SPINAL STENOSIS

JOHN EDWIN NIXON
M.A., M.B., Ch.B., F.R.C.S.

Ch.M. Thesis
University of Edinburgh
1987
## SPINAL STENOSIS

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DECLARATION

This thesis is composed and written entirely by myself as a result of work carried out over the last four years by myself with the assistance of the individuals acknowledged below.

The work is original and the concepts and opinions expressed in this thesis are my own based on review of the literature and the results of research described in this thesis.
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TO:

Bridget,
David, Susannah and Natasha
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Name of Candidate             John Edwin NIXON
Address                      116, Staunton Road,
                             Headington, Oxford, OX3 7TN
Degree                       Ch.M.
Date                         1987
Title of Thesis              Spinal Stenosis

AIMS AND OBJECTIVES

This thesis describes in detail the clinical spectrum of spinal stenosis in a series of two hundred and twenty-one patients at the Nuffield Orthopaedic Centre. It depicts those conditions with which spinal stenosis may be confused, and other conditions with which it is associated. Characteristic symptoms and physical signs are reported and the role and value of different methods of investigation are discussed.

The aetiology and pathogenesis of spinal stenosis is discussed and the emphasis turned away from absolute measurements of the dimensions of the bony spinal canal, towards the role of the soft tissues and the dynamic response of the canal and its neural contents to postural change and loading, as evidenced by erect flexion and extension radiculography. The spinal reserve capacity measurement on CT approaches more closely the ideal of measurement of volumetric disproportion of canal and contents, but it takes no account of the dynamics of the canal. Magnetic Resonance Imaging may, in the future, provide the most objective criteria for diagnosis if section thickness can be reduced.

Experimental spinal stenosis was produced in a group of immature New Zealand white rabbits. This was induced by sublaminar wiring at three levels at the age of eight weeks and allowing the animals to grow for twenty-four months before sacrifice and analysis of the spines. The effect of sublaminar wiring on the growth and development of the lamina and spinal canal was analysed using a Kontron Ibas Image Analysis Computer, and the results described and statistically analysed.

The results of surgery were analysed in detail in a group of seventy-two patients with spinal stenosis at the Nuffield Orthopaedic Centre. The long-term results were compared with the initial post-operative result and two groups were identified: the stable result and the unstable result. The indications for and results of re-operation were also analysed in a group of twelve patients.

Improved understanding of the aetiology of spinal stenosis has enhanced surgical management and results. The extent of surgical decompression must be precisely planned pre-operatively from radiographic and CT studies, and the surgeon must be able to execute this plan at operation. There is now no place for exploratory operations. The objective of surgery is adequate nerve root decompression without spinal de-stabilisation and when this is achieved, re-operation is redundant.
CHAPTER 1

HISTORY OF SPINAL STENOSIS

The first recognisable example of lumbar spinal stenosis is provided by the achondroplastic Greek god Hephaestus, who limped as a result of trauma to an already narrowed spinal canal (Preller 1860).

Hephaestus was the only Greek god to be deformed, and this was an unusual trait since the culture dictated that the gods were created not just in the image of man but of beautiful man. However Hephaestus was "the god of the crooked foot" and was lame, hobbled, and limped.

There are two explanations for the limp. The most widely quoted is that Hephaestus was injured as a result of being hurled from Olympus by Zeus because he was interfering in a domestic quarrel (Homer 1961).

"Why, once before when I was trying hard to save you, he seized me by the foot and hurled me from the threshold of Heaven. I flew all day, and as the sun sank I fell half dead in Lemnos, where I was picked up and looked after by the Sintians."

However, another version is that Hephaestus' mother Hera was ashamed of having given birth to him because he was congenitally deformed, ugly and misshaped, and the result of an illicit love affair, and that his "wicked mother had tried to do away with me because I was a cripple" (Homer 1961).

It is probable that Hephaestus was originally a dwarf, probably achondroplastic, of ancient lineage. Hephaestus was most likely a direct descendant of the Egyptian gods Ptah and Bes, both of whom were depicted as deformed, bow-legged dwarfs, and there seems little doubt that Hephaestus was "lame in both legs" (Ἀμβλώνης), and bow-legged (Κυλλοποδίως).

Statuettes representing the Egyptian gods Ptah and Bes, leave little doubt that they were achondroplastic dwarfs, and that Hephaestus was also a dwarf was borne out by a number of incidents one of which was his unsuccessful attempt to rape Athene ending in an ejaculation "against her thigh, a little above the knee" (Graves 1959). He could be good-natured.
and amusing so that he became a figure of fun for the gods in whom he provoked "helpless laughter". (This role remains today traditionally associated with the achondroplastic circus clown.) He could, on the other hand, have the twisted mentality of the deformed dwarf and at times become ugly and ill-tempered, vindictive and malicious, as for example when he traps his unfaithful wife in bed with Ares by means of a finely forged net, or his unloving mother in a golden chair from which only he can release her. He was surprisingly married to the beautiful Aphrodite and fathered two sons, both of whom were handicapped by a crippling deformity of both legs. There seems little doubt that these are the physical features of achondroplasia, a disorder of great antiquity (Fig. 1:1 & 1:2).

An alternative hypothesis was advanced by Robert Graves who felt that since Hephaestus became a smith-god he was purposely lamed by his own tribe to prevent him from running off and joining the enemy tribes. It was felt in the Bronze Age that artefacts made of metal were endowed by special magical properties by the smith who therefore became a "sorcerer" or "magician" (Graves 1959). This ritual crippling and laming of smiths however is difficult to accept and the evidence is not convincing. One puzzle remains and that is the change in appearance of both Ptah and Hephaestus through successive early pictorial representations. The early pictorial representations of Ptah as illustrated show a youthful appearance and clearly achondroplastic deformity of the statuette. Later reproductions of Ptah however, which became more customary, show a normally-proportioned individual who became known as "the god of the beautiful face" (Fig. 1:3).

Sixth century B.C. pictorial representations of Hephaestus showing his return to Olympus demonstrate a short stature and deformed feet and claw-like toes. This is shown in two separate representations (Fig. 1:4 & 1:5). Later representations however show a change in the appearance of Hephaestus, who is now shown as a bearded man riding a horse with no obvious lower limb deformity. Other representations of about 470 B.C. show that although Hephaestus who is still bearded is requiring support to walk, he is not the "god of the crooked foot" previously shown (Fig. 1:6) and the drawings of 435-430 B.C. illustrate a well-developed body and a pensive dignified expression of the mature Hephaestus. Possibly one leg is thinner than the other from muscle wasting due to chronic denervation, but there is no other obvious deformity to be seen but again there is a hint that he needs the support of a satyr (Fig. 1:7).
Figure 1: Early representations depict Ptah with youthful but clearly achondroplastic features. It was unusual that a Greek god should be deformed and in any way imperfect.
Figure 1: The Egyptian god Bes, the protector of children and the buffoon of the gods. This statuette demonstrates certain achondroplastic features which Hephaestus may have inherited.
Figure 1:3. A reproduction of the more customary representation of Ptah, the "god of the beautiful face".
Figure 1: The Return of Hephaestus to Olympus. Note the bilateral foot deformities, possibly neurogenic in origin.
Figure 1:5. The Return of Hephaestus to Olympus. Note the short stature, deformed feet and claw-like toes. Caeretan Hydria from Cerveteri, second half of the sixth century B.C. He was lame, hobbled and limped possibly as a result of injuries sustained at the hands of Zeus.
Figure 1:6. By 470 B.C. Hephaestus has grown in stature as a god. As a result he is depicted by vase makers and painters with a well-developed body and a pensive dignified expression. One leg is possibly thinner than the other, however, and he still requires help walking. The thin leg may represent muscle wasting from spinal stenosis.
Figure 1:7. Representations of Hephaestus of 435-430 B.C. Again one leg appears thinner than the other but the only evidence of disability is the hint that he needs the support of a satyr. This disability was probably the result of trauma to the spine as he was hurled from Olympus by Zeus (Homer) causing stenosis of an already narrowed spinal canal.
We see therefore a remarkable transition from an uncouth markedly deformed youthful early Hephaestus to the serene dignified later Olympian. Malten's thesis is that the crude twisted deformities of the feet occasionally found in reproductions of Hephaestus are simply attempts of the vase makers and painters to express and to symbolise the dwarf-like nature and deformity which they know to have existed from ancient myths, and do not represent the actual deformity but that they are merely an indication of an important underlying defect (Malten 1913). As Hephaestus increased in stature and importance as a Greek god and Olympian the artists represented him as a well-proportioned muscular individual with normal facial features, but still requiring support, presumably through weakness in the legs on walking or intermittent neurogenic claudication (Fig. 1:6 & 1:7).

Long before Charcot's first description in 1859 of intermittent claudication of vascular origin (Charcot 1859), Portal had in 1803 already focused attention on changes in the size of the spinal canal as possibly being responsible for compression of its contents (Portal 1803) (Fig. 1:8 & 1:9). However Charcot's first description of intermittent claudication in 1859 became more well-known. He described lower extremity vascular insufficiency in a man with an aneurysm of the aorta, and he compared this with a similar situation found in a carriage-horse. Walking a short distance produced a painful limp, rest brought prompt relief. He stated this was due to ischaemia of the muscles and nerves of the legs. In 1862 Raynaud introduced the element of vasomotor spasm due to dysfunction of the sympathetic nervous system into this consideration (Raynaud 1862), and in 1898 Erb added a third type of peripheral vascular disease known as thrombo-angiitis obliterans (Erb 1898). Intermittent claudication was explained by some observers on the basis of inadequate blood supply to muscles and by others to a disturbance of innervation. Bramwell in 1886 stated that:

"paraplegia sometimes results from the sudden stoppage of the blood supply to the lower end of the cord, an accident which occurs in the course of some abdominal aneurysms in consequence of detachment of the clot or obstruction of the abdominal aorta. In some cases of aortic regurgitation, weakness of the lower extremities - never so far as I know amounting to true paralysis - is seen. The motor weakness probably results from anaemia of the lumbar region of the cord" (Bramwell 1886).

This concept was taken a step further by Dejerine in 1894 and
SUR LA

CLAUＤIСATION INTERMITTENTE

OBSERVÉE

DANS UN CAS D’OBLITÉRATION COMPLÈTE DE L’UNE DES ARTÈRES
ILIQUES PRIMITIVES;

Note lue à la Société d’Histoire.

PAR M. LE DOCTEUR CHARCOT,

 Médecin du bureau central des hôpitaux, secrétaire de la Société de Biologie,
 ancien chef de clinique à la Charité.

L’observation intéressante, à plus d’un titre, qui fait l’objet de la pré-
sente communication, nous parait pouvoir être rapprochée des cas de
claudication intermittente par oblitération artérielle, bien connus
aujourd’hui en pathologie hippique (1), et sur lesquels nos collègues,
MM. Goubaux et H. Bouley, auxquels on doit d’importants travaux sur

(1) M. Bouley jeune a le premier, je crois, appelé l’attention sur la claudi-
cation intermittente par oblitération artérielle chez le cheval. (Acad. de Mé-
decine, séance du 18 octobre 1831, et Arch. génér. de médecine, t. XXVII,
p. 472, 1831.) — M. Goubaux a ensuite traité le sujet d’une manière très-com-
plée dans son important mémoire sur les paralysies du cheval causées
par l’oblitération de l’aorte postérieure. (Recueil de médecine vétéri-
naire, t. XXIII, p. 576, 1846.) — On trouve aussi plusieurs articles concer-
cnant cette espèce de claudication dans le Journal de Gurlt et Hertwig (Nagazer
— Voir aussi Romberg, Lehrbuch der Nervenkrankheiten, etc. 2. A. M.,
p. 13.

Figure 1:8. The front sheet of Charcot’s publication of 1859 describing
intermittent claudication of vascular origin.
Fig. 7. Portal (1803) was the first to mention narrowing of the vertebral canal, even in the absence of signs or symptoms (arrow).

Figure 1:9. More than fifty years before Charcot's classic publication on Intermittent Claudication, Portal realised that narrowing of the spinal canal could cause compression of its contents with resulting weakness, numbness and paralysis of the lower extremities. This was the first report in the medical literature of spinal stenosis, in 1803.
again in 1911 when he described a rare form of "intermittent claudication of the spinal cord" (Dejerine 1911). This he stated mimicked vascular claudication. He initially described a thirty-seven year old woman with a variable spastic paraparesis of one leg with increased reflexes and extensor plantar response brought on by walking. It was relieved by standing still for a few minutes and he deduced that there was intermittent ischaemia of the spinal cord due to vasculitis which could be syphilitic. He felt there was a progressive occlusion of the arteries to the lower cord attributable to arteritis (presumably syphilitic) with the legs becoming weak unilaterally on exertion, and bilaterally promptly on walking. He noted on examination of the resting patient that nothing abnormal was found, but during a period of fatigue a certain degree of spasticity was evident. Tendon reflexes became hyper-active in response to walking. Babinski's sign appeared on exertion and disappeared on rest. Deep and superficial sensibility was intact, but urinary symptoms sometimes appeared with loss of libido or with hyper-excitability and premature ejaculation. The sphincter may or may not escape. Finally permanent spastic paraplegia supervened.

At approximately the same time, the first published case of cauda equina compression produced by a herniated disc was described by Oppenheim and Kause in 1909 (Oppenheim and Kause 1909), and in 1911 Goldthwaite observed that cauda equina compression could be precipitated by hyper-extension of the low back (Goldthwaite 1911). Sumita in 1910 described stenosis of the spinal canal in chondrodystrophia fetalis and noted that others had previously described "frontal" stenosis of the spinal canal in such cases (Sumita 1910). As early as 1913 Thomson measured the AP diameter of the vertebral foramen (Thomson 1913).

Elsberg in 1913 operated on a patient following trauma for nerve root compression and found marked enlargement of the ligamentum flavum. He removed this at operation and the patient had complete relief of symptoms (Elsberg 1913).

It was not until 1925 however that Donath and Vogl first described important morphological changes in the achondroplastic spine (Donath and Vogl 1925). These consisted of shortening of the pedicles and reduction of the interpedicular distance with consequent narrowing of the spinal canal in both diameters. These bony changes caused by the basic failure of cartilage growth were thought to be confined to that disorder and to account for the high incidence of cord and nerve root compression.
Junghanns in 1930 described another cause of lumbar spinal stenosis when he described eleven patients with degenerative spondylolisthesis or "pseudospondylolisthesis" (Junghanns 1930). He found that in many of the specimens the angle between the pedicle and the plane of the facet approached 180°. In 1934 Elsberg and Dyke began to calculate the transverse diameter of normal vertebral canals by measuring the interpedicular distance as seen on the AP radiograph (Elsberg and Dyke 1934).

By this time however Dr. Barr, working one Sunday morning in 1932 at the Massachusetts General Hospital Pathology Laboratory in Boston, had begun to appreciate that the excised material thought to be a chondroma pressing on the nerve root was in reality a herniated disc (Barr 1961).

Howard Brown writing in the Journal of Bone and Joint Surgery in 1938 however found it impossible to distinguish between patients with a prolapsed intervertebral disc and those with an enlarged ligamentum flavum. He stated that:

"the history and symptoms of patients with enlarged ligamenta flava are essentially the same ... as those with prolapsed discs ... and clinical differentiation between the two conditions is extremely difficult or impossible. In many instances too the syndrome cannot be differentiated clinically from lumbo-sacral or sacro-iliac disorders and careful study is required before a diagnosis can be reached" (Brown 1938).

The situation became further confused when Frederick Reichert reported four non-syphilitic patients with radiographic evidence of marked arteriosclerosis of the lower abdominal aorta with recurring weakness in the lower limbs on exertion, unaccompanied by cramps or by obliteration of arterial pulsation in the foot and ankle. He again postulated ischaemia of the spinal cord due to arterio-sclerosis involving the segmental branches from the aorta. To confirm this hypothesis he ligated these arteries in an experimental animal and claimed to have produced an identical syndrome (Reichert 1934).

However Grafton Love in 1940 emphasised again the importance of the hypertrophied ligamentum flavum using graphic descriptions of the pathological changes observed (Love 1940). He stated that:

"these changes consisted of marked thickening, increased firmness and decreased elasticity of the ligamentum such that when it was cut the ligament imparts an entirely different type of resistance from that encountered in the resection of the normal ligamentum flavum, and the cut surface appears whitish-grey rather than yellow".

However
Grossly the affected ligaments he stated were thicker than normal measuring 4-6 mm in cross-section against an established normal of 2-5 mm, with white bands running throughout. Microscopically, examination revealed fragmentation usually longitudinal of the elastic tissue fibrils, with the ends of the frayed fibrils appearing shrivelled. There was replacement of damaged fibrils by more primitive mesoderm, namely fibrous connective tissue which in many instances ran as wide bands throughout the length of the affected ligament. He noted the blood vessels were few and small in calibre with a hyaline substance deposited within the walls of the muscularis, and he postulated that vascular change as a result of trauma might explain the other changes. This vascular change he referred to strangely affected exclusively the ligamentum flavum.

Sarpyener in 1945 reminded surgeons of the "congenital stricture of the spinal canal" (Sarpyener 1945). He noted peripheral nerves may be compressed by sudden pressure from a fractured bony fragment and gradual pressure of exuberant callus or the slow envelopment of scar tissue. He considered that the spinal canal may also become narrow in certain other conditions. He stated that the spinal canal is normally narrow in three regions that is above, below and between the cervical and lumbar enlargements. He postulated that in spina bifida occulta there is a stricture of the cord which caused enuresis of varying grades, pains similar to those in lumbago and sciatica with atrophy of the muscles, skin changes similar to vitiligo, and perforating ulcer. He reasoned that the congenital stricture may cause spastic or flaccid paralysis and that this was dependent not on the location of the lesion, but on the degree of pressure exerted on the cord, and he described four forms of congenital stricture.

1. Ring-like stricture at one or more levels.
2. More extensive stricture usually associated with spastic paralysis similar to that seen in Little's disease.
3. Localised stricture. He felt this type was responsible for club foot deformity.
4. An atypical form causing a cleft either in the cord alone or in the cord below and the dura mater.

He introduced lipiodol into the spinal theca and noted a block at the level of the second or third lumbar vertebra which he understood was the region of the cord controlling bladder function. He noted also that strictures may
occur in the absence of spina bifida occulta, and argued that many club feet of the paralytic type were due to congenital strictures of the canal and could be cured by laminectomy. He described ten patients with spastic diplegia similar to that seen in Little’s disease, in whom there was a lipiodol block. Congenital strictures or narrowing of the canal he stated were responsible for many cases of enuresis, club foot, and spastic and flaccid paralysis leading to abnormalities of gait and contractures. The narrow bony canal through pressure on the cord and by the Law of Delpech prevented its normal development. He stated it was also possible that congenital strictures of the intervertebral foramina may cause pressure on the nerve roots, but that these did not cause pain since they developed very slowly and inhibited the growth of nerves. This was unlike inflammatory constrictions of the nerve which occurred in later life causing sudden pressure against a fully developed nerve and therefore pain. In 1945 Sarpyener enumerated the indications for laminectomy:

1. Enuresis in which, when uncomplicated, laminectomy may be postponed until puberty.
2. All children with enuresis who have reached adolescence without improvement.
3. Children of four or five who without other assignable cause had never learned to walk.
4. Abnormal gait which was getting worse at five or six years old.
5. Atrophy or paralysis combined with abnormalities of the lower limbs.

He described the surgery stating that:

"the stricture must be completely relieved and pulsation return to the dura below the level of the lesion".

This remains good surgical practice. His second recommendation which also is current practice is that:

"if by accident the dura is opened it should be sutured in such a way as not to cause any constriction".

A later publication by Sarpyener in 1947 stated that he had

"made a number of observations which seemed to indicate a causal relationship between stricture of the spinal canal and certain developmental conditions of the hips and knees such as coxa plana, coxa vara congenital, subluxation of the hip, malum coxa juvenalis (Legg-Calve-Perthes disease), malum coxa senilis, and Osgood-Schlatter's disease. It seems to the author that if stricture of the spinal canal can produce atrophy of the soft parts, pain and spasticity, it could also affect the bones by exerting pressure against their trophic nerves" (Sarpyener 1947).
He considered that the occurrence of these conditions during childhood and early adolescence supported this theory. He reasoned that it was during this time, particularly adolescence when the narrow spinal cord was contained within a growing spinal canal, that the lesions would tend to heal. In Sarpyener's experience narrowing of the canal was often found at a considerable distance from spina bifida when this was present.

In 1949 however Verbiest reported narrowing of the lumbar vertebral canal not in association with any other anomaly of the spine in seven male patients whose ages ranged from thirty-seven to sixty-seven (Verbiest 1949). The patients he described had complaints which could be misinterpreted as intermittent claudication of vascular origin and he felt that the anomaly could not be recognised on plain radiographs. Neurological examination was normal, Laségue's test was negative in four patients and positive in only one with a marginal positive result in two patients. The interpedicular distance was normal in these patients and he concluded that the narrowing was confined to the sagittal plane producing a complete block as revealed by myelography or that the constriction of the canal occurred behind the pedicles. He considered the lesion to be developmental and quoted the cervical rib and Arnold-Chiari mal-formation, and the narrowing of the achondroplastic vertebral canal to explain why the disturbance only became manifest in adults. He felt that:

"in the writer's humble opinion the ligamentum flavum is most unlikely to contact any spinal root unless this root is distorted from its regular path," and this was quoted by Jackson (Jackson 1948). He noted arthritis of the vertebral joints radiographically but although he felt at operation that these changes appeared to have no part in the causation of the narrowing, he stated that changes were most pronounced in the L3 and L4 areas which accounted for the normal canal being narrowed in this region, and he did postulate that it seemed justifiable to ascribe the cause of the narrowing of the spinal canal to encroachment of the articular processes on the vertebral canal. Verbiest described graphically the clinical picture, radiographic findings, and myelographic complete block of the extradural compression of spinal stenosis.

In 1961 Blau and Logue described an unusual syndrome resulting from central protrusion of a lumbar intervertebral disc which gave rise to symptoms suggesting "intermittent claudication of the cauda equina" (Blau and Logue 1961). In 1966 Joffe and others described two further patients with intermittent claudication of the cauda equina (Joffe 1966).
A search was subsequently made for features suggestive of this diagnosis which would make it easily recognisable on plain radiographs. Schlezing and Taveras in 1953 regarded an abnormal reduction of the interpedicular distance as of great significance in their patients (Schlezing and Taveras 1953), but in 1954 Verbiest found the interpedicular distance normal, and concluded that the narrowing was in the sagittal plane (Verbiest 1954). In 1962 Epstein reported twenty-nine patients with symptoms related to narrowing of the lumbar spinal canal (Epstein 1962). In eighteen the sagittal diameter was 1.5 cm. or less, and the interpedicular distance was at the lower limit of normal in ten of their patients, and they stressed the significance of relative flattening of the intervertebral foramina as shown in the lateral radiographs. Teng and Papatheodorou in 1963 reported thirty patients with spondylosis of the lumbar spine, in whom they describe narrowing of the lumbar canal due to reduced antero-posterior diameter (Teng and Papatheodorou 1963), and in 1967 Hancock recorded the finding of short pedicles in the narrowed lumbar canal, but he also noted a reduction in the interpedicular distance at L4 and L5 in one patient (Hancock 1967). Schatzker and Pennal in 1968 noted that several of their patients described as having developmental stenosis had in fact a reduction in the antero-posterior diameter of the lumbar canal (Schatzker and Pennal 1968), and in 1969 Clark stressed that the interpedicular distance may be reduced at all levels and may account for considerable difficulty at operation (Clark 1969).

Finally in the 1970's two outstanding changes have influenced the management of spinal stenosis. Firstly, significant advances have occurred in investigative methods. Safer water-soluble contrast agents have become available leading to improved definition of nerve roots. Computerised axial tomography has heralded a revolution in non-invasive imaging of the spinal canal, and ultrasonic measuring devices have been applied to the spine and in places epidural venography has attained some popularity. The role of magnetic resonance imaging remains to be defined in this condition.

Secondly, there has been a growth of interest in the clinical problems attributable to spinal stenosis and an emerging awareness of the different symptoms and signs produced by stenosis of the central part of the spinal canal, of the lateral recess and of the nerve root foramen.

It is essential that the terminology used in description of spinal stenosis should be clear and unambiguous to permit accurate classification of the condition and precise planning of operative procedures.
CHAPTER 2

DEFINITION OF SPINAL STENOSIS

The word "stenosis" is a Hellenised Latin word derived from the Greek "stegnosis". The Greek word "stenos" probably meant "narrow, closed, or locked-up", and the suffix "-osis" was used in the Greek language to express a pathological condition. The word "stenosis" subsequently became less popular however and does not reappear prominently in the medical literature until the early nineteenth century. At this time there was considerable interest in etymology and in a study of comparative and historical linguistics. In 1831 it received mention for the first time in the second edition of the Medical Lexicon produced by the German critic Kraus (Kraus 1831).

The adjective "stenos" was linked with other words by Galen and Aristotle to indicate pathological conditions of abnormal narrowness, such as "stenopores" for small orifices, "stenotos" for narrowing of the oesophagus, or "steno-phlebos" for small veins (Skinner 1970). The word "stegnosis" was used by Dioscorides in the first century A.D. to express "the fact of rendering constipated" and by Galen to signify a complete obstruction of the passages of substances through anatomical ducts caused either by constriction, by adherence of all parts of their walls or by condensation of their contents.

Morgagni used the word frequently to describe a great variety of pathological narrowing of vascular or mucosal channels at a time when great interest was being shown in the relationship between stricture, function, and pathology (Morgagni 1761). The word seemed ideal to describe the narrow channels observed. When used to describe the narrowing of mucosal ducts, the term "stenosis" was applied loosely and synonymously with "constriction", "striction", or "narrowness". However in the description of the orifices of the cardiac valves or of arteries the term "stenosis" was applied more strictly, and became the only word used to describe narrowing of these structures. Throughout this period the term was limited to conditions in which the transportation of fluids, solids or gases was impeded.

During the first half of the twentieth century a semantic drift became evident with the use of words such as "craniostenosis". Such words did not retain favour for long however and were replaced: "craniostenosis" thus became "craniosynostosis".
The term "stenosis" was first applied to the spinal canal by Sumita in 1910 to describe the narrowing of the canal in the chondrodystrophia foetalis (Sumita 1910). This is the first time the word "stenosis" was used to describe the narrowing of a bony structure which did not directly transmit fluids, solids or gases. One could however of course note that the spinal canal does allow room normally for the transportation of blood, cerebrospinal fluid, and axonoplasm. Nonetheless its use as a descriptive term for the spinal canal does indicate a semantic drift which on this occasion became more widely adopted.

More recently in 1976 an international group of orthopaedic surgeons with a particular interest in the spine produced a definition which was designed to clarify and delineate the term "lumbar spinal stenosis". This they defined as:

"any type of narrowing of the spinal canal, nerve root canals (or tunnels) or intervertebral foramina. It may be local segmental or generalised. It may be caused by bone or by soft tissues, and the narrowing may involve the bony canal alone or the dural sac or both. Disc herniations which have in the past been considered as a distinct and separate entity are included in this classification when they occur together with other types of stenosis. Space occupying lesions due to the products of inflammation or neoplasm are in the strictest sense types of "stenosis" but are considered to be beyond the scope of this definition" (Arnoldi 1976).

The term "stenosis" when applied to the lumbar spine thereby became all inclusive covering not only bony stenosis but also arachnoiditis, intrathecal neoplasms, as well as disc prolapses in association with bony narrowing. This makes the Oxford English Dictionary definition of "stenosis" as "the contraction or stricture of a passage, duct or canal" appear outdated. That is unless one considers that stenosis occurs at the microscopic level and the duct which is undergoing contraction or stricture to be axon which normally allows a flow of axonoplasm. Alternatively it may be the flow of blood, arterial or venous, or cerebrospinal fluid which is affected by the stricture of the canal. I hope to demonstrate in this thesis that these are in fact profitable perspectives from which to view spinal stenosis.

This internationally agreed classification again stretched the word "stenosis" beyond recognition by applying the qualifying adjectives "central and peripheral". If the term "stenosis" is applied to a single canal, then clearly narrowing can occur in the peripheral zone which forms the walls of the canal. Alternate descriptions have been suggested, "medial" and
and "lateral" stenosis or "central" and "lateral" stenosis. Fortunately however this anatomical conundrum did not last. The term "central canal stenosis" remained and the more descriptive term "nerve root canal stenosis" became popular, and between the two the term "lateral recess stenosis" became acceptable.

The lateral recess is certainly not a canal or duct by any stretch of the imagination being simply a narrowing of the lateral part of the central canal as it becomes trefoil, and therefore having only two and in places at the most three walls.

Thus the borrowing of scientific words from classical language has not always been successful. Many classical words have such explicit meaning that confusion cannot be avoided. Unfortunately current use of the term "stenosis" is an example of the adoption of an old medical term which has been poorly defined from the start. The ambiguity of its meaning has been compounded by semantic drift which has given rise to a great number of competing synonyms. Why the term "stenosis" has become so firmly attached to descriptions of narrowing of the vertebral canal however remains unclear and may be more a reflection of fashion in medical circles of the type ridiculed as early as the 17th century by Moliere and Montaigne, than on any strict scientific or etymological justification.

Attempts have been made to define narrowing of the spinal canal both radiologically and directly in absolute values. As recently as 1976 Verbiest stated that interpedicular distances smaller than 17 mm and mid-sagittal diameters smaller than 12 mm are too short (Verbiest 1976). He based this conclusion on a "fair number of measurements" made on the vertebrae of American, Dutch, Norwegian, Lapp and Zulu skeletons. Absolute measurements seem unimportant however when dealing with either the individual patient with symptoms, signs and myelographic findings of spinal stenosis or with society at large where screening procedures have been proposed to aid job selection, for instance the ultrasonic screening of the lumbar spine of miners. What is clearly important in symptomatic spinal stenosis are the neurological effects.

Nerve root size differs from individual to individual, and between the races and the sexes. Nerve roots not bony canals must be the ultimate arbitor of the presence or absence of spinal stenosis. It is therefore not unreasonable to suggest that the ducts or canals which become stenosed or narrowed are in fact the axons.
The warning of Bronowski must be heeded: "a science which orders its thoughts too early is stifled" (Bronowski 1959).
CHAPTER 3

CLASSIFICATION OF SPINAL STENOSIS

Recently lumbar spinal stenosis has attracted more attention because of the important correlations observed between structural changes within the spines of cadaver specimens, radiological studies, and the clinical symptoms and signs of affected patients. The recognition and management of this condition demands a clear understanding of diverse anatomical changes within the spine and an appreciation of the wide spectrum of fluctuating clinical manifestations and a careful correlation between these and their radiological representations.

The adoption in the literature of colourful terminology has not helped in this clear understanding of pathology. For instance, spinal canal stenosis is known also variously as pseudoclaudication, intermittent cauda equina compression, spondylotic caudal radiculopathy and neurogenic intermittent claudication to name but a few, whilst lateral recess stenosis is also known as lateral recess entrapment or "occult spinal stenosis". The term "entrapment" was used because it was uncertain whether pain or neurological deficit was due to compression, traction, irritation, or inflammation of the nerve roots. The process may involve not only the spinal nerve but also the branches of the sinuvertebral nerve of Luschka. Disc herniation has been included in the classification when it occurred together with other types of stenosis, but not when it occurred alone in an otherwise spacious spinal canal. Space occupying lesions such as infections or neoplasms can produce stenosis of the canal, but are not generally considered within the scope of most classifications of spinal stenosis (Grabias 1980).

Verbiest in 1954 introduced the term "developmental stenosis" and distinguished this from congenital stenosis (Verbiest 1954) to indicate a genetic disturbance that revealed its pathological effects only when growth was complete and the vertebra had attained its full size. Verbiest felt that most cases of so-called "acquired stenosis" were in fact superimposed on pre-existing "developmental stenosis". It would seem reasonable to distinguish between congenital and developmental stenosis, with congenital stenosis due to disturbed foetal development occurring in such instances where there is a congenital malformation of the lumbar spine, as described by Sarpyener (1945, 1947) and later Verbiest (1976).

It must be appreciated however that little is known about the
normal size of the canal during the years of growth (Bowen 1978).

When a distinct localised congenital anomaly of the lumbar spine is not evident however spinal stenosis can still exist. Five different types of congenital and developmental anomalies have been recognised (Bowen 1978).

1. The constriction ring and localised area of narrowing described by Sarpyener (Sarpyener 1945, 1947), but these appear extremely rare and many authors claim to have no experience of them.

2. Uniform stenosis involving the whole of the lumbar spinal canal. This is the most common type. The antero-posterior diameter seen on the lateral radiograph is more affected than the lateral diameter seen on the antero-posterior view. When the antero-posterior diameter is less than 15 mm. this is described as mild stenosis, and when it is less than 10 mm. it is described as severe stenosis (Bowen 1978).

3. Uniform stenosis involving the L4 and L5 segments only.

4. Uniform stenosis involving the L2 and L3 segments only. This is uncommon.

5. Shingling of the lamina usually at the L4-5 or L5-S1 level or both of these. The laminae pass upwards and forwards at an angle of 30°-45°. This is not uncommonly seen in spondylolisthesis and appears to be a developmental phenomenon. The localised stenosis thus produced is never very marked.

On the basis of measurements of spinal canal antero-posterior diameters, Verbiest identified three types of lumbar stenosis.

1. "Pure absolute stenosis." In this group all mid-sagittal diameters of the narrowed portions of the spinal canal were 10 mm. or less and he identified forty-one cases of pure absolute stenosis.

2. "Pure relative stenosis." All mid-sagittal diameters of the narrow portion of the spinal canal varied between 10 mm. and 12 mm. (twenty cases).

3. "Mixed stenosis." Both absolute and relative stenosis existed at different levels and he identified twenty-eight cases of mixed stenosis (Verbiest 1975).

The mid-sagittal diameter at the caudal border of the vertebral arch was usually larger than that of the cephalad side. The ratio between the mid-sagittal diameters at the cephalad and caudal borders (the RMD) was usually less than 1. Verbiest felt that values greater than 1 could be considered abnormal or dysmorphic, but he admitted that the lower limit
of normal for this value was not known. He felt that the occurrence of
an abnormal RMD ratio was characteristic of lumbar stenosis.

Verbiest also noted that the interpedicular distances were normal in
all cases and the vertebral bodies did not contribute to the stenosis, and
so he attributed stenosis to changes within the vertebral arch resulting
from short pedicles, hypertrophic medially displaced articular processes, or
disproportionate or thickened lamina. He again classified these into:

1. Wholly stenotic arches with relative or absolute stenosis existing at
   both the cephalad and caudal borders.
2. Half a stenotic arch with stenosis present at one of the borders
   whereas the mid-sagittal diameter at the other border was within
   normal limits.
3. He did identify a third group in which absolute stenosis was found
   at one border and relative stenosis at the other and he called these
   "mixed stenosing arches". More often the area of stenosis involved
   two vertebral arches and in these the upper and lower terminal
   stenosing arches he felt marked the transition from normal to
   stenotic areas.

He also noted that the interlaminar space was important particularly
with regard to disc protrusions which become more readily compressive
when localised to a narrowed interlaminar space. Of all the cases he
described, half the patients with pure relative stenosis presented with
stenosis of only one vertebral arch, whereas this was noted in only one
case of mixed stenosis. With pure absolute stenosis, solitary stenosing
arches occurred in nine out of forty-one cases. Involvement of two or
three vertebrae was most frequent with pure stenosis (twenty-seven out
of forty-one cases) and mixed stenosis (twenty out of twenty-eight cases).

Epstein described spinal stenosis much more simply as either general,
segmental, or local, and also emphasised the importance of the lateral
recess and foramenal canals, pointing out that the intervertebral foramen
was not simply an opening but was a definite canal (Epstein 1977).

Three types of pathological changes are identified in lateral canal
"stenosis" (Bowen 1978).

1. Developmental narrowing of the lateral recess medial to the foramen
   at the L4-5 or L5-S1 level. This is one part of the change seen in
developmental stenosis.
2. Enlargement and anterior subluxation of the superior articular
   process of L5 or of the upper sacrum. Degenerative changes in the
disc result in loss of disc height, and over-riding of the articular processes.

3. When, in the way described above, disc and posterior joint instability is marked, this permits recurrent rotational movements of one vertebra upon another. In this way the superior articular process moves upwards and forwards with recurrent narrowing of the recess and entrapment of the nerve root laterally.

To balance the complexity of absolute measurements of the spinal canal be it at the caudal or cephalad end of the vertebral arch in an attempt to classify spinal stenosis, the basic definition of stenosis must be recalled. The Oxford English Dictionary defines stenosis as "the contraction or stricture of a passage, duct or canal". The history of this term is given in Chapter 2.

In common with strictures of the common bile duct, narrowing of the coronary arteries and compression of the median nerve at the carpal tunnel, stenosis of the spinal canal becomes important only when it results in interference with the normal function of the contents of the canal. Measurements of bony canal diameter represent merely one side of the equation, and the other side of the equation is the volume of the nervous tissue contained within the canal, and the variations that exist between individuals of the same and different racial groups and sexes. A more detailed analysis of these differences is provided in Chapter 5.

Suffice it to say that at present narrowing of the spinal canal seems to be a normal part of advancing age, and absolute measurements of the spinal canal diameter either radiologically or at operation or in cadaver spines can act only as a rough guide to the condition.

The classification of spinal stenosis which has been most widely adopted is known as the "International Classification". This was founded on a practical and clinically orientated basis. The aetiological factor determines the type of stenosis rather than absolute measurements of the degree of narrowing or levels or location. This is by far the most practical and useful classification and is shown in Table 3:1.

Clearly the proportion of patients falling under each category of this classification varies enormously from one centre to another. An example of a breakdown of patients into this classification in a specialised spinal centre in the United States of America is shown in Table 3:2, and in England is shown in Table 3:3.
TABLE 3:1

INTERNATIONAL CLASSIFICATION OF SPINAL STENOSIS

1. Congenital - developmental stenosis
   a) Idiopathic
   b) Achondroplastic

2. Acquired stenosis
   a) Degenerative
      i) Central portion of spinal canal
      ii) Peripheral portion of canal, lateral recesses and nerve root canals (tunnels)
      iii) Degenerative spondylolisthesis
   b) Combined
      Any possible combinations of congenital/developmental stenosis, degenerative stenosis and herniations of the nucleus pulposus
   c) Spondylolisthetic/spondylolytic
   d) Iatrogenic
      i) Post-laminectomy
      ii) Post-fusion (anterior and posterior)
      iii) Post-chemonucleolysis
   e) Post-traumatic, late changes
   f) Miscellaneous
      i) Paget's disease
      ii) Fluorosis

(Arnoldi 1976)

TABLE 3:2

<table>
<thead>
<tr>
<th>No. of Patients</th>
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<tbody>
<tr>
<td>1a) Congenital developmental idiopathic</td>
</tr>
<tr>
<td>2a) Acquired stenosis, degenerative</td>
</tr>
<tr>
<td>i) Central portion of spinal canal</td>
</tr>
<tr>
<td>ii) Peripheral portion of canal, lateral recesses and root tunnels</td>
</tr>
<tr>
<td>iii) Degenerative spondylolisthesis or pseudospondylolisthesis</td>
</tr>
<tr>
<td>2b) Any possible combination</td>
</tr>
<tr>
<td>2c) Spondylolisthetic or spondylolytic</td>
</tr>
<tr>
<td>2d) Post-operative</td>
</tr>
<tr>
<td>i) Post laminectomy</td>
</tr>
<tr>
<td>ii) Post fusion</td>
</tr>
<tr>
<td>iii) Post chemonucleolysis</td>
</tr>
<tr>
<td>2e) Post-traumatic late changes</td>
</tr>
<tr>
<td>2f) Paget's disease and fluorosis</td>
</tr>
</tbody>
</table>

(Brodsky 1976)
Figure 3:1. Diagramatic representations of the seven major types of spinal stenosis. The stippled areas represent the pathological areas.
TABLE 3:3  CLASSIFICATION OF SPINAL STENOSIS MANAGED SURGICALLY AT THE
NUFFIELD ORTHOPAEDIC CENTRE

<table>
<thead>
<tr>
<th></th>
<th>MALES</th>
<th>FEMALES</th>
<th>TOTAL NUMBER OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital - developmental stenosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Idiopathic</td>
<td>4</td>
<td>1</td>
<td>5 (7%)</td>
</tr>
<tr>
<td>(b) Achondroplastic</td>
<td>0</td>
<td>1</td>
<td>1 (1%)</td>
</tr>
<tr>
<td><strong>Acquired stenosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Degenerative</td>
<td>16</td>
<td>5</td>
<td>21 (30%)</td>
</tr>
<tr>
<td>i) Central stenosis</td>
<td>3</td>
<td>2</td>
<td>5 (7%)</td>
</tr>
<tr>
<td>ii) Lateral stenosis</td>
<td>4</td>
<td>9</td>
<td>13 (18%)</td>
</tr>
<tr>
<td>(b) Combined - any combination principally disc herniation and degenerative</td>
<td>9</td>
<td>4</td>
<td>13 (18%)</td>
</tr>
<tr>
<td>(c) Spondylolisthetic - spondylolytic</td>
<td>0</td>
<td>0</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>(d) Post-operative</td>
<td>9</td>
<td>2</td>
<td>11 (16%)</td>
</tr>
<tr>
<td>i) Post-laminectomy</td>
<td>0</td>
<td>1</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>ii) Post-fusion</td>
<td>0</td>
<td>0</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>(e) Post-traumatic, late changes</td>
<td>1</td>
<td>0</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>(f) Paget's disease</td>
<td>1</td>
<td>0</td>
<td>1 (1%)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>47</td>
<td>25</td>
<td>72</td>
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</tbody>
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CHAPTER 4
AETIOLOGY OF SPINAL STENOSIS

INTRODUCTION

In this chapter the factors responsible for the development of spinal stenosis will be discussed using the International Classification described in Chapter 3. Details of anatomical work, physiological studies, biomechanical factors, and the immunology of disc degeneration will not be considered here since they will be covered in some detail in subsequent chapters.

HYPERTROPHY OF THE LIGAMENTUM FLAVUM

As early as 1913 Elsberg described a syndrome consisting predominantly of low back pain with pain radiating into one or both lower extremities (Elsberg 1913). As a result of anatomical studies he considered that this was at least partly due to a greatly increased thickness of the ligamentum flavum. The ligamentum flavum, composed normally of yellow elastic tissue, connects the laminae of contiguous vertebra, and may at times undergo hyperplastic change becoming so increased in thickness that it encroaches upon the spinal canal, thereby compressing its contents (Elsberg 1913). Spurling in 1937 reported seven cases in which this lesion was he considered the pathological entity (Spurling 1937). In each patient there was a history of trauma which the patient considered to be the causative factor, but in none of the patients was pain in the back relieved by the recumbent position. Most of them were more comfortable whilst sitting or standing, whereas coughing, sneezing or straining at stool frequently augmented the pain. Radiation of the pain into one or both lower extremities was present in all seven cases. In two instances it was referred to the hip and testicle. Sexual impotence was complete in three patients. Numbness, either in the legs or buttocks, was present. Urinary incontinence was present in all but one patient of the group. One patient had a trophic ulcer on the heel. The Achilles tendon reflex was abolished or diminished in six of the seven cases (Spurling 1937).

Spurling recommended treatment of the lesion by removal of the involved lamina and ligament. He stated that he had regularly removed the lamina above and below the lesion for the purpose chiefly of adequate exposure. The lamina of the fourth lumbar vertebra was completely
sacrificed, including its superior articular facets. The wide exposure was necessary

"in order that the lateral portion of the thickened ligament may be removed".

He also noted that the lamina of the fourth lumbar vertebra was thickened, and this was in fact the first gross abnormality noted at surgery. He considered the lamina was fifty to one hundred per cent thicker than normal, and predural fat was always normal above and below the lesion and absent beneath it. Spurling concluded that the ligamentum flavum had probably been traumatised in the beginning, and in the process of healing or as the result of continuous trauma an excess of scar tissue was laid down.

It is clear from Spurling's description that although he considered the most important priority of surgery was to remove the thickened ligamentum flavum, he was in fact performing a full bony decompression by laminectomy and going as far as facetectomy to ensure total removal of the ligamentum flavum. Apart from the one fatality from streptococcal meningitis, he claimed good results with resolution of the lesion following removal of the lamina and ligament.

