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Obstetric brachial plexus palsy: a comprehensive review of evaluation and management

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ABSTRACT

Obstetric brachial plexus palsy (OBPP) is the most common peripheral nerve injury associated with childbirth, with an incidence of 0.4–1.6 per 1000 live births. It typically results from traction on the brachial plexus during difficult deliveries and is associated with risk factors such as shoulder dystocia, fetal macrosomia, breech presentation, prolonged labour, and maternal diabetes. The severity of neurological involvement ranges from isolated upper trunk lesions (Erb's palsy) to complete brachial plexus paralysis with associated Horner syndrome. Early and accurate clinical evaluation is critical for prognostication and treatment planning. Serial clinical examinations remain the cornerstone of assessment, while imaging and electrophysiological studies may aid in defining the level and severity of injury. Although spontaneous recovery occurs in a majority of infants, a significant subset requires timely surgical intervention. Advances in microsurgical techniques, including nerve grafting and distal nerve transfers, have improved functional outcomes. In addition to neural deficits, OBPP is associated with secondary musculoskeletal sequelae such as shoulder contractures and glenohumeral dysplasia. This review provides a comprehensive overview of the pathoanatomy, clinical evaluation, prognostic indicators, and contemporary management strategies for OBPP, with emphasis on surgical reconstruction and management of secondary deformities.

Keywords: Obstetric brachial plexus palsy, Brachial plexus birth palsy, Erb's palsy; Nerve grafting, Nerve transfer

INTRODUCTION

Obstetric brachial plexus palsy (OBPP) is a birth-related peripheral nerve injury with a reported incidence ranging from 0.38 to 1.56 per 1,000 live births.[1] The observed variation in incidence across populations is likely influenced by differences in obstetric practices, maternal and neonatal characteristics, and regional variations in average birth weight.[2,3] Numerous perinatal risk factors have been associated with the development of OBPP, including fetal macrosomia, multiparity, a previous history of OBPP, maternal diabetes, prolonged labour, breech presentation, shoulder dystocia, and operative vaginal delivery.[4-11]

The underlying mechanism of injury is multifactorial. In addition to mechanical traction on the brachial plexus during delivery, fetal distress may predispose to nerve injury by causing generalized muscle hypotonia, thereby reducing the protective support normally provided by surrounding musculature against excessive stretch or compression forces.[12] Although caesarean delivery does not entirely eliminate the risk of OBPP, it has been shown to markedly reduce its occurrence in selected high-risk pregnancies, with reports suggesting an almost 100-fold reduction in incidence.[13]

The clinical presentation of OBPP varies according to the extent of nerve root involvement. Isolated involvement of the C5 and C6 nerve roots results in Erb's palsy, whereas involvement of the C5, C6, and C7 roots is referred to as extended Erb's palsy.

Injury affecting the entire brachial plexus (C5–T1) results in global palsy, which is typically associated with the most severe functional impairment.

ANATOMY OF THE BRACHIAL PLEXUS

The brachial plexus is typically formed by the ventral rami of the fifth cervical (C5) through the first thoracic (T1) spinal nerve roots. Anatomical variations are not uncommon. In approximately 22% of individuals, a prefixed plexus receives an additional contribution from the C4 nerve root, whereas a post-fixed plexus, involving contribution from T2, is considerably less frequent and occurs in about 1% of cases.[14]

The C5 and C6 roots unite to form the upper trunk, the C7 root continues as the middle trunk, and the C8 and T1 roots join to form the lower trunk. Each trunk subsequently divides into anterior and posterior divisions. The posterior divisions of all three trunks converge to form the posterior cord, while the anterior divisions of the upper and middle trunks form the lateral cord. The anterior division of the lower trunk continues as the medial cord.

