Obstetrical Brachial Plexus Palsy

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Summary: In this article, the authors review their approach to evaluation, operative management, and reconstructive technique. Brachial plexus injuries in the newborn are usually managed nonoperatively. The timing and indications for primary surgery vary significantly between institutions. The motor examination is used to determine which infants would benefit from operative management. Patients are selected based on established criteria, such as the Toronto Test Score, applied at age 3 months. However, some cases are initially less clear, and we may recommend delaying operative management until age 6 months or as late as age 9 months if the child fails the cookie test. Neuroma excision, sural nerve grafting, and nerve transfers are performed when indicated by clinical motor examination. The use of selective motor nerve transfers, either in combination with nerve grafting or alone, has allowed nerve coaptations to be performed closer to the neuromuscular junction, which may further improve regeneration. Children undergoing primary surgery experience low rates of perioperative morbidity, and they experience gains in motor function until 3 or 4 years postoperatively, at which point recovery stabilizes. (*Plast. Reconstr. Surg.* 124 (Suppl.): 144e, 2009.)

HISTORICAL BACKGROUND

Although brachial plexus injuries were described in antiquity, the first description of obstetrical brachial plexus paralysis in the modern era was by William Smellie in 1768.[1] He noted bilateral arm paralysis in a newborn that he had delivered by applying traction to the head with forceps. Narakas proposed an injury severity classification system based on his experience with over 1000 cases of obstetrical brachial plexus palsy.[2–4] A type I injury heals spontaneously within weeks. A type II injury does not recover full shoulder function, but elbow function is adequate. Tendon transfers are sometimes needed for wrist and digital extension. A type III injury is composed of an upper trunk injury with avulsion of the C7 spinal root plus a more mild injury to the lower trunk. The clinical presentation at birth may include a Horner syndrome, which resolves. A type IV injury presents like a type III injury but the Horner syndrome persists, indicating likely avulsion of C8-T1. There may be partial recovery of C5-6. A type V injury involves C5-T1. The Horner syndrome does not resolve. He considered paralysis of both the rhomboid and serratus anterior muscles a particularly poor prognostic sign for spontaneous recovery.

Gilbert subsequently popularized the most common indication for operative management in obstetrical brachial plexus paralysis. He advocated neuroma excision and interpositional nerve grafting in cases where no biceps function was present at age 3 months (see later under Indications).[5–7]

EPIDEMIOLOGY

The incidence of obstetrical brachial plexus palsy is between 0.5 and 3 injuries per 1000 live births.[8–12] The injury is usually a result of increasing the neck-shoulder angle, producing longitudinal stretching forces that exceed the nerves' tensile stress tolerance. It remains controversial to what extent uterine expulsive forces or external manipulations (or both) produce the palsy, but the majority of authors agree that the lesion is the result of the birth process. Compression injury can be produced by hematoma, associated fractures of the humerus or clavicle, or instrumentation during delivery. A mixed type of injury is often produced, ranging from neurapraxic injury to complete rupture or root avulsion from the spinal

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cord. Early operative management, consisting of nerve grafts and nerve transfers, has been accepted as the standard of care for total plexus injuries. The indications for and timing of surgical intervention in cases of subtotal injury remain without consensus. There are limited multidisciplinary brachial plexus centers, and each one uses different indications and techniques, making comparison between centers difficult.

EVALUATION

Early referral is preferred. Patients are seen as early as possible in the postpartum period, ideally in the newborn nursery. Predisposing factors are determined, including those that may have been present prenatally and during labor. The immediate postpartum history is also obtained. The baby is examined to determine whether there is a true brachial plexus lesion: sometimes, other conditions such as fractures or brain injury can present in ways that may prompt referral for brachial plexus palsy. In addition, many children who do have brachial plexus lesions have other confounding problems as well.

Maternal factors include prenatal factors, such as diabetes, preeclampsia, prolonged duration of labor, and previous obstetrical complications. The delivery history includes the following: delivery presentation (vertex, breech, or other); whether the delivery was complicated or not; whether forceps, vacuum, or other instruments were used; whether a cesarean section was performed; and whether shoulder or head dystocia was present.

Child-related factors included in the history include the size and gestational age of the baby, and the presence of complications, including respiratory complications, fractures, Horner syndrome, diaphragmatic paralysis, shoulder subluxation, and torticollis. The parents are asked whether they can recall the child's Apgar scores at 1, 5, and 10 minutes. Information regarding developmental milestones is obtained. Shoulder dislocations and clavicular, rib, and humeral fractures can also present a picture that can be confused with a brachial plexus injury.

