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Original article

The impact of neurodynamic testing on the perception of experimentally induced muscle pain

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Abstract

Neurodynamic tests such as the straight leg raising (SLR) and slump test are frequently used for assessment of mechanosensitivity of neural tissues. However, there is ongoing debate in the literature regarding the contributions of neural and non-neural tissues to the elicited symptoms because many structures are affected by these tests. Sensitizing manoeuvres are limb or spinal movements added to neurodynamic tests, which aim to identify the origin of the symptoms by preferentially loading or unloading neural structures. A prerequisite for the use of sensitizing manoeuvres to identify neural involvement is that the addition of sensitizing manoeuvres has no impact on pain perception when the origin of the pain is non-neural. In this study, experimental muscle pain was induced by injection of hypertonic saline in tibialis anterior or soleus in 25 asymptomatic, naïve volunteers. A first experiment investigated the impact of hip adduction, abduction, medial and lateral rotation in the SLR position. In a second experiment, the different stages of the slump test were examined. The intensity and area of experimentally induced muscle pain did not increase when sensitizing manoeuvres were added to the SLR or throughout the successive stages of the slump test. The findings of this study lend support to the validity of the use of sensitizing manoeuvres during neurodynamic testing.

1. Introduction

The ability to identify the tissue origin of symptoms and to recognize the neurophysiological mechanisms involved in a patient's pain state is often challenging, even for skilled clinicians. The straight leg raising (SLR) is traditionally considered an important diagnostic test for lumbar intervertebral disc herniation and nerve root inflammation (Thelander et al., 1992; Jonsson and Stromqvist, 1995; Rebain et al., 2002). Only recently has this test been used to assist in the identification of more distal nerve entrapments such as entrapment of the common fibular nerve at the head of the fibula or the plantar nerves at the heel (Butler, 1991; Meyer et al., 2002).

Neurodynamic tests, also termed neural provocation tests (Elvey, 1997), are sequences of movements designed to assess the mechanics and physiology of part of the nervous system (Shacklock, 1995; Butler, 2000). The mechanical components include the ability of the nerve to move and strain in relation to surrounding tissues, and the physiological components relate to, for example, inflammation, ischaemia, and altered ion channel activity resulting in sites of abnormal impulse generation. The underlying concept for these tests is that sensitized and painful neural tissues may become non-compliant to an increase in relative length of the nerve bedding to which the nerve must accommodate (Elvey, 1997).

A neurodynamic test is considered positive if symptoms can be reproduced, if responses on the involved side differ from the uninvolved side or from known normal responses, and if structural differentiation supports a neurogenic source (Butler, 2000). In this

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study, we focused on the use of sensitizing manoeuvres to structurally differentiate peripheral tissues involved in nociception. Previous studies have demonstrated that neurodynamic tests are able to reproduce the symptoms of patients with peripheral nerve disorders and that differences between the involved and uninvolved side can be objectively measured (Shacklock, 1996; Coppieters et al., 2003a; Coppieters et al., 2003b).

The key concept for use of sensitizing manoeuvres (also called qualifying tests (Breig and Troup, 1979)) to identify neurogenic disorders is that the nervous system is continuous and that certain movements load the peripheral nervous system more than the overlying muscles or fascia (Elvey, 1994; Butler, 2000). Hip adduction, for example, is regarded as an effective sensitizing manoeuvre to add to the SLR (Breig and Troup, 1979) because the sciatic nerve runs lateral from the origin of the hamstrings and is loaded by this manoeuvre. Further load can be placed on the sciatic and tibial nerve by adding medial hip rotation or ankle dorsiflexion. In a cadaveric study, Breig and Troup (1979) demonstrated that medial hip rotation resulted in increased tension of the sacral plexus and movement relative to the greater sciatic notch of up to 1 cm. Other examples of sensitizing manoeuvres are the addition of ankle dorsiflexion and eversion to the SLR to assess possible entrapment of the medial calcaneal nerve or lateral plantar nerve in patients with heel pain (Meyer et al., 2002) and the addition of plantar flexion and inversion to assist in the differential diagnosis of fibular nerve problems (Rebain et al., 2002).

It has been demonstrated that the available range of movement of a particular joint is dependent on the position of other body segments. For example, the range of SLR decreases when ankle dorsiflexion is added (Gajdosik et al., 1985; Boland and Adams, 2000) and the addition of cervical flexion, ankle dorsiflexion or medial hip rotation reduces knee extension range during the slump test (Fidel et al., 1996; Johnson and Chiarello, 1997). Although it was hypothesized that neuromeningeal structures were the most likely structures to cause the change in range of motion (ROM), decisive conclusions could not be made. Other structures that have been suggested to contribute to a limitation in ROM are subcutaneous connective tissues, skin, blood vessels and fascia (Gajdosik et al., 1985).

