Obstetric Brachial Plexus Injuries

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KEYWORDS

• Brachial plexus • Obstetric brachial plexus lesion
• Brachial plexus repair • Nerve surgery

An obstetric brachial plexus lesion (OBPL) is thought to be caused by traction to the brachial plexus during labour.[1,2] In most cases, delivery of the upper shoulder is blocked by the mother's symphysis (shoulder dystocia). If additional traction is applied to the child's head, the angle between neck and shoulder may be forcefully widened, further stretching the ipsilateral brachial plexus.

The incidence of OBPL varies from 0.42 to 2.9 per 1000 births in prospective studies.[3–5] Risk factors that have been identified for the occurrence of OBPL reflect the disproportion between the child and the birth canal. The main fetal risk factor is macrosomia;[6,7] maternal factors include gestational diabetes and multiparity.[6] Shoulder dystocia and assisted delivery by forceps or vacuum cup are well-known risk factors for the development of OBPL.[6] A less common delivery pattern concerns infants, usually with low birth weight, born in a breech position. This pattern carries a high risk for root avulsion.[8]

The debate is ongoing about whether OBPL is preventable and whether the obstetrician can be held responsible. This debate is fed by numerous malpractice suits in which large sums of money are compensated.[9] Case reports of spontaneous deliveries without traction applied to the child during labor with the occurrence of brachial plexus injury have been reported, suggesting that these injuries may occur in the absence of negligently performed delivery maneuvers.[10] The upper brachial plexus is most commonly affected, resulting in paresis of the supraspinatus, infraspinatus, deltoid, and biceps muscles, as first described by Erb and Duchenne. Typically, in the C5-C6 lesion type, the affected arm is resting on the surface in adduction, internal rotation, and extension. The wrist and fingers are continuously flexed when C7 is also damaged (**Fig. 1**). Hand function is additionally impaired in approximately 15 % of patients;[3,11,12] isolated injury to the lower plexus (Déjèrine-Klumpke type) is rare.[13]

The traction injury may vary from neurapraxia to axonotmesis, to neurotmesis, or to avulsion of rootlets from the spinal cord.[14] The severity of neural damage can only be assessed by evaluation of recovery in the course of time, because nerve lesions of different severity initially present with the same clinical features. Neurapraxia and axonotmesis eventually result in complete or near-complete recovery. Neurotmesis and root avulsion, on the other hand, result in permanent loss of arm function and, in time, development of skeletal malformations, cosmetic deformities, behavioral problems, and socioeconomic limitations.[15–19]

NATURAL HISTORY

The prognosis of OPBL is generally considered to be good, with complete or almost-complete spontaneous recovery in more than 90% of patients.[20–25] However, these data are based on a limited number of series,[26,27] without considering important methodologic aspects of these studies. In a systematic literature review, we discussed the methodologic flaws in the available natural history studies.[28] We found that no study presented a prospective, population-based cohort that was scored with a proper scoring system with adequate follow-up. In other words, no scientifically sound evidence exists to support the common perception of complete spontaneous recovery

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Neurosurg Clin N Am 20 (2009) 1–14
doi:10.1016/j.nec.2008.07.024
1042-3680/08/$ – see front matter

Fig. 1. An infant who has a C5-C6-C7 lesion. The affected left arm is held in adduction, internal rotation, and extension. The wrist and fingers are continuously flexed.

from OBPL. The often-cited excellent prognosis may be too optimistic. Analysis of the most methodologically sound studies led us to estimate the percentage of children who have residual deficits to be 20% to 30%.

NEUROPATHOPHYSIOLOGY

In OBPL infants, the damaged nerves are usually not completely ruptured, in the sense that a gap exists between two stumps (neurotmesis), which is most likely caused by the gradual exertion of traction forces over a small distance that act during a long period. The two crucial factors that determine good functional recovery are the number of damaged axons that successfully elongate past the lesion site and their routing. Axonal outgrowth and restoration of connections with their original motor or sensory end organs can only take place when the basal lamina tubes surrounding the axons, which are in this context the crucial anatomic structures, remain intact. The distance from the lesion site, which in OBPL is almost always at root and trunk level, to the end organ determines the length of the time required for recovery. Proximal muscles recover, therefore, at an earlier stage than more distally located ones. Recovery of predominantly axonometic OBP lesions is usually seen within the first 3 to 4 months of life.

When the traction lesion is severe, the basal lamina tubes are ruptured, but the perineurium and epineurium remain more or less intact. Outgrowing axons may not then end up directly in any tube. Typical for the OBPL is that the stretched and damaged nerve forms a ''neuroma-in-continuity'' (ie, a tangled mass of connective scar tissue and outgrowing, branching axons). The local environment encountered by the axonal growth cone may impede outgrowth and may ultimately block the restoration of axonal continuity. Even in the most severe OBPL C5-C6 lesions, at least some axons will pass through the neuroma-in-continuity and reach the tubes distal to the lesion site. The number of axons, though, may not be sufficient to yield a clinically significant functional result. The number of axons that will not pass the lesion site depends on the severity of the lesion, which is determined by the magnitude and angle of the exerted traction forces. A minimum number of axons should reconnect with an end organ to regain function. In addition, adequate recovery requires a minimum of axons that should be properly routed to their original end organ. We presume that those axons in the OBPL neuroma-in-continuity are particularly prone to abnormal branching and misrouting. Because the direction of outgrowth after severe lesions is essentially random,[29] outgrowing axons growing through a neuroma-in-continuity are likely to end up in the wrong tube. Each OBPL case is unique on an axonal level in that the number of ruptured axons and basal laminal tubes differ for each intraplexal element, which subsequently leads to the wide variety in level of functional recovery found in individual cases. Branching and misrouting can also explain cocontraction,[30] a typical feature of OBPL at a later age, in which shoulder abduction and elbow flexion, or elbow flexion and extension, become irreversibly linked.

