4

Neurodynamics

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Chapter Objectives

Neurodynamics addresses the peripheral and central nervous systems in a unique way. Historically, the nervous system has been viewed as a communicating and coordinating organ for the rest of the body. Little attention has been focused on the fact that as the body moves so the nervous system has to accommodate and adapt to the movements it paradoxically produces.

After reading this chapter you should be able to:

1 Appreciate that the nervous system moves and has many design elements that allow this to happen. This includes both the central nervous system (CNS) and the peripheral nervous system (PNS);
2 Appreciate that joint movements in one area can have quite far-reaching mechanical effects on neural tissues;
3 Understand that neurodynamics looks at the effects of movement/posture on the nervous system. This includes both movements and postures that tend to compress neural tissue

and those that tend to elongate it. Most movements and postures are likely to produce combinations of compression and elongation effects;

4 Appreciate that neural compression and elongation produce physiological effects on neural tissue;
5 Appreciate that pathological processes in tissues that surround the nervous system may have detrimental consequences on its mechanical and physiological health and that alterations in compliance and sensitivity may be a consequence;
6 Appreciate that the nervous system in general is relatively insensitive, yet if it is injured or physiologically compromised in some way it has the potential to become an extremely hypersensitive tissue and a potent source of ongoing pain;
7 Have a good understanding of possible pathophysiological processes that can underlie enhanced neural sensitivity.

Introduction

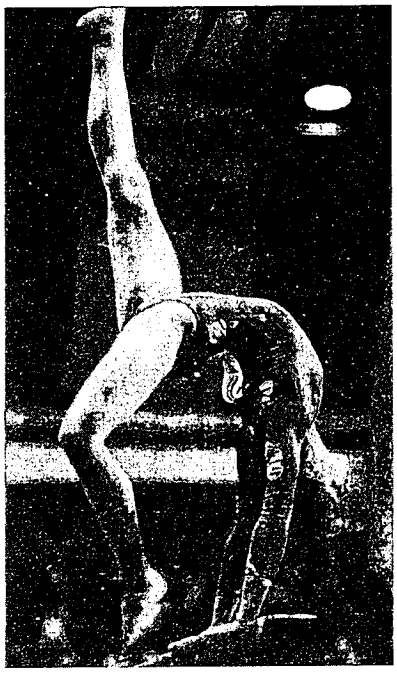
That the nervous system moves is beyond doubt. In 1978 Alf Breig published a book called *Adverse Mechanical Tension in the Central Nervous System* which presented clear evidence that nerve trunks and roots, the spinal cord and its coverings, as well as the brain, are capable of quite remarkable movement. Professor Henk Verbeist, one of the world's leading neurosurgeons, wrote in the foreword to Breig's book:

> In my opinion his work should not only be read by specialists in neuroscience and orthopaedic surgery, but also by anaesthetists whose activities regularly involve the positioning of defenceless patients, and last but not least by physiotherapists for reasons which need no further precision. (Breig, 1978)

That was back in 1978. For physiotherapy it was not until the late 1980s and early 1990s that the notion that the nervous system has subtle design features that allow it to move, was more widely appreciated (Butler and Gifford, 1989; Butler, 1991).

The prime purpose of the nervous system is one of continuous communication, whatever the situation and whatever the body happens to be doing at the time. Thoughts about its anatomical design must take into account the need for vital electrical and chemical processes to be able to continue unhindered during movement. For instance, observe the highly coordinated yet extreme movements of gymnasts and dancers and consider the need for the nervous system to be able to physically adapt (Figure 4.1).

Figure 4.1 Extreme joint movement and muscle stretch requires considerable physical adaptation of the peripheral nervous system and the neuraxis. The elongation and compressive forces that the nervous system structures have to cope with in this gymnast are impressive.



In terms of gross structure the nervous system appears as a well organized cord-like meshwork branching away from the core central nervous system (CNS) structures, the brain and spinal cord. The term 'neuraxis' (Bowsher, 1988) in place of CNS helps to focus us on this component of the nervous system from a biomechanical perspective. However, Butler (1991) introduced the idea that we should consider the nervous system as a continuum, in other words, get away from the traditional anatomical descriptive concepts of central, peripheral and autonomic nervous systems and move towards a view that the whole structure is closely linked. In this way, the nervous system is unique among organs and systems in that it has a pretty straightforward mechanical, electrical and chemical 'connectedness' (Butler, 1991). The implication is that mechanical, electrical and chemical changes in one part of the nervous system may have far-reaching effects for the rest of it.

Clinical Example

Consider a subject who gets nasty calf pain in the full slump test position (see Figure 4.2). Lifting the head relieves the pain in the calf and allows the knee to be fully extended without any major discomfort. Putting the head flexion back on again causes a dramatic return of calf symptoms. Without knowledge of structural connections and sensitivity of the nervous system it would be difficult to explain such a clear-cut phenomenon in terms of other tissue systems.

In a grossly mechanical sense the nervous system can be seen as a massive ligament or tendon that just happens to contain a system of specialized conducting and communicating cells. In this extreme slump 'test' position (Maitland, 1986) (Figure 4.2) the loss of knee extension could be analysed in purely mechanical terms, thus, lift the head, put some slack into the neuraxis/sciatic system and allow the knee to extend. In some situations like this the nervous system *may* physically limit movement. The reality though, is that at any limit of range, noxious forces cause nociceptive messages that make most people call a halt to a movement as well as offering up an appropriate protective motor reflex that further prevents movement (Elvey, 1995; Hall *et al.*, 1995). Perhaps performing the slump test under a general anaesthetic would help clarify whether or not pure nerve mechanics/tension was the key limiting structure to this position.

Clinical reality forces us to consider both

Figure 4.2 The slump test: note how release of the head flexion allows greater knee extension range. The slump test is fully described in Maitland (1986), Butler (1991) and Butler and Gifford (1998). (Adapted, with permission, from Butler, 1991, *Mobilisation of the Nervous System*. Churchill Livingstone, Melbourne.)

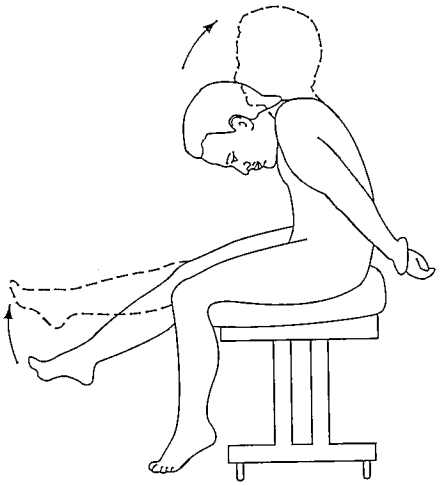
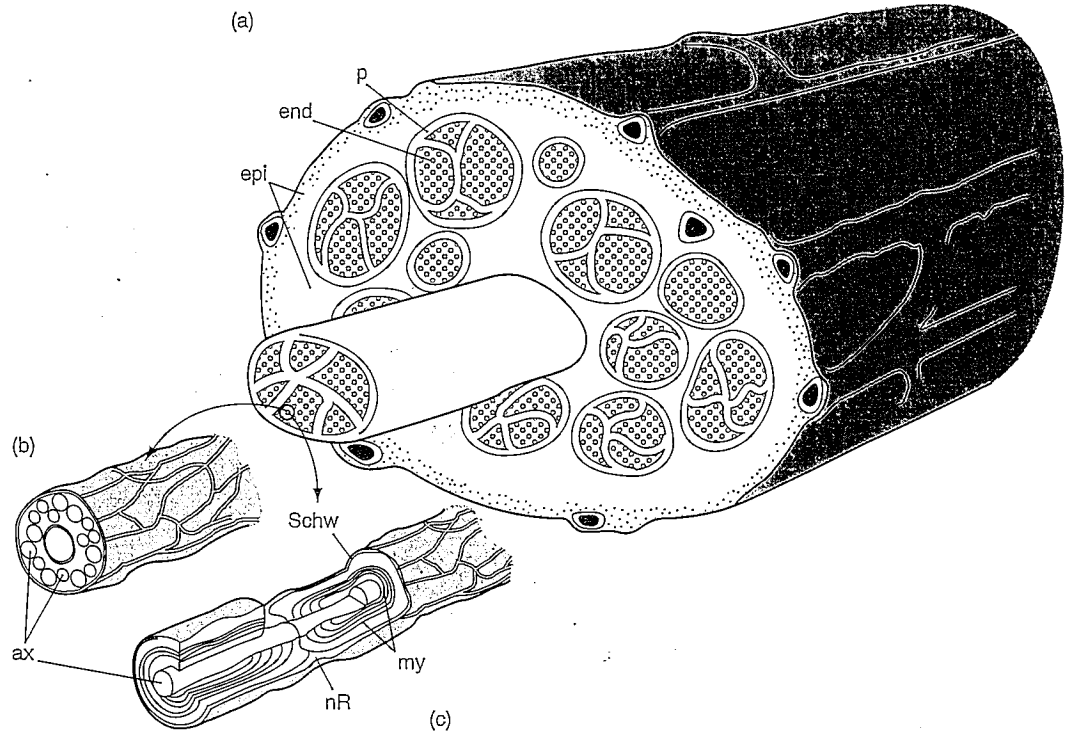


Figure 4.3 Microanatomy of a peripheral nerve trunk and its components. (a) Fascicles surrounded by a multilaminated connective tissue perineurium (p) are embedded in a loose connective tissue, the epineurium (epi). The outer layers of the epineurium are condensed into a sheath. (b) and (c) illustrate the appearance of unmyelinated and myelinated fibres respectively. Nerve fibres are surrounded by the endoneurial connective tissue (end). Schw, Schwann cell; my, myelin sheath; ax, axon; nR, node of Ranvier. (Adapted, with permission, from Lundborg, G (1988) *Nerve Injury and Repair.* Churchill Livingstone, Edinburgh.)



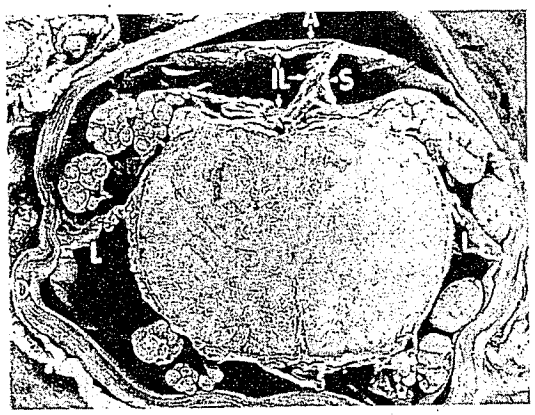
mechanics and sensitivity in parallel. In order to fully appreciate this we must recognize that the nervous system consists of four major tissue types, the first two of which are generally given most attention:

1 *Conducting nerve fibres, the neurones;*
2 *Collagenous connective tissues* whose important protective role is the major consideration;
3 Nonconducting *glial cells;*
4 The *vasculature.*

In the peripheral nervous system the conducting and connective tissue elements combine to form nerve trunks and nerve roots (Figure 4.3), whereas in the CNS the major protective connective tissues envelope and remain external to the neuraxis (Figure 4.4).

The existence of nonconducting cells in the CNS, the glial cells, was first recognized in the 1800s. For a long time these cells were considered as uninteresting putty that packs out spaces between the conducting cellular elements. What is now clear is that they have very important roles to play that include an immunological function, reabsorption of unused transmitters and providing the axons of conducting fibres with myelin (Streit and Kincaid-Colton, 1995).

Figure 4.4 Scanning electron micrograph of the lower spinal cord of a 15-month-old child. L, denticulate ligaments that suspend the cord within the sub arachnoid space. D, Dura; note the thickness and the layers. A, Arachnoid; note how it has come away from the dorsal dura in the preparation. S, dorsal septum. IL, intermediate leptomeningeal layer. Note the sectioned multifascicular nerve roots within the subarachnoid space. The pia can be seen adhering to and surrounding the cord. (From Nicholas, DS, Weller, RO, 1988, *Journal of Neurosurgery* 69: 276–282, with permission.)



The last tissue type of importance is the vasculature. The recent upsurge in interest in the detrimental effects of ischaemia on nervous system structures means that more than passing attention should be paid to the whereabouts of feeder vessels and veins and, vitally, the important effects that movement and sustained posturing has on their patency. For now, consider that movements that compress or stretch blood vessels will tend to decrease the lumen size and hence deprive the local or regional tissues of blood.

