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PEDIATRIC ORTHOPEDICS (B HEYWORTH, SECTION EDITOR)

The natural history and management of brachial plexus birth palsy

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Abstract Brachial plexus birth palsy (BPBP) is an upper extremity paralysis that occurs due to traction injury of the brachial plexus during childbirth. Approximately 20 % of children with brachial plexus birth palsy will have residual neurologic deficits. These permanent and significant impacts on upper limb function continue to spur interest in optimizing the management of a problem with a highly variable natural history. BPBP is generally diagnosed on clinical examination and does not typically require cross-sectional imaging. Physical examination is also the best modality to determine candidates for microsurgical reconstruction of the brachial plexus. The key finding on physical examination that determines need for microsurgery is recovery of antigravity elbow flexion by 3–6 months of age. When indicated, both microsurgery and secondary shoulder and elbow procedures are effective and can substantially improve functional outcomes. These procedures include nerve transfers and nerve grafting in infants and secondary procedures in children, such as botulinum toxin injection, shoulder tendon transfers, and humeral derotational osteotomy.

Keywords Brachial plexus · Birth palsy · Erb's palsy · Microsurgery · Tendon transfers · Glenohumeral dysplasia

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Introduction

Brachial plexus birth palsy (BPBP) is an upper extremity paralysis that occurs due to traction injury of the brachial plexus during childbirth. BPBP is typically diagnosed at birth due to lack of spontaneous upper extremity movement (Fig. 1). Clinical examination may also reveal a loss of the Moro reflex. The incidence of BPBP is frequently cited as 0.4 to 4 per 1000 live births, although more recent data suggests that incidence may actually be decreasing in the setting of increasing rates of multiparity, labor induction, and cesarean section as well as decreasing rates of macrosomia [1–4]. The variation in reported incidence may be attributed to geographic differences in obstetric care and birth weights. Approximately 10–30 % of infants will have residual neurologic deficits, resulting in permanent alteration in upper limb development and function [5].

Multiple risk factors for BPBP have been identified, including gestational diabetes, macrosomia, shoulder dystocia, prolonged labor, instrumented delivery, breech delivery, and fetal distress resulting in hypotonia [1, 6–8]. Shoulder dystocia increases the risk of brachial plexus birth palsy by 100 times [3]. However, only 46 % of patients have one or more identifiable risk factors [3]. Cesarean section appears to be protective, as it reduces the risk of injury to 0.2 per 1000 live births [3].

Anatomy and classification

The brachial plexus is formed by the C5-T1 nerve roots, which give rise to trunks, divisions, cords, and, finally, terminal branches. Roots are not covered by the protective conjunctive tissue found at the level of the trunks. BPBP lesions more commonly present with involvement of the C5 and C6 nerve roots, as they are anatomically more vertically oriented and

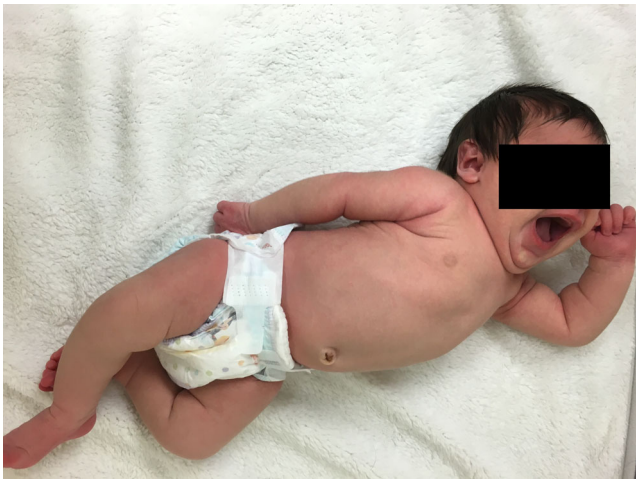

Fig. 1 Typical presentation of Erb's palsy in a female infant. The right arm lies in adduction with the shoulder in internal rotation, the elbow in extension, the forearm in pronation, and the wrist in flexion. The contralateral left side is functioning well with active shoulder external rotation and elbow flexion

vulnerable to traction injury [9]. The most common location of injury is at the upper trunk, also known as Erb's point. Injuries that occur at Erb's point are commonly referred to as Erb's palsy and impact the suprascapular nerve and the anterior and posterior divisions of the upper trunk.

Brachial plexus injuries can be classified into two types: pre-ganglionic and post-ganglionic lesions. Pre-ganglionic lesions occur proximal to the dorsal root ganglion and involve avulsion of the nerve rootlets from the spinal cord. Avulsion injuries most often occur at the lower portion of the brachial plexus (C8 and T1 nerve roots). Pre-ganglionic avulsion injuries cannot be repaired by direct surgery and portend a poor prognosis.

Post-ganglionic lesions occur distal to the dorsal root ganglion. These are more common, have a better prognosis, and can be further classified by the Seddon/Sunderland classification (Table 1). It is important to understand that spontaneous recovery can occur following neurapraxia or axonotmesis but will not occur following neurotmesis or avulsion.

Table 1 Seddon/Sunderland classification

Type I (neuropraxia), temporary paralysis due to stretch or traction injury of peripheral nerve and loss of conduction, local damage to the myelin sheath with intact axons, and no loss of structural continuity
Type II (axonotmesis), peripheral nerve injury with damage to the axons and their myelin sheath and peripheral nerve sheath intact; axons can spontaneously recover at rate of 1–2 mm per day
Type III–V (neurotmesis), complete peripheral nerve rupture with neuroma formation, preservation of perineurium in type III, preservation of epineurium in type IV, and complete transection in type V; will not spontaneously recover but nerve repair or reconstruction are possible

Source: [10]

Natural history

There is significant variability in the natural history of BPBP. Of those diagnosed, 66–92 % of infants will have experience spontaneous recovery of elbow flexion against gravity in the first 2 months of life, which strongly suggests eventual complete neurologic recovery in the remainder of the upper limb [5, 11, 12]. However, 10–30 % of patients are left with permanent upper extremity weakness or alteration in function [1, 5]. The Narakas classification system stratifies prognosis based on the extent of nerve root involvement [13]. Narakas type I injuries involve C5 and C6 (also known as an Erb's palsy) and represent 46 % of patients. Approximately 80 % of these patients make a full spontaneous recovery. Narakas type II injuries involve C5–C7 (extended Erb's palsy) and represent 30 % of patients. Only 60 % of patients in this group make a full spontaneous recovery. Narakas type III injuries represent a total plexopathy (flail extremity). Narakas type IV injuries are the most severe and present with a flail extremity and Horner's syndrome. Narakas type III and IV injuries occur 20 % of the time.

