist seeks to recognise the many factors responsible for each syndrome, in order to modify them and reduce the size of the back pain problem to society.

The are four syndromes related to mechanical disorders of the lumbar spine where there are some generally agreed criteria, — symptomatic disc protrusion, root entrapment syndrome, segmental instability and neurogenic claudication.

Symptomatic Disc Protrusion

Clinical Presentation

McCulloch (1), described several criteria which he thought should be present in order to make the diagnosis of a symptomatic disc lesion. (It is described as symptomatic, because a disc protrusion can be present without symptoms). He expected three of the following five criteria to be present:

1. Unilateral leg pain in a typical sciatic root distribution, including discomfort below the knee.
2. Specific neurological symptoms incriminating a single nerve.

From the Royal Infirmary, Doncaster, England.
Address: Richard W. Porter, M.D., F.R.C.S., F.R.C.S.E., Doncaster Royal Infirmary, Doncaster, England DN5 2LT.

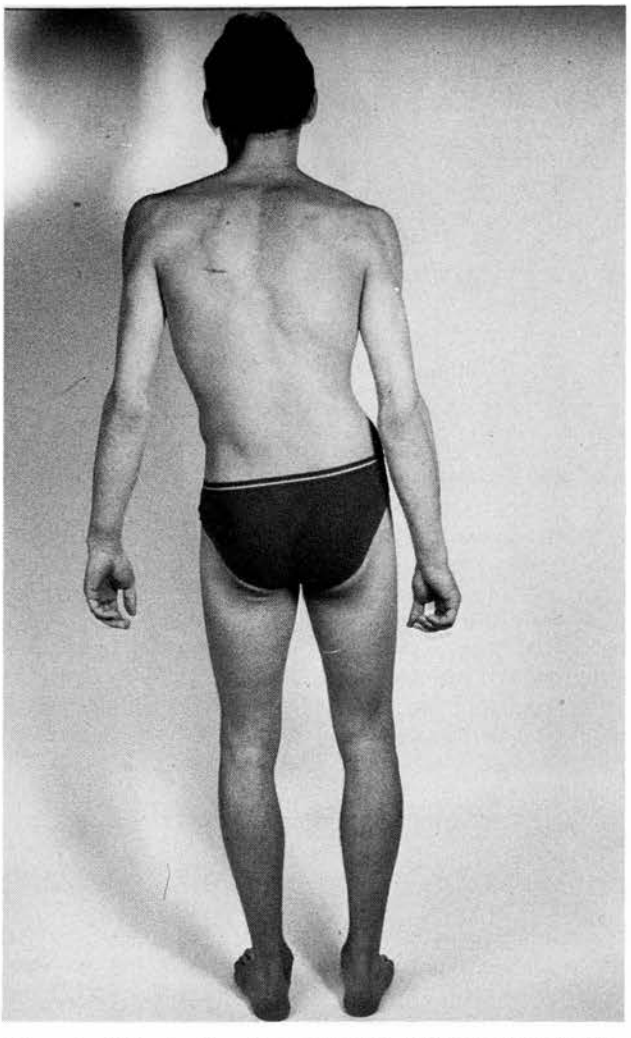

Figure 1. Patient with a disc protrusion, listing to the left when standing.

3. Limitation of straight leg raising by at least 50 % of normal.
4. At least two neurological changes of muscle wasting, muscle weakness, sensory change or hyporeflexia.
5. Myelographic evidence of a disc protrusion. (We might add computed tomography evidence also).

An interesting phenomenon observed in about a third of the patients with a disc protrusion is a gravity induced list. The pelvis remains horizontal with the floor, but the lumbar spine deviates to one side with a "windswept" appearance (Fig. 1). The list is abolished by lying down, and by hanging on a bar. It is not known if it serves a useful function. We observed the list in 5.6 % of patients attending a back pain clinic (2). Of 100 listing patients 71 had pain in a sciatic distribution below the knee, and 49 fulfilled McCulloch's criteria for a symptomatic disc lesion. Twenty subsequently had surgical excision of the disc. It has a poor prognosis for conservative management.

A further useful sign is cross leg pain, where straight leg raising of the painless leg produces pain on the opposite affected side (3). The pain is generally felt in the buttock or thigh. This also has a poor prognosis for conservative management. Almost half of our patients with both a list and cross leg pain subsequently required surgery. These two signs are, however, two of the best predictors for a good surgical result (4).

A common symptom associated with a disc protrusion that is producing root signs, is an aggravation of the pain when coughing or straining. Similarly many patients with root pain from disc protrusion experience difficulty stooping down in a morning, but the mechanism has not been explained.

These symptoms and signs, however, are sufficient for the clinician to recognise a symptomatic disc protrusion when it is involving a lumbar root, and the criteria are adequate for the epidemiologist to investigate it's multifactoral aetiology.

Multifactoral Aetiology

The disc fails when the nucleus pulposus extrudes through a fissure in the annulus or as a Schmorl's node through the vertebral end plate. It is the annular fissure with a space occupying lesion in the vertebral canal, that is responsible for the symptoms we have described above. The inner annulus may tear with intact outer fibres, resulting in a disc protrusion. The fissure may extend through the annulus with an extruded nucleus into the vertebral canal, or a complete fragment of nucleus may sequestrate through the annulus. There has been much debate about the forces required to tear the annulus. Hickey and Hukins (5) suggested that rotation was important, whilst Adams and Hutton (6, 7), believed that compression of the flexed spine is particularly damaging. It may be that compression in flexion or torsion is likely to constitute the first injury to an intact annulus (5). This mechanism of injury fits the known facts of the functional anatomy of the spine, and also the clinical explanation of the first injury, often recounted by many patients — a fall or an unexpected strain causing the patients to twist whilst in the stooping position.

Disc resilience is probably as important as the forces applied to the disc. There is some evidence that disc resilience is related to the spinal stress in the developing years. In other anatomical sites, form and function go hand in hand, and hard work may be expected to produce a strong annulus. Kelsey (8) showed that there was a greater than expected incidence of disc protrusion in sedentary workers. We also showed that coal miners has a lesser incidence of disc protrusion than other workers in the population (9).

There is increasing interest in disc nutrition. The disc is the largest avascular structure in the body, and its resilience to withstand injury depends on adequate nutrition. It depends on a satisfactory capillary bed in the vertebral end plate, and can be affected by bony sclerosis, from raised intraosseous venous pressure or the age related changes in the intraosseous arterial anatomy (10). Frymoyer and his colleagues (11) have shown that back pain is more frequent in smokers and in individuals subjected to high frequency vibrations, which may affect disc nutrition.

There are diurnal variations in the disc volume dependant on the hydrostatic pressure. This creep effect on the disc is probably beneficial to nutrition, but will make the disc most vulnerable to annular damage when the disc height is greatest, in the early morning.

There is a familial association in disc protrusion with a higher occurrence than expected in first degree relatives of affected patients (12). It has not been shown that this is a genetic association.

It is unrealistic to consider only the many factors that affect disruption of the annulus, because annular tears and disc protrusion can occur without significant symptoms. The space available for the protrusion, and the mobility of the nerve root is equally important. There is a large weight of evidence that subjects with small vertebral canals are at greater risk of symptomatic disc protrusion than those with larger canals (13—16). A small midsagittal diameter, a trefoil shape, and a small root canal, is an unhealthy anatomy, should that subject develop a disc protrusion.

Nerve roots tethered by dural ligaments (17) and anomalous roots (18), will be more affected by disc protrusion than freely mobile roots.

Root Entrapment Syndrome

Clinical Presentation

Entrapment of the lumbar nerve root in the root canal, or in the lateral recess of the central canal, produces a clinical syndrome distinct from that of the acute disc lesion. In this condition the root is usually affected by bony and soft tissue encroachment into the canal. Both conditions produce pain in the leg, but their presentation and management are distinctly different.

The sciatic pain is different in character. It is described as a severe pain often unremitting day and night. Whilst pain from a disc is frequently relieved by lying down, this pain is so troublesome it can make the patient get up and walk about. Sitting is uncomfortable

and driving far impossible. Some patients demonstrated a claudication element to the root pain, with discomfort walking far, relieved by stopping and changing posture. The claudication pain is in a root distribution. One then suspects a vascular component, either venous engourgement or arterial insufficiency of one root. There are other characteristics which differ from disc symptoms. The pain is not affected by sneezing or coughing. It is not difficult to reach down to the foot in a morning. There is no list.

The signs also differ. Straight leg raising is generally good (19). There may in fact be no abnormal neurological signs, though the symptoms can be severe. Lumbar flexion can be good, but extension is usually limited.

Most have radiological evidence of degenerative change, reduction of the L5/S1 disc space being the most consistent finding. In expert hands, electromyography can provide evidence of root dysfunction, and it will sometimes identify the root affected (20). Other studies suggest the nerve root can not be well predicted with electrical studies (21). Radiculography has a poor sensitivity (22). Computed tomography (CT) is probably the best method of demonstrating the degenerative encroachment, but of course, demonstrable pathology does not mean it is the cause of the symptoms. Nerve root infiltration with contrast medium and local anaesthetic can help identify the affected root (23). The absence of fat in the root canal and in the lateral recess can be demonstrated by magnetic resonance imaging, and will support the diagnosis of root entrapment syndrome (24).

Multifactoral Aetiology

The size of the root canal, and the shape and size of the central canal at maturity is highly significant. The development of degenerative change in bone and soft tissues will compromise the nerve root in a developmentally small canal. A variety of pathology can affect the root in the lateral recess of the central canal, in the root canal, or at the foramen (25). Posterior vertebral bars, osteophytes at the margins of the apophyseal joint, an overhanging medial lip of the superior facet, thickened ligamentum flavum, capsular thickening of the apophyseal joint, fibrosis from previous disc protrusion, hypertrophy at the margins of a spondylolysis, iatrogenic scarring, — all can contribute to reducing space, and excursion of the root.

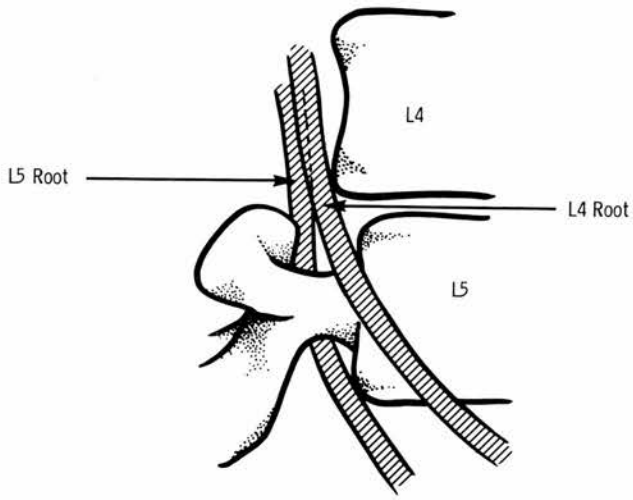

Figure 2. Diagram to show that posterior displacement of an L4 vertebra can affect the L root in the root canal, or the L5 root in the lateral recess of the central canal.

A dynamic component to the pathology is added by segmental displacement in any of the three planes of rotation, and by excessive or erratic segmental movement (Fig. 2). Postural movement, especially extension, can compromise the root and precipitate symptoms.

The patho-physiological changes in the nerve root have yet to be resolved. The neural elements are probably vulnerable to arterial ischaemia, the hypoxia of venous engourgement, and alteration in the axonal transport system. Root claudication probably results when the dynamic and vascular component are significant.

Wiltse and his colleagues (26) described an unusual cause of root entrapment — the far out syndrome — where gross displacement of an isthmic spondylolisthesis compresses the L5 root between the fifth lumbar transverse process and the alar of the sacrum.

Although the pathological changes of root entrapment must persist, it is suprising that in the majority of patients the symptoms slowly resolve (27). The peak incidence occurs in the sixth decade, suggesting that some age-related factors are beneficial in later life.

Segmental Instability

Clinical Presentation

The definition, pathology and clinical manifestation of instability enjoys less agreement than the other three syndromes. It is accepted, however, that in health the spinal segments have a defined arc of rotation, and that abnormal segmental movement can develop. There may be excessive movement, or erratic movement in any of the three planes of rotation. A displaced vertebra should probably not be considered unstable, unless there is associated unnatural movement.

Excessive or erratic movement may compromise the canals contents, producing the symptoms of root entrapment or neurogenic claudication, but these symptoms should probably not be considered instability syndromes. Rather, instability is one factor in the many factors responsible for these syndromes.

Instability symptoms are of two types:

a) Symptoms of fatigue in the structures which restrain shear. The posterior bony elements of the vertebral arch, with the coronally orientated lower lumbar apophyseal joints, are the major restraining structures. When these fail, either by deficiency of the pars interarticularis, or by disorganisation of the apophyseal joints, then the ligamentous and muscular structures are liable to fatigue (28, 29). This probably results in backache, with or without referred pain round the pelvis or into the posterior things. It is aggravated by walk-

ing far, especially shopping, which involves stopping and starting and carrying. Neither can these patients stand for long. They prefer to sit down, or even lie on the floor for relief, when the pain resolves.

b) Symptoms of momentary subluxation. The pain source may be in the apophyseal joints, in the ligamentous structures or in the dura. It is experienced as the deformed spine returns to the pre-deformation position. Typically the patient experiences pain rising from the stooped position, or pain getting out of a low chair. When rising they have to support themselves by taking their weight with their hands on their knee, or on the arms of a chair.

The patient with symptoms of instability frequently exhibits an exceptionally good range of forward flexion. We have found that many can easily reach down to place their hands flat on the floor, which is uncommon in other patients with back pain (30). They may have an "extension catch", when the normal smooth motion of standing upright after stooping, is broken by a jerky movement in the middle of the range. To prevent this, some patients use their hands to support the spine by "climbing up their legs".

MacNab described radiological signs of an unstable segment, with some disc space narrowing and traction spurs of the adjacent vertebrae (31) (Fig. 3). Herkowitz et al. (32) suggested that more than 2 mm of segmental displacement of flexion and extension radiographs is evidence of instability, but this is probably not a very reliable sign. More promising is the clinical application of standard lateral flexion radiographs measuring the length of the centrode (33).

Multifactoral Aetiology

Instability results from a failure of structures to restrain shear. These are the apophyseal joints, the intervertebral disc the intact neural arch, and the musculo-ligamentous complex.

The apophyseal joints effectively restrain shear in the lower lumbar spine, by their coronal orientation, and at L5 the torque is butressed by the broad pedicles. There is no convincing evidence that asymmetrical facets predispose to instability. We found no useful correlation between facet trophysm and traction spurs in the vertebral bodies of archaeological spines (34).

a) The lumbar segment is a three joint system and unnatural movement must occur both at the apophyseal joints and at the disc. The posterior joints will remodel as degenerative change progresses, which will contribute to, and be affected by, instability.

b) An intact disc restrains shear (35). Instability can probably occur with a healthy disc, but *in vitro,* mild disc degeneration is associated with excessive movement. Some stability is regained with gross disc degeneration. Should the surgeon incise the annulus when decompressing a degenerative spondylolisthesis, there is a risk of increasing the displacement.

c) An intact neural arch resists shear. Many patients with spondylolysis do not have symptoms, and their spines remain stable, but when back pain does occur, the most common presenting syndrome is instability (36). Root entrapment syndrome is not uncommon, but the symptomatic disc lesion, and neurogenic claudication are unusual because of the deep vertebral canal (Fig. 4). We have observed that many patients with instability symptoms have a spina bifida occulta.

d) The musculo-ligamentous complex is as important as the bony structures in maintaining stability. This includes the apophyseal joint capsules, the anterior and posterior longditudinal ligaments, the lumbar fascia with muscle attachments, and the strong lumbo-sacral ligament. The para-spinal muscles within the envelopes of the three layers of the lumbar fascia, and the psoas muscle reinforced by intra-abdominal pressure, provide an active splint for the spine. Even the powerful latissimus dorsi with its insertion into the humerus, tenses the lumbar fascia and will influence the lumbar spine stability when lifting. It is probably because of the efficient musculo-ligamentous complex that many subjects with bilateral defects of the pars, complain of no instability back symptoms.

Ligamentous laxity that accompanies the last trimester of pregnancy (37) increases the risk of segmental injury from the increasing shear forces. Not a few women date their first onset of instability symptoms from pregnancy.

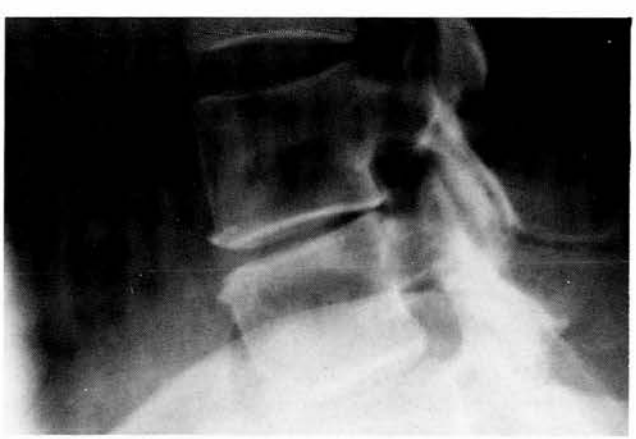

Figure 3. Lateral radiograph showing radiological features of an unstable segment. Disc space narrowing, traction spurs, and a little posterior rotational displacement.

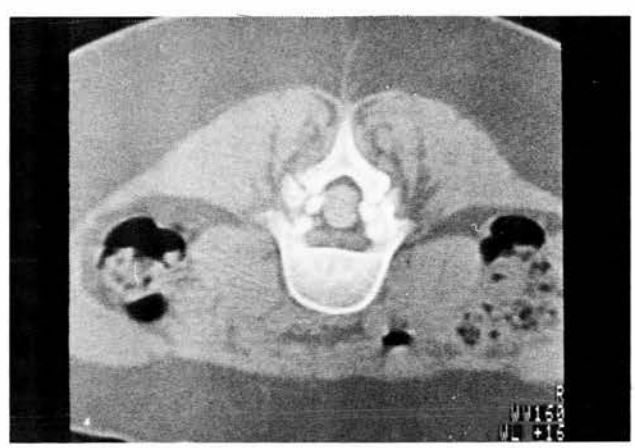

Figure 4. Computed tomography scan of an isthmic spondylolisthesis, showing the deep vertebral canal.

Neurogenic Claudication

Clinical Presentation

This intriguing syndrome usually affects men over 50 years of age, who have done manual work in the past. They complain of discomfort in the legs when walking, affecting both legs, in the thighs, calves and feet. They have difficulty describing their symptoms, speaking of "heavy legs" or tiredness. There is a threshold when the symptoms develop, and a tolerance, which is about twice the threshold, when they stop. They are limited to less than 500 m, often much less. The walking distance may vary during the day, from one day to the next, and even during one stretch of walking. They find they gradually reduce their walking speed, and stoop forwards until they finally stop, — the stoop test (38). They will lean forwards for a while, and after a few minutes the feeling in the legs recovers and they start walking again. For many patients the flexed position relieves the discomfort, and for that reason they can walk up a hill better than down, and some can cycle but not walk. Nights are often troublesome, with sleep being disturbed by restless legs and cramps when they get up and walk about. There is usually, but not invariably, a long history of back pain, sometimes with previous surgery, and claudication symptoms for some years before they seek help.

Apart from posture, the examination is remarkable for its lack of gross abnormality. Flexion can be good, but extension is absent. In fact, they can rarely stand erect, adopting the "Simian stance" (39), with the hips and knees slightly flexed. Straight leg raising (SLR) is good, reflexes normal, with power and sensation normal. The peripheral circulation is normal, excluding intermittent claudication, though peripheral arteriosclerosis can co-exist.

It is helpful to assess walking objectively on a treadmill, to record speed, posture and distance. A plain radiograph raises the suspicion of a shallow canal, and half the patients have a degenerative spondylolisthesis. A myelogram is necessary to confirm the diagnosis, and a CT scan shows the lateral extent of the stenosis.

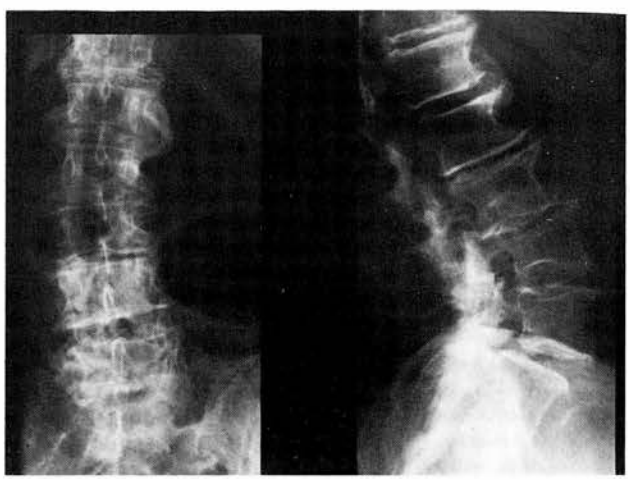

Figure 5. Antero-posterior and lateral radiographs of a 68-year-old lady with a lumbar scoliosis, and rotational displacement. She had neurogenic claudication, and could walk no more than 100 m.

Multifactoral Aetiology

Verbiest (40) recognised that neurogenic claudication was associated with a shallow canal. In fact the term "spinal stenosis" has become synonymous with neurogenic claudication, when a shallow canal is only one factor in the pathology.

The canal size is of course important. The claudicating patient has probably grown up with a developmentally shallow canal, and there are many causes for this. There is a genetic component, obvious in the achondroplastics, but there is probably a more significant environmental factor with early impairment of growth, which leaves its mark in adult life as a shallow vertebral canal (41, 42).

In addition to the developmentally small canal, degenerative change will further reduce the canal's size, with posterior vertebral bars and a rolled ridge of annulus posteriorly, thickened laminae and ligamentum flavum posteriorly, and facet arthritis and capsular thickening postero-laterally.

Vertebral displacement with an intact neural arch will critically narrow an already small canal (43, 44). Rotatory displacement with lumbar scoliosis is a particularly sinister event with a small canal (Fig. 5).

There is a dynamic component to the pathophysiological process. It is not known how much the rotatory movement of walking affects the root, but Schonstrom (45) has shown that simple axial loading will reduce the cross sectional area of the canal by 50 mm^2, and extension of the spine by 40 mm^2. Some spines are stabilised by gross degenerative change, and one might expect a number of different mechanisms to produce claudication symptoms. It is interesting to observe that most patients have multi-level lesions.

If space in the vertebral canal is important, then so is the cross sectional area of the canal's contents, which shows little correlation to the bony canal size (45). We need to know why some individuals have small canals yet a large cauda equina.

Symptoms are probably the result of inadequate oxygenation of the cauda equina, but the mechanism at present is speculative. There may be arterial ischaemia or venous engourgement which just permits adequate nerve function at rest, but inadequate function during exercise. The fact that patients are generally over 50 years of age when arteriosclerosis is becoming more common, is compatible with an ischaemic component to the pathology. There is probably localised vasodilatation of the radicular arteries in response to exercise. If space is at a premium, and segmental movement further reduces that space, the cauda equina will become vulnerable to ischaemia. An engourged extradural plexus of veins from an increased lower limb venous return, will be a further embarrassment.

The axonal transport system, and the effects of a semi-closed sac of cerebro-spinal fluid, are worthy of study in stenotic patients, but it is apparent that there are many known and unknown factors that contribute to this syndrome.

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Reprinted from SPINE, Vol. 14 • No. 6, June, 1989

Cross Leg Pain and Trunk List

B. KHUFFASH, FRCS, and R. W. PORTER, MD, FRCS, FRCSE

The prognostic significance of gravity-induced trunk list and cross leg pain was investigated in 113 patients who had root tension signs from a lumbar disc lesion. Cross leg pain, (a positive contralateral straight leg raising sign) and list was associated with poor prognosis for conservative management. There was a high incidence of disc sequestration and extrusion in the operated patients with cross leg pain. It was concluded that cross leg pain is probably a contraindication to chymopapain injections, and the surgeon should be aware of the possibility of a migrated disc fragment during operation on patients with cross leg pain. [Key words: cross leg pain, trunk list, lumbar disc, sequestration]

GRAVITY-INDUCED LIST and cross leg pain are signs of lumbar disc protrusion.[6,7] The former may be present without any root involvement, though the latter is a sign of root irritation. They are two of the best predictors of good surgical result.[2] We have examined patients attending a back pain clinic with defined criteria of disc protrusion to estimate the prevalence of these two signs and their prognostic signficance.

METHODS

Patients attending a back pain clinic were examined for criteria of symptomatic disc protrusion. They had to satisfy three or more of the criteria described by McCulloch[4]: unilateral leg pain in a typical sciatic root distribution, with discomfort below the knee; specific neurologic symptoms incriminating a single nerve; the limitation of straight leg raising by at least 50% of normal; at least two neurologic changes of muscle wasting, muscle weakness, sensory change, or hyporeflexia; and myelographic or computed tomography (CT) evidence of disc protrusion. The presence of a trunk list was noted when patients were listing in the standing position, but the list was abolished by lying down. Trunk list was not recorded if patients had measurable leg shortening, or a structural lumbar scoliosis, or if they listed only when flexing forward. Cross leg pain also was recorded if pain was increased in the contralateral leg or hip when raising the asymptomatic leg with the knee extended. Cross leg pain usually was experienced in the buttock and sometimes in the thigh. We compared the presence of these signs in those patients whose symptoms settled at home, those whose symptoms had not settled after 2 weeks of recumbency at home and required inpatient bed rest, and those whose symptoms persisted after 2 weeks' hospitalization and required diskectomy. In the operated patient, we noted whether the disc was a protrusion, extrusion, or sequestration, and also recorded the topographic position of the disc in relation to the nerve root.

RESULTS

One hundred thirteen consecutive patients with evidence of symptomatic disc protrusion were included in the study. The mean age was 33 years (rage, 23–49 years); 86 were male. Sixty patients had neither cross leg pain nor trunk list, 20 patients had a list and no cross leg pain, 21 patients had cross leg pain and no list, and 12 patients had both cross leg pain and a list. Only 10% of the patients with neither list nor cross leg pain required surgery, while 30% of those with list and no cross leg pain, 48% of those with cross leg pain and no list, and 58% of those with both list and cross leg pain, required surgery (Table 1).

The operative findings of the 17 patients with cross leg pain who required diskectomy are shown in Table 2. Only one of these patients was found to have a disc protrusion, the remainder having either a sequestration or an extrusion of the disc material. There was no consistency in the topographic position of the disc in these patients.

DISCUSSION

In this series, cross leg pain was present in 30% of patients attending the clinic with symptomatic disc protrusion and 59% of those requiring surgery. The compares with 32% of surgically treated patients of Woodall and Hayes (1950),[8] and 44% of diskectomies of Edgar and Park (1974).[1] Many patients with cross leg pain fail to respond to conservative treatment.[3,5]

Gravity-induced list is known to carry a poor prognosis.[6] In this study, a trunk list in a patient with signs of disc protrustion reduced the chance of resolution by conservative management. Cross leg pain was associated with an even less favorable prognosis, but when combined, these two signs were poor prognostic indicators for nonoperative management.

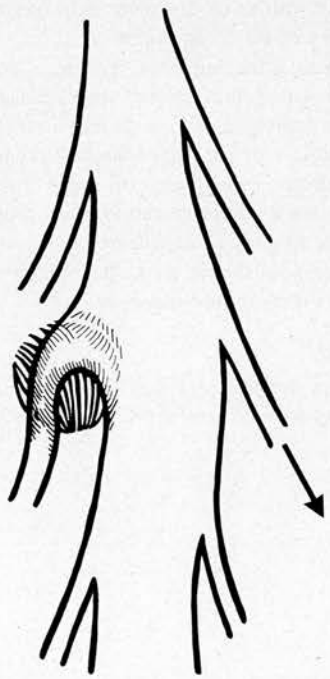

Fig 1. Diagram to show how tension on a root to the asymptomatic leg can displace the cauda equina and increase the pressure on the root overlying the disc herniation.

From the Doncaster Royal Infirmary, South Yorkshire, England.
Submitted for publication March 17, 1988, and revised October 20, 1988.
The authors would like to thank Mr. Swann for preparing the illustration and Miss Elizabeth Key for typing the manuscript.

Table 1. The Response to Conservative Treatment and Incidence of Diskectomy in 113 Patients with Symptomatic Lumbar Disc Protrusion

	Number	*Settle at home*	*Admitted*	*Diskectomy*
1. Patients without list and cross leg pain	60	28 (47%)	26 (43%)	6 (10%)
2. Patients with list	20	4 (20%)	10 (50%)	6 (30%)
3. Patients with cross leg pain	21	1 (5%)	10 (48%)	10 (48%)
4. Patients with list and cross leg pain	12	—	5 (42%)	7 (58%)

The χ^2 P values for diskectomy between Group 1 and Groups 2, 3, and 4 were $P < 0.001$; between Group 1 and Group 3 were $P < 0.006$; between Group 1 and Group 4 were $P < 0.006$.

Group 2 was significantly different from Group 1 at $P < 0.86$, and was not significantly different from the other groups.

Table 2. Operative Findings in 17 Patients with Cross Leg Pain

Level	*No. of patients*	*Operative findings*	*No. of patients*	*Site*	*No. of patients*
L5-S1	9	Sequestration	11	Axilla/anterior	9
L4-L5	8	Extrusion	3	Lateral	2
		Protrusion	1	Not recorded	6
		Not recorded	2		

We found that patients with cross leg pain generally had a sequestrated or extruded disc at operation. It is perhaps the association with disc herniation that earns this sign a poor reputation for conservative management.

It has been suggested that cross leg pain is present when the disc lesion is in either the axilla of the root, or directly anterior to the root,[3,8] when displacement of the dura toward the side of the flexed "well leg" increases tension on the opposite root (Figure 1). Edgar and Park, however, did not find the position of the disc in relation to the root to be consistent.[1] We also found no consistent topographic position of the disc in these patients. We did however, find a very high incidence of sequestration or extrusion in the operated patients with cross leg pain, only one patient having a protrusion with an intact outer anulus.

We suggest that cross leg pain is a sign that should cause the clinician to suspect that conservative management will fail, especially when combined with a gravity-induced list. It is not surprising that these also are two of the best predictors for a good surgical result.[2] Being associated with sequestration or extrusion, this sign is probably a contraindication to chymopapain injection. Failure to find a disc lesion at a time of operation in a patient who has cross leg pain should alert the surgeon to the possibility of migration of a sequestrated fragment.

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Address reprint requests to

R. W. Porter, MD, FRCS, FRCSE
Consultant Orthopaedic Surgeon
Doncaster Royal Infirmary
Armthorpe Road
Doncaster DN2 5LT
England

Accepted for publication December 28, 1988.

Reprinted from SPINE, Vol. 14 • No. 2, February, 1989

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Physical Activity and the Strength of the Lumbar Spine

R. W. PORTER, MD FRCS,* M. A. ADAMS, PhD,† and W. C. HUTTON, DSc‡

The cadaveric lumbar spines of nine young men killed in road accidents were subjected to a range of mechanical tests, and the results compared with the men's occupational and recreational histories. It was found that the compressive strength of the spines tended to increase with the level of physical activity in life, but the increase was significant only in the eight spines aged 18 or over. Compressive failure usually occurred in the vertebral body, but in three cases, the disc prolapsed into the vertebral canal; these discs came from three of the four most physically active individuals. It is concluded that physical activity strengthens both the vertebrae and the discs. A high level of activity can cause vertebral strength to exceed that of the discs. [Key words: intervertebral disc, mechanics, strength, exercise]

THE RELATIONSHIP between physical activity and the strength of the spine is still poorly understood. Vertebrae are probably strengthened by heavy labor, since weightlifters' vertebrae have a high bone mineral content[10] and this is associated with high compressive strength.[11]

Spinal ligaments should be considered dynamic structures by virtue of their attachments to the lumbar fascia,[6] and their strength therefore may be related to the strength of the abdominal and spinal muscles. They may also be strengthened by activities that flex and extend the spine throughout the full physiologic range. However, over-vigorous movements could cause joint sprains and ligament weakness.

There is little information concerning the intervertebral discs. It might be expected that their compressive strength would match that of the adjacent vertebrae, and that their resistance to bending would similarly keep pace with ligament strength, but this is by no means certain. Discs have no blood supply, and collagen turnover time is probably several years.[15] Consequently, repair processes may not be fast enough to reverse the effects of mechanical fatigue, and physical activity may lead to the disc becoming weaker, not stronger.

The epidemiologic evidence is rather confusing, possibly because most studies consider only workplace activity. Low-back pain is associated with heavy labor,[8,9,19] but disc prolapse is more common in sedentary occupations.[14,16]

The purpose of the present experiment was to investigate directly the effect of physical activity on spinal strength. The cadaveric spines of young men killed in road accidents were examined and their mechanical properties compared with the men's occupational and recreational histories.

From the *Doncaster Royal Infirmary, Doncaster, the †Comparative Orthopaedic Research Unit, University of Bristol, England, and the ‡Department of Orthopaedics, Emory University School of Medicine, Atlanta, Georgia.

Supported by a grant from the Back Pain Association.
Submitted for publication November 13, 1987, and revised May 3, 1988.

MATERIALS AND METHODS

Cadaveric Material. Nine lumbar spines were obtained from young white men who had been killed in road accidents in the Doncaster area. The restrictions imposed were that the lumbar spine should show no gross evidence of trauma, that the men should have been in good health prior to death, and that they should be aged between 16 and 40 years of age. In practice, many of them were young motorcycle riders.

The lumbar region from L1 to S2 was excised within 24 hours of death and stored in a sealed plastic bag at −17 C for up to 3 months. Prior to testing, the spine was defrosted in its bag for 12 hours at 2 C, and then dissected into two "motion segments," each consisting of two vertebrae and the intervening soft tissue. Care was taken not to damage the intervertebral disc or ligaments during dissection, and tissue dehydration was prevented by covering cut surfaces with thin polythene film whenever possible.

Mechanical Testing. Each motion segment was set in two cups of dental plaster and loaded on a Mayes servo-controlled materials testing machine. The following tests were performed:

1) Range of flexion. The specimen was subjected to combined compression, shear, and bending so that it flexed forwards in a physiologic manner, and the flexion angle was measured at which inelastic (irreversible) deformation first occurred.[5]

2) Resistance to flexion of the disc and ligaments. The specimen was flexed to the limit of its range of flexion after each of the following structures had been cut through in turn: the interspinous and supraspinous ligaments, the ligamentum flavum, and the apophyseal joints. The resistance to flexion of each structure was calculated from force-deformation curves, as described previously.[1]

The remaining vertebral body-disc-vertebral body unit was then compressed to failure. One specimen from each spine was loaded as in Test 3 and the other as in Test 4.

3) The specimen was wedged in moderate flexion (to simulate the "flat-back" posture normal in heavy lifting) and compressed to failure at 3,000 N/s. The "peak force" recorded was the compressive force resisted when the gradient of the force-deformation curve first dropped to zero.[12]

4) The specimen was wedged in hyperflexion, up to 4° beyond the end of the range determined in Test 1, and then compressed to failure at 3,000 N/s. This loading regimen previously caused posterior disc prolapse in 26 out of 61 specimens tested.[2]

After testing, the discs and vertebrae were cut through in several planes and examined for evidence of mechanical failure. The cross-sectional area of the disc was measured by counting the squares of superimposed graph paper. Disc degeneration was assessed macroscopically on a scale of 1 to 4. Vertebral body "waisting" was recorded as "absent," "slight," or "pronounced"; "waisting" may be related to mechanical loading of the spine since it increases the cross-sectional area of the discs and reduces the compressive stress acting on them.

Physical Activity of Subjects. The next of kin were interviewed (by RWP) in order to obtain a comprehensive occupational, sporting, and recreational history of each subject. On the basis of these histories, and in ignorance of the results of the mechanical tests,

Table 1. Details of the Nine Young Men whose Spines Were Used in this Study

Subject No.	Age (yrs)	Bodymass (kg)	Activity scores			Lumbar spine failure stress (N/sq cm)
			Work	Sport	Total	
1	20	70	4	5	9	558
2	18	70	1	2	3	580
3	25	73	8	9	17	752
4	20	67	1	2	3	492
5	24	86	8	7	15	639
6	19	73	4	3	7	476
7	16	76	7	6	13	464
8	23	70	4	2	6	540
9	32	67	7	4	11	590

The lumbar spine failure stress was the higher of the two values obtained for the two motion segments from that spine.

two scores for physical activity were assigned to each subject. The "sport" score was a measure of participation in sport and exercise for recreation, and it depended on both the intensity and frequency of the activities. Scores of 1, 2, and 3 were awarded for activities that could be described as "light" (eg, walking, cycling), "medium" (eg, running, racquet sports), or "heavy" (eg, rugby, football, martial arts). The score was multiplied by 2 for participation three or more times per week. The score was halved if the participation had been for less than 2 years.

The "work" score similarly indicated the intensity, frequency, and duration of physical activity required at work. Scores of 1, 2, or 4 were awarded for work that was "light" (sedentary), "medium" (light manual), or "heavy" (involving repeated or sustained stooping, lifting, or carrying). The score was multiplied by 2 if the activity was for more than 4 hours per day. Unemployed people received a score of 1.

RESULTS

Details of the nine subjects and their activity scores are shown in Table 1. In this particular sample, several of the subjects with a low "work" score also took part in little sporting activity. Since the "work" and "sport" scores showed a similar distribution, they were added to give a single measure of activity.

Table 2 gives full details of the cadaveric spines and the results of the mechanical tests. All discs were found to be nondegenerated (Grade 1 or 2), so disc grade was omitted from the table. Vertebral body "waisting" was pronounced in two spines with high activity scores, but in most cases was absent.

The bending properties of the discs and ligaments showed considerable variation, but no significant dependence on activity or age or bodymass. Compressive failure usually occurred in the vertebra, either by crushing of the vertebral body or by fracture of the end-plate. However, there were three disc prolapses, one of them occurring simultaneously with an end-plate fracture. Each prolapse involved nucleus pulposus material being extruded through the posterior annulus in a manner described previously.[2] The affected discs were from the first, second, and fourth most active individuals.

The final column in Table 2 gives the failure stress (peak force divided by the cross-sectional area of the disc) for each motion segment. The higher of the two failure stress values obtained for each spine was considered to give the best measure of that spine's compressive strength, since strength is probably related to the flexion angle used in the test[12] and the angle corresponding to maximum strength was not established. These failure stress values are shown in the final column of Table 1 and are plotted against the activity scores in Figure 1. Because of the somewhat arbitrary nature of the activity scores, a statistical test was applied that depended only on the ranking order of the scores rather than their numerical values (Spearmans rank correlation test). This showed that the increase in motion segment compressive strength with activity was not significant; however, if the 16-year-old spine was excluded (on the grounds that it might not have been skeletally mature), then the increase was significant ($P < 0.05$).

DISCUSSION

The interviews and requests for permission to examine the spines involved considerable distress for the next of kin, which made it

Table 2. The Full Results from the Mechanical Tests on Nine Cadaveric Spines

Spine	Lumbar level	Disc area (sq cm)	VBW	Range of flexion (degrees)	Bending moment resisted (Nm)				Compressive strength test			
					Disc	Facet joints	Ligamentum flavum	S/I ligament	Flexion angle	Peak force (N)	Mode of failure	Stress (Ncm^{-2})
1	2-3	17.3	−	13	11.0	16.1	15.8	6.3	16	8500	EPF	490
	4-5	20.8	−	13	6.1	17.9	9.3	9.3	6	11600	EPF	558
2	2-3	17.6	−	14	17.7	20.4	7.5	5.6	6	10206	EPF	580
	4-5	18.7	−	16	7.7	13	6.2	4.7	18	10330	EPF	552
3	2-3	18.5	++	15	11.2	17.6	8.3	3.8	6	13954	VBC	752
	4-5	20.3	++	18	8.2	12.2	4.9	6.8	20	10140	PID	499
4	2-3	16.6	−	13	2.4	6.3	6.1	6.3	6	8179	EPF	492
	4-5	19.5	−	15	3.0	14.5	8.5	7.2	19	7631	EPF	390
5	2-3	14.8	+	10	5.3	12.9	3.6	1.7	6	9460	PID/EPF	639
	4-5	15.5	+	14	3.6	15.6	9.1	3.4	18	9760	VBC	628
6	2-3	15.3	−	15	5.7	43.7	9.4	9.6	6	7310	EPF	476
	4-5	16.8	−	17	2.5	21.0	7.1	3.0	20	7710	VBC	456
7	2-3	15.9	−	18	7.5	30.8	10.3	14.1	6	5120	EPF	321
	4-5	18.7	−	22	4.1	25.6	8.2	8.8	22	8680	VBC	464
8	2-3	16.7	+	16	1.7	15.4	7.0	5.3	6	7675	EPF	459
	4-5	17.0	++	18	2.0	9.5	4.8	9.2	21	9206	VBC	540
9	2-3	18.7	−	11	16.7	21.1	6.4	3.2	6	8404	VBC	449
	4-5	19.2	−	13	10.3	19.8	9.2	6.3	16	11322	PID	590

The spine number corresponds to the subject number in Table 1.
VBW = vertebral body waisting; − = absent; + = slight; ++ = pronounced; S/I = supraspinous/interspinous; EPF = end-plate fracture; VBC = vertebral body crushing; PID = prolapsed intervertebral disc.

