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Brachial Plexus Injury in the Newborn

Trenna L. Sutcliffe, MD, MSc, FRCPC*

Objectives  After completing this article, readers should be able to:

1. Distinguish among perinatal brachial plexus palsy (PBPP) classification categories based on clinical findings.
2. Identify risk factors for PBPP.
3. Describe other injuries commonly seen in infants who have PBPP.
4. Evaluate clinical features that predict long-term outcome.
5. Assess the need for referral to a specialized clinic.

Abstract

Brachial plexus palsy in the neonate is classified according to the anatomic location and type of injury. Upper plexus lesions are observed most frequently. The overall rate of perinatal brachial plexus palsy (PBPP) has remained stable for the last 3 decades, although risk factors for the injury are well described. The true rate of full recovery after PBPP remains controversial. Knowledge of an infant’s PBPP classification can assist in predicting long-term outcome. A thorough physical examination is essential to make the diagnosis, evaluate for comorbidities, determine the treatment plan, and assess the need for referral. Additional investigations typically are not indicated. Initial management of the infant who has PBPP includes parent counseling, physical or occupational therapy, and clinical observation of the infant. A proportion of affected infants eventually requires surgical intervention or alternative therapeutic approaches, including botulinum toxin injections.

Introduction

Perinatal brachial plexus palsy (PBPP) is a peripheral nerve injury detected in the newborn. It is commonly but not exclusively associated with a traumatic birth and potentially can lead to long-term disability. Physicians who care for newborns should be familiar with the presenting signs, comorbid conditions, and prognosticating features. Historically, PBPP injuries have been classified with the eponyms Erb palsy or Klumpke palsy. More recent classification systems describe lesions based on location and type of injury. Knowledge of these newer PBPP classification categories and the anatomy of the brachial plexus can assist the physician in making decisions in the nursery and communicating accurate information to the family. This article discusses these issues and provides information on long-term outcome and current management approaches important for parent counseling in the neonatal period.

Classification of PBPPs

Understanding the brachial plexus anatomy is important to classify PBPP injuries and evaluate the clinical presentations. The brachial plexus arises from the spinal cord at cervical levels C5, C6, C7, and C8 and thoracic level T1. Brachial plexus nerves run from the neck to the axilla, passing under the clavicle, to provide motor and sensory function to the shoulder, arm, and hand (Fig. 1). Above the clavicle, the nerve roots reconfigure to form three trunks of the plexus: upper trunk from roots C5 and C6, middle trunk from C7, and...
lower trunk from C8 and T1. Nerve root T1 also gives rise to the sympathetic supply to the head and neck.

One classification system describes the location of the lesion (upper lesion versus lower/total lesion) and the type of lesion (avulsion versus rupture) (Table 1). (1) Lesion location is determined by clinical examination. Abnormal postures and loss of movement in specific muscles are associated with specific location patterns. Lesion type is determined with monitoring of the rate of recovery.

For injuries classified as upper lesion, poor shoulder function is ubiquitous and hand function is variable. Accordingly, the phenotype frequently is referred to as the “bad shoulder, good hand” scenario. The injury known as Erb palsy is an example of an upper lesion and involves the upper trunk (roots C5 and C6) and occasionally the middle trunk (root C7). The muscle groups involved in this injury are the shoulder external rotators and abductors, elbow flexors, forearm supinators, and occasionally wrist extensors. This results in a classic phenotype referred to as the “waiter’s tip” posture, with shoulder adduction and internal rotation, elbow extension, forearm pronation, and wrist flexion (Fig. 2). In this injury, in addition to the affected biceps muscle, the elbow sometimes is partially flexed when the triceps muscle is weak or absent. If the injury is isolated to C5 and C6, the elbow may be in full extension without flexion due to a functioning triceps muscle that opposes the nonfunctional brachialis and biceps. Wrist and hand function may be normal in this case.