A number of factors have been associated with obstetrical brachial plexus injury. A history of maternal diabetes leading to fetal macrosomia is also associated with brachial plexus injury.[13] Shoulder dystocia can lead to lateral traction on the infant's head, especially during the last phase of labor, resulting in an increase in the neck-shoulder angle, producing a stretch injury to the brachial plexus.[14] A brachial plexus injury in a previous delivery has also been associated with subsequent deliveries complicated by brachial plexus injury.[15] Use of forceps or vacuum techniques during delivery[16] and clavicular and humeral fractures are also associated with brachial plexus injuries.[17,18] Shoulder dislocations and clavicular, rib, and humeral fractures can all present a picture that can be confused with a brachial plexus injury (pseudoparalysis). It has been hypothesized that a clavicular fracture may be protective, because it would allow downward rotation of the shoulder in the case of dystocia.[19] However, no statistical association has been found correlating long-term prognosis with humeral or clavicular fracture.[17]

Large infants are at risk.[20] Birth weight among infants with a brachial plexus lesion is significantly greater than average, by 500 to 1000 g.[8] Unfortunately, current prenatal ultrasound techniques are unable to determine prenatal weight accurately enough to recommend cesarean section in borderline cases. Most brachial plexus injuries occur in vertex presentation vaginal deliveries, but there have been reports of brachial plexus injury with breech and cesarean deliveries as well. Breech delivery brachial plexus lesions are frequently associated with C5-6 avulsions.[21] As noted by Eng et al.,[8] affected babies with vertex presentation weighed more than normal (between 3807 and 5000 g), whereas affected babies with breech presentations weighed less than normal (1820 to 3700 g). It should be emphasized that low-birthweight babies can suffer from palsy if the presentation is breech. Although it may seem puzzling that brachial plexus injuries sometimes occur in the setting of cesarean section,[10,14,22] in unsuccessful attempts at vaginal delivery followed by cesarean section, the lesion may have been present before the cesarean section or it may be difficult to extract the infant through the abdomen if the labor has already progressed sufficiently. It is also possible that injury may occur on extracting the infant from the uterine incision.

The physical examination helps to determine the severity of the lesion. Confounders, including fractures and other factors suggestive of a non–brachial plexus origin of motor weakness, are determined. The involvement of an experienced physical or occupational therapist is critical in assessing motor function in these very small infants. The infant is placed on a clean sheet on a padded floor or large low bench in the examination room. In this manner, the brachial plexus team can examine the infant efficiently and critically in a safe environment. Older, cooperative children may stand for the examination. To appreciate all of the

subtleties of the infant physical examination, it is important to remove all of the child's clothing except for the diaper or other undergarments, depending on the age and developmental stage of the child. The posture of the head and neck is assessed for torticollis. Children with brachial plexus lesions tend to look away from the side with the lesion. In patients with obstetrical palsy, torticollis may be the result of trauma to the ipsilateral neck muscles at the time of birth or may be the result of persistently positioning the baby on one side with the injured extremity uppermost. Significant birth trauma that results in obstetric palsy could also cause concomitant torticollis secondary to injury to ipsilateral neck muscles. In contrast, torticollis could occur independently because of factors related to abnormal intrauterine head positioning.[23] An association of torticollis with obstetric palsy would then be coincidental. When present, torticollis should be managed with physiotherapy to minimize long-term sequelae, such as sternocleidomastoid muscle contracture and positional head deformity.[14] Persistent torticollis may require release of the sternocleidomastoid muscle in rare cases.

The posture of the upper extremity is suggestive of the level of the lesion. In upper plexus injury (Erb palsy, involving the fifth, sixth, and sometimes the seventh cervical roots), the shoulder is adducted and internally rotated at rest. The elbow is extended, the forearm is pronated, and the wrist and digits are held in flexion. Formerly, waiters would discretely ask for a tip using a similar posture, and so this finding has become known as the "waiter's tip" position. In total plexus palsy (C5, C6, C7, C8, ±T1), a flail limb is present, in which there is no motor activity. Pure lower plexus palsy (Klumpke paralysis, involving C8 and T1)[24] is generally not seen in obstetrical brachial plexus palsy. However, intermediate plexus palsy (C7 ± C8 and T1) has been described.[25] In intermediate palsies, the upper extremity is not held in a stereotypical posture, which varies depending on the level of the lesion. Isolated C7 avulsion has also been reported, presenting with a flexed posture of the elbow.

Abdominal and thoracic symmetry with breathing is assessed to detect deficits in phrenic nerve function. If the phrenic nerve is injured, it suggests an injury to the upper plexus, because it arises from C3, C4, and possibly C5 (although more often than not the phrenic nerve contributes to C5).[26] Fractures involving the humerus, clavicles, and ribs are suggestive of trauma to the brachial plexus. The shoulder is evaluated for dislocation. Winging of the scapula indicates injury to the long thoracic nerve, which arises from the C5, C6, and C7 roots.

Horner syndrome indicates concomitant injury to the T1 root proximal to the level at which the sympathetic fibers (gray and white rami communicantes) separate from the somatic motor fibers. Preganglionic sympathetic fibers travel along the white rami communicantes from T1 (and sometimes T2) and then enter the sympathetic chain. Disruption of these fibers leads to the classic presentation of ipsilateral ptosis, miosis, anhidrosis, and the appearance of enophthalmos.

The motor examination is central to the prognosis and treatment plan. We use the Active Movement Scale developed at The Hospital for Sick Children. Fifteen selected upper extremity motions are assessed, including those of the shoulder (abduction, flexion, internal rotation, and external rotation); elbow (flexion and extension); forearm (pronation and supination); and wrist, fingers, and thumb (flexion and extension). Each movement is assigned a score from 0 to 7 according to the Active Movement Scale (Table 1).[14] The advantage of using this system instead of the more familiar Medical Research Council scale (Table 2) is that cooperation on the part of the infant is not required. The Active Movement Scale measures

Table 1. The Hospital for Sick Children Active Movement Scale*

Observation	Muscle Grade
Gravity eliminated	
No contraction	0
Contraction, no motion	1
Motion ≤½ range	2
Motion >½ range	3
Full motion	4
Against gravity	
Motion ≤½ range	5
Motion >½ range	6
Full motion	7

*Reprinted with permission fromClarke HM, Curtis CG. An approach to obstetrical brachial plexus injuries. *Hand Clin.* 1995;11:563–580.