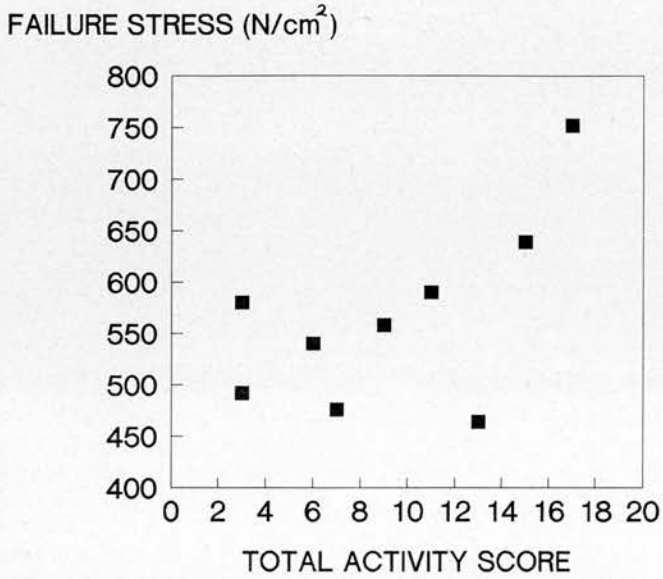

Fig 1. Graph showing how the compressive strength of the cadaveric spines varied with the "activity" score of the subject before death. The failure stress values are from the final column of Table 1.

necessary to limit the study to a small number of spines. The results are therefore of low statistical significance, and this may explain the lack of correlation between activity and the spines' bending properties. Nevertheless, certain trends can be identified that may provide the basis for future study.

The increase in motion segment compressive strength with physical activity (Figure 1) is in agreement with previous results for vertebral bodies,[10,11] and it suggests that the strength of both disc and vertebra usually keep pace with the loads applied to them. We suggest that this is the most reasonable explanation, but accept that an alternative hypothesis is possible: that subjects with strong spines engage in heavy physical work and enjoy vigorous recreational persuits.

The most active individual (Spine Number 3), who had worked as a coal miner and was a karate expert, had the strongest spine recorded in the literature. It showed pronounced vertebral body waisting. It is probably not surprising that many patients with spinal stenosis have previously been involved in heavy manual work[17,18] when bony hypertrophy and thickened ligamentum flavum contribute to a developmental stenosis. Several roentgenogram studies[7,13] and a survey of low-back pain syndromes in a mining community[16] lend support to this view.

The spine of a sedentary worker, conversely, can be relatively weak and therefore vulnerable to damage by high unexpected loading. This could explain why acute disc prolapse is more common in sedentary workers.[14,16]

In the present experiment, disc prolapse occurred in the spines of three of the four most "active" subjects. If the in vitro situation is comparable to disc prolapse in life, we could postulate that high levels of physical activity increase vertebral strength more (or more rapidly) than disc strength, so that the discs become the weak link, even though they are stronger than the discs of inactive people. The discs will then be most vulnerable to injury in the early months after starting a physically demanding job or new recreational activity, since by that time the muscles and vertebrae will be stronger than the discs, which will be in the process of adapting to increased loads. Occupational surveys will not detect these disc problems if early injury leads to premature change of employment.

When levels of activity are severe, then the discs may not be strengthened at all; instead they can accumulate fatigue damage leading to a "gradual prolapse."[3,4].

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Address reprint requests to

Dr. M. A. Adams
Comparative Orthopaedic Research Unit
University of Bristol
Park Row
Bristol BS1 5LS
England

Accepted for publication September 15, 1988.

kyphosis seems to cause problems even outside the area of fusion. To prevent these secondary changes, operative reduction of lumbosacral kyphosis may be indicated in such cases. It may be that new developments in instrumentation, such as transpedicular fixation, will make it possible to achieve and maintain an adequate correction.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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DIURNAL CHANGES IN SPINAL MECHANICS AND THEIR CLINICAL SIGNIFICANCE

M. A. ADAMS, P. DOLAN, W. C. HUTTON, R. W. PORTER

From the University of Bristol

Diurnal changes in the loads acting on the spine affect the water content and height of the intervertebral discs. We have reviewed the effects of these changes on spinal mechanics, and their possible clinical significance.

Cadaveric lumbar spines subjected to periods of creep loading show a disc height change similar to the physiological change. As a result intervertebral discs bulge more, become stiffer in compression and more flexible in bending. Disc tissue becomes more elastic as its water content falls, and its affinity for water increases. Disc prolapse becomes more difficult. The neural arch and associated ligaments resist an increasing proportion of the compressive and bending stresses acting on the spine. Observations on living people show that these changes are not fully compensated for by modified muscle activity.

We conclude that different spinal structures are more heavily loaded at different times of the day. Therefore, the time of onset of symptoms and signs, and any diurnal variation in their severity, may help us understand more about the pathophysiology of low back pain and sciatica.

During the recumbency of sleep, the loading on the intervertebral discs is reduced, and their relatively unopposed swelling pressure results in them absorbing fluid and increasing in volume (Urban and McMullin 1988). The absorbed fluid is expelled during the day when the loading of the spine is increased. There is, thus, a diurnal variation in the fluid content and height of the discs which causes a variation in the mechanical properties of the spine.

We review the experimental evidence concerning these changes, and then discuss the changes in loading of different spinal structures at different times of the day. We then suggest that the time of onset of symptoms and signs, and any diurnal variation in their severity, might be an aid to diagnosis.

M. A. Adams, PhD, Research Fellow
P. Dolan, PhD, Research Fellow
Comparative Orthopaedic Research Unit, University of Bristol, Park Row, Bristol BS1 5LS, England.

W. C. Hutton, DSc, Professor
Department of Orthopaedics, Emory University, Atlanta, Georgia, USA.

R. W. Porter, MD, FRCS, Consultant Orthopaedic Surgeon
Doncaster Royal Infirmary, Armthorpe Road, Doncaster, South Yorkshire DN2 5LT, England.

Correspondence should be sent to Dr M. A. Adams.

0301-620X/90/2027 $2.00
J Bone Joint Surg [Br] 1990; 72-B:266–70.

CADAVERIC SPINES

Periods of creep loading of cadaveric lumbar spines cause a change in disc height similar to the diurnal change seen in vivo. Certain mechanical properties have been measured before and after the period of loading.

Disc height. Constant loading at 1 000 N for six hours (simulating light manual labour: Nachemson 1981) causes disc height to decrease by 1.53 ± 0.34 mm (Adams, Dolan and Hutton 1987). The height loss is rapid at first but much slower by the end of the six hours (Fig. 1). Similar results have been reported in other experiments (Adams and Hutton 1983; Koeller, Funke and Hartmann 1984). If the applied compressive force is increased at hourly intervals from 1 000 N to 2 000 N and then to 3 000 N in order to simulate manual labour of increasing severity, then the height loss shows no sign of slowing down, and the cumulative loss after three hours is 2.13 ± 0.35 mm (Adams et al 1987, see Fig. 1).

The average diurnal variation in human stature is about 19 mm (Tyrrell, Reilly and Troup 1985) which is mostly attributable to changes in disc height (De Puky 1935). A 19 mm change in stature corresponds to a change of about 1.5 mm in the height of each lumbar disc (Adams et al 1987), so the loading regimes discussed above are sufficient to simulate physiological diurnal reduction in disc height.

Changes in disc height are caused by fluid exchange and creep deformation of the annulus fibrosus (Koeller

et al 1984). The relative importance of each mechanism probably depends upon the severity and duration of loading (Adams et al 1987) and factors such as age and the degree of disc degeneration.

The diurnal disc height change of 1.5 mm is of a similar magnitude to the normal narrowing of the lumbar discs expected with age (Koeller et al 1986). It could have a significant effect when there is pathology in the nerve root canal since the total height of the lumbar intervertebral foramen averages only about 15 to 20 mm (Panjabi, Takata and Goel 1983).

Disc water content. Creep loading reduces the water content of the discs. After four hours loading at about 700 N, when disc height is reduced by about 1.5 mm, the average fluid loss is 12% from the annulus and 5% from the nucleus (Adams and Hutton 1983). Discs from people under the age of 35 years lose almost twice this amount. Most of the fluid loss probably occurs in the first few hours of loading because longer-term creep tests cause only a little more fluid loss: 24 hours loading at about 1 000 N reduces the fluid content of annulus and nucleus by 11% and 8% respectively (Kraemer, Kolditz and Gowin 1985).

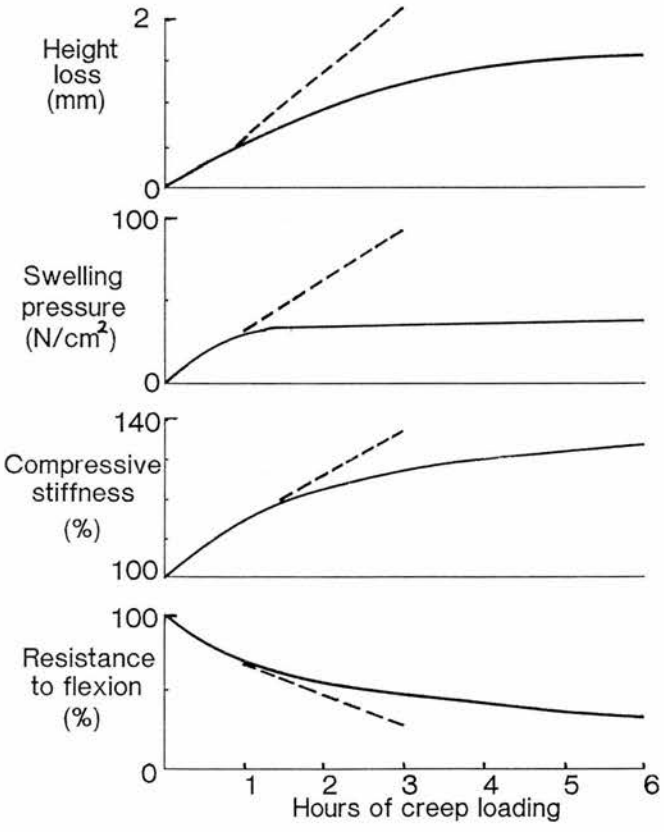

Fig. 1

The effect of compressive creep loading on some mechanical properties of lumbar motion segments. The solid lines refer to a constant compressive force of 1 000 N applied for six hours. The broken lines refer to a compressive force of 1 000 N in the first hour, 2 000 N in the second, and 3 000 N in the third. Resistance to flexion was measured at the physiological limit determined before creep loading. The 'swelling pressure' of an intervertebral disc is defined in the text.

Fluid loss is accompanied by a reduction in energy dissipation during a loading/unloading cycle (Koeller et al 1984). This means that the dehydrated disc behaves more like an elastic solid and less like a viscous fluid.

Disc swelling pressure. Disc swelling pressure can be defined as that physical pressure which must be applied to the disc in order to prevent it from swelling up in saline (Urban and McMullin 1988). It is a measure of the tissue's affinity for water.

Swelling pressure can be measured by adjusting the compressive force acting on a motion segment until there is no detectable change in disc height. This force is then divided by the cross-sectional area of the disc. Swelling pressure increases rapidly during creep loading as shown in Figure 1, and its rise can be accelerated by more intense loading (unpublished results from our laboratory, 1988). The clinical significance of swelling pressure is that it determines the rate at which a disc recovers lost height (and mechanical properties) at the end of a period of high loading.

Compressive stiffness. Creep loading increases the disc's compressive stiffness. The increase is about 50% after two to three hours of physiological cyclic loading (Koeller et al 1984) and can rise to 100% after 28 hours (Smeathers 1984). Motion segment stiffness is of clinical significance because it determines how much the disc and surrounding soft tissues deform during physiological dynamic loading of the spine.

Disc bulging. Radial bulging of the disc has been observed to increase after creep loading (Koeller et al 1984). The size of this increase has not been measured directly, but it may be inferred from the results of Brinckmann and Horst (1985). They altered the volume of the disc, either by injecting fluid into it or by fracturing the vertebral body end-plate, and found that the change in radial bulging was about one-third of the change in disc height. This suggests that the diurnal reduction in disc height of 1.5 mm should be accompanied by an increased radial bulge of about 0.5 mm. For comparison, the increased radial bulge caused by increasing the compressive force on the spine from 300 N (lying in bed) to 1 000 N (light manual work), is only about 0.2 mm (Brinckmann and Horst 1985).

Diurnal disc bulging will have clinical implications when the central or root canal is stenotic. The width of the intervertebral foramen is normally about 8 to 10 mm (Panjabi et al 1983) but it can be much less in the root canal and the lateral recess.

Loading of the apophysial joints. The compressive force on the apophysial joints has been measured when motion segments were loaded to simulate a typical sitting posture (lumbar spine slightly flexed) and an erect standing posture (spine in slight extension). Before creep, the apophysial joints resist little, if any, compressive force in either posture (Adams and Hutton 1980). After creep, there is little resistance in the flexed spine, but in the simulated standing posture the apophysial joints resist

an average of 16% of the applied compressive force. In some cases, the proportion can be as high as 70%. In more extended postures, compressive creep loading can result in high stress concentrations on the lower margins of the apophysial joint surfaces (Dunlop, Adams and Hutton 1984) and capsule (Yang and King 1984).

Forward bending properties. Creep loading increases a motion segment's range of flexion by 2° or 3° (Adams et al 1987) which is equivalent to about 12.5° extra movement for the whole of the lumbar spine. At the physiological limit of flexion (as determined before creep loading) the motion segment's resistance to bending is reduced by about 70% (Fig. 1). The reduction for the disc and ligaments, measured separately, is 85% and 45% respectively.

performed in the early morning generate much higher stresses in the osteoligamentous lumbar spine than do similar movements performed later in the day. Also, the discs resist a higher proportion of these increased stresses in the morning.

Backward bending properties. Creep loading reduces the disc's resistance to backward bending by about 40% (Adams, Dolan and Hutton 1988). This is balanced by increased resistance from the apophysial joints and spinous processes, so that the resistance to backward bending of the whole motion segment, and the range of movement, are unaltered by creep loading.

Prolapsed intervertebral disc. Some cadaveric discs can be induced to prolapse posteriorly by loading them in combined bending, shear and compression. Of 61 motion segments loaded in this way, 26 failed by posterior disc prolapse (Adams and Hutton 1982). The experiment was repeated on a further group of 19 motion segments from cadavers of a similar age range after they had been creep loaded (Adams et al 1987). Only two of these discs prolapsed, and they were both from the same spine.

Creep-loaded discs, in vivo, may also be less susceptible to prolapse, perhaps because of the reduced fluid content of the nucleus pulposus and the reduced flexion stresses in the posterior annulus.

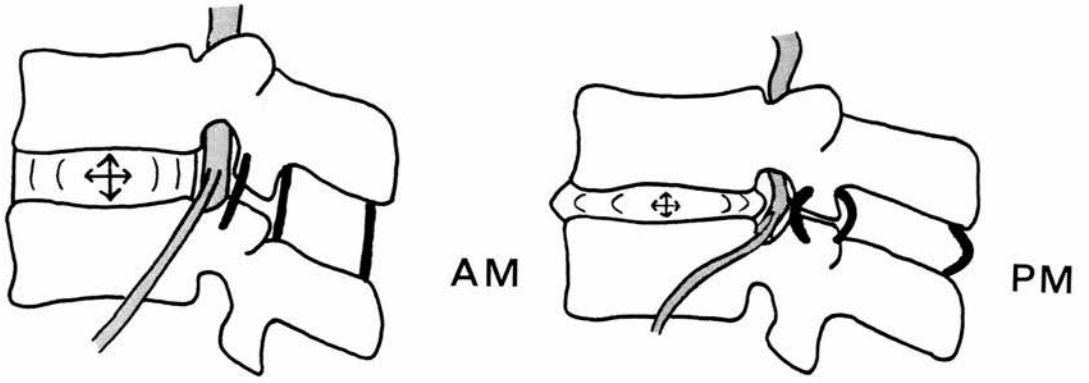

Fig. 2

Diagrams showing the diurnal changes in spinal mechanics (see Table I).

Table I. Diurnal variation of maximal stress on various structures in the lumbar spine

	Period	Comment
Intervertebral disc	AM	Especially in bending
Posterior longitudinal ligament	AM	Especially in flexion
Vertebral body end-plate	AM	
Segmental nerve root	AM	Increased tension
	PM	Increased compression
Apophysial joint		
Articular surface	PM	
Capsule and ligaments	AM	Flexion
	PM	Extension
Supra/interspinous ligaments	AM	Increased tension in flexion
	PM	Increased compression in extension

These effects can probably be attributed to the reduce fluid content of the nucleus pulposus. If the fluid content is artificially raised (by injecting saline) or lowered (by injecting chymopapain) then there is a corresponding increase or decrease in the disc's resistance to bending (Andersson and Schultz 1979; Dolan, Adams and Hutton 1987).

These results indicate that, in life, flexion movements

CLINICAL DIURNAL VARIATION

Spinal posture and mobility. It is possible that, in vivo, the muscles of the back and abdomen may modify spinal posture in order to compensate for some of the diurnal changes in the underlying spine. However, unpublished results from our laboratory show that the lumbar lordosis increases by about 3° during the day. This would increase the loading of the apophysial joints and compound the effects due to loss of disc height.

It could be thought that the higher bending stiffness of the osteoligamentous spine in the early morning would

be offset by the trunk muscles restricting the range of bending, so that the bending stresses on the spine remain the same. However, the experimental evidence suggests that this does not happen to any significant extent. In vivo, the range of lumbar flexion is reduced by only 5° in the early morning (Adams et al 1987) whereas the range of flexion of the underlying spine is reduced by about 12.5° before creep loading (see above). Calculations comparing the in vivo and in vitro evidence suggest that, in life, bending stresses on the lumbar discs and ligaments can be increased by about 300% and 80% respectively in the early morning (Adams et al 1987).

The slight diurnal variation in the range of spinal movement in vivo may be partly attributable to muscle 'warm-up' (Baxter 1987). A similar effect has been observed in hip movements (Adams et al 1987) even though there is unlikely to be any variation in the mechanical properties of the underlying joints, since normnal articular cartilage does not swell up overnight like the disc.

Low back pain. There is little published information about diurnal variations in symptoms and signs, nor in the time of onset of low back pain. Varma (1987) recorded that 47% of 'first episodes' of back pain occurred in the early part of the working day. This agrees with a study by Evans et al (1980) who found that mine workers sustained spinal injury more commonly in the morning.

Accidents in general tend to become more frequent towards the end of a working shift, when people are tired and inattentive. These effects will tend to mask any trends due to changes in spinal mechanics and they must be taken into consideration in any surveys of diurnal variation in spinal injuries.

DISCUSSION

The experimental evidence can be summarised as follows: with creep loading, the intervertebral discs lose height, bulge more, become stiffer in compression and more flexible in bending. Disc tissue becomes more elastic as its water content is reduced, and its affinity for water increases. Disc prolapse becomes less likely. The neural arch and associated ligaments resist an increasing proportion of the compressive and bending stresses acting on the spine. These results are summarised in Figure 2.

In life, these changes will occur mostly in the first few hours of the day, but the time scale and the magnitude of the changes will depend upon the severity of loading on the spine: heavy labour will have a greater effect, and in less time, than sedentary activity. The swelling pressure results suggest that the effects of intense loading will be reversed more rapidly than the effects of less intense activity of longer duration. Alternating periods of rest and activity throughout the day probably cause minor changes in spinal mechanics similar to the diurnal changes.

We suggest that diurnal variations in spinal mechanics are of clinical significance. Since different structures are more heavily loaded at different times of the day, the time of onset of a patient's symptoms, and any diurnal changes in their degree of severity, might help us to understand the pathophysiology of different back pain syndromes.

Table I lists some of the structures thought to be responsible for low back pain and sciatica, and indicates when they are most heavily loaded. The *disc* resists all of the compressive force on the spine in the morning, and in addition, is much more highly stressed during flexion and extension movements. A herniated disc, however, may behave differently from one with an intact annulus. The *posterior longitudinal ligament* is stretched more in the morning because of the increased height of the disc, although reduced radial bulging of the disc will counteract this effect to some extent. *Vertebral body end-plates* can be ruptured by increasing the fluid content of the nucleus pulposus (Jayson, Herbert and Barks 1973) so it is likely that the end-plates are more highly stressed in the morning when the discs are swollen with fluid.

The *segmental nerve roots* are stretched more in the morning because the spine is about 19 mm longer; but they are compressed more in the afternoon by extra radial bulging of the disc, by the reduced height of the intervertebral foramen, and by the buckling of the ligamentum flavum. The *apophysial joint surfaces* are pressed closer together in the afternoon, especially in lordotic postures and during backward bending, and at this time joint pain may be expected to increase. However, the *capsular ligaments* and the *supraspinous and interspinous ligaments* will all be most stretched during forward bending movements performed in the early morning because the increased disc height allows them less slack.

As we attempt to understand more about spinal pathology, clinical studies into the diurnal changes of spinal symptoms and signs could be rewarding.

This work was supported by the Arthritis and Rheumatism Council, Action Research for the Crippled Child and the Back Pain Association.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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Reprinted from SPINE, Vol. 15, • No. 2, February, 1990

Diurnal Changes in Straight Leg Raising

R. W. PORTER, MD, FRCS, FRCSE, and I. F. TRAILESCU, MD

Diurnal changes in straight leg raising were measured in 28 patients with criteria of lower lumbar disc protrusion, having first established an acceptable intra-observer repeatability using an oil-filled precision goniometer. Eight patients showed little change between the straight leg raising after a night of recumbancy, and after 180 minutes in the upright posture. Twenty patients, however, had 10 or more degrees of improvement in straight leg raising, with a mean improvement of 16.9° (SD 7.1°); most of the increase occurred in the first hour of being upright. The measurement returned again to the base level after 120 minutes of further recumbancy. Five of the eight patients who showed little diurnal change had discectomy, and four had a complete annular tear. Only one of the 20 patients with more than 10° of diurnal change required surgery. Diurnal change in straight leg raising is probably related to the disc's proteoglycan content, its hydration, the tension of the protrusion and the presence of intact peripheral annular fibers. [Key words: disc, protrusion, sequestration, diurnal, straight leg raising]

DIURNAL CHANGES in the intervertebral discs have been extensively studied *in vitro*,[9] with the disc's fluid content depending on the proteoglycan in the tissues, and on the relationship between the external load and the disc's swelling pressure. *In vivo* also, the diurnal variation in stature is largely attributable to changes in disc hydration and height.[1,8] Little is known, however, about diurnal changes in the spine in the presence of pathology, and the effects these may have on symptoms and signs. Straight leg raising in particular, being a root tension sign, may change with disc hydration and the tension of a protrusion.

In order to measure diurnal changes in straight leg raising, the test's repeatability must be adequate. We have therefore performed an intra-observer repeatability study, and then recorded diurnal changes in straight leg raising in a group of patients with lower lumbar protrusion.

METHODS

Straight leg raising was performed with the patient resting supine on a firm surface, and the head supported on a flat pillow. An oil-filled precision "Rippstein" goniometer was placed on the anterior surface of the lower tibia at a site where the pointer recorded "zero" (Figure 1), and its position was marked on the skin with a felt-tipped pen. The examiner placed one cupped hand beneath the heel, the other supporting the goniometer. With the patient maintaining an extended knee, and avoiding pelvic movement,[3] the leg was lifted slowly until it was stopped voluntarily by pain in the back or leg.

We conducted an intra-observer repeatability study using 60 paired measurements from six patients with root tension signs. The examiner (I.F.T) was blind, as the face of the goniometer was directed towards the scribe (R.W.P) (Figure 2).

From the Doncaster Royal Infirmary, Doncaster, England.

Submitted for publication March 29, 1989, and revised October 5, 1989.

The goniometer was designed and manufactured by J. Rippstein, Pierrettes 54, CH-1093 La Conversion, Switzerland, from whom similar goniometers may be purchased.

We are grateful to Mrs. J. Reynolds for her secretarial assistance, and to Mr. G. Swann and Mr. A. Bryden for the illustrations.

Diurnal changes in straight leg raising were then examined in a series of patients who had criteria of lower lumbar disc protrusion. They had unilateral leg pain below the knee in a typical sciatic root distribution incriminating a single nerve; straight leg raising was 50° or less; they had either a gravity-induced trunk list, or at least two neurological changes of muscle weakness, muscle wasting, sensory change or hyporeflexia.

Straight leg raising was recorded after the patients had a night of recumbancy, and before they got out of bed. This was then repeated after they had assumed the upright posture (sitting, standing or walking) for 15, 30, 60 and 180 minutes, and after a further 120 minutes of recumbancy in those patients whose straight leg raising had improved by at least 10°.

RESULTS

The intra-observer repeatability of straight leg raising for 60 paired measurements from six patients was 1.6° SD 1.4° (Table 1).

Thirty patients with criteria of disc protrusion were examined for diurnal changes in straight leg raising. Two were excluded because of difficulty in obtaining repeatable measurements. Eight patients showed less than 10° change by assuming the upright posture for 180 minutes (Table 2A, Figure 3).

Twenty patients, however, after being upright for the same period of time, had 10° or more improvement in straight leg raising (Table 2B) with a mean improvement of 16.9° (SD 7.1°). Most of this increase occurred in the first hour (Figure 4).

Six patients failed to improve after 2 weeks of hospital bed rest, and were treated by discectomy. Four of these had sequestrated or extruded discs with a complete annular tear, and these had shown little diurnal change in straight leg raising (Table 2). Two with protrusions, where the outer annulus was intact, had 6° and 14° of change, respectively.

Twelve patients had a radiculogram; eight with disc lesions at L4/5 had a mean improvement in straight leg raising of 16.1°; four with L5/S1 lesions had a mean improvement of 6.3°.

DISCUSSION

The intra-observer repeatability of straight leg raising is poor. Nelson et al[5] recorded about 10° between observers; Kosteljanetz found that 70% of his measurements were within 10° of his colleagues,[4] but they were recorded at intervals of several hours, and could have been influenced by diurnal change. With one examiner, we were able to measure to a surprising mean repeatability of 1.6°, which we attribute to the use of a precision oil-filled goniometer, a standard technique marking the position of the goniometer on the skin, and measurements in sequence.

It has been suggested that straight leg raising improves by consecutive testing over a short period of time.[4] Though we observed this in two patients it was not generally our experience. We found that although the second of the paired measurements in the repeatability study was greater than the first on 20 occasions, on 7 it was less. We are confident that the improvement we observed when the patient became upright was not due to repetitive measurements. Between the first and third hour, the test was not repeated, and yet the initial improvement was maintained. Twenty of 28 patients showed improvement of 10° or more which, after 2 more hours of recumbancy, invariably returned towards the baseline

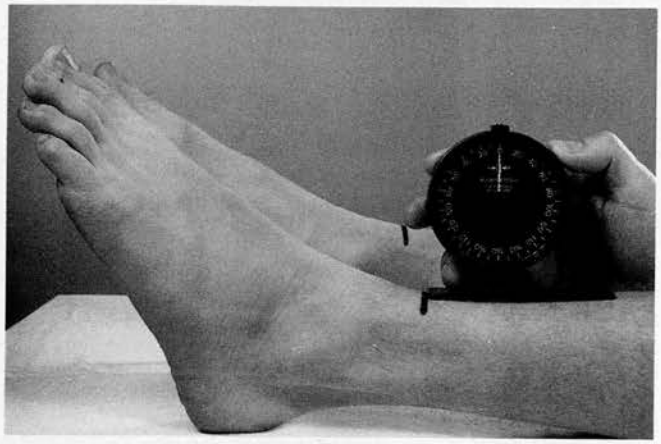

Fig 1. A "Rippstein" goniometer positioned on the lower surface of the tibia with the pointer at zero.

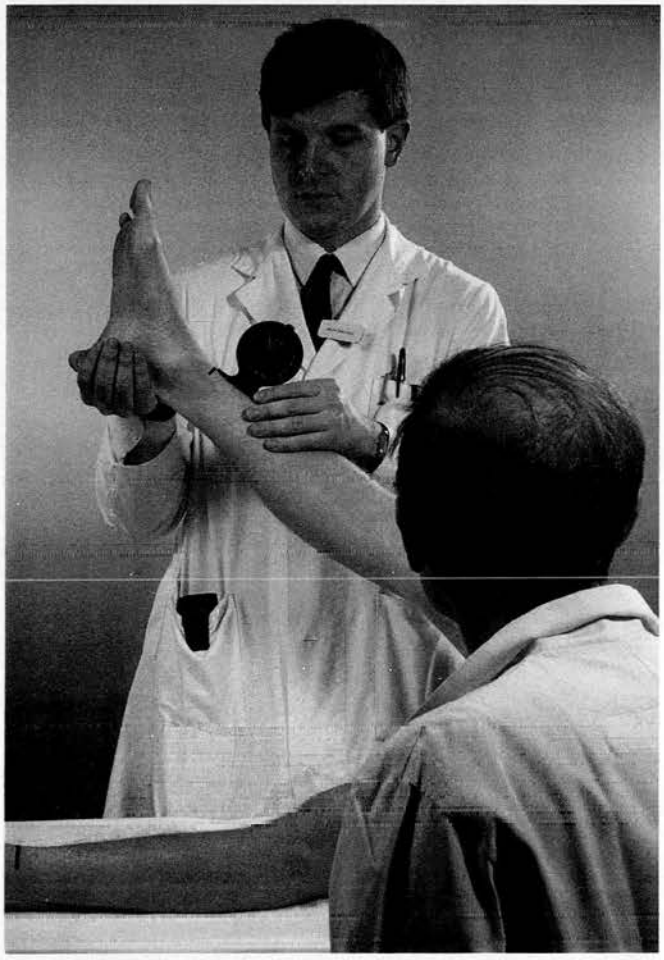

Fig 2. Straight leg raising for the repeatability study recorded with the goniometer facing away from the examiner.

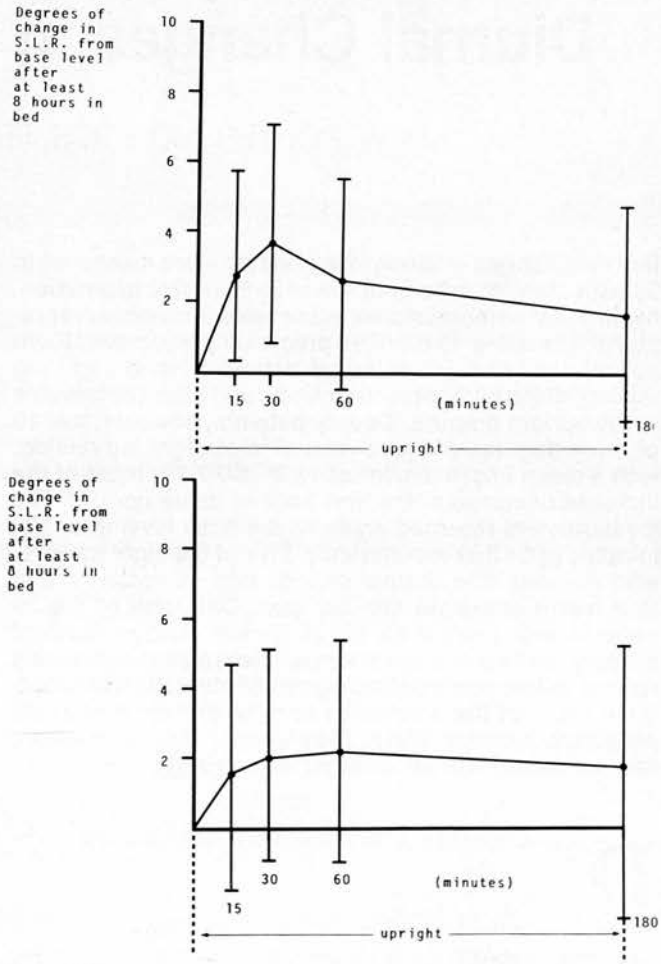

Fig 3. The mean change in straight leg raising (SLR) and standard deviation (in degrees) after 180 minutes in the upright posture following recumbancy, for eight patients who had less than 10° of change, **A**, for the symptomatic (most affected) leg, and **B**, for the asymptomatic (least affected) leg.

Table 1. Intra-Observer Repeatability of 60 Paired Measurements on Six Patients

Patient number	*Number of paired measurement*	*Mean error (degrees)*	*SD (degrees)*
1	12	1.6	1.5
2	8	1.4	1.6
3	12	1.3	1.3
4	8	2.5	1.4
5	8	1.5	1.4
6	12	1.8	1.3
Combined	60	1.6	1.4

again. These results can be explained in terms of disc hydration and changes in the tension of a protrusion.

The disc imbibes fluid during recumbancy when the external load is reduced, and the osmotic pressure is relatively unopposed. The disc space increases with increasing hydration, and the bulge of a healthy annulus becomes less.[2] By contrast, a weakened disc protrusion acts like a safety valve, bulging further with increasing hydration, the outer fibers becoming tense at the expense of increasing the disc space. When the patient stands upright, initially there is even more tension in the outer annular fibers as the external load is increased, but gradually the fluid is expressed from the disc with a reduction in its volume, until a new equilibrium is reached. When the patient then lies down for examination of straight leg raising, a reduction in the external load produces a relatively flaccid protrusion. Straight leg raising, if it is related to the tension of the protrusion, will then be improved. This cycle requires a protrusion which has intact outer annular fibers. There are, however, alternative explanations for changes in straight leg raising. Changes in disc height will increase tension in a root tethered by

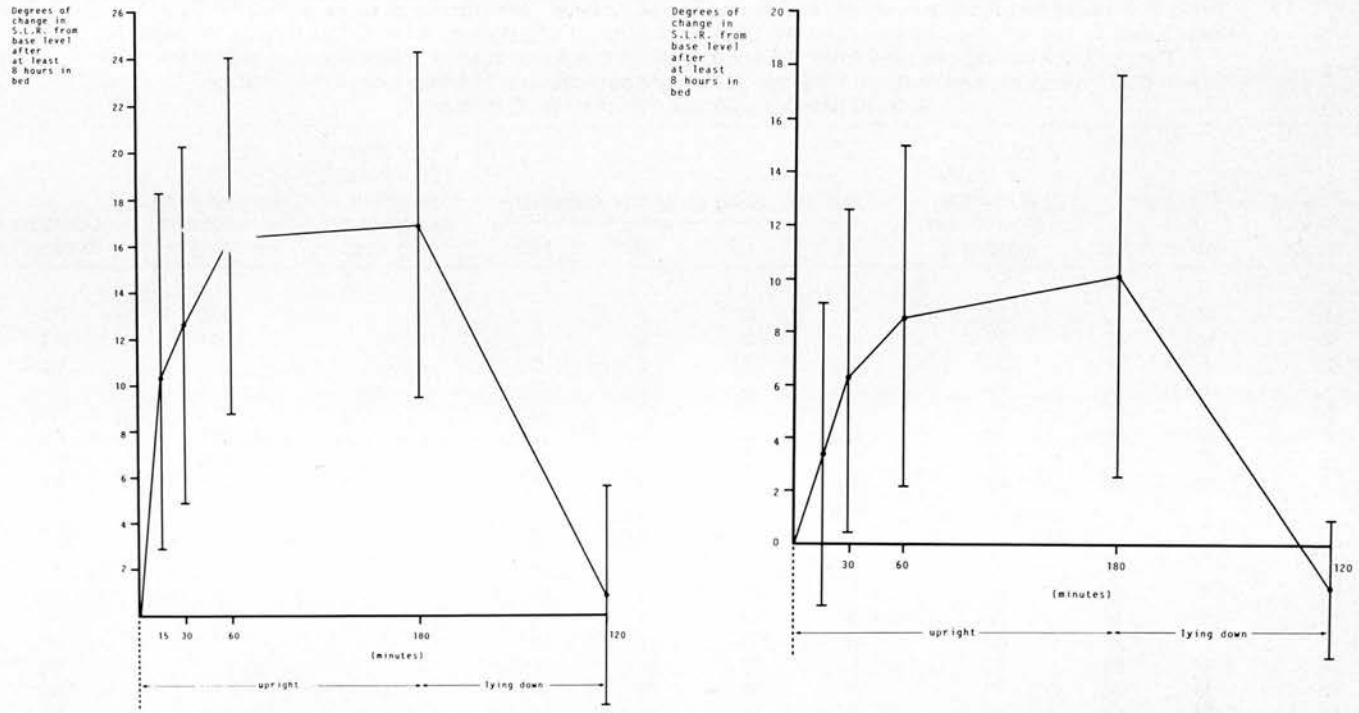

Fig 4. The mean change in straight leg raising (SLR) and standard deviation (in degrees) after 180 minutes in the upright position following recumbancy, and then after 120 minutes recumbancy, for 20 patients who had 10° or more of change, **A**, for the symptomatic (most affected) leg, and **B**, for the asymptomatic (least affected) leg.

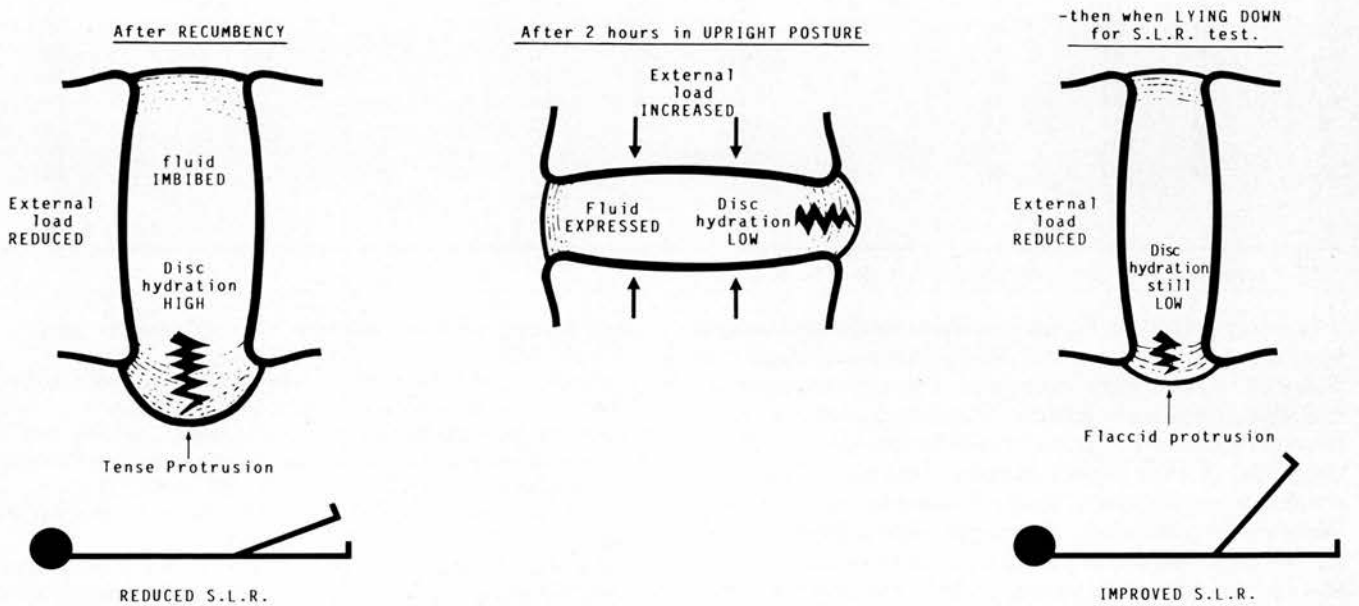

Fig 5. Diagram to show that after a period of recumbancy, increased hydration will cause a disc protrusion to bulge. Straight leg raising (SLR) will then be reduced. After 2 hours in the upright posture, the increased external load causes fluid to be expressed, and when lying down for the SLR test, the protrusion will then be flaccid, with improvement in the SLR.

dural ligaments,[7] and diurnal changes in lumbar lordosis will also affect the root. These factors may act in combination.

If the size of a protrusion changes, then imaging a disc also may be affected by diurnal changes, perhaps explaining some negative radiculograms in patients with surgically proven discs. It should now be possible to examine diurnal disc changes by Magnetic Resonance Imaging.

Certain clinical phenomena may be explained by diurnal and postural changes. Trunk list is a sign that often accompanies a disc protrusion. It is worse in a morning, and disappears quickly or slowly when the patient lies down.[6] It is probably related to the tension of the outer annulus. Similarly diurnal changes might explain why many patients with a disc protrusion need help with their socks or stockings in a morning, but have little difficulty later in the day.

Table 2. Straight Leg Raising in the Most Affected Leg in Patients With Criteria of Lumbar Disc Protrusion, After at Least 8 Hours of Recumbancy, and the Change in Straight Leg Raising After 15, 30, 60 and 180 Minutes in the Upright Posture, and then After 120 Minutes of Further Recumbancy; Myelographic Level of the Disc Protrusion, and Surgical Findings. **A,** In eight patients who had less than 10° of change. **B,** In 20 patients who had 10° or more of change.

Patient number	Sex	Age	SLR after at least 8 hours of recumbancy (degrees)	SLR after being upright for (minutes)				SLR after 120 minutes of further recumbancy (degrees)	Myelographic level of disc protrusion	Operative findings*
				15	30	60	180			
A										
1.	M		20	0	0	2	0	—	4/5	AT
2.	M	42	53	1	2	0	0	—	5/1	AT
3.	F	51	34	6	8	6	6	—		No
4.	F	43	75	0	0	−2	−2	—	5/1	No
5.	M	40	30	6	8	7	0	—		No
6.	M	26	20	0	0	0	0	—	5/1	AT
7.	M	43	36	4	6	4	6	—	4/5	P
8.	M	23	40	6	6	4	4	—	4/5	AT
			Mean — 38.5	2.87	3.75	2.62	1.75			
			SD — 17.05	2.11	3.38	2.95	2.90			
B										
9.	F	35	58	7	12	12	12	—		No
10.	M	47	70	10	10	8	8	−10		No
11.	F	27	62	8	8	18	18	3		No
12.	M	28	58	8	12	10	12	—		No
13.	M	36	30	39	30	30	30	10		No
14.	M	53	50	8	14	12	14	—		No
15.	M	42	67	3	−1	4	10	−2		No
16.	M	28	38	12	14	12	12	—	5/1	P
17.	F	43	20	20	26	24	20	—	4/5	No
18.	M	33	50	12	14	15	20	0	4/5	No
19.	M	38	60	8	10	10	10	0		No
20.	M	42	56	10	14	18	18	4		No
21.	F	29	42	6	8	13	13	—	4/5	No
22.	M	39	40	20	30	38	38	0	4/5	No
23.	F	41	40	10	10	20	20	0	4/5	No
24.	M	43	60	8	5	10	10	0		No
25.	M	24	26	4	9	14	14	—		No
26.	M	37	58	8	10	23	23	—		No
27.	M	31	60	2	4	20	20	6		No
28.	M	37	60	8	12	16	16	0		No
			Mean — 50.3	10.5	12.5	16.3	16.9	0.9		
			SD — 13.7	7.9	7.7	7.8	7.1	4.6		

*P = protrusion; AT = complete annular tear; No = no surgery.

