Neural tension technique is no different from random passive movements in reducing spasticity in patients with traumatic brain injury

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Purpose: Neural tension technique (NTT) is a therapy believed to reduce spasticity and to increase range of motion (ROM). This study compared the ability of NTT and random passive movements (RPMs) to reduce spasticity in the knee flexors in 10 spastic patients with brain injury. Methods: An RCT study with crossover design evaluated muscle tone measured by: 1) hand-held dynamometer; 2) Modified Ashworth Scale (MAS); 3) and ROM by; 4) angles of resistance onset “catch” (R1) compensatory movement (R2); and 5) ‘subjectively perceived reduction in muscle tone’. Outcome measures were recorded by three raters before and after a single treatment session. Results: Objective stiffness measured with the hand-held device showed no significant changes for the NTT or RPM ($p \geq 0.09–0.79$). The subjective measures showed significant changes after the NTT for the non-blinded rater (MAS: $p < 0.05$; $R_1: p < 0.05$; $R_2: p < 0.05$), but for the blinded rater a significant reduction was found only for $R_1$ ($p < 0.05$) and $R_2$ ($p < 0.05$). For the non-blinded rater intervention effects were found for $R_1$ ($p < 0.01$), $R_2$ ($p < 0.01$) and subjectively perceived tone reduction ($p < 0.01$). For the blinded rater no intervention effect was found. Conclusions: An objective evaluation of NTT demonstrates that it does not reduce spasticity. However, it increases ROM with the same effect as RPM.

Keywords: Spasticity, neural tension, traumatic brain injury

Implications for Rehabilitation

- Neural tension techniques do not reduce spasticity in patients with traumatic brain injury when evaluated with objective biomechanical evaluation methods.
- Neural tension techniques may improve range of motion with the same effect as random passive movements.

Introduction

Neural tension technique (NTT) is a therapeutic concept based on the idea that the mechanoreceptors and their connectivity to the central nervous system can be clinically assessed and treated by mobilisation of the peripheral nerves [1,2]. According to the NTT theory, the assessment can be done by the so-called “neural tension” tests where passive mobilisations of the extremities are supposed to provoke (stretch) the peripheral nervous system. The response to the test is thought to give information about the mobility of the nerves in relation to the surrounding tissues and about physiological abnormalities such as ischemia and inflammation in the nerves [3,2]. The lack of mobility of the peripheral nerves in relation to the surrounding structures is suggested to be caused by variations in blood flow, axonal transport and impulse traffic [2].

The primary treatment objective for NTT is thus to “restore the natural movement of the neural tissue and surrounding mechanical tissue” and thereby “reduce the intrinsic pressure on the neural tissue to regain natural physiological function” [1,2]. The treatment consists of manually induced movements of the limbs with the objective of inducing “sliding of the nerves relative to the surrounding structures by elongation of the structures that surrounds the nerves” by so-called “nerve gliding exercises” [4].

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The efficacy of NTT in terms of pain reduction has been investigated in individuals with carpal tunnel syndrome [6,7,8], with lower back pain [9] and with cervicobrachial neurogenic pain [4]. The efficacy illustrated in these studies is not conclusive, and a recent systematic review on the therapeutic efficacy of neural mobilisation found that there was a lack of quality study on this topic and concluded that there is limited evidence to support treatment efficacy of NTT [10]. To our knowledge only one small study, with five participants and no control group, has investigated the use of NTT as an antispastic treatment in participants with damage to the central nervous system [11]. The results suggested a small reduction in muscle activity after NTT treatment. Although the NTT method appears not to have been validated scientifically for patients with damage to the central nervous system, it is used extensively by physiotherapists in neurological rehabilitation centres throughout Europe with the aim of reducing muscle tone and increasing range of motion (ROM) in participants with brain injury. In this study we perform a quantitative assessment of the effect of NTT in relation to change in muscle tone and ROM in traumatic brain injury (TBI) individuals with spasticity defined as “velocity-dependent increase in tonic stretch reflexes to phasic stretch, in the absence of voluntary activity” [12].

Methods
Participants
Ten patients with TBI and clinically identified spasticity in the knee flexors (Modified Ashworth Scale (MAS) ≥ 1) [13] admitted to the Department of Neurorehabilitation TBI Unit, Copenhagen University Hospital, Glostrup, Denmark participated in this blinded RCT study with cross over design. After inclusion the participants received, in random order, either the NTT treatment or the random passive movement (RPM) treatment on two different occasions (cross over design) with at least 1 day and a maximum of 6 days between each treatment. The randomisation procedure consisted of tossing a coin and was carried out by the person who included the patient. Participants were not included if: 1) they had pain during clinical assessments 2) had orthopaedic problems in the lower limb that made tests or intervention impossible or 3) were identified with clinically manifest contractures in the knee joints.