Table 2. Medical Research Council Muscle Grading System*

Observation	Muscle Grade
No contraction	0
Flicker or trace of contraction	1
Active movement, with gravity eliminated	2
Active movement, against gravity	3
Active movement, against gravity and resistance	4
Normal power	5

*Reprinted with permission fromBritish Medical Research Council. *Aids to the Investigation of Peripheral Nerve Injuries.* London: Her Majesty's Stationery Office; 1943.

motion within the range of joint motion, not voluntary strength. It incorporates gravity as a standard; movements are tested both with gravity and with gravity eliminated to document early recovery with precision. It is a validated instrument that has been used in clinical outcome studies.[14,22,27–29] It can be used for young infants and also for children old enough to cooperate. Mallet introduced a scale to measure outcomes following brachial plexus injury.[30] Unfortunately, the specific movements required in this scale cannot be generated by infants (Fig. 1) and so the scale cannot be used to guide management in children younger than 3 years in general. Thus, the Active Movement Scale is valuable as a single scale applicable to patients of any age. The evaluation of older patients for

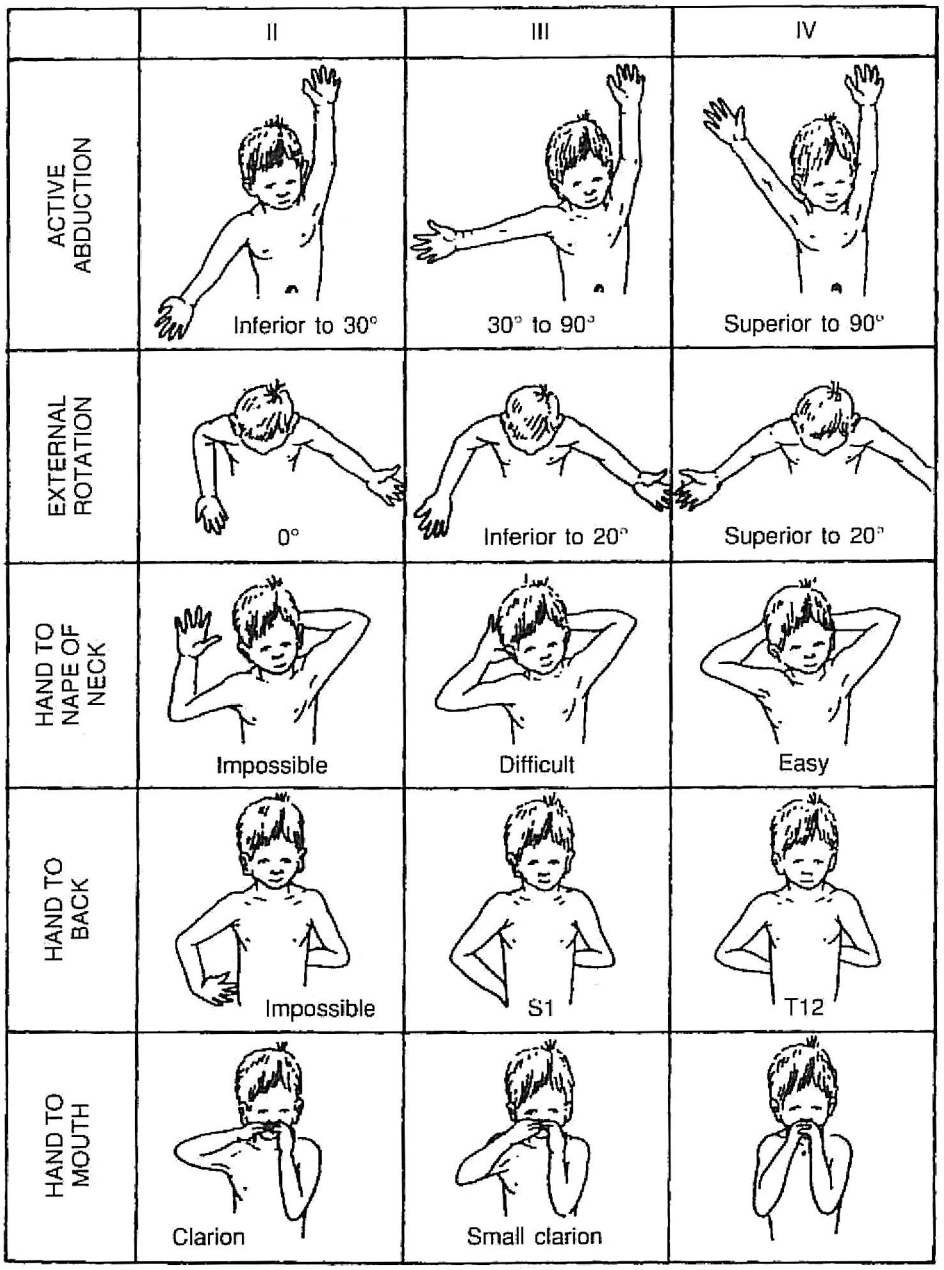

Fig. 1. The Mallet classification of upper extremity motion. This classification is useful in older children (approximately age ≥4 years). In younger children, this classification system is less useful because the child must follow specific commands. (Reprinted with permission from Gilbert A. Obstetrical brachial plexus palsy. In: Tubiana R, ed. *The Hand*. Philadelphia: Saunders; 1993:579.)

possible secondary treatment is beyond the scope of this review.