There may be diagnostic importance in the diurnal changes in straight leg raising, disc protrusion at L5/S1 showing less diurnal change than those at L4/5 because of the relatively reduced proteoglycan content and low hydration of the lumbosacral disc.[9] Perhaps of greater importance is the poor prognosis of the eight patients who had less than 10° of diurnal change, five of whom required discectomy, and four of these had a complete annular tear. Diurnal changes in straight leg raising probably require an intact outer annulus and are inhibited by a complete annular tear. We suggest that a recumbancy test, comparing straight leg raising after a period in contrasting postures, might be a useful supplement to the usual single measurement.

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Address reprint requests to

R. W. Porter, MD, FRCS, FRCSE
Consultant Orthopaedic Surgeon
Doncaster Health Authority
Doncaster Royal Infirmary
Armthorpe Road
Doncaster DN2 5LT
England

Accepted for publication October 9, 1989.

International Orthopaedics (SICOT) (1992) 16: 152–156

International
Orthopaedics

Failed lumbar spinal surgery

S. Dhar and R. W. Porter

The Department of Orthopaedic Surgery, Doncaster Royal Infirmary, England

Summary. *Failures and poor results of 160 patients after lumbar spinal surgery between 1980 and 1984 were analysed retrospectively. A self-rated questionnaire carried out 12 months after operation revealed 20 poor results; these occurred most commonly after multiple operations, decompression and fusion as compared to disc excision. The commonest cause was failure to recognise abnormal pain behaviour before operation. A more careful preoperative assessment should reduce the incidence of failure.*

Résumé. *L'étude rétrospective de 160 opérés du rachis lombaire, de 1980 à 1984, à l'hôpital général de district, a permis d'analyser les échecs et les mauvais résultats. Grâce à un questionnaire adressé à ces malades un an aprés l'intervention on a pu retrouver 20 mauvais résultats. Ces vingt cas ont été réexaminés afin de déterminer la cause de ces échecs. Il y avait davantage de mauvais résultats aprés chirurgie itérative, aprés décompression et arthrodèse qu'aprés simple discectomie. La cause habituelle de ces échecs était une mésestimation, lors de l'examen pré-opératoire, d'une réaction anormale à la douleur. Il semble qu'un examen plus attentif des patients lombalgiques puisse diminuer le nombre des échecs du traitement chirurgical.*

Every reported series of lumbar spinal surgery for low back pain and sciatica contains a hard core of patients with poor results. This group are severely disabled and difficult to manage [3, 19]. Beside the morbidity, the immense socioeconomic implications are self-evident.

The present review analyses the patients' subjective results one year after operation. An attempt is made to identify the patients with poor results and the reasons for failure, and to propose suitable management for them.

Patients and methods

We reviewed the medical records and radiographs of patients who had spinal operations between January 1980 and December 1984 at our hospital. This included a self-rated questionnaire, completed by each patient twelve months after operation, which recorded the degree of their recovery of normal function at home, at work and in recreation, the presence or absence of back or leg pain, their current drug therapy, and their satisfaction with the result of the operation. Each patient was then classified as excellent, good, fair or poor (Table 1).

All the operations were carried out by the senior author (RWP). The results of disc excision, decompression for central or lateral canal stenosis, fusion for spinal instability, and multiple spinal operations were compared. Those with poor results were then reviewed in an attempt to identify the cause of failure. This review was conducted in three parts with a survey of the clinical course before operation; an assessment of the operative procedure, and an analysis of the postoperative course. The original diagnosis was reconsidered, and the adequacy of the surgery, with the possible failure to deal with the pathological lesion, was taken into account.

Evidence of abnormal pain behaviour [27] was sought with particular attention to inappropriate signs (Fig. 1) and any psychological disturbance was assessed by the Minnesota multiphasic personality inventory (MMPI). Specific questions were asked about the financial implications of the disability.

When necessary, further investigations were carried out including radiography, CT scanning, radiculography and electrodiagnostic tests, such as fibrillation potentials and ankle reflex latencies [11].

Reprint requests to: S. Dhar, University Department of Orthopaedic and Accident Surgery, Royal Liverpool Hospital, Prescot Street, PO Box 147, Liverpool L69 3BX, UK

Table 1. Criteria for assessment of results

Result	Criteria
Excellent	No pain, no functional limitation
Good	Intermittent pain in the back or leg, no functional limitation at work, home or in recreation, no analgesics
Fair	Intermittent pain in back or legs, and either affecting function at work, home or in recreation, or use of analgesics
Poor	Constant pain in back or legs, or daily analgesics, or would not have had the operation in retrospect

Results

One hundred and sixty patients who had spinal operations completed the questionnaire; 26 had been lost to follow up. The results are shown in Table 2. Most operations were excision of discs and there were fewer poor results in this group than after other procedures. Repeated operations were the most disappointing.

Twenty patients with poor results were reviewed and the causes of failure are suggested in Table 3. They all complained of low back pain, 18 had more than 50% limitation of lumbar movement, and one had signs of nerve root irritation or tension.

Two patients had a persistent motor neurological had deficit which was slight in one; both had had further operations.

Three patients, who were treated by decompression for spinal stenosis, had persistent stenosis and would have benefited from a more extensive decompression initially (Fig. 2).

Two others were thought to have symptoms due to a central canal stenosis; radiculography showed dural encroachment and a further decompression was carried out in spite of signs of abnormal pain behaviour.

One patient benefited temporarily from an anterior fusion, but a subsequent posterior fusion, although sound, failed to relieve her pain. Another was thought to have an unstable segment, but continued to have pain after a sound fusion. A pseudarthrosis followed one attempted fusion.

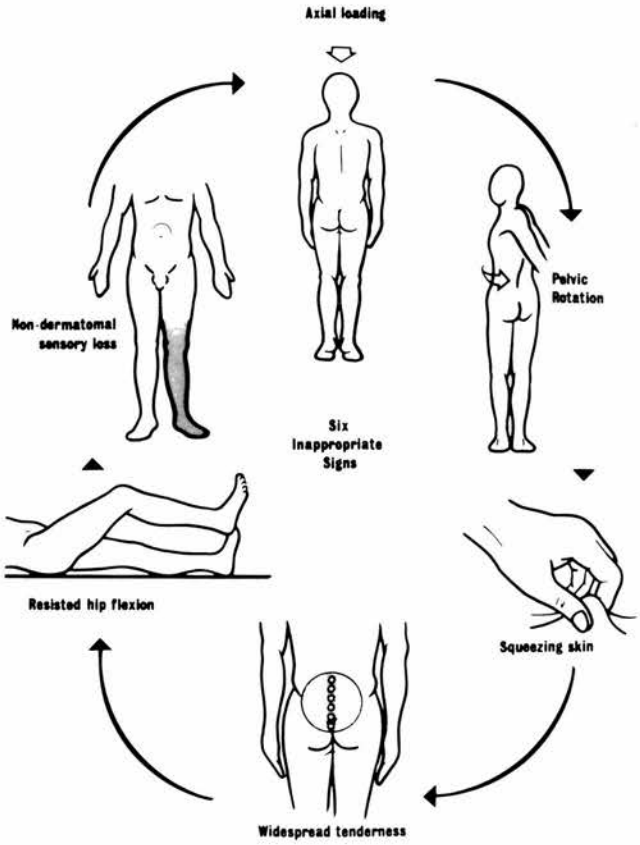


Fig. 1. The six inappropriate signs

Two had signs of irreversible root damage on electrical testing, in spite of an adequate decompression on a second occasion.

Four had evidence of epidural fibrosis, but in only one was it considered to be severe enough to be responsible for symptoms.

Two patients would have lost financially if their operation had been successful.

Thirteen of the 20 patients showed a significant psychological disturbance as judged by the symptoms, clinical examination, and investigations, in-

Table 2. Comparison of results of each operative category

Operations	Number	Result (%)			
		Excellent	Good	Fair	Poor
Discectomy	86	25 (29)	44 (51)	14 (16)	3 (4)
Decompression	36	4 (11)	7 (19)	18 (50)	7 (20)
Fusion	16	2 (12)	8 (50)	3 (19)	3 (19)
Multiple spinal Surgery	22	1 (5)	4 (18)	10 (45)	7 (32)

Table 3. Causes of failure in 20 patients

No.	Operation	Age (years)	Iatrogenic causes			IRD	APB	Psych. causes	Financial implications
			EF	ID	PSEUD				
1.	Discectomy	32	–	–	–	–	+	–	–
2.	Discectomy	27	–	–	–	+–	+	–	–
3.	Discectomy	43	+	–	–	–	+–	–	–
4.	Decompression	59	–	+	–	–	–	–	–
5.	Decompression	62	–	+	–	–	+–	–	–
6.	Decompression	53	–	+	–	–	–	–	–
7.	Decompression	60	–	–	–	–	+–	–	+
8.	Decompression	49	+–	–	–	–	+–	+	–
9.	Decompression	58	–	–	–	–	+–	+	–
10.	Decompression	69	–	–	–	–	+	–	–
11.	Fusion	52	–	–	+	–	–	–	–
12.	Fusion	41	–	–	–	–	+	–	–
13.	Fusion	45	–	–	–	–	+–	+	–
14.	Multiple operations	59	+–	–	–	+	–	–	–
15.	Multiple operations	60	–	–	–	+	–	–	–
16.	Multiple operations	43	–	–	–	–	+–	–	+
17.	Multiple operations	52	–	–	–	–	+–	+	–
18.	Multiple operations	45	–	–	–	–	+	–	–
19.	Multiple operations	40	–	–	–	–	+	–	–
20.	Multiple operations	51	+–	–	–	–	+	–	–

EF: epidural fibrosis, ID: inadequate decompression, Pseud: pseudarthrosis, IRD: irreversible root damage, APB: abnormal pain behaviour, Psych.: psychiatric, +: present, –: absent, +–: maybe present

cluding MMPI. They suffered from depression, anxiety, hypochondriasis and conversion manifestations. As this was a retrospective study, it was not possible to determine whether or not these symptoms were the cause or the effect of the poor outcome of the operations.

Discussion

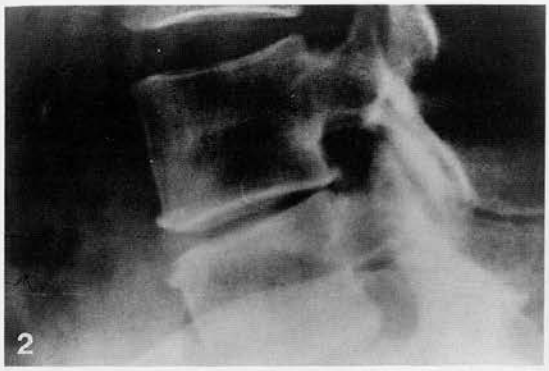
The incidence of failure after lumbar spinal operations is still disappointingly high, in spite of sophisticated investigations and improved methods of assessment now available. Failure is easily recognised by the patient who is the best judge of the result. Short term results can give a false impression of success [17] and its incidence can be manipulated by the questionnaire [8]. It has been shown that limited information improves the reliability of the assessment [18]; our questionnaire was therefore simple and our criteria few. Failure at twelve months is unmistakable with continuous pain, the daily use of analgesics, or a patient, who with hindsight, says he would not have had the operation.

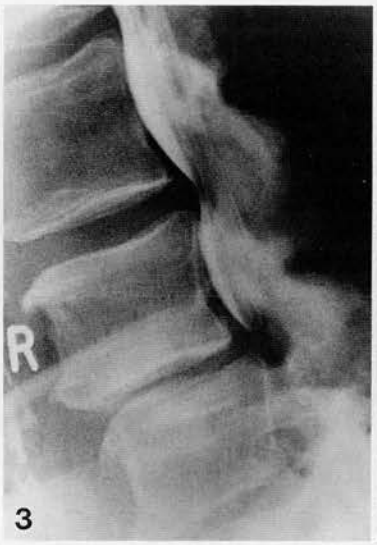
The results were less poor after disc excision, only 4% were unsatisfactory, because the indications for operation are now clearly defined [14, 29]. If pain is not relieved by adequate rest, and the level is confirmed by radiculography or a CT scan, an operation carried out to relieve root tension will be followed by rapid recovery [6].

One of our patients, who had a disc excision, was relieved of root pain, but continued with back pain referred to the leg from an unsuspected unstable segment. The two patients with irreversible root damage, confirmed by electrodiagnostic testing [11, 15], failed to respond to adequate decompression.

Unfortunately, the criteria for fusion and decompression are less exact. It is difficult to estimate how extensive a decompression needs to be from myelography and CT scans. The canal will have been narrowed for several years before symptoms develop, and although stenosis may be present at several levels, the lesion causing symptoms is probably localised (Fig. 3). It is essential to ensure that this segment is decompressed [22, 31], and it is tempting to rely on the impression during operation that the tight dura and roots are given adequate space. On the grounds of safety, it may be wise to be more radical than seems necessary [12], but better results have been reported after a one- or two-level decompression for localised segmental stenosis than for a three-, four- or five-level decompression for multisegmental disease [5]. Somato-sensory evoked potentials may help in assessing the correct level during operation [2].

The incidence of iatrogenic lesions was small, but may increase with a longer follow up. Extradural fibrosis was responsible for symptoms in one of the four patients in whom it occurred (Fig. 4), in spite of the dura being covered with a fat graft [10]. The





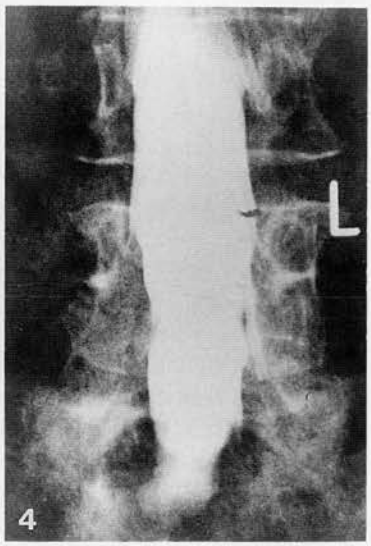


Fig. 2. Lateral radiograph showing disc degeneration with traction spurs and retrolisthesis. The double shadow of the posterior vertebral border suggests some rotational displacement. One root can be affected in the root canal, and another in the central canal at the cranial lip of the lamina and in the lateral recess; both need to be decompressed

Fig. 3. Lateral radiculogram of a 60 year old man with symptoms of neurogenic claudication. Degenerative spondylolisthesis occludes the metramazide column. Should L 3/4 also be decompressed?

Fig. 4. Radiculogram of a patient who still has root symptoms after decompression for spinal canal stenosis. There is epidural fibrosis, and the root sheaths on the right side particularly failed to fill

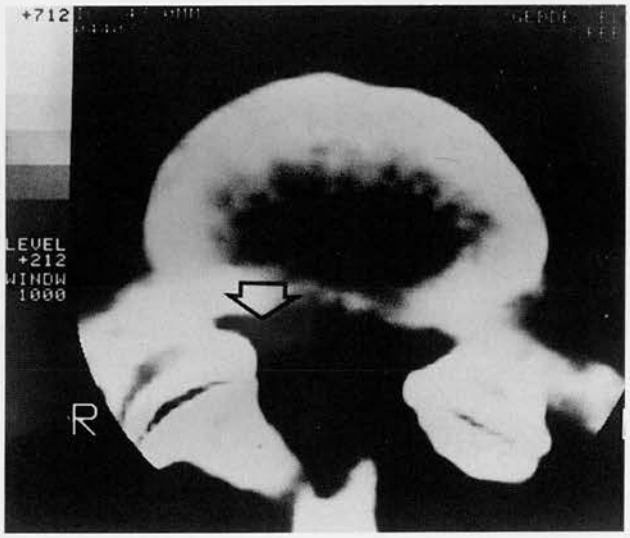


Fig. 5. A CT scan of a 34 year old women who had a left sided disc protrusion excised through a fenestration 2 years previously. Symptoms had been completely relieved until right sided root symptoms developed. She had no inappropriate signs. The scan showed a new right sided disc protrusion (*arrow*) which was excised with a good result

significance of the fibrosis is debatable as it was found in 75% of CT scans after operation [24].

The commonest cause of failure was not due to inadequate operation or iatrogenic complications, but to incorrect assessment of the patient. In seven failures, a lesion was found at operation which was thought to be causing symptoms, but abnormal pain behaviour had been ignored. Four of these patients had a depressive illness. A population with low back pain has a high proportion of individuals with psychological and psychotic disturbances [13] which may only become apparent when chronic invalidism has followed operation [16].

Litigation and a treatable condition can coexist, but a pending claim for compensation may have a negative effect on the result of operation [4, 26].

The problem is to interpret the significance of a lesion in a distressed patient; abnormal pain behaviour may mask a treatable condition, but the abnormality found by radiculography or CT scan may not be causing symptoms. Failure may follow when the decision to operate is influenced by the degree of distress, or by the presence of abnormal investigations, rather than by abnormal objective signs [28]. Thirteen of the twenty poor results had signs of abnormal pain behaviour twelve months after operation; this may be the effect of having to cope with a painful back, but most had the same problem before operation when a treatable lesion was thought to coexist.

The result of a second intervention after a failed lumbar spinal operation is unpredictable [3, 23]. Although four of our failures had possible physical causes, most failures had been re-operated on be-

cause of the presence of a lesion which was mistakenly thought to be responsible for their distress, and there were inappropriate signs (Fig. 1). A detailed psychological assessment should be carried out before all re-operations. There is no evidence that the MMPI low back scale will differentiate between those patients who will have a poor, fair or good outcome after spinal fusion [30], or from decompression [7], nor does it identify non-organic from organic low back pain [25], but an abnormal profile directs the surgeon's attention to factors other than the spine which may be responsible for low back pain. It does not necessarily mean that a second operation will fail, but the operation can only be justified if both patient and surgeon are aware of the risks (Fig. 5). There are possible benefits for a multidisciplinary approach before a second operation, involving a psychologist, anaesthetist, radiologist, general practitioner, spouse and surgeon [1]. Assessment will take time, but nevertheless it should be comprehensive as further failure will compound the patient's problems.

Management of patients in whom a spinal operation has failed is difficult. The most recalcitrant problem is the patient who has had several operations, but surgeons should not operate on pain as such. A further operation will only help if the original diagnosis was wrong or the surgery inadequate, or if there are iatrogenic complications. One of our patients with epidural fibrosis was referred to a pain clinic for epidural injections [9], but most of our failures showed abnormal illness behaviour for which invasive treatment would have been counterproductive. Four patients had psychiatric help and three entered a behaviour modification programme with moderate success [20, 21]. The remainder were told frankly, after full investigation, that further treatment was not likely to be effective and they were discharged to their general practitioner.

Failure will always be with us. We suggest that its incidence will be reduced, not by more sophisticated investigation of the patient's back, but by a more careful clinical and psychological assessment.

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International Journal of Industrial Ergonomics, 9 (1992) 157–160
Elsevier

Review article

Is hard work good for the back? The relationship between hard work and low back pain-related disorders

R.W. Porter

Dept. of Orthopaedics, University of Aberdeen, University Medical Buildings, Foresterhill, Aberdeen AB9 2ZD, UK

(Received July 4, 1990; accepted in revised form November 13, 1991)

Abstract

The high incidence of absenteeism from low back pain in heavy manual workers may be related more to the effect of low back pain than to its cause. This review examines the relationship between heavy work and both PID and degenerative low back pain disorders, concluding that hard work in adult life and early working life probably reduces the incidence of disc prolapse, but increases the risk of symptoms from degenerative disease. Hard work probably has physiological benefits and has a long-term role in rehabilitation.

Relevance to industry

Hard work is not necessarily harmful to the back. It can predispose to symptoms from degenerative disease, but there is evidence that physical activity strengthens the spine and protects the disc. Graduated exercise and work has a role in rehabilitation.

Keywords

Low back pain, prevention, rehabilitation.

The epidemiology of back pain has been studied intensively within and between industries. There is a high work-handicap from back pain in the iron and steel industry, mining, forestry, dock work, nursing and driving (Hult, 1954; Anderson et al., 1962; Blow and Jackson, 1971; Kelsey, 1975; Daniel et al., 1980; Andersson, 1981; Afacan, 1982; Burkart, 1983; Stubbs et al., 1983) which might suggest that hard work is not good for the spine.

It is possible however, that the high incidence of reported back pain in manual workers is related not so much to the heavy nature of the work causing back pain, but the inability to do hard work when the back is painful. A sedentary worker may continue at work with a painful back which would cause a heavy manual worker to take leave.

In addition, back pain is but a symptom of a number of different syndromes, and it is possible that there is a high incidence of heavy manual workers with one syndrome, and a low incidence with another.

Underground coal-mining is still demanding on the spine (Dales et al., 1986), and in order to assess the relative importance on heavy manual work in various back pain syndromes, we compared the prevalence of coal miners and non-miners attending hospital with back pain and with three defined sciatic syndromes (Porter, 1987). Men attending a back pain clinic over a three-year

period were asked if they had been underground coal-miners from leaving school, or had worked underground for at least 5 years. The percentage of these miners fulfilling criteria for three defined sciatic syndromes was then recorded.

(1) *Lower Lumbar Disc Protrusion (PID)*. Three or more of the criteria described by McCulloch (1977); unilateral leg pain in a typical sciatic root distribution below the knee; specific neurological symptoms incriminating a single nerve; limited straight leg raising, at least two neurological changes of muscle wasting, muscle weakness, sensory change or hyporeflexia; and radiculographic evidence of disc protrusion.

(2) *Root Entrapment syndrome (RE) – Lateral Canal Stenosis*. Pain in the leg below the knee incriminating a single root, severe and constant, unrelieved by rest, spinal extension less than third of normal range, straight leg raising better than 70 degrees, over 40 years of age, and radiological evidence of degenerative change (Getty et al., 1981; Porter et al., 1984).

(3) *Neurogenic Claudication (NC)*. Discomfort in both legs above and below the knees when walking with less than 500 metres tolerance, relieved by rest, and a positive myelogram or CT scan showing central canal stenosis (Verbiest, 1954; Ehni, 1969; Blau and Logue, 1978).

More miners than non-miners attended hospital with back pain than would be expected (2.78% of the miners compared with 1.99% of the non-miners, $p < 0.001$, table 1). Significantly less miners attended with criteria of lumbar disc protrusion (0.32% compared with 0.40% $p < 0.001$), but significantly more had criteria of root entrapment syndrome from lateral canal stenosis (0.58% compared with 0.31%, $p < 0.001$), and also significantly more had symptoms of neurogenic claudication (0.22% compared with 0.04%, $p < 0.001$). Thus there were more coal miners with syndromes related to degenerative change, but less than expected with disc protrusion. We also observed that over the three-year period, 0.6% of the non-miners required discs excision, compared with only 0.02% of the miners. Coal mining seemed to protect a subject from the need for disc surgery.

We conducted a second study assessing the early work experience of patients with disc protrusion, and those with root entrapment from lateral canal stenosis, using an assistant administered occupational questionnaire. 196 patients with criteria of symptomatic disc protrusion, and 53 with root entrapment syndrome were asked about heavy physical work between fifteen and twenty years of age. Significantly more subjects with disc protrusion had done no heavy physical work in those early years, when compared with patients with root entrapment syndrome (39.7% compared with 22.6% $p < 0.001$). Significantly more with root entrapment syndrome had done five full years of heavy work from fifteen to twenty years of age when compared with those with disc protrusion (65.6% compared with 30.1% $p < 0.001$).

Kelsey (1975) showed that there was a greater than expected incidence of disc protrusion in sedentary workers. Our studies support this and also support an association with sedentary activity in early working life.

The relationship between physical activity and the strength of the spine is still poorly understood. Vertebrae probably strengthen by heavy labour, since weight lifters' vertebrae have a high bone mineral content (Granhed and Hansson, 1986), and this is associated with a high compressive strength (Hansson et al., 1980). Spinal liga-

Table 1

Number of miners and non-miners in the population, attending the back pain clinic, with three defined back pain syndromes and 'other causes', expressed as a percentage, of miners and non-miners in the population. (PID: Prolapsed Intervertebral Disc, RE: Root Entrapment, NC: Neurogenic Claudication).

	Workers in the population	Attended back clinic in a three-year period	PID	RE	NC	Other
Miners	16,000	445 (2.78%)	52 (0.32%)	93 (0.58%)	35 (0.22%)	265 (1.65%)
Non-miners	49,100	977 (1.99%)	198 (0.40%)	152 (0.31%)	21 (0.04%)	606 (1.23%)
		$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$	n.s.

ments, important in their protective role for the spine, are dynamic structures by virtue of their attachments to the lumbar fascia (Bogduk and McIntosh, 1984; Tesh et al., 1987), and their strength may be related to the strength of the abdominal and spinal muscles; they may be strengthened by activities that flex and extend the spine through the full physiological range. There is, however, little information about the intervertebral discs. It might be expected that their compressive strength will match that of the adjacent vertebrae, and that their resistance to bending will keep pace with ligament strength, but this is not certain.

We investigated the effect of physical activity on spinal strength by examining cadaveric spines of young men killed in road accidents, and compared the mechanical properties with the men's occupational and recreational histories (Porter et al., 1989). The compressive strength of nine spines tended to increase with the level of activity in life, physical activity strengthening both the vertebrae and the discs ($p < 0.05$).

It is possible that the strong annulus of a manual worker resists forces that would disrupt the weaker disc of a sedentary worker. Strong musculo-ligamentous structures and an efficient reflex mechanism may also be important.

The concept that heavy work is related to spinal pathology has influenced therapy. A generation of physicians have recommended rest and sedentary activity for patients with back pain. They have been asked to note and avoid activity that produces pain, which becomes more anticipated pain and suffering than the actual experience of pain (Fordyce et al., 1982; Linton, 1985). It has been suggested however, that avoidance behaviour is as unproductive in reducing pain as it is in reducing fear (Phillips and Hunter, 1981). Not only does passive avoidance lead to psychological reinforcement of the invalid status, but it also has adverse physical effects. Joints become stiff, soft tissues weak, and protective reflexes are impaired. An acute episode of back pain that should resolve within a short period, may persist as chronic disability, with psychological disturbance and physical disuse phenomena.

The concept of unnecessary rest and avoidance of physical stress has been challenged (Nachemson, 1983). Hard work and functional rehabilitation can in fact be therapeutic for the patient with backache. Functional rehabilitation aims to improve physical fitness and influence low pain by improving disc nutrition, assist healing of connective tissues, and increase endorphines. There is a correlation between fitness and recovery from back pain (Cady et al., 1979; Mellin, 1986). Patients who have been off work for more than eight weeks with an acute episode of back pain and who are treated with a vigorous exercise regime, not only return to work more quickly than an untreated control population, but they are also significantly better than the control group at one year (Lindstrom et al., 1989). Functional rehabilitation is also effective for patients with chronic back pain when surgery is not suitable and traditional therapies have failed (Mayer et al., 1987; Hazars et al., 1989). For the patient with abnormal pain behaviour, when many inappropriate signs are an exhibition of distress, functional rehabilitation can result in a dramatic conversion to 'wellness behaviour' (Porter et al., 1989).

Is hard work good for the spine? A high rate of reported back pain in heavy manual occupations belies the fact that physical stress to the spine has selective value. Hard work is associated with a strong annulus, and seems to protect from symptomtic disc protrusion. This bonus is balanced, however, by increased risk of syndromes related to degenerative change in later decades (root entrapment syndrome from lateral stenosis and neurogenic claudication). As a therapeutic regime, graduated return to fitness with a programme of functional rehabilitation can be helpful both after the acute episode and also in the presence of chronic disability, and a sympathetic employer can undoubtedly ease a graduated return to full activity. Hard work can be good for you.

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Reprinted from SPINE, Vol. 17•No. 9, September, 1992

Briefly Noted

■ Effect of Straight Leg Raising on Blood Pressure

A. H. M. A. Ismaiel, FRCS, MSc Orth, and
R. W. Porter MD, FRCS, FRCSE

Hypertension has been reported as a complication of bone lengthening procedures in children,[2,7,9—11] after correction of talipes equinovarus,[1,8] and with traction treatment for congenital hip dislocation, femoral fractures,[3,5] and flexion deformities in the knee joint.[4,7] It has been attributed to stretching of peripheral nerves. We have examined the effect of sustained straight leg raising (SLR) on the blood pressure and pulse rate in adult patients who have low back pain and root tension signs to determine if there are also blood pressure changes associated with these signs.

Method

Ten patients admitted for treatment of disc protrusion entered the study. They all had restriction of SLR recorded by goniometer.[6] After at least 1 day of bed rest, and at a similar time of the day,[6] the same examiner obtained the baseline blood pressure and pulse recordings in the supine position, using the Dynamap machine (Johnson & Johnson Co., FL) until a minimum of three blood pressure recordings were within 5 mmHg. Similar measurements were then taken with the patient lying on the side until there was a stable hemodynamic state. Horizontal SLR was conducted on the uppermost leg to the maximum permitted level for at least one minute. The least affected leg was examined first, recording the blood pressure and pulse rate. Similar measurements were then recorded with the patient lying on the opposite side. Statistical analysis was by a two-tailed paired sample Student t test.

Results

Ten patients were examined, 6 male and 4 female. The range of resting systolic pressure was from 114 to 143 mmHg and diastolic blood pressure of 60 to 91 mmHg. There was no significant difference in blood pressure or pulse rate between the supine and right and left positions.

After maintaining full SLR of the most symptomatic leg, the systolic blood pressure remained the same in one patient, it increased by between 7 and 17 mmHg in 3 patients and it decreased by between 2 and 20 mmHg in 6 patients (Table 1). The results were similar when carrying out SLR on the least affected leg. The pulse rate showed similar changes. In one patient only there was marked rise of all readings. This patient was originally mildly hypertensive and anxiety could not be eliminated during performance of his examination.

There was a small but significant rise in the pulse rate with SLR of the most affected leg of ($+5.3$ minutes $\pm$ 6.4 minutes, 9 degrees of freedom). There was no significant rise in systolic or diastolic pressure in either leg with SLR. There was a fall in systolic pressure with SLR of the least affected leg (-4.5 mmHg, $P < 0.05$). The tendency for blood pressure to drop rather than rise is contrary to the effect of stretch of nerves in children.

Discussion and Conclusion

Several studies suggest that a rise in blood pressure accompanies stretching of peripheral nerves in children. If a similar rise in blood pressure accompanied sustained SLR in patients with disc protrusion, this might provide more information about the pathophysiologic significance of this sign.

The test was carried out after a period of bed rest to ensure a stable baseline. Then in order to eliminate any hemodynamic effect of increasing the venous return by elevating the leg, the SLR test was carried out with the patients lying on their side. However, no consistent rise in blood pressure was observed when fairly forceful SLR was maintained. The significant rise in pulse rate with SLR of the most affected leg, may have been related either to anxiety or to the vascular changes of sustained hamstring spasm.

It is possible that stretching of the peripheral nerves is associated with a rise in blood pressure only in children. Alternatively it may be necessary to maintain maximum straight leg raising for a longer period or at a higher level to produce blood pressure changes, but even with quite forceful SLR and maintaining it for a longer period, we were not able to produce a consistent rise in blood pressure. A further hypothesis may be that in patients with the so called root tension signs, SLR is not in fact limited because the roots of the sciatic nerve are under

From the Department of Orthopaedic Surgery, Aberdeen, Scotland, UK.
Accepted for publication April 13, 1992.

Table 1. Blood Pressure and Pulse Measurements at Rest and on SLR in Patients with Low Back Pain and Root Tension Signs

Resting Pulse and Blood Pressure lying on the Side Before Examining SLR				Changes in Pulse & Blood Pressure After 1 Minute of SLR of the Least Affected Leg				Changes in Pulse and Blood Pressure After 1 Minute of SLR of the Most Affected Leg			
Pulse Rate	Systotic Blood Pressure	Diastotic Blood Pressure	Mean Blood Pressure	Pulse Rate	Systolic Blood Pressure	Diastolic Blood Pressure	Mean Blood Pressure	Pulse Rate	Systolic Blood Pressure	Diastolic Blood Pressure	Mean Blood Pressure
68	120	60	80	−5	−1	3	3	−6	−9	−9	−9
58	121	69	85	10	−3	5	5	0	7	−9	−1
81	116	76	88	0	2	11	2	4	−11	−3	−3
63	130	74	94	6	11	1	−3	12	17	31	33
61	133	81	101	5	3	7	7	8	−2	−2	−2
57	143	91	115	1	−5	−9	−9	0	−3	13	5
74	114	66	80	−8	−7	3	16	14	−20	2	−4
88	132	88	95	−5	−2	−14	17	8	12	−24	−7
81	126	74	90	4	−18	−2	−10	2	0	0	0
73	123	79	95	12	−3	−3	−3	11	−15	−7	−11
Mean				2	−4.5*	0.2	2.5	5.3*	−2.4	−0.8	0.1
SD				6.6	6.3	7.5	9.2	6.4	11.9	14.6	12.4
t value				0.95	−2.27	0.08	0.86	2.64	−0.64	−0.64	0.03

*$P < 0.05$.

tension, but limited SLR may be a protective reflex to avoid that root tension. A significant rise in pulse rate during SLR of the most affected leg may be related either to pain, or to the involuntary muscle activity preventing full SLR.

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Address reprint requests to

R. W. Porter, MD, FRCS, FRCSE
Department of Orthopaedic Surgery
Polwarth Building, Foresterhill
Aberdeen AB9 2Zd
Scotland

Aberdeen University Review, no. 187, Spring 1992

UPRIGHT MAN

Inaugural Lecture delivered on 23 April 1991

By Richard W. Porter

I HAVE selected this title, partly because of the derivation of the term Orthopaedics, a word first coined by Nicholas Andry in 1741 from two Greek words, the first meaning straight, upright, free from deformity, and the second meaning child—upright child. The elder Sayre believed the second word was to be translated educate or teach, to educate about being upright. Orthopaedic surgeons have been true to their tradition, even though the practice and teaching of orthopaedics has changed with the years.

In 1936 Alexander Mitchell was given 36 beds to start the specialty of orthopaedics in this city. He was succeeded a decade later by Alec Rennie, and by 1965 he had four colleagues. In 1971 Alec Rennie was given a personal chair. Today there is a team of nine orthopaedic surgeons. One common denominator in the practice and teaching of orthopaedics, has been an interest in maintaining good function in the upright posture.

My second reason for selecting this title is because Action Research has most generously sponsored this chair. Historically known as Action Research for the Crippled Child, they deserve much credit for many useful advances in the prevention and correction of deformity. This chair also bears the name of their past President Sir Harry Platt, that distinguished orthopaedic centenarian, and one time president of the British Orthopaedic Association and of the English Royal College of Surgeons. On behalf of the orthopaedic department, Professor Eremin who worked so hard to establish this chair, and of the University, I would like to express our thanks to Action Research for making this chair possible.

So to my title, *'Upright man'*.

The one physical attribute that makes man unique amongst his vertebrate cousins, is his upright stature. Other primates may enjoy the canopy of the tropical forest, sometimes climbing, sometime semi-upright or quadrupedal, but man alone is comfortable and confident with his upright bipedal stance.

We could examine the energy saving characteristics of the hip or the knee in the upright posture, or the dynamic activity of the arch of the human foot, but I prefer to focus on the lumbar spine and examine how its form matches its function. The lumbar spine has three complementary roles, to be strong and supportive, to protect the nerves within the vertebral canal, and to be flexible for useful function in a variety of postures. I would like to examine to what degree these three roles are compatible with each other and with the demands of the upright posture.

FIRST, THE STRENGTH OF THE SPINE.

1. Each vertebra has to carry the load of the segments above, and it follows that there is an increasing load carried by each succeeding vertebra. The load at the base of the spine is considerably greater than that above. How does the spine accommodate these increasing loads?

I would like to demonstrate the load bearing characteristics of the spine, by using some models. When I suggested to the Principal that I hoped to use some models he was somewhat concerned until he learnt that they were to be constructed in the Department of Biophysics. I thank Eddie Stephenson for making them.

The first model is a tower construction of five cubes of equal size but different density. To provide a stable system we place the cube of greatest density at the base, and cubes of lesser density above (Fig. 1). This seems a reasonable load bearing system for the spine, with the vertebrae at the base of the spine being of relative greater density that those above. Bone certainly has the capacity to modify its density to the stresses of load bearing.[1] In Aberdeen, and elsewhere studies have shown that women who are obese, or have been obese, have greater total bone density that other women of similar age and stature. Conversely, disuse produces osteoporosis; loss of load bearing produces loss of bone density.

However, Paul Brinkmann in Munster has examined the density of human vertebrae and noted that the density in the lower spine is the same as in the middle and upper spine.[2] Its mass per unit volume does not change throughout the spine, so although the spine has the capacity to change its density in response to load, it does not do so.

A second model is a tower construction, this time with material of the same density, but with blocks of different size. To provide a stable system, the largest block is placed at the base, building upwards with blocks of decreasing size (Fig. 2). This is the system adopted by the spine. Brinkmann observed that although the bone of the spine did not vary in density, it matched the increasing load with an increasing cross sectional area.

In the human skeleton, the lumbar vertebrae are considerably larger than those in the neck. Chimpanzees which are semi-upright have less difference in size (Fig. 3), whilst quadrupedal sheep have neck vertebrae equally as large as those in the lumbar region. If human vertebrae had accommodated load bearing by increasing the density, this would have been compatible with a recent change from quadrupedal to an upright stance, but this is not what we observe. The relative increase in load bearing at the base of the spine is matched by an increase in size, and this is also pre-formed. Before the infant stands on his feet, the spine has large lower lumbar vertebrae, ready for the expected upright posture.

2. The internal structure of the vertebrae also has special qualities for carrying load. This can be demonstrated by another model (Fig. 4). A piece of card represents a sheet of compact bone. When placed across two supports, and trying to support a weight, the card readily

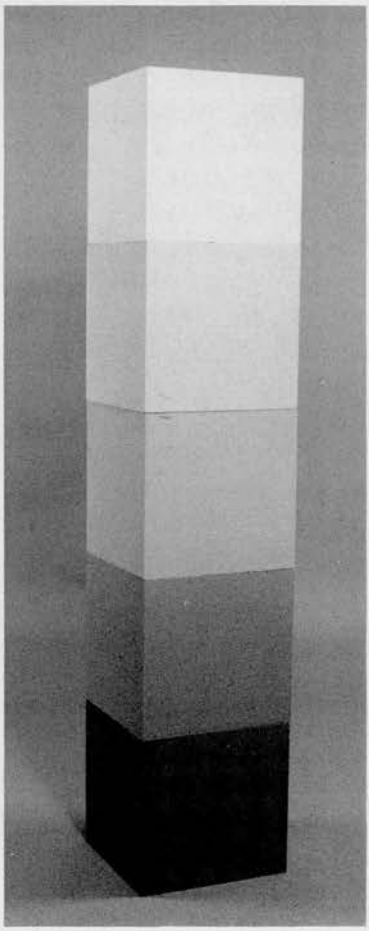


Fig. 1 A model of a potentially stable tower system of five cubes of equal size. The cubes of greatest density are at the base, with those of lesser density above.

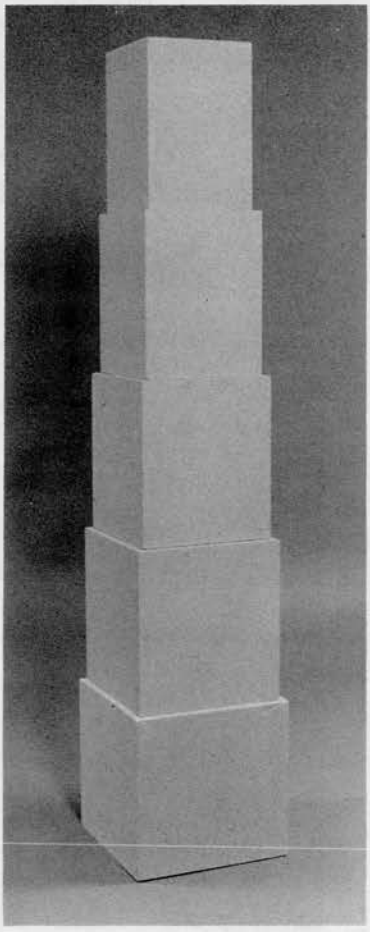


Fig. 2 A model with an alternative design, showing a potentially stable tower system of five cubes each of the same density, but this time of different volume. The larger cubes are at the base, with the greater load supported by an increase in the cross sectional area.

Fig. 3 The chimpanzee is semi-upright. The lower lumbar vertebrae are relatively larger than the more proximal vertebrae, but the difference is not so marked as in man. Note the absence of lumbar lordosis, which is a feature of man's upright stance, and the close proximity between the lower ribs and the rim of the pelvis.