Continuity of the fascial system may provide an alternative explanation for changes in ROM and pain perception during neurodynamic testing (Gajdosik et al., 1985; Barker and Briggs, 1999). A continuous fascial network has been reported to extend via the thoracolumbar fascia to the gluteal muscles, the sacrotuberous ligament and biceps femoris (Vleeming et al., 1995; 1996). The deep crural fascia of the leg has connections with the iliotibial band, which is connected to the tendinous insertion of the caudal part of the gluteus maximus to the gluteal tuberosity (Gerlach and Lierse, 1990). This continuity of the facial system may allow effective load transfer between spine, pelvis and legs (Vleeming et al., 1995). In addition, the posterior layer of the thoracolumbar fascia has attachments to the tendons of splenius capitis and cervicis (Barker and Briggs, 1999). It has been argued that this fascial network may account for positive findings such as pain and limited ROM when cervical flexion is added to the slump test (Barker and Briggs, 1999).

There is no doubt that neurodynamic tests not only load the nervous system, but also challenge non-neural structures. This contributes to the controversy regarding the origin of the elicited symptoms and the difficulty with which the tests are interpreted. To validate the use of sensitizing manoeuvres, it would be ideal to compare the impact of sensitizing manoeuvres between patients with isolated peripheral nerve disorders characterized by an increased mechanosensitivity and patients in whom the peripheral nervous system is not the origin of the pain perception. However, nerve disorders rarely occur in isolation, like median nerve entrapment in the carpal tunnel due to inflammation of the flexor tenosynovium (Gerritsen et al., 2002), and the aim of neurodynamic tests is often to determine the relative contribution of the peripheral nervous system in the origin of the symptoms. A prerequisite for use of sensitizing manoeuvres to identify neural involvement is that the addition of sensitizing manoeuvres has no impact on pain perception when the origin of the pain is non-neural and when an upregulated central nervous system or pathological central mechanisms do not play a dominant role in the patient's symptoms (Shacklock, 1996).

This condition was tested by analysis of the impact of the SLR and slump test on the perception of experimentally induced muscle pain elicited by injection of hypertonic saline. Intramuscular injection of hypertonic saline induces isolated sensitization of muscle nociceptors which leads to muscle tenderness and hyperalgesia (Graven-Nielsen and Mense, 2001). We hypothesized that the perception of this pain of non-neural origin would not change when sensitizing manoeuvres were added to the SLR and slump test.

2. Methods

Two studies were undertaken to assess whether postures or specific positions of the lower limb influenced the perception of pain in distal body segments when pain is induced by injection of hypertonic saline. In the first experiment we investigated the SLR and the impact of sensitizing manoeuvres on experimentally induced muscle pain in tibialis anterior. In the second experiment, we investigated the effect of the different stages of the slump test on experimental muscle pain in the soleus.

2.1. Evaluation of pain response

A single bolus of 0.5 ml of hypertonic saline (5% NaCl) causes a rapid increase in pain, followed by a rapid decrease (Graven-Nielsen and Mense, 2001). In most subjects, the pain almost completely resolves within 5 min. As this period was too short for our experiments, we conducted a pilot study to analyse the effect of an intramuscular injection of 1.2 ml of hypertonic saline (5% NaCl) in tibialis anterior. Seven asymptomatic volunteers participated (4 males, 3 females; age (mean ± SD): 29.4 ± 4.4 years; height: 177.9 ± 10.3 cm; weight: 72.6 ± 11.3 kg). In a supine position, subjects were asked to indicate the intensity of the pain on a visual analogue scale (VAS) every 20 s. Recordings started ten seconds after the injection and continued for ten minutes, or longer if the pain had not yet disappeared. No trunk, neck or lower limb movements were performed during the experiment. Fig. 1 shows the course of the intensity of experimentally induced muscle pain over time. A more gradual decrease could be observed and the duration of the pain was substantially longer than has been reported for a single bolus of 0.5 ml with the same concentration. Based on this pilot study, a single bolus of 1.2 ml of hypertonic saline (5% NaCl) was used for the experiments.

2.2. Subjects

Fifteen asymptomatic volunteers participated in the first experiment (13 males, 2 females; age (mean ± SD): 23.9 ± 4.6 years; height: 179.9 ± 9.3 cm; weight: 78.0 ± 15.3 kg) and 10 asymptomatic participants volunteered for the second experiment (9 males, 1 female; age: 24.9 ± 3.9 years; height: 182.2 ± 6.3 cm, weight: 81.0 ± 9.3 kg). To be suitable for inclusion, each participant had to be free of back, neck and right leg pain in the last year and had to be naïve to the concept of neurodynamic testing. Subjects with a history of neurogenic disorders or any known contraindication to injections were excluded. Participants were given a full explanation of the procedure, without disclosing information regarding the hypothesis of the study. Consent was ascertained prior to the commencement of the study and all procedures were approved by the Institutional Ethics Committee.