MANAGEMENT

Indications for Primary Nerve Surgery

Sometimes the decision to operate is obvious. A child with a Horner syndrome and a flail limb clearly needs a full exploration of the brachial plexus, excision of the lesion, and nerve grafts and transfers within the first few months of life to maximize function. A Horner sign alone is a reliable indicator for operative management (Fig. 2).[31] In addition, patients with clinical evidence of T1 avulsion (a limp hand resting in the intrinsic minus position) (Fig. 3) should have surgery within the first 3 months of life, as soon as it is clear that the avulsion exists. On the other extreme, an infant with a mild paralysis who recovers substantial motor function in the first month of life clearly does not need operative management. These children go on to full or nearly full recovery. The majority of infants with brachial plexus lesions are best managed nonoperatively. The difficulty arises in determining which infants will benefit from operative management, and how early the operations can be performed with reasonable certainty that surgery is necessary. There currently is no consensus. The most commonly used indication is lack of biceps function at age 3 months, as proposed by Gilbert et al.,[32–35] who observed that shoulder outcomes at 5 years were poor in those infants that failed to spontaneously develop elbow flexion by age 3 months. In a detailed analysis of the natural history of obstetrical brachial plexus injuries, Michelow et al. determined that absent elbow flexion alone at age 3 months incorrectly predicted a poor recovery 12 percent of the time. When elbow flexion and elbow, wrist, thumb, and finger extension at 3 months were combined into a Test Score, the proportion of patients whose recovery was incorrectly predicted was reduced to 5.2 percent.[10] Waters determined that infants that failed to recover biceps function by age 3 months went on to develop abnormal global upper extremity function.[36] Operative management for lack of biceps function at 6 months produced better outcomes than those managed nonoperatively

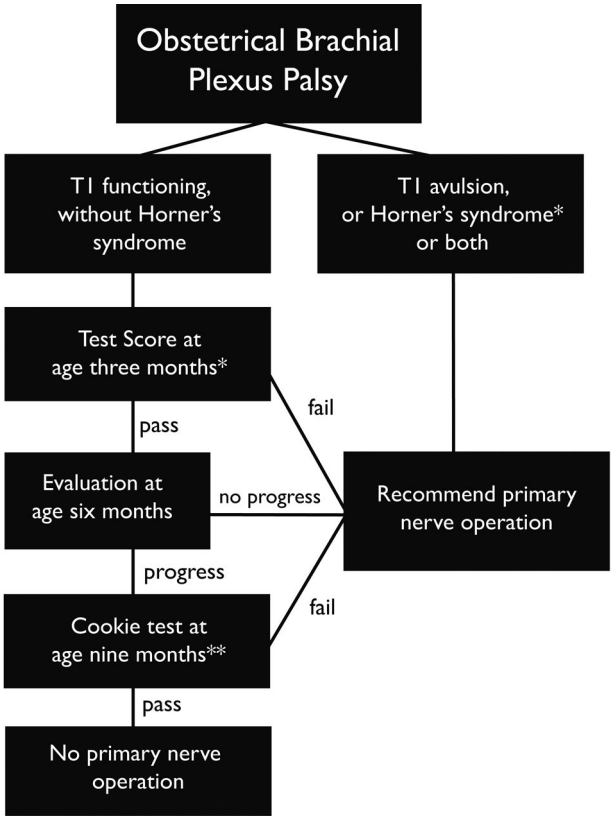

Fig. 2. Flow diagram depicting the diagnostic sequence used in our clinic. All patients with Horner syndrome and flail arms (total plexus injuries, with clinical evidence of T1 avulsion) are offered primary nerve surgery. Those with subtotal palsy are evaluated with the Test Score at age 3 months. Those who fail the Test Score are offered surgical management. Those who pass are critically evaluated at age 6 months. If they demonstrate progress in recovery, they are not offered surgical management at that time. If progress has plateaued, and the examiners believe the child will be unlikely to pass the cookie test in a further 3 months, selected individuals are offered surgical management. At age 9 months, the cookie test is administered, and those who fail are offered primary nerve surgery. *Test based on statistically analyzed data. **Test based on published empirical evidence.

Fig. 3. A 1-month-old infant with a brachial plexus injury.

after recovering biceps function at 5 months. He therefore recommended surgery for those patients not recovering biceps function at 5 months and those with flail upper extremities with Horner syndrome.[37]

The 3-month Test Score of Clarke and Curtis has been shown to be a validated instrument for use as an indication for operative intervention.[14,24,27] A Test Score is determined at age 3 months, based on the statistical discriminants that were shown to limit the false prediction rate for poor recovery to 5.2 percent (elbow flexion and extension, and wrist, finger, and thumb extension). The Test Score is determined by first converting the scores from the Active Movement Scale (Table 3) and adding up the total converted scores for elbow flexion; elbow extension; and wrist, finger, and thumb extension. A Test Score of less than 3.5 is strongly predictive of poor recovery without surgical intervention (it incorrectly predicts poor recovery 5 percent of the time, compared with using elbow flexion alone, which incorrectly predicts poor recovery 12 percent of the time). For example, if the scores for elbow flexion and extension, and wrist, finger, and thumb extension are 2, 2, 3, 5, and 5, respectively, the converted scores would be 0.3, 0.3, 0.6, 0.6, and 0.6, for a total Test Score of 2.4. If the 3-month Test Score is less than 3.5, we recommend operative management. This hypothetical child would be considered for primary brachial plexus exploration. Conversely, if the 3-month Test Score is greater than 3.5, there is no Horner syndrome, and T1 is clinically intact, we defer making a final decision regarding surgical management until a later visit.