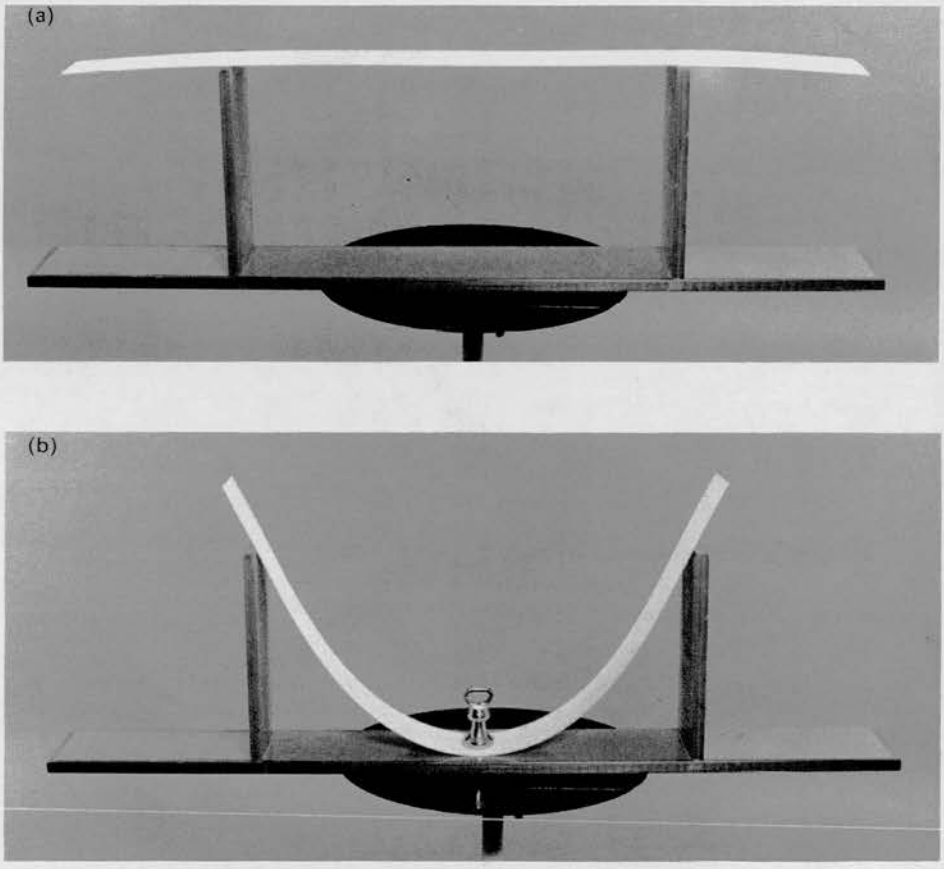
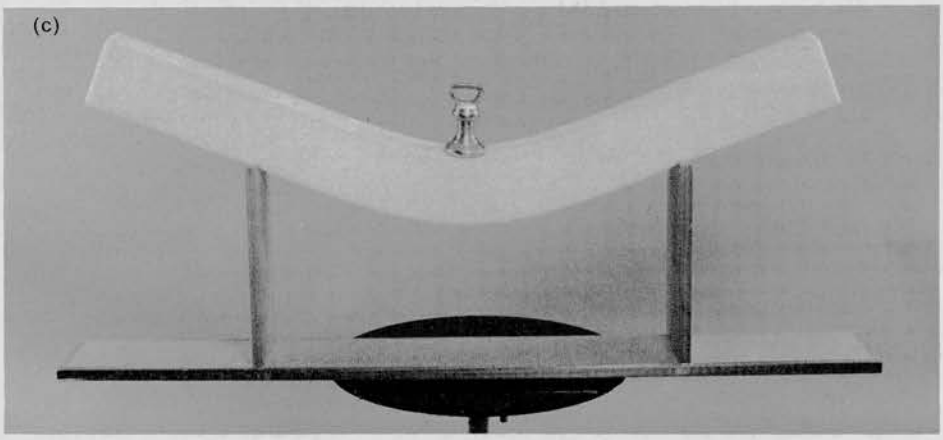


Fig. 4 a) A sheet of thin card on two supports. b) This easily deforms with a small load. c) A block of foam rubber also deforms with the same load. d) A sandwich of the same foam rubber between two sheets of card hardly deforms with a greater load. Many bones have this structure, combining strength with lightness of weight.



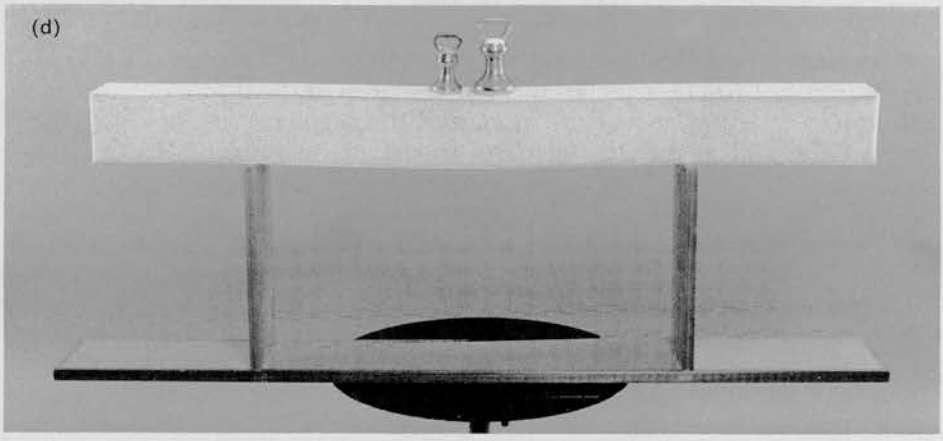


Fig. 4—*continued.*

deforms. Even two pieces fare little better. If we examine a thick sheet of sponge, which could represent spongy bone, it is also easily deformed by a similar weight. However, if we apply the same test to a composite sandwich of these materials—a sponge between two sheets of card—there is hardly any deformation at all. In the construction industry this is called 'foam filling'. It is light in weight, and remarkably strong. Many bones adopt this design, but it is particularly effective for the vertebral bodies in the lower spine, which have to carry considerable load. They are made of spongy cancellous bone sandwiched between two plates of thin cortical bone, which combines lightness of weight with strength.

3. One of the characteristics of the upright posture is the hollow at the base of the spine, the lumbar lordosis. The chimpanzee has one long kyphotic curve from the base of the neck to the lower back, but upright man has a secondary curve. It develops when the infant stands on his feet, pushing the lumbar spine forwards into the abdominal cavity. Richard Aspden, Research Fellow in the Department of Orthopaedics in Aberdeen, has been the first to recognise the biomechanical significance of this curve.[3] It accounts for the considerable strength of the human spine, enabling man to lift loads in excess of loads lifted by a gorilla, when compared with his body mass.

An arch is remarkably strong in its load bearing characteristics (Fig. 5). It matters not whether the arch is horizontal or vertical provided there is abutment at either end of the arch, and the segments are held together stiffly. The arch is then by virtue of its design, very strong. In the lumbar spine, the abutment is provided at the lower end, as the arch rests on the sacrum and pelvis, and at the upper end by the load of the body and the pull of the muscles. The segments are made stiff by the small muscles closely applied to the spine, which pass from one segment to the next, holding them tightly together. Far from being a mistake, or a disadvantage, the hollow at the base of the human spine is an integral part of its strength.

If the spine is so well designed, why does it sometimes fail? The upper part of the spine at the level of the shoulders does sometimes collapse with the development of round-shoulders in the elderly. Is this a failure of the spine to accommodate the upright posture? We think not, and perhaps the quality of the bone in general, rather than the structure of the spine or the upright posture in particular, is responsible for this occasional spinal failure.[4] Osteoporotic fracture is not restricted to the spine, and species other than man suffer from these fractures. Owners of racehorses at Newmarket and breeders of battery hens also seek a solution for osteoporotic fractures. In the Orthopaedic Department in Aberdeen we are sharing in the hunt for environmental factors that might affect the quality of bone and cause these features, but provided bone quality is satisfactory, the gross structure of the spine is not only adequate for carrying the loads of the upright posture, but it also has a useful margin of safety.

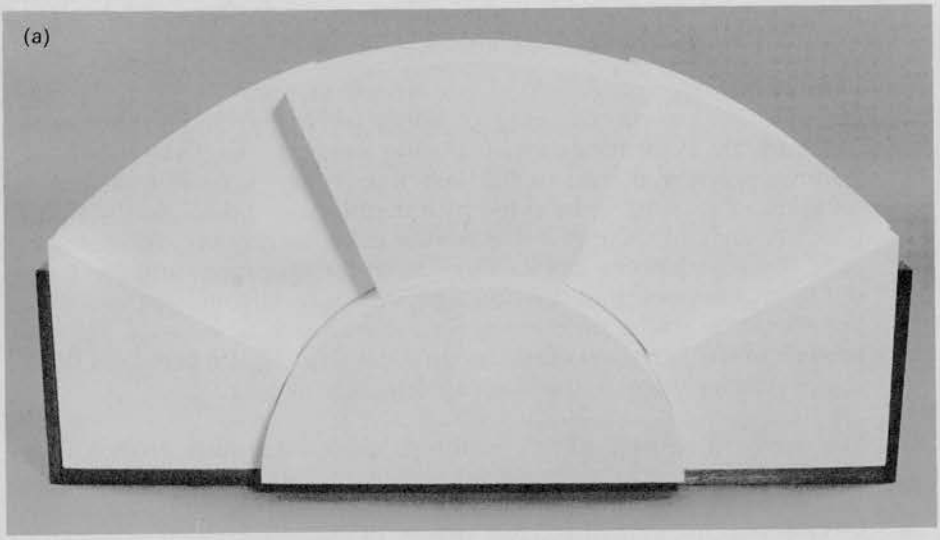


Fig. 5 a) A model of polystyrene blocks shaped into an arch. b) The system is strong if there is satisfactory abutment at each end, and if the segments are held stiffly together.

The SECOND ROLE OF THE SPINE IS TO PROTECT THE NERVES within the vertebral canal.

1. This triangular space behind the vertebral bodies (Fig. 6) is surrounded by a protective ring of bone, with projections for muscle attachments. It forms a segmental tube along the length of the spine, housing the spinal cord in the upper part, and a bundle of nerves bathed in fluid and surrounded by a membrane of dura, in the lower part. In early life, the growing nerves have an intimate relationship with the surrounding bone. They have an epigenetic influence on the bone, ensuring that the bones provide not only enough space for comfort, but also a little more for safety. By four years of age the growth of the vertebral canal is complete (Fig. 7); the vertebral body is still relatively small, but the canal is fully mature.

2. We have measured many hundreds of spines, and observed as expected, that the adult vertebral canal steadily reduces in size down to the fourth lumbar level. At each segmental level, a pair of nerves leave the spine, and with fewer nerves in the canal, perhaps not surprisingly the space reduces. At the fifth lumbar level however, the canal increases in size.[5] The mechanism for increase at the fifth level has not been explained, but it is convenient at a site where the spine has an acute backwards bend, and where the spine is particularly prone to pathological change. Extra space for the nerves at this level is a bonus.

Although the space for the nerves in the vertebral canal is generally adequate, with an additional margin for safety, some individuals have an unusually small canal, called 'spinal stenosis' (Fig. 8). The space is then just adequate in health, but super-added pathology rapidly causes symptoms.[6] There is no margin for safety in the unfortunate stenotic patient who has a disc protrusion, or bony projections from degenerative change which encroaches into the small vertebral canal. The nerves are soon compromised with resulting symptoms in the legs. Stenosis then becomes one factor amongst many accounting for disability. The vertebral canal is protecting the nerves from outside insult, but has it failed in its protective role from injury within?

Spinal stenosis may have a causative genetic component, but the studies of George Clark in Boston,[7] and our work,[8] has shown that there are also environmental factors responsible for stenosis. We suspect that environmental insults before four years of age can permanently stunt the canal. It fails to reach a mature size, and remains stenotic. Growth in pregnancy is particularly critical and is probably inhibited by factors such as toxins, cigarette smoking, alcohol, drugs, infections whether virus or bacterial, and a poor placental circulation. If the canal can be stunted at an early age, like a late running train, it will never catch up. Other systems may grow, to produce a six foot man, with large bones muscles and nerves, but a spinal stenosis can remain.

Given a good start in life however, the canal is large enough to protect the nerves in health, with a margin to spare in disease.

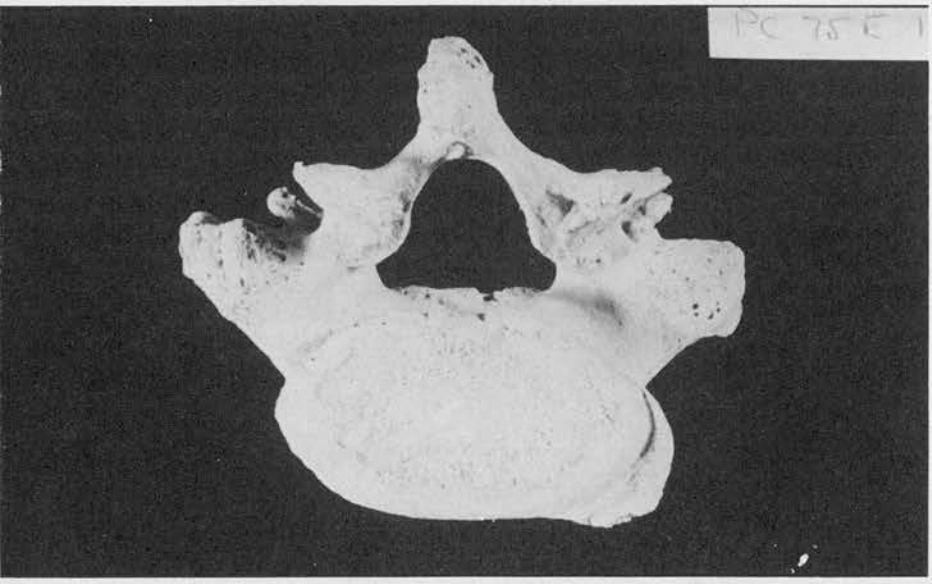


Fig. 6 A photograph of a fifth lumbar vertebra, showing a large vertebral canal behind the vertebral body.

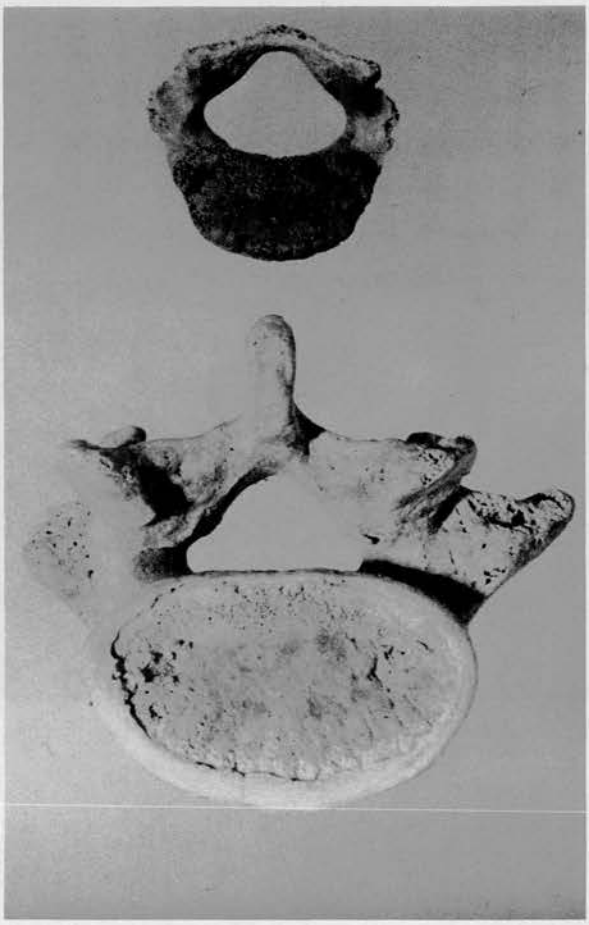


Fig. 7 Photographs of two fifth lumbar vertebrae, the upper vertebra being from a four year old, and the lower from an adult. There is a marked difference in the size of the vertebral bodies, but the vertebral canal of the four-year-old vertebra is largely mature, with the same cross sectional area as the adult.

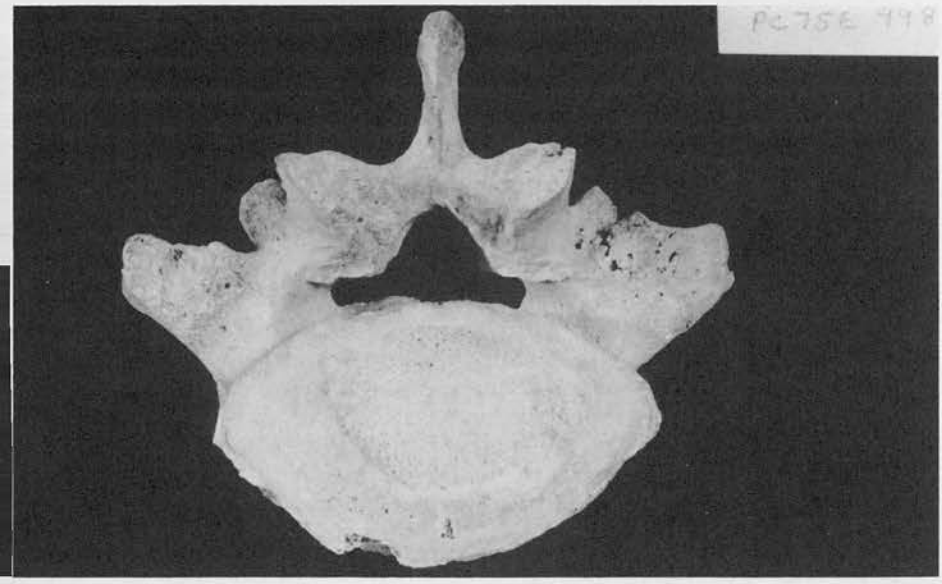


Fig. 8 Photograph of a fifth lumbar vertebra, with spinal stenosis. The vertebral canal is shallow and trefoil in shape, a phenomenon that occurs in about 15 percent of the population.

The THIRD ROLE OF THE SPINE IS TO BE FLEXIBLE.

The spine is flexible because of its segmental nature, with intervertebral discs between each vertebra, but flexibility may appear to be incompatible with strength and protection.

1. The discs are composed of collagen, large molecules of proteoglycan, cells, and depending on age the disc's water component is between 60 and 90 percent. Being the largest avascular structures in the body, their nutrition is of interest, particularly because the cells require adequate nutrition to replace degraded collagen and proteoglycan. Nutrients reach the disc from the small blood vessels in the vertebral end plate above and below the disc, and are transported by diffusion in the disc matrix. Waste products leave by the same route.
2. Although the upright posture increases the stress on the lower lumbar discs, there is a small but significant bonus to disc nutrition from being upright. The large interlacing molecules of proteoglycan exert a strong osmotic pressure attracting water into the disc, and this is balanced by the hydrostatic pressure, or the external load which squeezes water out of the disc. When we get up in a morning, the disc is juicy and full of water. Over the next two hours, the vertical load squeezes water from the disc until an equilibrium is reached between the hydrostatic pressure and the osmotic pressure.[9] At night when we lie down, with a reduction in the hydrostatic pressure, the osmotic pressure is then relatively unopposed, and over two hours, the disc imbibes fluid. We wake up in the morning approximately 17 millimetres taller than the night before. With the fluid entering the disc at night, nutrients are gained, and as water is extruded in the morning, metabolites are lost. This is only a minor component of the water transfer system, but it does have some significance. Communities who have the good sense to enjoy an afternoon siesta have a double benefit to disc nutrition.
3. Cellular activity within the disc is also greatly influenced by pressure, and although our understanding of cellular response is still in embryo, alternating change from recumbency to the upright posture is probably beneficial to disc health.

Disc nutrition can be impaired, and degeneration is very common. Studies of identical and non-identical twins by Tapio Videmann in Helsinki suggest that the genetic component of disc disease is small, when compared with environmental factors. One environmental factor is vibration. Epidemiological work by Pope and his colleagues in Vermont have shown that men in occupations exposed to vibration such as truck drivers, tractor drivers and crane drivers have a greater incidence of disc disease, when accounting for other factors.[10] A second adverse environmental factor is smoking. Magnetic Resonance studies of identical twins has shown that there is impaired hydration of the discs when one twin smokes.[11]

Smoking and vibration probably cause spasm in the small vessels which feed the disc, interfering with the start of the fluid transport mechanism. There are probably other environmental factors yet to be identified, and other areas where nutrition may be affected—the edge of the disc, the

matrix pathway, the cell membrane. We need to understand how discs remain healthy and what are the differences between natural age-related disc changes, and pathological degeneration. Our present knowledge suggests that disc are well endowed with a nutritional transport mechanism to ensure healthy function provided environmental insults are avoided.

4. The disc is responsible for segmental motion, and it may be considered the potentially weak region of the spine. It is necessary to understanding the gross structure of the disc to appreciate how motion is still compatible with the spine's role of strength and protection.

The central part of the disc is homogenous in nature and is described as the nucleus. The outer disc is a layered material, like layers of onion skin. It is composed of parallel collagen fibres, aligned 70 degrees to the vertical, with each alternate layer having fibres in opposing directions. This produces a radial ply effect, like the radial ply of a motor-car tyre, with the ability to withstand large compression forces. A simple model demonstrates the load that can be applied to this type of construction (Fig. 9). Two circular plates represent the vertebrae above and below a disc, with a balloon between representing the central nucleus of the disc. The outer annulus is represented by two layers of rubber bands in opposing directions. Loading this model will not burst the balloon, which is readily contained by the tension in the outer rubber bands.

In the laboratory is not possible to rupture a healthy disc by applying a vertical load to the spine; the vertebral bodies will fail first.[12] In life, if we have the misfortune to fall from a height and land on our feet, we fracture the vertebra before we damage a disc. The discs, far from being the weak link in the chain, are in fact the strongest part of the spine.

5. The orientation of the outer fibres of the disc allow movement in a backward and forward direction, and movement sideways, but by reason of the 70 degree angle of the collagen fibres, rotation is restrained. Clockwise rotation of the disc will tighten one layer of fibres, whilst anticlockwise rotation will tighten the opposing layers. The discs permit movement with rotational restraint. David Hukins in Manchester, has shown that the outer fibres of the annulus allow 3 degrees of rotation without damage.[13] Between 3 and 5 degrees, the fibres suffer plastic deformation, stretching and failing to return to the pre-deformed position. After 5 degrees, some of the fibres will tear. Thus although there is considerable freedom for each segment to move forwards and sideways, twisting movement is restrained to no more than 3 degrees.

Prevention of rotation is the protective role of the disc, protecting the nerve roots from compression as they leave the spine at each segmental level. More than three degrees of rotation will pinch the nerves in the outer part of the vertebral canal, where the nerves leave the spine.

6. Provided the spine is not loaded, and provided the disc is healthy, rotation is easily restrained by the outer fibres of the annulus. When the disc is loaded however, the fibres of the annulus may not be able to restrain rotation unaided, and they benefit from two guardians—the small posterior facet joints, and the musculo-ligamentous system.

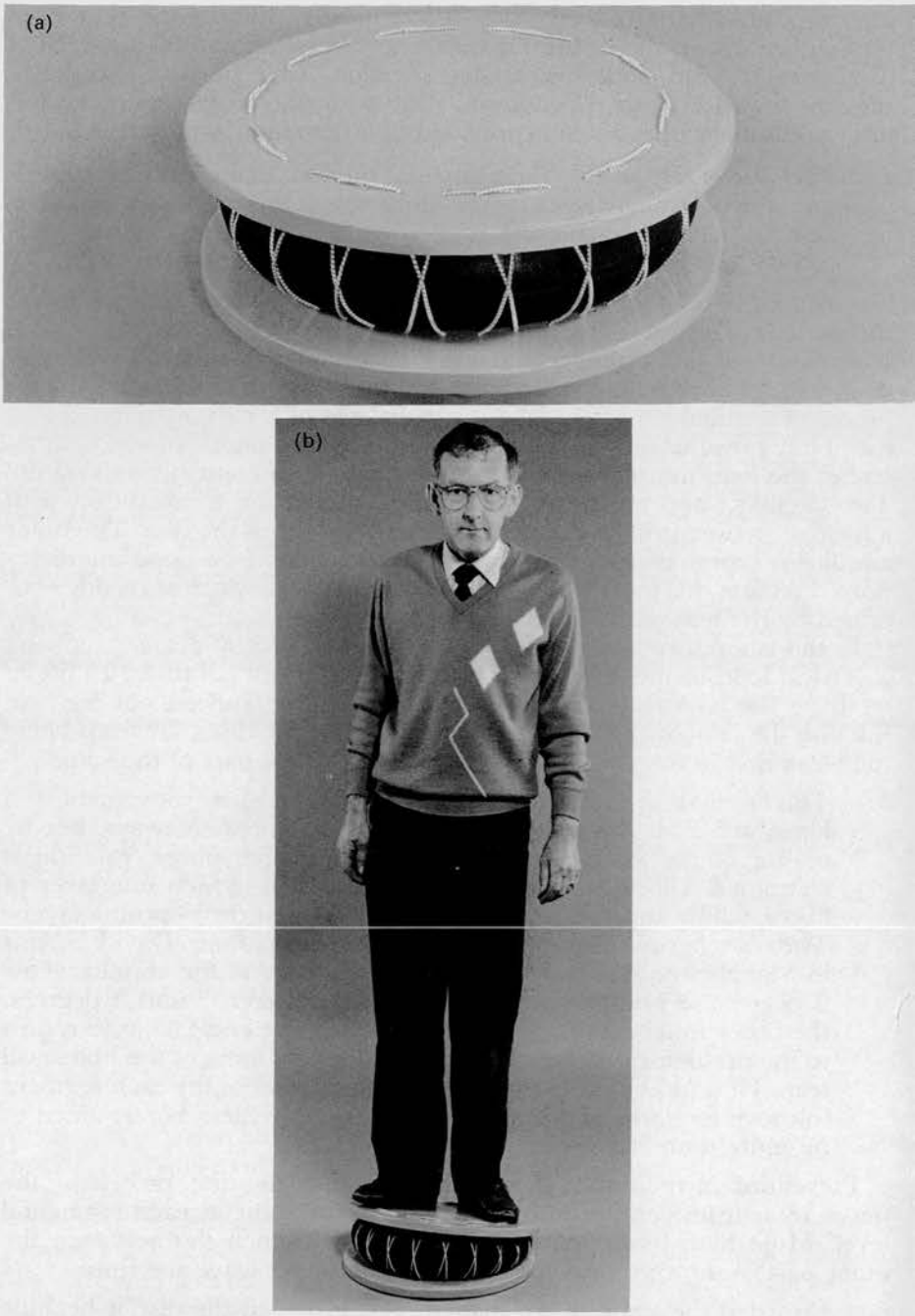


Fig. 9 a) A model demonstrating the radial ply effect of the annulus of the disc. A balloon represents the nucleus of the disc, and two circular plates the vertebrae above and below.Two layers of rubber bands orientated at 70 degrees to the vertical in opposing directions, represent two of the layers of the annulus. b) The system can support considerable axial load partly by the tension in the outer fibres.

7. At the back of the vertebrae two small linking joints stop rotation of more than 3 degrees. They are lined up in the sagittal plane, front to back, permitting forward and backward movement and some sideways bend, but not rotation—that is provided they are locked together with the spine in the upright position. If by chance the spine were bent forwards, the joints would be unlocked, and rotation would not then be opposed by these joints.

8. The second guardian of the disc, inhibiting more than three degrees of rotation is the musculo-ligamentous system.[14] It is worth thinking about how we lift a heavy weight. We take in a deep breath, hold our breath and the muscles of the diaphragm contract. At the same time we contract the abdominal muscles, and the muscles of the pelvic floor. This compresses the abdomen, causing a great rise in the intra-abdominal pressure. In upright man, the lumbar lordosis has thrown the lower spine forwards into the abdominal cavity, which means that the lumbar spine is surrounded on three sides by the abdomen. On three sides it is splinted by the great rise in intra-abdominal pressure, so that it is not possible to lift a heavy weight without the pressure of the abdomen holding the spine stiffly in a splint. It is like the ambulance driver applying a pneumatic splint to the patient's fractured leg at the roadside, in order to hold the leg stiffly together during transport to hospital. The spine is splinted, holding the posterior facet joints locked together during a lift.

Furthermore, behind the spine are muscles which also assist in the spinal splint. The bundles of muscles which are closely applied to the spine are wrapped in envelopes of fascia, called the lumbar fascia (Fig. 10). This becomes a thick sheet of fascia laterally, and is attached to the abdominal muscles. In the process of lifting, when the abdominal muscles contract, they pull on the lumbar fascia, tightening the fascial envelope around these posterior spine muscles, applying them even closer to the vertebrae, and stiffening the segments of the spine. This has a double advantage. It prevents the spine flexing forwards which would unlock the facet joints, risking damage to the disc and the nerve roots. And by stiffening the spine it reinforces the arch for load bearing. The spine's flexibility, rather than being in conflict with strength and protection, is compatible with these two roles, and with the demands of being upright.

When compared with other primates, man in his upright posture has a more robust abdominal musculo-ligamentous system. Man has five lumbar vertebrae, with considerable space between the pelvic brim and the lower ribs, whilst many other primates have only three lumbar vertebrae, and a lesser space.

Discs are sometimes damaged of course. The annulus can tear with a twisting injury in an unguarded moment. A slip, or a fall, can occur before the muscle reflexes have time to respond. The spine is flexed, the joints unlocked, and the annulus of the disc may not restrain the twisting moment, especially if the spine is loaded. Damage can occur, but this is the result of injury, and poor disc nutrition rather than inadequacy of the spine.

I would attempt to draw three conclusions from the observations I have shared with you on the lumbar spine. First, there is an excellence about

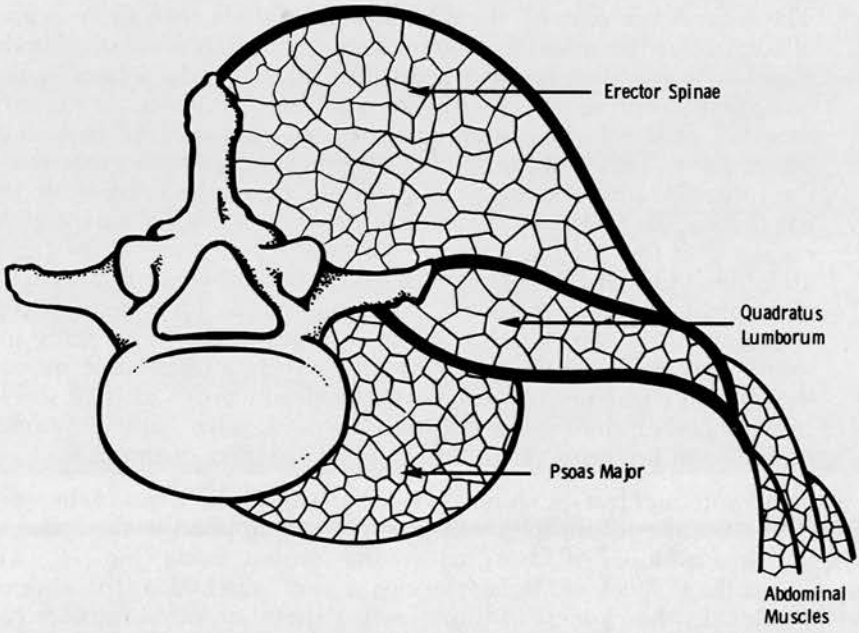


Fig. 10 Diagram to show the posterior muscles of the spine, closely applied to the vertebrae, and wrapped in three layers of the lumbar fascia. This fascia extends laterally to be attached to the abdominal muscles. When lifting and contracting the abdominal muscles, the lumbar fascia is in tension, applying the spinal muscles even more closely to the spine.

the system which echoes the words of the psalmist, *'I am wonderfully and marvellously made'*. It is as though we have been examining pieces of china from a set of porcelain, each stamped with the maker's mark of excellence. Secondly, there is no evidence of a so-called vestigial structure, that the spine was only half adequate, that man was only half upright, or semi-upright. And thirdly, when spinal pathology does occur, and the spine is no more exempt from pathology than any other system, that pathology, rather than being innate to the system, is generally the result of preventable environmental factors for which we are in part responsible.

In research we make observations, and then interpretations. Our observations may be correct, and we can be mistaken in our interpretation. I believe the observations I have shared with you are a true representation of the science of the lumbar spine as we know it today. The interpretation is a matter of opinion and choice. How did we attain the upright posture? Is it a matter of time, perhaps long periods of time, and chance? Or is it the work of design and plan? Here is the interface between science and theology, and here I must stop.

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Aberdeen University Review, no. 187, Spring 1992

'HERE'S TAE US. WHA'S LIKE US?': THE ABSENT SOCIOLOGY OF SCOTLAND

Inaugural Lecture delivered on 28 October 1991

By Steve Bruce

AS Bertie Wooster would have said, 'Tricky blighters, inaugurals': I intend to use my single chance of a hearing outside the circles of my own discipline to complain and to advertise. To help the time pass as entertainingly as possible, I will begin with a very long introduction and then make just one very simple point.

The Mythical Kings

Let me begin by introducing you to a curious episode in the evolution of Scottish intellectual life. Anyone who has toured Holyroodhouse Palace may have been struck by the paintings of the Scottish kings in the Gallery. The guides, Philistines to a man, generally whip parties through with a few coarse jokes about the painter, a Dutchman called Jacob De Wet, having modelled most of the faces on a few tramps he hired from the slums of the High Street. What is forgotten is just how expensive was this act of 18th century propaganda; in relative terms the paintings probably cost more than even this government spends on public relations! The portraits were commissioned by James, Duke of York, who had been sent to Edinburgh by his brother Charles the Second to get him out of the way while the British Parliament quarrelled over the succession. James, who in addition to being an enthusiastic and public Roman Catholic, was a vain and silly man, was not popular and there were moves to exclude him from the succession. The Holyroodhouse portraits were part of his reply. They were commissioned to represent the glorious history of Scotland as having flowed from its unbroken monarchy. Despite the fractured history of the succession, all the kings are there in neat order. To fit the arrangements of the Gallery, most are painted as head and shoulders but eighteen are painted life size. Naturally, since a Stewart was paying for it, all the Stewarts are life size but that left room for a few more big ones and they were chosen to represent kingly virtues. Robert Bruce as the Liberator is full-size as is Kenneth McAlpin, the uniter of Scots and Pics. David the Saint, who did much for the Christian Church in Scotland, is also full-size. The point of the portraits is obvious. The Scottish lords who assembled to await an audience with the king were supposed to be stunned by the antiquity of the monarchy and reminded that the Stewarts (and in particular James) were the rightful heirs to it.

What is unusual about this representation of Scotland's monarchs is that there are too many of them! The series begins in 330 BC, about eight

SPINE Volume 19, Number 2, pp 228–235

A Laboratory Model of Lumbar Disc Protrusion

Fissure and Fragment

49

P. Brinckmann, PhD,* and R. W. Porter, MD, FRCS, FRCSE†

Discs of 20 human lumbar motion segments from donors between 20 and 52 years of age were subjected to a procedure that effected a radial fissure of the anulus, sparing a peripheral layer of approximately 1 mm in thickness. In addition, fragmented tissue pieces that resembled those retrieved at surgery for prolapse were created in the center of the disc. The disc contour was measured under pure axial load as well as in flexion and extension. In the intact specimen, the disc contour shifts in ventral direction in flexion and in dorsal direction in extension. In the 'fissure and fragment' discs a broad-based protrusion develops dorsolaterally at the location of the fissure. The magnitude of the protrusion is independent of flexion or extension angles in the range of ±5°. The 'fissure and fragment' discs exhibit disc prolapse at loads between 0.9 and 6.1 kN and flexion angles below 10°, i.e., under loading conditions well in the physiologic range. The findings of this experiment support the hypothesis that disc prolapse—aside from the hyperflexion trauma described in the literature—has to be preceded by generation of radial fissures and tissue fragmentation within the disc. Thus, prolapse appears to be a late event during the course of a long-term degenerative process. [Key words: human lumbar discs, disc protrusion, disc prolapse]

A lumbar disc prolapse occurs when a separate tissue fragment extrudes or sequestrates through a complete tear of the anulus. By definition therefore a prolapse requires both a fissure and a fragment. The temporal sequence of these events has yet to be established. There are two possible hypotheses, the first that the prolapse is a single event or second that the prolapse is the final episode in a degenerative process.

Disc prolapse from a single event might result from a mechanical overload of the disc from high external forces or high tissue stresses. Under the unfavorable conditions of combined axial loading, side bending, and twisting, a prolapse of disc material could occur suddenly. On the other hand, a slow degenerative process could gradually alter the mechanical properties and the macroscopic integrity of the disc. An innocent load could then trigger disc prolapse as a final event, extruding a tissue fragment through a completely torn anulus.

Epidemiologic data and case histories do not distinguish between these two hypotheses. Some studies show an association between the risk of prolapse with lifting and twisting.[16,17,18] Patient histories can record the onset of disc symptoms with a strenuous episode, yet others show no such association. Our ignorance about preexisting disc pathology, which may be symptomless before the final prolapse, makes it difficult to distinguish between the two hypotheses, or in fact whether both processes occur. The answer has both therapeutic and medico-legal implications.

Laboratory studies have attempted to answer the question by modeling disc prolapse *in vitro*. Early studies suggested that the vertebral body rather than the disc failed when the motion segment was subjected to axial compression.[9,19,21] This was confirmed by Hutton et al[15] and Brinkmann et al,[7] who applied single loading episodes, and by Hardy et al[14] and Brinkmann et al[6] using fatigue loading.

In flexion beyond the physiological limit, the posterior anulus will rupture or sometimes tear from the vertebral endplate. Adams and Hutton[1] demonstrated that hyperflexion combined with high axial load can sometimes produce a prolapse *in vitro*. Such a mechanism may account for the prolapse occasionally seen in athletes, sometimes in combination with a fracture of the posterior rim of the vertebral body,[11] but would probably be unusual in everyday life activity.

Adams and Hutton[2] also investigated gradual disc prolapse with cyclical compression and flexion. Existing radial fissures did not progress to prolapse, but peripheral migration of nuclear pulp was observed in nondegenerate discs from young persons.

Brinkmann[5] was unable to model a disc prolapse by an incision of all the fibers of the inner posterior anulus vertically between the two endplates. The radial contour of these specimens did show a small localized radial bulge (0.3 mm peak height at 1 KN load). However, as the load increased up to the fracture load of the verte-

From the *Institut für Experimentelle Biomechanik, Universität Münster, Germany, and the †Department of Orthopaedics, University of Aberdeen, Aberdeen, Scotland.
Supported by grants from the Deutsche Forschungsgemeinschaft and the Wolfgang Ritter-Stiftung.
Accepted for publication January 18, 1993.

Table 1. Demographic Data of the Spine Specimens

Spine No.	Age	Sex	Disc Degeneration
1	50	m	III
2	52	m	III
3	35	m	II
4	45	m	II
5	51	f	II
6	49	m	III
7	20	m	I
8	49	m	II
9	48	f	II
10	38	m	II
11	25	m	I
12	38	m	II

Disc degeneration was graded according to Galante (1967).

bra, no prolapse occurred. The results suggested that a radial fissure alone does not lead to prolapse.

Gordon et al[13] loaded motion segments with intact posterior elements cyclically in a combination of flexion, rotation, and compression. In 4 of 14 specimens, the authors stated that "nuclear extrusion was shown by complete radial fissure between the central nucleus and an intact or disrupted anulus." Their studies described a fissure but not the extrusion of central or peripheral disc material outside the confines of the disc, which makes interpretation difficult. The majority of their discs showed fissures of the anulus and separation of the peripheral lamellae. Some of these changes may have been present before the testing, and yet the authors conclude that these loading conditions can lead to primary injury of the peripheral anulus and to disc prolapse.

Others investigating the role of axial rotation on the etiology of unilateral prolapse in an experimental and finite-element study conclude that without facet damage, axial rotation is insufficient to cause disc injury leading to prolapse.[4,10]

This study extends previous work,[5] which suggested that a radial fissure alone will not cause disc prolapse *in vitro*. A new model is designed incorporating fragmented disc material with a fissure, and examining the disc contour under different loading conditions. It tests the hypothesis that a disc protrusion will be greater when a fragment is associated with a fissure than when there is a fissure alone, and that pre-existing fragments within a partial anular fissure will prolapse and extrude as the outer anulus ruptures under minimal load.

■ Materials and Methods

Intact motion segments of human lumbar spines were obtained from donors between 20 and 52 years of age. They were tested immediately after autopsy or after immediate storage at −20°C in vacuum-sealed bags. They were dissected into motion segments of two vertebrae, and the intervening disc, stripped of all muscle, but the posterior elements and ligaments were left intact. Disc degeneration was graded according to Galante.[12] Demographic data are listed in Table 1.

The specimens were mounted in an electromechanical materials testing machine. A force was applied via a roller to a metal plate cemented to the upper endplate of the superior vertebra (Figure 1). Thus the load was either purely axially compressive, or by eccentric application, it simulated compressive loading plus a flexion or extension moment. The moments with respect to the lower fixed vertebra are calculated as the product of the axial force multiplied by the eccentricity. The roller gave the upper vertebra freedom to move with respect to the lower vertebra in compression, anterior-posterior shear, flexion, or extension. Two linear transducers measured the angulation of the upper vertebra in the sagittal plane to 0.05°. The specimens were kept moist during preparation and testing, and the experiments were carried out at room temperature.

The radial extension of the disc in its midplane, the "disc contour," was measured by a new three-joint goniometer (Figure 2). It consisted of three arms (A, B, and C) connected by axes perpendicular to the plane of measurement. The relative angles of the arms (U, V, and W) were measured by angle transducers mounted on these axes. From the length of the arms and their angles, the center coordinates (x,y) of the tip P could be calculated. The accuracy of the coordinate measurement, determined from the measurement of an object of known shape was 0.015 mm.