It is clear that the nervous system moves and that its anatomy strongly reflects this to be a built-in feature of its design. In order to be complete, the nervous system has to be examined from all its functional perspectives, that is, in terms of its ability to conduct and its ability to move in a normal and symptom-free way.

The Nervous System Responds to Movement

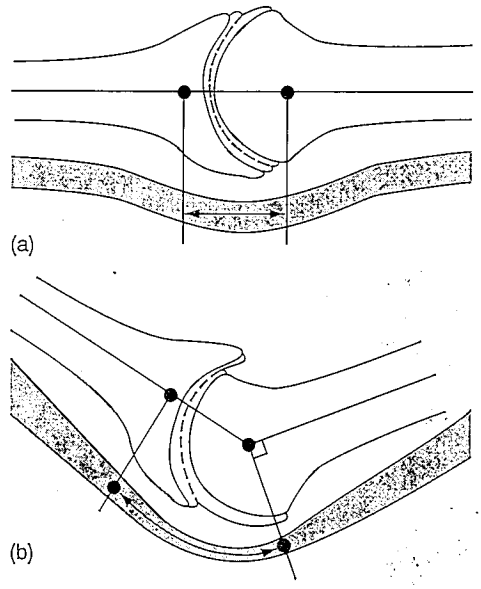
Basic Concepts

1 *The relationship of nerve position to the axis of a joint helps establish the effect a movement may have on neural tissue*: Many of the modern neurodynamic tests have been developed as a result of the anatomical and biomechanical appraisal of nerve trunks in relation to their surrounding tissues. In particular, the relationship to joint axes of movement. It is a matter of mentally drawing the known position and course of a peripheral nerve on the body and then moving the various joint components that it traverses in a way that will exert increased tension on the nerve under consideration. There has also been much reflection on the literature (Butler and Gifford, 1989; Butler, 1991). For instance:

- From the work of Millesi (Millesi, 1986; Millesi *et al.*, 1990) it has been calculated that from wrist and elbow flexion to wrist and elbow extension, the 'bed' of the median nerve gets approximately 20% longer.
- Beith *et al.* (1995) have shown that the sciatic nerve bed increases in length by 8–12% during the straight leg raising (SLR) manoeuvre.
- As long as 100 years ago it was considered self-evident that the length of a nerve must undergo changes during joint movement and that these changes create intraneural tension when a nerve's length is increased (Dyck, 1984; Beith *et al.*, 1995).

A key consideration is the position of the nerve in relation to the axis of movement of

Figure 4.5 Diagram of a nerve as it traverses a joint while joint is in neutral position. Arrows indicate length of nerve bed at level of joint. (a) Neutral position – nerve is slack. (b) Angulated position – nerve bed has elongated, causing nerve to be lengthened and bent across joint. (Reproduced from Shacklock M, 1995, *Physiotherapy* 81 (1): 9–16, with permission.)



the adjacent joint (Figure 4.5). Thus during elbow flexion the ulnar nerve at the elbow will tend to elongate and the median and radial nerves on the ventral aspect of the joint will tend to shorten, buckle and be compressed. End range neurodynamic tests like the SLR and the upper limb tension tests (ULTT) must be considered in terms of rather gross elongation effects of whole nerve trunks, plexi and nerve roots with further possible repercussions in the neuraxis. What must be emphasized is that even localized joint movements will have quite marked effects on adjacent neural tissues.

2 *Sliding, elongation and compression*: In order to adapt to body movements the nervous system is known to slide over adjacent tissue, elongate and be compressed. For example:

- Normal nerve roots are known to be compressed by spinal extension due to the decrease in size of the intervertebral foramen (Yoo *et al.*, 1992; Farmer and Wisneski, 1994).
- At the wrist the median nerve can be simultaneously compressed and elongated in the carpal tunnel during wrist and finger extension (LaBan *et al.*, 1989; Yoshioka *et al.*, 1993).
- The median nerve in the upper limb of normal volunteers has been shown to slide an average of 7.4 mm during movements of the wrist alone (McLellan and Swash, 1976).

Key Point

The fundamental physiological effects of sliding, elongation and compression due to normal movements are that they will load the nervous system and hence cause an increase of pressure within it. Changes in pressure cause changes in circulation and prolonged changes in circulation are likely to have detrimental effects on a tissue that is relatively blood thirsty (see below).

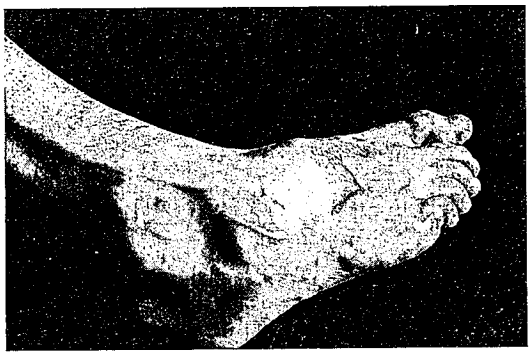
3 *Anatomical and attachment considerations in neurodynamics*: Concepts of nervous system movement must embrace the fact that its structure, its attachments and the interfacing structures around it, are continuously changing and very variable from one site to the next. The implications of this are that simple movement of one part of it does not have a uniform spread of effects throughout the whole, as it would if one considered the peripheral nerves and neuraxis as a homogenous string-like structure of uniform thickness and

elasticity and having no attachments to neighbouring tissues. The reality is that the nervous system gets ever thinner and more branching as it reaches towards its target tissues and it has varying amounts of connective tissue within and around it. It has considerable attachments to adjacent tissues that may prevent dispersal of forces further proximally or distally and it has contracting and moving structures around it that may have varying effects on neural load dissemination (Sunderland, 1978).

Thus, the effects of joint movement have non-uniform effects on the nervous system (Shacklock, 1995). For example:

- Full spinal flexion induces a 15% dural strain at L1–2 whereas at L5, strain approaches 30% (Louis, 1981). Further, the load on the nervous system will be greatest in the neural tissue adjacent to the site of joint movement (Shacklock, 1995).
- Ankle plantar flexion – inversion will produce quite a marked effect on the tension and movement of the superficial peroneal nerve as it courses over the anterior aspect of the ankle and foot. This is very easy to demonstrate on a thin foot (Figure 4.6). Proximal movement and tension repercussions of ankle plantar flexion inversion on the peroneal nerve tract have been observed as far as the thigh (Borges *et al.*, 1981).
- Smith (1956) in monkeys and Breig and Troup (1979) observing fresh human cadavers, have demonstrated that ankle dorsiflexion with the leg and trunk in a neutral position can have mechanical influences as far afield as the lumbosacral nerve roots. Performing the same movement in a SLR may have tension repercussions as far as the cerebellum (Smith, 1956).

Figure 4.6 Demonstrating the superficial peroneal nerve on the dorsum of the foot. (From Butler and Gifford, 1998, *The Dynamic Nervous System*. Adelaide, NOI Press, with permission.)



It seems that movements of one part of the body can have quite far-reaching repercussions for the nervous system elsewhere. This biomechanical consideration can be utilized in management approaches. For instance it may be desirable to physically influence nerve roots in a pain-free way post laminectomy. Moving the lumbar spine and hips as in performing SLR may be far too noxious, but moving the foot or knee may be tolerated well. One of the uses of this knowledge of the nervous system is that it can easily be influenced from a distance. It is also often worth giving some consideration to the order or sequence that tests and movements are performed in.

A key principle is that the greatest effect of a joint movement on a nerve will occur in the part of the nerve that is immediately adjacent to the joint being moved (Shacklock, 1995).

Thus ankle plantar flexion inversion will have most physical effect on the peroneal nerve over the dorsum of the ankle and less and less higher up the leg. Alterations of sequencing can be useful clinically when trying to localize tension to a particular segment of nerve trunk (Shacklock, 1995; Butler and Gifford, 1998).

It is now worth taking a more focused look at a few specific anatomical sites that illustrate some major neurodynamic principles and relate them to the pathological state.

The Effects of Movement and Sustained Posture on the Peripheral Nervous System

Trunk, head and limb movements and postures produce compression and elongation effects on the peripheral nervous system.

COMPRESSION

Nerves get relatively compressed when the cross-sectional area of the surrounding interface material diminishes in size. To demonstrate this, consider three anatomical zones that are commonly implicated in pain states associated with the nervous system. The first two zones relate to nerve roots, the third to a nerve trunk.

1 The L5–S1 nerve roots in the radicular canal;
2 The nerve roots generally in the intervertebral foramen (IVF);
3 The median nerve in the carpal tunnel.

L5–S1 Nerve Roots in the Radicular Canal

The L5–S1 roots are special because they cause a great many problems. It is not surprising that they have unique features and relationships that make them vulnerable to compressive distress and consequently the nerve root pain of sciatica.

Normal Anatomy/Biomechanics Anatomically the lumbar spinal canal contains only the peripheral nerve roots of the cauda equina encased in their dural sac (Figure 4.7). (The cord terminates at approximately L1–2.) From the point at which they emerge from the spinal cord to their exit in the IVF the lumbosacral nerve roots may be as long as 16 cm (Grieve, 1988). The next important anatomical consideration is that nerve roots are positioned more centrally in the spinal canal when they emerge from the cord but, as they descend towards their exiting foramen, they course more and more laterally into less and less space (Figure 4.7) (Wall *et al.*, 1990).

Figure 4.7ab Dural sac contents at L2–3 and L4–5 levels. (a) Diagrammatic representation of contents of lumbar dural sac at the L2–3 level. (b) Diagrammatic representation of contents of lumbar dural sac at the upper L4–5 level. (Adapted from Wall et al., 1990, *Spine* 15 (12): 1244–1247, with permission.)

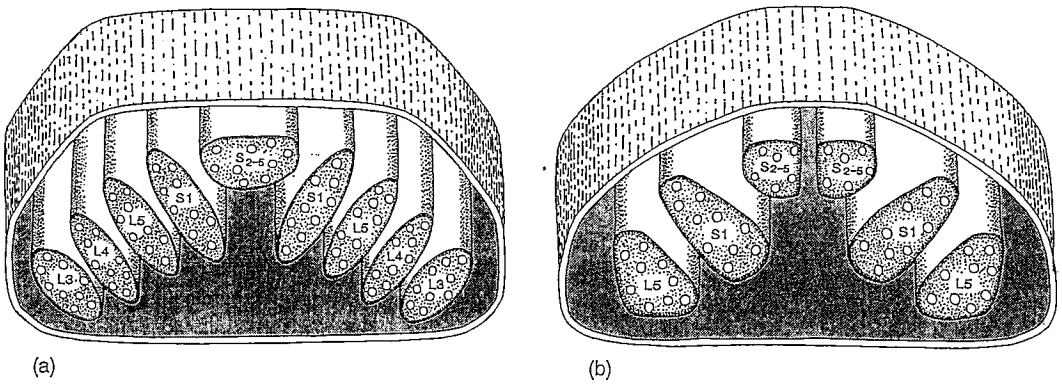
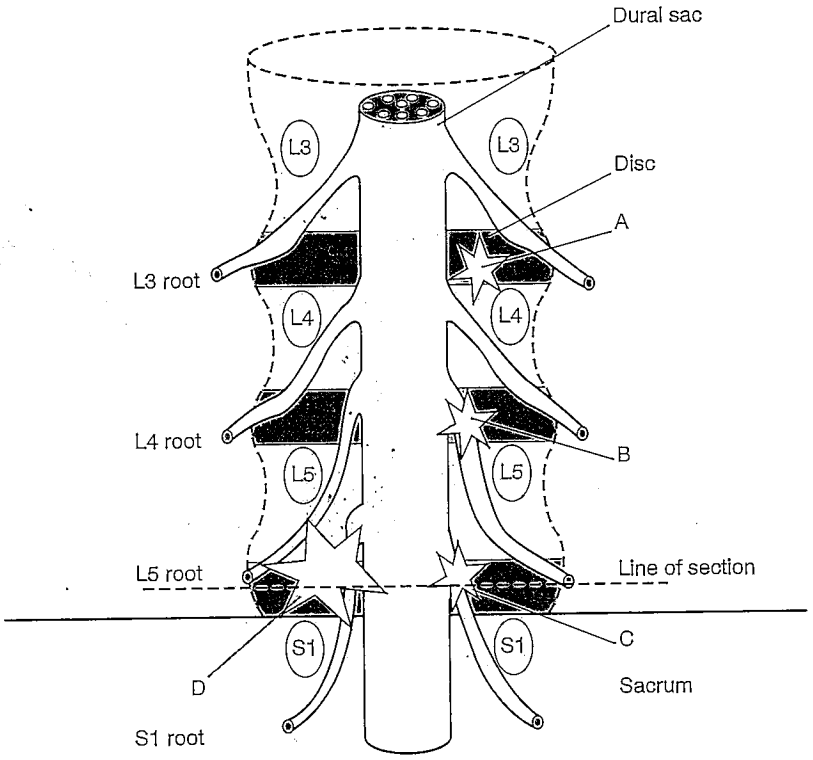


