SCHOLARLY PAPER

Neurodynamics

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Key Words

Neurobiomechanics, neurophysiology, neural tension tests.

Summary

Mobilisation of the nervous system is an approach to physical treatment of pain. The method relies on influencing pain physiology *via* mechanical treatment of neural tissues and the non-neural structures surrounding the nervous system. Previous descriptions of this method have not clarified the relevant mechanics and physiology, including interactions between these two components. To address this, a concept of neurodynamics is described.

The body presents the nervous system with a mechanical interface *via* the musculoskeletal system. With movement, the musculoskeletal system exerts non-uniform stresses and movement in neural tissues, depending on the local anatomical and mechanical characteristics and the pattern of body movement. This activates an array of mechanical and physiological responses in neural tissues. These responses include neural sliding, pressurisation, elongation, tension and changes in intraneural microcirculation, axonal transport and impulse traffic.

Because many events occur with body movement, in addition to tension, the term 'neural tension' is incomplete and requires expansion to include both mechanical and physiological mechanisms. 'Neural tension tests' may be better described as 'neurodynamic tests'. Pathomechanics and pathophysiology in neural tissues and their neighbouring structures may be regarded as pathodynamics.

Introduction

Mobilisation of the nervous system (MOTNS) has recently emerged as an adjunct to assessment and treatment of pain syndromes (Fahrni, 1966; Elvey, 1986; Maitland, 1986; Butler and Gifford, 1989; Butler, 1991). An important aspect of this approach is that healthy mechanics of the nervous system enable pain-free posture and movement to be achieved. However, in the presence of pathomechanics of neural tissues (eg nerve entrapment), symptoms may be provoked during daily activities.

The use of neural tension tests is a major part of the MOTNS approach. An aim of using these tests in assessment is to stimulate mechanically and move neural tissues in order to gain an impression of their mobility and sensitivity to mechanical stresses. In the presence of abnormality, the purpose of treatment *via* these tests is to improve their mechanical and physiological function.

Tension tests are limb and trunk movements which are passively performed by a physiotherapist. Structures which can be moved with these tests include the neuraxis, meninges, nerve roots (Breig, 1960, 1978; Louis, 1981) and peripheral nerves (Goddard and Reid, 1965; McLellan and Swash, 1976; Millesi, 1986).

Commonly used tests that move neural structures include the straight leg raise (SLR) (Breig and Troup, 1979), passive neck flexion (PNF) (Reid, 1960; Adams and Logue, 1971), prone knee bend (PKB) (O'Connell 1946), slump (Cyriax, 1942; Maitland, 1986) and upper limb tension (ULT) tests (Frykholm, 1951; Elvey, 1980; Kenneally *et al*, 1988; Butler, 1991). There are also more refined versions of tension tests which direct stress toward specific peripheral nerves, including the radial, radial sensory (Mackinnon and Dellon, 1988), ulnar, common peroneal (Kopell and Thompson, 1976), sural and posterior tibial (Butler, 1991).

Proficient application of MOTNS requires an understanding of neural mechanics and physiology. It is difficult for clinicians to make good use of these subjects because they are large, contain more information than clinicians need, and are not always easily linked to clinical decision making. There is also much information on each subject which does not relate to the other, so that mechanics and physiology of the nervous system have traditionally been considered to be quite separate domains.

In reality, nervous system mechanical and physiological events are dynamically interdependent. For example, mechanical stresses applied to nerves evoke physiological responses such as alterations in intraneural blood flow, impulse traffic and axonal transport. Conversely, physiological misbehaviour of nerves renders them predisposed to mechanical disturbances, as in diabetes (Mackinnon and Dellon, 1988).

There is no single subject in which the interactions between nervous system mechanical and physiological mechanisms are described. However, there is much fragmented reference to these interactions in the literature. Information on these connections may be assimilated to form a subject which covers the necessary information without including superfluous material. The value of this is that clinicians may understand and use the information more easily. This subject may be called 'neurodynamics'. Thus, an aim of this paper is to present a concept of neurodynamics for physiotherapists interested in MOTNS.

Another purpose of this paper is to challenge the use of some terms which are commonly employed when considering MOTNS. These terms consist of 'tension tests', 'neural tension' and 'adverse mechanical tension'. The author believes that there is adequate clinical and scientific material to support the notion that 'tension' is an incomplete term, and so alternative words are suggested.