The probe P consisted of a vertically orientated metal cylinder of 2.5-mm radius and 5-mm height. This ensured that the probe maintained contact with the disc at the point of its

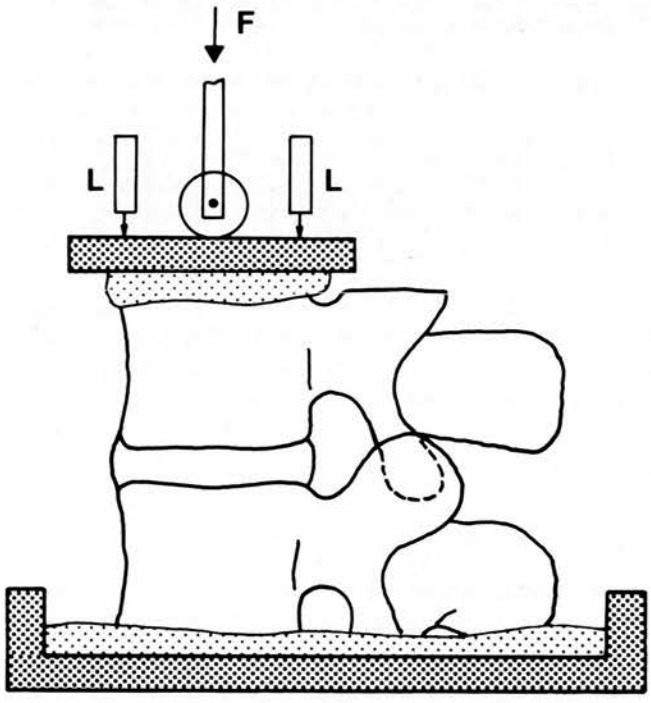

Figure 1. Mounting of a motion segment specimen in the materials testing machine. The compressive force F is applied via a roller to a stainless steel plate mounted on top of the superior vertebra. The point of application of the force can be shifted in the sagittal plane. The superior vertebra is unrestrained to movement in compression, flexion, extension, and anterior-posterior shear. The angulation of the upper vertebra is measured by two linear transducers L.

largest radial extension irrespective of small changes in disc height or in disc geometry when the segment was flexed or extended. A mathematical correction was applied to the measured contours to account for the probe/disc contact occurring at the periphery of P, while the measured coordinates were calculated at the center of P (Appendix).

The disc contour was measured by guiding the probe manually around the disc. Because the posterior elements were intact, the complete disc contour was measured in two phases, introducing the probe first through one foramen and then through the other. A foraminotomy was necessary to provide free access for the probe, removing 2 or 3 mm of bone without disturbing the integrity of the apophyseal joints.

When measuring the disc contour and its radial extension, the dorsal margin of the foramen was also measured on the ventral surface of the right and left processi articulares craniales of the inferior vertebra to provide a fixed landmark. These two contour sections provided fixed landmarks to evaluate a shift of the whole disc during the experiment.

Measurements were obtained as an attempt was made to simulate a disc protrusion and prolapse as follows:

1) The contour of the disc was measured in the intact specimen under axial compression at 1 and 2 kN, and in flexion and extension at 1 kN load, with flexion or extension moments of 15 Nm.

2) A sharp cutting instrument was introduced through a ventral incision, dividing all the dorsolateral anular fibers except the outermost 1 mm of anulus. The cutting tool and the auxiliary tools used for insertion of the cutting tool are identical to the tool used in a former experiment;[5] for illustrations of the type of injury produced by this tool, the reader is also referred to this publication. The position and the depth of the radial fissure was controlled on the computer screen by measuring two landmarks on the cutting tool (Figure 3). The incision divided not only the fibers vertically between the endplates, but laterally for approximately 1 cm, including the fiber insertions at the endplates within this channel. It produced a broad radial fissure but spared the outer anulus.

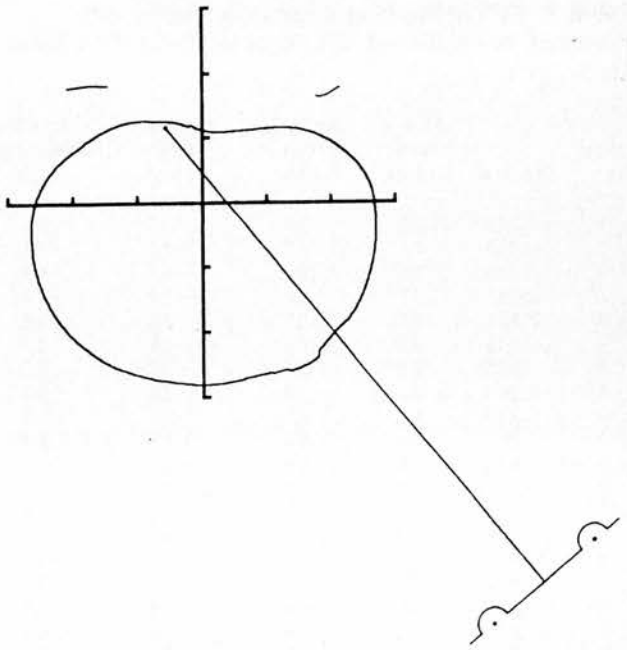

Figure 3. Example of a graph obtained when controlling the location and depth of the artificial radial disc fissure. The graph displays the contour of the intact disc and the position of the cutting instrument calculated from the measured coordinates of two landmarks on its handle. Also shown is the calculated minimum distance between the cutting edge of the tool and the disc contour.

3) The disc contour was then measured again, under a load of 1 kN in axial compression and also in flexion and extension. In a preparatory test, four motion segments were then subjected to cyclic loads varying between 0 and 1 kN applied eccentrically at a frequency of 0.25 Hz. After 1000 cycles of 0° to 5° of flexion and 1000 cycles of 0° to 4° of extension, no change was observed in the disc contour, and cyclical tests were therefore discontinued.

4) A discectomy was then performed with a rongeur inserted through the same ventral incision, excising between 2.3 and 3.1 g of disc material. Discrete fragments of disc material approximately 8 × 3 × 2 mm were cut from the central and inner anular disc tissue from another disc of the same spine, and these were then introduced into the disc space of the discectomy segment. The size of pieces of disc material was selected to imitate the size of fragments frequently removed at surgical discectomy, and the weight was matched to within 0.1 g of tissue previously excised from the specimen. The height of the specimen (under 1kN axial load) after refill was within 0.2 mm of the original disc height.

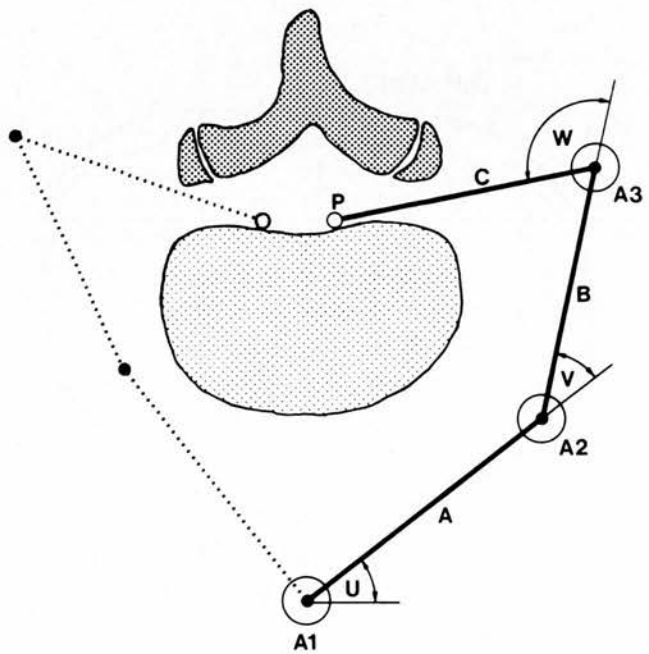

Figure 2. Graph of a horizontal section through the disc and the zygoapophyseal joints. The disc contour is traced by the probe P mounted at the tip of the three-arm goniometer (A,B,C). To measure the contour in its dorsal part, the probe is introduced through the foramina first from one and then from the other side (dotted position of the goniometer).

Table 2. Shift of the Disc Contour in Flexion and Extension in Relation to Disc Contour Under Pure Axial Load

Spine No.	Specimen	Flexion Angle (deg)	Contour Shift in Flexion (mm)	Extension Angle (deg)	Contour Shift in Extension (mm)
1	L2/L3	5.0	0.3	−3.4	−0.3
2	L2/L3	7.1	1.2	−2.1	−0.2
2	L4/L5	7.9	1.1	−4.7	−0.9
2	T12/L1	3.7	0.6	−3.4	−0.5
3	T12/L1	4.0	0.3	−3.0	−0.4
3	L4/L5	7.4	0.9	−4.9	−0.7
4	L4/L5	6.8	0.7	−5.2	−0.6
4	T12/L1	3.4	0.2	−2.8	−0.3
4	L2/L3	5.4	0.6	−3.6	−0.5
5	L2/L3	5.6	0.4	−4.9	−0.6
5	L4/L5	5.4	0.5	−4.3	−0.5
6	L4/L5	7.2	1.0	−5.3	−0.5
6	L2/L3	7.0	0.7	−4.2	−0.4
7	L4/L5	7.7	0.7	−2.9	−0.3
8	L4/L5	5.5	0.8	−4.2	−0.6
9	L4/L5	9.2	0.9	−4.9	−0.5
10	L2/L3	4.3	0.8	−4.0	−0.5
10	L4/L5	5.2	0.8	−4.5	−0.8
11	L4/L5	6.1	0.6	−2.0	−0.2
12	L2/L3	5.3	0.6	−4.2	−0.6

The value quoted is the mean value of the contour shift measured dorsomedially and at the location of the left and right foramina. A positive value indicates a shift in ventral direction; a negative value indicates a shift in dorsal direction (resulting in reduced space for the nerve tissues). Loading conditions: 1 kN axial load, or 15 Nm of flexion or extension moment respectively in addition to 1 kN axial load. Angles are counted positive in flexion and negative in extension.

5) The ventral aperture of the anulus through which the cutting tool and the rongeur had been introduced was then closed effectively with a five-stitch suture. Three stitches ran over the defect in horizontal direction (like the bars of an E); two stitches crossed the defect (like an X). Without such a suture loose fragments of disc material would extrude through the aperture at very low loads of less than 100 N. Other attempts to seal the ventral anulus incision with a metal implant and with cyanoacrylate tissue adhesive ("Histoacryl") were unsuccessful.

6) The contour of the disc was again measured, under axial compression and in flexion and extension.

7) The load was increased in increments of 1 kN up to failure. In the first ten specimens, the load was applied first centrally and then 1.5 cm eccentrically with a flexion and then extension moment (magnitude: load × 0.015 Nm). In the second ten specimens, extension was tested before applying flexion to document a correlation of disc failure with bending angle. The disc contour was measured at each stage of loading.

■ Results

In all specimens tested, the initial axial loading at 1 and 2 kN showed no asymmetry of disc contour or any localized radial bulge that might have suggested a preexisting protrusion.

When the intact motion segment was subjected to flexion, the dorsal and dorsolateral bulge of the disc decreased when compared with the contour under pure axial load (Table 2, Figure 4). At 6° of flexion, there was an average decrease of 0.7 mm. The decrease of the dorsal bulge in flexion is due to the stretching of the posterior anular fibers.

In extension, there was an increase in the dorsal bulge (Figure 5), on average of 0.5 mm at 4° of extension. This implies less space for the nerve tissues in the central and root canal during extension.

The experimental radial fissure of the inner anulus produced a small eccentric radial protrusion at that site (Table 3). Under 1 kN axial load, the artificial protrusion had an average height of 0.8 mm. The size of the protrusion in relation to the corresponding contour of the intact disc did not change in flexion or extension. This broad-based inner anular fissure produced a protrusion three times larger than that of an earlier experiment[5] when the inner anular fibers were incised with a single sharp cut.

After discectomy and reinsertion of fragments of disc material, there was a further increase in the size of the protrusion (Table 3, Figure 6) to an average of 1.1 mm under 1 kN axial load. Again the contour did not change in flexion or extension when compared with the initial intact state.

As the load was increased in these "fissure and fragment" specimens, increasing in increments of 1 kN in axial compression, flexion and extension, 18 of the 20

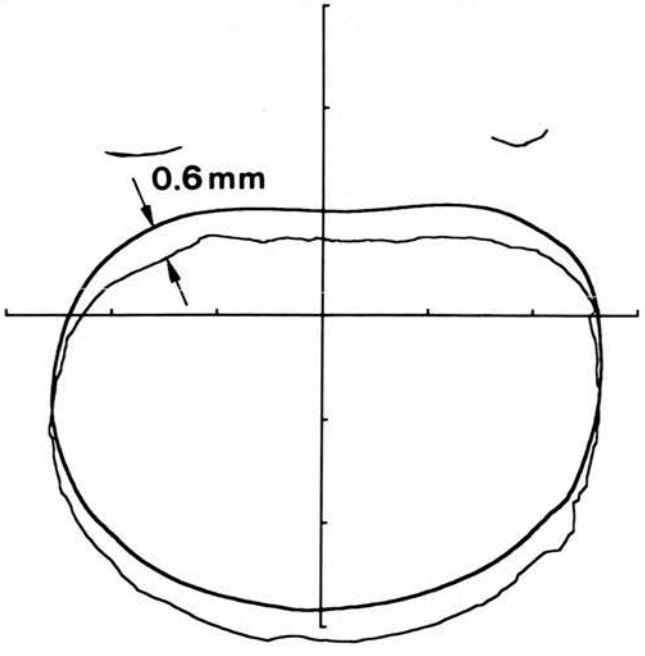

Figure 4. Example of the shift of the disc contour in flexion (1 kN plus 15 Nm flexion moment) in relation to the contour under axial load (1 kN). Motion segment L4/5, flexion angle 5.4°, 5× magnification of difference between the two contours. In this graph as well as in Figures 5 to 7 the two contour parts dorsal to the disc designate the dorsal margin of the foramen, i.e., the ventral surface of the left and right processi articulares craniales of the inferior vertebra. Perfect superposition of these contours proves that the specimen as a whole did not move between the two contour measurements shown.

specimens exhibited a disc prolapse, with rupture of the anulus and extrusion of the fragments. This occurred at loads between 0.9 and 6.1 kN and at flexion angles below 10 degrees (Table 4). In all the specimens tested, the prolapse occurred in either axial compression or axial compression combined with flexion. Prolapse did not occur in the extension mode. Sometimes the prolapse occurred while raising the load a further kN, and sometimes after 1 or 2 minutes in static load. Figure 7 shows a disc contour measurement only a few seconds before prolapse. Prolapse always occurred at the site of the inner anular fissure, and all the previously inserted fragments extruded at once.

■ Discussion

This experiment has shown that *in vitro,* a broadly based fissure of the inner anulus, leaving intact 1 mm of outer anulus, results in only a small bulge (mean 0.8 mm). When the fissure is associated with loose fragments of disc material there is a slight increase in the bulge (to a mean of 1.1 mm). It then takes only a small load of between 0.9 and 6.1 kN, and a flexion angle of less than 10 degrees, for the bulge to rupture with a prolapse of extruded fragments through a complete anular tear. This type of loading is well within everyday physiological conditions.

A similar situation exists *in vivo* when a fissure extends to the outer anulus. Under load and in flexion and extension there is only a small bulge that will compromise neurologic structures only in the presence of stenosis. A fissure may be present for months or years without compromising an adequate canal.

When a fissure is associated with a loose fragment, however, there is a slightly larger bulge that readily ruptures under physiological loads. It is not known how the center or the periphery of the disc develops fragments *in vivo*. The mechanism is open to speculation—asymptomatic endplate fractures, fatigue, impaired blood supply near the endplates, and changes in diffusion barriers. Histologic investigations[8,20] show that in a large percentage of cases extruded fragments consist of endplate material and not just of (former) nuclear material. Cadaveric discograms demonstrated that the nucleus can coalesce into fibrous lumps, separated from the endplates by parallel fissures.[3] Further degeneration could cause the tongue of tissue to separate into an isolated fragment, when a minimal load will precipitate prolapse.

Once present, the fragment suddenly and significantly alters the status of the disc. A minimal stress, such as a cough or a stumble would be sufficient to cause a disc prolapse with extrusion of the fragment. To the patient it seems a significant event, but in pathologic terms it is only the end of a long developing process with prolapse as the final event.

Table 3. Magnitude of the Localized, Dorsolateral Disc Protrusion After Creation of the Radial Fissure and After Subsequent Discectomy and Refill with Fragmented Pieces of Disc Tissue

Spine No.	Specimen	Localized Protrusion (mm) After Radial Incision			Localized Protrusion (mm) After Incision, Discectomy and Refill		
		Ax	Flex	Ext	Ax	Flex	Ext
1	L2/L3	0.2	0.2	0.3	0.3	0.4	0.5
2	L2/L3	0.1	0.2	0.0	0.2	0.3	—
2	L4/L5	0.0	0.2	−0.1	0.0	0.0	0.3
2	T12/L1	0.5	0.5	0.5	0.5	0.5	0.7
3	T12/L1	0.5	0.6	0.6	1.4	1.4	1.7
3	L4/L5	0.5	0.3	0.7	1.5	1.1	1.7
4	L4/L5	0.5	0.5	0.3	0.8	0.5	1.1
4	T12/L1	1.3	1.0	1.2	*	*	*
4	L2/L3	1.3	1.2	1.5	1.8	1.8	1.9
5	L2/L3	1.0	0.9	1.1	1.1	1.2	1.3
5	L4/L5	0.8	0.7	0.7	0.7	0.8	—
6	L4/L5	1.0	0.9	1.0	1.0	1.1	1.1
6	L2/L3	0.4	0.5	0.4	1.3	1.5	1.3
7	L4/L5	0.6	0.5	0.5	0.7	—	0.5
8	L4/L5	0.6	0.5	0.7	0.6	0.4	0.7
9	L4/L5	1.3	0.9	1.2	1.3	1.4	1.4
10	L2/L3	2.0	1.7	1.9	2.3	2.5	2.6
10	L4/L5	2.0	1.9	1.9	2.2	2.2	2.0
11	L4/L5	1.0	0.9	1.0	1.9	2.2	1.7
12	L2/L3	0.5	0.2	0.6	1.1	0.7	1.6

* Not measured because prolapse occurred at 0.9 kN axial load.
The protrusion was measured in axial compression (ax), flexion (flex) and extension (ext) in relation to the initial state under axial compression, flexion or extension respectively. Loading conditions: 1 kN axial load, or 15 Nm flexion or extension moment respectively in addition to the axial load. Flexion and extension angles equalled those listed in Table 2.

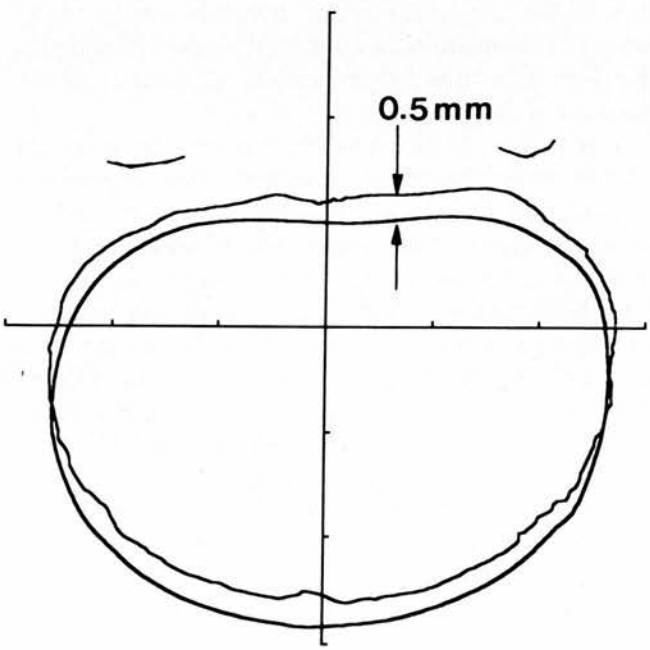

Figure 5. Example of the shift of the disc contour in extension (1 kN plus 15 Nm extension moment) in relation to the contour under axial load (1 kN). Motion segment L4/5, extension angle 4.3°, 5× magnification of difference between the two contours.

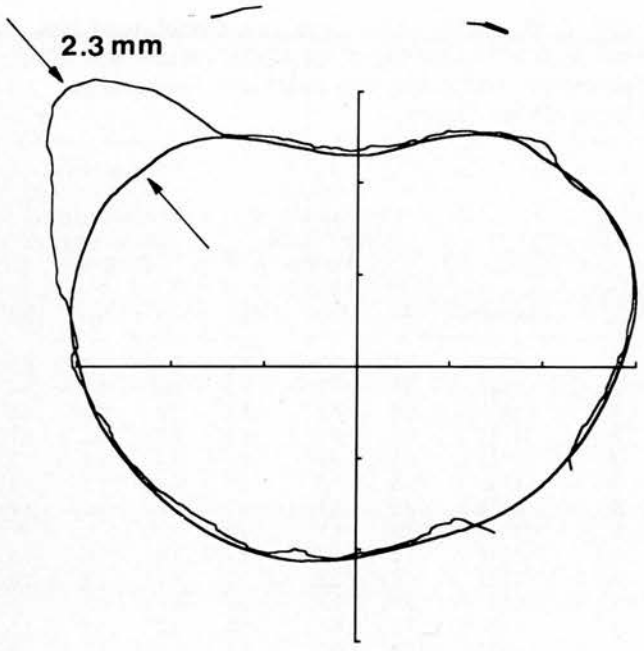

Figure 6. Example of protrusion in a disc with a radial fissure and loose fragmented tissue pieces in the central volume of the disc ('fissure and fragment' model). Motion segment L2/3, 1 kN axial load, 5× magnification of difference between the two contours.

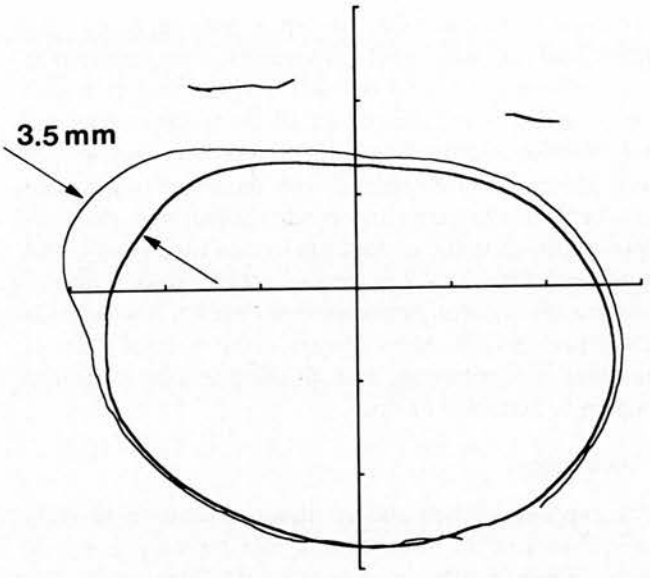

Figure 7. Example of protrusion in a disc with a radial fissure and loose fragmented tissue pieces in the central volume of the disc immediately before prolapse. Motion segment L4/5, load: 4 kN plus 6 Nm flexion moment in relation to initial state loaded 1 kN plus 1.5 Nm; 2× magnification of difference between the two contours.

We do not suggest that the magnitude of the load is unimportant in every disc prolapse. This experiment standardized the fissure of the inner anulus to within 1 mm of the periphery. With less complete fissures, fragments will probably prolapse only under greater loads. Many combinations of conditions in which the fragment is still contained may exist.

Table 4. Loading Conditions Which Produced a Disc Prolapse in Specimens with a Radial Fissure and Loose, Fragmented Tissue at the Center of the Disc

Spine No.	Specimen	Load at Prolapse: Axial Load (kN)	Load at Prolapse: Flexion Moment (Nm)	
1	L2/L3	2.0	0.0	
2	L2/L3	1.9	0.0	
2	L4/L5	5.6	0.0	
2	T12/L1	3.1	46.5	
3	T12/L1	1.7	25.5	
3	L4/L5	2.0	0.0	
4	L4/L5	6.1	0.0	
4	T12/L1	0.9	0.0	
4	L2/L3	2.4	0.0	
5	L2/L3	2.0	30.0	
5	L4/L5	3.0	0.0	
6	L4/L5	3.0	0.0	
6	L2/L3	—	—	Fracture of L2 at 3 kN
7	L4/L5	4.0	40.0	
8	L4/L5	3.8	0.0	
9	L4/L5	—	—	Fracture of L4 at 4 kN
10	L2/L3	3.0	0.0	
10	L4/L5	3.0	0.0	
11	L4/L5	3.6	0.0	
12	L2/L3	2.0	0.0	

If prolapse occurred under application of a flexion moment, the flexion angle at the onset of prolapse ranged between 5° and 8°.

We have to explain how at surgery some disc protrusions are incised and no loose fragment is found. The diagnosis may be incorrect, and the protrusion may be insignificant clinically. Alternatively there may be a fragment still unidentified, or another process may be responsible for a large soft protrusion, not yet successfully modeled in the laboratory.

Surgeons are unsure how much disc material should be removed at the time of surgery. This experiment suggests that care should be taken to identify and remove all the loose fragments. Once the anulus had ruptured, all the loose pieces of disc material extruded at loads below 100 N, which would explain how a hidden loose fragment can be responsible for a recurrence of symptoms in the early postoperative period. Having removed the loose fragments, however, there may be no need to excise the remainder of the nucleus. The fragment, not the fissure, is the problem.

If the disc fragment is the key to protrusion and prolapse, then therapy must address the problem of the fragment. Chymopapain, laser treatment, and percutaneous discectomy will succeed if they eradicate the free fragment. If the fragment remains *in situ* they will probably fail.

In conclusion, the key to the pathology of disc protrusion and prolapse is the loose fragment. This implies a chronic rather than an acute process, and it suggests

that prevention and therapy should focus on the fragment.

■ Appendix

Mathematical Procedure to Document Disc Contours

The goniometer designed for measurement of the disc contour consisted of three arms (A,B,C) joined by axes perpendicular to the plane of coordinate measurement (Figure 8). Arms A and B had a length of 100 mm, and C a length of 70 mm; all dimensions known with an error of ±0.02 mm. Angle transducers were mounted on the axes. Measurement of angles was accurate to 0.01 degree. If lengths of A, B and C as well as relative angles U, V and W are known, the coordinates (x,y) of the center of the probe P at the tip of arm C can be calculated. Under the assumption of an error of 0.1 mm in each arm length and 0.01° in each angle, a Monte Carlo calculation predicted a maximum error in x and y of 0.06 mm and a mean error of 0.22 mm (SD = 0.01 mm) within the measurement area, a circle of radius r = 270 mm.

The angle transducers, however, had no built-in zero mark so that angles U, V and W could not be measured directly but only relative to an unknown zero-position as U-Uo, V-Vo, W-Wo, where Uo, Vo and Wo were unknown constants. In addition, the origin of the goniometer at the free end of A could be shifted by unknown

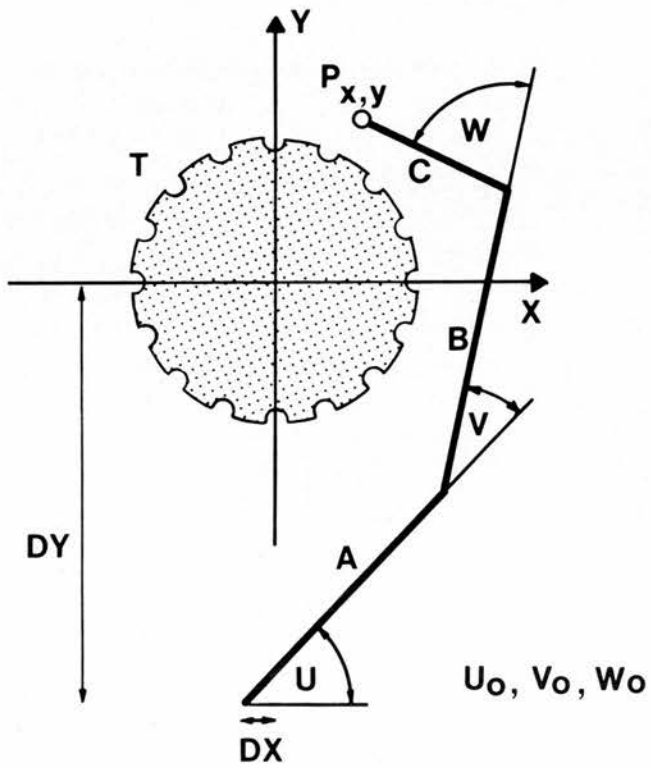

Figure 8. Calibration of the goniometer by measurement of a known test object T. A,B,C: goniometer arms; U,V,W: relative angles of the arms; Uo,Vo,Wo: zero position of the angle transducers; DX,DY: shift of the origin of the goniometer with respect to the x,y coordinate system defined by the test object T.

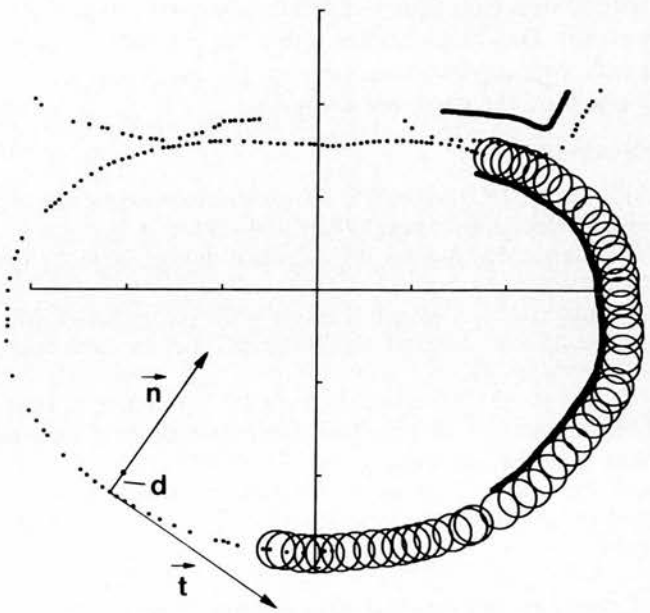

Figure 9. The graph illustrates the correction applied to the raw data of the goniometer. Small dots: center coordinates of the probe P; circles: actual outline of the probe P in different positions. The true contour is given by the thick line, tangential to the outlines of the probe P in successive positions. The true contour is located at a distance d (radius of the probe P) normal to the tangent t to the raw coordinate data.

increments DX and DY with respect to coordinate origin. For this reason, a calibration object was placed on the specimen fixture before the start of each experiment. The calibration object had 16 grooves of semicircular cross section, exactly matching the shape of the probe P. The center coordinates of the grooves were known to 1/100 of a mm. For calibration, the probe P was placed in each of the grooves in succession and the 3 gonimeter angles were recorded. With knowledge of the true values of the 16 coordinate pairs, the 5 unknown constants (Uo, Vo, Wo, DX, DY) were adjusted by means of a least square fit. The quality of the fit could then be checked indirectly by docking the probe P into one of the grooves and measuring the same point 20 times, though with different angulations of the goniometer. This procedure reproduced the known coordinates with an error (SD) of typically 0.015 mm.

A correction had to be applied to the raw coordinate data due to the fact that contact between the probe P and the disc surface occurred at the periphery of P. This is illustrated in Figure 9, showing in its left half the raw data, i.e. the midpoint coordinates of P, and in the right half the actual outline of the probe P in successive locations along the disc contour. The true disc surface is tangential to this set of probe locations. To correct each measured probe coordinate pair, a second order polynomial was fitted to five consecutive raw data points, the tangent t and the normal vector n were calculated and the raw data point was shifted by a distance d in the

normal direction n; d = 2.5 mm was the radius of the probe P. The samples of the outline of the superior facet joints were corrected accordingly. The thick lines in Fig. 9 illustrate the corrected contours.

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Address reprint requests to

Prof. Dr. P. Brinckmann
Institut für Experimentelle Biomechanik
Domagkstraße 11
D-48129 Münster

Drugs 48 (2): 189-198, 1994
0012-6667/94/0008-0189/$10.00/0

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Pharmacological Management of Back Pain Syndromes

Richard W. Porter[1] and *Stuart H. Ralston*[2]

1 University of Aberdeen, Department of Orthopaedics, Aberdeen, Scotland
2 University of Aberdeen, Department of Medicine and Therapeutics, Aberdeen, Scotland

Contents

Summary

The symptom of back pain may be the result of many different pathologies. As such, patients with back pain require careful assessment to determine whether the cause is from the spine or other systems. For acute mechanical back pain, treatment is often symptomatic.

Symptomatic treatment may include analgesics, anti-inflammatories and/or muscle relaxants. Patients may also need hypnotics in the short term to help them sleep at night. However, drug therapy should be reduced and stopped as soon as possible. Furthermore, too much bedrest may be counterproductive.

Paracetamol (acetaminophen) is the standard treatment for transient back pain. More severe pain may require the addition of an opioid, such as codeine or dextro-

propoxyphene. Morphine and pethidine (meperidine) may be necessary in patients with back pain due to neoplastic disease or osteoporotic fracture. However, the opioid analgesics are associated with dependence, tolerance and adverse effects.

Nonsteroidal anti-inflammatory drugs (NSAIDs) have analgesic efficacy comparable with paracetamol. Individual patients respond differently to different NSAIDs, and several agents may have to be tried. Long term therapy with NSAIDs is necessary in diseases with an inflammatory component such as ankylosing spondylitis.

Calcitonin reduces bone resorption and bone blood flow, and has been suggested to have central analgesic effects. As such, it has been used successfully in patients with Paget's disease, osteolytic bone disease and osteoporosis.

Bisphosphonates also inhibit osteoclastic bone resorption and may be useful in Paget's disease, osteolytic metastases and osteoporotic fractures.

Other drugs which may be useful in relieving back pain associated with specific circumstances include the tricyclic antidepressants, anxiolytics, antiepileptic agents, corticosteroids, colchicine and chymopapain.

Back pain and sciatic pain can be one of life's most unpleasant experiences. In the acute form it may be the worst pain the patient has ever experienced. Chronic pain in the lower back is less severe, yet it can cause great distress and seriously reduce quality of life.

This paper reviews the differential diagnosis and management of patients with acute and chronic back pain and abnormal pain behaviour. The place of various agents in the general management of back pain are discussed along with practical recommendations for management of back pain associated with specific underlying disease states.

1. Diagnosis

Back pain is of course only a symptom. It may be the result of many different pathologies, each requiring individual management specific to the underlying cause (table I).

It is obvious that the patient whose back pain is from an aortic aneurysm requires more than pain relief. Similarly, pain due to osteolytic bone lesions from myeloma needs different management from ankylosing spondylitis. Therefore, every patient with back pain requires careful assessment to distinguish whether the pain is from the spine or from other systems. If from the spine, assessments should be made to determine whether it is from some sinister lesion in the bone, or is of an inflammatory or mechanical nature. The last is the most common, but the diagnosis should not be taken for granted.

Mechanical back pain is defined as back pain which was precipitated by and is affected by mechanical factors. It can usually be distinguished from other types of back pain by a good history and examination. Diagnosis can sometimes be more specific. The diagnostic criteria are fairly well de-

Table I. Common causes of back pain

Cause of back pain	Age (years); gender
Acute	
Disc prolapse	20-50; male > female
Vertebral fracture	60-80; female > male
Intrathoracic or abdominal aneurysm	50; both genders
Pancreatitis	40+; both genders
Myocardial infarction	40+; both genders
Chronic	
Degenerative lumbar spine disease	50+; both genders
Osteoporosis	60+; female > male
Ankylosing spondylitis	20+; male > female
Paget's disease	50+; both genders
Soft tissue	30+; both genders
Neoplasia	any age; both genders

Symbol: > indicates greater prevalence in the former than the latter group.

fined for disc protrusion or sequestration, especially if there is sciatica. Also, root pain from degenerative root canal stenosis is well recognised, as is neurogenic claudication. However, in the absence of root pain, it may be difficult to understand the mechanism of back pain of mechanical origin. The pain may be related to the disc, to the facet joint or to musculoligamentous structures. Sometimes the diagnosis gets no further than acute mechanical back pain, and treatment is then symptomatic.

2. Investigations

Most patients with back pain will require some investigation to determine the underlying cause. Although several investigations may be required, the extent of investigation will depend on the clinical history and physical evaluation in individual patients. Some commonly used investigations and their indications are shown in table II.

Table II. Investigation of back pain

Investigation	Suspected disorder
Plain x-ray of the spine	Osteoporotic fracture
	Paget's disease
	Osteoarthritis
	Degenerative disc disease
	Osteolytic lesions[a]
	Ankylosing spondylitis
x-Ray of sacroiliac joints	Ankylosing spondylitis
Abdominal ultrasound	Aneurysm
MRI spine CT myelography	Disc prolapse
	Degenerative stenotic pathology
Radionuclide bone scan	Paget's disease
	Osteolytic lesions[a]
Bone mineral density	Osteoporosis
Biochemistry	
Immunoglobulins	Myeloma[b]
Cardiac enzymes	Myocardial infarction
Amylase	Pancreatitis
Blood count/ESR	Myeloma
	Ankylosing spondylitis

a Bone scan is more sensitive than x-ray in most cases.

b False negatives may occur in myeloma.

Abbreviations: CT = computerised tomography; ESR = erythrocyte sedimentation rate; MRI = magnetic resonance imaging.

3. Mechanical Back Pain

This is defined as back pain which was precipitated, and is affected, by mechanical factors.[1] Although many causes of back pain (ankylosing spondylitis, osteoporosis and osteolytic lesions) may have these characteristics, we define mechanical back pain in this article as pain arising from degenerative disc disease or osteoarthritis.

Most patients with an acute episode of mechanical low back pain make a good recovery in a few days. They may have recurrent episodes, and they may or may not be left with a vulnerable back. In only a minority of patients does pain persist for more than a few weeks, but when it becomes a chronic problem it taxes the skill of the clinician. In cases of chronic back pain, clinicians must try to answer several questions.

- Is it still an organic mechanical problem, or is there now an element of abnormal pain behaviour, in which case drugs are inappropriate?
- Is this still a problem than can be managed conservatively, or is there a lesion requiring surgery?

A working diagnosis and an understanding of the natural history is a prerequisite to pharmacological management. The patient is counselled, the anxiety level decreases and appropriate drug therapy is considered.

When patients are *immobilised* with severe back pain, they need analgesics, perhaps with an anti-inflammatory component, and possibly a muscle relaxant. They need to get to sleep at night. Drugs, however, should be reduced and stopped as soon as possible. Too much bedrest is counterproductive. Two days in bed for acute back pain is as good as 2 weeks.[2]

In the *recovery period* it is better to mobilise and experience some back pain, than risk the complications of bedrest and the sedating effects of too many drugs. This is no problem when the patient is getting better, but it is quite a dilemma for clinicians treating patients whose back pain may become chronic.

Drug treatment for patients with *chronic* back pain is complex. Can too many drugs reduce the patient's endorphin production? Can this encourage abnormal pain behaviour? How much should the patient be permitted to suffer? The clinician learns to resolve these questions by the hard road of experience. The present authors believe that many patients with chronic back pain suffer from an excessive prescription of analgesics.

Patients with *abnormal pain behaviour* are frequently taking far too many drugs including analgesics, opioids and anti-inflammatory agents. Inappropriate prescribing frequently compounds the difficulty of coping with distress, and addiction can be a problem. The drugs may have lost their efficacy, may have become addictive and may be producing adverse effects. There is sometimes a place for prescribing a cocktail of drugs in increasingly weaker doses, in order to wean the patient off excessive medication.[1]

3.1 Indications for Surgical Referral

Absolute indications include patients with evidence of disc protrusion and associated bladder dysfunction. In such patients, early surgery is imperative. Relative indications include patients with root signs from disc protrusion which fail to improve over a 2-week period, and patients with recurrent episodes of disc protrusion interfering with quality of life. Older patients with severe root pain from degenerative lateral canal stenosis will benefit from surgical decompression. Patients with neurogenic claudication require investigation and perhaps spinal decompression. Fusion benefits patients with chronic back pain due to isthmic spondylolisthesis. However, random fusion for chronic back pain is unrewarding.

4. Ankylosing Spondylitis

This should be suspected in young patients (especially male) who give a history of low back pain and stiffness that is worse on waking and eases off during the day. Associated features may include a history of psoriasis, inflammatory bowel disease or uveitis. The diagnosis can be confirmed by finding evidence of sacroileitis on x-ray of the pelvis and other typical radiological features (syndesmophytes, vertebral squaring and erosions) on spinal radiographs. Other suggestive features on laboratory tests include an elevated erythrocyte sedimentation rate or C-reactive protein level.[1]

Patients with ankylosing spondylitis require full doses of nonsteroidal anti-inflammatory drugs (NSAIDs) to give adequate control of their symptoms and these should be given long term. Indomethacin (150 mg/day) or naproxen (1500 mg/day) are our agents of first choice, but other NSAIDs may also be effective. However, individual patients respond to particular NSAIDs differently and the final choice of agent should be based on the personal responses of each patient.

Simple analgesics may also be required if pain control is inadequate with NSAIDs alone. An equally important part of management is the institution of an exercise programme, to prevent the development of flexion deformity of the spine. Rarely, surgical treatment (multiple osteotomies of the vertebral bodies) may be indicated in patients with severe fixed flexion deformities of the spine.

5. Osteoporotic Fracture

Osteoporotic fracture is suspected when there is a history of acute back pain in the thoracic or lumbar regions. The pain may be very severe and often radiates laterally to the anterior chest wall. Some patients, who presumably have intermittent episodes of minor vertebral collapse, may not identify an acute episode, but rather, present with a more gradual onset of pain. Many fractures are unassociated with trauma, but close questioning may reveal a precipitating event such as lifting or bending.