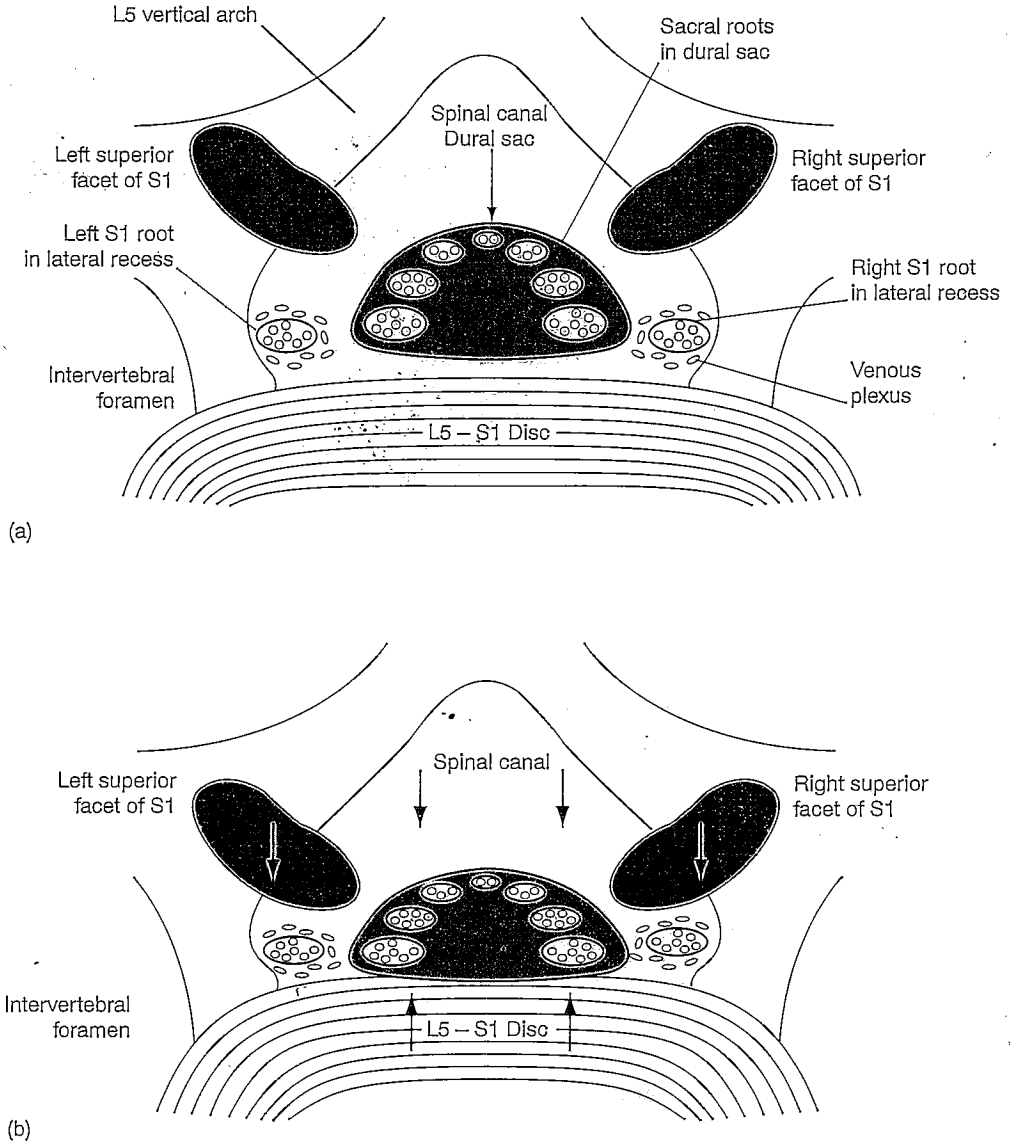
Figure 4.8 Diagrammatic representation of lumbar dural sac – nerve root anatomical relations and vulnerable zones. A, L4 root is less vulnerable to facet impingement here because the root exits the dural sac below the disc/facet level. B, L5 root vulnerable in standing/extension here – from superior facet of L5 coming down on it and from disc bulging backwards. C, S1 root vulnerable in standing/extension here – from superior facet of S1 coming down on it and from disc bulging backwards. D, L5 and S1 roots can both be influenced by movement-related compressive effects in this zone, especially if degenerate changes have caused narrowing of foramen and spinal canal. Line of section refers to Figures 4.9 and 4.10. (From Butler and Gifford, 1998, *The Dynamic Nervous System*, Adelaide, NOI Press, with permission.)



- Before emerging at the S1–2 intervertebral foramen the S1 nerve root lies in the lateral recess or 'radicular canal' medial to the articular pillar of S1 (Figure 4.8).
- Just above this it travels over the back of the L5 disc where it is in close relation to the overlying superior facet of S1.
- The salient feature is that the nerve root is isolated in its own dural sleeve having left the dural sac just above to the L5–S1 disc (Figure 4.8). A similar situation occurs with the L5 root at the L4–5 disc level.
- Note how the more rostral roots leave the dural sac just *below* the disc level.
- The significance of this is that the two 'isolated' roots (L5 and S1) are particularly vulnerable to compression effects within the lateral recesses of the spinal canal during movements of the spine.

Penning (1992) has convincingly shown that at the level of the disc/superior facet the size of the lateral recess is movement and posture dependent. For instance in standing, and increasingly in extension, the combined effects of the back-

Figure 4.9 (a) Horizontal section through L5–S1 disc in neutral position non-weight-bearing. (Line of section in Figure 4.8.) Image of vertebral arch of L5 is in background. (b) Horizontal section through L5–S1 disc in upright or extended position. (Line of section in Figure 4.8.) Image of vertebral arch of L5 is in background. Note how the contents of the spinal canal, the S1 facet and the L5–S1 disc move (arrows) to relatively compress the dural sac, the S1 root and surrounding tissues (adipose tissue and venous plexus). (From Butler and Gifford, 1998, *The Dynamic Nervous System*, Adelaide, NOI Press, with permission.)



ward bulging disc and the forward-moving superior articular facet significantly reduces the size of the recess (Figure 4.9a,b). The major effects of this in the normal spine are a modest squashing of the dense venous plexus surrounding the root and a medial sliding of the root. However, it has been suggested that this medial sliding may be strongly curtailed due to ligamentous distal fixation of the root in the intervertebral foramen (Spencer *et al.*, 1983).

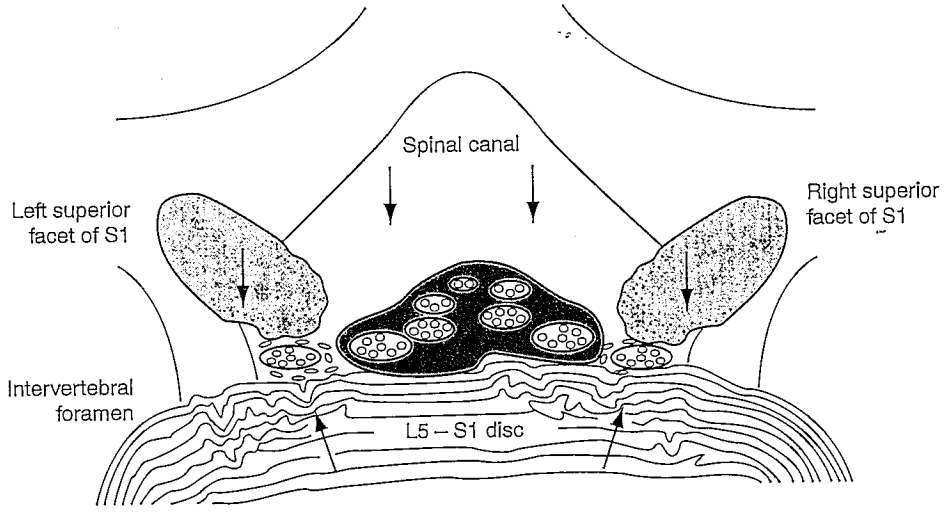
Pathological Anatomy/Biomechanics Pathological encroachment of the radicular canal area by a protruding disc or as a result of degenerative facet enlargement and flaval ligament thickening may have dire compressive consequences in standing and extension (Figure 4.10). This may go a long way to explaining why so many patients with sciatic nerve root distribution symptoms so often dislike standing for long, especially standing upright and extending, and prefer to be in varying degrees of flexion.

The important point is that we so often think of pathology affecting the root at the IVF, yet it should be apparent that if we follow the L5–S1 roots upwards there is a second zone where they are also vulnerable to compressive 'subarticular' forces (Vanderlinden, 1984).

Degenerative changes, congenitally limited radicular canal size and nerve root anomalies (Grieve, 1988) are important factors to consider when assessing any contributing factors to a disorder.

Nerve Roots and the Intervertebral Foramen According to Hoyland *et al.* (1989) the roots of the lumbar spine occupy the upper pole of the foramen, *above the disc*, and occupy a maximum of 35% of the area of the IVF. These workers found that the average area occupied by the root was

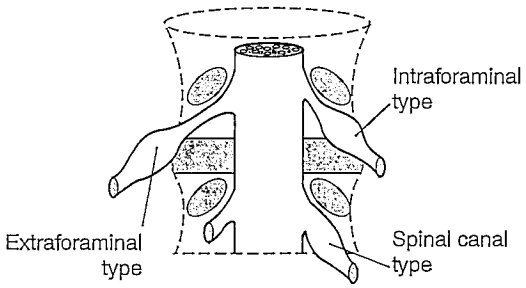
Figure 4.10 Degenerate changes: horizontal section through L5–S1 disc in upright or extended position. (Line of section in Figure 4.8.) Image of vertebral arch of L5 is in background. Note how the thickened spinal canal contents, S1 facet and L5–S1 disc move (arrows) to compress the dural sac, the S1 root and surrounding tissues (venous plexus and adipose tissue). (From Butler and Gifford, 1998, *The Dynamic Nervous System*, Adelaide, NOI Press, with permission.)



21% and that it ranged from as low as 2%. The rest of the apparantly ample space is mainly occupied by adipose tissue, radicular arteries and a venous plexus meshwork. Hoyland *et al.* (1989) stressed that relatively few of the cadavers they studied showed evidence of direct compression of the nerve roots by the disc. They highlighted the fact that the disc protrusions they observed more frequently compressed and severely distorted the venous plexus situated predominantly in the lower pole of the IVF overlying the disc protrusion. The pathophysiological importance of this observation is supported by Jayson (1992) who noted that careful examination of radiculograms in patients with disc prolapse often showed dilated veins and swelling of the nerve root indicating the presence of oedema. Unfortunately observing dissected cadavers does not take into consideration the effect of posture and movement on the disc–superior facet–root structure relationship in this area.

Dorsal Root Ganglion The position of the dorsal root ganglion (DRG) relative to the IVF is of some interest as this structure is recognized by some as a key player in the generation of radicular pain (Howe *et al.*, 1977; Devor and Rappaport, 1990; Devor, 1994). The reason for this is that the DRG is normally exquisitely mechanosensitive, yet the rest of the root tissue proximally and distally is relatively insensitive unless it is in an inflamed state (Garfin *et al.*, 1991; Kuslich *et al.*, 1991). The clinical implication of this is that symptoms of nerve root compression may only occur if the DRG gets compressed, or the adjacent insensitive areas of the root become inflamed – a process that does not invariably happen, and if it does, takes time to build up. It is very tempting to argue that sciatic pain that occurs at the instant of injury must be the result of DRG compression since normal nerve root takes time to become inflamed and generate pain.

