# Central Adaptation following Brachial Plexus Injury

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**Key words**
- Apraxia
- Brachial plexus injury
- Central adaptation
- Nerve trauma
- Neuroplasticity

**Abbreviations and Acronyms**
**BPT**: Brachial plexus trauma
**CIMT**: Constraint-induced movement therapy
**CNS**: Central nervous system
**EMG**: Electromyography
**ES**: Electrical stimulation
**H-reflex**: Hoffman reflex
**MRI**: Magnetic resonance imaging
**OBPP**: Obstetric brachial plexus palsy
**PNI**: Peripheral nerve injury

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**Brachial plexus trauma (BPT) often affects young patients and may result in lasting functional deficits. Standard care following BPT involves monitoring for clinical and electrophysiological evidence of muscle reinnervation, with surgical treatment decisions based on the presence or absence of spontaneous recovery. Data are emerging to suggest that central and peripheral adaptation may play a role in recovery following BPT. The present review highlights adaptive and maladaptive mechanisms of central and peripheral nervous system changes following BPT that may contribute to functional outcomes. Rehabilitation and other treatment strategies that harness or modulate these intrinsic adaptive mechanisms may improve functional outcomes following BPT.**

## INTRODUCTION

Brachial plexus trauma (BPT) occurs in two different age groups. Obstetric brachial plexus palsy (OBPP) is a recognized complication of childbirth, with an incidence of 0.3% of deliveries.[1] The next peak in the incidence of BPT is in younger (usually male) adults, and it is often caused by motor vehicle accidents or penetrating wounds.[2]

BPT can result in severe and lasting neurologic impairments, and because the affected patients are young, it has a long-term effect on work and societal roles. As such, optimizing recovery from brachial plexus injury is critical.

## RECOVERY FROM BPT

Recovery of nerve tracts following BPT relies on a complex cascade of peripheral nerve regenerative processes, culminating in target muscle reinnervation.[3] In instances of severe injury to the brachial plexus or nerve roots, successful nerve regeneration might not be possible without surgical brachial plexus reconstruction.[4,5]

Even patients in whom regenerating nerve fibers reach the target muscles, there are substantial barriers to a good functional outcome. The process of axonal regeneration is inefficient, and sprouting fibers may not reconnect with the appropriate fascicle beyond the injured segment.[6,7] Fibers that reach the target muscle do so in substantially reduced numbers, resulting in incomplete reinnervation and a reduced number of functional motor units.[8] Although the dynamics of motor unit recruitment can recover from nerve injury if the muscle is reinnervated by the same motor nerve,[9] aberrant reinnervation can affect this process.[10]

### Maladaptive Central Processes following BPT

Beyond peripheral nerve regeneration, central processes are involved in determining successful or failed functional recovery following BPT.

***Developmental Apraxia.*** The majority of patients with OBPP make an excellent functional recovery, with no residual deficits identified on serial clinician or physiotherapist review.[1] However up to 15% of patients have persisting significant deficits,[1] and the proportion of patients with spontaneous functional recovery may be overestimated.[11]

In patients with residual deficits, a pattern of recovery may be seen, with most deficits seen in C5- to C7-innervated muscles. Forearm pronation typically improves before forearm supination.[12] The degree of recovery of shoulder external rotation, elbow flexion, and forearm supination at 3 months predicts which children will retain persistent functional deficits.[12] In children with residual impairment, functional tasks can typically be performed using the injured limb, but with asymmetric movement relative to the uninvolved limb. Elbow flexion and shoulder abduction are the functional movements that prove most challenging in patients after severe OBPP, but shoulder external rotation and forearm supination are usually the most affected movements and recover last.[12,13] These described patterns of recovery and residual functional impairment inform rehabilitation strategies, although therapy paradigms are largely empiric and are based on splinting of flail wrists and elbows to prevent dislocation and passive range-of-motion exercises to prevent contractures.

In a small subgroup of patients with residual symptoms, electromyography

(EMG) studies of the affected limb demonstrate active motor units in weak muscles, suggesting at least partial reinnervation, but clinical function remains poor.[14] This unique issue has been termed *developmental apraxia* and is considered to be an example of maladaptive central motor programming early in infancy following OBPP.[15] The presence of EMG activity in these clinically weak muscles has been apportioned to "luxury innervation," which refers to the phenomenon of additional muscle innervation from spinal segments beyond those typical for the individual muscle. As such, a typically C5- and C6-innervated muscle, such as biceps, may have luxury innervation from C7, resulting in the presence of active motor units on EMG with minimal functional movement. Luxury innervation begins between weeks 16 and 25 of gestation and is usually lost after the age of 3 months.[16,17] Limb immobilization in the period of sensory and motor brain organization, such as following OBPP, can result in incomplete realization of this reserve function, and harnessing this reserve capacity may benefit recovery.[18]

Investigations of patients with developmental apraxia identified that reduction of motor skills and muscle strength were exaggerated when compared with neurophysiological and physical muscle responses to peripheral nerve stimulation.[15] This finding was interpreted as being indicative of impaired voluntary motor unit activation, suggesting defective motor programming development in early infancy.

