1 1.1 Neural Tension Background

1.1.1 Tests for Neural Tension

Test procedures are required for abnormal neural tension in the lower quadrant. This is not a new concept. In 1864 sciatica was recognised by Lasègue and his student. Forst published a description of the Lasègue test now known as the straight-leg-raise (SLR) test (Woodhall & Hayes, 1950). The SLR test is performed with the patient lying and the leg is raised with the knee straight until pain is felt (Woodhall & Hayes, 1950). This test is recognised as a good diagnostic test of lumbar intervertebral disc and nerve root lesions (Farhni, 1966).

Petren (1909) presented the earliest description of a sitting lower limb neural tension test based on extension of the knee in sitting (Woodhall & Hayes, 1950). Cyriax, (1942) also described neural tension tests in sitting whereby the knee was extended passively or cervical flexion was performed (Cyriax, 1942). The modern form of these sitting SLR tests known as the ‘slump test’ combines trunk and neck flexion with the SLR (Maitland, 1979). Both the SLR and the slump tests include sensitising movements. Remote testing procedures such as neck flexion, medial rotation of the hip or ankle dorsiflexion can be added to the base neural tension test to further tension the neural system from either the distal or proximal segment (Butler & Gifford, 1989a; Troup, 1981). Troup (1981) described these qualifying tests of the SLR test as sensitive diagnostic and predictive tests that indicated the
severity of episodes of back pain or sciatic pain (Troup, 1981). This emphasises the importance of adding sensitising manoeuvres to the base neural tension test.

The diagnostic value of neural tension tests is well recognised but their use as treatment techniques is a more recent development (Butler & Gifford, 1989b). Physiotherapists have modified clinical examination and treatment techniques designed to assess the mechanosensitivity of the major nerve trunks in the upper and lower limbs (Butler & Gifford, 1989a; Butler & Gifford, 1989b; Elvey, 1992a; Maitland, 1979; Shacklock, 1995l). The slump test is one such test designed to produce movement of the pain-sensitive neuromeningeal structures within the vertebral canal and was first described by Maitland (1979) in the current form (Maitland, 1979).

1.1.2 Slump Test

The slump test is a neural tension test of the lower quadrant that tests neural tissue in the spine and lower limb and it combines the sitting SLR tests described above with the addition of trunk flexion and neck flexion (Maitland, 1979). The test is widely used by physiotherapists to test for suspected involvement of the peripheral nervous system (PNS) in many lower quartile conditions including low back pain, hamstring strains, calf strains and ankle sprains (Kornberg & Lew, 1989; Maitland, 1985; Pahor & Toppenberg, 1996). The slump test applies tension to neural tissue through the full range of spinal and extremity motions (Louis, 1981; Maitland, 1985). It is often more
applicable to functional situations such as slouched sitting, getting in and out of a car and raising one leg with the trunk flexed when dressing than the SLR test. The slump test has been shown to be consistent with high inter-therapist reliability when the patients symptoms form the criterion for a positive or negative test (Philip, Lew, & Matyas, 1989). Figure 1.1 provides a pictorial description of the slump test. The amount of knee extension gained during the test and the impact that knee extension has on pain along with the effect of neck flexion are the main factors of interest during the slump test.

Tension applied to neural tissue during the slump test can be divided into three levels:

• Slight tension of neural tissue is produced by neck flexion performed in sitting with the trunk upright.

• Moderate tension of neural tissue is produced by trunk flexion performed in sitting with the head up.

• Maximum tension of neural tissue is produced by trunk flexion with the addition of neck flexion performed in sitting.

The assumption that these positions place tension on neural tissue is based on the anatomy of neural tissue being the major structure that is a continuous tract from the head to the foot. Anatomical and biomechanical studies provide evidence that neck flexion, trunk flexion and combined trunk and neck flexion in a sitting position with the hip flexed and the knee extended creates movement and tension in neural tissue (Breig, 1978; Breig
Stage One: Full cervical, thoracic and lumbar flexion with overpressure from the therapist.

Stage Two: Knee extension with the ankle dorsiflexed (maximum slump).

Stage Three: Cervical flexion is released and the neck is extended relative to the thoracic spine (moderate slump).

