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ORIGINAL ARTICLE

Peripheral Nerve

Current Concepts in Brachial Plexus Birth Injuries: A Comprehensive Narrative Review

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Background: Brachial plexus birth injury (BPBI) encompasses a spectrum of upper extremity paralysis cases following childbirth. The etiology of BPBI is multifactorial, involving maternal, obstetric, and neonatal associative factors. Despite opportunities for spontaneous recovery, recent literature demonstrates that a significant proportion of infants experience residual deficits and functional limitations as they age. Understanding the complex anatomy of the brachial plexus, clinical presentations of the pathology, diagnostic workup, current treatment options, and common secondary sequelae is instrumental for appropriate management of BPBI.

Methods: Following a comprehensive search strategy used by the authors to identify relevant literature relating to the progression, patho-anatomy, clinical presentation, management, and treatment of BPBI, this comprehensive narrative review outlines current approaches to assess, manage, and advance BPBI care.

Results: We advocate for prompt referral to specialized multicenter brachial plexus clinics for accurate diagnosis, timely intervention, and individualized patient-centered assessment. Further research is needed to elucidate mechanisms of injury, refine diagnostic protocols, and optimize long-term outcomes.

Conclusions: Collaboration between healthcare providers and families is paramount in providing comprehensive care for infants with BPBI. This review offers insights into the current understanding and management of BPBI, highlighting the importance of tailored approaches and intraoperative decision-making algorithms to optimize functional outcomes. *(Plast Reconstr Surg Glob Open 2024; 12:e6083; doi: 10.1097/GOX.0000000000006083; Published online 22 August 2024.)*

INTRODUCTION

Brachial plexus birth injury (BPBI) represents a spectrum of upper extremity paralysis following childbirth. Although the exact mechanism of injury remains unclear, factors causing traction or compression of the brachial plexus during fetal development or delivery likely contribute to BPBI.[1] The incidence of BPBI ranges between 0.42 to 5.1 per 1000 live births, with variations influenced by obstetric care.[2,3] Recent literature challenges previously reported spontaneous recovery rates of greater than 90%, instead suggesting a less favorable course where up to 36% of infants experience lasting deficits.[4–8]

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Preventative strategies may mitigate BPBI, including prenatal education to reduce factors associated with BPBI and advanced imaging to decrease the risk of fetal malpositioning.[9] Emphasis on regular antenatal visits is essential for determining safe modes of delivery. However, despite comprehensive prenatal care, some BPBI associative factors may go unnoticed until delivery, such as shoulder dystocia.[9] Thus, ongoing research is essential to enhance preventive interventions and improve prenatal risk stratification.

The economic impact of BPBI encompasses both direct healthcare costs, such as diagnostic workup and treatment, and indirect costs, such as long-term functional disability.[10] Disparities in diagnostic and treatment access in low-resource areas may exacerbate these challenges.[10] For instance, under-resourced groups face significant delays in brachial plexus surgery.[11] Addressing such disparities is imperative for promoting equitable care and outcomes in BPBI management.

Disclosure statements are at the end of this article, following the correspondence information.

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This article provides a review of BPBI, covering patho-anatomy, clinical presentation, diagnostic workup, treatment, recent advancements, and disparities in BPBI care. It is based on a comprehensive survey of peer-reviewed literature and the clinical experience of the senior authors in BPBI management.

ETIOLOGY

The American College of Obstetricians and Gynecologists consensus guidelines and other seminal articles highlight significant neonatal, maternal, and obstetric risk factors for BPBI.[12–14] These variables include shoulder dystocia, fetal weight greater than or equal to 4500 g (macrosomia), gestational diabetes, instrumented vaginal delivery, clavicle or humerus fractures, prolonged second stage of labor, cephalopelvic disproportion, maternal obesity, fetal maneuvers, preeclampsia, multiparity, and a prior child with BPBI.[15,16] Shoulder dystocia is the most significant risk factor, presenting a 100-fold increased risk of BPBI.[17] Shoulder dystocia and fetal macrosomia in a previous child are independent risk factors for shoulder dystocia.[18] However, 50%–75% of shoulder dystocia cases occur without associated risk factors, making it an unpredictable obstetric emergency.[18] Although a risk factor for BPBI, shoulder dystocia does not reliably indicate its development. In a 10-year review of 340,322 births across 60 hospitals, 3356 infants weighing 4500 g or more were identified. Shoulder dystocia occurred in 11.7% of births, whereas BPBI was observed in 0.36%.[14] Furthermore, the definition of macrosomia, a minimum birth weight of 4500 g, relies on a limited series of patients.[19] Macrosomia proved an unreliable predictor of BPBI, with BPBI rates ranging from 0.5% to 25.9%. Moreover, concurrent shoulder dystocia and brachial plexus injury varied among macrosomic infants, ranging from 4.6% to 22%.

Overall, the etiology of BPBI remains unclear with ill-defined contributors. This raises the question: are there true risk factors or merely associations for its development? In a series of BPBI infants, 89% were born to nondiabetic mothers, 76% were born to nonobese mothers, 91% had a normal labor, and 76% did not undergo assisted delivery.[19] Therefore, the authors argue that these "risk factors" are simply statistical associations because no single factor can be held accountable based on current evidence. Hence, BPBI arises from an interplay of neonatal, maternal, and obstetric factors.