More recently Crock, describing root canal stenosis, indicated that the apex of the superior facet of the inferior vertebral body moved upwards to abut on the pedicle of the superior vertebra at the interspace (Crock 1976). He stated that this results inevitably in deformation of the intervertebral foramen, and

"buckling of the ligamentum flavum into the foramen and into the nerve root canal".

Crock therefore emphasised the importance of the ligamentum flavum in compromising the intervertebral foramen. He stated that the intervertebral foramen is a portal with dimensions set in a single plane somewhat oblique to the sagittal plane of the body, but by contrast the nerve root canal must be viewed in three dimensions. By definition it commences superiorly at the origin of the nerve root sleeve from the dural sac. Its roof at this point is formed by the inner margin of the superior articular facet, the contiguous attachments of the ligamentum flavum and the superior margin of the lamina.

The ligamentum flavum is therefore implicated not only in central canal stenosis but also in root canal stenosis. More recent detailed anatomical studies of the ligamentum flavum will be described in Chapter 5.
CONGENITAL AND DEVELOPMENTAL STENOSIS

The features here are of uniform narrowing throughout the length of the lumbar spinal canal which is more marked in the antero-posterior plane than in the transverse plane. It should be noted that congenital and developmental stenosis by themselves rarely if ever cause symptoms, even in achondroplasia, and it is the superimposition of the degenerative process which precipitates symptoms. Myelography in this form of stenosis reveals a generally narrow theca. In the opinion of Paine the lower limit of normal of the antero-posterior dimensions seen on myelography is 14 mm. (Paine 1972). Eisenstein however using measurements of the bony spinal canal rather than the theca seen on myelography, put the figure at 15 mm. as seen radiographically (Eisenstein 1978).

When hypertrophy or enlargement of the articular processes is observed with narrowing of the lateral recess, this represents the development of superimposed degenerative change. McIvor considers that enlargement of the articular processes is not one of the features of developmental stenosis (McIvor 1976).

CONGENITAL SPINAL STENOSIS

This may take the form of a localised spinal stenosis due to a congenital mal-formation of the vertebral column but more commonly exists in the generalised form. Localised congenital stenosis is due to a congenital mal-formation of the vertebral body and/or posterior arches. The term congenital implies that stenosis is present at birth, but in fact stenosis is not caused entirely by the mal-formation alone. It may well be present at birth, but this has not yet been described. Compression commonly manifests itself in adult life when additional factors arise such as thickening of the laminae, hypertrophy of the articular facets or ligamenta flava or slight protrusion of the annulus fibrosus which in themselves would not be sufficient to cause compression symptoms in a normal spinal canal. (Fig. 4:1).

The classical example of congenital spinal stenosis is achondroplasia. The spondylo-epiphyseal dysplasias such as Morquio’s syndrome can give rise to congenital spinal stenosis (Postacchini 1983) (Fig. 4:2). A spur projects downwards from the inferior facet into the foraminal canal. The vertebra are moderately diminished in height because of impaired epiphyseal end plate growth while increased periosteal growth results in broad squat
Figure 4:1. Lateral radiograph of the thoraco-lumbar spine of a fifteen-year old boy with Morquio's disease to show the characteristic features of spondylo-epiphyseal dysplasia. Note the kyphosis with spondylolisthesis at the thoracolumbar junction. He also had subluxing patellae, unstable genu varum bilaterally, hypermobile wrists and suffered recurrent chest infections.
Figure 4:2. Spondylo-epiphyseal dysplasia due to Morquio's disease. At the age of fifteen this boy with unstable genu valgum bilaterally began to go off his feet, developed urinary difficulties, and upper motor neurone signs in the lower limbs only. The myelogram shows significant anterior indentations at each disc level of the thoraco-lumbar junction.
vertebra. Premature fusion of the neurocentral synchondroses results in short heavy pedicles (Lee 1978). The lamina are thick and broad. The dorsal surfaces of the centra are concave, while the intervertebral discs become prominent and project into the spinal canal (Fig. 4:3 and 4:4).

Epidural fat is practically absent. The spinal cord is disproportionately large in as much as it develops normally and available space is diminished. The lowermost two lumbar vertebrae descend deeply between the iliac bones and the sacrum becomes tilted horizontally increasing the lumbar lordosis. As a consequence of diffuse spinal stenosis even slight intrusions can prove catastrophic (Epstein B.S. 1977). Gibbous formation can exaggerate the stenosis. Disc degeneration and spondylo-arthritis change augment compressive changes on the cord, the cauda equina, and the nerve roots. The upper and mid-thoracic segments are rarely affected, and the cervical spine which shows similar characteristics is also frequently a cause of severe symptoms. The other dyschondroplasias apparently are not causative of lumbar or other spinal stenosis (except for atlanto-occipital displacements) (Fig. 4:5).

DEVELOPMENTAL STENOSIS

Developmental stenosis is not associated with the vertebral malformations described in achondroplasia, but may also be subjected to superimposed degenerative changes. The pedicles tend to be short and the lamina convergent (Fig. 4:6a and 4:6b). The embryological basis for failure of the pedicles to attain normal height and orientation is association with inadequate width of the laminar arch remains unexplained (Lee 1978). The intervertebral foramina are moderately narrowed and angulated. Weinstein described eight female patients with developmental stenosis all of average height and weight. In only one case was the patient unusually short and heavy set, as might be expected if developmental lumbar stenosis were associated with atypical body growth pattern. None of these women had a history of having sustained serious spinal trauma or of having engaged in strenuous physical labour (Weinstein P.R. 1983).

The shape and size of the normal spinal canal vary enormously and the borderline between normal and abnormal is frequently ill-defined, however it appears from operative and radiological observations that three different types of developmental narrowing occur: concentric stenosis, sagittal flattening and abnormal articular processes (Fig. 4:7).
Figure 4:3. Spondylo-epiphyseal dysplasia with upper motor neurone signs in the lower limbs. At the level of spondylolisthesis at the apex of the thoraco-lumbar kyphosis there is a complete block in the contrast column. This fifteen-year old boy with Morquio's disease also had atlanto-axial instability and electrophysiological evidence of upper motor neurone changes in the upper limbs.
Figure 4:4. Spondylo-epiphyseal dysplasia with partial block on the myelogram at the level of spondylolisthesis and lateral vertebral translocation seen on the antero-posterior radiographs.
Figure 4:5. The block in the contrast column became partial in the supine position, and the patient, a fifteen year old boy with mucopolysaccharidosis responded to a period of bed rest and bracing.
Figure 4:6 (a) The antero-posterior radiograph of lumbar spine of the fifty-five year old teacher (E.T.) with developmental stenosis. Note six lumbar vertebrae, facet joints close to the mid-line, and shingling of the laminae, but in this case no reduction of inter-pedicular distance. The arrow indicates a developmental anomaly of a facet joint. The patient was six feet seven inches tall and of normal proportions suggesting no reduction of longitudinal growth, despite the definite failure of the pedicles to achieve full height.
Figure 4.6 (b)  

Radiculogram of the same patient (E.T.) confirming the presence of generalised developmental degenerative stenosis. Symptoms are frequently precipitated by disc bulging or sequestration as shown here at the L5-6 level resulting in a one level block in a generally narrowed canal. Surgical decompression should be confined to this level and a full longitudinal decompression not attempted.
DEVELOPMENTAL SPINAL STENOSIS

Figure 4:7.  
A. Normal spinal canal  
B. Concentric stenosis: reduced pedicle height and interpedicular distance  
C. Sagittal stenosis: reduced pedicle height  
D. Unilateral facet joint anomaly  
E. Bilateral facet joint anomaly
1. Concentric Stenosis

This type of pathological condition represents a growth error of the spinal canal with a disproportionately retarded growth of the posterior elements (pedicles, articular processes, and laminae) compared to its neural contents. The spinal canal is contracted concentrically in both the sagittal and coronal planes. The typical myelographic picture shows a narrow column of contrast medium on both the antero-posterior and lateral projections. Plain radiographs demonstrate narrowness of both the inter-pedicular distance and the sagittal diameter of the canal (Lee 1978).

2. Sagittal Flattening

In this instance the error of growth results in disproportionately retarded growth of pedicles. Normal inter-pedicular distance but narrow sagittal diameter is seen on plain radiographs. Myelographic examination shows absent or decreased epidural space between the dura and the posterior surface of the vertebral bodies. On the lateral projection there are smooth ventral indentations of the column of contrast medium at disc level without actual disc protrusion (Fig. 4:6(b)). A.P. projection of the myelogram shows prominent nerve root shadows at these levels.

3. Abnormal Articular Processes

The third type of disease process is probably a developmental error of articular process growth. Stenosis is caused by an abnormally large or abnormally located articular process or processes. Occasionally, this type of stenosis is associated with either of the first two types of growth abnormalities (Fig. 4:8). A developmental anomaly of the articular process(es) is the basis of this type of stenosis, although hypertrophic degenerative arthritis frequently supervenes (mixed type). Myelographic examination demonstrates typical large and smooth lateral indentations of the column at disc level on A.P. projection. When the defects are multiple and bilateral, a "wasp-waist" appearance of the column of contrast medium will be produced. Differentiation from disc pathology may become difficult when the defect is single and unilateral.

This condition generally exists for many years before disc degeneration, spur formation, and spondylo-arthritis change (singly or in combination), together with ligamentous thickening, vertebral displacements, and increased lordosis bring about sufficient structural change to produce clinically apparent stenosis. There are frequently changes in the lumbar and cervical spine.
Figure 4:8. Antero-posterior radiograph and CT scan to illustrate a forty-six year old patient with developmental spinal stenosis from one enlarged facet joint.
Trauma may precipitate these changes and spinal fusion of one segment is usually followed by alterations of the adjacent segments causing localised stenosis at the non-fused segment above and below. It must be remembered that patients with spinal stenosis in the lumbar region may also have stenosis of the cervical spine. One however usually predominates giving either an upper motor neurone or lower motor neurone pattern of signs, followed possibly many years later by the other level predominating.

There have been only three reports of familial lumbar stenosis and none in the orthopaedic literature prior to 1985. Verbiest reported the cases of two brothers (Verbiest 1973) and two sisters (Verbiest 1976) with developmental stenosis, and Varughese and Quartey described the cases of four brothers with acute lumbar disc herniation or herniations and myelographic evidence of narrowing of the lumbar spinal canal in the sagittal plane (Varughese 1979).

Postacchini reported in 1985 the cases of two brothers and a sister with severe developmental or combined lumbar stenosis (Postacchini 1985). In addition, developmental narrowing of the lumbar spinal canal probably existed in at least one other member of the family (Fig. 4:9).

In these cases of familial lumbar stenosis, the stenosis was developmental or combined; all of the patients had abnormal narrowing of the lumbar spinal canal in the sagittal plane at one or more levels. These findings suggest that the dimensions of the spinal canal may be regulated by genetic factors. This is consistent with morphometric studies showing that the mean dimensions of the spinal canal and the lower normal limits vary in different races as well as in different populations of the same race.

In all three siblings described by Postacchini, symptoms of nerve root compression became apparent in the fifth decade of life or later. This supports the view that developmental narrowing alone of the osseous vertebral canal is insufficient to produce compression of the cauda equina. In other words, a distinction must be made between a developmentally narrow canal not producing cauda equina compression, and a stenotic canal causing compression of the neural elements.

Since a developmentally narrow canal has the potential to become stenotic, recognition of a family predisposition to developmental and combined lumbar stenosis might be of practical use. Porter has used diagnostic ultrasound screening of mining recruits and nurses to identify those with reduced spinal canals who may be at risk (Porter 1978 a). Decompressive laminectomy completely relieved the radicular symptoms in
FAMILIAL DEVELOPMENTAL LUMBAR SPINAL STENOSIS

Figure 4:9. Reports in the literature of Familial Lumbar Stenosis are rare. This family pedigree was reported by Postacchini (1985).
Postacchini's patients, but only partially improved the marked and long-standing neurological deficits in one of them.

DEGENERATIVE STENOSIS

This is the most common type of stenosis. The severity of the stenosis usually parallels that of the arthritic change. It is usually asymptomatic in the early years, but causes progressive radiculopathy and myelopathy as disc and facet degeneration progresses. It produces a segment of "hour glass" type of stenosis, and may be localised affecting usually the lowermost two lumbar segments, but occasionally L2 or L3 levels may also be affected. Two or more contiguous segments are often involved.

When present as a localised lumbar stenosis the L4 vertebra is most frequently involved, followed by L3 and L5. At times all three are affected and a more diffuse form of stenosis is one which affects three or more levels. The symptoms and signs may be exaggerated when spondylolisthesis develops particularly at the L4-5 level. The acquired form of localised stenosis is associated with a wide variety of degenerative, granulomatous, inflammatory and discal disturbances, often augmented by derangements of vertebral alignment. It may alternatively follow trauma or vertebral fusion. The anatomy and configuration of the neural arch often has a profound effect on the canal when associated with spondylotic and inflammatory changes.

The stenosis may be either central or lateral or both. The central type of stenosis affects the theca and cauda equina from thickening of the lamina, and thickening of the ligamentum flavum (due primarily to contraction from decrease in the inter-laminar distance). The laminae are sometimes more than 1 cm. thick and widened so that there is no inter-laminar space left (Martens 1984). Central narrowing is accentuated by bulging of the disc from disc degeneration, and enlargement of the articular processes especially the inferior facet, and occasionally a slip caused by degenerative spondylolisthesis.

When stenosis is purely peripheral or lateral it is usually associated with abnormalities of the facet joints. Hypertrophy of the superior facets narrows the nerve root canals. Disc narrowing leads to subluxation of the facets and the tip of the superior facet encroaches on the intervertebral foramen. The arthritic articular facets are major contributors to both central canal and nerve root canal narrowing. (Fig. 4:10).
NORMAL  FACET JOINT HYPERTROPHY  PEDICLE DESCENT  DISC PROLAPSE  LATERAL DISC PROLAPSE

Diagrams to illustrate some of the causes of nerve root compression indicating the nerve root(s) affected (shaded). The pathological areas are stippled.

Figure 4:10. Possible causes of nerve root compression which may precipitate or aggravate symptoms and signs of spinal stenosis.
On the whole the inferior facet hypertrophy results in narrowing of the central part of the spinal canal, whilst the superior facet hypertrophy and enlargement results in narrowing of the nerve root canal. Great care must be exercised when placing such a patient with lumbar stenosis into a position of exaggerated lordosis on the operating table following full muscle relaxation. Any excessive lordosis or forcible hyper-extension may compress the cauda equina or the conus and result in permanent damage. The patient is particularly vulnerable during periods of full relaxation prior to surgery.

The importance of the posterior vertebral articulations in the production of low back pain and sciatica, was well known before recognition of the syndrome of the herniated lumbar disc. Ghormley in 1933 described the clinical and pathological aspects of degenerative disc disease and osteoarthritis of the posterior facets with neural compression (Ghormley 1933). Mitchell described the production of arthritic changes in the apophyseal joints of patients with transitional vertebra and unilateral sacralisation (Mitchell 1936). Epstein described twelve patients with lumbar nerve root compression at the intervertebral foramina caused by arthritis of the posterior facet joints (Epstein J.A. 1973). These were patients with disc narrowing, spondylolisthesis and transitional vertebra, and he stated that these features placed additional stresses on the facet joints. Root compression is more common in patients over the age of forty, resulting in unremitting sciatic pain without evidence of a herniated disc. (Fig. 4:11).

Epstein considered that the increased frequency of arthritis and spondylolisthesis at the L4-5 level related to the higher range of mobility of the fourth lumbar vertebra compared with the other lumbar vertebrae, the fifth being the next most mobile vertebra (Epstein J.A. 1973). This disposition occurred because of the alignment of the facet joints obliquely to the transverse plane, those at the lumbo-sacral level being in a coronal plane. The developmental variations of the articular processes involving the size, shape and angle of projection occur particularly at the lumbo-sacral region, but were also encountered at the fourth lumbar vertebral level. Spinous processes which were poorly developed and asymmetrical also predisposed to mechanical instability, ligamentous injury and osteo-arthritis especially when unilateral resulting in abnormal motion (Epstein J.A. 1973).

However the commonest cause of degenerative arthritis of the facet joints is undoubtedly narrowing of the lumbar disc space. In such patients the vertebral body descends resulting in anterior or posterior displacement.
Figure 4:11. A lumbar vertebra with unilateral facet arthrosis to demonstrate the resultant narrowing of the lateral recess and nerve root entrapment.
Instead of the normal rocking or rotational movement between vertebra seen with the normal disc, a backward and forward or translocating movement occurs which increases the detruisive effect on the disc and facet joints.

Unilateral saclarisation of the fifth lumbar vertebra occurs frequently. When unilateral, abnormal strain is thrown upon the upper facets of the opposite side resulting in degenerative changes often to an advanced degree (Mitchell 1936). Mitchell considered that excision of the superior articular facet of the fifth vertebra may be required to relieve the marked narrowing of the lateral recess and foramen (Mitchell 1934). A similar operation was described in a group of patients by Newman (Newman 1963). Gill and White considered that since little or no motion existed at a severely degenerate disc, that facet removal in such cases could be carried out safely with no perceptible increase in mobility (Gill G.G. 1955).

The Superior Facet Syndrome

In 1972 Epstein described fifteen patients with intense sciatic pain in whom surgical exploration disclosed no evidence of a herniated disc but were found to have entrapment of the L5 or S1 nerve roots in a stenotic lateral recess beneath the superior articular facet of the inferior vertebra (Epstein J.A. 1972). It was unusual to find an objective neurological deficit, but Lasegue's sign was frequently positive and this was the most consistent finding.

Radiographs of the spine were often non-contributory and myelography was negative or showed only minimal change because the normal dorso-ventral diameter of the spinal canal was preserved. The most notable feature was failure of filling of the narrow lateral recess. Electromyography was positive in five of the fifteen patients, and surgical decompression of the lateral recess with removal of the overhanging horizontal portion of the superior facet decompressed the incarcerated nerve root and relieved the symptoms. Review of the myelogram showed that in fact the spinal canal had normal dorso-ventral and transverse diameters, but the narrowing was confined to the lateral recess giving an impression of "cup-shaped" and tightly embraced nerve roots. The fifteen patients with superior facet syndrome appeared in a group of four hundred who were operated on during a five year period with a presumed diagnosis of herniated disc. Their ages ranged from seventeen to fifty-one, but the majority were between twenty and forty. There were eight men and seven women.
Hadley had already described the decreased dorso-ventral diameter of both the foramen and the canal in patients with reverse spondylolisthesis (Hadley 1936). In this instance the tip of the superior articular facet projected into the foramen compressing the nerve root against the unyielding pedicle above. In a large number of cases, clinical presentation with careful analysis of plain films is sufficient to reach an almost certain diagnosis. In spinal stenosis, the superior articular process is more sagittally orientated than normal (Chynn 1978). (Fig. 4:12-4:14).

Disc herniation beneath the articular facet is well described and results in marked neural compression. Hirsch found herniated discs beneath the superior facets in patients with characteristic neurological symptoms and signs but in whom both myelography and previous operation had been negative (Hirsch 1948). The use of computerised tomography and magnetic resonance imaging now allows excellent visualisation of the lateral recess and nerve root foramen pre-operatively.

SPONDYLOLISTHETIC STENOSIS

In 1950 MacNab described spondylolisthesis with an intact neural arch, the so-called pseudo-spondylolisthesis (MacNab 1950). He divided these into three groups, which determined treatment. Group one had backache with or without sciatica, but with no signs of nerve root compression. Group two had sciatica with or without backache, but with signs of nerve root compression. In this group there may be (a) compression in the intervertebral foramen, (b) compression by the lamina of the displaced vertebra, (c) compression of a nerve root by an associated prolapsed intervertebral disc. Group three had signs of compression of the cauda equina. This group requires a laminectomy and MacNab recommended that this should be followed by spinal fusion (MacNab 1950).

Fitzgerald and Newman analysed the mechanism resulting in the development of spondylolisthesis, and root compression (Fitzgerald 1976). They considered that the check given by one facet lying behind the other lost its efficiency owing to degenerative change and the inferior facet or the vertebra above gradually ground its way between the superior facets of the vertebra below permitting forward displacement.

An important aspect of the development of spondylolisthesis is the presence of an unstable joint. In patients with degenerative spondylolisthesis radiographic and operative observations indicate that the facet joint is
Figure 4:12 (a). A photograph taken to demonstrate the pear-shape of the nerve root canal at the lumbar four-five level. The volume, cross-sectional area and shape of this canal is preserved by the height of the intervertebral disc and presence of articular cartilage on the articular facets.

Figure 4:12 (b). In the absence of disc degeneration or osteophyte formation in the articular facet the segmental nerve root is permitted to exit from the spinal canal unimpeded.
Advancing age and loss of disc height and articular facet cartilage reduce also the cephalad-caudal dimension (height) of the nerve root canal. Osteophyte formation on the margins of the vertebral body and articular facets compromise the dorso-ventral dimension (width) of the nerve root canal. During spinal flexion (a combined rotation and translocation when the disc is degenerate) the volume of the nerve root canal (and the spinal canal) are considerably increased. Increasing the overall length of the spinal canal stretches the nerve roots of the cauda equina to their normal length and thereby reduces their cross-sectional area, both individually and when combined in the cauda equina.

Spinal flexion may thereby relieve compression of the nerve root both in the nerve root canal and the spinal canal.
Figure 4:14 (a). Extension of the lumbar intervertebral joint is a combined movement of rotation around a transverse horizontal axis, and posterior translocation. In this illustration, posterior translocation is well seen and the resulting migration of the superior articular facet of the vertebra below into the nerve root canal clearly shown to compromise the width of this canal.

Figure 4:14 (b). The nerve root is therefore trapped in the nerve root canal by the guillotine effect of the superior articular facet against the lower margin of the pedicle above (which has descended already through loss of disc height) and the postero-lateral aspect of the vertebral body above.
orientated more toward the sagittal plane and that it may actually present a $90^\circ$ angle to the horizontal (Weinstein P.R. 1983). Latent segmental instability as demonstrated in flexion and extension radiographs, is more common in women particularly at the L4-5 level. Allbrook has shown that the fourth lumbar vertebra is the more mobile, whereas the fifth vertebra is supported between the iliac crest and anchored by the ilio-lumbar ligaments. The fourth is only partially protected in this way, and when the sacrum is relatively high it is more exposed to stress causing instability (Allbrook 1957).

In the normal spine the level of the supra-cristal plane is most frequently at or above the lowermost level of the body of the fourth lumbar vertebra, whereas in degenerative spondylolisthesis it is usually at or below this level. This observation suggests that spines destined to develop this type of spondylolisthesis tend to have a congenitally high base. This reduces the protection to the fourth lumbar vertebra given by the soft tissues between the iliac crest, exposes the joints between the fourth and fifth vertebra to increased stress and leads to instability. Once instability has developed it is countered by increased tone of the sacrospinalis giving a lordotic posture. If this develops as a chronic state an extension contracture develops, with abnormal weight stress transmitted to the neural arches and zygapophysial joints and eventual degenerative change and frequently spondylolisthetic spinal stenosis.

Rosenberg considered that with an unduly stable lumbo-sacral joint, more stress is placed on articulations between the fourth and fifth lumbar vertebra (Rosenberg 1976). Decompensation of that disc and its ligaments add to instability, hyper-mobility, and degenerative disease of the articular processes, which normally resist the tendency of the upright spine to slip forward. The degenerative changes cause wear and deformation of the anterior part of the articular processes and ultimately allow forward slipping, until the isthmus of the upper vertebra abuts on the upper margin of the superior articular process of the vertebra below.

Since the ilio-lumbar ligaments restrict rotation of the lumbo-sacral joint they protect it from rotational stress and leave the level above most susceptible to torque. The length of the ilio-lumbar ligaments may be judged by the length of the fifth transverse process and by the location of the fourth lumbar vertebra with regard to the inter-cristal line. A transverse process that extends further laterally than that of L3 usually indicates a short ligament with stability. Also, a spine in which the inter-
cristal line passes through the middle third of the fourth lumbar vertebra or higher must have short ilio-lumbar ligaments and therefore a stable lumbo-sacral motion segment. In these instances, the L4-5 intervertebral joint is more likely to be affected by torsional strain.

FIBROUS SPINAL STENOSIS

The advent of water-soluble contrast materials has made it possible to examine the thecal sac in much greater detail and correlate these findings with the clinical features. As a result of such a study in 1976 by De Villiers of eight hundred and fifty myelograms using water-soluble contrast medium, a clear description of fibrous spinal stenosis was provided (De Villiers 1976). De Villiers measured the normal size of the thecal sac in twenty-five men and twenty-five women. He also measured the vertebral bodies. He noted that the thecal sac fitted fairly snugly into the upper part of the bony lumbar spinal canal, but that it was often surrounded by a fairly wide soft tissue "space" in the lower lumbar region.

The space between the contrast column and the posterior surfaces of the vertebral bodies varied from 1 mm. to 4 mm. at the fourth and from 1 mm. to 7 mm. at the fifth lumbar vertebral body. He stated that the presence of excessive amounts of fibrous tissue in a bony canal of normal size was confirmed at operation in several cases. The length of the involved segment varied from 1 cm. to 10 cm. and in some cases the sac was completely obliterated. The lower end of the sac was most frequently involved. In cases of severe stenosis the individual nerve roots of the cauda equina were often not visualised at all and some were replaced by a single thick "cord" adherent to parts of the sac wall. In all patients with fibrous stenosis the associated nerve root sheath failed to fill with contrast medium.

In every case of severe and extensive fibrous spinal stenosis, symptoms had been present for years, and the patients had been subjected to multiple surgical procedures of laminectomy, and/or spinal fusion. In some cases an earlier myelogram using an oily medium had been performed, and in some patients who had been subjected to laminectomy, nerve root sheaths were obliterated in the absence of stenosis of the thecal sac.

This was not however the first description of these myelographic findings. Epstein in 1969 and others had described spinal arachnoiditis leading to fibrosis, with numerous aetiological factors including operative trauma (Epstein B.S. 1969). De Villiers considered that fibrous spinal
stenosis was due to subarachnoid adhesions (or senecchia), or to fibrous thickening involving the dural sac or the extra-dural tissues. He considered it could result from blood, inflammatory exudate or chemical irritants, all of which would tend to settle in the most dependent part of the sac, possibly accounting for the common site of stenosis. He demonstrated what he called "fibrous spinal stenosis" in six per cent of the eight hundred and fifty myelograms using 10 ml. doses of water-soluble Dimer-X (May and Baker). Mild involvement resulted in obliteration of the nerve root sheaths only. More severe fibrosis produced in addition stenosis of a segment of the thecal sac.

He also made some interesting observations on the anatomy of the thecal sac. Measurements revealed the thecal sac to be of similar size in both sexes regardless of the larger skeleton of the male. He considered it to be a uniform tube throughout the lumbar region, except at the fifth lumbar vertebral level where it often becomes considerably wider or narrower. In view of the normal variations he considered actual measurement had no value in the diagnosis of thecal sac stenosis (De Villiers 1976).

**POST-OPERATIVE SPINAL STENOSIS**

Schlesinger and Tavaras pointed out that the developmentally small spinal canal can magnify the clinical symptoms of even small disc herniations (Schlesinger 1953). A spinal canal of normal calibre can also become stenotic post-operatively by marked hypertrophy of the posterior bone graft, or by secondary changes at the first free joint above the fusion. In one series from a specialised spinal unit in the United States of America of five hundred and fifty-two patients with all types of spinal stenosis, there was a high incidence of post-operative stenosis (forty-one per cent) (Brodsky 1976). Spinal stenosis may develop after laminectomy alone, particularly in susceptible individuals who demonstrate the following anatomical features. (Fig. 4:15).

1. Vertical placement of the lamina especially at L5, producing a shingling effect.
2. Ventral projection and "beaking" of the superior edge of the spinous process.
3. Enlargement and medial projection of the facet joints with consequent narrowing of the transverse diameter of the neural arch.
4. Thickened ligamentum flavum, even in relatively young people.
Figure 4:15. A diagram to illustrate the most common causes of postoperative spinal stenosis. The possibility of the patient developing spinal stenosis following laminectomy or spinal fusion should always be remembered when surgery is being considered. If the risk is significant, as defined in the text, it is advisable to inform the patient of this possibility.
5. A trefoil-shaped spinal canal with narrow posterior recesses which predispose to foraminal entrapment and magnify the effect of lateral disc herniations.

6. A combination of any of these factors especially in the presence of increased lumbar lordosis.

POST-LAMINECTOMY STENOsis

Two important factors must be considered.

1. Sub-periosteal stripping of the lamina has been noted to produce ligamentous thickening and bony thickening similar to the new bone formation which follows elevation of the periosteum of long bones.

2. Heavy scar tissue at the site of a previous sub-total laminectomy may create postero-lateral pressure on the cauda equina.

Brodsky, after reviewing two hundred and thirty-one patients with post-operative spinal stenosis (Brodsky 1976) emphasised the importance of Paine's recommendation to perform a complete and adequate decompressive laminectomy even for lesser degrees of post-laminectomy spinal stenosis (Paine 1972).

POST-FUSION STENOsis

This may develop as a severe concentric stenosis just above the fusion mass. It may be associated with disc herniation, marked thickening and infolding of the ligamentum flavum both posteriorly and laterally, and hypertrophy and medial projection of the articular processes and ventral projection of the upper margin of the fusion mass and spinous process. The cauda equina may be reduced to a diameter smaller than a lead pencil. There is usually a lesser degree of diffuse narrowing under the fusion mass.

Very rarely a disc herniation may be found under the fusion mass (Brodsky 1976). This must be decompressed even if it involves sacrificing the facet joint to free the nerve in the foramen. Mechanical instability so produced is usually minor and fusion is usually not mandatory. In some cases a secondary fusion has become necessary. A few of these patients develop slight spondylolisthesis or retrolisthesis, but Brodsky's experience with post-fusion stenosis led him to avoid laminar fusions. His experience was that no significant mechanical weakness of the back developed in those patients where the articular facets had not been sacrificed.
ARACHNOIDITIS

Arachnoiditis complicating chronic arthritis of the spine was first described by Vincent in 1930 (Vincent 1930). Arachnoiditis with persistent pain after multiple back operations virtually precludes a successful long-term outcome from any procedure. The unique annular type of adhesive arachnoiditis which produces myelopathy and radiculopathy has responded to laminectomy and micro-dissection of the constricting bands on the spinal cord (Epstein J.A. 1978). In the majority of cases however the process is so diffuse that it defies treatment. Whilst adhesions may be separated they tend to reform in uninvolved areas extending the pathology. The presence of widely disseminated arachnoiditis contra-indicates surgery.

Spinal arachnoiditis is a protean disorder of multiple aetiology. The most common causes include spinal anaesthesia, the use of intra-thecal medications, infections and parasitic diseases, neoplasms and trauma. Most cases are currently related to multiple myelographic and operative procedures. Unfortunately aetiological factors other than iatrogenic with important diagnostic, therapeutic, and medico-legal implications have received little attention (Epstein J.A. 1978).

POST-TRAUMATIC SPINAL STENOSIS

Repeated minor trauma or a specific major incident may be sufficient to precipitate the clinical syndrome of spinal stenosis in those patients who have already a compromised spinal canal because of congenital, developmental or degenerative factors. Secondly at the other end of the spectrum major trauma may result in acute compression of the contents of the spinal canal through fracture dislocation, and disruption of the normal vertebral alignment. This may result in the sudden onset of a complete or partial neurological lesion. When partial this may be central or lateral with symptoms occurring with changes of posture or with exercise (Hasue 1980).

Thirdly, it appears that a minor back injury in childhood can lead to progressive degenerative change of the spine (Bowen 1978). Some patients presenting with spinal stenosis give histories of old or recent trauma. Trauma to the back may have occurred many years previously, and consist of a vertebral fracture of the upper lumbar spine with subsequent fibrous tissue formation, which many years later may result in post-traumatic
spinal stenosis due to laminar thickening, and posterior osteo-arthritic spur protrusion (Bailey 1911). Alternatively trauma may initiate progressive traumatic disc degeneration with subsequent localised osteo-arthritis and spinal stenosis. Children falling from a height landing on their buttocks can cause fractures of the cartilage end plate which leads to degeneration of the disc and other components of the three joint complex. Rotational strains may initiate changes in discs and posterior facets. The final result many years later is a one level central or lateral stenosis or sometimes multiple level stenosis (Bowen 1978).

MISCELLANEOUS

Paget's Disease

The aetiology of the neurological complications of Paget's disease is not entirely clear. The most common sites for Paget's disease apart from the sacrum is the lumbar spine (Schmorl 1959), but paraparesis is more common with thoracic involvement. This may be because the canal is narrower in comparison with the diameter of the spinal cord in the thoracic region (Hartman 1966). Dysfunction of the cord however can occur at any level below the occiput. At operation the cord is usually found to be compressed by the expanded bodies of the vertebra, as was noted in two of the patients reported by Douglas (Douglas 1981). Occasionally expansion of the pedicles and lamina can cause compression (Turner 1940). In some cases no convincing cord compression can be demonstrated by radiography, and the cause of paraparesis may then be related to vascular changes in the adjacent soft tissues and bone. In one reported case of paraparesis without complete myelographic obstruction medullary arteriography demonstrated delay in filling and emptying of the anterior spinal artery at the level of the involved vertebra (Mathe 1976). The speed of response to medical treatment within two weeks in some of the patients reported by Douglas, suggests that the response could not have been due to remodelling of bone, but rather to a decrease in soft tissue swelling or to a redistribution of blood flow. In three patients reported with Paget's disease of the spine, clinical improvement took place with medical treatment, although repeated ultrasound examination showed no change in the bony diameter of the spinal canal (Porter 1978).
Extra-dural "Plumboma"

Gunshot injuries to the spine and spinal cord are relatively common in war, and also in some countries such as the United States, where they comprise twelve per cent of traumatically acquired acute cord injuries. Staniforth in 1982 however reported one patient in whom neurological complication evolved eight years after the initial injury (Staniforth 1982). Usually the late development of neurological signs following spinal cord or cauda equina injury occurs as a result of syrinx formation or focal arachnoiditis. Other complications however are the absorption of lead from closed cavities presenting as lead poisoning (Ellis 1875), but can also cause the diffuse opacification of synovium, the so-called lead arthrogram or plumbogram (Weston 1978). Not only may the lead pellets cause pressure on neurological structures but the joints which have been damaged by trauma or lead may hypertrophy causing spinal stenosis.

PATHOGENESIS OF NEUROGENIC CLAUDICATION

There are therefore three essential factors in the production of the spinal stenosis syndrome. (Fig. 4:16).

Firstly, a basic congenital or developmental defect reduces the space in the central part and lateral recesses of the spinal canal, and this space is further reduced by superimposed degenerative or other changes. Narrowing of the spinal canal may be present for many years without symptoms, and axial tomography is necessary to demonstrate changes in the lateral recess.

Secondly, fixity of the nerve root together with a long angular course around the larger arthritic facet joints which interferes with vascularisation.

Thirdly, symptoms arise on walking because of bulging of the annulus into the nerve root canal, and rotatory and lateral instability due to degeneration of the intervertebral disc.

It goes without saying therefore that to produce adequate relief of stenotic symptoms simple laminectomy is inadequate. The nerve root canals on each side must be decompressed by either partial or total facetectomy which allows the nerve root to regain its mobility by restoring the normal course of the nerve. If the disc is found to be contributing to lateral recess stenosis, it should be removed, but discectomy per se should be avoided since it adds to the instability of the decompressed motion segment.
Vertebral translocation forwards and backwards and possibly rotational during walking, particularly when discs are degenerate.

Nerve root fixation by adhesions or fibrous tissue so it is not free to glide during walking or standing causing constriction and traction on the nerve root during activity.

Congenitally or developmentally short pedicles: a growth disturbance following trauma or a constitutional or inherited disorder.

Ligamentum flavum buckling into spinal canal during extension on standing or walking.

Bulging and increased transverse diameter of disc on weight-bearing and walking.

Increased metabolic rate of nerve root during activity which makes blood supply critical when nerve is compressed.

Figure 4:16. Aetiological mechanisms of neurogenic claudication, both postural and ischaemic.
Wilson in 1971 postulated three mechanisms by which neurogenic claudication might be produced (Wilson 1971).

1. The transmission of neural impulses through the cauda equina roots causes reactive hyperaemia with dilation of the radicular vessels, but blood flow through radicular arteries becomes inadequate with activity through impairment at points of constriction.

2. Actively conducting nerve roots demand an increased supply of oxygen which puts them in competition with blood flow to actively contracting muscles.

3. The relative ischaemia of nerve roots of the cauda equina may be accentuated by other occult sites of compression suggesting a cumulative effect of nerve root compression.

There is really very little evidence to support these statements, but the work of Blau and Logue in 1961 is frequently mis-quoted in this context (Blau 1961). They considered that pain and sensory disturbances were ischaemic in nature produced by compression of the spinal cord which prevents the reactive hyperaemia that would normally occur following exercise. However significant stimulation was required to produce histological changes within the spinal cord (please note NOT the nerve roots as is frequently mis-quoted). This would certainly be much more than the provocative stimulus which is normally required to produce symptoms in patients in the clinical situation, even if one were allowed to extrapolate from mice to human beings. Also it should be noted that symptoms may develop when lying in bed or standing for prolonged periods of time, and not simply with exercise (static or postural claudication) (Wilson 1971).

Evans in 1964 measured the claudication times of patients using varying tensions of inspired oxygen (Evans 1964). Claudication times increased as the oxygen saturation increased suggesting that pain or paraesthesia was due to relative ischaemia of the cauda equina roots during exercise. Davson noted that prolonged standing or lying in certain positions could increase the pressure of the confined cerebro-spinal fluid below the site of the block (Davson 1967). He considered that in such cases an increase in the pressure of the cerebro-spinal fluid could in turn result in the collapse of strategic venous return, and result in stagnant anoxia to selected nerve trunks. This he considered would account for postural or nocturnal claudication (Davson 1967). The next chapters consider in some detail the anatomical, physiological and mechanical factors underlying the development of symptomatic stenosis. (Fig. 4:17).
Figure 4:17. MECHANISMS OF PATHOGENESIS OF SPINAL STENOSIS
CHAPTER 5
ANATOMICAL STUDIES

INTRODUCTION

This chapter sets out to look in some detail not only at the anatomical structure of the spinal canal, nerve root canals, and intervertebral foramina, considering both racial variations and those found in conditions such as achondroplasia, but also to look at the innervation of the lumbar spine itself and to try to identify those structures which may be primarily responsible for the production of low back pain and pain referred to the legs.

It is clearly important in discussion of spinal stenosis to describe the anatomy of the blood supply to the cauda equina, and in this discussion variations in the level of termination of the spinal cord during normal and abnormal development are described. The blood supply of the vertebral column itself is also considered since this is important in the discussion of possible complications of surgery. Certain anatomical variations affecting the facet joints and the ligamentum flavum will be described, together with the anatomical structure of the nerve roots of the cauda equina. This is important when considering tension or compression of nerve roots. Finally the relationship between the spinal canal and the spinal cord and dural sac is described, including discussion of the concept of spinal reserve capacity.

COMPARATIVE ANATOMY

For many years the daschund, Pekinese and bassett have been bred for their achondroplastic features. The spinal canal is relatively narrower in dogs than in man, and the spinal cord terminates at the L6-7 level. Degenerative changes are more marked in chondrodystrophied dogs leading to premature calcification of up to ninety per cent of the discs in a two year old daschund (Yates 1981). Disc lesions occur mainly at the thoracolumbar junction, with annular rupture causing haemorrhage and an ischaemic cord lesion. The position therefore is not analogous to man, but the problem is enthusiastically tackled by canine neurosurgeons in the United States of America. Myelography, venography and discography are employed in diagnosis, and extensive decompression operations are performed. Considerable ingenuity is shown in rehabilitation methods, including corsets and wheeled supports (Hoerlein 1978).
THE VERTEBRAL LEVEL OF TERMINATION OF THE SPINAL CORD DURING NORMAL AND ABNORMAL DEVELOPMENT

The adult cord may terminate anywhere between the last thoracic and third lumbar vertebra, though the great majority end opposite the first and second lumbar vertebra. Female cords and those of Negroes tend to be slightly longer on average than those of white males (Needles 1935). Current text books of anatomy all state that the cord terminates opposite the third lumbar vertebra at birth. Kunitomo noted that the conus medullaris receded from S1-2 in a 52 mm. (twelve week) embryo to L4-5 in a 67 mm. (fourteen week) embryo. It appears from this that the ascent of the cord takes place most rapidly between 30 mm. and 100 mm. (nine to sixteen weeks) (Kunitomo 1918).

The immature human spinal cord was examined by Barson in two hundred and seventy four individuals, to determine the level of termination of the cord. Twenty-two of these had mal-formations of the vertebrospinal axis, and their ages ranged from thirteen weeks of gestation to fifteen years, with most being in the latter half of gestation. Barson confirmed that although there was a normal variation at any particular age, he demonstrated a relatively rapid ascent of the conus medullaris to the fourth lumbar vertebra at the nineteenth week of pregnancy. This was followed by a slower ascent of the cord which ended approximately opposite the lower border of the second lumbar vertebra at full term attaining the 'adult' level about two months post-natally (Barson 1970).

Those specimens with mal-formations of the vertebral column or spinal cord tended to be associated with a cord ending at a lower level than normal for a particular age. For instance in patients with diastematomyelia one would theoretically expect severe paralysis at birth, since most of the ascent occurred before this date. Alternatively, the bony spike could produce a lengthening cleft as the cord ascends and the ascent of the cord is slowed in this condition but not prevented so that it continues into childhood beyond the normal time of cessation. It is conceivable that immature neuronal tissue is capable of developing alternative pathways so that neurological damage is not evident at birth or early childhood, but as this capacity is slowly lost neurological damage becomes evident later (Barson 1970).

The level of termination of the spinal cord is clearly of relevance in patients with upper lumbar spinal stenosis who may develop a combination of upper motor neurone and lower motor neurone changes.
EMBRYOLOGICAL DEVELOPMENT OF THE SPINAL CANAL

Sclerotome cells of mesenchymal origin migrate from the somites in the third foetal week to form the membranous vertebral column. In the fourth and fifth foetal week, this is succeeded by the cartilaginous vertebral column. What appears to be two cartilaginous centres have been described by Tondury as microscopically uniting into one core (Tondury 1958). There is also one cartilaginous centre for each half of the developing vertebral arch, which is clearly visible in the seventh week of gestation (Fig. 5:1). At the same time centres for the ribs have appeared. Ossification starts simultaneously in the vertebral bodies and in the arches in the seventh to eighth week after gestation. It does however commence at different locations in the spine. Ossification of the vertebral bodies begins in the lower thoracic region and spreads cranially and caudally whilst ossification of the arches starts cervically and proceeds downwards. In the lumbar spine ossification of both bodies and arches begins in the fifteenth week of gestation (Fig. 5:2).

In an attempt to assess the influence of the spinal cord and dural sac upon the development of the spinal canal, Tondury examined foetuses of crown rump lengths of 10, 12 and 17 cm. These correspond to sixteen week, eighteen week and twenty-two week foetuses, and he found that in all foetuses the lumbar spinal canal was oval in outline, but the oval shape was least prominent at the L5 level (Tondury 1958). Larsen measured the sagittal and interpedicular distances of foetal spinal columns (Larsen 1981). He found less individual variation at the upper three lumbar levels, than at L4 and L5. He also found that segmental variation in the interpedicular distance between L1 and L3 was smaller compared with that between these three levels and L4 and L5, with L4 and L5 displaying large variations in interpedicular distance. This confirmed the previous findings, and it is tempting to conclude that the upper part of the lumbar spinal canal is influenced greater in its embryological development by the presence of the dural sac and its contents.

The spinal cord is lengthened in spinal flexion and shortened in extension. Breig and Marions noted however that there was no axial displacement of the cord when the entire spinal column either flexes or extends, but when the cervical spine alone flexes and extends there is respectively stretching and slackening of the cord and this movement is transferred to the lumbar nerve roots (Breig 1963). The nerve roots therefore move within the intervertebral foramina. It is possible that the
OSSIFICATION OF THE LUMBAR VERTEBRA

AT BIRTH

Vertebral Arch
Primary Centre

Centrum

Three primary centres of ossification joined by cartilage.