For injuries classified as lower lesion, shoulder function generally is good, and poor hand function is ubiquitous. The phenotype, therefore, frequently is referred to as the “good shoulder, bad hand” scenario. The injury known as Klumpke palsy is an example of a lower lesion and involves the lower trunk (roots C8 and T1). The involved muscle groups are wrist flexors, finger flexors,
and intrinsic hand muscles. Horner syndrome can be associated with lower lesions if the sympathetic fibers of T1 are injured. This injury pattern is extremely rare in infants.

For injuries classified as total lesion, poor function is observed in the entire arm and hand. The lesion includes injury to all nerve roots in the brachial plexus. The clinical presentation is a flail arm and a hand that may be flail, cupped, or in a “claw” configuration, with metacarpophalangeal joints extended and interphalangeal joints flexed.

Injury type can be determined once injury location is known. Borrero (1) describes injury type as either avulsion or rupture. This classification system resembles the injury severity classification described by Seddon in 1943, who described injury severity as either: 1) a stretch injury of intact nerve fibers, 2) physical disruption of axons or nerve fascicles, or 3) disruption of a nerve trunk or root. (2) Borrero describes avulsion lesions as tearing of the spinal nerve roots, which are considered most severe. Avulsion lesions are true spinal cord injuries and may include injury to motor or sensory nerves. Alternatively, rupture lesions refer to injury at any point along the nerves of the brachial plexus. These are true peripheral lesions. Rupture lesions may include stretching of intact nerve fibers or physical tearing and interruption of the fibers.

Epidemiology

The incidence of PBPP has not decreased over the last 3 decades, which may be due partly to an increase in population birthweights. The rate of PBPP increases with birthweight, as shown by a meta-analysis demonstrating the median incidences to be 0.9 per 1,000 for infants weighing less than 4,000 g, 1.8 per 1,000 for infants weighing 4,000 to 4,500 g, and 2.6 per 1,000 for infants weighing more than 4,500 g. (3) It is estimated that 5,420 new cases of PBPP occur each year in the United States. (4)

The rate of lower plexus lesions has decreased significantly with the decline in vaginal breech births that can result in shoulder hyperabduction. al-Qattan and associates (5) found only 20 cases of lower plexus lesions among 3,308 cases of PBPP. Total plexus lesions reportedly make up 18% of PBPP cases. (6)

Etiology

PBPP is believed to be due most frequently to force or traction on the fetal brachial plexus during vaginal delivery. Shoulder dystocia (impaction of the fetal anterior shoulder against the maternal pubic symphysis) during vaginal delivery can cause stretching of the fetal neck and increase the angle between the head and neck, leading to plexus injury. Evidence to support the hypothesis that PBPP may occur for reasons other than excessive force during vaginal delivery and the presence of shoulder dystocia is accumulating. Cases of PBPP following cesarean section deliveries (7)(8) and intrauterine malposition (9)(10) have been reported. In addition, PBPP of the posterior shoulder has been documented, which may be related to maternal forces during labor and pressure as the fetus passes over the maternal sacral prominence. (11)

Risk Factors

Risk factors for PBPP can be maternal, fetal, and parturitional (Table 2). An estimated 45% of PBPP injuries are associated with shoulder dystocia. (12) Most commonly, shoulder dystocia occurs without warning. Factors associated with shoulder dystocia include: fetal macrosomia, maternal gestational diabetes, maternal short stature, abnormal pelvic anatomy, history of shoulder dystocia, postterm delivery, assisted vaginal delivery, prolonged active phase of first-stage labor, and protracted second-stage labor.
Many believe that the rate of full recovery following PBPP is high. A number of studies have suggested full recovery rates as high as 90%. However, this may be an overestimation, as outlined in the systematic review by Pondaag and associates. (13) Studies measuring long-term outcome after PBPP often are flawed with selection bias, insufficient follow-up time, patients lost to follow-up, or lack of standardized outcome measures. Pondaag and colleagues concluded that the true rate of spontaneous recovery is unknown. However, two studies that best met the inclusion criteria for systematic review demonstrated residual deficits after PBPP in 20% to 30% of cases. (6)(14)

Little is known about predictive factors for recovery. Spontaneous recovery may occur within 48 hours or up to 6 months after birth. Predicting which children will recover spontaneously is extremely difficult early in the course. Lesion classification can help to determine long-term prognosis, with 21% of upper plexus lesions resulting in permanent impairments compared with 66% of total plexus lesions. (6) Therefore, clinical evidence of C8 or T1 injury or the presence of Horner syndrome, which suggests lower or total plexus lesions, is a poor prognostic sign.

