Intraventricular baclofen for multifocal spasticity

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Introduction.– The natural target of the botulinum neurotoxin type A (BoNT-A) is the neuro-muscular junction. Botulinum neurotoxins induce flaccid paralysis by inhibiting synaptic transmission in cholinergic synapses. BoNT-A is known to block central synapses after muscular injections due to retrograde transport in animal models. In humans, the question of a possible direct central action of BoNT-A is still debated. The present study was designed to address whether BoNT-A modifies the activity of the spinal recurrent inhibitory pathways, when injected at muscular level.

Patients and methods.– Experiments were performed on 14 post-stroke patients exhibiting spasticity in ankle plantarflexors, before injection then one month after BoNT-A. The protocol for the study of heteronomous recurrent inhibition from soleus to quadriceps motoneurones is based on the pattern of distribution of recurrent inhibition in human lower limbs. Inhibition was revealed by testing the influence of PTN stimulation on quadriceps Hoffmann reflex amplitude (VL).

Results.– One month after BoNT-A, the level of recurrent inhibition was depressed significantly; average – 45.2 ± 9.2% (P < 0.001). No relationship was found between the date of stroke and loss of inhibition. Loss of inhibition was stronger in patients who had not received BoNT-A before the investigation (P < 0.02) and in patients who had 5–6 injections sites than in patients who had 3–4 injections sites (P < 0.05).

Discussion.– We suggest that catalytically active BoNT-A may act presynaptically, reducing acetylcholine release in motoneurone recurrent terminals projecting on Renshaw cells impinging on VL motoneurones. Recurrent inhibition is normally depressed to assist upright position and stance phase of walking, partly via the inhibitory corticospinal control on Renshaw cells, which may contribute to muscle synergy. After stroke, an altered descending drive may be responsible for the lack in task-dependent modulation of Renshaw cell activity, that may make muscle synergies less flexible. The BoNT-A induced depression of recurrent inhibition could thus compensate for the lack of inhibitory corticospinal control after stroke and contribute to improve motor synergy in the process of functional recovery of locomotion and upright position.

Further reading

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Pain assessment during injection sets of botulinum toxin for upper limb spasticity treatment

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Introduction.– Injection sets of botulinum toxin are painful. What is the part due to the four times of the technique: skin breaking, electrical stimulation for localization, injection, needle withdrawal? Although clinically important, this question has been little addressed. This was the objective of this study.

Method.– Prospective study including 26 patients (16 M/10 W, age 61.4 ± 14years) treated without analgesia by Botox® or Dysport® for upper limb spasticity (5th injection set in average), 9.1 years after a stroke. Pain intensity was assessed using a verbal scale from 0 to 10, after each of the 4 steps (skin breaking, electrical stimulation, product injection, withdrawal of the needle) of every injection. Sensory loss was quantified using Semmes-Weinstein filaments. Statistics were nonparametric and data expressed as follows: average [95% LCL and UCL].

Results.– The average number of muscles injected per patient was 3.4 and the average number of injections per muscle 1.6. Electrical stimulation was the most painful time (4.4 [3.3–5.4]; P < 0.001), followed by skin breaking (3.1 [2.1–4.1]; P < 0.01). Pain at injection was not negligible (1.6 [0.9–2.3]), greater than pain accompanying the withdrawal of the needle (0.8 [0.3–1.4]; P < 0.05). No significant correlation was found between pain intensity and clinical characteristics of patients, including sensory loss.

Discussion.– This study specifies the nature and intensity of pain during treatment by botulinum toxin without analgesia of the upper limb spasticity in adults with stroke. The penetration of the product into the muscle and the withdrawal of the needle may be painful. Stimulation time is the most painful, followed by skin breaking. These findings argue for analgesia associated with an adaptation and a learning of therapeutic techniques in order to reduce pain.

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