The major peripheral nerves of the upper limb arise from these cords. The musculocutaneous nerve originates from the lateral cord, the ulnar nerve from the medial cord, and the axillary and radial nerves from the posterior cord. The median nerve is formed by contributions from both the lateral and medial cords. In addition to these terminal branches, several important collateral branches arise at different levels of the plexus. The dorsal scapular nerve originates from the C5 root, while the long thoracic nerve receives contributions from the C5–C7 roots. The suprascapular nerve arises from the upper trunk, whereas the lateral and medial pectoral nerves originate from the lateral and medial cords, respectively. The thoracodorsal nerve, along with the upper and lower subscapular nerves, arises from the posterior cord.

From proximal to distal, the brachial plexus is organized into roots, trunks, divisions, cords, and terminal branches. The divisions are situated posterior to the clavicle, an anatomical relationship that is important when considering the location and extent of injury. Through its terminal and collateral branches, the brachial plexus provides motor innervation to virtually all muscles of the upper limb and serves as the principal neural pathway for upper extremity function (Figure 1).

INJURY PATTERNS AND CLINICAL EXAMINATION

Obstetric brachial plexus palsy encompasses a wide spectrum of nerve injuries, ranging from transient neurapraxia to complete root avulsion. Based on the Sunderland classification, lesions may be categorized as stretch injuries (type I), varying degrees of nerve rupture (types II–V), or avulsion injuries. The pattern of injury is influenced by the mechanism of delivery. Extraforaminal ruptures involving the upper trunk are more commonly associated with vertex deliveries complicated by shoulder dystocia, whereas C5–C6 root avulsions are seen more frequently in breech presentations and may occasionally occur bilaterally.[15-17] In cases of total plexus involvement, different regions of the brachial plexus may simultaneously sustain stretch, rupture, and avulsion injuries. The diagnosis of OBPP is primarily clinical. A careful examination is essential not only to confirm the diagnosis but also to exclude other causes of apparent upper-limb weakness. Differential diagnoses include pseudoparalysis secondary to fractures, central nervous system or cervical spinal cord injury, neuromuscular disorders, and congenital anomalies affecting the upper limb. Because clavicular fractures may coexist with brachial plexus injury, radiographic evaluation is often warranted. Although humeral shaft fractures can mimic brachial plexus palsy, the two injuries rarely occur together in the same extremity.

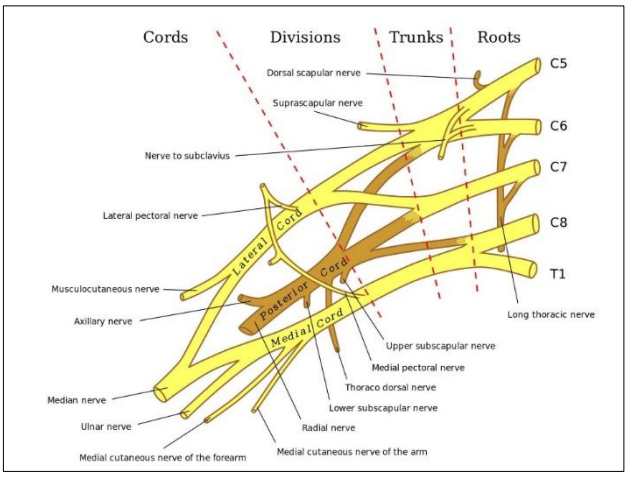

Figure 1: Anatomy of the brachial plexus illustrating the roots, trunks, divisions, cords, and terminal branches.[63]

Clinical findings vary according to both the severity of nerve injury and the age at assessment. Early examination should include observation of spontaneous limb movement, evaluation of neonatal reflexes and assessment of active motor responses. Recovery during infancy is dynamic, with neurological findings often evolving over the first weeks and months of life. Consequently, serial clinical assessments are critical for monitoring recovery and identifying infants who may benefit from surgical intervention.[17-19] Regular examinations at intervals of approximately one to three months during infancy provide valuable prognostic information and help guide treatment decisions. Among the clinical signs associated with severe injury, Horner syndrome, characterized by ptosis, miosis, enophthalmos, and anhidrosis, is particularly important because it suggests involvement of the lower cervical and upper thoracic roots and is generally associated with a poorer prognosis. The presence of Horner syndrome often raises suspicion for root avulsion and extensive plexus injury.