Physical examination may be unremarkable, but thoracic kyphosis is common in patients with multiple fractures. The diagnosis is confirmed by finding generalised osteopenia on x-ray with anterior wedging or central collapse of one or more vertebrae. In all cases, thyrotoxicosis and myeloma should be excluded by the appropriate laboratory investigations. If a bone scan is performed (it is

seldom necessary in uncomplicated cases) the typical pattern of a linear area of increased uptake in one or more vertebrae may be seen. Bone mineral density measurements, although not necessary for diagnosis, may be of value in assessing the response to treatment.

Patients with osteoporotic fracture need adequate analgesia in the acute stages with opioids if necessary. A short period of bedrest may also help, but patients should be mobilised as soon as possible since prolonged immobility makes things worse in the long term by increasing the rate of bone loss. Pharmacological therapy to increase bone mass is indicated in all patients with osteoporotic fracture. Many agents have been found to increase bone density in osteoporotic patients including calcium supplements, fluoride, vitamin D analogues, calcitonin, hormone replacement therapy and anabolic steroids.[3,4] However, only for cyclical etidronic acid (etidronate) is there evidence of benefit in terms of secondary prevention of vertebral fracture. The role of etidronic acid in the treatment of osteoporotic fracture is discussed in more detail in the section on bisphosphonates (see section 8.11).

6. Osteolytic Metastases

This is suspected when there are signs, symptoms or a previously established diagnosis of neoplasia. Confirmation is by radiographical and/or bone scan examination. Treatment of the underlying tumour with specific anticancer therapy may improve osteolytic bone pain if tumour regression is achieved. In cases where this is not possible (or where antitumour treatment is ineffective) local radiotherapy often gives symptomatic relief. When these measures are ineffective, or only partially effective, patients may require a cocktail of opioids, nonopioid analgesics, NSAIDs and antidepressants.[5] Inhibitors of bone resorption, such as calcitonin and the bisphosphonates, have also been tried with some success in this situation.

7. Paget's Disease

Paget's disease may be suspected on the basis of clinical features (bone deformity and pain), in the presence of elevated serum alkaline phosphatase levels, and the typical radiographical and bone scan abnormalities.[6] Since Paget's disease is often asymptomatic and is frequently associated with other pathology, such as degenerative disc disease and osteoarthritis, a common clinical problem is deciding whether the patient's pain is indeed due to the Paget's disease or not. This is an important distinction to make, since Pagetic pain usually responds to anti-osteoclast therapy (bisphosphonates or calcitonin), unlike pain due to other conditions.[7] The corollary of this is that pain in a Pagetic patient should always first be treated with antiosteoclast therapy rather than analgesics or NSAIDs.

Unfortunately, there are no pathognomonic features of Pagetic pain. However, a useful practical approach is to give a trial of antiosteoclast therapy and monitor the response. If the pain fails to improve despite adequate biochemical control of the disease (i.e. alkaline phosphatase levels return to normal or near normal values), it must be assumed that the pain is non-Pagetic, and treatment should be based on symptomatic relief with analgesics.

8. Pharmacological Management of Back Pain

This section provides an account of pharmacological agents which may be used to treat the symptom of back pain as well as some specific underlying diseases associated with back pain.

8.1 Non-Opioid Analgesics

Paracetamol (acetaminophen) is the standard treatment for transient back pain. It has no anti-inflammatory properties but does not cause the gastric irritation which may be a problem with NSAIDs. Combinations of paracetamol with a fixed dose of opioid, such as codeine or dextropropoxyphene, are available. These are associated with the same adverse effects as opioid (nausea,

vomiting, constipation and drowsiness). These drugs are suitable for short term treatment of moderate pain and are helpful for the patients who are immobilised with acute back pain. The combination of paracetamol, an opioid and bedrest commonly induces constipation, which may necessitate coprescription of a laxative. Straining to defecate with a bad back is quite unpleasant.

8.2 Opioid Analgesics

These drugs are ideal for short term relief of severe pain, but they can cause dependence and tolerance. Opioids are frequently necessary to control back pain associated with neoplastic disease, and may also be required in the short term management of acute osteoporotic fracture.

Opioid adverse effects can be a problem. Constipation is almost invariable and may require coprescription of a laxative particularly in elderly patients. Larger doses cause respiratory distress and hypotension. Morphine and pethidine (meperidine) are valuable for patients admitted to hospital with acute back pain. Diamorphine (heroin) is a μ-receptor agonist and is seldom indicated except in severe pain due to osteolytic lesions. Buprenorphine (sublingual) is a partial agonist and is very useful for patients with severe acute back pain causing immobility. In instances where non-opioid analgesics are not adequate, buprenorphine 200 to 400μg every 8 hours for a few days can provide great pain relief. Nausea is generally not a problem for resting patients.[8] It is a good maxim that the patient who visits the doctor with back pain can gain satisfactory relief by non-opioid analgesics, but if the doctor has to visit the patient then a partial agonist like buprenorphine is required.

8.3 Nonsteroidal Anti-Inflammatory Drugs

NSAIDs have analgesic activity similar to that of paracetamol. A wide variety of these drugs is now available and they are appropriate for a few days in combination with analgesics for acute back pain, and during a short exacerbation of chronic low back pain. The difference between the anti-inflammatory activity of different NSAIDs is highly variable, but a more important factor is the considerable difference in individual patient response. It is often necessary to find which drug suits a particular patient. Long term therapy should be avoided if possible in elderly patients with osteoarthritis because of the risk of adverse gastrointestinal effects.

Long term NSAID therapy is required when back pain has a significant inflammatory component. Such a situation occurs in ankylosing spondylitis, which responds poorly to simple analgesics. In our experience, full dosages of potent NSAIDs such as indomethacin (up to 150 mg/day) or naproxen (up to 1500 mg/day) are required in ankylosing spondylitis, although other NSAIDs may also be effective in particular patients. However, the literature is conflicting about the benefits of NSAIDs in mechanical back pain.[9] Furthermore, the nociceptive source of back pain of mechanical origin has not been shown to be inflammatory. Some clinicians, however, in spite of slender evidence, choose to give NSAIDs.

8.4 Muscle Relaxants

Painful muscle spasm is sometimes associated with acute low back pain, and a muscle relaxant in combination with an analgesic can give symptomatic relief.[10]

8.5 Antidepressants

Antidepressant drugs can elevate mood, and may increase pain tolerance in depressed patients reducing chronic low back pain.[11] Amitriptyline is more effective at relieving back pain than other antidepressants, but also has more adverse effects such as dry mouth, constipation and drowsiness. The analgesic effect of antidepressants is distinct from the antidepressant effect, occurring at lower doses.[11] A typical dosage regimen for amitriptyline would be 10 to 25mg at night gradually increasing up to 100 to 150mg until an effect is seen or adverse effects occur.

Other antidepressant drugs such as dothiepin (dosulepin), doxepin, clomipramine, desipramine may also be tried, but amitriptyline has the best

documented analgesic actions.[5,11] The onset of analgesia with antidepressants may be delayed for up to 3 months. Low dose tricyclic antidepressants may enhance the analgesic effects of transcutaneous nerve stimulation.

8.6 Anxiolytics

Diazepam has a place for very short term treatment of back pain when a definite anxiety state is a significant factor in the back pain problem. It should be given in the lowest dose for the shortest time.

8.7 Antiepileptic Drugs

Antiepileptic drugs such as carbamezapine, phenytoin and clobazepam have been used for neuropathic 'lancinating' (i.e. shooting or darting) pain such as trigeminal neuralgia.[12] They are used in the same doses as for epilepsy. Therapy should be continued for 6 to 8 weeks before they are discarded as being ineffective.

8.8 Corticosteroids

These are administered locally as an epidural injection or into the facet joints. In the epidural space they are indicated when there is root pain, but we wait for adequate clinical trials to confirm their efficacy. There is some evidence that the patient with an acute disc protrusion can be managed conservatively and avoid surgery with epidural corticosteroids.[13] Similarly, patients with root entrapment syndrome from root canal stenosis seem to benefit. Furthermore, in postdiscectomy syndrome, where epidural scarring may contribute to back and leg pain, corticosteroid injections may often be beneficial.[14] However, there are few trials to confirm that this procedure is any better than doing nothing. Similarly, the therapeutic benefits of injections into facet joints have yet to be proved. Corticosteroids should not be used in back pain due to osteoporosis, since they may increase bone loss.

8.9 Colchicine

Several studies have shown that oral and intravenous colchicine produce clinical improvement in patients with discogenic pain. However, a double-blind trial[15] did not demonstrate any statistically significant difference between colchicine and placebo.

8.10 Calcitonin

Calcitonin has many effects. It produces capillary vasodilatation of the skin and the vessels of the cauda equina.[16] It reduces the blood flow in bone, and reduces bone resorption. The latter effects make calcitonin of particular value in treating conditions where back pain is associated with increased osteoclastic bone resorption. Such conditions include Paget's disease,[17] osteolytic bone disease and osteoporosis.[18] Calcitonin has been shown to reduce bone pain in Paget's disease.[19] A beneficial effect on pain in patients with osteolytic metastases has also been claimed, although most observations have been anecdotal.

The beneficial effects of calcitonin are probably due to reductions in bone resorption and bone blood flow.[17] Calcitonin may also improve back pain by a central analgesic effect. In some patients with spinal Paget's disease, calcitonin has improved back pain as well as their paraparesis.[19] This prompted us to use calcitonin for patients with spinal stenosis who did not have Paget's disease. We observed that about 40% of over 100 of these patients with neurogenic claudication responded with a reduction in leg pain and an increase in walking distance.[20] Calcitonin 100U was administered subcutaneously 4 times a week for 4 weeks. We have not been able to prove that this is more than an analgesic effect nor even a placebo response,[21] but patients will often have a dramatic improvement in their walking ability 2 weeks after starting calcitonin.[20-23]

8.11 Bisphosphonates

These agents are inhibitors of osteoclastic bone resorption and, like calcitonin, have been mainly

used in the treatment of conditions associated with accelerated bone resorption such as Paget's disease, osteolytic metastases and osteoporosis.[24] The main advantages of the bisphosphonates over calcitonin are their greater potency, longer duration of action, ability to be given by the oral route and lower cost.

Three bisphosphonates are currently available for routine clinical use: etidronic acid (ethane-hydroxy-1,1, bisphosphonate), pamidronic acid (pamidronate, 3-amino-1,1-hydroxyprolylidene bisphosphonate) and clodronic acid (clodronate, dichloromethylene bisphosphonate). All have inhibitory effects on osteoclast activity.[24]

In Paget's disease, all 3 agents have been shown to be effective by the oral and intravenous routes, both in terms of biochemical response (decreased alkaline phosphatase and urinary hydroxyproline levels) and pain relief.[7] In 1 case report, clodronic acid was also found to reverse paraparesis in a patient with spinal Paget's disease.[7] Although pamidronic acid and clodronic acid are used in preference to etidronic acid in many specialist centres for the treatment of Paget's disease,[25] only etidronic acid is currently licensed for treatment of Paget's disease in the UK and US. A typical regimen is 5 mg per kilogram bodyweight per day (typically 400 mg/day) orally for 3 months, depending on the biochemical response.

In resistant cases, etidronic acid has been successfully combined with calcitonin. Etidronic acid must be used with caution in the treatment of Pagetic patients with deformities or osteolytic lesions of the long bones, since a mineralisation defect (osteomalacia) can occur and increase the risk of pathological fracture.[16]

Bisphosphonates have also been used in the treatment of osteolytic metastases. Both clodronic acid and pamidronic acid have been used in the management of bone lesions associated with cancer.[27,28] Evidence to date suggests that both agents slow, but do not prevent, the progression of osteolytic lesions in this situation when given on a long term basis by the oral route. They have also been associated with reductions in the need for radiotherapy and chemotherapy and a modest improvement in bone pain.[27,28]

In a recent large study,[28] oral clodronic acid was found to reduce the rate of development of vertebral fractures in breast carcinoma. Anecdotal reports and a randomised placebo-controlled study suggest that the 'analgesic' effect of bisphosphonates seems to be enhanced by intravenous administration, although the mechanism by which bisphosphonates improve bone pain in this situation is unclear. Although the data on bone pain and progression of bone lesions with bisphosphonates are encouraging, neither clodronic acid nor pamidronic acid is currently licensed for this indication in the UK or US.

Bisphosphonates are of value in the treatment of osteoporotic fractures. Two large randomised studies have confirmed that etidronic acid, when given as a cyclical regimen alternating with calcium (2 weeks etidronic acid, 11 weeks calcium), is of value in the secondary prevention of osteoporotic fractures in patients with postmenopausal osteoporosis.[29,30] Patients treated with cyclical etidronic acid frequently report a significant improvement in bone pain. However, whether this is a specific effect or simply related to healing of the fracture that commonly precipitates the patients' referral for specialist advice is unclear.

Etidronic acid is well tolerated.[29,30] However, there is a theoretical risk that sustained inhibition of bone remodelling which results from the above treatment may increase bone fragility as a result of impaired healing of microfractures. Because of this theoretical risk, the current recommendation is that cyclical etidronic acid should be continued for a maximum of 3 years. Other bisphosphonates have been used in the treatment of osteoporosis, but long term experience is limited.

8.12 Chymopapain

This lysing agent is used in the treatment of disc herniation.[31] It is not a treatment for back pain but for root pain as an alternative to surgery. It enjoyed a decade of popularity,[32] but because of painful muscle spasm that can accompany intradiscal in-

jection, and the occasional instances of anaphylactic shock, cerebral haemorrhage and paraplegia, many of the original advocates have been directed to other percutaneous techniques for disc symptoms.[1] Encouraging long term results are giving chemonucleolysis a renaissance,[33] but most feel that surgical discectomy is still the gold standard.

9. Conclusions

Since back pain has several pathologies, patients must be carefully assessed to determine the cause. The symptoms may be treated with analgesics (e.g. paracetamol), anti-inflammatory agents and/or muscle relaxants. NSAIDs are often indicated for long term treatment of back pain of inflammatory origin, such as ankylosing spondylitis.

More severe pain may necessitate opioid treatment, but the dependence and tolerance risks of this therapy must be considered. Hypnotics may be needed to assist sleep, but should be tapered and withdrawn as soon as possible. Excessive bedrest also must be avoided.

With its effects in reducing bone resorption and bone blood flow, and possible central analgesic effects, calcitonin has been a successful treatment of patients with Paget's disease, osteolytic bone disease and osteoporosis. By reducing osteoclastic bone resortion, bisphosphonates have shown efficacy in Paget's disease, osteolytic metastases and osteoporotic fractures.

Tricyclic antidepressants, anxiolytics, antiepileptic agents, corticosteroids, colchicine and chymopapain may also be used in patients with back pain under certain circumstances.

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Correspondence and reprints: *Professor R W Porter*, University of Aberdeen, Department of Orthopaedics, Medical School, Foresterhill, Aberdeen, AB9 2ZD, Scotland.

Journal of Orthopaedic Rheumatology 8, 75–78 (1995)

The sock test for patients with disc protrusion

S.R. Murali,* I.A.C. Lennox and R.W. Porter

Department of Orthopaedics, University of Aberdeen, Aberdeen, UK

Patients with disc protrusion often complain that they have difficulty in putting on their socks, especially when getting up in the morning. The clinical significance of this symptom has been investigated in patients with symptoms of low back pain radiating down one leg to the ankle. Their ability to reach towards their foot with flexed hip and knee was compared with straight leg raising (SLR) and with range of spinal flexion.

There was a significant correlation between the sock test and SLR ($p = 0.00004$) in patients with symptomatic disc protrusion, confirmed by CT scan or myelography.

Keywords: disc protrusion, straight leg raising, spinal flexion, sock test.

Introduction

Low back pain radiating down the leg is a common symptom in clinical practice. There are several causes, one of the commonest being a disc protrusion affecting a nerve root. However the clinical diagnosis is not always straightforward. We have noticed that several patients with sciatic pain also complain of inability to put their socks on especially in the morning and have attempted to investigate the diagnostic significance of this symptom.

Method

Patients with low back pain radiating down one of their legs in a root distribution below the knee, severe enough to be admitted for bed rest in the orthopaedic department, were examined in this study. They had a CT scan or myelogram of the lumber spine and had at least 24 h bed rest prior to being examined.

They were asked to hold a sock in both hands and attempt to put it on, first on the foot of the asymptomatic leg and then on the painful leg. The test was recorded first in the sitting and then in the supine posture. They were asked to flex the hip and knee and draw their leg towards them between the two hands (Figures 1 and 2). The distance reached by the hands, i.e. to the lower third of the thigh, to the knee, to the upper third, middle third, or lower third of the leg, to the ankle, to the dorsum of the foot or to the toes was recorded (the 'sock test').

Straight leg raise (SLR) was recorded using a precision oil-filled goniometer (Rippstein, plurimeter-V). One of the authors blind to the goniometer reading held the goniometer on the anterior border of the lower tibia and performed the SLR to the maximum permitted level while another author recorded the reading blindly. The SLR was carried out three times on each side and the average taken.

Spinal flexion was measured using a kyphometer (Strauman Ltd AZB CH-4802) placed between the upper thoracic vertebrae and the sacrum (Salisbury and Porter, 1987).

In a separate group of eight patients with similar complaints, inter- and intra-observer variability of the sock test was carried out. The results are shown in Table 1 and confirm the reproducibility of the test.

Results

Of the 36 patients examined, 14 could not put on their socks when sitting up and nine patients could not do this when lying down. There were 20 patients with a definite disc protrusion on CT scan or myelogram and there were 16 who did not have a disc protrusion. The results of the sock test, the SLR and of the myelogram or CT scan are shown in Table 2.

In the 20 patients with CT or myelographically confirmed disc protrusion, there was a Pearson correlation coefficient of $r = 0.784$ between the sock test supine and SLR ($p = 0.00004$) (Table 3 and Figure 3). This was not so well correlated when the patients were sitting up ($r = 0.55$), but it was still significant at the 0.01 level.

*Correspondence should be addressed to: Dr S.R. Murali, FRCSEd, Department of Orthopaedics, Medical School Buildings, Foresterhill, Aberdeen AB9 22D, UK; fax: 01224 685373.

Figure 1. Demonstrates the sock test in the sitting position. Patient is able to reach to the middle third of the leg.

Table 1. Results of intra- and inter-observer variability of the sock test

	Results of observer variability of sock test			
Patient	*Observer 1*		*Observer 2*	
	Attempt 1	*Attempt 2*	*Attempt 1*	*Attempt 2*
1	9	9	9	9
2	4	4	3	3
3	9	9	9	9
4	8	8	8	8
5	5	6	6	6
6	7	8	8	8
7	4	4	4	4
8	5	5	5	5

Subsequent references to the sock test will refer to the test being carried out in the supine position.

There was no relationship between the sock test and lumbar flexion, nor between SLR and lumbar flexion (Table 3).

In the 20 patients with disc protrusion there were two distinct groups. 12 could reach at least to their ankle, and their SLR was more than 40°. Eight patients could not reach to their ankle, and their SLR was less than 40° (Figure 3). The 16 patients with root pain due to other causes, however, showed no correlation between the sock test, SLR and lumbar flexion (Table 3). Six of these patients with a negative CT scan or myelogram could not reach to the lower third of the tibia but they had SLR of over 40°. Figure 4 shows that all the patients with good SLR and poor sock test results (greater than 40° SLR and not be able to reach the ankle), did not have a disc protrusion. There were no patients who had a poor SLR and yet a good sock test (less than 40° SLR and yet able to reach to the ankle or distal to the ankle).

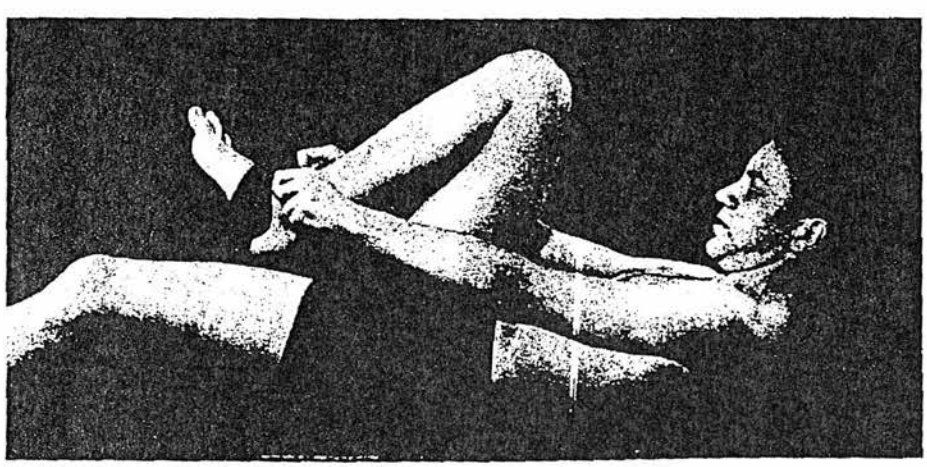

Figure 2. Demonstrates the sock test in the supine position. Patient is able to reach to the ankle.

Table 2. Results of straight leg raise (SLR), sock test (lying down) of the affected leg and CT scan/myelogram in 36 patients

SLR (°)	Sock test (see key)	CT scan/myelogram	
		Findings	Level
62	7	+	L 3,4
5	1	+	L 4,5
36	4	+	L 4,5
38	3	+	L 4,5
42	8	+	L 4,5
40	5	+	L 4,5
64	9	+	L 4,5
68	6	+	L 4,5
70	9	+	L 4,5
22	3	+	L5, S1
28	2	+	L5, S1
28	5	+	L5, S1
32	3	+	L5, S1
42	9	+	L5, S1
42	8	+	L5, S1
46	8	+	L5, S1
64	9	+	L5, S1
66	8	+	L5, S1
72	9	+	L5, S1
48	9	+	L5, S1
20	2	–	
28	1	–	
30	1	–	
32	3	–	
48	8	–	
50	8	–	
50	9	–	
54	3	–	
56	9	–	
58	4	–	
58	9	–	
58	3	–	
60	2	–	
60	2	–	
60	8	–	
70	2	–	

Key:
1 = able to reach the lower third of the thigh; 2 = able to reach the knee; 3 = able to reach the upper third of the leg; 4 = able to reach the middle third of the leg; 5 = able to reach the lower third of the leg; 6 = able to reach the ankle; 7 = able to reach the dorsum of the foot; 8 = able to reach the toes; 9 = able to put the socks on.
+ = disc protrusion present; – = disc protrusion absent.

Discussion

The good correlation between the sock test and SLR in patients with disc protrusion suggests that there may be a similar pathophysiological mechanism for both these signs. The SLR involves flexion of the straight leg at the hip and is considered to be a root tension sign. The sock test, however, involves flexion of both hip and knee and does not apply distal tension on the nerve root. When putting on the socks there may be proximal tension on

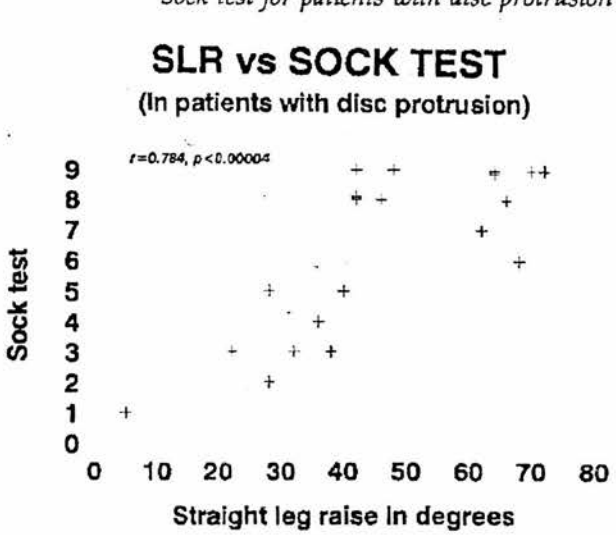

Figure 3. Correlation between SLR and sock test in patients with disc protrusion.

Table 3. Results of correlation between straight leg raise (SLR), sock test and spinal flexion in 36 patients

	Pearson correlation between		
	Sock test and SLR	Sock test and spinal flexion	SLR and spinal flexion
20 patients with disc protrusion	$r = 0.784$ $p < 0.00004$	$r = 0.397$ p = NS	$r = 0.283$ p = NS
16 patients without disc protrusion	$r = 0.32$ p = NS	$r = 0.11$ p = NS	$r = 0.51$ p = NS

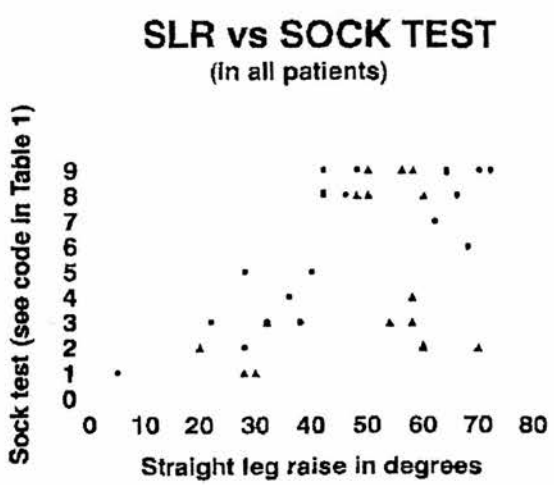

Figure 4. Results of SLR vs sock test in all patients. ●, with disc protrusion; ▲, without disc protrusion.

the nerve root and the dura as the spine and neck are flexed. The greater difficulty which some patients experienced putting their socks on when sitting up compared with when lying down, may be related to the raised intra-abdominal pressure or to the increased hydrostatic pressure affecting the disc bulge.

In this population, it was not possible to identify patients with disc protrusion either by the SLR or the sock test alone. Neither was it possible to use both tests in combination to predict a positive disc protrusion on CT or myelography because some patients with positive sock test and poor SLR did not have a protrusion. However no patient was found to have a protrusion who could not reach to the ankle and yet had a SLR greater than 40°. Neither was there a patient with a protrusion who could reach to the ankle or distal to the ankle and had a SLR less than 40° (Figure 4). As a negative predictor therefore, the sock test has value. Thus a discrepancy between the tests was not compatible with disc protrusion.

The discrepancy we observed in patients without a disc protrusion may be explained by the sock test being totally subjective. The SLR, however, is to some degree influenced by the examiner. It is possible that some of the patients with a poor sock test yet good SLR had an abnormal response to pain.

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(*Accepted for publication 7 March 1995*)

J. R. Coll. Surg. Edinb., **40,** June 1995, 200–202

Orthopaedic section

Review

Pathology of symptomatic lumbar disc protrusion

R. W. PORTER

Department of Orthopaedics, University of Aberdeen, UK

There has been considerable advance in our understanding of lumbar disc protrusion. Contrary to popular belief, the healthy lumbar disc is remarkably resilient to trauma. Its nutrition is the key to failure. A fissure alone will not predispose a disc to protrusion, unless a free fragment also develops. The first symptoms of protrusion are often dramatic (the free fragment is displaced posteriorly), but this has been preceded by a long period of symptomless pathology. Protrusion is often inevitable: an incident and not an accident. Space in the vertebral canal is a major factor determining whether there is nerve root compromise. Mediators from the nucleus cause root inflammation and root tension signs. These inflammatory changes and the symptoms can resolve even though the protrusion remains. Imaging to identify the level, and surgery, are required only when conservative measures fail.

INTRODUCTION

Lower lumbar disc protrusion is the most common spinal condition treated by surgeons, who need to keep abreast of the major advances in understanding its pathophysiology. New concepts on the function of the disc in health and disease, and the significance of the abnormal signs, have important therapeutic and medico-legal implications, and there is a danger that new imaging techniques might outpace surgeons' clinical skills.

PHYSIOLOGY

Morphology

The remarkable structure of the intervertebral disc makes the spine both supple and strong. When subjected to axial loads, the disc is stronger than the vertebrae. Bone will fracture before the disc fails. The radial ply design of the annulus is responsible for its strength. Each layer has parallel fibres at approximately 30 degrees to the horizontal, but in the opposite direction to the adjacent layer. When the disc is loaded the laminated collagen is in tension, and the annulus merely bulges. The large molecules of proteoglycan in the nucleus attract water, achieving a balance with the spine's hydrostatic pressure. In health this is a very strong system.

However, the collagen fibres fail in more than three degrees of disc torsion[1]. One layer of the annulus will deform beyond its elastic limit. This is normally restrained by the sagittal orientation of the apophyseal joints, and by the splinting effects of the spinal muscles and the intra-abdominal pressure.

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Nutrition

The disc is the largest avascular structure in the body. The cells in the disc turn over collagen and proteoglycan, and must receive nutrients and eliminate metabolites. They require an efficient diffusion pathway[2]. It is through the vertebral end-plate that diffusion largely occurs, and its blood supply is therefore critical.

Passive diffusion depends on a balance between the osmotic and hydrostatic pressure of the disc. During recumbency, with a reduced hydrostatic load, fluid is imbibed into the disc, with 17 mm diurnal change in stature. This will benefit small molecular diffusion, and thus a post-prandial slumber may keep the discs healthy.

However, there may also be an active component to disc nutrition. The vessels supplying the end-plate are terminal branches of the lumbar arteries and they have an unusual structure. Many are tortuous, resembling the glomerular vessels, and are probably under autonomic control[3]. Acetylcholine produces a significant increase in end-plate blood flow, suggesting the presence of muscarine receptors in the vascular bed[4]. Nicotine may also have a potent effect on end-plate blood flow. Studies of identical twins, only one of whom smokes, have shown significant reduction in disc hydration in the smoking twin[5]. Smoking is also known to be a risk factor for back pain. There is also a high incidence of back pain in occupations subject to vibration[6–8], which may also affect end-plate circulation and disc nutrition.

Disc failure and pathological change may therefore result more from impairment of disc nutrition than from mechanical causes.

PATHOLOGY

Mechanical stress

Disc nutrition cannot be the whole explanation of disc failure, otherwise one would expect all the lumbar discs to be equally affected by pathological change. MRI studies, however, frequently show one or two degenerate discs, while adjacent discs are well hydrated. It is probable that poor nutrition makes a disc vulnerable to damage by either peak loads or repetitive minor loads. The disc begins to fissure, and once damaged, because of the low metabolic rate, repair is unlikely to keep pace with further pathological change[2]. Fissures progress throughout the nucleus to the periphery of the annulus. Of clinical significance is the double fissure, which produces a 'doughnut' appearance, with the formation of a loose fragment.

Disc protrusion

It is difficult in the laboratory situation to produce a protrusion by applying an axial load to an isolated spine. Enormous loads

in non-physiological hyperflexion can rupture a healthy disc, but this probably does not occur in life. Even if a fissure is artificially created in a disc, it is not possible to produce a protrusion by loading the segment. However, if a fragment of disc material from another disc is introduced into that fissure, it takes only minimal physiological loads (1 kN) to produce a protrusion[9]. The posterior annulus will bulge and sometimes rupture as the free fragment is extruded, much like a pea squeezed out of a pod. If the laboratory situation is true to life, it is probable that a disc protrusion cannot occur unless there is both a disc fissure and also a free or almost-free fragment. Once the fragment has formed, it does not take much of a load to cause a protrusion, or even a complete rupture of the annulus.

Medico-legal implications of the 'fissure and fragment'

Many patients with symptomatic disc protrusion can recollect an acute onset to their first symptoms—more than patients with other back pain syndromes.[10] They remember an incident when they twisted or bent forwards, perhaps carrying a load, which they describe as an 'injury'. The first pain can be agonizing. It is imprinted on the memory, and if it occurred at work is thought worthy of compensation. However, the first symptom is merely the end-result of a long-standing symptomless process, which would inevitably have occurred whenever the spine was subject to this degree of load. Even lesser repetitive loads could cause the protrusion. Thus when the first symptoms occur when lifting, twisting or bending they are an incident in a long-standing process, and not an accident. It would probably not have been long before a similar load produced a similar problem.

The vertebral canal

The size and the shape of the vertebral canal are critical in the development of symptoms from a disc protrusion. If the vertebral canal is sufficiently wide, the nerve roots of the cauda equina are spared from compression. The posterior annulus is stretched, causing back pain and/or referred pain, but there is no root pain. However, if the vertebral canal is shallow in the sagittal plane, or trefoil in shape, the nerve root is rapidly compromised. Fifty per cent of patients whose symptomatic disc protrusion causes sciatic pain have vertebral canals in the bottom tenth percentile of the normal population.[11] Patients with wider canals may have disc protrusion, but they escape disabling root symptoms. By 60 years of age, one-third of the population has sustained a disc protrusion, but most are symptomless.[12] This is because most of them are fortunate enough to have a wide vertebral canal.

Inflammation

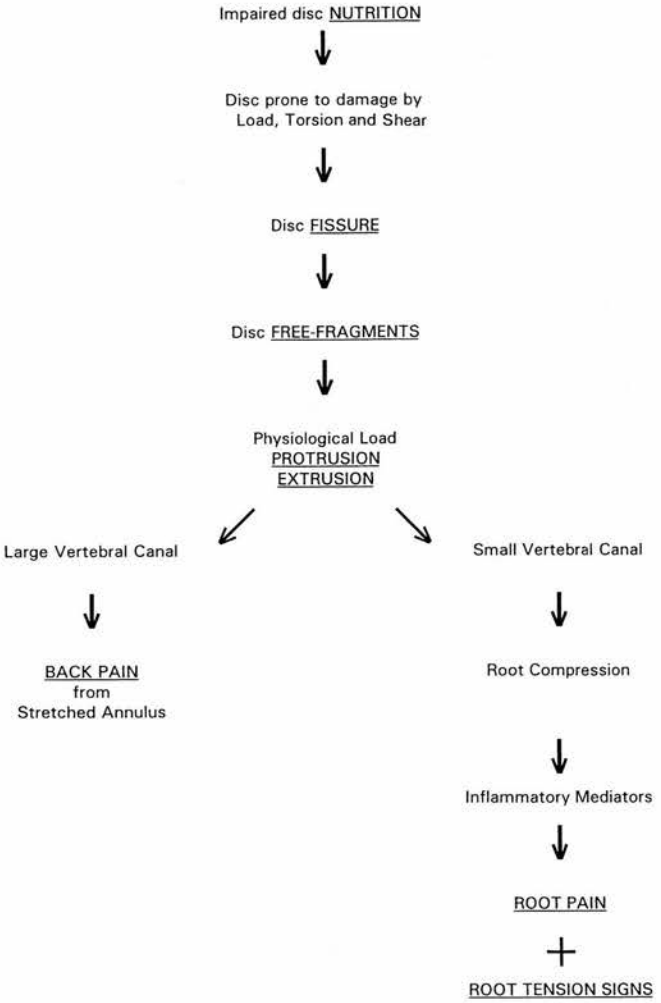
Disc protrusion with mechanical compression of the nerve root does not explain the whole mechanism of root symptoms. Nerve roots are frequently compressed by degenerative bone, capsule, or ligamentum flavum, without developing root-tension signs. Similarly, root-tension signs vary with time in spite of a constant protrusion size on imaging. Furthermore, straight leg raising recovers only slowly after surgical removal of a disc protrusion, even though the compression is relieved. The phenomenon of the root-tension sign can best be explained by an inflammatory process mediated by the nucleus pulposus on a compressed root.

In a pig model, nucleus pulposus has a profound effect on the cauda equina, inducing chemotactic signals that attract inflammatory cells.[13] It is probable that these inflammatory changes are responsible for the acute root symptoms, and for the root oedema frequently seen on MRI for many months after a successful discectomy. The pathological process responsible for symptomatic disc protrusion is summarized in Fig. 1.

SYMPTOMATOLOGY

Trunk list

The only objective sign that is pathognomonic of disc protrusion is a trunk list. Other signs can be influenced by subjectivity, but the trunk list is beyond the patient's control. Its mechanism is unknown. It is present in about 50% of patients with a protrusion. It is gravity induced, being abolished when the patient lies down or hangs from a bar. Twice as many patients list to the left than to the right. It is unrelated to the side of the sciatica, to the level or the side of the lesion, and it is unrelated to the topographical position of the disc—axillary, anterior or lateral.[14] It carries a poor prognosis for



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Bladder dysfunction is the only absolute indication for early surgery in a patient with disc protrusion. Surgery is unnecessary when straight leg raising is better than 50 degrees, provided the patient is prepared to wait for recovery. These patients ultimately do as well treated conservatively as by operation.[17] However, many are impatient and early expeditious surgery will relieve the root pain. Risks are remote but significant.

Imaging

Only when the decision to operate has been made is imaging requested, not to diagnose the condition but to identify its level. The choice of imaging depends on resources, but MRI is the method of choice. Using imaging as a 'fishing trip' to make a diagnosis is poor practice. It is likely to confuse the issue and reveal protrusions that may be clinically irrelevant. However, we can now anticipate excellent results with improved assessment, precise imaging and expeditious surgery.

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J. R. Coll. Surg. Edinb., **40,** June 1995, 200–202

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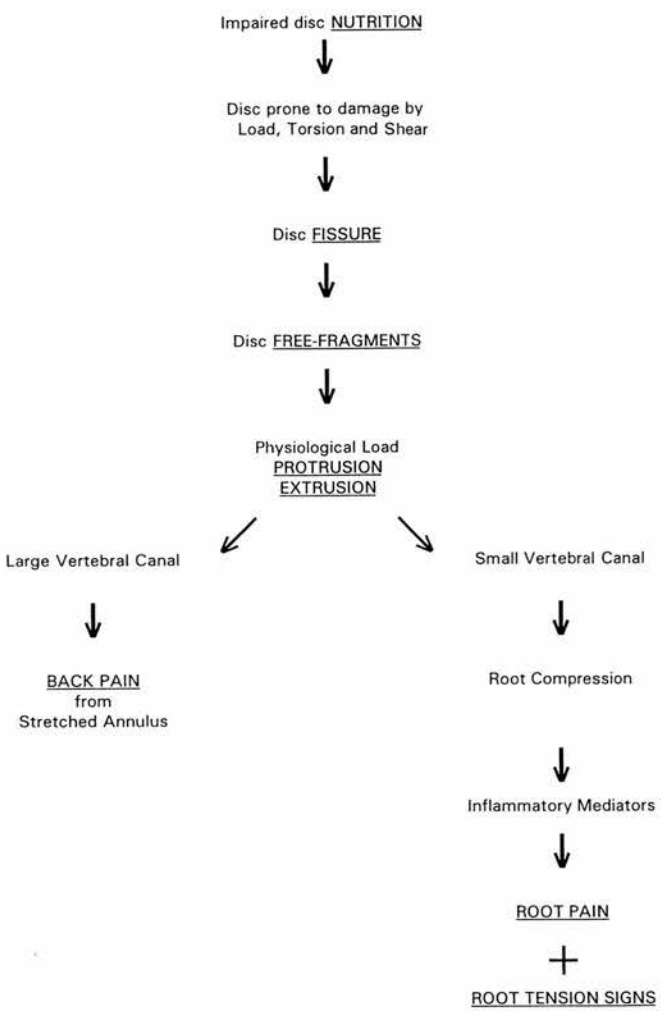

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Eur Spine J (1995) 4:145–147

European Spine Journal

Sudden onset of back pain*

K. M. K. Varma[1] **and R. W. Porter**[2]

[1] 7/2 Richmond Road, Bungalore 560025, India
[2] Department of Orthopaedics, University of Aberdeen, UK

Summary. Patients attending a back pain clinic completed a structured interview, with particular reference to whether onset of the first episode of back pain was sudden or insidious. They were classified into four diagnostic groups. A significantly higher proportion of patients who had experienced a sudden onset of pain suffered from sciatic pain with positive root tension signs than was the case with patients whose pain had started more insidiously (50.8% vs. 20.6%; $P < 0.001$). These patients were also more likely to be male, to have been lifting or twisting with a weight, and to have been confined to bed or hospitalised, and have undergone manipulations, than patients with insidious onset of their pain.

Key words: Backpain – Onset – Disc

Back pain due to mechanical derangement of the spine may be associated with a specific event and therefore occur suddenly, or it may develop slowly and insidiously. In the hope of understanding the significance of these two types of onset, we compiled a structured history of patients attending a back pain clinic. We identified patients who recollected that their first episode of back pain was associated with a specific event and was sudden in onset, and compared their history and examination findings with those of patients who said that their initial back symptoms were unrelated to any event specific and that pain had developed slowly.

Methods

Patients attending a secondary referral hospital-based back pain clinic were assessed by means of a structured interview and examination by one clinician (R.W.P.). The data were analysed by a second doctor (K.M.K.V.). The interview included questions about the first episode of back pain: Was it associated with a specific event and therefore sudden? When and where did it occur, at what time of day, and what was their posture at the time? What were their first symptoms? What initial and subsequent treatment did they receive? What was their occupation at the time? The patients were also asked whether or not the pain initially or subsequently affected the leg below the knee in a nerve root distribution with symptoms incriminating a single nerve [3]. The presence of root tension signs at the time of examination were noted (straight-leg raising using a standard technique [4] of 70° or less, positive bowstring sign, positive Lasague sign. Neurogenic claudication was defined as no leg pain at rest, but leg pain below the knee when walking [5] that limited walking to less than 500 m.

Four diagnostic goups were identified:

Group 1: Pain below the knee in a root distribution with positive tension signs
Group 2: Pain below the knee in a root distribution without root tension signs
Group 3: Neurogenic claudication
Group 4: Back pain with or without referred pain, not below the knee

Exclusion criteria were back pain of nonspinal origin, inflammatory back pain, tumour, infection, bony trauma and peripheral neuropathy.

Comparisons were made between 500 consecutive patients seen over a 27-month period who recollected that their first onset of back pain was sudden and associated with a specific event, and 300 patients seen in the same period whose onset of back pain was slow and insidious. The distribution of these two types of patients across the four diagnostic groups was compared. Statistical analysis was performed using Student's *t*-test.