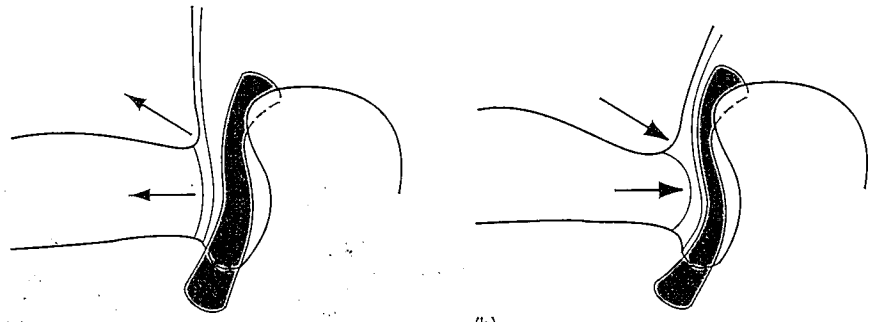
Figure 4.11 Variation in anatomical position of the dorsal root ganglion. Classification according to Sato and Kikuchi (1993). (Adapted from Sato and Kikuchi, 1993, *Spine* 18 (15): 2246–2251, with permission.)



It seems likely that some people may be more prone to DRG compression. A recent cadaver study by Sato and Kikuchi (1993) revealed that the position of the ganglion relative to the foramen was remarkably variable. They classified the posiiton of the DRG as being either located medially (spinal canal type), within the foramen, or extraforaminal (see Figure 4.11) and found that the extraforaminal type was the least likely to suffer adverse pathological compression. As far as the L5 ganglion was concerned, it appeared more vulnerable if it was situated more proximally.

Sato and Kikuchi (1993) interestingly noted 'indentations' on the ganglia due to impingement by the disc and adjacent superior facet in elderly specimens which showed space encroaching pathology (enlarged degenerate facets and bulging disc material). The authors made no comment as to whether the ganglia were physically pinched by these structures when they dissected them (a dissected out lumbar spine is likely to be in a neutral position and not subjected to any compressive forces that would alter the relevant

Figure 4.12 Diagram of lumbar nerve root dynamics in the intervertebral foramen. (a) Flexion: posterior wall of disc annulus is stretched and tightened, the body of the vertebra above moves forwards and away from the root and the superior facet moves backwards in relation to the root. The intervertebral foramen area thus enlarges. (b) Extension: the opposite occurs; note how the disc bulges into the foramen, the facet tip and the posterior rim of the vertebra above come close together and the nerve gets relatively compressed by this pincer mechanism. (From Penning, 1992, *Clinical Biomechanics* 7 (1): 3–17, with permission.)



structures position and shape). According to Penning's (1992) findings, it is reasonable to assume that standing postures and extension movements would dramatically decrease the already pathologically limited space in the foramen and even physically pinch the ganglion/root there (Figure 4.12).

Thus the dorsal root ganglion/nerve root in the IVF can be compressed in the 'jaws' formed by the tip of the superior facet behind and the bulging disc and the inferior rim of the adjacent vertebral body in front. The fundamental movements to consider are those that tend to 'close' the IVF, i.e. going to standing (since erect standing compresses and causes the disc to bulge), extension and movements towards the side under consideration (Panjabi *et al.*, 1983). This principle also applies to the cervical spine (Yoo *et al.*, 1992; Farmer and Wisneski, 1994) and more than likely in the thoracic spine, judging by clinical findings.

The Carpal Tunnel

Since the carpal tunnel contains the median nerve to the hand, as well as nine flexor tendons, it has little room for intrusions. Thus any process that tends to cause narrowing of the space, or an increase in the size of the contents, will increase the tissue pressure in the tunnel.

Normal pressure in the carpal tunnel is around 2.5 mmHg, in wrist flexion the pressure increases to about 30 mmHg, a pressure which corresponds well to the critical pressure known to induce the first changes in intraneural microcirculation and axonal transport (see below) (Gelberman *et al.*, 1981).

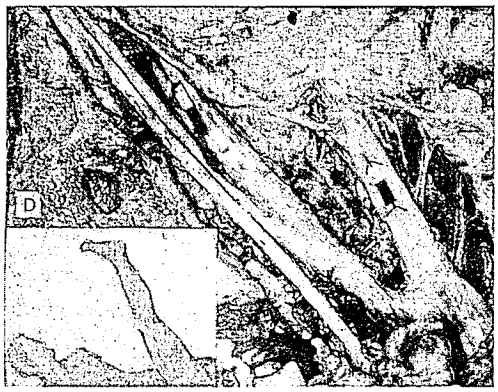
Movements of the wrist alter the size of the carpal tunnel. For example, Yoshioka *et al.* (1993) have shown that in wrist flexion the carpal tunnel gets 16% smaller at the pisiform level. It is hardly surprising that sustained wrist flexion begins to cause hand paraesthesia in many who are otherwise normal.

SLIDING AND ELONGATION

Nerves get relatively elongated by limb and trunk movements that increase the length of their nerve

Figure 4.13 Anterolateral view of right sacral plexus and roots L4 and L5 as they emerge from their intervertebral foramen. Paper markers 1 cm long have been sutured to the nerves. In (a) with the trunk in neutral and hip in flexion the neural structures are relatively slack and the markers lie within (L4) and just distal (L5) to the intervertebral foramen. In (b) the right knee has been extended into the straight leg raise position and the amount of root movement relative to the foramen is clear. (From Breig, A, 1978, *Adverse Mechanical Tension in the Central Nervous System*, Almqvist and Wiksell, Stockholm, with permission.)

(a)



(b)

beds (i.e. the tissues which surround them and which they lie in). They adapt to this in two ways:

1 By sliding;
2 By elongation.

The remarkable movement of the L4 and L5 spinal nerves during the SLR manoeuvre is shown in Figure 4.13. The S1 nerve root complex has been reported to slide as much as 10 mm (Goddard and Reid, 1965) and tension transmitted as far rostrally as the mid brain (Smith, 1956). An important principle to consider with regard to neurodynamics is that in some situations more sliding of a nerve will occur, yet in others more tensioning and elongation will occur, and that what occurs when, is likely to be hugely variable between individuals.

Examination of fresh cadavers (Goddard and Reid, 1965) has revealed that during straight leg raising, when the heel is only 5 cm above the horizontal, movement/sliding of the nerve at the greater sciatic notch has already begun. The movement spreads proximally the more the leg is lifted and does not start to affect the nerve roots in the IVF until around 35° of SLR. As the leg raising continues so the sliding effect diminishes and the tension/elongation effects grow, so that by 70° there is little sliding and the adaptation has to be borne by the intrinsic elastic capabilities of the nerve. Considerable stresses are imparted on nerve roots during spinal movements. For instance, Louis (1981) calculated that during flexion greatest strain (16%) was taken by the S1 nerve root.

It should be noted, though, that cadaver observations must be interpreted with caution since close scrutiny of nerve trunks and roots reveals quite marked attachment tissues that often strongly anchor the nerve to the surrounding interfacing tissues (e.g. see Spencer *et al.*, 1983). *These tissues may well be dissected away during exposure of the nerve and hence give a false representation of the amount of movement available* (Beith *et al.*, 1995). For example, Lombardi *et al.* (1984) have criticized the notion that the L5 nerve root slips

in and out of the IVF during SLR since it is securely bound to the side of the body of S1.

There is ample evidence that upper limb nerve trunks slide and move. For instance, the ulnar nerve migrates proximally during elbow flexion (Macnicol, 1980), extension of the index finger has been shown to move the median nerve in the carpal tunnel 4 mm distally (LaBan *et al.*, 1989) and wrist and finger extension will pull the median nerve in the upper arm downwards by an average of 7.4 mm (McLellan and Swash, 1976). This last result was achieved on normals by inserting fine needles into their median nerves that protruded out of the skin! The excursion of the nerve was calculated by observing the amount of movement produced by the tip of the needle.

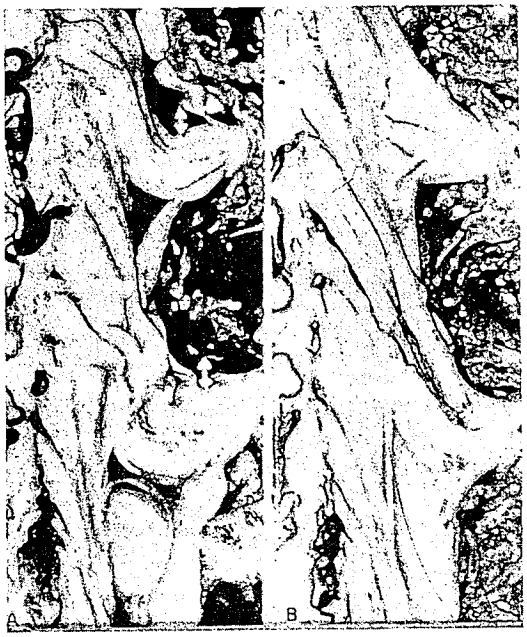
In summary, the nervous system is capable of quite remarkable adaptations in response to the body's movement and postural demands. Peripheral nerve roots and nerve trunks are being continuously squashed and stretched and in situations where there is relatively little room or where they are anatomically tethered they are vulnerable to adverse forces and pathological tissue encroachment.

The Effect of Movement on the Neuraxis

EVIDENCE FOR DYNAMIC EFFECTS DUE TO SPINAL MOVEMENT

The neuraxis is housed in the spinal canal and cranium. Since the spinal canal increases in length by 5–9 cm from full extension to full flexion (Breig, 1978; Louis, 1981; Inman and Saunders, 1942) there are quite marked physical effects on

Figure 4.14 Normal deformation of the dura, cord and nerve roots in the cervical canal in the cadaver due to full extension (left) and flexion (right) of the cervical spine. A total laminectomy has been performed and the dura opened and retracted although still able to transmit tension. In cervical extension, the nervous system is slack, the root sleeves have lost contact with the pedicles (lower arrows) and the nerve roots with the inner surfaces of the dural sleeves (upper arrows). In flexion, the nervous system including the dura mater has been stretched and moved in relation to surrounding structures. Note change in shape of the blood vessels. (From Breig, A, 1978, *Adverse Mechanical Tension in the Central Nervous System.* Almqvist and Wiksell, Stockholm, with permission.)



the connective tissue sheaths and the cord and brain structures themselves. Most lengthening occurs in the most mobile cervical and lumbar regions (28 mm each) and least in the thoracic region (3 mm) (Louis, 1981). The dynamic adaptations of the cervical cord and roots can be seen in the dissection photograph (Figure 4.14). Figure 4.15 illustrates the effects of flexion–extension on the brainstem. Further examples include:

- Neck flexion causing the floor of the fourth ventricle to elongate;
- The thoracic cord decreasing its diameter during spinal flexion (Breig, 1978);
- The folding and straightening of individual neurones in the dorsal column tracts of the cord during flexion and extension (Breig, 1978; Butler, 1991). This emphasizes the need for physical adaptations to movement right down to the cellular level.
- During spinal flexion the key considerations are that the whole neuraxis contained in the spinal canal tends to move anteriorly, elongate along its entire length and slide relative to the canal interface (see Shacklock *et al.*, 1994).

MOVEMENTS OF NEURAXIS RELATIVE TO THE SPINAL CANAL

Butler (1991) first brought more widespread attention to the fact that movements of the neuraxis *relative to the surrounding tissues* was not at all uniform and not necessarily in directions one would expect. For instance, during flexion of the monkey spine the dura at the level of L4 moves 3 mm caudally yet at L5 it moves 3 mm rostrally. In the neck the dura at C5 tends to move caudally and at C6–7 rostrally. In the thoracic region the dura above and below T6 tends to move away from it (Louis, 1981).

Figure 4.15 The remarkable movement of the brain stem in extension (a) and flexion (b) is illustrated by reference to the markers. Marked changes in tension of the eleventh cranial nerve (spinal accessory) can also be seen anterior to the cord/brainstem as it passes up from its origins on the cord to exit through the jugular foramen on the upper right of the picture. (From Breig, A, 1978, *Adverse Mechanical Tension in the Central Nervous System.* Almqvist and Wiksell, Stockholm, with permission.)