Due to the variability in presentation, multiple investigations have attempted to better prognosticate the potential milestones for neurologic recovery. At 2 months, return of partial antigravity elbow flexion suggests that the patient will have a full neurologic recovery [13]. However, Waters demonstrated that if biceps function has not returned by 3 months, the patient will rarely have complete recovery and will likely have decreased shoulder strength and range of motion [14]. Phrenic nerve injury (resulting in paralysis of the hemidiaphragm) and Horner's syndrome are individually considered prognostic for poor motor recovery and are associated with nerve root avulsion [15, 16]. Paralysis of the hemidiaphragm can be diagnosed with plain radiography or ultrasonography. Horner's syndrome is evident on initial examination and characterized ptosis, miosis, and anhidrosis. Functionally, patients with permanent neurologic injury have been found to have slightly smaller dimensions of the affected limb, along with delayed skeletal maturation [17, 18]. These children can expect the affected limb to be approximately 95 % the length and girth of the contralateral limb [19].

Multiple investigations have sought to characterize the sequelae of permanent neurologic injury, particularly in the shoulder. Mallet noted that global shoulder function worsens with increasing delay in return of biceps function [20]. Incomplete neurologic recovery leads to muscle imbalance (greater shoulder internal rotation from intact pectoralis major and subscapularis motor function, compared to external rotation, affected by impaired supraspinatus and infraspinatus motor function), internal rotation contracture, and glenohumeral

dysplasia—three sequelae that are intimately related and have been the subject of significant attention in recent years.

Glenohumeral dysplasia (and posterior shoulder dislocation) is associated with BPBP and may occur as early as 3 months of age [21•]. Approximately 60–80 % of children who do not recover full motor function develop some degree of glenohumeral deformity [22]. This typically presents as retroversion of the glenoid, flattening of the humeral head, and posterior subluxation. Posterior displacement of the humeral head center of rotation beyond 50 % leads to a high likelihood of developing a pseudoglenoid [23]. Eismann has noted that a majority of these patients also have glenohumeral abduction deformities, with contractures as severe as 65°, a deformity that may actually improve global shoulder function by positioning the glenohumeral joint in abduction [24].

The majority of patients with glenohumeral dysplasia develop concomitant internal rotation contracture [25]. Many of these patients demonstrate difficulty in bringing their hand to mouth or reaching the back of their head and neck (Fig. 2a, b). Multiple studies have attributed contractures and bony deformity to muscle imbalance due to weak extensors and external rotators [26–28]. However, recent murine and computational studies have found that impaired longitudinal muscle growth results in more pronounced osseous deformity than muscle imbalance [29, 30].

Like internal rotation contractures of the glenohumeral joint, elbow flexion contractures in BPBP have received significant attention. The incidence of significant elbow flexion contractures in BPBP may be greater than clinicians perceive, and prevalence increases with patient age [31]. Nikolaou has shown that functional shortening of denervated muscle plays a primary role in contracture pathogenesis [32]. However, the mechanism of contracture development in denervated muscle remains unanswered [33, 34].

Distally, decreased forearm supination and wrist deformity can occur. In patients with Narakas type III and IV injuries, significant functional impairment of the hand often persists, even in children who have regained good shoulder function [35].

Evaluation

Physical examination is the primary diagnostic tool for BPBP. Inspection will reveal a lack of spontaneous upper extremity movement (Fig. 1). By careful evaluation of each major muscle group in the upper limb, the clinician can determine the pattern of nerve root involvement. The clinician should also examine for ipsilateral ptosis or miosis in order to establish the presence or absence of a Horner's syndrome. Serial examination of the child allows the clinician to assess any ongoing motor recovery. There are multiple examination scoring systems available to track recovery and function. The most commonly used and validated are the Modified Mallet Score, the Active Movement Score (AMS) system, and the Toronto Score Test (Table 2). The Modified Mallet Score assesses for ability to perform activities of daily living with functional movements, such as global shoulder abduction, global shoulder external/internal rotation, hand to neck, hand to spine, and hand to mouth. AMS identifies 15 spontaneous movements of the affected limb and scores them based on active movement without gravity and against gravity. The Toronto Score Test attempts to prognosticate neurologic recovery by scoring the following five movements: elbow flexion, elbow extension, wrist extension, digital extension, and thumb extension. No scoring system is comprehensive, and they are difficult to compare. For example, Modified Mallet Scores do not correlate well with AMS or Toronto Scores due to its focus on shoulder motion [36].

The most important single prognostic sign is recovery of antigravity elbow flexion. Clinical examination can help distinguish BPBP from radial nerve palsy in a newborn. Neonatal radial nerve palsy should be suspected in infants presenting with absent wrist and digital extension but intact deltoid, biceps, and triceps function. The presence of ecchymosis and/or fat necrosis along the posterolateral brachium is pathognomonic for neonatal radial nerve palsy and is less commonly seen in BPBP.

