Adverse Mechanical Tension in the Nervous System: A Model for Assessment and Treatment

The functional anatomy of the nervous system includes mechanisms to allow adaption to body movements. Injury or impairment of these mechanisms may lead to symptoms. Clinicians using tension tests as part of assessment and treatment have noted that altered nervous system movement and extensibility is a very frequent finding in many disorders.

This paper describes a new model for assessment and treatment of mechanical disorders of the nervous system that is based on clinical observations and interpretations of anatomical, biomechanical and pathological literature. A broad approach is outlined which provides an insight into the possible mechanisms by which the nervous system can be responsible for symptom production. The concepts of intraneural and extraneural pathology are put forward and related to assessment and treatment.

DAVID S. BUTLER

David Butler, B.Phty, Grad.Dip.Adv.Manip.Ther., is a private practitioner in Adelaide, and a tutor on the postgraduate Diploma in Advanced Manipulative Therapy course, South Australian Institute of Technology, North Terrace, Adelaide.

This article includes some material and diagrams previously published in the Proceedings of the Fifth Biennial Conference of the Manipulative Therapists Association of Australia, Melbourne, 1987, and reprinted with permission. The assistance of Megan Dalton and Paul Ryan in preparation of this paper is gratefully acknowledged.

'Neural tension' as a possible source of a variety of signs and symptoms has attracted much attention in recent years. In Australian manual therapy, the Slump test (Maitland 1978) and the Brachial Plexus Tension test (Elvey 1979) now rank with other well established 'neural tension' tests such as the Straight Leg Raise (SLR), Prone Knee Bend (PKB) and Passive Neck Flexion (PNF). Initially these tests were used as diagnostic aids; for example, a positive PKB was thought to implicate an L3 disc lesion (Estridge *et al* 1982). However, judicious use and variations of the tests can also implicate mechanical disorders of the nervous system as a component of many common 'musculoskeletal' disorders. The tests and their derivatives may also be used as treatment techniques.

The model presented in this paper results from the interpretation of clinical findings with as much assistance from the available literature as possible. Rather than the anatomically incorrect term 'neural tension' (since the connective tissues of the nervous system are tensioned far more than the neural elements), adverse mechanical tension in the nervous system (AMT) (Breig 1978) is considered a more correct term.

This model applies to the entire nervous system - peripheral, central and autonomic components - and regards it as a continuum. The nervous system has two intimately related components: a *connective tissue* component and a *neural tissue* component.

Anatomy - The Movement Role of the Nervous System

The connective tissues protect the neural component of the nervous system in a manner that allows impulses to be sent while the body is in any desired posture or movement. These tissues also protect neurones from compressive forces.

The importance of the dynamic role of the nervous system is emphasized by the specialized anatomy of the connective tissue components. Spinal dura mater, with layered and axially directed collagen fibres, possesses great axial strength and also a little elasticity (Tunturi 1977, Breig 1978). Collagen fibres of the pia and arachnoid are arranged in a lattice pattern that allows lengthening and shortening (Breig 1978). Differing collagen arrangements in peripheral nerve appear to give the perineurium and endoneurium tensile strength with the epineurium more resistant to compressive forces (Sunderland 1978). Tension in peripheral nerves is transmitted to spinal dura mater via the perineurium which is continuous with the dural sleeves, and to epidural tissues via the epineurium (Murphy 1977, Sunderland 1978). As well as protection via the meninges, spinal cord tracts and peripheral nerve fasciculi are wavy and folded and allow elongation when stretched (Breig 1978). Segmental pairs of denticulate ligaments linking pia mater to dura mater allow tension dissipation and effectively suspend the cord centrally in the spinal canal (White and Panjabi 1980). As well as its nutritive role, cerebrospinal fluid cushions the spinal cord during movement (Louis 1981) as does the extensive network of flow reversible veins in the spinal canal (Penning and Wilmink 1981).

The nervous system is attached to surrounding tissue in a manner that allows the system some mobility yet can impart restraint. Attachments within the spinal canal, such as the dural ligaments from anterior dura to disc and posterior longitudinal ligament (Spencer *et al* 1983, Tencer *et al* 1985), and the dorsal plicae from posterior dura to the ligamentum flavum (Blomberg 1986, Savolaine *et al* 1988) (Figure 1), are quite specialized, apparently for a movement role. Considerable mobility of peripheral nerve within the nerve bed is allowed by epineurial attachments that differ in strength depending on the area of the body (Sunderland 1978).

Biomechanics

The nervous system must adapt to a wide variety of ranges, speeds and combinations of movement. It also has the anatomical structure to mechanically limit some movement combinations. For example, when seated and in full spinal flexion, the usual inability to extend the knees and dorsiflex the feet (Slump test) is thought to be due to the nervous system at its mechanical limit (Maitland 1978).

The nervous system adapts to and mechanically controls movement in two ways that invariably overlap:
- by the development of tension or pressure within the system.
- by movement relative to its mechanical interface.

The *mechanical interface* should be regarded as the most anatomically adjacent tissue to the nervous system that can move independently to the system. For example, the ligamentum flavum may be considered the mechanical interface to the posterior aspect of the dura mater or the supinator muscle as the mechanical interface to the radial nerve as it passes through the radial tunnel.

The relationship of the nervous system as a whole to interfacing tissue during movement is not the only movement warranting consideration. Within the nervous system, the neural component has a movement relationship with the connective tissue component, for example, movement of the cord in relation to dura mater, interfascicular sliding (Park and Watanabe 1985, Millesi 1986) or movement of myelin sheaths and Schwann cells in relation to the endoneurium. Breig's (1978) photograph makes an excellent summary of some of these adaptive mechanisms (Figure 2).

The adaptive mechanisms can be analysed further and are best explained by reference to the SLR test. It is well known that on performing a SLR, there is considerable caudad movement of lumbosacral nerve roots in relation to their intervertebral foramina (Goddard and Reid 1965), yet little consideration has been given to the rest of the sciatic tract. Smith (1956) has shown that the tibial nerve proximal to the knee also moves caudad in relation to its mechanical interface. Distal to the knee the tibial nerve moves cranially in relation to its mechanical interface (Figure 3). Thus there is a point posterior to the knee where the nerve/interface relationship is constant during movement. In this paper an area where the nerve/interface movement remains constant during movement is referred to as a *'tension point'*.

