

## Strategies for the Abnormal Co-Contraction of Internal Rotators of the Shoulder in Children with Obstetrical Brachial Plexus Palsy

Chan Chen, MD, PhD, Fang Li, MD, PhD

Chan Chen, Fang Li, Department of Rehabilitation Medicine, Huashan Hospital, Fudan University, Shanghai, 200040, China

Fang Li, Department of Rehabilitation Medicine, Huashan Hospital, Fudan University, Shanghai. Renhe Hospital Baoshan district of Shanghai. China.

Correspondence to: Fang Li, MD, PhD, Department of Rehabilitation Medicine, Huashan Hospital, Fudan University, Shanghai. Renhe Hospital Baoshan district of Shanghai. China.

Email: [fangli@fudan.edu.cn](mailto:fangli@fudan.edu.cn)

Telephone: +86-21-52887820

Fax: +86-21-52887820

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### ABSTRACT

Obstetrical brachial plexus palsy (OBPP) is caused by traction of the brachial plexus during delivery. The incidence of OBPP was once reported to be 2.9 per 1000 live births, and the incidence of persisting OBPP was 0.46 per 1000. Although most patients with OBPP recover spontaneously in the first 2 months, up to 35% are left with varying degrees of shoulder weakness, contracture, or joint deformity. Even after neuro-reconstructive surgery, children with incomplete recovery often have abnormal motor performance.

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**Key words:** Co-Contraction; Internal Rotators; Shoulder; Obstetrical Brachial Plexus Palsy

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### LETTER TO THE EDITOR

Obstetrical brachial plexus palsy (OBPP) is caused by traction of the brachial plexus during delivery. The incidence of OBPP was once reported to be 2.9 per 1,000 live births, and the incidence of persisting OBPP was 0.46 per 1,000<sup>[1]</sup>. Although most patients with OBPP recover spontaneously in the first 2 months, up to 35% are left with varying degrees of shoulder weakness, contracture, or joint deformity<sup>[2]</sup>. Even after neuro-reconstructive surgery, children with incomplete recovery often have abnormal motor performance<sup>[3]</sup>.

The upper trunk of the brachial plexus (C5 and C6) is most commonly affected in OBPP<sup>[3,4]</sup>. Paresis of the deltoid, supraspinatus, infraspinatus, and teres minor, which are innervated by the upper trunk, results in poor performance of the shoulder abduction/external rotation. The relatively unaffected or better recovered internal rotators can lead to adduction/internal rotation contracture of the shoulder due to the muscular imbalance. Besides, the internal rotators are often overactive while the patients flex, abduct, or externally rotate their shoulders. The phenomenon of this overactivation is called abnormal co-contraction, which is something like unilateral synkinesis after facial palsy that can be treated by botulinum neurotoxin<sup>[5]</sup>.

On physical examination, the muscle tightness of the latissimus dorsi and teres major can often be palpated. Active range of flexion and abduction are limited. The patients perform movements of arm elevation usually with elbow flexion, scapular wing protruding outward, and trunk extension. The complex movements such as hand-to-mouth and hand-on-neck are hard to accomplish. Co-contractions can be identified by physical examination and further confirmed by the electromyography (EMG) study. Abnormal co-contractions of the internal rotators impair the active range of motion of shoulder abduction/external rotation, impede the rehabilitation of the affected arm, and cause the functional loss of daily life activities such as putting food into the mouth, dressing, high reaching, and caring of hair.

In our preliminary observation, abnormal co-contraction of internal rotators tends to be found in older children with OBPP and become an obstacle to further functional progress. A popular theory for the explanation of co-contraction is the abnormal branching and misrouting of peripheral nerves during regeneration and reinnervation. When co-contraction appears, the antagonist is abnormally activated with the agonist. Improper exercise may enhance the power of the antagonist as well as the agonist, which could impede the progressive functional recovery at later age. Besides, the deafferentation of the affected prime muscles and the aberrant outgrowth of motor axons to the depressor muscles may hinder the proper central motor programming by presenting conflicting information<sup>[6]</sup>. In these patients, the serial and smoothly coordinated movements to do specific tasks are replaced by “trick movements” such as scapular rotation instead of glenohumeral rotation as a functional adaptation<sup>[6,7]</sup>. The abnormal co-contraction phenomena in OBPP can also be observed in the biceps-triceps couple, which is not discussed here.