To facilitate prognostication, Narakas and Slooff proposed a four-group clinical classification system for OBPP.[20-22] Group I corresponds to the classic upper trunk (C5–C6) or

Erb's palsy, characterized by loss of shoulder abduction and external rotation, elbow flexion, and forearm supination, while wrist and finger function remain preserved.[23] This group has the most favourable prognosis, with spontaneous recovery reported in up to 90% of affected infants.[22] Group II represents involvement of C5–C7 and is distinguished by additional weakness of wrist and finger extension, resulting in the characteristic "waiter's tip" posture. Outcomes are generally less favourable than those observed in isolated C5–C6 injuries. Group III consists of a flail upper extremity without Horner syndrome, whereas group IV represents the most severe form of injury, characterized by a flail limb in association with Horner syndrome. Infants in the latter group may also demonstrate phrenic nerve involvement, resulting in diaphragmatic paralysis and elevation of the ipsilateral hemidiaphragm.[24]

An important objective of the clinical evaluation is determining whether the lesion is preganglionic or postganglionic, as this distinction has significant prognostic and therapeutic implications. Preganglionic injuries occur proximal to the dorsal root ganglion and represent root avulsions from the spinal cord. Because the motor neuron cell bodies remain within the spinal cord, these injuries lack the potential for spontaneous regeneration. Clinical signs suggestive of a preganglionic lesion include Horner syndrome, an elevated hemidiaphragm due to phrenic nerve involvement, scapular winging resulting from long thoracic nerve dysfunction, and weakness of the rhomboids indicating dorsal scapular nerve involvement.

The management options differ substantially between preganglionic and postganglionic injuries. Preganglionic lesions require reconstruction using nerve transfers, commonly employing intercostal nerves, branches of the spinal accessory nerve, or other donor nerves.[25-29] In contrast, postganglionic ruptures preserve proximal and distal nerve segments outside the zone of injury, allowing reconstruction with interposition nerve grafts. Nerve transfers may also be used in selected cases. Unlike preganglionic injuries, postganglionic lesions retain the potential for spontaneous functional recovery, likely owing to partial nerve continuity and the presence of overlapping neural innervation pathways during early development.[30]

CLINICAL EVALUATION OF RECOVERY

Accurate assessment of motor recovery in infants with obstetric brachial plexus palsy remains challenging because conventional muscle strength grading systems depend on voluntary patient participation. The Medical Research Council (MRC) grading system, widely used in adults, classifies muscle strength on a scale from 0 (no contraction) to 5 (normal power). However, its application in infants is limited by the inability of young children to reliably perform voluntary motor tasks. To address this limitation, Gilbert and Tassin proposed a modified grading system tailored for infants, consisting of four grades: 0 (no contraction), 1 (muscle contraction without movement), 2 (movement with gravity eliminated), and 3 (full movement against the weight of the limb).[31] Several additional assessment tools have subsequently been developed to quantify recovery and guide treatment decisions.

The modified Mallet classification is commonly used to evaluate global upper-extremity function, particularly shoulder performance, rather than assessing individual muscle groups in isolation.[32] In contrast, the hospital for sick children active movement scale (AMS), developed by Clarke and colleagues, provides a more detailed assessment of motor recovery by grading movements both with gravity eliminated and against gravity on an eight-point scale (0–7). The Toronto test score evaluates selected elbow, wrist, and hand functions during infancy and is primarily used to identify patients who may benefit from microsurgical reconstruction rather than to monitor long-term functional recovery. Longitudinal studies have demonstrated that the pattern and timing of motor recovery are among the most important predictors of outcome.[33] Nevertheless, each assessment tool has inherent limitations, and no single scoring system has emerged as the definitive standard. Consequently, clinical decision-making is typically based on a combination of serial examinations, functional assessments, and the trajectory of neurological recovery over time.