The most severe lesion type, which is specifically related to traction to the spinal nerves forming the brachial plexus, is a root avulsion. The result is a complete discontinuity of the neural connections of the central nervous system to the peripheral nervous system. Outgrowth of axons, and thus neuroma formation or misrouting, will not take place in the case of a root avulsion.

In addition to the inadequate number of outgrowing axons and misrouting that may reduce

functional regeneration, improper central motor programming may occur.[31] The formation of motor programs may fail in OBPL for various reasons. First, OBPL causes deafferentation and weakness; many functions in the central nervous system depend on afferent input in a specific time window or else they are not formed correctly. Second, aberrant outgrowth of motor axons may present the central nervous system with conflicting information. A motor command for shoulder abduction may, for instance, cause elbow flexion in addition to abduction, through misrouted motor axons. The resulting feedback may well hamper the formation of a selective abduction program, because the central nervous system probably has no way to identify the ''misbehaving'' motor units.[32,33] Third, sensory axons might also be prone to misrouting, compounding the problem. A final hurdle for the central nervous system may be the severity of paresis. In such cases, the only way to effect certain movements may be through ''trick movements'' (such as scapular rotation instead of glenohumeral rotation), which then represent a functional adaptation.

CONSERVATIVE TREATMENT THE FIRST FEW MONTHS OF LIFE

In the past, the tendency has been to immobilize the arm directly after birth to prevent secondary damage to the injured nerve elements of the brachial plexus. It is highly unlikely that secondary damage to the brachial plexus can occur during the passive movements of the arm in a physiologic range of motion during exercises or care taking. We advocate the early and frequent mobilization of the affected extremity to prevent joint contracture and enhance the potential for future recovery. Not only does no scientific proof exist that immobilization is of any benefit to the nerve regeneration process but joint contracture formation might be detrimental to the final functional outcome when contractures limit the effective contraction of reinnervated muscles. It may also lead to improper modeling of the joints, of which the glenohumeral joint is most frequently affected.[18] Contracture formation may start as early as 2 to 3 weeks after birth. These typically restrict internal rotation, flexion, and pronation of the upper limb. Exercises that focus on prevention of contracture formation and optimization of joint mobility consist of passive external rotation in adduction and supination with an elbow flexed at 90°, just to the point where a certain tension can be felt. The arm should be held in this position for a few seconds and then released. This passive movement should be repeated frequently during one session. We advise the parents to mobilize the affected arm as frequently as possible during the day, but at least every time the diaper is changed. In addition, we recommend that the parents move both arms in a symmetric fashion. In this way, the parents can use the unaffected arm range of motion as a reference value and therapy target of reach for the affected arm. The joint mobility should be evaluated every week by a specialized child physiotherapist.

SURGICAL TREATMENT

Indications

Surgery should be restricted to severe cases in which spontaneous restoration of function is not likely to occur (ie, in neurotmesis or root avulsions).[14] The clinical difficulty lies in the ability to distinguish these patients from those who have similar deficits but who have a high likelihood of spontaneous recovery. At present, the earliest accepted indication of the severity of the lesion can be obtained at 3 months of age. Paralysis of the biceps muscle at 3 months is associated with a poor prognosis[34] and is considered an indication for nerve surgery by some investgators.[35–39] However, biceps paralysis at age 3 months does not preclude satisfactory spontaneous recovery.[40–43] Additionally, biceps muscle testing may not be reliable in infants.[43–45] Alternative tests[39,44,46] are complex or are done at an even later age. These difficulties in the diagnostic process may also lead to parental distress.[47] We are in favor of concentrating the treatment of severe OBPL in specialized referral centers. Early diagnosis of severe OBPL lesions and admission to a specialized center opens opportunities to start appropriate and rigorous child physiotherapy or (appropriately) early surgery, if necessary.

Electromyography and Prognosis

Ancillary testing, in particular, electromyography (EMG), is not considered reliable enough for prognostication of OBPL.[32,48] A needle EMG might seem a useful tool in this respect, but at present, its role is debated. A main reason for this is that EMG findings may be discordant with clinical findings at 3 months of age, at which the biceps test is performed.[37] In a paralytic biceps brachii muscle, the expected findings are an absence of motor unit potentials (MUPs) and the presence of positive sharp waves or fibrillation potentials (to be called ''denervational changes''). But in a typical OBPL case, MUPs are present and denervation is absent in a paralytic biceps muscle at 3 months of age. This confusing finding has been noted by others,[49,50] and may have contributed to the

opinion that the EMG is not useful in OBPL.[24,51] We previously outlined several possible explanations for ''inactive MUPs,'' (ie, MUPs in a paralytic muscle),[32] which suggest that the presence of inactive MUPs may depend on time after injury because they reflect incomplete outgrowth of damaged axons and the formation of motor programs in the central nervous system.

Spontaneous recovery of useful extremity function has been observed in patients who do not have elbow flexion at 3 months of age.[43] In one study, even 20 of 28 infants who had no biceps function at 3 months had developed biceps contraction at 6 months.[41] Together with our findings[52] that MUPs can almost always be found in the biceps muscle at 3 months, this finding strongly suggests that the age of 3 months does not represent a stable state in OBPL. In fact, the outgrowing axons may well have only just arrived in the various muscles, and the central nervous system may not yet have learned to cope with the situation. In nerve lesions in adults, one may expect all motor programs to be ready and waiting for the restoration of peripheral connections. In OBPL, axonal outgrowth may only be the starting point for restoration of function because formation of central nervous system motor programs may only commence after enough axons have arrived at their muscle targets. At the same time, forming such central motor programs may be more difficult and thus may take longer than in healthy children because the central nervous system must somehow take aberrant outgrowth and the confusing feedback it causes into account. Faced with a degree of inescapable co-contraction, it may not be easy to program effective elbow flexion, abduction, or rotation. In this hypothetic view, the age of 3 months may well be the worst period imaginable to correlate the EMG with clinical findings: it is late enough to show evidence of axonal outgrowth, but too early for the brain to control contraction efficiently. Therefore, the role of the EMG for prognosis at 3 months remains undetermined at present. We showed that severe cases of OBPL can be identified reliably at 1 month of age based on clinical findings and needle EMG of the biceps.[52] These findings will be reported in full after independent validation, which is currently in progress.

