

Management of brachial plexus injuries

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With 9 Figures

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Abstract

Most brachial plexus lesions are traction injuries sustained during birth, but in adolescents and older people they are usually caused by traffic accidents or following a fall in the home. A minority are the result of penetrating injury after civilian assault or trauma encountered during wartime.

Birth palsy cases (obstetric brachial plexus palsy) and the remaining cases (traumatic brachial plexus palsy) are viewed differently with regard to treatment and outcome and so these two groups are usually discussed in separate chapters. In this paper we treat both groups in parallel because as far as primary (= nerve) surgery is concerned, many treatment problems and solutions are present in both groups and are therefore comparable.

Keywords: Brachial plexus injuries; birth palsy; obstetric brachial plexus palsy; traumatic brachial plexus palsy; management of brachial plexus injuries.

Introduction

In the nineteen-sixties, there was a revival of primary (= nerve) surgery of brachial plexus injuries in some neurosurgical centres. The wide application of the surgical microscope, new techniques for nerve repair, innovative imaging techniques, etc. gave rise to a fresh interest in the surgical treatment of brachial plexus palsy cases, because there was an expectation that the results of surgery could now be rewarding. But surgery was prolonged, results were modest and interest soon waned in most centres. In a few centres, however, the realization of a functional concept governing surgical treatment gave a new impetus to continue, because the value of the outcome was taken into account. As a logical consequence, secondary surgery, such as osteotomy, tendon- or muscle transfer, was included in the total treatment policy as soon as the results of the nerve surgery had become clear. The introduction of treatment of birth palsy cases gave an extra incentive, because results were often much more rewarding in these children.

In this paper we shall discuss indications and surgical techniques for brachial plexus injuries from a functional point of view. In addition to this overview, the reader is referred to the extensive literature for further information. Although both obstetric and non-obstetric brachial plexus palsy are traumatic in origin (mostly traction injuries), it is quite normal for clinical features and surgical approach to be discussed separately.

Epidemiology

The incidence of obstetric brachial plexus injury is better documented than that of traumatic, non-obstetric injury. Official registration institutions in The Netherlands (SIG) quoted an incidence of 1.02‰ in 1995, 1.34‰ in 1996, and 0.90‰ in 1997 [11]. The reported incidence from other countries in the

last three decades of the twentieth century varies considerably from 0.42 [24] to 2.5 per 1000 births [4, 31, 33, 62, 65]. Risk factors are breech presentation, macrosomia and shoulder dystocia [43].

The reported incidence of obstetric brachial plexus injury in different countries varies from 0.42 to 2.5 per 1000 births.

The incidence of traumatic, adult lesions is difficult to evaluate. In The Netherlands, numbers ranging from 82 (1992) to 119 cases (1994) have been reported, constituting complete or partial supraclavicular lesions or infraclavicular lesions; sometimes double lesions are present. Most adult lesions are the result of motorcycle accidents, but the number of open lesions due to shotgun- or stabbing injuries is increasing. About 30% of the patients have concomitant injuries of the head, thorax or internal organs, and fracture of the humeral shaft or injury to the glenohumeral joint is usually present. The subclavian artery is torn in 15% of supraclavicular and in 30% of infraclavicular lesions; damage to the spinal cord is found in 5% of complete lesions [8].

According to Bonnard *et al.* [15], the palsy is complete in 60%, nearly complete in 20% and partial in 20% of cases.

Anatomical features

Although significant intra- and interindividual variability exists, the basic feature is that the brachial plexus is formed by the anterior spinal nerves from the four lowest cervical roots and the first thoracic root (Fig. 1). The spinal nerves result from a fusion of multiple small spinal rootlets originating from the anterior and posterior horns. The rootlets leading to the posterior horn have all passed through the cells in the spinal ganglion. The rootlets emanating from the anterior horn are axons emerging from cells in the grey matter of the anterior horn. Berthold *et al.* [6] described the junction between roots and spinal cord, identifying a central-peripheral transition zone lying outside the spinal cord at varying levels along the roots. The zone demarcates clear histological differences and is relevant to the level of intradural injury. This and the possibility of selective avulsion of either ventral or dorsal rootlets, makes it clear that different types of intradural lesion of the spinal roots may be present (Table 1). This accounts for the varying rates of recovery illustrating the terms partial and total avulsion [55].

On emerging from the foramen, the spinal nerves undergo a complicated process of joining and dividing of nerve elements to form the brachial plexus. The nerves unite to form three trunks: upper (C5 and C6), middle (C7), and lower (C8 and T1). Each trunk divides into two branches: anterior and posterior. The anterior branches (divisions) join to form the two anterior cords, lateral and medial. The musculocutaneous nerve and the lateral root of the median nerve stem from the lateral cord. The medial cord gives rise to the

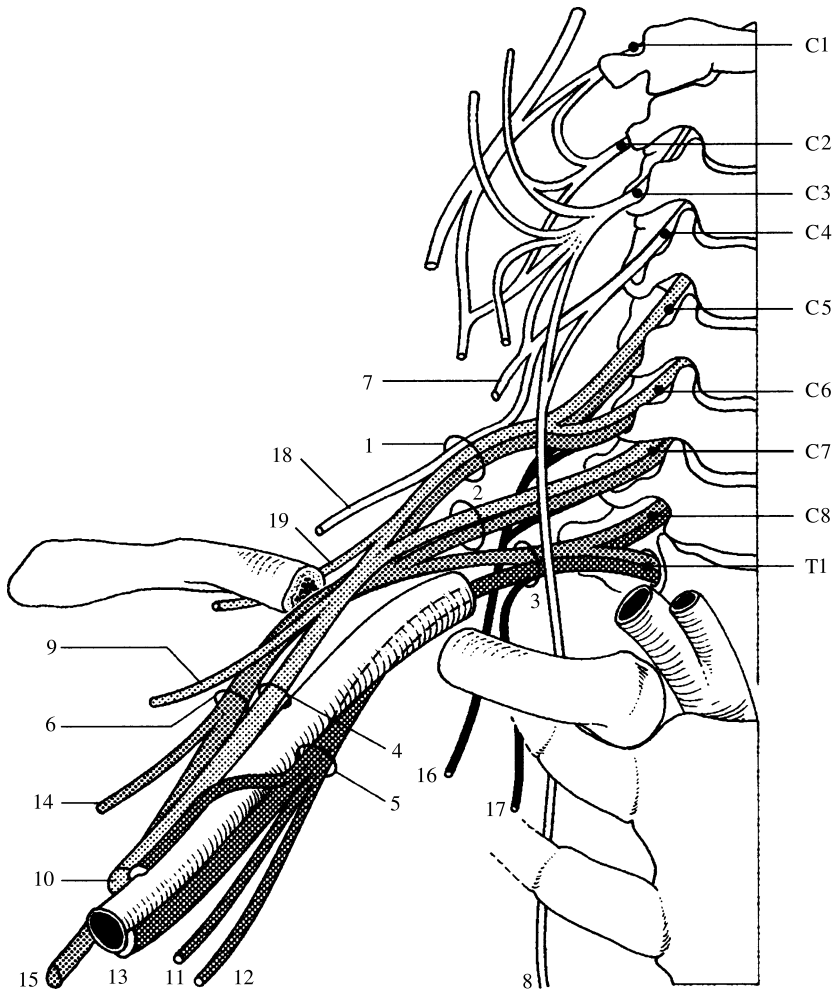


Fig. 1. Anatomical diagram of the brachial plexus and significant nerve branches (from Blaauw and Pons, 1999; by courtesy of De Tijdstroom). ■ Lateral cord, ▣ posterior cord, ▣ medial cord, 1 upper trunk (from C5 and C6), 2 middle trunk (from C7), 3 lower trunk (from C8 and D1), 4 lateral cord (from anterior divisions of upper and middle trunk), 5 medial cord (from anterior division of lower trunk), 6 posterior cord (from posterior divisions of all three trunks), 7 supraclavicular nerve, 8 phrenic nerve, 9 musculocutaneous nerve, 10 median nerve, 11 medial cutaneous antebrachial nerve, 12 medial cutaneous brachial nerve, 13 ulnar nerve, 14 axillary nerve, 15 radial nerve, 16 long thoracic nerve, 17 medial pectoral nerve, 18 dorsal scapular nerve, 19 suprascapular nerve

medial root of the median nerve, the ulnar nerve, the medial cutaneous nerve, and the medial antebrachial cutaneous nerve. The lateral and medial roots of the median nerve unite to form the median nerve. The three posterior divisions unite to form the posterior cord, which gives rise to the axillary and radial

Table 1. Classification of preganglionic injury shows the types graphically [55]. For example myelographic figure 3 is illustrative of type B4

Type	
A	Roots torn central to transitional zone. True avulsion
B	Roots torn distal to transitional zone
Type 1	Dura torn within spinal canal, DRG displaced in neck
Type 2	Dura torn at mouth of foramen: DRG more or less displaced
Type 3	Dura not torn. DRG not displaced
Type 4	Dura not torn. DRG not displaced, either ventral or dorsal roots intact

DRG Dorsal root ganglion.

nerves. For detailed information of anatomical features the reader is referred to the exemplary book by Kline *et al.* [37].

