Obstetric Brachial Plexus Injuries: Evaluation and Management

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Abstract

Most infants with brachial plexus birth palsy who show signs of recovery in the first 2 months of life will subsequently have normal function. However, infants who do not recover in the first 3 months of life have a considerable risk of long-term limited strength and range of motion. As the delay in recovery extends from 3 months to beyond 6 months, this risk increases proportionately. The presence of a total plexus lesion, a partial plexus lesion with loss at C5–C7, or Horner's syndrome carries a worse prognosis. Microsurgery is indicated for failure of return of function by 3 to 6 months. The exact timing of intervention is still open to debate. With microsurgical reconstruction, there is improvement in outcome in a high percentage of patients. However, the neural lesion is too severe and complex for present methods of reconstruction to restore normal function. Secondary correction of shoulder dysfunction with either latissimus dorsi–teres major tendon transfer or humeral derotation osteotomy is clearly beneficial for patients with chronic brachial plexopathy, as is reconstruction of supination forearm contracture with biceps rerouting transfer and/or forearm osteotomy. Reconstruction of the hand is also indicated for the patient with chronic disability. All of these procedures improve, but do not completely normalize, function.

J Am Acad Orthop Surg 1997;5:205-214

Brachial plexus birth palsy occurs in 0.1% to 0.4% of live births.[1,2] Most infants with brachial plexus birth palsy who show signs of recovery in the first 2 months of life will subsequently have normal function. However, infants who do not recover in the first 3 months of life have a considerable risk of long-term limited strength and range of motion. As the delay in recovery extends from 3 months to beyond 6 months, this risk increases proportionately, and microsurgery may be indicated.

Central to the controversy of treatment of brachial plexus birth palsies is predicting the natural history of recovery of the neural lesion. In general, this depends on the type of nerve lesion (stretch, rupture, or avulsion), the level of injury (partial [i.e., upper, lower, or mixed] or total), and the severity of the injury (Sunderland grades I through V). Many researchers have attempted to address the predictive value of physical examination, plain and interventional radiography, and electrodiagnostic testing in determining the severity of injury. However, it has been difficult to predict long-term recovery on the basis of information obtained in early infancy. At present, the decision to allow for spontaneous reinnervation and muscle recovery or to undertake microsurgical reconstruction of the injured plexus remains dependent on the physical findings. The purpose of this article is to review the present knowledge of the natural history of brachial plexus birth palsies, the indications for microsurgical intervention during infancy, and the indicators for tendon transfers and osteotomies in the child with chronic plexopathy.

Etiology

Perinatal risk factors include large size for gestational age, multiparous pregnancy, prolonged labor, and difficult delivery. Fetal distress may contribute to relative hypotonia and less protection of the plexus due to

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stretch injury during delivery. Mechanically, shoulder dystocia in vertex deliveries and difficult arm or head extraction in breech deliveries increase the risk of neural injury.[3]

Brachial plexus birth palsy usually involves the upper trunk (C5 and C6 Erb's palsy), although there may be an additional injury to C7. Less often, the entire plexus (C5–T1) is involved.[4] In rare instances, the lower trunk (C8-T1) is most seriously involved. Obstetric injuries to the upper trunk are generally postganglionic. The exception is upper trunk lesions after breech delivery, which tend to be preganglionic injuries of C5-C6. When the lower plexus is involved, it is usually a preganglionic injury of C8-T1.

Anatomy

Essential to any discussion regarding the natural history and treatment of a brachial plexus lesion is a thorough understanding of the anatomy (Fig. 1). The brachial plexus most commonly (77% of cases) receives contributions contiguously from the anterior spinal nerve roots of C5 to T1. Prefixed cords (22% of cases) receive an additional contribution from C4. The much less common postfixed cords (1% of cases) receive a contribution from T2.[5]

The C5 and C6 nerve roots join to form the upper trunk; the C7 nerve root continues as the middle trunk; and the C8 and T1 nerve roots combine to form the lower trunk. Each trunk bifurcates into anterior and posterior divisions. The posterior divisions of all three trunks make up the posterior cord. The anterior divisions of the upper and middle trunks form the lateral cord. Finally, the anterior division of the lower trunk forms the medial cord. The major nerves of the upper extremity are terminal branches from the cords, with the ulnar nerve arising from the medial cord, the radial and axillary nerves from the posterior cord, the musculocutaneous nerve from the lateral cord, and the median nerve from branches of the medial and lateral cords.