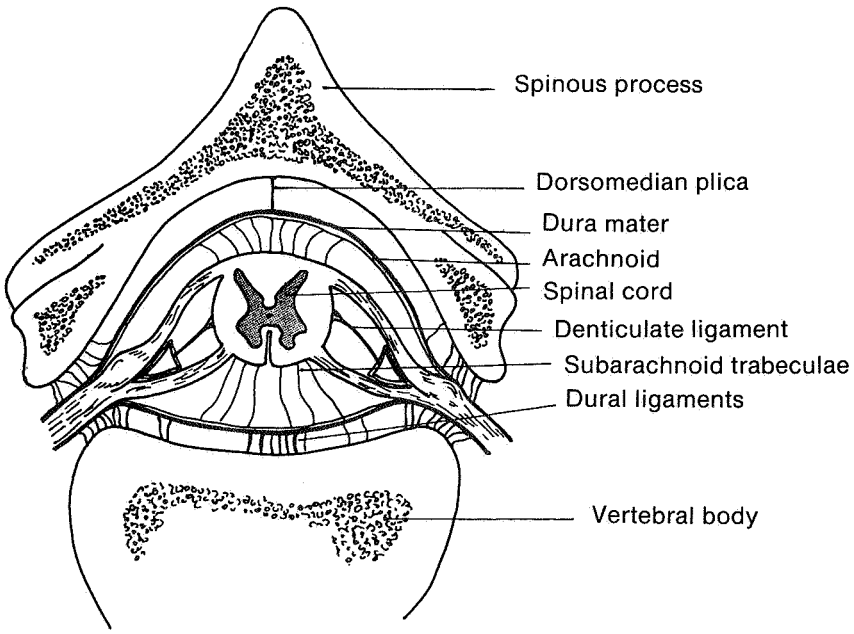


Figure 1: Diagrammatic representation of meningeal attachments in the spinal canal (transverse section)

Similar dynamics exist in the spinal canal to cater for the 5-9 cm length difference from flexion to extension (Breig 1978, Louis 1981). Extensive cadaver studies by Louis (1981) have shown directions of neuromeningeal movement in relation to the spinal canal and areas where the neuromeningeal/spinal canal movement relationship was constant during spinal flexion and extension. According to Louis (1981), these areas approximate C6, T6 and L4 (Figure 4). Of likely relevance to these tension points is that, at the L4,5 level, the ventral dura mater is usually firmly attached to the posterior longitudinal ligament (Blikna 1969, Spencer *et al* 1983). The exact location of tension points may well vary with the way in which tension is taken up during testing.

In the upper limb, McLellan and Swash (1976) placed needles in the median nerve and observed sliding of up to 2 cm in relation to interfacing tissue in the upper arm of volunteers during wrist and neck movements. Millesi (1986) pointed out that the median nerve has to be capable of adapting to a nerve bed made 20 per cent longer from wrist and elbow flexion to wrist and elbow extension.

Pechan and Julis (1975) were able to markedly affect intraneural ulnar nerve pressure at the elbow by altering shoulder and wrist positions with the elbow position constant. Using buckle transducers, Reid (1987) documented tension changes in the cords of the brachial plexus during the Upper Limb Tension Test (ULTT), a test presumed to test the mechanical integrity of the nervous system in the arm and neck (Elvey 1979).

It is very likely that tension points may occur in nerves at the elbow and shoulder during arm and neck movement combinations. Rubenach (1987) noted very little movement of the median nerve at the elbow during ULTT manoeuvres in a cadaver, and Sunderland (1978) has suggested that

where nerves branch or enter a muscle at an abrupt angle, movement was likely to be less.

In adapting to body and limb movements, the nervous system does not only move parallel to its interface. Penning and Wilmink's dissections (1981) have revealed the antero/posterior movement of the dural sac in the lumbar spine during flexion and extension and Apfelberg and Larsen (1973) noted dorso-medial sliding of the ulnar nerve in the cubital tunnel during elbow flexion and extension.

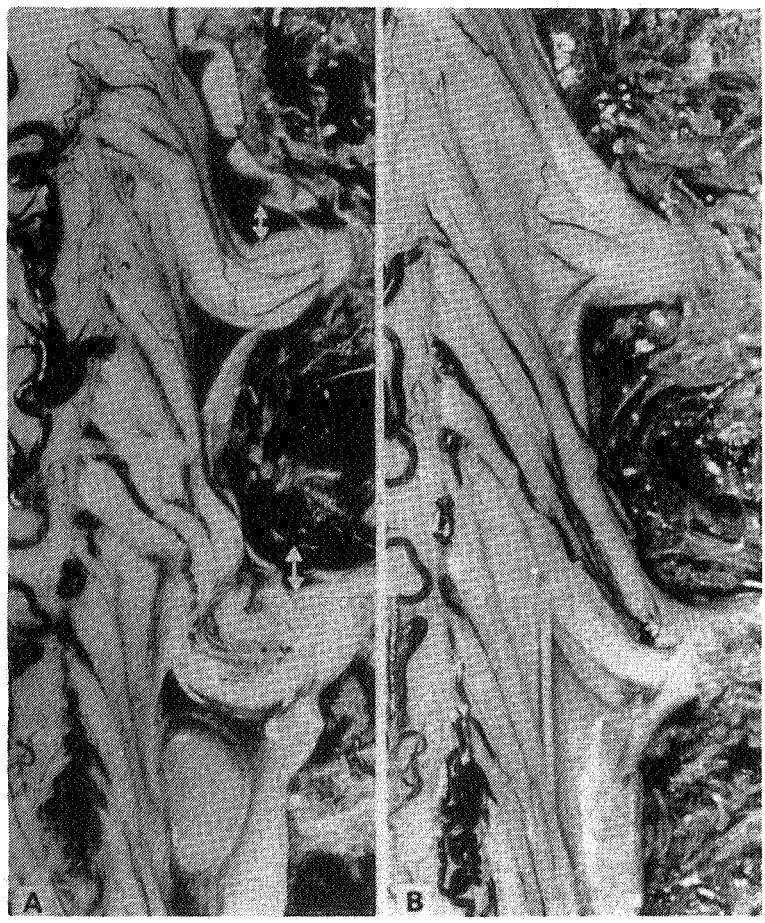
Overall the most important biomechanical consideration is that nervous system movement in one part of the body will create tension and movement in the nervous system in other more remote body parts. However, there will be less movement and tension development further away from the force application site as Tani *et al* (1987) have shown in cats and Reid (1987) in humans. It should be apparent that the nervous system can have a mobility of its own, independent of the interfacing tissues.

A clinically relevant but poorly researched area is that of the biomechanics of the autonomic nervous system (ANS). Breig (1978) demonstrated the considerable movement of the lumbar sympathetic trunk during a SLR and the ANS has been implicated as susceptible to injury in the extension phase of 'whiplash' in animal models (MacNab 1964).

Pathology

Clinically, it appears that there are vulnerable sites in the body where lesions that affect the elasticity and movement of the nervous system often begin. Soft tissue, fibro-osseous or osseous tunnels such as the carpal tunnel or the intervertebral foramen are such sites. Sunderland (1978) and Dawson *et al* (1983) also include areas where nerves branch, *eg* the radial nerve at the elbow, or where the nervous system is relatively fixed, *eg* the common peroneal nerve at the head of the fibula, or dura mater at the L4 spinal level (Blikna 1969). Many of these vulnerable areas could be considered as 'tension points'.

In patients with manually proven components of adverse tension, the



A. Extension. The dura, cord and nerve-roots in the cervical canal are slack: the root-sleeves have lost contact with the pedicles (lower arrows) and the nerve-roots with the inner surfaces (upper arrows).

B. Flexion. The dura, cord and nerve-roots are drawn out, the root-sleeves come into contact with the pedicles, and the nerve-roots with the inner surfaces of the sleeves.

Note that neural tissue moves in relation to dura and that dura moves in relation to canal. Tension changes are evident in the shape of the blood vessels. (Reproduced with permission from Breig A (1978), *Adverse Mechanical Tension in the Central Nervous System,* Almquist and Wiksell, Stockholm, p 17.)

Figure 2: Normal deformation of dura, cord and nerve-roots in the cervical canal in the cadaver due to full extension and flexion of the cervical spine.

