Review article

Brachial plexus birth palsy: Management during the first year of life

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ABSTRACT

Brachial plexus birth palsy (BPBP) is defined as an injury to any nerve root of the brachial plexus during difficult delivery. BPBP is relatively rare; its incidence has remained constant over the last few decades, mostly due to unpredictable risk factors, such as shoulder dystocia. Both diagnosis and assessment of spontaneous recovery is based on clinical examination. Electromyography is difficult to interpret in the newborn and is therefore not meaningful. MRI of the cervical spine requires sedation or general anesthesia. Searching for a pre-ganglion tear prior to surgery is indicted. Prognosis depends on the level of the injury (pre- or post-ganglion), size and severity of the post-ganglion tears, speed of recovery, and quality of initial management. Although spontaneous recovery is frequent, some children suffer various degrees of sequelae, up to complete loss of function of the affected upper limb. Recent publications have improved general knowledge and indications for surgery. However, some aspects, such as indication and timing of nerve repair continue to be debated.

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1. Introduction

Brachial plexus birth palsy (BPBP) is a neuromotor flaccid paralysis secondary to injury to one or several brachial plexus (BP) roots occurring during delivery. It is relatively rare, with an incidence between 0.04 and 0.4% live births [1–3].

It was first reported in 1764 by William Smellie [4]. However, the term “obstetrical paralysis” was used for the first time in 1872 by Duchenne de Boulogne in his treatise “De l’électrisation localisée” [5]. In 1877, Erb described obstetrical paralysis of the proximal brachial plexus, which now carries his name [6]. In 1885, Klumpke reported the first description of an isolated involvement of the distal roots of the brachial plexus [7]. The first experimental studies challenging the obstetrical cause by direct traction on the nerve roots appeared at the end of the 19th century [8]. In 1898, Duval and Guillain reported the first anatomical studies [9]. Most particularly, they studied the direction of the nerve roots, explaining the predominance of the proximal C5 and C6 nerve involvement.

The first surgical repairs of the BP were reported at the beginning of the twentieth century in the United States. They initially consisted of resection of the neuroma associated with a simple direct nerve suture. The first cases of nerve grafting were described a few years later, in 1930, in response to the failure of direct suture techniques. However, surgical treatment rapidly lost interest because of poor functional results, high morbidity, and substantial mortality. Conservative treatment therefore remained the standard until the end of the 1960s. The development in particular of microsurgery of the brachial plexus in adults and imaging techniques providing better preoperative analysis resulted in renewed interest in surgical treatment of BPBP. In France, Gilbert et al. [10] are credited with having developed BPBP surgery toward the end of the 1970s.

BPBP was already presented in an instructional lecture by Dr. Jean-Paul Métairie in 1993. This article will not cover certain aspects, notably the etiopathology that was fully described during this lecture. We will focus mainly on treatment during the first year of life, emphasizing three main aspects: injury diagnosis, assessment of the prognosis, and therapeutic management.

2. Anatomy

2.1. Descriptive anatomy (Fig. 1)

The brachial plexus is formed by the anastomosis of the ventral branches of the spinal nerves from C5 to T1, which give rise to seven terminal nerves and approximately ten collateral branches. It most often receives a ramus of C4 (prefixed plexus) and more rarely a ramus of T2 (post-fixed plexus). The C5 and C6 nerve roots join to form the upper trunk, C7 alone forms the middle trunk, whereas C8 and T1 anastomose to form the lower trunk. Each upper trunk is then divided into two anterior and posterior branches that form the secondary trunks. The two anterior branches of the upper and
middle trunks anastomose to form the lateral cord, which gives rise to the musculocutaneous nerve and the lateral root of the median nerve, whereas the anterior branch of the lower trunk alone forms the medial cord, which gives rise to the following nerves: medial cutaneous nerve of the arm, ulnar nerve, and the medial root of the median nerve. The posterior branches of the three upper trunks join to form the posterior cord to give rise to the axillary and radial nerves.

Among the collateral branches, the suprascapular nerve, stemming from the upper trunk, must be distinguished. It innervates the later rotator muscles of the shoulder.