To predict outcome, it is important to determine whether the lesion is preganglionic or postganglionic. The ganglion is adjacent to the spinal cord and contains the

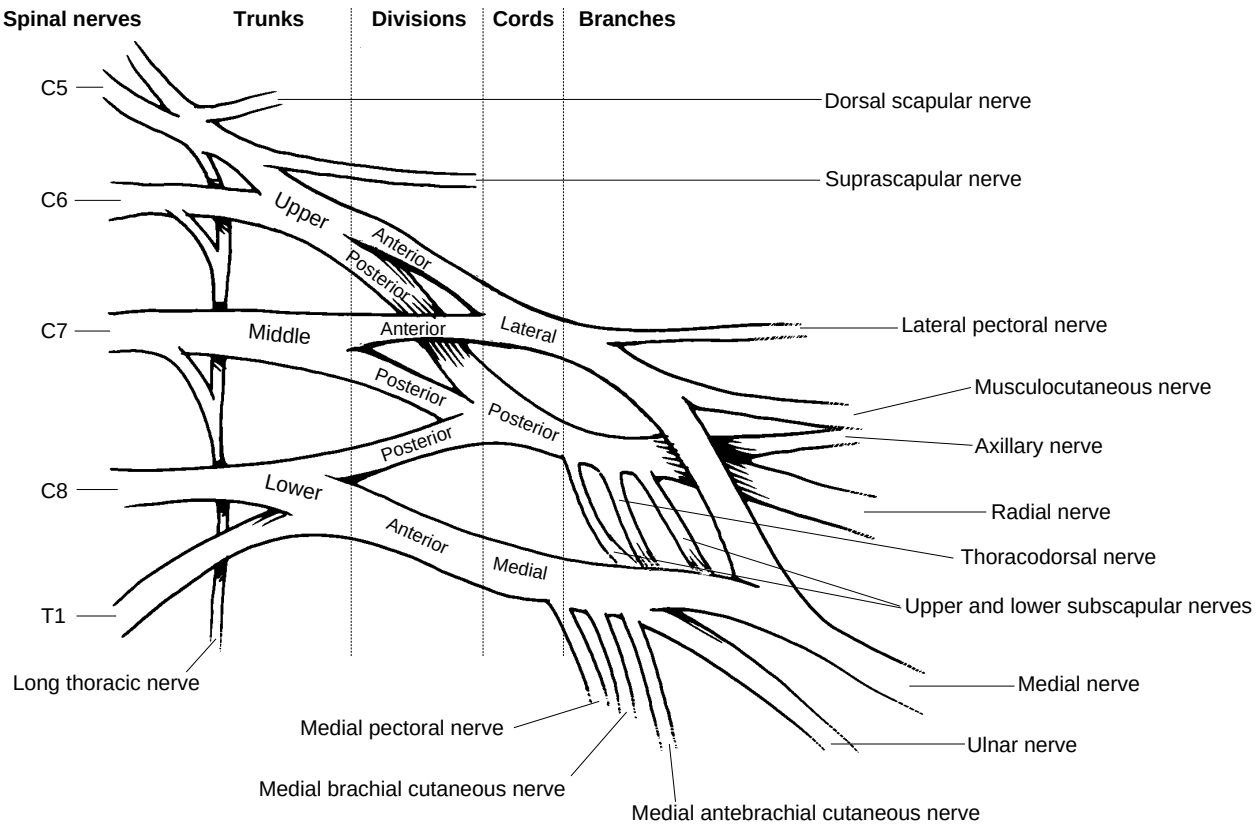

Fig. 1 Structures of the brachial plexus.

sensory cell body. The motor cell body is in the spinal cord. Preganglionic lesions are avulsions from the cord, which will not spontaneously recover motor function. By assessing the function of several nerves that arise close to the ganglion, careful physical examination can determine the level of the lesion. Specifically, the presence of Horner's syndrome (sympathetic chain), an elevated hemidiaphragm (phrenic nerve), or a winged scapula (long thoracic nerve) raises serious concern about a preganglionic lesion, as does the absence of rhomboid (subscapular nerve), rotator cuff (suprascapular nerve), and latissimus dorsi (thoracodorsal nerve) function.

Classification Systems

A modification of the Mallet classification system[6] can be used to define the recovery of upper-trunk function in infants. It has five separate categories for global abduction, global external rotation and hand-to-neck, hand-to-mouth, and hand-to-sacrum function. Grading is on a scale of 0 to 5, with 5 being normal and 0 being no muscle contraction. Grades II through IV are illustrated for each category in Figure 2. Preliminary studies on natural history, microsurgical plexus reconstruction, and secondary reconstructive shoulder surgery have used the Mallet classification. Unfortunately, it does not measure individual motor strength or separate joint function or provide a comparative scoring system. Its usefulness is primarily in upper-trunk assessment of infants. It cannot be used to assess forearm, wrist, and hand function.

Michelow et al[7] proposed a scoring system for surgical indications for nerve reconstruction of the infantile brachial plexus. Scoring is based on return of (1) shoulder abduction, (2) elbow flexion, (3) extension of the wrist, (4) extension of the fingers, and (5) extension of the thumb. A score of 0 to 2 is given for each of those five motor functions. (A score of 0 represents no function; a score of 1, partial function; a score of 2, normal function.) A total score of less than 3.5 beyond 3 months of life is an indication for microsurgery.

There are several other proposed systems of measuring function and outcome, but none has been validated or is widely accepted. The absence of a uniform, accepted measure of outcome makes comparison of results of natural history, microsurgery, and reconstructive surgery studies difficult. Obviously, this is essential for defining the indications and results of surgical procedures.