(a) (b)

Effects on Nerve Roots

These relative movements will have repercussions for the tension and angulation of the nerve roots as they leave the dura and as they exit in the IVF. For example, in flexion, nerve roots above L4 will tend to be angled more horizontally and those below more vertically. Further, any deviations from such 'normal' movement adaptations, due to for instance abnormal tethering following injury, may have serious mechanical overload repercussions at local and distant sites which may already be somewhat vulnerable. These thoughts have been used to hypothesize why symptoms often spread or jump to remote sites (Butler, 1991). For example, it is not uncommon for pain from whiplash injuries to appear in the midthoracic and lumbar regions long after the event.

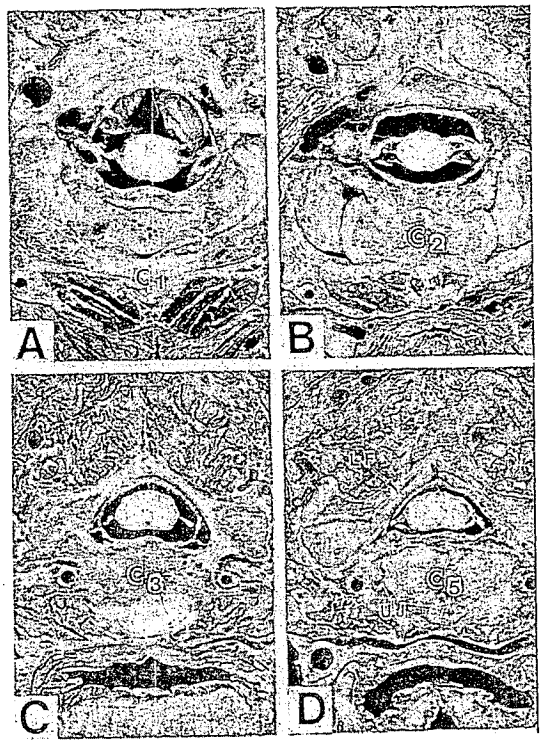
EFFECTS OF SPINAL EXTENSION

- During spinal extension the whole canal shortens causing the neuraxis to slacken and increase in cross-sectional area. Observable folds can be seen in the cervical dural sac in extension.
- In the lumbar spine in particular, the spinal canal gets smaller in diameter in extension due to the posterior bulging of the discs and the forward bulging of the interflaval fat pad and the ligamentum flavum. This effect increases greatly in the presence of degenerative encroachment (Penning, 1992; Penning and Wilmink, 1981).
- This may be a reason why lumbar extension causes an increase in lumbar cerebrospinal fluid pressure (Hanai *et al.*, 1985).

Zones of the spinal canal where there is relatively little space, like C5 compared to C1 or C2 (Figure 4.16), may be significantly more vulnerable to:

1 Pathological encroachment;
2 The addition of movements into extension.

Figure 4.16 The changing spaces available for the neuraxis and meninges in the cervical spinal canal at C1(A), C2(B), C3(C) and C5(D). LF, ligamentum flavum; UJ, uncovertebral joint. (Reproduced from Parke, WW, 1988, *Spine* 13: 831–837, with permission. Lippincott Raven Publishers, Philadelphia.)



Think how common it is for the elderly degenerate spine to become relatively flexed. One way of viewing this is to see it as an adaptive process that helps maintain the least possible pressure on the threatened neuraxis and its peripheral roots and trunks.

SIDE FLEXION AND GRAVITY

Side flexion of the spine tends to produce slackening on the concave side and tightening on the convex side, and gravity displaces the spinal canal structures downwards (see Breig, 1978; Shacklock et al., 1994).

When interpreting symptom responses it is wise to consider the relative flexion/extension

position of the spine as well as compression effects in the canal and IVF. It is apparent that it is not easy to find a position where there isn't some degree of mechanical impact on any one part of the nervous system. Knowledge of neurodynamic influences does help rationalize the difficulties patients have in finding relief when confronted by neurogenic pain.

Design Features and Nervous System Dynamics

Since it seems well established that movement of the nervous system occurs it is pertinent to highlight a few anatomical features that add weight to the notion of it having this dynamic capacity.

Neurones, Fascicles and Plexi

- Neurones, the individual nerve fibres, tend to run an undulatory course within the fascicles of peripheral nerves and within the tracts of the spinal cord (Breig, 1978).
- If a nerve fibre is stretched the myelin sheath lamellae slide on each other and little clefts (incisures of Schmidt–Lantermann) in the myelin part to accommodate the increased stress (Friede and Samorajski, 1969).
- The fascicles that contain the nerve fibres are capable of sliding within their epineurial sheaths as well as following an undulating and branching course (Figure 4.17) that appears geared to a function of load dispersal.
- The reason for the complex branching of the various peripheral nerve plexi may relate to load dispersal (Figure 4.18) (Butler, 1991).

Figure 4.17 The fascicular branching in the musculocutaneous nerve. (Redrawn, with permission, from Sunderland, S, 1978, *Nerves and Nerve Injuries*. Churchill Livingstone, Edinburgh.)

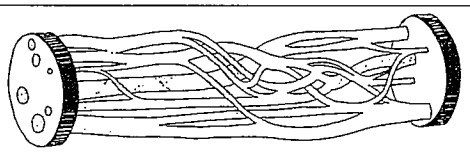
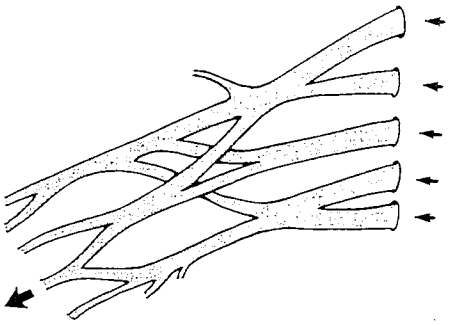


Figure 4.18 The brachial plexus as a force distributor. Tension on one trunk will be distributed throughout the whole plexus. (From Butler, DS, 1991, *Mobilisation of the Nervous System*, Churchill Livingstone, Melbourne, with permission.)



Connective Tissue

The peripheral and central nervous system contains connective tissue with viscoelastic properties, similar to ligaments and tendons (Bora *et al.*, 1980; Kwan *et al.*, 1992; Sunderland and Bradley 1961a,b). These tissues serve to allow and control nerve trunk motion in parallel with a protective role when forces reach physiological limits. It appears that in regions where the nervous system is more vulnerable to injury there is a higher density of connective tissue.

Clinical Example

The size of the superficial peroneal nerve over the top of the foot (Figure 4.6) is quite remarkable when one considers that it has only a relatively small dermal

innervation field to reach that is very close by. Its size here has to be considered in terms of its obvious vulnerability to injury and therefore its need for a relatively large connective tissue protection component.

Other examples are the sciatic nerve at the buttock where it gets compressed in sitting. Here it contains in the region of 80% connective tissue (Sunderland, 1978). The peroneal nerve at the head of the fibula contains 17% more connective tissue than the same nerve a few centimetres proximally in the relatively sheltered area of the popliteal fossa (Sunderland and Bradley, 1949). Sunderland (1978) also demonstrated how the number of fascicles in vulnerable areas tends to increase in parallel with the increase in connective tissue, yet another mechanism thought to afford better protection to the nerve fibres from adverse compression.

Inside the spinal canal attention is focused on the tough dura mater connective tissue covering. This structure is particularly strong and well designed to cope with longitudinal forces (see Butler, 1991 and Shacklock *et al.*, 1994 for good overviews of the neuraxis connective tissues).

Blood Supply

There are many dynamic design features in the nervous system. It is designed to elongate and recoil and it is designed to cope with intermittent compression and distortion. During all this it must still continue to conduct and connect physiologically to its target organs and tissues – a condition only possible if adequate blood supply is provided. Although only accounting for 2% of the body mass the nervous system requires 20% of the available oxygen (Domisse, 1994).

Maintaining an adequate blood supply, whatever the posture or movement, is imperative for the metabolic demands of normal neural function. It is hardly surprising therefore that the blood vessels appear relatively slack and coiled, follow meandering courses along and within nerves, and enter the domain of the nervous system in zones where the system is relatively fixed in relationship to its adjacent structures (see Breig, 1978; Lundborg, 1988; Parke and Watanabe, 1985). For example, major feeder vessels enter the cervical cord between C5 and C7 – a region that does move in relation to the interface, but relatively little, due to the fixation effect of the brachial plexus and the strong tethering of these lower cervical nerve roots to the gutters on the transverse processes just distal to the IVF (Sunderland, 1974; Butler, 1991). Even though there is good design for continuous adequate blood supply, no matter what the posture or position, there are still plenty of opportunities that lead to its compromise (see below).

The Normal Nervous System as a Sensitive Tissue

Generally, tissues that are most likely to be exposed to extreme physical stress are likely to have a good sensory innervation – compare the mechanical sensitivity of the contents of the gut to the sensitivity of the skin or the musculoskeletal system for instance. Further, any tissue that is weakened or vulnerable following injury requires a greatly enhanced sensory mechanism to help protect it from further damage and to promote appropriate behaviour for it to recover (see Chapter 5). Coupled with a good sensory relay is a good reflex system, and in many higher

vertebrates, an active consciousness that provides the background for appropriate adaptive action/behaviour.

The normal nervous system copes with physiological movements in a remarkably silent way; however, extreme ranges of movement and direct forces often endanger neural tissue. The nervous system has the means to respond via its own sensory system. Just as the heart has its own blood supply so the nervous system has its own nerve supply. The innervation is specifically to the protective connective tissues of the peripheral and central nervous systems (see Butler, 1991, Shacklock *et al.*, 1994, for good overviews).

It is well known that brain tissue itself is mechanically insensitive and that normal peripheral nerve fibres will only modestly respond to intense physical probing (Melzack and Wall, 1996). In fact there are many who report that peripheral nerve trunks are more or less insensitive to mechanical pressure and manipulation (e.g. Kuslich *et al.*, 1991; Howe *et al.*, 1977). However, anyone who has spent time learning to palpate peripheral nerves will know that some nerves are far more sensitive than others and that where any one nerve is sensitive depends on its anatomical site (Butler and Gifford, 1998). For example, a vulnerable nerve like the superficial peroneal on the foot (Figure 4.6), is pretty insensitive – you have to tap it or 'twang' it vigorously to get a modest tingling in its distal innervation field. Compare that to palpating the ulnar nerve at the elbow or the tibial nerve just behind the medial malleolus; in both cases gentle palpatory pressure reveals a rather sickening 'nervy' discomfort.

The important practical point is that peripheral nerves are variably sensitive – depending on the anatomic site and depending on the individual. Some people's nerves have great sensitivity over others.

Senstitivity to Stretch/Elongation

We should also consider sensitivity to stretch/elongation. Again there is great variability in sensitivity and some people have far more slack in their nervous systems than others.

Practical Task

Try this: place your arm down by your side, extend your elbow hard and keep it extended hard, extend your wrist with your fingers as straight as you can. Now depress your shoulder slowly and focus on what you feel in your hand and arm. Many of you will get a deep generalized ache vaguely in the elbow/forearm region plus or minus feelings of pulling and paraesthesia in the hand. Adding neck side flexion away normally increases the response still further, but go carefully. Those who have good mobility of their joints often have extreme mobility of their nervous systems and may get very little with a manoeuvre like this; others will easily get many of the symptoms described and more. Note that since the symptoms can be changed in the forearm/hand with scapular depression and neck movement, the assumption is that neural tissue is responsible (see Chapter 6). It is argued that fascia and vasculature that also traverse these regions are unlikely to produce such 'nervy' sensations as deep diffuse ache and paraesthesia in such a clear-cut way.

Type of Sensitivity

There are two significant issues with regards to sensitivity:

1 Mechanical sensitivity;
2 Sensitivity to ischaemia.

Mechanical sensitivity
This exists through

- Mechanical stimulation of mechanically sensitive *nerve endings* in the protective connective tissue layers of the nerve;
- Mechanical sensitivity of the *nerve fibres* (neurones) themselves.

Nerve fibre axons are generally considered to be designed to convey impulses not generate them – nerve impulses traditionally originate from the ends of nerves that reside in target tissues, not mid-axon somewhere. Impulses that do originate mid-axon are said to be 'ectopic' (see Chapter 5) and if derived from afferent fibres may cause the generation of odd sensations – hence pins and needles are commonly felt when nerve trunks that supply cutaneous sensation are firmly tapped.