AGE 6 YEARS

Neurocentral growth plate active. Fuses at seven years. Inherited or constitutional factors or trauma may interfere with growth at this plate resulting in permanently short pedicles.

AGE 18 YEARS

The C-shaped ring apophysis is starting to fuse. The cartilaginous vertebral end plate which contributes to growth in height of the vertebral body is ossifying. The apophyses of the transverse and spinous processes have appeared.

Figure 5:1. Diagrams to illustrate the embryological development and post-natal growth of the lumbar vertebra.
OSSIFICATION CENTRES OF LUMBAR VERTEBRA

Superior Ring Apophysis
Centre for Mamillary Process
Centre for Transverse Process

Inferior Ring Apophysis Neurocentral Growth Plate Centre for Spinous Process

Figure 5:2. A purely diagramatic representation of the ossification centres since they are not all present at one time. The neurocentral growth plate has fused by the seventh year. Ring apophyses appear around twelve years of age, those of the transverse and spinous processes appear around eighteen years of age. All have fused between eighteen and twenty-five years of age.
innumerable sliding motions of the nerves in and out of the foramina could easily mould the medial and lower surfaces of the pedicle, leading to an increase in the interpedicular distance seen in the upper lumbar spine particularly. Larsen felt that the influence of the nerve root upon the intervertebral foramen and the pedicles was demonstrated by the different shapes of the foramen at different spinal levels. He considered that the greater the angle of exit of the emergent nerve with respect to the longitudinal axis of the spine, the greater the tendency would be towards pedicular divergence (Larsen 1981).

Eisenstein however noted that spinal stenosis was relatively rare in the Negro race, at least in the South African Negro, but in these individuals the bony lumbar canal was not particularly wide (Eisenstein 1977). It has in fact been shown to be slightly less capacious than in Caucasians when measured on the basis of antero-posterior and transverse diameters. It is known however that the spinal cord extends further caudally in adult Negroes than in Caucasians (Louis 1978 and Needles 1935), and although little is known of the ascent of the cord in Negroes compared with Caucasians, its lower position could be sufficient to influence the development of the spinal canal more distally in Negroes. The anatomical result would be an oval configuration of the canal extending more distally which would better accommodate the spinal cord and the dural sac, than the triangular canal of the same interpedicular and antero-posterior distance.

This indirect evidence suggests that a slower and more incomplete ascent of the spinal cord within the spinal canal may possibly reduce the frequency of symptomatic spinal stenosis in later life.

Scalloping of the Vertebral Bodies

Larsen later measured the degree of scalloping in concavity of the posterior surface of the lumbar vertebral bodies (Larsen 1985). In the median sagittal plane he found an increase in scalloping from L1 to L4, with a subsequent decrease at L5. In the lateral plane the scalloping increased from L1 to L5 and was greater laterally than centrally at all levels. Scalloping in the lateral sagittal planes was particularly prominent at the fourth and fifth lumbar levels raising the possibility that this was the result of pressure exerted by the spinal nerves. On the other hand, medial scalloping is probably partially the result of hydrostatic pressure of the cerebrospinal fluid in the dural sac during growth and development. At the level of the disc, the insertion of the outer layers of the annulus fibrosus
superiorly and inferiorly into the vertebral end plates, and the bulging of the disc during compression and loading probably prevents scalloping from occurring at the vertebral end plates (Larsen 1985). Failure of scalloping at the level of the motion segment may be important in some individuals in the development of spinal stenosis at this level.

INNERVATION OF THE LUMBAR SPINE

Numerous structures of the lumbar spine are richly innervated with pain fibres whilst other areas are devoid of pain innervation. The spine is innervated by the sinuvertebral nerve and posterior primary ramus. (Fig.5:3). Herbert Luschka first described the sinuvertebral nerve, which passes through the intervertebral foramen, close to the pedicle on the cranial side of the corresponding disc (Luschka 1850). It is unlikely that this nerve is implicated in simple disc herniation, except where sequestration occurs (Edgar 1976). The lateral branches of the posterior primary rami from the upper three lumbar levels have cutaneous nerves which reach distally as far as the greater trochanter. Interestingly, there is no skin supply from the lower two lumbar posterior primary rami.

Innervation of the Intervertebral Discs

Nerve endings are confined to the peripheral part of the annulus. Stilwell (1956) and Pederson (1956) and others were only able to observe these nerve endings on the surface, whereas Malinsky (1959), Hirsch (1963), and Jackson (1966) observed that nerve fibres penetrated into the outer layers of the disc. Shinohara agreed that only the outer layer of the annulus fibrosus was innervated in a normal disc, but that in a degenerate disc fine nerve fibres accompanying granulation tissue could be found in the deeper layers (Shinohara 1970). Most of the nerve terminations were naked nerve endings and probably mediated pain sensation. This is consistent with the pain provocation of saline acceptance test of discography (Lindblom 1951).

The Innervation of the Posterior Longitudinal Ligament

This ligament is supplied by the sinuvertebral nerve and contains more nerve endings than the anterior longitudinal ligament. It is possible that this ligament might be responsible for much of what is called discogenic pain. During mechanical stimulation at operation under local anaesthesia, pain is felt adjacent to the midline and radiates to the lumbar region and
SITES OF NERVE COMPRESSION IN SPINAL STENOSIS

Thickening of fibrous tissue at site of spondylosis with or without spondylolysis.

Capsular thickening of facet joint and degenerate cyst formation.

Thickening of dura mater and arachnoid lining in constrictive arachnoiditis.

Sequestrated disc fragment in spinal canal or nerve root canal.

Spondylolisthesis causes traction and compression of nerve roots particularly when degenerate in origin.

Tumour or infection in vertebral body, disc space or epidural space may cause nerve root compression with an inflammatory component.

Laminar thickening.

"Thickening" of ligamentum flavum.

Inferior articular facet hypertrophy (central stenosis).

Superior articular facet hypertrophy (lateral recess and nerve root forameno stenosis).

Medial branch of posterior primary ramus supplying sensation to facet joint.

Nerve root canal narrowing compressing nerve root.

Sinuvertebral nerve supplying anterior dura and disc compressed in root canal.

Sagging of degenerative annulus fibrosus centrally and laterally.

Osteophyte formation on margin of vertebral body.

Migration posteriorly and possible sequestration of nucleus pulposus.

Figure 5:3. This diagram illustrates firstly at least fourteen possible causes of nerve root compression, and secondly the innervation of the facet joints, dura and annulus fibrosus through which pain impulses are mediated.
upper buttock (Murphey 1968). (Fig. 5:3).

The Innervation of the Ligamentum Flavum

Interspinous and supraspinous ligaments and the ligamentum flavum are innervated by fibres derived from the overlying muscles and on its deep surface by nerves from the posterior epidural space (Stilwell 1958). Edgar could find no nerve fibres ending within the substance of the ligaments (Edgar 1976). Hirsch however found free and complex unencapsulated nerve endings in the supraspinous and interspinous ligaments, but free endings only were seen in the outermost layers of the dorsal surface of the ligamentum flavum, and never again in its substance (Hirsch 1963). The innervation of the lumbar dorsal fascia is from the posterior primary rami.

Innervation of the Dura Mater

The sinuvertebral nerve is confined to the anterior dura, these fibres ascend one level and descend two levels. No nerves were found by Edgar in the posterior dura (Edgar 1976). Stilwell did observe some fine nerve fibres in the posterior dura, but these were exclusively autonomic (Stilwell 1958). The anterior dura is pain sensitive, and stimulation gives rise to a pattern of pain, similar to that from the posterior longitudinal ligament.

Innervation of the Vertebral Periosteum, Bone and Facet Joints

Two types of fibres exist: one is the free fine fibres mediating pain, and the other is the complex unencapsulated fibres most likely mediating proprioception. These are from the autonomic rami and paravertebral plexuses. The facet joints of monkey spines are innervated from the posterior ramus which supplies its own level and sends a branch to the joint of the level below (Stilwell 1956). Lewin confirmed on human material the findings of Stilwell. Both pain and proprioception are mediated by these fibres (Lewin 1962).

Clinical Relevance of Innervation of the Lumbar Spine

There is a marked overlap of innervation in the lumbar spine with each sinuvertebral nerve and posterior primary ramus supplying at least two levels.
The poor localisation of much low back pain and its tendency to radiate may be related to this neurological pattern. Newman's concept appeared attractive that the posterior rami were concerned with the stability of the spine, whilst the sinuvertebral nerve protected the nerve tissue itself (Newman P.H. 1959). Irritation of the sinuvertebral nerve endings would therefore have been most relevant in patients with spinal stenosis.

Kaplan suggested that low back pain and sciatica could be caused by irritation of the sinuvertebral nerve (Kaplan 1947). On the other hand, Arnoldi and Ficat have suggested from their studies that intra-osseous venous hypertension may produce pressure on nerve endings within the bone substance (Arnoldi 1972, Ficat 1973). This is clearly relieved at the time of laminectomy by performing bony decompression as well as neurological decompression. Clearly surgery for spinal stenosis which is designed to relieve pressure on neurological tissues, may frequently be associated with the relief of low back pain, and the hypothesis of lowering intra-osseous pressure would clearly provide a convenient explanation for this relief of back pain.

One other concept worth considering is that claudication pain may be referred from the lower lumbar facet joints which may be arthritic and can certainly refer pain into the legs. As with other osteo-arthritis synovial diarthroidal joints, pain comes on with activity. This provides a rationale for facet joint blocks in patients with clear claudicant symptoms but with unimpressive radiological features. Facet joint injections frequently affect also the emergent nerve roots and so may be effective partly because of this side effect.

THE BLOOD SUPPLY OF THE CAUDA EQUINA AND LUMBAR NERVE ROOTS

In 1946 Feeney and Watterson observed:
"there exists a very close relationship between the metabolic requirements of the nervous tissue and the final distribution of intraneural vessels in the adult, a relationship which functions in such a way as to provide the nervous system with a blood supply just adequate for its minimal needs" (Feeney 1946).

The hydrostatic pressure within small vessels is normally 30-35 mm. of mercury at the anterioal end of the capillary, and 10-15 mm. of mercury at the venular end of the capillary (Dommsse 1976). Clearly the arterial supply of the nerve root which is dependent upon large numbers of capillary-sized vessels, is threatened or obliterated by intra-spinal lesions of various kinds, during the course of operative intervention and other forms of trauma,
and arising from post-operative scar tissue formation (Seddon 1972 and Bertrand 1975).

Pressure on arteries, veins and capillaries can impair conduction with changes in sensation and loss of motor power in one or both limbs. If the pressure is prolonged, irreversible changes take place in the substance of the nerve root, and recovery of sensation and motor power following adequate surgical decompression will then be incomplete (Dommisse 1975). The intervertebral foramen appears to be a site of high vulnerability in nerve root vascularisation (vide infra) since this is the level at which the radicular as well as the medullary arteries arise. Rising pressure within the spinal canal is likely to produce variable results in the different contents of the canal such as fatty tissue, neural and vascular structures which together constitute an intricate anatomical complex. The neural structures within the spinal canal are less vulnerable to increased pressure because of the positive subarachnoid fluid pressure. On the other hand, the vascular structures within the spinal canal are more vulnerable because of low intra-arteriolar and intra-capillary pressure and the low pressure venous system of Batson's plexus (Batson 1940).

Parke carried out vascular injection studies of eleven perinates looking at the lumbo-sacral spinal nerve root blood supply (Parke 1981). He concluded that the intrinsic blood supply of each nerve root was derived from two sources, firstly distally from the segmental arteries, and secondly proximally from the radicular arteries which received their blood from respectively the anterior or dorso-lateral spinal arteries (Fig. 5:4). The level of the anastomosis of these two sources was in the proximal one-third of the root. This represented an area of relative hypo-vascularity just below the conus, which Parke suspected contributed to the neuro-ischaemic manifestations occurring in degenerative disease of the lumbar spine. The proximal supply via the anterior or dorso-lateral spinal arteries was derived from the medullary vessels. It is well known that Adamkiewicz in 1882 described the nutritive vessels of the cord and noted a relatively consistent large medullary artery which supplies most of the cord (Adamkiewicz 1882), but Dommisse pointed out that this does not occur at a constant level, and in fact may occur at any intervertebral level (Dommisse 1975). (Fig. 5:5).

The thoraco-lumbar area provides one of the segments of vascularity of the cord, the other areas being the cervico-thoracic and the mid-thoracic areas. There is minimal collateral exchange between the three areas with only a single feeder artery in each of the lower two regions,
Figure 5:4. The Relationships between the Segmental Artery, the Medullary Vessels and Nerve Root and Meningeal Blood Supply.
Diagramatic representation of conus medullaris and cauda equina to illustrate the U-shaped area of relative hypo-vascularity of each nerve root in the upper third of its course. This occurs at the site of the anastomosis of the proximal medullary derived radicular arteries with the distal segmentally derived radicular arteries. The nerve roots may be particularly vulnerable to pressure and surgical manipulation at this level.
but as stated the level of this is variable, so that the direction of blood flow within the longitudinal vessel is also variable. Parke however demonstrated that in the human as in the dog and the monkey, the lumbo-sacral enlargement of the spinal cord is one area which possesses a most conspicuous collateral vascularity, in the form of enlarged longitudinal spinal arteries (Parke 1981). Lazorthes described three types of branches from the segmental artery entering the intervertebral foramen (Lazorthes 1971).

1. **Proper radicular branches** which do not reach the cord, but terminate within the root or the dura.
2. **Pia mater branches** which anastomose with the vasa corona, but do not supply the substance of the cord.
3. **Spinal branches** which are feeder arteries to the cord tissue, via the longitudinal spinal arteries.

The terminology proposed by Parke is shown in Figure 5:6 (Parke 1981).

This consists of:-

1. **The medullary arteries** which course along the internal aspects of their respective roots, but give no branches to them in their mid-course.
2. **The distal radicular arteries** (dorsal and ventral) which supply the distal part of the root and are branches of the ganglionic plexus of the segmental (spinal) artery when the medullary vessels are not present; or they are an intermediate branch of the medullary vessel as it enters the dura.
3. **The ventral proximal radicular arteries** branch from the anterior vasa corona, and receive their blood supply from the anterior spinal artery.
4. **The dorsal proximal radicular arteries** are immediate branches of the posterior spinal artery.

To emphasise the importance of Dommissé's statement that there is no vascular "safe area" of the cord I shall quote directly (Dommissé 1975).

"Because of the variability every intervertebral foramen is a potential site of origin of any anterior and/or posterior feeder vessel. Accordingly the intervertebral foramen is a vulnerable point in the system of arterial supply of the cord, and should be treated with caution during the course of surgical procedures. It is also a point of maximal concentration of blood vessels and one at which bleeding is likely to be brisk from arteries and veins. Rough handling, heavy retraction, plugging in event of haemorrhage, and the application of cautery at this level are threats which should be avoided. Ischaemia and neurological deficits could be precipitated in the event of involvement of a major medullary feeder. Structures other than the spinal medulla, in particular the segmental nerve root, and the
Figure 5:6. A diagramatic representation of the intrinsic blood supply of the nerve root illustrating both the primary and the secondary compensating coils which permit inter-fascicular and intra-fascicular longitudinal movements.
paravertebral muscles could be involved in the same manner."

The motor, sensory and sphincter impairment which may present quite suddenly in patients with degenerative spinal stenosis is most likely of vascular origin. It has been believed that surgical intervention within twelve hours of the onset of acute symptoms would reduce the incidence of permanent neural deficit, but Onkey has studied a number of these cases which revealed that there is in fact no specific interval in which decompression can be reliably expected to forestall irrevocable damage (Onkey 1978). Nevertheless the symptoms are seldom relieved without early decompression.

Vascular studies of the cauda equina reveal that there is a U-shaped pattern across the cauda equina below the tip of the conus where there is relative hypo-vascularity. At this point of anastomosis the delivery system to the root substance is most attenuated (Fig. 5:5).

Having considered the anatomy of the blood supply to the cauda equina and nerve roots, the patho-physiology of ischaemia of the cauda equina or claudication will be described later in Chapter 6 on Physiological Studies.

THE NUTRITION OF THE NERVE ROOT

The lumbar nerve root therefore has a much more precarious blood supply than a peripheral nerve (Lundborg 1968). The nerve root firstly lacks a collateral mesoneurial blood supply and secondly has only a meager connective tissue support for its intrinsic longitudinal blood vessels. During flexion and extension the spinal canal lengthens and shortens respectively (Breig 1960), causing the lumbo-sacral nerve roots to become taut in flexion and slack and undulant during extension (Breig 1963). These movements of loosely bound fascicles must result in considerable inter-fascicular motion both longitudinally and laterally. It is not surprising therefore that Parke and Watanabe noted the presence in the vessels of what they termed primary compensating coils both laterally and inter-fascicular to allow for this movement without jeopardising the blood supply of the nerve fibre bundles (Parke 1985). Secondary compensating coils of vessels are found intra-fascicular to permit longitudinal elastic movements within the nerve fibre bundles (Fig. 5:6).

The compensating coils of vessels are not seen in the pig or rabbit (Parke 1985) and probably represent adaptive features which probably occur in post-natal development to protect the nerve roots from the unique
biomechanical stresses which occur in the adult human lumbo-sacral nerves.

There are also large slowly spiralling radicular veins which not only permit root elongation but also frequently anastomose via wide bore vessels with the arterial supply of the root thereby protecting the nerve root's blood supply right up to the point of any localised root compression. At the point of nerve root compression the veins are compressed but the higher pressure arterial system remains patent. Although venous drainage is therefore not possible at the site of compression, venous engorgement can be seen at either side of the compression draining blood both proximally and distally thereby maintaining blood flow to the compressed root. Numerous arterio-venous anastomoses are essential for this protective function (Parke 1985).

The cerebrospinal fluid bathing the nerve roots also contributes to their nutrition as demonstrated by the elegant work of Rydevik, Holm, Brown and Lundborg (1984). The pial sheaths of the nerve root comprise a fine gauze-like envelope which permits the free diffusion of metabolites between cerebrospinal fluid and nerve root fibres.

Of all these systems concerned with nutrition of the nerve root, the large thin-walled radicular veins seem to be the most vulnerable component particularly when the nerve root is tethered in the inter-vertebral foramen. These thin-walled veins may also collapse during nerve root longitudinal tension resulting in a functional block of the afferent supply to the nerve root. Such venous insufficiency would probably compromise only half of the nerve root's metabolic requirements (Parke 1985), but perineural inflammation and thickening may also "block" the diffusion pores of the pial sheath.

CAUDA EQUINA AND NERVE ROOT STRUCTURE

Certain anatomical features of the cauda equina and nerve roots predispose them to injury and damage by fibrosis. The peripheral nerve is protected from mechanical pressure, especially at areas of risk such as the fibular neck and the peroneal nerve, by the elasticity of the epineurium. This causes the undulations in a peripheral nerve as it lies in its normal relaxed state. This epineurium however is very poorly developed in the nerve root (Murphy 1977). Not only is the total amount of elastic tissue small, but the collagen structure is sparse and more fragile in composition when compared with a peripheral nerve.

The perineurium is important as a diffusion barrier for the nerve, but also in maintaining intra-fascicular pressure, and is the major structure
resisting tensile forces applied to a peripheral nerve. Again the nerve root has no perineurium. Moreover the fibres within the nerve root do not branch as in a peripheral nerve, but run in parallel in non-plexiform strands which are loosely held together by connective tissue.

In summary, the differences between a nerve root and the peripheral nerve are three-fold.

1. The epineurium which provides a mechanical cushion to compressive forces is less abundant in the nerve root.
2. The fasciculi of nerve roots do not branch.
3. The perineurium which is important in resisting tension forces and as a diffusion barrier against chemical irritants is missing in the nerve root.

The innervation of nerve root has already been described (vide supra).

The posterior nerve root is provided with sensory innervation primarily from fibres originating from cell bodies within the spinal ganglion, whilst the anterior nerve root receives fibres entering its epineural tissue by way of fine twigs from the sinu-vertebral nerve. The sinu-vertebral nerve also innervates structures of the epidural space. Increased intraneural pressure, changes in electrolyte equilibrium or altered nerve or root mechanics will stimulate intrinsic nocicepter (pain receiver) systems resulting in pain.

Finally the peripheral nerve has a fine plexus of intraneural lymphatics which drain the epineural and perineural spaces, and probably enhance the reversibility of oedema of the peripheral nerve. A good lymphatic drainage has not been demonstrated within the connective tissue layers of the nerve roots. It has been postulated that serum protein exchange with the surrounding cerebro-spinal fluid may help to alleviate oedema, but it seems clear that any inflammatory response which gained access to the endoneural space is cleared with great difficulty predisposing the nerve to invasion by fibroblasts, and the development of intra-neural fibrosis (Table 5:1).

NERVE ROOT MECHANICS

The axis of rotation of the spine during flexion and extension is located anterior to the spinal canal. This means that the spinal canal lengthens in forward flexion and shortens in extension. Breig has shown that this change in length is in the order of 7 cm. in the average adult (Breig 1960). Breig has also shown that the neural tissue does not slide in the canal during spinal motion, but rather adapts to length change by passive deformation (Breig 1963). Marten's studies on autopsies showed
<table>
<thead>
<tr>
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<th>NERVE ROOT</th>
<th>PERIPHERAL NERVE</th>
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<tr>
<td>EPINEURIUM</td>
<td>Sparse, providing little protection from compression</td>
<td>A strong mechanical cushion protecting against compression and bony prominence</td>
</tr>
<tr>
<td>PERINEURIUM</td>
<td>Sparse, providing minimal protection from tension, and chemical irritants</td>
<td>An important mechanical and chemical barrier resisting diffusion of chemical irritants</td>
</tr>
<tr>
<td>FASCICULI</td>
<td>No branching</td>
<td>Frequent branching</td>
</tr>
<tr>
<td>INNERVATION</td>
<td>Richly innervated with pain fibres</td>
<td>Well innervated with pain fibres</td>
</tr>
<tr>
<td>BLOOD SUPPLY</td>
<td>Precarious at level of anastomosis in cauda equina</td>
<td>Rich blood supply</td>
</tr>
<tr>
<td>LYMPHATICS</td>
<td>Ineffective, such that any inflammatory exudate is organised into fibrous tissue causing intra-neural fibrosis and adhesion formation</td>
<td>Abundant</td>
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Table 5:1. A comparison of the structure and function of the lumbar nerve root compared with the peripheral nerve
more recently that when the lumbar spine moves from flexion to extension the spinal canal shortens by an average of 2.2 cm., so that the contained nerve tissue also shortens and becomes wider. At the same time the fibres of the yellow ligament become slack and their cross-sectional area increases (Marten 1984). (Fig. 5:7 and 5:8).

It is clear that the dura is firmly anchored at its cranial end to the posterior fossa, and distally it is relatively fixed by way of fibrous connections between the root sleeves and the intervertebral foramina. The nerve root itself however in both the cervical and lumbar regions passes freely through the subarachnoid space of the dural sac and root sleeve (Murphy 1977). Within the root pouch the space between the arachnoid and the surface of the nerve root is bridged by fine fibres and the distance between the arachnoid and the pia is short. These bridging fibres may be dense particularly in older patients (Sunderland 1974). In young subjects there is some to and fro motion of the spinal nerves at the foramen (Inman 1942). Goddard however demonstrated that similar nerve root mobility occurred in the older age group as well in whom dense adhesions are common in the presence of disc degeneration (Goddard 1965).

The sciatic nerve moves during straight leg raising. When the heel was raised only one to three inches from the horizontal position the sciatic nerve moved at the level of its exit from the pelvis (Goddard 1965). At 20°–30° stretching of the nerve occurred at the intervertebral foramen and movement continued until after 70° when little additional movement took place. These results were dependent upon the patient's age and the presence of adhesions.

The length to which a spinal nerve may be stretched before nerve conduction is lost on the purely mechanical basis has not been investigated but is assumed to occur at or below the elastic limit, which is approximately fifteen per cent in spinal nerves (Sunderland 1961). The degree of deformation required to inhibit conduction is probably considerably reduced in conditions of inflammation and oedema accompanying disc herniation and degeneration. It has been noted in cadaver dissections of patients experiencing sciatica at the time of death that the inflammatory response around the nerve root and the development of adhesions may extend for a considerable distance beyond the nerve root (Goddard 1965).
Figure 5:7. The influence of changes in the soft tissues with advancing age on the dimensions of the nerve root canal. 'A' represents a young spine and 'B' an older spine with loss of disc height. Similar changes affect the central canal also.
Figure 5.8. Two drawings to illustrate the influence of flexion and extension on the dimensions of the nerve root canal. These factors of translocation and distortion of soft tissues also influence the central part of the spinal canal.
Redundant Nerve Roots in Degenerative Spinal Stenosis

The neurosurgical literature has reported over a number of years that some patients develop redundant nerve roots (Sorensen 1975, Thulin 1978). Following investigation of one hundred and seventeen cadaver spines, one hundred and seventeen patients with degenerative lumbar canal stenosis, operative inspection of the nerve roots of the cauda equina on fifty-six patients and electrophysiological testing, Tsuji concluded that there were three types of redundant nerve roots: (i) undulant, (ii) fine wrinkled, and (iii) combined (Tsuji 1985). At operation with dural incision in twelve patients the nerve roots tended to be coiled just caudal to the conus medullaris. Redundant nerve roots were more often sensory than motor. Myelography revealed that twenty-two of the fifty-six patients (thirty-nine per cent) with degenerative spinal stenosis had redundant or slack nerve roots.

A possible mechanism of production of redundant nerve roots in degenerative spinal stenosis is that with loss of disc height and loss of overall length of the lumbar spine, the nerve roots of the cauda equina become relatively slack. These slack nerve roots may be gripped tightly in the nerve root foramen and caudal spinal canal such that just caudal to the conus medullaris the redundancy of the nerve roots becomes most prominent with at times the development of coils of nerve roots as first described by Ehni (1971).

THE BLOOD SUPPLY OF THE LUMBAR VERTEBRAL COLUMN

The blood supply to the vertebral bodies is very rich and not easily damaged. The blood supply to the posterior elements however is more precarious and susceptible to impairment following routine surgical approaches for firstly exploration of the spinal canal and decompression of the spinal canal stenosis, and secondly posterior spinal fusions.

In the lumbar region there are usually four lumbar arteries which arise in pairs. Each lumbar artery gives off two sets of branches, a short central branch and longer ascending and descending branches. These give rise to three main sets of branches, firstly to the vertebral body wall, secondly into the spinal canal, and thirdly to the posterior spinal elements. The intermediate or spinal canal branches divide into three again, firstly the anterior spinal branches, secondly the branches to the nervous system, and thirdly the posterior spinal branches to the vertebral column (Crock 1976).
Venous drainage is via a confluence of the anterior and posterior internal vertebral venous plexus, which emerges from and surrounds the nerve root in the intervertebral foramen. These vessels drain the vertebral body.

Surgical Relevance

It is important to avoid damage to the intermediate or spinal canal branches in particular during nerve root decompression. These vessels give rise to branches which are level with or anterior to the pars interarticularis, and may easily be damaged by diathermy at this point.

In the upper lumbar region spinal canal decompression may result in damage to the great radicular veins which lie just inside the spinal canal. A proper appreciation of the site of origin of the arteries and veins which supply the vertebral column and are connected with the supply to the nervous system is of great practical significance if permanent damage to the cauda equina or even the conus medullaris is to be avoided (Crock 1976). During surgery magnification is of great help to identify these vessels which should be protected during surgery, but if damage occurs haemorrhage should be controlled using bipolar coagulation and not routine diathermy which may earth via the important neural vessels. The second technical point is that it is obviously essential that the accurate localisation of the bleeding point should be achieved before any coagulation is attempted.

THE ANATOMY OF THE FACET JOINTS

Piersol stated that the outer portions of the ligamentum flavum are inseparably attached to the loose articular capsules of the facet joints preventing the infolding of the capsule during movement (Piersol 1930). The ligamentum flavum encroaches upon the medial part of the facet joint capsule and it was considered necessary to prevent the capsule from being "nipped between the articular surfaces during movement". The ligamentum flavum also, according to Cunningham, restored the facet articular surfaces to their normal position after movements of the spinal column (Cunningham 1912).

Goldthwaite recognised two well known variations from the normal skeleton liable to produce backache (Goldthwaite 1911):

1. The tall slender visceroptic type with long narrow flexible spines and flat lumbo-sacral facets.
2. The short thick heavy type in whom the diarthroidal facets are large and crescentic and limit the motion of the vertebra particularly in flexion.

Putti stressed the importance of asymmetrical development of the articular facets regarding it as an anomaly of articular tropism (Putti 1937), and Goldthwaite also pointed out that if the facets were asymmetrical then movements were irregular (Goldthwaite 1911).

In a review of three thousand radiographic studies of the lumbo-sacral spine Brailsford found that fifty-seven per cent of the lumbo-sacral facets pointed backwards, twelve per cent pointed inwards, and thirty-one per cent were mixed or asymmetrical (Brailsford 1928). It has been shown that in cases of facet joint tropism, there is an increased incidence of disc prolapse on the contra-lateral side, since the anomalous joint blocks rotation on the ipsilateral side (Farfan 1967).

The orientation of the laminae and facet joints of the lower lumbar spine has been analysed by Van Schaik at two hundred and twelve vertebral levels in one hundred and twenty-three patients, using computerised tomography with slice thickness of 3 mm. or 4.5 mm. The relationship between the facet joint and caudal part of the lamina was investigated (Van Schaik 1985). At the L3-4 level, both facet joints and caudal portions of lamina tend to be sagittally orientated, whilst at the L5-S1 level they are more coronally orientated with L4-5 intermediate. The only link between the inferior articular facet and the vertebral body is via the pars inter-articularis and the lamina, and the lamina at this point acts as a buttress to the inferior facet.

ANATOMY OF THE LIGAMENTUM FLAVUM

Verbiest described the hypertrophic ligamentum flavum as a compressive agent in lumbar spinal canal stenosis (Verbiest 1975). In most cases of "pure absolute stenosis", the inter-laminar spaces were too small to allow the hypertrophic ligament to bulge into the spinal canal on extension of the spine. In one case of "pure absolute stenosis" described by Verbiest, the ligamentum flavum showed irregular thickening, calcification and cysts producing additional narrowing of the spinal canal. In three cases of "pure relative stenosis" the ligamenta were hypertrophied to a degree such that they might have bulged into the spinal canal with extension.

A further study of the anatomy of the ligamentum flavum showed that even a moderate enlargement could cause compression of the nerve
roots (Brown 1938). The ligaments were described as arising from the dorsal and upper margins of each lamina, passing upwards beneath the lamina immediately above and entering the neural canal. They fuse in the midline and laterally have expansions which extend well down towards the antero-lateral aspects of the neural canal, and form the posterior margins of the intervertebral foramina. As described the ligaments lie close to the articular facets and to some degree form a capsular covering for the articulations, particularly anteriorly.

Directly anterior to each ligament lies the intervertebral disc and the very narrow space between the two transmits the nerve root after it emerges from the dural canal. An increase in the size of the ligament, a posterior protrusion of the disc, or a combination of the two may easily compress the nerve root at this point.

The normal ligament is composed entirely of yellow elastic fibres and grossly has considerable elasticity. Post-mortem examination of patients who have undergone surgical removal of the enlarged ligaments reveals bulbous expansions of the ends of the ligaments which appear to have retracted following partial removal and undergone further thickening through scar tissue formation (Brown 1938). It was recommended that removal should be performed as short a distance as possible from the attachment to bone, so that further compression by the retracted ligamentum flavum does not occur and result in further nerve root compression. Calcification may occur in residual ligamentum and further embarrass the nerve root.

The ligamentum flavum is often the structure contributing most significantly to constriction of the dural sac in the midline and the nerve roots in the lateral recesses or foramina. Using special histological staining techniques, scanning electron microscopy, and biochemical analysis for elastin, collagen, lipid, and DNA content, Weinstein examined surgical specimens in sixteen patients for comparison with normal tissue obtained at autopsy (Weinstein 1983). Despite a four-fold increase in thickness of the ligamentum, there was no statistically significant evidence of disproportionate increase in any of its components. Although focal areas of granulation or collagen scar deposition and disruption of the normal elastin fibre pattern were commonly observed, there was no significant alteration in the elastin to collagen ratios comparing surgical spondylosis specimens with autopsy controls. Although focal inflammation was occasionally seen in association with microscopic areas of haemorrhage within the ligament, the overall ratio of cells to ground substance was also not altered in the pathological material. In three
cases amyloid-like deposits were seen without evidence of any systemic disorder. Islands of cartilaginous and osseous metaplasia were also occasionally identified. Thus, it would appear that pathological enlargement of the ligamentum flavum in lumbar stenosis and spondylosis patients reflects excessive growth of this strategically located inter-laminar ligament without occurrence of fibroblast hyperplasia or selective increase in any of its components.

According to scanning electron microscopic observation of the ligamentum flavum removed from spines with no abnormal ossification on radiographs, elastic fibres formed a dense and regular pattern with interconnecting micro-fibrils.

Observation of the ossification of the thoracic ligamentum flavum showed degenerative change of fibres, followed by the appearance of numerous osteocyte lacunae with granular substance. Finally osteocytes appeared resulting in enchondral ossification (Oka 1982).

Ossification and calcification of the ligamentum flavum are two completely different conditions.

PARASPINAL MUSCLES

It has been shown that the lumbar muscles change their histological staining characteristics in patients with sciatica (Jowell 1975). The normal harmonic mosaic pattern of rapid and slow fibres changes to a predominantly slow fibre pattern, most likely the result of partial denervation or inactivity.

ANATOMY OF CONGENITAL SPINAL STENOSIS

The pedicles are short and the lamina thick in patients with achondroplasia. Although there are forty-two reported descriptions of surgery of the spine in achondroplasia, there is unfortunately very little anatomical detail given in these. The anatomical features of a fifty-five year old achondroplastic male with severe non-vascular claudication, who died from a myocardial infarct, were however studied by Lutter and others (Lutter 1977). Comparisons were made with a normal control.

The cross-sectional area of the spinal canal was measured at different levels and compared with the control. The size of the canal decreased from L1 to L5, being thirty-nine per cent of the control at L1 and twenty-seven per cent of the control at L5. Six parameters were measured to
determine which were responsible for this reduction in cross-sectional area.

1. **Antero-posterior dimension and pedicle height**
   There was a significant reduction in the antero-posterior height of the canal at the L4 and L5 levels compared with the control. Direct measurements of the pedicles showed shortening.

2. **Lamina**
   The lamina at comparable levels appear to be equal.

3. **Interpedicular distance and pedicle thickness**
   The interpedicular distance was a third smaller for the achondroplastic spine than controls, whilst the pedicles were in the range of thirty to forty per cent thicker in the achondroplast, giving approximately the same outer interpedicular distance in the achondroplastic patient as in the control.

4. **The inferior facet size**
   A marked protrusion of the inferior facet to the nerve root canal was seen in the achondroplastic patient. This was due to degenerative change.

5. **Nerve root canal size**
   The nerve root canal was found to be 1 mm. to 2.8 mm. smaller in the achondroplast than in the normal control.
   Clearly when surgical decompression of an achondroplastic patient with spinal stenosis is performed, these anatomical features must be borne in mind.

**THE NORMAL LUMBAR SPINAL CANAL WITH RACIAL VARIATIONS**

The lumbar spinal canal is principally oval at the L1 and L2 levels in cross-section, but this shape is lost with increasing frequency at the lower levels. In particular it is the trefoil canal which is found in association with spinal stenosis mostly at the lower lumbar levels. The shallow lateral recesses are occupied almost entirely by the nerve roots, with a result that any further intrusion into the space will affect their mobility and produce nerve root damage. Spinal flexion increases the size of the vertebral canal, and the size of the inter-vertebral foramina. Conversely extension of the spine decreases the cross-sectional area of the canal and the size of the inter-vertebral foramina (Jayson 1982). Eisenstein in 1976 measured the spinal canal and vertebral bodies of two hundred and seventy-five skeletons, one hundred and thirteen of these were "hite" Caucasian skeletons, and one hundred and sixty-two were Zulu skeletons (Eisenstein 1976). The
antero-posterior and transverse diameters were measured in one thousand three hundred and forty lumbar vertebra using a Vernier scale. The spinal index was calculated according to the method of Jones and Thomson (Jones 1968), that is the product of the antero-posterior and transverse diameters of the spinal canal expressed as a ratio of the product of the same diameters of the vertebral body. In radiological measurements the posterior limit of the spinal canal was established by placing metal markers within the canal, and from the L1 to the L4 vertebra it was found to coincide with a line drawn from the apex of the superior articular facet to the apex of the inferior articular facet. At the L5 level the posterior limit of the spinal canal was found by a line placed 1-2 mm. in front of the anterior border of a large radiolucent "lake" within the spinous process.

Little difference was found between the canals of males and females, notwithstanding the difference in size of the vertebral bodies, but the Negro canal was found to be slightly less capacious than the Caucasian. Interestingly enough however, spinal claudication is not reported in the South African Negro. (Fig. 5:9 and 5:10)

When radiological measurements were made on the plain film, the lower limit of normal was 15 mm. for the antero-posterior diameter, and it was found that there was a magnification factor of 1.23 (twenty-three per cent) for the antero-posterior diameter, and 1.14 (fourteen per cent) for the transverse diameter. The spinal index, that is the size of the spinal canal relative to the size of the vertebral body, was found to be an unreliable index because of considerable variations within the normal individuals and in the sexes (Eisenstein 1976). This study therefore relied on absolute measurements using the Vernier scale to make comparisons.(Fig.5:11)

ANATOMICAL RELATIONSHIP BETWEEN THE LUMBO-SACRAL NERVE ROOTS AND SURROUNDING TISSUES

It is interesting to note that the space around the nervous tissue both in the spinal canal and the intervertebral foramen is narrower in the male than in the female (Hasue 1983). Hasue dissected and carefully examined cadavers of fifty-nine elderly people of average age seventy-one point eight years. Dissections and radiographs were photographed and subsequently analysed. The results were based essentially on the analysis of horizontal sections.

1. **Topographic anatomy of the lumbo-sacral nerve roots**

The course of the nerve root was found to be influenced by the disc,
Figure 5:9. Photographs taken to illustrate the transition between the first lumbar and first sacral vertebrae in the shape of the spinal canal. The first lumbar canal is clearly rounded including scalloping of the vertebral body, whilst the first sacral canal is clearly flattened in the sagittal plane and widened transversely. This shape reflects accurately the orientation of nerve roots in the cauda equina and the shape of the spinal cord influencing the shape of the spinal canal. The cephalad surface of the pedicle is not smooth (see Fig.5:10)
The undersurface of the pedicle appears extremely smooth.

The normal sliding effect of the nerve root immediately caudal to the pedicle during embryological development and growth is probably the most important influence on the shape and smoothness of the undersurface of the pedicle. This is well shown in Figure 5:10 D, which is the view from below of the fifth lumbar vertebra.

Figure 5:10. Photographs taken from below of the four lowermost lumbar vertebrae illustrate the transition from the oval shaped canal found in the thoracic spine to the more triangular shaped canal of the fifth lumbar vertebra.
ANTERO-POSTERIOR AND TRANSVERSE SPINAL CANAL MEASUREMENTS

Figure 5:11. The antero-posterior and transverse diameters of all three spinal canals are identical. Nonetheless spinal canal 3 is severely stenotic. The cross-sectional area of the spinal canal particularly when related to the cross-sectional area of the contained nerve tissue is more relevant in the diagnosis of spinal stenosis than either diameter.
the articular facet and the pedicle of the vertebral arch. This may correspond to the findings of the "superior facet syndrome" proposed by Epstein (Epstein 1972). Congenital anomalies of the nerve root were found in five cadavers (eight point five per cent). A communicating branch between two roots was found in one cadaver, and conjoined roots in which two roots shared the same root pouch were found more frequently and occurred in four cadavers (Hasue 1983).

2. Cross-sectional anatomy of the spinal canal
Jacobsen divided the spinal canal into two alternating parts, the intra-osseous, and the articular segments (Jacobsen 1975). The articular segment is clearly clinically more important. At the intra-osseous segment of the L5 level the spinal canal is trefoil in shape, having deep lateral recesses on both sides. At the bottom of the recess the cross-sectional nerve roots can be observed. Microscopic examination of the nerve bundles showed separation with proliferation of fibrous tissue between them. This is a characteristic feature of intraneural fibrosis (Hasue 1983).

3. Spatial relationship of the nervous tissue to osseous and non-osseous elements of the spinal canal and the intervertebral foramen
The mean area of both the non-osseous and the osseous spinal canals in the male are largest at the L5 level and the smallest in the middle of the lumbar spine. In the female however no such tendency was clearly observed. On the other hand, the mean area of the nervous tissue becomes smaller from L1 to L5 in both sexes, except for the L5 level in the female. The non-osseous and osseous spinal canals are wider in the female except for the L5 level. On the other hand the nervous tissue is larger in the male, except for the L5 level.

Microscopic examination of the intervertebral foramen with an intact non-osseous element indicate that the shape is oval both at the internal and external ostia. However, the shape is almost triangular after removal of the soft tissues. The ratio of nerve tissue to the osseous intervertebral foramen and of nerve tissue to the non-osseous intervertebral foramen increases from the L1-2 level to the L5-S1 level when measured at the internal ostium. These ratios are largest at the L5-S1 level. That is the available space around the nerve root diminishes caudally especially in the male, and especially at the L5-S1 level. Also the space around the L5 root is much narrower at the lateral recess in the male. This relationship is not established in the female however.
4. **Topographic anatomy of the tissues surrounding the nerve roots**

The "epidural membrane" consists of fibres running from the surface of the dura to the superficial layers of the posterior longitudinal ligament. In the intervertebral foramen around the nerve root there is a rather thick membranous structure and this epidural membrane is continuous with the epiradicular sheath forming a sort of envelope around the nerve root.

Ossification of the spinal ligaments is frequently found in clinical presentations. Ossification of the yellow ligament is most frequently seen in the thoraco-lumbar spine, and that of the posterior longitudinal ligament in the cervical spine. The posterior longitudinal ligament may however sometimes be calcified in the lumbar spine, and may be a cause of radicular symptoms.

Developmental stenosis or degenerative stenosis without spondylolisthesis is more frequent in the male. It is also most common at the L5 or L5-S1 levels, and it is at this level that the space around the nerve root is maximum within the spinal canal, but narrowest within the intervertebral foramen. Again this is more marked in the male. The L5 root is also most frequently involved in the narrow lateral recesses of degenerative stenosis.

The epidural membrane or extradural membrane described by Dommisse (Dommisse 1975) is continuous with the epiradicular sheath identified by Hasue and appears to have principally a protective role for the nerve root (Hasue 1983), but further studies are required to determine its physiological role and significance in the development of spinal stenosis.

**SPINAL CANAL RESERVE CAPACITY**

It has already been stated that a reduction in the absolute dimensions of the spinal canal when measured radiologically (antero-posteriorly and transversely) does not necessarily indicate active spinal stenosis. Histological studies and the increased use of computerised tomography have enabled identification of a detectable space between nervous tissue and bony canal. It is most likely the obliteration of this space which induces clinical symptoms in the elderly and in persons with developmental or acquired lumbar spinal stenosis. The ratio of the volume of the spinal canal to the volume of its contents is therefore clearly the most critical measurement (Weisz 1983).
The situation is further complicated by the fact that variations in the position of the spine can influence the distance between the subarachnoid space and the bony canal, as shown myelographically by Larsen and Smith (Larsen 1980). The study by Larsen and Smith represents a significant attempt to revise the static view of stenosis towards the more functional dynamic view.

This dynamic view of spinal stenosis brings into consideration the concept of mobility of the nerve roots. Clearly there is a form of "constrictive stenosis" in which symptomatic spinal stenosis is brought about by bony or ligamentous reduction in the absolute dimensions of the spinal canal. On the other hand, however, when there is restriction of nerve root mobility, most frequently caused by adhesions, symptoms can occur even in the absence of bony canal stenosis (Weisz 1982). This type of "restrictive stenosis" presents problems of management (Chapter 22).
CHAPTER 6

PHYSIOLOGICAL STUDIES

INTRODUCTION

One of the major ways in which the practice of medicine differs from the application of a pure science is that frequently treatment is provided on an empirical basis for conditions which are poorly understood. Few instances illustrate this better than the management of patients with low back pain. This chapter discusses a number of studies, some in humans and some in animals, which have attempted to localise the source of pain in patients with low back pain and spinal stenosis, and provide a rationale for the observed symptoms and signs upon which treatment can be based.