ANATOMY

Understanding BPBI patho-anatomy is crucial for management. The brachial plexus, an intricate nerve network, arises from cervical nerves C5-C8 and thoracic nerve T1 (occasionally includes C4 and/or T2). It governs movement and sensation in the shoulder, arm, hand, and fingers. The upper trunk (C5-C6) controls the shoulder, elbow flexion, and elbow supination. The middle trunk (C7) controls pectoralis function, latissimus function, and elbow extension. The lower trunk (C8 - T1) governs hand function. The trunks divide into anterior and posterior divisions, which merge to create the lateral, posterior, and medial cords, ultimately forming five terminal motor/sensory nerve branches: musculocutaneous, axillary, radial, median, and ulnar nerves (Fig. 1). Most cases adhere to this anatomical description, but documented variations in brachial plexus anatomy exist.[20]

BPBIs are classified by nerve injury severity and anatomical location.[21,22] The Seddon classification categorizes nerve injury severity into neuropraxia (stretching), axonotmesis (severed axon but intact epineurium), and neurotmesis (complete nerve disruption).[21] Axonometric lesions are graded by the Sunderland classification.[22] Neuropraxic lesions often resolve spontaneously within two months.[23] However, axonotmetic or neurotmetic injuries, leading to neuromas-in-continuity, nerve root rupture, or avulsions, often require surgical intervention.[24] Nerve conduction studies and electromyography help assess injury severity, but definitive diagnosis relies on intraoperative visualization.

Takeaways

Question: What are the current approaches and recent advancements in the diagnosis, management, and treatment of brachial plexus birth injuries (BPBI)?

Findings: This article discusses the importance of early management and intervention in patient progression, patho-anatomy, clinical presentation, management, and treatment of BPBI.

Meaning: We advocate for prompt referral to specialized multicenter brachial plexus clinics for accurate diagnosis, timely intervention, and individualized patient-centered assessment. This review offers insights into the current understanding and management of BPBIs, highlighting the importance of tailored approaches and intraoperative decision-making algorithms to optimize functional outcomes.

CLINICAL FINDINGS

Infants with BPBI display diverse clinical presentations, varying by injury severity and location. Signs in the

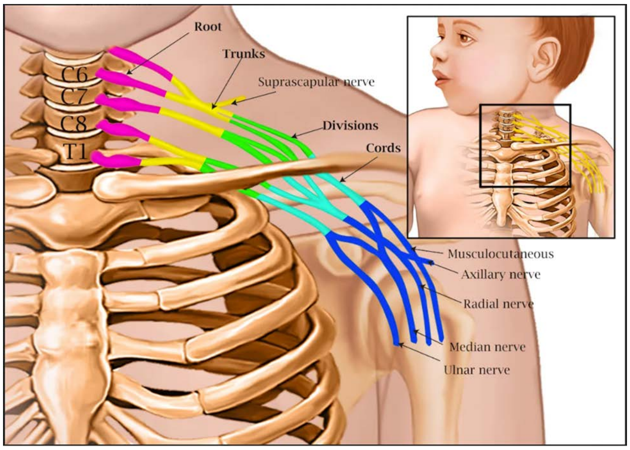

Fig. 1. An illustration of the anatomy of the brachial plexus.

affected upper extremity include paralysis, hypotonia, reduced reflexes, sensory deficits, abnormal posturing, flaccidity, or contractures. Notably, BPBI is highly suspected among neonates with unequal Moro reflexes after shoulder dystocia. Additionally, concurrent clavicular and humeral fractures are common in BPBIs, with an incidence of approximately 8%–9% and 7%, respectively.[25] Infants suspected to have BPBI should also be examined for Horner syndrome, signs of other nerve injuries (ie, phrenic nerve), and torticollis.

Clinical presentation can help localize damaged nerves, whereas the Narakas classification categorizes the extent of the lesion (Table 1).[26] BPBI can manifest as upper trunk palsy (C5-C6), upper/middle trunk palsy (C5-C7), lower trunk palsy (C8-T1), or global palsy (C5-T1) (Fig. 2). Upper trunk palsy (Erb palsy), seen in about 60% of cases, is characterized by the "waiter's tip" position due to deficits in muscles innervated by C5-C6.[20] These injuries result in up to 90% spontaneous recovery, although some studies have challenged this figure.[5,6,27,28] Combined upper and middle trunk palsy (extended Erb palsy), accounting for 20–30% of cases, includes deficits in shoulder internal rotators, triceps, wrist, and finger extensors.[16] Lower trunk palsy (Klumpke palsy) is relatively rare and results in deficits in hand flexors and intrinsics. Total or global plexus palsy, representing 15%–20% of cases, presents as a complete lack of upper extremity function and carries the poorest prognosis. The additional presence of Horner syndrome indicates lower root avulsions and a worse prognosis.[29]

DIAGNOSTIC WORKUP

Prompt evaluation is warranted, as BPBI is often evident at birth and can lead to irreversible functional deficits without timely intervention. Neonates with BPBI associative factors, such as shoulder dystocia, clavicular or humeral fractures, or breech delivery require a focused upper extremity examination. Initial assessment includes a thorough medical history (including pregnancy and birth details), physical examination, and radiography for suspected fractures.

Early signs necessitate immediate referral to specialized BPBI centers. Delay in referral by pediatricians, typically around the age of 6 months, may stem from assumptions that BPBIs resolve spontaneously, highlighting the need for raising awareness and early referral protocols. At our institution, a system automatically flags patients with BPBI or associated factors to prompt referrals to the senior author. This screening system, in partnership with the risk management team and department of obstetrics and gynecology, prioritize streamlining referrals for timely intervention (Fig. 3). Institutions lacking such processes may risk overlooking BPBI cases.