Sensitivity to ischaemia or to blood loss, i.e. 'Ischaemosensitivity' (Butler and Gifford, 1998). Falling asleep with your arms above your head and later waking up with a numb and tingling arm that does not respond to demands for movement is an extreme example that is not uncommon. Holding continuous pressure on the anterior wrist over the carpal tunnel produces a slowly building paraesthesia in many otherwise asymptomatic subjects. The fact that symptoms are not immediate suggests loss of blood supply to the underlying median nerve as a likely mechanism. Some people are more susceptible than others and they often find constant end-range positions in bed (wrist flexion for example) or during the day (crossed leg sitting) a common source of irritation by way of numbness and pins and needles type sensations.

When individual nerve fibres are deprived of circulation their function is impaired and many of the large metabolically demanding Aβ sensory fibres begin to fire ectopically – hence pins and needles if they innervate skin. Ectopically firing sensory fibres (due to mechanical force or loss of blood) that innervate deeper tissues, like muscle, may produce incongruent sensations that relate to their innervated tissues. Thus persistent pressure over the radial nerve in the radial groove may produce deep aching sensations vaguely in the forearm muscles as well as paraesthesia in the skin over the back of the lateral hand. These areas relate to the muscles and skin innervated by the radial nerve.

Although not proven, it seems that nerve trunk mechanosensitivity in the normal physiological state depends firstly on vulnerability and secondly on design that includes connective tissue density as well as specialized local innervation characteristics, and the senstivity of the contained nerve fibres themselves (Lundborg, 1988). Thus, many nerves are in extremely vulnerable places near the surface of the skin, like the many little geniculate nerves on the side of the knee, the superficial peroneal nerve on top of the foot and the radial nerve in the radial groove on the lateral aspect of the humerus. These nerves seem particularly insensitive to palpation and are probably hugely adapted to cope by consisting of large quantities of protective connective tissue (Sunderland, 1978). By contrast, some superficially placed nerve trunks are remarkably mechanosensitive (Butler, 1991), the ulnar nerve at the elbow, the median nerve in the axilla and just distally, and the proximal cords of the brachial plexus in the supra clavicular

fossa are examples. It appears that these nerves are less well endowed with connective tissue, have relatively exposed nerve fibres and are hence more sensitive (Butler, 1991). To a degree, these areas are situated in regions less likely to be directly injured in the rough and tumble of daily life. Deeper nerves often reside in zones prone to pressure changes with normal movements and postures, and seem to cope in a remarkably restrained and silent way. For example, nerve roots in the intervertebral foramen and the median nerve in the carpal tunnel get squashed and stretched all the time yet we are totally unaware of it unless pathological changes cause them to become acutely ischaemosensitive or mechanosensitive.

Physiological Effects of Movement on Nervous Tissue

The Importance of Blood

The importance of an uninterrupted blood supply to the nervous system has already been discussed. Like all other cells, neurones require blood for their metabolic demands, but in particular for powering axoplasmic transport and impulse generation and conduction (Lundborg and Dahlin, 1996; Lundborg, 1988). Loss of conduction due to ischaemia is most notable by the appearance of paraesthesia, numbness and weakness as when you fall asleep with your arms above your head.

AXOPLASMIC FLOW OR TRANSPORT

Many neurones in the peripheral nervous system are unique in that they are extemely long cells, for example an individual nerve cell from the foot to the spinal cord may be 1 metre long. To put this in perspective, consider if this neurone's cell body was increased in size to 100 cm in diameter, it would have an axon diameter of 10 cm and a length of around 10 kilometres (Rydevik *et al.*, 1984).

The viability of any cell is largely dependent on the activities of the nucleus and cell body and its ability to communicate with the rest of the cellular constituents.

In neurones the specialized flow of cytoplasm from the cell body to its distant peripheral sites and back is termed *axoplasmic* flow. This flow of neurochemicals and cellular structural components within the axoplasm is essential not only for the health and functioning of the cell itself, but also for the health of the tissues that the cell innervates. The nonmyelinated afferent C fibres are now thought to have a particularly important role to play in the maintenance of their target tissues' health and in aiding any healing process via a direct chemical contribution to the inflammatory cascade (see Chapter 5). Importantly, neurones are not just designated to an impulse-conducting role, many also have the ability to chemically sample the tissues they innervate and relay these chemicals as a type of 'messenger' back to their nucleus and cell bodies (Donnerer *et al.*, 1992). The nucelus/cell body then alters its activity to produce a specific chemical response that is transported back to the target tissues in order to rebalance the trophic disturbance that was detected earlier (Donnerer *et al.*, 1992; Heller *et al.*, 1994; McMahon and Koltzenburg, 1994). In this way the nerve cell is constantly monitoring the health of its target tissues, responding to any abnormality and as a result helping to maintain tissue viability. The implications of this are that anything that disturbs the bidirectional axoplasmic flow between cell body and the axon and its terminals will have repercussions for the health of

the nerve cell itself as well as the target tissues it supplies. If the loss of communication is severe enough the distal axon undergoes Wallerian degeneration or the whole cell may even die (Devor, 1994).

POSTURE AND MOVEMENT EFFECTS ON CIRCULATION TO NERVE

Axoplasmic flow is particularly sensitive to changes in circulation (Okabe and Hirokawa, 1989), and circulation to and within nerve tissues is influenced by changes in pressure produced by postures and movements of the interfacing tissues.

Circulation to a nerve, like any other tissue, is dependent on a pressure gradient existing between the incoming arterial supply and the outgoing venous return (Sunderland, 1978). Thus a greater pressure is required at the arterial side in order to push blood flow through the nerve and out into the veins. Further, any changes in pressure around the veins or nerve will upset this gradient and will cause back-pressures that lead to vascular stasis and ischaemia/hypoxia.

Pressure changes that influence circulation to nerve can be brought about by relative compression or elongation of nerve tissue. The influences of pressure changes produced by wrist flexion on the contents of the carpal tunnel have already been noted. Olmarker's group (Olmarker *et al.*, 1989) have found that pressures as low as 5–10 mmHg can stop venular blood flow in the lumbar nerve roots of pigs, pressures probably easily achieved at the IVF and radicular canal due to compression during normal extension or perhaps even during prolonged standing.

When *elongation* forces are applied to a peripheral nerve the supply vessels tend to straighten up and narrow; the intraneural vessels similarly unfold, stretch and reduce their lumens, and the intraneural pressure steadily increases the more the nerve is stretched (Pechan and Julis, 1975). In the rabbit peripheral nerve, venous return starts to decline at 8% elongation and by 15%, arterial, capillary and venous flow is completely occluded. If we consider that the length of the nerve bed of the median nerve can increase by as much as 20% in full arm, hand and finger extension, it is not surprising that many of us get building ischaemia-related neural responses when maintaining these positions.

Key Points:

- *It is important to understand that the nervous system is designed to accommodate to continuous pressurizing and stretching forces and that recovery after short-term modest loading is normal.*
- *What the nervous system does seem to find unacceptable is ongoing adverse physical stress – it far prefers movement. Long-term increased pressures and stretching that many of our relatively static lives foist upon the nervous system may well be quietly damaging it more than it otherwise should be.*
- *Pechan and Julis (1975) showed that mere elbow flexion doubles the pressure in the ulnar nerve when compared to elbow extension.*
- *Dahlin and McLean (1986) showed that modest prolonged pressures on rabbit vagus nerves will completely block axoplasmic flow and that its return to normal may take anything from 24 hours to 1 week depending on the amount and time of pressure.*

- *Axoplasm tends to increase its viscosity if nerves are not moved (Baker et al., 1977); it thus gets thicker, less fluid and its flow properties will be compromised at the expense of the health of the nerve and its target tissues. By contrast movement will enhance its flow properties and hence nerve and tissue health.*

Many of the features discussed may also be pertinent to the neuraxis. For example, the longitudinal stress imparted during spinal flexion may increase the pressure within the cord since it decreases its diameter in flexion (Breig, 1978). Longitudinal vessels may be narrowed (Figure 4.14) and movements of the limbs may influence feeder vessel lumens by directly or indirectly pulling or angulating them. Adequate cerebrospinal fluid circulation in the subarachnoid space of the cord and brain is maintained by movement and since much nutritional delivery is via this route it underlines yet again the importance of regular movement for adequate neural health and function.

The student is reminded that far more can be achieved in tissue physiological terms by the patient performing simple uncomplicated active movements than can be achieved by purely passive techniques.

Pathophysiology of Nerve and its Influence on Movement: 'Neural Pathodynamics'

Pathodynamics: Integrating Pathomechanics and Pathophysiology

So far the emphasis has been on the influence and interaction of mechanical forces on the physiology of the nervous system. Shacklock's (1995) timely introduction of the term 'neurodynamics' has helped focus attention away from pure mechanics (Butler and Gifford, 1989) and towards a perspective that powerfully includes the inseparable influences of mechanics and physiology on each other. Further, in order to emphasize thoughts of pathophysiology and pathomechanics when injury or disease occurs Shacklock has introduced the term 'neural pathodynamics' (Shacklock, 1995).

Many pain states that physiotherapists encounter are the result of some mechanical event which varies from the extreme of sudden insult to more minor sustained and prolonged adverse pressures or tensions of some kind. Adverse mechanical events lead to burgeoning pathophysiological responses, typically inflammation, healing and repair plus or minus a pain state, which taken as a whole, is the body's means of restoring function. That full or perfect repair in many of the more complex and metabolically sluggish tissues of the body can be achieved has to be questioned since most adult musculoskeletal tissues repair by scar tissue formation not regeneration (Butler and Gifford, 1998). Thus, it is to be expected that as a result of physiological reparative processes

(pathophysiology/pathobiology), some mechanical dysfunction (pathomechanics) will inevitably occur during the later stages of healing and much may well remain permanently. Inevitably this means that tissues in the functional proximity of a given lesion will have to adapt accordingly and may well predispose the individual to further problems later on. It has to be assumed that if a repaired tissue is mechanically compromised it will also be physiologically compromised in some way too. For example, a tissue that remains scarred after healing may have a less than perfect vasculature and this could have futher negative consequences. In considering pain states local adverse physiology must be interpreted in terms of continued effects on the firing and sensitivity of sensory nerve endings in the neural connective tissues and on any local damaged nerve fibres.

Pain Mechanisms Relating to Injury of Nervous Tissue

The nervous system is a special tissue in that it must be considered as having two distinctly separate mechanisms that cause pain when it is physically injured (see Chapter 5 for review of mechanisms):

1 A nociceptive mechanism, which is due to injury and/or stimulation of the sensory endings of nerve fibres in its connective tissues; for example, injured and inflamed dura, in the spinal canal, or perineurium in a peripheral nerve trunk.
2 A neurogenic mechanism, which is due to injury and alteration of sensitivity of the conducting fibres themselves. *'Peripheral neurogenic mechanisms'* consider abnormal responsivity of peripheral neurones, and *'cental neurogenic mechanisms'* consider lesions in the central nervous system as well as abnormalities of information processing (see Chapter 5 for expanded discussion) (Devor, 1996).

Clinically, the fact that several different mechanisms of pain production can operate when the nervous system is injured may in part account for the very complex pain patterns, pain qualities and pain behaviour that is so often seen.

Injuring Forces

Peripheral nerve trunks and roots can be injured and hence become pain sensitive as a result of direct mechanical force or as a result of alterations in pressure around the nerve. The key to understanding the influences of alterations in pressure (vascular factors) and direct mechanical injury really revolves around a consideration of length of time of the forces involved. As a generalization, sudden high forces cannot be tolerated well by the nervous system and, like any other tissue, lead to injury, inflammation, healing and repair plus or minus a pain state. More sustained forces can be tolerated surprisingly well, especially if the load is dispersed over a wide area. Long-term focal increases in pressure may not be tolerated without some expense. Alteration of normal circulation by pressure change may lead to degenerative changes in nerve tissue *without* necessarily any inflammation (Olmarker *et al.*, 1995) and more importantly *without* necessarily any pain. This is a rather 'occult' form of pathophysiology that is probably quietly accumulating in all of us as we progress through life.

SUDDEN MECHANICAL FORCE

Sudden mechanical force considers adverse stretching/compression of nerve, for example:

- Sudden twisting of an ankle which may injure the superficial peroneal nerve over the ankle (Nitz *et al.*, 1985) or as far proximally as the lower part of the thigh (Nobel, 1966);
- Any sudden direct physical force on nerve, for example an injury to the radial nerve following a fracture dislocation of the radial head;
- A whiplash involves huge forces that may well rip and squash many major or minor nerve trunks and plexi (Jeffreys, 1980), as well as the cord (McMillan and Silver, 1987) and brain (La Rocca, 1978).