Mechanics

Neural Container, Mechanical Interface and Responses to Movement

General Aspects

The body is the container of the nervous system. Within the body, the musculoskeletal system is the mechanical interface (MI) to the nervous system. The MI consist of central and peripheral components. Centrally, the MI is formed by the cranium and spinal and radicular canals which collectively house the neuraxis, cranial nerves, meninges and nerve roots. Peripherally, the MI consists of the nerve bed in the limbs and torso where the nerves are presented with bone, muscles, joints, fascia and fibro-osseous tunnels, against which the neural structures contact during daily movement and postures. As the body or container moves, the MI changes its dimensions which in turn imposes forces on neural structures (Goddard and Reid, 1965; Millesi, 1986).

In order that the nervous system is protected against compromise due to the dimensional changes of its container, the neural elements undergo distinct mechanical events which must occur harmoniously with body movement. Elongation, sliding, cross-sectional changes, angulation and compression of neural tissues are such occurrences. These dynamic features occur at many sites including the central and peripheral nervous systems (Breig, 1960, 1978; Goddard and Reid, 1965; McLellan and Swash, 1976) (fig 1).

When the dynamic protective mechanisms fail or are exceeded, symptoms may result. Several examples of musculoskeletal pathomechanics which may cause neural consequences are disc protrusion, spondylolisthesis, joint instability, high intramuscular pressure, and overuse. These disorders may impart altered mechanical stresses to the nearby neural structures. Pathomechanics may lead to pathophysiology in neural tissues, resulting in pain and disability.

Combined Neuromechanical Mechanisms

Two features which combine to cause neuromechanical responses are joint angulation and anatomical destination of the nerves.

Joint angulation increases the length of the nerve bed on the side of the axis of rotation which opens. This causes the nerve on the elongated side to slide and lengthen in response to that joint movement (fig 2).

Tension tests for different peripheral nerves may be deduced from their position relative to the joint axis and how the limb must be moved to exert tension. For example, the median nerve passes along the ventral surface of the elbow and wrist, therefore extension of these joints stresses the nerve (McLellan and Swash, 1976). Plantarflexion/inversion of the ankle moves the common peroneal nerve distally (Kopell and Thompson, 1976), while the radial nerve spirals around the lateral aspect of humeral shaft, hence internal rotation of the arm and pronation of the forearm are likely to stress this nerve (Butler, 1991). Since the spinal canal is situated behind the axis of rotation of the motion segment, flexion induces as much as 7 cm elongation of the canal. This results in longitudinal stress and movement in the neuraxis and meninges (Breig, 1978; Louis, 1981). For a review of mechanics of the neuraxis and meninges, see Shacklock *et al* (1994).

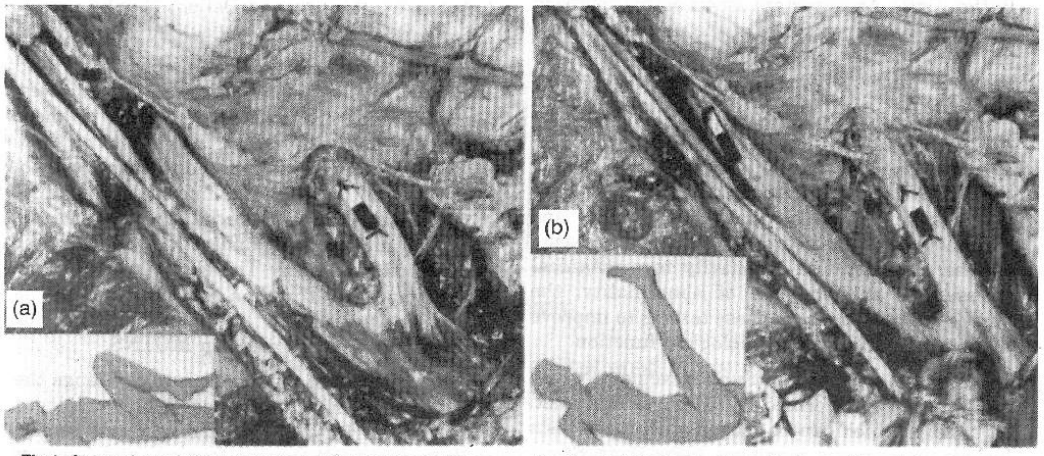

Fig 1: Antero-lateral view of right L4 and L5 spinal nerves as they emerge from respective intervertebral foramina and join more distally to form lumbosacral trunk. Markers 1 cm long placed on spinal nerves indicate position of neural structures relative to foramina. Sympathetic trunk, with one of its ganglia, passes between the two spinal nerve roots