While physical exam is the cornerstone of the diagnostic workup in BPBP, imaging can be used to augment the workup. Radiographs should be considered to evaluate for clavicle and humerus fractures, particularly in infants with a history of shoulder dystocia [9]. Fracture can result in pseudoparalysis of the ipsilateral limb, important in the differential diagnosis of BPBP. The role of electromyography (EMG) for prognosticating neurologic recovery is controversial, and its use in clinical practice remains limited. Heise found that EMG overestimated clinical recovery [37]. However, more recently, Van Dijk found that the absence of biceps motor unit potentials in early EMG at 1 month accurately predicted elbow flexion paralysis, although EMG of biceps at 3 months again overestimated recovery [38]. Computed tomography (CT) myelography and magnetic resonance imaging (MRI) can help characterize injury patterns and can distinguish between pre-ganglionic and post-ganglionic injury. Both modalities are highly correlated (no advantage to using both), although MRI eliminates the radiation exposure [39]. Currently, BPBP remains a clinical diagnosis based on exam. There are no strong indications for routine advanced imaging. It should be reserved as an adjunct tool for unusual cases rather than a screening tool for all infants, particularly due to sedation requirements in infants. However, MRI of the shoulder may be useful in toddlers and older children to assess glenohumeral dysplasia. MRI permits clear visualization of the shape of the glenohumeral joint and can therefore distinguish candidates for shoulder tendon transfers (mild glenohumeral dysplasia)

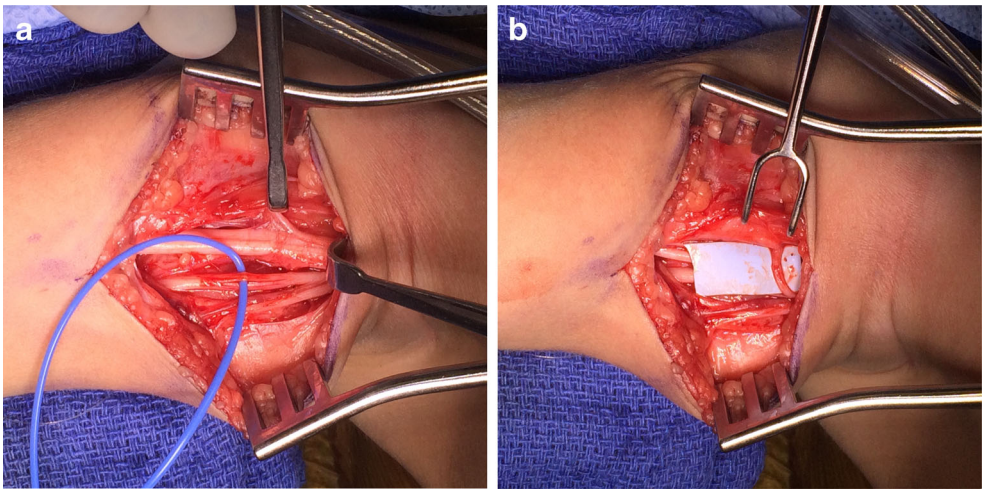

Fig. 2 Intra-operative photographs demonstrating an Oberlin's transfer (single fascicle ulnar nerve transfer to motor branch of the biceps brachii). In **a**, the principal extrinsic fascicle of the ulnar nerve (marked with the vessel loop) has been identified following epineurotomy, microsurgical dissection, and interoperative nerve stimulation. This fascicle is transferred to the motor branch of the biceps brachii (**b**)

versus humeral osteotomy (moderate or severe glenohumeral dysplasia) [40]. Ultrasound can be used to detect infantile shoulder dislocation [41]. An ultrasound of the shoulder is recommended in all infants with incomplete recovery at 6 months and particularly in infants with progressive loss of shoulder external rotation with the arm at the side as this may indicate glenohumeral subluxation or dislocation.

Management

Treatment of BPBP begins with physical or occupational therapy. Passive shoulder, elbow, and digital range of motion therapy should begin immediately unless the patient is also diagnosed with a clavicle or humerus fracture, in which case, therapy should begin at 3–4 weeks [42]. Frequent stretching of the shoulder is critical during infancy in order to prevent glenohumeral dysplasia or infantile shoulder dislocation. Shoulder and elbow strengthening through guided play is also important for maximizing functional neurologic recovery. During this period of time, serial examination by an orthopedic surgeon is critical in order to detect infants at risk of shoulder dislocation.

Microsurgical indications

Microsurgical reconstruction of the brachial plexus is recommended for infants that do not demonstrate sufficient spontaneous neurologic recovery. It is generally accepted that surgical intervention is contraindicated in patients who recover antigravity biceps function by 3 months. However, clinicians continue to debate how to accurately identify the population of patients who will have a poor outcome with continued conservative management. Although strong evidence-based indications for microsurgery are lacking, there is general consensus that microsurgery is indicated at 3 months of age for infants with Narakas type III and IV lesions (global lesions with or without Horner's syndrome). Haerle recommends surgical intervention in Narakas type IV patients if they show no recovery of forearm and hand function at 3 months even if biceps function is present [43]. However, indications for microsurgery and timing of surgery for patients with Narakas type I and type II lesions (upper trunk or combined upper and middle trunk lesions) continue to be debated.

Many authors have focused on biceps function to guide decision-making. Absence of return of antigravity biceps function (AMS score ≥5) from 3 to 9 months has been cited as indication for microsurgery [14, 44–46]. There is no

Table 2 Scoring systems for recovery and function

	Pros	Cons
Modified Mallet	Incorporates shoulder abduction and external/internal rotation	Not suitable for infants, requires cooperation and communication
AMS	All ages	Increased time requirements
	Most extensively validated	Complex grading guidelines
Toronto	Prognostic value	Oversimplification
	Simple grading system	

Source: [36]

consensus on optimal timing. Chuang asserts that the absence of biceps function with little or no hand function is an indication for early exploration at 3 months; improvements in hand function have been poor with delayed reconstruction [47]. Finally, a recent study by Argenta argued that nerve grafting is indicated from 9 to 12 months of age if there is weakness in shoulder function or forearm supination even in patients with near-normal AMS scores for elbow flexion [48].