ONGOING MECHANICAL FORCE

As already discussed, ongoing mechanical forces result in the disruption of the subtle pressure gradient required to maintain adequate vascular supply to nerves. The spectrum of physical factors that can influence supply range from normal postural forces and ongoing inactivity to pathological intrusions such as disc protrusions, osteophytes, thickened ligamentous, muscular and tendinous tissues, scar tissue, oedema and inflammatory exudate and haematoma (Butler, 1991; Butler and Gifford, 1998).

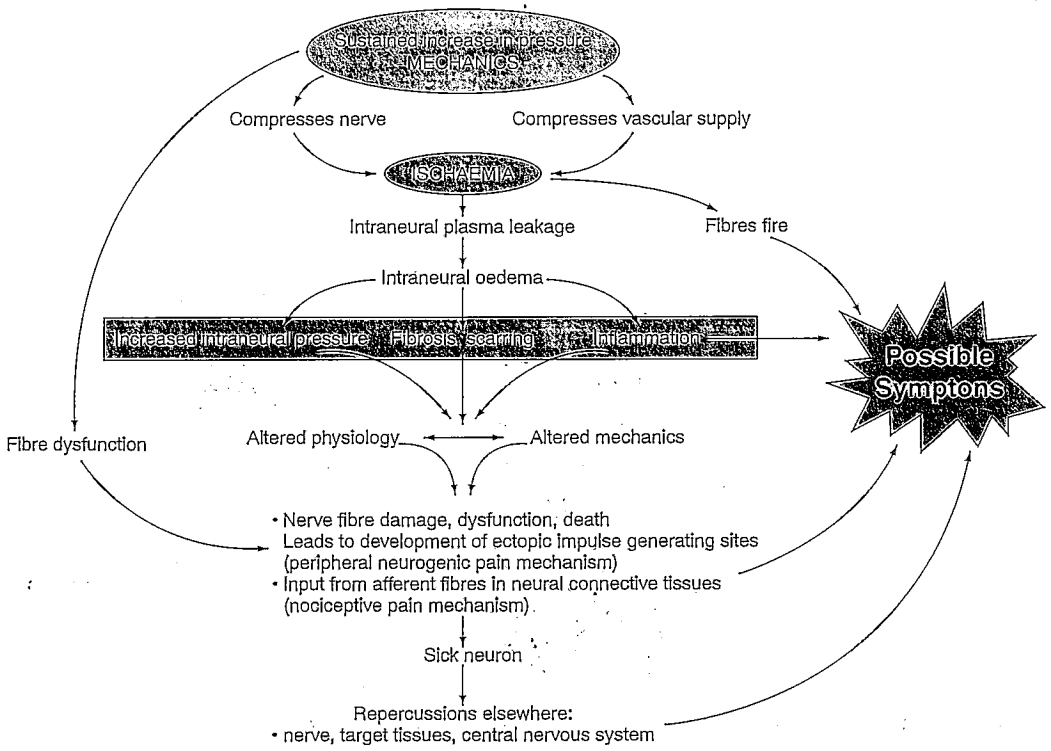
Local pressure changes affecting the immediate nerve may not be the sole influencing factors. Thus, consideration of vascular 'control systems' focuses attention on sympathetic tone to supply vessels – if tone is increased the tissues in the supply field will suffer. There is also the possibility of pathologies in more remote regions adversely influencing the supply of blood to the area. This could range from heart disease (LaBan and Wesolowski, 1988) to localized adverse pressures on supply vessels due to for instance sustained postures, tight muscles, scar tissue, tumours or simple swelling.

The Repercussions of Neural Ischaemia

Loss of blood to a nerve results in local hypoxic or ischaemic conditions (Figure 4.19). This in itself may cause nerve fibres to start to fire ectopically and produce abnormal sensations that include paraesthesias, numbness and pain depending on the type of fibre affected and the central nervous system processing of the abnormal impulse discharges that are generated. Continuing hypoxia leads to plasma leakage from the endothelial walls of capillaries within the nerve itself (Lundborg, 1988; Lundborg and Dahlin, 1996). This intraneural oedema formation leads to a further increase in pressure within the nerve that adds to the problems of inadequate circulation. The nerve may even swell proximally or distally. The end result, originally proposed by Sunderland (1976), is that this protein-rich oedema promotes the formation of fibrosis both around the fascicles and within them. This must have detrimental affects on the normal mechanics and physiology of the connective tissue as well as on the health of the nerve fibres themselves.

It is easy to see how local loss of circulation can lead to dysfunction and damage to nerve fibres and even to their death (Jayson, 1992). Rather puzzlingly, this may or may not lead to a pain state. Examination of lumbar nerve roots post mortem often reveals quite severe nerve fibre damage and fibrosis yet the patients' medical records make no mention of back-related pain problems (Hoyland *et al.*, 1989). On the other hand, conditions like those describe must surely be adequate to produce or predispose to a neurogenic pain state. Ultimately, or at least from a purely tissue-based perspective, it depends on whether the physiological changes are enough to sensitize and fire

Figure 4.19 Pathobiological effects of compression on nerve.



nociceptors or create ectopic impulse generating sites in damaged nerve fibres (Hasue, 1993). The other perspective to consider is that many of us may actually have many nociceptors and ectopic impulse-generating sites firing, but our CNS chooses to pay no attention to the activity.

The Importance of Inflammation

Inflammation, or at least the presence of irritative chemicals, may be a vital determinant as to whether or not a neuropathy is painful. There may be a marked difference in the chemical environment of a nerve that is being slowly compressed by developing degenerative invasion and one where inflammation has occurred due to a more acute injury or where local interfacing tissue has been damaged and becomes inflamed.

Disc disruption is a powerful example of the many diverse influences on a nerve tissue.

- Any disc protrusion *may mechanically* damage the adjacent nerve root – compression of the dorsal root ganglion is likely to be immediately symptomatic since it is normallly mechanosensitive. Neural connective tissue may be damaged (perineurium, dura) which leads to an inflammatory response in this tissue and more than likely a pain state.
- The protrusion, even without contacting the nerve root tissue, may increase the pressure around the nerve and cause an increase in venous pressure. This may be sufficient to

produce ischaemic conditions both around and within the nerve – a situation that has the potential to lead to inflammation within the nerve (Hoyland *et al.*, 1989), or more likely to oedema and fibrosis in and around the nerve as described above (Cooper *et al.*, 1995).

- The disc injury itself may precipitate inflammation in the immediate environment of the nerve. Leakage of nuclear fluid is thought to create an inflammatory response (McCarron *et al.*, 1987; Saal, 1995), and chemicals so produced may get rapidly transported into the local nerve (Byrod *et al.*, 1995) and thus produce an intraneural pain-generating environment (Olmarker *et al.*, 1993, 1994).
- As a result of possibly profound intraneural physiological changes, nerve fibres become damaged and develop abnormal ectopic impulse generating sites that can be responsible for ongoing and often very disturbing pain states (see Hasue, 1993, for useful summary).

In summary,

- Acute disc injury has many potential repercussions when creating a pain state – from nociceptive mechanisms in damaged tissues of the disc and adjacent tissue, to peripheral neurogenic mechanisms as a result of direct or indirect nerve root irritation and damage (see Chapter 5). The mechanisms are complex and may develop over time which warns clinicians to progress cautiously with very early management of low back disorders.
- Physiotherapists should be aware that disc injury related pain and therapy is more complex than discs mechanically 'going out' and being 'put back'. Appreciation of the complexity and timing of the recovery process and the conditions that enhance the natural resolution arguably promote a rehabilitative physiotherapy approach coupled with a more rational use of the best that modern medicine can offer.

Implications of Peripheral Nerve Pathophysiology

The wider implications of any nerve injury seem quite daunting (Figure 4.19):

- Impulse barrages from ectopic impulse generating sites are potent modulators of central nervous system sensitivity (Chapter 5).
- Damage in one area of a nerve has been shown to influence the sensitivity and health of distal or proximal areas on the involved and related nerve trunks (Mackinnon, 1992). Thus healthy neurones effectively become 'sick neurones' and their seemingly malevolent influences can spread.
- Loss of, or poor communication, between the neurone nucleus and its target tissues may have dire consequences for target tissue health (see above).

Nerve contraction, loss of elasticity or any tethering as a result of pathophysiological processes may have far-reaching adverse mechanical influences on related tissues. For example, it is easy to visualize how a tethered, fibrous and inelastic segment of a peripheral nerve trunk would put more strain on its proximal and distal segments during movements that stretched it. Adverse mechanical tension in one area may thus lead to pathophysiological processes with the potential to become the source of symptoms at distant sites (Breig, 1978; Butler and Gifford, 1989, 1998; Butler, 1991). Thus, it is not uncommon to find that following a single neuropathy other pain states later crop up in 'neurally' related areas. For instance, carpal tunnel syndrome has been strongly related to nerve injury in the neck.

- Upton and McComas (1973) found approximately 80% of patients with carpal tunnel syndrome or lesions of the ulnar nerve at the elbow had neural lesions in the neck. These authors termed the phenomenon 'double crush' syndrome, i.e. where a proximal nerve compression predisposes a nerve to pathology distally.
- The literature also describes reversed double crush (Lundborg, 1988) and multiple crush syndromes (Mackinnon, 1992) that really highlight how the health of a whole nerve can be affected by modest forces that start in one area. Spreading of symptoms and signs is very common in chronic pain development.

Mechanical injury and changes in pressures and circulation to the spinal cord, brain and its lining connective tissues are less easy to study (Butler, 1991) than the peripheral nervous system. There is no reason not to assume that issues similar to those that influence peripheral nerves could operate in the CNS (see Butler, 1991; Butler and Gifford, 1998).

From Theory to Practice: Neural Sensitivity

Pathological Mechanosensitivity

Clinically, mechanosensitivity can be seen as an immediate symptom response that bears a direct relationship with a physical force. Nerves can be influenced mechanically by any movement, static muscular contractions, even the pulsating of arteries if extremely sensitive (Butler, 1991; Butler and Gifford, 1998).

It may make clinical analysis easier to think in terms of movements that mechanically stretch nerves – as in the neural tension or 'neurodynamic' tests (Shacklock, 1995), and those which compress nerves – as in the closing of the intervertebral foramina in spinal extension or ipsilateral rotation/side-flexion movements, or the compression of the median nerve in the carpal tunnel by wrist flexion or extension.

Butler (1996) has used the terms:

- 'Container dependent' for symptoms evoked by compressive effects;
- 'Neural dependent' for symptoms evoked by neural elongation/neurodynamic tests like the slump, SLR and ULTT (see Chapter 6).

Analysis of symptom-provocative postures and movements with these thoughts in mind is often very useful.

The frequent straightforward link between a performed movement or posture and symptom reproduction are often complicated by the fact that impulse discharges from ectopic impulse generators in mechanosensitive segments of nerve respond in a great variety of ways (Devor, 1994; Butler and Gifford, 1998). For example:

- You do a test, it hurts for a couple of seconds and then disappears, you repeat the test and nothing happens. Five minutes later you repeat the test and it responds again, and so on.
- A test may produce a fleeting symptom immediately tension or compression is applied and then again when the force is removed, there being no response when the force is maintained.
- A test may produce a response in parallel with the force applied but continue long after the test force is removed.

Odd and clinically frustrating reactivity like this makes one suspect a peripheral neurogenic pain mechanism (see Chapter 5).

Pathological Ischaemosensitivity

Further evidence of sensitivity is found when a delay and then a slow build-up and spread of symptoms is produced. This may be an indicator that the nerve is more ischaemosensitive than mechanosensitive, especially when the test performed could well be adversely influencing the pressure in the nerve or the circulation to it (Butler and Gifford, 1998).

- A classic example is an acute or subacute cervical or lumbar nerve root problem where sustained rotation towards the side of pain is at first symptom free but then after a few seconds a slow build-up of discomfort occurs that frequently spreads down the arm/leg to produce distal paraesthesia.
- Carpal tunnel syndrome symptoms are often heightened by Phalen's test. Here, sustained wrist flexion slowly produces an increase and spread of symptoms.

