

LETTER TO THE EDITOR

BOTULINUM NEUROTOXIN TREATMENT FOR SPASTICITY:  
THE ROLE OF ELECTROMYOGRAPHY GUIDANCE

Various guidance methods have been used to increase the accuracy of botulinum neurotoxin (BoNT) injections. Initially, guidance by anatomical landmarks or “manual placement” was used. However, this method is inaccurate compared with guidance by electromyography (EMG), as demonstrated by Molloy et al. (1) and compared with guidance by electrical stimulation of the muscles, as demonstrated by Chin et al. (2). The introduction of ultrasound guidance has increased the accuracy of the treatment, and this method is now used by many centres involved in BoNT treatment of spasticity.

Electrical stimulation of the muscles and ultrasound guidance are used to localize the muscles that are selected for treatment and are, in this respect, directly correlated. EMG examination fulfils a different role; it provides little information about localization of the specific muscle, but it uniquely provides information about the involuntary activity of the muscles. Use of ultrasound guidance for accurate injection has decreased the use of EMG guidance. This raises questions about the future role of EMG guidance: Can we afford to exclude information about the involuntary EMG activity of the muscle? Does EMG guidance still have a role in treating spasticity with BoNT?

It was pointed out 30 years ago, that the main gait impairment in children with cerebral palsy is not related to spasticity, but to changes in the properties of the passive elastic elements in muscles, tendons and joints (3). Subsequent experiments showed that changes in passive muscle properties are also dominant and, to some extent, indistinguishable clinically from spasticity in adults with stroke, spinal cord injury, multiple sclerosis and hereditary spastic paraparesis (4–8).

Without appropriate determination of the cause of hypertonia, which requires biomechanical and electrophysiological (EMG) evaluation (4), there is a risk of injecting BoNT into a muscle that is not hyperactive but only shows alterations in passive muscle properties. The mechanism of action of BoNT alone makes it unlikely that BoNT would have an effect on these passive properties (9).

New findings have demonstrated that exaggerated stretch reflexes (spasticity) are not responsible for the majority of functional problems in the patients (10). There is therefore a risk that BoNT injection into a silent muscle, based only on the finding of spasticity in the neurological examination, will not be effective.

Exaggerated stretch reflex activity is generally only present in spastic patients at rest and not during voluntary activation (11). This has to do with the normal integrative control of stretch reflex activity in relation to movement. In healthy subjects, low stretch reflex activity is maintained in the resting state through a number of inhibitory spinal mechanisms (12).

In relation to voluntary movements, this spinal inhibition is removed and stretch reflex excitability is increased in order for sensory feedback mechanisms to contribute efficiently to activation of the muscles (12). In spastic patients, because the spinal inhibitory mechanisms are deficient, stretch reflex excitability is abnormally high in the resting state, but during voluntary movement the lack of inhibition is advantageous since the sensory feedback may thereby contribute efficiently to muscle activation, as in healthy subjects. Injection of BoNT into a silent muscle showing hyper-reflexia would therefore not necessarily be expected to have beneficial functional effects.

Exaggerated stretch reflex activity is also unlikely to cause sustained (involuntary) muscle activity and inappropriate postures, since the stretch reflex is highly dynamic and EMG activity is generally seen for only a very restricted period following a sensory stimulus. The stretch reflex circuitry also generally does not provide much force and therefore is unlikely to produce the strong sustained muscle contractions observed in many spastic patients. The sustained, involuntary muscle activity in persons with spasticity shares characteristics with dystonia and is, similar to dystonia, in all likelihood caused by descending motor activity secondary to lesions of the basal ganglia. Hence, it has been proposed that the sustained involuntary muscle activity in spastic patients should be named “spastic dystonia” (13, 14).

At present, there is no direct evidence showing that the effect of BoNT on spasticity depends on the EMG activity of the injected muscle. Thus, further research is needed to clarify this issue.

If it is demonstrated that BoNT does have a beneficial effect on the “EMG silent” muscle, then EMG investigation may be omitted and ultrasound guidance and/or guidance by electrical stimulation may be applied without EMG.

On the other hand, if BoNT does not affect the “EMG silent” muscle, then EMG examination should be used to identify those patients who are likely to benefit from the treatment, and it would then be rational to apply EMG examination in combination with ultrasound guidance and/or guidance by electrical stimulation for localizing the right place to inject BoNT in the selected muscles.

Until the effect of BoNT on “EMG silent” muscles in spasticity has been further clarified, it is suggested to apply a combination of ultrasound and EMG. This approach also makes it possible to apply electrical stimulation together with ultrasound for localization of the muscles.

In daily clinical practice, as well as in the setting of a clinical study, differentiation between muscles with and without involuntary EMG activity is suggested.

*Conflicts of interest*

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*Ole Jakob Vilholm, PhD\* and Jens Bo Nielsen, MD*  
 From the Department of Neurology, Lillebaelt Hospital, Vejle and Department of Nutrition, Exercise and Sport Sciences and Department of Neuroscience and Pharmacology, Panum, University of Copenhagen, Denmark.  
 \*E-mail ole.vilholm@rsyd.dk