Physiotherapists employed at the rehabilitation centre identified and included participants with signs of spasticity in the knee flexors (4 female, 6 male; age 31.5 ± 12.1 years). One of the raters tested the legs for increase in stiffness (based on MAS) to select for the treatment and stiffness test the one most affected by increase in muscle tone. Clinical details of the participants are shown in Table I. All participants or their relatives gave written consent before they were included in the study. The study was approved by the local ethics committee, and performed in accordance with the Declaration of Helsinki.

Clinical evaluation of stiffness
All tests were conducted immediately before and after each treatment. Three experienced physiotherapists evaluated knee joint spasticity. Two of them were blinded to the treatment protocol while the third was the one carrying out the treatments and was therefore not blinded. This test design, where some raters were blinded and one was not, was chosen to gain information about the extent to which the blinding of the raters influenced the clinical measurements. Spasticity was evaluated clinically by the MAS [13]. Ankle clonus was evaluated on a three-point scale (0 = no clonus; 1 = extra beats (1–4 beats); 2 = sustained clonus (>4 beats)). Achilles and patella reflexes were also evaluated on a three-point scale (0 = no clonus; 1 = hyporeflexia; 2 = normal reflex activity; 3 = hyperreflexia; and Ankle clonus, 0 = no clonus, Eb = extra beats (1–10), perm = permanent clonus (>10 beats). Columns 7–8 MAS score (0–4) on knee flexors and knee extensions; Column 9–11 number of months between the onset of disease and test; examined side and use of anti spastic drugs (BTX = botulinum toxin; Bac = baclofen; NA = no anti spastic drug). Column 12 functional and cognitive scores based on early functional ability (EFA)/Functional Independent Measure (FIM)/Ranchos Los Amigos (RLA).
Neural tension techniques do not reduce spasticity

Knee stiffness evaluation with hand-held device

The test apparatus (The Neurokinetics RA1 Rigidity Analyzer, Edmonton, Canada) used to measure the stiffness of the lower limb consisted of two opposing hand-held force-sensing air-filled pads connected by tubes to a pressure sensitive diaphragm. The resultant force applied to the lower leg was measured by the difference in the amount of air pressure in the tubes connected to the two air filled pads. A gyroscope attached to the force pad placed on the anterior part of the lower leg provided information about the joint displacements and velocity during movements. For details about the device see Prochazka et al. [14]. All tests were made in a quiet room at ~20°C.

The participants were positioned supine with the tested knee placed in a splint at a height that stabilised the knee side-ways in 90° flexion. The centre of the gyroscope was placed 33 cm distal to the rotating centre of the knee joint on the anterior aspect of the lower leg with the force pads on the fore and back sides of the lower leg. The movements imposed by the raters were identical to a normal clinical examination of spasticity in the lower limb except that the movements were made through the force measuring device.

The stiffness measurements were made at two velocities with ~20 alternating movements per set. The operators were instructed to complete each oscillation in ~1 sec or less for the fast movements (~120°/s) and 3 sec for the slow movements (~20°/s). Each oscillation was followed by a 1 sec pause and each set of 20 movements was followed by a 5-min break.

For further details of the hand-held stiffness evaluation method see Lorentzen et al. [15] or Prochazka et al. [14]. As an additional test to the test method described in Lorentzen et al. [15] a “full range of motion” set of movements was made at the low velocity to investigate changes in stiffness at the end point of the knee joint, where the “passive stiffness” contribution to muscle tone was expected to be greater.