Further Considerations

Many peripheral nerve problems are far from predictable, are ongoing in nature or occur spontaneously often with agonizing ferocity that can be very worrying to the patient. There are many possible explanations that can be derived from current tissue pathophysiological knowledge. For example:

- A consideration of mechanisms that produce 'ectopic pacemaker' capability must be included (see Chapter 5).
- Ongoing normal or pathological pressures on sensitized nerve may be factors. Consider oedema, muscle tone, haematomas, or any pathology/defect/congenital defect that could occur adjacent to sensitized neural tissue and that will put pressure on it or its vascular supply.
- Many ectopic impulse generating sites have increased sensitivity to circulating chemicals. Thus, anxiety and 'stress' may increase pain/symptoms since ectopic sites can become sensitive to adrenaline and noradrenaline released as a result of sympathetic nervous system activity (see Chapter 5 and Devor and Rappaport, 1990; Devor, 1994, 1996).

Neural mechanical sensitivity and ischaemosensitivity can be investigated by analysing pain quality and pain behaviour, pain response to normal movements, sustained movements and responses to tests that attempt to focus mechanical forces on neural tissues. These include tests that have a bias to elongation and tension (see Chapter 6) and to those that emphasize compression via joint movement or via direct 'palpatory' techniques (Butler and Slater, 1994; Butler and Gifford, 1998).

Restricted Range of Neurodynamic Tests – A Wise Stance?

Physiotherapists must consider the nervous system as capable of becoming a very mechanically sensitive system and rarely one that becomes so mechanically compromised that it is *physically* responsible for *major* losses of range of movement. It can be argued that in the majority of cases it limits range by producing pain/symptoms that then call a halt to movement via the active will of the patient and reflex protective muscular contraction in concert.

This now has support from current work reported by Elvey (1995) and Hall *et al.* (1995) that demonstrates increased muscle activity during straight leg raising in patients with sciatica.

They found that the electromyographic (EMG) activity of the hamstring muscle increases in parallel with the pain response felt by the patient.

The key point is that the nervous system, especially if it has become injured and hence sensitized, mobilizes adaptive motor protective mechanisms if it is physically challenged.

A blocked straight leg raise in a subject with chronic sciatica may be a point of argument for some, in that the block to movement can be seen as being due to tethered and noncompliant lumbosacral nerve roots, especially if sciatic pain is reproduced. The root may well be tethered and not moving as well as it once did, but can this tethering be responsible for as much as 30–40° of loss of SLR range?

Consider that:

- The amount of movement observed in fresh cadavers in the lumbar nerve roots is at best 10 mm (Goddard and Reid, 1965) (Figure 4.13), which is hardly enough to cause such a large loss of range.

It seems more likely that the nervous system may protect itself by:

- Becoming mechanically sensitive;
- Inducing powerful protective reflexes in the hamstrings/glutei when mechanically threatened;
- Later, more long-term protective processes may be added, e.g. adaptive physiological/mechanical changes in the hamstrings/glutei, the nerve root itself and other relevant tissues.

Thus, in the acute and subacute situation loss of range is related more to physiological processes and pain responses (sensitivity), and in the more chronic situation is related to both neural sensitivity and some mechanical compromise of multiple tissues that includes the nerve roots.

It is complex and often difficult to sort out clinically. The easiest way would be to place the patient under a general anaesthetic and observe the range devoid of any pain response or protective muscular activity. Although this is rarely feasible it does occasionally help to think when examining a patient, 'would this range be normal if there was no pain?' Ultimately, the nearest we can get to the answer comes from the highly skilled analysis of the 'end-feel', or the resistance felt during testing and the rate of improvement of range when attempts are made to mobilize it. However, Butler reports (1996) that he has had the opportunity to examine a few *apparently* mechanically 'blocked' straight leg raises under general anaesthetic and was surprised to find some quite normal in range and yet others that were indeed tight and blocked. Our thoughts must always be open.

The purpose of making these points about mechanical block and sensitivity is that there are great dangers in viewing disorders labelled as neural 'tension' solely in terms of mechanical compromise of the nerve, and that pushing hard into resistance at end of range is a necessary procedure to overcome a pain problem that presents with limited range of motion of a neruodynamic test.

Even if a nerve is mechanically compromised there are many inherent dangers in strongly mobilizing it since it is likely to be far more physically vulnerable and more easily resensitized than it was before injury.

Understanding Neurally Safe 'Stretching' or Mobilizing

It is possible to move into tissue resistance in 'neurally safe' ways.

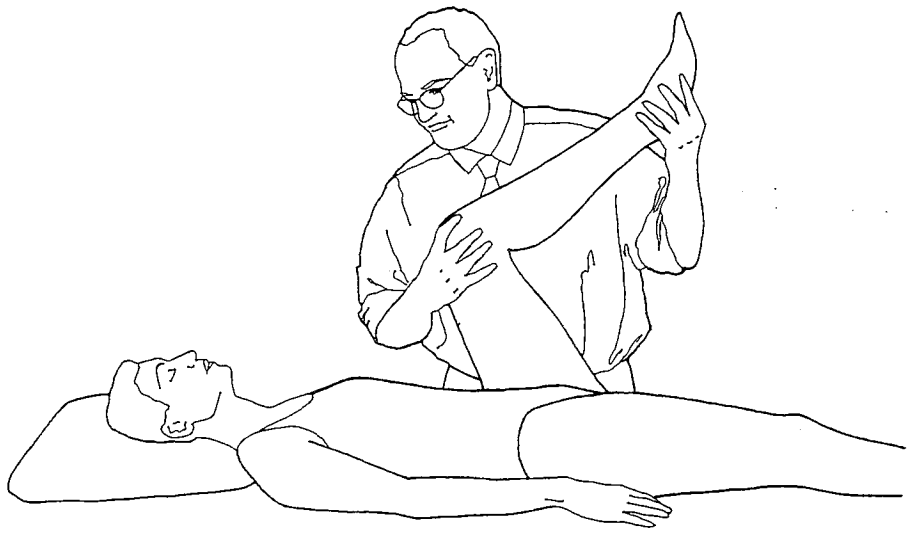
Practical Task

Try this simple test observation on anyone who has difficulty reaching their ankles with straight knees in the standard forward-bending test and who has no symptom problems at all.

- *Perform a standard SLR test and ask carefully about their symptoms at a reasonably firm end-range position – the quality and distribution of the symptoms are important. In my experience there is commonly a deep diffuse aching sensation distributed anywhere from the back of the hip, back of thigh, back of knee into the calf and sometimes into the foot. Get the subject to focus on the symptoms and remember them so that they can be compared to the next test.*
- *On the same leg perform hip flexion to around 100°, add knee extension into modest resistance but do not allow it to go to full extension. If the knee does reach full extension take the hip into further hip flexion and add the knee extension again (see Figure 4.20).*
- *Keep the knee in exactly the same position while further flexing the hip and ask about the area and type of response in comparison to the first test. The classic response to this test is a well localized pulling sensation in the hamstring muscles – it 'feels' muscular.*

It seems that in the last position muscle tension and a muscle tension sensation come into play far earlier than neurally related tension and symptom response. The concept then is to use mobilization techniques that tend to produce more muscle tension feelings rather than neural ones.

Figure 20 Hip flexion test with knee short of full extension (see text for explanation). (Reproduced, with permission, from Butler and Gifford, 1998, *The Dynamic Nervous System*. NOI Press, Adelaide.)



Practical Tips

It is quite often possible to mobilize a chronic or subacutely restricted SLR into resistance by going into flexion with hip abduction and external rotation and knee flexion as described above – the patient feels hamstring pulling, no discomforting neural symptoms, and small progressive gains in range can be made. The patient can also perform home stretches utilizing similar components. For example, in sitting, symptomatic leg forward, heel on floor, foot in neutral, knee in 15–20° of flexion, hip in abduction and modest lateral rotation – perform gentle flexion movements from the back and hips.

In the SLR with the knee-flexed position described it is often possible to see the tibial division of the sciatic nerve standing out at the back of the popliteal fossa. It is certainly easy to palpate here even if it cannot be seen (Figure 4.21). The nerve in this position is very tight, and is a reasonable indication of quite marked dynamic changes along the length of the nerve that probably includes the nerve roots. However, in terms of symptom response, it seems that SLR with a fully extended knee is far more 'nerve provocative' than when the knee is flexed 15–20°.

Hopefully this illustrates one way of addressing resistance in a neurally safer way, i.e. perform the restricted movement into resistance so that a sensation of muscle stretch is produced (knee slightly flexed SLR) rather than one that produces strong neural symptoms (knee extended SLR). The essential element of safety is provided by focused symptom enquiry and analysis and cleverly adjusting starting positions.

Figure 4.21 The tibial and common peroneal nerve at the back of the knee are sometimes very visible in the position shown in Figure 4.20. The examiner's thumb is on the lateral aspect of the subject's right knee. The peroneal nerve lies medial to the biceps femoris tendon and the tibial nerve runs centrally into gastrocnemius. (Reproduced, with permission, from Butler and Gifford, 1998 *The Dynamic Nervous System*, NOI Press, Adelaide.)

However, we should always be aware that symptoms reporting by consciousness is not always as accurate a reflection of what may be happening at tissue level as we would wish (see Chapter 5).

Restoring range of movement is an important tenet of physiotherapy that should be achieved in the safest possible way. Since it is argued here that in the majority of patients neural sensitivity issues are much more dominant than mechanical block to neural movement issues, it is suggested that physiotherapists use neurodynamic techniques and exercises:

1 With thoughts of promoting better physiology to help decrease sensitivity and restore range;
2 With thoughts of slowly stretching/elongating related musculature that may be reflexly more alert, or modestly mechanically shortened, due to the reasoning discussed above.

Final Comments

Butler and I published a paper in 1989 entitled 'The concept of adverse mechanical tension in the nervous system' (Butler and Gifford, 1989) which with hindsight viewed and addressed disorders of the nervous system in a predominantly mechanistic way – just as joints and muscles were examined at the time. Sensitivity of the nervous system was addressed by attempting to judge the irritability of the system as per Maitland (Maitland, 1986). This is no longer adequate. New biologically based knowledge now allows reasoned judgements to be made on the class of pain a patient may be suffering (see Chapter 5 and Butler and Gifford, 1998), and provides an understanding of the complex issues of sensitive neural tissues and their clinical correlates. For many patients, mobilizing neural tissue may well be as appropriate as mobilizing any tissue. However, under an expanded reasoning framework (see Chapters 5, 6 and Butler and Gifford, 1998) that pays due attention to the manual therapist's findings in relation to the pathobiological process involved in the patient, modern management now calls for better and gentler techniques, decisions on hands on or hands off, more patient self-management, empowering the patient with knowledge, and techniques and exercises done with the full understanding of the patient and therapist (see Chapter 15).

These are exciting times for physiotherapy. It is essential that clinicians take a forward step from the old mechanistic systems of manual therapy and integrate the pain-related sciences to provide a better and safer delivery of physiotherapy whose aims are to reduce pain, relieve suffering, restore range and enhance overall function.

Conclusions and Key Points

- The nervous system moves and this is reflected in many of its anatomical features.
- Movement of the nervous system is not at all simple. Over and above many fundamental gross movement features and principles there is great variability between one individual and the next.
- The nervous system is designed to cope with remarkable elongation and compression effects due to posture and movement. Sudden elongation or compression forces extend the nervous systems' safety adaptations to their limits and may easily cause injury. Long-term compression/elongation is detrimental too, but the system has time to adapt. Thus many pathological abnormalities are commonly found that *may* have no symptomatic sequelae.
- The nervous system has modest normal sensitivity in some areas, but if injured it can become the source of very disabling pain and hypersensitivity states.
- Treatment reasoning models based purely on mechanics of tissues without due respect for pathophysiology, enhanced sensitivity and a diagnostic framework that includes analysis of pain mechanisms, are arguably inadequate and incomplete.

FURTHER READING

Current texts

Butler, DS, Gifford, LS (1998) *The Dynamic Nervous System.* NOI Press, Adelaide.
Probably the most up-to-date in-depth examination of neurodynamics and pain relevant to clinical diagnosis and physiotherapy management.

Classic texts and chapters:

Breig, A (1978) *Adverse Mechanical Tension in the Central Nervous System.* Almqvist and Wiksell, Stockholm.
The classic pictorial atlas illustrating movement of the nervous system in freshly dissected cadavers.

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