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Relevance of botulinum toxin injection and nerve block of rectus femoris to kinematic and functional parameters of stiff knee gait in hemiplegic adults

Johanna V.G. Robertson [a,*], Didier Pradon [b], Djamel Bensmail [b], Christophe Fermanian [d,e], Bernard Bussel [b], Nicolas Roche [c,e]

[a] CNRS UMR 8119, Laboratoire de Neurophysique et Physiologie, 45 rue des St Pères, 75 006 Paris, France
[b] Service de Médecine Physique et Réadaptation, Hôpital Raymond Poincaré, AP-HP, 104 boulevard Raymond Poincaré, 92380 Garches, UVSQ, France
[c] Service d'Explorations Fonctionnelles; Hôpital Raymond Poincaré, AP-HP, 104 boulevard Raymond Poincaré, 92380 Garches, UVSQ, France
[d] Unité de Recherche Clinique Paris-Ouest, Hôpital Ambroise Paré, 9 avenue Charles-de-Gaulle, 92100 Boulogne-Billancourt, France
[e] Centre d'Investigation Clinique et d'Innovation Technologique, Hôpital Raymond Poincaré, AP-HP, 104 boulevard Raymond Poincaré, 92380 Garches, France

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ABSTRACT

Stiff knee gait (SKG) is common in hemiplegic patients. The main focus of treatment is rectus femoris (RF) spasticity. The aims of this study were to evaluate the effect of botulinum toxin injection (BTI) in the RF muscle on peak knee flexion during swing phase and its quantitative and functional impact on gait. We also wished to evaluate the correlation between the effects of nerve block and BTI on peak knee flexion. 10 adult hemiplegic subjects (> 6 months post stroke or traumatic brain injury) with SKG and inappropriate RF EMG activity during mid-swing phase were included. 3D gait analysis, clinical and functional assessments (Timed Up and Go test, 10 m walk test, 6 min walk test and the time taken to ascend and descend a flight of stairs) were performed initially, 30 min after anaesthetic block of the RF nerve and one month post BTI. After BTI, there was a significant increase in knee flexion (8° average) and a tendency towards improvement in gait and functional parameters. The effect of the nerve block on peak knee flexion was significantly correlated with the effect of BTI (11° average increase in peak knee flexion after nerve block). We challenge the relevance of RF nerve blocks in this population when EMG and kinematic data are available. Our results indicate that BTI is an effective treatment for SKG in adult hemiplegic subjects, with a significant increase in peak knee flexion, no reduction in hip flexion and a tendency towards functional improvements.

1. Introduction

Stiff Knee Gait (SKG) is characterised by a decrease in peak knee flexion during swing phase of gait and is commonly observed in patients following stroke, traumatic brain injury or cerebral palsy.

Various mechanisms have been attributed to SKG, the most common being spasticity of the rectus femoris muscle (RF). Rectus Femoris spasticity has been associated with an increased knee extension moment and decreased knee flexion velocity at toe-off, both of which potentially decrease peak knee flexion [1]. Goldberg et al. (2003) [2] suggested that knee flexion angular velocity at toe-off has an important influence on range of knee flexion during swing. Indeed, this author has suggested that conditions during pre-swing phase are important in determining events during swing phase [2,3]. Other causes of SKG cited in the literature are spasticity of the vastii [3], decreased hip flexion moments [4,5] and decreased ankle plantar flexion moments [6,7]. The exact mechanisms remain, however, unclear and seem to be variable.

RF spasticity is the most common focus of treatment for SKG. Surgical procedures such as RF tenotomy with or without transfer are carried out in children with cerebral palsy [8,9] but are not considered as a treatment of choice in adults. Botulinum toxin injection (BTI) is a common treatment for spasticity in adults and children however; its effect has rarely been studied in the RF muscle for the treatment of SKG [10]. BTI blocks synaptic transmission at the neuro-muscular junction. Its effect is temporary, lasting around 3 months. This treatment has few side effects and would seem to be a treatment of choice for adult patients with SKG and inappropriate RF activity.