The patho-physiology of nerve root dysfunction produced by compression, tension, chemical irritation, or vascular impairment is described, together with the chemical effects of chymopapain on intra-neural microcirculation and axonal transport of proteins. The pathogenesis of spondylosis and stenosis will be considered. An attempt has been made to monitor the pressure required to produce a spinal block on myelography and this will be described.

NERVE ROOT TRACTION

In 1958 Smyth and Wright described an elaborate clinical experiment in which loops of nylon thread were placed around the exposed nerve roots at operation and brought out through the skin, so that at the end of surgery the nerve roots could be stimulated by pulling on the nylon thread (Smyth 1958). The description of pain or discomfort experienced by the patient was documented.

In the first part of the experiment, nylon thread was pulled gently which caused the patient to jump and experience a severe pain shooting down the buttock and thigh into the calf above the lateral malleolus when the first sacral nerve root was stimulated. This was relieved immediately the tension was released. After a continuous but gentle pull for a quarter of a minute the toes and dorsum of the foot began to feel numb. With tension applied on the thread it was not possible to perform a straight leg raise test because of sciatic pain, but when the tension was relaxed the straight leg raise was to 50° and 60° without pain.
In a second part of the experiment the thread was applied around the dura and tension on the thread produced pain in the buttock. This was not a severe pain unlike the thread around the nerve root itself, and it was concluded that the dura mater appeared virtually insensitive whilst the nerve root appeared hypersensitive. The third part of the experiment was traction on the ligamentum flavum, and this produced a dragging sensation after pulling hard for a few seconds, felt deep in the back in the region of the wound. It was not painful; the patient could not clearly distinguish between periods when the thread was pulled and periods when the thread was not pulled.

Traction on the interspinous ligament caused only minimal discomfort and when a steady pull was maintained the patient was virtually unaware of what was happening, but was aware only of the thread being pulled through the wound.

It was considered that the nerve root had not been stretched by pulling on the thread since the thread was pulled only enough to take up the slack. The nerve root had simply been touched by the nylon (Smyth 1958). The nerve roots appeared exquisitely sensitive, whereas the ligamentous structures appeared less sensitive. Further, traction on the annulus fibrosus did not elicit pain.

It was noted that the longitudinal extent of the pain appeared proportional to the amount of pressure exerted on the nerve root, whilst the length of time the pressure was maintained did not appear to be of any significance. It may be that the extent of the pain was an indication of the degree of pressure on the sciatic nerve roots, and that those patients who present with sciatic pain in the thigh alone should respond to conservative treatment, whilst those with more extensive sciatica involving the whole extremity may respond less well to conservative treatment.

Electromyographic studies indicated that sciatic pain in the leg was not produced as a result of muscle spasms when the nerve root was irritated (Smyth 1958). Some degree of tolerance developed after a few stimulations, and it was clear that the nerve root which had been under pressure within the spinal canal was much more sensitive to the touch of the thread than a neighbouring nerve root which had been uninvolved. Prolonged irritation of the nerve root appeared to have made the root hypersensitive. This did not settle immediately following surgery, and the root remained hypersensitive for some time, possibly related to the development of reactionary fibrosis within the root or the tethering of the nerve root.
which prevented it moving freely and adapting itself to spinal movements. It is possible that the tethering of the nerve root associated with movement is analogous to the pulling of the nerve root with the nylon thread.

NERVE ROOT IRRITABILITY

Not only does the tethering or mechanical irritation of a nerve root provoke pain, but in addition the chemical milieu bathing the nerve root may be partly responsible for nerve root irritability. It is possible that since the glycosaminoglycans of the degenerating disc is acidic, it would cause pain if it leached out. Measurement of the pH around the nerve root of some patients undergoing surgery revealed a very high hydrogen ion concentration in a few patients who demonstrated many scars and adhesions around the nerve roots (Nachemson 1969). It is clear that a high hydrogen ion concentration in tissues causes pain (Lindahl 1961). Subsequently however it was demonstrated that this was not due to the glycosaminoglycan concentrations, but rather to an increased amount of lactate (Diamant 1968). Lactate in large amounts is produced when fibroblasts and other cells metabolise under anaerobic conditions. The disc is the largest avascular structure in the body being devoid of directly penetrating vessels from the age of fifteen onwards. The area between the nucleus and the annulus posteriorly is an area under much stress, and this is the part of the disc where metabolic requirements are probably least well met.

THE PHYSIOLOGY OF NERVE ROOT INJURY

It is frequently found during surgical decompression procedures that the nerve root is quite often swollen and inflamed with an injected surface, and is exquisitely sensitive and irritable to even the slightest manipulation, provoking muscle contraction during nerve root mobilisation. This contrasts distinctly with manipulation and mobilisation of nerve roots which are neither inflamed nor irritable. On occasions when laminectomy has been performed under local anaesthetic even the slightest tension on an inflamed nerve causes radicular pain. If tension is increased the patient develops symptoms of paraesthesia in the extremity, and when tension is prolonged or increased sensory deficits develop and motor loss occurs (Murphy 1977).

According to Sunderland simple compression of nerve tissue in the absence of inflammation results in painless loss of motor and sensory
function (Sunderland 1968). Holt correlated the histology of cervical disc degeneration with adjacent spinal nerve fibrosis in a cadaver study (Holt 1966). The nerve was normal in ninety-two per cent of cases. However if the disc was degenerate the adjacent nerves showed intraneural fibrosis in over sixty per cent of cases.

It seems reasonable to conclude that the experience of pain occurring in nerve root compression or tension is mediated through alterations in the neural structures themselves, and that these alterations involve complex chemical and physiological events, that are not simply a reflection of mechanical forces. Following injury to the nerve root there is an immediate outpouring of serum albumin into the endoneural space at the level of injury, and for a distance both proximally and distally. There has been no evidence of the existence or abundance of mast cells in human spinal nerves or nerve roots. The leakage of serum proteins into the endoneural space poses a major problem to the nerve root because this space is devoid of a lymphatic system. With time the exudate is invaded by inflammatory cells with the eventual conversion to intraneural fibrosis. (Fig. 6:1)

The presence of intraneural fibrosis creates a permanent and irreversible focus of potential irritation because:

1. It alters the biomechanical properties of the nerve. Essentially it prevents the "accordion" effect. This normally occurs as the nerve root is relaxed, such as during lumbar spine extension with the hips and knees also extended.

2. The presence of intraneural fibrosis interferes with the normal intrinsic vascular supply of the root.

3. Replacement, constriction, and loss of axons which disrupts the normal balance of neural transmission through the involved segment of the nerve root.

THE ARTIFICIAL SYNAPSE

Under normal conditions the myelin sheath functions as an insulator which prevents the action currents associated with the passage of a nerve impulse along one fibre from spreading to interfere with electrical activity in neighbouring fibres. It was first recognised by Hering in 1882 (Hering 1882) and subsequently other investigators including Renshaw and Therma (Renshaw 1941). Sensory fibres have a lower threshold for stimulation. Acute inflammation may result in artificial synapse formation whereby neural activity in the spinal nerve is funnelled into low threshold
pain-carrying fibres (Fig. 6:1). It is probable that paraesthesia and tingling as well as pain are expressions of such abnormal patterns of neural activity due to short circuiting of waves of depolarisation.

It may be therefore that the increased pain in the sciatic nerve distribution which occurs during activity in patients with spinal stenosis may in part be due to the development of an artificial synapse. In this event, waves of depolarisation travelling down motor axons would be short-circuited and result in waves of depolarisation in afferent pain or sensory nerve fibres.

Intraneural fibrosis affects the elasticity and deformability of the neural tissues, resulting in abnormal mechanical forces within the nerve generated during movement, which in turn starts a vicious cycle resulting in further "chronic mechanical inflammation" of the nerve root. The intrinsic vascular supply of the nerve root is affected by fibrosis resulting in chronic ischaemia, which again makes the nerve root tissue more irritable and lowers its threshold of firing. Finally fibrosis causes loss of axons which at present is irreversible and untreatable.

It may be that many patients who present late with spinal stenosis and are treated by adequate and extensive decompression, fail to gain benefit symptomatically because of such permanent changes already present within the nerve root. It is clear therefore that the effective treatment of patients with spinal stenosis involves not only prompt decompression, but also adequate control of nerve root inflammation, which may persist even following decompression.

Changes in nerve function during acute graded compression have been observed in the rabbit vagus nerve (Rydevik 1980) and the rabbit tibial nerves (Rydevik and Nordborg 1980). These nerves were chosen because of their accessibility. It is clear however from the preceding chapter on anatomy that the structure and function of the nerve roots and the cauda equina cannot be compared with peripheral nerves. The results of these studies therefore will not be quoted, and there remains no satisfactory study of the results of graded compression on nerve root function and structure.

THE CIRCULATORY RESPONSE OF THE CONTENTS OF THE SPINAL CANAL TO EXERCISE

A well known principle of physiology is that as the metabolic rate of an organ increases with activity, there is an accompanying local vaso-
Large motor neurone heavily insulated with high depolarisation threshold

Small fine unmyelinated pain fibres with low depolarisation threshold

At site of ischaemia and inflammation (which may be traumatic or chemical) intraneural oedema is organised by fibrous tissue ingrowth into the nerve fibre with adhesion formation (there are no intraneural lymphatics)

Leg Pain on Exercise may be caused by: (a) spontaneous depolarisation of pain fibres on activity; (b) artificial synapse in nerve root converting motor neurone action potential into pain fibre action potential

Figure 6:1. To illustrate, diagrammatically, the effects of compression or tension on the nerve root with possible mechanisms of production of neurogenic claudication on exercise. Intraneural fibrosis with loss of myelin sheath insulation short-circuits motor efferents to become pain afferents. Partial depolarisation of pain fibres results in spontaneous action potentials with nerve root motion or compression.
dilation and increased blood flow. This has been demonstrated in the secreting submandibular gland by Claude Bernard (Bernard 1858). Leonard Hill first suggested that there was a relationship between brain metabolism and its blood flow (Hill 1896). The animal experiments of Cobb and Talbot (Cobb 1927) and Serota and Gerard (Serota 1936) confirmed the thesis that nerve cell activity in cerebral cortex is accompanied by a local increase in blood flow.

Many years previously Gowers inferred such a relationship in the spinal circulation (Gowers 1899). He stated:

"an active congestion which may be called secondary seems to result from prolonged and violent action of the nerve elements of the spinal cord. Thus local vasodilation and distension of vessels, migration of leucocytes into the sheath and into adjacent tissues and even small extravasations are found in some cases of hydrophobia, of tetanus and of strychnine poisoning, and it is probable that similar congestion attends all violent physiological activity, violent and continued exercise, coitus, etc. Coitus several times repeated has been known to cause haemorrhage within the cord which must be accompanied by intense congestions. Vascular dilation with an increased blood supply is the physiological attendant of functional activity in all organs, and doubtless also in the cord, but it is morbid only when excessive."

Trueta and Hodes have shown that extensive haemorrhage in the lumbar region of the spinal cord may result from the injection of highly irritant substances (such as ten per cent formol saline or croton oil) into the thighs of mice. They also indicated that intense hyperaemia of the lumbar cord accompanied swimming to exhaustion (Trueta 1954).

The relative simplicity of the vascular hilum of the brain can be used to deduce the blood flow in absolute values (i.e. millilitres per minute per one hundred grammes of tissue). Unfortunately such methods are impracticable in the case of the spinal cord which draws its arterial supply from many different levels and from which venous return occurs in an equally complex manner.

Blau and Rushworth chose the spinal flexor reflex elicited by single shock to a hind limb in an anaesthetised animal as a means of comparing the size and density of the blood vessels on the two sides of the cord, and related these to changes in the level of motor activity (Blau 1958). The control animals which were not exercised showed no definite pattern with random scatter of scores for density of blood vessels on each side of the cord. In the exercised animals however there was a well-defined maximum of intensity of blood vessels in the lumbar region, extending into the sacral
segments of the mouse cord. This suggested that vascular dilation and increased density of blood vessels was localized to the homolateral region of the spinal cord segments activated. They were unable to demonstrate a simple relationship between the duration of exercise and the vascular response. But in the cords of mice exercised for thirty minutes the increased density and dilation of blood vessels on the homolateral side was sometimes as intense as in mice exercised for much longer periods up to on hundred and five minutes. There was a great deal of individual variation from one animal to another.

From the work of Blau and Rushworth it would appear that neural tissue might have an adequate blood supply to maintain normal structure, but inadequate for physiological function. The neurological deficit in cases of spastic paraplegia of undetermined origin (Marshall 1955), Arnold Chiari mal-formation, basilar impression and cervical spondylosis (Mair and Druckman 1953), might be considered in terms of inadequacy of the blood vessels supplying the nervous tissue. This at least was the view of Blau and Rushworth (Blau 1958).

It was subsequently postulated that when a person walks there is an increase in blood flow in the vessels of the cauda equina and nerve roots, with an increase in size and possibly number of afferent and efferent blood vessels (Blau 1961). In those patients with firm constriction of the nerve roots by a disc protrusion or stenosis, the dilation and increased blood supply in response to activity of the roots is prevented by comparison, and the symptoms of ischaemic neuritis result. This accords well with the clinical observation of the constant amount of activity needed to produce symptoms and its rapid resolution as soon as activity ceases, as well as with the physiological data of the evolution of sensory before motor symptoms in ischaemic nerve compression (Lewis 1931).

Lumbar spondylosis usually spares the highest lumbar spinal segments (Wilson 1971), and therefore the great anterior medullary artery which enters most often by the first or second lumbar segment on the left, is only rarely involved in narrowing of the spinal canal, and degenerative change. Certainly reports of ischaemic claudication give no reason to suspect involvement of this vessel and impairment of radicular arterial flow at a more caudal level through root compression would be consistent with clinical observations and experimental studies.

An interesting observation in patients with spinal stenosis was that symptoms could be reproduced by ergometric exercises of the leg whilst
lying in bed (Evans 1964). The symptoms were not affected in severity or
time of onset by increasing or decreasing the lumbar lordosis by means of
pillows. The onset of symptoms was related to the rate of exercise as
well as its duration. This evidence again supports the work of Blau and
Rushworth, that there may be an increase in pressure on a normally
confined nerve root during exercise due to vasodilation (Blau 1958). There
may also be an increase in volume of the active nerve fibres during
exercise (Hill 1950). The claudication time of spinal stenosis patients has
also been shown to vary directly with the oxygen tension of inhaled gas
(Evans 1964). There was in this experiment however a lag before the
effect of change in the respired gas became apparent. When air alone was
breathed claudication occurred between one and four minutes, when twelve
per cent oxygen was inspired claudication time extended to five to seven
minutes, and when one hundred per cent oxygen was inspired the claudication
time extended further to between eight and eleven minutes.

Unfortunately the work of Blau and Rushworth has been frequently
misquoted. The statement that:
"the vessels of the spinal nerve roots were particularly
prominent and often widely dilated in exercised animals"
appears frequently in the literature. Clearly this is not so, since Blau and
Rushworth did not examine the vessels of the nerve roots but only the
vessels of the spinal cord (Personal Communications 1983).

One rather crude experiment was performed on adult dogs to produce
ischaemia to not only the cord but the nerve roots by occlusion of one or
more lumbar arteries which caused ipsilateral claudication (Reichert 1934).
The signs of weakness of the hind limbs especially of the hips and thighs
was accompanied at times by temporary incontinence and associated with
delayed spasticity invariably following either ligation of the lower abdominal
aorta or ligation bilaterally of one or more lumbar segmental arteries.
Ligation of one or more lumbar segmental arteries on one side only
invariably produced weakness of the ipsilateral hind limb. In these
experiments exercise resulted in weakness only and never any apparent pain.
Bismuth arterial injections made through the heart filled the entire arterial
system as demonstrated by stereoscopic radiographs, and revealed that the
weakness was not caused by inadequate blood supply to the involved muscle
but could only be due to ischaemia of the spinal cord. With the
development of adequate collateral circulation through adjacent lumbar
arteries, the signs rapidly improved. The author also presented four
patients with weakness of the thigh muscles on exertion (Reichert 1934). In each patient the signs and symptoms of generalised arteriosclerosis were obtained and all exhibited calcification of the terminal abdominal aorta on the radiograph. Reichert postulated that the cord and cauda equina were ischaemic during activity resulting in weakness, but clearly it was not possible from this report to decide how much was in fact the result of vascular claudication in the lower limbs.

**PATHOGENESIS OF NEUROGENIC CLAUDICATION**

The symptoms of neurogenic claudication occur in a large proportion of patients with spinal stenosis during any activity or body position that involves extension of the lumbar spine, and this may be termed "postural spinal stenosis". In a larger group of patients, symptoms appear only after exercise and are related to exertion rather than posture, and may be more appropriately termed "ischaemic claudication" (Wilson 1971). This however may be an artificial distinction since postural claudication may itself be caused by ischaemia in the final analysis. Postural claudication appears to represent a more severe form of neurogenic claudication.

**Postural Claudication**

Postural claudication occurs at rest, and sometimes when the patient reclines depending on the spinal posture he adopts. Breig has shown that extension of the lumbar spine causes bulging of the intervertebral discs, with dorsal displacement of the cauda equina root (Breig 1960). Extension also, by decreasing the length of the spinal canal, forces an increase in the cross-sectional diameter of the cauda equina roots. Ehni has demonstrated the effect of changes in lordotic position during myelography, with extension producing total block and flexion permitting the contrast medium to pass through the blocked area (Ehni 1965). When the spine is immobile, leg movements such as Lasègue's manoeuvre do not move the cauda equina roots (Breig 1960).

Although weight bearing in the upright position normally produces slight bulging of the lumbar intervertebral discs, this factor probably plays an insignificant role in the production of the symptoms of postural neurogenic claudication, because these symptoms can also occur when the patient is recumbent. Ischaemia of the involved nerve roots may occur as a passive event secondary to mechanical compression, if as proposed by Denny-Brown
and Brenner, ischaemia produced by a tourniquet bears a close resemblance to the effects of prolonged local pressure on nerve roots (Denny-Brown 1944).

Ischaemic Claudication

The work of Cranefield, Brink, and Bronk to determine the rate of oxygen uptake in peripheral nerves (Cranefield 1957), and the work of Larrabee (Larrabee 1958) both established the increased oxygen demand of conducting peripheral neural tissues during exercise.

Exercise of the gastrocnemius and soleus muscles on a foot ergometer in patients with spinal stenosis reproduced the patient's symptoms but pain was not maximal in the exercise muscle but in the fifth lumbar dermatome. Wilson found that in two patients whose gastrocnemius muscle was exercised whilst they lay in bed, neither the severity nor the time of onset was affected in the slightest by increasing or decreasing the lumbar lordosis by means of pillows (Wilson 1971).

However measuring the time to claudication whilst the patient breathed air, pure oxygen and a mixture of nitrogen and twelve per cent oxygen did influence the claudication time. The claudication time varied directly with the oxygen tension and on this basis Evans postulated relative ischaemia of active cauda equina roots during exercise (Evans 1964). Blau and Logue also favoured an ischaemic basis for neurogenic claudication because the symptoms could be abolished very rapidly, and because sensation was impaired before motor function (Blau 1961).

EFFECTS OF CHYMOPAPAIN ON NERVE TISSUE

Hirsch first suggested the possibility of injecting a chondrolytic enzyme into the disc to cause a connective tissue reaction thereby enhancing the normal process of disc ageing and thus stabilising the disc (Hirsch 1959).

The first clinical experience of chymopapain injection into the disc space was published by Smith in 1964, who referred to the procedure as chemonucleolysis (Smith 1964). Since then more than fifteen thousand patients have undergone chemonucleolysis as treatment for various low back pain syndromes. During the last few years there has been intensive debate on the efficacy of this kind of treatment as well as on the genesis and frequency of complications, such as anaphylaxis, arachnoiditis, and post-injectional neurological deficits. The intrathecal toxicity of chymopapain
is well known from a large number of animal investigations (Widdowson 1967). Leakage of the injected enzyme from the disc space into the extradural space has been shown to occur in about one in four human beings (Wiltse 1975). Experience from discography further indicates that leakage often occurs when the disc space is injected.

Smith reported that chymopapain had no acute effects on the conductivity of electrical impulses in frog and rabbit sciatic nerves when tested at concentrations of up to forty per cent applied during one hour (Smith 1963). McNab reported experiments where nerve roots had been bathed in chymopapain, and then tested with electrical stimulation. They found no change in conduction time or motor latency time (McNab 1971).

Rydevik noted that chymopapain produced the following effects on the peripheral nerve tissue in rabbits after local application (Rydevik 1976). Four weeks after the application there was widespread nerve fibre degeneration. There was considerable axonal destruction and in transverse sections a severe loss of myelinated fibres was noticed in seven nerves, and a moderate loss in two nerves. Nine out of the ten nerves showed epineural and endoneural fibrosis, whilst perineural fibrosis was seen in one specimen. Four weeks after the application of 1 ml. of 0.4% chymopapain, seven out of nine nerves showed a marked increase in stimulus threshold for eliciting action potentials, compared to the control operated nerves.

The enzyme induces intraneural oedema by affecting the microvessels of the nerve tissue and the protective barrier of perineurium. These structures are made more permeable resulting in intraneural oedema formation and subsequent intraneural fibrosis. The long-term effects are degeneration of nerve fibres and formation of intraneural fibrosis with impaired impulse conducting properties.

It is interesting to speculate that some patient might have been relieved of their symptoms by a "chemical rhizotomy" of nerve root lesions after chymopapain injection. This could conceivably be a possible mechanism whereby sectioning of one nerve root relieves pain without causing any major neurological deficits. Even if the enzyme did not come into contact with the nerve root, there are other neural elements in the areas surrounding the discs, for instance, nerves from the ligaments, dura and joints and the outer part of the annulus fibrosus which might be affected by leakage of chymopapain, resulting in pain relief and possibly explaining some of the beneficial clinical results.
CLINICAL RECORDING OF PRESSURE ON THE SPINAL CORD AND CAUDA EQUINA

Kavanaugh and colleagues suggested that a spinal block might prevent egress of spinal fluid regardless of body position (Kavanaugh 1968). Elevation of pressure below the block would collapse radicular veins, leading to stagnant anoxia of involved cauda equina roots.

Attempts have been made in patients with spinal stenosis to measure the pressure acting on nervous tissue which is sufficient to cause symptoms. Forty-two patients with clinical symptoms and myelographic findings of central lumbar spinal stenosis were selected for the measurement of intrathecal pressure to determine the site, degree and dynamics of mechanical compression on the spinal nerve roots (Magnaes 1982). Pathological pressure on the cauda equina was found in sixty-seven per cent of these patients. The pressure in the region of the spinal block was high during standing and walking, and in several patients exceeded mean arterial blood pressure. In these patients the block pressure appeared to be the main mechanical factor affecting the central part of the spinal canal and causing pain and paresis. In thirty-three per cent of the patients normal pressures on the cauda equina were found and lateral compression of multiple nerve roots seemed to be the only causative factor.

These measurements were made by performing a lumbar puncture at the lumbo-sacral junction, and then performing an infusion test, with the rate of infusion at 1.5 ml per minute. The spinal block pressure was recorded with the patient standing erect and relaxed, and then with the lumbar spine in a semi-flexed and then a fully flexed position. The patient then laid down and the pressure was recorded with the lumbar spine in the same position as when he was standing erect. To determine the relative importance of spinal block pressure and caudal compartment pressure in causing pain, the caudal compartment pressure was artificially elevated by infusion of fluid in seven patients with a rather constant pain time and spinal block pressure of 1400 mm. water or above when standing erect. The pain time was determined at a caudal compartment pressure of 200 mm. (normal), 600 mm., and 1,000 mm. water. At the same time arterial blood pressure was recorded using a sphygmomanometer.

Dynamics of Cerebro-spinal Fluid Flow

There was free flow of cerebro-spinal fluid when the spine was fully
flexed. In the semi-flexed position there was a partial block and the infusion curve began to assume an exponential gradient. In the erect position there was a complete block with an exponential volume-pressure curve, and a point of deviation from this curve at about 1200 mm. water which indicated the pressure on the cauda equina. The highest pressure recorded in the erect position was 2700 mm. of water. The pressure on the cauda equina was invariably lowered or returned to normal when the lumbar spine was flexed. In six patients the block pressure in the erect position exceeded mean arterial blood pressure. One patient was unable to micturate whilst standing erect. Whilst standing the spinal block pressure on the cauda equina was found to be 1900 mm. water. When sitting however, he was perfectly able to void urine, and in this position the block pressure reduced to about 900 mm. of water.

The highest pressure recorded in a patient lying straight was 2060 mm. of water. The highest pressure in the walking phase was 2800 mm. of water. The spinal block pressure whilst standing erect was compared with the pressure in the walking position. Six more patients developed a spinal block and the pressure was usually higher when walking than when simply standing. On the whole the shorter the claudication distance the higher the block pressure, but in three patients there was quite a short claudication distance but no measurable block pressure, probably due to lateral recess stenosis rather than central stenosis.

These results seem to point to three conclusions. The first is that pressure on the cauda equina in the region of the block (up to 2800 mm. of water) was the main pathogenic mechanism in the central part of the spinal canal. The block pressure regularly increased with extension of the lumbar spine, and the difference in pressure between the horizontal and upright body position was small when the degree of extension was kept constant. Secondly, the rise in the fluid pressure caudal to the block (up to 750 mm. water), with larger fluctuations in this pressure on walking (up to 1420 mm. water) may represent an additional pathological mechanism. Thirdly, only sixty-seven per cent of patients had pathological pressures of the cauda equina. The other thirty-three per cent of patients most likely had compression from multiple level lateral stenosis, a condition which can be difficult to distinguish clinically from central canal stenosis.

In several patients the block pressure exceeded the mean arterial blood pressure, whilst walking and standing. This means that a segment of the nerve root was without its blood supply during walking and standing.
Even with this "tourniquet" effect around the cauda equina, patients were able to stand and walk for several minutes. This can be compared with the pressure on peripheral nerves for example during hand surgery. When a tourniquet is applied around the limb in excess of mean arterial pressure for up to twenty or thirty minutes there may be preservation of sensitivity and fine movement.

EPIDURAL VENOUS STASIS IN SPINAL STENOSIS

Computed tomography permits reliable demonstration of the spinal canal and its contents. Measurements of the sagittal diameter of the bony canal do not take into consideration size, shape and state of the intraspinal soft tissue structures such as the thecal sac and its contents, epidural fat and blood circulation patterns. Epidural venous engorgement may be visualised in spinal stenosis. Kaiser considers that epidural venous stasis occurring in segmental spinal stenosis is a computerised tomography sign of clinically significant narrowing of the neural canal (Kaiser 1984).

High intraspinal pressures in stenosis act on all the intraspinal contents. These consist of three compartments:

a) The dura and its own contents, with the cerebrospinal fluid pressure as internal pressure.
b) Extradural fat without internal pressure.
c) The vascular compartment, in particular the epidural venous plexuses with the same internal pressure as the remaining venous system.

These compartments react differently when submitted to high pressure. Fat is inert and is passively displaced towards regions where the pressure is normal. The dura may be deformed or displaced in cases of severe compression. When a vein undergoes compression its lumen collapses because of the laxity of its wall and the lower internal pressure. Blood flow deviation, venous dilation and stasis above and around the compression may be observed as a result.

THE PATHOGENESIS OF LUMBAR SPONDYLOSIS AND STENOSIS

The following concepts have developed through a study of cadaver spines, operative dissections, and radiological anatomy. Farfan and co-workers have drawn attention to three important facts (Farfan 1969).

1. At each level of the lumbar spine the two posterior joints and the disc form the "three-joint complex". Lesions affecting the
posterior joints also affect the disc and vice versa.

2. At the lower levels the posterior joints are aligned obliquely. This puts these joints at greater risk of recurrent rotational strain.

3. At the same two levels the discs are wedge-shaped, greater in height anteriorly than posteriorly. This also places these joints at risk.

Two types of injury are described as principally responsible for initiating degenerative changes.
1. Recurrent rotational strain.

Changes starting at one level usually the L4-5 level, later on in life place the level above and the level below at risk of strain, and in this way the process spreads. The initial lesion becomes more severe, and the degenerative changes become generalised (Kirkaldy-Willis 1978). A more detailed analysis of the spinal mechanics involved will be given in Chapter 9.

Posterior Joint Changes

The medial and anterior capsule formed by lateral extensions of the ligamentum flavum is composed of eighty per cent elastin and twenty per cent collagen. We do not know whether the elasticity of the medial capsule makes the joint stronger or weaker, and thus more or less liable to strain.

Changes which can be observed in the facet joints include the following:
1. A synovial reaction.
2. Fibrillation of articular cartilage.
4. The formation of osteophytes.
5. Fracture of an articular process.
6. Loose bodies in the joint.
7. Laxity of the joint capsule resulting in instability.

The Intervertebral Disc

Changes occurring in the intervertebral disc with age are of course impossible to distinguish between those changes which result from repeated minor trauma. The changes consist of the development of radial annular tears concentrated in the postero-lateral quadrant of the disc. Internal
disruption of the nucleus pulposus may occur. Discography shows a marked correlation between loss of disc height, the presence of traction spurs, and disruption of the disc. At this stage of advanced age and degeneration, the disc resembles a deflated football and herniation is most unlikely because the intradiscal pressure is so much reduced. Disc resorption occurs and the disc contents may be also completely resorbed. The narrow space between the vertebral bodies becomes occupied by a small amount of fibrous tissue. The vertebral body bone on either side of the disc becomes sclerotic.

**Intersegmental Motion**

Upward and forward displacement of the superior articular process results in marked narrowing of the lateral recess just medial to the intervertebral foramen. The type of nerve entrapment in the lateral recess is clearly seen in specimens described as early as 1932 by Williams (Williams 1932). This was two years before Mixter and Barr published their classic paper on disc herniation (Mixter 1934). The importance and frequency of this complication of loss of disc height is not always fully appreciated. (Fig. 4:14 and 5:7)

Instability superimposed on structural change can also cause recurrent spinal nerve entrapment. For example, in degenerative lesions affecting the L4-5 segment, changes in the posterior joints lead to loss of articular cartilage, osteophyte enlargement of the articular processes, stretching of the capsule, and an abnormal degree of movement of these joints. If the L4 vertebra is rotated on L5 on one or other side depending on the direction of rotation, the posterior joint opens up, the superior articular process of L5 moves forward, and the lateral recess is further narrowed. This is a dynamic process.

Experimental work supports the view that abnormal mobility and decreased mobility at one level predisposes to strains at levels above and below. The final stage in this process, seen at the end of the spectrum, is severe degenerative disease of the whole lumbar spine, often accompanied by some degree of scoliosis, with a rotatory element. In some instances both central and lateral stenosis occur with severe nerve entrapment at several levels.
Degenerative Spondylolisthesis

This is seen more often in women than in men. The main pathological finding is very marked erosion of the superior articular process of L5. This allows the inferior articular process of L4 to slip forwards as the disc yields. The anterior portion of the inferior articular process of L4 is almost in contact with the posterior aspect of the vertebral body of L5. The L5 nerves are entrapped at this site between the articular processes and the vertebral body.

Developmental Stenosis

Kirkaldy-Willis believed that this type of stenosis alone did not cause symptoms, but that when a small disc herniation occurred or a minor degree of degenerative change occurred, superimposed on developmental abnormalities, stenosis was more likely to cause nerve entrapment than when these developmental anomalies were not present (Kirkaldy-Willis 1978).

Major fracture of a vertebral body, isthmic spondylolisthesis, post-fusion stenosis, and rarely Paget's disease and fluorosis may also result in nerve entrapment syndromes. When these lesions are present there is usually some degree of concomitant degenerative change which precipitates or exacerbates entrapment of the spinal nerves.

INTRA-OSSEOUS HYPERTENSION

Intra-osseous hypertension has been found in patients with low back pain and may also, by causing venous congestion, result in some compression of the cauda equina in patients with spinal canal stenosis (Arnoldi 1976).

It is well recognised from intra-osseous venography that the bone marrow adjacent to osteo-arthritic joints has a disturbed venous outflow (Helal 1962). Intra-osseous stasis is accompanied by a rise in intra-medullary venous pressure. Intra-osseous hypertension may be relieved by osteotomy or cortical fenestration and this may be followed by prompt disappearance of pain arising from the arthritic joint.

Arnoldi in 1976 measured the pressure within the spinous processes of three vertebrae (Arnoldi 1976). The radiologically normal vertebra had intra-osseous pressures varying within narrow limits (2-13 mm. of mercury) with a mean value of 8.3 mm. of mercury. But in the levels affected radiologically by spondylotic change, the pressures were significantly higher (28.1 mm. of mercury mean, with a range of 14-49 mm. of mercury).
High pressures in the group with severe chronic lumbago were found, and a relationship was postulated between the degree of spondylotic change and the height of the intra-osseous pressure. It was found that these vascular changes were segmental. That is, intra-osseous hypertension might be present in one or several vertebra, whilst other vertebra even adjacent vertebra may show normal intra-osseous pressures. Intra-osseous hypertension was postulated by Arnoldi to be a reason for failure of "standard" fusions in osteo-arthritic spines, since the intra-osseous hypertension was not relieved in these procedures.

More recently dynamic measurements of intra-osseous pressure in lumbar vertebra were performed to determine the effects of positional change on lumbar vertebral circulation (Hanai 1980). Twenty-three patients were used, twelve of whom had spinal canal stenosis and eleven had spondylosis deformans. The pressures within the spinous processes of L3 and L5 were measured continuously whilst patients were prone, lying in the lateral position, and standing. The pressures ranged from approximately 10 mm. to 30 mm. of mercury in the prone position, and showed substantial changes with changes in position. In each subject they were lowest in the prone, intermediate in the lateral, and highest in the standing position. It was postulated by Hanai that increases in epidural venous pressure might be responsible for the increased intra-osseous pressures, and act as a possible cause for the symptoms of spinal stenosis.

It was found that the pressure in L5 was always higher than the pressure in L3 whatever the position of the patient. No correlation was observed between the ages of the subjects and the pressures. The mean pressures in the group of patients who had spinal canal stenosis tended to be lower than in the group of patients who had spondylosis deformans. On the other hand, patients with spinal stenosis were found to have a higher pressure increase compared with those with spondylosis alone. This implies that patients with spinal stenosis have a higher resistance to venous outflow from the lumbar vertebrae possibly resulting from venous obstruction in the extradural venous plexus.

The measured elevation of intra-osseous pressure could well occur in association with congestion of the extradural venous plexus. It may be congestion of this plexus which in turn causes compression of the cauda equina in patients with spinal stenosis.
CHAPTER 7
THE IMMUNOLOGY OF DISC DEGENERATION
AND NERVE ROOT IRRITATION

INTRODUCTION

The formation of nerve root adhesions frequently encountered during surgical explorations of the spinal canal may be the result of a chronic inflammatory reaction. This reaction may clearly be initiated by mechanical irritation of the nerve root, or in response to chronic ischaemia of the root, or as a result of chemical radiculitis, and it has also been proposed that an auto-immune process may play some part in perpetuating chronic inflammation around the nerve root.

The nucleus pulposus is normally tightly contained within the annulus, and after its embryological formation it no longer normally makes any vascular contact with the systemic circulation (Hirsch 1952). This situation is analogous to the enclosed thyroglobin in Hashimoto's disease, and vitreous humour of the eye in the production of sympathetic ophthalmia.

It may be that fragments of nucleus pulposus which escape from the annulus may therefore not be recognised as "self" by the body's own immune recognition system and may stimulate an auto-immune response. The same may be true for breakdown products of a degenerate disc.

Experimental and clinical evidence to support this concept is presented in this chapter.

THE PRODUCTION OF AUTO-ANTIBODIES TO NUCLEUS PULPOSUS IN THE RABBIT

Auto-antibodies to autogenous nucleus pulposus have been experimentally produced in the rabbit (Bobechko 1965). These antibodies are cell bound within lymphoid cells and are greatest in primary lymph nodes. The auto-immune antibody response within the primary lymph node reaches a rapid maximum at four days, but then unlike most homograft reactions which are short-lived, remains at a high level for three weeks. In addition, a secondary rise in cell-bound antibody appears in all lymph nodes in six weeks as shown by strong pyroninophilia and fluorescence. This may be part of a generalised systemic antibody response, but no check was made for a humeral antibody titre in this report.

An interesting observation by Bobechko and Carl Hirsch in two human
subjects who underwent anterior lumbar interbody fusion for failed posterior disc surgery, was that a marked hyperplasia and enlargement of the regional para-aortic lymph nodes about the lumbar spine was noted at the time of surgery.

AUTO-IMMUNITY IN DEGENERATIVE DISC DISEASE OF THE LUMBAR SPINE

The role of inflammation in the pathogenesis of symptoms and signs of lumbar disc degenerative disease has been implicated by certain clinical observations.

1. The dramatic relief in some patients with the local and systemic use of anti-inflammatory drugs, such as steroids.
2. The predominance of pain, which is a feature of an inflammatory reaction, as opposed to other pure entrapment syndromes in which the predominant symptom is numbness.
3. The finding at surgery of inflammation and fibrosis around the nerve roots.
4. The microscopic picture of degeneration, vascular ingrowth, granulation and fibrosis in discs removed at the time of surgery.

Gertzbein and others noted that the following tissues are isolated from the body's immune recognition systems, which are occurring in early development (Gertzbein 1975). These are the cornea, the uvea, cartilage, thyroglobulin, sperm and myelin and the nucleus pulposus of the intervertebral disc. The nucleus pulposus contains proteoglycans which are known to contain certain antigenic determinants.

Using the lymphocyte migration inhibition test as a measure of the development of cellular immunity in patients with prolapsed intervertebral disc, Gertzbein carried out thirty-three such tests in twenty-four patients, ten males and fourteen females with an average age of fifty-five and forty-four years respectively. They found that the majority of patients (seventy-two point seven per cent) showed significant inhibition with the leucocyte migration inhibition test suggesting a cellular immune response to the nucleus pulposus. On the other hand, only twenty-six point three per cent of patients whose discs were herniated showed no inhibition. In nine patients the test was repeated at four to six week intervals, and these tests almost invariably reproduced the same results. That is, those tests which were positive remained positive and those tests which were negative remained so. Gertzbein came to four conclusions as a result of this study.
1. Degenerative disc disease of the lumbar spine is mediated in some patients by an inflammatory component.

2. The chronicity of the inflammation may have an auto-immune basis.

3. The leucocyte migration inhibition test demonstrated the presence of a cellular immune response in patients whose discs were found to be sequestrated at the time of surgery.

4. No human humeral antibody could be demonstrated (Gertzbein 1975).

   Arthur Naylor in 1976 reported the release of appreciable quantities of chemical substances from discs which were treated by freezing or thawing, or by the addition of Triton X100 (Naylor 1976). This he stated was due to the rupture of the lysosomal membrane. Lysosomal enzymes such as acid phosphatase, β-glucoronidase, galactosidase, β-glucosamonidase, and acid proteases were all released. This lysis could be depressed by cortisone treatment which stabilises cell membranes. Five species of acid glycerophosphatase were isolated from the disc tissue separated by chromatography.

   He correlated these results with clinical finding that the symptoms of lumbar disc herniation could frequently be relieved by the intradiscal or epidural injection of hydrocortisone. He stated that it was possible that the cell membrane stabilisation effect of hydrocortisone prevents further lysosomal enzyme release, and prevents further protein polysaccharide breakdown.

   Naylor also examined the concentrations of IgM, IgG, and IgA in subjects prior to surgery for prolapsed intervertebral disc with those of normal healthy adult populations. He found a significant increase of IgM and IgG in patients with lumbar disc prolapse, but he was unable to find immunoglobulins in the disc tissue itself (Naylor 1976).

The Lymphocyte Transformation Test

   As stated Gertzbein has shown, using the leucocyte migration inhibition test, that sequestrated disc inhibits migration whereas non-sequestrated disc showed no significant inhibition (Gertzbein 1975). He then went on to confirm this work using the lymphocyte transformation test, and another parameter of cellular immunity (Gertzbein 1977).

   Gertzbein tested twenty-five patients and used forty-three controls. The average transformation index of the controls was 3.6 (S.D. ± 2.4). There were sixteen patients with positive myelograms, ten of whom underwent surgery, and were found to have sequestrated or very large herniated
discs. These ten were considered to have had discs in contact with the body's immune mechanism for a significant period of time. These patients had a significantly greater mean transformation index of 16.6 (S.D. ± 16.3), as compared with the control group using the logarithmic transformation of the student's test.

Six patients had moderate to large defects on their myelograms, but did not undergo surgery. These patients showed a transformation index of 6.0 (S.D. ± 5.1). This group was found to be statistically different from the overall control group (p < 0.0025).

Nine patients had small myelographic defects or negative myelograms. None of these patients underwent surgery. This group was not found to be statistically different from the control group, with a mean transformation index of 4.8 (S.D. ± 3.8).

Gertzbein considered the antigenic stimulation was originating from the proteoglycan molecule. The test disc material was taken from cadavers, but antigenic stimulus from cells was considered unlikely because the purification process destroyed cells, and also there were very few of these cells within the tissue. Also centrifugation eliminated the likelihood of H-LA antigens on cell fragments from contaminating the antigen in the supernatant. He therefore considered that the lymphocyte transformation test was useful in augmenting the diagnostic criteria for establishing a diagnosis of disc herniation. It was also on this evidence that the use of cortico-steroids in the acute treatment of disc prolapse patients gained a more rational basis.

Again the evidence was that an auto-immune response was mounted to the nucleus pulposus in patients with sequestrated discs.

THE ROLE OF CHEMICALS IN THE PRODUCTION OF NERVE ROOT IRRITATION

Pressure on a normal nerve root causes not pain but paraesthesia. It is only the inflamed root which responds to pressure with a symptom of pain. Armstrong described the oedematous reddened nerve root containing petechial haemorrhages in the early stages of compression, and later fibrosis of the nerve root occurring in association with degenerative change of the spine (Armstrong 1967).

It was postulated by Marshall that fluid from the nucleus pulposus could be ejected sometimes under pressure into the peridiscal tissues and
could track down the nerve root (Marshall 1977). The proteoglycan content of nuclear fluid made it highly irritant to nerve tissue and this sudden ejection of fluid could cause sudden severe sciatic pain. The glycoprotein of the nuclear fluid following its escape from the annulus fibrosus, took on an antigenic role and stimulated the production of antibodies which were detectable in high titre in the blood stream after a three week interval.

Leon Marshall set out to answer the following questions:

1. Can nuclear fluid reach the nerve root? Lindblom and Rexed dissected one hundred and sixty cadavers and demonstrated a connecting pathway between the nucleus and the nerve root via annular rupture. This is now well established (Lindblom 1948).

2. Is nuclear fluid in a liquid form at some stage of the degenerative process? It has been noted by many observers at operation and verified by Armstrong and Walk, that nuclear fluid can be exuded as a liquid under pressure at the time of operation (Walk 1956). Armstrong states that he has incised the annulus on occasions and the fluid has squirted out of the wound (Armstrong 1967).

3. Is nuclear fluid irritant when in contact with nerve tissue? Marshall searched the literature and no answer to this question was found.

4. If nuclear fluid is liberated into the tissues from its enclosing annulus fibrosus theoretically it should produce an antibody response which can be detected in the blood.

To test this theory an abstract was made of nuclear material which was then injected into the pulmonary artery of a lung preparation. A carbohydrate glycoprotein was isolated from nuclear fluid. A concentration of glycoprotein was then injected into a heart-lung preparation. The result was a severe reaction consisting of broncho-constriction followed by oedema of the lung.

Marshall then extracted the fluid from the heart-lung preparation and again tested it on the jejunum of guinea pig. The irritant perfusate produced contractions which were recorded in the usual manner. A small measured amount of perfusate from glycoprotein showed a strong reaction equal to a small amount of histamine.