Figure 1.1: Slump test.
2 1.2 Restriction of Movement in Neural Tension Tests

The term neural tension implies reduced mobility of the nervous system but evidence has been provided that there is normal compliance in the neural system with neural tension tests until the onset of protective muscle activity (Hall et al., 1998). The focus on the mechanical aspect of stretch on neural tissue does not address the importance of the central nervous system (CNS) and neurophysiological changes that may be occurring in response to movement of a nerve trunk. Neurophysiological changes in the CNS are considered to contribute to the mechanism where reflex muscle spasm limits the neural tension test (Zusman, 1998).

1.2.1 Muscle Activity during Lower Limb Neural Tension Tests

Recent research has indicated muscle activity as the factor that restricts movement during neural tension tests. In an attempt to identify the cause of the restriction to movement during neural tension tests studies to date have used electromyography (EMG) to measure muscle activity in lower limb neural tension tests (Fidel, Martin, Dankaerts, Allison, & Hall, 1996; Goeken & Hof, 1994; Hall et al., 1995; Hall et al., 1998; Hall et al., 1993; Lew & Puentedura, 1985). The majority of studies relating to neural tension tests of the lower limb have been performed during the straight leg raise (SLR) test. These studies along with those assessing the slump test support the argument for a reflex muscle contraction being the restricting factor of neural tension tests.
**Slump Test**

Changes in EMG activity recorded from the hamstring muscle group during the slump test were inconclusive. EMG activity during the slump test demonstrated that hamstring muscle activity was generally present during knee extension in normal subjects (Fidel et al., 1996; Lew & Briggs, 1997). There was no consistency in whether EMG activity accompanied the first or second point of pain during the knee extension movement in the slump position (Fidel et al., 1996) and readings fluctuated within baseline levels irrespective of whether the neck was flexed or extended (Lew & Briggs, 1997). When the knee was flexed and extended in the slump position as it would be during a neural mobilisation treatment technique, the EMG amplitude was reduced with each repetition of knee extension (Fidel et al., 1996). This may indicate neurophysiological accommodation with the mobilisation technique and suggests that the tension evoked on neural tissue during the slump test may influence the motoneuron pool. Substantive evidence that the slump test can influence α-motoneurons, however, has not been provided with these studies.

**Straight Leg Raise Test**

Studies performed on the SLR test also show conflicting results as to the presence and amount of EMG activity recorded, and the effect of the sensitising manoeuvres of cervical flexion and ankle dorsiflexion on activity
(Goeken & Hof, 1994; Hall et al., 1995; Hall et al., 1998; Hall et al., 1993; Lew & Puentedura, 1985). When active and passive straight leg raising was measured in normal subjects with the ankle in relaxed plantar flexion and then dorsiflexed, the angle of SLR with the ankle dorsiflexed was less than with the ankle relaxed (Gajdosik, LeVeau, & Bohannon, 1985). The loss of motion, however, was unrelated to the EMG activity of the hamstring muscles (Gajdosik et al., 1985). Other authors have also found that a passive SLR elicited negligible EMG in the hamstring muscle group (Norton & Sahrmann, 1981).

EMG activity recorded in the hamstring muscles during SLR coincided with the onset of pain in normal subjects. The onset of pain associated with SLR was most rapid in subjects that were inflexible (Goeken & Hof, 1993). These authors suggested that the evoked EMG activity may be considered a physiological phenomenon that protects the connective tissue from too much tension (Goeken & Hof, 1994). The variation in muscle activity measured by EMG recordings of the hamstring muscles during SLR testing indicates there is a difference between a SLR test that provokes pain in normal subjects compared with a non-pain provoking test (Gajdosik et al., 1985; Goeken & Hof, 1993; Norton & Sahrmann, 1981).

Subjects with abnormal neural tension demonstrated an increase in muscle activity with neural tension tests (Hall & Quinter, 1996; Hall et al., 1995; Hall et al., 1998). The type of muscle activity that occurred during the neural
tension test was important and in particular the differentiation between normal and abnormal muscle contractions limiting the test (Goeken & Hof, 1994). Abnormal muscle contractions occurred in subjects who had low back and leg pain and was evidenced by early onset contractions in one or more muscles during the SLR test (Goeken & Hof, 1994).