It is important to distinguish two segments of the brachial plexus: the supraclavicular segment (roots and upper trunks) and the subclavicular segment (cords and terminal branches). Finally, it should be remembered that the BP is proximally closely related to the phrenic nerve (a branch of C4) whose injury is responsible for diaphragm paralysis. Distally, there are connections of the two last roots, C8 and T1, with the cervical sympathetic chain whose injury is responsible for Bernard Horner syndrome.

### 2.2. Functional anatomy (Table 1)

The most classically used statistical correspondences of the myotomes is Bonnel and Rabischong’s [11]. However, there is great anatomical variability. Several nerve roots participate in the formation of a nerve. A function can depend on several roots and a root can participate in several functions.

Briefly, C5-C6 injury manifests by a deficit in abduction and lateral rotation of the shoulder and by a deficit in elbow flexion and supination. If the injury extends to the C7 root, a deficit in elbow, wrist, and finger extension and a deficit in radial inclination are associated. Injury to the C8-T1 roots results in a deficit in flexion of the wrist and fingers as well as the intrinsic function of the hand.

### 3. Physiopathology

#### 3.1. Injury mechanism

The most common mechanism is stretching of the BP during the second stage of a dystocic delivery, either through traction on the head (cephalic presentation) or traction on the upper limb (breach presentation). The causes are probably multifactorial, but the two main risk factors are dystocia of the shoulders and macrosomia [12]. Delivery with instrumentation (forceps/vacuum extractor), prolonged labor, primiparity, prematurity (breach deliveries), a history of BPBP during a preceding birth, and excessive maternal weight gain are also potential risk factors [13].

In very rare cases, the BP can be stretched during the initial phase of delivery during the passage of the fetus at the sacral promontory [14]. Finally, certain uterine anomalies have been reported (fibroma, bicornuate uterus) to be potentially responsible for intruterine involvement in the BP [14].

Four anatomical characteristics explain the chronology, the type, and the seat of BP lesions [11,15]:

- orientation of the BP: the C5 and C6 roots take a nearly vertical descending direction, which makes them more vulnerable than the distal roots that are nearly horizontal. Excessive traction on the BP will first lead to proximal root involvement and then secondary involvement of the distal roots. Consequently, proximal lesions are more frequent;
- the posterosuperior ligament: it is present at the C5 and C6 roots and anchors these two roots to the transverse apophysis, thus protecting them from radicular avulsions. At the C7-C8T1 roots, when this ligament is absent, any traction force is transmitted directly to the rootlets. This explains the frequency of distal avulsions and the infrequency of distal avulsions;
- conjunctive tissue: a protective element, conjunctive tissue is richer at the trunks than at the roots, which explains the more frequent seat of these lesions in the roots;
- pre-fixed BP: the participation of the C4 branch can be considered as a predisposing factor for proximal lesions, whereas the post-fixed plexus (participation of a T2 branch) instead plays a protective role for the distal roots.

### 3.2. Types of lesions

To determine the severity of the problem, it is important to distinguish two types of lesions (Fig. 2):

- the pre-ganglion or avulsion lesion, located upstream of the dorsal root ganglion, is a veritable tearing of the rootlet at the spinal cord. This lesion, which most often involves the C8 and D1 roots, is particularly serious because it cannot be repaired by direct surgery. It should be systematically sought on MRI or CT-myelography for better surgical planning;
- the post-ganglion lesion is located downstream of the dorsal root ganglion. Three types are described in the Sunderland classification [16]:

![Diagram of the brachial plexus with labeled roots and nerves.](image-url)
5.1. The clinical exam

Diagnosis of BPBP at birth is generally easy and based solely on clinical examination.

Questioning covers the obstetrical history looking for risk factors. Elements of dystocic delivery are often found. Macrosomia with a birth weight over 4 kg is frequently associated.