Research on the relationship between muscle function in infancy and long-term outcome is emerging. Hand grasp and movement in the forearm are favorable prognostic signs. The importance of biceps function during infancy in predicting outcome has been shown by Waters, (15) who demonstrated that infants who recovered biceps function within the first month after birth had full neurologic recovery and functional use, as measured with the Mallet Scale, at 2-year follow-up. Biceps recovery 2 or 3 months after birth was associated with full recovery in only two of 13 infants; the remainder continued to have deficits at 3 years of age. Recovery of biceps function after 3 months of age was associated with long-term neurologic deficits, and it was rare for such infants to have full recovery. Waters’ results are consistent with others, who have described poor prognosis in infants who have poor recovery of the wrist, fingers, thumb, and biceps at 3 months of age. (16)(17)

The outcomes of PBPP following breech presentation are less studied, although they have been described. (18)(19) Isolated injury to roots C5 and C6 is common in this group. The injury is more severe than that seen in vertex presentation and frequently is associated with complete root avulsion. Bilateral injuries also are more common in infants born via breech deliveries.

**Comorbidities**

Infants who have PBPP are at risk for a number of associated injuries, including clavicular fractures, shoulder girdle fractures, epiphyseal humeral fractures, torticollis, phrenic nerve palsy, facial nerve palsy, and Horner syndrome. If the sympathetic fibers of T1 are involved, an ipsilateral Horner syndrome may be observed.

**Impact on Child Development**

Impaired arm and hand function during infancy due to PBPP may have significant long-term sequelae for motor skills and other developmental domains. Brown and associates (20) suggested that poor nerve regeneration in children who had PBPP may be only one reason for poor motor outcome. By measuring muscle action potentials, these investigators documented impaired motor abilities out of proportion to neurophysiologic test results. Motor difficulties were believed to be due to an inability to recruit available muscle units. These findings are consistent with observations made by Strombeck and colleagues, (21) who reported impaired hand grip and bimanual hand function in children who had C5 to C6 plexus injuries and intact hand innervation. Brown and associates (20) postulated that suboptimal cortical development occurs for the affected hand and arm due to prolonged paralysis during a critical time in an infant’s development. If true, this emphasizes the importance of early intervention for arm and hand mobility.

Other developmental domains also may be affected negatively in children who have PBPP. Typical arm and
Major hand movement during infancy is important not only to attain motor milestones, but also influences overall child development. Considerable evidence demonstrates the importance of hand manipulation and manual exploration in young children for later cognition, perception, and the ability to acquire information from the hands. Alternative diagnoses for poor unilateral arm function should be suspected when forearm weakness and absent hand grasp are detected. Assessment for respiratory compromise due to phrenic nerve palsy and the presence of Horner syndrome (enophthalmos, ptosis, miosis, anhidrosis, and heterochromia) should be included in the examination.

Alternative diagnoses for poor unilateral arm function also need to be evaluated, including humeral and clavicular fractures. Humeral fractures are associated with intact biceps reflex and little active arm movement. Clavicular fractures are associated with an absent Moro reflex; intact biceps reflex; and crepitus, bony irregularity, and possible bruising over the clavicle. A chest radiograph may aid in assessing for these fractures while also helping in the evaluation for diaphragmatic paralysis. Both of these fractures are associated with PBPP in 10% of cases.