A lesion to the brachial plexus can be situated at any level from the origin of the nerve root to the division in the axillary region. The most common lesion is caused by traction and usually results in a combination of rupture of upper plexus elements and avulsion of one or several lower roots supplying the brachial plexus. Extraforaminal lesions are as a rule postganglionic lesions and are referred to as nerve ruptures.

Microscopic neuroanatomical examination of the posterior horns initially only defined a few well delimited regions, such as the substantia gelatinosa and Lissauer's zone. The unique pattern of spinal grey organization in the cat consisted of juxtaposed cell layers, as specified in the original description by Rexed [54]. The second layer corresponded exactly to the earlier description of the substantia gelatinosa Rolandi. Later, physiological experiments on the Rexed layers of the dorsal horn revealed their importance as specific regions for the modulation of sensory information from all regions of the body. Six Rexed layers are recognized, each with its particular cytological feature and typical cellular organization. The neurochemical features of each layer are also different. Afferent and specific nociceptive fibres take care of the input and modulate the neurochemical processes. Damage to the posterior horn deregulates the systems in the layers, causing the typical pain in non-obstetric brachial plexus injury.

Clinical features

Obstetric palsy

Clinical examination usually allows a diagnosis of the extent and level of the injury. In obstetric injury, the birth weight of the neonates born after breech delivery contrasts with that of the cases of brachial plexus palsy following vertex presentation [69], the latter cases being almost invariably macrosomic neonates.



Fig. 2. Boy with a bilateral upper palsy following breech delivery. On the left side the lesion shows early proximal recovery. On the same side he has a Horner's syndrome and a phrenic palsy, which required plication of the diaphragm

In most children, a functional disturbance of the arm is noted directly after birth, although in some the cause is initially suspected to be a fracture. A local haematoma may point to the site of the trauma. A fractured clavicle is more frequent than a fractured humerus. Dislocations and/or fractures of the cervical spine emphasize the traumatic birth. Sometimes there are also fractures of the lower limbs. Hemiphrenic nerve palsy may be present. Bilateral lesions mainly occur following breech delivery (Fig. 2); phrenic nerve lesions and especially bilateral brachial plexus lesions are typical of a breech delivery [13].

On the whole the cases can be divided into Narakas' classification system [46]. This classification is based on an examination 2–3 weeks of age and on the clinical course of children during the first 8 weeks after birth. These observations were correlated with nerve injuries classified in five degrees following Sunderland [64], and they are depicted in Table 2.

In a later publication Narakas classified the patients in four groups, because previously group IV was divided into two groups, but Narakas' experience showed that there were no significant differences between them regarding late sequelae [47]. Narakas' classification assesses the future of the palsy and excludes many mild cases, which recover full function in a matter of days.

Table 2. *Narakas classification of obstetric brachial plexus palsy*

	Clinical picture	Pathology grades (Sunderland's degrees [64])	Recovery
Type I	C5–C6	1 and 2	Complete or almost in 1–8 wks
Type II	C5–C6	Mixed 2 and 3	Elbow flexion: 1–4 wks Elbow extension: 1–8 wks
Type III	C7	Mixed 1 and 2	Limited shoulder: 6–30 wks
	C5–C6	4 or 5	Poor shoulder: 10–40 wks Elbow flexion: 16–40 wks
	C7	2 or 3	Elbow extension: 16–20 wks Wrist: 40–60 wks
Type IV	C8–T1 (no Horner's sign)	1	Hand complete: 1–3 wks
	C5–C7	4 and/or 5	Poor shoulder: 10–40 wks Elbow flexion: 16–40 wks
	C8	Mixed 2–3	Elbow extension: incomplete, poor: 20–60 wks or nil
Type V	T1 (temporary Horner's sign)	1 and 2	Wrist: 40–60 wks Hand complete: 20–60 wks
	C5–C7	5	Shoulder and elbow as above
	C7	or avulsed	
	C8	3 or avulsed	Wrist poor or only extension; poor flexion or none
	T1 C8–T1 (Horner's sign usually present)	2 and 3 avulsed	Very poor hand with no or weak flexors and extensors; no intrinsics

From Narakas AO (1986) *Injuries to the brachial plexus*. In: Bora FW Jr (ed) *The pediatric upper extremity: diagnosis and management*. WB Saunders, Philadelphia, p 247.

Group I: This includes paralysis of shoulder abduction and external rotation, of elbow flexion and of supination of the forearm resulting in adduction-endorotation posture of the shoulder, extension of the elbow and fixed pronation of the forearm. It is caused by a lesion to roots C5 and C6, or upper trunk, and is often called Duchenne's or Erb's paralysis (Fig. 3).

Group II: This is an extended Duchenne's/Erb's palsy: spinal nerve C7 is involved, as indicated by a palsy of the elbow, wrist and finger extensors and paralysis of the latissimus dorsi. The elbow may lie slightly flexed and waiter's tip position of the hand is pronounced (Fig. 4). In groups I and II pectoralis major, finger and thumb flexors are usually active, but muscle atrophy often



Fig. 3. A child with a C5–C6 lesion



Fig. 4. A child with a C5–C6–C7 lesion. Note waiter's tip position of hand



Fig. 5. A child with a total lesion

develops early. Distal sensation and vasomotor control are usually unaffected. In the majority of obstetric lesions there is paralysis of the upper roots only, of C5 and C6, or of C5, C6 and C7.

Group III: The injury has extended to spinal nerves C8 and T1. There is no Horner's syndrome. In some cases where the injury to the lower nerves of the brachial plexus is of a lesser degree, partial recovery of the hand function usually takes place and serious deficit of the muscles to shoulder and elbow persists. Sometimes the opposite is true and recovery of function is more pronounced proximally than distally. This is referred to as lower brachial plexus or Klumpke's palsy.

Group IV: There is (almost) complete motor and sensory deficit of the arm. A Horner's syndrome is present (Fig. 5).

Non-obstetric, traumatic palsy

In non-obstetric palsy we must take into account:

1. *The type of lesion:* is there a stretch and/or avulsion injury or is there a focal penetration or laceration involving the plexus as in gunshot wounds and a variety of mechanisms of injury such as those resulting from iatrogenic causes.

2. *Age:* although we obtained some surprising successes in older patients, recovery patterns after nerve surgery in children revealed a degree of recovery which was superior to that observed after nerve repair in adults. Sunderland reported on this issue in 1968 [64].

3. *Level of lesion*: the attitude to a total lesion is different from that to an upper plexus lesion; the latter is usually surgically treated at an early stage with the aim of controlling shoulder function and elbow flexion; infraclavicular lesions are more amenable to reconstructive surgery and thus seem to have a better prognosis.

4. *Time of surgery*: early repair has a greater chance of success, preferably no later than 2 months post-injury for blunt injuries and immediately for open injuries; repair carried out after more than 6 months is regarded as useless.

5. *Pain*: which is always present in preganglionic lesions, can sometimes be relieved by nerve transfers.

In traumatic brachial plexus palsy, important features to be obtained from the case history include the violence of the injury and the mode of application of force to the damaged limb.

Infraclavicular lesions due to shoulder dislocation or to humeral fracture are low impact lesions and they have a better prognosis than high impact lesions such as motor cycle accidents. The distinction between supra- and infraclavicular lesions due to a penetrating injury is not difficult but it is sometimes less clear, especially when a longitudinal force is present as in traction injuries. In these cases, double lesions may be present. There are usually special features which make it easier to distinguish between supra- and infraclavicular lesions.

The findings of sensory examination are very important. Partial or full sensory function in the presence of motor paralysis clearly points to good prognosis: neurapraxia is considered to be present. This may be found in skin areas supplied by one or several nerves or roots.

Partial or full sensory function in the presence of motor paralysis is indicative of a good prognosis.

Lesions in each group can be complete or partial and are often a combination of preganglionic (damaged roots) and postganglionic lesions. The latter can be supra- and retroclavicular lesions, in which case trunks are involved. Infraclavicular lesions involve damage to cords and/or nerves.

Special investigations

These include neurophysiological and image producing techniques, such as CT-myelography, MRI and sonography.