Inspection of the newborn finds asymmetric active movements between the two upper limbs with an overall spontaneous position in adduction and internal rotation. One must immediately search for signs of severity, most particularly the Horner syndrome triad: ptosis, myosis, and enophthalmia (Fig. 3). Mobilization can be painful, particularly in the 1st week of life. This confirms the flaccidity of the monoplegia. At this stage, there is no limitation of passive range of movement. Active range of movement, difficult to obtain at this age, can be assessed using archaic reflexes (Moro for the external rotators of the shoulder, the grasping reflex for the finger flexors), the myotatic reflex (response contraction after stretching a muscle), and stimulation of the cutaneous zone opposite the muscle concerned. The motor recovery score, a vital parameter for monitoring neurological recuperation, can be assessed at this age with the British Medical Council classification up to a score of 3 (0: no contraction; 1: contraction without movement or with a slight movement; 2: active movement with gravity eliminated, perceptible intrinsic contraction; 3: active movement against gravity; 4: active movement against resistance, some intrinsic weakness; 5: contraction with normal power).

The contralateral upper limb is also examined looking for any bilateral involvement or an associated fracture. Finally, a central location can be ruled out by a general neurological exam.

At the end of this clinical workup, three possibilities can be encountered:

- paralysis of the upper roots with C5C6 ± C7 involvement: this accounts for more than 75% of BPBP with most often post-ganglionic lesions. The limb involved has a characteristic spontaneous position: shoulder in adduction and internal rotation, elbow in extension contrasting with the usual physiological hyperflexion at this age, forearm in pronation and wrist in extension in C5C6 lesions or in flexion and ulnar inclination when the lesion extends to the C7 root. The fingers retain their physiological tonicity in flexion. In terms of active range of movement of the shoulder, the Moro reflex is absent and no response is found on stimulation of the deltoid. At the elbow, there is no active flexion. At the wrist and the hand, active extension is preserved, except in cases with associated C7 involvement (Figs. 4 and 5). At the hand, the grasping reflex is present and finger function is preserved. In this context, paralysis of the hemidiaphragm caused by phrenic nerve damage should be systematically sought by the presence of abdominal asymmetry during respiratory movements;
- total paralysis: this accounts for approximately 20% of cases. Its diagnosis is even easier when an upper limb is completely inert and dangling (Fig. 6). There are sensory problems and no active range of movement is possible beyond the presence of a few shoulder movements at the scapulothoracic joint and flessum of the fingers’ distal interphalangeals. In this context, the signs

Fig. 2. Diagram of proximal pre-ganglionic lesion (1) distal post-ganglionic lesion (2).

o type 1 or Seddon neurapraxia [17]: a simple elongation of the PB without interrupting the nerve continuity, leading to transitory paralysis that is spontaneously resolved,
- type 2 or axonotmesis: partial nerve rupture touching the axon but keeping the nerve sheath intact. Spontaneous recuperation is possible but with a risk of a “switching” error,
- type 3 or neurotmesis: complete nerve rupture with neuroma formation. Spontaneous recuperation is impossible but the lesion remains accessible to nerve repair.

4. Epidemiology

Despite progress in obstetrics, the incidence of BPBP has remained stable over the last few decades. This may be essentially related to the unpredictability of shoulder dystocia and the increase in mean birth weight [18].

The incidence of BPBP varies greatly between series and is estimated between 0.04 and 0.4% of live births [1–3]. The largest epidemiological survey, conducted at the national level in the United States in 2008, reported a 0.15% incidence of live births [1].

The frequency of the anterior left occipitoitlia presentation, which places the right shoulder under the maternal pubis, explains the predominance of these lesions on the right side. In 4% of cases, the lesions can be bilateral [18].

Proximal C5C6 paralysis (Erb–Duchenne), by far the most frequent, accounts for 50–60% of cases. In approximately 30% of cases, proximal C5C6 involvement is extended to the C7 root. Total C5T1 paralysis, a severe condition often with consequential sequelae, is found in 15–20% of cases [19]. Distal CBT1 paralysis (Klemplke–Déjerine), occurring during breech delivery, is exceptional and accounts for less than 2% of cases [18].

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indicating involvement of the sympathetic nervous system (vaso-motor problems with blotches, local coldness, and sweating, Horner syndrome) are often associated. Their presence often indicates a radicular avulsion of the distal roots, which worsens the prognosis [20];

- distal paralysis (Klumpke palsy): this is exceptional and accounts for less than 2% of cases [18]. The lesional mechanism is direct traction on the upper limb, with the shoulder in abduction. This situation can be encountered in cephalic presentations with the hand first and in certain caesarian deliveries. Clinically, the wrist and the hand are inert, whereas the elbow and shoulder retain normal function.