ROLE OF IMAGING AND ELECTROPHYSIOLOGICAL STUDIES

Imaging and electrophysiological investigations serve as valuable adjuncts in the evaluation of obstetric brachial plexus injuries, particularly when surgical intervention is being considered. Their principal role is to help differentiate preganglionic root avulsions from postganglionic nerve ruptures and to assist with preoperative planning.

Historically, myelography and CT myelography were the primary imaging modalities used to identify nerve root avulsions.[34-39] Advances in magnetic resonance imaging (MRI) have led to its widespread adoption because it provides comparable diagnostic accuracy while also allowing visualization of the extraforaminal components of the brachial plexus.[40] Modern techniques, including high-resolution spin-echo MRI, MR myelography, and MR neurography, have further improved visualization of neural structures and may aid in detecting complex lesions, including double-crush injuries.[41,42] Despite these advances, imaging findings should be interpreted cautiously. Although they provide important anatomical information, definitive confirmation of root avulsion often occurs only during surgical exploration. Therefore, imaging is best regarded as a complementary tool rather than a substitute for careful clinical assessment.

Electrophysiological studies, including nerve conduction studies and electromyography (EMG), have also been used

to assess the severity and level of nerve injury.[43] The presence of preserved sensory nerve conduction in the absence of motor conduction is highly suggestive of a preganglionic avulsion injury. Similarly, failure to demonstrate evidence of reinnervation by approximately three months of age may indicate a severe lesion with limited potential for spontaneous recovery. The H-reflex has also been investigated as a potential prognostic marker.[44]

However, the interpretation of electrophysiological findings in infants can be challenging. Detection of electrical activity within a muscle does not necessarily correlate with meaningful functional recovery, and partial reinnervation may produce findings that are difficult to reconcile with the clinical examination. For this reason, most specialized brachial plexus centres continue to place greatest emphasis on serial physical examinations when determining prognosis and deciding on the timing and necessity of surgical reconstruction.

TIMING OF SURGICAL INTERVENTION

The optimal timing of microsurgical reconstruction in obstetric brachial plexus palsy remains a subject of debate; however, most treatment algorithms are based on the pattern and rate of spontaneous motor recovery during infancy. Historically, the absence of meaningful neurological recovery within the first few months of life has been considered an indication for surgical exploration.[45,46] As early as 1917, Wyeth and Sharpe recommended operative intervention in infants who failed to demonstrate recovery by three months of age.[47]

Subsequent studies have focused on specific motor milestones as predictors of outcome. Gilbert and Tassin identified the return of antigravity biceps function by three months as a key indicator of favourable spontaneous recovery, a finding later supported by Waters.[16,19] Other investigators have emphasized the importance of assessing additional muscle groups, including the deltoid and triceps muscles, when determining prognosis.[48] Clarke and colleagues proposed a more comprehensive evaluation using the hospital for sick children active movement scale (AMS), incorporating recovery of elbow flexion, elbow extension, wrist extension, finger extension, and thumb extension into a composite score that helps identify candidates for microsurgical reconstruction.[49,50]

For infants with equivocal recovery, Clarke further introduced the “cookie test” at approximately nine months of age. Failure to bring a cookie to the mouth with the arm held at the side suggests inadequate upper-limb function and may support the decision to proceed with surgical reconstruction. Although practice patterns vary among centres, commonly accepted indications for early nerve surgery include total plexus palsy associated with Horner syndrome and failure of biceps recovery in upper plexus lesions. In general, reconstruction is performed between 3 and 9 months of age, with many surgeons’ favouring earlier intervention to minimize prolonged denervation and irreversible motor end-plate degeneration. Early surgery may also facilitate parental acceptance before compensatory movement patterns and joint deformities become established. A summary of the described indications for microsurgical intervention is shown in Table 1.