Temporary anaesthetic motor nerve blocks are carried out in order to determine the therapeutic management of spasticity [11,12]. The effect lasts approximately 4 h making it a useful evaluation tool. In SKG, RF nerve block is used to determine this muscle's role in diminishing knee flexion but its usefulness has

* Corresponding author. Tel.: +33 1 42 86 20 80.
E-mail address: johanna.robertson@univ-paris5.fr (Johanna V.G. Robertson).

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rarely been studied. Indeed, the relationship between the effect of RF nerve block compared with BTI on peak knee flexion, is little-known.

This study was carried out in a population of adult hemiplegic subjects with SKG and inappropriate RF EMG activity during mid-swing phase. There were two main aims. The first was to evaluate the effect of BTI on peak knee flexion during swing phase, and to assess the quantitative and functional impact on gait. We also wished to verify that decreased hip flexion did not occur as a result of decreasing RF activity. Second, we aimed to verify if the effect of a nerve block was correlated with the effect of BTI on peak knee flexion during swing and thus to evaluate its relevance in this population.

2. Methodology

2.1. Subjects

157 gait analyses (131 patients) were performed in our gait lab between July 2005 and July 2006. During this time, 18 consecutive individuals with hemiplegia were referred by a physician for evaluation of SKG in our gait lab. Of these 18 patients, 12 fulfilled the inclusion criteria: over 18 years old, more than 6 months post stroke or traumatic brain injury (TBI), decrease in peak knee flexion of more than 2 standard deviations (normal = 58.3 ± 3.8°), ability to walk 10 m without walking aids, RF EMG activity in mid swing (considered as inappropriate) and successful nerve block (less that 50% of EMG RMS remaining). Two subjects, however declined participation: 1 was concerned about unwanted effects with BTI and 1 moved home. Ten subjects consented, and were included (Table 1).

Gait analysis and a clinical and functional evaluation were carried out on three occasions: at inclusion (PRE), half an hour after nerve block (BLOCK) and 1 month after botulinium toxin injection (BTI). Three subjects only carried out the gait analyses (and not the clinical and functional tests) due to constraints regarding the fact they were seen as outpatients.

2.2. Clinical and functional analysis

Saggital hip, knee and ankle joint range of motion was assessed in order to check for muscle contractures. Spasticity of the quadriceps and RF was evaluated using the modified Ashworth scale [13].

Functional ability of each subject was assessed using the Timed Up and Go Test (TUG) [14], 10 m walk test [15], 6 min walk test (6 MWT) [16] and the time taken to ascend and then descend a flight of 13 stairs.

Subjects were asked to rate their perception of improvement or worsening of their gait using a visual analogue scale.

2.3. Gait analysis

Three-dimensional gait analysis was performed using the Motion Analysis System (60 Hz, Motion Analysis Corporation) and 2 forceplates (1000 Hz, AMTI). The Helen Hayes marker set was used. The subjects walked barefoot at their selected pace. A minimum of 10 gait cycles was averaged for the kinematics analysis. The signal was filtered using a low-pass Butterworth filter with a cut off frequency of 6 Hz [17]. Orthotrak 6.2.8 was used to perform the inverse dynamics calculations.

Motion curves for each joint were established and angular velocity was calculated by derivation of the displacement data. Phases of the gait cycle were defined according to Perry [18]. Peak joint moments during pre-swing were calculated for the ankle, knee and hip using inverse dynamics and were normalised for body weight.

Table 1
Demographic characteristics of each subject

Subject	Sex	Age (years)	Height (cm)	Weight (kg)	Pathology	Time since onset (years)
1	M	27	180	74	CVA L-hemi	4
2	M	40	188	73	CVA L-hemi	0.5
3	M	58	160	69	TBI R-hemi	5
4	M	33	180	85	CVA L-hemi	3
5	M	29	166	72	CVA L-hemi	5
6	M	37	182	82	CVA R-hemi	5
7	M	40	181	72	TBI L-hemi	3
8	F	52	162	68	CVA R-hemi	36
9	M	40	173	76	CVA L-hemi	8
10	F	40	163	51	CVA L-hemi	7
MEAN		40	174	72		8

M = male, F = female, CVA = cerebrovascular accident, TBI = traumatic brain injury, L-hemi = left-sided hemiplegia, R-hemi = right-sided hemiplegia.