### 5.2. Additional investigations

#### 5.2.1. Initial phase

In the initial phase, the clinical exam is crucial both to establish the diagnosis and to assess lesion severity. Therefore, no complementary exam is indicated here.

However, a standard radiological workup will be useful in searching for associated lesions. Most particularly, an X-ray of the scapular girdle and the humerus should be requested to search for any associated clavicle or humerus fracture, which may not be visible on the X-ray if there is physeal injury. A chest X-ray can be requested if phrenic paralysis is suspected. In this case, it shows a raised homolateral hemidiaphragm associated with inertia on the radioscopic exam.

#### 5.2.2. Cases in which function is not recuperated by the 3rd month

In cases in which function is not recuperated by the 3rd month, two complementary exams can be requested:

- electromyography (EMG), an exam that is difficult to perform and to interpret in the newborn or the infant, currently plays a very limited role in the diagnosis of BPBP. Most studies often describe it as “overly optimistic” because of the frequent underestimation of the severity of the nerve lesions. However, it can be requested in the preoperative workup, with an essentially medical-legal goal, when surgical exploration of the BP is deemed necessary;

- MRI of the cervical spine is a very useful exam in preoperative planning. Compared to the CT-myelography, it has the advantage of direct visualization of the spinal cord and in certain cases the BP itself. The presence on MRI of a pseudo-meningocele (Fig. 7) is highly suggestive of radicular avulsion. It can significantly influence the nerve repair strategy. False-positive and false-negative results are found in 10–15% of cases. However, its reliability remains close to that of the CT-myelography [21]. MRI has the advantage of being a direct, non-invasive exam that is increasingly easy to perform with simple sedation, whereas CT–myelography always requires general anesthesia.

### 5.3. Differential diagnosis

It is mainly the physeal injury of the proximal humerus that can cause a problem in the differential diagnosis with BPBP. This lesion, responsible for painful unilateral pseudoparalysis, is not visible on the X-ray because the epiphysis has not yet ossified (Fig. 8). When in doubt, an ultrasound can be requested. Radiographic verification 10–15 days later retrospectively confirms the diagnosis by the presence of bone callus formation.

The other fractures (clavicle, humeral diaphysis) are easily diagnosed on imaging studies. They can be isolated, but also associated with BPBP.

Septic osteoarthritides of the shoulder, much rarer, can also simulate BPBP, but the clinical context (prematurity, emergency or
neonatal unit care) and ultrasound of the shoulder rapidly orient the diagnosis.

Relatively rare, congenital deformities are unfortunately only diagnosed when exploring the BP surgically. In our practice, this situation has been encountered in two cases.

Central nervous system involvement may be suggested but will be rapidly ruled out by a general neurological examination.

6. Progression and prognosis

In the literature, the natural history of BPBP is still debated. Selection bias, the heterogeneity of the BP lesions, and the diversity of the evaluation measurements explain the great variability of these data.

Most authors agree that PB lesions are most often transitory, with 75–95% of cases advancing to complete recuperation [22–25]. The most recent studies report a lower rate of 66%, with a residual deficit in 20–30% and considerable alteration of function in 10–15% of cases [2,23,26].

The final prognosis of BPBP is directly related to the type of initial nerve lesions. Although the extent of the lesions can be assessed clinically, today no exam can specify the type of lesion (simple elongation, rupture, or avulsion). The prognostic assessment at birth is based only on clinical criteria. Total paralysis and the presence of Horner syndrome are the main factors announcing a poor prognosis. The prognostic value of diaphragm paralysis is, however, controversial. It is often cited in publications as a factor of poor prognosis. In a study analyzing the prognostic value of phrenic nerve involvement, Al-Qattan et al. [27] observed that it had only a low predictive value, with 13% poor results. In our practice, the cases of BPBP associated with phrenic paralysis – including two complicated cases of respiratory distress requiring hospitalization, were all proximal and all progressed favorably on the neurological level.

After birth, it is the quality and most particularly the speed of recuperation that will provide the greatest amount of information on prognosis and will consequently guide the therapeutic indications [23]. This requires that the clinical exam be repeated within the outpatient clinic at least once a month. The assessment of recuperation is based for the most part on biceps muscle testing, which remains, particularly for the first 6 months, the simplest and the most reliable monitoring indicator. The main objective is to identify the cases in which early nerve repair could contribute to a better functional result than conservative treatment.