Neurophysiology

In *adult lesions*, the EMG can help in the analysis of the severity and extent of the lesion. For this purpose, the EMG investigation has to be based upon a

Table 3. Neurophysiological protocol*Sensory nerve action potentials (SNAPs)*

- Radial nerve to first digit (C6, upper trunk)
- Median nerve to first and third digit (C6, C7 upper and middle trunk)
- Ulnar nerve to fifth digit (C8, lower trunk)
- Medial (C8/T1, lower trunk) and lateral (C6, upper trunk) cutaneous nerves to forearm (not routinely in neonates)

Needle EMG

Muscle examined	Root	Trunk	Cord	Nerve(s)
Abductor pollicis brevis	(C7), C8, T1	(middle), lower	medial	median
First dorsal interosseus	C8, T1	lower	medial	ulnar
Flexor carpi radialis	(C6), C7 , (C8)	upper, middle , (lower)	lateral , medial	median
Flexor carpi ulnaris	C8, T1	lower	lateral, medial	ulnar
Extensor digitorum communis	(C6), C7 , C8	(upper), middle , lower	posterior	radial
Brachioradialis	(C5), C6	upper	posterior	radial
Biceps brachii	C5, C6	upper	lateral	musculocutaneous
Triceps brachii	(C6), C7 , C8	(upper), middle , lower	posterior	radial
Deltoid	C5 , C6	upper	posterior	axillary (circumflex)
Pectoralis major	C5, C6 , C7 , C8 , T1	upper , middle, lower	lateral, medial	lateral and medial pectoral
Infraspinatus	(C5), C6	upper		suprascapular
Trapezius				accessory
Rhomboids	C4, C5			dorsal scapular
Serratus anterior	C5, C6 , C7			long thoracic

The main innervation is indicated in normal and bold, bold indicating the most important; innervation sporadically found is shown between brackets.

combination of measuring the sensory nerve action potentials (SNAPs) for finding extraforaminal lesions and the needle-EMG for finding lesions of the motor fibres in a myotome (Table 3). If the lesion is complete, all muscles innervated by this root (normal SNAP), trunk or cord (both absent SNAP) will show pathological spontaneous muscle fibre activity. In the first case the sensory ganglion is positioned extramedullary and the lesion is preganglionic, thus the SNAP is normal. When the lesion is in the trunk or cord it is distal to the ganglion and SNAP is thus absent. Bonney and Gilliatt have as early as 1958 shown that in a preganglionic lesion there is evidence of conductivity in

the intact sensory axons (no Wallerian degeneration), but this may not have contact with the central nervous system [16]. Basically from each root, trunk or cord, at least 2 muscles innervated by different nerves should be sampled in order to pinpoint the exact location. Problems appear when there are multiple lesions, the most important of which is root avulsion. Root avulsion can be missed when there is also a more peripheral lesion in the same myotome. Using these basic principles, however, we were able to analyze correctly more than 80% of the adult traumatic brachial plexus lesions; sometimes we also found more distal lesions, information which proved important for the surgeon when deciding what to do during the operation [74].

In *obstetric lesions*, EMG can make an important contribution to the solution of the lesion's aetiology and the likely prognosis [51]. Generally, the neurophysiological data at 3–4 months of age seem to indicate a much smaller lesion than found during operation if interpreted in the same way as in adults. This stimulated an investigation into these differences. It was found that there are many differences between adults and neonates quite apart from the size:

1. The pathological spontaneous muscle fibre activity, sometimes also called denervation activity, appeared much earlier than in adults: in neonates as early as days 4–5, and also disappeared much sooner when collateral innervation was possible [19]. This means that for the EMG-analysis of a lesion, the search for reinnervation is even more important than just looking for pathological spontaneous muscle fibre activity.

2. Focusing on the biceps brachii muscle, innervation could be demonstrated from more roots than in adult patients. We called this “luxury innervation” [72]. In the biceps brachii muscle the C7 root appeared to be most important in neonates, whereas in adults innervation from this root of the biceps brachii muscle is only minimal or absent [73].

3. Also at the level of the muscle fibres, polyneural innervation could be demonstrated by the Groningen group of Gramsbergen *et al.* [32].

4. Using the somatosensory evoked potential technique (SEP), “luxury innervation” at the root level could also be demonstrated for the somatosensory pathways [20].

5. Smith [60] reported that mixed nerve action potentials (NAPs) of the median nerve appeared to offer prognostic information in obstetric lesions. She suggested an important role for electrodiagnosis in obstetric brachial palsy: firstly to determine the extent and level of involvement of individual components of the plexus; secondly to identify root avulsion; and thirdly, to identify the nature of the lesion in terms of neurapraxic, axonotmetic and neurotmetic injury, and thus to assist in making a prognosis [60].

These median nerve-NAPs give an estimation of the number of functioning nerve fibres, both sensory and motor. The fact that the median nerve contains many fibres from C7 also indicates the possibility of recovery of the

biceps brachii in neonates, based upon possible spontaneous reinnervation by the “luxury innervation”: a low amplitude of the median nerve-NAP means severe loss of C7 fibres, thereby loss of the “luxury innervation”, and hence loss of possibility of spontaneous reinnervation.

6. We think that central factors also play an important role in obstetric brachial palsy. Although innervation of muscles may exist – e.g. in root avulsions of both C5 and C6, the EMG may show considerable innervation of the biceps brachii in neonates, but not in adults; in most cases, this innervation cannot be used by the infant at the age of 4 months. Using magnetic cortical stimulation (Magstim) and measuring the motor evoked potentials (MEPs) from the biceps brachii muscle, we could demonstrate the existence of corticospinal connections to the “luxury innervation”, although the infant could not use these voluntarily. This indicates that cortical plasticity may be responsible for the, sometimes limited, spontaneous recovery seen in many of these infants.

Van Dijk *et al.* [70] also reported from their patient material, that EMG performed close to the time of possible intervention (3 months) usually reveals a discrepancy: motor unit potentials are seen in clinically paralyzed muscles. They explained this in five ways: an overly pessimistic clinical examination; overestimation of EMG recruitment due to small muscle fibres; persistent fetal innervation; developmental apraxia; or misdirection, in which axons reach inappropriate muscles.

Bisinella *et al.* [10] reported results of neurophysiological prediction of outcome in 73 children who showed slow recovery (biceps function returning after 3 months of age) and whose results of neurophysiological investigations were relatively favourable. Following the protocol defined by Smith in the London Peripheral Nerve Injury and Congenital Hand Unit, the cases were regarded as favourable on the basis of their NAP and EMG findings. These cases did not, therefore, undergo nerve surgery but later showed recovery of muscular function. A number did, however, require surgery for medial rotation contracture (11 cases) or posterior dislocation (21 cases) of the shoulder.

In summary: the EMG in neonates must be interpreted differently from that in adults, but the EMG can make important contributions to the likely prognosis [51]; also the search for reinnervation is very important. In case of doubt, the median nerve-NAP can provide prognostic information indicating the possibility of spontaneous recovery, even at an early age. We also tried to use the MEP for this purpose, but this technique did not give reliable information about prognosis below the age of 3–4 months.

Pathological spontaneous muscle fibre activity appears in neonates as early as 4–5 days following brachial plexus birth injury. Interpretation of electromyographic data in peripartal plexus lesions is quite different from the results gained from traumatic lesions.
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Myelography, CT-myelography, MRI and ultrasonography

Since the accidental demonstration of a traumatic meningocele in a patient who had sustained a brachial plexus injury [45], many radiological findings have been published. These were reviewed by Rankine [53]. CT-myelography remains useful for detecting intradural lesions i.e. total and/or partial avulsions (Figs. 6–8) particularly in obstetric cases. The accuracy of MRI for determining nerve root avulsion matches CT-myelography only in adults [22].

It remains to be seen whether ultrasonography can reliably identify post-ganglionic brachial plexus injuries, when significant soft-tissue disruption to the normal landmarks is expected. Another difficulty is that nerves may become

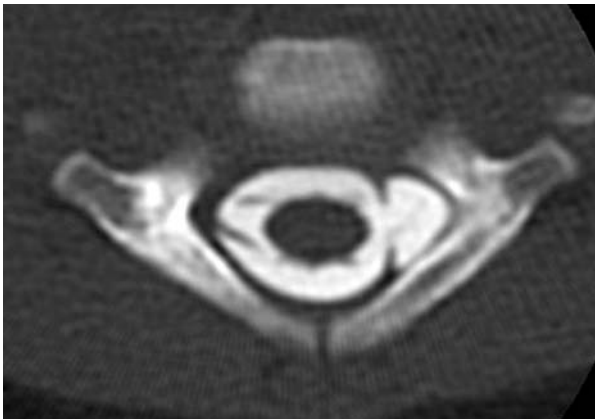


Fig. 6. At the left side dorsal and ventral rootlets are absent and a meningocele is present, illustrating the picture of complete root avulsion