Table 1. Narakas Classification of Brachial Plexus Birth Palsy

Group*	Description	Roots Injured	Site of Weakness/Paralysis
1	Sunderland injury of degrees 1 and 2 and exceptionally degree 3 to the upper trunk.	C5-C6 Degrees of injury 1–2 C7-C8-T1 Normal	Paralysis of abduction and external rotation in the shoulder, or elbow flexion, supination of forearm and, frequently, a palsy of wrist extensors. There is no Horner sign and the fingers and wrist have normal flexors; the intrinsic muscles of the hand are not affected.
2	Sunderland's second- and third-degree injury involves the upper trunk, and degrees 1–2 to the C7 root.†	C5-C6 Degrees of injury 2–3 C7 Degrees of injury 1–2 C8-T1 Normal	As above, but active elbow extension is not as strong.
3	Ruptures or severe injury in continuity of the upper trunk (degrees 4 and 5), a third and sometimes fourth injury to C7, while C8 and T1 are less affected.	C5-C6 Degrees of injury 4–5 C7 Degree of injury 3 C8-T1 Degrees of injury 1–2	Paralysis of the whole limb; the infant has a flail shoulder with indifferent rotation of the humerus, no elbow flexion and weak extension. The wrist is flexed, and the fist is tightly closed. There is no Horner sign.
4	Complete paralysis of C5-T1 with or without a Horner sign.	C5-C6-C7 Degree of injury 5 C8 Degrees of injury 2–4 to T1 OR C5-C6 Degrees of injury 4–5 C7-C8 Degrees of injury 3–4 to T1	Flail extremity with a half-open hand showing scarcely any finger movements.

*As per Narakas,[1] this classification is based on a physical examination 2–3 weeks following birth. This classification excludes many mild cases of brachial plexus birth injuries, which may recover full function in a matter of days.

†Differential diagnosis between groups 1 and 2 is made at 6 weeks of age.

Table created by the authors from descriptions contained within Narakas AO. Obstetrical plexus injuries. In: Lamb DW, Ed., *The Paralysed Hand*. Vol. 2; 1987: 116–135.

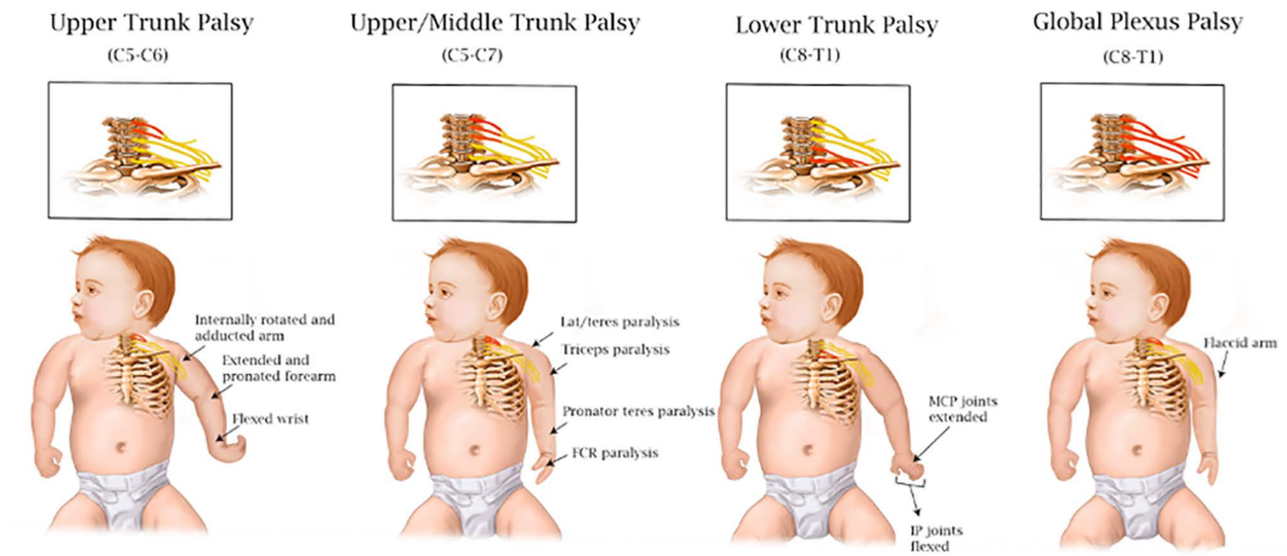

Fig. 2. Clinical presentations of upper trunk palsy (C5-C6 lesion), extended upper/middle trunk palsy (C5-C7 lesion), lower trunk palsy (C8-T1 lesion) and global plexus palsy (C5-T1 lesion).