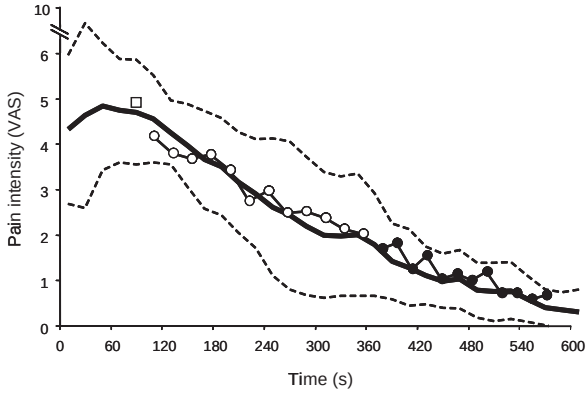

Fig. 1. Time course of experimentally induced pain following injection of hypertonic saline (1.2 ml; 5%NaCl). The solid line represents the mean intensity and the dotted lines represent ± 1 SD. The pain intensity recorded during the first (○) and second (●) series of the SLR experiment have been added to the figure.

2.3. Experimental muscle pain

An experimental pain model was used to ensure that pain was isolated in muscle tissue. Injection of hypertonic saline has been used extensively because the quality of the induced pain is considered comparable to acute clinical muscle pain and shows localized as well as referred pain characteristics (Graven-Nielsen and Mense, 2001). A 1.2 ml bolus of hypertonic saline (5% NaCl) was injected intramuscularly. In the first experiment, hypertonic saline was injected in the tibialis anterior, approximately 2 cm lateral and 7 cm caudal to the tibial tuberosity. Pain is reported to be felt over the anterolateral side of the lower leg and dorsum of the foot (Graven-Nielsen and Mense 2001), which corresponds with the symptomatic area in superficial or common fibular nerve entrapment and with the L5–S1 dermatome. In the second study, soleus was injected at the dorsomedial side of the lower leg, distal to the muscle belly of the gastrocnemius muscle, which is reported to cause pain in the calf (Matre et al., 1999).

2.4. Neurodynamic tests

2.4.1. Straight leg raising

According to the recommendations for standardization of the passive SLR (Rebain et al., 2002), subjects were positioned supine on a plinth with the trunk and neck in a neutral position (Fig. 2). For each subject, the submaximal range of SLR was determined for the right leg. The submaximal range was defined as the maximal range of SLR, with medial hip rotation or adduction, without causing any discomfort. The rationale for moving the limb to a position that did not provoke symptoms, such as a pulling sensation at the posterior thigh or calf, was that pilot trials demonstrated that these additional symptoms distracted the subject, reducing their ability to concentrate on the experimental muscle pain.

Six tests were performed in random order: (1) SLR to the submaximal range, (2) SLR to half of this range, (3,4) the addition of 30° medial or lateral hip rotation to the SLR and (5,6) the addition of 30° hip adduction or abduction to the SLR (Table 1A).

An electronic clinometer (Accustar, Schaevitz Sensors, Virginia, USA) was placed proximal to the lateral

femoral condyle to measure the angle of hip flexion during the SLR. An ankle splint was used to limit change in muscle length of the tibialis anterior. Twin axis electrogoniometers were attached with double sided adhesive tape to the lateral side of the ankle and knee to monitor possible changes in knee and ankle position (SG65 and SG110, Penny and Giles Biometrics Ltd, Blackwood Gwent, UK). To measure the amount of hip rotation, one endblock of a third electrogoniometer (SG65) was placed on a wedge attached to the heel of the ankle splint and a weight (65 g) was attached to the other freely suspended endblock to create a pendulum-type electrogoniometer. Strips of tape were applied to the plinth to mark the target range of hip abduction and adduction.

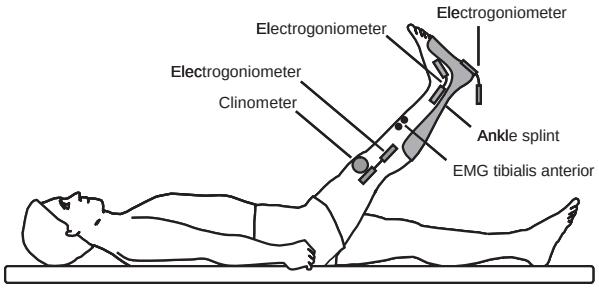

Fig. 2. Set-up for Experiment 1 (SLR). A screen was placed across the subject's abdomen to make the subject visually unaware of the movements of the leg.