All in all, the combination of these three factors (the type and extent of the lesions, Horner syndrome, and the speed of recuperation) will provide the assessment of the patient’s progression and guide the therapeutic indications.

7. Course to follow during the first year (Fig. 9)

7.1. Course to follow during the first 3 months

Children with BPBP, and most particularly those who do not recover during the first 2 months, should be managed in a tertiary care facility preferably by a multidisciplinary team.

Treatment is first conservative. It is based essentially on functional rehabilitation. During the first phase, it is advisable to undertake a 10–15 day period of immobilization of the elbow to the body to relieve pain and to foster healing of any neurapraxic lesions.

BPBP includes muscle imbalance between the agonists and the antagonists that may be responsible for retraction and joint stiffness with very short-term development of osteoarticular deformities. The typical example is encountered at the scapulohumeral joint, particularly in partial loss of function with incomplete recuperation. The weakness of the lateral rotator and abductor muscles leads to muscle imbalance favoring the medial rotators resulting in progressive fixation of the shoulder in medial rotation.

Stiffness in medial rotation of the shoulder is the most frequent sequel. It can appear very early and rapidly set in by retraction of the periarticular soft tissues. It is responsible for growth problems of the glenohumeral joint, leading to retroversion of the glenoid and progressive subluxation of the humeral head. These deformities can appear as early as 5 months of age and rapidly evolve toward posterior dislocation of the humeral head [28]. It should be screened and treated rapidly before osteoarticular deformities have the time to set in. Stiffness is located in the glenohumeral joint and physical therapy should focus on this joint. This requires emphasizing work on the shoulder in the elbow-to-body position while maintaining the scapula in place, thus preventing any parasitical movement in the scapulothoracic joint.

Functional rehabilitation is therefore crucial and should be pursued throughout neurological recuperation. Its objectives are maintenance of joint range of movement, prevention of retractions, and muscular reinforcement.

![Fig. 7. Cervical spine MRI demonstrating hypointensity related to pseudo-meningocele highly suggestive of avulsion at the left C8 and T1 roots.](image)

![Fig. 8. Physeal fracture at birth of the right proximal humerus. Radiographic view at birth.](image)
Corrective braces, not highly effective and often poorly tolerated by infants, have a very limited use during this early phase.

7.2. Course to follow between the 3rd and 6th month

Depending on the patient’s progression and particularly on the speed of recovery, several situations are possible. For some the course to follow is unanimous, but for others it continues to be debated [3].

7.2.1. First situation

The most obvious situation and by far the most frequent is when recovery of biceps strength to +3 occurs within the first 4 weeks. It is the clinical expression of a simple elongation of the BP (neurapraxia), and rapid and complete recovery is the rule.

7.2.2. Second situation

Conversely, in cases of total paralysis, especially when associated with SCBH, in the majority of cases, spontaneous progression is unfavorable. Most authors agree on early surgical repair at the age of 3 months, when biceps function is not recovered [29]. To achieve this, we advise organizing a preoperative workup, in particular with MRI, as early as the 1st month and to begin preparing the family for the surgical intervention.

7.2.3. Third situation

When recovery of biceps strength to +3 occurs between the 1st and the 3rd month, progression is more often favorable. However, complete recovery of overall function of the upper limb sometimes can only occur late over the child’s first 2 years, and even when recovery is incomplete, the sequelae remain minimal without hindering the function of the limb involved. Treatment remains resolutely conservative, while pursuing rehabilitation and monitoring [30].

7.2.4. Fourth situation

In cases of proximal paralysis in which no biceps recovery has been noted by the 3rd month, opinions are divided between early repair [31,32] and waiting for the 6th [33,34] or even the 9th month for some authors [35].

Among those who plead for early surgery, Gilbert and Tassin [29] were the first to recommend nerve surgery at the age of 3 months if biceps function had not been recovered. They argue that children who do not manage to recuperate biceps strength at the 3rd month have little chance of complete recovery and that later nerve surgery may compromise the final functional result.

