Analysis of electromyographic activity in spastic biceps brachii muscle following neural mobilization

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SUMMARY

Introduction: Hypertonia is prevalent in anti-gravity muscles, such as the biceps brachii. Neural mobilization is one of the techniques currently used to reduce spasticity. Objective: The aim of the present study was to assess electromyographic (EMG) activity in spastic biceps brachii muscles before and after neural mobilization of the upper limb contralateral to the hemiplegia. Materials and Methods: Repeated pre-test and post-test EMG measurements were performed on six stroke victims with grade 1 or 2 spasticity (Modified Ashworth Scale). The Upper Limb Neurodynamic Test (ULNT1) was the mobilization technique employed. Results: After neural mobilization contralateral to the lesion, electromyographic activity in the biceps brachii decreased by 17% and 11% for 90° flexion and complete extension of the elbow, respectively. However, the results were not statistically significant (p > 0.05). Conclusions: When performed using contralateral techniques, neural mobilization alters the electrical signal of spastic muscles.

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Introduction

Cerebral vascular accident (stroke) is defined by the World Health Organization as a clinical sign of the rapid development of a focal disturbance in brain function that persists beyond 24 h (Cesário et al., 2006). The sensory, motor and/or cognitive sequelae of a stroke can lead to impaired functional capacity, diminished independence and a reduction in
quality of life (Aguiar et al., 2008). The incidence of stroke doubles every decade after 55 years of age, with prevalence values ranging from 0.5% to 0.7% of the population worldwide (Rodgers, 2004).

Due to compromised neurons in the corticospinal plexus, contralateral paresis is one of the most common manifestations of a stroke. Approximately 50% of affected individuals recover functional independence after six months, whereas the remaining half experience hemiparesis or hemiplegia (partial and complete loss of voluntary movements, respectively) stemming from the event, which can lead to mild, moderate or severe motor disability (Marcucci et al., 2007).

Spasticity is another characteristic consequence of central nervous system injuries (Pontes et al., 2000). This motor disorder is characterized by an increase in resistance to passive muscle stretching, dependent on the velocity of the stretch, causing hyper-excitability of the stretch reflex, elastic hypertonia and changes in proprioceptive sensitivity, often accompanied by clonus, flexor and/or extensor spasms and contractures. Spasticity can lead to incapacity, affecting the musculoskeletal system and limiting normal motor functions (Felice and Santana, 2009).

Secondary disturbances related to spasticity include the extreme difficulty of muscle function rehabilitation, joint disorders, pain and compensatory movements (Corrêa et al., 2005). Spasticity among hemiplegic patients is clinically recognized by predominant hypertonia in the anti-gravity muscles. The hemiplegic upper limb generally remains in a position of adduction and internal rotation of the shoulder, elbow flexion, forearm pronation and finger flexion (Teive et al., 1998).

Neural mobilization is one of the techniques currently used to reduce spasticity in individuals suffering from neurologological disorders (Marinzeck, 2010). The term refers to a group of techniques the aim of which is to place the neu- raxis in tension and stretch it by means of appropriate mobilization through certain postures, followed by the application of slow, rhythmic movements directed toward the peripheral nerves and spinal cord. These treatment techniques evolved from the diagnostic tests proposed by Elvey to assess adverse neural tension (Butler, 2003; Junior and Teixeira, 2007).

When a nerve is mobilized, its cross-sectional area is gradually reduced and this deformation affects the intraneuronal microvascular flow, causing an improvement in neural function; moreover, the length of the spinal canal undergoes a change during movement (Walsh, 2005). Damaged nerves cause adverse tension in the entire nervous system, which limits movement and affects the adaptation capacity of the entire body and not only the injured area (Zamberlan and Kerppers, 2007). Davies (1997) stresses the importance of early treatment and the prevention of adaptive injuries to the peripheral nervous system following a stroke.

The aim of the present pilot study was to determine the influence of neural mobilization on spasticity in patients with stroke sequelae.

Materials and methods

The present study was conducted in compliance with the Brazilian National Health Council guidelines contained in Resolution 196/96 and received approval from the local Human Research Ethics Committee. All participants were properly informed regarding the objectives and procedures of the study and signed a statement of informed consent.

The participants in the present pilot study were enrolled at the CEFISIO Physiotherapy Clinic of the Universidade Estadual do Centro-Oeste (Brazil). The sample consisted of six adults with stroke sequelae (four men and two women) with a mean age and standard deviation of 54.16 ± 7.9 years, height of 1.70 ± 0.09 m, body mass index (BMI) of 24 ± 6.5 kg/m² and an average of four years elapsed since the stroke.

The inclusion criterion was a diagnosis of stroke with spasticity in the elbow flexors, according to the modified Ashworth Scale, which quantitatively evaluates the degree of passive movement during muscle stretching and determines the degree of spasticity in stroke victims through a scale ranging from 0 to 4, subdivided into Grade 0, Grade 1, Grade 1+, Grade 2, Grade 3 and Grade 4, with 0 denoting an absence of resistance to passive stretching and 4 denoting joint rigidity and no range of motion. The exclusion criteria were joint deformities of the upper limb and cognitive deficiency that would impede the testing procedures.