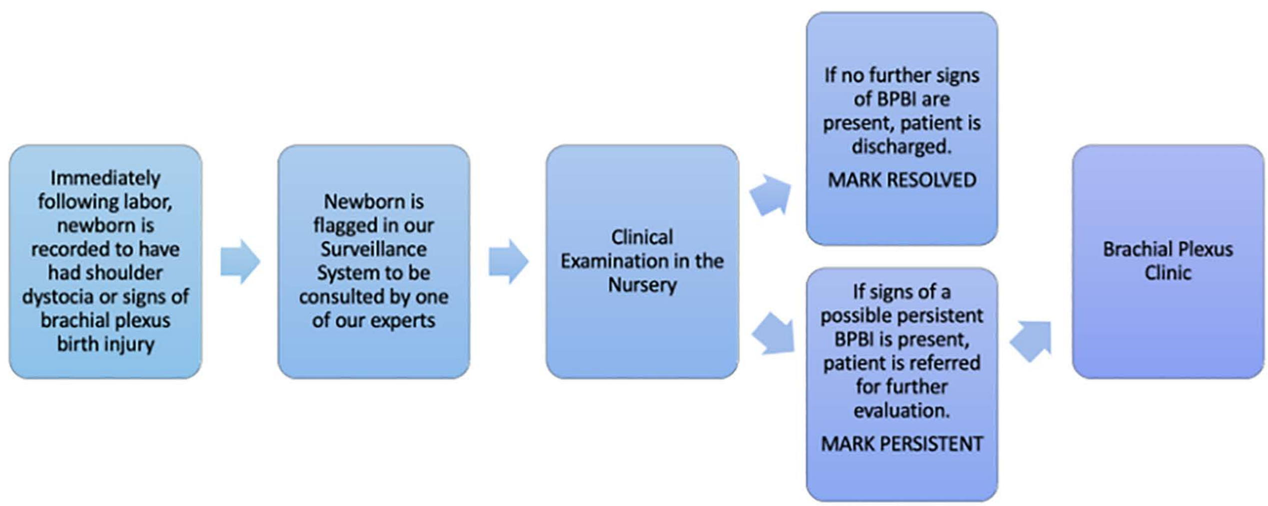

Fig. 3. Quality improvement algorithm to streamline care for patients suspected of BPBI at birth.

At the specialty brachial plexus clinic, a multidisciplinary team should conduct a comprehensive physical examination to ascertain BPBI diagnosis. The team consists of an orthopedic upper extremity surgeon specializing in brachial plexus care, a pediatric neurosurgeon, and a certified occupational hand therapist. Additional members may include a social worker, a neurologist addressing central causes, and a research team. During assessment, the team must rule out potential differential diagnoses such as fractures, septic shoulder, cerebral palsy, and isolated radial nerve palsy.

Standardized examinations like the Active Movement Scale (AMS), Toronto Test Score (TTS), and modified Mallet classification demonstrate both intra- and interobserver reliability for evaluating BPBIs.[30–34] The AMS is particularly useful for neonates and infants suspected of BPBI.[33] The TTS is not recommended before three months of age, while the modified Mallet classification is not recommended before 2 years of age, and requires modification with the ABC loops test between 2 and 3 years old.[31,35,36]

The AMS assesses 15 upper extremity movements (Table 2).[32,33] Functional motion is a score of 6 or higher, whereas full motion is a score of 7.[37] Surgeons or certified hand therapists perform initial grading. Serial AMS scores at each clinic visit track recovery progress, informing clinical decision-making on conservative therapy versus surgery. The TTS assesses five movements on a scale of 0 (no movement) to 2 (full movement), yielding an aggregate score of 0 to 10. A total score below 3.5 at 3 months of age indicates the potential need for surgery.[31] The Mallet classification assesses children

Table 2. Hospital for Sick Children AMS

Movement Grade		Observation
0	Gravity Eliminated	No muscle tone or contraction
1		Muscle contraction, no motion
2		Joint motion ≤ ½ range
3		Joint motion >½ range
4		Full joint motion
5	Against Gravity	Joint motion ≤½ range
6		Joint motion >½ range
7		Full joint range

Scores are assigned for the following upper extremity movements: shoulder abduction, shoulder adduction, shoulder flexion, shoulder external rotation, shoulder internal rotation, elbow flexion, elbow extension, forearm pronation, forearm supination, wrist flexion, wrist extension, finger flexion, finger extension, thumb flexion, and thumb extension.

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aged 3 years and older.[30] The child imitates six postures demonstrated by the clinician (Fig. 4). Graded on a five-point Likert scale, higher scores reflect enhanced upper extremity function. Serial scoring aids in tracking BPBI progression or resolution.

Multiple imaging techniques are available for diagnostic evaluation of BPBI. At initial presentation, plain radiographs are used to rule out fractures (ie, humeral, clavicular). A chest radiograph may assess for elevated hemidiaphragm. The senior author performs point-of-care dynamic ultrasonography of the diaphragm to evaluate phrenic nerve function. Additionally, magnetic resonance imaging (MRI) detects root avulsion and injury severity.[38] However, imaging of the infant brachial plexus may be limited due to size relative to the quality of the scanner. Thus, we focus on the cervical spine to evaluate for root avulsions. Moreover, MRI may be challenging to obtain as infants require anesthesia.[15] Efforts to optimize MRI include the Nonanesthetized Plexus Technique for Infant MRI Evaluation (NAPTIME) study, recently completing recruitment (NIH NCA1703).

Ultrasound is increasingly used in BPBI evaluation due to its availability and superior spatial resolution, offering real-time imaging without sedation or contrast.[39] Further, ultrasound effectively screens for glenohumeral joint dysplasia, with monthly screening starting at 6 weeks old at our institution.[40] [**See table, Supplemental Digital Content 1,** which displays a photograph of a clinician conducting a point-of-care ultrasound (POCUS) on an infant's shoulder with diagnosed BPBI to assess for glenohumeral dysplasia. **http://links.lww.com/PRSGO/D438**.][40] Ultrasound has shown noninferiority to MRI and is the preferred imaging modality at many centers, including our own.[40]

TREATMENT AND MANAGEMENT

Nonoperative Management

The principle guiding BPBI management is timely referral to multidisciplinary specialists for individualized treatment. Treatment requires continuous follow-up from early infancy until musculoskeletal maturity and optimal function are achieved. Although previous literature indicates that most infants spontaneously recover, recent reports suggest incomplete neurological recovery.[5,6] Thus, early occupational therapy is essential for passive range of motion exercises and regular monitoring to prevent contractures, strengthen muscles, stimulate sensory nerves, and promote normal developmental milestones.[41,42]