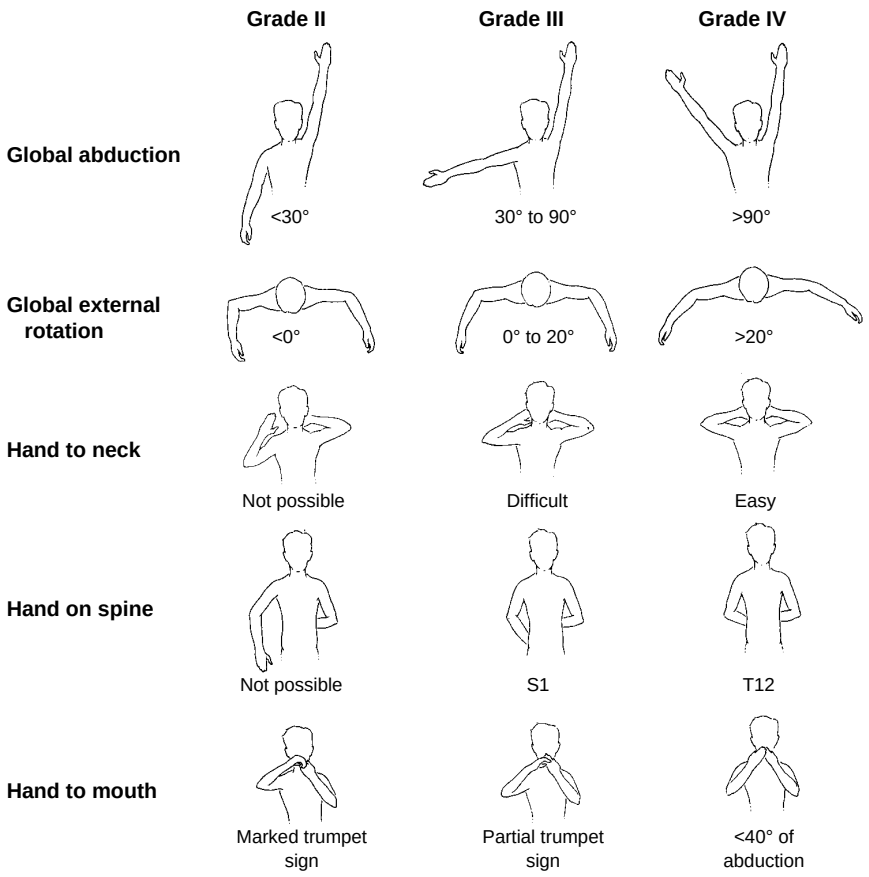

Fig. 2 Modification of the Mallet classification for assessing upper trunk function in young children. Grade I is no function, and grade V is normal function. Grades II, III, and IV are depicted for each category.

Diagnosis

The most important reason to define the level and severity of neural injury is to predict the potential for spontaneous recovery. Physical examination of the infant is the most reliable method of assessing the severity of neural injury. Spontaneous shoulder,

elbow, wrist, and finger motion are evaluated. Provocative testing by stimulating neonatal reflexes (Moro, asymmetric tonic neck, and Votja reflexes) to induce elbow flexion and wrist and digital extension is used. The presence or absence of Horner's syndrome is recorded. Serial examinations are necessary over the first 3 to 6 months of life.

Gilbert and Tassin[6] first pointed out the importance of monitoring the return of biceps function as an indicator of brachial plexus recovery. In their original work, they found that if normal biceps function (as determined with the modified Mallet classification) failed to return by 3 months of age, the outcome at 2 years of age was not normal (Fig. 2). This was generally confirmed by subsequent studies.[1,7-9] However, Michelow et al[7] found that return of biceps function at 3 months had a 12% rate of failure in detecting poor outcome. By combining return of elbow flexion with return of wrist extension, digital extension, thumb extension, and shoulder abduction, they were able to decrease their error rate to 5%. In all studies, the presence of total plexus involvement, C5–C7 involvement, and/or Horner's syndrome meant a poorer prognosis for spontaneous recovery.

Invasive radiologic studies with myelography, combined myelography–computed tomography (CT), and magnetic resonance (MR) imaging have been used in an attempt to distinguish between avulsions and extraforaminal ruptures. Kawai et al[10] compared the findings obtained with all three techniques with the operative findings in infants. Myelography had an 84% true-positive rate, a 4% false-positive rate, and a 12% false-negative rate. The addition of CT to myelography increased the true-positive rate to 94%. The presence of small diverticula was only 60% accurate for an avulsion. However, the presence of large diverticula or frank meningoceles was diagnostic. Magnetic resonance imaging had a true-positive rate similar to that of myelographic CT studies and also had the additional benefit of allowing more distal imaging of the plexus. These findings agreed with those of similar studies in adults with traumatic brachial plexus lesions.