Table 1
Different positions for the SLR and slump test

A. Experiment 1: Straight leg raising	
Starting position	End position
SLR_{ZERO}	$SLR_{1/2}$
SLR_{ZERO}	SLR
SLR	SLR + 30° Medial hip rotation
SLR	SLR + 30° Lateral hip rotation
SLR	SLR + 30° Hip adduction
SLR	SLR + 30° Hip abduction

B. Experiment 2: Slump test
Progressive stages
1. Neutral sitting
2. Neutral sitting, Knee extension
3. Thoracolumbar flexion, Knee extension
4. Thoracolumbar flexion, Knee extension, Cervical flexion
5. Neutral sitting

SLR_{ZERO}, zero degrees of hip flexion, no rotation and no abduction or adduction; SLR, submaximal straight leg raising (maximal range without causing any discomfort), no rotation and no abduction or adduction; $SLR_{1/2}$, half of the ROM of the SLR, no rotation and no abduction or adduction.

Electromyographic activity (EMG) was recorded from the tibialis anterior by Ag–AgCl surface electrodes placed 1/3 of the distance between the head of the fibula and the medial malleolus (Hermens et al., 1999). Because muscle activity may impact on pain, EMG recordings were made throughout the experiment to ensure that the participants remained relaxed.

2.4.2. Slump test

The slump test (Maitland, 1979, 1985) involves a combination of knee extension, ankle dorsiflexion, slouched sitting and neck flexion. The aim of the test is to assess the peripheral nerves of the lower extremity, the neural structures in the spinal canal and the connective tissues, such as the meninges.

Prior to the start of the experiment, the maximal range of knee extension in the slumped position (including cervical flexion) without causing any discomfort was determined and identified as the submaximal ROM. The subject sat on the edge of the plinth while the different stages of the slump test were performed: (1) neutral sitting, (2) stage 1 plus passive knee extension, (3) stage 2 plus thoracolumbar flexion, (4) stage 3 plus cervical flexion, and (5) neutral sitting (Table 1B).

An ankle splint was used to prevent changes in muscle length of the soleus (Fig. 3). An electrogoniometer was used to verify the position of the ankle (SG65, Penny and Giles Biometrics Ltd) and to measure the range of knee extension (SG110). EMG electrodes were placed over the soleus at 2/3 of the distance between the medial condyle of the femur and the medial malleolus (Hermens et al., 1999) to verify that the muscle was relaxed throughout the experiment.

2.5. Pain measurement

Because pain is subjective, self-reports are regarded to provide the most valid measure of the experience (Katz and Melzack, 1999). Participants indicated pain intensity on a 10 cm VAS anchored with "no pain" and "worst possible pain". The VAS was mounted on a 10 cm sliding potentiometer, which was connected to the data acquisition system. To indicate the size of the area of the elicited pain, subjects were shown a diagram depicting a series of 10 circles increasing in size from 1 to 10 cm in diameter. This scale has been used previously (Bennell et al., 2004).

2.6. Procedure

In both experiments, three series of tests were performed. The first series was performed prior to injection of hypertonic saline, and the subsequent two

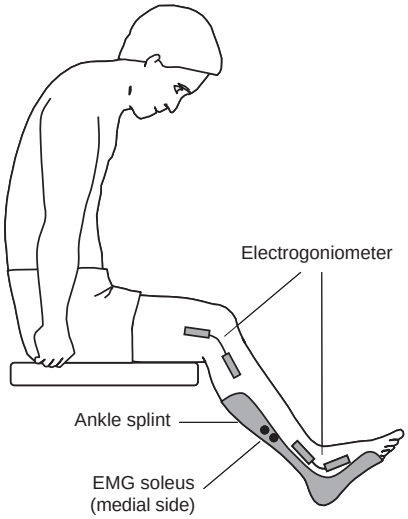

Fig. 3. Set-up for Experiment 2 (Slump test).

series were performed with experimental pain. Following injection, participants were asked to describe the quality of the pain, mark the location of the pain on a body chart and indicate the pain intensity and size. Tests with induced muscle pain started once the level of pain had reached a plateau. In the first experiment, the subject was asked to compare the pain intensity and size in the end position with the ratings in the starting position (for starting and end positions: Table 1A). Participants were made aware that the pain could increase, decrease or remain unchanged in one position relative to another. The slider knob on the VAS was returned to zero before each starting position. In the second experiment, pain size and intensity were compared between the four successive stages of the slump test (Table 1B). The VAS was returned to zero when returning to the final test position (neutral sitting). A one-minute pause was provided after the first series with experimental muscle pain.