Additionally, splinting has proven to minimize deformities, prevent contractures, and enhance motor control.[43] The supination and external rotation (Sup-ER) protocol, which includes an orthosis worn by the infant for about 22 hours daily, aims to preserve shoulder development until nerve-generated movement is restored. [**See table, Supplemental Digital Content 2,** which displays a photograph depicting an infant with BPBI wearing the Sup-ER orthosis. **http://links.lww.com/PRSGO/D439**.][44] Application of the Sup-ER splint, as early as age six weeks, may improve arm function.[45]

The use of onabotulinum toxin type A (BTX-A) injections may enhance active motion by targeting imbalanced antagonistic muscles in children with BPBI.[46] BTX-A injections are typically performed alongside spica casting or splinting to maintain closed reduction of the glenohumeral joint. Greenhill et al demonstrate that most patients receiving BTX-A injections go on to require subsequent surgical intervention.[47] Thus, the utility of BTX-A injection remains subject to ongoing debate.[48,49]

Operative Management

Surgical intervention is pursued after exhausting nonoperative treatment. Early surgery (< 6 months) is associated with improved outcomes, while delayed surgery (> 18 months) may diminish nerve regeneration potential and result in complications.[50,51] Indications for surgical exploration relies on surgeons' expertise and literature-based guidelines.[52] Our protocol recommends surgical exploration (1) at the earliest opportunity for global palsy (by 3 months of age); (2) if there is an AMS score of 0 for biceps brachii elbow flexion at 3 months of age or no antigravity function (AMS score of elbow flexion ≤4) at 6 months of age with plateaued scores; (3) if there is no antigravity shoulder function at 6 months of age (AMS score ≤4); or (4) if glenohumeral dysplasia worsens despite Sup-ER orthosis (via ultrasound assessment). Monthly assessment by the multidisciplinary team is crucial to identify indications for surgical intervention.

Microsurgical interventions for operative treatment of BPBI include neurolysis, neuroma-in-continuity resections with nerve grafting, and nerve transfers. Although surgical approach previously varied by surgeon preference, recent studies provide clearer evidence-based guidance. Patients with over 50% conduction across the neuroma during intraoperative testing benefit from neurolysis alone, whereas those with less than 50% conduction, indicating severe disease, are recommended nerve transfers.[37,53]

Surgeons select between nerve grafts and transfers for reconstruction based on intraoperative nerve viability.[54] Nerve allograft is another option, but it is