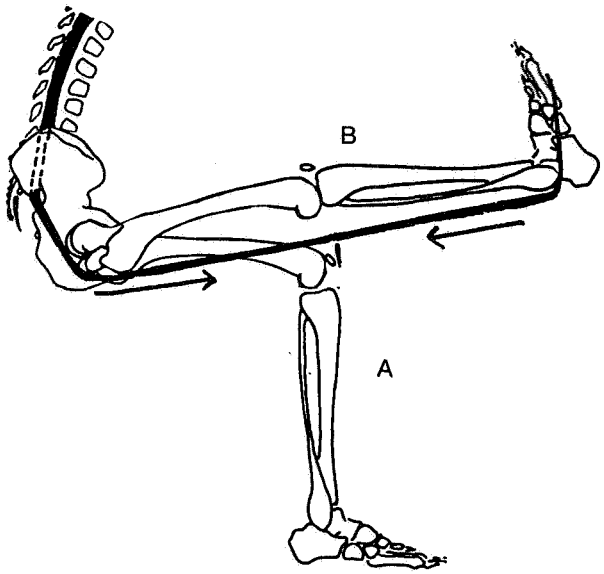


Figure 3: From position A to position B, movement of the tibial nerve in relation to the tibia and femur is the direction of the arrows. There is no movement posterior to the knee (adapted from Smith 1956)

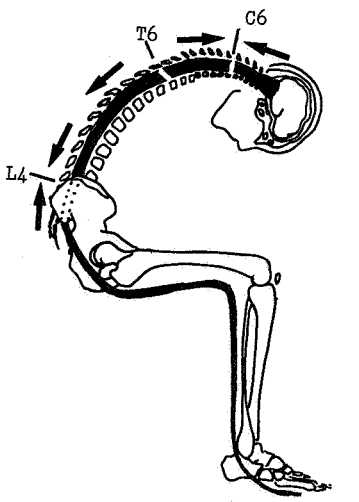
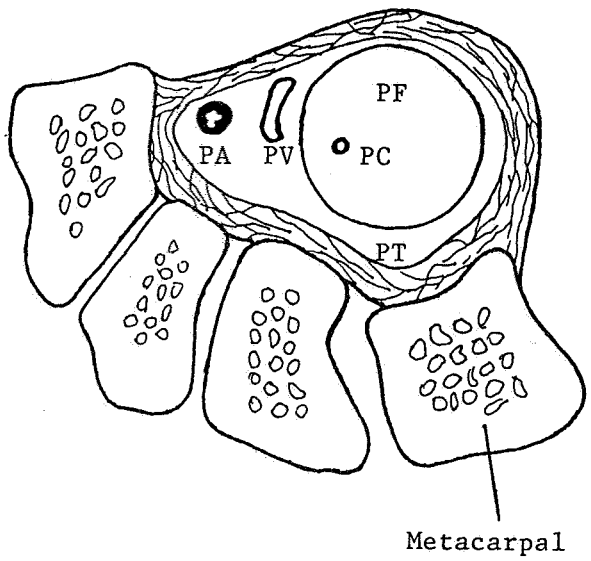


Figure 4: Movement of the dura mater and nerve roots in relation to the spinal canal during flexion. There is no movement at C6, T6, and L4 (adapted from Louis 1987)

area of symptoms frequently matches these vulnerable areas, especially early in the development of a disorder.

Sunderland's Pressure Gradients

Sunderland (1976) has documented a logical sequence of events that follows continued altered pressure in the carpal tunnel and considers that similar events could occur in other tunnel sites (Figure 5). Sunderland insists that for adequate intrafascicular circulation and thus neural function, a series of pressure gradients must exist within the tunnel. The pressure must be greatest in the epineurial arteriole, and progressively less in the capillary, fasciculus, epineurial venule and tunnel. The pressure gradient system is in delicate balance. If the tunnel pressure increases to greater than that in the venous system, then venous drainage will be impaired. Perhaps some clinical examples are: oedema in the carpal tunnel after overuse (Faithfull *et al* 1985), blood in the lumbar extradural space after a motor vehicle accident (Twomey and Taylor 1987) and altered anterior compartment pressure after overuse



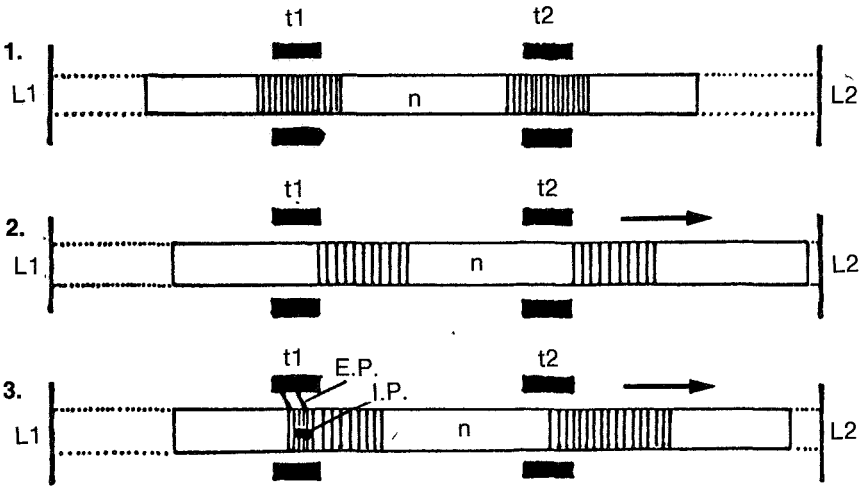
PA = pressure in the nutrient artery
PC = pressure in the capillary
PF = pressure in the fasciculus
PV = pressure in the vein
PT = pressure in the tunnel

Figure 5: Diagrammatic representation of one fasciculus of the median nerve in the carpal tunnel.

For adequate intraneural circulation, the pressure gradient must be
PA > PC > PF > PV > PT
(Redrawn from Sunderland 1976)

of the foot (Styf 1988). Another potent and underestimated effect is that of the sympathetic nervous system. Selander *et al* (1985) showed in rabbits that stimulation of the lumbar sympathetic trunk could reduce intraneural blood flow to less than 10 per cent. Lundborg (1988) suggests that sympathetically induced intraneural vasoconstriction may be an important factor in reflex sympathetic dystrophy and some chronic pain syndromes.

Sunderland details three distinct stages that may occur with persistent tunnel pressure - hypoxia, oedema and fibrosis. In the hypoxic state, embarrassed intrafascicular circulation causes impairment of nerve fibre nutrition and may lead to local pain. With continuing hypoxia, damage to capillary endothelium results in leakage of protein rich oedema. As perineurium is not crossed by lymphatics (Lundborg 1975), oedema cannot disperse and a further rise in intrafunicular pressure results. The nerve may swell, usually proximal to the damaged area and neuroma formation is quite common. Fibroblastic proliferation, enhanced by the protein rich oedema, results in intraneural fibrosis within the nerve. The increased volume of connective tissue again increases intraneural pressure and a self-perpetuating cycle of irritation may be established. Sunderland (1976) refers to the affected segment as becoming a 'fibrous cord' and a further development as 'friction fibrosis' elsewhere along the tract, most likely at vulnerable tunnel sites. Sunderland (1978) also considers that a 'friction fibrosis' may be more painful and damaging than the original lesion. In an area of friction fibrosis, intraneural fibrosis and extraneural tethering may be the eventual outcome of a similar sequence of events as in the original lesion (Figure 6).



t1, t2 = tunnel sites
n = nerve
L1, L2 = position that n can reach during normal movement with t1 and t2 constant

▥ = surface of n that can be in contact with t1 or t2 during normal movement

1. Neutral
2. to reach L2, n has moved in relation to t1 and t2 and increased intra n tension
3. with extraneural pathology (E.P.) and/or intra neural pathology (I.P.) at t1, for n to reach L2 requires increased intra n tension and increased friction as ▥ can now never be free of t2 during movement

Figure 6: The possible effect of intraneural and extraneural pathology at one site on other sites along the nerve trunk.

