

CASE REPORT

QUANTITATIVE ASSESSMENT OF ANAESTHETIC NERVE BLOCK AND NEUROTOMY IN SPASTIC EQUINUS FOOT: A REVIEW OF TWO CASES

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Objective: To quantitatively evaluate the effect of motor nerve branch block and neurotomy of the soleus nerve on triceps surae spasticity, reviewing 2 cases.

Methods: Beside clinical assessment, we carried out a quantitative measurement of the stiffness of the ankle flexor muscles. The path length of the phase diagram between elastic and viscous stiffness quantifies the reflex response to movement and reflects the importance of the spasticity. The assessments were carried out before and 30 min after motor nerve branch block of the upper soleus nerve and more than 7 months after neurotomy.

Results: Both patients presented with pronounced ankle plantar flexor spasticity: their path lengths were more than 6 times greater than normal values at baseline (#1: 354 N m rad⁻¹; #2: 409 N m rad⁻¹). Motor nerve branch block and neurotomy allowed a near-normalization of elastic and viscous stiffness of ankle plantar flexor muscles in the 2 patients. Their path length was almost similarly improved by motor nerve branch block (#1: 127 N m rad⁻¹; #2: 231 N m rad⁻¹) and neurotomy (#1: 60 N m rad⁻¹; #2: 162 N m rad⁻¹).

Conclusion: These case reports highlight the fundamental role of the soleus muscle in triceps surae spasticity in our patients, the predictivity of motor nerve branch block in the preoperative assessment, and the effectiveness of soleus neurotomy in spastic equinus foot.

Key words: nerve block, muscle spasticity, stroke.

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Diagnostic soleus motor nerve branch block (MNBB) with anaesthetics is a method frequently used in patients after stroke to assess the role of the soleus muscle in spastic equinus foot, as described by Decq et al. (1). In the case of positive response to MNBB, a partial neurotomy of the soleus nerve can be performed. In theory neurotomy has the same impact on spasticity as MNBB; however, in clinical practice, the spasticity assessment is unfortunately limited to ordinal scales with low reliability, as shown by Platz et al. (2) in a recent systematic review. A quanti-

tative method, such as the one described by Lehmann et al. (3), seems to be a reliable indication of ankle plantar flexor muscles spasticity, as previously shown by Detrembleur & Plaghki (4) and Deltombe et al. (5). The objective of this study was quantitatively to evaluate the effect of MNBB and neurotomy of the soleus nerve on spasticity, reviewing 2 cases.

METHODS

Clinical evaluation

Clinical assessment included the passive range of motion (ROM) measurement of the ankle, Manual Muscle Testing (MMT) of the ankle dorsal and plantar flexors (Medical Research Council scale), and spasticity assessment with the Tardieu scale (6).

Quantitative assessment

The muscles can be modelled as a spring and a viscous damper connected in parallel. As a spring, the elastic stiffness of the muscle is independent of velocity and will produce a torque response in phase with the displacement. As a damper, viscous stiffness increases with velocity and will develop a resistive force in proportion to the velocity applied to it. Elastic and viscous stiffness of the ankle plantar flexor muscles can be assessed by a method described by Lehmann et al. (3). This method was previously used by Detrembleur & Plaghki (4) to assess the effect of intrathecal baclofen administration in 11 spastic patients. The same experimental procedure was followed in this study.

Motor nerve branch block and neurotomy

Using 1.5 ml of 2% lidocaine, we performed MNBB of the upper nerve of the soleus following the method described by Deltombe et al. (7). A partial and segmental selective neurotomy of the soleus nerve was performed following the method described by Decq et al. (8).

Subjects

The first patient was a 47-year-old woman with right spastic hemiparesis. Her Stroke Impairment Assessment Set (SIAS) (9) was 49/76. She presented with a permanent ankle clonus, which impaired her gait. She did not have any orthopaedic contracture; in particular, the ROM of the ankle in dorsiflexion was above 0°, as shown in Table I. We performed MNBB of the upper nerve of the soleus 2 months after stroke, and the patient underwent surgery 16 months after stroke. Given that the patient meanwhile developed a slight contracture of the gastrocnemius muscles, we performed a combined surgery (80% fascicular resection of the upper nerve of the soleus associated with 30% resection of the lower nerve of the soleus and slight lengthening of gastrocnemius muscles). Clinical evaluation and quantitative assessment of spasticity were carried out before MNBB, 30 min after MNBB, and 7 months after surgery.