Microsurgical techniques

There are three types of microsurgical techniques that can be used in BPBP, neurolysis, nerve grafting, and nerve transfers. It was traditionally accepted that neurolysis alone was not sufficient in regaining neurologic function [49]. However, neurolysis alone has been revisited and may be indicated in infants who show signs of recovery intra-operatively with nerve stimulation or in a subgroup of patients with >50 % nerve conduction pre-operatively [12, 50]. Superior results with neuroma resection and nerve grafting compared with neurolysis have been consistently demonstrated in the literature. Nerve grafting is typically performed with autograft (most commonly the sural nerve) but can alternatively be performed with synthetic nerve conduits or allograft. Nerve grafts are designed to bypass the zone of injury by permitting axonal regrowth from the nerve root level to distal targets (typically, divisions or cords of the brachial plexus). Nerve grafting can result in improved motor function and sensibility. Although difficult to compare due to variable indications and scoring systems, studies have consistently demonstrated significant functional gains in elbow flexion in patients who undergo surgery between 3 and 9 months [14, 51, 52].

Nerve transfers have gained popularity over the past decade. Unlike nerve grafting, nerve transfer builds a direct motor-to-motor neural connection close to the muscle target. Nerve transfers have been found to be effective for late presentation, isolated deficits, failed primary reconstruction, and multiple nerve root avulsions [53•]. The following three nerve transfers are among the most popular and can be performed in conjunction with one another to augment shoulder abduction and external rotation as well as elbow flexion. Transfer of the terminal motor branch of the spinal accessory nerve (cranial nerve XI) to the suprascapular nerve reliably restores some infraspinatus and supraspinatus function [54, 55]. Transferring the long head of the triceps motor branch of the radial nerve to the anterior branch of the axillary nerve can restore deltoid function [56]. Finally, transfer of a single extrinsic fascicle of the ulnar nerve (primarily supplying the flexor carpi ulnaris) to the motor branch of the biceps (Fig. 2a, b)—better known as Oberlin's transfer—improves elbow flexion [57]. As with nerve-grafting outcomes, it is difficult to compare existing studies. Studies comparing spinal accessory to suprascapular nerve transfer versus C5 nerve grafting show equivalent results [58, 59]. Of the patients, 65–90 % demonstrate functional recovery with the Oberlin transfer [60–62]. There is insignificant data to assess isolated outcomes of triceps branch to axillary nerve transfer in children, as it is more commonly done in conjunction with other nerve transfers [63]. Other nerve transfers have also been developed, but are less commonly used, including intercostal nerve transfer and contralateral C7 nerve root transfer [64–67].

There is a dearth of significant data directly comparing nerve grafting to nerve transfer for primary reconstruction in BPBP. A recent retrospective case series by Seruya evaluated outcomes in 74 patients with BPBP undergoing suprascapular nerve reconstruction—46 patients with spinal accessory nerve transfer and 28 patients with nerve autograft reconstruction. There was a trend towards diminished shoulder abduction and external rotation with a twofold increase in secondary shoulder surgery with nerve grafting, when compared to nerve transfer [68]. In cases of BPBP involving the C7-T1 nerve roots, Tse maintains in a recent position paper that nerve transfers alone are inadequate to address all functional deficits and, notably, will not improve sensibility [53•]. Both nerve grafting and nerve transfers are acceptable options for surgical reconstruction in BPBP. Surgeon preference continues to dictate whether nerve grafting, nerve transfer, or both will be used.

Delayed secondary procedures

Beyond reinnervation strategies in infants, there are a number of secondary procedures that have been developed for toddlers or children with persistent functional deficits. Limited shoulder function in children with plateauing of neural recovery could be related to persistent internal rotation contracture, progressive glenohumeral deformity, infantile dislocation, and insufficient abduction and external rotation power. These concerns have resulted in the development of a number of intra-articular and extra-articular secondary procedures for the shoulder [22]. These correct internal rotation contractures, improve shoulder function, and limit progression of glenohumeral dysplasia.

The predominant intra-articular procedures include open and arthroscopic anterior joint capsule release. A recent meta-analysis of functional status in 405 patients demonstrated greater improvement in shoulder abduction with an open technique in comparison to an arthroscopic technique (67 versus 28 %). There was no difference between open and arthroscopic techniques for improvement in external rotation, or aggregate Mallet scores was seen [69].

Musculotendinous extra-articular procedures include tendon lengthening and tendon transfers. Historically, subscapularis and pectoralis major lengthening was the workhorse procedure, although recent studies demonstrate functional improvements in Mallet Score at 1 year but not at

long-term follow-up [70]. Lengthening can be performed in conjunction with tendon transfers. The most popular procedure is the L'Episcopo procedure, a transfer of the teres major and latissiumus dorsi to the rotator cuff. Noaman demonstrated an average gain of 85° in shoulder external rotation and 75° in shoulder abduction in 76 patients [71]. Although tendon transfers reliably improve shoulder range of motion, without concurrent intra-articular procedures, correction of glenohumeral dysplasia will not occur. Waters and Bae demonstrated improvement in glenohumeral dysplasia in 83 % of patients with combined intra-articular and extra-articular procedures, confirmed with post-operative MRI [72].

In children with severe glenohumeral dysplasia, humeral derotational osteotomy can be used to reposition the arc of shoulder motion and improve function. According to Price, if a patient is less than 4 years old, there is greater potential for the glenohumeral joint to remodel, so anterior contracture release and tendon transfers are generally indicated. However if a child is greater than 3–4 years old, MRI is indicated to assess joint congruity and dysplasia, and the ability of a child to reach the mouth and back of the head should be assessed (Fig. 3c, d). If the joint is incongruent and the child has difficulty reaching the mouth without moderate to severe trumpeting and is unable to reach the back of the head, a humeral external rotation osteotomy is often indicated instead of tendon transfers and soft tissue release [73].