To improve standardization in the first experiment, the examiner who performed movements of the leg observed a monitor, which displayed the target range of SLR and hip rotation. Knee and ankle angles were also shown on the screen to provide feedback of confounding movements. In the second experiment, one examiner sat next to the subject in order to guide the subject through the different stages of the slump test. In the tests with knee extension, the subject's foot was placed on a height-adjustable surface in order to maintain a constant range of knee extension.

2.7. Statistical analysis

A two-way, repeated measures analysis of variance (ANOVA) with two repeated factors ('Test' [6 levels: see Table 1A] and 'Position' [2 levels: starting and end position]) was performed to analyse the data of the first experiment. For the second experiment, a one-way, repeated measures ANOVA was performed (5 levels: 'Progressive stages': see Table 1B). Tukey tests were used for the post-hoc analysis. The level of significance was set at $P<0.05$.

3. Results

3.1. Straight leg raising

3.1.1. Test characteristics

The mean range $\pm$ SD of SLR without causing any discomfort was $52.9\pm11.4^\circ$ (range: 36.1–72.1°). All target positions for the different tests were achieved within a few degrees. The examiner achieved hip rotation within $1.3\pm0.8^\circ$ from the target position and SLR within $2.3\pm3.0^\circ$. The mean changes in ankle and knee position were $0.4\pm2.3^\circ$ and $-0.3\pm3.0^\circ$, respectively. These results demonstrate that the tests were performed accurately. The position of the ankle in the splint was $37.1\pm8.7^\circ$ plantar flexion and was held constant.

3.1.2. Pain measures

Apart from one position for one participant, all positions without hypertonic saline were reported to be pain-free. Following intramuscular injection of hypertonic saline into tibialis anterior, pain was predominately perceived over the middle shin around the injection site, with a separate area of referred pain over the ankle joint (Fig. 4). The participants described the pain as a throbbing, dull, deep aching and sometimes cramping pain. The pain intensity plateaued approximately one and a half minutes after the injection (92 ± 25 s) at 4.9 ± 1.6 cm on the VAS. The initial series with experimental pain lasted approximately 5 min (268 ± 42.1 s) with a mean VAS of 3.0 ± 1.2 cm. The second series lasted for approximately three and a half minutes (211.3 ± 51 s) with a mean VAS of 1.1 ± 0.7 cm.

The pain intensity and size of pain for the different tests in the three series are presented in Figs. 5 and 6. For the experimental pain conditions, the Position x Test interaction was not significant for pain intensity or pain size ($F_{5,84}\leqslant1.86$, $P\geqslant0.11$), demonstrating that the tests, which load the neuromusculoskeletal structures had a similar effect on the pain perception as the tests that unload the structures. For the initial series with experimentally induced pain, the main effect for Position was significant for both pain intensity and pain size ($F_{1,84}\geqslant8.89$, $P\leqslant0.004$). Pain ratings were significantly lower in the end position than in the starting position. However, the decreases in VAS (-0.4 ± 0.4) and pain size (-0.4 ± 0.3) were small and the changes were not significant in the second series (VAS: 0.2 ± 0.2; size

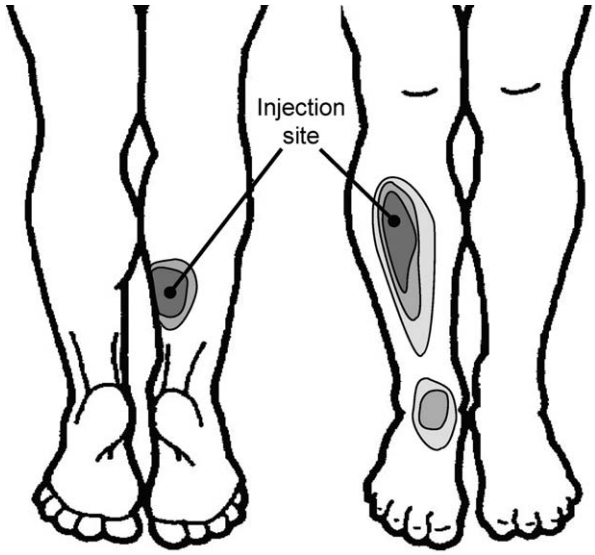

Fig. 4. Location of the experimentally induced pain following injection of soleus (left panel) and tibialis anterior (right panel). A higher grey scale represents a larger number of subjects reporting pain in that area.