There is experimental as well as clinical support for these theories. Beel *et al* (1984) demonstrated in mice that damaged peripheral nerve rapidly increased strength and stiffness and showed decreased elasticity which the authors considered to reflect intraneural structural changes. Millesi (1986) notes similar findings in humans. Beel *et al* (1984) also emphasized that altered mechanical properties in one area of the nerve trunk (and thus altered mechanical interfacing) could lead to further damage to the whole nerve and to the mechanical interfacing. Earlier, Lishman and Russell (1961) used a similar explanation for the regular occurrence of symptoms along nerve trunks following 'brachial neuritis'. Further, McLellan and Swash (1975) who had the opportunity to view the considerable excursion of the median nerve in the forearm during arm movements, warned of the potential for friction lesions and the consequences for the entire nerve trunk. Based on their experiments using sciatic nerves from mice, Triano and Luttges (1982) suggest that 'intermittent mechanical agitation' from longitudinal sliding of a nerve trunk across an irritant is a major and underestimated factor in the production of inflammatory changes.

In the nerve entrapment literature, there are many references to the

'double crush' phenomenon, a concept introduced by Upton and McComas (1973). These authors examined 115 patients with either carpal tunnel syndrome or lesions of the ulnar nerve at the elbow and found that 81 had electrophysiological and clinical evidence of neural lesions at the neck. Dyro (1983) pondered on how 27% of young people (N=50) with brachial plexus lesions could develop carpal tunnel syndrome. Crymble (1968) had also noted this occurrence. Electrophysiological abnormalities were found in the ulnar nerve at the wrist in 46% (N=63) of patients with unilateral carpal tunnel syndrome and in 88% of patients (N=185) with bilateral carpal tunnel syndrome by Cassvan *et al* (1986). Similar percentages had been shown by Bendler *et al* (1977). Such findings are often considered due to 'underlying subclinical neuropathy' (Sedal *et al* 1973, Neary and Eames 1975, Silver *et al* 1985). Most authors refer to Upton and McComas' (1973) explanation of altered axoplasmic flow. It is suggested that an altered mechanical tension, either instigated by injury such as 'whiplash' or disease such as diabetes, could also be a precipitating factor. Sunderland (personal communication, 1988) considers such an explanation feasible.

The relationship of nerve anomalies and AMT should be considered. Werner *et al* (1985) demonstrated in 9 patients that pronator syndrome (high median nerve compression) was far more common when the median nerve pierced the humeral head of the pronator teres muscle rather than its usual passage between the humeral and ulnar heads of the muscle. Ten to twenty-five per cent of the population have ulnar and median nerve connections in the forearm according to Guttman (1977); fifteen per cent of the population have aberrant lumbosacral nerve roots (Kadish and Simmons 1984) and intradural connections between adjacent cervical nerve roots occur frequently enough for Marzo *et al* (1987) to consider them normal variations rather than anomalies. These may well have repercussions in the presence of, or development of, AMT.

A clinical paradox is occasionally encountered where a patient's signs and symptoms are obviously emanating from the nervous system, yet electrodiagnostic tests are negative. It must be realized that it is possible to have symptoms arising from irritated, even scarred connective tissues yet with unaffected or unmeasurable alterations in impulse conduction. An uneven distribution of fascicular pathology may also lead to normal electrical tests even if there is some nerve fibre involvement (MacKinnon *et al* 1986).

A time factor exists with the development of AMT. Once an intraneural fibrosis is established, there is likely to be an irreversible component to the disorder and treatment may be more difficult. Murphy (1977) warned of this with regards to nerve roots, Ford and Ali (1985) with nerve trunks and Fernandez and Pallini (1986) demonstrated, in mice, the vast proliferation of intradural connective tissue once an inflammatory process gained access to the dural sac. The nervous system will adapt very well to compressive and tensile forces if these forces are applied over a long time. However, a rapid force such as a motor vehicle accident or a bullet injury may cause huge damage even if the nervous system is not directly hit (Sunderland 1978). A force is created in the nervous system that is too rapid for all the adaptive mechanisms to be deployed.

In summary, it is postulated that the nervous system adapts to movement by the development of tension within the system and by movement in relation to its mechanical interface. Pathological processes may interfere with both of these mechanisms; *extraneural pathology* will affect the nerve/interface relationship and *intraneural pathology* will affect the intrinsic elasticity of the nervous system. It is suggested that once an area of AMT is established in the nervous system then the likely spread of symptoms is to an adjacent 'tension point' or to vulnerable areas.

The clinical consequences of such a hypothesis are not entirely clear. Most relevant literature dwells on nerve compressive injuries involving Wallerian degeneration, not an irritative lesion of connective tissues where neural conduction may be minimally, or not, affected.

The Mechanism of Symptom Reproduction

There is evidence that most of the connective tissues of the nervous system are innervated. Bogduk (1985) has confirmed earlier findings that the sinuvertebral nerve innervates the contents of the spinal canal. Hromada (1963) demonstrated that the connective tissue investments of peripheral nerve are innervated with sensory and autonomic fibres from local axonal branching. These tissues can thus be considered culpable for patients symptoms. There is no documentation of possible symptom referral from peripheral nerve although nerve root referral or 'radicular pain' is well known (Grieve 1981). The possibility of peripheral nerve referral should be entertained. It is considered that 'lines of pain', for example over the deltoid or medial wrist, may be examples. Sunderland (personal communication 1988) acknowledges such a possibility. There is only anecdotal (Cyriax 1982) and clinical evidence of the extrasegmental referral of symptoms from the meninges.

The nervous system displays a mechanosensitivity that is different to other tissues. Unsensitised nerves and neuromeningeal tissues may be moved and handled without response. However, if sensitised by mechanical or chemical means, only a very small stimulus is needed for a reaction (Smyth and Wright 1958, Triano and Luttges 1982, Loeser 1985). Triano and Luttges (1982) refer to the 'exquisite sensitivity' of irritated peripheral nerve. Clinically, nervous system mechanosensitivity may be evident in patients whose arm symptoms can be brought on by SLR, or those where palpation of the cervical spine can create a lumbar pain and vice versa. This relatively common clinical finding will usually be in the presence of a positive Slump test.

As outlined previously, inflammation of the nerve roots and/or peripheral nerves results in intraneural oedema which directly impairs nerve function as well as compromising the blood supply and venous drainage. Since a correlation exists between nerve fibre diameter and metabolic activity, the large diameter mechanoreceptors with

high metabolic demands are those fibres first and most severely affected by ischaemia (Bogduk 1980). The inhibitory action of the mechanoreceptor's afferent fibres is therefore lost and nociceptive impulses predominate. Bogduk (1980) also offers an alternative explanation in that ischaemia may damage the dorsal root ganglion which then becomes a source of pain. Perl (1976) states that inflammation can also decrease a nociceptor's threshold for depolarisation, causing weak and ordinarily harmless stimuli to be perceived as noxious. A concomitant increase in their baseline activity also occurs which could be perceived as noxious stimuli in the absence of any such stimuli.