As a result of these studies it was postulated that there may be a group of patients usually young patients, in whom chemical radiculitis may occur in the absence of disc prolapse. In these individuals where there is more nerve root irritation than compression, the symptoms may be relieved by the administration of cortisone or its derivatives. This would allow
early mobilisation of the patient and help to disperse and aid absorption of toxic substances in contact with the nerve root and prevent the formation of nerve root adhesions.

Marshall also confirmed that the liberation of disc fluid into the tissues evokes a circulating antibody response and auto-immune reaction. A high titre to glycoprotein was noted at three weeks after an acute attack of back pain, and this was taken as evidence of a significant disc lesion. In selected patients immediate relief of pain occurred after the administration of cortisone or a suitable cortisone derivative. This was more likely when the nerve root pain arose from chemical radiculitis and not nerve root compression. It is possible that a thin watery fluid may escape from the disc to produce such chemical radiculitis in the absence of sequestration of the nucleus pulposus.

Prolonged rest may be contra-indicated because of the risk of formation of nerve root adhesions.
CHAPTER 8
EXPERIMENTAL SPINAL STENOSIS

INTRODUCTION

Sub-laminar segmental spinal instrumentation as developed by Luque is now used more frequently in the treatment of complex neuromuscular spinal deformities, and for many has become the treatment of choice. Segmental sub-laminar wiring provides more secure fixation of the spine to a pre-contoured rod which can be fashioned to restore the normal thoracic kyphosis and lumbar lordosis. Correction of the scoliosis segment by segment (with the exclusion of the rotational deformity) provides not only stronger support for the spine but better overall correction. Fixation is frequently so secure at the time of surgery that no external orthosis is required post-operatively (Allen 1985).

If surgeons are experienced with the sub-laminar wiring technique, idiopathic scoliosis can be safely treated with the Luque method (Shufflebarger 1985, Thompson 1985), particularly now that spinal cord monitoring is more reliable (Wilber 1984, Hardy 1973). Sub-laminar wiring to, for instance, the Hartshill rectangle is now used to provide a stronger more rigid method of stabilising the lumbar spine whilst bony fusion occurs (Dove 1986).

The sub-laminar passage or manipulation of wires may be complicated most commonly by the development of sensory dysaesthesia (Allen 1985, Thompson 1985). This may be the result of an expanding epidural haematoma or of oedema from a mild dural contusion.

Intra-operative spinal cord monitoring using somatosensory cortical evoked potentials, may not detect the changes which result in such sensory dysaesthesia (Wilber 1984). It is however, very reliable in detecting major cord damage which is sufficient to produce motor loss.

Delayed paraplegia after segmental sub-laminar wiring has been reported in two patients with congenital scoliosis (Johnston 1986). It was postulated that the sub-laminar wire caused neural irritation in a narrow kyphotic segment of the spinal canal through dural impingement, with the onset of paraplegia thirty hours and six days post-operatively. One patient improved partially following removal of the instrumentation, but the other permanently lost walking ability.
THE LONG-TERM EFFECTS OF SUB-LAMINAR WIRING

The long-term effects of sub-laminar wires on the contents of the spinal canal have only recently been considered (Schrader 1986, Gillespie 1986). From work in dogs it does appear that the passage of sub-laminar wire may result in damage to both dura and underlying neurological tissues. In the long-term, histological evidence of chronic progressive long tract degeneration has been noted (Hafer 1986).

There is however no work to date to indicate the long-term effects of sub-laminar wiring on growth and development of the immature spine and in particular the spinal canal (Luque 1986). Does wire between the lamina and the dura provoke, in addition to chronic dural irritation, bony overgrowth of the lamina? Circumferential wiring of a long bone for fracture fixation has been shown to devitalise the underlying bone and possibly retard new bone formation, but the blood supply of the lamina is different from that of a long bone. Does the lamina fail to develop normally or does the wire stimulate overgrowth? Most important of all, if sub-laminar wiring is performed in the skeletally immature juvenile or adolescent, does the presence of wire within the spinal canal stimulate the formation of new bone on the under-surface of the lamina? If new bone formation does occur within the spinal canal, is it so minimal that it can be safely ignored, or is it possible that in the long-term it could produce sufficient narrowing to result in the development of symptoms and signs of spinal stenosis?

Bony thickening which occurs with advancing age, particularly new bone formation around the facet joints, may lead to degenerative spinal stenosis in certain individuals, whilst others whose vertebrae are thickened by Paget's disease may also develop the syndrome. Could the presence of sub-laminar wiring accelerate the development of the bony hypertrophy of advanced age, not in every patient but in those who are susceptible by virtue of having already relatively small, under-developed spinal canals?

No experimental work so far, including the studies quoted above, have addressed this question. Since sub-laminar wiring is frequently performed on skeletally immature individuals, a model was developed of segmental sub-laminar wiring in eight week old rabbits to assess its effects on growth and subsequent development of the spine. The rabbits were allowed to reach skeletal maturity and were sacrificed at two years of age before analysis of laminar thickness and spinal canal deformity was
performed. The aim of this experiment was to determine whether or not the presence of sub-laminar wiring predisposes to sub-laminar bony overgrowth, with subsequent development of spinal stenosis (Fig. 8:1).

OPERATIVE METHOD

Sub-laminar wiring was performed on thirty-four eight week old New Zealand white rabbits at three levels on one side only of the lower thoracic spine. A midline incision was made under general anaesthetic using fentanyl citrate and diazepam and only the left side of the spine was exposed by paravertebral muscle stripping. This stripping was continued one segment above and one segment below to assess the results of stripping and retraction alone (Fig. 8:2). At the uppermost level the sub-laminar wire was secured tightly to itself around the lamina. At the middle level the wire was intentionally secured only loosely, and the cut end of the wire was left about 0.5 cm. long, protruding into the erector spinales muscle such that any muscle movement would result in wire movement also. The most caudal wire was secured around the lamina tightly, in fact so tightly that a laminar fracture occurred (Fig. 8:3). Where a laminar fracture could not be produced by reasonable wire tightening, then the lamina was scored adjacent to the wire using a scalpel to cause damage to the outer cortex simulating trauma to the lamina by wire or by instruments. The wounds were closed without drainage, and an antero-posterior and lateral radiograph of the spine were taken to confirm the position of the wires and for documentation purposes.

The rabbits were allowed to recover from the general anaesthetic which most did satisfactorily. Four rabbits showed some evidence of neurological deficit following this procedure, such as dragging a leg or failing to hop and developed diarrhoea. These were put down within one week of surgery.

The rabbits were allowed to attain full size and skeletal maturity and at the age of twenty months were sacrificed using pentobarbitone sodium. The entire thoraco-lumbar spine was harvested and photographs of the fresh specimens taken together with antero-posterior and lateral radiographs of the fresh spines (Fig. 8:4). The entire spine was then fixed in formal saline for four weeks. The segments containing sub-laminar wires were then dissected from the remainder of the spine and underwent dessication using alcohol.
Figure 8:1. A flow chart to illustrate briefly the method used to determine the effect of sub-laminar wiring on growth and development of the immature spinal canal.
At operation under a general anaesthetic a blunt aneurysm needle was carefully passed beneath the lamina, fed with 32 gauge wire, and was then withdrawn taking the wire beneath the lamina. This was performed at three adjacent levels on one side of the spine. Ipsilateral levels above and below were stripped of soft tissue to act as exposed but non-instrumented controls. Contralateral levels were not exposed and so acted as non-instrumented non-exposed controls.

Prior to wound closure the three instrumented levels can be seen (arrows indicate the wires). The cephalic wire is tight, the middle wire is loose, and the caudal wire is tight around a fractured lamina.
Figure 3. Lateral radiographs of the fresh isolated spine following sacrifice of the rabbit prior to fixing. Figure 4 (a) illustrates no deformity. In Figure 4 (b) a kyphosis has been produced, and Figure 4 (c) illustrates one case in which a lordosis was produced by posterior fusion of the bone mass around the wires.
METHOD

Embedding Technique

L-R white resin was selected because of its low viscosity and ability to permeate fully a relatively large specimen. The portion of the rabbit spine containing the wires, with two normal vertebrae at each end was placed in the low viscosity L-R white resin and kept at room temperature under a vacuum of minus 400-500 mm.Hg. for three months. Our experimental work had demonstrated that L-R white resin "cured" more effectively using heat rather than using the supplied catalyst which would not mix evenly with the resin and produced only patchy polymerisation. After three months the impregnated specimen was therefore placed in an oven at 30°C for seven days. It was then possible to section the specimen into 2 mm. sections or discs using a band saw.

Sectioning Technique

During preliminary trials using L-R white resin and non-essential rabbit spine, it became apparent that on polymerisation the resin frequently lost its translucent properties making it impossible to see the specimen and select appropriate planes for sectioning. To overcome this problem, each wired rabbit spine was secured to a short Kirschner wire using three sutures at the junction of the annulus fibrosus of the intervertebral discs at the top, bottom and middle of the specimen and the anterior longitudinal ligament of the spine which was easily discernable in each specimen. The Kirschner wire thereby lies in the longitudinal axis of the vertebral column and the spinal canal. Only in one specimen (N2) was the spine so deformed (by the formation of a posterior inter-laminar fusion on one side) that it was not possible to secure the Kirschner wire throughout the spine in contact with the discs and vertebral bodies. Following polymerisation of the L-R white resin, the attached Kirschner wire could be seen at the top and bottom of the embedded specimen indicating the longitudinal axis of the spinal canal of the specimen contained within the opaque resin.

Using the Kirschner wire as a guide it was possible to secure the specimen to be sectioned in a rig with the longitudinal axis of the spinal canal perpendicular to the cutting blade of a band saw. Sections or discs of the specimen 2 mm. thick were then cut slowly and because of the thickness of the band saw blade, this produced two different sections for analysis with each cut, corresponding to each side of the band saw blade.
A segment of the stout Kirschner wire was present in each 2 mm. disc of the specimen and examination of this under 80x magnification revealed that there had been no movement of the wire during sectioning. Despite the fact that the stout Kirschner wire was obviously the hardest part of the specimen/resin/wire complex for the band saw blade to cut there was no evidence of cracking of the resin or crescentic defects around the wire to indicate loosening, and indeed the wire could not be pushed out of the specimen using another Kirschner wire.

Similarly it was apparent on microscopy that the sub-laminar wires and the fragile laminae and vertebrae had been sufficiently supported and secured by the L-R white resin that sectioning had not disturbed the relationship between the wire and the bone. Had the embedding technique been inadequate then the bone would have fractured during sectioning, and the wires either migrated or pulled out.

Some specimen discs were polished after sectioning but on subsequent microscopic examination it was found that this did not reveal any more detail of the spinal canal, lamina, or wire/bone relationship than was apparent without polishing. The sections were therefore not routinely polished.

**Staining Techniques**

After careful brushing of the specimen using a soft bristled lens brush to remove remnants of resin and bone saw-dust, a freshly prepared solution of 5% w/v aqueous silver nitrate was dropped on to the specimen, which was then placed in bright sublight (the Oxford weather was unusually favourable for this part of the experiment). The osseous tissue of the vertebrae were therefore stained dark brown and surrounding muscle and soft tissues, including ligament and intervertebral disc took up less of the silver nitrate stain and were therefore lighter brown in colour. The demarcation between bone, wire, soft tissue and resin thus became clear and distinct. It was immediately apparent macroscopically that abundant new bone formation had occurred around and in relation to the sub-laminar wires. (Fig. 8:5)

Each specimen disc was washed using distilled water and the stain fixed in the specimen using 5% w/v sodium thiosulphate (‘HYPO’) solution. Approximately fifteen discs were produced from each one of the fifteen spines. Each disc had two surfaces which were both stained and fixed, providing thirty sections for analysis per specimen (Fig. 8:5).
Figure 8:5. Three typical sections of rabbit spine after fixation and staining. Figures 5 (a) and 5 (b) illustrate the extent of new bone formation mostly outside the spinal canal in response to sub-laminar wiring (wires indicated by arrows). Figure 5 (c) illustrates a non-instrumented control.
COMPUTER-ASSISTED MORPHOMETRIC ANALYSIS OF THE SPINAL CANAL AND LAMINAR RESPONSE TO SUB-LAMINAR WIRING

The purpose of this study was to determine the effect of sub-laminar wiring of the juvenile rabbit spine on the subsequent growth and development of the spinal canal. It was assumed that this effect would be observed only on the half of the lamina encircled by wire. This was measured in a number of different ways: firstly on laminar thickness, secondly on spinal canal radius, and thirdly on cross-sectional area of the two halves of the spinal canal. An Ibas Kontron Analytical Image Analysis Computer was used to obtain these measurements (Fig. 8:6).

(a) Laminar Hypertrophy

The first part of the analysis consisted of measurement of the thickness of the lamina at fourteen separate sites (Fig. 8:7). As already described, only the left half of the lamina and spinous process was exposed at each vertebral level, so it was possible to use the unexposed half as a control, and seven measurements of its thickness were made. Other controls consisted of the vertebrae above and below the instrumented segments: these were surgically exposed on one side and unexposed on the other side. Two different series of controls were therefore employed, one contralateral and the other ipsilateral and contralateral at a different non-instrumented vertebral level.

The three vertebral segments instrumented using sub-laminar wiring were each measured in a similar fashion. That is, fourteen measurements of laminar thickness were made at each level. Since however more than one section was made of each vertebral segment, in practice more measurements were made. Up to six separate sections of one vertebral level were available for analysis, providing forty-two measurements of one half of one lamina, which could be compared with forty-two contralateral control measurements and up to one hundred and sixty-eight control measurements from the non-instrumented levels above or below the sections. Again, in practice, the measurements from the instrumented side of the lamina were compared with the measurements from the non-instrumented contralateral control of the same level and a ratio of instrumented to non-instrumented obtained.

Absolute measurements of laminar thickness in micrometres were made after calibration of the computer, but it was considered that
Figure 8.6. The Ibas Kontron Analytical Image Analysis Computer used for morphometric analysis of sections of the rabbit spines.
Figure 8:7. An example of a print-out from the Ibas Kontron computer of one section (8:9) to illustrate the fourteen measurements of laminar thickness and the fourteen measurements of spinal canal radius made on the instrumented (left) and non-instrumented sides. (Reduced in size by fifty per cent.)
absolute values had less significance than the ratio of the two halves of the lamina (RA 23). Between twenty-eight and forty-two such ratios were available for each vertebral segment and for control segments above and below the instrumented levels. In total, one thousand five hundred and eighty-two measurements of laminar thickness were made of both experimental specimens and controls (Fig. 8:8).

The question arises of how can laminar thickness be most accurately measured. A number of different methods were attempted, and one method chosen because it was reproducible and measured the lamina as close to perpendicular to its deep surface as possible. This method consisted of finding the central point of the spinal canal and constructing a line from this point to the central point of the vertebral body. From this line fourteen further lines were constructed from the centre of the spinal canal at 10° angles from the midline up to and including 70° to the right and to the left of the midline (Fig. 8:7).

The normal spinal canal is ellipsoid in shape and therefore does not have one centre. The centre of gravity of a normal elliptical spinal canal would correspond to the central point of the spinal canal, but when the spinal canal is deformed, as it is usually following sub-laminar wiring, then the centre of gravity was found to be an unreliable indicator of the anatomical centre of the spinal canal. The anatomical centre of the spinal canal is determined more by reference to the vertebral body which is least affected by sub-laminar wiring than to the centre of the spinal canal which is modified by sub-laminar instrumentation.

To obtain the anatomical centre of the spinal canal therefore, a line joining the inner aspects of the two pedicles and a line joining one side of the vertebral body posteriorly to the other are bisected. This indicates the midline of the anterior part of the vertebral column, and from there a line can be continued posteriorly to the neural arch. This midline is easily determined at levels not instrumented, and with practice it can also be reliably and reproducibly obtained at instrumented levels. A line was then constructed joining the centre of the anterior vertebral column to the centre of the neural arch, and this formed the axis from which other measurements were made. To find the point on this axis from which the fourteen angles could be constructed it was simply necessary to bisect the distance between the posterior margin of the vertebral body and the deep surface of the neural arch along this line (Fig. 8:9). See below for details of reproducibility studies on this point and axis.
Figure 8.8. A photograph taken from the computer monitor to illustrate the type of image created from the specimen which can be analysed firstly by measurement of laminar thickness on the instrumented and then the control side, and secondly by measurement of spinal canal radius on instrumented and control sides. The extent of firstly laminar thickening and secondly intrusion of thickened lamina into the spinal canal can be calculated from these measurements.
Figure 8.8. A computer print-out of one specimen (12;5) upon which an acetate onlay is superimposed to indicate the method used to determine the central point of the spinal canal and the neurocentral axis from which measurements can be made. (Half the normal size of the print-out.)
(b) Laminar Intrusion into the Spinal Canal

The second part of the computer-assisted morphometric analysis consisted of measuring the radius of the spinal canal at fourteen separate sites and comparing the instrumented side with the non-instrumented side. Measurements of seven radii were made on each side at 10°, 20°, 30°, 40°, 50°, 60° and 70° angles from the midline and the instrumented 20° radius was compared with the non-instrumented 20° radius, the instrumented 30° radius with the non-instrumented 30° radius and so on. The object of these comparisons was to determine if sub-laminar wiring provoked new bone formation on the deep anterior surface of the lamina, thereby reducing the cross-sectional area and consequently the volume of the spinal canal.

Since the normal spinal canal of the rabbit is ellipsoid in cross-section the radius increased on each side from 10° to the midline to 70° to the midline. The rate of increase of radius from medial to lateral was, however, considered to be less important than the ratio of comparable radii from the experimental and contralateral control sides (RA 45) (Fig. 8:7 and 8:9).

(c) Cross-sectional Area of Each Half of Spinal Canal

The sagittal midline of the spinal canal determined by reference to the vertebral body and pedicles as described above, was used to divide the spinal canal into two halves. One half was contained by midline, half the posterior wall of the vertebral body, one pedicle, and one half of the lamina which was either instrumented or the non-instrumented control (Fig. 8:10 and 8:11).

Measurement of each half of the spinal canal and comparing one half with the other at each level (RA 6) provided a second method of looking for intrusion of the lamina into the spinal canal in response to sub-laminar wiring, in addition to method (b) above. Method (b) was considered more accurate than method (c) for a number of reasons, viz:

(i) Measurement of a radius of the spinal canal represents direct measurement of any intrusion by the wall, and would be detected even if the intrusion occurred over a relatively small extent of the wall of the canal.

(ii) Measurement of half the area of the spinal canal may at first sight appear to represent the more accurate method of calculating intrusion by the lamina into the spinal canal, since it is measuring the cumulative
Figure 8:10. The computer image of the rabbit spine specimen subjected to analysis of the two halves of the spinal canal, one experimental and the other control, to look for evidence of reduction in cross-sectional area of the canal on the experimental side.
Figure 6:11. Computer print-out of one specimen (34;11) to illustrate the measurement of cross-sectional area of each half of the spinal canal (6-1 is experimental and 6-2 is control) in square microns.
effect of either a reduction or increase in seven, or an infinite number of radii. In practice, however, the cross-sectional area of half the canal was determined more by the overall elliptical shape of the canal than by a specific reduction of the radii in one quarter segment of the canal, that is the lamina. Asymmetric vertebral bodies and pedicles and spinal canal shape were found to influence this measurement making it less reliable than the measurement of canal radii [measurement (b)].

(iii) Small deviations of the midline to either the right or the left had a marked influence on cross-sectional area of each half of the spinal canal, since the midline shift was occurring at the widest antero-posterior dimension of the canal.

(iv) Minor deviations of the mid-point of the spinal canal anteriorly or posteriorly within the limits of error of the measurement made no difference whatsoever to either the laminar thickness (a) or canal intrusion (b) measurements when they were expressed as a ratio. Clearly such a shift would alter the absolute measurement in microns, but the error on the experimental side would be identical to the error on the control side and would therefore be cancelled out in the ratio.

(v) Minor deviations of the mid-point of the spinal canal to the right or the left would produce significant changes in the measurement of cross-sectional area of half the canal, but its effect on measurement of thickness of the lamina would be minimal, since it would alter by only a few degrees the section of the lamina being measured. The effect of such a shift would be marginally greater on measurement of the radius of the canal at fourteen separate sites, tending to increase the radius most in the 60° and 70° measurements and have negligible effect on the 10°, 20° and 30° measurements.

In practice, reproducibility studies were carried out on one hundred sections to determine observer error and inter-observer error in firstly determining the central point of the spinal canal and secondly determining the neurocentral axis.

REPRODUCIBILITY STUDIES

One hundred specimens were analysed independently by two different observers (A and B) providing four hundred results. Each observer was required to identify the central point of the spinal canal and the line representing the neurocentral axis. These results were then subjected to further analysis to determine firstly the repeatability of the measurements
or (i) observer error, and secondly the difference in results between the two observers to indicate (ii) inter-observer error.

(i) Assessment of observer error
(a) Centre point analysis

<table>
<thead>
<tr>
<th>TABLE 1. Percentage error measurements</th>
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</thead>
<tbody>
<tr>
<td>OBSERVER</td>
</tr>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
</tbody>
</table>

Deviation of the central point of the spinal canal to either right or left would adversely affect the accuracy of RA 45 and RA 6 measurements. It was considered that a ± 1% difference would be an acceptable difference between measurements. A 1% error was equivalent to a deviation of the central point by one-hundredth part of the transverse diameter of the spinal canal to either right or left. A 2% error would represent a two-hundredths shift. Any measurement outside this was considered an error. From Table 1, which details percentage error of these measurements, Table 2 can be constructed.

<table>
<thead>
<tr>
<th>TABLE 2. Percentage error measurements with ± 1%</th>
</tr>
</thead>
<tbody>
<tr>
<td>OBSERVER</td>
</tr>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
</tbody>
</table>

To test whether or not the two observers have significantly different numbers of error-free measurements, the two proportions of 0.96 and 0.89 are considered as binomial random variables. Thus the two proportions are tested for equality using a Z statistic and looking up the significance in tables of the normal distribution.

Results

\[ Z = 1.8792 \quad (p<0.05) \]

Conclusions

The two proportions are not significantly different from each other at the 5% level. This then means that observers A and B do not differ significantly from each other in their accuracy of measurements to within a 1% of error. It should be noted that the two observers have very accurate results.
(b) Neurocentral axis analysis

TABLE 3. Percentage error measurements (degrees)

<table>
<thead>
<tr>
<th>OBSERVER</th>
<th>MEASUREMENT ERROR</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>0°</td>
</tr>
<tr>
<td>A</td>
<td>79%</td>
</tr>
<tr>
<td>B</td>
<td>67%</td>
</tr>
</tbody>
</table>

When determining the neurocentral axis which influences all measurements including RA 23 and RA 6, a 1° error was considered acceptable. From Table 3, which indicates percentage error of measurement in degrees, Table 4 can be constructed.

TABLE 4. Percentage error measurements (degrees) with ±1° considered acceptable

<table>
<thead>
<tr>
<th>OBSERVER</th>
<th>MEASUREMENT ERROR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;1°</td>
</tr>
<tr>
<td>A</td>
<td>0.91</td>
</tr>
<tr>
<td>B</td>
<td>0.90</td>
</tr>
</tbody>
</table>

As with the previous measurements the above are considered as binomial random variables, the test statistic again having a normal distribution.

Results

\[ Z = 0.2412 \ (p<0.05) \]

Conclusions

There is therefore no significant difference between the two observers when considering their accuracy of axis measurements. Both observers have a high level of accuracy, both significantly different from 0.5 at the 1% level.

The results indicate that the measurements of both observers are reproducible when considering measurements less than or equal to 1% error and 1° as acceptable. The two observers do not differ significantly in the accuracy of their results. It thus appears that there is a high degree of confidence in the ability of each observer to be highly accurate, indicating that the results are repeatable for each observer.

(ii) Assessment of inter-observer error

(a) Centre point

The difference between the observer's estimates of the centre point was calculated for seventy-six specimens viewed independently by each observer. The difference between observers is shown in Table 5.
TABLE 5. Results of inter-observer error measurements (% of total in brackets)

<table>
<thead>
<tr>
<th></th>
<th>&lt;1°</th>
<th>&gt;1°</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1°</td>
<td>45 (59.2)</td>
<td>13 (17.1)</td>
<td>58</td>
</tr>
<tr>
<td>&gt;1°</td>
<td>10 (13.2)</td>
<td>8 (10.5)</td>
<td>18</td>
</tr>
<tr>
<td>TOTAL</td>
<td>55</td>
<td>21</td>
<td>76</td>
</tr>
</tbody>
</table>

The aim of the analysis of these figures was to test whether or not the majority of the differences between the observers were within 1% error of the point and 1° error of the axis. This was tested by comparing the proportion of observations within 1° and 1% error.

Results
1. Is the proportion of results within 1° and 1% error significantly greater than that for measurements within 1° but greater than 1% error?
   \[ Z = 5.3431 \text{ (p <0.05)} \]
   The result of the Z test shows that it is significantly greater at the 5% level.
2. Is the proportion of results with 1° and 1% error significantly greater than the proportion within 1% error but not within 1°?
   \[ Z = 5.9078 \text{ (p <0.05)} \]
   The result of the Z test shows that it is significantly greater at the 5% level.
3. Is the proportion of results within 1% error and 1° significantly greater than the proportion of results greater than 1% error and 1°?
   \[ Z = 6.2975 \text{ (p <0.05)} \]
   The result of the Z test shows that it is significantly greater at the 5% level.

Conclusions
The results show that the majority of the results are within 1° and 1% error of the zero axis and centre point. Whilst only 8 (10.5%) of the measurements are greater than 1° and 1% of error.

From these results there is clearly a high degree of confidence in stating that the majority of the measurements are within the accepted limits for error. This then gives further validity to the conclusions of the subsequent analysis. Both observer error and inter-observer error are therefore insignificant and the measurements of RA 23, RA 45 and RA 6 are reproducible both by an individual observer and by different observers.
RESULTS

Thirty-four eight week old New Zealand white rabbits underwent sub-laminar wiring at three levels totalling one hundred and two sub-laminar wiring procedures. Fifteen of these rabbits survived for longer than eighteen months thereby attaining full skeletal maturity. Twelve rabbits died or were sacrificed within one month of surgery. Of these, four rabbits failed to recover from the general anaesthetic, or suffered intra-operative respiratory and cardiac arrest. Four rabbits developed uncontrolled diarrhoea and four developed a hind limb neurological deficit which was apparent immediately post-operatively resulting in the rabbit becoming unable to hop or move a hind limb normally. These eight rabbits were sacrificed for humane reasons. Six other rabbits died suddenly and for no apparent reason usually during the night at ages between seven months and sixteen months. Fifteen rabbits therefore survived fit and healthy to full maturity, and were then sacrificed at between eighteen and twenty-seven months of age. They were therefore sacrificed at between sixteen and twenty-five months post-operatively.

Immediately following sacrifice the entire length of the thoracic and lumbar spine was harvested from each of the fifteen specimens and was photographed and X-Rayed prior to preservation in formal saline. The radiograph indicated the precise location of the wires, so it was then possible to remove the spine proximal and distal to the segmentally instrumented section without having to disturb the wires to identify them. The muscle overlying the wire was left undisturbed to avoid interfering with the wire-bone interface. The segments of instrumented spine together with at least one non-instrumented segment above and one below were then placed in large containers of formal saline for eight weeks.

Radiographs of the specimens confirmed the initially gross observation that not all the spines had grown straight (Fig. 8:4). Many were straight on both the antero-posterior and lateral radiograph, but many spines had developed scoliosis with the sub-laminar wires on the convex surface, and some had developed an exaggerated kyphosis at the point of instrumentation. One spine developed a pronounced lordosis at the site of sub-laminar wiring and on close inspection of the lateral radiograph this appeared to be the result of hypertrophic bone around the wires of one segment fusing with the next segment thereby acting as a posterior spinal fusion in preventing longitudinal growth of the posterior spinal elements causing the development of a pronounced lordosis (Fig. 8:4c).
In six spines the sub-laminar wires appeared to leave growth undisturbed and the spines developed normally in both antero-posterior and lateral dimensions. Two spines did not develop a scoliosis but did develop a prominent kyphosis at the level of wiring (Fig. 8:12). The spine where fusion occurred posteriorly (N2) resulting in lordosis did not become scoliotic.

Six spines developed a scoliosis (Fig. 8:13). In each case the unilateral sub-laminar wiring had been performed on the convex side of the curve. This would suggest that the wire caused excessive growth of the lamina on that side. As will be seen later, there is no doubt that sub-laminar wiring resulted in abundant new bone formation adjacent to the lamina, mostly on the dorsal surface, and it may be that not only is the lamina thickened by sub-laminar instrumentation, but also the longitudinal growth of the lamina is stimulated. Three of the spines which became scoliotic developed no kyphosis, whilst three of the scoliotic spines also became kyphotic. Lamina overgrowth in response to sub-laminar wiring may occur in both antero-posterior and lateral dimensions of the spine resulting in the development of a kyphoscoliosis.

The growth pattern of nine spines was therefore sufficiently disturbed to produce some form of spinal deformity. Six spines remained free from deformity. The growth of six spines was such that a deformity developed in only one plane, either sagittal or coronal, whilst in three spines deformity was present in both dimensions (Fig. 8:12).

**STATISTICAL ANALYSIS OF LAMINAR THICKNESS**

Statistical analysis of the measurements described above was performed to determine whether or not the presence of sub-laminar wiring influenced (i) laminar thickness, (ii) laminar intrusion into the spinal canal and (iii) the cross-sectional area of the spinal canal. Laminar thickness on the wired side will from now on be known as Channel 2, containing five separate counts. Only five of the seven counts made were in fact used for analysis, since counts 1 and 2 tended to extend along the transverse process. Channel 3 represents laminar thickness on the control side. Channel 4 measures spinal canal radius on the instrumented side, and Channel 5 the radius on the control side. Channel 6:1 contains measurements of the cross-sectional area of the instrumented half of the spinal canal, and Channel 6:2 the cross-sectional area of the control half. Channels 2, 3, 4 and 5 each contain five separate counts or measurements (Fig. 8:14).
SPINAL DEFORMITY OBSERVED IN 15 NEW ZEALAND WHITE RABBITS, 18 - 27 MONTHS AFTER SUBLAMINAR WIRING

![Histogram to illustrate the gross deformity of the spine induced by surgery and sub-laminar wiring.](image)

**Figure 8:12.** Histogram to illustrate the gross deformity of the spine induced by surgery and sub-laminar wiring.

**Figure 8:13.** Antero-posterior radiograph of the fresh specimen to illustrate the various effects of sub-laminar wiring on spinal growth. Figure 8:13 (a) illustrates an analysis whilst Figure 8:13 (b) demonstrates that when scoliosis did develop sub-laminar wiring was always on the convex side.
Figure 8:13. Antero-posterior radiograph of the fresh specimen to illustrate the various effects of sub-laminar wiring on spinal growth. Figure 8:13 (a) illustrates no scoliosis whilst Figure 8:13 (b) demonstrates that when scoliosis did develop sub-laminar wiring was always on the convex side.
<table>
<thead>
<tr>
<th>CHANNEL/COUNT</th>
<th>LENGTH</th>
<th>LAMINAR THICKNESS: INSTRUMENTED</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>4011.43</td>
<td></td>
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<tr>
<td>1</td>
<td>4800.</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>4125.40</td>
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<tr>
<td>2</td>
<td>4100.</td>
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<tr>
<td>2</td>
<td>3862.</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3417.</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2922.43</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3173.</td>
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<tr>
<td>2</td>
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<td>2</td>
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<tr>
<td>2</td>
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<td>2</td>
<td>1455.</td>
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<tr>
<td>2</td>
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<tr>
<th>CHANNEL/COUNT</th>
<th>AREA (µm²)</th>
<th>PERIM (µm)</th>
<th>DCIRCLE (µm)</th>
<th>SPINAL CANAL HALF AREA: INSTRUMENTED</th>
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</thead>
<tbody>
<tr>
<td>8</td>
<td>1.100E+08</td>
<td>1.140E+05</td>
<td>3742</td>
<td>Instrumented</td>
</tr>
<tr>
<td>8</td>
<td>1.130E+08</td>
<td>1.170E+05</td>
<td>3777</td>
<td>Control</td>
</tr>
</tbody>
</table>

Figure 8:14. An example of the computer print-out of measurements obtained from one specimen (8:9). The channel number is illustrated in Figure 8:7 and the count number shown in Figure 8:9. The lengths indicating laminar thickness and canal radius are measured in microns. The cross-sectional areas of each half of the spinal canal (8-1 and 8-2) are measured in square microns. (Print-out size reduced by fifty per cent.)
Four groups of specimens were analysed using these six channels. Group 1 specimens had tight sub-laminar wires on one side, Group 2 wires were slack, Group 3 wires were tight and the lamina fractured and Group 4 consisted of non-instrumented controls taken from above and below the instrumented segments. Control levels were exposed surgically in the eight week old rabbit, and therefore subjected to sub-periosteal stripping but no instrumentation.

It was initially considered that the recorded lengths and areas measured in microns and square microns could be analysed directly. However, a more representative measure of asymmetry was considered to be provided by analysis of a standardised difference between the experimental and control channels.

The measure of asymmetry subsequently used in the analysis was as follows, taking as an example Channels 2 and 3.

$$RA_{23} = \frac{\text{Length Channel 2} - \text{Length Channel 3}}{\text{Length Channel 3}}.$$  
Thus $RA_{23}$ is the experimental channel length minus the control channel length, divided by the control channel length. Thus a value of zero would indicate equal lengths, whilst a value greater than zero indicates asymmetry with the experimental length greater than control length.

**STATISTICAL METHODS USED IN THE ANALYSIS**

The statistical analyses of the data takes the form of the analyses of variance (ANOVA). Essentially this is an extended form of Students t test. Using the Students t test, two means may be compared, but using ANOVA, more than two means may be tested. The test demonstrated whether or not the means of more than two series are equal and can therefore be used to compare five counts in one channel with the comparable five counts in the other channel, and to compare channels from different groups. If the result of the ANOVA is significant then Scheffe's test is used. This compares all pairs of means and gives an indication of which means contributed to the significant ANOVA results. If the ANOVA result is non-significant then there is no need to use Scheffe's test.

Initially groups 1 - 3 are compared, and the five counts within each group are compared in addition. Then the control group 4 is included in the comparison.

The ANOVA also tests for interaction. This is a test of, for example, whether the difference between means of say count 3 and 4 is the same for all four groups.
Assumptions in ANOVA

All statistical tests are based upon assumptions. The main assumptions upon which ANOVA is based are:

1. Constant variance (in this case for all group/count combinations).
2. The data must come from a normal distribution.

Assumption 1 was checked in the analyses and if the variances were not equal then a weighted analysis was used.

Assumption 2 was checked using a normal probability plot of the residuals from the fitted model. Basically, ANOVA is designed to fit a model. Parameters in the model are estimated and the residuals are the differences between the actual and predicted values (using the fitted model). If the data comes from a normal distribution, then a frequency histogram of the residuals should appear like that of the normal distribution and also the normal probability plot of the residuals should be roughly linear. In the subsequent analysis the normality assumption was checked in this way and adjusted in one case by removal of outliers. An outlier or influential observation is one which does not seem to 'fit in' with the rest of the data, being much larger or smaller than the majority of the values. If a comparison in the ANOVA table was found to be significant, then Scheffe's statistic was used to compare pairs of means. This was considered the most appropriate test for the above measurements.

COMPARISON OF CHANNELS 2 AND 3 USING RA 23 (FOR GROUPS 1-3)

An exploratory analysis revealed that there were to be several RA 23 values of greater than 4, some greater than 6. After detailed investigation it was found that this was due to several small control lengths compared to experimental lengths. It was assumed that since these only occurred several times and in different groups, then these were 'outliers' and were removed so the analysis proceeded without them. It was considered that had this not been done, their inclusion would have biased the results.

Figures 8:15 to 8:18 show frequency histograms for groups 1 - 4. It is clear that, in general, experimental lengths tend to be greater than control lengths. Negative values for the control group merely show natural variation. That is, in some cases in the non-instrumented control group 4, the distances on the two sides vary only as one would expect from natural variation in spinal symmetry.

Table 6 shows summary statistics for group means and count means
HISTOGRAM FOR RA23 GROUP 1
TIGHT SUBLAMINAR WIRES

Figure 8:15. Frequency histogram of laminar thickness ratio (RA 23) for tight sub-laminar wires (Group 1). -0.5 indicates the lamina on the instrumented side was 50% thinner than on the control side. 3.5 indicates the instrumented lamina was 350% thicker than that on the control side.
Figure 8:16. Frequency histogram of laminar thickness ratio (RA 23) of segments instrumented with slack sub-laminar wires compared with controls (Group 2).
Figure 8.17. A frequency histogram of laminar thickness ratio (RA 23) of the specimens with tight wires and broken lamina. 0.5 is equivalent to a 50% increase in laminar thickness on the instrumented side.
Figure 8:18. Frequency histogram of laminar thickness ratio of control Group 4. In this control neither side of the spine was instrumented, but one side of the spine was surgically exposed and sub-periosteal muscle stripping performed. This had no effect on laminar thickness since there is no significant difference between the two sides.
### TABLE 6  SUMMARY STATISTICS FOR RA 23 BY GROUP

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>MEAN</th>
<th>S.E.MEAN</th>
<th>MIN</th>
<th>MAX</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>149</td>
<td>1.3600</td>
<td>0.0696</td>
<td>-0.1608</td>
<td>3.9823</td>
</tr>
<tr>
<td>2</td>
<td>160</td>
<td>1.2529</td>
<td>0.0714</td>
<td>-0.3353</td>
<td>3.6391</td>
</tr>
<tr>
<td>3</td>
<td>177</td>
<td>1.1747</td>
<td>0.0694</td>
<td>-0.3540</td>
<td>3.7389</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>0.0932</td>
<td>0.0442</td>
<td>-0.5512</td>
<td>1.2210</td>
</tr>
</tbody>
</table>

### TABLE 7  RESULTS OF ANOVA ON RA 23 FOR GROUPS 1 - 3

<table>
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<tr>
<th>SOURCE</th>
<th>Degrees of Freedom D.F.</th>
<th>Sum of Squares of Values S.S.</th>
<th>Mean of Squares M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
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<td>2.7824</td>
<td>1.3912</td>
<td>1.91</td>
</tr>
<tr>
<td>COUNTS</td>
<td>4</td>
<td>40.7390</td>
<td>10.1848</td>
<td>14.00***</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>8</td>
<td>2.8856</td>
<td>0.3607</td>
<td>0.50</td>
</tr>
<tr>
<td>ERROR</td>
<td>471</td>
<td>342.6417</td>
<td>0.7275</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>485</td>
<td>389.0487</td>
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<td></td>
</tr>
</tbody>
</table>

### TABLE 8  RESULTS OF ANOVA FOR RA 23 FOR ALL FOUR GROUPS

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>S.S.</th>
<th>M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
<td>3</td>
<td>75.1498</td>
<td>25.0499</td>
<td>37.71***</td>
</tr>
<tr>
<td>COUNTS</td>
<td>4</td>
<td>37.6167</td>
<td>9.4042</td>
<td>14.16***</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>12</td>
<td>6.1934</td>
<td>0.5161</td>
<td>0.78</td>
</tr>
<tr>
<td>ERROR</td>
<td>526</td>
<td>349.3824</td>
<td>0.6642</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>545</td>
<td>468.3424</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*** = p <0.001  
** = p <0.01  
*  = p <0.05
by group. It is obvious that the means are similar for groups 1 – 3 but that for group 4 (control group) is significantly smaller. This means that if the control group is regarded as the normal situation, then the effect of sub-laminar wiring is to produce asymmetry between experimental and control regions with significant thickening of the instrumented lamina.

Figure 8:19 shows the above data in graphical form. One would conclude from this that there is no difference between the means for groups 1 – 3, but probably a significant difference between groups 1 – 3 and group 4. Group 4, the control group, has a mean value near zero as one might expect, that is no real difference between right and left sides. The two sides are clearly not the same on average with respect to the channels but the value is near enough to zero for a valid comparison of the means of groups 1 – 3.

Table 7 shows the results of ANOVA analysis. It is clear that there is no significant difference between groups 1 – 3, but a highly significant difference between counts (p <0.001). There is no significant interaction. The normal probability plot showed the data to be from a normal distribution.

Scheffe's test found there to be a significant difference only between count 7 and the rest (p <0.05). In general count 7 had a mean much smaller than the other counts. Further details of analysis of the counts will not be described since count-analysis contributed little to the overall results and understanding of the effects of sub-laminar wiring.

Table 8 shows the results of the ANOVA of RA 23 for all four groups. The introduction of group 4 produces a very highly significant difference between groups and counts with no significant interaction (p <0.001). Scheffe's test for groups showed that only group 4 was significantly different from the rest (p <0.05) and in general group 4 had smaller means than the other three groups. This is shown clearly in Figure 8:19.

Scheffe's statistic for count means showed that as before, count 7 tended to have significantly smaller means than any of the other counts (p <0.05).

**ANALYSIS OF LAMINAR INTRUSION INTO SPINAL CANAL BY COMPARISON OF CHANNELS 4 AND 5 USING RA 45 (GROUPS 1 – 4)**

Figures 8:20 to 8:23 show frequency histograms for group means. It can be seen that these are less skewed than those for RA 23, with a tendency to cluster around the zero point. This suggests that there is no
Figure 8:19. This illustrates the data from Table 6 in graphical form. The control Group 4 shows no difference in laminar thickness between right and left sides, but experimental Groups 1 - 3 all demonstrate a highly significant thickening of the lamina on the experimental side compared with the control group.
Figure 8:20. Frequency histogram to illustrate the effect of sub-laminar wiring on laminar intrusion into the spinal canal measured by comparison of the radius of the canal on the instrumented side with the radius of the canal on the control side. Group 1 consisted of tight wire. -0.15 represents a 15% increase in the radius on the instrumented side. 0.15 indicates a reduction by 15% of the canal radius on the operated side.
Figure 8:21. Frequency histogram illustrating the slack wiring on spinal canal radius. 0.25 represents a 25% reduction on canal radius.
Figure 8:22. Frequency histogram to demonstrate the influence of tight sub-laminar wires with fractured lamina on laminar intrusion into the spinal canal. 0.05 indicates a 5% intrusion into the spinal canal on the instrumented side.
Figure 8:23. Frequency histogram to show laminar intrusion in the control group. The figures centre around zero change suggesting no significant difference between the two sides of the spinal canal in control group 4.
significant difference between control and experimental regions.

Table 9 shows means by group. From this table it can be seen that group 2 has a higher mean than group 1 and 3 and a larger standard error. A tentative conclusion would be that group 2 may be different from the other groups 1 - 3. Figure 8:24 illustrates this clearly. From this graph it can be seen that group 2 (slack sub-laminar wiring) and the control group have roughly equal mean values. This implies that the slack sub-laminar wiring has no appreciable effect with respect to intrusion into the spinal canal, compared with the control side. The reason for this is unknown but may be of some clinical interest.

Table 10 shows the results of the ANOVA analysis. There is a very highly significant difference between groups (p <0.001) and a highly significant difference between counts (p <0.05). Scheffe's statistic for groups showed that group 2 had a significantly greater mean than group 1 (p <0.05) and group 3 (p <0.05). Scheffe's statistic for counts showed count 7 to be significantly greater than count 3 (p <0.05) and count 4 (p <0.05).

Table 11 shows the results of the ANOVA for comparing all four groups. There is a significant difference between groups and between counts but no significant interaction.

Scheffe's statistic for group means showed there to be a significant difference between groups 2 and 1, and 2 and 3. Scheffe's statistic for count means showed the same results for groups 1 - 3. The mean value of group 4 is not significantly different from the means of groups 1, 2 and 3.

ANALYSIS OF INTRUSION FACTOR BY MEASURING SPINAL CANAL CROSS-SECTIONAL AREA AND COMPARISON OF CHANNELS 6:1 and 6:2 USING RA 6 (GROUPS 1 - 4)

If the effect of sub-laminar wiring is to produce laminar intrusion into the spinal canal (as shown by RA 45 analysis) then the result of analysis of RA 6 would be to demonstrate a smaller area. The larger the negative RA 6, the smaller the area of the experimental side compared to the control side.