Position (a) Hip flexion/knee flexion — Neural structures are loose and markers are situated in or near IVF

Position (b) Hip flexion/knee extension — Spinal nerves are drawn distally from their IVF and pulled taught. Sympathetic trunk with its interposing ganglion is also stressed by the manoeuvre. From Breig (1978), with permission

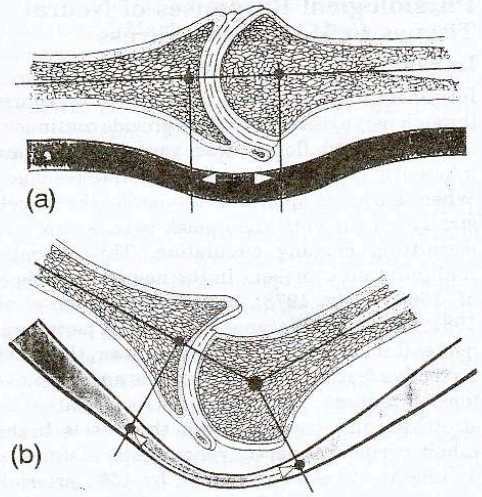

Fig 2: Diagram of a nerve as it traverses a joint while joint is in neutral position. Hatched arrows indicate length of nerve bed at level of joint.

Position (a) Neutral — Nerve is slack

Position (b) Angulated — Nerve bed has elongated, causing nerve to be lengthened and bent across joint

As mentioned, anatomical destination of nerves provides the second means of stressing nerves. Tension is transmitted to a nerve by stressing the structure in which the nerve terminates. The PKB serves as an example, where stretching the quadriceps muscle applies tension to the femoral nerve and mid-lumbar nerve roots (O'Connell, 1946).

Site of Movement Initiation

Early in the range of a tension test, for example the SLR, the nerves are wrinkled and sit loosely in their bed. When movement which exerts tension in the nerves occurs, the nerves lose their slack (Sunderland and Bradley, 1961a, b) and begin to slide (Breig, 1978). These dynamic events begin at the joint where movement is initiated and, with further limb movement, the mechanical effects spread progressively along the nerve to remote areas. Neural movement remote from the joint where movement is initiated will start only when the slack along the nerve has been taken up. As limb movement continues through the mid range, the nerves slide more rapidly because there is sufficient tension to cause their movement. Towards the end range of limb movement, the amount of available neural sliding becomes depeleted, causing neural tension to increase more markedly (Charnley, 1951).

Non-uniform Mechanics

Specific Features

Body movement causes non-uniform strain in neural tissues. Spinal flexion induces 15% dural strain at L1-2 whereas, at L5, strain approaches 30% (Louis, 1981). Nerves are also exposed to different forces along their course as they make contact with neighbouring bone, muscle and fascia (Goddard and Reid, 1965). For example, the ulnar nerve is compressed when the fingers are flexed because the nerve passes under the flexor carpi ulnaris muscle (Werner *et al*, 1985). Pressure on nerves is also increased when neighbouring muscles are passively stretched (Werner *et al*, 1980) or when joints are positioned in a way which decreases the available space in the adjacent nerve tunnel (Apfelberg and Larson, 1973; Gelberman *et al*, 1981; Maffulli and Maffulli, 1991; Spinner, 1968). For example, elbow flexion induces a four-fold increase in pressure around the ulnar nerve at the cubital tunnel (Pechan and Julis, 1975).

Displacement, strain, intra-neural pressure and tension vary at different neural sites, depending on the local anatomical and mechanical characteristics. Clinically, knowledge of regional anatomy and biomechanics is important so that assessment and treatment can be adapted to the individual disorder.