Many disorders that can be clinically shown to have a significant component of adverse tension, such as 'whiplash', repetition strain injury or chronic low back pain, exhibit symptoms that may defy interpretation and perhaps lead to practitioner suspicion of their validity. Symptoms resulting from mechanical disorders of the nervous system may be easily explained on the basis of *local* and *remote* origins.

Possible local origins
- hypoxic, damaged, regenerating or immature axons
- connective tissue irritation (*eg* dura mater, epineurium, attachments)
- mechanical interface irritation.

Possible remote origins
- extrasegmental referral from the dura mater and nerve root sleeves
- mechanical interface referral (*eg* disc, zygapophyseal joint)
- referral from the autonomic nervous system

Once an altered mechanical tension is set up then both nervous system and interfacing tissue are more vulnerable. To further complicate the symptom presentation there may be an alteration of afferent input from non-neural tissue, there may be pain from postural syndromes and there may be the manifestations of chronic pain syndromes. If the patient's symptoms originate from any more than one of the above, then the clinical presentation may be complicated.

Assessment

There are many clues that allow identification of AMT as a disorder component.

Subjective Examination

Symptom Area and Nature
- Symptoms may present that do not fit familiar patterns and distributions. It was Cyriax's suggestion (1982) that when symptoms have no 'localizing value' then the nervous system should be suspected as a component. The clinician should also be aware AMT is often clearly demonstrable in many familiar disorders such as nerve root trespass or tennis elbow and may well mimic the 'capsular patterns' described by Cyriax (1982).
- 'Lines' of pain may be present often, but not necessarily over peripheral nerves (*eg* along the ulnar nerve in the upper arm or along the sciatic nerve in the buttocks). There may be vague 'clumps' of pain over known 'tension points'. The patient uses the whole hand to identify the area. The whole limb may ache. Some catching pains, burning pains and even very localized spot pains, such as in tennis elbow, can be manually proven to have all or part of their origins in AMT.
- The pains from a sensory nerve are more localized, less vague and less of a general ache than that from a primarily motor or mixed nerve (Koppell and Thompson 1976).
- The patient may use quite bizarre terms to describe the symptoms *eg* burning, crawling, strings pulling, strangling, dragging.
- Sensations of swelling especially in the web spaces of the hand, the MCP row and the feet are common complaints.

Behaviour of Symptoms
- May increase with known tension-altering positions such as getting into a car, reaching up to a clothes line, kicking a football.
- May demonstrate activity-specific mechanosensitivity. For example, typing, repetitive sports or musical instrument action. It is suggested in these cases that one small segment of nervous system has a symptomatic relationship in part of the total nerve/interfacing tissue relationship.
- May increase at night.
- May exhibit latency.
- May behave with interfacing tissue *eg* cervical zygapophyseal closing may aggravate arm symptoms; flexed lumbar spine could increase disc symptoms.
- May be postural; patient cannot settle into a static position and continually wriggles, or sustained posture may aggravate.
- When there is more than one symptom area, there may be a behavioural relationship in that all symptoms may come on together or the patient reports one area or the other.

History
- May be a history of severe trauma, such as a motor vehicle accident. Another example is a person slipping and falling directly on their buttocks, thus replicating the Slump position. Patients may not even consider the incident as related to the current symptoms. Clinically, it appears that an initial tension-creating incident can have occurred years previously. Pretensioning of the nervous system may predispose a person to symptoms.
- May be a history of repetitious use.
- Chronicity of symptoms.
- A history of 'jumping' or moving symptoms.
- Failure of other treatments. Traditional joint based treatments may not 'get at' a nervous system component effectively.

Objective (Physical) Examination

Posture
- The patient may prefer to adopt out-of-tension postures, such as slight hip flexion, abduction and lateral rotation, knee flexion and plantar flexion; cervical lateral flexion towards the side of pain; arm above the head; scoliosis.

Active Movement
- Nervous system lengthening movements aggravate (though can also be shortening). 'Irregular patterns' of active movement can suggest that structures other than joints are involved (Edwards 1985).
- It is the belief of the author that the 'feel' of passive accessory intervertebral movements (PAIVM) influenced by AMT may be different to the 'feel' of a joint restriction. The 'feel' doesn't

change during oscillation and has a through-range 'rubbery' kind of resistance.

• Certain anatomical sites are more likely to display palpable and testable changes due to their proximity to the nervous system. For example, the intervertebral joints, muscles such as the scalenes or supinator, the radio-humeral and superior tibio-fibular joints, and the transverse carpal ligament.

• A neurological examination may reveal changes in conduction although not in specific dermatomal or myotomal distributions. Often the whole limb can be weak or demonstrate a loss of sensation.

It may be possible, using the interpretation of subjective and objective examination to identify extraneural or intraneural components of a disorder (Table 1).

Table 1:
Signs and symptoms that may indicate extraneural or intraneural pathology

Extraneural	*Intraneural*
Catch or twinges of pain	Persistent symptoms
Short duration of symptoms	Increased duration of symptoms
'Lines' of pain	'Blocks' of pain
Through range pain and resistance with tension tests	End range pain and resistance with tension tests (tension applied from 'both ends')
Symptoms provoked by a tension test, then eased by tension at the 'other end' *eg* cervical flexion easing SLR symptoms	
	Neurological changes

Objective Examination: Handling and Interpretation of AMT: Expansion of the Routine Examination

• In interpreting tension tests, what is more important than positivity is the *relevance* of the symptoms provoked and the range of movements exhibited to the overall disorder presentation. For example, hamstring area pain on full slump that is only slightly different from left to right may well be relevant in a patient whose symptoms come on only after a full day's work or a long run.

A relevant tension test usually means that all or part of the patient's symptoms have been reproduced. (This may not yet implicate the nervous system). It can also mean that symptoms produced are different to what is known to be normal and, in the case of limb testing, different to the contralateral limb. There may also be an abnormal resistance to the movement when compared with the contralateral limb.

• Sensitising additions assist in implicating the nervous system. For example, adding hip adduction (Sutton 1979) or dorsiflexion (Breig and Troup 1979) to a SLR will increase tension in the tract. Sensitising additions require careful consideration of nervous system anatomy. For example, it is obvious and clinically apparent that the addition of plantar flexion and inversion will tension the common and deep peroneal nerves and perhaps replicate antero/lateral shin pain. The tension will be more if the limb is in SLR.

Sensitising or desensitising additions help in proving or disproving nervous system involvement, especially if the addition is some distance away from the symptom site. For example, if a SLR reproduces thoracic pain and ankle dorsiflexion further increases that pain, then adverse mechanical tension in the nervous system is a likely culprit, as thoracic spine structures other than nervous system have not been altered. Because of nervous system connections to other tissues and inevitable movement of some of these tissues during tension testing, all available subjective and physical evidence must be used in diagnosis. Essentially, it is 'making the features fit' (Maitland 1986).

• A relevant tension test does not necessarily indicate that there is a mechanical disorder of the nervous system. The tension test could be placing a force on a surrounding symptomatic structure. Another consideration is that part of the nervous system may be irritated and symptom provocative, but the mechanics are normal. Elvey (1987) makes the suggestion that nervous system mobilisation may have a physiological effect as well as a mechanical effect.

• A positive tension test gives the physiotherapist a valid reason to examine away from the symptom area and known referral sites. For example, with a Slump test including knee extension positive for headache, the entire spine may need examination. Sources of altered nervous system movement and tension may be a considerable way from the area of symptoms. Areas more relevant to adverse tension have been discussed.