Figures 8:25 to 8:28 show the frequency histograms for groups 1 - 4. It does appear that the majority of values lie around zero. It is considered that this measure of asymmetry is not as sensitive as RA 45.

Table 12 shows summary statistics by group. From these we can see that group 2 tends to have a smaller mean than other groups but a similar
### Table 9: Summary Statistics for RA 45 by Group

<table>
<thead>
<tr>
<th>GROUP</th>
<th>MEAN</th>
<th>S.E. MEAN</th>
<th>MIN</th>
<th>MAX</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-0.0480</td>
<td>0.0086</td>
<td>-0.3195</td>
<td>0.0086</td>
</tr>
<tr>
<td>2</td>
<td>0.0072</td>
<td>0.0113</td>
<td>-0.4866</td>
<td>0.0113</td>
</tr>
<tr>
<td>3</td>
<td>-0.0569</td>
<td>0.0068</td>
<td>-0.3252</td>
<td>0.2853</td>
</tr>
<tr>
<td>4</td>
<td>-0.0095</td>
<td>0.0069</td>
<td>-0.1126</td>
<td>0.1391</td>
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</tbody>
</table>

### Table 10: Results of ANOVA on RA 45 for Groups 1 - 3

<table>
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<tr>
<th>SOURCE</th>
<th>D.F.</th>
<th>S.S.</th>
<th>M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
<td>2</td>
<td>0.3129</td>
<td>0.1565</td>
<td>11.14***</td>
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<tr>
<td>COUNTS</td>
<td>4</td>
<td>0.2526</td>
<td>0.0632</td>
<td>4.50**</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>8</td>
<td>0.0902</td>
<td>0.0113</td>
<td>0.80</td>
</tr>
<tr>
<td>ERROR</td>
<td>481</td>
<td>6.7558</td>
<td>0.0140</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>495</td>
<td>7.4115</td>
<td></td>
<td></td>
</tr>
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</table>

### Table 11: Results of ANOVA for RA 45 Groups 1 - 4

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<th>SOURCE</th>
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<th>S.S.</th>
<th>M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
<td>3</td>
<td>0.3457</td>
<td>0.1152</td>
<td>8.92***</td>
</tr>
<tr>
<td>COUNTS</td>
<td>4</td>
<td>0.2397</td>
<td>0.0599</td>
<td>4.64**</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>12</td>
<td>0.1075</td>
<td>0.0090</td>
<td>0.69</td>
</tr>
<tr>
<td>ERROR</td>
<td>536</td>
<td>6.9220</td>
<td>0.0129</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>555</td>
<td>7.6150</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*** = \( p < 0.001 \)
**  = \( p < 0.01 \)
*   = \( p < 0.05 \)
Figure 8:24. This illustrates graphically the figures given in Table 9. The differences between the groups when considering laminar intrusion into the spinal canal appears to be minimal, but subsequent analysis using ANOVA and Scheffe’s statistic revealed a significant difference between groups 2 and 1, and 2 and 3. The practical or clinical significance of slack sub-laminar wires resulting in reduced bony intrusion however requires classification.
Figure 8:25. Frequency histogram to illustrate the effect of tight sub-laminar wiring on the cross-sectional area of half the spinal canal when compared with the control half. -0.25 is equivalent to a 25% reduction in cross-sectional area on the instrumented side.
Figure 8.26. Frequency histogram to illustrate the effect of slack sub-laminar wires on the cross-sectional area of half the spinal canal compared with the control half. -0.05 is equivalent to a 5\% reduction in cross-sectional area.
Figure 8:27. Frequency histogram to illustrate the effect of tight sub-laminar wiring with a broken lamina on cross-sectional area of half the spinal canal.
Figure 8:28. Frequency histogram to illustrate the change in cross-sectional area of half the spinal canal which occurs at non-instrumented control levels.
### Table 12
**Summary Statistics for RA 6 by Group**

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>MEAN</th>
<th>ST. ERROR</th>
<th>MIN</th>
<th>MAX</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>-0.0652</td>
<td>0.0135</td>
<td>-0.2530</td>
<td>0.0566</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>-0.0227</td>
<td>0.0151</td>
<td>-0.2830</td>
<td>0.1635</td>
</tr>
<tr>
<td>3</td>
<td>36</td>
<td>-0.0654</td>
<td>0.0153</td>
<td>-0.2635</td>
<td>0.1709</td>
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<tr>
<td>4</td>
<td>12</td>
<td>-0.0485</td>
<td>0.0160</td>
<td>-0.1173</td>
<td>0.0853</td>
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### Table 13
**Results of the ANOVA for Comparing Groups 1 - 3**

<table>
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<tr>
<th>SOURCE</th>
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<th>M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
<td>2</td>
<td>0.0400</td>
<td>0.0200</td>
<td>2.76</td>
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<tr>
<td>ERROR</td>
<td>96</td>
<td>0.6945</td>
<td>0.0072</td>
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</tr>
<tr>
<td>TOTAL</td>
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<td>0.7345</td>
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</table>

### Table 14
**Results of the ANOVA for Comparing Groups 1 - 4**

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>S.S.</th>
<th>M.S.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUPS</td>
<td>3</td>
<td>0.0401</td>
<td>0.0134</td>
<td>1.96</td>
</tr>
<tr>
<td>ERROR</td>
<td>107</td>
<td>0.7284</td>
<td>0.0068</td>
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<tr>
<td>TOTAL</td>
<td>110</td>
<td>0.7685</td>
<td></td>
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</tbody>
</table>
standard error. Figure 8:29 shows more clearly than table 12 that group 2 has a value nearer to the control group mean than the other two groups as was the case with RA 45. The reason why this mean is less than the mean for the control group might be purely natural variation.

Table 13 shows the results of the analysis of variance comparing groups 1 - 3. Clearly there is no significant difference between groups (p <0.7). This data was therefore not analysed further. The probability plot showed the data to be from a normal distribution.

Table 14 shows the results of the analysis of variance for all four groups. There is still no significant difference between groups (p <0.1) as Figure 8:29 would indicate.

There was therefore no significant difference between the groups with respect to RA 6. This might be due to the fact that this is not as sensitive a measure of asymmetry as RA 45 when looking specifically for intrusion of the lamina on the experimental side into the spinal canal when compared with the contralateral and non-instrumented control. Figure 8:29 clearly shows this.

Figure 8:30 illustrates the percentage increase or decrease in the instrumented half of the spinal canal for the three experimental groups. It can be seen that there is considerable variation in the effects of sub-laminar wiring, but more intrusion is noted than expansion.

CONCLUSIONS

It appears that RA 23 is the best indication of asymmetry due to the effects of sub-laminar wiring. RA 45 and RA 6 are measuring similar parameters though RA 45 is more sensitive than RA 6. That is, RA 45 is a direct measure of intrusion of the lamina of the experimental side into the spinal canal compared with the control side, whilst RA 6 is a more indirect measurement using area.

The results of the ANOVA to compare groups 1 - 4 were:-

RA 23: No significant difference between groups 1 - 3 but a very highly significant difference between groups 1 - 3 and group 4, that is between experimental and control groups. (p <0.001)

RA 45: Highly significant difference between groups 1 - 3, and between groups 1 - 3 and group 4. (p <0.001)

RA 6: No significant difference between groups 1 - 3 and groups 1 - 3 and group 4. (p <0.07)

From the results of these analyses the only significant difference
Figure 3.29. This graph illustrates the figures for mean and standard error of the mean of RA 6 (or change in cross-sectional area) given in Table 12. It indicates that the cross-sectional area of the half of the canal which is instrumented is marginally reduced.
Figure 8:30. A frequency histogram to illustrate the effect of sub-laminar wiring on the cross-sectional area of half the spinal canal. Most specimens showed a 5%-10% reduction in area of the spinal canal on the instrumented side but this varied from a 20% increase to a 30% reduction in cross-sectional area.
between the groups 1-3 was in laminar intrusion into the spinal canal. The slack wire resulted in significantly less bony intrusion into the spinal canal than either the tight wire, or the tight wire around a broken lamina. In all other respects groups 1-3 were identical, but significantly different from group 4 (control) in all respects other than cross-sectional area of spinal canal measurement. From these results it can be concluded that:

1. Sub-laminar wiring results in the development of significant laminar thickening during growth and maturation of the New Zealand white rabbit.

2. Most of this laminar thickening occurs beneath the paraspinal muscles outside the spinal canal.

3. Significant intrusion of bone into the spinal canal was observed on the experimental side when compared with the contralateral control and non-instrumented control.

4. The radius of the spinal canal is reduced by a mean of 6% of the control value by bony intrusion following sub-laminar wiring (maximum 25%).

5. The cross-sectional area of the instrumented half of the spinal canal is reduced by approximately 5%, but the range varies considerably between a 25% and a 15% increase and this reduction is not statistically significant.

6. Measurement of the radius of the spinal canal is a more sensitive indicator of laminar intrusion than measurement of the cross-sectional area.

7. There is no significant difference between the experimental groups 1-3 in either laminar thickness or canal intrusion. There appears to be less intrusion when the sub-laminar wires were not fully tightened but when the difference between groups 1-3 and the control group 4 is taken into account, this difference becomes less significant.

8. From these results it is postulated that intrusion of bone into the spinal canal is insufficient to produce developmental spinal stenosis in the short term, since the cross-sectional area of the spinal canal is not significantly reduced. Clinical studies are required to observe whether such changes occur in man following sub-laminar wiring. It is proposed that laminar intrusion in man could be assessed using computerised tomography.
9. It is postulated that the relatively small intrusion of bone into the spinal canal compared with the large amount of new bone forming outside the canal may in some way be due to the inhibitory influence of the sliding or gliding movement of the neurological structures combined with the pulsatile action of the dura and blood vessels within the canal. This effect would be similar to the proposed influence of neurological structures on the growth of the vertebral column and spinal canal during embryological development (Larson 1981).
CHAPTER 9
THE BIOMECHANICS OF LUMBAR SPINAL STENOSIS

INTRODUCTION

Musculo-skeletal problems account for up to twenty-five per cent of General Practitioner consultations (Orthopaedic Services 1981, Morbidity Statistics 1972). Out of every hundred patients on the General Practitioner's list, four will each year initiate a consultation for back pain (OPCS 1974). Low back pain is responsible for over thirty million working days lost in Britain per annum (Department of Health and Social Security 1985). Between twenty-one per cent (Rowe 1969) and eighty-four per cent (Leavitt et al. 1971) of such "lumbago" occurs without a definite diagnosis, but it seems likely that degenerative disease of the spine underlies much of this absenteeism. Some occupations are affected more than others (Kelsey and White 1980). A study by the Royal College of Nursing reports the incidence of low back pain in nurses to be as great if not greater than amongst manual workers (Royal College of Nursing 1980).

Following skeletal maturation, the spine begins to degenerate both biochemically and biomechanically, and this process continues throughout life at a rate which may be genetically predetermined (Woessner 1981). Auto-immune mechanisms may also play a part in this process (Gertzbein et al. 1975, Bisla et al. 1976). Degeneration occurs most rapidly at the L4-5 and L5-S1 levels. These are the levels subjected to the greatest mechanical stresses, so it is reasonable to assume that mechanical forces play a major role in disc degeneration (Fig. 9:1).

This chapter considers a number of mechanical influences on the spine, some physiological, some pathological. The mechanical factors for consideration are shown in Table 1.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>INTERVERTEBRAL DISC MECHANICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Spinal Column Flexibility</td>
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<tr>
<td>2.</td>
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Figure 9:1. A simplified version of intervertebral joint mechanics at work on site in the intervertebral disc.
SPINAL COLUMN FLEXIBILITY

The rigid elements of the spinal column consist of twenty-four pre-sacral vertebra which increase in size from above downwards. Mechanically each vertebra can be divided into four parts.

1. The body which transmits most of the applied load.
2. Posterior facet joints which control and restrict intervertebral joint motion and transmit a variable load. Normal facets carry between three per cent and twenty-five per cent of the applied load, whilst arthritic facet joints can carry as much as forty per cent (Yang and Kind 1984).
3. Pedicles and laminae which complete the spinal canal.
4. Transverse and spinous processes which provide mechanically advantageous sites for muscle and ligamentous attachments.

Flexibility of the spinal column is provided by the disc which is composed of the central nucleus pulposus and surrounding annulus fibrosus. Each layer of the annulus has fibres directed at about 25° to 45° to the vertebral end plate, and this angle diminishes with age (Galante 1967). The direction of the fibres alternates in each successive layer, providing flexibility whilst maintaining strength. If one layer of the annulus is relatively slack during movement, its adjacent layers will be taut.

SPINAL COLUMN STABILITY

The spinal column has both intrinsic and extrinsic stability (Fig. 9:2). Intrinsic stability results from the opposing forces of (a) ligaments restraining vertebral motion and (b) pressure within the nucleus pulposus tending to push them apart. This latter is known as the "imbibition" pressure. Hydrophilic proteoglycans of the nucleus pulposus imbibe and retain water molecules to produce a turgid resilient nucleus. The vertebrae are pushed apart with a force of about 100K Pa: (Nachemson et al. 1979), which essentially pre-stresses the ligaments. Extrinsic stability results largely from trunk musculature, and intra-abdominal pressure which is in turn maintained by abdominal wall musculature.

With ageing the imbibition pressure falls. At birth the nucleus contains about eighty-eight to ninety per cent water (Hirsch et al. 1954), but by seventy years of age it is only sixty-five to seventy per cent water (Gower and Pedrini 1969). These values are similar for articular cartilage (approximately seventy-five per cent by weight) where water content is
SPINAL STABILITY

Figure 9:2. The imbibition pressure of the nucleus pulposus pre-stresses the fibres of the annulus fibrosus and the longitudinal ligaments to provide intrinsic stability even without muscle action. Muscle co-ordination and control and the pneumatic pressure of the abdominal viscera provide the extrinsic stability required for the spine to withstand the considerable loads to which it is subjected.
highest next to the articular surface (Stockwell 1970). In a sense therefore we do "walk and move on water". The gradual reduction with age in the water content of the nucleus occurs following proteoglycan breakdown, and associated ingrowth of collagens tissue. Although collagen is largely responsible for the tensile strength of the disc, proteoglycan is the major component resisting compression and providing resilience (Greenwald et al. 1978).

The mucopolysaccharide-protein complexes of young discs are large molecules composed primarily of chondroitin sulphate A and C. These macro-molecules are strongly hydrophilic and increase disc viscosity (Meachim and Freeman 1979). During the early twenties they begin to break down into smaller molecules possibly under the influence of changes in oxygen tension (Holm et al. 1981) and blood supply (Fig. 9:3). Mucopolysaccharide production is switched to chondroitin sulphate B and keratosulphate, smaller molecules which bind less water. The annulus fibrosus does not lose water with age (Urban et al. 1977), but changes in the glycosaminoglycan ground substance "lubrication" between adjacent lamellae may generate shear stresses with movement (Fig. 9:4) (Szirmai 1970).

The imbibition pressure of the nucleus is largely responsible for the variation in a person's height from morning to evening. Up to the age of ten a child is two per cent shorter at the end of the day than at the beginning. Young adults are on average 1.1 cm. taller in the morning, whilst a seventy year old individual shows little change (Fig. 9:5). A more bizarre example is an increase in height of over 5 cm. of an astronaut on return to earth through imbibition pressure unopposed by gravity (Fig. 9:6).

As imbibition pressure falls with age, the nucleus pulposus becomes less turgid, more fibrotic, and less efficient at re-distributing vertical compressive loads centrifugally to the inner layers of the annulus. A larger proportion of the load is therefore taken directly by the annulus itself. Bulging of the annulus occurs with loading (Hirsch and Nachemson 1954), and this may compress adjacent nerve roots or cauda equina (Naylor 1979). Loss of disc height with advancing age results in relative lengthening of the ligaments with consequent reduction of intrinsic spinal stability. This occurs at a time when the tone of trunk and abdominal musculature is reduced compromising extrinsic stability also.
Figure 9.3. During the early twenties biochemical degeneration begins within the nucleus pulposus with mucopolysaccharide production switched from chondroitin sulphate A and C, which are strongly hydrophilic molecules, to chondroitin sulphate B and keratosulphate which are less hydrophilic. This may be the result of changes in oxygen tension within the disc.
Cement layer of annulus weakens

Figure 9:4. The ageing process brings about changes in the glycosaminoglycan ground substance "lubrication" or "cement" between adjacent lamellae of the annulus fibrosus which may result in the generation of shear stresses during movement.
Figure 9:5. Diurnal variation in height of the subject varies with age. A male in his early twenties is an average of 1.1 cm taller in the morning than the evening, whereas no change occurs in a seventy year old individual.
Figure 9:6. During space travel the imbibition pressure of the disc is unopposed by gravity. An astronaut may gain 5 cm. in height within the first few days of leaving earth's gravitational field.
The intervertebral joint (one disc and two facet joints) is a type of universal joint with six degrees of freedom (Posner et al. 1980): three of translation (sideways, backwards and forwards, up and down) and three of rotation (side-to-side, bending forwards and backwards, and longitudinal rotation (Fig. 9:7). In practice however the joint does not perform simple movements in isolation, but always couples one translational with one rotational movement. For example, as the spine axially rotates there is always lateral flexion and vice versa. Three-dimensional radiographic studies of spinal movement (Pearcy & Tibrewal 1984) have shown that relatively little rotation occurs in the lumbar spine due to facet joint orientation. In the thoracic spine the centre of rotation lies within the nucleus so the disc is subjected to rotational forces (Gregersen & Lucas 1967), whereas in the lumbar spine this axis lies posterior to the disc subjecting the disc to translational shear forces. Shear and rotational stresses are probably the most significant ones contributing to degeneration.

Facet joint tropism, a rotational mal-alignment, displaces the axis of rotation of the intervertebral segment away from the midline, thus increasing the translational and rotational stresses imposed upon the opposite facet joint and contra-lateral side of the disc. This probably accelerates their degeneration, and it has been noted that the incidence of facet joint degeneration and annular rupture is increased on the side opposite the blocked facet joint (Farfan & Sullivan 1967).

Stress analysis of the intervertebral joint by Farfan (1972) indicated that the greatest stresses are generated during rotation when only half of the oblique fibres of the annulus are under tension, and the others are relaxed. At this time, the nucleus pulposus is contained by only half of the annular fibres. Many patients with acute disc prolapse do report a twisting of the trunk in addition to forward bend as a precipitating factor.

TECHNIQUES FOR THE MEASUREMENT OF SPINAL MOVEMENT

Accurate three-dimensional measurements of the six degrees of freedom of motion of the intervertebral joint have been made possible by the development of modern measurement techniques. Standard engineering principles cannot always be applied however, since they usually involve systems where the design specifications are known from the outset, unlike the human spine.
The intervertebral joint consisting of one disc and two facet joints is a type of universal joint with six degrees of freedom, three of translation, and three of rotation. One translational movement is always coupled with a rotational movement.
There are basically two general types of measurement techniques, firstly stereometry, and secondly electro-mechanical methods (Punjabi 1981).

1. Stereometry

The position of the points on the spine are determined in a global co-ordination system with the help of two or more views utilising some portion of the electromagnetic spectrum, such as visible X-Rays or infra-red. The images of the points (markers) are recorded on film, and digitised. Using geometric procedures the three-dimensional co-ordinates of the three points of the body in two positions are determined.

In a method developed by Selcom A.B. in Gothenburg, Sweden, the markers consist of light emitting diodes (LED's) working in the infra-red region of the light spectrum. These are imaged by two video cameras suitably positioned. Another on-line stereometric system, using acoustic waves, is that of Graf/Pen, by Scientific Research Accessories, Southport, Connecticut, U.S.A. In this system the markers consist of spark generators. Generation of each spark produces an acoustic wave which is picked up by three suitably placed microphones.

2. Electro-mechanical Methods

Various motion transducers have been utilised to measure three-dimensional motion. Kinzel et al. (1972) designed an all rotatory transducer consisting of seven linkages connected in series by single revolute (one rotational degree of freedom) joints (Kinzel 1972). The angular motion at each of the six revolute joints was measured by rotatory potentiometer. Another three-dimensional six degree of freedom motion transducer measures the six distance changes between three points on one body, and three points on the other body (Koogle 1977). These changes are measured by length transducers made of highly compliant, mercury-filled elastometer tubes. Utilising six Wheatstone bridges, electrical signals are produced that vary with the strain of the transducers. The strain-signal relationship however is both non-linear and unstable over time.

Punjabi in 1981 described a measuring system consisting of six uni-axial translation transducers arranged with respect to a fixed global co-ordinate (Punjabi 1981). The measuring jig was made of three balls A, B and C arranged non-collinearly and rigidly attached to each other and to the moving body. The motion measuring procedure consisted of recording the fixed transducer readings before and after the body undergoes a step of motion. Thus twelve readings are recorded to define a single
motion step. The initial position vector of the centre of the three balls was measured in the space-fixed global co-ordinate system. X-Rays were taken to establish the relationship between the three balls, and an anatomic co-ordinate system of the joint under study. Punjabi found that this photogrammetry system offered various advantages such as performing measurements without contact with the spine, and a large number of points can be monitored simultaneously. The main problem was that the digitisation and analysis of the points of interest was extremely tedious and time consuming. He did, however, claim that it utilised low-cost off the shelf items and had a high resolution and accuracy (0.4-0.9% error for 2-10 mm. or degrees), that it was electrically stable, and was capable of measurements in real time at reasonably high speeds using ultra high speed cameras.

LOADING MECHANICS

The ligamentous cadaver spine devoid of muscle control can support a compressive weight of only 20N (4 lb. or approximately 2 kg.) before collapse of the column occurs (Lucas & Bresler 1961) (Fig. 9:8). It is the trunk musculature which provides extrinsic stability and enables the spine to support tremendous loads. Vector diagram analysis at the lumbo-sacral junction of a person bending over to pick up a weight indicate forces in the order of ten times the lifted load are generated (Fig. 9:9). This implies that lifting a load of only 20 kg. would be sufficient to fracture the vertebral end plate (Armstrong 1965), since cadaver studies have shown end plate failure to occur under loads as small as 10kN (Perey 1957, and Evans & Lissner 1959). The normal disc is much stronger than the vertebral end plate of the cadaver spine on vertical loading (Jayson et al. 1973). Mechanisms must exist therefore to protect the vertebral end plate from tremendous loads transmitted by the disc. A number of protective mechanisms have been postulated.

THEORIES OF PROTECTIVE MECHANISMS

1. Hydrostatic Properties of the Disc

The hydrostatic model of the disc was first postulated by Schmorl (Schmorl 1926, Virgin 1951), and demonstrated in vivo by Nachemson (Nachemson 1960) both in normal and slightly degenerate discs. Pressure within the nucleus pulposus under loading conditions is redistributed both
The ligamentous cadaver spine devoid of muscle control can support a compressive load of only 20N (4 lb. or approximately 2 kg.) before the whole column buckles. This emphasises the part played by muscle control in maintaining spinal stability.
Figure 9.9. Vector diagram analysis of the forces acting on the lumbo-sacral joint of a person bending over to pick up a weight indicate that forces generated are in the order of at least ten times the load lifted.
radially to the inner layers of the annulus fibrosus as well as vertically to the vertebral end plates. This effectively converts compressive loading of the nucleus to tension within the annulus helping to unload adjacent vertebrae. Pressure is always present within the disc even at rest (Nachemson & Morris 1964), so the annulus is effectively pre-stressed.

2. **Pneumatic Properties of Abdominal Cavity**

Cross-sections through the trunk and C.T. scans demonstrate the abdominal cavity lies laterally as well as anterior to the spinal column. It was first postulated by Bartelink (Bartelink 1957) that this cavity may, under loaded conditions, act as a semi-rigid cylinder capable of unloading to some extent the spinal column. To test this hypothesis subjects swallowed tubes into the oesophagus and stomach and electromyographic measurements of various paraspinal muscles were taken (Morris et al. 1961). The subject then lifted weights from 50 lbs. to 200 lbs., and as muscle activity increased, so did intra-abdominal pressure and to a lesser extent intra-thoracic pressure. The same occurred on static loading of the spine when the subject pulled on a strain ring. On lifting 91 kg. (200 lbs.) the increased intra-abdominal pressure resulted in a reduction of the calculated forces at the lumbo-sacral junction from 939 kg. (2,071 lbs.) to 673 kg. (1,483 lbs.), a drop of thirty per cent.

Eie (1966) used Nordic ski-jumpers who swallowed intra-abdominal tubes linked to tape recorders with a retarder in the pocket to measure the gravitational force on landing. He found the landing force to be 10G which would theoretically have generated 420 kg. of compression at the lumbo-sacral junction, but this was reduced to 310 kg. by intra-abdominal pressure elevation since the jumper held his breath at the moment of impact.

3. **Dorso-lumbar Fascia**

Selective layers of the anterior abdominal wall arise posteriorly from the dorso-lumbar and illo-lumbar fascia and maintain its shape and length during lifting. Gluteus maximus and the ham-strings also generate forces in this fascia during lifting. The posterior layer of fascia converges upon the tips of the spinous processes posterior to the erector spinalis muscle (Bogduk & MacIntosh 1984). Its action through a long lever arm is therefore to extend the spine and possibly to reduce the force required in the erector spinae muscles during lifting.
4. **Blood Diversion**

During lifting, blood is diverted from the inferior vena cava via Batson's plexus to the epidural veins. From here it flows freely into the spongiosa of the vertebral bodies to increase the intra-osseous venous pressure. This may strengthen the vertebral body against compressive loads, but the hypothesis awaits experimental confirmation.

Weight lifters attempting to break their record wear a broad belt pulled tightly around the waist which increases abdominal pressure. It seems likely however that the "turgor hypothesis" would be more effective in conditions of acute compression such as ski-jumpers landing or aircraft pilots ejecting whilst performing a valsalvar manoeuvre (Andersson 1977).

5. **Measurement of Intra-discal Pressure**

Intra-discal pressures have been measured in normal subjects (medical students) by inserting a needle attached to a pressure transducer posterolaterally (Nachemson 1960). The pressure was increased during sitting, and especially when slumped forwards. This may help to explain a patient's increased back pain and sciatica in these positions. Lifting a heavy object with the knees bent and trunk straight generates a lower pressure than lifting whilst bending at the waist (Andersson et al. 1977). The distance between the centre of gravity of the lifted load and the body is of most significance in reducing intra-discal pressure. Intra-abdominal pressure is conversely much higher when lifting properly. This type of pressure measurement has been employed to aid the design of car seats in Sweden.

Some physiotherapy exercises recommended for patients with low back pain were found to generate enormous intra-discal pressures, particularly sit-up exercises. Isometric exercises produced least elevation of intra-discal pressure (Nachemson & Elfstrom 1970). Pressures were measured in two patients following spinal fusion and were found to be reduced during lifting.

**THE MECHANICAL EFFECTS OF PROLONGED LOADING IN FLEXION**

This section deals with the physical properties of creep, deformation and hysteresis in the lumbar vertebral column. Clearly loading in flexion like axial loading is a physiological state for the lumbar spine. However a number of occupational groups, for instance bricklayers and stone masons, regularly submit their spines to conditions of prolonged loading in flexion.
1. **Creep**

This is the progressive deformation of a structure under constant load when the materials are stressed well below their fracture points. In the study by Twomey it was the mechanism by which structural units of the spine deform in response to a given load and through which stress is redistributed until the structure deforms no further (Twomey 1982).

2. **Hysteresis**

This term describes the phenomenon whereby energy is absorbed or dissipated by a distorted structure. It describes how much of the strain was inelastic. Virgin showed that hysteresis of the vertebral column varied with both age and the region tested (Virgin 1951).

3. **Stiffness**

This is the load required to produce unit deformation. It may be conveniently related to Young modulus (E) of the material of the structure. This is the value of the applied stress divided by the material’s elastic strain. The larger the modulus, the stiffer the structure. The elastic limit of the material is the stress beyond which plastic deformation occurs resulting in a permanent set. Kazarian showed that as creep occurs in a vertebral segment, its stiffness increases (Kazarian 1975).

4. **Immediate Range of Movement**

This is a range of movement which is initial or immediate under a given load applied to the vertebral column and under-estimates the complete movement potential of the region, since it does not include the creep component.

5. **Total Range of Movement**

This includes both the immediate range of movement and the amount of creep occurring over a period of time.

Twomey established values for creep and hysteresis using nine male cadaver spines aged six to seventy-on (Twomey 1982). Using a standard flexion test he found that the average creep deflection was 3.8° (range 1.5°-6.5°). Creep was rapidly arrested in the young, but continued throughout the hour of the test in four older specimens. There is also an age change with hysteresis testing. Hysteresis in the young showed a common recovery curve independent of the initial load applied, but in the elderly hysteresis recovery was much slower and the recovery curve varied
with the size of the load applied. The range of movement was also noted to be markedly decreased in the elderly.

Twomey postulated that creep probably occurred as a result of progressive polymer distortion and fluid displacement from intervertebral discs. Virgin observed the appearance of beads of moisture on the surface of discs subjected to heavy axial loading (Virgin 1951). The decreased range of movement observed in the elderly may be due to changes in the intervertebral discs rather than in the apophyseal joints and their ligaments.

Virgin considered that repeated cyclical loading of the disc improved its "efficiency" because of an associated reduction in hysteresis. The study of Twomey however supports the view of Kazarian that hysteresis is unaffected by cyclical loading, implying that Virgin's conclusions are invalid because he did not allow the spinal columns to return to their original pre-test conditions before re-testing.

**FATIGUE FAILURE**

On histological examination of cadaver discs of people who had not had significant back pain, Farfan and Hirsch demonstrated distortion of the lamellae of the annulus fibrosus particularly posteriorly, and in ten to fifteen per cent radial fissures were seen postero-laterally (Farfan et al. 1972, Hirsch & Schajowicz 1953).

Recently nerve endings have been visualised in the annulus fibrous, and it may be that fissures or the products of degeneration associated with them are a source of pain (Yoshizawa et al. 1980). Fissures appear to be more a fatigue phenomenon than the result of acute trauma (Adams & Hutton 1983). Although theoretical stress analysis would suggest that the posterior annulus is subjected to greater forces than the anterior annulus, the proximity and orientation of the facet joints does to some extent protect the posterior annulus during axial rotation. During spinal flexion however the annulus becomes highly stressed posteriorly (Fig. 9:10).

To assess the fatigue effect of flexion on the loaded spine simulating heavy manual labour such as digging, a cadaver spine was cyclically loaded in a jig within the physiological range of flexion (Fig. 9:11). A compressive force of between 1,500 and 6,000 Newtons, depending upon age and sex, was applied forty times per minute to simulate the fastest a British workman could possibly work, and this was continued to a maximum of four hours giving 9,600 loading cycles (Adams & Hutton 1983). Some vertebral bodies failed through fatigue fracture within four hours of testing.
Figure 9:10. To illustrate the effect of spinal flexion on the loading mechanics of the disc.
Figure 9:11. The jig illustrated allows a compressive load of between 1,500 Newtons and 6,000 Newtons to be applied to the intervertebral joint forty times a minute with the spine in flexion. This is considered to be the fastest a British workman could possibly work. Over four hours this provides 9,600 loading cycles to test the effect of fatigue on the intervertebral joint.
Following this, dissection of the discs revealed that in over fifty per cent the lamellae were closely packed anteriorly, widely spaced laterally, and tightly curved postero-laterally. Postero-lateral radiate fissures were found in seventy per cent of all discs, and in one hundred per cent of L4-5 and L5-S1 discs from thirty to fifty year olds. These occurred most noticeably where the annulus was most stretched by an in-built eccentric setting of the flexion apparatus.

The degree of distortion of the lamellae is determined by the centrifugal force of the nucleus against the inner later of the annulus, posterior displacement of the nucleus during flexion, and vulnerability of the annulus postero-laterally to "creep" or stretching. It may be that when radial fissures become confluent a pathway is provided by which nuclear material can escape usually postero-laterally (Fig. 9:12).

Clearly in vivo the long term effects of fatigue are modified by the cellular repair mechanisms. Chondrocytes of a mature disc can manufacture collagen and large proteoglycan molecules. The turnover of proteoglycans is five hundred days in dogs, and collagen production is probably even slower (Urban et al. 1978), such that the repair processes will have only minimal effect over days or weeks, but may be substantial over a period of months or years.

ACUTE MECHANICAL FAILURE

The mechanism underlying acute extrusion of the nucleus pulposus must be clarified. As stated, pure compression of the intervertebral joint leads invariably to vertebral body failure, even with a fatigue pattern of loading (Perey 1957). Flexion just beyond the physiological limit results in damage to the ligaments of the neural arch but not the disc (Adams et al. 1980). Torsion damages the articular facets and if carried beyond the physiological range, produces circumferential tears of the annulus fibrosus (Adams & Hutton 1982). However when the intervertebral joint is flexed a few degrees beyond the normal range and then compressed with about 8,000 Newtons, comparable to the compressive forces generated within the discs of a healthy young man straining to lift a heavy weight, acute disc prolapse has been produced in cadaver spines in twenty-six out of the sixty-one joints (Adams & Hutton 1982). Adams and Hutton found the intervertebral joint could fail in one of four ways.
The postero-lateral part of the annulus fibrosus is particularly vulnerable during combined flexion and loading. Stretching or "creep" of the fibres occurs and when radial fissures develop and become confluent, nuclear migration follows with sometimes eventual nuclear sequestration from the disc space.
1. Nuclear extrusion - with nuclear material appearing on the posterolateral edge of the vertebral body or within the canal.
2. Annular protrusion with an annular ring disruption occurring at a point of bulging and the nucleus displaced posterolaterally behind the bulge.
3. Compression fracture - anterior crushing of the vertebral body and occasional end plate fracture with nuclear material sometimes entering the vertebral body.
4. Hyperflexion fracture - even before compressive loads were applied, the posterior rim of the vertebral body could become detached and the anterior rim of the body fracture. Sufficient flexion had to occur to render the annulus vulnerable and this is achieved by over-stretching the interspinous ligaments. It was reported many years ago that the spinous and interspinous ligaments were frequently found to be ruptured in patients undergoing disc surgery; the so-called "sprung back" described by Philip Newman (Newman 1952).

Across the age range, twenty-six out of sixty-one discs tested failed by nuclear extrusion, but on looking at the L4-5 and L5-S1 discs of cadaver specimens from thirty to fifty year old patients, the rate of prolapse rose to fourteen out of the seventeen discs tested. It is possible that there is a greater degree of pre-existing degeneration at these two levels resulting from their increased range of flexion. Alternatively these levels may be more susceptible to prolapse because of morphological or biochemical variations.

SPINAL MOTION AND LOADING IN THE PRODUCTION OF DEGENERATIVE CHANGE AND SPINAL STENOSIS

Normal Spinal Motion

Estimates of the range of flexion/extension motion of the lumbar spine vary from 121° in the young male acrobat (Wiles 1935), to 21° in elderly women (Tanz 1953). Begg and Falconer (1949) considered 70° to be the "normal" average total range of lumbar flexion/extension (Begg 1949). Axial rotation of the lumbar spine probably does not occur in isolation from other movements (Tondury 1971; Kapandji 1974). The facet joints are in fact designed to prevent it (Lewin 1961). Other sources estimate the range varies from 5° to 36° (Gregersen & Lucas 1967; Farfan 1973; Loebl 1973).

Taylor demonstrated that adolescents and young adult females are
thirteen to twenty-six per cent more mobile in the flexion/extension plane than are males of the same ages, but both sexes have substantially the same ranges of flexion/extension in middle aged and elderly groups (Taylor 1980). Adolescent females are more mobile (by 4°) than males of the same age, but adult males of all ages are a little more mobile in rotation (1°-2.5°) than adult females. This was based on work in both cadavers and human subjects. In all instances flexion was found to be greater in cadavers than in the living, suggesting an important role for muscles in limiting the amount of possible movement, as well as the possibility that the deformable but compressible "balloon" of the abdominal cavity restricted movement in the flexion plane. "Warm-up" exercises in living subjects increased the range of movement possible by approximately 4° of flexion and was assumed to be due to the stretching of the erector spinales muscles. A simulated "warm-up" of the cadaver specimens resulted in no increase in movement.

Advanced age produces not only osteoporotic bones and bony proliferative changes, but also the intervertebral discs and spinal ligaments show an increase in collagen and a decrease in elasticity due to increased cross-linkages of collagen molecules, and because elastic fibres are replaced by collagen. At the same time a decrease in the amount of muscle reduces the forces producing movement, while the decreased fluidity of the nucleus pulposus reduces the plasticity and compressibility of the discs (Taylor 1980).

THE EFFECT OF TORSION ON THE PRODUCTION OF DEGENERATION AND MECHANICAL DERANGEMENT OF THE LUMBAR SPINE

Twisting can damage the intervertebral joint and the joints between the articular processes act to some extent as a protective mechanism. Loading the intervertebral joint with body weight increased this protective function of the articular processes, rendering the whole intervertebral joint more resistant to torsion (Farfan 1970).

Farfan and others in 1970 established three experimental models to determine the torque strength and the degrees of rotation required to cause failure of firstly the intact intervertebral joint, secondly the isolated disc and thirdly the posterior intervertebral joint complex by dividing sequentially the annulus, the interspinous, supraspinous, and inter-transverse ligaments as well as the ligamentum flavum, finally leaving only the articular process intact. He used sixty-six necropsy specimens.
The intact intervertebral joint with a normal discogram failed at an average torque of $881.4 \times 10^6$ dyne-centimetres. The disc of this joint supplied thirty-five per cent of the resistance to the torque whilst the remaining sixty-five per cent of the resistance could be attributed to the posterior column comprising the articular process, their capsules and the interspinous ligaments. The whole intervertebral joint was found to be weaker when the disc was degenerate compared with normal discs.

The torsional strength of the disc was found to depend on the shape and area of the disc as well as on the integrity of its annulus, and on the rate at which loading was applied. It was also found that injury to the joint could be produced by slowly applied rotation in amounts within the range of normal lumbar movement. Annular fibres stated Farfan did not deteriorate purely as a result of age, but deteriorated principally due to damage and scarring of the annular fibres.

He postulated that in vivo disc degeneration and secondary intervertebral joint change was due to imposed torsional strains rather than compressive loading. Since the joints between the articular processes stabilise the intervertebral joint against torsion, it was suggested that any impairment of the function of these joints between the articular processes might result in a higher risk of disc degeneration.

Farfan obtained failure of the intervertebral joint after about $10^\circ$ to $30^\circ$ of torsion, and demonstrated irreversible damage to the apophyseal joint beyond $3^\circ$ of rotation. It should be noted that the physiological range of rotation motion has been measured in life to be about $10^\circ$ for the whole lumbar spine, or about $1^\circ$ for each side of each joint. These measurements were taken from young men asked to rotate with maximum effort (Adams 1981).

Adams and Hutton more recently demonstrated that the compression facet during rotation is the first structure to yield at the limit of torsion, and this occurred after about $1^\circ$-$2^\circ$ of rotation in joints with non-degenerate intervertebral discs (Adams 1981). Much greater degrees of torsion were required to damage the intervertebral disc, and Adams and Hutton concluded that torsion seemed unimportant in the aetiology of disc degeneration and prolapse.

Problems encountered were that the centre of axial rotation varied greatly from joint to joint, and required individual definition within any one specimen. The rig in fact constructed allowed the specimen to find its own centre of rotation.
Degenerate specimens with advanced apophyseal joint degeneration allowed extra mobility. This might well have been due to thinning of the articular cartilage of the apophyseal joints, thus allowing more "play" before the facets came into firm apposition. For example, if one degree of rotation brought the compression facet surfaces about 0.5 mm. closer together, a total loss of 3 mm. of articular cartilage would allow 8° of extra movement. Increased motion at the apophyseal joints might be responsible for acceleration in disc degeneration, and loss of disc height, and consequently compromise the spinal canal and nerve root foramina.

Sullivan demonstrated this point rather elegantly in an animal experiment (Sullivan 1971). Using thirty-nine white domestic immature rabbits he performed a facetectomy of the inferior articular facet. As the rabbits matured, it was clear that resection of one articular process of one posterior facet at the intervertebral joint level, altered the mechanics of the whole joint and precipitated a progressive change in the disc and also the contralateral facet joint. The appearance of the experimental joint was quite distinct from that of controls. The development of pathological changes was reflected in the radiological appearance of the joints. Changes in these rabbits resembled those noted in humans at disc surgery, confirming the effect of torque on the whole intervertebral joint in the cadaver and correlating disc prolapse with sciatica in those patients with facet joint asymmetry.

Sullivan had therefore produced degenerative changes in the disc and posterior facet joints by an indirect surgical assault. He confirmed that the main role of the posterior facet joint was to protect the disc against rotational stresses, and that removal of one facet joint produced rotational instability and subsequent degeneration of the whole joint.

SPINAL MOVEMENT AND INSTABILITY IN THE DEVELOPMENT OF SPINAL STENOSIS

When lumbar disc degeneration allows rotatory and lateral instability, postero-lateral bulging of the annulus fibrosus into the root canal occurs when weight is taken on the ipsilateral lower limb (Hirsch 1954). Symptoms of spinal stenosis do not occur according to Naylor until the development of this instability (Naylor 1979).

Naylor noted that gross lumbar spondylosis associated with spinal rigidity was not usually accompanied by spinal stenosis. Hirsch and Nachemson showed that when an intervertebral disc was compressed on one
side bulging appeared on the side of the compression (Hirsch 1954). If an isolated disc preparation was loaded from one hundred to four hundred kilograms, bulging due to compression normally caused a change in the radius of 0.8 per cent. In the degenerate disc however the bulging was at least thirty per cent. In patients with stenotic symptoms, annular bulging of the incompetent intervertebral disc postero-laterally into the lateral recess and nerve root canal is seen on radiography and at operation, giving a segmental waisting appearance and lack of filling of the nerve root sleeve.

Inefficient distribution of the load by the degenerate disc (vide supra) associated with alternative lateral compression during walking results in antero-posterior rotational and lateral instability of the segment, and increased intermittent annular bulging on the side of compression. This may interfere with the blood supply of the nerve root. Support for this view is afforded by the relief of stenotic symptoms by a successful intervertebral fusion, although the space of the spinal canal and lateral recess is unaltered by the procedure. This can well be demonstrated in patients suffering from both lytic and degenerative types of spondylolisthesis. In the latter, developmental mal-rotation of the intervertebral body contributes to stenosis of the lateral recess.

FORCES ACTING ON THE INTERSPINOUS LIGAMENTS AND THE NEURAL ARCH AND THEIR RELEVANCE TO LOW BACK PAIN

Punjabi measured directly the in vitro strains within the supraspinous ligament during flexion (Punjabi 1982). He found the computed values to be higher than those directly measured by about ten per cent at the highest loads applied. This difference could be attributed to deformation of the neural arch itself. In such measurements it was important to define exactly the points of ligamentous attachments to the vertebra. The interspinous ligament for instance does not run a pure cephalo-caudal direction as indicated by most standard anatomical texts, but runs obliquely as indicated by only one anatomical work, that is "Grant's atlas" (Grant 1952).

The stress-strain relationship of a ligament is, in general, highly non-linear and biphasic. A very low modulus of elasticity occurs in the early stages of loading and a very high modulus of elasticity occurs at load just prior to failure. This implies that in the initial stages of stretching only small stresses are produced in the ligaments, although the strains
themselves are large. Such a design by evolution decreases the possibility of damage to the ligaments and provides at the same time ample spinal mobility, with minimum expenditure of muscle energy. The opposite is true at high strains giving high resistance to motion and large energy absorption capacities providing stability and protection of the spinal cord and nerve roots. As stated above, spinal ligaments especially the capsular and posterior longitudinal ligaments are highly innervated.

The forces acting across the neural arch in the fully flexed position reach a maximum with high ligamentous stress. Maintenance of this posture may be important in the production of degenerative changes and low back pain. The lordotic standing posture as well as this fully flexed posture, may also be important in the aetiology of low back pain and degenerative change in the apophyseal joints (Hutton 1980).

Both extremes of flexion and extension have therefore been implicated in the production of degenerative change within the apophyseal joints, and the initiation of low back pain, and subsequently spinal stenosis.

**SPINAL INSTABILITY**

The surgeon is often faced with the dilemma of determining whether or not the lumbar or lumbo-sacral spine is clinically stable. This problem may be encountered following trauma, tumour or infection, or during a particular surgical procedure. In some circumstances the answer may be obvious but often the decision is very difficult.