Clinical Examination for Diagnosis

PBPP is diagnosed at birth or shortly afterwards and usually is obvious, based on history and physical examination findings. The history may be significant for PBPP risk factors. Physical examination may demonstrate an infant who has a flail arm, inequality in upper arm movement, and absent biceps and Moro reflexes on the affected side. Injury to the lower plexus should be suspected when forearm weakness and absent hand grasp also are detected. Assessment for respiratory compromise due to phrenic nerve palsy and the presence of Horner syndrome (enophthalmos, ptosis, miosis, anhidrosis, and heterochromia) should be included in the examination.

Alternative diagnoses for poor unilateral arm function also need to be evaluated, including humeral and clavicular fractures. Humeral fractures are associated with intact biceps reflex and little active arm movement. Clavicular fractures are associated with an absent Moro reflex; intact biceps reflex; and crepitus, bony irregularity, and possible bruising over the clavicle. A chest radiograph may aid in assessing for these fractures while also helping in the evaluation for diaphragmatic paralysis. Both of these fractures are associated with PBPP in 10% of cases.

Clinical Examination for Making Treatment Decisions

The clinical examination remains the cornerstone in determining the need and urgency for referral. A complete assessment of shoulder, elbow, wrist, thumb, and fingers is needed. Flexion and extension are assessed in the elbow, wrist, thumb, and fingers. Assessment of the shoulder includes anterior flexion, abduction, internal rotation, and external rotation. Ideally, all infants who have palsies that persist after 1 month should be referred to a clinic specializing in PBPP. Infants who have hand paralysis or Horner syndrome should be seen as soon as possible and preferably by 1 month of age. Infants who have essentially normal hand function at birth should be seen within the first 3 postnatal months. The type and severity of injury is determined by monitoring clinical evolution. Many cases of PBPP present with similar clinical pictures immediately after birth, but the extent of injury and decisions around surgical intervention can be determined only after close clinical monitoring over time.

Investigations

Investigations beyond the clinical examination are not essential in determining the need for surgery. However, further investigations may be helpful in determining the extent and location of injury, which may assist the surgeon in planning the operative procedure. Nerve conduction velocity and electromyography testing sometimes are considered. Such tests are not standard procedures for many centers because results can be unreliable and difficult to interpret in young infants and do not predict how many motor fibers will recover over the long term. Imaging with magnetic resonance (MR) or computed tomography (CT) myelography may help the surgeon determine root avulsion and aid in planning the surgical procedure. MR is noninvasive and allows visualization of the plexus, neuromas, and disruptions within the plexus. CT myelography provides visualization of the nerve rootlets exiting the spinal cord. Imaging may be ordered by the surgeon for surgical planning; results do not affect decisions in the nursery.

Management

Initial Handling of the Infant

Management of PBPP begins in the nursery. Families can be given instructions from a therapist familiar with PBPP for active and passive range-of-motion exercises. Such exercises reduce the risk for contractures and muscle atrophy over the long term and can become part of a family’s routine. Intermittent and partial immobilization may be included in the initial management of a flaccid arm if symptoms persist beyond the first week after birth. Such immobilization simply may involve pinning of the arm sleeve to the trunk to relieve the forces of gravity that act on the arm and contribute to further traction on the brachial plexus.

Management also includes discussions with the family. Complete and accurate information should be communicated. It is important that parents be informed of the potential risk for long-term sequelae. Although rapid improvement is observed in most cases, information regarding the injury, natural history, and management plan should be discussed. Physicians need to be cautious...
at early assessments in counseling that full recovery will occur. Close monitoring of affected infants after discharge is required to ensure timely referral if necessary. Assessments every 3 to 4 weeks after birth are suggested. The use of a movement scale to monitor arm and hand motion is recommended. The movement scale from the Hospital for Sick Children in Toronto is used commonly (Table 3). (32) The persistence of symptoms beyond 1 month of age suggests that the injury may require treatment, and affected children should be referred to a specialized clinic.