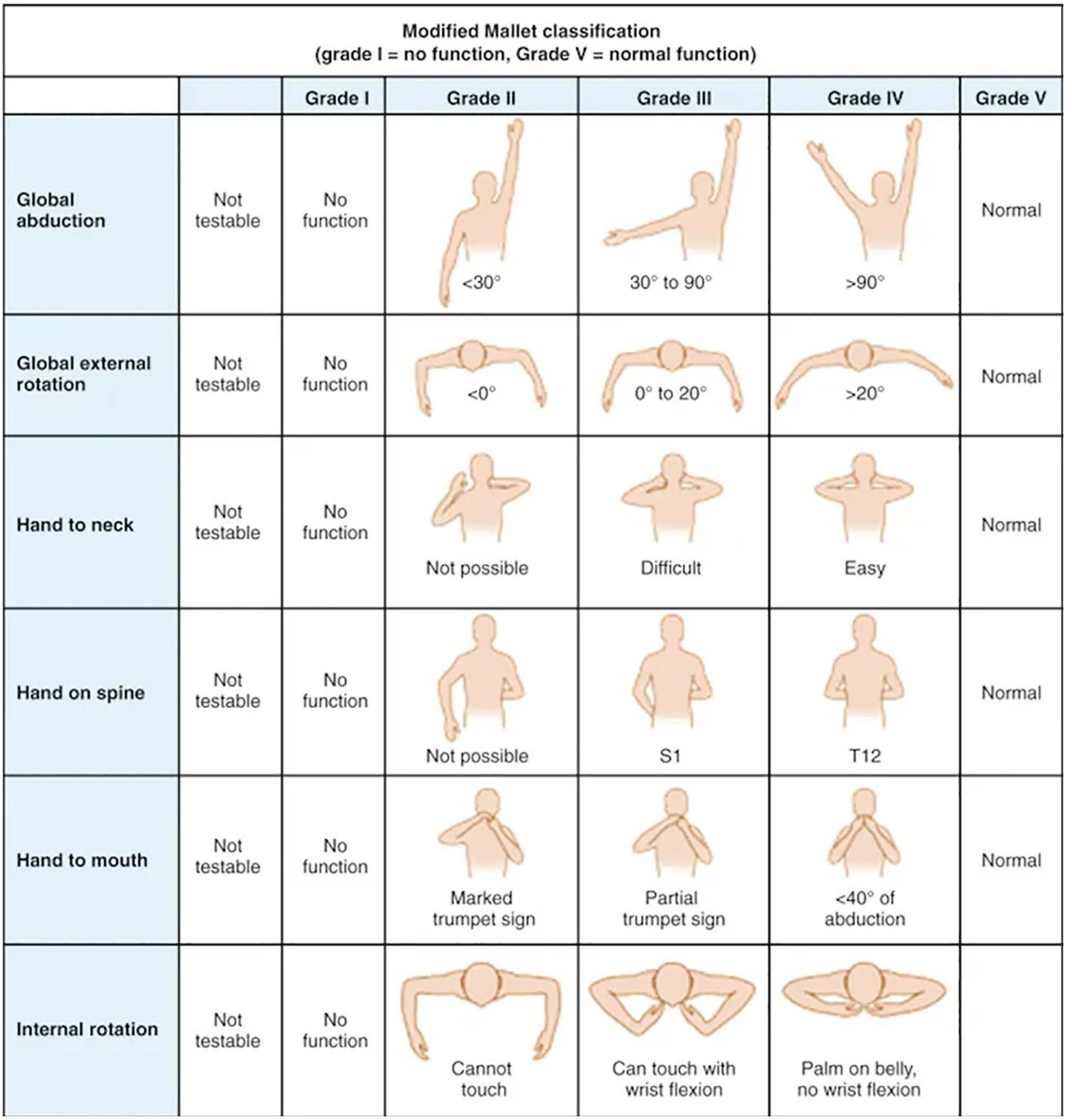

Fig. 4. Modified Mallet classification. Modified permission from Russo SA, Richardson RT, Richards JG, et al. Effect of Glenohumeral reduction type combined with tendon transfer for brachial plexus injury on objective, functional, and patient-reported outcomes. *J Hand Surg Am*. 2021;46:624.e1-624.e11. doi:10.1016/j.jhsa.2020.11.021.

not recommended by the authors. In one case series examining acellular allografts after iatrogenic nerve injuries to the median or ulnar nerve, axons failed to regenerate into the allograft in two cases, while axonal regeneration diminished or ceased in the other three.[55] In a retrospective case series of peripheral mixed nerve reconstruction with nerve allografts, all 14 patients experienced complete failure with no motor or sensory improvement.[56]

In addition, a comparative study of nerve autograft versus nerve transfer found similar postoperative AMS scores and shoulder external rotation recovery.[57] However, the nerve transfer group demonstrated a higher proportion (24%) achieving an AMS score of over five for shoulder external rotation compared with the nerve graft group (5%), indicating advantages in select cases. The nerve transfer group was 42% less likely to require secondary shoulder surgery compared with the nerve graft group.[49] Additional literature supports the superiority of nerve transfers over grafting for restoring active external rotation.[58] Nerve transfers have demonstrated improvement in shoulder abduction, external rotation, and elbow flexion.[58–61] Microsurgical technique selection is tailored to the specific lesion (Fig. 5). The authors' practice incorporates both end-to-end and end-to-side nerve transfers.[58,61] As such, we developed an algorithm to guide individualized intraoperative decision-making (Fig. 6). It is important to note that end-to-side nerve transfers are new in the field of BPBI and are currently being studied.[61] Although they are widely used in the authors' practice, it is important to highlight that this is not a normative practice.

Late peripheral nerve repair yields varying results. One study on patients older than nine months demonstrated improved functional outcomes with nerve transfer and grafting procedures, despite the notion of reduced nerve plasticity in older patients.[62] Another study on nerve repair beyond 12 months demonstrated recovery of elbow flexion and shoulder external rotation, but limited improvement in finger flexion and wrist extension.[63] These findings underscore discrepancies in age cut-offs for peripheral nerve repair.

Patients older than 2 years who missed the opportunity window for nerve transfers may alternatively receive tendon transfers. Limited literature supports tendon transfers in children younger than 2 years.[64] The consensus is to reserve tendon transfers of the latissimus dorsi and teres major muscles for patients ages 2–5 years for restoration of active external rotation.[65,66]

Arthroscopic and open methods address passive external rotation limitations in BPBI with direct visualization.[67,68] Recent research proposes that arthroscopic release of the glenohumeral joint capsule and subscapularis tendon can enhance passive shoulder external rotation and humeral head centering, promoting glenohumeral joint remodeling.[69,70]

Overall, peripheral nerve reconstruction of BPBI via nerve grafting or nerve transfer facilitates functional recovery. Recent studies challenge traditional age cut-offs, indicating potential benefits of nerve transfer beyond the critical window. This underscores the importance of individualized decision-making for optimal outcomes, regardless of age at presentation.

SECONDARY CONSEQUENCES

Musculoskeletal complications of delayed management or untreated BPBI are well documented in the literature. Shoulder dysfunction is the most common consequence and is described using Water classification, which denotes severity of glenohumeral deformity (Table 3), or Zancolli classification, which describes functional limitations due to contractures with or without joint deformities (Table 4). Glenohumeral dysplasia, affecting approximately 33% of BPBI patients, results from musculoskeletal changes in the glenohumeral cavity.[71,72] Other secondary shoulder abnormalities include paralysis of internal and/or external rotator muscle groups and contractures, leading to a shoulder that is either externally rotated and abducted or internally rotated and adducted.

Surgical interventions encompass a spectrum of techniques, including contracture release, muscle transfers, humerus and glenoid osteotomies, and shoulder arthrodesis (Fig. 7). These interventions are aimed at addressing mechanical shoulder dysfunction through case-specific needs to optimize functional outcomes. The timing of surgical intervention plays an essential role in ensuring the effectiveness of the treatment approach. We suggest considering these surgical procedures for toddlers through young adults (approximately 3–12 years old) without degenerative changes. It is important to emphasize that shoulder arthrodesis is considered as a final resort and is recommended for older patients with evidence of a closed physis.