In addition to secondary procedures of the shoulder, a number of secondary procedures of the elbow and wrist have been developed to correct contractures and improve function. The Steindler flexorplasty allows for improvement in elbow flexion by detaching the medial epicondyle and the flexor-pronator origin and transposing them proximally on humerus. This has demonstrated improved

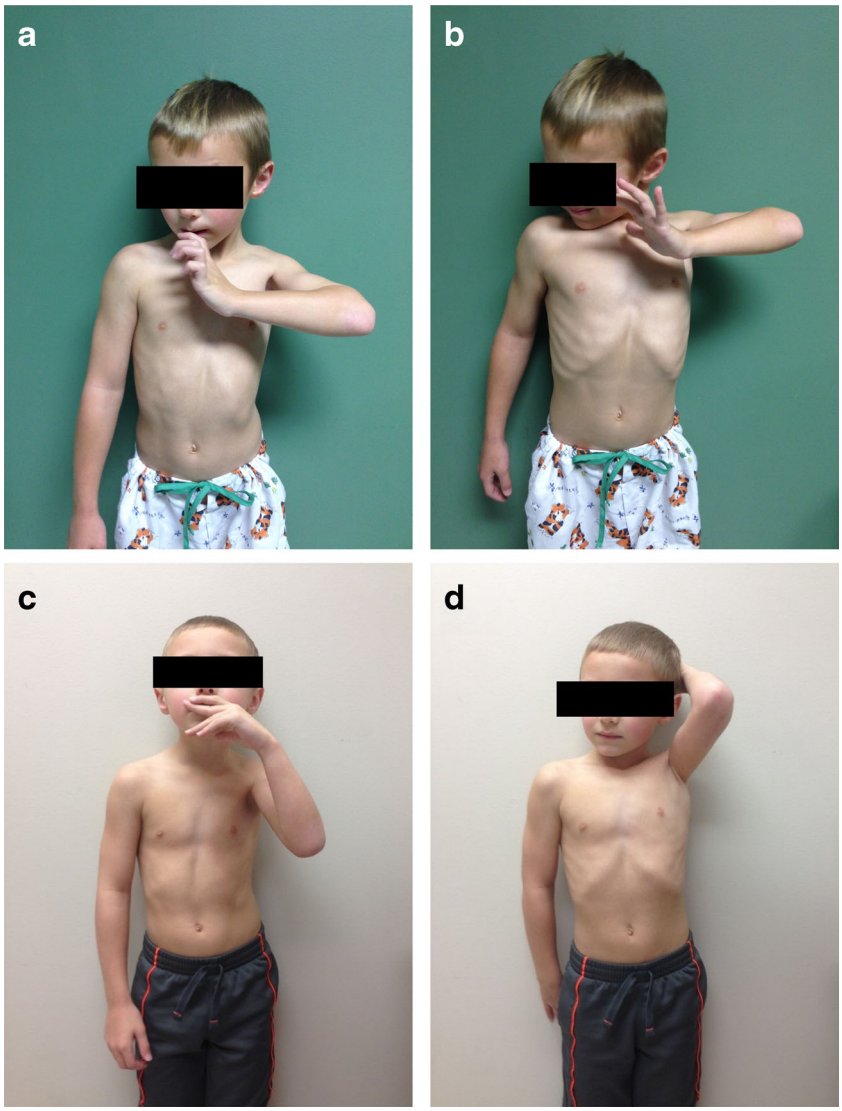

Fig. 3 **a**, **b** The natural history of shoulder muscle imbalance and incomplete neurologic recovery in brachial plexus birth palsy. This 10-year-old child lacks external rotation beyond neutral and is only able to bring the affected hand to his mouth with significant trumpeting (shoulder abduction) and wrist extension. The child is unable to reach the back of his head. **c**, **d** Shoulder functioning 2 months following an external rotation humeral osteotomy. The child is now able to reach his mouth with mild trumpeting and without wrist extension. The child is also able to reach the back of his, although this does require continued cervical spine rotation

flexion power and range of motion, increasing from an average of 14 to 97° flexion at minimum of 2 years [74].

Recent studies have evaluated the use botulinum toxin as an adjunct to both non-surgical and surgical treatments of BPBP. In particular, it has been used following closed reduction and casting of the shoulder to facilitate external rotation and prevent glenohumeral dysplasia and need for reconstructive shoulder surgery [7576•]. Additionally, botulinum toxin has been used to prevent post-operative contractures [77]. The efficacy of botulinum toxin remains unclear. A recent study by Arad looked at the use of botulinum toxin for elbow and shoulder contractures in BPBP with follow-up at 1 month and 1 year. Elbow flexion strength demonstrated with sustained improvement at 1 year, but initial improvements of shoulder external rotation strength were not sustained at 1 year [78]. More comparative studies are needed to better elucidate the potential benefit of adjunctive botulinum toxin.

Outcomes

Prognosticating functional recovery in BPBP remains difficult, and outcomes reflect this challenge. Zuckerman recently compared two groups of infants with Narakas grade 1 BPBP between the ages of 5 and 9 months. One group underwent nerve transfers, while the other progressed past the point of needing intervention. Both demonstrated similar long-term function at 2 years [79]. Despite this challenge, microsurgical procedures have demonstrated reliable return of shoulder function in 60–80 % of cases and return of elbow flexion against gravity in 80–100 % of cases [11, 14, 42, 80, 81]. When evaluating long-term outcomes, the importance of functional recovery cannot be overstated. Nearly 90 % of children with BPBP participate in sports, which is similar to the published normative pediatric data [82]. Finally, in a study surveying quality of life in adolescents with BPBP, functional and aesthetic factors were responsible for the majority of perceived differences in quality of life [83].

Future directions

Timing of microsurgery in Erb's palsy at 3 versus 6 months continues to be a controversial debate and will likely remain a topic with significant research attention until a reliable consensus can be reached. Additionally, the value of botulinum toxin and external rotation casting to prevent shoulder internal rotation contractures is an area of great clinical interest, as efficacy remains unclear. Finally, as our understanding of the pathophysiology of muscle contracture improves, future translational research should hopefully minimize the downstream effects of muscle imbalance, fibrosis, and impaired longitudinal growth.

Conclusions

BPBP can result in permanent neurologic injury, a devastating outcome for parents and patients, which continues to spur interest in optimizing the workup and management of a problem and with high variable natural history. BPBP is best diagnosed on clinical examination, and exam findings are also used to determine candidates for microsurgical reconstruction of the brachial plexus. The key exam finding is return of antigravity elbow flexion. Both microsurgery and secondary shoulder and elbow procedures are effective and can substantially improve function.

Compliance with ethical standards

Conflict of interest Kristin L. Buterbaugh and Apurva S. Shah declare that they have no conflict of interest.