**Surgery**

Infants who have total or lower plexus injuries require surgery, which frequently occurs after the second month of age. A limited number of infants who have upper plexus injuries require surgery when resolving biceps function is delayed. The timing of surgery in such infants remains controversial, and different centers may follow different protocols. Decisions about proceeding with surgery versus waiting to observe the course begin at 2 months of age.

Surgical procedures for infants who have PBPP include meticulous exploration of the brachial plexus to determine the location and extent of injury and reconstruction of the plexus. Reconstruction may include nerve grafting. Autologous nerve grafts from the sural nerve in the lower leg or cutaneous nerves of the arm are used. Randomized, controlled trials measuring the benefits of surgery have not been performed. McNeely and Drake (33) completed a systematic review that supported the use of surgical Intervention as a valid approach for PBPP based on level III and V evidence. Shenaq and associates (34) provide further review of surgical procedures used in infants who have PBPP.

**Treatment With Botulinum Toxin**

Additional therapies, such as botulinum toxin injections, are being studied in children who have PBPP. (35) It is believed that children learn atypical motor patterns prior to surgical intervention, and such motor patterns rely on activity from the antagonist muscles, which are not affected by the plexus injury. Despite surgery and reinnervation of affected muscles, the compensatory and atypical motor patterns may persist. Botulinum toxin, when injected into overactive antagonist muscles, allows for a transient period of motor training and strengthening of the affected muscle groups.

**Conclusion**

PBPP injuries in infants are not common but must be recognized and treated appropriately. For infants born with asymmetric arm and hand function, the clinical examination is used to determine diagnosis and PBPP classification. Such information is important for counseling of parents, predicting prognosis, and determining the management plan.

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# NeoReviews Quiz

1. Perinatal brachial plexus palsy (PBPP) can be classified according to the location of the nerve injury. Each type of nerve injury has a specific pattern of involvement of motor and sensory function of the upper limb as well as associated neurologic abnormalities. Of the following, Horner syndrome (ptosis, miosis, anhidrosis, enophthalmos, and heterochromia) that involves sympathetic nerve fibers is most likely to represent brachial plexus injury at the level of:

   A. Cervical nerve root 5.
   B. Cervical nerve root 6.
   C. Cervical nerve root 7.
   D. Cervical nerve root 8.
   E. Thoracic nerve root 1.

2. Risk factors for PBPP can be maternal, fetal, or parturitional in origin. Of the following, the most common risk factor for PBPP is:

   A. Assisted forceps delivery.
   B. Fetal macrosomia.
   C. Maternal diabetes mellitus.
   D. Shoulder dystocia.
   E. Uterine abnormality.

3. A term newborn has “waiter’s tip” posture indicative of Erb palsy following a difficult vaginal delivery. You are discussing with medical students the epidemiology and outcome of PBPP. Of the following, the most accurate statement regarding PBPP is that:

   A. Initial arm and hand involvement in PBPP may affect subsequent cortical development even after full recovery of innervation.
   B. Lower brachial plexus injury is much more common than upper or total brachial plexus injury.
   C. Residual neurologic deficits following PBPP occur in fewer than 10% of cases.
   D. Spontaneous neurologic recovery following PBPP generally is complete within 3 months after birth.
   E. The incidence of PBPP has decreased over the last 3 decades with improvements in perinatal care.

4. Neurologic recovery from PBPP can be ascertained by examination of muscle function of the upper limb within the first 3 months after birth. Of the following, the most favorable sign for full neurologic recovery from PBPP, as described by Waters, is the recovery of:

   A. Biceps function.
   B. Finger movements.
   C. Forearm supination.
   D. Hand grasp.
   E. Shoulder abduction.

5. The need for surgical reconstruction of the brachial plexus is influenced by the extent of nerve injury and the recovery of neurologic function over time after birth. Of the following, the most essential determinant of the need for surgery in PBPP is:

   A. Clinical examination.
   B. Computed tomography myelography.
   C. Electromyography.
   D. Magnetic resonance imaging.
   E. Nerve conduction velocity.