Intervention

Both interventions (NTT and RPM) were conducted by an experienced physiotherapist trained to use NTT as an antispastic treatment. The NTT treatment lasted for 20 min and consisted of passive movements of the hip, knee and ankle joints with the objective of applying dynamic movements of the hamstring muscle by “straight leg raise (SLR)” [1] and—according to the NTT-concept—attempts to apply tension to the nerve and movement of the nerves in relation to the surrounding tissues. The participants were positioned in a supine position on a couch with the non-tested leg lying straight. The movements were made according to the directions from the NTT guidelines [1]. Four series of 30 passive oscillating movements were made. The first series consisted of hip flexion with the knee extended starting from 0° hip flexion. The second series consisted of hip flexion with the knees extended and the ankle in dorsiflexion starting from 0° hip flexion. The third series consisted of knee extension with the hip flexed to 90° and the knee in maximal flexion. The fourth series consisted of knee extension with the hip flexed to 90°, the knee in maximal flexion and a small rotation applied to the lower back during the movements. The repetitive movement was continued until a gentle stretch at the endpoint between R1 and R2 was reached in the hamstrings (see description of R1 and R2 above). The movements were made at velocities lower than the expected threshold for the onset of the velocity dependent reflexes based on previous observations where the stretch reflex is present in the electromyogram at ~75°/s for the ankle [16] and 60–270°/s in the hamstring [17]. The RPM treatment was chosen with the aim of having no impact on the stiffness and thus had characteristics similar to placebos as used in drug studies. Unlike the passive movements made in the NTT treatment no attempt was made to apply tension to the tibial nerve during the movements in the RPM treatment. The movements were made randomly at ankles, knees and hips and lasted for 20 min and—similar to the NTT treatment—consisted of passive movements with the patients lying in supine position on a couch. The unstructured movements at random velocities (above and below the expected threshold for the velocity dependent reflex) were all within a small ROM that never approached the angle for R1.

Statistics

Two-way ANOVA was used to test the difference in stiffness between pre- and post-interventions and between the two interventions measured with the hand-held device.

All average values are presented as median (for MAS) or mean ± SD.

Differences in MAS between the pre- and post-intervention tests were presented as average change in MAS score. All MAS data were changed to numbers in order to calculate medians before and after the different treatments (MAS 1 = 1; 1+ = 2; 2 = 3; 3 = 4; 4 = 5). The difference in reduction in MAS score after the NTT and RPM treatment was tested with the Wilcoxon signed-rank test.

Difference in R1/R2 angle and subjective effect measured on the VAS scale between the NTT and RPM treatment was tested with the pared t-test for repeated measures unless the data were not normally distributed, in which case the Wilcoxon signed-rank test was used.
The significance levels were set to $p < 0.05$. All statistical analyses were made with SigmaPlot 11.0.

Results

Change in stiffness in the knee measured with the hand-held device after treatment

Figure 1 shows the stiffness at the knee joint measured with the hand-held device at high velocities (Figure 1A; 77.1 ± 25.1°/s (range: 54–119); 27.1 ± 6.9°), low velocities (Figure 1B; 31.0 ± 14.0 (range: 7–48)/s; 28.9 ± 28.9°)) and large movement amplitudes (Figure 1C; 54.7 ± 23.2°/s (range: 10–79); 60.8 ± 13.4°) before and after NTT (closed circles) and RPM treatment (open circles) in 10 spastic patients.

The stiffness measured with the rapid movements before the interventions was not significantly different from that measured after the interventions ($p = 0.71$) (Figure 1A). Likewise, no difference was found in the effect of the two treatments on the stiffness ($p = 0.71$) nor between the before- and after-measurements when the intervention effects were taken into account (NTT: mean stiffness pre: 0.258 ± 0.208 Nm/°; post: 0.238 ± 0.151 Nm/°; RPM: mean stiffness pre: 0.283 ± 0.185 Nm/°; post: 0.259 ± 0.179 Nm/°) ($p = 0.97$).

For the stiffness measured with the slow movements (Figure 1B) a small but insignificant trend, ($p = 0.09$) was observed towards a reduction in stiffness after the interventions. No difference was found in the change of stiffness following the two interventions ($p = 0.17$) nor between the before and after measurements due to the intervention effect (NTT, mean stiffness pre: 0.340 ± 0.152 Nm/°; post: 0.239 ± 0.152 Nm/°; RPM, mean stiffness pre: 0.410 ± 0.189 Nm/°; post: 0.317 ± 0.159 Nm/°) ($p = 0.90$).

Finally, no significant reduction in stiffness was found after the treatments for the large test amplitude ($p = 0.29$) (Figure 1C). Also, no difference was found between the treatments ($p = 0.11$) nor between the measurements before and after the interventions when the intervention effects were taken into account (NTT: pre: 0.284 ± 0.125 Nm/°; post: 0.254 ± 0.115 Nm/°; RPM: pre: 0.353 ± 0.122 Nm/°; post: 0.304 ± 0.100 Nm/°) ($p = 0.82$).

Change in clinical stiffness measures for the knee after treatment with NTT or RPM

The reflex and clonus measures shown in Table I were unchanged following both the NTT- and RPM-treatment for the 10 patients.