ROLE OF NERVE SURGERY

The goals of microsurgical reconstruction are to restore critical upper-limb functions, particularly elbow flexion, shoulder abduction, and external rotation. Surgical options include neurolysis, neuroma resection with nerve grafting, and nerve transfers. The choice of procedure depends on the type, location, and severity of the injury identified during surgical exploration. Neurolysis alone has a limited role and is generally reserved for lesions demonstrating satisfactory intraoperative conduction across a neuroma-in-continuity. When less than 50% of the expected muscle action potential is preserved following neurolysis, resection of the neuroma and reconstruction with nerve grafts is typically recommended. Several studies have demonstrated superior outcomes with nerve grafting compared with neurolysis alone in appropriately selected patients.[51] For postganglionic ruptures, the standard reconstructive strategy involves excision of the neuroma and interposition grafting using sural nerve autografts.[52] In isolated C5–C6 injuries, grafts are commonly directed toward the anterior division of the upper trunk, lateral cord, musculocutaneous nerve, suprascapular nerve, and posterior division pathways supplying the axillary and radial nerves. Additional grafting from C7 may be required when the middle trunk is involved.

Over the past two decades, nerve transfers have gained increasing popularity, particularly in upper plexus injuries involving the C5–C6 or C5–C7 roots.[53,54,59-61] Commonly performed transfers include transfer of intercostal nerves or a partial ulnar nerve fascicle to the motor branch of the biceps (Oberlin transfer), spinal accessory nerve transfer to the suprascapular nerve, and transfer of the motor branch to the long head of the triceps to the axillary nerve. Functional outcomes following these procedures appear comparable to those achieved with nerve grafting for restoration of shoulder and elbow function.[53,54,60] In complete brachial plexus avulsions, nerve transfers represent the only viable reconstructive option because no proximal nerve stump is available for grafting. Donor nerves may include intercostal nerves, the spinal accessory nerve, the contralateral C7 root, and, in selected cases, the hypoglossal nerve. Compared with adults, infants possess a greater capacity for neurological recovery and cortical reorganization, and the shorter distance between the site of reconstruction and target muscles may contribute to improved functional outcomes, including the potential for meaningful hand recovery.

Despite ongoing controversy regarding the ideal timing and technique of reconstruction, current evidence supports

individualized treatment planning based on the extent of injury, pattern of recovery, and functional deficits. Distal nerve transfers have become an increasingly important component of modern OBPP reconstruction, with procedures such as the Oberlin transfer and spinal accessory-to-suprascapular nerve transfer demonstrating encouraging results in restoring elbow flexion and shoulder function.[62] Recent advances in microsurgical reconstruction have led to increasing use of selective distal nerve transfers, either as standalone procedures or in combination with nerve grafting. These transfers provide a shorter regeneration distance to the target muscle and may facilitate earlier functional recovery. Commonly employed transfers include the Oberlin transfer for restoration of elbow flexion and spinal accessory-to-suprascapular nerve transfer for shoulder function. Growing evidence suggests that carefully selected distal nerve transfers can achieve outcomes comparable to traditional nerve grafting while potentially reducing donor-site morbidity and operative complexity.

Current commonly accepted indications for surgery include: total plexus palsy with Horner syndrome and minimal recovery by 2–3 months, absence of meaningful biceps recovery in upper plexus lesions by 3–6 months, failure to achieve functional upper-limb recovery on serial clinical assessment, and poor performance on validated functional assessment tools, including the AMS and cookie test.

CONTRACTURES AND SECONDARY MUSCULOSKELETAL DEFORMITIES

Despite neurological recovery, many children with obstetric brachial plexus palsy develop secondary musculoskeletal deformities resulting from persistent muscle imbalance, impaired muscle growth, and abnormal joint loading. These deformities may progress over time and can significantly affect upper-extremity function. The shoulder is the most commonly affected joint, although contractures and deformities may also develop at the elbow, forearm, wrist, and hand. A summary of the common secondary deformities and their brief management is given in Table 2.