Results

Compared with the patients whose pain developed insidiously, a significantly higher percentage of the patients who had experienced a sudden onset of pain were in diagnostic group 1, with positive route tension signs, and a significantly lower proportion were in groups 2 and 4 (Table 1).

There was a significantly higher proportion of men among patients, who described a sudden onset of their symptoms than among the patients with an insidious onset of pain (63.8% vs. 55.3%; $P < 0.001$). The occupational profiles of the men with different types of pain onset were similar, among the women, there was a higher percentage in unpaid work among those whose onset of back pain was insidious (Table 2). Of the 333 patients with sudden

Received: 29 July 1994
Revised: 25 October 1994
Accepted: 25 October 1994

* Research was carried out at the Department of Orthopaedics, Royal Infirmary, Doncaster, UK

Correspondence to: Department of Orthopaedics, University Medical Buildings, Foresterhill, Aberdeen AB9 2ZD, UK

Table 1. The distribution among four diagnostic groups of patients with sudden onset of their first episode of low back pain and those whose low back pain developed insidiously

Diagnostic group	Sudden onset (n = 500)			Insidious onset (n = 300)			P
	Men	Women	Proportion	Men	Women	Proportion	
1	174	70	50.8%	34	25	20.6%	$P < 0.001$
2	33	15	10.0%	23	35	20.2%	$P < 0.001$
3	21	7	5.8%	12	4	5.5%	NS
4	91	89	33.0%	63	104	53.5%	$P < 0.001$

Table 2. Patients' occupations at the time of the onset of back pain

Occupation	Sudden onset (n = 500)		Insidious onset (n = 300)	
	Men (64%)	Women (36%)	Men (55%)	Women (45%)
Manual	75%	28%	75%	18%
Sedentary	4%	13%	4%	6%
Housewife	–	31%	–	68%
Non-paid/unemployed	12%	18%	7%	4%
Unknown	9%	11%	13%	3%

Table 3. The place where the first episode of pain occurred in 500 patients with sudden onset of low back pain

Place	Men (n = 319)	Women (n = 181)
Work	63%	28%
Sporting activity	11%	12%
Road traffic accident	5%	5%
Shopping	1%	2%
Home	7%	26%
Bathroom	2%	5%
Kitchen	1%	7%
Bedroom	0%	6%
Living room	5%	7%
Garden	10%	4%
Driving (not work)	2%	1%
Stairs	4%	17%
Unknown	12%	27%

onset of back pain whose litigation status was known, only 17.8% had pursued a claim.

Among the patients with sudden onset of pain, 50.6% said that is had occurred at work, 14.2% at home, 11.0% during sporting activity, 5.0% in a road accident and 1.6% while shopping (Table 3). The most common activities at the time of the onset of pain wer lifting a weight (27.8%) and twisting (17.2%). In 23% h of patients, the first experience of pain occurred when they were in a bent position. The most common combined activity was reported as lifting and twisting (29.9%) (Table 4).

The time of day for the onset of pain was accurately described by 38% of the patients whose pain started suddenly, and for 46% of these pain began between 8am and noon.

Most patients with sudden onset of pain first experienced pain in the back (n = 378; 75.6%); only 14 (2.8%) said their pain had started in the leg; 108 patients (21.6%) could not remember where their pain had first started.

The initial management of the two groups is shown in Table 5. More of the patients with an acute onset of pain rested in bed, either at home or in hospital, than did those with an insidious onset of pain (38.2% vs 9.1%, $P <$ 0.001).

Discussion

The first symptoms of back pain can be very severe and frightening, and it is not surprising that many patients clearly remember their first episode of pain, even to the day and the hour. However, it is difficult for the clinicial to know how to interpret this history. If litigation is involved, it might influence the patients' recollections. However, we suspect that this confounding factor was not significant, because financial gain was involved in only 18% of those questioned.

This study suggests that patients attending a hospital back pain clinic who remember their first pain as an acute episode are 2.5 times more likely to have had subsequent root pain with root tension signs and to have had a period of bed rest, than those whose onset of pain was insidious. They were also half as likely to develop root pain without root tension signs and were less likely to have back pain only.

Patients with root tension signs (Group 1) fulfilled clinical criteria of lower lumbar disc protrusion. The preponderance of these patients having an acute onset to their pain is consistent with our understanding of disc pathology, i.e. that a sudden protrusion can occur as a loaded spine is stressed by bending and twisting.

We know that, for many patients with disc protrusion, the first symptom is an event of great significance. For the clinician, however, it is no more than an incident in a pathology which has been silently developing for some

Table 4. The activity at the time of the first episode in 500 patients who had sudden onset of low back pain (some patients cited more than one activity)

Activity	Percentage of patients (n = 500)
Trip falling onto back	6.6
Trip falling onto bottom	4.2
Trip undefined	7.8
Slip onto back	3.0
Slip onto bottom	3.8
Slip undefined	4.6
Bending alone	11.4
Bending with weight	11.8
Lifting weight	27.8
Pushing weight	1.4
Pulling weight	1.6
Kneeling and lifting	0.2
Twist	17.2
Sneeze/cough	1.0
Direct injury	9.0
Jarred back	1.4
Unknown	6.4
Combined activities	**No. of patients (n = 79)**
Trip falling onto back/other direct injury	5
Trip falling onto bottom/other direct injury	1
Trip undefined/other direct injury	2
Trip falling onto back/twist	1
Trip falling onto bottom/twist	4
Trip undefined/twist	1
Slip onto bottom/twist	1
Slip onto back/other direct injury	1
Slip undefined/twist	1
Bend/twist	6
Bend/lifting weight	4
Bend/pulling weight	2
Bend with weight/twist	11
Bend with weight/lifting weight	10
Bend/pushing weight	1
Lifting weight/twist	17
Lifting weight/pushing weight	1
Lifting weight/slip onto back	1
Lifting weight/slip undefined	5
Twist/sneeze or cough	2
Lifting weight/direct injury	2

time. In the laboratory it is difficult to damage a healthy disc by loading the spine. The vertebrae tend to fracture first, unless the spine is hyperflexed [1]. However, if a fissure is created and a fragment is inserted into it, the disc

Table 5. Treatment received (after the first episode of pain) by patients with sudden and patients with insidious onset of low-back pain

Treatment	Sudden onset (n = 500)	Insidious onset (n = 300)
None	8.0%	28.6%
Bed rest at home	30.4%	6.4%
Physiotherapy	35.8%	33.7%
Bed rest in hospital	7.8%	2.7%
Epidural injection	6.0%	9.2%
Jacket	9.2%	3.7%
Corset	26.2%	26.9%
Chiropractic/osteopathy	5.8%	1.7%
Surgery	6.2%	4.75%

will protrude under minimal load, and the fragment will frequently herniate [2]. If this also occurs in vivo, it is probable that the process leading to protrusion starts when a fragment of disc detaches or begins to detach, and then under suitable load that fragment protrudes or herniates, precipitating pain. The episode is acute and severe for the patient, often described as an accident, but it may in reality be only an inevitable incident at the end of a chronic pathological process.

These were some patients fulfilling criteria of disc protrusion who remembered an insidious onset to their pain, and it is possible that, if they did in fact have a protrusion associated. with an acute onset, which is compatible with the pathological concept of sudden displacement of a fragment.

It is interesting that many patients who recollected a gradual onset of pain were in diagnostic Groups 2 and 4. The heterogeneous nature of the pathologies responsible for root pain without tension signs, and for low back pain without leg pain, gives room for speculation about slow onset, but it makes an explanation invidious.

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Eur Spine J (1995) 4:148–152

European Spine Journal

Active treatment programs for patients with chronic low back pain: a prospective, randomized, observer-blinded study

A. F. Bendix, T. Bendix, S. Ostenfeld, E. Bush, and A. Andersen

Copenhagen Back Center, National University Hospital, Copenhagen, Denmark

Summary. Several new studies have indicated that an active approach to patients with chronic disabling low back pain (LBP) seems effective. Some of these studies emphasize the importance of dealing with the patient's total situation in comprehensive multidisciplinary programs – the bio-psycho-social model. However, these programs are expensive. The aim of this study was to evaluate the rehabilitation outcome from three different active programs in terms of: (1) return-to-work rate, (2) days of sick leave, (3) health-care contacts, (4) pain and disability scores, and (5) staying physically active. The subjects included 132 patients randomized to the study, of whom 123 started one of the treatment programs. They had all had at least 6 months of chronic LBP. The patients were randomized into one of three programs: group 1 – a full-time, intensive 3-week multidisciplinary program, including active physical and ergonomic training and psychological pain management, followed by 1 day weekly for the subsequent 3 weeks; group 2 – active physical training, twice a week for 6 weeks, for a total of 24h; group 3 – psychological pain management combined with active physical training, twice a week for 6 weeks, also for a total of 24h. The results presented here are based on data collected 4 months following treatment, which shows an 86% response rate. The initial examination and the follow-up evaluation were performed by a blinded observer. The results show that 4 months after treatment, the intensive multidisciplinary program is superior to the less intensive programs in terms of return-to-work rate, health-care contacts, pain and disability scores, and staying physically active. In conclusion, it seems that although the multidisciplinary program is initially expensive compared to the less intensive programs, the savings in sick pay, early retirement pensions, and health care contacts make it economically worthwhile. Long-term follow-up will show whether this effect continues.

Received: 23 December 1993
Revised: 3 September 1994
Accepted: 28 September 1994

Correspondence to: A.F. Bendix, Copenhagen Back Center, 7521, National University Hospital, Tagensvej 20, DK-2200 Copenhagen N, Denmark

Key words: Low back pain – Rehabilitation – Physical fitness – Psychological pain management – Occupational medicine

Chronic low back pain (LBP) problems have increased tremendously in Western societies during the past decades. In Great Britain, for example, days of sick leave due to back pain have increased tenfold during a 30-year period [29]. In Sweden, work days lost through sick leave and early retirements due to back pain have increased 40 times over the last 30 years [21]. The same tendency has been observed in Denmark [3]. One of the main explanations for the increase of the problem might be a reduced acceptance of (back) pain; due to the high level of technology, many pain conditions can be treated effectively today, and pain can often be reduced or even removed. This makes it more difficult to accept those types of pain that can not be cured.

Another explanation might be the large variety of different medical and paramedical treatments, many of these being passive [29]. This could leave the message: "A back problem is a serious thing and has to be taken care of by others (i.e., experts)." People with even minor back problems may therefore tend to be inactive and leave the responsibility for their back pain to others.

Attitudes to treatment have waved between active and passive during the past decades. Active, intensive treatment with specific back exercises was described by Hoffa as long ago as 1925 [13]. In the 1960s the attitude changed towards being more passive, encouraged in part by the observation that disc pressure is lower when lying down and higher when sitting, standing, and lifting [20]. This may have been interpreted by many professionals as a reason for avoiding loads on the back, because pressure-induced disc degeneration was thought to be one of the major factors in back problems. The treatments in the 1960s and 1970s were therefore dominated by passive modalities: hot packs, ultrasound, massage, injections, etc. This in spite of the fact that passive treatments have no documented long-term benifit to chronic back pain patients [4, 9, 10, 14, 15, 23, 27]. In contrast recent studies

Reprinted from SPINE, Vol. 21, No. 14, July 15, 1996

Printed in the U.S.A.

A Comparison of Methods for Measuring Trunk List

A Simple Plumbline Is the Best

Ian P. McLean, FRCS,* Maureen G. C. Gillan, PhD,† Jennifer C. Ross, MEd,‡ Richard M. Aspden, PhD,† and Richard W. Porter, FRCS†

Study Design. Trunk list was measured using three different techniques to compare accuracy, precision, and ease of use.

Objective. To obtain a reproducible technique for further studies of the nature, cause, and clinical relevance of trunk list.

Summary of Background Data. Gravity-induced trunk list is a clinical sign that is frequently observed in patients with low back pain and has been associated with intervertebral disc lesions.

Methods. Patients with trunk list participated in a comparison of three techniques to determine list magnitude and direction. Paired measurements of trunk list were obtained from each patient using three techniques: a plumbline, a projected shadow, and the 3SPACE Isotrak (McDonnell Douglas Electronics Company, Colchester, VT). In addition, intra- and interobserver reliability of list measurement was assessed by comparison of paired measurements by each of two observers.

Results. List measurements assessed by the plumbline and the projected shadow techniques were not significantly different, but the Isotrak produced data that differed significantly ($P < 0.05$) from both of these techniques. Comparison of intra- and interobserver repeatability of list measurement using the plumbline technique indicated no significant difference between repeated measures by each observer or between two observers.

Conclusions. A plumbline is the most useful instrument for measuring static trunk list, but its limitations and the need for standardization of measurement technique must be recognized. [Key words: intervertebral disc, low back pain, lumbosacral list, observer variation]
Spine 1996;21:1667–1670

Gravity-induced trunk list is a sign that is frequently observed in patients with acute low back pain and has been associated with an intervertebral disc lesion.[7] In the clinical assessment of back pain, Waddell et al[8] advocated quantification of trunk list on the basis that simple inspection identified only gross examples. Using a tape measure as a plumbline to measure the midline deviation of the lower thoracic convexity from the gluteal cleft, they showed that patients could be categorized according to whether the magnitude of the list was more or less than 1 cm.

The clinical diagnosis, management, and scientific analysis of lumbar function and dysfunction require the collection of valid and reliable data.[8] Several studies have highlighted the need to assess the consistency of repeated measures by one observer and by different observers using the same technique.[2,4]

The objective of the present study was to find a reproducible technique for measuring trunk list as a preliminary to further studies of the nature, cause, and clinical usefulness of this physical sign.

Methods

List Measurement. Adult patients (aged 22–64 years) with trunk list at the Orthopaedic Outpatient Clinic at Aberdeen Royal Infirmary gave informed consent to participate in a comparison of three techniques for measuring the magnitude of trunk list. List was defined as the lateral displacement, in millimeters, of a surface marking of the spinous process of T12 from that of S1. Identification and skin marking of the spinous processes were performed with the subjects lying prone. Three techniques were used to determine list magnitude and direction:

1. a plumbline;
2. a shadow projected from a vertical wire onto the skin of the back; and
3. the 3SPACE Isotrak (McDonnell Douglas Electronics Company, Colchester, VT).

Displacement to the left was deemed positive and was chosen to conform with the orientation of the reference axes of the Isotrak.

Seven patients were measured using all three techniques, and an additional five were measured using only the plumbline and the shadow. Twenty-seven patients were measured using the plumbline.

From the *Princess Margaret Rose Orthopaedic Hospital, Edinburgh, the †Department of Orthopaedic Surgery, University of Aberdeen, Aberdeen, and the ‡Department of Physiotherapy, School of Health Studies, Robert Gordon University, Aberdeen.
Support was received from the Sir Jules Thorn Charitable Trust (IPM), London, United Kingdom, and the New Zealand Back Pain Foundation (MGCG), Waikanae, New Zealand.
Acknowledgment date: April 21, 1995.
First revision date: September 5, 1995.
Acceptance date: November 14, 1995.
Device status category: 1.

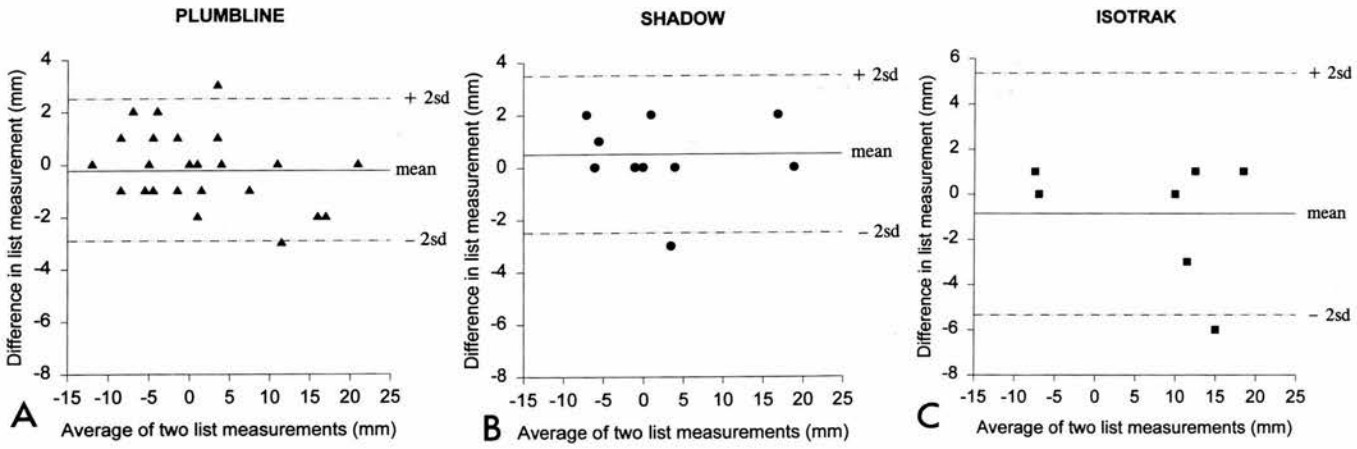

Figure 1. Trunk list was measured using (**A**) a plumbline (n = 27), (**B**) a projected shadow (n = 12), and (**C**) the 3SPACE Isotrak (n = 7). For each technique, the difference between each pair of readings is plotted against the average of each pair.

The plumbline consisted of a brass weight on a thin wire that was held to overlie the line at S1. List was measured as the horizontal distance of the marking at T12 from the plumbline, using a tape measure. To produce a projected shadow, a similar thin wire was attached vertically to a frame. This wire was placed between a point light source and the subject so that a sharp shadow of the wire was projected onto the skin of the back. The position of the frame was adjusted so that the shadow passed through the marking at S1, and the perpendicular distance from the shadow to the marking at T12 was measured using a tape measure. The 3SPACE Isotrak has been used for measuring movements of the lumbar spine.[5] It consists of an electromagnetic device for measuring the position and orientation of a sensor in space. The source and sensor were attached to each subject while prone, thus abolishing the gravity-induced list, and secured with double-sided tape. A molded plastic plate was used to attach the source over the sacrum, and a polystyrene cup and adjustable strap gave additional support to the fixation of the sensor at T12. Data were recorded using a laptop computer with the subjects initially lying prone, to obtain a zero-position, and then in a standing position.

For all three methods, list was measured with the subjects standing comfortably erect with their feet together and arms resting by their sides. Two measurements were made with the subjects performing their pain-free range of lumbar flexion between each reading. List was measured by one observer in the order of Isotrak, shadow, and then plumbline.

Intra- and Interobserver Agreement. Using 14 volunteers, the authors assessed the intra- and interobserver reliability of plumbline measurements. With the subjects lying prone, skin markings were made at S1 and T12. To simulate the presence of trunk list, markings at T12 were laterally displaced from the midline. Two measurements were made on each subject by each of two observers: Observer 1 made one measurement on each subject followed by Observer 2, followed by a repeat measurement by Observer 1 and Observer 2.

In addition, to determine how much of the variation resulted from subject variability, one observer made 10 repeated measurements of markings on a vertical board. Two marks were made on the board at arbitrary lateral displacements of between 5 and 25 mm and separated vertically by about 10 cm.

Statistical Analysis. One-way repeated measures analysis of variance was used for comparison of results using the different techniques followed by pairwise multiple comparison by the Student-Neuman-Keul's method.

Paired measurements from a number of subjects enables the repeatability of the measurement technique to be determined.[1] A plot of the difference between each pair of readings against their mean should have a mean difference of zero; a non-zero result indicates that one reading is affecting the other. The standard deviation of the differences and a repeatability coefficient,[1] defined by the British Standards Institution to be two standard deviations, can be calculated. This implies that 95% of any pair of measurements made by the method under test should differ by no more than this value. Patient data from the plumbline, shadow, and Isotrak methods were analyzed by this method. The agreement between two measurements by one observer (intraobserver) and between two observers (interobserver) were assessed in the same way.[1]

■ Results

Repeatability

Because no standard reference measurement is available to compare the different techniques for accuracy, the repeatability of each technique only can be measured. For the three measurement techniques, the mean and the difference of each pair of measurements were calculated, and the differences were plotted as a function of the

Table 1. Comparison of List Measurement Techniques*

Method of Measurement	Number of Patients	Mean Difference (mm)	Standard Deviation (mm)
Plumbline	27	0.1	1.9
Shadow	12	0.5	1.4
Isotrak	7	–0.8	2.7

* Pairs of observations were made on each patient, and the mean differences and standard deviations were calculated.

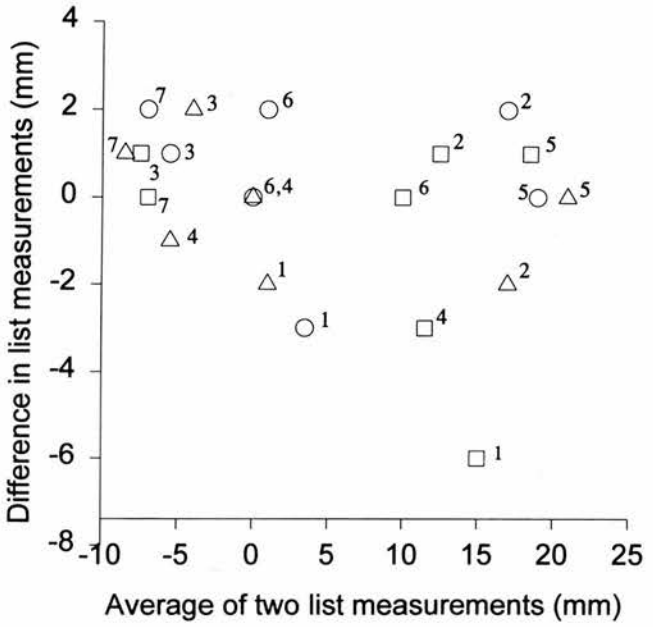

Figure 2. For seven patients two measurements of trunk list were made using all three measurement techniques. For each patient, the average of each pair of list measurements was plotted against the difference between the two measurements. △ = plumbline; ○ = shadow; □ = Isotrak.

mean (Figure 1).[1] The mean of the differences in list measurement and the standard deviation of these differences is given in Table 1. From these data, the coefficients of repeatability can be calculated.[1] Thus for the plumbline method, 95% of the second measurements would be expected to be within 4 mm of the first measurement with corresponding values for the shadow and the Isotrak of 3 mm and 5 mm, respectively.

Comparison of Techniques

The differences in the results obtained using the plumbline and the shadow method were not significantly different. However, the Isotrak produced results that were different from those obtained by the plumbline and the shadow ($P < 0.05$ for both). Trunk list was measured in seven patients using all three techniques, and the data for each patient were plotted as before as the difference *versus* the mean. For good agreement between the three techniques, data points for each patient should be clustered together. Horizontal displacement of the corresponding points in Figure 2 is the best estimate of the measurements. It is apparent that the shadow and the plumbline results are generally separated by little horizontally, whereas the corresponding Isotrak point is considerably displaced. This is especially true for Patients 1, 4, and 6.

Because the results from 12 patients using the shadow and the plumbline showed no significant difference, for the convenience of the patients, the remainder of the study was done using only the plumbline.

Intraobserver Repeatability

From the 14 volunteers, list measurements were made by two observers using the plumbline method. Intraobserver repeatability was assessed by calculating the difference between the pair of readings and their mean for each observer. Plots of the difference *versus* the mean showed no systematic variation, and the mean difference and its associated standard deviation ($x_1 \pm s_1$, $x_2 \pm s_2$) could be calculated for each observer (Table 2).

Pairs of measurements by one observer of 10 separate marks on a board were analyzed in the same way. The mean difference was found to be 1 mm, and the standard deviation of the mean difference was 1 mm, giving a repeatability coefficient of 2 mm.

Interobserver Repeatability

Interobserver repeatability was determined from the means of the paired observations of the two observers. The difference between the two means was plotted against their mean, and again the mean difference and its standard deviation (s_d) was calculated. This is an underestimate of the standard deviation of the differences, and the corrected value is given by $s_c = \sqrt{(s_d^2 + \frac{1}{4}s_1^2 + \frac{1}{4}s_2^2)}$.[1] These results are summarized in Table 2.

■ Discussion

A simple plumbline is the best way of measuring trunk list. There is little to choose in terms of repeatability or precision between the plumbline and the shadow methods, but the former has the advantage of simplicity and ease of use as a suitable bedside test. The set-up and use of the Isotrak is time-consuming, and the authors found that even with careful attachment of the source and sensor erroneous results could not be avoided: for one patient, the Isotrak recorded a list in the opposite direction from that indicated by the other techniques and by simple inspection, and for a second patient, the magnitude measured using the Isotrak was unrealistically large. In preliminary investigations, the authors noted that after the zero position of the Isotrak had been set, movement of the source or sensor by a few millimeters altered the relative orientation of the source and sensor and resulted in the recording of erroneous values for lateral deviation. Therefore, poor agreement between

Table 2. Intra- and Interobserver Repeatability of List Measurement*

Observer	Mean Difference (mm)	Standard Deviation (mm)	Repeatability Coefficient (mm)
Observer 1	−0.1	3.8	8
Observer 2	0.1	2.6	5
Interobserver	−0.3	5.1	10

* Paired measurements of trunk list were made on each of 14 subjects using a plumbline. Repeatability coefficients for each observer and between observers were calculated from the standard deviations of the differences.

the Isotrak and the plumbline and shadow methods may result from displacement of the source or sensor when patients move from the prone position to standing. Comparison of list magnitude and direction measured by each technique suggests there is poor agreement between the Isotrak and the plumbline and shadow methods. However, the Isotrak may prove useful in the clinical assessment of dynamic lateral deviation of the lumbar spine during flexion–extension movements.[5]

In assessing the repeatability of a method, the mean difference between two consecutive measurements made by one examiner or between one measurement by each of two observers would ideally be zero.[1] The authors' data from the comparison of list measurement techniques together with the data from intra- and interobserver differences in list measurement indicate that it is possible to measure trunk list to within 4 mm using the plumbline technique. Their observation that, without a patient, the plumbline measurement technique has a repeatability coefficient of 2 mm suggests that a major inconsistency can be attributed to subject variability. In particular, accurate assessment of trunk list in patients with low back pain may be compromised by their inability to maintain a steady posture or to adopt a neutral position between repeat measurements.

When patients are studied over a period of several weeks, months, or even years, the reliability of measurements is notoriously difficult to assess. The reliability of clinical measurements in orthopedics depends not only on the precision of the measurement technique and patient compliance but on consistency in the skill and measurement technique of the observer(s).[3,9] For example, identification of the spinous processes of T12 and S1 depends on the training and experience of the observer, and inconsistency in measurements can occur if observer assessment techniques have not been standardized.[3,4,6] These factors could be of particular significance in multicenter studies.

In summary, the authors consider that a plumbline is the most useful instrument for measuring a static trunk list, but its limitations and the need for standardization of measurement technique must be recognized.

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Address reprint requests to

Maureen G. C. Gillan, PhD
Department of Orthopaedic Surgery
University of Aberdeen
Aberdeen AB9 2ZD
UK

SPINE Volume 22, Number 2, pp 151–155

An *In Vitro* Study of the Biomechanical Effects of Flexible Stabilization on the Lumbar Spine

55

Tibor Papp, MBChB,* Richard W. Porter, MD, FRCS, FRCSE,* Richard M. Aspden, PhD,* and John A. N. Shepperd, MA, FRCSE†

Study Design. Lumbar motion segments were tested *in vitro* to examine biomechanical changes after posterior fixation by a flexible device.

Objectives. To assess changes in load distribution and conformation of vertebral structures after a flexible stabilization. This should provide the foundations for a scientific understanding of the immediate effects of this surgical procedure.

Methods. Hooks were placed over the proximal spinous process and the distal laminas of a motion segment and connected by a polyester braid. Tension applied to the braid then generated a compression of the posterior elements. The force between the articular facets, the displacement of the posterior anulus fibrosus of the intervertebral disc, and the change in the relative position of the adjacent vertebrae were measured as the applied tension was increased.

Results. Facet joint force, disc bulge, and vertebral angulation increased with applied tension until a position of "locking" was achieved, apparently when the bony margin of the superior half of the facet joint contacted the inferior pars interarticularis. A tension of between 50 to 100 N in the braid was required for this. Facet joint force was less than 40% of this, and disc bulge was only 0.15 mm. The extension of the motion segment was between 2° and 8°.

Conclusions. The results suggest that if such a system is applied surgically, stabilization is produced by compaction of the bony margins of the facet joints. Only a relatively small proportion of the posteriorly applied load is carried by the facet joints themselves, and little angulatory change is expected with minimal disc bulge. [Key words: artificial ligament, biomechanics, flexible stabilization, lumbar spine] **Spine 1997;22:151–155**

Spinal fusion has been without rival in the surgical management of chronic low back pain. However, its unpredictability and the complications of this major surgery have directed surgeons to consider alternative procedures.[2] Flexible fixation systems that could add stability to a motion segment without fusion have been developed[6] and used with encouraging results.[3,4,7] The mode of action is unknown, and there are few biomechanical studies of the effect of such systems on the spine.[13] This study was designed to assess some of the biomechanical effects of an artificial ligament-like implant and its ability to reinforce the posterior structures of a lumbar motion segment.

From the Departments of Orthopaedic Surgery, *University of Aberdeen, Aberdeen, and †The Conquest Hospital, Hastings, United Kingdom.
Supported by the Sir Jules Thorn Charitable Trust.
Acknowledgment date: May 26, 1995.
First revision date: October 11, 1995.
Acceptance date: November 24, 1995.
Device status category: 1.

Materials and Methods

Twelve lumbar motion segments (three each of L1–L2, L2–L3, L3–L4, and L4–L5) were examined from six cadaveric lumbar spines. The specimens were devoid of bony diseases. They ranged from 64 to 75 years of age. The laboratory model consisted of two adjacent vertebrae, the intervertebral disc, and the segmental ligaments. A motion segment was clamped into a jig holding the distal vertebra, whereas the proximal vertebra could move freely. The samples were kept moist at room temperature throughout the tests.

Forces were applied to the posterior elements of the motion segment through a system of hooks and cords. This system uses a hook over the spinous process of the proximal vertebra, one hook over the caudal aspect of each lamina of the distal vertebra, and two polyester braids looped around bollards on the hooks on both sides of the mid-sagittal plane (Figure 1). This device is similar to one being developed for clinical use in which the cords can be independently tensioned, after which braids are then crimped individually to secure the chosen position of the motion segment.

In the current study, for the purpose of measurement, the cords were extended distally through a pulley and attached together to the crosshead of a materials testing machine (Instron 5564) to apply a controlled force. Figure 2 shows the laboratory model in position before an experiment.

Four measurements were recorded:

1. The force applied to the cords was measured by the load cell of the testing machine.
2. The facet joint force was determined in one of the two joints of a motion segment using a force-sensitive resistor. This device is a flexible sensor, 0.35-mm thick, that exhibits a nonlinear decreasing resistance with increasing force applied to the device surface. The sensitive area of this transducer was a circle 9.5 mm in diameter, which is sufficiently small to cover the whole surface of a lumbar facet. A similar

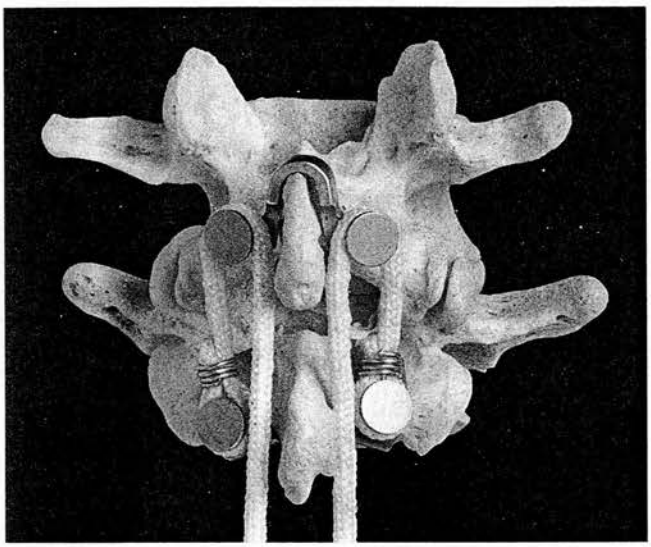

Figure 1. Hooks and cords on a model as they were positioned on cadaveric motion segments. Load is applied through the cords, which are attached together to a materials testing machine.

device has been used previously to measure forces in the facet joints.[8]

3. The posterior disc bulge was measured using a displacement transducer. The measuring pin of the transducer was introduced through a fenestration in the lamina, and it rested on the posterior surface of the disc.

4. The angulation of the motion segment was measured using an oil-filled precision goniometer attached to the centrum of the freely moving proximal vertebra.

To examine changes in the measurements with time with the segment subjected to the final load, the measurements were recorded at 10-minute intervals over a 2-hour period. At the end of this period, the load was increased to 500 N to investigate whether and where failure would occur.

Trends across segmental levels were analyzed using linear regression. For results in which there was a steady increase before a plateau region was reached, linear regression was performed for data on the rising part of the curve.

■ Results

As the tension in the cords was increased, the facet joint force, posterior rotation of the proximal vertebra, and disc bulge all increased up to a point that we shall describe as "locking." In this position, no further change was observed in any of these measurements with increasing applied load, and the tip of the inferior facet of the proximal vertebra made bony contact with the pars interarticularis of the distal vertebra.

1. The load that was necessary to achieve locking of the facet joints was found to be between 57 to 102 N (Table 1), and tended to be smaller for the proximal segments than for the distal ones (Figure 3A).

2. The facet joint force increased approximately linearly with the applied load until the joint locked, when it remained constant (mean of all levels, 26 N) despite an increasing load applied to the cords (Table 1, Figure 4). The maximum facet joint force was found to be smaller at the distal segments than at the proximal ones (Figure 3B). A preload of between 22 to 56 N was observed in five of the 12 segments (two at L1–L2, one at L2–L3, and two at L3–L4) in the facet joints. However, the observed force at locking did not seem to be affected by the presence of preload. The fraction of the applied load transmitted through the facet joints also decreased at more distal levels (Figure 5).

3. The posterior rotation of the proximal vertebra increased as the load applied to the cords increased until the facet joints locked (Figure 6). The mean angulation was least at L1–L2 and increased successively at more distal levels (Figure 3C).

4. The mean displacement of the posterior anulus of the intervertebral disc was found to be 0.15 mm in the locked position, and there was no significant difference between levels (Table 1, Figure 3D). The intervertebral disc is viscoelastic, so time-dependent changes under a constant applied load, or creep might be expected. This was investigated by maintaining the applied load for 120 minutes while the displacements were recorded. The bulge increased by no more than 0.05 mm during the first hour, after which there was no measurable change. The posterior angulation did not change during this time.

5. Increasing the load to 500 N produced failure in only four samples. In three cases, failure occurred by the spinous process hooks slipping off, and in one case by fracture of the spinous process, which could be directly attributable to damage incurred during removal from the cadaver.

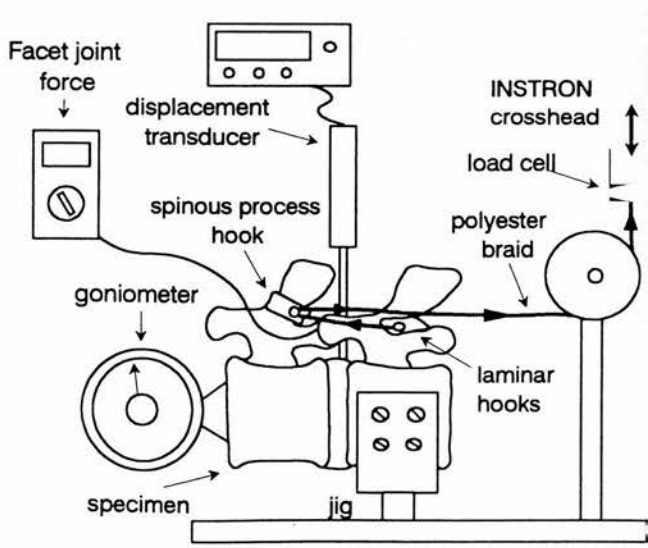

Figure 2. Diagram of a motion segment prepared for testing.

Table 1. Mean and Standard Deviation of the Applied Load to Produce Locking and the Resulting Facet Joint Force, Extension Angulation and Disc Bulge at Each Spinal Level*

	L1–L2	L2–L3	L3–L4	L4–L5	Mean	r^2	*P*
Loan (N)	59 ± 11	74 ± 6	87 ± 18	78 ± 22	74 ± 15	0.52	0.018
Facet j.f. (N)	31 ± 7	29 ± 13	26 ± 14	17 ± 3	26 ± 11	0.69	0.028
Extension (°)	2.3 ± 0.6	5.0 ± 3.0	5.0 ± 1.0	6.3 ± 0.6	4.6 ± 2	0.84	0.048
Disc bulge (mm)	0.14 ± 0.13	0.12 ± 0.06	0.18 ± 0.12	0.17 ± 0.09	0.15 ± 0.09	0.72	0.075

The column marked mean is the mean of all the levels combined and the final column is the squared correlation coefficient for a linear regression analysis of the variation with spinal level shown in Figure 3 with its associated significance.

■ Discussion

The clinical benefits of a flexible fixation system have not been explained. They may result from restriction of motion or from supplementation of soft tissue support. It is important, however, to understand the biomechanical changes that accompany locking of the facet joints by a flexible ligament stabilization system. Locking in this way is apparently what is achieved initially by the Graf ligament.

Locking of the facet joints was determined by observing the changes in angulation and facet joint force. In this position, there is direct contact between the bony margin of the inferior facet joint of the proximal vertebra and the pars interarticularis of the distal vertebra. There is, therefore, little further possibility of increasing the angulation. The proximal vertebra may try to pivot around the point of contact, but this stretches the capsular ligaments, the anterior longitudinal ligament, and the anterior anulus fibrosus, which restricts any additional movement. Overextension *in vivo* is probably best avoided because Jacob and Suezawa[9] have shown that bony contact between the articular process and the caudal pars interarticularis can lead to fracture of the pars (spondylolysis) if an additional axial load is applied.

It might be expected that a tight posterior ligament would significantly add to the load across the facet joints. However, the force required to lock the facet joints is small compared with body weight. This force clearly is a function of the lever arm (*i.e.*, how far from the bending axis it is applied). The positions chosen are those that could be used in surgery and are close to the vertebrae. The aim was to apply a controlled force to a motion segment at a clinically reasonable position. The results show that only a fraction of this is borne by the facet joints themselves. The load carried by a vertebra is distributed between the vertebral endplates (measurable in

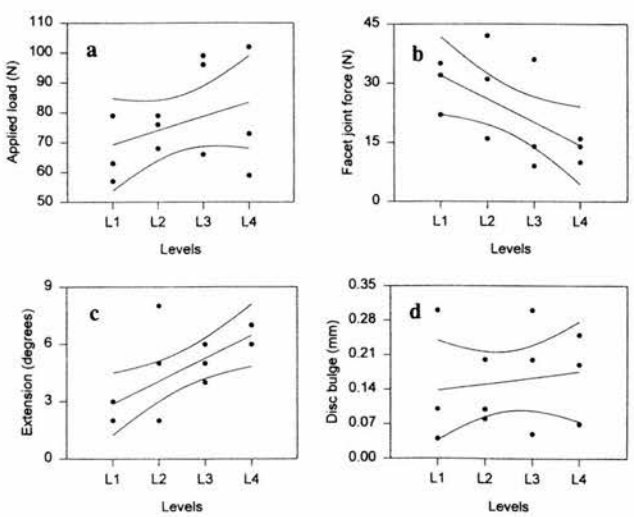

Figure 3. The applied load to produce locking is shown **(A)**, along with the facet joint force **(B)**, extension **(C)**, and disc bulge **(D)** in this position. (Regression line and 95% confidence limits.)

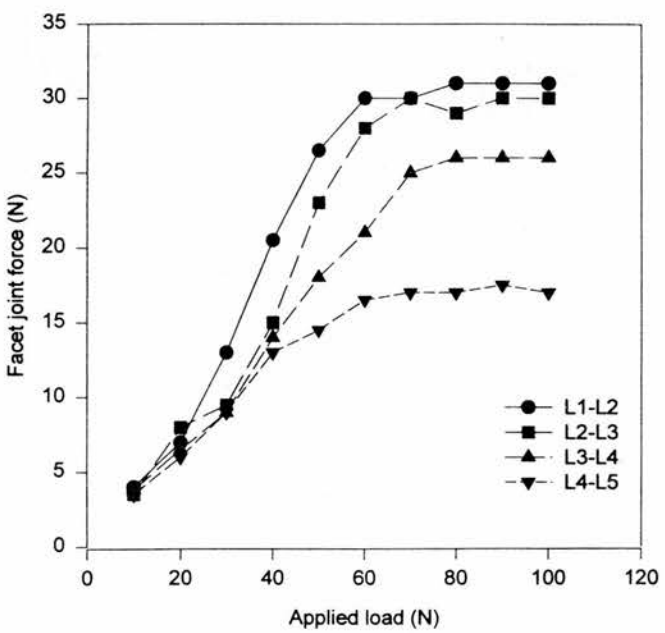

Figure 4. Mean curves from measurement of facet joint force with increasing applied load. The force increases approximately linearly until locking of the facet joints occurs, after which there is no further increase with increasing load (L1–L2: $r^2 = 0.82$, L2–L3: $r^2 = 0.89$, L3–L4: $r^2 = 0.91$, L4–L5: $r^2 = 0.86$, $P < 0.05$).