For less extreme cases (ie, most OBPL cases), the challenge lies in predicting whether function will be better after spontaneous outgrowth through a neuroma-in-continuity, resulting in reinnervation through tangled paths, or after nerve grafting, in which the grafts serve as a straight path that can be targeted. Results achieved by surgery are claimed to be superior to the outcome in conservatively treated patients who have equally severe lesions.[37,42,53] However, this comparison relies on historical controls;[54] no randomized study has been performed.[55,56] The best way to answer this question may be by way of a controlled trial comparing nerve surgery to spontaneous recovery. In view of the current standard of treatment practice, it seems extremely difficult to perform such a prospective randomized trial.

Selection for Surgery

In the Leiden University Medical Center, surgery for OBPL is rarely performed before 3 months of age (for anaesthesiologic reasons) but almost always before the age of 7 months. In selecting infants for surgery, we seek to identify all cases of neurotmesis or avulsion. Infants are selected for surgery when external shoulder rotation and elbow flexion with supination remain paralytic after a 3- to 4-month period to await spontaneous recovery. Impaired hand function is an absolute indication for nerve surgery as soon as the infant turns 3 months old.[57] If the quality of shoulder and elbow joint movements is doubtful, surgical exploration is performed hoping that errors would consist of not finding neurotmesis or avulsion during surgery rather than letting such lesions go unoperated. Preoperative ancillary investigations in all patients consist of ultrasound of diaphragm excursions to assess phrenic nerve function and CT myelography under general anesthesia to detect root avulsions (**Fig. 2**).[58,59]

Surgical Exposure

In OBPLs, supraclavicular brachial plexus exploration is the first step, to localize the site and extent of the lesion. In most cases, this exposure will suffice for proper evaluation and reconstruction, if needed. The supraclavicular incision runs parallel and superior to the clavicle at the basis of the lateral cervical triangle. After opening the deep fascia colli media, the phrenic nerve is identified in its course over the ventral surface of the anterior scalene muscle. The site where the phrenic nerve crosses the lateral border of the anterior scalene muscle consistently lies on top of the spinal nerve C5 where it emerges from the C4-C5 foramen; hence, it is a reliable surgical landmark. The phrenic nerve is then neurolysed in its trajectory running on top of the anterior scalene muscle to allow a medial transposition without traction on the phrenic nerve. Resection or partial resection of the scalene muscles is indicated in order to make a good inspection of the proximal, intraforaminal, part of the spinal nerves possible. Because such proximal exposure involves a risk for opening

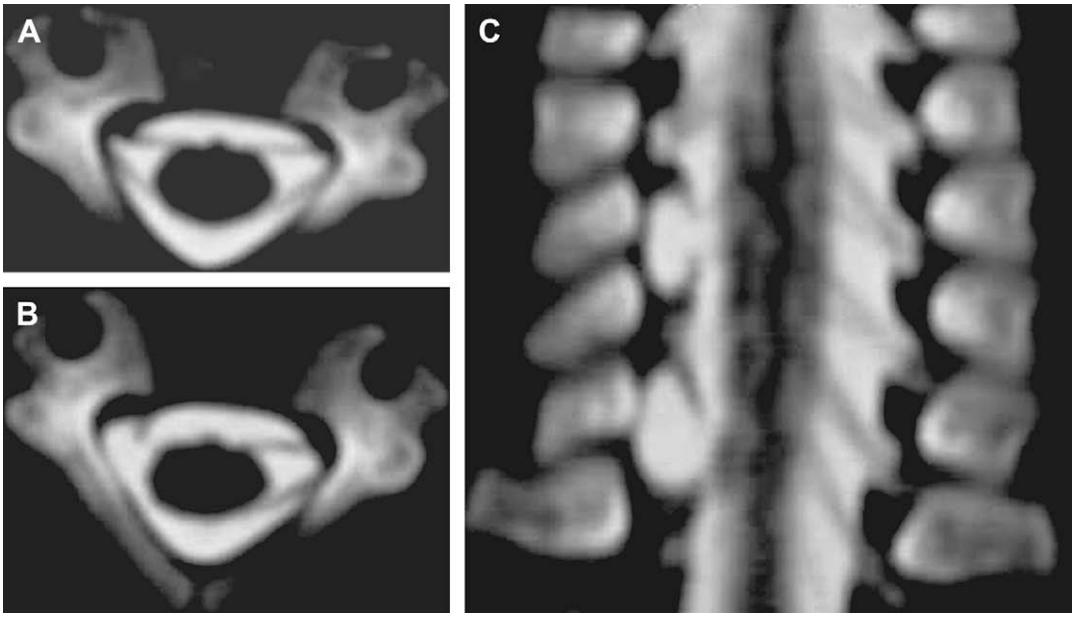

Fig. 2. CT myelography. (*A*) Normal CT myelography. (*B*) Avulsed root on the right side. Note the absence of root filaments and the small pseudomeningocele. (*C*) Coronal reconstruction of the CT myelogram demonstrating right-sided pseudomeningoceles.

a pseudomeningocele that extends extraforaminally, care should be taken to identify such pseudomeningocele on CT myelography or MRI. The omohyoid muscle is identified as the caudal aspect of the exploration. We do not resect the omohyoid muscle but instead, mobilize it and retract it downward to the clavicle. Occasionally, the transverse cervical vein and artery are ligated and divided. The suprascapular nerve (SSN) originating at the lateral side of the superior trunk normally follows a slightly oblique cranial-caudal course to the scapular notch. Caudal displacement of the superior trunk changes this course in a more or less horizontal direction.