Direction of Neural Movement

Neural sliding does not always occur in one direction during a limb or body movement. In the position of hip flexion, knee extension movement causes distal movement of the sciatic nerve toward the knee. However, the tibial nerve slides proximally (Smith, 1956). The nerves converge toward the joint where the elongation is initiated, in this case, the knee (fig 3).

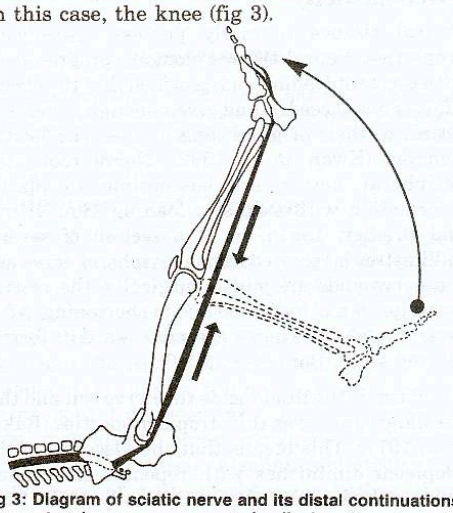

Fig 3: Diagram of sciatic nerve and its distal continuations. Knee extension causes nerves to be displaced convergently towards knee

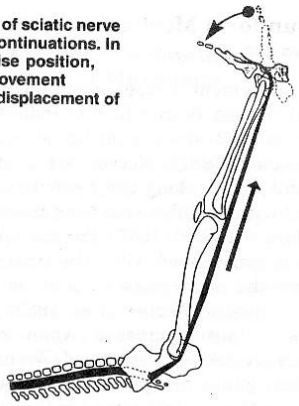

Fig 4: Diagram of sciatic nerve and its distal continuations. In straight leg raise position, dorsiflexion movement induces distal displacement of nerves

The neuraxis and meninges also show convergent behaviour (Louis, 1981), where flexion of the whole spine causes the neural structures in the canal to displace toward C6 and L4, as illustrated in Butler (1991, fig 2.9 page 41).

Sequence of Movement

The direction of neural sliding is also influenced by the sequence of body movements. As stated, knee extension causes the neural structures to slide toward the knee. However, when dorsiflexion is performed, the nerves at the knee slide toward the ankle (Smith, 1956) (fig 4).

Similar sequencing effects occur in the spine where, in the lumbar region, the neural structures slide rostrally when cervical flexion alone is performed (Breig and Marions, 1963) instead of converging toward L4 as with flexion of the whole spine (Louis, 1981).

Viscoelasticity

Neural tissues normally possess viscoelastic properties. Neural tissues elongate progressively with constant loading and, provided that the elastic limit is not exceeded and given enough time, they return to their original length when the load is removed (Kwan *et al*, 1992). Nerve roots and peripheral nerves are susceptible to plastic deformation with excessive loading (Sunderland and Bradley, 1961a, b). If a section of several millimetres is removed from a peripheral nerve and the nerve ends are joined surgically, the resting nerve tension is increased due to shortening. After several weeks, the nerve extends toward its former relaxed state (Bora *et al*, 1980).

Axoplasm is the fluid inside the nerve cell and this substance possesses thixotropic properties (Baker *et al*, 1977). This means that the viscosity of the axoplasm diminishes with repeated movement, causing the liquid to flow more easily. If the fluid is left to stand, it becomes more viscid.