• Using one traditional tension test for examining a limb is a crude method of examining nervous system mechanics. For example, it is not enough to examine the ULTT, find it negative and declare that adverse tension has no part in the patient's arm symptoms. Symptoms may not be in the shoulder abduction and elbow extension position. Subjective clues and a knowledge of biomechanics must be used to make up new tension positions to fit the test to the patient's complaint. Elvey (1986) and Butler (1987) have suggested different ULTT positions. More suggestions for better testing are included in the treatments section. Just as present day manual therapy encourages the examination of a joint in many directions (Maitland 1986), then so too the nervous system. Physiotherapists must also consider the possibility that changes in connective and neural tissue are likely to be present before the tension tests can be identified as relevant.

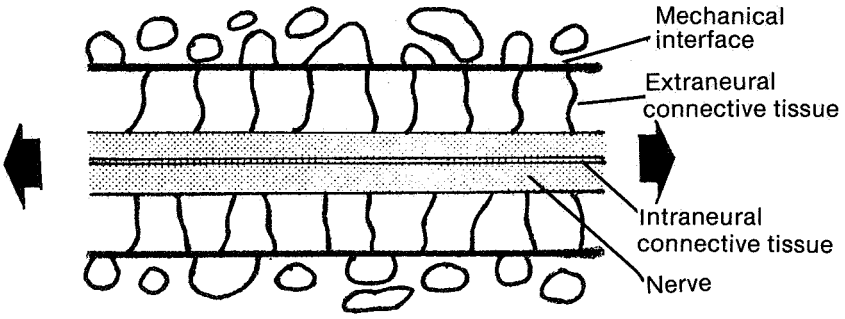
• Often with tension testing, 'the' pain or the entire pain cannot be completely reproduced. This may well be a manifestation of the complexity of nervous system anatomy and crudity of testing. It stresses the need to carefully interpret symptoms and the resistance encountered during testing.
• In complex, widespread and irritable disorders, the examination of distal components is recommended. For example, the effect of the contralateral arm or a SLR on arm pain should be examined.
• Palpation along nerve trunks can be useful as it may reproduce relevant symptoms.
• A neurological examination for all patients treated via tension tests is considered essential. Subjective and objective knowledge of cord function should also be considered mandatory prior to spinal cord mobilisation. This is not only for safety, but also for diagnosis and prognosis. Neurological signs can make excellent reassessments.
• Physiotherapists should be aware that they are capable of producing movement of nervous system in relation to mechanical interface and also an alteration in intraneural pressure or tension (Figure 7).

Treatment Via Passive Movement

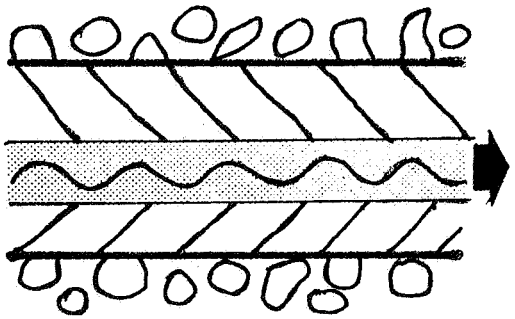
There are three ways to consider treatment of a disorder that involves AMT:
1. Selective nervous system mobilisation.
2. Treatment of interfacing and related tissues.
3. Indirect treatment – consideration of posture, ergonomic design of furniture and equipment.

1. Selective Nervous System Mobilisation

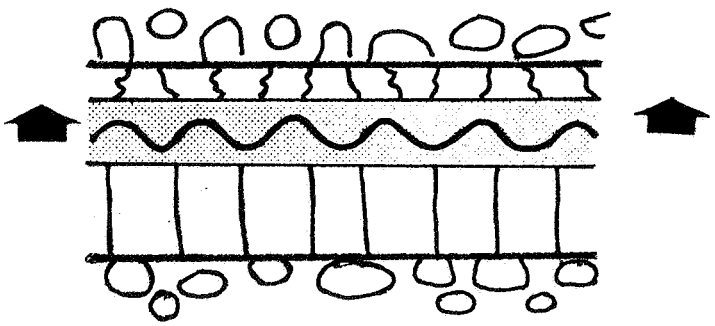
Nervous system mobilisation fits perfectly into the Maitland (1986) concept. Just as for a joint, the presenting irritability, severity and nature of the disorder are the essential components affecting the initial treatment decision. A key to successful treatment is to consider *mobilising* the nervous system, rather than just



Increased tension in the intraneural component, *eg* 'tension from both ends' as in the complete Slump test



Increased tension in the extraneural component, *eg* movement of the nerve in relation to the mechanical interface as in the SLR



Movement in another plane, *ie* tension in part of the extraneural component as in 'frictions'

Figure 7: Diagrammatic representation of passive movements of the nervous system available to examination and treatment.

stretching it. The all-encompassing term 'mobilisation', as physiotherapists use it with regard to joint treatment, encourages consideration to gentle and strong treatment, through-range and end-range treatments, movements in relation to pain and resistance and continual reassessment.

Consideration should be given to the 'nature' of the disorder. Extraneural and intraneural components may be identified and treated accordingly. It is logical that extraneural pathology may be better examined and treated with through range (Grade II and III) movements and attention to the interfacing tissue, and intraneural pathology more by grade IV movements. For definitions of grades consult Maitland (1986).

Treatment can be modified for the irritable and the non-irritable disorders. Many of the treatment techniques will be extensions of the techniques used in the objective examination.

The irritable disorder (treatment directed at pain)

As in a joint problem, it is possible and clinically effective to treat pain via nervous system mobilisation. Burton *et al* (1987) have shown that mobilisation of the SLR with regard to pain reproduction can lead to improved painfree range in normals.

- The suggested treatments are Grade II through range movements and Grade IV - - . The initial treatment may be as little as 20 to 30 seconds. Most interfacing tissue is muscle and better nerve/interfacing tissue movement will be possible if the patient is relaxed and comfortable.
- An indirect component could be selected. For example, in the case of left shoulder pain, SLR or right ULTT or left hand mobilisation may be the desired starting treatment.
- Progression is by repeating the same technique for longer, respecting pain less, or doing the same technique with another component altered to increase tension. For example, repeating Grade II shoulder depression with the cervical spine in some lateral flexion away from the symptomatic side.

The non-irritable disorder (treatment directed at resistance and pain)

- Treatment techniques will be more into resistance, *ie* Grade IIIs and IVs.
- Progression is the same as for the irritable disorder.
- Treatment positions away from the standard tension tests are likely to be more useful than the standard tests. Elvey (1986) and Butler (1987) have suggested alternative ULTT positions.

Physiotherapists are encouraged to make up their own tension tests by using subjective information, known biomechanics and 'hunting out' during the objective examination. It may be beneficial to use combined movements *eg* treat SLR in hip adduction and medial rotation, or to just treat the sensitising movement *eg* treat hip adduction in SLR. It is often useful to treat in differing joint/muscle relationships, *eg* treat SLR in lumbar rotation, the ULTT in scalenes stretch or to mobilise the radiohumeral joint while the arm is in an ULTT position. Treating and examining 'from the other end' *ie* taking up the distal components first, can be beneficial. If the treatment is done in an out-of-routine tension test position, then the routine tests, with their known normal responses make excellent reassessments.