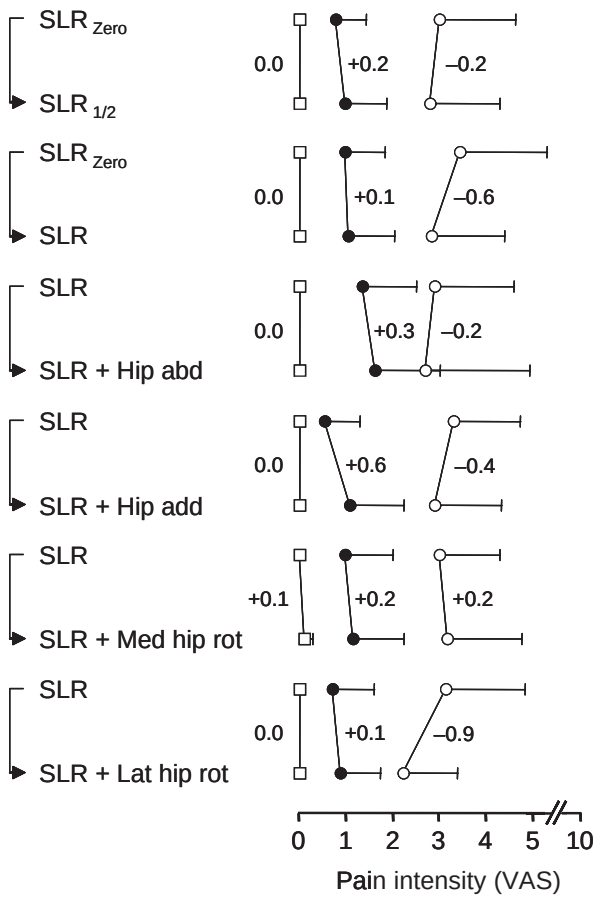

Fig. 5. Pain intensity (mean and SD) for the different SLR positions. Numbers indicate the mean difference in VAS between the start and end position for each manoeuvre. □ = No experimental pain; ○ = experimental pain (series 1); ● = experimental pain (series 2). See Table 1 for description of abbreviated start and end positions.

0.0 ± 0.1). There was no difference between the tests which intend to load or unload the neural structures.

3.2. Slump test

3.2.1. Test characteristics

The maximal angle of knee extension in the slump position without causing any discomfort was $22.6 \pm 7.9^{\circ}$ from full knee extension. This position altered only slightly after the addition of thoracolumbar flexion (knee: $-1.9 \pm 1.9^{\circ}$) or cervical flexion (knee: $0.4 \pm 0.8^{\circ}$). The position of the ankle in the splint was $22.9 \pm 7.9^{\circ}$, which changed minimally throughout the experiment ($0.0 \pm 2.1^{\circ}$ during knee extension; $0.2 \pm 0.8^{\circ}$ after thoracolumbar flexion; and $-0.1 \pm 0.4^{\circ}$ after the addition of neck flexion).

3.2.2. Pain measures

Before injection of hypertonic saline in soleus, all stages of the slump test were pain free. The experimentally induced pain was predominantly perceived around

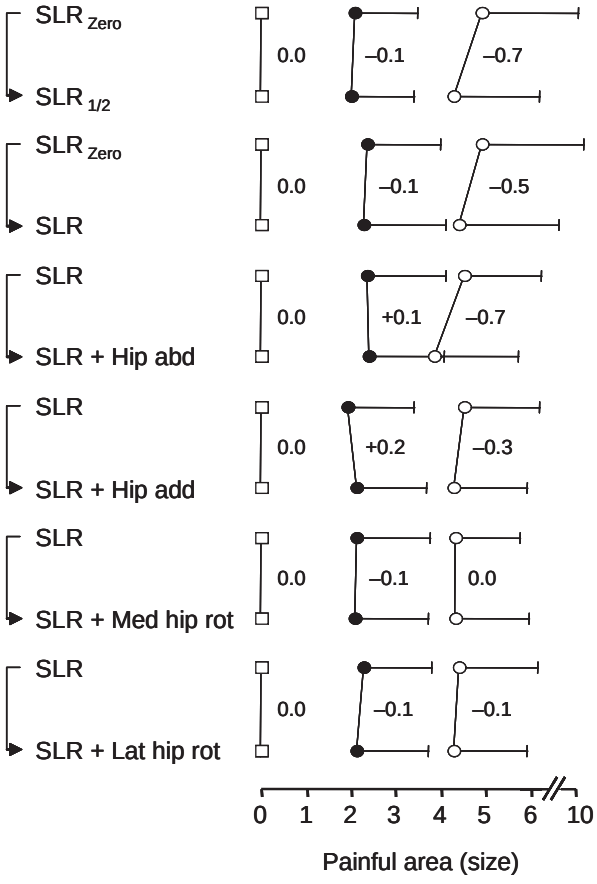

Fig. 6. Size of painful area (mean and SD) for the different SLR positions. Numbers indicate the mean difference in painful area between the start and end position for each manoeuvre. □ = No experimental pain; ○ = experimental pain (series 1); ● = experimental pain (series 2). See Table 1 for description of abbreviated start and end positions.

the injection site in the lower third of the medial calf (Fig. 4). Pain intensity reached a steady state approximately one and a half minutes after the injection (84.3 ± 26.8 s) and equalled a mean VAS score of 4.9 ± 1.3 cm. The first series with experimental pain lasted approximately one and a half to 2 min (103.0 ± 19.0 s) with a mean VAS of 4.1 ± 0.3 cm. The second series lasted for approximately one and a half minutes (92.7 ± 14.0 s) with a mean VAS of 2.6 ± 0.3 cm.