Shoulder

The shoulder is particularly vulnerable to secondary deformity following upper trunk injuries. Persistent weakness of the shoulder abductors and external rotators, combined with relatively preserved internal rotators, leads to progressive internal rotation and adduction contractures. Over time, abnormal joint mechanics contribute to glenohumeral dysplasia, characterized by posterior humeral head subluxation, glenoid retroversion, and progressive joint incongruity.[55,56] Although muscle imbalance has traditionally been considered the primary mechanism underlying contracture formation, current evidence suggests a multifactorial process involving impaired muscle growth, altered muscle architecture, capsular tightness, and abnormal joint development. As a result, shoulder deformity is increasingly recognized as a dynamic process that begins early in infancy rather than a late sequela of paralysis alone.[56]

Early recognition is critical because glenohumeral dysplasia may develop within the first year of life, and dislocation has been reported in infants as young as three months of age.[57,58] Regular clinical and imaging surveillance allows timely intervention before irreversible joint deformity occurs. Initial management focuses on maintaining passive range of motion through physiotherapy, stretching programmes, and splinting. Botulinum toxin injections into internal rotator muscles may be used as an adjunct in selected patients to reduce muscle imbalance and facilitate stretching. When progressive contracture or glenohumeral deformity develops despite conservative treatment, surgical intervention may be indicated.

Common indications for surgery include infantile glenohumeral dislocation, persistent internal rotation-adduction contracture, progressive loss of shoulder abduction and external rotation despite neurological recovery, and worsening glenohumeral dysplasia. Surgical options include soft-tissue releases, subscapularis lengthening or slide procedures, tendon transfers, and reduction of glenohumeral dislocations. Tendon transfer procedures, most commonly involving transfer of the latissimus dorsi and teres major, are used to improve active shoulder abduction and external rotation in children with persistent functional deficits.

In advanced cases with severe glenohumeral dysplasia, soft-tissue reconstruction alone may not restore functional alignment. Derotational humeral osteotomy can improve the functional arc of motion by repositioning the hand into a more useful orientation, thereby facilitating activities such as feeding, grooming, and personal care. Following reconstructive procedures, immobilization in a shoulder spica cast or brace is typically maintained for four to six weeks before rehabilitation is resumed.

Surveillance imaging of the glenohumeral joint

Early identification of glenohumeral dysplasia has become an important component of contemporary management of obstetric brachial plexus palsy. Clinical examination alone may underestimate the severity of underlying joint deformity, particularly in infants with persistent internal rotation contractures. Consequently, imaging surveillance is increasingly used to detect early changes in glenohumeral alignment before irreversible dysplasia develops. Ultrasonography offers a non-invasive and radiation-free method for evaluating humeral head position in young infants and can facilitate serial monitoring during the first year of life. Magnetic resonance imaging provides a more comprehensive assessment of glenoid version, humeral head subluxation, muscle atrophy, and associated soft-tissue abnormalities.

MRI is particularly valuable when surgical intervention is being considered because it allows detailed characterization of the severity of glenohumeral deformity. Early recognition of dysplasia enables timely intervention through stretching programmes, splinting, botulinum toxin injections, soft-tissue balancing procedures, or joint reduction techniques. Increasing evidence suggests that treatment instituted before the development of advanced glenoid deformity may improve joint remodelling and preserve long-term shoulder function.

Elbow

The elbow is commonly affected by both weakness and contracture. Persistent weakness of elbow flexion can significantly impair upper-limb function and may necessitate secondary reconstructive procedures. Tendon transfers using the pectoralis major, latissimus dorsi, triceps, or flexor-pronator muscle mass (Steindler flexorplasty) have been employed to restore active elbow flexion in selected patients. Elbow flexion contractures are another frequent sequela and may progressively interfere with function and limb positioning. Management strategies vary according to severity and patient age and range from observation and serial casting to surgical release, gradual distraction techniques, tendon balancing procedures, and corrective osteotomies. Treatment should be individualized based on functional limitations and the degree of fixed deformity.