Occasionally, the lesion extends to the infraclavicular part of the brachial plexus. The infraclavicular brachial plexus may be surgically exposed in several ways. The major and minor pectoral muscles cover the brachial plexus, which runs medial to the coracobrachial muscle and short head of biceps brachii muscle. A straight incision is made above the deltoid-pectoral groove. The major pectoral muscle is retracted downwards to expose the infraclavicular brachial plexus. The minor pectoral muscle is alternately retracted upward and downward allowing inspection of the entire infraclavicular plexus. The brachial plexus, with accompanying vessels, is initially identified at the inferior border of the pectoralis minor muscle. To facilitate retroclavicular exposure, if indicated, the retroclavicular space can be widened for inspection by upward clavicular retraction by an assistant or a suspended, table-mounted retractor. An alternating supra- and infraclavicular view allows dissection or repair of the retroclavicular nerve bundles (**Fig. 3**).

In its upper infraclavicular course, the posterior cord runs lateral and posterior, rather than medial and posterior, to the lateral cord. The latter course is frequently depicted incorrectly in schematic anatomic drawings. The axillary nerve runs through the quadrilateral space above the latissimus dorsi and teres major tendons. This nerve can be identified more easily by external rotation of the humerus.

When nerve transfers are applied, the donor nerves are dissected free. An absolute imperative of donor nerves is that these nerves have normal function. Their identification and function can be tested with direct electric stimulation. The accessory nerve (XIN) can be identified on the medial-anterior surface of the trapezius muscle. The nerve gives off a branch to the superior part of the trapezius muscle, which has to be kept intact. The accessory nerve is dissected free distally as far in its course as possible and cut. The proximal stump is then passed through the fascia colli media. In this way, a direct coaptation with the SSN can almost always be performed. The medial pectoral nerves can be identified running below the minor and major pectoral muscles. Because the medial pectoral nerve originates from the

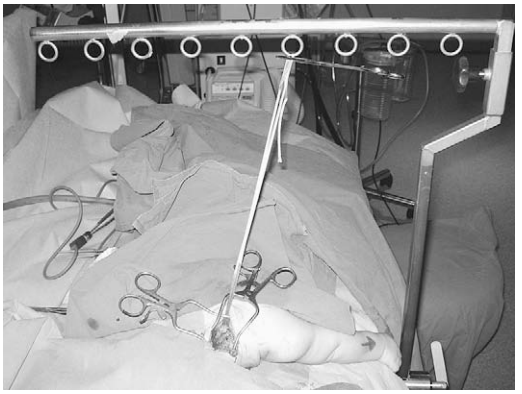

Fig. 3. Exposure of the retroclavicular part of the brachial plexus can be easily improved by pulling the clavicle upwards by a lace that is attached to a bar above the operating table. An additional incision is not needed. When using these additional, table-mounted retractor systems, care must be taken to guard against positioning injuries, with the use of sufficient padding between the system and the patient.

medial cord,[60] its function is intact in C5-C6 or C5-C6-C7 lesions. Nerve stimulation is an indispensable step in the identification of the medial pectoral nerve because small vessels may occasionally have an almost similar aspect and course. Routinely, two individual medial pectoral nerve branches exist. They should be cut as distally as possible and then transferred laterally to the musculocutaneous nerve (MCN). Depending on the cross-sectional area of the medial pectoral nerve branches, the MCN is cut completely or only partially. Direct pectoral-musculocutaneous coaptation is feasible in most cases.

We previously described the technique for intercostal nerve (ICN) transfer in adults.[61] We apply the same surgical technique in OBPL infants. In short, three ICNs are dissected through an undulating skin incision from the inferior border of the major pectoral muscle to the costosternal junction. The ICNs are transected as close as possible to the sternum to obtain sufficient length to be tunneled to the axilla for direct coaptation to the MCN.

Surgical Assessment of the Severity of the Lesion

Surgery is performed under general anesthesia without muscle blocking agents. The brachial plexus is exposed in the lateral neck triangle through a straight incision parallel to the clavicle. Depending on the extent of injury, the infraclavicular part is also exposed. The severity of the lesion of each clinically involved spinal nerve is subsequently assessed. A distinction is made between axonotmesis, neurotmesis, and root avulsion based on (1) inspection of the status of nerve continuity at the intraforaminal level in combination with the presence or absence of root filaments on CT myelography; (2) the extent and location of neuroma formation; (3) selective electric stimulation of all the involved spinal nerves using a bipolar forceps in combination with a 2.5-Hz pulse generator with increasing voltage (maximum 6 V).

A spinal nerve root is considered avulsed when the nerve at the intraforaminal and juxtaforaminal levels exhibits root filaments, the dorsal root ganglion is visible, neuroma formation is absent, and no muscle contractions occur following direct stimulation. In most spinal nerves, these findings correspond with the absence of root filaments as demonstrated by CT myelography. Avulsed roots are cut as proximally as possible. When the dorsal root ganglion can be morphologically identified, it is dissected from the ventral root and removed. Following confirmation by frozen section of the presence of ganglion cells, one may be certain that the distal stump consisted only of the ventral root. This ventral root can be the target for nerve grafting, or the ventral root can be attached to a qualitatively good nerve stump directly, without a nerve graft.

A spinal nerve is considered neurotmetic when the following features are present: a normal appearance at the intraforaminal level, a clear increase of the cross-sectional diameter at the juxtaforaminal level, abundant epineural fibrosis, loss of fascicular continuity, increased consistency, and increase in the length of the nerve elements with concomitant distal displacement of the trunk divisions. Electric stimulation of the spinal nerve proximal to the neuroma might cause weak muscle contractions that are detectable with palpation but are not strong enough to move the limb. Resection of neurotmetic tissue is performed, and the proximal and distal stumps are prepared for nerve reconstruction.