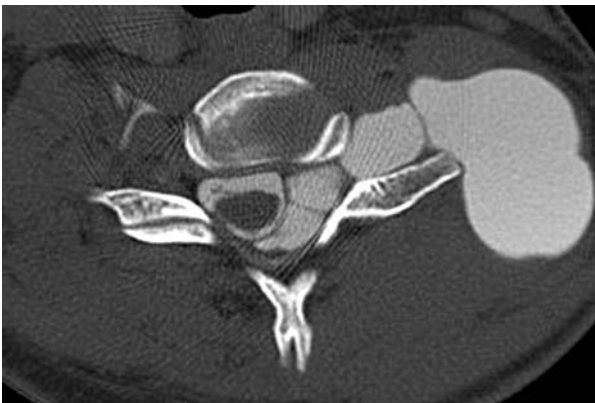


Fig. 7. Large traumatic meningocele in the presence of root avulsion extending into the soft tissues of the neck

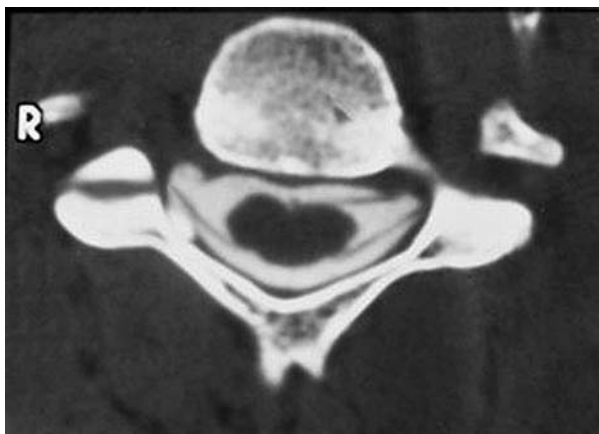


Fig. 8. Partial avulsion: at the right side the dorsal rootlet is normal, while the ventral rootlet is absent in the presence of a small traumatic meningocele

invisible when they are situated beneath bone such as the brachial plexus under the clavicle.

Fluoroscopy of the chest is necessary to confirm the absence of phrenic paralysis.

Indication and surgical approach

Obstetric palsy

When we advise surgery in a case of obstetric brachial plexus injury, it is important to operate at an age when there is no anaesthesia risk in a medical environment well suited to the care of the very young, because the risk of complication is high [38]. It is important to realise that obstetric brachial plexus injuries are mainly supraclavicular lesions. This means that we cope in general with two types of injury: upper and total lesions of roots and/or trunks.

In a recent extensive review on the natural history of obstetric brachial plexus palsy [52], no restriction for language was applied and articles published in medical periodicals not identified in the initial Medline query were added if these had appeared after 1965; a total of 1020 articles was found. Criteria for inclusion were as follows: the study had to be prospective; the study population had to be on a demographic basis; a follow-up of at least three years; and assessment of end-result preferably using a pre-defined assessment protocol. It was concluded that no study had the width of scope necessary to answer the expected natural course of obstetric brachial plexus palsy. Two studies which came closest to these strict criteria showed that 20–30% residual deficits can be expected. The authors of the review concluded that the often-cited excellent

prognosis for this type of birth injury could not be (considered to be) based on their criteria for scientifically sound evidence.

The often-cited excellent prognosis following the natural course for brachial plexus birth injury is not based on scientifically sound evidence.

The difficulty is that at present there are no universally accepted criteria for the categorization of function for surgical or non-operative treatment. Bae *et al.* [3] determined the intraobserver and interobserver reliability of the modified Mallet Classification [40], Toronto Test Score [41], and Hospital for Sick Children Active Movement Scale [21] in the evaluation of patients with brachial plexus birth palsy. They concluded that these three systems are reliable instruments and can be used to assess functional outcomes. They stressed that reliability does not imply validity of these instruments as this was not tested.

The modified Mallet Classification, the Toronto Test Score and the Active Movement Scale are reliable methods of quantifying upper-extremity function in patients with brachial plexus birth palsy and may be used clinically and to assess functional outcomes.

At present, surgical exploration of the brachial plexus is considered to be indicated if spontaneous recovery is insufficient at a preset age. Absence of biceps function at 3 months of age is regarded by many as the key indicator for surgery in upper brachial plexus lesions [27], in persisting total lesions, surgery should be performed earlier. Others use a combined score of different movements to decide whether nerve surgery may be performed at a later date but not later than 9 months [18].

Which methods of nerve surgery are available? These consist of nerve repairs and nerve transfers. Nerve repairs are possible when adequate proximal and distal stumps are available following resection of neuromas, but interposition of sural or other grafts is usually necessary to cover the gap. Nerve transfers are an adjunct if nerve repair is only partially possible (Tables 4 and 5). The transfer of an uninjured nerve to the distal stump of an injured nerve can be very successful. Favourable transfers are: accessory to suprascapular nerve for recovery of shoulder functions and medial pectoral branches to musculo-cutaneous nerve if the pectoralis muscle is contracting [12] or intercostals to musculo-cutaneous nerve if the pectoralis nerve is not working. Several other transfers are available, but these are not as widely used [8]. We do not believe that the sacrifice of such an important function as exerted by the hypoglossal nerve is balanced by the gain demonstrated in our series [14].

If an upper brachial plexus palsy is present, we often find a neuroma of the upper trunk, sometimes also of the middle trunk; the neuroma is resected until a healthy zone is reached. When avulsion is present this is recognized and it

Table 4. *Nerve transfers which are frequently used (Narakas [48])*

Nerve	Introduced by	Remarks
Spinal accessory nerve (No. XI)	Mentioned by Tuttle (U.S.A) in 1913. Reintroduced by Kotani (Japan) 1963	Frequently applied for the shoulder (spinati and deltoid) and for elbow flexors
Pectoral nerve	Stoffel (Germany) 1920, Lurje 1948	Particularly for elbow flexors
Intercostal nerves	Ciasserini (Italy), Yeoman and Seddon (1963), Kotani, Tsuyama, Hara perfected this method (1972)	Particularly for the musculocutaneous nerve but also for the median nerve and other nerves
Healthy parts of the brachial plexus itself or proximal roots (intraplexal neurotization) for elbow flexors	Harris en Low (U.K.) 1903, Narakas (1972)	This may cause synkineses

Table 5. *Infrequent transfers*

Nerve	Introduced by	Remarks
Hypoglossal nerve (No. XII)	Narakas (Switzerland) 1980, Slooff (Netherlands) 1992	Used for shoulder and elbow function. Serious clinical morbidity, poor results and abandoned (Blaauw <i>et al.</i> 2006)
Phrenic nerve	Gu (China) 1983	Not routinely used in Western countries. In OBPP now abandoned by Chen
Long thoracic nerve	Förster (Germany) 1928, Lurje (Russia) 1948, Narakas (Switzerland) 1972	Causes scapula alata and instability of the shoulder
Contralateral pectoral nerve or brachial plexus parts (particularly part of medial trunk)	Gilbert (France) 1984, Gu (China) 1989	Long transplants are necessary; associated movements

means that reconstruction to the proximal root is impossible. Fortunately C5 usually withstands avulsion and may thus be used for reinnervation.

In complete lesions the principle of repair is that everything should be done to improve hand function. As the lower roots are avulsed in most of these

cases, it will be necessary to use upper roots for reinnervation of distal arm muscles by connecting upper roots to the lower trunk.

In obstetric lesions the principle of repair is that everything should be done to improve hand function: hand function is more important than proximal extremity function.

Non-obstetric, traumatic brachial palsy

Emergency surgery is indicated in the case of life-endangering concomitant lesions, such as vascular trauma. At the time of repair of vascular and bone lesions the extent of the nerve lesion can be estimated and nerve surgery need not be delayed. If deemed necessary following initial surgery, the patient should be referred as quickly as possible to a centre capable of handling brachial plexus surgery. Penetrating injuries must also be surgically explored as soon as possible. Figure 9a and b are graphical representations of the management in

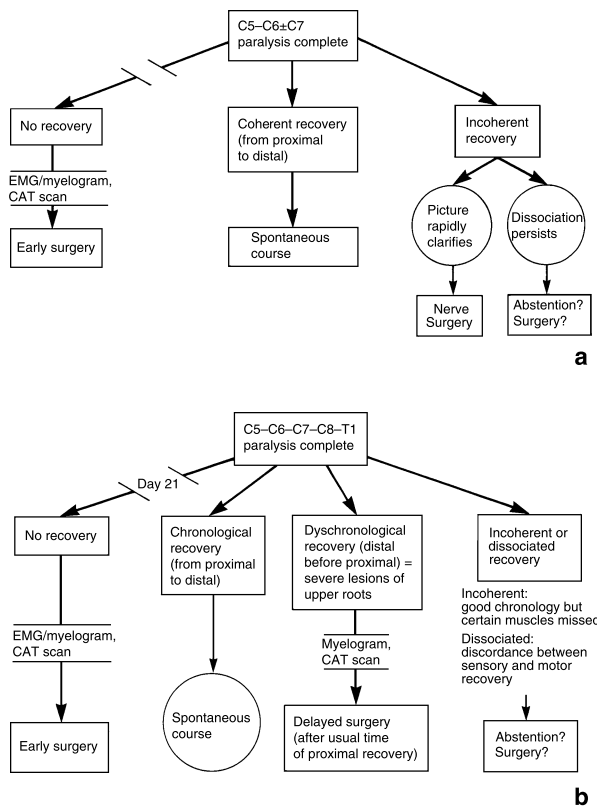


Fig. 9(a and b). Alnot's algorithms for the care of upper and total lesions

total and upper supraclavicular lesions [2]. Kline digresses extensively upon the significance of other injury categories [36].