Forearm

Abnormal forearm rotation is a common consequence of incomplete neurological recovery. Children may present with either excessive pronation or fixed supination depending on the pattern of muscle imbalance. Mild deformities often respond to physiotherapy, splinting, and activity-based rehabilitation. In more severe cases, particularly among children with global plexus involvement, fixed supination deformities may coexist with elbow flexion contractures, resulting in substantial functional impairment. Surgical correction may involve musculotendinous lengthening procedures, tendon transfers, or rotational osteotomies aimed at restoring a more functional forearm position. Because isolated loss of supination or pronation rarely causes major disability, operative intervention is generally reserved for patients with significant functional limitations.

Wrist and hand

Residual weakness of the wrist and hand can significantly compromise upper-extremity function, even when proximal recovery is satisfactory. Wrist instability may reduce the effectiveness of tendon transfers and impair grasp and release activities. Surgical stabilization procedures include capsulodesis, partial wrist fusion in skeletally immature patients, and wrist arthrodesis after skeletal maturity.

Ulnar deviation of the wrist is a frequently encountered deformity and typically results from imbalance between preserved and weakened wrist stabilizers. Tendon transfer procedures, commonly redirecting the extensor carpi ulnaris or flexor carpi ulnaris to a more central insertion, can improve wrist alignment and enhance overall hand function. These procedures are often performed in conjunction with corrective surgeries at the elbow or forearm to optimize upper-limb mechanics.

Table 1: Described indications for microsurgical reconstruction in obstetric brachial plexus palsy.

Author/group	Assessment criterion	Suggested timing	Surgical indication
Wyeth and Sharpe[47]	Absence of clinical recovery	3 months	Consider surgical exploration
Gilbert and Tassin[16]	Absence of antigravity biceps function	3 months	Nerve reconstruction recommended
Waters[19]	Delayed biceps recovery	Early infancy	Predictor of poor spontaneous recovery
Laurent et al[48]	Recovery of biceps, triceps, and deltoid function	Serial assessment	Persistent weakness may warrant surgery
Clarke et al[49]	Active movement scale (AMS) score incorporating elbow, wrist, finger, and thumb function	Serial assessment during infancy	Composite score used to identify candidates for surgery
Clarke et al[49]	Failure of the “cookie test”	Approximately 9 months	Consider microsurgical reconstruction

Table 2: Secondary musculoskeletal deformities in obstetric brachial plexus palsy.

Anatomical region	Common deformity	Pathophysiology	Treatment options
Shoulder	Internal rotation-adduction contracture, posterior	Muscle imbalance, impaired muscle growth, capsular	Physiotherapy, splinting, botulinum toxin injections, soft-tissue release,

Continued.

Anatomical region	Common deformity	Pathophysiology	Treatment options
	subluxation, glenohumeral dysplasia	contracture, abnormal joint development	tendon transfer, humeral derotational osteotomy
Elbow	Flexion weakness, flexion contracture	Persistent denervation, muscle imbalance, soft-tissue shortening	Tendon transfers, serial casting, contracture release, corrective osteotomy
Forearm	Fixed pronation or supination deformity	Imbalance between forearm pronators and supinators	Therapy, splinting, tendon transfers, rotational osteotomy
Wrist	Instability, ulnar deviation deformity	Imbalance of wrist flexors and extensors	Tendon transfers, capsulodesis, partial wrist fusion, arthrodesis
Hand	Weak grasp, intrinsic muscle imbalance, clawing	Lower plexus involvement and residual denervation	Tendon transfers, joint stabilization procedures, occupational therapy

CONCLUSION

Obstetric brachial plexus palsy encompasses a spectrum of neurological injuries with variable patterns of spontaneous recovery and long-term functional impairment. Serial clinical examination remains the cornerstone of assessment and guides decisions regarding the need and timing of surgical intervention. Infants with severe plexus involvement or inadequate motor recovery should be identified early and considered for microsurgical reconstruction, including nerve grafting and nerve transfers. Long-term management should also address secondary musculoskeletal deformities, particularly shoulder contractures and glenohumeral dysplasia, through appropriate surveillance, rehabilitation, and reconstructive procedures when indicated. Optimal outcomes require individualized, multidisciplinary care aimed at maximizing neurological recovery and preserving upper-limb function.

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