Human and animal rights and informed consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance

1. Hoeksma AF, ter Steeg AM, Nelissen RG, van Ouwerkerk WJ, Lankhorst GJ, de Jong BA. Neurological recovery in obstetric brachial plexus injuries: an historical cohort study. Dev Med Child Neurol. 2004;46:76–83.
2. Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. Am J Obstet Gynecol. 2005;192:1795–800. discussion 1800–1792.
3. Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the United States. J Bone Joint Surg Am. 2008;90:1258–64.
4. National Vital Statistics Report 2012 Vol. 61, Centers for Disease Control and Prevention (CDC).
5. Pondaag W, Malessy MJ, van Dijk JG, Thomeer RT. Natural history of obstetric brachial plexus palsy: a systematic review. Dev Med Child Neurol. 2004;46:138–44.
6. Waters PM. Obstetric brachial plexus injuries: evaluation and management. J Am Acad Orthop Surg. 1997;5:205–14.
7. van Ouwerkerk WJ, van der Sluijs JA, Nollet F, Barkhof F, Slooff AC. Management of obstetric brachial plexus lesions: state of the art and future developments. Childs Nerv Syst. 2000;16:638–44.
8. Sibiński M, Synder M. Obstetric brachial plexus palsy—risk factors and predictors. Ortop Traumatol Rehabil. 2007;9:569–76.
9. Abid A. Brachial plexus birth palsy: management during the first year of life. Orthop Traumatol Surg Res. 2016.
10. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain. 1951;74:491–516.

11. Laurent JP, Lee R, Shenaq S, Parke JT, Solis IS, Kowalik L. Neurosurgical correction of upper brachial plexus birth injuries. J Neurosurg. 1993;79:197–203.
12. Greenwald AG, Schute PC, Shiveley JL. Brachial plexus birth palsy: a 10-year report on the incidence and prognosis. J Pediatr Orthop. 1984;4:689–92.
13. Narakas AO. Injuries of the brachial plexus and neighboring peripheral nerves in vertebral fractures and other trauma of the cervical spine. Orthopade. 1987;16:81–6.
14. Waters PM. Comparison of the natural history, the outcome of microsurgical repair, and the outcome of operative reconstruction in brachial plexus birth palsy. J Bone Joint Surg Am. 1999;81:649–59.
15. Yoshida K, Kawabata H. The prognostic value of concurrent phrenic nerve palsy in newborn babies with neonatal brachial plexus palsy. J Hand Surg [Am]. 2015;40:1166–9.
16. El-Sayed AA. The prognostic value of concurrent Horner syndrome in extended Erb obstetric brachial plexus palsy. J Child Neurol. 2014;29:1356–9.
17. Gosk J, Wnukiewicz W, Urban M. The effect of perinatal brachial plexus lesion on upper limb development. BMC Musculoskelet Disord. 2014;15:116.
18. Oktay F, Cömert D, Gökkaya NK, Ozbudak SD, Uysal H. Bone age in children with obstetrical brachial plexus palsy: effect of peripheral nerve injury on skeletal maturation. J Child Neurol. 2014;29:203–9.
19. Bae DS, Ferretti M, Waters PM. Upper extremity size differences in brachial plexus birth palsy. Hand (N Y). 2008;3:297–303.
20. Mallet J. Obstetrical paralysis of the brachial plexus. II. Therapeutics. Treatment of sequelae. Priority for the treatment of the shoulder. Method for the expression of results. Rev Chir Orthop Reparatrice Appar Mot. 1972;58 Suppl 1:166–8.
21.• Iorio ML, Menashe SJ, Iyer RS, et al. Glenohumeral dysplasia following neonatal brachial plexus palsy: presentation and predictive features during infancy. J Hand Surg [Am]. 2015;40:2345–51.e2341. **Comprehensive detailed description of the natural history of glenohumeral dysplasia, its effect on functional outcomes and the importance of screening.**
22. Hale HB, Bae DS, Waters PM. Current concepts in the management of brachial plexus birth palsy. J Hand Surg [Am]. 2010;35:322–31.
23. Pearl ML, Woolwine S, van de Bunt F, Merton G, Burchette R. Geometry of the proximal humeral articular surface in young children: a study to define normal and analyze the dysplasia due to brachial plexus birth palsy. J Shoulder Elb Surg. 2013;22:1274–84.
24. Eismann EA, Little KJ, Laor T, Cornwall R. Glenohumeral abduction contracture in children with unresolved neonatal brachial plexus palsy. J Bone Joint Surg Am. 2015;97:112–8.
25. Aly A, Bahm J, Schuind F. Percutaneous humeral derotational osteotomy in obstetrical brachial plexus palsy: a new technique. J Hand Surg Eur Vol. 2014;39:549–52.
26. Brochard S, Alter K, Damiano D. Shoulder strength profiles in children with and without brachial plexus palsy. Muscle Nerve. 2014;50:60–6.