EMG was recorded simultaneously during the kinematic data collection using bipolar surface electrodes (MA 311) placed on the rectus femoris, vastus lateralis, semimembranosus, tibalis anterior, gastrocnemius medialis and soleus muscles. Vastus lateralis activity was assumed to be representative of the three vastii [18,19]. Digitalised EMG (1000 Hz) was analysed to determine the periods of activity of each muscle.

2.4. Interventions

Nerve block was performed according to the technique described by Sung et al. [20] using lidocaïne 2%. The effectiveness of the block was tested by comparing surface EMG activity recorded before and after the block during a maximal voluntary contraction of hip flexion. A reduction of at least 50% of activity (calculated using the RMS) was considered effective. EMG of a maximal voluntary knee extension contraction was compared before and after block in order to verify that the vastus lateralis nerve had not received lidocaïne.

200U of Botulinum Toxin type A (Botox®) were injected in four anatomical points into the RF muscle belly under electrical stimulation (5 mA).

2.5. Data analysis

Quantitative data from the three conditions investigated (PRE, BLOCK and BTI) were compared using a non parametric test (Sign test) and variables were grouped according to type: kinematic and dynamic; temporo-spatial and functional; and perceived improvement in gait post intervention. A Holm correction [21] was used to take into account the number of comparisons performed in each group. The adjusted p (p_{adj}) was compared with a significance level of 0.05.

Spearman's rank correlation (univariate) was used to test for correlations between peak knee flexion angle in BLOCK and peak knee flexion angle in BTI and between percentage change in peak knee flexion and percentage of subjective improvement in both conditions.

Mean values are given in the text to allow comparison with values in other studies. The median values on which the statistical analyses were carried out are given in Table 2.

If the mean value of a parameter for a hemiplegic subject was within two standard deviations of mean data from 10 healthy subjects collected in our lab, it was considered normal.

3. Results

3.1. Effect of interventions on kinematics

Average peak knee flexion in swing phase significantly increased by 11° in BLOCK and 8° in BTI (PRE = 27.4 ± 8.2°, BLOCK = 38.2 ± 10.7°, BTI = 35.0 ± 8.8° ($p_{adj} < 0.05$) (Table 2, Fig. 1).

Peak knee flexion angle was not significantly different in BLOCK compared to BTI.

A correlation coefficient of 0.77 ($p < 0.05$) was found between peak knee flexion following BLOCK and peak knee flexion following BTI (Fig. 2).

Average peak hip flexion angle was within normal limits in PRE (24.6° ± 9.9) and tended to increase in BLOCK and BTI (BLOCK = 29.0 ± 8.2°, BTI = 27.6 ± 8.4°) (Table 2).

Knee angular velocity at toe off significantly increased in BLOCK and BTI (PRE = 119.6 ± 69.5°/s, BLOCK = 162.1s ± 53.7°/s, BTI = 172.5 ± 53.7°/s ($p_{adj} < 0.05$)).

The angular velocity curve of saggital knee flexion (Fig. 3) in PRE demonstrates a low peak velocity and low velocity at toe off compared to healthy subjects. As angular velocity changes direction from flexion to extension during swing phase, there is a stalling phase which is particularly obvious in PRE but diminishes in BLOCK and BTI. This phase probably relates to the characteristic dimpling observed in saggital knee displacement curves in SKG.

3.2. Effect of interventions on kinetics

Average peak hip flexion moment in pre swing phase was within normal limits in PRE and was not significantly altered in BLOCK or BTI (PRE = −0.49 ± 0.23 N m kg^{-1}, BLOCK = −0.52 ± 0.29 N m kg^{-1}, BTI = −0.56 ± 0.28 N m kg^{-1}).