Electrodiagnostic studies with electromyography and measurement of nerve-conduction velocities have also been used in an attempt to improve the accuracy of evaluating the severity of the neural lesion. Unfortunately, the presence of motor activity in a given muscle has not been accurate in predicting an acceptable level of motor recovery. The absence of reinnervation at 3 months is indicative of an avulsion, but the presence of reinnervation seems only to confuse the clinical picture.[11-14]

At present, most clinicians rely on clinical examination for determination of the level and severity of the lesion. The rate and extent of spontaneous recovery of elbow flexion, shoulder abduction, and extension of the wrist, fingers, and thumb in the first 3 to 6 months of life help predict outcome.[7] The presence of Horner's syndrome indicates a poorer prognosis.[4,6,7,9,11,12,14]

Nonsurgical Treatment

During the period of observation for neural recovery, passive range of motion of all joints should be maintained. This often requires the assistance of a physical therapist. In particular, glenohumeral motion should be maintained by passive therapy while stabilizing the scapulothoracic joint. This may prevent the development of glenohumeral capsular tightness or lessen its severity. Votja techniques attempt to induce the normal infantile reflexes of elbow flexion and wrist and digital extension with specific stimulation. It is postulated that this stimulates reinnervation, although supportive data are limited. Stimulation of the limb for sensory reeducation has been advocated.[11,12]

Microsurgery

Indications and Timing

Without question, the role and timing of microsurgery are the most controversial issues in the treatment of infants with brachial plexus injuries. At present, microsurgery is performed more commonly in Europe, South Africa, and Asia[4,12,13] than it is in North America. The original interventions (at the turn of the 20th century) were resection of the neuroma and direct repair. Early direct repair is currently performed only in Finland.

The present recommendations for care are transection of the neuroma and sural nerve grafting for extraforaminal ruptures. In the treatment of upper-trunk ruptures, grafts are performed from the C5 and C6 roots to the musculocutaneous nerve or lateral cord, suprascapular nerve, and upper-trunk posterior division to the posterior cord. In the case of avulsions, nerve transfers are performed with the use of the thoracic intercostals and/or a branch of the spinal accessory nerve beyond the point at which it innervates the trapezius. For the treatment of total avulsions, Gilbert[14] advocates prioritizing microsurgical reconstruction of the median and ulnar nerves to reinnervate the hand.

Unlike adults, infants with brachial plexopathy may have the potential to regain hand function after nerve grafting or transfers.

Although there is an ongoing debate about the timing of microsurgical intervention, the criteria for use in clinical practice have been established. Brachial plexus exploration followed by reconstruction with sural nerve grafts is indicated (1) for infants with total plexopathy, Horner's syndrome, and no return of biceps function at 3 months or a Toronto score less than 3.5; and (2) for infants with upper-trunk plexopathy, no return of biceps function at 3 to 6 months, and a Toronto score less than 3.5[4,6,7,9,11,12,15] (Fig. 3). Reconstruction is usually performed between 3 and 6 months of age, although the range in various studies extends from 1 to 24 months.

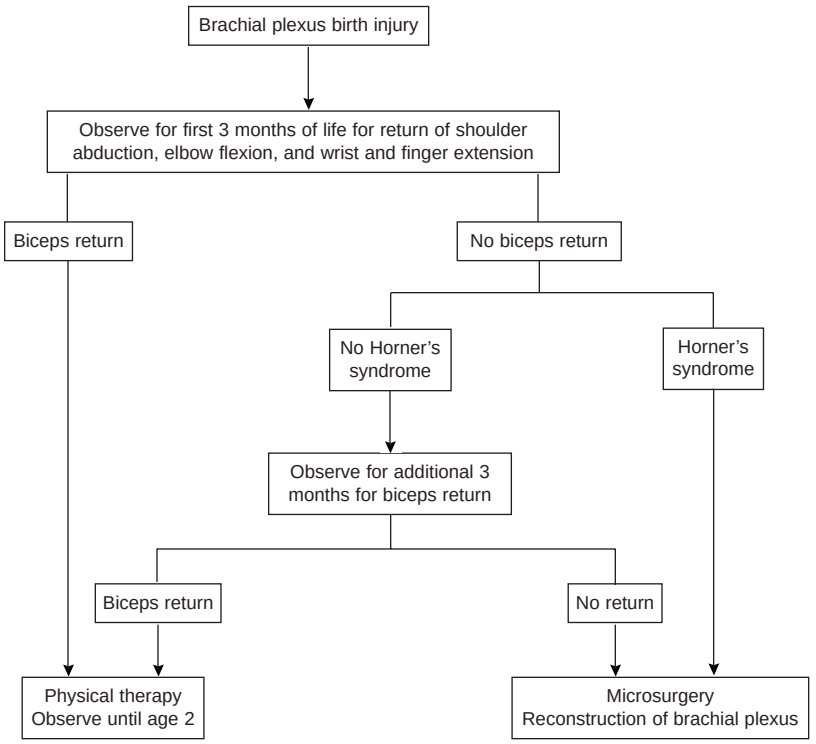

Fig. 3 Algorithm for treatment of infants with incomplete recovery of neural function.