A separate study used transcranial magnetic stimulation to examine a cohort of patients with OBPP without evidence of functional recovery.[18] Motor evoked potentials were recordable in all patients, confirming the presence of an intact connection between the motor cortex and muscle, and suggesting that the failure of functional recovery could be due to abnormalities of central motor control processes.

## Illustrative Case 1: Developmental Apraxia

A 17-month-old baby boy suffered a severe left brachial plexus injury at birth resulting in a complete upper trunk palsy on clinical examination. Magnetic resonance imaging (MRI) showed no evidence of nerve root avulsion. EMG showed no voluntary units in his left biceps and shoulder abductor muscles, but little active muscle denervation. A postganglionic injury of the left upper trunk was diagnosed.

He was taken to surgery and after being anesthetized, electrodiagnostic testing was performed. Transcutaneous stimulation of his left Erb's region gave rise to contraction of his left biceps, supraspinatus, infraspinatus, and deltoid muscles. Transcranial electrical stimulation (ES) of the right motor cortex also gave rise to motor evoked potential responses in these muscles (Figure 1).

He underwent surgical exploration of his infraclavicular brachial plexus. A supraclavicular brachial plexus exploration was not performed. Direct stimulation of the left musculocutaneous nerve gave rise to contraction in the biceps muscle. Similarly, direct stimulation of the axillary nerve gave rise to a contraction in deltoid.

Because of a lack of functional recovery in elbow flexion, a side-to-side median to musculocutaneous nerve repair was performed to provide a supplementary source of axons to biceps with minimal iatrogenic neurological morbidity.[19] Postoperatively he

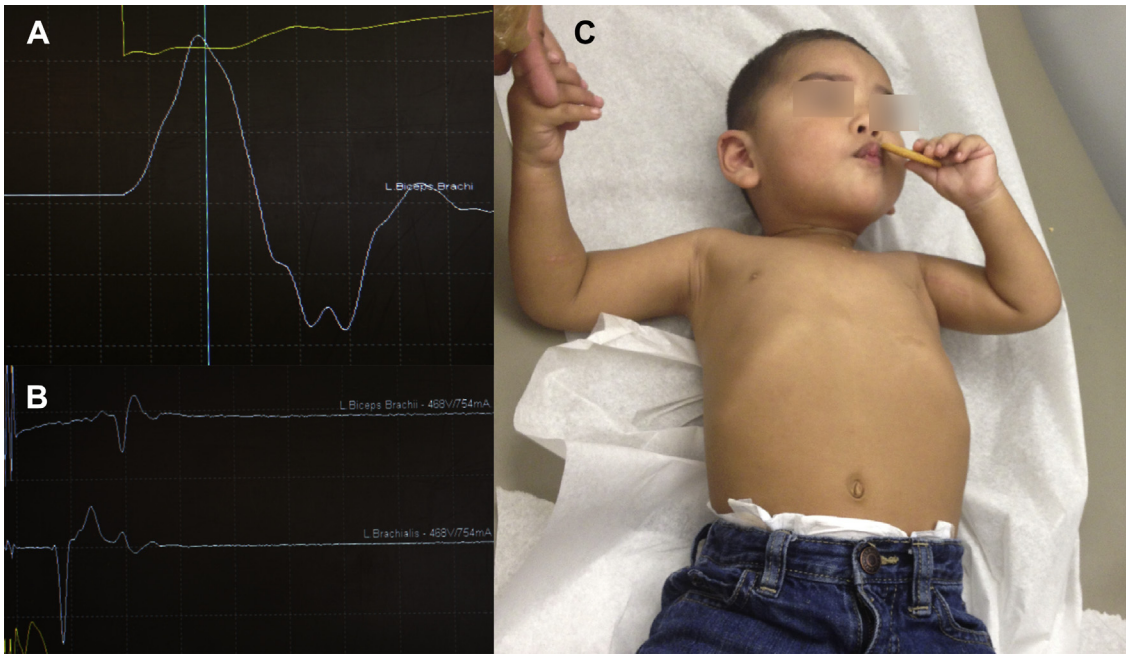


**Figure 1.** Developmental apraxia following obstetric brachial plexus palsy (Illustrative Case 1). Intraoperative electrophysiology (**A**, **B**) identified retained responses from biceps to brachial plexus and cortical stimulation in the absence of voluntary activity of the muscle. (**C**) Three months after a side-to-side anastomosis of the median to musculocutaneous nerve, functional elbow flexion returned.