The Test Score is intended to guide early treatment, not to dictate final operative or nonoperative management or to replace clinical judgment. Brachial plexus injuries are complex lesions, and the patterns of reinnervation are neither completely understood nor completely predictable. In highly selected cases where the child passes the Test Score at age 3 months but does not show substantial improvement in elbow flexion by age 6 months, we may recommend surgery at age 6 months if we feel that the child will be unlikely to improve sufficiently by age 9 months to pass the cookie test. The 6-month decision for surgery is based on the experience of the surgeon and the therapist in conjunction with evaluation of the rate of change of the Active Movement Scale scores over time. Again, these cases are highly selected. It is uncommon to recommend surgery at 6 months for patients who have passed the Test Score. The cookie test is performed at age 9 months. If the child fails the cookie test, operative management is recommended. The cookie test is performed by placing a lightweight cookie in the child's hand, holding the humerus at the child's side, and allowing the child to attempt elbow flexion sufficient to bring the cookie into the mouth without flexing the neck beyond 45 degrees.[22,27] If the child successfully reaches the mouth with the cookie, he or she passes the cookie test, and nonoperative management is usually recommended. If the child does not reach the mouth with the cookie, operative management is recommended.

In both operative and nonoperative cases, we institute passive range-of-motion therapy under the supervision of a physical therapist or occupational therapist beginning at the time of presentation. Therapy is aimed at maintaining or improving passive range of motion of the shoulder, elbow, and hand.

Table 3. The Hospital for Sick Children Active Movement Scale Conversion for Use in Calculating Test Score*

Muscle Grade	Converted Score
0	0
1	0.3
2	0.3
3	0.6
4	0.6
5	0.6
6	1.3
7	2.0

*Adapted, with permission, fromClarke HM, Curtis CG. An approach to obstetrical brachial plexus injuries. *Hand Clin.* 1995;11:563–580.

Preoperative Radiologic Evaluation

Preoperatively, patients undergo diaphragmatic ultrasound and computed tomographic myelography. The diaphragmatic ultrasound is used for documentation of phrenic nerve function preoperatively. The computed tomographic myelogram is used primarily to help determine which roots are avulsed. If a pseudomeningocele is seen without demonstrable ventral rootlets, that root is probably avulsed (specificity, 0.98).[38]

Brachial Plexus Exploration, Nerve Grafting, and Nerve Transfers

Sural Nerve Harvest

After nasal intubation, the endotracheal tube is secured to the membranous septum with a 3-0 or 2-0 silk suture to minimize the possibility of intraoperative extubation.[39] If the surgeon is confident that sural nerve grafting will be required, the operation begins with the child in the prone

position in preparation for bilateral sural nerve harvest. The infant is placed on an abdominal support to facilitate positioning. Both lower extremities are prepared, sterile tourniquets are applied, and the sural nerves are harvested through three transverse incisions measuring 2 cm in length (one posterior to the lateral malleolus, one at the popliteal fossa, and one at the distal belly of the gastrocnemius muscle belly, anticipating the point at which the nerve becomes subfascial).[40] The use of a 0-degree, 4-mm, 18-cm endoscope facilitates identification of the peroneal branch and other potentially useful branches when placed in the distalmost incision. Usually, approximately 13 to 15 cm of sural nerve can be harvested from each leg in a 10-kg infant. The nerves are marked with ink to preserve orientation and then placed in a damp sterile container in a refrigerator.[41]

Approach to the Brachial Plexus

The child is then turned supine and placed at the extreme head of the table, as close to the side of the table as possible. All pressure points are meticulously padded. "Egg crate" type padding, without an additional ring for the head, works well. A small beanbag is placed under the padding beneath the scapula to provide moderate neck extension. Intraoperative fluids are minimized, to prevent pulmonary edema.[39] Urine output, measured by Foley catheter, is anticipated to be low. The ipsilateral hand, arm, chest, neck, and head are prepared into the field. The head is turned away from the side with the lesion. A clear drape is used to facilitate the anesthesia team's visualization of the head and endotracheal tube. The hand is secured in a sterile towel clipped to the drapes, to allow easy visualization of the hand during intraoperative nerve stimulation.

There are a number of incisions available to approach the brachial plexus. We favor marking an incision posterior to the sternocleidomastoid muscle, curving gently at the insertion on the clavicle and continuing laterally toward the deltopectoral groove. An extensile exposure is marked for use if necessary along the deltopectoral groove, but this is usually not needed. The V-shaped incision is sufficient for exposure of the contents of the posterior triangle and heals quite well.

