Effect of botulinum toxin injection in rectus femoris in stroke patients with stiff knee gait: Analytical effect and in gait re-organization

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Keywords: Stroke; Spasticity; Botulinum toxin; Motion analysis; Isokinetic dynamometer

Objective.– Over the past few years, several studies have shown that a botulinum toxin (BTX-A) injection in the rectus femoris muscle (RF) could enhance stroke patients’ walking capacity (Robertson et al., 2009). However, the mechanisms behind these benefits are still unknown. The aim of this study was to compare the analytical effect of BTX-A in the RF muscle (passive stretch and voluntary strength assessed by an isokinetic dynamometer) and functional repercussions on gait (assessed by three-dimensional motion analysis) in spastic hemiparetic patients with stiff knee gait (SKG) caused by RF spasticity.

Methods.– Ten stroke patients with SKG caused by RF spasticity were included and evaluated before and after a BTX-A injection in RF. Analytical evaluations consisted of: (i) a clinical examination; (ii) an assessment using an isokinetic dynamometer with surface electromyography (in order to quantify passive stretch and knee flexor and extensor voluntary strength; (iii) a three-dimensional motion analysis gait assessment (in order to quantify spatiotemporal and kinematic gait parameters).

Results.– One month after BTX-A injection in RF, the following results were found: (i) a decrease in knee extensor strength and an increase in knee flexor strength; (ii) no change in the intensity of extensor activity during passive stretch; (iii) an increase in the angle at which the stretch reflex occurred. 3D analyses showed an increase in gait speed, which was related to an increase in cadence and an increase in peak knee flexion during swing phase.

Discussion.– The improvement in knee flexion following BTX-A injection in RF seems to be related to an increase in the angle at which the stretch reflex occurs with no change in intensity of the contraction. This change in the threshold of the sensitivity of neuromuscular spindles infers that BTX-A affects the motoneuron γ-neuromuscular spindle synapse (Tronquetto et al., 2006).

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Biomechanical, clinical and gait analysis’ effects of BotNTA injection in the rectus femoris muscle of incomplete SCI patients

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Keywords: Botulinum toxin; Rectus femoris; Spasticity; Stretch reflex; Stiff-knee gait; Torque

Background.– An understanding of the mechanical effects of botulinum toxin types A (BoNTA) on spastic and voluntary muscle contraction may help predict functional responders.

Objective.– To compare the effect of BoNTA on voluntary and stretch reflex-related torque (and angle at peak torque) produced by activation of the rectus femoris (RF) muscle.

Methods.– A prospective open pilot study. n = 15 incomplete SCI patients, impaired by a specific RF spasticity (n = 20), with RF hyperactivity in mid-swing and a stiff-knee-gait quantified by formal Gait Analysis (GA), were assessed before and after BoNTA injection (Botox, 200 UI). Main outcome

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Generalized fatigue after Botulinum neurotoxin A (BoNTA): Research of systemic diffusion with single fiber EMG

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Keywords: Botulinum toxin; Botulism-like syndrome; General weakness; Single fiber EMG

Aim.– To establish if patients reporting unusual fatigue present generalized diffusion of BoNTA. Fatigue after BoNTA injection is frequently reported in literature (up to 18% of injected patients).

Material and methods.– Retrospective, monocentric study. Generalized diffusion has been searched with a single fiber EMG (or Neuroumuscualr Jitter) on striated muscles away from BoNTA injected muscles. Neuromuscular jitter (NMJ) is compared between 16 patients with unusual fatigue after BoNTA injection, 17 asymptomatic patients treated with BoNTA (control group), 19 patients suffering botulism-like syndrome having muscular deficit distant from BoNTA injection site and three patients suffering botulism food poisoning. Indications were treatment of neurogenic detrusor overactivity or spasticity. NMJ is the gold standard for evaluating neuromuscular junction dysfunction. Mean jitter, percentage of pathological fibers and number of conduction blocks are compared between groups.

Results.– Mean jitter, percentage of pathologic fibers and conduction blocks for asymptomatic patients differs from those of patients presenting distant muscular deficit (P = 0.0001) and patients suffering botulism (P = 0.017). Mean jitter and number of conduction blocks differs between patients presenting unusual fatigue and patients presenting distant muscular deficit (P = 0.0005). No significant difference appears between asymptomatics and patients relating unusual fatigue after BoNTA treatment.

Discussion.– This study doesn’t give rise to generalized BoNTA diffusion in patients reporting unusual fatigue after BoNTA treatment although jitter clearly differs between patients presenting distant muscle weakness and control group. Generalized neuromuscular disorder doesn’t seem to explain those fatigue events. Several hypotheses can account for it: not enough patients in the group reporting unusual fatigue, fatigue could be due to autonomic nervous system disorder that is not explored with this single fiber EMG, immune mechanism. Fatigue could be reported to initial disease.

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