Table II shows the changes in MAS score following the NTT- and RPM-treatment for the three raters. For the knee flexors no significant change in MAS was found after the NTT treatment for the two blinded raters (mean change: 0.4–0.6; $p = 0.10–0.31$) nor after the RPM treatment (mean change: 0.4–0.5; $p = 0.1–0.3$). The non-blinded rater found significant reduction after the NTT (mean change: 0.7; $p = 0.02$), but not after the RPM (mean change: 0.2; $p = 0.5$).

The reductions in MAS after the NTT treatment were not significantly different from those after the RPM treatment for any of the three raters ($p = 0.12–0.71$).

For the knee extensors no significant reduction in MAS scores was found for any of the three raters after the NTT treatment (0.1–0.3) ($p = 0.33–0.80$) nor after the RPM treatment (0–0.11) ($p = 0.80–1.0$). As for the knee flexors, no significant difference due to the intervention effect was found for the extensors for any of the raters ($p = 0.44–1.0$) (Table II).

Change in the muscle-reactions to passive movement after NTT or RPM

Figure 2 shows the change in muscle reactions (R1 angles of resistance onset “catch” and R2 the angle at which no further ROM could be achieved without pain or compensatory movements) after the NTT- and RPM-treatment measured by one of the blinded raters and by the non-blinded one.

For R1 both raters found that the average angle at which the R1 was identified increased after the NTT treatment by 8 ± 7° and 9.5 ± 4°, respectively ($p < 0.05$). After the RPM treatment the blinded rater found a similar significant increase (9.5 ± 8°) ($p < 0.05$), whereas the non-blinded one found no significant increase after the RPM treatment (1.5 ± 3°) ($p = 0.50$). A significant difference due to intervention effects was found for the non-blinded rater ($p < 0.01$), but not for the blinded one ($p = 0.3$).

Both raters found similar significant average increases in ROM before onset of R2 after the NTT treatment 10.0 ± 4°.
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and 8.0 ± 4° (p < 0.05). After the RPM treatment the blinded nor the non-blinded rater found a significant change in ROM (blinded: 10.5 ± 9°; p = 0.13; non-blinded: 1.5 ± 3°; p = 0.51) (Figure 2). An intervention effect was observed for the non-blinded rater (p < 0.01), but not for the blinded one (p = 0.6).

Figure 3 shows the average effect of the NTT- and RPM-treatment in terms of subjectively perceived reduction of muscle tone in the tested knee in millimeters on a VAS scale. The blinded raters reported similar changes following RPM and NTT (12 ± 13 mm vs. 13 ± 11 mm (rater 1), 16 ± 18 mm vs. 20 ± 22 (rater 2); p > 0.32), whereas the non-blinded one reported a much larger change following NTT than RPM (15 ± 8 mm vs. 5 ± 5 mm; p < 0.01).

Discussion

The aim of this double blind, randomised controlled study with cross-over design was to evaluate the efficacy of NTT in terms of reducing muscle tone and increasing ROM in spastic patients. By a combination of biomechanical and

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Median: 2.5 ± 1.5 −0.4 2.5 ± 2 −0.5 1 ± 1 −0.3 1 ± 0.5 0 ± 0

Mean (SD): (0.5) (0.9) (0.8) (0.8)

Discussion

The aim of this double blind, randomised controlled study with cross-over design was to evaluate the efficacy of NTT in terms of reducing muscle tone and increasing ROM in spastic patients. By a combination of biomechanical and
clinical measurements we assessed the resistance to passive movements, clinical measures of spasticity, ROM and the subjective perception of change in muscle tone. We found no significant change in spasticity related stiffness parameters after the NTT treatment: 1) resistance measured with the hand-held device for the fast, slow or large test amplitude movements, 2) MAS and the clinical reflex and clonus evaluations. The efficacy of the NTT treatment was no larger than that of the RPM treatment for any of the measured parameters for the blinded raters. However, measurements of the MAS, ROM and perceived reduction in muscle tone appeared to be highly influenced by whether the blinded or non-blinded raters conducted the test.