A supraclavicular exposure is used.[41] An incision is made through the skin and the platysma in a single layer. It can be difficult in very young patients to visualize the platysma. The triangular flap of skin and platysma is retracted laterally. Supraclavicular nerve branches are seen crossing the inferior, transverse limb of the incision. These are divided as they cross the clavicle. Following them proximally will lead to the fourth cervical root of the cervical plexus, which marks the superior limit of the dissection and serves as a landmark when identifying the C5 root. Other branches of the cervical plexus, including the great auricular nerve and lesser occipital nerves, are preserved. The C4 contribution to the cervical plexus can be used as graft material if needed. The sternocleidomastoid muscle is identified and dissection is begun just lateral to it. The clavicular head of the sternocleidomastoid muscle is divided to facilitate exposure. The external jugular vein lies over the sternocleidomastoid muscle at its lateral border, and it may or may not be necessary to divide this vein. The omohyoid muscle is located, dissected, and divided in the tendinous condensation found in the middle of the muscle. Deep to the omohyoid muscle lies the fat pad of Brown, an adipolymphatic layer that is dissected and retracted laterally to expose the neuroma. The transverse cervical artery and vein are located centrally in the field, dissected, and divided to facilitate exposure. A lateral sweeping motion with a gauze sponge draped over the index finger is very effective in separating this fat pad from the underlying clavicle. The suprascapular artery and vein are located at the superior margin of the clavicle or just deep to the bone itself and are divided to facilitate exposure. The neuroma is usually quite prominent and firm. The phrenic nerve, the only nerve that courses from lateral to medial as it descends the neck, can be draped across the external surface of the neuroma at this level. The phrenic nerve can be seen coursing from C4 distally. It often gives contributions to C5, as evidenced by the nerve being thicker proximal to C5 and thinner distally, indicating that is has given axons to C5 before entering the thorax. The phrenic nerve is stimulated with a nerve stimulator, and the twitch of the diaphragm can be palpated and visualized. The phrenic nerve must be dissected carefully from the neuroma and, in most cases, from the C5 root to allow access to a healthy C5 stump. The neuroma is then dissected. The anterior scalene muscle, which lies anterior to the brachial plexus, may be adherent to the neuroma. The middle scalene, lying posterior to the plexus, may also be scarred. The long thoracic nerve of Bell lies posterior to the plexus within the middle scalene muscle. It should be preserved during the dissection if possible but may be heavily encased in scar, depending on the level of the lesion. The C4 root serves as a landmark for locating the C5 root, which is dissected up into its foramen. Once C5 is dissected, C6 and C7 are located and dissected. Proceeding inferiorly in the neck, each foramen

progressively appears closer than the previous one (i.e., the distance between C5 and C6 appears greater than the distance from C6 to C7). The more inferior the foramen, the more posteriorly it is located. In the case of an empty foramen, the root has likely been avulsed. The injured plexus can be distorted by progressive contraction of the neuroma along its length. Avulsions and ruptures also distort the remaining plexus. The clavicle does not need to be routinely divided to facilitate exposure. Inferior retraction is usually sufficient. In the case of very long lesions, the dissection can be carried inferior to the clavicle by separating the fibers of the pectoralis major muscle from the lower border of the clavicle and, if necessary, detaching the origin of the pectoralis minor muscle from the coracoid process.

The upper trunk should be visible at this point. The suprascapular nerve lies along the lateral border of the plexus. The middle trunk may be adherent to the upper trunk, depending on the level of the lesion. The subclavian artery is located overlying the lower trunk. It should be safely dissected away from the plexus to allow inspection of the C8 and T1 roots and their foramina. The dorsal scapular artery is found either between the upper and middle trunks or between the middle and lower trunks and should be divided to facilitate exposure. The C8 and T1 roots lie deep in the operative field but, with retraction of the clavicle, can be readily visualized without dividing the clavicle or extending the incision down into the deltopectoral groove. The T1 root lies immediately superficial to the parietal pleura. If the pleura is violated, as demonstrated by air bubbles in a saline-flooded field, it may be possible to seal the leak with fibrin glue (Tisseel; Baxter International, Deerfield, Ill.) or it may become necessary to insert a chest tube. Once the T1 and C8 roots are identified, they are traced proximally. Dissection continues distal to the neuroma to define where the nerve appears undamaged.

The roots are then stimulated directly with a nerve stimulator, and the responses in the extremity are noted. A root that has been avulsed will not conduct distally even though the nerve may appear normal in the case of an intraforaminal avulsion. The neuroma is then transected at its midportion. The structures distal to the neuroma are separated. The lesion can vary significantly both in level and in overall length. The nerves are transected with a fresh no. 15 blade at levels where they feel supple and appear fascicular and healthy. It is helpful to cut against a tongue blade or an empty scalpel handle to obtain a clean cut. The cut sections of the stumps are inked for orientation and sent for toluidine blue frozen section staining. It is often helpful to tag the epineurium of the distal stump of the suprascapular nerve with a 6-0 polypropylene suture to facilitate subsequent retrieval.