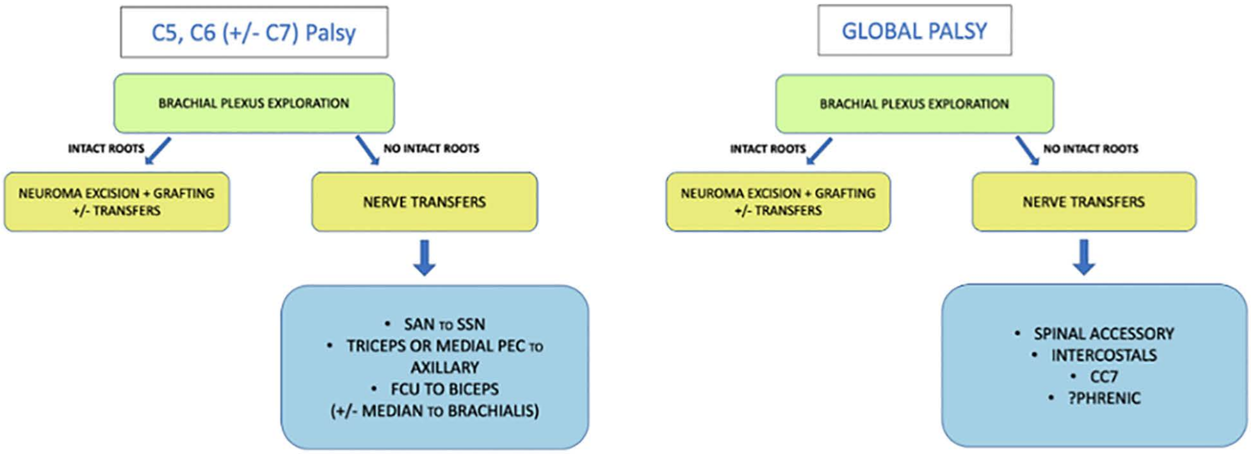

Fig. 5. The technique selection algorithm for nerve transfer based on intraoperative exploration findings. SAN, spinal accessory nerve; SSN, suprascapular nerve; FCU, flexor carpi ulnaris; CC7, contralateral C7.

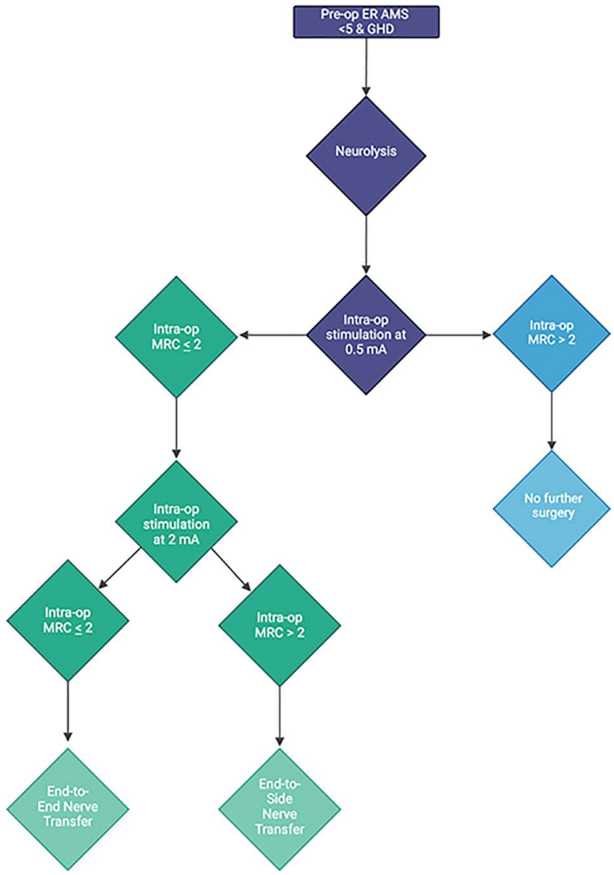

Fig. 6. Microsurgical algorithm for intraoperative decision-making in infants with BPBI. This algorithm is recommended for patients with preoperative external rotation AMS scores of less than 5 of 7 and evidence of glenohumeral dysplasia. Following neurolysis, nerve stimulation is conducted via a hand-held nerve stimulator at 0.5 mA. If the corresponding muscle moves against gravity intraoperatively (translating to an MRC score of >2), no further surgery is recommended. If the corresponding muscle does not move against gravity, nerve stimulation is conducted at 2 mA. If the corresponding muscle moves against gravity intraoperatively (corresponding to an MRC score of >2), an end-to-side nerve transfer is performed. If there is still no antigravity movement of the innervated muscle (corresponding to an MRC score of ≤2) following stimulation at 2mA, an end-to-end nerve transfer is performed. ER, external rotation; GHD, glenohumeral dysplasia; MRC, Medical Research Council Scale for Muscle Strength; mA, milliamps.

Some argue that correcting these shoulder deformities may render previously compensatory scapular winging detrimental, necessitating subsequent correction.[73,74] Despite controversy surrounding the topic, addressing scapular winging either conservatively or surgically, via transfer of the contralateral trapezius or pectoralis major muscle to the affected scapula, is dependent on the provider and the caregivers.[75]

Disparities in BPBI Care

Children in low-resource settings experience higher rates of BPBI and delayed referral to specialists.[10] Due to late presentation, secondary procedures such as tendon transfer and rehabilitation are used to improve functionality, but do not optimally restore upper extremity function.[74] Improved access to BPBI care and treatment is necessary to reduce lifelong functional deficits and promote equitable treatment.