27. Soldado F, Fontecha CG, Marotta M, et al. The role of muscle imbalance in the pathogenesis of shoulder contracture after neonatal brachial plexus palsy: a study in a rat model. J Shoulder Elb Surg. 2014;23:1003–9.
28. Crouch DL, Plate JF, Li Z, Saul KR. Computational sensitivity analysis to identify muscles that can mechanically contribute to shoulder deformity following brachial plexus birth palsy. J Hand Surg [Am]. 2014;39:303–11.
29. Crouch DL, Hutchinson ID, Plate JF, et al. Biomechanical basis of shoulder osseous deformity and contracture in a rat model of brachial plexus birth palsy. J Bone Joint Surg Am. 2015;97:1264–71.
30. Cheng W, Cornwall R, Crouch DL, Li Z, Saul KR. Contributions of muscle imbalance and impaired growth to postural and osseous shoulder deformity following brachial plexus birth palsy: a computational simulation analysis. J Hand Surg [Am]. 2015;40:1170–6.
31. Sheffler LC, Lattanza L, Hagar Y, Bagley A, James MA. The prevalence, rate of progression, and treatment of elbow flexion contracture in children with brachial plexus birth palsy. J Bone Joint Surg Am. 2012;94:403–9.
32. Nikolaou S, Peterson E, Kim A, Wylie C, Cornwall R. Impaired growth of denervated muscle contributes to contracture formation following neonatal brachial plexus injury. J Bone Joint Surg Am. 2011;93:461–70.
33. Nikolaou S, Liangjun H, Tuttle LJ, et al. Contribution of denervated muscle to contractures after neonatal brachial plexus injury: not just muscle fibrosis. Muscle Nerve. 2014;49:398–404.
34. Weekley H, Nikolaou S, Hu L, Eismann E, Wylie C, Cornwall R. The effects of denervation, reinnervation, and muscle imbalance on functional muscle length and elbow flexion contracture following neonatal brachial plexus injury. J Orthop Res. 2012;30:1335–42.
35. Immerman I, Alfonso DT, Ramos LE, et al. Hand function in children with an upper brachial plexus birth injury: results of the nine-hole peg test. Dev Med Child Neurol. 2012;54:166–9.
36. Greenhill DA, Lukavsky R, Tomlinson-Hansen S, Kozin SH, Zlotolow DA. Relationships between 3 classification systems in brachial plexus birth palsy. J Pediatr Orthop. 2015.
37. Heise CO, Siqueira MG, Martins RS, Gherpelli JL. Clinical-electromyography correlation in infants with obstetric brachial plexopathy. J Hand Surg [Am]. 2007;32:999–1004.
38. Van Dijk JG, Pondaag W, Buitenhuis SM, Van Zwet EW, Malessy MJ. Needle electromyography at 1 month predicts paralysis of elbow flexion at 3 months in obstetric brachial plexus lesions. Dev Med Child Neurol. 2012;54:753–8.
39. Tse R, Nixon JN, Iyer RS, Kuhlman-Wood KA, Ishak GE. The diagnostic value of CT myelography, MR myelography, and both in neonatal brachial plexus palsy. AJNR Am J Neuroradiol. 2014;35:1425–32.
40. Waters PM, Smith GR, Jaramillo D. Glenohumeral deformity secondary to brachial plexus birth palsy. J Bone Joint Surg Am. 1998;80:668–77.
41. Somashekar DK, Di Pietro MA, Joseph JR, Yang LJ, Parmar HA. Utility of ultrasound in noninvasive preoperative workup of neonatal brachial plexus palsy. Pediatr Radiol. 2015.
42. Abzug JM, Kozin SH. Evaluation and management of brachial plexus birth palsy. Orthop Clin North Am. 2014;45:225–32.
43. Haerle M, Gilbert A. Management of complete obstetric brachial plexus lesions. J Pediatr Orthop. 2004;24:194–200.
44. Gilbert A, Tassin JL. Surgical repair of the brachial plexus in obstetric paralysis. Chirurgie. 1984;110:70–5.
45. Bain JR, Dematteo C, Gjertsen D, Hollenberg RD. Navigating the gray zone: a guideline for surgical decision making in obstetrical brachial plexus injuries. J Neurosurg Pediatr. 2009;3:173–80.
46. Clarke HM, Curtis CG. An approach to obstetrical brachial plexus injuries. Hand Clin. 1995;11:563–80. discussion 580–561.
47. Chuang DC, Mardini S, Ma HS. Surgical strategy for infant obstetrical brachial plexus palsy: experiences at Chang Gung Memorial Hospital. Plast Reconstr Surg. 2005;116:132–42. discussion 143–134.
48. Argenta AE, Brooker J, MacIssac Z, et al. Obstetrical brachial plexus palsy: can excision of upper trunk neuroma and nerve grafting improve function in babies with adequate elbow flexion at nine months of age? J Plast Reconstr Aesthet Surg. 2016.
49. Clarke HM, Al-Qattan MM, Curtis CG, Zuker RM. Obstetrical brachial plexus palsy: results following neurolysis of conducting neuromas-in-continuity. Plast Reconstr Surg. 1996;97:974–82. Discussion 983–974.
50. Chin KF, Misra VP, Sicuri GM, Fox M, Sinisi M. Intra-operative neurophysiological prediction of upper trunk recovery in obstetric