The proximal level of transection is then determined. Sometimes a root avulsion is clear. For example, if there is an empty foramen, then there is no proximal stump available for grafting. Also, if the dorsal root ganglion is visible, then that root is clearly avulsed and not usable. The dorsal root ganglion normally lies within the bone. The presence of the dorsal root ganglion can be verified on intraoperative histology, in which dorsal root ganglion cells can be readily visualized. For nerves in continuity, dissection is carried out to what appears to be healthy tissue and the nerve is transected. Sometimes, this can mean removing portions of the anterior and middle scalene muscles purely to improve visualization to the level of the foramen. The nerves are transected proximally and stained for orientation, and the stumps are sent to the neuropathology department for histologic evaluation along with the distal stumps. The slides are evaluated by a neuropathologist to determine whether or not the proximal and distal stumps are adequate sources or targets for reinnervation. Essentially, all of the neuroma must be cleared from the potential nerve coaptation site, or scarring will prevent axonal regeneration. If the distal nerve stump appears appropriate histologically, it is suitable to receive axons by means of grafts or transfers. Determining the suitability of the proximal stumps as potential donors is more problematic. Even if the proximal stump appears suitable on histologic examination (very little scarring, good fascicular pattern, and no ganglion cells), an intraforaminal rupture cannot be ruled out. Multiple factors must be considered in making the diagnosis of an intraforaminal avulsion, including the clinical examination, computed tomographic myelography, intraoperative direct nerve stimulation, histology, and the "feel" of the nerve at transection. The clinical examination also gives one an indication of what to expect at operation. If there is evidence of an upper trunk lesion without a Horner sign, at least the T1 root should be left in continuity, undergoing only neurolysis. In cases where surgery is indicated by the clinical examination, neurolysis alone, without neuroma excision and grafting, is inappropriate.

Reconstruction of the Brachial Plexus

Once the proximal and distal targets are adequately prepared, nerve reconstruction is begun. Reconstruction is performed using nerve grafts

and nerve transfers. Priority must be given to reinnervation of the hand first, followed by restoration of elbow flexion, and finally shoulder stability.

The distances from the available proximal stumps to the distal stumps are measured and the distribution of the available grafts is planned. Because the distances between the proximal and distal stumps are typically 2.5 to 4.5 cm and one can usually obtain approximately 25 to 30 cm of graft material, six to 10 sural nerve grafts are typically feasible. Grafts from the cervical plexus are used where additional graft material is needed. Grafts are preferentially directed from the original root to the originally intended target (anatomical grafting). When the proximal stump is not available to innervate the intended distal nerve, other sources of axons must be used, either from within the plexus (intraplexus neurotization) or from outside the plexus (extraplexus neurotization). Sural nerve grafts are reversed to minimize axonal dropoff from side branches. Grafts placed using anatomical grafting are arranged to take advantage of the gross internal topography of the proximal stumps. Therefore, in a situation suitable for anatomical grafting, the most cephalad portion of the C5 root would be coapted to the suprascapular nerve, the posterior aspect to the posterior division of the upper trunk, and the anterior aspect to the anterior division of the upper trunk.

Grafts are arranged in the desired location beginning in the depths of the wound, and fibrin glue (Tisseel) is used for nerve coaptations (Fig. 4). No sutures are necessary for the nerve graft coaptations. The use of fibrin glue allows sectioning of the proximal stump deep within the intervertebral foramen, where suturing would be difficult if not impossible. Fibrin glue also allows the grafts to be distributed easily and selectively over the cut face of the appropriate stump.

Nerve transfers are indicated in cases in which multiple root avulsions have occurred, resulting in a paucity of donor axons from the proximal stumps. Transfer of the distal part of the spinal accessory nerve to the suprascapular nerve is the most commonly performed nerve transfer. This transfer is usually performed through the existing supraclavicular exposure. The nerve is located on the deep surface of the trapezius muscle and traced distally into the upper back beyond the proximal branches serving the upper and middle trapezius to prevent shoulder droop (Fig. 5). It is divided as far distally as possible to provide sufficient length for direct coaptation to the suprascapular nerve. As an alternative, one may approach the accessory-to-suprascapular nerve transfer through a separate posterior incision with the patient prone.[42] The advantage of the posterior approach is that it allows one to obtain a more distal coaptation between the accessory to the suprascapular nerve, thus reducing the distance from the coaptation to the motor end plates. The disadvantage is that it requires a separate exposure. Intercostal nerve transfers can also be used where insufficient donor axons are available, par-

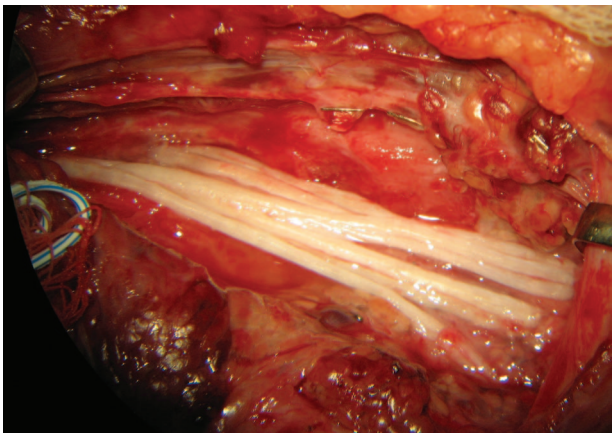

Fig. 4. Sural nerve grafts are arranged in the desired location and then fibrin glue is used for nerve coaptations. No sutures are necessary for the nerve graft coaptations.