The current operative management of the residual shoulder sequela is to release the contracture such as subscapularis slide from its origin, or restore the active external rotation by muscle transfer, usually the latissimus dorsi/teres major complex, into the rotator cuff. Although the shoulder motion is improved after operations, the long-term outcome and the contribution to the joint remodeling and the synergistic motor function need to be determined<sup>[8]</sup>.

Recently, an increasing number of reports on the treatment of botulinum neurotoxin A (BTX-A) for OBPP have been published<sup>[3]</sup>. BTX-A is a reversible neurotoxin. It reduces the contract strength of a muscle by preventing release of acetylcholine at the presynaptic neuromuscular junction. As one of the 7 serotypes produced by *Clostridium botulinum*, BTX-A is the most studied in therapeutic application. The classical indications of BTX-A treatment include wrinkles, dystonia, and spasticity. For the shoulder movement disorder in OBPP, both the muscle imbalance and the abnormal co-contraction are reasons for BTX-A treatment. By temporarily weakening the strong internal rotators, BTX-A can contribute to better muscle balance of the shoulder. The most commonly injected muscle in previous reports was the pectoralis major, and in varying combination with the latissimus dorsi, teres major, and subscapularis. The active range of shoulder abduction/external rotation increased after BTX-A treatment in most of the studies<sup>[3]</sup>. We applied BTX-A treatment for abnormal co-contraction of the latissimus dorsi/teres major complex and subscapularis confirmed by the surface EMG. At 1 month after BTX-A treatment, most of the patients had better performances of arm elevation and hand-on-neck. The procedure of injection is less invasive compared with surgery. By carefully setting the clear inclusion and exclusion criteria, accurate dosage (6U/kg for children), and injecting under guidance of EMG, the adverse effects can be minimized. BTX-A treatment has been a promising therapeutic choice to treat the abnormal co-contraction, thus facilitate the maintenance of shoulder abduction/external rotation and aid the rehabilitation of shoulder function.

As the effect of BTX-A wears off in about 6 months, the antagonist may return in force at that time. The long-time effect of BTX-A injection needs to be assessed. A 12-month of follow-up study showed the improvement in external rotation was significantly reduced but still present compared with before treatment, and internal rotation had returned to pretreatment values<sup>[9]</sup>. There are also reports of significant improvement of dexterity evaluated by the nine-hole peg test after BTX-A treatment and that persisted for 12 months<sup>[10]</sup>. In our experience, although the abduction of the shoulder improved after

BTX-A treatment with the internal rotators, it was still difficult for the patients to do more complex movements such as hand-on-waist as consistently and efficiently as the unaffected upper limb. Up to now, the mechanism of improvement by BTX-A treatment is thought due to the peripheral muscle balance. It may facilitate the reciprocal muscle activity. While the long-time effect of BTX-A, presumed as facilitating the learning of synergistic relaxation of antagonists, needs to be further investigated, repeated treatment of BTX-A is necessary in some cases with recurrent imbalance and abnormal co-contraction, which can be well monitored by the EMG study. The outcome assessment and contribution of repeated injections to the shoulder function and the coordination movements in OBPP have not been well documented yet.

Last but not the least strategy in the management of the shoulder sequela in OBPP is the physiotherapy, which should never be neglected whether operation or BTX-A treatment is applied. The rehabilitation therapy should start as soon as possible for OBPP. The goal of initial therapy is to maintain passive range of motion and muscle strength. Actually, the more we learn about the neuropathophysiological basis of OBPP, the more challenges to the present maneuvers. A case report suggested a combination of BTX-A treatment of abnormal co-contraction and constraint-induced movement therapy had potential to promote functional gains for children with OBPP possibly through the hypothetical mechanism of an increased peripheral feedback to the sensorimotor cortex for motor pattern learning<sup>[11]</sup>. With a basis of peripheral nerve lesion, the concept of brain plasticity in OBPP is still under evaluation.

In conclusion, the treatment and rehabilitation of the abnormal co-contraction of internal rotators of the shoulder in OBPP are discussed. Key factors to understand and evaluate the present strategies include: (1) the innervation and reinnervation after lesion, (2) balance between the agonist and the abnormal co-contracted antagonist, and (3) outcome assessment at the ICF level of functions and activities. As a promising strategy, BTX-A treatment needs further investigations.

## CONFLICT OF INTERESTS

The authors have no conflicts of interest to declare.

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