Physiological Responses of Neural Tissues to Mechanical Stress

Intraneural Blood Flow

Intra-neural blood vessels take a tortuous course through nerve tissue in order to provide continuous adequate blood flow. These vascular curls are inherently relaxed before elongation takes place. When tension is applied to the nerve, the vessels straighten out until their slack is taken up, still permitting ongoing circulation. This vascular configuration is present in the neuraxis (Breig *et al*, 1966; Breig, 1978), nerve roots (Parke *et al*, 1981; Parke and Watanabe, 1985) and peripheral nerves (Lundborg, 1975, 1988). However, the above protective features have limitations and excessive tension reduces intra-neural microcirculation by stretching and strangulation of the vessels. In the rabbit peripheral nerve, venous return starts to decline at 8% elongation and, by 15%, arterial, capillary and venous flow is completely occluded. At these values, circulation returns to normal once the load is removed. If the vascular capabilities are overwhelmed by excessive stretch, nerve damage occurs (Lundborg and Rydevik, 1973; Ogata and Naito, 1986). These observations in peripheral nerve correspond well with studies of the spinal cord where impaired blood flow and impulse conduction have been linked directly to increased tension (Cusick *et al*, 1977; Tani *et al*, 1987; Owen *et al*, 1988). It is unclear whether human physiological movement alters intraneural blood flow significantly but there are arguments that, in some situations, circulation changes may occur. Millesi (1986) found that the median nerve bed changed length by 20% from full wrist and elbow extension to flexion. This percentage is greater than that needed to produce experimentally total ischaemia in nerve tissue (15%) (Lundborg and Rydevik, 1973). Human evidence for neural ischaemia lies in holding the arm in the ULT test position for a sustained period, much like 'Saturday night palsy'. Neurogenic symptoms in the form of pins and needles appear with time because the neural elongation strangles the intra-neural blood vessels. The time-dependent nature of the symptoms suggests that, with ongoing vascular compromise, the axons become hypoxic and produce symptoms.

Axonal Transport

Axoplasm contains cellular organelles and many substances which are essential for neuronal function (Shepherd, 1988). Intracellular movement of axoplasm (axonal transport) is achieved by an energy-consuming process which is sensitive to hypoxia (Ochs and Hollingsworth, 1971; Leone and Ochs, 1978; Okabe and Hirokawa, 1989).

Nerve compression causes hypoxia (Sunderland,

1976) and forms a mechanical barrier to axonal transport (Mackinnon and Dellon, 1988). Importantly, axonal transport is reduced at pressures as low as 30 mm Hg (Rydevik *et al*, 1980; Rydevik *et al*, 1981; Dahlin *et al*, Dahlin and McLean, 1986). This pressure is only approximately 25% of normal systolic blood pressure and, when sustained, is sufficient to cause carpal tunnel syndrome (Gelberman *et al*, 1981, 1988). Pressure of this magnitude on adjacent nerves is also reached when asymptomatic subjects perform wrist flexion/extension movements (pressurising median nerve) (Gelberman *et al*, 1981) and when passive stretch of the supinator muscle is performed (posterior interosseus nerve) (Werner *et al*, 1980). Carpal and cubital tunnel pressures as high as 238 mm Hg (double normal systolic blood pressure) during active contraction of the local muscles in nerve entrapment sufferers have been recorded (Werner *et al*, 1983, 1985). Thus, daily movements and many physical techniques are likely to induce at least temporary changes in axonal transport.

Mechanosensitivity

Mechanosensitivity refers to the activation of impulses when a neural structure is subjected to mechanical stimuli such as pressure or tension. The dorsal root ganglion (DRG) is normally mechanically sensitive to gentle manual pressure at surgery and Lasègue's manoeuvre. Action potentials can be activated by mechanically stressing peripheral nerves in animals (Gray and Ritchie, 1954). Impulses are more easily evoked when the nerves are irritated or injured (Calvin *et al*, 1982; Howe *et al*, 1976, 1977). In patients with irritated nerve roots, pain can be reproduced at surgery by gentle manipulation of the nerve roots (Smythe and Wright, 1958; Lindahl, 1966; Kuslich *et al*, 1991). Furthermore, with microneurographic techniques, Nordin *et al* (1984) were able to measure nerve impulses in patients with neuropathies. They noted that mechanically evoked impulses correlated directly with the symptoms described by the patients. Action potentials and symptoms were preferentially stimulated by the performance of movements which place mechanical stress on neural structures, including the dorsal columns of the spinal cord, dorsal nerve roots and peripheral nerves.

Sympathetic Activation

Manual stretching and compression of nerves have been shown to cause action potentials in sympathetic nerve fibres, evoking increased sweating in the skin (Lindquist *et al*, 1973). Futhermore, tension tests have been shown in humans to induce alterations in blood flow and sweating in peripheral tissues (Kornberg, 1992; Slater *et al*, 1994) (fig 1).