brachial plexus palsy with neuroma in continuity. Bone Joint J. 2013;95-B:699–705.
51. Lin JC, Schwentker-Colizza A, Curtis CG, Clarke HM. Final results of grafting versus neurolysis in obstetrical brachial plexus palsy. Plast Reconstr Surg. 2009;123:939–48.
52. El-Gammal TA, El-Sayed A, Kotb MM, et al. Total obstetric brachial plexus palsy: results and strategy of microsurgical reconstruction. Microsurgery. 2010;30:169–78.
53.• Tse R, Kozin SH, Malessy MJ, Clarke HM. International Federation of Societies for Surgery of the Hand Committee report: the role of nerve transfers in the treatment of neonatal brachial plexus palsy. J Hand Surg [Am]. 2015;40:1246–59. **Editorial that elaborates on the current controversy on the role of nerve transfers versus nerve grafting.**
54. Terzis JK, Kostas I. Outcomes with suprascapular nerve reconstruction in obstetrical brachial plexus patients. Plast Reconstr Surg. 2008;121:1267–78.
55. Midha R. Nerve transfers for severe brachial plexus injuries: a review. Neurosurg Focus. 2004;16:E5.
56. Bertelli JA, Ghizoni MF. Reconstruction of C5 and C6 brachial plexus avulsion injury by multiple nerve transfers: spinal accessory to suprascapular, ulnar fascicles to biceps branch, and triceps long or lateral head branch to axillary nerve. J Hand Surg [Am]. 2004;29:131–9.
57. Oberlin C, Béal D, Leechavengvongs S, Salon A, Dauge MC, Sarcy JJ. Nerve transfer to biceps muscle using a part of ulnar nerve for C5-C6 avulsion of the brachial plexus: anatomical study and report of four cases. J Hand Surg [Am]. 1994;19:232–7.
58. Tse R, Marcus JR, Curtis CG, Dupuis A, Clarke HM. Suprascapular nerve reconstruction in obstetrical brachial plexus palsy: spinal accessory nerve transfer versus C5 root grafting. Plast Reconstr Surg. 2011;127:2391–6.
59. Pondaag W, de Boer R, van Wijlen-Hempel MS, Hofstede-Buitenhuis SM, Malessy MJ. External rotation as a result of suprascapular nerve neurotization in obstetric brachial plexus lesions. Neurosurgery. 2005;57:530–7. discussion 530–537.
60. Siqueira MG, Socolovsky M, Heise CO, Martins RS, Di Masi G. Efficacy and safety of Oberlin's procedure in the treatment of brachial plexus birth palsy. Neurosurgery. 2012;71:1156–60. Discussion 1161.
61. Little KJ, Zlotolow DA, Soldado F, Cornwall R, Kozin SH. Early functional recovery of elbow flexion and supination following median and/or ulnar nerve fascicle transfer in upper neonatal brachial plexus palsy. J Bone Joint Surg Am. 2014;96:215–21.
62. Al-Qattan MM, Al-Kharfy TM. Median nerve to biceps nerve transfer to restore elbow flexion in obstetric brachial plexus palsy. Biomed Res Int. 2014;2014:854084.
63. McRae MC, Borschel GH. Transfer of triceps motor branches of the radial nerve to the axillary nerve with or without other nerve transfers provides antigravity shoulder abduction in pediatric brachial plexus injury. Hand (N Y). 2012;7:186–90.
64. El-Gammal TA, Abdel-Latif MM, Kotb MM, et al. Intercostal nerve transfer in infants with obstetric brachial plexus palsy. Microsurgery. 2008;28:499–504.
65. Gu YD. Contralateral C7 root transfer over the last 20 years in China. Chin Med J. 2007;120:1123–6.
66. Carlstedt T, Grane P, Hallin RG, Norén G. Return of function after spinal cord implantation of avulsed spinal nerve roots. Lancet. 1995;346:1323–5.
67. Amr SM, Essam AM, Abdel-Meguid AM, Kholeif AM, Moharram AN, El-Sadek RE. Direct cord implantation in brachial plexus avulsions: revised technique using a single stage combined anterior (first) posterior (second) approach and end-to-side side-to-side grafting neurorrhaphy. J Brachial Plex Peripher Nerve Inj. 2009;4:8.
68. Seruya M, Shen SH, Fuzzard S, Coombs CJ, McCombe DB, Johnstone BR. Spinal accessory nerve transfer outperforms cervical root grafting for suprascapular nerve reconstruction in neonatal brachial plexus palsy. Plast Reconstr Surg. 2015;135:1431–8.
69. Louden EJ, Broering CA, Mehlman CT, Lippert WC, Pratt J, King EC. Meta-analysis of function after secondary shoulder surgery in neonatal brachial plexus palsy. J Pediatr Orthop. 2013;33:656–63.
70. Kruit AS, Choukairi F, Mishra A, Gaffey A, Jester A. Subscapularis Z-lengthening in children with brachial plexus birth palsy loses efficiency at mid-term follow-up: a retrospective cohort study. Int Orthop. 2015.
71. Noaman HH. Anterior shoulder release and tendon transfer as 1-stage procedure for treatment of internal rotation contracture deformity in obstetric brachial plexus injuries. Ann Plast Surg. 2013;71:510–8.
72. Waters PM, Bae DS. The early effects of tendon transfers and open capsulorrhaphy on glenohumeral deformity in brachial plexus birth palsy. Surgical technique. J Bone Joint Surg Am. 2009;91(2):213–22.
73. Bae DS, Waters PM. External rotation humeral osteotomy for brachial plexus birth palsy. Tech Hand Up Extrem Surg. 2007;11:8–14.
74. Gilbert A, Valbuena S, Posso C. Obstetrical brachial plexus injuries: late functional results of the Steindler procedure. J Hand Surg Eur Vol. 2014;39:868–75.
75. Ezaki M, Malungpaishrope K, Harrison RJ, et al. OnabotulinumtoxinA injection as an adjunct in the treatment of posterior shoulder subluxation in neonatal brachial plexus palsy. J Bone Joint Surg Am. 2010;92:2171–7.
76.• Greenhill DA, Wissinger K, Trionfo A, Solarz M, Kozin SH, Zlotolow DA. External rotation predicts outcomes after closed glenohumeral joint reduction with botulinum toxin type A in brachial plexus birth palsy. J Pediatr Orthop. 2016. **Describes the current use and limitations of botulinum toxin for glenohumeral dysplasia and internal rotation contracture. Highlights the use of botulinum toxin as an adjunct to closed reduction of the glenohumeral joint in order to optimize development.**
77. Price AE, Ditaranto P, Yaylali I, Tidwell MA, Grossman JA. Botulinum toxin type A as an adjunct to the surgical treatment of the medial rotation deformity of the shoulder in birth injuries of the brachial plexus. J Bone Joint Surg (Br). 2007;89:327–9.
78. Arad E, Stephens D, Curtis CG, Clarke HM. Botulinum toxin for the treatment of motor imbalance in obstetrical brachial plexus palsy. Plast Reconstr Surg. 2013;131:1307–15.
79. Zuckerman SL, Allen LA, Broome C, et al. Functional outcomes of infants with Narakas grade 1 birth-related brachial plexus palsy undergoing neurotization compared with infants who did not require surgery. Childs Nerv Syst. 2016.
80. Gilbert A. Long-term evaluation of brachial plexus surgery in obstetrical palsy. Hand Clin. 1995;11:583–94. Discussion 594–585.
81. Hentz VR, Meyer RD. Brachial plexus microsurgery in children. Microsurgery. 1991;12:175–85.
82. Bae DS, Zurakowski D, Avallone N, Yu R, Waters PM. Sports participation in selected children with brachial plexus birth palsy. J Pediatr Orthop. 2009;29:496–503.
83. Squitieri L, Larson BP, Chang KW, Yang LJ, Chung KC. Understanding quality of life and patient expectations among adolescents with neonatal brachial plexus palsy: a qualitative and quantitative pilot study. J Hand Surg [Am]. 2013;38:2387–97.e2382.