Hall et al (1998), found that abnormal muscle activity was present in all subjects with radiculopathy when SLR was tested (Hall et al., 1998). These subjects demonstrated normal through range neural tissue compliance until the onset of muscle activity. Further support for the production of muscle activity with neural tension tests in subjects with abnormal neural tension was demonstrated in subjects with cervical spine radiculopathy where palpation over major nerve trunks and tendon reflexes elicited a widespread increase in EMG activity often in muscles unrelated to the spinal level affected (Hall & Quinter, 1996). In contrast, palpation over normal peripheral nerve trunks produced no pain and no EMG activity in muscles (Hall & Quinter, 1996).

The increase in EMG activity in the SLR test and upper limb tests in normal subjects when pain was provoked and in subjects with abnormal neural tension suggests that a protective reflex muscle contraction occurs which prevents movement of sensitive neural structures (Goeken & Hof, 1994; Hall & Quinter, 1996; Hall et al., 1998). The onset of muscle activity during neural tension tests rather than an increased stiffness of neural tissue more accurately represents neural pathophysiology (Hall et al., 1998).
There appears to be contrasting EMG responses to neural tension tests in normal subjects and subjects with abnormal neural tension. No studies have assessed muscle activity during the slump test in abnormal neural tension subjects. The questions remain as to whether normal subjects exhibit muscle activity during the slump test and to what extent muscle activity occurs in abnormal subjects during the slump test.
3 1.3 Neurophysiology of Neural Tension

The consequences of abnormal neural tension cannot be explained solely by the anatomy and biomechanical behaviour of neural tissue when it is placed under tension. Neurophysiological influences of tension are of great importance especially with regard to abnormal neural tissue.

1.3.1 Peripheral Nerve Damage

Neurogenic pain is described by the International Association for the study of Pain as “pain initiated or caused by a primary lesion, dysfunction or transitory perturbation in the peripheral or central nervous systems” (Merskey & Bogduk, 1994). Pain that has its origin in nerve tissue is easily identified when the damage to the nerve is great enough to impair axonal conduction, the resultant sensory loss and motor weakness presents no obstacle to diagnosis (Greening & Lynn, 1998). Neuropathic pain creates permanent changes in the nervous system (Woolf, 1987) and it is doubtful that physical treatment of nerve injuries with associated morphological changes are appropriately treated with physical techniques (Elvey, 1998).

Minor peripheral nerve injuries under the category of neurogenic pain that do not show changes to nerve function present a greater challenge to diagnosis. These injuries are capable of responding to physical treatment, however, and can be usefully assessed and treated using neural tension techniques (Hall & Elvey, 1999; Klingman, 1999; Kornberg & Lew, 1989; Uth, 1999; Wise,

1.3.2 Nervi Nervorum

The nervi nervorum supply local innervation of the connective tissue of nerve trunks (Hromada, 1963). Hromada (1963) made observations on the innervation of peripheral nerve trunks and identified the unmyelinated nervi nervorum with mainly free nerve endings and some encapsulated endings in the epineurium, perineurium and endoneurium. The nervi nervorum take origin from the perivascular plexuses and nerve bundles within the sheath they are in and have a vasomotor innervation function (Hromada, 1963).

Bove and Light (1995) in a study on rats confirmed that these nerve fibres were nociceptors that responded to mechanical, chemical and thermal stimulus (Bove & Light, 1995). The nervi nervorum were found to respond to longitudinally applied stretch on the nerve. These authors also found that the nervi nervorum had receptive fields in deep structures like muscle or tendon. This finding indicates that pain stemming from the nervi nervorum may not be restricted to one area, stimuli from the muscle or tendon in the corresponding receptive fields could be transmitted by the nervi nervorum.