A spinal nerve is considered axonometic when neurolysis reveals no substantial increase in the cross-sectional diameter, only limited epineural fibrosis and intact fascicular continuity. Furthermore, on C5 stimulation, abduction with movement of the limb and some external rotation should be present, and on C6 stimulation, elbow flexion against gravity with supination should be found. These movements, of course, will only occur if sufficient time has elapsed for spontaneous recovery to occur, which is often not the case with fairly early surgery at 3 to 5 months. Axonotmetic nerves are left in situ because

spontaneous nerve regeneration is in process, although as yet clinically not clearly apparent. Axonotmesis is confirmed by the occurrence of good spontaneous recovery after at least 2 years of follow-up.

Intraoperative Electrodiagnostic Studies

In adults, recording of intraoperative nerve action potentials (NAP) and evoked compound motor action potentials (CMAP) is advocated to distinguish objectively between nonconducting and recovering lesions.[62,63] The presence of a NAP across the lesion site requires at least 3000 to 4000 nerve fibers with a diameter more than 5 μm. The presence of these fibers in a recovering nerve indicates that spontaneous functional recovery will take place, and that, therefore, resection and grafting are not indicated.[64]

We analyzed the results of intraoperative NAP and CMAP recordings in 95 patients who had OBPL to assess the predictive values for the diagnosis of axonotmesis, neurotmesis, avulsion, or normal spinal nerves, respectively.[65] We found statistically significant differences among diagnosis groups. For the individual patient, however, a clinically useful cutoff point for NAP and CMAP recordings to differentiate between avulsion, neurotmesis, axonotmesis, and normal could not be found. The sensitivity for an absent NAP or CMAP was too low for clinical use. Intraoperative NAP and CMAP recordings, therefore, do not add to the decision making during surgery. Direct electric stimulation remains important to obtain a nonquantitative evaluation of the presence of axons in a regenerating nerve segment.

Surgical Reconstruction

The first goal of nerve repair is restoration of hand function if necessary; the second priority is restoration of elbow flexion, and the third goal is recovery of shoulder movements.[57] The sources of outgrowing axons for reinnervation are viable proximal nerve stumps, which we evaluate with frozen section examination. The total quantity of myelin in the entire cross-sectional area of the donor stump, which corresponds to the viability of the proximal stump, is expressed semiquantitatively: (1) less than 25%; (2) 25% to 50%; (3) 50% to 75%; (4) more than 75%.[66] The neuropathologist can additionally assess the presence of ganglion cells (indicative of total avulsion) and fibrosis/neuroma in the proximal and distal stumps. In our center, proximal stumps are only used as an outlet for nerve grafting and thus, are considered viable when the total myelin quantity is greater than or equal to 50%.

In most cases, nerve grafts are led out from a viable proximal nerve stump to distal target stumps after resection of a neuroma-in-continuity (**Fig. 4**). The preferred option in the case of a root avulsion is direct coaptation (so without the use a graft) between an available proximal nerve stump and the avulsed root (intraplexal nerve transfer) (**Fig. 5**). In such cases, the ventral and dorsal roots are separated, and the dorsal root ganglion is resected before direct coaptation or nerve grafting. When the number of proximal stumps is limited, intraplexal transfer or nerve grafting is used to reinnervate the hand and extra-intraplexal nerve transfers are performed to restore shoulder function and elbow flexion. A direct coaptation without a nerve graft is performed in cases in which the ICNs, medial pectoral, or spinal accessory nerves are used as the donor nerves.

Selection of the distal target stumps is determined by the aforementioned goals. For that reason, the first goal of hand function restoration is pursued by neurotization of C8, T1, inferior trunk, or middle trunk, aiming at restoration of median or ulnar nerve–innervated functions. Preferably, a direct coaptation to C8 without a nerve graft is performed (**Fig. 6**).[57]

The second priority is restoration of elbow flexion; the anterior division of the superior trunk, the lateral cord, or the MCN is chosen as the target nerve. The third goal is to recover shoulder movements and involved neurotization of the posterior division of the superior trunk, the suprascapular, or the axillary nerve. If possible, a graft is led out from C5 to the SSN to reanimate external rotation. The XIN–SSN transfer is performed when the available stumps have been used for nerve reconstruction, aiming at reanimation of the hand or biceps muscle. In all patients, both sural nerves are harvested as grafts, a procedure that is routinely performed with the aid of an endoscope (**Fig. 7**). In addition, the cutaneous cervical plexus or the cutaneous nerves of the arm or forearm are used in some cases.

Postoperative Treatment

Postoperatively, the child's upper body is placed in a prefabricated bay cast for a period of 2 weeks to limit movements of the head and affected arm. Patients are examined at our outpatient clinic at 6-month intervals. The active and passive range of joint movements is noted in degrees and Medical Research Council (MRC) grade. In addition, the Mallet score[67] is assessed to evaluate shoulder function and the Raimondi hand score is assessed to evaluate hand function.[68]