The pain intensity and size of pain for the different stages of the slump test in the three series are presented in Figs. 7 and 8. The ANOVA revealed significant differences for pain intensity and pain size for both series with experimentally induced pain (VAS: $F_{4,36} \geqslant 2.52$; $P \leqslant 0.04$, size: $F_{4,36} \geqslant 3.71$; $P \leqslant 0.01$). The post-hoc analysis revealed no significant differences between two consecutive stages of the slump test, but did reveal a significant decrease for the pain measures for the neutral sitting position at the start and end of each series (VAS: $P \leqslant 0.048$; size: $P \leqslant 0.002$).

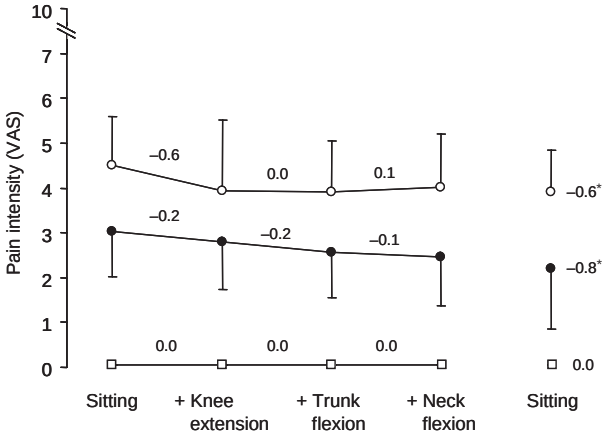

Fig. 7. Pain intensity (mean and SD) for the different stages of the slump test. Numbers indicate the mean difference in VAS between the progressive stages of the slump test. An asterisk indicates a significant difference between the pain intensity in sitting at the start and end of the series. □ = No experimental pain; ○ = experimental pain (series 1); ● = experimental pain (series 2).

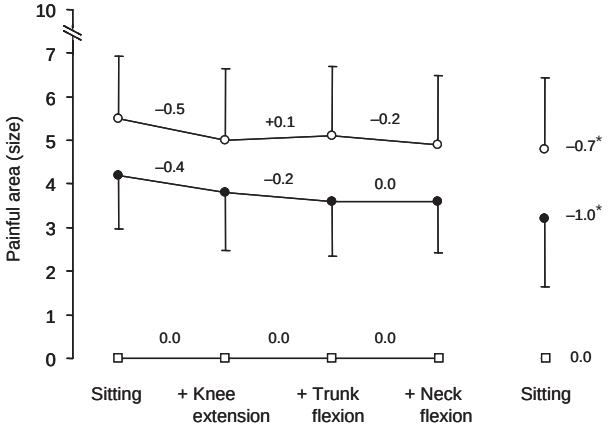

Fig. 8. Size of painful area (mean and SD) for the different stages of the slump test. Numbers indicate the mean difference in painful area between the progressive stages of the slump. An asterisk indicates a significant difference between the size of the painful area in sitting at the start and end of the series. □ = No experimental pain; ○ = experimental pain (series 1); ● = experimental pain (series 2).

4. Discussion

The first experiment demonstrated that the SLR did not increase the perception of experimentally induced muscle pain in tibialis anterior. Furthermore, sensitizing manoeuvres that have been demonstrated to further increase tension in the peripheral nervous system, such as medial hip rotation or hip adduction, did not increase the perception of pain. Results from the second experiment revealed similar findings. The addition of knee extension, thoracolumbar flexion, and cervical flexion to the sitting position did not increase the perception of experimental muscle pain in the soleus. These findings support the hypothesis that different stages of the slump test and the SLR have no impact on pain perception when the origin of the pain is not neural in origin and when the pain mechanism is predominantly related to a peripheral sensitization of muscle nociceptors.

Although we anticipated no change in pain with the addition of sensitizing manoeuvres, the progressive stages of the slump test and the addition of sensitizing manoeuvres to the SLR showed a trend of decreasing pain perception. This occurred despite the accumulation of load, which the SLR and slump test have been reported to place on the neuromusculoskeletal structures. Several mechanisms may account for this unexpected trend. For instance, spinal and supraspinal analgesic effects have been reported from increased discharge of joint and ligament afferents (Wright, 1995). However, the most likely explanation for the decrease in pain is the dispersion of intramuscular saline over time. Three factors support this hypothesis. First, for the slump test, pain in the neutral sitting position at the end of the series was significantly lower than at the start. Second, pain scores at the intermediate stages of the slump test lay between the ratings at the start and end of the series. Third, the pilot study demonstrated that the rate of decrease of pain over time when the leg was not moved was similar to the reduction in the SLR experiment (Fig. 1).