It has been theorised that the nervi nervorum mediated local nerve inflammation where there was no axonal damage (Zochodne & Ho, 1993). A local inflammatory reaction that was independent of central connections was
demonstrated in peripheral nerves. Capsaicin application to the epineurium of rat sciatic nerve resulted in the release of substance P and calcitonin gene-related peptide along with other neuropeptides from ‘capsaicin-sensitive’ afferents and resulted in vasodilation and plasma extravasation (Zochodne, 1993; Zochodne & Ho, 1993). The nervi nervorum may also be an ongoing generator and ‘refresher’ of the central changes associated with central sensitisation (Bove & Light, 1997).

1.3.3 Central Sensitisation – Influence on the Ventral Horn

Nociceptive input from the nervi nervorum may initiate sensitisation of central neurons but this remains to be established (Zochodne & Ho, 1993). Injury to any of the structures of the spinal motion segment would, however, initiate sensitisation of central neurons. Sensitised dorsal horn neurons abnormally process mechanical input from nerve, muscle, cutaneous or joint afferents (Dubner & Ruda, 1992). The known correlates of central sensitisation are a lowered threshold to firing, increased frequency in background firing, increased responsiveness to noxious and innocuous stimuli and increased receptive field size in the periphery so that a larger area in the periphery would be capable of triggering central neurons (Dubner & Ruda, 1992). Changes to the flexor reflex demonstrated that sensitised dorsal horn neurones are able to generate an increase in the excitability of α-motoneurons in the ventral horn (Woolf, 1984; Woolf & McMahon, 1985; Woolf & Wall, 1986). Thus afferent input into the sensitised dorsal horn of the spinal cord will more readily activate α-motoneurons producing aberrant
muscle activity. Receptors within neural tissue capable of responding to tension have been identified and the influence of central sensitisation on motoneuron excitability has also been recognised. The question remains, however, as to whether tension applied to neural tissue is capable of influencing motoneuron excitability, thus causing an alteration in muscle activity during a neural tension test.

1.3.4 Receptors that may Influence Alpha-Motoneuron Excitability

Aside from receptors within the nerve there are many other receptors that may be activated by the slump positions and thereby influence the α-motoneuron pool. Muscle afferents would be activated by stretch on paravertebral muscles (Burke, Gandevia, & Macefield, 1988) in the moderate slump position and this stretch may be increased with cervical flexion by the attachments of the thoraco-lumbar fascia (Macintosh, Bogduk, & Gracovetsky, 1987; Vleeming, Pool-Goudzwaard, Stoeckart, van Wingerden, & Snijders, 1995). Joint afferents would be activated by end range stretch on the lumbar spine joints (Burke et al., 1988; Gandevia, DI, & Burke, 1992) in the moderate slump position but this stretch would not increase with the addition of neck flexion in the maximum slump as the lumbar spine joints would remain unchanged. Cutaneous receptors are likely to be activated with stretch in both the moderate and maximum slump positions and are capable of modulating the motoneuron pool (Burke et al., 1988; Walk & Fisher, 1993).
4 1.4 Methodology Used to Measure Muscle Activity

The use of EMG to record changes in muscle activity during neural tension tests has had mixed results. Several studies have shown that normal subjects did not show an increase in EMG activity during the Slump or SLR test (Fidel et al., 1996; Gajdosik et al., 1985; Goeken & Hof, 1993; Lew & Briggs, 1997; Norton & Sahrmann, 1981) and sometimes no EMG activity was measurable (Gajdosik et al., 1985; Goeken & Hof, 1993; Norton & Sahrmann, 1981). This indicates that either the muscles were inactive or an inhibitory effect of the neural tension test may have occurred in these normal subjects. When pain was provoked with the neural tension test in normal subjects (Goeken & Hof, 1993) and in pathological subjects where pain was a feature, an increase in EMG activity was recorded (Goeken & Hof, 1994; Hall & Quinter, 1996; Hall et al., 1998).