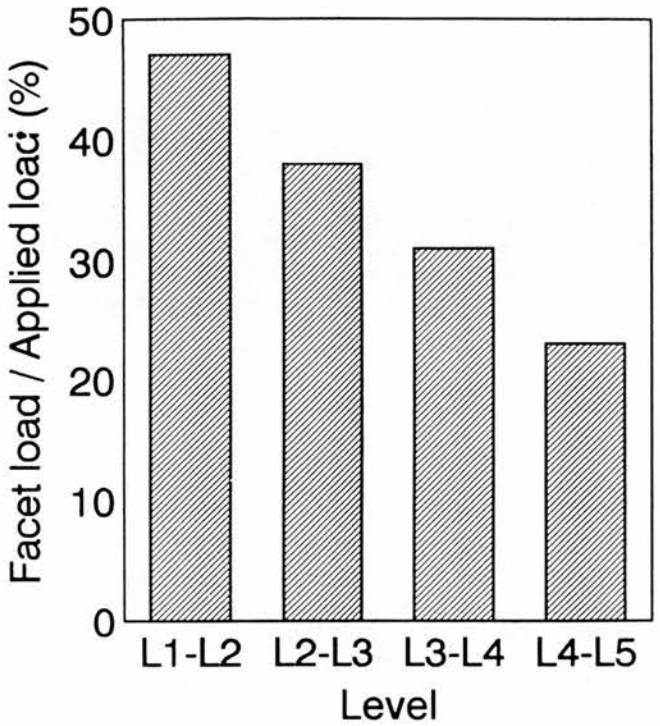

Figure 5. At locking, a smaller fraction of the applied load is transmitted through the facet joints of the more distal segments, implying that a greater load is carried by the disc.

the disc) and the facet joints. Previous studies have shown the load measured in the L3 disc of a living subject in an upright posture is more than body weight, and in just 20% of forward flexion can be more than twice body weight.[10] *In vitro* studies have shown that the facet joints bear between 5% to 60% of an applied compressive force.[8] The applied load required to lock the facet joints in the current study was found to be only about 10% of an average body weight (700 N), and less than one half of this is carried by the facet joints. Segmental differences in this load probably result from the different anatomy of the facets at different levels. The articular processes limit extension more markedly at the proximal levels than at the distal ones because of the more coronal facet orientation.

It has been shown that 2° of extension for a lumbar motion segment corresponds to erect standing in life.[1] We observed a posterior angular motion of between 2° to 8° that was limited by locking of the facets. As might be expected, it therefore appears that an implant that locks the facet joints puts the spine into an extension greater than the normal lordosis. However, this angulation was in the isolated segment, and in the intact spine it might be considerably less because of the attachment of neighboring levels. It is anticipated that application of such a tension band will itself restrict the relative movement of adjacent vertebrae, which may be abnormal in a patient and may produce a locking of the facet joints. Limitation of movement may be the cause of the reduction of pain, but additional studies are required to investigate dynamic effects that occur *in vivo*.

In extension, the posterior anular bulge is expected to be approximately 1 mm; during lateral bending in an asymptomatic individual, the normal disc bulge has been shown to be as much as 1.5 mm into the root canal.[12] The neural elements usually occupy less than 30% of the available space in the lumbar central spinal canal,[5] and there is ample room for the cauda equina with normal disc bulge. The additional displacement measured here of 0.15 mm is only 10% of the maximum bulge measured *in vivo* and less than 1% of the mid-sagittal diameter of the vertebral canal.[11] As a result of the posterior compression, however, the capacity for lateral bending of the particular motion segment is reduced. Hence, the root canal would probably be subjected to less reduction in size after stabilization than it is in an unfixed spine. There appears, therefore, to be no requirement to decompress the spine before attaching such an artificial ligament unless there are already existing neurologic signs.

This model provides the first biomechanical results of the effects of applying a force such as may be exerted on the posterior elements of the lumbar spine by a flexible stabilization system. Although the data are limited by having an isolated segment and by the temporary nature of the experiment, the model has provided some useful information and suggests a direction for future studies. This study should, therefore, provide some encouragement to clinicians.

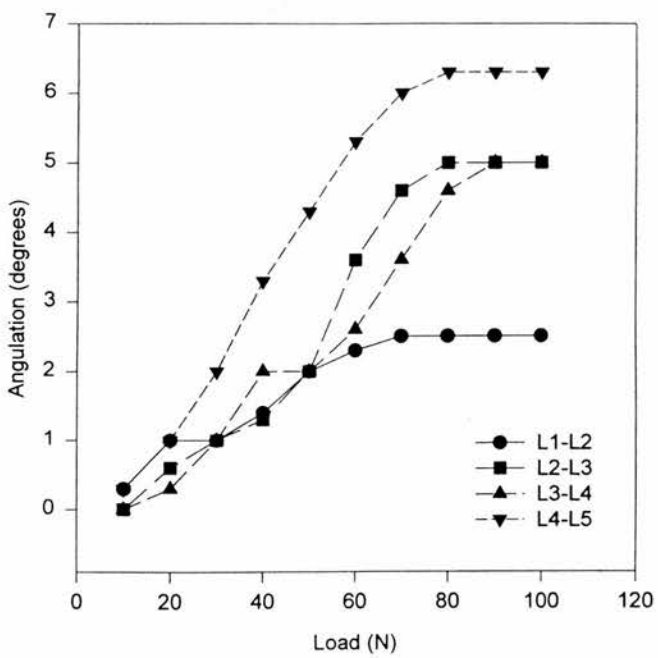

Figure 6. The angulation of the motion segments increased approximately linearly with increasing applied load until the facet joints locked (L1–L2: $r^2 = 0.87$, L2–L3: $r^2 = 0.89$, L3–L4: $r^2 = 0.97$, L4–L5: $r^2 = 0.92$, $P < 0.05$). After this, there is no further measurable change. The mean of all results at each level is shown.

Acknowledgments

The authors thank the Medical Research Council for a Senior Fellowship for RMA, and Surgicraft Ltd. for kindly providing the hooks and cords.

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Address reprint requests to

Richard W. Porter, MD, FRCS, FRCSE
Department of Orthopaedic Surgery
University of Aberdeen
Polwarth Building
Foresterhill, Aberdeen
Scotland, United Kingdom

J. R. Coll. Surg. Edinb., **42**, December 1997, 376—380

Review article

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Spinal surgery and alleged medical negligence

R. W. PORTER

Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh EH8 9DW

More than 20 000 spinal operations are carried out in the UK each year. The decision when and whether to operate requires mature judgement. Spinal surgery is technically difficult, demanding a high level of surgical skill. It is learnt only by lengthy apprenticeship. The after-care is equally important. The personal supervision of the surgeon who leads a coordinated team of clinicians, nurses and physiotherapists will ensure the best results.It is inevitable and unfortunate that mistakes will occasionally be made and only careful attention to detail in the pre-operative assessment, meticulous surgical care and supervised post-operative management will ensure consistently good results. The spinal surgeon needs to remain up-to-date, be disciplined with a systematic and careful approach and lead a coordinated team to maintain the highest standards.

Keywords: negligence, spinal surgery

PRE-OPERATIVE MANAGEMENT

What is a patient entitled to expect at the present time and what are the standards that are recognized by the general body of spinal surgeons today?

Avoid unreasonable haste

Spinal surgery offers the patient a life-threatening procedure for a non-life-threatening condition. There are always potential surgical complications, and although the benefits of surgery can be considerable, all reasonable conservative approaches should have been attempted first. When to operate is a difficult decision demanding fine judgement. There is a spectrum of opinion amongst surgeons for most spinal conditions. Some prefer a radical approach offering early surgery in order to hasten recovery of normal function. Others adopt a more conservative attitude. In the long term, most spinal conditions have a good natural history but disability can be protracted with conservative management. For example, there is no absolute indication for a surgical discectomy unless the patient has bladder symptoms. Even with severe sciatica and marked root tension signs, surgical treatment is not mandatory. With time symptoms will resolve, perhaps not completely, but at least to a manageable level. If symptoms have been present for only 2 weeks, it is not possible to predict the natural resolution. Without surgery, many of these patients will recover over a 3-month period with complete relief of symptoms and return to work. If there has been no improvement over a 6-week period, however, the prognosis is less optimistic. Most spine surgeons would argue therefore that disc surgery for a patient who has had less than 6 weeks of symptoms is not good practice. They would say that unless there were bladder symptoms it was operating with unreasonable haste to offer surgery to a patient with less than 2 weeks of symptoms and this would not be a practice adopted by any responsible spine surgeon.

Avoid unacceptable delay

Patients are sometimes distressed because they believe that there has been unnecessary delay in referral to a spinal unit. They may have suffered for many years with chronic back pain until finally they find an expert who is able to offer them successful spine surgery. They may remember the years of pain and suffering, and at some time having been told the problem is psychological, and they might seek redress. Such a claim, however, is unlikely to succeed because surgery for chronic low-back pain is very unpredictable. There is always a body of reputable clinicians who would not recommend surgery for chronic low-back pain. It is reasonable to operate but it is also reasonable to encourage patients to avoid surgery.

A more frequent claim for potential medical negligence is an unacceptable delay in a patient who has a bladder problem. Spinal pathology which compromises the sacral nerves supplying the bladder generally requires emergency treatment. Every surgeon knows that a patient who has a spine problem and is unable to pass urine or alternatively is incontinent of urine has a potentially serious condition. Involvement of the L5 nerve may leave a patient with a foot drop and some numbness of the foot. It is a disability, but not serious even if it does not recover. However, involvement of the sacral nerves which supply the bladder is a major problem. When the symptoms have been there for more than a few hours the problem tends to be permanent, with loss of normal bladder function for a lifetime. The same nerves supply the bowel and sexual function, which are also permanently affected.

Pathology which affects the conus can cause these problems. The conus is the lower end of the spinal cord at the level of the second lumbar vertebrae and this can be compressed by fractures at this site. The conus may be compressed mechanically or its blood supply may be affected by the fracture. The sacral nerves can also be affected in the mid- or lower-lumbar spine. These nerves form the posterior part of the cauda equina. They lie fairly close to the lamina

and they can be damaged by decompressive surgery at this level. A massive disc herniation can so compress the cauda equina that the sacral roots are pressed tightly against the lamina (cauda equina lesion).

It is therefore an emergency situation when a patient presents with back pain and bladder symptoms. These patients require rapid and comprehensive neurological assessment. The sacral nerves also supply the skin around the anus and perineal region. They are responsible for the tone of the anal sphincter muscles. Clinical assessment of this region confirms that there is a significant problem with the sacral roots and early surgery is imperative.

The patient with the massive disc protrusion and bladder problems requires urgent imaging with MRI and rapid removal of the herniated disc, decompressing the cauda equina. The patient who has had spine surgery and in the post-operative period has evidence of sacral nerve root dysfunction also requires urgent investigation and re-exploration for a treatable condition. The patient with an upper lumbar fracture and a conus lesion similarly requires urgent decompression after appropriate imaging.

It is agreed that the longer the sacral nerve roots are compressed the less likely is their recovery and therefore for the best result early surgery is required. It takes only a few minutes of complete loss of blood supply to the nerve roots for permanent damage to occur. Thus, if compression is complete, in practice early surgery is generally too late. However, the majority of patients have incomplete compression, when surgery in the first few hours can be fairly effective. The longer the delay, the less chance there is of recovery.

Adequate pre-operative investigation

The diagnosis of back pain syndromes is made from the history and the examination. Investigations are supplementary, usually to identify the level at which surgery will be required. A plain radiograph is helpful in a negative sense. It can exclude advanced ankylosing spondylitis where the sacroiliac joints are sclerosed. It can demonstrate a spondylolisthesis which may be a hidden cause for back pain. It can also show spinal metastasis from primary tumours elsewhere when these have progressed to an advanced stage. It will also show the presence of an osteoporotic fracture. However, for the majority of patients with back pain syndromes the plain radiograph is negative. There is no good correlation between degenerative change in the lumbar spine and back pain.

The image of choice is the MRI scan. This will identify a disc protrusion, degenerative discs, spinal stenosis, infection and neurological tumours. It is so sensitive that pathology is often demonstrated which is asymptomatic. It is only of positive value when the images correlate with the clinical features in the history and examination.

When MRI is unavailable, a CT scan is the next best imaging modality, followed by myelography. It is unacceptable to operate on a patient without one of these supplementary investigations. Spinal surgery should be a once-in-a-lifetime experience. It is not an exploratory procedure in order for the surgeon to make a diagnosis; rather, there should be as much information available to the surgeon as possible prior to the operation.

There are no reliable procedures which will tell us where the pain is coming from. Some clinicians will carry out facet joint injections or provocative discography in the hope that this will identify the pain source by reproducing the pain. Others will use a diagnostic external fixator and, if the pain is relieved, fuse that spinal segment. Others recommend pain probing to seek the source of the pain. However none of these are totally reliable.

Depending on the patient's clinical problem, other investigations may be necessary. For example, a patient with osteoporosis requires blood investigations to exclude other sources of demineralization. They need blood tests for osteomalacia and hyperparathyroidism, tests of the liver function and renal studies. However, for most of the patients having spine surgery, the history and examination often followed by an MRI scan is the main pre-operative assessment. It may sometime be supplemented by other studies.

Operate in the patient's best interest

It may seem obvious that surgery should be offered only when it is in the patient's best interest. However, it is unusual to have an absolute indication for surgery on the spine. The natural history of spinal disorders without surgery is generally good although disability can be protracted. It is necessary therefore for the surgeon to have a complete understanding of the patient's past history and social history to understand their lifestyle and occupational requirements before recommending surgery. It is important to understand the patient's expectations and whether these are realistic, and to ask whether surgery should in fact be carried out at all. Simply because it is possible to change pathology by a surgical approach, does not mean that it is indicated. It is justified, however, in the patient who requires a rapid return to normal function because of the family or occupational situation.

OPERATIVE MANAGEMENT AND COMPETENT SURGERY

Competent surgeon

At the present time, there are few full-time spine surgeons in the UK. Most of the spine surgery is carried out by orthopaedic surgeons or neurosurgeons who besides having a general practice take an interest in the spine. In previous years, most orthopaedic and neurosurgeons would do an occasional spine operation but it is now becoming an acceptable practice for spine surgery to be the preserve of those who have a special interest. This means that the majority of spinal surgery for degenerative conditions is carried out by a surgeon doing at least 20 spine operations per year. There is no agreed minimum limit but competence is maintained by continued surgical practice.

There are occasions when a surgeon can predict technical difficulties. For example, obesity will add to the operative difficulties and repeat spinal surgery is more complicated than the first procedure. It can be anticipated that a grossly displaced spondylolisthesis will be difficult to fuse, and extensive spinal stenosis with gross degenerative change is not easy to decompress. With such problems a good surgeon will refer to one of his more experienced colleagues. Failure to do so, and particularly failure to inform the patient of potential hazards, is poor practice.

Correct level

One of the most common causes for medical negligence is operating at the wrong spinal level. Most patients have five lumbar

vertebrae followed by the solid segments of the sacrum. About 5% of the population will have six lumbar vertebrae where one of the sacral segments is lumbarized or four lumbar vertebrae where the lowest lumbar segment is sacralized. This can confuse the surgeon. The radiographs may show the disc protrusion say at L4/5 level. If the fifth lumbar vertebrae is sacralized and fixed to the sacrum, the surgeon may be confused and operate at the L3/4 disc by mistake. It is imperative therefore that the surgeon adopts a safe practice to identify the operative level. There are two methods. One is to expose the sacrum in the operative field. It is possible to identify the solid sacrum by vision and by palpation and then work up the spine identifying each of the lower levels. When using a minimal exposure, the correct level can be identified radiographically on the operating table by introducing a needle down to the appropriate segment and checking the level on the radiograph. Sometime, methelene blue dye can be injected through the needle and this mark recognized when the area is exposed. There is really no defence for operating at the wrong level.

Correct side

Human error is responsible for operating on the wrong side. It is not defensible, but the surgeon can mistake the laterality when the patient is lying prone. Good practice requires a skin marker on the back identifying the side requiring surgery.

Sufficient surgery

There has been long debate about how much disc material should be removed in a patient who has a disc protrusion. A consensus is developing that only the loose fragmented material needs to be removed, along with any other loose fragments within the disc space. The surgeon is operating through a deep hole and cannot visualize the centre of the disc space. Loose fragments are extracted by rongeurs and sometimes by a blunt curette. Previously, surgeons would remove large amounts of disc material from within the disc space to avoid a recurrence, but this is now considered unnecessary. It is a balance of clinical judgement as to how much or how little material should be removed and there are as yet no absolute guide lines.

When carrying out a discectomy the symptomatic lesion is usually at one level. Not uncommonly, imaging will show protrusion at perhaps two levels, and there is then a dilemma about which one is symptomatic. The surgeon usually operates at the level which is compatible with the clinical features of the nerve root involved. It is good practice to limit the surgery to as little as possible compatible with the clinical features.

Spinal decompression for spinal stenosis requires removal of the tight bony lamina to allow more room for the underlying nerves of the cauda equina. In previous years surgeons were very radical, sometimes removing the lamina of all five lumbar vertebrae. In the last few years, surgery has become much more conservative, frequently recommending only partial laminectomy at a selected level where the stenosis is most significant. If decompressive surgery is too extensive, it runs the risk of the development of post-operative scar tissue causing further stenosis and also the risk of instability and post-operative back pain. However, if the decompression is too limited, the nerve roots may not be adequately decompressed. In addition, bony ridges can develop post-operatively, tightening up the canal again.

Damage to nerves

Disc surgery is surgery for the nerve root, and decompressive surgery removes bony structures where spinal stenosis is causing nerve problems. These nerves are already vulnerable, having been partially compressed, and they need to be handled with great care. Even gentle retraction of the nerves in order to expose a disc or to remove tight bone can further affect nerve root function. Even when these nerves are satisfactorily decompressed the patient may be left with some abnormal nerve function. This can occur even in the best hands and is unavoidable. There are, however, occasions when nerves are crushed or bruised by surgical instruments and sometimes a nerve can be severed. This is the result of poor surgical technique and is generally not to be expected from a competent spine surgeon. There are occasions, however, when an experienced surgeon is attempting to decompress a tight spinal canal, the nerves cannot be fully visualized and unavoidable contusion can occur. The surgeon may or may not be aware of this injury at the time.

The most popular method of performing a spinal fusion is to support the bone graft by instrumentation using pedicle screws. These screws are inserted posteriorly through the pedicle of the vertebrae into the vertebral body. This is done blindly with an understanding of the direction of the pedicles. Studies have shown that even in expert hands 20% of the pedicle screws transgress the pedicle to some degree. Frequently this is not clinically important but from time to time there may be a serious transgression of the pedicle and the screw can damage one of the nerve roots. This occurs when the anatomy of the spine is slightly abnormal and the surgeon will not usually be aware that the screw is not within the pedicle. It is only when the patient recovers from the anaesthetic complaining of leg pain that the surgeon is aware of this complication.

Radiographs do not usually help to confirm whether or not the screw has transgressed the pedicle because of so many overlapping shadows. CT scans and MRI are similarly unhelpful because the metal scatters the images and impairs good definition. Nerve root damage as a result of a pedicle screw is a clinical diagnosis which can sometimes be difficult. Nerve root pain can be present post-operatively because of mechanical disturbance to a nerve root during an associated decompression. The spinal mechanics may have been slightly altered, compressing the nerve root, and it is sometime difficult to be confident that a pedicle screw is responsible. If a screw has completely transfixed a root then surgical removal of the screw is not likely to significantly affect the symptoms. If, however, the nerve root is being irritated or contused by the screw, the removal will be beneficial. The surgeon tends to re-operate because of a high level of suspicion and sometime is rewarded with a relief of the symptoms. On other occasions permanent damage will have resulted from this misfortune. Although the surgeon can not reasonably be blamed for this problem, the patient should be warned pre-operatively that there is a slight risk of nerve damage when pedicle screws are to be inserted.

Damaged dura

The dural membrane surrounds the cauda equina and the nerve roots bathed in cerebrospinal fluid. There are two layers of the

dura. If both layers are cut or torn cerebrospinal fluid will leak into the wound. The extradural veins are usually compressed by the tight dura and when the cerebrospinal fluid leaks, the dural pressure falls and the veins become congested. The operation then becomes difficult because of cerebrospinal fluid and venous blood filling the wound. Damage to the dura occurs in about 5% of lumbar spine operations and it is considered an accepted complication that is sometimes difficult to avoid. Some surgeons then recommend a wider exposure followed by suturing the dura and others recommend that the dura not be repaired. These surgeons rely rather on a secure muscular repair to avoid a post-operative leak.

If the dura is contused and torn, the nerve roots which lie posteriorly in the cauda equina are the sacral roots which supply the bladder and bowel and sexual functions. The surgeon is therefore particularly cautious when decompressing at the back of the spinal canal. Rough handling of the tissues at this level is of course poor practice, but the surgery can be very difficult in the obese patient with marked degenerative change.

A few patients who have a dural leak at the time of operation continue to have a discharging sinus of cerebrospinal fluid. In the majority of these the sinus will become dry over a few days, while on other occasions it will produce a chronic leaking sinus which requires further surgery. Occasionally a pseudo-meningocele will form with a large cyst of cerebrospinal fluid, which requires surgical closure.

Damage to blood vessels

Most patients have minimal blood loss during spine surgery and do not require a blood transfusion. Extensive decompression and a spinal fusion using a bone graft from the pelvis will, however, frequently cause sufficient blood loss to require a blood transfusion.

It is only when a major vessel is damaged that serious complications occur. The superior gluteal artery leaves the pelvis into the buttock through the lower part of the pelvis (the greater sciatic foramen). When taking a bone graft from the back of the pelvis it is possible to damage this vessel and the surgeon will therefore avoid this particular region. Bleeding from the superior gluteal artery is not the result of poor practice, but failure to ligate the vessel is not acceptable. Sometimes the help of a vascular surgeon is required to identify and secure the bleeding vessel.

It is possible to damage the aorta or the inferior vena cava when carrying out a discectomy, if the disc extractor penetrates through the anterior annulus of the disc. If the surgeon relies on removing only the loose fragment this complication is not likely to occur. However, if there is a radical excision of the disc space it is possible to penetrate through the anterior annulus. This may already be torn pathologically. The surgeon is therefore particularly cautious when operating towards the front of the disc space. It is difficult to defend this injury.

The surgeon is aware that there has been some damage to a blood vessel anterior to the disc space when there is a small amount of blood on the instruments. The disc space is avascular and the instruments should be dry. The anaesthetist may note that there is a drop in blood pressure and an increase in the pulse rate. There should be no delay in turning the patient into the supine position and with the help of a vascular surgeon exposing the damaged vessels. Tragedies occur when the surgeon procrastinates and hopes the injury is minimal. It is then frequently too late to avoid a fatal outcome.

Infection

Infection can occur from time to time. This can be an airborne infection or bacteria transmitted from instruments. The surgeon's gloves can perforate with infection being transmitted from the surgeon's hands. Sometimes bacteria in the patient's own blood (bacteraemia) can settle in the wound, producing the infection. These are occasional hazards which are difficult to avoid. Some surgeons recommend perioperative prophylactic antibiotics, but this is not routine general practice.

POST-OPERATIVE MANAGEMENT

The surgeon and the surgical team need to be vigilant in the post-operative period. As soon as the patient is awake from the anaesthetic it is important to carry out a neurological assessment, particularly to confirm that there is not abnormal neurology which was not present before surgery. If there is an area of anaesthesia in the lower leg and foot or some weakness of the lower leg, this suggests some nerve root damage by surgery. If the patient has severe pain in the root distribution which was not present before surgery this is again evidence of some nerve root damage. A complete transection of the nerve root is not usually painful but will give some motor weakness and sensory loss. Root pain, however, suggests some degree of nerve compression or irritation.

Sacral anaesthesia and some loss of anal tone suggests sacral nerve root damage and possible bladder disfunction. This is a serious sign which usually requires urgent repeat surgery. If there is sacral nerve damage there may be a haematoma pressing on the nerve roots which can be relieved by decompression. However, if the sacral roots have been contused by the surgery, further exploration will not help.

In the next few days, if the patient continues with severe root pain or root pain which was not previously present, it is worth re-exploring the spine after suitable imaging. It is possible that the surgery has been carried out at the wrong level or the wrong side or that the decompression, although at the correct site, has not been adequate. Further surgery is likely to be helpful. If, however, nerve root symptoms develop some days after surgery, after a pain-free interval, this suggests that there has been a fragment of disc material previously missed which has now extruded and is pressing on a nerve root. It is no fault of the surgeon. Further surgery for this fragment after suitable imaging can be helpful. However, depending on the severity of the symptoms, the pain may be left to resolve naturally.

If there is a leak of cerebrospinal fluid after surgery the patient should remain in bed receiving appropriate antibiotics and routine dressings until the wound becomes dry. If after a few days the wound is continuing to discharge cerebrospinal fluid, exploration may be appropriate.

Post-operative infection can be the result of a chest infection or urinary infection. If these can be excluded then infection of the wound is the most likely source. There should be no delay in identifying the source and giving appropriate antibiotics. If there is a high temperature a blood culture is indicated. Post-operative

discitis is an infection in the disc space. It is associated with severe low-back pain and usually spasm of the spinal muscles. The patient is in severe pain when attempting to stand. The radiographs are normal for a few weeks but the MRI scan is very sensitive to discitis and is the image of choice. A blood culture or needle biopsy will identify the organism and its sensitivity.

Repeat surgery

It is a difficult decision to know whether or not to operate again in the post-operative period. The complications of the second operation are greater than the first, but provided the indications are correct there can be a considerable bonus in performing this procedure. The inexperienced spine surgeon should seek a second opinion before embarking upon repeat surgery.

INFORMED CONSENT

One of the most common causes of patient dissatisfaction is failure to receive sufficient information about the surgery and its risks. At the present time, spine surgeons would agree that every patient should be given information about risks and benefits. In broad terms patients should be informed of the changes of improvement by surgery. For example, when performing a discectomy for nerve root pain there is something like a 90% chance of relief of leg pain by surgery. When decompressing the spine for neurogenic claudication there is a 60% chance of reducing the symptoms and perhaps a 60% chance of relieving chronic back pain by a spinal fusion.

The patient should also be told, in general terms, the risk of not being improved by surgery. For example, in discectomy there is a 10% chance that some leg pain will persist in the short term. The nerve has been bruised for a long time and even though the fragment of disc has been removed the nerve can remain sensitive and painful. When offering fusion for low-back pain, 40% may continue with their pain.

There is always a remote risk that the patient could be worse. They should be told of this possibility. In disc surgery 2 or 3% can be worse as a result of damage to a nerve root, a leak from the dura or some post-operative infection, and there is always the chance of some anaesthetic complication. Patients are not usually told about these remote risks in great detail, or why they could be worse by surgery. If, however, they specifically ask how they could be worse, then it is the surgeon's responsibility to explain some of these problems. When carrying out decompression for neurogenic claudication or a fusion for chronic low back pain, patients are told that not only may their symptoms not be relieved but they could have more pain and over a period of time things could get steadily worse. There is about a 5% chance of having repeat surgery after discectomy and 10 to 20% chance of having further surgery after decompression for neurogenic claudication. There is a similar risk of repeat fusion for chronic low-back pain. The broad concept of improvement, failure to relieve symptoms and the change of being worse should be explained to every patient and also recorded.

In spite of these many pitfalls most patients do well. Fortunately those patients who have careful pre-operative selection, competent surgery and good post-operative management are significantly helped by their operation, and provided they are aware of potential risks, patients usually accept that the surgeon and the surgical team have done their best in a very difficult area of medical care.

through the six dorsal compartments of the wrist forms an ulnarly directed obtuse angle which results in ulnar translational stress with muscle contraction. In full supination and wrist ulnar deviation, this angle may become even more acute, resulting in a greater force vector against the fibro-osseous sheath. With complete disruption of this sheath, the tendon may bowstring and dislocate across the ulnar styloid.

We observed two types of disruption of the fibro-osseous sheath. When a tear of the fibro-osseous sheath occurs at the ulnar wall, the ECU tendon may come to lie beneath the torn fibro-osseous sheath when returning to its ulnar groove. When a tear occurs at the radial wall, the ECU tendon may come to lie on its ulnar groove superficial to the torn fibro-osseous sheath, preventing any chance of healing. Burkhart *et al*[3] and Rayan[4] suggested that acute injuries can be treated by cast immobilisation with the forearm in pronation and the wrist in radial deviation, although they have had no experience with treating acute dislocation of the ECU tendon. Rowland[5] , however, operated on an acute case and found that there was a considerable gap between the torn edges of the fibro-osseous sheath no ma position the wrist was placed. This gest that there may be no p 57 anatomical healing of the fibro-oss in an acute dislocation of the ECU tendon. The findings of the present two cases and the case reported by Rowland[5] provide a strong argument in favour of surgical treatment for symptomatic dislocation of the ECU tendon, even in an acute case.

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Achilles tendon rupture and sciatica: a possible correlation

Nicola Maffulli, Andrew S Irwin, Michael G Kenward, Francis Smith, Richard W Porter

Department of Orthopaedic Surgery, University of Aberdeen Medical School, Polwarth Building, Foresterhill, Aberdeen, Scotland, United Kingdom
N Maffulli
A S Irwin
R W Porter

Department of Radiology, MRI Centre, Woodend Hospital, Eday Road, Aberdeen
F Smith

Department of Medical Statistics, University of Kent, Canterbury, Kent, United Kingdom
M G Kenward

Correspondence to:
Nicola Maffulli, Department of Orthopaedic Surgery, University of Aberdeen Medical School, Polwarth Building, Foresterhill, Aberdeen AB25 2ZD, Scotland.

Accepted for publication
1 December 1997

Abstract

The association between Achilles tendon rupture and sciatica was investigated by questionnaire in 138 patients who underwent repair of an Achilles tendon rupture, and in a group of individuals nominated by the patients, matched for age, sex, and occupation. A total of 102 patients (74%) and 128 peer nominated controls (71%) replied to the questionnaire. Of the 102 respondent patients, 18 had an office-based job, 47 were involved in skilled non-manual work, and 16 were retired. Back pain had been experienced by 63 of the patients who replied to the questionnaire, and by 91 (75%) of the individuals in the control group (difference not significant). In about 30% of both groups, the pain confined them to bed for at least two days, and resulted in absence from work. Thirteen of the patients and 16 of the controls had undergone thoracic, lumbar, or sacral radiography. One individual in each group had received surgery for back pain. However, 35 of 102 patients had experienced sciatic pain before Achilles tendon rupture. Pain of a similar nature had been experienced by only 15 individuals in the control group (12%) ($p<0.001$). Using this study design, we found a highly significant association between Achilles tendon rupture and sciatica. We propose that this association could be due to impaired afferent signals from the lower leg, or to similar collagen or vascular anomalies of the vertebral disc and the Achilles tendon.
(*Br J Sports Med* 1998;**32**:0–0)

Keywords: Achilles tendon; sciatica; back pain

Achilles tendon rupture and prolapsed intervertebral disc are relatively common musculoskeletal ailments, both showing peaks of prevalence in the fourth and fifth decades.[1] Achilles tendon rupture can be associated with systemic disorders such as systemic lupus erythematosus and diabetes, and with local and systemic steroid administration.[2 3] Vascular alterations can play a role in the degenerative process preceding Achilles tendon rupture.[4]

Previous studies have also shown that, in fresh samples of ruptured Achilles tendons, there is a pathological increase in the content of type III collagen.[5] Alterations in Achilles tendons and intervertebral disc biochemistry have also been hypothesised to be important in causing pathological changes eventually leading to rupture and prolapse respectively.[6–8] We

had previously noted that a proportion of patients presenting with an Achilles tendon rupture gave a past history of sciatic pain. We have therefore sought a possible association between these two conditions.

Patients and methods

PATIENTS

One hundred and fifty consecutive patients admitted to the Aberdeen Royal Hospital NHS Trust for a unilateral Achilles tendon rupture in the period January 1988 to June 1994 were retrospectively identified using the operating theatre registers.

QUESTIONNAIRE

A questionnaire was mailed to each patient for whom admission and operation notes were available (see below) asking (1) whether they had ever experienced low back pain (LBP) prior to the Achilles tendon rupture, (2) whether the LBP had confined them to bed, (3) whether the LBP had warranted consultation of a doctor, and (4) whether they had had any LBP related (*a*) admission(s) to hospital, (*b*) lumbosacral spine radiographs, or (*c*) surgery to the lumbar spine.[9] Patients were also asked (5) whether, with the LBP or independently from it, they had experienced sciatic pain, extending into the thigh and below the knee,[10] (6) whether the sciatic pain was (*a*) ipsilateral or (*b*) contralateral to the side of the Achilles tendon rupture, (7) whether the sciatica had confined them to bed, (8) whether the sciatica had warranted consultation of a doctor, and (9) whether they had had any sciatica related (*a*) admission(s) to hospital, (*b*) lumbosacral spine radiographs, or (*c*) surgery for their sciatica. Finally, patients were also asked (10) to provide details of their occupation at the time of injury.

CONTROL GROUP

Patients were asked to nominate two persons of the same sex and within two years of their own age, who had not suffered from a non-traumatic rupture of a lower limb tendon, and who were in the same or similar occupation at the time of injury. Not all patients nominated two such persons, and a total of 179 people were peer nominated. The control group was mailed the same questionnaire investigating their past medical history of LBP and sciatica.

OPERATIVE DETAILS

The patients suffering from an Achilles tendon rupture were treated operatively either by an open repair method[11] or by percutaneous repair.[12 13]

STATISTICS

Descriptive statistics were calculated. The previous history of back pain and sciatica was compared between the patients with Achilles tendon rupture and the controls. Because there are incomplete sets of matched cases and controls, the use of conventional conditional analysis would lose information on unmatched observations. Hence, the groups were compared using the generalised estimating equation technique.[14] Significance was set at $p<0.05$.

Results

CLINICAL DATA

Admission and operation notes from 138 patients were available for analysis. There were 75 right- and 63 left-sided Achilles tendon ruptures, and the overall median age of the patients at the time of injury was 46 years (range 24–84 years). The ruptures were repaired using an open method through a medial para-tendinous approach in 106 patients, and using a percutaneous method in the remaining 32 patients.

The mechanism of injury was recorded in 105 patients. In 48 (46%), the Achilles tendon rupture occurred during sport. In the remaining 57, the rupture was incurred at work or during household or gentle leisure activity. Nine patients were diabetic, six had asthma, and two had inflammatory bowel disease. Three patients had received systemic steroid therapy, but in no case had the Achilles tendon ever been injected with steroids. Patients were not aware of symptoms related to tender Achilles tendon before the rupture.

QUESTIONNAIRE DATA

After three mailings, 102 patients (74%) and 128 of the 179 peer nominated controls (71%) replied to the questionnaire. The occupations of the patients and the peer nominated controls were comparable. They were largely sedentary workers. Eighteen of the 102 respondent patients had an office based job, 47 were involved in skilled non-manual work such as teaching or being officers in the army, 16 were retired at the time of Achilles tendon rupture, and the remaining 21 were manual workers.

Previous back pain had been experienced by 63 (62%) of the patients who replied to the questionnaire, and by 91 (71%) of the control group (difference not significant). In about 30% of individuals in both groups, this pain had been of sufficient magnitude to confine them to bed for at least two days and to result in absence from work. Thirteen patients and 16 individuals in the control group had had thoracic, lumbar, or sacral radiographs (difference not significant). One individual in each group had received surgery for back pain. However, 35 of 102 patients (35%) had experienced previous pain extending into the thigh and below the knee that had resulted in bed rest or consultation of a doctor. In 24 of these 35 patients, the pain was ipsilateral to the site of Achilles tendon rupture ($p = 0.0001$). Three patients could not remember the side on which the sciatic pain had occurred. Pain of a similar nature had been experienced by only 15 individuals in the control group (12%) ($p = 0.001$).

Discussion

Sciatic pain has been reported in 19% of coal workers.[9] The incidence of sciatic pain is recorded as 22% for machine operators, 24%

for carpenters, and only 14% for office workers.[9] Manual work is a risk factor for sciatic pain.[10] For this reason, our patients and controls were matched not only for age and sex, but particularly for occupation. We have observed a statistically significant association between Achilles tendon rupture and sciatic-like pain: 35% of individuals who had had an Achilles tendon rupture recollected previous sciatic pain compared with only 12% of the controls.

The two most common causes of sciatic pain are a prolapsed intervertebral disc and root entrapment from degenerative changes in the root canal. Both are frequently the consequence of intervertebral disc pathology. The cause of disc degeneration is not understood, but the role of the blood supply to the vertebral end plate is thought to be highly significant.[8] Smoking[15] and vibration[16] may adversely affect blood supply to the disc. Impaired disc nutrition probably predisposes to degeneration with the final symptomatic protrusion generally following an innocent physiological stress.[17] Whether the collagen changes cause nutritional impairment or are a consequence of it is unknown.[18 19]

It is also uncertain why the Achilles tendon ruptures. Even with subclinical involvement, the afferent signals from joints, ligaments, and muscles of the lower leg supplied by the L5 and S1 roots[20] may be impaired and improperly interpreted. This would produce altered proprioception from these structures. Malcoordinated motion could occur at the ankle joint, resulting in dorsiflexion of the ankle while the gastrocnemius-soleus complex is still contracted, with overstretching of the Achilles tendon and its subsequent rupture. The high frequency of sciatica in the same leg as the Achilles tendon rupture makes this a plausible explanation.

An Achilles tendon rupture can occur with a physiological load, and may be the result of a nutritional problem similar to that of the intervertebral disc.[4] Alternatively, there may be general collagen abnormality affecting both disc and tendon, making them both prone to disruption in some individuals.

The present study has several weaknesses, some of which are connected. There is an inevitable selection bias using peer nomination, and it is at present unknown whether the controls were less likely to recall illness and episodes of pain than the patients with an Achilles tendon rupture. As in any retrospective study, the individuals involved in the present work may have an inevitable recall bias. It could be argued that, with or without knowledge of the purpose of the study, patients would have been inclined to select particularly healthy individuals as controls, and that hospital patients might have been more appropriate. However, the prevalence of sciatica varies in the different age groups and occupations, and we would have introduced a further bias in the form of the conditions for which such a control group, composed of patients, was hospitalised.

This is a retrospective study, and, although sciatic pain is distinctive,[10 16] a single episode could have been forgotten. However, the controls had clear recollection of LBP. It is unlikely that sciatic pain episodes had been forgotten, especially as patients often require bed rest and a doctor consultation for such ailment. The data collected from our peer nominated control group were probably not biased in this respect.

The patients were not examined by an orthopaedic surgeon, neurosurgeon, or neurologist. We did pilot the questionnaire extensively, but our definition of sciatica could still have been misinterpreted. Some patients may have complained of sciatica whereas the pain was really an Achilles tendon pain. Also, both patients and controls may have interpreted arthritic pain from the hip radiating to the thigh and the knee as sciatica. The same concern could be raised, for example, regarding conditions such as spinal stenosis, Baker's cyst, or pyriformis syndrome. An interview and a formal clinical examination by a trained physician could have clarified the question, but a further bias, i.e. the presence of an interviewer, would have been introduced. Imaging could also have helped, but the costs and logistics of the study would have been prohibitive. Also, a significant number of asymptomatic individuals show lumbar disk herniation on magnetic resonance scans.[21]

In retrospect, we should also have controlled for activity level in both our patients and the control group. It is unlikely that the control group was different from our study group in this particular aspect (see Patients and methods), but we cannot prove that this is the case. Probably, future research should address this question as well.

In conclusion, a previous history of sciatic pain appears to be significantly more common in patients with Achilles tendon rupture than in matched controls. The fact that this relationship exists does not establish a causal effect, and one should not be led to overspeculate on such a finding, at least at this stage. Achilles tendon rupture may be predisposed by previous sciatica when there is impairment of the afferent signals from tendons, muscles, and joint structures carried by the sciatic nerve. Alternatively, the association may be due to similar nutritional vascular impairment of the intervertebral disc and the Achilles tendon, or to abnormal collagen present in both structures.

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Commentary

This study deals with the interesting topic of a possible association between Achilles tendon rupture and sciatica. The study construction is relevant, and the selection of peer nominated control subjects (with age, sex, and occupation all matched) as well as the sample size make the statistical testing feasible, especially as the equation analysis model was employed. The correlation of tendon rupture and sciatica is, no matter how you analyse it in a clinical setting, evidence based and hypothetical, but according to the present results clearly possible.

There are, however, some points, that I would like to pay attention to.

(1) This is a questionnaire study, which the authors admit is a weakness. The questionnaire is well designed, but leaves some uncertainty about the correctness of the diagnosis of sciatica. The patient may interpret for example arthritic hip and its referred pain to the thigh and knee as LBP (the age of some of the patients was high enough for coxarthrosis to occur). Spinal stenosis, Baker's popliteal cyst, and piriformis syndrome can also sometimes mimic symptoms of LBP. Only thirteen patients and 16 controls had lumbar or thoracic *x* ray pictures taken. However, despite these weaknesses, I can accept the study as a first effort to analyse the possible association between Achilles tendon rupture and sciatica. Therefore the questionnaire set up can be justified.

(2) Another weakness of the study, which the authors themselves point out, is the selection of the control group. I agree with the authors about this, but, on the other hand, I do not think that any other cohort of control subjects (rather than peer nominated) would have given a different trend in the results.

I would like to urge the authors to carry on their research on this topic. Why not look at the possible association from the opposite point of view. What would the results be if the occurrence of Achilles tendon rupture was studied in patients operated on (or treated conservatively) for a verified disc prolapse and compared with a proper control group! This kind of study may, or may not, support the findings of the present one.

Finally, as the association of Achilles tendon rupture and sciatica still remains speculative, I would be fascinated to explain the impairment of the afferent signals from tendons, muscles, and joint structures as the mechanism leading to tendon rupture. The predisposing changes in the Achilles tendon vascularity and connective tissue structures in the background are already well established.

MATTI U K LEHTO