The problem with reviewing the results of microsurgery is that very few patients have had long-term follow-up and microsurgery has usually been combined with other methods of treatment. Gilbert and Tassin's original study[6] compared the data on cases in which microsurgery was performed with the data on cases in which spontaneous recovery occurred. In the cases of C5-C6 lesions, 100% of the infants treated nonoperatively had class III recovery (modified Mallet classification). Of the infants treated microsurgically, 37% had class III recovery, and 63% had class IV recovery. In the cases of C5–C7 lesions, 30% of the infants in the nonsurgical group had class II recovery, and 70% had class III recovery. Of the infants treated with microsurgery, 35% had class II recovery; 42%, class III; and 22%, class IV.

More recently, Gilbert and Whitaker[4] reported the results of reconstruction at 2-year follow-up in terms of modified Mallet scores for abduction. Of the infants with C5-C6 reconstructions, 81% had class III, IV, or V recovery. Of the infants who underwent total plexus reconstruction, 64% had class III or IV recovery.[14] At 5-year follow-up, after performance of secondary shoulder reconstructions, these results improved such that 70% of the infants with C5-C6 reconstructions had Mallet class IV or V abduction recovery.[14] The results were similar for total plexopathy reconstructions, in which nerve grafting for the hand was prioritized. At 2-year follow-up, only 25% of patients had grade III or IV shoulder function; 70% had grade III, IV, or V elbow function; and 35% had grade III or IV hand function. With the addition of secondary tendon transfers and stabilization procedures, 77% had good shoulder function, and 75% had good hand function at 6-year follow-up.[14] Gilbert[14] maintains that microsurgery not only improves function in selected patients over what would be expected from the natural history but also increases the possibilities for secondary tendon transfers.

These results are comparable with the limited natural history data. Benson et al[8] examined the data on 142 patients to assess the natural history of brachial plexopathy. Seventy-one patients had full recovery by 6 weeks. The other 71 were older than 6 weeks when biceps function returned. At final follow-up, 67% had excellent shoulder function; the results were good in another 12%, fair in 5%, and poor in 10%.

Waters[9] addressed the same issue prospectively and found that

of 49 infants with no biceps recovery at 3 months, 42 recovered biceps function by 6 months. In infants with biceps recovery between 3 and 6 months, there was a progressive decrease in Mallet grades for abduction, external rotation, and hand-to-mouth and hand-to-neck activities with each successive month. None of the children with biceps recovery after 3 months of age had normal function by Mallet criteria.

Like microsurgery, secondary shoulder tendon transfers and osteotomies significantly improve function in patients with residual deficits. In a subgroup of 20 patients with shoulder reconstructions,[13] there was a significant ($P<0.0005$) improvement for all Mallet classes. Therein lies the basis for another of the present controversies. Clearly, patients with no biceps function by 6 months or a Toronto score less than 3.5 have a poor prognosis and will benefit from microsurgical reconstruction of the plexus.[4,6,8,9] But how different are patients who undergo microsurgery at 3 months from those who recover biceps function between 3 and 6 months and undergo secondary reconstructions? As Gilbert and Whitaker's microsurgery results[4] include secondary procedures, this controversy is presently unresolved. Although there are many believers in the importance of microsurgical intervention at 3 months, we know of no current studies randomizing entry to treatment protocols that will answer these questions.

Technique

Standard exposure of the brachial plexus is performed with a Z-plasty skin incision extending from adjacent to the mastoid process, parallel to the sternoclaviculomastoid muscle, and across the clavicle and descending into the axilla. Supraclavicular exposure of the roots and trunks is performed between the anterior and middle scalene muscles. In infants, the clavicle is not osteotomized, but rather is retracted.

The major nerves are identified distally after appropriate takedown of the pectoralis major and minor muscles. Proximally, the extent of injury is defined as an avulsion or extraforaminal rupture for each nerve root. In the presence of extraforaminal rupture, proximal transection of the neuroma is performed. This is generally at the C5-C6 root or the upper-trunk level. The viability of the proximal nerve is confirmed by (1) microscopic inspection of the fascicles, (2) histologic examination of the myelin fibers, and (3) peripheral-to-central somatosensory evoked potentials or central-to-peripheral motor stimulation. Sural nerve grafts from the lower portions of both legs are placed from the proximal C5 and C6 roots to the lateral cord or musculocutaneous nerve, the suprascapular nerve, and the posterior division of the upper trunk to the posterior cord.[4,11,12,15]

In the presence of upper-root avulsions, nerve transfers are necessary. The spinal accessory nerve beyond the point at which it supplies the trapezius is transferred to the suprascapular nerve. Thoracic intercostal nerves (T2–T4) are used for repair of the musculocutaneous nerve or lateral cord and the posterior cord.

In the presence of a total plexopathy with a combination of C5-C6 rupture and distal avulsion, the hand is prioritized. The C5 and C6 nerve roots are used for grafting to the median nerve and the medial cord or ulnar nerve. Transfers of spinal accessory and intercostal nerves are used for the suprascapular nerve and the posterior and lateral cords.