Surface EMG measures muscle activity occurring directly below the surface electrode in primarily in superficial muscles (De Luca, 1997; Gilmore & Meyers, 1983). One of the limitations of measuring EMG during neural tension tests is that in asymptomatic subjects a reduction in EMG cannot be measured. When using EMG recording during neural tension tests EMG activity was typically calibrated as a percentage of an isometric contraction of the muscle being recorded (Gajdosik et al., 1985; Goeken & Hof, 1993). Therefore, a positive EMG activity level must be present before a reduction in EMG can be measured. In manual therapy research the influence of a treatment technique on muscle spasm has been measured with EMG (Thabe,
In asymptomatic subjects, however, when the muscle being recorded is completely relaxed with no spasm present, then a reduction in muscle activity cannot be calculated and an inhibitory effect cannot be measured. If an inhibition of motoneurons to the muscle being recorded occurs then it is not possible to record ‘negative’ EMG. EMG, therefore, may not be a suitable measure to detect subtle changes in muscle activity following neural tension tests.

The Hoffmann-reflex (H-reflex), however, can measure inhibitory and facilitatory effects on the α-motoneuron pool (Hugon, 1973). There have been no studies investigating the effect of the slump test and the cervical sensitising manoeuvre on the H-reflex recorded from the soleus muscle. In the field of manual therapy the H-reflex has been used to assess excitability changes in response to spinal manipulative therapy (Murphy, Dawson, & Slack, 1995), manual traction (Bradnam, Rochester, & Vujnovich, 2000), massage (Morelli, Chapman, & Sullivan, 1999; Morelli, Seaborne, & Sullivan, 1990; Morelli, Seaborne, & Sullivan, 1991; Morelli, Sullivan, & Chapman, 1998; Sullivan, Williams, Seabourne, & Morelli, 1991) and muscle stretch (Etnyre & Abraham, 1986; Misiaszek, Cheng, Brooke, & Staines, 1998; Robinson, McComas, & Belanger, 1982; Vujnovich & Dawson, 1994).

Evidence of change in α-motoneuron excitability may be used to either support or refute the proposal that reflex muscle activity is responsible for the restriction of movement in neural tension testing.
5 1.5 H-Reflex Methodology

Hoffmann first described the delayed reflex response in the calf muscle following stimulation of the tibial nerve in 1918. The reflex was subsequently named the H-reflex by Magladery and McDougal in 1950 (Braddom & Johnson, 1974). The H-reflex is used in both clinical and research settings. In the clinical setting it is used as a diagnostic tool to detect neurological abnormalities (Braddom & Johnson, 1974). As a research tool, the H-reflex, used experimentally is a means of measuring the excitability of the α-motoneuron pool (Hugon, 1973).

A 0.5-1.0 ms duration electrical pulse that preferentially stimulates Ia afferents in the mixed tibial nerve at the popliteal fossa evokes the soleus H-reflex. The Ia afferent volley produces excitation of homonymous α-motoneurons and a reflex muscle contraction is recorded by EMG from the soleus muscle (Hugon, 1973). The H-reflex is an essentially monosynaptic reflex (Burke, Gandevia, & McKeon, 1983; Burke, Gandevia, & McKeon, 1984) that occurs at a latency of approximately 21-38ms in the lower limb soleus reflex arc (Buschbacher, 1999; Fisher, 1992; Maryniak & Yaworski, 1987; Sabbahi & Khalil, 1990a). The amplitude of the H-reflex gives an indication of the number of motoneurons recruited from the α-motoneuron pool in response to a given stimulus.
1.5.1 H-Reflex Modulation

The amplitude of the H-reflex reflects activation of a portion of a segmental motoneuron pool and can be enhanced by manoeuvres that increase motoneuron pool excitability and attenuated by factors that decrease motoneuron pool excitability. The effect of manual therapy treatment modalities on α-motoneuron excitability has been studied in an endeavour to establish why these treatments are effective. Joint manipulation, mobilisation and soft tissue massage in normal subjects has been shown to inhibit α-motoneurons (Bradnam et al., 2000; Goldberg, Sullivan, & Seaborne, 1992; Morelli et al., 1999; Morelli et al., 1990; Morelli et al., 1991; Morelli et al., 1998; Murphy et al., 1995; Sullivan et al., 1991). Inhibition of α-motoneurons has also been demonstrated with stretch of homonymous muscles (Burke et al., 1984; Etnyre & Abraham, 1986; Robinson et al., 1982; Romano & Schieppati, 1987; Vujnovich & Dawson, 1994). Additionally, stretch of muscles remote from the soleus produced an inhibition of α-motoneurons when passive movement of the hip and/or the knee was performed (Misiaszek et al., 1998). Muscle contraction facilitated the α-motoneuron pool (Abbruzzese, Reni, Minatel, & Favale, 1998; McHugh, Reeser, & Johnson, 1997; Miller, Newall, & Jackson, 1995) and passive pedalling and stepping movements have been shown to inhibit the α-motoneuron pool (Brooke, Cheng, Misiaszek, & Lafferty, 1995; Misiaszek, Brooke, Lafferty, Cheng, & Staines, 1995). During gait, α-motoneurons of various leg muscles were also inhibited (Brooke, Collins, Boucher, & McIlroy, 1991).
1.5.2 H-Reflex Parameter Used to Measure Changes