Which techniques are currently used for reinnervation? This depends largely on the extent of the brachial plexus injury. Millesi has provided detailed graphic instructions concerning the surgical techniques [42]. In his book he shows the technique of nerve repair and the details of individual nerves and their significance.

Are root avulsions present and how many? Does the injury consist of rupture of nerve elements? Are they supra- and/or infraclavicular? It is important to realise that one is not dealing with a uniform type of plexus injury in all the patients. The brachial plexus injury is not a lesion in one nerve element but in a complex nerve structure. Thus the surgical treatment in an individual patient is a combination of the surgical possibilities and the functional goals to be achieved. We must accept that in the case of total lesions, which usually include multiple avulsions especially of the lower roots, manual function is seldom achieved following surgery.

Associated vascular lesions must be repaired immediately. Fractures and dislocations should be dealt with at the same time. Further treatment consists of the prevention of trophic changes and joint stiffness. Active mobilization is started as soon as possible, as is passive mobilization. The palsy is assessed by repeated motor and sensory examinations and by progression of Tinel's sign. If there are no signs of recovery from injury after a short period of time (± 3 weeks), cervical CT-myelography, MRI and neurophysiological studies are performed, because there is an indication for surgery.

Surgical treatment of brachial plexus injury patients was performed a few decades ago by Seddon and his co-worker Brooks and they demonstrated the efficacy of transfer of intercostal nerves to the musculocutaneous nerve, but the rationale and techniques of surgery in brachial plexus injuries may still be difficult to comprehend. Several surgical techniques are available but their application depends on functional goals and not on reinnervation of individual muscles. More vigorous attempts are made to achieve recovery of motor function than recovery of sensation. This is assumed to occur more or less simultaneously. Thus, following the evaluation of the patient's functional status, goals are formulated and means are sought.

Early surgery is essential; surgical treatment after 6 months is useless.

In cases with total paralysis, management depends on the presence of root avulsion. Myelography often shows traumatic meningoceles at C8 and T1, and often at C7; C6 may have an abnormal aspect, and C5 is usually normal. In these cases at surgery, the one or two normal roots are used to obtain proximal reinnervation. If only one root is to be grafted, repair of the suprascapular nerve and the lateral cord or the anterior division of the upper trunk is

performed. The goal is to obtain stabilization of the shoulder, an active pectoralis major muscle for thoraco-humeral grip, flexion of the elbow, and some palmar sensibility. Motor recovery of the hand will not be achieved. If two roots are available it is sometimes possible to graft parts of the posterior cord to improve radial nerve or axillary nerve function. If surgery reveals meningoceles on all roots, one will find total avulsion of C5–T1. In these cases neurotizations are performed using intercostal nerves, the spinal accessory nerve, or superficial cervical plexus in order to obtain some shoulder control and elbow flexion. In Table 3 a summary of nerve transfers is shown. Reimplantation of avulsed nerve roots may be useful for selected cases [17].

When spontaneous recovery takes place in the first month from proximal to distal, this may indicate that we have a case which can achieve significant recovery. This occurs especially in patients with shoulder dislocation. Early recovery from distal to proximal may indicate the presence of a repairable supraclavicular lesion which will often be operated on at an early stage. This management is graphically represented in Fig. 9a and b (from Alnot [2]).

In closed adult traumatic brachial plexus palsy, nerve surgery is usually postponed, but not for too long; early exploration is preferable when there is doubt about prognosis.

In cases of paralysis of C5, C6 ± C7, the prognosis is dominated by the fact that the hand is normal or only partially involved but can still be used. Surgery must be performed at an early stage and a satisfactory functional result is achieved by nerve repair or graft and/or nerve transfer. The results of direct reinnervation are better than those of (later) secondary surgery.

In infraclavicular lesions distal to the level of the terminal branches of the plexus, such as the axillary nerve, the musculocutaneous nerve, and the radial nerve, a sensory and motor paralysis implies a complete rupture, and it is necessary to operate before the third month. In lesions at the level of the cords, diagnosis and indication for surgery may be difficult.

In adult complete traumatic brachial plexus palsy, the treatment goal is to obtain stabilization of the shoulder, an active pectoralis major for thoraco-humeral grip, active flexion of the elbow, and some palmar sensibility.

Secondary surgical techniques

Primary (nerve) surgery is followed by careful observation of functional recovery. At least two years must elapse before the results of brachial plexus repair can be properly analyzed, because reinnervation after grafting or following nerve transfer is always a long process. Amongst other things it depends on

the age of the patient, the time between injury and surgical repair, the type of injury and the distance between graft and end-organ.

When the results of reinnervation are clear, the requirement to improve shoulder-, elbow-, wrist- and hand function is evaluated. What are the options for improving these functions?

Obstetric lesions

It was expected that primary surgery would diminish the sequelae of obstetric birth palsy. Although primary surgery achieved greater functionality, a number of joint problems persisted due to imbalance of muscular function which may cause serious malformations during growth, in particular gleno-humeral deformities.

Upper brachial plexus lesion (Erb's palsy) is the most common type. An isolated lower lesion (so-called Klumpke's palsy, although she did not in fact describe this) is extremely rare (if at all present [1]); it may be the clinical presentation of a total lesion with significant recovery of the upper part of the brachial plexus and persisting palsy of the lower plexus parts. Thus we are left with upper lesions and total lesions with lesion of upper plexus elements as the common denominator; hence it is understandable that impairments and disabilities by glenohumeral deformities are most common, although they are less frequent in patients who had early nerve surgery. It is, therefore, important to offer nerve reconstruction when the prognosis is known.

At the beginning of the last century, when nerve reconstruction was not yet widely practiced, the occurrence of a fixed internal rotation contracture of the shoulder in 40–60% of cases was described [35, 57, 67, 68, 75]. In 1934, L'Episcopo [39] concluded that the disability resulting from upper obstetrical palsy is in fact often due to a severe internal contracture of the shoulder and this opinion is still valid.

In obstetric birth palsy, prevention of internal rotation contracture of the shoulder is very important because the contracture may give rise to a number of serious gleno-humeral deformities.

From these experiences we are familiar with many aspects of the shoulder problems and some surgical solutions. Neurosurgeons are usually not trained to offer these surgical solutions, and so hand-, plastic- or orthopaedic surgeons are often called in to assist. A number of deformities may also develop secondarily, such as:

1. Posterior subluxation of the humeral head: passive lateral rotation of the arm in the shoulder is restricted. There is no secondary deformity of acromion, coracoid or glenoid.

2. Posterior dislocation; the humeral head can be seen and palpated behind the glenoid. Reposition of the humeral head is possible causing a “click”. Passive lateral rotation is more restricted.

3. Complex subluxation/dislocation of the humeral head: at this stage marked skeletal deformities are present such as elongated coracoid, elongated and downward hooked acromion, bifacetal glenoid, retroversion of the glenoid, torsion deformity of the humerus. Passive external rotation is usually impossible [8].

It is clear that prevention of internal rotation deformity of the shoulder is necessary in obstetric brachial plexus palsy. Thus the parents are instructed to exercise the arm. Sometimes this is not sufficient to prevent contracture, necessitating surgical measures. First of all, persisting shoulder impairment due to internal rotation contracture and concomitant external rotation weakness around the shoulder needs attention. Subscapular release or subscapular tendon lengthening is sometimes required at an early stage when contracture is evident. Then patients with rotator cuff muscle imbalance, minimal joint contracture and no glenohumeral deformity are treated with latissimus dorsi transfer, sometimes with additional teres major tendon transfer or humeral derotation osteotomy. Patients with a glenohumeral deformity or dislocation are treated with some form of surgical reduction supplemented by tendon transfers and musculocutaneous lengthenings.

In the elbow, insufficient flexion, extension or pro- and supination may be present. For elbow flexion a flexor-pronator plasty (Steindler's operation [63]) may be indicated. Our group reported results in 26 patients, showing a good result in 23 cases [71]. To improve pronation, especially in supination contracture, osteotomy of the radius is undertaken, forcing the hand in pronation, eventually supplemented with biceps rerouting transfer. Elbow extension is usually sufficiently enhanced by the force of gravity.