Secondary Reconstruction of Internal Rotation Contractures of the Shoulder

Open Reduction for Posterior Glenohumeral Dislocation

Treatment of posterior glenohumeral dislocation varies according to the age of the child at diagnosis and the extent of glenoid deformity (Fig. 4).

In rare instances, infants less than 1 year of age have a posterior dislocation of the glenohumeral joint. There is limitation of external rotation, and the humeral head is palpably dislocated posteriorly. Ultrasonography, arthrography, CT, or MR imaging can be used to confirm the diagnosis (Fig. 5).

If dislocation is detected in infancy, open reduction and capsulorrhaphy are indicated. There must be an anatomic glenoid for stable reduction of the humeral head. Simultaneous anterior and posterior approaches to the glenohumeral joint are used. An anterior release and posterior capsulorrhaphy are performed as outlined by Troum et al.[16] Whether a simultaneous latissimus dorsi transfer should be performed is unclear. Postoperative immobilization in a spica cast is maintained for 4 weeks. Passive and active exercises for maintaining

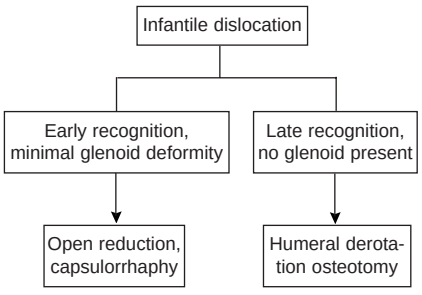

Fig. 4 Algorithm for treatment of patients with infantile dislocation.

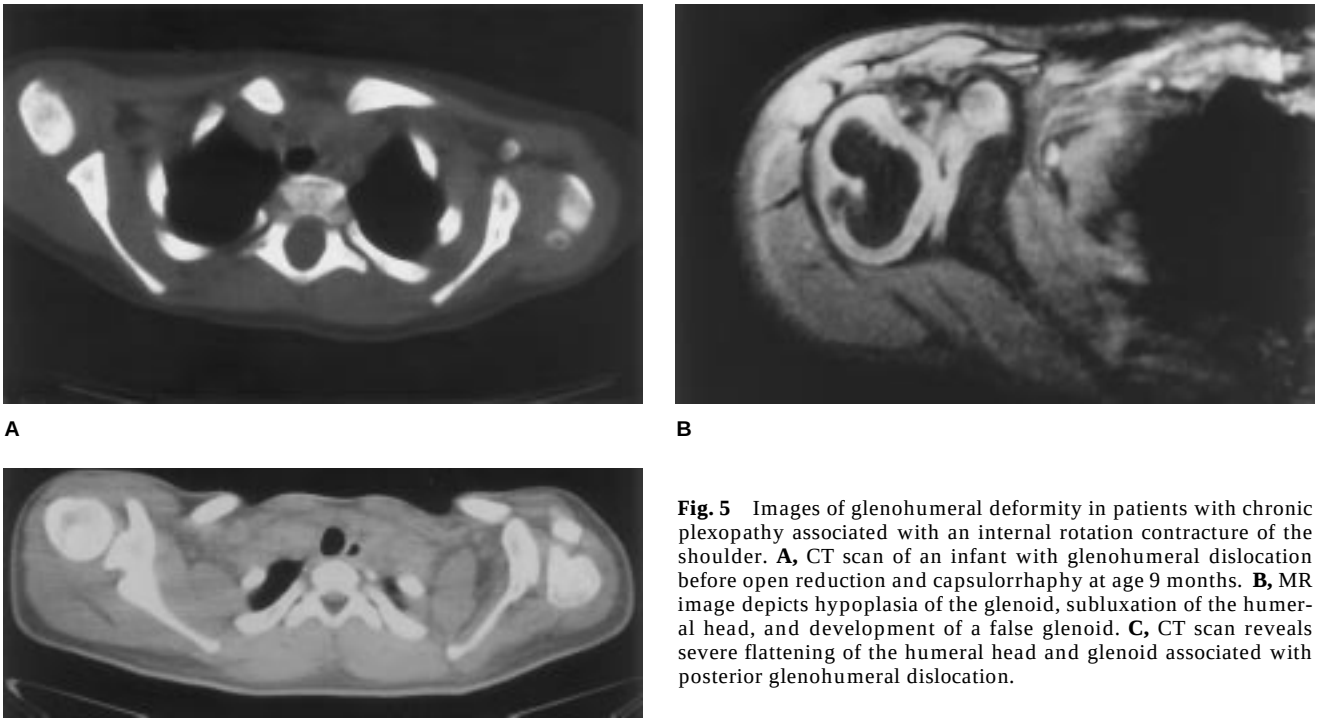

Fig. 5 Images of glenohumeral deformity in patients with chronic plexopathy associated with an internal rotation contracture of the shoulder. **A,** CT scan of an infant with glenohumeral dislocation before open reduction and capsulorrhaphy at age 9 months. **B,** MR image depicts hypoplasia of the glenoid, subluxation of the humeral head, and development of a false glenoid. **C,** CT scan reveals severe flattening of the humeral head and glenoid associated with posterior glenohumeral dislocation.

range of motion are started immediately thereafter.

