

Mobilization of the contralateral limb in Slump position: effects on knee extension in healthy adult subjects

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Summary. *Background and aim:* In the acute phase of neuropathic pain due to nerve root disorders, the neurodynamic approach proposes the mobilization of the contralateral limb to decrease the pain and increase the range of motion in the affected limb. The aim of this study was to evaluate the effect of the contralateral knee mobilization on the ipsilateral knee extension range of motion in Slump position in healthy adult subjects. *Methods:* Thirty-eight healthy subjects underwent a placebo, control and experimental manoeuvres that included a passive contralateral knee mobilization into extension. The knee, hip and cervical angles and distance between glabella and femoral condyle achieved in Slump test position were measured with an optoelectronic motion analysis system before and after each manoeuvre. *Results:* Experimental manoeuvre produced a statistically significant increase of the knee extension ROM when compared to the control ($p=.017$) and placebo ($p=.007$) manoeuvres. A significant increase of the hip angle and distance between glabella and femoral condyle was detected after the experimental manoeuvres ($p<.001$), but not after the placebo and control manoeuvres. *Conclusions:* The contralateral mobilization in Slump position increases the ipsilateral knee extension ROM. Further research is required to confirm that the knee increment ROM was due to the neural component. (www.actabiomedica.it)

Key words: contralateral mobilisation, neural mobilization, neurodynamics, neurodynamic test, Slump test

Introduction

Neurodynamics studies the relationship between the mechanical and physiological properties of the nervous system (1). Accordingly, it postulates that the exposure of the nerve tissue to different mechanical stresses, like tensile or compressive loads, impairs its extra- and intra-neural blood flows and may cause neural ischemia. Furthermore, it has been demonstrated that inflammation of the neural tissue alters its histological structure (2). The clinical sign of these alterations is an increased mechanosensitivity of the nerve itself, therefore the nerve is more sensitive to

mechanical stimuli, like pressure or tension (1). The neural mechanosensitivity is usually assessed with the provocation (i.e. Tinel's) and neurodynamic test, in which movement of a neural mechanically sensitive structure can reproduce the symptoms reported by the patient. In neurodynamic testing, the nerve tissue is assumed to be involved when the symptoms reported by the patient change after moving a segment distant from the symptomatic region, otherwise the symptoms are believed to originate in non-neural tissues (i.e. soft tissues) (3).

The Slump test is an example of a neurodynamic test, used for the assessment of the increased mecha-

nosensitivity of the meningeal and neural tissues (4) in patients with low back pain (5, 6) and with lumbar disc herniation (7). Despite the Slump test is described in several ways and there is not a universally accepted procedure (8); the Slump position is achieved with a maximal passive thoraco-lumbar and cervical flexion; afterwards, a passive or active knee extension and ankle dorsiflexion are performed in order to increase tension to the neural and meningeal structures (9). Irrespective of the procedure used to perform the test, if patient complaints of symptoms in the back of the thigh in Slump position that change after a cervical extension, the sciatic nerve is assumed to be involved. As passive knee extension range of motion measurements in Slump position have shown to be reliable in healthy subjects, (10) in the case of neural impairment, the degree of knee extension in Slump position may be considered a direct indicator of the nerve mechanosensitivity, since this movement causes considerable strain on the sciatic nerve. Therefore, a reduced knee extension would indicate a higher mechanosensitivity.

Even though neural techniques have been shown to be useful in the management of neural symptoms (11-13) and limited evidences support their use in the treatment of low back pain, (5, 6, 14), the research in this field needs to gather further insights into the underlying mechanisms (15). The effects of neural mobilization of the lower limb, (i.e. a knee extension mobilization in Slump position) on the knee range of motion (ROM) have been studied by several authors. Ten passive knee extension have been reported to produce a significant increase of knee ROM in Slump position in asymptomatic subjects (16). A similar ROM increase was also observed after an active knee extension protocol lasting 6 weeks (17). Moreover, Herrington (18) found a positive effect of either slider or tensioner neural mobilizations aimed at improving knee extension ROM.

Although these studies may support the use of knee mobilization in Slump position to decrease the mechanosensitivity in the symptomatic lower limb, this technique may be considered too irritating in patients with a neuropathic pain due to an impairment of the peripheral nervous system. Therefore, the mobilization into extension of the knee contralateral to the affected limb has been suggested to decrease neuropathic pain

(19). The lumbar roots' anatomy supports the rationale of contralateral knee mobilization. As the lumbar roots arise from the spinal cord with an angle that can be divided into horizontal and axial components, it is postulated that tensioning the contralateral root will cause a movement of the spinal cord in the caudal direction, which will reduce the tension of the ipsilateral nerve root through the axial component (19).

