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LETTER TO THE EDITOR

POTENTIATION OF BOTULINUM TOXIN TYPE A WITH ORAL ANTI-SPASTICITY MEDICATIONS IN THE MANAGEMENT OF FOCAL SPASTICITY

Sir,

We read with interest the recently published study by Bergfeldt et al. (1) describing focal spasticity therapy with botulinum toxin. However, there are some important points that need to be clarified.

When botulinum toxin is injected into the neuromuscular junction of a spastic muscle, that muscle will relax for a period of 4–6 months. Other interventions, such as physiotherapy should be used to improve muscle strength and coordination. An important point to be stressed is the role of physical therapy. There is general agreement that physical therapy is necessary to rehabilitate patients with focal spasticity. However, formal evidence is still lacking in order to establish the real benefit of physical and occupational therapy in the rehabilitation of focal muscle spasticity (2).

Another treatment approach that was not mentioned in the study by Bergfeldt et al. (1) is the efficacy of oral anti-spasticity agents. Most cerebral palsy conditions are managed with a combination of modalities. General consensus is to reduce the spasticity first with botulinum toxin type A (BTX-A) or orthopaedic surgery, and then to fit an ankle–foot orthosis. A variety of oral medications have been used to diminish the sensitivity of local nerves and muscles to control their reactions to environmental stimuli that result in muscle spasticity. Baclofen and tizanidine are examples of the most frequently prescribed oral agents, which have been studied extensively in adults with spasticity from a variety of causes and have been found to be of proven benefit (3). The use of intramuscular injections of BTX-A and oral medication to manage focal spasticity may potentiate the anti-spasticity effect and also lower the required doses of orally administered drugs to reduce spasticity. All these have different modes of action and clinical efficacy. Combination treatment, such as BTX-A and oral agent, commonly used in clinical practice can potentiate their effects remarkably. BTX-A can be used in conjunction with oral anti-spasticity treatments and this adjunctive treatment works quite adequately, and with remarkably good tolerance, with BTX-A.

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RESPONSE TO LETTER TO THE EDITOR BY ALPER I. DAI

We thank Dr Dai for the interest in our work, and welcome the opportunity to respond.

With regard to the importance of physical and/or occupational therapy, we concur with Dr Dai's comment, and again emphasize that the results were obtained by combining the botulinum toxin injections with 260 additional therapeutic interventions. These interventions were physiotherapy (70), assisted home-training (43), occupational therapy (18), speech therapy (1), and different orthoses/orthopaedic shoes (88), on average 2.6 per patient. We also agree that only a randomized, controlled study comparing combination therapy with physiotherapy or occupational alone would provide an answer to the issue raised. However, based on the results of our (approximately 90% response to therapy) and other studies in similar adult patient groups, such a study would probably be based on testing a non-inferiority hypothesis regarding physiotherapy alone.

Approximately 50% of the patients in our study group received oral anti-spasticity therapy, which thus was both insufficiently effective with regard to their principal therapy targets, and was kept unchanged throughout the study. Again only a randomized controlled study could provide an answer as to both positive and negative effects of combining oral and focal spasticity therapy. Because of some well-known adverse effects of the presently available oral alternatives, a focal agent with longer-lasting effect and without systemic effects would be welcome.

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