Weakness of wrist and/or finger extension is a recurring problem both in upper brachial plexus lesions and in total lesions following initial nerve surgery. If strong wrist and finger flexors are present, it is possible to deal with this problem using tendon transfer. The results of hand function following nerve reconstruction in total lesions are often modest but are greatly appreciated by the patients and their environment and are useful [34]. Secondary surgery is not standardized for these patients, but is tailored to the functional result in the individual patient.

Non-obstetric lesions

Secondary surgery in patients with brachial plexus palsy who underwent primary surgery is intended to achieve greater functional results; only in obstetric palsy is it also performed to redress malfunction due to growth disturbances.

Thus growth does not cause a problem in the non-obstetric palsy group and as the results of primary surgery are not as good as in obstetric cases, the role of secondary surgery is not equal in the treatment procedure.

If sufficient shoulder function is not regained following nerve surgery, arthrodesis of the shoulder is a useful alternative. Normal function of the trapezius and serratus muscles is required. The strength of the arthrodesed shoulder is better than that obtained by muscular transfers. The procedure is indicated when at least some recovery of elbow function is achieved.

Elbow flexion plays a key role in the global function of the upper extremity. If, following primary surgery, elbow flexion is not regained, surgery is mandatory. In upper palsies a flexor-pronator plasty (Steindler's operation) is indicated and is usually successful, when active wrist extension is adequate. If this operation is not possible, bipolar latissimus dorsi transfer or triceps-to-biceps transfer may be considered.

Especially for the distal motor functions of the paralyzed arm, free muscle transfers may be undertaken. In special cases wrist and finger flexion and extension may be regained [22].

It is our contention that amputation is very seldom indicated and should not be undertaken because of pain. Functional braces are rarely utilized, and cosmetic prostheses for social use are always less satisfactory than the paralyzed arm.

Results of both primary and secondary surgery

The results of brachial plexus reconstruction in adults are generally modest, especially in cases of total lesions. Although the same techniques are used in neonates the results are far better, because of shorter regeneration distances, stronger potential for regeneration, and the greater capacity for brain adaptation.

Obstetric lesions

One of the biggest problems is being able to compare the results of the different treatment policies because of lack of consensus about the method of assessment and how to use the various scoring systems. This of course makes it complicated to compare, for example, the results of a more conservative attitude in treating obstetric palsy with a more aggressive surgical approach. The Brachial Plexus Group in Heerlen uses several assessment methods [44]. Bae *et al.* [3] have demonstrated that the modified Mallet Classification [40], the Toronto Test Score [41] and the Active Movement Scale [21] are reliable methods of quantifying upper-extremity function in patients with brachial plexus birth palsy and may be used clinically and to assess functional outcomes.

Using the Mallet scale, Gilbert [29] reviewed 237 patients who had undergone brachial plexus reconstruction before 1996. Of these, 103 patients were operated for C5–C6 lesions. Including those patients (one-third) who had required secondary surgery, he concluded that 80% had a good or excellent result (grades IV–V) and 20% were average (grade III). In 61 patients who had C5–C6–C7 defects (7 patients had subscapularis release and 25 had tendon transfers for the shoulder following primary surgery), the results were excellent or good (grade IV–V) in 61%, average (grade III) in 29%.

Complete paralysis and associated root ruptures and avulsions are severe, and the results cannot be evaluated before completion of growth. From Gilbert's patient group, a series of 73 patients with complete paralysis operated on from 1978 to 1994 were followed with a mean follow-up of 6.4 years [34]. Secondary operations (mainly on the shoulders) were necessary on 123 occasions. Although the results show that the shoulders and elbows did not do as well as in upper-type lesions, because in these cases upper roots were used to reinnervate lower roots, the results at the level of the hand were encouraging, showing 75% useful results after 8 years, even in patients with avulsion injuries of the lower roots. In this paper, scoring scales for elbow and hand function were used.

In our series of more than 500 neurosurgical obstetric cases, we examined functional results of 171 cases who had undergone accessory nerve transfer to the suprascapular nerve with at least a 2-year follow-up. We evaluated active external glenohumeral rotation, because this is particularly exerted by the infraspinatus muscle which is innervated by the suprascapular nerve and thus reflects specifically the result of the transfer. Results were evaluated in patients who had had the transfer more than 24 months earlier using the Mallet scale. In 4 cases, data were lost and 14 cases, which had required a latissimus dorsi transfer to improve external rotation, had to be excluded. Thus, we could evaluate 153 cases. Results were not good in 47 cases (Mallet scale 2 in 38 cases (22%) and Mallet scale 3 in 9 cases (5%)), but in 106 cases (62%) the Mallet scale was 4. Possible reasons for the failures could be technical, lack of neurotization of the infraspinatus muscle for unknown reason, or lack of central learning.

Non-obstetric lesions

As mentioned earlier and supported by international colleagues the results in severely injured patients are modest [15], partly because surgery is often performed late and now we know that timing is everything. In these cases we aim to achieve some recovery of shoulder and elbow function, in particular elbow flexion, and in a few cases – especially in adolescents – some strength is regained in wrist flexors and extensors. A certain degree of protective sensibility in the hand may also be regained but the distal muscles remain paralysed.

Particularly in upper (partial) palsies the ability to actively move the shoulder and elbow so that the good and functionally not injured wrist and hand may be used, is important. Thus surgical measures to induce key functions in the shoulder and elbow either by nerve surgery sometimes followed by secondary surgery or by secondary surgery alone, are essential.

Summary of management of patients with brachial plexus lesions

In *obstetric lesions* Gilbert's biceps rule in upper lesions is applied, while total lesions can be operated earlier. Thus we recommend that surgery should take place before three months of age for global palsy cases and at three months in upper (Erb's type) lesions, for best functional results. Even in complete lesions the end results at the level of the hand are encouraging [29]. When spontaneous recovery takes place in the first month from proximal to distal this may indicate that we have a case which can achieve significant recovery. In partial lesions operation is advised when paralysis of abduction of the shoulder and of flexion of the elbow persists after the age of three months and neurophysiological investigations predict a poor prognosis. Operation is carried out earlier in complete lesions showing no sign of clinical recovery [9].

In *non-obstetric* lesions, associated vascular lesions must be repaired immediately at the same time as the nerves [8]. In closed lesions without a vascular rupture, nerve surgery is usually postponed, but not too long and early exploration is preferable when there is doubt concerning prognosis. Fractures and dislocations should also be dealt with early. Further treatment consists of the prevention of trophic changes and joint stiffness. Active mobilization is started as soon as possible, as is passive mobilization. The palsy is assessed by repeated motor and sensory examinations, and by progression of Tinel's sign. Tinel's sign on day 1 is an early indication of the presence of nerve rupture.

If recovery does not take place during the first weeks following the injury, this may be an urgent indication to operate, because otherwise it may be too late to achieve good results. When there is a surgical indication, cervical CT-myelography and neurophysiological studies are performed.

Pain following traumatic brachial plexus injury

Pain is common after non-obstetric traction lesions of the brachial plexus. Also postganglionic lesions are not associated with pain. Pain almost exclusively can create a serious problem in patients with avulsion of the lower roots supplying the brachial plexus. It seems reasonable to suppose that the more

violence exerted on the spinal cord, the greater the deafferentation and the greater the change in abnormal circuits after sprouting, with a larger number of cells firing spontaneously and upsetting some of the Rexed layers in the posterior horn.

In 1911, Frazier and Skillern [26] set out to perform dorsal rhizotomy in a patient, a physician, with intractable pain from closed traction lesion of the brachial plexus. They exposed the cervical cord by laminectomy and found the anterior and posterior rootlets of C6, C7 and C8 to be absent: “the roots evidently had been torn completely from the cord”.

The patient’s description of the pain was characteristic: “the pain is continuous; it does not stop a minute either day or night. It is either burning or compressing . . . In addition, there is, every five minutes, a jerking sensation similar to that obtained by touching . . . a Leyden jar. It is like a zigzag made in the sky by a stroke of lightning. The upper part of the arm is mostly free of pain; the lower part from a little above the elbow to the tip of the fingers, never.”

It is all there, the characteristic pain in extensive traumatic brachial plexus palsy. The pain is severe, there are two parts to it (one constant, the other intermittent and unpredictable), and it is worst in the hand and forearm. The typical pain following brachial plexus injury often develops very quickly, within hours, but in some cases not until several weeks after the accident. It is not clear whether the neurochemical mechanisms underlying these pain sensations are the same in all these conditions.

Treatment with tegretol, tryptizol, peripheral analgesics or opiates frequently only has limited success. Cannabis seems to be more effective. Alcohol is helpful, probably because it relaxes the patient and helps to induce sleep. A fairly characteristic feature is that a really good night’s sleep can often give marked relief of pain. None of our patients have become alcoholics or drug addicts because they soon learned that apart from other social and psychological mechanisms, work is the best method of pain relief, and they cannot therefore risk being drowsy.