If posterior glenohumeral dislocation is detected beyond infancy and there is marked glenoid deficiency, a humeral derotation osteotomy is a more appropriate means of treatment than open reduction and capsulorrhaphy.

Tendon Transfers and Osteotomies

Reconstructive surgery is clearly beneficial for children with chronic plexopathy, an internal rotation contracture, and external rotation weakness of the shoulder[13,14,17,18] (Fig. 6). The long-standing muscle imbalance from an upper-trunk lesion with intact adductors and internal rotators and weak abductors and external rotators leads to progressive glenohumeral deformity.[13] Early release of the subscapularis muscle origin[19] at 1 year of age may improve passive external rotation and lessen the risk of progressive glenohumeral subluxation in infants with a contracture that is unresponsive to physical therapy. Anterior release of the pectoralis major tendon and transfer of the latissimus dorsi and teres major muscles is appropriate for patients with minimal glenohumeral deformity and a debilitating contracture. Humeral derotation osteotomy is best for patients with an internal contracture and advanced glenohumeral deformity.[13,18]

Subscapularis Release

Release of the origin of the subscapularis muscle[19] may be indicated when intensive physical therapy fails to improve an internal rotation shoulder contracture in an infant. Therapy should be directed at increasing the humeroscapular angle in external rotation by stabilizing the scapulothoracic joint.[11,12] A subscapularis release may be indicated if there is less than 30 degrees of external rotation in adduction by 1 year of age.

Carlioz and Brahimi[19] have outlined a procedure that exposes the subscapularis origin posteriorly along the medial border of the scapula. A muscle slide is performed to improve passive external rotation to more than 30 degrees. Postoperative immobilization in a shoulder spica cast is maintained for 3 to 4 weeks.

Anterior Release of Pectoralis Major and Latissimus Dorsi–Teres Major Transfer

Anterior release of the pectoralis major insertion and transfer of the latissimus dorsi and teres major muscles to the rotator cuff is indicated for patients with (1) persistent internal rotation contracture,

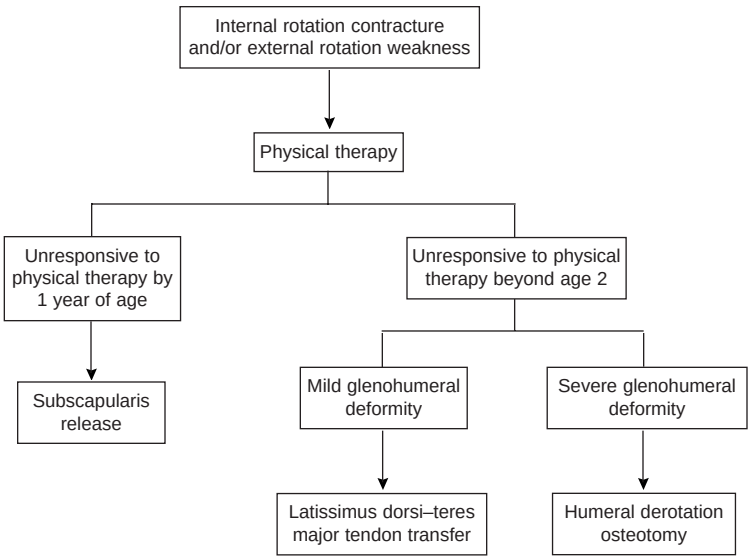

Fig. 6 Algorithm for treatment of patients with disabling internal rotation contractures.

(2) external rotation weakness, (3) limited abduction, and (4) posterior subluxation of the glenohumeral joint without glenoid deformity (Fig. 6). Transfer can be successfully performed between the ages of 2 to 7 years, depending on the severity of glenohumeral deformity.[13,17] In rare instances, the transfer may be of insufficient strength to provide effective abduction and external rotation; a supplemental derotation osteotomy of the humerus or shoulder arthrodesis may be necessary.

Hoffer et al[17] have outlined an approach through an anterior incision in which the pectoralis major tendon is lengthened at its humeral insertion. Through a posterior incision, the latissimus dorsi–teres major insertion is then transferred to the greater tuberosity of the humerus. Others have modified the approach of Hoffer et al by leaving the pectoralis major intact, releasing the teres major, and transferring only the latissimus dorsi muscle. Postoperative immobilization in a shoulder spica cast in abduction and external rotation is maintained for 4 to 6 weeks, followed by physical therapy for transfer education.

Humeral Derotation Osteotomy

The indications for humeral derotation osteotomy are the same as those for latissimus dorsi–teres major tendon transfer except that patients are selected for osteotomy if there is more severe glenohumeral deformity with flattening of the glenoid and humeral head. This presentation is most common in adolescents.[11,13,18]

Anterior humeral exposure is performed in the distal aspect of the deltopectoral interval. The pectoralis major and deltoid muscle insertions are identified. Subperiosteal dissection is then performed proximal to the deltoid muscle insertion. The radial nerve is protected with this exposure, as it crosses posterior to the deltoid at this level. The osteotomy is performed proximal to the deltoid insertion in transverse fashion.