Vibration

Vibration is another form of mechanical stimulus which induces physiological changes in neural tissues. Vibration at 5 Hz, a frequency similar to that experienced by truck drivers, causes altered production of neuropeptides (substance P and vasoactive intestinal peptide) in the DRG. Abnormal amounts of these bioactive materials are moved by axonal transport to the target structure, where these substances mediate trophic functions, including degeneration and inflammation of joints and intervertebral discs (Weinstein *et al*, 1988; Pedrini-Mille *et al*, 1990).

Concept of Neurodynamics

Although mechanical and physiological functions of the nervous system interact closely, there is no specific subject which includes both these aspects and their relationships. There is a need for such a field because both components ought to be considered together when assessing and treating a patient *via* nervous system mobilisation and manual therapy. This may be addressed by neurodynamics, which encompasses the interactions between mechanics and physiology of the nervous system. The term 'pathodynamics' may be used to describe the combination of pathomechanical and pathophysiological events in disorders. In treating pain syndromes, clinicians will aim to improve the pathodynamic changes which cause the symptoms and disability (fig 5).

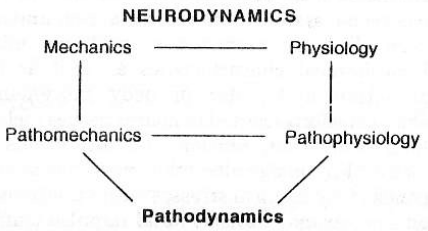

Fig 5: Neurodynamics encompasses interactions between mechanics and physiology of the nervous system. Changes in neural mechanics or physiology may lead to pathodynamics

Inadequacies of 'Tension'

The word 'tension' is frequently used when considering neuromechanical dysfunction (Butler, 1989; Butler and Gifford, 1989; Elvey, 1980, 1986). While adverse neural tension may be a component of some clinical disorders, a problem is that there is a focus on 'tension' as the dominant aspect. This component is only a fragment of what occurs during neural disorders and human movement. There may also be occasions where the disorder is one of increased mechanosensitivity or disturbance of pain mechanisms rather than adverse mechanical tension in the nerves. Furthermore, in some situations it would be

inaccurate to encourage the use of the word tension clinically because production of tension in neural tissues in the patient is at times inappropriate or contra-indicated. Instead, passive movement techniques which are aimed at sliding or reducing tension in neural tissues or omitting passive movement would be preferable. Neural tension tests may therefore be called neurodynamic tests, since they will evoke many mechanical and physiological reactions which ought to be included in clinical thinking (fig 6).

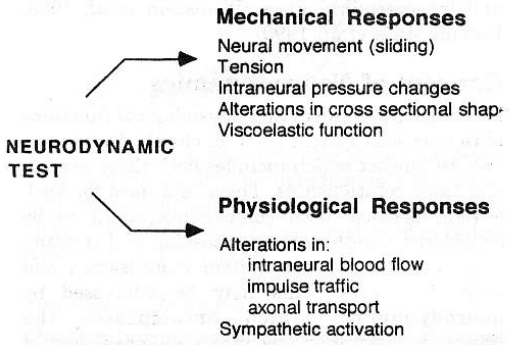

Fig 6: Mechanical and physiological effects of neurodynamic tests on neural tissues

Conclusion

During body movement, there are inevitably interactions between mechanical and physiological mechanisms of the nervous system. Mechanically, the nervous system behaves in a non-uniform pattern which is determined by local anatomical and mechanical characteristics as well as the combination and order of body movements. Mechanical effects exerted in neural tissues include sliding, elongation, tension and alterations in pressure. Physiologically, the nervous system responds to mechanical stresses with variations in blood flow, axonal transport and impulse traffic.

The term 'neurodynamics' may be employed to include the link between mechanical and physiological types of mechanisms. Neural tension tests should thus be regarded as neurodynamic tests. This is because these procedures will evoke many mechanical and physiological responses in addition to tension, so that the word 'tension' does not encompass well enough the broad nature of responses produced by the tests. The term 'pathodynamics' may be used in the presence of abnormality, because pathomechanics may produce painful pathophysiological changes and both types of events must be included in clinical reasoning.

In the presence of neural dysfunction, a patient's presentation will often reflect the nature of the pathodynamics. Clinicians may attempt to link clinical presentation and treatment needs with hypotheses about the pathodynamics in a patient.

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