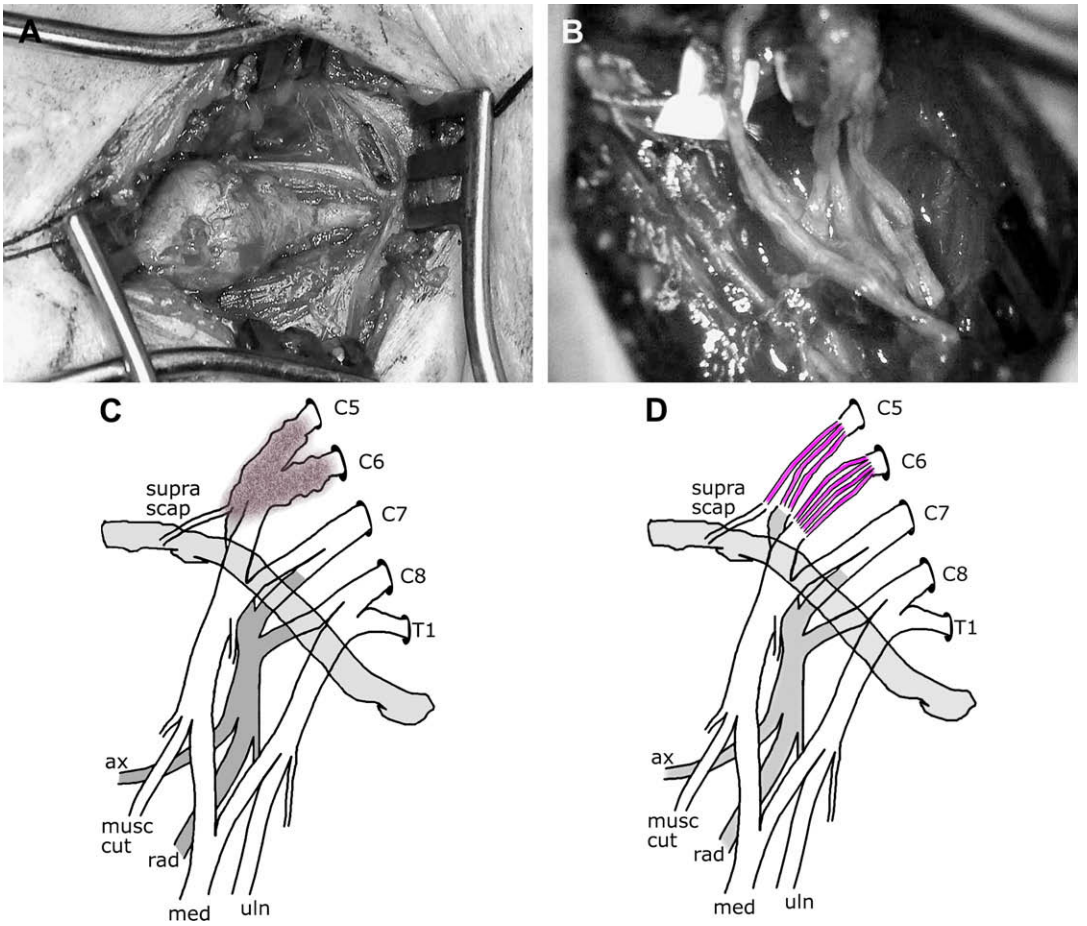

Fig. 4. (*A*) Intraoperative photograph of neuroma-in-continuity of the superior trunk. (*B*) Intraoperative photograph of nerve grafting of the spinal nerve C5 to the posterior division superior trunk and SSN and C6 to the anterior division superior trunk. (*C*) The most common lesion type: supraclavicular neuroma-in-continuity of the superior trunk. (*D*) Nerve grafting after resection of the neuroma of the spinal nerve C5 to the posterior division superior trunk and SSN and C6 to the anterior division superior trunk. ax, axillary nerve; med, median nerve; musc, musculocutaneous nerve; rad, radial nerve; supra scap, suprascapular nerve; uln, ulnar nerve.

RESULTS OF NERVE SURGERY

Shoulder Function

The results of nerve repairs to improve shoulder function have been published in several series, from which at first glance it can be concluded that global shoulder function recovery is good.[35,38,42,69,70] We performed a study (n = 86) that focused on the recovery of true glenohumeral external rotation as a solitary movement in to determine specific factors affecting recovery after neurotization of the SSN (**Table 1**).[71] During the neurologic evaluation of these children, trick movements were eliminated for a clean comparison of two surgical techniques and of other prognostic factors (**Fig. 8**). We found that only 20% of the patients gained more than a 20° range of true external rotation and that restoration of true glenohumeral external rotation failed in as many as 41% of the patients. In contrast to this disappointing result of true external rotation, functional evaluation showed that 87% of the patients could reach their mouths and 75% of children could reach the backs of their heads, which illustrates the great ability of the infants to compensate for their limited true external rotation by thoracoscapular movements. We found no difference between nerve grafting C5-SSN and nerve transfer of XIN to SSN.

Elbow Flexion

In the previously mentioned cohort, biceps muscle force against gravity or more was gained in 92% of patients.[71] In most of these patients, nerve grafting had been performed. Recently, we analyzed

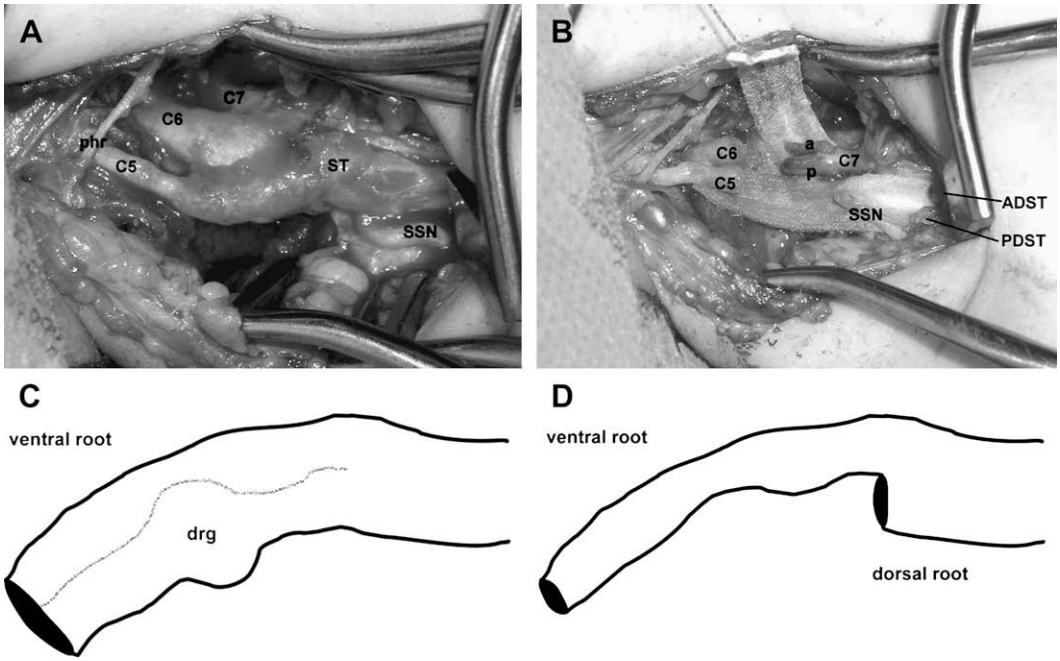

Fig. 5. (*A*) Intraoperative photo showing neuroma of the upper trunk. phr, phrenic nerve; ST, superior trunk. (*B*) After resection of the upper trunk, the ventral and dorsal rootlets of C7 are separated. a, anterior root filaments C7; p, posterior root filaments C7; ADST, anterior division superior trunk; PDST, posterior division superior trunk. (*C*) The structure of the avulsed root. drg, dorsal root ganglion. (*D*) After separation of ventral and dorsal roots, the dorsal root ganglion is resected.