Table 3. Waters Classification of Glenohumeral Deformity Secondary to BPBI

Classification	Deformity Severity	Description of Deformity
Type I	Normal glenoid	Difference of less than 5 degrees between affected and unaffected glenoid
Type II	Minimal deformity	Difference of more than degrees between affected and unaffected glenoid with no posterior subluxation
Type III	Moderate deformity	Posterior subluxation of the humeral head with less than 35% of the humeral head anterior to the scapular line
Type IV	Severe deformity	Presence of a false glenoid
Type V	Humeral head deformity	Progressive or complete dislocation of the humeral head with flattening of the humeral head and glenoid
Type VI	Infantile dislocation	Glenohumeral joint dislocation in infancy
Type VII	Growth arrest	Growth arrest of the proximal humerus

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Table 4. Classification of Secondary Shoulder Deformities following BPBI by Zancolli

Type	Subtype	Pathology	Treatment
I: Shoulder contracture			
(a) Internal rotation/adduction contracture	(i) Normal joint	Subscapularis contracture	Subscapularis release, L'Episcopo transfer
	(ii) Joint deformity	Posterior joint subluxation	Humerus osteotomy
(b) External rotation/abduction contracture	(i) Normal joint	Infraspinatus/teres minor contracture	Release of external rotators
	(ii) Joint deformity	Anterior joint subluxation	Humerus osteotomy
(c) Combine internal and external rotation/abduction contracture		Combined pathology	Release both subscapularis and external rotators
(d) Pure abduction contracture	(iii)	Contracted supraspinatus	"Z" plasty of supraspinatus
II: Flaccid paralysis		Paralysis of all muscles	Shoulder arthrodesis

FUTURE RESEARCH

Firstly, as present literature demonstrates ill-defined contributors to BPBI, further investigation is needed to identify true BPBI risk factors and elucidate mechanisms underlying injury. Moreover, further research should

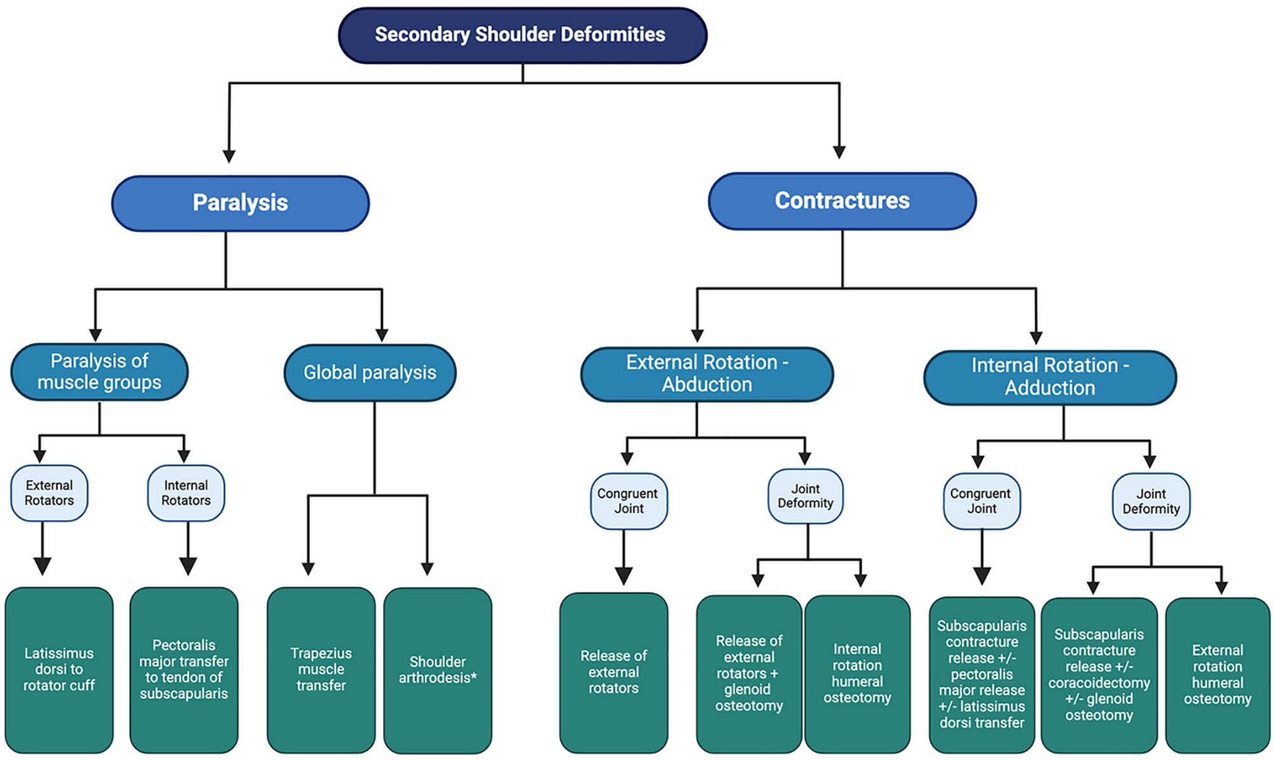

Fig. 7. Algorithm of our recommended surgical techniques for correcting secondary shoulder deformities for children between the ages of 3 and 12 years old. *Shoulder arthrodesis is reserved for older patients with evidence a closed physis.

refine clinical guidelines for peripheral nerve repair timing, as discrepancies in patient age cut-offs exist among present studies. Future studies should also investigate parameters of BPBI care for underserved communities, particularly lower socioeconomic and under-resourced groups, to promote equitable BPBI care.

CONCLUSIONS

BPBI has the propensity for long-term disability without prompt treatment. Clinical presentation varies based on injury severity and location, requiring early assessment and physical examination. Nonoperative approaches facilitate recovery for most cases, yet severe instances warrant surgical intervention. Early referral to multidisciplinary teams specializing in BPBI is imperative for optimal functional outcomes and adequate follow-up care.

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DISCLOSURES

Steven M. Koehler is a committee member of the American Society for Surgery of the Hand (ASSH), and a stockholder and member of the medical advisory board for Reactiv, Inc. Erin Meisel is a paid consultant for Tissium, Inc and a stockholder of Joint Development LLC. The other authors have no financial interest to declare in relation to the content of this article.

ETHICAL APPROVAL

Ethical approval to report these cases were obtained from Montefiore Medical Center's institutional review board (Study ID: 2022-14122).

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