- Axial and transverse tension considerations. Nervous system biomechanics dictate that, for optimum axial tension, lateral movement must also be free and vice versa (*eg* the ULTTs must be clear before optimum Slump).
- Speed of movement. Clinically, it appears that the results are better when the treatment is sustained (suggest up to 10 seconds). Increasing time is a way of progressing treatment and it fits in a small way to the temporal properties of the nervous system with regard to stretch. In some patients, replication of symptoms and thus treatment may require rapid movements.
- Accessible areas such as the ulnar nerve above the elbow may be treated effectively by frictions.
- Treatment pain. In the non-irritable disorder, no matter how strong the treatment, the pain caused should go within seconds of stopping. This may relate to the instant replenishment of blood to nerve fibres.

If pain persists, then the physiotherapist may have misjudged the assessment or another tissue may be affected. It may well be therapeutic (as it is for a joint) to do gentle through range movements after the treatment.

- Effect on other signs. It is a basic manual therapy tenet, and one that is crucial to continued learning, that if using selective tissue mobilising then the effects on other tissues must be re-assessed.
- Some examples of self mobilising via the nervous system are described by Butler (1987).

2. Treatment Via Interfacing and Related Tissues

In many cases, it may be better to 'get at' mechanical disorders of the nervous system by treating interfacing and related tissues, such as joint, muscle, or fascia. If so, then the relationship between signs must be continually reassessed. For example, in treatment of a limited SLR in the presence of joint signs at the L4 level, initial treatment of the L4 may give good relief, but results may plateau and require nervous system mobilisation and vice versa. Both components may have to be treated. It is suggested that physiotherapists learning treatment via nervous system mobilising, begin by treating related joint and muscle signs first and reassessing the effect on the nervous system.

3. Indirect Treatment

The application to posture should flow on from a knowledge of biomechanics and be as applicable in dynamic posture as it is in the unconscious patient. Butler (1987) has suggested some postural applications. It is logical that ergonomic design considerations should include the movement and tension mechanics of the nervous system.

Precautions and Contra-indications

[This list should be read in conjunction with Maitland (1986)]

Precautions

- Other structures involved in testing, for example, lumbar discs during the Slump test, symptomatic zygapophyseal joints during the ULTT.

• Irritability related to the nervous system. The inherent mechanosensitivity of the nervous system needs consideration. Clinically, it appears easier to aggravate arm symptoms than leg. Irritable disorders may demonstrate latency.
• Neurological signs. In chronic, stable disorders where nervous system mobilisation is possible, the neurological signs must be continually monitored.
• General health problems. Pathologies that affect the nervous system, for example, diabetes, multiple sclerosis, Guillian Barre. Recent surgery and medical considerations.
• Dizziness in cervical spine problems.
• Circulatory disturbances. (In many areas of the body, the nervous system is connected to the adjacent artery).

Contraindications

• Recent onset of, or worsening, neurological signs.
• Cauda equina lesions.
• Cord signs. Physiotherapists treating via Slump and SLR should be aware of Tethered Cord Syndrome (Pang and Wilberger 1982).

Conclusions

A model of adverse mechanical tension in the nervous system, based on biomechanics, pathology and clinical experience has been introduced. This model can be easily integrated into the Maitland approach.

The implications for physiotherapists are that:
• use of the model will allow rapid identification of adverse tension as a component of many 'musculoskeletal' disorders.
• it highlights the considerable potential of the nervous system to participate in the production of signs and symptoms.
• knowledge of biomechanics and pathology enhances assessment and the results of treatment via passive movement. Better prognoses are allowed.
• complexity of anatomy, biomechanics and pathology dictates that there cannot be one single tension test for a limb. As well as routine tests, examination must be adapted to the signs and symptoms presented by the patient.

References

Apfelberg DB and Larsen SJ (1973), Dynamic anatomy of the ulnar nerve at the elbow, *Plastic and Reconstructive Surgery*, **51**, 76-81.

Beel JA, Groswald DE and Luttges MW (1984), Alterations in the mechanical properties of peripheral nerve following crush injury, *Journal of Biomechanics*, **17**, 185-193.

Bendler EM, Greenspun DO, Yu J and Erdman WJ (1977), The bilaterality of carpal tunnel syndrome, *Archives of Physical Medicine and Rehabilitation*, **58**, 362-368.

Blikna G (1969), Intradural herniated lumbar disc, *Journal of Neurosurgery*, **31**, 676-679.

Blomberg R (1986), The dorsomedian connective tissue band in the lumbar epidural space of humans; an anatomical study using epiduroscopy in autopsy cases, *Anaesthesia and Analgesia*, **65**, 747-752.

Bogduk N (1980), The lumbar zygapophyseal joints; their anatomy and clinical significance, in *Proceedings Low Back Pain Symposium, Manipulative Therapists Association of Australia Conference*, Sydney.

Bogduk N (1985), The innervation of the vertebral column, *The Australian Journal of Physiotherapy*, **31**, 81-89.

Breig A (1978), *Adverse Mechanical Tension in the Central Nervous System*, Almqvist and Wiksell, Stockholm.

Breig A and Troup JDG (1979), Biomechanical considerations in the straight-leg-raising test, *Spine*, **4**, 242-250.

Burton S, Brunsdon J and Bleicher K (1987), An investigation of different methods of stretching the structures which limit straight leg raise, in *Proceedings 5th Biennial Conference, Manipulative Therapists Association of Australia*, Melbourne.

Butler DS (1987), Adverse mechanical tension in the nervous system: application to repetition strain injury, in *Proceedings 5th Biennial Conference, Manipulative Therapists Association of Australia*, Melbourne.

Cassvan A, Rosenberg A and Rivera LF (1986), Ulnar nerve involvement in the carpal tunnel syndrome, *Archives of Physical Medicine and Rehabilitation*, **67**, 290-292.

Crymble B (1968), Brachial neuralgia and the carpal tunnel syndrome, *British Medical Journal*, **3**, 470-471.

Cyriax J (1982), *Textbook of Orthopaedic Medicine*, Volume 1, 8th edition, Bailliere Tindall, London.

Dawson DM, Hallet M and Millender LH (1983), *Entrapment Neuropathies*, Little, Brown & Co. Boston.

Dyro FM (1983), Brachial plexus injuries and carpal tunnel syndrome, *Electromyography and Clinical Neurophysiology*, **23**, 251-256.

Edwards BC (1985), Combined movements in the lumbar spine; their use in examination and treatment, in Greive GP (Ed), *Modern Manual Therapy of the Vertebral Column*, Churchill Livingstone, Edinburgh.

Elvey RL (1979), Painful restriction of shoulder movement - a clinical observational study, in *Disorders of the Knee, Ankle and Shoulder*, Western Australian Institute of Technology, Perth.

Elvey RL (1986), Treatment of arm pain associated with abnormal brachial plexus tension, *The Australian Journal of Physiotherapy*, **32**, 224-229.

Elvey RL (1987), Pathophysiology of radiculopathy, in *Proceedings 5th Biennial Conference, Manipulative Therapists Association of Australia*, Melbourne.

Estridge MN, Stanley MD, Rouhe A and Johnson N (1982), The femoral stretching test, *Journal of Neurosurgery*, **57**, 813-817.

Faithfull DK, Moir DH and Ireland J (1985), The micropathology of the typical carpal tunnel syndrome, *Journal of Hand Surgery*, **11B**, 131-132.