An expectative management is adopted in all these cases because the majority of the patients will be able to cope with the situation with acceptable remaining pain one year after the injury, only for it to return in situations of stress, underlying illness, or marked changes in the weather. Surgical repairs and reconstructions are regarded as playing a role in the diminution of pain. About 10% of the patients request further measures one year or more after the injury because of intractable pain. According to Wynn Parry [76], the most useful modality for the management of avulsion pain was regarded to be prolonged transcutaneous electrical stimulation, but we now doubt this. Berman *et al.* [5] concluded that nerve repair can reduce pain from spinal root avulsions and that the mechanism may involve successful regeneration,

and/or restoration of peripheral connections prior to their function, possibly in muscle.

Sadly, there is a significant number of patients who do not respond to nerve reconstruction, electrical stimulation, drugs, or distraction by work and hobbies and who do not lose their pain over a period of time.

The hypothesis that abnormal electrophysiological phenomena in the posterior horn are responsible for the pain syndromes, has led to the introduction of neuromodulation on the one hand, and on the other to surgical destruction of circumscribed areas in the posterior horn by a so-called DREZ-lesion (Dorsal Root Entry Zone-lesion).

We have used neuromodulation in a certain number of cases with only modest success. An important condition for optimal pain diminution is a complete overlap of the pain area and the area in which paraesthesias are achieved by stimulation. This congruence is rarely reached, and so in almost all cases insufficient pain diminution occurred. This is probably caused by the deafferentation which arises following avulsion of multiple roots causing atrophy of the posterior horn. Although in some cases the patients achieved pain diminution of more than 70%, they became dissatisfied with this result in the course of their ailment, making them less motivated to continue neuromodulation treatment.

In 1974, Sindou *et al.* [58] described the neuroanatomical basis for DREZ lesioning as well as certain clinical indications for the procedure. Nashold and Ostdahl indicated the possibility that thermal destruction of the denervated neurones in the posterior horn could be a solution for persisting pain in brachial plexus lesions [49]. Thomas and Kitchen [66] concluded from their follow-up study that DREZ thermocoagulation is an effective procedure for relieving deafferentation pain. Sindou thinks that the long-term efficacy of this procedure strongly indicates that pain after brachial plexus avulsion originates from the deafferented (and gliotic) dorsal horn [59].

Dorsal root entry zone thermocoagulation is an effective procedure for relieving deafferentation pain following brachial plexus injury.

We agree that long-term pain relief is good or complete in a high percentage of these patients. However, DREZ lesioning is an invasive procedure and we have found persistent, albeit mild, neurological deficits at the time of follow-up. The side-effects are rarely severe or functionally significant, but a few patients who are suffering permanent side-effects – locomotor deficits, in particular, impotence and proprioceptive loss – have doubts as to whether they were right in choosing the operation, because patients soon forget having had pain. It is useful to make a recording of the patient's feelings about his pain before the operation so that, if necessary, he can be reminded of his once desperate state.

Acknowledgements

We are grateful to Professor Rolfe Birch, London, UK, for his helpful discussions. Mr Koen Koenders, Oud-Turnhout, Belgium, assisted in the preparation of the illustrations.

References

1. Al-Qattan MM, Clarke HM, Curtis CG (1995) Klumpke's birth palsy: does it really exist? *J Hand Surg (Br)* 20: 19–23
2. Alnot J-Y (1988) Traumatic brachial plexus palsy in adults. In: Raoul Tubiana (ed) *The hand*, vol. III. WB Saunders, Philadelphia, pp 607–644
3. Bae DS, Waters PM, Zurakowski D (2003) Reliability of three classification systems measuring active motion in brachial plexus birth palsy. *J Bone Joint Surg* 85: 1733–1738
4. Bager B (1997) Perinatally acquired brachial plexus palsy – a persisting challenge. *Acta Paediatr* 86: 1214–1219
5. Berman JS, Birch R, Anand P (1998) Pain following human brachial plexus injury with spinal cord avulsion and the effect of surgery. *Pain* 75: 199–207
6. Berthold CH, Carlstedt T, Corneliuson O (1993) Anatomy of the mature transitional zone. In: Dyck PJ, Thomas PK, Griffin JW, Low PA, Poduslo JF (eds) *Peripheral neuropathy*, 3rd edn. WB Saunders, Philadelphia, pp 75–80
7. Birch R (1996) Brachial plexus injuries. *J Bone Joint Surg* 78-B: 986–992
8. Birch R, Bonney G, Wynn Parry CB (1998) *Surgical disorders of the peripheral nerves*. Churchill Livingstone, Edinburgh, pp 398–404
9. Birch R, Ahad N, Kono H, Smith (2005) Repair of obstetric brachial plexus palsy. Results in 100 children. *J Bone Joint Surg (Br)* 87-B: 1089–1095
10. Bisinella GL, Birch R, Smith SJM (2003) Neurophysiological prediction of outcome in obstetric lesions of the brachial plexus. *J Hand Surg* 28B: 148–152
11. Blaauw G, Pons C (1999) *Letsels van de plexus brachialis*. Elsevier/Bunge, Maarssen, pp 36–38
12. Blaauw G, Slooff ACJ (2003) Transfer of pectoral nerves to the musculocutaneous nerve in obstetric upper brachial plexus palsy. *Neurosurgery* 53: 338–341
13. Blaauw G, Muhlig RS, Kortleve JW, Tonino AJ (2004) Obstetric brachial plexus injuries following breech delivery: an adverse experience in The Netherlands. *Semin Plast Surg* 18: 301–307
14. Blaauw G, Sauter Y, Lacroix CLE, Slooff ACJ (2006) Hypoglossal nerve transfer in obstetric brachial plexus palsy. *J Plast Reconstr Aesthetic Surg* 59: 474–478
15. Bonnard C, Allieu Y, Alnot J-Y, Brunelli G, Merle M, Santos-Palazzi A, Sedel L, Raimondi PL, Narakas A (1996) Complete palsies and supraclavicular lesions. In: Alnot J-Y, Narakas A (eds) *Traumatic brachial plexus injuries, Monographie de la Soci t  Fran aise de Chirurgie de la Main (GAM)*. Expansion Scientifique Fran aise, Paris, pp 126–155
16. Bonney G, Gilliatt RW (1958) Sensory nerve conduction after traction lesion of the brachial plexus. *Proc R Soc Med* 51: 365–367
17. Carlstedt T, Anand P, Hallin R, Misra PV, Nor n G, Seferlis T (2000) Spinal nerve root repair and reimplantation of avulsed ventral roots into the spinal cord after brachial plexus injury. *J Neurosurg Spine* 93: 237–247

18. Clarke HM, Curtis CG (1995) An approach to obstetrical brachial plexus injuries. *Hand Clin* 11: 563–581
19. Colon AJ, Vredevelde JW, Blaauw G, Zandvoort JA (2003) Spontaneous muscle fiber activity appears early in cases of obstetric brachial plexopathy. *Muscle Nerve* 28: 515–516
20. Colon AJA, Vredevelde JW, Blaauw G, Slooff ACJ, Richards R (2003) Extensive somatosensory innervation in infants with obstetric brachial palsy. *Clin Anat* 16: 25–29
21. Curtis C, Stephens D, Clarke HM, Andrews D (2002) The active movement scale: an evaluative tool for infants with obstetrical brachial plexus palsy. *J Hand Surg* 27A: 470–478
22. Doi K (2001) Palliative surgery: free muscle transfers. In: Gilbert A (ed) *Brachial plexus injuries*. Martin Dunitz, London, pp 137–147
23. Doi K, Otsuka K, Okamoto Y, Fujii H, Hattori Y, Baliarsing AS (2002) Cervical nerve root avulsion in brachial plexus injuries: magnetic resonance imaging classification and comparison with myelography and computerized tomography myelography. *J Neurosurg* 96 Suppl 3: 277–284
24. Evans-Jones G, Kay SPJ, Weindling AM, Cranny G, Ward A, Bradshaw A, Hernon C (2003) Congenital brachial palsy: incidence, causes, and outcome in the United Kingdom and republic of Ireland. *Arch Dis Child Fetal Neonatal Ed* 88: F185–F189
25. Fairbank HAT (1913) Birth palsy: subluxation of the shoulder-joint in infants and young children. *Lancet* 1: 1217–1223
26. Frazier CH, Skillern PF (1911) Supraclavicular subcutaneous lesions of the brachial plexus not associated with skeletal injuries. *J Am Med Ass* 57: 1957–1963
27. Gilbert A, Tassin JL (1984) Réparation chirurgicale du plexus brachial dans la paralysie obstétricale. *Chirurgie* 110: 70–75
28. Gilbert A (1995) Long-term evaluation of brachial plexus surgery in obstetrical palsy. *Hand Clin* 11: 583–594
29. Gilbert A (2001) Results of repair to the brachial plexus. In: Gilbert A (ed) *Brachial plexus injuries*. Martin Dunitz, London, pp 211–215
30. Gilbert A, Pivato G, Kheiralla T (2006) Long-term results of primary repair of brachial plexus lesions in children. *Microsurgery* 26: 334–342
31. Gordon M, Rich H, Deutschberger JD, Green M (1973) The immediate and long-term outcome of obstetric birth trauma. *Am J Obstet Gynecol* 117: 51–56
32. Gramsbergen A, IJkema-Paassen J, Nikkels PGJ, Hadders-Algra M (1997) Regression of polyneuronal innervation in the human psoas muscle. *Early Hum Dev* 49: 49–61
33. Greenwald AG, Schute PC, Shively JL (1984) Brachial plexus birth palsy: a 10-year report on the incidence and prognosis. *J Pediatr Orthop* 4: 689–692
34. Haerle M, Gilbert A (2004) Management of complete obstetric brachial plexus lesions. *J Pediatr Orthop* 24: 194–200
35. Hoffer MM, Phipps GJ (1998) Closed reduction and tendon transfer for treatment of dislocation of the glenohumeral joint secondary to brachial plexus birth palsy. *J Bone Joint Surg* 80A: 997–1001
36. Kline DG, Hudson AR (1995) Nerve injuries. Operative results for major nerve injuries, entrapments, and tumors. W.B. Saunders, Philadelphia, pp 345–471
37. Kline DG, Hudson AR, Kim DH (2001) Atlas of peripheral nerve surgery. W.B. Saunders, Philadelphia, pp 1–75
38. La Scala GC, Rice SB, Clarke HM (2003) Complications of microsurgical reconstruction of obstetrical brachial plexus palsy. *Plast Reconstruct Surg* 111: 1383–1390