Recently a randomized clinical trial (20) assessed the effects of the contralateral mobilization in Slump position compared with a sham mobilization and no mobilization on asymptomatic subjects. The effects were measured using a Numerical Rating Scale that scored the tension sensation reported by the subjects in the posterior thigh region. The authors found that the contralateral mobilization reduced the tensile sensation compared to no movement, whereas the sham mobilization did not. The purpose of this study was to further investigate the effects of contralateral knee mobilization using a different outcome measure, i.e. the knee extension in the Slump position. Our experimental hypothesis was that the mobilization into extension of the left knee will decrease the tension in the right nerve roots and, accordingly, increase the right knee extension in the Slump position.

Methods

Participants

A repeated measure design was performed at the Motion Analysis Laboratory of the Piero Palagi Hospital, Florence, Italy. Voluntary participants, recruited through a sample of convenience, were included if they met the following inclusion criteria: being healthy, aged >18 years, naive to manual therapy and neurodynamics, and having a limited right knee extension in Slump position that increased after an active cervical extension (3, 21). Participants were excluded if they had neck or back pain in the previous 3 months, a history of major trauma to the lumbar, hip or knee regions or if they suffered from neurological conditions. Subjects suffering from diabetes were also excluded, since a reduced mechanosensitivity has been found in this patients (22).

Prior to participating in any study-related procedures, participants read and signed an informed written consent form and were informed about the procedure but not about the aim of the study. The study protocol was approved by the local Institutional Ethical Committee.

Procedure

In all participants the right knee extension ROM was measured in the Slump position before and after an experimental, a placebo and a control manoeuvre, as described later. In the present study we used the positioning proposed by Maitland (4). The subject sat on a table with the pelvis fixed against a support to maintain the sacrum in vertical position. The distance between the support and the anterior edge of the seat was adjustable in order to fit the subject's thigh length. The popliteal fossae were put against the edge of the table and the thighs were parallel. The subject was asked to "slump" the trunk while keeping the cervical spine in neutral position. Then, the examiner passively flexed the cervical spine at the end of movement and applied a slight pressure over the shoulders in order to further increase thoracic and lumbar spine flexion. Finally, a belt was fastened across the thighs to ensure that no hip flexion took place, and across the shoulders just below the seventh cervical vertebra to keep the subject's position stable during each test (Figure 1).

Kinematic measurements were performed by using an optoelectronic motion analysis system (SMART-E 600; BTS, Milan, Italy) with five infrared cameras and five passive markers, applied on the following anatomic landmarks: right lateral malleolus (M1), lateral condyle of the right femur (M2), greater trochanter of the right femur (M3), spinous process of the seventh cervical vertebra (M4) and glabella (M5). In order to avoid any discrepancy in the markers placement among the three tests, the markers were attached on each subject at the beginning of the experimental session and were removed only after the completion of the three manoeuvres. The error of the system in the recognition of the markers position within the calibrated volume was less than .3 millimetres for all acquisitions.

In the Slump position three manoeuvres (experimental, placebo and control), each lasting for 2