Measurement of the slope of the rising portion of the H-reflex recruitment curve (H_{slp}) is a relatively new parameter used to assess H-reflex change. H_{slp} has been shown to be a valuable indicator of the excitability of the α-motoneuron pool (Bradnam et al., 2000; Funase, Higashi, Yoshimura, Imanaka, & Nishihira, 1996; Funase, Imanaka, & Nishihira, 1994; Komiyama, Kawai, & Fumoto, 1999). By analysing the central two-thirds of the rising portion of the H-reflex recruitment curve, the variability in responses associated with threshold level H-reflex responses (Crone et al., 1990) and the possible extinction of H-reflexes at or near H_{max} due to the collision effect are avoided (Hugon, 1973).

It is argued that analysis of an H-reflex / M-wave (H/M) ratio or using the M-wave as a covariate in analysis gives the change in H-reflex that is due to the intervention and removes any variation due to the change in the M-wave (Bell & Lehmann, 1987; Funase et al., 1994; Ruegg, Krauer, & Drews, 1990). The analysis of the H/M ratio however, is flawed since changes in the numerator (central H-reflex changes) or the denominator (peripheral M-wave changes) can result in a changed ratio and the investigator is unable to distinguish to what extent either value is responsible for the ratio change. Using the M-wave as a covariate is also an unsound method for the same reasons.

The most satisfactory alternative to using a ratio or covariate analysis is to ensure that accurate methods are used to establish M-wave stability. When
the M-wave is stable during testing, consistency in peripheral conditions is assured and it is possible to analyse the H-reflex data alone without the complicating factor of the M-wave included in a ratio or covariate analysis. Bradnam et al (2000) have demonstrated that in subjects where the limb being stimulated and recorded from was not moved, the $H_{slp}$ parameter alone was able to accurately measure changes in $\alpha$-motoneuron excitability (Bradnam et al., 2000).

1.5.3 M-Wave Stability Analysis

The M-wave is a short latency response that occurs as a result of direct stimulation of the motor fibres of the tibial nerve during H-reflex recording (Hugon, 1973). M-wave recruitment curve stability during recording of H-reflex recruitment curves is an intrinsic means of controlling for possible changes in the H-reflex due to factors affecting the peripheral stimulating and recording environment. If a consistent number of motor fibres are recruited in response to a given stimulus as determined by a stable M-wave, this will result in a constant monosynaptic bombardment of the $\alpha$-motoneuron pool by Ia impulses (Taborikova, 1973). Similarly when identical M-wave recruitment curves are recorded in separate experiments on the same subject, the technical conditions for stimulation have been stable. In this manner changes to the H-reflex can safely be attributed to a change in excitability of $\alpha$-motoneurons indicating excitatory or inhibitory influences on the motoneuron pool (Hugon, 1973; Taborikova, 1973).
M-wave changes do occur using H-reflex experimentation in human subjects (Allison & Abraham, 1994; Bell & Lehmann, 1987; Funase et al., 1994; Ruegg et al., 1990). Change in the M-wave may indicate an alteration in the stimulus intensity delivered to the nerve or a change in the population of motor efferents stimulated. Thus a possible change in the population of Ia afferents activated would confound the results of the experiment. M-wave changes may also indicate a change in the conduction velocity of nerve fibres (Bell & Lehmann, 1987) or a variation in the orientation of muscle fibres under the recording electrode (Gerilovsky, Tsvetinov, & Trenkova, 1989; Myklebust, Gottlieb, & Agarwal, 1984).