30 consecutive patients (1995–2005) in whom nerve transfers for biceps reanimation had been applied (data not yet published). From 1995 to 2000, only intercostal-musculocutaneous nerve (ICN-MCN) transfers were performed, and from 2001 to 2005, the pectoral-musculocutaneous nerve (PEC-MCN) transfer was preferentially applied, when the C8/T1 trajectory to the inferior trunk was intact. In 15 of 16 ICN-MCN transfers, three ICNs were coapted directly to the MCN; in one patient, a 1-cm graft proved necessary. In all patients with PEC-MCN transfers, we were able to perform a direct coaptation. Elbow flexion greater than or equal to MRC 3 was achieved in 87% of patients after a mean follow-up of 40 months. The results in the PEC-MCN group were better than those of the ICN-MCN group (93% versus 81%, respectively), which may be explained by the more severe brachial plexus lesions that were included in the ICN-MCN group (8/16 patients in the ICN-MCN group had a flail arm). In the ICN-MCN group, one secondary surgery was performed (a Steindler flexorplasty). No adverse effects were noted in either group. We did not see any rib cage deformity after ICN-MCN transfer. Such deformities have been occasionally mentioned by some colleagues, but we are not aware of any reports in the literature. We think that the crucial factor for avoiding rib cage deformity is to leave the periosteum of the ribs untouched during dissection of the ICNs. In this way, the growth of the ribs of the infant remains undisturbed over time.

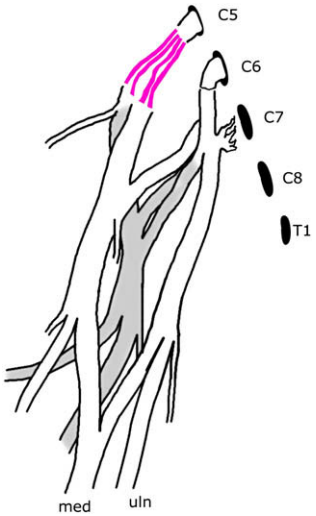

Fig. 6. Example of reconstruction of hand function: grafting C5-superior trunk and direct coaptation C6-C8. med, median nerve; uln, ulnar nerve.

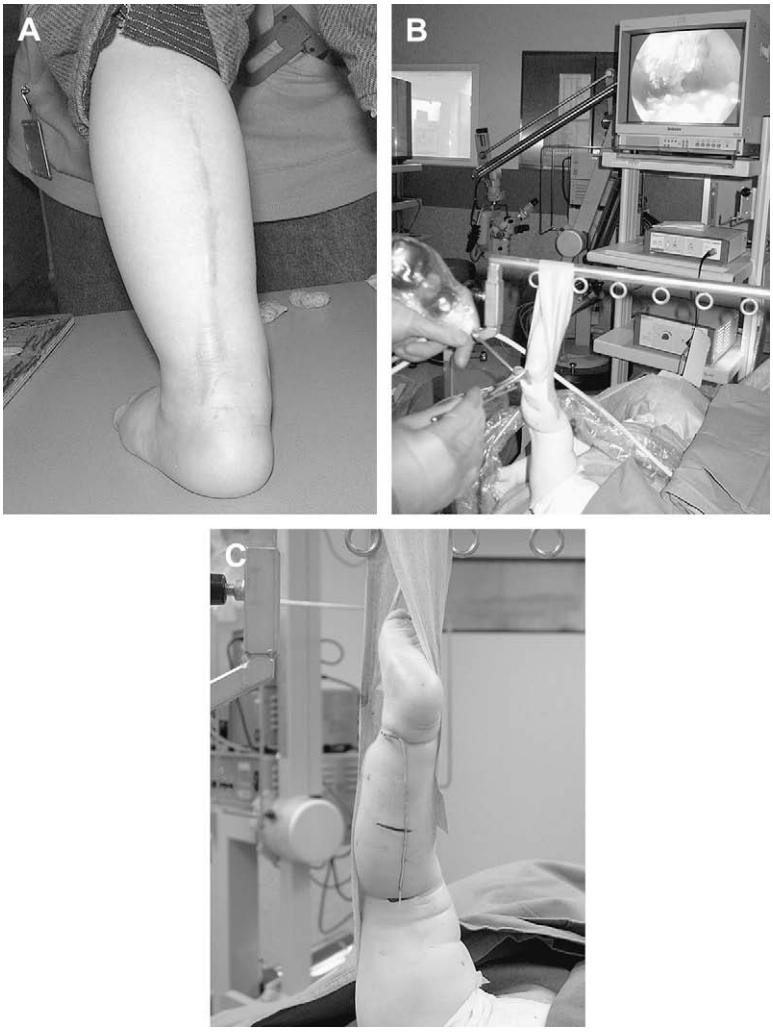

Fig. 7. (*A*) The scar after the "traditional" longitudinal incision of the calf for harvesting the sural nerve. (*B*) Endoscopic assistance for neurolysis of the sural nerve. (*C*) End result after harvesting the sural nerve using three short incisions; the nerve has been cut proximally.