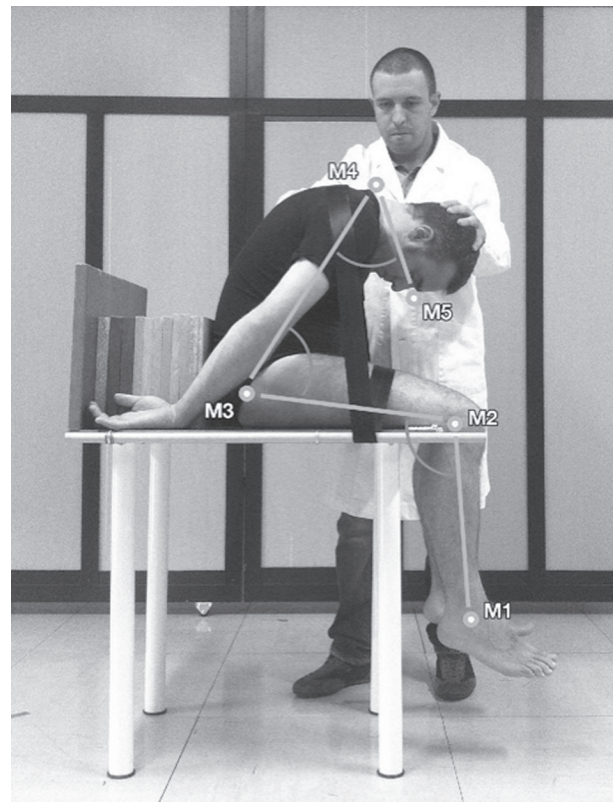


Figure 1. Markers and subject positioning

minutes, were performed with a 10 minutes interval between one another. The use of a rest period of 10 minutes between each manoeuvre was based on a cadaveric study on the median nerve (23). However, the same time span has been demonstrated adequate also for restoring the viscoelastic properties of the soft tissues (24) and neural tissues (25) in living humans. The order of the three manoeuvres was randomized among participants.

The experimental manoeuvre (Figure 2) consisted of 12 passive left knee mobilization into extension (while maintaining the ankle in maximum dorsiflexion) until the achievement of the second resistance (R2), i.e. the range where the maximum resistance to movement is perceived (26). To standardize the mobilization speed, the operator reached R2 every 10 seconds following the rhythm paced by auditory cues. The neural tissue R2 has been found to be reliable in healthy subjects (27).

The placebo manoeuvre (Figure 3) consisted in mobilising the left ankle joint into dorsiflexion with



Figure 2. Experimental manoeuvre. The researcher carried out a passive mobilization of the left knee into extension (while maintaining the ankle in dorsiflexion maximum) from the Slump position (A) until the achievement of the second resistance (B)



Figure 3. Placebo manoeuvre. The researcher carried out a passive mobilization of the left ankle joint into dorsiflexion in Slump position with the knee flexed at 90° (A) until second resistance was perceived (B)

the flexed knee until R2 was perceived. Although the dorsiflexion of the ankle is considered a neurodynamic movement, the mobilization of the ankle to end-range with the knee flexed to 90° should not be able to transmit tension along the sciatic nerve and reach the nerve roots lumbar (19, 20). The ankle mobilization was performed with the same amount and frequency described above for the experimental condition. In the control manoeuvre, the subject did not receive any mobilization but kept the Slump position for 2 minutes.

A physiotherapist with a postgraduate degree in manual therapy (TG) performed both the subject positioning and the different manoeuvres in all participants. Before and after each manoeuvre, a different examiner, (17) blinded to the performed manoeuvre, passively moved the subject's right knee into extension until perceiving R2. When the examiner perceived the maximum resistance to knee extension movement, he ordered the start of kinematic acquisition, that lasted 5 seconds. All tests were performed in a single session lasting approximately 60 minutes.

Statistical analysis

From kinematic data, three angles in the sagittal plane were computed: the knee angle (KE), defined as the angle between the thigh segment (identified by M2 and M3), and the leg segment (identified by M1 and M2), the hip angle (HA), defined as the angle between the trunk segment (identified by M3 and M4) and the thigh segment, and the cervical angle (CA), defined as the angle between the head segment (identified by M4 and M5) and the trunk segment. Moreover, the distance between M2 and M5 (head-knee distance, HKD) was measured (Figure 1). It is noteworthy that the HA, as defined in this study, includes hip, pelvis and trunk movement. For each angle, data from the first and the last 1000 milliseconds of recording were ignored and the mean value during the middle 3 seconds was calculated and used for all analyses.

As the Shapiro-Wilk test found that all variables were normally distributed, analyses were conducted using parametric tests. An ANOVA for repeated measures with manoeuvre as within-subjects factor (3 levels: experimental, placebo, control) was used to assess the stability of the initial position among the three manoeuvres. In order to verify whether the interval between each manoeuvre (10 minutes) was sufficient to restore the viscoelastic properties of the soft and neural tissues that had been elongated, the analysis was repeated considering the temporal sequence of the different manoeuvres, i.e. comparing the knee extension ROM measured in the Slump position before the first, the second and the third manoeuvre. Paired t-tests were also used for pairwise comparisons. Mauchly's sphericity test was used prior the ANOVA; whenever

the sphericity test was not met, Greenhouse–Geisser correction was made.