Volunteers first underwent an anthropometric examination and an assessment of the degree of spasticity in the elbow flexor muscles using the Modified Ashworth Scale. A score of 1–4 was awarded for passive movement of elbow flexion-extension (Borhannon and Smith, 1987). An electromyographic assessment of the biceps brachii muscle of the affected upper limb was also carried out. Trichotomy and skin cleansing with ethyl alcohol (70%) were performed to reduce bioimpedance at the sites on which the electrodes were to be placed, as recommended by Surface Electromyography for the Non-Invasive Assessment of Muscles (Hermens et al., 1999). Electromyographic studies were performed using an 8-channel signal recording system (EMG System Brasil LTDA) connected to a data acquisition and analysis system (WinDaqXL). The signal was then submitted to a 20–500 Hz band filter, amplified 1000 times and converted by an A/D plate with a sampling frequency of 2000 Hz per channel and input variation of 5 mV. Active bipolar electrodes were used, separated by a distance of 20 mm (De Luca, 1997). The root mean square (RMS) electronic media was used to measure the amplitude of the electromyographic signal. All procedures used to capture and analyze the electromyographic signals in this study are recommended by the International Society of Electrophysiology and Kinesiology (Solomonow, 1995).

Electromyographic signals were recorded under two conditions: 90° flexion (F) and maximal extension (E). Under the first condition (F), the patients were instructed to lie in the supine position with the affected upper limb held passively at 90° elbow flexion. Under the second condition (E), the individuals remained in the supine position and actively extended the elbow, seeking the maximal range of motion. The procedure lasted 30 s under both conditions. As the biceps brachii muscle has fast muscle fiber characteristics, the 30-second collection time was based on the principle that the central nervous system does not affect the inhibition of Gamma hyperactivity regarding myoelectrical activity. The capturing of the electromyographic signals was performed in an isolated location following the neural mobilization procedure by a single operator with skill in handling both the hardware and software.
After the initial electromyographic activity was recorded, neural mobilization of the median nerve was carried out (Upper Limb Neurodynamic Test or ULNT1) on the non-affected upper limb. According to Elvey apud Butler (2003), this procedure can alter symptoms in the limb undergoing treatment. Neural mobilization of the affected limb was not performed in the present study in order to avoid the tendency toward a reduction in electrical activity due to the positioning of the spastic limb in a muscle stretching situation.

The neural mobilization was performed by a professional physiotherapist with experience in this technique. To attain the required tension and neural mobilization, the patients were instructed to begin with passive depression of the shoulder girdle, 90° shoulder abduction, hands and forearms in a neutral position, wrist extension in the neutral position, forearm in supination, external rotation of the shoulders and elbow extension. These movements were performed consecutively without allowing the anterior segment to return to its original position. After holding this position, neural mobilization was performed with oscillations of slow, rhythmic wrist flexion and extension. Twenty oscillations were performed per minute for 3 min, repeated three times in the same session, with 1 min of rest between each effort (Butler, 2003). All procedures were conducted by the same investigator. The electromyographic signal of the biceps brachii muscle of the affected upper limb was recorded again after neural mobilization (at 90° flexion and maximal extension).

For the statistical analysis, the percentage difference in RMS before and after neural mobilization was determined using the following calculation: percentage difference = 100 (RMS value pre-neural mobilization – RMS value post-neural mobilization)/original RMS value (Stell, 1997). The data were then analyzed using the Origin 7.0 program. The D’Agostino and Pearson test was used to assess the normality of the data, followed by Student’s t-test. The significance level was set at 5% (p ≤ 0.05) (Figure 1).

**Results**

Figure 2 displays the mean electromyographic activity of the biceps brachii muscle before and after mobilization of the median nerve (at 90° flexion and maximal extension). Electromyographic activity in the biceps brachii muscles decreased by 17% and 11% during flexion and extension, respectively, after neural mobilization of the unaffected limb. However, these changes were not statistically significant (p = 0.27 and p = 0.30, respectively).

Figure 3 displays the mean values in relation to the degree of spasticity. Individuals with grade 1 + spasticity exhibited decreased electromyographic activity (from 16.03 μV to 14.77 μV) during elbow flexion after neural mobilization and increased electromyographic activity (from 29.60 μV to 30.13 μV) during elbow extension after neural mobilization. This small increase in myoelectrical activity may have been generated by facilitation of the biceps brachii muscle in the stretched position. Patients with grade 2 spasticity exhibited decreased electromyographic activity (from 13.95 μV to 13.05 μV) during elbow flexion after neural mobilization and decreased...
electromyographic activity (from 33.70 μV to 30.85 μV) during elbow extension after neural mobilization. Moreover, patients with grade 1 spasticity exhibited decreased electromyographic activity (from 36.50 μV to 23.30 μV) during elbow flexion after neural mobilization and decreased electromyographic activity (from 46.60 μV to 30 μV) during elbow extension after neural mobilization.

Discussion

The present pilot study demonstrated that the application of the technique did not result in significant differences. A reduction in myoelectrical activity was found in the individuals submitted to the neural mobilization technique on the side contralateral to the stroke sequela. However, an increase in myoelectrical activity was found when the signal capturing was performed with the arm in extension. This corroborates the statement by Butler (2003) that the central capturing was performed with the arm in extension. This increase in myoelectrical activity was found when the signal side contralateral to the stroke sequela. However, anuals submitted to the neural mobilization technique on the reduction in myoelectrical activity was found in the individualsthe technique did not result in significant differences. A

Conclusion

The present study on the effect of neural mobilization using the Upper Limb Neurodynamic Test 1 demonstrates a reduction in myoelectrical activity in certain cases of spasticity and an increase in activity in other cases. The small number of individuals in the sample may have contributed to the lack of statistically significant results. Thus, there is a need for studies involving a larger number of individuals in a more homogeneous sample with both males and females in order to determine whether the mobilization of the median nerve through neural mobilization on the side contralateral to the sequela can truly be used for the control of different degrees of spasticity.

Although the application of this technique did not result in significant differences, a reduction in electrical activity was found in the spastic muscle after neural mobilization of the contralateral limb, demonstrating that this technique could be an additional tool in the control of spasticity, particularly in cases of lesser intensity.

References