These results correspond well to the few reports in literature. Kawabata[72] reported the results of 31 ICN-MCN transfers in OBPL patients: 94% reached greater than or equal to MRC 3. For the PEC-MCN transfer, a success rate of 88% greater than or equal to MRC3 was reported by Blaauw.[73] The transfer of a single fascicle of the ulnar nerve to the biceps motor branch has been proposed by some investigators as alternative intraplexal transfer, after satisfactory results in adults with this technique were published.[74,75] We have included this technique in our overall surgical strategy but feel that indications in OBPL infants may be limited, especially because the ICN-MCN and PEC-MCN transfers are almost always an option and provide satisfying results.

Recovery of Hand Function

The objective of surgical treatment of the infant who has a flail arm is significantly different from that in adult brachial plexus lesions. The main target is to establish the ability to use the affected hand to assist in bimanual activity. Combined with good elbow flexion, strong finger flexion is mandatory for a supportive role in the bimanual execution of daily life tasks. Without reanimation of the hand, the maximal function that can be obtained is the use of the affected limb as a ''hook.'' In the past, reanimation of hand function in adults who had a total brachial plexus lesion has been tried, but it did not result in useful function.[76] Because of better nerve regeneration and neural plasticity in infants compared with adult patients, restoration of hand function in OBPL infants is feasible. The primary aim of surgery in patients who have a flail arm due to OBPL is, therefore, restoration of hand function. In our OBPL series, we identified and subsequently analyzed 16 patients who had a flail arm, in whom discontinuity of the outflow of the spinal nerves C7, C8, and T1 was

Table 1
Retrospective analysis of results at the Leiden University Medical Center

Subject	n	Year	Inclusion Criteria	Results
External rotation	86	1-1-1990 to 12-31-2000	Surgical reconstruction of SSN function Grafting C5-SSN or transfer XIN-SSN Follow-up: 3 years	20% true external rotation >20° 87% can reach mouth 75% can reach back of head 94% elbow flexion ≥MRC 3
Elbow flexion	20	1-1-1995 to 12-31-2005	Nerve transfer for elbow flexion ICN-MCN or PEC-MCN transfer Follow-up: 2 years	86% elbow flexion ≥MRC 3 PEC-MCN: 93% ICN-MCN: 81%
Hand function	16	1-1-1990 to 07-01-2002	Reconstruction of C8 and T1 Follow-up: 3 years (n = 15), 2 years (n = 1)	69% Raimondi hand score ≥3

Abbreviations: ICN-MCN, intercostal-musculocutaneous nerve; PEC-MCN, pectoral-musculocutaneous nerve.

present due to avulsion injury or neurotmetic parts of the outflow were resected, followed by neurotization of C8/T1/inferior trunk or median nerve was performed. The postoperative recovery of hand function could, therefore, only be attributed to the nerve reconstruction.[57] The analysis of our surgical results showed that useful reanimation of the hand was obtained in 69% of patients (Raimondi score ≥3).[68] Only a few other reports concerning recovery of hand function were published. Haerle and Gilbert[77] reported 76% good recovery of hand function, but in this series, secondary surgery (ie, tendon transfers) had also been performed on several patients. In Birch and colleagues'[35] series of 47 patients, 57% regained a Raimondi score greater than or equal to 4 and 93% regained a Raimondi score greater than or equal to 3.

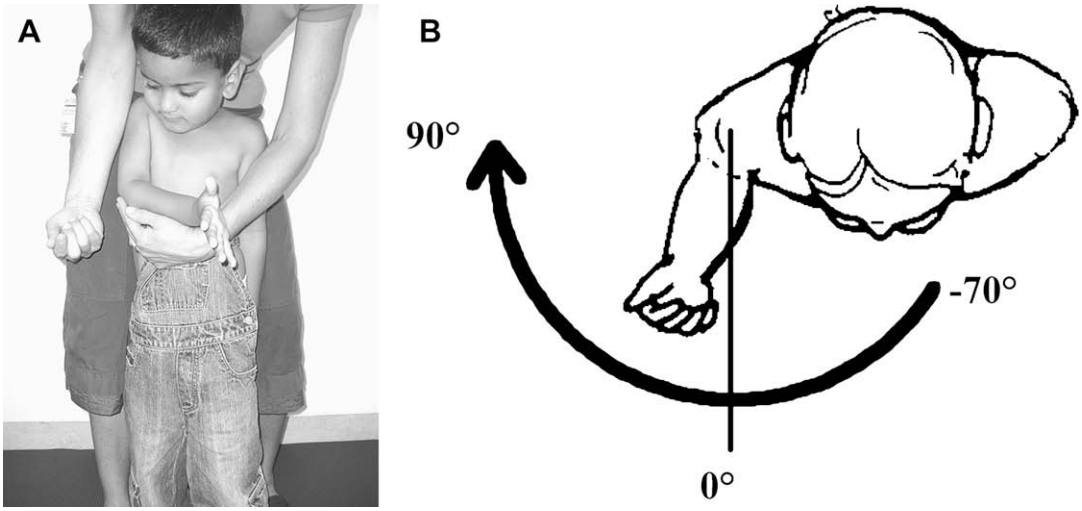

Fig. 8. (*A*) Example of measurement of true glenohumeral external rotation in adduction; trick movements are eliminated. Note the extended wrist to compensate for a lack of external rotation. (*B*) Measurement angle of external rotation.

SUMMARY

An OBPL is not an uncommon birth injury; 20% to 30% of infants who have this condition may have incomplete spontaneous recovery. As a consequence, functional disability remains, which might affect their upper limb function for the rest of their lives. The level of functional loss depends on the extent of the nerve lesion. Selection for appropriate surgical treatment is challenging and requires experience, as does the nerve reconstructive surgery. Good results with nerve reconstructive surgery have been obtained, significantly improving the functionality of the arm to a level that would probably not have been reached through spontaneous regeneration and conservative treatment. Specialized centers with a multidisciplinary approach are probably best suited for the treatment of these infants.

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