The second patient was a 68-year old man with a left spastic hemiparesis. His SIAS was 54/76. He also presented with a permanent ankle clonus, which impaired gait, without any contracture (Table I). We performed MNBB of the upper nerve of the soleus 25 months after stroke, and the patient underwent selective neurotomy (75% fascicular resection of the upper nerve of the soleus) 32 months after stroke. Clinical evaluation and quantitative assessment of spasticity were carried out before MNBB, 30 min after MNBB and 19 months after surgery.

This study was approved by the local ethics committee, and both patients provided written informed consent.

RESULTS

At baseline, both clinical evaluation and quantitative assessment of ankle plantar flexor muscles stiffness demonstrated a marked spasticity in the 2 patients (Table I and Fig. 1). The elastic and

viscous components of stiffness are shown as a function of oscillation frequency in Fig. 1A and 1B. Before treatment, elastic stiffness followed an abnormal V-shaped curve, with maximum values at the low and high frequencies of oscillation and minimal values at intermediary frequencies. As stated earlier, this is not normal; normal subjects exhibit elastic stiffness that is independent of velocity. Viscous stiffness also showed pathological results, with higher values at high and low frequencies of oscillation and lower values at intermediate frequencies (“S-shaped path”). In normal subjects, viscous stiffness increases as a function of velocity. The path length (L_{path}) of the phase diagram between elastic and viscous stiffness (Fig. 1C and Table I) quantifies the reflex response to movement and reflects the importance of the spasticity. Before treatment, the phase diagram followed a C-shaped path with abnormal high values for the L_{path} .

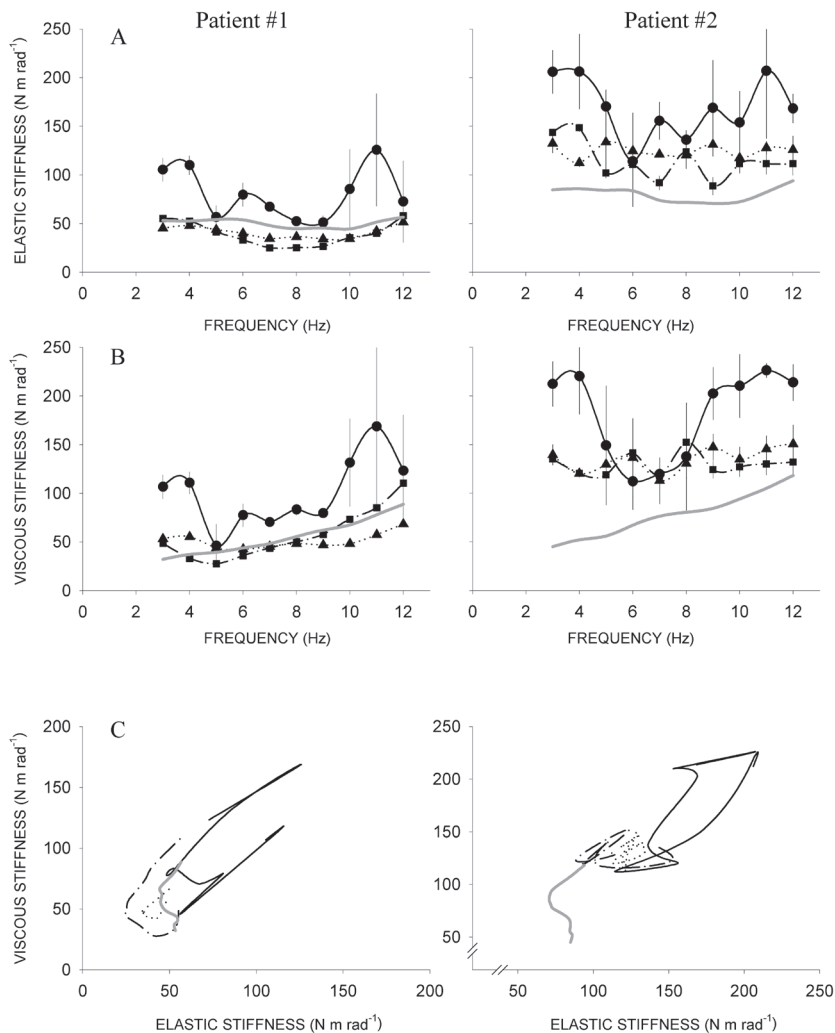


Fig. 1. The left-hand graphs show data for patient 1 and the right-hand graphs for patient 2. For both patients, the 2 curves in the upper panels (A and B) trace the time course of elastic and viscous stiffness of the ankle plantar flexor muscles expressed in N m rad⁻¹ by oscillation in frequency (in Hz). The lower panel (C) shows the phase diagram of viscous stiffness as a function of elastic stiffness. Circles (●) represent the median value for stiffness before motor block. Squares (■) represent the median value for stiffness 30 min after selective motor block. Triangles (▲) represent the median value for stiffness after neurotomy. The grey line is the median value for stiffness in normal subjects.