39. L'Episcopo JB (1934) Tendon transplantation in obstetrical paralysis. *Am J Surg* 25: 122
40. Mallet J (1972) Paralysie obstetricale du plexus brachial: traitement des sequelles. Primaute du traitement de l'épaule – Méthode d'expression des résultats. *Rev Chir Orthop Reparatrice Appar Mot* 58 Suppl 1: 166–168
41. Michelow BJ, Clarke HM, Curtis CG, Zuker RM, Seifu Y, Andrews DF (1994) The natural history of obstetrical brachial plexus palsy. *Plast Reconstruct Surg* 93: 675–681
42. Millesi H (1992) *Chirurgie der peripheren Nerven*. Urban & Schwarzenberg, München, pp 79–120
43. Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L (2005) Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstetric Gynecol* 106: 913–918
44. Muhlig RS, Blaauw G, Slooff ACJ, Kortleve JW, Tonino AJ (2001) Conservative treatment of obstetrical brachial plexus palsy (obpp) and rehabilitation. In: Gilbert A (ed) *Brachial plexus injuries*. Martin Dunitz, London, pp 173–187
45. Murphey F, Hartung W, Kirklin JW (1947) Myelographic demonstration of avulsing injury of the brachial plexus. *Am J Roentgenol* 58: 102–105
46. Narakas AO (1986) Injuries to the brachial plexus. In: Bore FW (ed) *The pediatric upper extremity. Diagnosis and management*. W. B. Saunders Company, Philadelphia, pp 247–258
47. Narakas AO (1987) Obstetrical brachial plexus injuries. In: Lamb DW (ed) *The paralysed hand. (The hand and upper limb, vol. 2)*. Churchill Livingstone, Edinburgh, pp 116–135
48. Narakas AO (1991) Neurotization in the treatment of brachial plexus injuries. In: Gelberman RH (ed) *Operative nerve repair and reconstruction*. JB Lippincott Company, Philadelphia, pp 1329–1358
49. Nashold BS Jr, Ostdahl RH (1979) Dorsal root entry zone lesions for pain relief. *J Neurosurg* 51: 59–69
50. Ostdahl RH (1996) DREZ surgery for brachial plexus avulsion pain. In: Nashold BS, Pearlstein RD (eds) *The DREZ operation*. The American Association for Neurological Surgeons, Illinois, pp 105–124
51. Pitt M, Vredevelde JW (2005) The role of electromyography in the management of the brachial plexus palsy of the newborn. *Clin Neurophysiol* 116: 1756–1761
52. Pondaag W, Malessy MJA, Van Dijk JG, Thomeer RTWM (2004) Natural history of obstetric brachial plexus palsy: a systematic review. *Dev Med Child Neurol* 46: 138–144
53. Rankine JJ (2004) Adult traumatic brachial plexus injury. *Clin Radiol* 59: 767–774
54. Rexed D (1952) The cytoarchitectonic organization of the spinal cord in the cat. *J Comp Neurol* 96: 414–495
55. Schenker M, Birch R (2001) Diagnosis of the level of intradural rupture of the rootlets in traction lesions of the brachial plexus. *J Bone Joint Surg* 83-B: 916–920
56. Seddon H (1972) *Surgical disorders of the peripheral nerves*. Churchill Livingstone, Edinburgh London, pp 32–36, 174–198
57. Sever JW (1918) The results of a new operation for obstetrical paralysis. *Am J Orthop Surg* 16: 248–257
58. Sindou MP, Fischer G, Goutelle A, Mansuy L (1974) La radicellotomie posterieure selective. Premiers resultats dans la chirurgie de la douleur. *Neurochirurgie* 20: 391–408
59. Sindou MP, Blondet E, Emery E, Mertens P (2005) Microsurgical lesioning in the dorsal root entry zone for pain due to brachial plexus avulsion: a prospective series of 55 patients. *J Neurosurg* 102: 1018–1028

60. Smith SJM (1996) The role of neurophysiological investigation in traumatic brachial plexus lesions in adults and children. *J Hand Surg* 21: 145–148
61. Smith SJM (1998) Electrodiagnosis. In: Birch R, Bonney G, Wynn Parry CB (eds) *Surgical disorders of the peripheral nerves*. Churchill Livingstone, Edinburgh, pp 482–487
62. Specht EE (1975) Brachial plexus palsy in the newborn. Incidence and prognosis. *Clin Orthop* 110: 32–34
63. Steindler A (1918) A muscle plasty for the relief of flail elbow in infantile paralysis. *Inter Med J* 35: 235–241
64. Sunderland S (1968) *Nerves and nerve injuries*. Churchill Livingstone, Edinburgh London, 633 pp
65. Tan KL (1973) Brachial palsy. *J Obstet Gynaecol Br Commonw* 80: 60–62
66. Thomas DGT, Kitchen ND (1994) Long term follow up of dorsal root entry zone lesions in brachial plexus avulsion. *J Neurol Neurosurg Psychiatr* 57: 737–738
67. Torode I, Donnan L (1998) Posterior dislocation of the humeral head in association with obstetrical paralysis. *J Pediatr Orthop* 18: 611–615
68. Troum S, Floyd WED, Waters PM (1993) Posterior dislocation of the humeral head in infancy associated with obstetrical paralysis. A case report. *J Bone Joint Surg* 75-A: 1370–1375
69. Ubachs JHM, Slooff ACJ, Peeters LLM (1995) Obstetric antecedents of surgically treated obstetric brachial plexus injuries. *Br J Obstet Gynaecol* 102: 813–817
70. Van Dijk JG, Pondaag W, Malessy MJ (2001) Obstetric lesions of the brachial plexus. *Muscle Nerve* 24: 1451–1461
71. Van Egmond C, Tonino A, Kortleve JW (2001) Steindler flexorplasty of the elbow in obstetric brachial plexus injuries. *J Pediatr Orthop* 21: 169–173
72. Vredevelde JW, Blaauw G, Slooff ACJ, Richards R (1997) “Luxury innervation”: does it exist and play a role at the time of birth? *Electroencephalogr Clin Neurophysiol* 107: 1P
73. Vredevelde JW, Blaauw G, Slooff ACJ, Richards R, Rozeman CAM (2000) The findings in paediatric obstetric brachial palsy differ from those in older patients: a suggested explanation. *Dev Med Child Neurol* 42: 158–161
74. Vredevelde JW, Slooff BCJ, Blaauw G, Richards R (2001) Validation of an electromyography and nerve conduction study protocol for the analysis of brachial plexus lesions in 184 consecutive patients with traumatic lesions. *J Clin Neuromusc Dis* 2: 123–128
75. Wickstrom J, Haslam ET, Hutchinson RH (1955) The surgical management of residual deformities of the shoulder following birth injuries of the brachial plexus. *J Bone Joint Surg* 27A: 27–36
76. Wynn Parry CB (1984) Pain in avulsion of the brachial plexus. *Neurosurgery* 15: 960–965