In these experiments, the SLR and the range of knee extension during the slump test were taken to a submaximal range, i.e., the maximal range without causing sensory responses. The rationale for doing so was that testing toward the maximal range elicits additional symptoms which would distract the subject from the experimental muscle pain. Although this

reduced the ROM, it is likely that neuromusculoskeletal structures were still considerably challenged. For instance, the average range of SLR of $52.9 \pm 11.4^{\circ}$ equalled the ROM when symptoms were elicited in a group of patients reporting unilateral lumbar pain with or without ipsilateral leg symptoms (Boland and Adams, 2000). A knee extension position of $22.6 \pm 7.9^{\circ}$ (0° corresponds with full knee extension) in a fully slumped position, including neck flexion, should also load the neuromusculoskeletal structures considerably in most subjects. Furthermore, the addition of medial hip rotation and adduction are regarded as a useful sensitizing manoeuvres when performed near the limit of pain free SLR (Breig and Troup, 1979).

The present study was not designed to confirm that all symptoms elicited during neurodynamic testing are of neural origin. However, the concept of using sensitizing manoeuvres to identify neurogenic disorders would have been compromised if the perception of experimentally induced acute muscle pain would have been altered with the addition of sensitizing manoeuvres. From this perspective, the findings of this study contribute to the validity of neurodynamic tests, in particular, to the specificity of the SLR and slump test. Tests with a high specificity have few false positive results.

When analysing the slump test in healthy subjects, Lew and Briggs (1997) demonstrated an increase in posterior thigh pain with cervical flexion and a decrease with cervical extension. As measurements of biceps tendon strain revealed no differences in tension, it was hypothesized that a structure with links to the cervical spine, most likely the nervous system, was responsible for the posterior thigh pain rather than changes in hamstring tension. However, more recently, Barker and Briggs (1999) provided an alternative explanation for positive findings during neurodynamic testing by demonstrating continuity of the thoracolumbar fascia with the rhomboids and with the tendons of splenius cervicis and capitis. In addition, although studies vary manifestly regarding the anatomic description of the inferior muscle attachments to the thoracolumbar fascia (Bogduk and Macintosh, 1984; Vleeming et al., 1995), if the deep lamina is continuous with the sacrotuberous ligament, and via it with biceps femoris (Vleeming et al., 1995), there is continuity of the fascial system from the cervical spine well into the lower limb. As these attachments are capable of transmitting tension (Vleeming et al., 1995; Barker and Briggs, 1999), continuity of the fascial system may provide an alternative explanation for changes in symptoms during neurodynamic testing. An increase in tension may result in increased pain perception when the tension is sufficient to stimulate the nociceptors of which the threshold has been reduced due to inflammation. Alternatively, tension may also stimulate the mechanoreceptors from myelinated fibres embedded in the musculoskeletal structures which may result in inhibition of the small diameter nociceptive afferent input at the level of the dorsal horn which may reduce the perception of pain.

Although this study demonstrated no increase in pain perception with the addition of sensitizing manoeuvres when pain is of non-neural origin, a change in symptoms with the addition of sensitizing manoeuvres should still be interpreted with care in clinical practice (Zusman, 1992, 1994). While sensitizing manoeuvres may be used to structurally differentiate neurogenic disorders when the processes involved relate predominantly to peripheral sensitization of nerves, the elicited symptoms do not necessarily originate from the peripheral nerve or root. When processes of central sensitization are dominant, the increased pain response following the addition of a sensitizing manoeuvre may be triggered by a barrage of normal input to an already sensitized central nervous system (Gifford and Butler, 1997; Butler, 2000; Coppieters and Butler, 2001). When pathological central mechanisms are involved, signals from receptor types which are normally not associated with pain now acquire the capacity to evoke pain (Raja et al., 1999). This condition arises through augmentation of responsiveness of central pain signalling neurons to input from low-threshold mechanoreceptors. Therefore, the clinician's impression about the pathobiological pain processes in operation, based on the interview and physical examination, should always be taken into consideration when interpreting the results of neurodynamic tests and many other clinical tests.

5. Conclusion

Increasing tension in the neuromusculoskeletal structures with the slump test and with the SLR, including the addition of sensitizing manoeuvres, does not increase the perception of experimentally induced muscle pain in the lower leg. Although care should be taken when interpreting changes in symptoms induced by changes in the loading of neuromeningeal structures, the findings of this study support the validity of the use of sensitizing manoeuvres during neurodynamic testing.

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