Table I. Clinical assessment and path length in the phase diagram

	Before MNBB	After MNBB	After surgery*
<i>Patient 1</i>			
Ankle ROM in dorsiflexion, knee flexed (°)	5	5	10
Ankle ROM in dorsiflexion, knee extended (°)	0	0	5
Spasticity (Tardieu scale /4)	4	0	0
Manual Muscle Testing of ankle dorsal flexors (/5)	2	2	2
Manual Muscle Testing of ankle plantar flexors (/5)	1	1	1
Path length in the Phase Diagram (N m rad ⁻¹)	354	127	60
<i>Patient 2</i>			
Ankle ROM in dorsiflexion, knee flexed (°)	20	20	15
Ankle ROM in dorsiflexion, knee extended (°)	10	15	0
Spasticity (Tardieu scale /4)	4	2	1
Manual Muscle Testing of ankle dorsal flexors (/5)	5	5	5
Manual Muscle Testing of ankle plantar flexors (/5)	2	2	2
Path length in the Phase Diagram (Nm rad ⁻¹)	409	231	162

*Patient 1 = 7 months after surgery; Patient 2 = 19 months after surgery. MNBB: motor nerve branch block; ROM: range of motion.

Patient 1 showed a clinical improvement in spasticity after MNBB and after surgery, with a decrease in the Tardieu scale value from 4/4 to 0/4 (Table I). Ankle ROM was improved after surgery because of the associated orthopaedic gesture. Quantitative assessment of spasticity confirmed our clinical results. MNBB and neurotomy allowed a similar improvement in elastic and viscous stiffness (Fig. 1A and 1B) with a normalization of the curves. C-shaped path and L_{path} (Fig. 1C and Table I) exhibited closer to normal values after MNBB (from 354 N m rad⁻¹, 13.1 times normal value, to 127 N m rad⁻¹, 4.7 times normal value) (4) and, slightly more importantly, after surgery (60 N m rad⁻¹, 2.2 times normal value).

Patient 2 had a slightly greater clinical improvement in spasticity after neurotomy than after MNBB, with a decrease in Tardieu scale from 4/4 to 2/4 after MNBB and to 1/4 after neurotomy (Table I). Several months after surgery, we observed a decrease in ankle ROM in dorsiflexion, which was more significant when the knee was extended than when the knee was flexed. Quantitative assessment of spasticity showed a near-normalization of the elastic and viscous stiffness shape curves (Fig. 1A and 1B) with a downward shift, almost similarly after MNBB and after surgery. C-shaped path and L_{path} (Fig. 1C and Table I) exhibited values closer to normal after MNBB (from 409 N m rad⁻¹, 6.1 times normal value, to 231 N m rad⁻¹, 3.4 times normal value) and, slightly more importantly, after surgery (162 N m rad⁻¹, 2.4 times normal value).

DISCUSSION

Our results highlight the important contribution of the soleus muscle to the spasticity of the plantar flexor muscles. The two

patients described here were quantitatively assessed in a knee extended position, stretching both the soleus and gastrocnemius muscles. Motor block and neurotomy of the motor nerve branch to the soleus muscle allowed a normalization of elastic and viscous stiffness of the triceps surae muscle in the first patient, and results reaching closer to normal values in the second patient. The predominance of the soleus muscle over gastrocnemius in the pathogenesis of spastic equinus foot has already been described by Decq et al. (1) and Buffenoir et al. (10). This predominance can be explained by several histological and biomechanical arguments. Additionally, this predominance has been illustrated in a recent case report by Deltombe et al. (5). In this case report, we carried out an objective evaluation of a simulated effect of a motor block instead of a neurotomy on the spasticity of the triceps surae muscle. To our knowledge, this is the first time that this has been quantitatively assessed. Our results confirm the importance of predicting the effect of selectively blocking the upper soleus nerve in preoperative assessment.

In conclusion, these case reports highlight the fundamental role of the soleus muscle in triceps surae spasticity and the effectiveness of soleus neurotomy in spastic equinus foot.

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