The effect of the three manoeuvres was assessed by an ANOVA 3x2 for repeated measures with two within-subjects factors, time (2 levels: pre and post) and manoeuvre (3 levels: experimental, placebo, control). When a significant Time x Manoeuvre was found, a post-hoc analysis was conducted to compare the three manoeuvres to one another.

For all statistical analyses, the α value was set at $p < .05$ and the software used was SPSS, Version 17.0 (SPSS Inc., Chicago, IL, USA).

Results

Thirty-eight asymptomatic subjects (10 females, 28 males) participated in this study. The characteristics of the sample are reported in Table 1.

No significant differences were found among the experimental, placebo and control manoeuvres for the four variables (KA, HA, CA, HKD) measured at the initial assessment. Conversely, the analysis showed that the KA measured at the initial assessment differed among the first, the second and the third manoeuvre performed ($F=3.775$, $p=.028$). Indeed, the KA measured before the first manoeuvre was lower than the value measured before the second ($t=-2.657$; $p=.012$) and the third manoeuvre ($t=-2.034$; $p=.049$), whereas no differences was found between the second and the third manoeuvre ($t=-0.158$; $p=.875$).

The manoeuvres produced different effects on the KA, as shown by the significant Time x Manoeuvre interaction ($F=6.365$, $p=.003$). The post-hoc analysis showed that the experimental manoeuvre increased

Table 1. Subjects characteristics (N=38)

Gender	
Male	28 (73.7%)
Female	10 (26.3%)
Age (years)	41.5±16.9
Height (centimeters)	172.9±8.8
Weight (kilograms)	73.5±12.8
Dominant limb	
Right	33 (86.8%)
Left	3 (7.9%)
Ambidextrous	8 (5.3%)

Data are expressed as mean ± standard deviation or frequency with percentage

significantly the knee extension ROM compared to both the placebo ($F=8.222$, $p=.007$) and control ($F=6.283$, $p=.017$). Conversely, no differences were found among control and placebo ($F=0.593$, $p=.446$). However, a significant Time x Manoeuvre interaction was found also for the HA ($F=47.387$, $p<.001$) and for the HKD ($F=18.167$, $p<.001$), but not for the CA ($F=1.155$, $p=.322$). For both variables, differences were found when comparing the experimental manoeuvre with the placebo (HA: $F=69.365$, $p<.001$; HKD: $F=23.892$, $p<.001$) and with control (HA: $F=51.675$, $p<.001$; HKD: $F=21.497$, $p<.001$) manoeuvre, but not between placebo and control (HA: $F=3.820$, $p=.058$; HKD: $F=.988$, $p=.327$) (Table 2).

All the subjects in the study did not experience adverse events during any manoeuvre.

Discussion

The purpose was to investigate the effect of the contralateral neural mobilization on the knee exten-

Table 2. Mean and standard deviation of kinematic measures before and after each manoeuvre in the 38 participants

	Experimental		Placebo		Control		p value		
	pre	post	pre	post	pre	post	T	M	TxM
KA	160.8±7.1	163.3±7.2	160.8±7.2	161.6±.5	161.3±6.9	162.3±7.2	<.001	NS	<.005
HA	72.7±5.8	75.4±6.1	72±6.3	72.4±6.8	72.8±4.8	72.9±5.3	<.01	NS	<.001
CA	58.3±5.7	57.7±5	58.6±6.2	57.2±6.1	57.5±6.3	55.7±6.0	<.005	<.01	NS
HKD	.35±.0	.37±.0	.35±.0	.35±.0	.35±.0	.35±.0	NS	<.05	<.001

KA=knee angle; HA=hip angle; CA=cervical angle; HKD=head-knee distance; T=time factor; M=manoeuvre factor; TxM=time x manoeuvre interaction; NS=non significant

sion ROM in the Slump position. We observed a significant increase of the knee extension ROM after the neural mobilization of the contralateral knee, but not after a control and a placebo mobilization. It is reasonable to assume that the contralateral mobilization provoked a displacement of the ipsilateral nerve root because several studies have shown a displacement of the sciatic nerve after a neural mobilization in vivo through ultrasound imaging. In healthy subjects, the full knee extension in a modified Straight Leg Raise position produced a sciatic nerve mean longitudinal distal excursion of 12.4 millimetres (SD=4.4) (28). In fifteen asymptomatic participants, tensioning and sliding techniques provoked a sciatic nerve excursion of 3.2 (SD=2.1) and 17.0 millimetres (SD=5.2), respectively (29). Moreover, in Slump position knee extension produced distal longitudinal excursion of the sciatic nerve ranging from 2.6 (SD=1.5) to 3.2 (SD=2.0) millimetres (30). Such excursion, though minimal, might suffice to explain the increased knee extension ROM observed in the present study, that was also very limited and might be therefore attributed to the neural component.