Factors producing a change in the M-wave may be responsible for changes in the H-reflex amplitude. When this occurs it cannot be accepted that changes in the H-reflex result from the intervention alone. The stability of the M-wave, therefore, is an important component of experimental H-reflex methodology. The correct analysis of M-wave stability is vital to establishing the likelihood that H-reflex changes are attributable to central neural effects of the intervention and do not result from an alteration in peripheral stimulating and recording conditions.

1.5.4 Methods Used to Establish M-Wave Stability

Despite the importance of ensuring consistency in the M-wave amplitude during H-reflex recording there is disagreement in the experimental literature on the most valid method of M-wave stability analysis. Many experimenters
have not reported criteria used for monitoring M-wave stability or give insufficient information about the criteria used (Abbruzzese et al., 1998; Etnyre & Abraham, 1986; Gollhofer, Schopp, Rapp, & Stoinik, 1998; McHugh et al., 1997; Robinson et al., 1982).

Stimulus Adjustment
In previous work authors have controlled for expected M-wave variation in the M-wave by adjusting the stimulus intensity to the required percentage (eg. 25% $M_{\text{max}}$) of the previous $M_{\text{max}}$ recorded (Simonsen & Dyhre-Poulsen, 1999). This method is commonly used when analysing H-reflex changes during gait (Brooke et al., 1995; Brooke et al., 1991; Capaday & Stein, 1987; Simonsen & Dyhre-Poulsen, 1999).

Ratio or Covariate
Other studies have used the M-wave as a covariate with the H-reflex in analysis in an attempt to remove the variability in the H-reflex that may be due to variability in the M-wave (Bell & Lehmann, 1987). This method has a fundamental flaw as has previously been discussed. The analysis of changes to the H-reflex with the M-wave used as a covariate will indicate whether changes seen are due to M-wave variability but the extent of those effects cannot be established. By the same token, measuring the H-reflex as a ratio of the M-response when there are visible changes in the M-wave (Ruegg et
al., 1990) gives no indication of whether the H-reflex or the M-wave or both has changed and to what extent the change may have been.

**Percent Deviation**

Using a nominated percent deviation in the M-wave as a rejection criteria is an accepted method for establishing the stability of the M-wave (Andersen & Sinkjaer, 1999; Brooke et al., 1991; Misiaszek et al., 1995; Morelli et al., 1999; Paquet & Hui-Chan, 1999). The M-wave should not deviate in any one recording by more than 5-10% from the mean M-wave at the intensity level chosen (Morelli et al., 1999; Paquet & Hui-Chan, 1999). Some authors have used a variation of less than 2.5% (Brooke et al., 1995; Misiaszek et al., 1998).

**Statistical Analysis**

Allison et al (1994) suggested that the method of percent change variation for the M-wave was an inadequate rejection criterion when compared to using an analysis of variance (ANOVA) criterion with the 95% probability (Allison & Abraham, 1994). Testing for M-wave stability with ANOVA has been used by many authors (Goldberg et al., 1992; Misiaszek et al., 1998; Morelli et al., 1999; Morelli et al., 1991; Morelli et al., 1998; Sulllivan et al., 1991). A contention with this method, however, is the inherent neurophysiological variability in the population (Crayton & King, 1981; McIlroy & Brooke, 1987; Williams, Sulllivan, Seabourne, & Morelli, 1992). Large inter-individual
variability in H-reflex and M-wave amplitudes makes the detection of a significant difference within or between the group means extremely difficult unless the difference is exceptional.

A specialised method of stability analysis for the M-wave recruitment curve is lacking in the literature. Many of the above mentioned methods have been demonstrated for use with constant stimulus intensity recordings (Andersen & Sinkjaer, 1999; Brooke et al., 1991; Misiaszek et al., 1995; Morelli et al., 1999; Paquet & Hui-Chan, 1999) or for use during gait (Brooke et al., 1995; Brooke et al., 1991; Capaday & Stein, 1987; Simonsen & Dyhre-Poulsen, 1999) but it is questionable as to whether these methods are appropriate for use with the M-wave recruitment curve methodology.

6 References


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