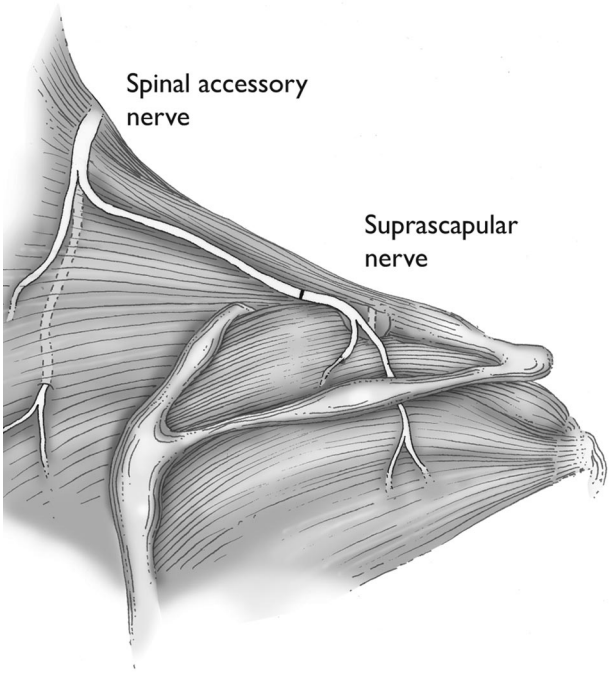

Fig. 5. Drawing depicting distal spinal accessory nerve transfer to the suprascapular nerve. It is important to preserve proximal fibers to the trapezius to prevent shoulder droop.

ticularly in cases with only one viable proximal root.[43,44] In that situation, three intercostal nerves can be used to reconstruct the musculocutaneous nerve, the accessory nerve is transferred to the suprascapular nerve, and the available proximal root is grafted to the lower trunk. Contralateral C7 transfer has been reported in obstetrical brachial plexus reconstruction, but this transfer is not widely practiced.[45] The indication for contralateral C7 transfer would be the rare case with five-root avulsions.

COMPLICATIONS

Complications arising from primary surgery have been documented in a retrospective series.[39] The overall complication rate was 33.5 percent, with no mortality. Accidental extubation occurred in 2.9 percent of cases; all of these occurred in the first half of the study. This problem was addressed by suturing the endotracheal tube to the membranous septum and by using transparent drapes for visualization of the tube. Postoperative fluid overload occurred in 14 patients (8.1 percent), three (1.7 percent) of whom developed pulmonary edema. Intensive care unit admission was required in two of those patients. Diuretic treatment was required in seven patients. No patient receiving less than or equal to 4 ml/kg/hour developed fluid overload, whereas 50 percent of the patients receiving greater than or equal to 10 ml/kg/hour did. Currently, the authors strictly limit intravenous maintenance fluids to 4 ml/kg/hour or less.

POSTOPERATIVE MANAGEMENT

The wound is then dressed and a soft Velpeau stockinette sling is applied (Fig. 6).[46] This sling maintains the shoulder in adduction for 3 weeks. The patients return for a wound check within the first 1 or 2 weeks postoperatively. After 3 weeks, the infant is allowed to move freely, and range-of-motion exercises are begun at 5 weeks. There is no need to use a plaster cast or prefabricated thermoplastic splint, because the majority of neck motion accounting for head movement occurs at C1 and C2.

Recovery of movement may begin as early as 3 months postoperatively, depending on the distance between the proximal stump and the neuromuscular junction. Restoration of preoperative movement is seen by 3 to 6 months postoperatively. By 6 to 9 months postoperatively, appreciable additional recovery is expected.[47] Further recovery is commonly seen until approximately 3 to 4 years postoperatively, when improvements in function plateau. Recovery is monitored entirely by clinical examination. No postoperative electrodiagnostic studies are typically performed. Sensory testing is also not routinely performed. Postoperative patients are followed nearly until skeletal maturity. Secondary procedures (beyond the scope of this review) are sometimes indicated to correct residual problems such as weakness of shoulder external rotation or wrist extension. Reconstructive goals include achieving elbow flexion sufficient to bring the hand to the mouth, and gaining adequate hand and shoulder function to accomplish activities of daily living. We encourage the families to involve the child in sports activities, especially those involving significant upper extremity and shoulder movement, such as swimming. We counsel them to have the child remain active in sports as long as possible.

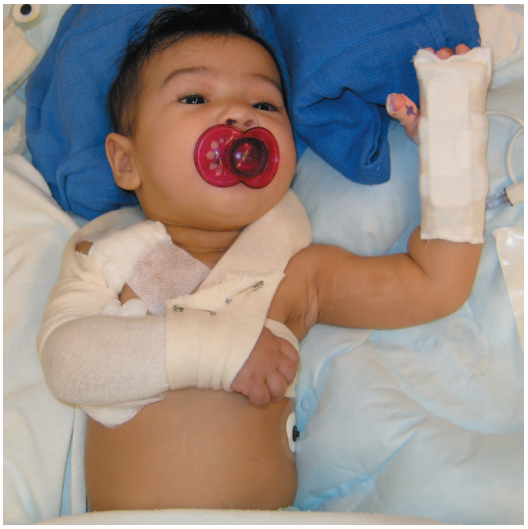

Fig. 6. A Velpeau-type sling garment is used for postoperative control of shoulder motion.

SUMMARY

Adequate diagnosis is fundamental to optimal treatment. Criteria such as the Test Score assist in making a timely, precise diagnosis, such that surgical treatment can be appropriately offered to those patients who would actually benefit from surgery. Surgical management consists of neuroma excision and sural nerve grafting, with nerve transfers as needed. Outcomes continue to improve for 3 or more years postoperatively.

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