Clinical instability was defined by White and Punjabi as:

"the loss of the ability of the spine under physiological loads to maintain relationship between vertebra in such a way that there is neither initial damage nor subsequent irritation to the spinal cord, or nerve roots, and in addition there is no development of incapacitating deformity or pain due to structural change" (White 1978).

Donald Nagel used five fresh human cadavers from the first and second lumbar vertebra to assess the degree of stability after progressive disruption of the joint followed by internal and external stabilisation (Nagel 1981). The disruption progressed from posterior to anterior leaving the anterior longitudinal ligament and anterior part of the annulus fibrosus intact. Flexion/extension range of motion was most sensitive to progressive disruptions and was significantly increased following disruption of the facets. A body cast was found to be effective in limiting all motions. Wire loops partially cut out through the spinous processes in all cases
with extreme flexion, whilst Harrington distraction rods were effective in limiting motion if under proper tension, but they did dislodge in three out of the five specimens.

In this study, axial rotation remained essentially constant until the facet joints were cut, and the increase in rotation was not statistically significant until maximal disruption. Axial rotation proved to be the most difficult to limit, and only the body cast reduced rotation to a statistically significant level. The Harrington rods were the only other device that appeared to be effective at all in limiting axial rotation.

Nachemson in cadaver material was able to demonstrate increased flexibility and increase in disc pressure following total laminectomy (Nachemson 1981). He noted however no difference when testing normal motion segments compared with degenerate ones.

Posner studied the mechanisms of the lumbar spine and lumbo-sacral region by transecting its components using flexion/extension and pre-load force alone or in combination. He confirmed a previous observation that the supraspinous ligament which is normally a strong structure measuring 5-7 mm. thick and 6-8 mm. wide, was not present in ninety-five per cent of spines below the L4 level (Posner 1982). Considerable care was made in preparation of specimens and in loading techniques and transection of the components as detailed in Posner's paper.

Posner's conclusions were that:
1. The site of failure of the anterior elements was at the disc, with the annulus fibrosus fibres and the cartilaginous end plate pulling off the vertebral body.
2. The site of failure of the posterior elements was at the attachment of the ligaments to the bone.
3. In extension loading with sequential component destruction the pre-load appeared to protect the spinal segments in excessive extension or sagittal plane rotation.
4. In flexion the pre-load added to the excessive flexion in sagittal plane rotation producing further instability.

Nachemson considered that 4 mm. of translatory movement between two vertebral bodies was on the safe side as an indication of abnormal motion (Nachemson 1981). Penning and Blickman, in radiological studies recently considered the measurement of angular changes which exceeded by 10° those of the adjacent vertebra were pathological (Penning 1980). These authors stressed the difficulty in obtaining accurate measurements.
of translatory instability, but their average of more than twenty spondylo-
listhetic segments in patients with good quality flexion/extension films
was 6 mm. (3-12 mm.). Nachemson considered that as of 1981 some
guide lines on the definition of instability were at least being obtained in
"semi-scientific terms". Clearly differences exist between the motion
segments from L1 to L5 and L5 to S1. In the lumbo-sacral segment up
to 6 mm. of forward translatory motion and more rotation can be
accepted as being within normal limits.

Therefore as a guide to lumbar motion segmental instability, using
flexion/extension radiographs, translatory motion in excess of 3 mm. at the
L1 to L5 segment, and in excess of 4 mm. at the L5-S1 segment, would
be abnormal, whereas an angular difference of more than 10° at the L1
to L5 segments, or more than 20° at the L5-S1 segments would be
considered abnormal.
CHAPTER 10

CLINICAL DIAGNOSIS OF SPINAL STENOSIS

INTRODUCTION

This chapter analyses the results of a clinical survey of two hundred and twenty-one patients treated at the Nuffield Orthopaedic Centre for spinal stenosis between 1976 and 1986. It utilises and summarises information gained from page one of the pro forma (Fig. 10:1), supplements this and combines it with a literature review to arrive at certain conclusions about the clinical manifestations of spinal stenosis.

The clinical presentation, symptoms and signs of lumbar spinal stenosis are remarkably constant regardless of the underlying cause of stenosis as outlined in the classification (Chapter 3). Some atypical presentations exist which will be described later in this chapter. The onset of symptoms is often quite insidious, and the clinician may find the patient's history unconvincing and the physical findings unimpressive in the early stages. This contrasts with the presentation of an acute disc prolapse in a younger individual.

Many elderly patients adapt to a limited exercise tolerance and may accept some discomfort in the lower limbs on walking for many years before seeking medical advice. However symptoms which have remained constant and tolerable for many years may suddenly increase following a lifting incident or some unaccustomed exercise. This may be the result of further nerve root irritation (Chapter 6), or excessive bulging of a degenerate annulus fibrosus into the spinal canal or nerve root canal during loading.

It can be seen from Table 10:1 that forty-three per cent (thirty-seven) of male patients managed medically described a lifting incident as the precipitating factor for their symptoms, twenty per cent (five) females treated surgically attributed their symptoms to a fall, but almost twenty per cent (nine) of males treated surgically could recall no specific injury and described an insidious onset of their claudication.
Figure 10:1. The proforma used for documentation of the pre-operative and post-operative symptoms, signs and functional disability.
1. A fall
2. Bending incident
3. Lifting incident
4. Twisting injury
5. Injury: mechanism unknown
6. Road traffic accident
7. Osteo-arthritis hips
8. Obesity
9. Previous spine surgery
10. Insidious onset: no injury

<table>
<thead>
<tr>
<th>INCIDENTS</th>
<th>MEDICAL</th>
<th>SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MALE</td>
<td>FEMALE</td>
</tr>
<tr>
<td></td>
<td>N=86</td>
<td>N=63</td>
</tr>
<tr>
<td>1. A fall</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>2. Bending incident</td>
<td>10%</td>
<td>5%</td>
</tr>
<tr>
<td>3. Lifting incident</td>
<td>43%</td>
<td>14%</td>
</tr>
<tr>
<td>4. Twisting injury</td>
<td>5%</td>
<td>0%</td>
</tr>
<tr>
<td>5. Injury: mechanism unknown</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>6. Road traffic accident</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>7. Osteo-arthritis hips</td>
<td>14%</td>
<td>14%</td>
</tr>
<tr>
<td>8. Obesity</td>
<td>5%</td>
<td>19%</td>
</tr>
<tr>
<td>9. Previous spine surgery</td>
<td>10%</td>
<td>19%</td>
</tr>
<tr>
<td>10. Insidious onset: no injury</td>
<td>5%</td>
<td>14%</td>
</tr>
</tbody>
</table>

Table 10:1. Incidents identified by the patient as marking the onset of symptoms, and other associations of possible relevance to the appearance of symptoms.

Clearly bulging of the annulus into the central or lateral part of the spinal canal may precipitate such acute narrowing of the canal that the cauda equina syndrome may be present at the first presentation of the patient. This obviously represents a surgical emergency. It does pre-suppose however that the spinal canal was already narrowed through developmental or degenerative change, with bony hypertrophy and thickening of the ligamentum flavum. There is clearly a broad overlap between the syndromes of disc prolapse and spinal stenosis and indeed eighteen per cent of all patients who were treated surgically at the Nuffield Orthopaedic Centre had a combination of degenerative and disc type of stenosis (Table 3:3). Indeed many young patients with prolapsed intervertebral disc complain of increasing leg pain on exertion or walking (Fig. 11:2 and 11:4). It is unlikely however that they would obtain relief through flexion of the spine.

SYMPTOMS OF SPINAL STENOSIS

In 1961 Blau and Logue reported six patients out of a series of three hundred with lumbar disc protrusions, who suffered intermittent claudication with pain and paraesthesia in the legs related quantitatively to exercise (Blau 1961). They noted that the symptoms usually had a constant relation to walking a certain distance, though in one patient they arose...
after standing for a fixed length of time. They were relieved completely after half to three minutes by standing still or sitting down. In some of the patients areas of skin over the feet or lower part of the legs were the sites of persistent tingling or numbness, which was over-shadowed by the appearance of a burning sensation on exercise. The burning disappeared completely on rest. None of these patients had sciatic pain, and they reported that "there were few symptoms which might have drawn attention to the intervertebral disc being the cause of the condition". Neurological signs were conspicuous by their absence, and the straight leg raise test was normal and painfree in five of the six patients. All the patients had a full range of spinal movement apart from one who had "extensive spondylosis".

Although Blau and Logue reported these patients as "an unusual syndrome resulting from central protrusion of the lumbar intervertebral disc", most clinicians would now recognise their description as spinal stenosis.

Six years earlier Verbiest had reported patients in whom such symptoms had "disappeared immediately" at rest (Verbiest 1955). The symptoms Verbiest described were typically tiredness and loss of power in both legs, anaesthesia, a feeling of numbness in some lumbo-sacral dermatomes, or bilateral sciatica. He considered this was due to a developmental narrowness of the bony lumbar vertebral canal.

Patients with spinal stenosis frequently give a history of trauma usually of a relatively slight degree (Table 10:1). The patient sometimes described a "snapping sensation" in the spine followed by pain low in the back and later gradual radiation of the pain over the sciatic distribution into the thigh and leg and foot. The interval between the onset of pain in the back and radiation into the leg varied in one series from a few hours to several months. This author proposed that the initial snapping sensation was due to rupture of the interspinous ligament, as proposed by Newman (Newman 1952).

Low back pain and neuritic pain of a mild intermittent nature may be present for many years before distressing symptoms appear. The combination of spinal stenosis, hypertrophic changes affecting the articular facets and lamina, gross thickening of the ligamentum flavum, discal ridges, and degenerative spondylolisthesis or retrolisthesis, may however combine to render the patient immobile through intractable pain and ultimately weakness. Intermittent neurogenic claudication begins with low
back pain and buttock pain at rest or during exertion. The pain radiates
down one or both legs, and is mainly sensory in nature with dyseaesthetic
and paraesthetic qualities quite different from those of vascular claudication.

Numbness, burning, and sometimes crampy sensations are likely to
appear whilst the patient is standing, and the condition is not influenced
by either strain or cough. An exaggerated lumbar lordosis augments the
symptoms. Activity is usually suspended before weakness appears, but
sometimes collapse occurs with prolonged exercise or standing. Relief
follows lying down, sitting or bending forwards, so that the lumbar lordosis
is relaxed. Claudication also occurs with narrowing of the foraminal and
lateral recesses without sagittal stenosis, and can be unilateral
(Epstein 1977).

The features differ from those of intermittent vascular claudication,
in that vascular claudication classically induces crampy, tired sensations
affecting exercised muscle groups. Relief is obtained by standing still, but
neither recumbency nor postural change are ameliorating. Impotence and
loss of sphincter control are variable and uncommon. Bruits are audible
over the groins and vascular pulsations become diminished (Fig. 11:2 and 11:4).

It is not possible to distinguish on clinical grounds between central
and lateral or nerve root canal stenosis. Lateral canal stenosis with the
superior facet syndrome is quite often unilateral, but may present with
bilateral pain. Again in nerve root canal stenosis which may result from
resorption over a number of years of an isolated disc, pain in the buttock
and leg is aggravated by physical exercise or walking. Again there may
have been numerous incidents of trauma, particularly involving falls onto
the buttocks causing severe bilateral buttock and leg pains. Repeated
bouts of low back pain may have been present for many years, but usually
resolving over three or four days, and associated with progressive
degeneration occurring in the facet joints (Crock 1976).

Details of the clinical presentation of patients with spinal stenosis
will now be described.

1. **Age at onset of symptoms**

Many patients with spinal stenosis have had low back pain for many
years, but the onset of spinal stenosis is marked by the development of
intermittent neurogenic claudication, and this frequently occurs in the
fifth and sixth decades. In those patients with spondylolisthesis it may
occur a few years earlier, but it is clearly one or two decades after the
peak incidence of disc prolapse.

The age of patients presenting at the Nuffield Orthopaedic Centre is shown in Table 10:2. It can be seen that patients in the surgical group present at a marginally younger age than those treated conservatively. This may be for two reasons. Firstly their symptoms and signs may be more severe such that they require, and by virtue of their age sometimes demand, surgical treatment. Secondly the conservatively treated group mean ages are biased slightly by the extreme elderly patients included because they are unfit for surgery.

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>63 (31-82)</th>
<th>Females</th>
<th>60 (30-84)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSERVATIVE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SURGICAL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>56 (18-82)</td>
<td>56 (18-82)</td>
<td>54 (12-80)</td>
<td>54 (12-80)</td>
</tr>
<tr>
<td>Females</td>
<td>54 (12-80)</td>
<td>54 (12-80)</td>
<td>54 (12-80)</td>
<td>54 (12-80)</td>
</tr>
</tbody>
</table>

Table 10:2. Mean age at presentation according to sex of patients at the Nuffield Orthopaedic Centre.

2. Sex

Males are more frequently affected than females, and this applies not only to degenerative but also to developmental stenosis (Table 3:3). Spondylolisthesis however is more frequently found in the female, and in the surgical group degenerative spondylolisthesis accounts for thirty-six per cent of spinal stenosis in the female.

Of the entire group of two hundred and twenty-one patients with spinal stenosis at the Nuffield Orthopaedic Centre, one hundred and thirty-three (sixty per cent) were male and eighty-eight (forty per cent) female. Thirty-five per cent of males were managed surgically and thirty per cent of females underwent surgical decompression (Table 10:3).
<table>
<thead>
<tr>
<th></th>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSERVATIVE</td>
<td>149</td>
<td>86</td>
</tr>
<tr>
<td>SURGICAL</td>
<td>72</td>
<td>47</td>
</tr>
<tr>
<td>TOTAL</td>
<td>221</td>
<td>133</td>
</tr>
</tbody>
</table>

35% males were treated surgically
30% females were treated surgically
65% of all patients surgically treated were male

Table 10:3. The sex and treatment category of the Nuffield Orthopaedic Centre patients.

3. Occupation

The occupation of two hundred and twenty-one patients in the series was reviewed and each patient placed into one of four categories illustrated in Table 10:4.

<table>
<thead>
<tr>
<th>OCCUPATION: CLASSIFICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sedentary e.g. Clerical, Solicitor, Inactive, Retired</td>
</tr>
<tr>
<td>2. Travelling e.g. Busdriver, Businessman: active but not lifting</td>
</tr>
<tr>
<td>3. Light manual e.g. Cleaner, Decorator, Motor mechanic: light lifting</td>
</tr>
<tr>
<td>4. Heavy manual e.g. Builder, Farmer, Nurse, Blacksmith: heavy lifting</td>
</tr>
</tbody>
</table>

Table 10:4. The occupational groups into which patients were classified.

Having classified patients by occupation and sex and again into medical and surgical treatment regimes, a number of observations were noted and were contrary to what was predicted (Table 10:5). Firstly, the majority of males (sixty-five per cent) and just over half the females (fifty-two per cent) in the surgical group belonged to the sedentary or travelling occupations, and did not engage in strenuous activities, although one quarter of females treated surgically were employed in heavy manual labour such as nursing or farming.

On the other hand the majority of males (sixty-one per cent) and females (seventy-two per cent) in the medical group were engaged in either light or heavy manual labour. This is compatible with forty-three
per cent of males in the conservative group identifying a specific lifting incident as the factor which precipitated their symptoms (Table 10:1).

<table>
<thead>
<tr>
<th></th>
<th>MEDICAL</th>
<th></th>
<th>SURGICAL</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>SEDENTARY</td>
<td>22%</td>
<td>14%</td>
<td>28%</td>
<td>42%</td>
</tr>
<tr>
<td>TRAVELLING</td>
<td>17%</td>
<td>14%</td>
<td>37%</td>
<td>10%</td>
</tr>
<tr>
<td>LIGHT MANUAL</td>
<td>30%</td>
<td>36%</td>
<td>19%</td>
<td>24%</td>
</tr>
<tr>
<td>HEAVY MANUAL</td>
<td>31%</td>
<td>36%</td>
<td>16%</td>
<td>24%</td>
</tr>
</tbody>
</table>

Table 10:5. Occupation of patients with spinal stenosis grouped according to sex and management regime.

4. Duration of symptoms

Since eighty per cent of people suffer low back pain at some stage in their life, it is hardly surprising that the majority of patients with spinal stenosis gave a history of low back pain which preceded the onset of claudication symptoms. Figure 10:2 is a histogram which illustrates the duration of symptoms of spinal stenosis before presentation to the orthopaedic out-patient department. In some patients the presentation was delayed by the patient's referral to a vascular surgeon for presumed ischaemic claudication, whilst in the majority the symptoms were usually not severe enough initially for the patient to feel justified in consulting their general practitioner and secondly for the general practitioner to consider an orthopaedic referral necessary. The natural history of the condition in those patients who finally require orthopaedic referral is often one of relentless and unremitting progression of symptoms until treatment is instituted. It is not known, however, how many patients with symptoms do not seek referral because of the spontaneous resolution or remission of their condition.

Table 10:6 is a summary of the results shown in the histogram of Figure 10:2. It is clear that the majority of patients have had symptoms for at least one year before requesting medical advice.
CONSERVATIVE GROUPS

Figure 10:2. Histograms to illustrate the duration of neurogenic claudication in each sex management group. The abscissa is time in months (m) or years (y). The ordinate indicates the number of patients in each group.
Table 10:6. The duration of symptoms of neurogenic claudication on first presentation to the orthopaedic surgeon, grouped according to sex and treatment regime.

The patient may, however, have suffered from low back pain, either constantly or more commonly relapsing, for many years before developing symptoms of neurogenic claudication and spinal stenosis. The interval between the patient's first recollection of low back pain and the onset of spinal stenosis is commonly between ten and twenty years (Table 10:7).

Table 10:7. The interval between low back pain and neurogenic claudication in the four groups.

5. Interval between onset of symptoms and first operation

This may vary between one month and forty years (Paine 1976). The average duration of symptoms before surgery at the Nuffield Orthopaedic Centre was six years. Surgery for this condition is not offered for patients with back pain, but may be appropriate when nerve root symptoms and signs develop.

6. Back pain

Painful stiffness of the spine may occur with inactivity, and may therefore wake the patient in the early morning. He may however quite often be able to work through this discomfort during the morning, and loosen up within two to three hours of activity. With increasing levels
of activity however, the pain may return and be troublesome in the evenings.

The majority of patients presenting with neurogenic claudication also complained of low back pain. This was usually experienced in the lower lumbar and lumbo-sacral regions in the midline and could radiate to the buttocks and backs of the thighs. Clearly this pain radiating to the backs of the thighs might increase on walking, but this was not classified as neurogenic claudication since it did not radiate below the knee in the fourth and fifth lumbar and first sacral dermatomes. Moreover its character was usually that of a dull ache and not the numbness, tingling or dysoesthesia characteristic of neurogenic claudication. Table 10:8 indicates the frequency of low back pain as either a major or a minor presenting symptom. Clearly patients with low back pain alone were not included in this series.

<table>
<thead>
<tr>
<th>LOW BACK PAIN:</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDICAL</td>
<td>73%</td>
<td>86%</td>
</tr>
<tr>
<td>SURGICAL</td>
<td>88%</td>
<td>94%</td>
</tr>
</tbody>
</table>

Table 10:8. The frequency of low back pain as one of the presenting symptoms of patients with spinal stenosis.

It is considered probable that the incidence and severity of low back pain is greater in patients with spinal stenosis than in the general population, but so far no specific data is available to confirm this.

7. Leg pain

Bilateral leg pains occur in over a third of patients with degenerative or developmental spinal stenosis. In both types pain usually comes on with increasing activity and disappears or improves with rest. This contrasts with patients with prolapsed intervertebral disc, who will be reluctant to even begin any activity. Although both legs may be involved, one leg is usually more affected than the other (Yates 1981). Pain and paraesthesia tend to start peripherally and extend upwards with exercise. In spinal stenosis more than one nerve root is more frequently involved than in patients with disc prolapse. With both disc prolapse and stenosis however, the commonest roots to be involved are the L5 and S1 roots. The L3 and
L4 roots are more commonly involved by stenosis than disc prolapse, whereas the L2 root is involved very much more frequently by stenosis than by disc prolapse.

The features of neurogenic intermittent claudication may descend from the buttocks or ascend from the feet as a "sensory march". Weakness generally follows the sensory symptoms if the patient continues the activity beyond the appearance of these unpleasant sensations. Sphincter disturbance, usually an inability to void, may accompany other symptoms brought on by appropriate activity.

The symptoms of neurogenic claudication are predominantly sensory appearing during the course of exercise or while maintaining a fixed posture and disappearing after rest or a change in posture. For a given patient the temporal pattern of activity - symptoms - relief is predictable. Sensory symptoms have distinctly paraesthetic and dysesthetic qualities, and do not simulate classical sciatica. The discomfort is described as numbness, coldness, or rarely burning, and the patient seldom mentions cramping. Some patients often deny pain, and instead select such terms as "unpleasant" and "uncomfortable".

Certain patients do not stand erect because extension of the lumbar spine produces pain, paraesthesia or weakness. Predictably the patient who walks stooped forwards will recline in a flexed posture, being unable to tolerate prone or straightened supine position. Each patient can relate his particular pattern of symptoms to specific activities. In all patients walking aggravates or precipitates symptoms. Careful questioning however reveals that many patients experience identical symptoms with hyperextension of the spine, or in some instances with even the slight extension required to stand erect.

I have already drawn attention to the fact that in the majority of patients with spinal stenosis pain in the legs follows many years of back pain (Table 10:7). All patients in the Nuffield Orthopaedic Centre series had neurogenic claudication on exercise. This is often described as a tingling, numbness, deadness of the leg, feeling of hot and cold running water along the leg, and unsteadiness on walking with the development of weakness if the patient attempts to walk through the sensory dysaesthesia. Alternatively the pain may be severe, sharp, stabbing and very distressing to the individual. Patients sometimes complain that they feel as though they are walking on broken glass, unlike the walking on marbles of metatarsalgia, or the walking on cotton wool of tabes dorsalis.
Although neurogenic claudication is predominantly sensory in nature, with motor weakness developing only in some patients who exceed their sensory claudication distance, in the Nuffield Orthopaedic Centre series a small number of patients presented with pure motor claudication — that is weakness on walking — and denied having pain or dysesthesia.

Fifty-one per cent of patients in the series experienced neurogenic claudication both above and below the knee, whilst twenty-four per cent complained of symptoms exclusively below the knee and twenty-six per cent of neurological-type symptoms purely above the knee (Figure 10:3). It can also be seen from Figure 10:3 that seventy-seven per cent of patients developed symptoms in one leg only (thirty-eight per cent right and thirty-nine per cent left) and only twenty-three per cent of patients had symptoms in both legs. This is surprising when one considers that thirty per cent of patients had central degenerative stenosis, and only seven per cent of patients had a pure lateral canal stenosis, with all other forms of stenosis in the series theoretically producing a central rather than lateral type of stenosis (e.g. degenerative spondylolisthesis). This may be anaalogous to the situation of bilateral osteo-arthritis of the hips when severe symptoms on the one side tend to mask symptoms from the less affected side, which then become severe and disabling following total hip replacement on the most painful side.

There were, however, a number of patients in this series in whom claudication symptoms changed from one leg to the other over a period of months or years. In other patients, following prolonged activity, the symptoms of claudication which began in one leg would then begin in the opposite leg producing bilateral claudication on exceeding the unilateral claudication distance.

Neurogenic claudication is usually precipitated by walking a certain distance, or when stenosis is more severe, may occur simply on standing, for instance on first getting out of bed in the morning. When claudication is provoked by walking, the claudication distance is variable between patients but relatively constant for each patient. The frequently quoted association of patients with neurogenic claudication being able to walk further uphill than downhill, whilst those with vascular claudication can walk further downhill than uphill was only rarely noted by patients in this series. This may be more a factor of the Oxford terrain than the condition of spinal stenosis. Claudication distance did vary considerably from twenty yards to two miles (Table 10:9).
Figure 10:3. The distribution of pain in the legs on exercise in each sex/management group. Each figure is the percentage frequency in each group for pain felt principally above the knee, below the knee, or in the entire lower limb (the middle figure). The figures printed between the legs indicate bilateral symptoms.
The range of claudication distances noted in the Nuffield Orthopaedic Centre series of patients with spinal stenosis, grouped according to sex, limb involvement, and conservative or surgical treatment group. The claudication distance ranged from 20 yards to 2 miles.

The concepts of "postural" and "ischaemic" claudication have been introduced in Chapter 6, but this is an appropriate point to describe further the clinical features of these two sub-groups of spinal stenosis. In the first group of patients symptoms appear only after exercising the legs and are related to exertion more than to posture. In the second group of patients symptoms appear during any activity or body position that involves extension of the lumbar spine, and this group is termed "postural".

8a. Ischaemic claudication

No matter how long these patients stand or what position the spine is in, claudication cannot be produced. Wilson examined two patients with ischaemic claudication in whom the pain was produced by ergometric exercise of the gastrocnemius muscle whilst the patient lay in bed (Wilson 1971). Neither the severity nor the time of onset was affected in the slightest by increasing or decreasing the lumbar lordosis by means of pillows. In these patients the onset of symptoms was directly related to the rate and duration of exercise.

Whilst the patient breathed air, pure oxygen, and a mixture of nitrogen and twelve per cent oxygen, the claudication time varied directly with the oxygen tension inspired. Blau and Logue favoured an ischaemic basis for neurogenic claudication because the symptoms could be abolished rapidly, and because sensation was impaired before motor function (Blau 1961).
8b. Postural claudication

In this group of patients symptoms are clearly related to the lordotic posture, appearing not only after walking but also after the patient stands quietly or kneels with his back extended. Depending upon his spinal posture symptoms may or may not appear when he is reclined. Symptoms are not precipitated by activities such as riding a bicycle or playing tennis, since neither of these activities involves continuous hyperextension of the lumbar spine. Symptoms are characteristically relieved when the patient bends forwards or squats.

Breig has shown that extension of the lumbar spine causes protrusion of the intervertebral discs, with dorsal displacement of the cauda equina roots (Breig 1960). Extension also by decreasing the length of the spinal canal forces an increase in the cross-sectional diameter of the cauda equina roots. Ehni has shown the effects of changes in the lordotic position during myelography (Ehni 1965), extension produces total block, whilst flexion permits the passage of contrast material through the previously blocked area. Patients with postural claudication can neither lie prone nor supine without discomfort unless they flex their knees.

In patients with postural claudication symptoms occur independently of muscular contraction. Ischaemia of the involved nerve root may occur as a passive event secondary to mechanical compression if, as proposed by Denny-Brown and Brenner, that ischaemia produced by a tourniquet bears a close resemblance to the effects of prolonged pressure on a nerve root. (Denny-Brown 1944).

In a smaller group of patients, neurogenic leg pains were experienced simply on standing. This postural claudication was noted almost immediately on rising from a chair or from bed, and indicates a must more severe form of spinal stenosis than ischaemic claudication which develops on exercise. It is not surprising therefore that only three per cent of patients managed conservatively experienced postural claudication, whilst twenty-four per cent of patients treated surgically complained of the symptoms of postural claudication on first presentation (Table 10:10).
Table 10:10. The percentage of patients who described postural or static (standing) claudication at the time of first presentation. Postural claudication indicates a more severe form of stenosis, as suggested by the larger number of patients in the surgical group with this complaint.

Patients with postural claudication are often housebound or may resort to the use of a wheelchair.

9. "Time to relief" of claudication symptoms

It is often stated, and now appears in some text books, that neurogenic claudication can be distinguished from vascular claudication by the time taken for symptoms to subside once they have become maximal. This is an erroneous concept, and indeed patients with severe spinal stenosis may obtain no relief by simply standing still, but rapid relief on squatting (the shoe-lace sign). In this series of patients, the "time to relief" varied considerably from one minute to thirty minutes, but it must be emphasised that not all patients obtained complete relief of symptoms even then (Table 10:11).

Table 10:11. "Time to relief" of neurogenic claudication in patients according to sex and treatment groups.

10. Impulse pain

Impulse pain is a neurological type of pain (tingling, electric shocks, shooting pains) experienced below the knee on coughing, sneezing, laughing or straining on the loo. It is commonly experienced in patients with irritability of the sciatic nerve roots which may be chemically or
mechanically mediated. It probably occurs by one or more of the three following mechanisms.

(a) Spasmodic and violent contraction of abdominal and paraspinal muscles causing loading of the spine.

(b) The valsalva manoeuvre raises intracranial pressure, and a wave of elevated cerebro-spinal fluid pressure travels along the spinal canal displacing nerve roots as it travels. Any nerve roots which are partially depolarised (irritable) may then initiate a wave of depolarisation. A normal nerve root will be unaffected.

(c) Increased intra-abdominal pressure diverts blood via Batson's plexus into the epidural venous plexus increasing venous congestion of the nerve roots augmenting their ischaemia, and ischaemic neuritis.

Table 10:12 indicates that approximately seventy per cent of patients with spinal stenosis do experience "impulse pain" on questioning, with little difference between the medical and surgical groups.

<table>
<thead>
<tr>
<th>&quot;IMPULSE PAIN&quot;:</th>
<th>PRESENT</th>
<th>ABSENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDICAL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>60%</td>
<td>40%</td>
</tr>
<tr>
<td>Female</td>
<td>80%</td>
<td>20%</td>
</tr>
<tr>
<td>SURGICAL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>71%</td>
<td>29%</td>
</tr>
<tr>
<td>Female</td>
<td>70%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Table 10:12. The percentage of patients according to sex and treatment group with and without impulse pain.

It is unlikely on this basis that impulse pain can be used as an important diagnostic or prognostic indicator.

11. Night pain

Low back pain worse at night, interfering with sleep, has historically been regarded as a harbinger of spinal metastasis. However, more often the converse is the case. Frequently nocturnal back pain is not pathognomonic of occult spinal metastasis and can well be associated with more benign conditions. In spinal stenosis, night pain can arouse patients from sleep (La Ban 1984). In the Nuffield Orthopaedic Centre the percentage of patients woken at night time by leg pains is shown in Table 10:13.
Table 10:13. The percentage of patients with spinal stenosis grouped according to sex and treatment group who claim to be "woken at night" by pains or discomfort in the legs.

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDICAL</td>
<td>5%</td>
<td>24%</td>
</tr>
<tr>
<td>SURGICAL</td>
<td>9%</td>
<td>20%</td>
</tr>
</tbody>
</table>

Nocturnal back and leg pain associated with spinal stenosis can occur unrelated to activity and whilst simply reclining. Venous distension adjacent to a pre-sensitised nerve root within the rigid, unyielding confines of a narrowed osseous passage triggers the pain arousing the patient from sleep.

Elevations in central venous pressure in patients with congestive cardiac failure are readily transmitted by collateral communicators to the paravertebral venous plexus, producing distension of the epidural plexus. Volume distension of this venous bed, a lack of valves and multiple interlinking tributaries may divert and effectively reduce flow and oxygenation. In many patients with spinal stenosis, sheets of epidural veins replace the normal paired bilateral anterior internal epidural veins, contributing to reduced flow and increased venous stagnation.

Additional mechanisms accounting for nocturnal increases in venous pressure are infusions of fluid moving from the extra-cellular space into the circulation as dependent oedema fluid moves into the venous bed, as well as diurnal reductions in systemic venous pressures in excess of twenty per cent.

Isolated anatomical or dynamic regional blocks to spinal fluid flow with subsequent increases in pressure within the confined fluid may also contribute to significant anoxia. The thin-walled venous plexus subjected to an increase in extrinsic spinal fluid pressure can collapse, contributing to localised stagnant ischaemia. Again, myelographic studies with spinal stenosis demonstrate blocks to the otherwise free flow of contrast material with even modest degrees of spinal extension.

Night pain secondary to vertebral medullary metastasis may in part be due to obstruction of the basi-vertebral venous branch draining the vertebral body with secondary intra-medullary venous hypertension. Distension of the paravertebral plexus might well provoke the uncontrolled
leg and toe movement of the restless legs syndrome. In this instance, the patient is aroused from sleep by an involuntary motor equivalent of lumbar spinal root irritability.

12. **Bladder and parasympathetic disturbance**

The problems of urinary incontinence in the elderly female and of difficulty initiating micturition, poor stream, terminal dribbling, and nocturia in the elderly male present diagnostic difficulties in spinal stenosis. Clearly these symptoms may well be the result of weakening of the pelvic floor musculature or benign prostatic hypertrophy respectively. In patients with neurogenic claudication, however, it should be assumed that the bladder disturbance is the result of pressure on the second, third and fourth sacral roots until proved otherwise.

In the series of two hundred and twenty-one patients with spinal stenosis at the Nuffield Orthopaedic Centre, two females presented with incontinence of urine, and nine males presented with genito-urinary disturbance. Five of the males were treated by prostatectomy, but the other four required surgical decompression for central stenosis. In addition to difficulties initiating micturition and dribbling overflow incontinence, one male had numbness of the penis, and the other male experienced erections on walking to his claudication distance. As the leg pain subsided so did the erection. This indicated disturbance of the sacral parasympathetic outflow in the second, third and fourth sacral roots.

13. **Functional disability**

The claudication distance, presence of numbness in the legs and absence of a reflex all indicate a significant degree of pressure on the nerve roots of the cauda equina. It is self-evident that the impact these findings will have on the patient's lifestyle depends upon the patient's occupation, social and domestic circumstances, and principally upon his or her resilience and ability to cope with disability. A minor disability may cause a less resilient person to lose their job particularly when litigation is involved if the symptoms developed after an industrial injury. A more stoical person, particularly if self-employed in his own business may soldier on with a major disability.

In an attempt to monitor to what effect the symptoms and signs of spinal stenosis were interfering with an individual's lifestyle, a
A functional disability score was devised. This is shown in Table 10:14.

<table>
<thead>
<tr>
<th>SCORE</th>
<th>FUNCTIONAL DISABILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Minimal or no discomfort. Normal work or recreation</td>
</tr>
<tr>
<td>2</td>
<td>Normal work, reduced recreation. Pain once or twice a year</td>
</tr>
<tr>
<td>3</td>
<td>Lighter work or time off work. No recreation. Out of work two or three times a year</td>
</tr>
<tr>
<td>4</td>
<td>Out of work. No recreation. Independent</td>
</tr>
<tr>
<td>5</td>
<td>Inactive and housebound. Dependent on others</td>
</tr>
<tr>
<td>6</td>
<td>Confined to bed most days through leg or back pain</td>
</tr>
</tbody>
</table>

Table 10:14. The functional grading scale used to assess the functional disability of the two hundred and twenty-one patients attending the Nuffield Orthopaedic Centre with spinal stenosis.

The functional grading score of the patient was noted on first presentation and then again on the completion of treatment. The results of treatment will be presented in Chapter 21. Table 10:15 illustrates the functional grading of the patients on presentation to hospital.

<table>
<thead>
<tr>
<th>FUNCTIONAL GRADING</th>
<th>FEMALE MEDICAL</th>
<th>MALE MEDICAL</th>
<th>FEMALE SURGICAL</th>
<th>MALE SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>6</td>
<td>33</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>53</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>24</td>
<td>50</td>
<td>53</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>12</td>
<td>17</td>
<td>24</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 10:15. The functional grading score of patients with spinal stenosis at first presentation to hospital as a percentage of each sex/treatment group.

It can be seen from table 10:15 that the majority of patients belong to the middle four functional grades. There is a higher proportion of patients (seventy-two per cent) in grades 4 and 5 amongst the surgical groups than amongst the medically treated groups (forty-five per cent).
14. The pain drawing

Many patients have difficulty describing the symptoms of neurogenic claudication, and the distribution of pain. On the other hand, patients with personality disorders or seeking compensation or with financial, sexual or family problems, may describe vividly pains in the legs on exercise. A useful guide to help the clinician distinguish between these two groups of patients with very different needs is used by the Problem Back Treatment Centre at the Rancho Los Amigos Hospital (Ransford 1976). The patient is asked to draw the site and indicate the quality of the pain in his legs and back. The drawing is then evaluated according to the following four criteria (Fig. 10:4).

Hypothesis for evaluating pain drawings

A patient with psychological difficulties may reveal this by:

1. On body drawings showing poor anatomic localisation. Bilateral pain is not weighted.
   a) total leg pain
   b) lateral whole leg pain (trochanteric area and lateral thigh allowed)
   c) circumferential thigh pain
   d) bilateral anterior tibial pain (unilateral allowed)
   e) circumferential foot pain
   f) bilateral foot pain
   g) use of all four modalities suggested in instructions. The patient is unlikely to have "burning areas", "stabbing pain", "pins and needles", and "numbness" all together.

2. Drawings showing "expansion" or "magnification" of pain (may also represent unrelated symptomatology; bilateral pain is not weighted)
   a) back pain radiating to iliac crest, groin, or anterior perineum: coccygeal pain is allowed
   b) anterior knee pain
   c) anterior ankle pain
   d) pain drawn outside the outline: this is a particularly good indication of magnification.

3. "I particularly hurt here" indicators:
   Some patients needing to make sure the physician is fully aware of the extent of symptoms may:
   a) add explanatory notes
Figure 10.4. The pain drawings of two patients with spinal stenosis taken from the series. Both drawings strongly suggest an organic disorder rather than a psychological disturbance. Patients with a psychological disorder usually produce bizarre, non-anatomical pain drawings.
b) circle painful areas

c) draw lines to demarcate painful areas

d) use arrows

e) go to excessive detail in demonstrating the pain areas

4. "Look how bad I am" indicators:

Additional painful areas in the trunk, head, neck, or upper extremities drawn in. Tendency toward total body pain.

The influence of the patient's psychological make-up on the results of surgery and treatment must be considered. In 1975 Wiltse and Rocchio published a double-blind study indicating that the hypochondriasis and hysteria scores on the Minnesota Multiphasic Personality Inventory questionnaire (MMPI) were overall the best prognostic indicators to the outcome of treatment for disc disease (Wiltse 1975). If the hysteria score was in excess of eighty-five then only ten per cent of these patients had a good result from treatment. However, if the score was less than fifty-four, ninety per cent of patients ultimately had a good result. Sternbach showed that a successful outcome to treatment can reduce the hypochondriasis score (Sternbach 1975). Alternatively, certain stressful situations can temporarily raise the profile.

The orthopaedic surgeon must consider the psychological make-up of the patient when assessing the degree of disability and making recommendations for treatment. Clearly this will, to some extent, become apparent during the clinical examination of the patient but can be assisted greatly by the use of the "pain drawing".

Of the two hundred and twenty-one patients with spinal stenosis, comments appeared more frequently in the case notes about the patient's psycho-social background and appearance than would occur in the case notes of patients considered for routine total hip replacement (Tables 10:16 and 10:17). These comments such as "difficult personality" and "demanding" may sometimes be expressions of the clinicians exasperation with the patient.
Table 10:16. Comments which appeared in the case notes of patients with spinal stenosis treated conservatively. The personality of the patient may significantly hinder the clinician in eliciting a good history and such a patient with spinal stenosis may remain undiagnosed because insufficient time has been spent with this "difficult" patient in a busy out-patient department.

<table>
<thead>
<tr>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor historian</td>
<td>Difficult personality</td>
</tr>
<tr>
<td>Imprisoned in Iran</td>
<td>Divorcing</td>
</tr>
<tr>
<td>Anxious</td>
<td>Anxious and depressed (6)</td>
</tr>
<tr>
<td>Depressed</td>
<td>Embellished history</td>
</tr>
<tr>
<td>Cancer phobia</td>
<td></td>
</tr>
</tbody>
</table>

Table 10:17. Comments appearing in the case notes of those patients with spinal stenosis treated surgically. Before considering elective surgery in spinal stenosis, the clinician is well advised to obtain a psychiatrist's and social worker's opinion on those patients he finds difficult to handle. In most spinal centres in North America this would be routine (Wiltse 1975).

<table>
<thead>
<tr>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strong functional element</td>
<td>Unwell and depressed</td>
</tr>
<tr>
<td>Depressed</td>
<td>Miserable</td>
</tr>
<tr>
<td>Large functional element</td>
<td>Large overlay</td>
</tr>
<tr>
<td></td>
<td>Demanding</td>
</tr>
<tr>
<td></td>
<td>Multi-symptomatic</td>
</tr>
</tbody>
</table>

THE PHYSICAL SIGNS OF SPINAL STENOSIS

In the early stages of spinal stenosis examination of the patient at rest discloses few neurological abnormalities, and this disparity between symptoms and signs has diagnostic value. The Achilles reflex is seldom normal, but its absence or depression in elderly patients lacks great significance. Sensory abnormalities may be found in lower lumbar and sacral dermatomes, but are said to be more often absent. A few patients will resist hyperextension of the lumbar spine, but most have normal spinal mobility. The Lasegue or straight leg raising manoeuvre is described as being almost invariably normal. Re-examination at the height of symptoms for instance after walking, may reveal motor reflex and sensory abnormalities not previously seen.
Wilson reported objective weakness in only thirty-one per cent of patients, and sensory abnormalities occurring with the same frequency (Wilson 1971). He found that depression of the Achilles reflex was the single most consistent sign and was depressed or absent bilaterally in forty-six per cent of patients and unilaterally in thirty-one per cent of patients, giving a total of seventy-seven per cent abnormal Achilles reflex.

It should be noted that patients frequently have abnormalities of the peripheral pulses and may have already had vascular surgical procedures performed prior to a diagnosis of cauda equina compression. Similarly it is important to recognise that patients with lumbar spinal stenosis may well have co-existent cervical spinal stenosis in association with cervical spondylosis.

Flattening of the lumbar lordosis occurs in sixty-three per cent of patients with stenosis, compared with seventy-one per cent of those with disc prolapse (Paine 1976). Scoliosis is less frequent in stenosis than disc prolapse occurring in twenty-eight per cent of patients with spinal stenosis, compared with thirty-eight per cent of patients with a prolonged intervertebral disc. Paine found that seventy-five per cent of patients with prolapsed intervertebral disc had spinal movements reduced to half or less, whereas seventy-five per cent of those with spinal stenosis had more spinal movement greater than fifty per cent of the normal range.

Although the ankle reflex is usually the one most seriously compromised the patellar reflex may be involved to a lesser extent. When weakness is present it is usually most evident in the distal musculature (Epstein 1962). Usually it is difficult to detect, but sometimes it may be so pronounced as to result in a drop foot in severely affected patients.

Many of the above observations were confirmed by a detailed review of the physical findings observed in two hundred and twenty-one patients with spinal stenosis treated at the Nuffield Orthopaedic Centre between 1976 and 1986. The following analysis was made of their physical signs:

(a) **Spinal Movement**

Only four per cent of patients were considered to have an increased range of spinal mobility, unlike Paine's observations (Paine 1976). The majority of patients had either a normal range of spinal flexion (forty per cent) or a decreased range of flexion (fifty-six per cent). Spinal extension, however, was invariably limited and this was a very useful sign. Lateral flexion also was limited in sixteen per cent of patients. Eight per cent of patients were noted to have a scoliosis, usually structural with vertebral
rotation. Table 10:18 illustrates the findings in more detail, analysed by sex and treatment groups.

<table>
<thead>
<tr>
<th>SPINAL FLEXION</th>
<th>INCREASED</th>
<th>NORMAL</th>
<th>DECREASED</th>
<th>SCOLIOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females managed medically (N=63)</td>
<td>12%</td>
<td>35%</td>
<td>53%</td>
<td>3%</td>
</tr>
<tr>
<td>Males managed medically (N=86)</td>
<td>0%</td>
<td>53%</td>
<td>47%</td>
<td>10%</td>
</tr>
<tr>
<td>Females managed surgically (N=25)</td>
<td>0%</td>
<td>40%</td>
<td>40%</td>
<td>10%</td>
</tr>
<tr>
<td>Males managed surgically (N=47)</td>
<td>4%</td>
<td>30%</td>
<td>66%</td>
<td>8%</td>
</tr>
</tbody>
</table>

Table 10:18. The spinal mobility in flexion expressed by sex and management group as a percentage of the total number (N) in that group.

Spinal extension was almost invariably reduced in patients with spinal stenosis and if spinal extension produced sciatic pain below the knees this was strongly suggestive of the diagnosis. Lateral flexion was also reduced significantly in some patients and when lateral stenosis was present, lateral flexion appeared to exacerbate the leg symptoms.