The distal humerus is positioned in 30 degrees of external rotation and is then stabilized with a four- to six-hole plate across the osteotomy. The degree of postoperative immobilization is dependent on the age of the patient and the stability of internal fixation. This can range from a shoulder spica cast for a young child to a sling and swathe for an adolescent. For a child, therapy is begun as soon as the osteotomy has healed; for an adolescent, therapy is begun when hardware provides sufficient stability.[11,18]

Secondary Reconstruction of Supination Contractures of the Forearm

It is common to have an elbow flexion and forearm supination contracture in the rare patients with residual C8-T1 neuropathy and recovery of C5-C6 function. These children have intact shoulder abduction, elbow flexion, and forearm supination and may have active wrist dorsiflexion and digital flexion. By surgically correcting the supination posture and repositioning the forearm into 20 degrees of pronation, the affected limb becomes a better assist (Fig. 7). When the posture of dorsiflexion of the wrist is corrected, gravity assists palmar flexion. The palmar flexion of the wrist aids digital extension by tenodesis.

Zancolli[20] advocated rerouting the biceps insertion to convert the biceps from a forearm supinator to a pronator. In the presence of a supination contracture, simultaneous interosseous membrane release was recommended. However, only 50% of his patients maintained the correction. Instead, Manske et al[21] recommend osteotoclasis of the radius and ulna. Most often, some variant of forearm osteotomy rather than soft-tissue release is performed for patients with a contracture.

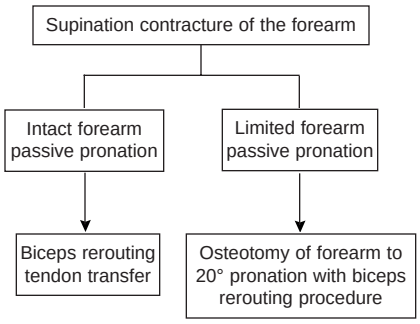

Fig. 7 Algorithm for treatment of supination contracture associated with predominant C7–T1 dysfunction. Intact wrist dorsiflexion is important preoperatively.

Biceps Tendon Transfer

Rerouting of the biceps tendon insertion to convert its muscle action from supination to pronation is indicated for patients with elbow flexion, forearm supination, and wrist dorsiflexion posturing from residual C7–T1 weakness and C5-C6 recovery (Fig. 7). Ideally, patients will have antigravity wrist dorsiflexion strength for effective postoperative wrist tenodesis to aid finger flexion. In the absence of at least 60 degrees of passive pronation, a simultaneous or sequential forearm osteotomy should be performed.[11,18,20]

The biceps rerouting transfer follows the procedure outlined by Zancolli.[20] A Z-plasty skin incision is made in the cubital fossa. The biceps tendon insertion is exposed laterally to protect the median nerve and the brachial artery. The tendon is lengthened in Z fashion. The distal insertion is rerouted around the radial neck while protecting the posterior interosseous nerve and is sutured to itself to act as a pronator rather than a supinator. Protective cast immobilization in 90 degrees of elbow flexion and 20 degrees of forearm pronation is maintained for 4 to 6 weeks. Active range-of-motion and strengthening exercises are begun thereafter.

Forearm Osteotomy

In the absence of forearm passive pronation, an osteotomy of the radius alone or of both the radius and the ulna is performed to correct the supination deformity. Manske et al[21] recommend a two-stage osteoclasis technique. A single-stage technique can be used if intramedullary fixation of the radius and ulna is accomplished before osteotomy. In the case of a less severe deformity, a distal radial osteotomy alone with internal plate fixation can be used. A simultaneous biceps rerouting procedure may lessen the risk of recurrent deformity with growth.

Summary

Most infants with brachial plexus birth palsy who show signs of recovery in the first 2 months of life should subsequently have normal function. However, infants who fail to recover in the first 3 months of life have a considerable risk of long-term limited function, especially about the shoulder. As the delay in recovery extends from 3 months to beyond 6 months, this risk increases proportionately. The presence of a total plexus lesion, a partial plexus lesion with C5–C7 loss, or Horner's syndrome all carry a worse prognosis.

Microsurgery may be indicated if function does not return in the first 3 to 6 months of life. The exact timing of intervention is still open to debate. With microsurgical reconstruction, there is improvement in outcome for a high percentage of patients. However, the neural lesion is too severe and complex for our present methods of reconstruction to result in normal function. Secondary reconstruction of a dysfunctional shoulder by means of a latissimus dorsi–teres major tendon transfer or humeral derotation osteotomy is clearly beneficial to patients with chronic brachial plexopathy, as is secondary reconstruction of a forearm supination contracture by means of biceps rerouting transfer and/or forearm osteotomy. Reconstruction of the hand is also indicated for patients with chronic disability. All of these procedures should improve, but will not completely normalize, function.

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