On the other hand, we observed that the HA and the HKD were also increased after the experimental manoeuvre. Even though the increased KA was observed uniquely after the experimental manoeuvre, it was associated with a similar amount of hip extension. This finding raises doubts about the hypothesis that the increased knee extension ROM observed after contralateral knee mobilization into extension might be attributed to the reduced neural tension. Indeed, the hamstrings stretching induced by the repeated, passive contralateral knee extension might have caused a posterior pelvic rotation, which in turn might have decreased the hamstrings tension in the right limb. Therefore, as we placed no markers on the pelvis landmarks, it is not possible to know whether the increase of the knee extension ROM was due to the neural or muscular component. Considering that the average increase of the knee and hip extension was quite similar (2.5° and 2.7°, respectively) the improvement of the knee extension ROM seems most likely attributable to the ischial tuberosities' posterior rotation induced by the contralateral hamstring tension. This distal pelvis motion might have therefore produced a distal shift of

the hamstring length, explaining the association found in the experimental condition between the increased knee extension and the HA. This argument raises doubts about the neural displacement that is produced with the contralateral mobilization, because the experimental manoeuvre induced a repetitive stretching of the hamstrings, which caused a posterior pelvic rotation. In light of this consideration, the results of previous studies (16, 17) claiming an increase of knee extension following an ipsilateral neural mobilization are questionable, as the posterior pelvic rotation was not considered a potential confounding factor.

Recently, Shacklock, et al. (20) studied the contralateral mobilization in Slump position in asymptomatic subjects. They found a reduction of stretch sensation, assessed with a Numerical Rating Scale, in the posterior thigh in the intervention group ($p \leq .001$), but no changes in the control and sham groups ($p = .996$). The experimental manoeuvre consisted in one contralateral knee extension mobilization to end-range, maintaining that position for 30 seconds. The authors concluded that the reduced perception reported by the subjects of the experimental group was attributable to the neural component rather than to the relaxation of the posterior thigh soft tissues, since the magnitude of the response reduction in the control and sham groups was not statistically significant. However, our data suggest that a different explanation for this finding is also possible, i.e. that the mobilization into extension of the contralateral knee could produce a posterior pelvic rotation which causes an ipsilateral hamstring relaxation and consequently a reduction of the stretch sensation in the back of the thigh; therefore, the conclusions of the authors raise questions.

A second finding of the present study is that the time interval between each manoeuvre (=10 minutes) seems to be not adequate to allow the complete restoration of the baseline viscoelastic properties of the tissues. Our results, in fact, showed the knee extension ROM of the pre-assessment of the first manoeuvre was significantly less when compared to the pre-assessment of the other two subsequent manoeuvres, whatever was the order established through the randomization. This result conflicts with other published data (24, 25). However, though all the structures elongated during the first knee extension did not

recover their full original resting length after the first pre-assessment, this unlikely biased increased ROM after the first pre-assessment, because it means that all the structures elongated during the first knee extension did not recover their full original length (viscoelastic properties) after the first pre-assessment. Actually, thanks to the randomized order of the three manoeuvres (experimental, control and placebo), no difference emerged among the pre-assessment measures. However, since the three conditions were randomized and differences among the pre-assessments were absent and this potential systematic error was minimized.

The present study has several limitations that should be discussed. Firstly, as mentioned, we did not study the pelvis motion. The finding of a slight increase of the HA associated with the increased knee extension strongly indicate that future studies should control for the posterior pelvic rotation by using radiographic imaging, as suggested by McHugh et al. (31) or by recording pelvic kinematics with additional markers on pelvic landmarks. Secondly, the present study included only healthy subjects, so the results cannot be generalized to a symptomatic population.

In conclusion, the results of this study demonstrated that mobilising the contralateral knee towards extension in Slump position increases the ROM of the extension if the ipsilateral knee. However, we cannot state with certainty that the increase of the knee extension ROM in Slump position is due to the effect of mobilization of neural tissue. In light of our findings and those of previous literature, we conclude that the underlying theory underpinning the contralateral neurodynamic mobilization has not been demonstrated in healthy subjects. Therefore, future research is needed to fill this grey area.

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