Table 10:19 summarises the finding expressed in Table 10:18, to allow comparison of conservatively and surgically treated groups.

<table>
<thead>
<tr>
<th>SPINAL FLEXION</th>
<th>INCREASED</th>
<th>NORMAL</th>
<th>DECREASED</th>
<th>SCOLIOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal stenosis patients managed medically (N=149)</td>
<td>6%</td>
<td>44%</td>
<td>50%</td>
<td>6%</td>
</tr>
<tr>
<td>Spinal stenosis patients managed surgically (N=72)</td>
<td>2%</td>
<td>35%</td>
<td>63%</td>
<td>9%</td>
</tr>
<tr>
<td>All patients with spinal stenosis (N = 221)</td>
<td>4%</td>
<td>40%</td>
<td>56%</td>
<td>8%</td>
</tr>
</tbody>
</table>

Table 10:19. Spinal mobility (flexion) of patients with spinal stenosis, grouped according to method of management.
(b) **Spinal Tenderness**

Spinal tenderness to either light palpation or firm percussion was not a prominent feature of these patients with spinal stenosis. Only ten per cent of patients managed conservatively and twenty-five per cent of patients managed surgically were found to have spinal tenderness. When present, tenderness was usually experienced at the fourth and fifth lumbar and first sacral levels and interspaces, but occasionally extended up to the first lumbar vertebra. Table 10:20 illustrates these findings.

<table>
<thead>
<tr>
<th></th>
<th>FEMALES</th>
<th>MALES</th>
<th>OVERALL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSERVATIVE GROUP</td>
<td>10%</td>
<td>9%</td>
<td>10%</td>
</tr>
<tr>
<td>SURGICAL GROUP</td>
<td>28%</td>
<td>23%</td>
<td>25%</td>
</tr>
</tbody>
</table>

Table 10:20. The proportion of patients found to have spinal tenderness on examination, expressed as a percentage of the whole group.

It is possible that a higher proportion of patients in the surgical group experienced spinal tenderness for a number of reasons. Firstly, the patients in the surgical group tended to have more severe stenosis; secondly there was a higher proportion of females with degenerative spondylolisthesis and males with combined degenerative and disc herniation types of stenosis in the surgical group; and thirdly, because the surgical group included a number of patients who had had previous surgery.

(c) **Straight Leg Raise Test**

The degree to which the straight leg could be raised varied considerably in the Nuffield patients, with two major groupings: firstly those patients who were only able to achieve 40° or less and those patients who could achieve 90°. Table 10:21 summarises these results.
Table 10:21. The degree to which patients with spinal stenosis could raise a straight leg, expressed as a percentage for each group according to sex and treatment method.

When the straight leg raise test was restricted, this was the result either of a positive sciatic stretch sign, or back pain, or generalised muscle tightness. The proportion of patients with a positive sciatic stretch test (Lasegue's sign) was evenly distributed across the range of straight leg raise from 40° to 90°. The vast majority of patients however (seventy to eighty per cent) had a negative Lasegue's sign confirming the observations of most published series. Table 10:22 illustrates these findings:

<table>
<thead>
<tr>
<th>STRAIGHT LEG RAISE</th>
<th>FEMALES MEDICAL</th>
<th>MALES MEDICAL</th>
<th>FEMALES SURGICAL</th>
<th>MALES SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40°</td>
<td>15</td>
<td>20</td>
<td>33</td>
<td>23</td>
</tr>
<tr>
<td>50°</td>
<td>3</td>
<td>7</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>60°</td>
<td>6</td>
<td>4</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>70°</td>
<td>15</td>
<td>4</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>80°</td>
<td>12</td>
<td>39</td>
<td>14</td>
<td>8</td>
</tr>
<tr>
<td>90°</td>
<td>49</td>
<td>26</td>
<td>39</td>
<td>41</td>
</tr>
</tbody>
</table>

Table 10:22. The proportion of patients with a positive or negative Lasegue's sign expressed as a percentage by sex and management group.

It is probable that the marginally increased proportion of positive Lasegue's signs in the males managed surgically is because of the higher frequency of combined degenerative/disc herniation type of stenosis in this group. One of the explanations of a positive Lasegue's sign is that it indicates a degree of sciatic nerve root irritability compatible with the chemical radiculitis adjacent to a disc herniation.
(d) **Sensory Disturbance**

Approximately one-third of all patients with spinal stenosis were found to have reduced sensation when tested for pin prick and light touch. Sensory loss or reduction occurred most frequently in the fifth lumbar and first sacral dermatomes. Table 10:23 summarises these findings.

<table>
<thead>
<tr>
<th>DERMATOME</th>
<th>L1</th>
<th>L2</th>
<th>L3</th>
<th>L4</th>
<th>L5</th>
<th>S1</th>
<th>SADDLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female: Conservative</td>
<td>0</td>
<td>6</td>
<td>6</td>
<td>29</td>
<td>41</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Male: Conservative</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>19</td>
<td>39</td>
<td>42</td>
<td>0</td>
</tr>
<tr>
<td>Female: Surgical</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>54</td>
<td>20</td>
<td>13</td>
</tr>
<tr>
<td>Male: Surgical</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>55</td>
<td>37</td>
<td>8</td>
</tr>
</tbody>
</table>

Table 10:23. Sensory loss or reduction indicated by dermatome and expressed as a percentage of the total number of patients in each sex/management group with sensory disturbance.

It can be seen that in the surgical groups a significant proportion of patients presented with saddle anaesthesia. These were managed by emergency decompression.

Not all patients with spinal stenosis were found to have sensory disturbance and Table 10:24 indicates the proportion in each group with sensory loss.

<table>
<thead>
<tr>
<th>CONSERVATIVE</th>
<th>SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEMALES</td>
<td>30%</td>
</tr>
<tr>
<td>MALES</td>
<td>25%</td>
</tr>
</tbody>
</table>

Table 10:24. The proportion of patients with objective sensory disturbance expressed as a percentage of the sex/management group.

It is not surprising that a greater proportion of patients in the surgical group presented with sensory disturbance since on the whole these patients had more severe stenosis.

(e) **Motor Weakness**

Approximately one quarter of the patients with spinal stenosis managed conservatively were found to have a motor deficit, whilst one half of those managed surgically were found, on detailed examination, to have motor weakness (Table 10:25).
Motor weakness expressed as a percentage of the entire sex/management group. Exercise-induced weakness was noted only in the surgical group and is expressed as a percentage of this group according to sex.

Contrary to a number of reports suggesting that motor weakness is rarely found, around half the patients in the surgical group were found to have some weakness. To elicit exercise-induced weakness, the patients were exercised to their maximum claudication distance and then re-examined. Weakness often involved multiple segmental levels and more than one joint. Table 10:26 summarises the levels of weakness.

<table>
<thead>
<tr>
<th></th>
<th>CONSERVATIVE GROUP</th>
<th>SURGICAL GROUP</th>
<th>EXERCISE INDUCED WEAKNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEMALE</td>
<td>28</td>
<td>55</td>
<td>11</td>
</tr>
<tr>
<td>MALE</td>
<td>25</td>
<td>46</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 10:25.

<table>
<thead>
<tr>
<th></th>
<th>FEMALE MEDICAL</th>
<th>MALE MEDICAL</th>
<th>FEMALE SURGICAL</th>
<th>MALE SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIP FLEXION</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>EXTENSION</td>
<td>4</td>
<td>0</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>KNEE FLEXION</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>EXTENSION</td>
<td>8</td>
<td>21</td>
<td>6</td>
<td>14</td>
</tr>
<tr>
<td>ANKLE DORSIFLEXION</td>
<td>19</td>
<td>26</td>
<td>19</td>
<td>7</td>
</tr>
<tr>
<td>PLANTAR FLEXION</td>
<td>0</td>
<td>5</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>INVERSION</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>EVERSION</td>
<td>15</td>
<td>16</td>
<td>19</td>
<td>10</td>
</tr>
<tr>
<td>FOOT E.H.L.</td>
<td>50</td>
<td>32</td>
<td>25</td>
<td>20</td>
</tr>
<tr>
<td>F.H.L.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 10:26. The distribution of motor weakness in each of the sex/management groups, expressed as a percentage of the total motor weakness of each group.

In some patients a solitary muscle weakness is noted. For instance, in one patient the only evidence of motor involvement was wasting of gluteus maximus on one side. In another patient the presenting feature was a dropped foot. In other patients however weakness was extensive and could involve the hip, knee, ankle, and foot. Patients treated surgically tended to have not only more severe weakness but also more extensive weakness, sometimes bilateral, and only occasionally requiring to be unmasked by
exercise. Table 10:27 shows the frequency with which multiple levels were involved.

<table>
<thead>
<tr>
<th>CONSERVATIVE GROUP</th>
<th>SURGICAL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEMALE</td>
<td>8</td>
</tr>
<tr>
<td>MALE</td>
<td>16</td>
</tr>
</tbody>
</table>

Table 10:27. The proportion of patients in whom motor weakness involved multiple segments and more than one joint, expressed as a percentage of the total number of patients in each group with motor weakness.

(f) Reflex Anomalies

Deep tendon reflexes are either present and normal, absent even with reinforcement, or diminished. Diminished indicates that the reflex is either reduced in amplitude or is slow in response. When a normal reflex is present on one side, this is easily detectable, but when the reflex is diminished or absent on the other side some degree of subjective interpretation is required. This, however, is usually not difficult, and by repeating the reflex a number of times with the patient as relaxed as possible or using reinforcement of the reflex, it is possible to obtain one of the most objective monitors of nerve root dysfunction. Tables 10:28, 10:29, and 10:30 indicate the reflex findings in this group of two hundred and twenty-one patients with spinal stenosis.

<table>
<thead>
<tr>
<th>CONSERVATIVE GROUP</th>
<th>SURGICAL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEMALE</td>
<td>42</td>
</tr>
<tr>
<td>MALE</td>
<td>50</td>
</tr>
<tr>
<td>COMBINED</td>
<td>46</td>
</tr>
</tbody>
</table>

Table 10:28. Summary of the proportion of patients in each group with reflex anomalies, expressed as a percentage of the total number of patients in each group.

Some patients had a solitary reflex abnormality, most commonly involving the ankle reflex, but other patients had multiple reflex abnormalities. Overall, forty-six per cent of patients managed conservatively had a reflex
abnormality, and fifty-nine per cent of patients managed surgically had some reflex anomaly.

<table>
<thead>
<tr>
<th></th>
<th>FEMALE MEDICAL</th>
<th>MALE MEDICAL</th>
<th>FEMALE SURGICAL</th>
<th>MALE SURGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>KNEE: diminished unilateral</td>
<td>15</td>
<td>15</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>KNEE: diminished bilateral</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>KNEE: absent unilateral</td>
<td>4</td>
<td>4</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>KNEE: absent bilateral</td>
<td>11</td>
<td>4</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>ANKLE: diminished unilateral</td>
<td>15</td>
<td>35</td>
<td>27</td>
<td>13</td>
</tr>
<tr>
<td>ANKLE: diminished bilateral</td>
<td>0</td>
<td>4</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>ANKLE: absent unilateral</td>
<td>33</td>
<td>8</td>
<td>27</td>
<td>27</td>
</tr>
<tr>
<td>ANKLE: absent bilateral</td>
<td>22</td>
<td>38</td>
<td>19</td>
<td>27</td>
</tr>
<tr>
<td>ANAL: reflex absent</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 10:29. Details of reflex abnormalities in the four groups of patients, with frequency expressed as a percentage of total number of patients with reflex anomalies in each group. Up to twenty per cent of patients had more than one reflex abnormality.

It can be seen that bilateral diminished knee reflexes were not diagnosed because of the difficulty clinically in making this observation. The findings expressed in Table 10:29 are summarised in Table 10:30 and expressed as a percentage of the total number of patients in each therapeutic group.

<table>
<thead>
<tr>
<th></th>
<th>CONSERVATIVE GROUP</th>
<th>SURGICAL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>KNEE: reflex diminished</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>KNEE: reflex absent</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>ANKLE: reflex diminished</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>ANKLE: reflex absent</td>
<td>25</td>
<td>34</td>
</tr>
<tr>
<td>ANAL: reflex absent</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 10:30. Reflex abnormalities expressed according to treatment group as a percentage of the total number of patients in each group.

It is clear that an absent ankle reflex is the most commonly found reflex abnormality in both groups of patients, being found in one-quarter of patients treated conservatively and one-third of patients managed
surgically. In subsequent chapters I shall describe the effect of surgical
decompression on these reflex (and motor and sensory) changes
(Chapter 21).

The "Simian Stance": A Sign of Spinal Stenosis

Patients with lumbar spinal stenosis may relieve compression on nerve
roots by adopting a stooped posture including flexion of the hips and knees
as well as the lower back. The resultant simian stance, which resembles
the bipedal gait of anthropod apes (Fig. 10:5), can be a valuable clue to
diagnosis (Simkin 1982).

Postural clues may be most helpful in interpreting such patients.
Walking downhill by increasing the lumbar extension may exacerbate
symptoms, whereas walking uphill may relieve them. It is useful to note
the tendency of such patients to obtain relief by leaning forwards and
resting their hands on their knees in a "leap-frog position". But a most
helpful sign is the simian posture which is characteristically adopted
both in the standing and walking attitudes.

The great apes maintain an erect posture with relative ease by means
of specially adapted hamstrings and quadriceps muscles. Man is not so
specially adapted for this posture, and in adopting a flexed knee stance
he accepts an inefficient posture requiring constant input of muscular
energy to stave off collapse. In spinal stenosis this disadvantage is offset
by the advantage of functional decompression of the lumbar spinal nerve
roots. When the adaptation is successful, nerve root function will be
preserved and neurological deficits either gross or electrophysiological may
be avoided (Simkin 1982). A simian stance may thus precede other
findings as a sensitive sign of early spinal stenosis.

The "Bicycle Sign" of Spinal Stenosis

Dixon reported an elderly woman with spinal stenosis due to Paget's
disease who was referred with "claudication". She could ride her
tricycle, even uphill, with no trouble, but on walking would collapse with
weak legs after about fifty yards (Dixon 1982). A much younger man, a
journalist on a local newspaper, was reported with a similar story. He
would cycle all day about his "parish" but could not walk without weakness
developing in his legs. The diagnosis was apparent from the history, and
was confirmed operatively by finding a large posterior osteophyte
Figure 10:5. The "Simian Stance". A chimpanzee stands with its knees and hips flexed. The centre of gravity passes far anterior to the hip joint and lumbar spine, and the pelvis is tilted forwards to obliterate or reverse the lumbar lordosis. This posture is normal for chimpanzees but pathological for humans and a characteristic of severe postural spinal stenosis. Fixed flexion deformity of the hip usually results in an exaggeration of the lordosis on standing.
Figure 10.6: The "Bicycle Sign". A patient with spinal stenosis whose walking distance is considerably diminished by neurogenic claudication may maintain independence and mobility by the fact that their cycling distance is unrestricted.
compressing the cauda equina. We cycle with flexed lumbar spines but normally walk with a lumbar lordosis (Fig. 10:6).

The "Shoe Lace" Sign

Not all people tie up their shoe laces in the street because the lace is undone and they wish to avoid tripping over it. Patients with spinal stenosis who have reached their claudication distance in a public place and do not wish to appear conspicuous and can find nowhere to sit down, may squat or bend over, deliberately untie their shoe lace and then proceed rather slowly to tie it up again. This may bring about relief of their leg pain since whilst they are bending over or squatting to tie the shoe lace the lumbar spine is flexed (Fig. 10:7) and the cauda equina functionally decompressed. Patients do not volunteer this information unless asked what they do when they are in a public place when there is nowhere to sit.

Tenderness of Motor Points as an Indicator of Nerve Root Involvement

The motor points of certain muscles are frequently tender in patients with low back pain. The motor points are fixed anatomical sites at which the major condensations of motor end plates occur, and vary only slightly from person to person (Fig. 10:8). Electromyography in patients with tender motor points has shown evidence of neuropathy (Gunn 1976). The degree of tenderness of the motor points may parallel the severity of symptoms of pain from day to day or even week to week. Moderately to acutely tender motor points are almost constantly found in patients with disc degeneration. The degree of tenderness and the number of tender points tend to parallel the patient's condition, and may serve as indicators to prognosis. Tenderness may be related to the irritation or degeneration of nerve fibres caused by trauma to the nerve root.

Pain and tenderness localised to the buttock and possibly attributed to gluteal bursitis or thought to be caused by sciatic nerve tenderness, may be due to tenderness of the motor point of gluteus maximum which is focal and not linear as sciatic nerve tenderness. Similarly, tenderness in the region of the trochanteric bursa may in fact be localised in the tensor fascia lata motor point, whilst tenderness thought to be due to "adductor strain" may arise from the motor points of the pectineus and adductor longus. The motor points of all the muscles of the lower limb are well described (Gunn 1976). The motor points of the paravertebral
Figure 10:7. The "Shoe Lace Sign". Not everyone stooping to tie their shoe lace in the high street has spinal stenosis, but when there is nowhere to sit down the stenotic patient may derive relief from the lumbar flexion afforded by this activity.
Figure 10.8. The anatomical location of the major motor points posteriorly in the lower limb. Tenderness at those sites may indicate nerve degeneration secondary to spinal stenosis.
muscles which are situated approximately 2.5 cm. from the midline, may also account for some paraspinal muscle tenderness in patients with stenosis.

UNUSUAL PRESENTATIONS OF SPINAL STENOSIS

1. The Neuropathic Bladder

Well recognised causes of a neuropathic bladder are disseminated sclerosis, central prolapsed lumbar intervertebral disc, and spinal tumour. A less well recognised cause of a neuropathic bladder is lumbar spinal stenosis (Sharr 1973). These patients tend to be referred to urological clinics rather than neuro-surgical or orthopaedic units. The features are initially intermittent and consist of recurrent infections nearly always associated with an atonic bladder, incontinence either overflow incontinence or total emptying, and episodes of acute retention (Sharr 1976).

The onset of urological symptoms does not always coincide with the onset of spinal symptomatology. Spinal symptoms preceded the onset of urinary troubles in half the patients reported by Sharr, whilst in half urological and spinal symptoms appeared at the same time. Two patients out of a series of seventeen had no spinal symptoms at all, although examination revealed neurological abnormalities. Considerable symptomatic improvement followed lumbar laminectomy in thirteen of the seventeen patients (Sharr 1973). In a later paper Sharr went on to describe three main groups, firstly true incontinence, secondly retention, and thirdly symptoms of urinary tract infection with bouts of dysuria, urgency and frequency. Infection was usually associated with an appreciable amount of residual urine (Sharr 1976).

Urological investigations including cystometry showed two main abnormal patterns, firstly the atonic or lower motor neurone bladder, and secondly the uninhibited or hypertonic upper motor neurone bladder.

Operative findings at laminectomy consisted of hypertrophied facet joints, thickened ligamentum flavum, and thickened sclerotic laminar bone. There was often a poor correlation between the degree of narrowing shown myelographically, and that found at operation. Eighty-one per cent of the patients gave a history of backache, sometimes for as long as thirty years or more.

Post-operatively normal bladder function recovered in patients whose symptoms, often severe, had been present for many years and whose pre-
operative cystometrograms were abnormal. All but three of thirteen patients with abnormal pre-operative cystometrograms showed striking cystometrographic and clinical improvement after surgery. Twenty-one patients had normal pre-operative cystometrograms, and fifteen of these showed clinical improvement after surgery. Sensory loss was found to be an adverse factor and only two of six patients with sensory loss improved.

2. **Spinal Stenosis Presenting as Spontaneous Priapism**

Disturbance of autonomic function is an unusual feature of compression of the cauda equina. A case was reported in 1979 of a sixty-six year old man who suffered a back injury whilst working on a car assembly line in 1973 (Ravindran 1979). From 1977 onwards he experienced numbness from the groin down after walking more than two hundred yards. He also experienced spontaneous penile erections on walking, which subsided with rest. This became embarrassing for the patient who had to limit many of his outdoor activities. He had no bladder or bowel problems. There was no associated pain in the lower limbs. The lower limbs showed marginal weakness of the right hamstrings and calf muscles, and radiographs of the lumbar spine showed spondylolisthesis of the L4 on L5 of about fifteen per cent. A lumbar myelogram showed complete block at the lower level of the fourth lumbar vertebra. A lumbar laminectomy was performed, and six weeks after surgery he was totally asymptomatic, tolerating physical activity well especially walking. Penile erection and numbness of the lower limbs both related to walking had cleared up completely.

Penile erection results from parasympathetic stimulation. The parasympathetic fibres arise in the intermediate lateral cells of the second, third, and fourth sacral segments of the spinal cord, and emerge in the pelvic nerves to form perivesicular prostatic, and cavernous plexuses (Chusid 1964). These plexuses supply vasodilator fibres to the corpora cavernosa and motor fibres to the compressor urethra, ischio-cavernosus and bulbo-cavernosus muscles, all concerned with penile erection and ejaculation. The parasympathetic outflow is contained within the S2 to S4 sacral nerve roots forming part of the cauda equina.
3. Atypical Presentation Resulting in Delayed Diagnosis

The diagnosis of spinal stenosis is frequently delayed. As already indicated this is most likely the result of an elderly patient's stoicism with advancing age and tolerance of leg pain on walking and a reduced exercise tolerance for other reasons such as shortness of breath. The second reason for delay in diagnosis is that the clinician may fail to be convinced of organic pathology through the vagueness of the patient's history, and the absence of hard objective neurological abnormalities.

The third reason for the delay in diagnosis is that the patient may be thought to have some other condition. Tuberculosis of the spine has been suspected as a result of narrowing of the disc space, and the presence of a paraspinal swelling (Douglas 1981). This has given rise to the mistaken diagnosis of Pott's disease (Siegelman 1968).

When the patient presents with acute retention of urine, sclerotic vertebra, and an elevated serum acid phosphatase, the diagnosis of carcinoma of the prostate may be erroneously made. It must be borne in mind that acid phosphatase level is often raised in Paget's disease.

Occasionally the patient may be thought to have a demyelinating disease (Hartman 1966), or a peripheral neuropathy.

SUMMARY

The assessment of patients with spinal stenosis pre-operatively and post-operatively remains therefore largely subjective. Attempts to produce objective rating scales for spinal stenosis have proved unsuccessful, and as will be seen the degree of disability which the patient experiences does not always correlate with the results of the more complex investigations such as myelography and computed tomography. The successful diagnosis of patients with spinal stenosis therefore continues to demand a high level of clinical suspicion, acumen, and expertise.
CHAPTER 11

THE DIFFERENTIAL DIAGNOSIS OF SPINAL STENOSIS

INTRODUCTION

The diagnosis of spinal stenosis is usually not in doubt when all investigations including myelography and C.T. scanning have been completed. Patients who are not significantly disabled or patients in whom surgery is not contemplated are usually not subjected to full investigation, however, and the diagnosis is arrived at on clinical grounds alone. It is important for these patients that the clinician is fully aware of other conditions which may mimic spinal stenosis in their presentation. Patients may be spared invasive, expensive, and time-consuming investigations for spinal stenosis when the astute clinician detects, for instance, a bruit on auscultation of the femoral artery.

It must be borne in mind also that neurogenic claudication may co-exist with vascular claudication, and it was as long ago as 1911 that Dejerine described a syndrome which he termed "intermittent claudication of the spinal cord" (Dejerine 1911). He noted that both upper and lower motor neurone lesions could be caused secondary to ischaemia of the spinal cord resulting from occlusive arterial disease of the aorta which involved the spinal arteries. Patients in whom both vascular and neurogenic claudication co-exist were also reported by Blau and Logue (Blau 1961), and Verbiest (Verbiest 1955). This chapter considers some of the commoner conditions which must be considered in the differential diagnosis of spinal stenosis (Table 11:1).

TABLE 11:1 CONDITIONS WHICH MUST BE INCLUDED IN THE DIFFERENTIAL DIAGNOSIS OF SPINAL STENOSIS

- Aorto-iliac occlusive disease
- Posterior facet syndrome
- Sacro-iliac syndrome
- Piriformis syndrome
- Herniation of the nucleus pulposus
- Cervical spondylosis with stenosis
- Peripheral neuropathy
- Prostatism
- Paget's disease and fluorosis
- Anterior tibial syndrome
- Neoplasia
AORTO-ILIAC OCCLUSIVE DISEASE

Patients with aorto-iliac occlusive disease describe painful sensations in the area of the hips, thighs and possibly buttocks, which may progress into the region of the calf, or which can begin in the calf and extend proximally. This comes on after walking a relatively constant distance. It is described as an aching or squeezing or cramp-like sensation which becomes more pronounced if exertion continues. If the patient continues walking he may experience paraesthesia or a sensation of weakness. He usually obtains relief by standing still for a few minutes. Sitting or lying down generally is not necessary to obtain relief.

The symptoms are aggravated by walking uphill, and walking at a slower pace delays the onset. The peripheral pulses may be normal when the patient is at rest, but often disappear or diminish after exercise (Fig.11:1). In addition exercise may produce loud bruits over the lower portion of the abdominal aorta and over the femoral arteries. Impotence is a common complaint. Reflex changes are unusual (Kavanaugh 1968).

The neurogenic claudication of spinal stenosis differs from these features in a variety of ways. The claudication distance may be variable and is influenced by the patient's posture. It may, for instance, result from prolonged standing or simply by straightening the back. This may occur either whilst lying in bed or standing or walking a few yards. It may come on whilst walking downstairs or down an incline, but not whilst climbing stairs, and walking distance may actually be increased by walking uphill.

The sensations described in neurogenic claudication are those of numbness or "tingling" and later weakness in co-ordination and "clumsiness" (Fig. 11:2). The pain is less of a feature than in vascular claudication. A saddle distribution of dysaesthesia is frequently experienced. Coughing and sneezing tends not to increase pain, and the straight leg raise manoeuvre frequently does not produce distress. The patient with neurogenic claudication must sit down or even lie down to obtain relief. Although standing still may be of some help on occasions, it may sometimes be an aggravating factor. At times the patient stoops forward to obtain relief. It may take as long as twenty or thirty minutes before relief is obtained. A comparison of the findings in arterial insufficiency and neurogenic claudication prepared by Wilson is shown in Figure 11:3 (Wilson 1971).
The presence of back pain and the finding of degenerative disease in the lumbar spine, often advanced, is extremely common and may not, in fact, be the underlying cause of the patient's symptoms of claudication. In this case, the patient's peripheral pulses were absent.
### Differential Diagnosis of Symptoms

<table>
<thead>
<tr>
<th></th>
<th>Spinal Stenosis</th>
<th>Prolapsed Intervertebral Disc</th>
<th>Peripheral Vascular Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
<td>Insidious</td>
<td>Rapid</td>
<td>Slow</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>Over 50</td>
<td>30 - 50</td>
<td>Over 60</td>
</tr>
<tr>
<td><strong>Precipitating Factor</strong></td>
<td>Fall or lifting</td>
<td>Lifting or Bending</td>
<td>Smoking - previous MI or CVA</td>
</tr>
<tr>
<td><strong>Leg Pain</strong></td>
<td>Neurogenic - numbness</td>
<td>Neurogenic - sharp shooting</td>
<td>Cramping</td>
</tr>
<tr>
<td><strong>Character</strong></td>
<td>aching</td>
<td>shooting</td>
<td>Dull ache</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>Sciatic distribution often</td>
<td>Sciatic distribution often</td>
<td>Calf or buttock</td>
</tr>
<tr>
<td><strong>Induced By</strong></td>
<td>Standing - postural : severe</td>
<td>Standing, walking, sitting : severe</td>
<td>Walking or other exercise</td>
</tr>
<tr>
<td></td>
<td>Walking - ischaemic : mild</td>
<td>Bending, lifting : mild</td>
<td></td>
</tr>
<tr>
<td><strong>Relieved By</strong></td>
<td>Sitting or squatting</td>
<td>Lying flat on boards</td>
<td></td>
</tr>
<tr>
<td><strong>&quot;March&quot;</strong></td>
<td>Lying in foetal position</td>
<td>Usually down</td>
<td></td>
</tr>
<tr>
<td><strong>Sitting</strong></td>
<td>Relieves leg pain</td>
<td>Aggravates leg pain</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Cycling</strong></td>
<td>Relieves leg pain</td>
<td>Aggravates leg pain</td>
<td>No difference</td>
</tr>
<tr>
<td><strong>Lateralisation</strong></td>
<td>Unilateral or bilateral</td>
<td>Usually unilateral</td>
<td>Aggravates leg pain</td>
</tr>
<tr>
<td><strong>Back Pain</strong></td>
<td>Usually present</td>
<td>Present or absent</td>
<td>Unilateral or bilateral</td>
</tr>
<tr>
<td><strong>Night Pain</strong></td>
<td>Present in 20%</td>
<td>Absent</td>
<td>Present or absent (non-contributory)</td>
</tr>
<tr>
<td><strong>Weakness</strong></td>
<td>May appear with exercise or present at rest when severe</td>
<td>Present at rest</td>
<td>Present in feet in severe ischemia</td>
</tr>
<tr>
<td><strong>Bladder and/or Bowel Disturbance</strong></td>
<td>Present when severe</td>
<td>Present when central disc prolapse</td>
<td>Absent</td>
</tr>
</tbody>
</table>

Figure 11.2. A comparison of the symptoms in the three conditions most often included in a differential diagnosis (Nixon 1987).
<table>
<thead>
<tr>
<th>Findings</th>
<th>Arterial Insufficiency</th>
<th>Neurogenic Claudication: Ischaemic and Postural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain: induced by:</td>
<td>characteristic muscular contraction</td>
<td>may be absent muscular contraction</td>
</tr>
<tr>
<td>location:</td>
<td>muscular contraction excised muscles</td>
<td>lumbo-sacral or sciatic</td>
</tr>
<tr>
<td>&quot;march&quot;:</td>
<td>absent cramping</td>
<td>up or down</td>
</tr>
<tr>
<td>character:</td>
<td>rest (standing)</td>
<td>dysaesthetic</td>
</tr>
<tr>
<td>relief by:</td>
<td></td>
<td>rest (sitting)</td>
</tr>
<tr>
<td>Motor deficit:</td>
<td>rare: during walking exercised muscles cramp and become tight</td>
<td>mild, variable: during walking appearance or exaggeration of motor and sensory deficits</td>
</tr>
<tr>
<td>Sensory deficit:</td>
<td>rare</td>
<td>mild, variable</td>
</tr>
<tr>
<td>Lasegue sign:</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Lumbar puncture:</td>
<td>normal</td>
<td>may be difficult, demonstrate block, CSF protein elevated</td>
</tr>
<tr>
<td>Pulse:</td>
<td>either femoral or distal pulse decreased; peripheral pulse may decrease during walking</td>
<td>normal; peripheral pulse unaffected during walking</td>
</tr>
<tr>
<td>Arterial murmur:</td>
<td>proximal (aorto-iliac)</td>
<td>absent</td>
</tr>
<tr>
<td>Plain X-Ray films:</td>
<td>arterial calcification</td>
<td>abnormal</td>
</tr>
<tr>
<td>Aortography:</td>
<td>diagnostic</td>
<td>normal</td>
</tr>
<tr>
<td>Myelography:</td>
<td>normal</td>
<td>diagnostic</td>
</tr>
</tbody>
</table>

Wilson 1971

Figure 11:3. The distinction between neurogenic and vascular claudication is frequently clarified by a detailed history and examination, without resource to further investigations.
POSTERIOR FACET SYNDROME

Farfan pointed out that the intervertebral joint consists of a "three joint complex" (Farfan 1973). These three joints are inter-dependent so that when the facet joints are degenerate and painful the intervertebral disc is also affected, and when the disc is degenerate and diseased the facet joints are also involved. It is therefore rather arbitrary to single out the facet joints as the sole cause of a syndrome responsible for pain in the back radiating down the legs. Also it is not surprising that in an attempt to obtain more precise anatomical location for low back pain and sciatica, it appears that "several quite distinct lesions commonly yield much the same symptom complex" (Kirkaldy-Willis 1979).

The posterior facet syndrome is said to be associated with limitation of spinal movement and an abnormal pattern of muscle contraction during lateral bending (Mooney 1976). Lateral bending in extension often causes most pain, but there is little loss of straight leg raising. Manipulative treatment over seven to ten days may establish the diagnosis. Alternatively an injection of Marcain into the facet joints at one or more levels as described by Mooney and Robertson often reduces or abolishes pain at least temporarily. I have already described (vide supra) how imprecise this injection usually is. It is probably no more specific than spinal manipulation, and yet this diagnosis of pain arising from osteoarthritic facet joints and radiating down the leg remains very probably as a definite entity.

SACRO-ILIAC SYNDROME

The patient quite often does not complain of low back pain. The presentation is of buttock pain, trochanteric pain and pain in the posterior thigh. This pain may radiate as far as the ankle, and rarely sensory changes are described (Norman G.F. 1956). The sacro-iliac joint may be the site of infection, with sometimes unusual organisms such as salmonella and brucella. It may be the first site of involvement in patients with ankylosing spondylitis, and it may be the site of chronic strain in sportsmen or heavy manual labourers. Useful clinical signs for sacro-iliac joint involvement are the "pump handle test" in which the patient lies supine on the examining couch and with the hip and knee flexed 90°, the clinician forces the hip joint into extreme adduction in flexion exerting a rotational torque on the pelvic ring; and Gaenslen's sign in which the patient holds
the thigh against the chest on one side whilst the clinician allows the other leg to hang off the side of the examining couch, thereby applying a torque to the pelvis.

PIRIFORMIS SYNDROME

In this condition external rotation of the hip against resistance produces pain. On rectal examination the piriformis muscle is found to be exquisitely tender. Injection of the muscle directly is done with one finger in the rectum over the tender muscle between the sacrum and the ischial spine. A long needle is introduced from the back midway between the lateral aspect of the sacrum and the greater trochanter. Two to three millilitres of Marcain is then introduced. When the piriformis muscle is involved striking relief of pain is obtained usually within ten minutes of the injection.

HERNIATION OF THE NUCLEUS PULPOSUS

Leg pain tends to be very much more acute in prolapse of the intervertebral disc compared with spinal stenosis. It is more frequently unilateral than spinal stenosis, although as stated the symptoms of spinal stenosis are not always bilateral. Spinal stenosis more frequently involves more than one nerve root, but a large prolapsed intervertebral disc may impinge on numerous nerve roots and in fact a central disc prolapse may be the cause of a "cauda equina syndrome".

Both disc protrusion and stenosis occur most frequently at the L4-5 and L5-S1 levels, with the fifth lumbar and first sacral nerves most frequently involved. Involvement of the third and fourth lumbar roots is more frequently seen in spinal stenosis, but may occur in prolapsed intervertebral disc. This produces anterior thigh pain, quadriceps weakness, and loss of the knee reflex. Again involvement of the L1 and L2 nerve roots is extremely uncommon in prolapsed intervertebral discs, but is more often seen in spinal stenosis (Yates 1981). This may produce lower abdominal and scrotal pain and may be confused with renal or ureteric disorders.

Patients with prolapsed intervertebral disc find on the whole that forward flexion increases pain, whereas this in fact may be the relieving posture in patients with spinal stenosis. Sitting therefore may be avoided in patients with prolapsed intervertebral disc, but is tolerated by patients with spinal stenosis (Fig. 11:4). Bed rest quite often brings about fairly
### Differential Diagnosis of Signs

<table>
<thead>
<tr>
<th>Posture</th>
<th>Spinal Stenosis</th>
<th>Prolapsed Intervertebral Disc</th>
<th>Peripheral Vascular Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>SITTING</td>
<td>Slipped stance</td>
<td>Loss of lordosis with sciatic scoliosis when acute</td>
<td>Normal</td>
</tr>
<tr>
<td>LYING</td>
<td>Unable to lie prone</td>
<td>Painful, tends to sit on one buttock</td>
<td>Comfortable</td>
</tr>
<tr>
<td>WALKING</td>
<td>Leans forwards, maybe supported by walking sticks</td>
<td>Comfortable if still</td>
<td>Comfortable</td>
</tr>
<tr>
<td>SPINAL MOBILITY</td>
<td>Normal flexion Reduced extension Reduced lateral flexion in lateral stenosis</td>
<td>Antalgic gait favouring pain-free side</td>
<td>Walks normally as far as claudication distance</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neurological Examination: Sensory</th>
<th>Sensory deficit appears or increases with exercise or present at rest when severe.</th>
<th>Sensory deficit present at rest dermatomal distribution</th>
<th>Usually no sensory deficit. Peripheral neuropathy (stocking distribution) of diabetes sometimes present</th>
</tr>
</thead>
<tbody>
<tr>
<td>MOTOR</td>
<td>Motor deficit appears or increases with exercise or present at rest when severe.</td>
<td>Myotomal distribution at rest</td>
<td>Rare: muscle may cramp and tighten on exercise.</td>
</tr>
<tr>
<td>Reflexes</td>
<td>Absent or diminished ankle reflex is most consistent finding.</td>
<td>May be diminished or lost ankle reflex.</td>
<td>Normal</td>
</tr>
<tr>
<td>LASÈGUE’S SIGN</td>
<td>Negative</td>
<td>Positive: crossed when severe</td>
<td>Negative</td>
</tr>
<tr>
<td>Vascular Examination: Peripheral Pulses</td>
<td>Normal</td>
<td>Normal</td>
<td>Absent or diminished. May disappear with exercise.</td>
</tr>
<tr>
<td>BRUITS</td>
<td>None</td>
<td>None</td>
<td>Present: aortic, femoral or popliteal</td>
</tr>
</tbody>
</table>

| General Features | Neuropathic ulcers on feet. May have co-existent vascular disease in this age group. | No ulcers | Trophic ulcers on feet, cardiac failure, arcosenilis, diabetes, previous cerebro vascular incident. |

**Figure 11:4.** A comparison of the physical findings in the three conditions most frequently included in the differential diagnosis (Nixon 1987).
rapid relief of leg pain in patients with a resolving disc prolapse, whereas patients with spinal stenosis may be woken at night time by pain and find extension of the spine in bed uncomfortable. Patients with acute disc prolapse tend to have positive impulse pain felt in the leg on coughing or sneezing or straining at defaecation.

It must be emphasised again that spinal stenosis and prolapsed intervertebral disc may co-exist. A patient with a narrow spinal canal may be asymptomatic until an unaccustomed bout of exercise or a lifting incident causes disc bulging and precipitates the spinal stenosis syndrome. The division between these two conditions is not black and white, but represents a broad grey zone.

CERVICAL SPONDYLOSIS WITH STENOSIS

Degenerative disease of the cervical spine frequently co-exists with degenerative disease of the lumbar spine. The clinician should always examine for evidence of an upper motor neurone lesion since cervical spinal stenosis has been associated with intermittent numbness and weakness in the legs aggravated by walking, but not by neck extension. Although the symptoms are usually characterised by paraesthesiae and incoordination rather than leg pain and weakness, a diagnosis of primary or associated cervical stenosis should be considered, especially when long tract signs or cervical radiculopathy are also present.

PERIPHERAL NEUROPATHY

The nerve roots and peripheral nerves of diabetics are probably more sensitive to ischaemia and pressure. It may be that the presence of diabetes increases the susceptibility of the cauda equina to compression in patients with spinal stenosis. Several patients have been reported who have had unusually severe weakness of the leg attributed to diabetic neuropathy, who have subsequently, as a result of radiculography, been found to have associated spinal stenosis (Yates 1981). Alternatively, patients suspected of having spinal stenosis were subsequently found to have painful weakness in the legs due to diabetic neuropathy. Other forms of neuropathies such as alcoholic neuropathy should be considered. A patient who deteriorates following spinal decompression may do so because the underlying diabetic neuropathy has progressed or the diabetes is out of control.
Conditions such as syringomyelia, lead poisoning, carcinomatous neuropathy, disseminated sclerosis, progressive muscular atrophy, and amyotrophic lateral sclerosis and rarely syphilitic amyotrophy, should be considered. Certain hereditary ataxias such as Friedreich's ataxia may present to the orthopaedic surgeon with unsteadiness on walking before the other features of intention tremor, dysarthria and nystagmus develop.

**PROSTATISM**

Retention of urine may occur as noted in Chapter 10 as a result of cauda equina compression. A patient who has tolerated back pain and neurogenic claudication for years without seeking medical advice may suddenly present to the clinician with retention and overflow incontinence. This requires urgent surgical intervention if bladder function is to be preserved.

**PAGET'S DISEASE AND FLUOROSIS**

These conditions will be considered in greater detail later. Spinal stenosis is reported in fluorosis in areas such as North Yemen where the water fluorine content is up to ten parts per million, and back pain is common.

**ANTERIOR TIBIAL SYNDROME**

Intermittent claudication may occur as a result of pre-infarctive anterior tibial syndrome (French 1982). The pain is usually quite clearly localised to one compartment, and may be associated with numbness in the toes through involvement of the deep peroneal nerve.

**NEOPLASIA**

In addition to carcinomatous neuropathy already mentioned, neurogenic claudication may be the presenting feature of the following conditions:

(a) Involvement of the lumbar vertebra most frequently by secondary malignant deposits;
(b) Involvement of the epidural space by spread from the vertebral bodies;
(c) Direct infiltration by a Hodgkin's type lymphoma occupying the posterior abdominal wall;
(d) A tumour of the cauda equina; these may all result in neurogenic claudication (Fagius 1979). Pain in the legs may be dependent upon posture, and come on with exercise. The onset of back pain with or without sciatica during recumbency most often nocturnally, compelling the patient to get up and walk around for relief, is said to be a helpful diagnostic feature (Wilson 1969), but as already described, up to twenty per cent of patients with spinal stenosis are found on careful questioning to have night pain.

The availability of technetium diphosphonate scanning, myelography, and CT scanning, now ensure that the correct diagnosis is made before operative exploration.

LABORATORY SCREENING TESTS

It goes without saying that a variety of screening tests should be performed on all patients with spinal stenosis to exclude associated conditions such as neoplastic or infective disease or metabolic bone disease. A basic minimum should consist of:

1. Erythrocyte sedimentation rate as a crude screen for inflammatory and neoplastic disease.
2. Serum concentrations of calcium phosphate and alkaline phosphatase to uncover any underlying metabolic bone disease, neoplasia, or Paget's disease.
3. Acid phosphatase in men who may have occult prostatic carcinoma.
4. Plasma and urinary proteins with electrophoresis should routinely be analysed as a screen for myelomatosis.

It is far preferrable to reach a clinical diagnosis of spinal stenosis by exclusion of other possible conditions, than to assume the patient has spinal stenosis and fail to diagnose the true cause of the patient's symptoms.

SUMMARY

The following conditions were identified during the clinical review of patients treated for spinal stenosis at the Nuffield Orthopaedic Centre (Table 11:2). Patients with these conditions were excluded leaving two hundred and twenty-one patients for further study.
Table 11:2. Spinal stenosis - patients excluded from the Nuffield Orthopaedic Centre series.

Other patients were included in the series but had co-existing conditions as outlined in Tables 11:3 and 11:4.

<table>
<thead>
<tr>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina (2)</td>
<td>Anaemia</td>
</tr>
<tr>
<td>Polymyalgia rheumatica</td>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Diabetes</td>
<td>On steroids (2)</td>
</tr>
<tr>
<td>Strokes (2)</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Obesity (2)</td>
<td>Angina</td>
</tr>
<tr>
<td></td>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td></td>
<td>Severe obesity (3)</td>
</tr>
<tr>
<td></td>
<td>Carcinoma rectum</td>
</tr>
<tr>
<td></td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Asthmatic</td>
</tr>
</tbody>
</table>

Table 11:3. Co-existing medical and surgical conditions in the Nuffield Orthopaedic Centre patients with spinal stenosis managed medically.
<table>
<thead>
<tr>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthmatic (2)</td>
<td>Malignant melanoma</td>
</tr>
<tr>
<td>On steroids</td>
<td>Obesity</td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>TIA's</td>
<td></td>
</tr>
<tr>
<td>Spastic paraparesis</td>
<td></td>
</tr>
<tr>
<td>Paget's (not spine)</td>
<td></td>
</tr>
</tbody>
</table>

Table 11:4. Co-existing medical and surgical conditions in the Nuffield Orthopaedic Centre patients with spinal stenosis managed surgically.