Fernandez E and Pallini R (1985), Connective tissue scarring in experimental spinal cord lesions: significance of dural continuity and the role of epidural tissues, *Acta Neurochirurgica*, **76**, 145-148.

Ford DJ and Ali MS (1985), Acute carpal tunnel syndrome, *Journal of Bone and Joint Surgery*, **65B**, 758-759.

Goddard MD and Reid JD (1965), Movements induced by straight leg raising in the lumbosacral roots, nerves and plexus and in the intrapelvic section of the sciatic nerve, *Journal of Neurology, Neurosurgery and Psychiatry*, **28**, 12-17.

Grieve GP (1981), *Common Vertebral Joint Problems*, Churchill Livingstone, Edinburgh.

Guttman L (1977), Median-ulnar nerve communications and carpal tunnel syndrome, *Journal of Neurology, Neurophysiology and Psychiatry*, **40**, 982-986.

Hromada, J (1963), On the nerve supply of the connective tissue of some peripheral nervous system components, *Acta Anatomica*, **55**, 343-351.

Kadish LJ and Simmons EH (1984), Anomalies of the lumbosacral nerve roots, *Journal of Bone and Joint Surgery*, **66B**, 411-416.

Kopell HP and Thompson WAL (1976), *Peripheral Entrapment Neuropathies*, Williams and Wilkins, Malabar.

Lishman WA and Russel WK (1961), The brachial neuropathies, *Lancet*, **2**, 941-947.

Loeser JD (1985), Pain due to nerve injury, *Spine*, **10**, 232-235.

Louis R (1981), Vertebroradicular and vertebromedullar dynamics, *Anatomica Clinica*, **3**, 1-11.

Lundborg G (1975), Structure and function of the intraneural microvessels as related to trauma, edema formation and nerve function, *Journal of Bone and Joint Surgery*, **57A**, 938-948.

Lundborg, G (1988), Intraneural microcirculation, *The Orthopaedic Clinics of North America*, **19**, 1-12.

Mackinnon SE, Dellon AL, Hudson AR and Hunter DA (1986), Histopathology of compression of the superficial radial nerve in the forearm, *Journal of Hand Surgery*, **11A**, 206-210.

MacNab A (1964) Acceleration injuries of the cervical spine, *Journal of Bone and Joint Surgery*, **46A**, 1797-1799.

Maitland GD (1978), Movement of pain sensitive structures in the vertebral canal in a group of physiotherapy students, in *Proceedings Inaugural Congress of Manipulative Therapists*, Sydney.

Maitland GD (1986), *Vertebral Manipulation*, 5th Edition, Butterworths, London.

Marzo JM, Simmons EH and Kallen F (1987), Intradural connections between adjacent cervical spinal roots, *Spine*, **12**, 964-968.

McLellan DC and Swash M (1976), Longitudinal sliding of the median nerve during movements of the upper limb, *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 556-570.

Millesi H. (1986), The nerve gap, theory and clinical practice, *Hand Clinics*, **2**, 4, 651-663.

Murphy RW (1977), Nerve roots and spinal nerves in degenerative disc disease, *Clinical Orthopaedics and Related Research*, **129**, 46-60.

Neary D and Eames RA (1975), The pathology of ulnar nerve compression in man, *Neuropathology and Applied Neurobiology*, **1**, 69-88.

Pang D and Wilberger JE (1982), Tethered cord syndrome in adults, *Journal of Neurosurgery*, **57**, 32-47.

Park WW and Watanabe R (1985), The intrinsic vasculature of the lumbosacral spinal nerve roots, *Spine*, **10** (6), 508-515.

Pechan J and Julis F (1975), The pressure measurement in the ulnar nerve: a contribution to the pathophysiology of the cubital tunnel syndrome, *Journal of Biomechanics*, **8**, 75-79.

Perl ER (1976), Sensitisation of nociceptors and its relation to sensation, in *Advances in Pain Research and Therapy*, Vol. 1, Bonica JJ and Alve-Fessard D (Eds), Raven Press, New York.

Penning L and Wilmink JT (1981), Biomechanics of the lumbosacral dural sac, *Spine*, **6**, 398-408.
Reid S (1987), The measurement of tension changes in the brachial plexus, in *Proceedings 5th Biennial Conference, Manipulative Therapists Association of Australia*, Melbourne.
Rubenach H (1987), The upper limb tension test, in *Tenth International Congress of the World Confederation for Physical Therapy*, Sydney.
Savolaine ER, Pandja JB, Greenblatt EH and Conover SR (1988), Anatomy of the lumbar epidural space: new insights using CT-epidurography, *Anasthiology*, **68** (2), 217.
Sedal L, McLeod JG and Walsh JC (1973), Ulnar nerve lesions associated with carpal tunnel syndrome, *Journal of Neurology, Neurosurgery and Psychiatry*, **36**, 118-123.
Selander D, Mannson LG, Karlsson L (1985), Adrenergic vasoconstriction in peripheral nerves of the rabbit, *Anesthesiology*, **62**, 6-10.
Silver MA, Gelberman RH, Gellman H and Rhoades CE (1985), Carpal tunnel syndrome: associated abnormalities in ulnar nerve function and the effect of carpal tunnel release on these abnormalities, *Journal of Hand Surgery*, **10A**, 710-713.
Smith CG (1956), Changes in length and posture of the segments of the spinal cord with changes in posture in the monkey, *Radiology*, **66**, 259-265.
Smyth MJ and Wright V (1958), Sciatica and the intervertebral disc, *Journal of Bone and Joint Surgery*, **40A**, 1401-1418.
Spencer DJ, Irwin JS, Miller JAA, (1983) Anatomy and significance of function of the lumbosacral nerve roots in sciatica, *Spine*, **8**, 672-679.
Styf J (1988), Diagnosis of exercise induced pain in the anterior aspect of the lower limb, *The American Journal of Sports Medicine*, **162**, 165-169.
Sunderland S (1976), The nerve lesion in carpal tunnel syndrome, *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 615-626.
Sunderland S (1978), *Nerves and Nerve Injuries*, Churchill Livingstone, Edinburgh.
Sutton JL (1979), The straight leg raising test, Unpublished thesis, South Australian Institute of Technology, Adelaide.
Tani S, Yamada S and Knighton RS (1987), Extensibility of lumbar and sacral cord, *Journal of Neurosurgery*, **66**, 116-123.
Tencer AN, Allen BL, Ferguson RL (1985), A biomechanical study of thoraco-lumbar spine fractures with bone in the canal, Part III, Mechanical properties of the dura and its tethering ligaments, *Spine*, **10**, 741-747.
Triano JJ and Luttges MW (1982), Nerve irritation: a possible model of sciatic neuritis, *Spine*, **7**, 129-136.
Tunturi AR (1977), Elasticity of the spinal cord dura in the dog, *Journal of Neurosurgery*, **47**, 391-396.
Twomey LT and Taylor JR (1987), Zygapophyseal joints of the lumbar spine: current research, in *Proceedings, Manipulative Therapists Association of Australia, 5th Biennial Conference*, Melbourne.
Upton ARM and McComas AJ (1973), The double crush in nerve entrapment syndromes, *Lancet*, **2**, 359.
Werner CO, Rosen I, and Thorngren KG (1985), Clinical and neurophysiological characteristics of the pronator syndrome, *Clinical Orthopaedics and Related Research*, **197**, 231-236.
White AA and Panjabi MM (1980), Basic biomechanics of the spine, *Neurosurgery*, **7**, 76-93.