Change in reflex vs. non reflex stiffness

The purpose of making the movements at fast and slow velocities was to separate the stiffness parameters for the velocity dependent reflexes from the passive stiffness parameters in the muscles and joints. In order to make that separation, test velocities above and below the reflex threshold were needed. In this study, the average velocity for the fast movements (77.1 ± 25.1°/s (range: 54–119)) were relatively close to the lowest velocity threshold for the hamstrings (60–270°/s) as found in a study with spastic CP children [17] and in the ankle in spastic stroke, multiple sclerosis and spinal cord injured participants [16]. Therefore, we may not have reached the reflex threshold for all participants and the results may reflect only to a limited extent the reflex contribution to knee stiffness. We may therefore potentially have overlooked a reduction in the reflex component to stiffness after the treatments. However, the clinical tests of the reflex activity confirmed the results and found no change in reflex excitability. Also, the clinical test of spasticity (MAS) showed no significant change after the treatments, except for the rater who was not blinded to the interventions. Small, insignificant reductions in reflex excitability after passive movements were found for the ankle in spastic stroke patients [18]. In this study, a single session intervention based on a combination of passive movements and stretching by a mechanical device was used. This resembles the movements used as the NTT intervention in our study except that the movements in our study were made manually. Goido et al. [11] in contrast found a reduction in EMG activity after treatment with NTT in five-stroke patients, but the lack of a control group and the low number of participants calls into question the validity of that study.

No significant reduction in resistance against passive slow movements was found in either the NTT or RPM groups. This indicates that the movements made during the interventions did not influence the velocity independent stiffness parameters. Although repetitive passive knee movements are suggested as a method of reducing stiffness by the mechanical properties in the knee [18] and in the ankle-by-ankle movements [19,20,21] the non-reflex components were not significantly influenced by the NTT- or the RPM-interventions. Furthermore, no significant reduction in stiffness was found with the hand-held device when the test movements with the large test amplitude were used for any of the interventions.

Only 10 participants were included in this study, and it might therefore be argued that we did not have sufficient power of the statistical tests to exclude a treatment effect. However, the observed reduction in spasticity due to the interventions was very small and all differences were similar
between interventions. Further studies of changes in stiffness in the knee with controlled movement velocities could be useful in identifying changes in stretch reflex activity after different treatments including NTT.

Clinical ratings of stiffness and ROM
No difference between the NTT and the RPM treatment was found in the clinical spasticity score MAS. This implies that the NTT treatment does not lead to a clinically detectable reduction of spasticity that goes beyond the reduction after RPMs. Also, no change in MAS and Ashworth score (AS) was found after weeks of stretching in participants with spasticity caused by spinal cord injury [22], stroke [23] and multiple sclerosis [24] as opposed to significant reductions in MAS found after single session stretch interventions in spastic stroke individuals [25,26]. One of the obvious reasons for the diverse results could be that both single session intervention studies were made with assessors who were not blinded to the intervention, whereas the two long term intervention studies [22,23] were made with blinded assessors. In the present study, two of the three assessors were blinded to the intervention. No significant differences in the MAS-ratings were found between raters, but for the measure of changes in tone the results were heavily influenced by the raters’ awareness of which intervention was used. No significant difference were found between the reductions in subjectively perceived muscle tone after the NTT and the RPM treatment for the blinded rater, whereas the non blinded rater found significantly larger reduction in tone after the NTT treatment than after the RPM treatment. The same trend was found for measurements of ROM: A significantly larger increase in ROM before onset of R1 and R2 was found after the NTT treatment compared to the RPM treatment for the non blinded rater, whereas no difference was found for the blinded rater. The results indicate that the anticipation of the efficacy of a treatment has a strong influence on the result of this clinical rating and emphasises the importance of blinding the assessors when testing efficacy of interventions.

The significant increase in ROM found for both treatment types by the blinded raters is similar to that found after single session stretch interventions in spastic patients [27] and after weeks of stretching [21,20]. No change in ROM was found after weeks of stretching by Cadenhead [28]. The stretch interventions from these studies are different from the interventions (NTT and RPM) used in the present study in terms of how the movements were applied. Most of the studies used a stretch and hold technique where the stretch was continued until maximum ROM and held until a stretch relaxation was observed. In the present study we made repetitive movements of the limbs where the endpoint just approached the maximum ROM, but the limb immediately moved away from the maximal end point following no stress relaxation for the NTT treatment. The movements in the RPM treatment were small and never close to the end point. The changes in mechanisms that lead to increase in ROM observed in the present study may not be very dependent on which movement- or stretch-intervention is given, but more on the movement itself. Therefore, both the NTT and RPM may have caused the observed increase.

Conclusion
An objective evaluation of the NTT therapy demonstrates that it does not reduce spasticity. However, it does increase knee joint ROM with the same effect as RPM.

Declaration of interest: The authors report no conflicts of interest.

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