Al-Qattan [36] proposed waiting until the 4th month to decide on the indication for surgery.

Other authors prefer waiting until the 6th month, believing that a surgical indication at the age of 3 months is excessive in children who may recover biceps function secondarily without BP surgery. In a series of 49 cases, Waters [33] found 42 patients whose biceps recovered between the 3rd and the 6th month. In a prospective study on 170 cases of BPBP, Smith et al. [34] reported 29 children (17%) who had not recovered biceps function at the age of 3 months. In this subgroup, 20 children (71%) recovered biceps function between 3 and 6 months of age.

However, other studies have shown that when the biceps recovered between the 3rd and 6th month, the children presented progressive deterioration of functional scores [33].

In addition, it has been recognized that children who recovered biceps function late between the 3rd and 6th month or who had early nerve surgery will always present incomplete recovery. It is in these two situations that a secondary surgery is the most often undertaken. Moreover, there have been no studies comparing the results of children whose recovery was spontaneous and who were...
operated secondarily with a similar group of children who had both primary repair and secondary surgery. Similarly, there has been no prospective study that compared the results of children operated at the age of 3 months and those who recovered biceps function between 3 and 6 months and who underwent secondary surgery.

In our practice, the absence of biceps recovery at 3 months in cases of proximal BPBP warrants planning a preparative workup (MRI and EMG) in view of surgical exploration. This is done between the 5th and 6th month if the biceps muscle has not recovered at this age.

7.3. Surgical treatment

The surgical procedure makes use of a supraclavicular approach for proximal BPBP or a super- and subclavicular approach in cases of total BPBP. The first exploratory phase is useful for establishing the lesional workup and defining the repair strategy. In case of brachial nerve rupture, the reference technique remains resection of the neurona and microsurgical nerve graft repair. Isolated neurorolyis and direct repair without grafting have been abandoned [29,33]. The grafts habitually used come from one of the two sural nerves. In avulsion cases, today there is no procedure that allows direct repair of this type of lesion and the avulsed roots will undergo neurorolyis.

Several nerve repair assemblies can be done depending on the type and the extent of the plexus lesions estimated intraoperatively. Here, we will discuss only the most frequent situations.

In isolated C56 ruptures, C5 is generally used for grafting the supraspacular nerve and the lateral cord, whereas the C6 root is used for the upper trunk graft. In C56C7 grafts, nerve fibers coming from C5 can be divided for the supraspacular nerve and anterolateral upper trunk grafting. The latter can also receive C6 nerve fibers, whereas the C7 root is used to graft the upper trunk. Extraplexus neurorolyis techniques (spinal nerve, interosseous nerves, fascicles of the ulnar nerve) are reserved for the exceptional proximal lesions with avulsion of at least two of the three roots.

In total paralysis, with rupture of the proximal roots (C5, C6, and C7) and avulsion of the two last roots (the most frequent situation), recovery of hand function should be the primary objective of the reconstruction strategy. In this case, the external branch of the spinal nerve is usually used for neurorolyis of the supraspacular nerve and the three proximal roots are divided over the entire BP. Thus, the C5 root is used for the lateral cord graft. The upper trunk is grafted from C6, whereas C7 is reserved for medial cord grafting. Extraplexus neurorolyises are reserved for total paralysis with avulsion of at least four roots.

After surgery, the nerve sutures should be protected by immobilization lasting 3 weeks. Two types of immobilization can be used: a simple plaster cast made in the immediate postoperative period or a thermoformed cervicothoracic brace associated with immobilization of the arm with the elbow against the body.

The results of nerve repairs in proximal paralysis are encouraging, with recovery of shoulder function in 60–80% of cases and recovery of biceps strength in 80–100% of cases [37]. If need be, secondary surgery can improve these functional results.

In cases of total paralysis, the results remain fair regardless of the type of treatment [29].

8. Conclusion

BPBP remains a current subject because of problems preventing and managing this malformation. Its prognosis depends for the most part on the severity of the initial lesions, the recovery speed, and the quality of the management. Partial proximal paralysis has a better prognosis with spontaneous progression that is most often favorable in the first 3 months of life. Treatment is first conservative, based on functional rehabilitation. In case of unfavorable progression, surgical repair is performed between the age of 3 and 6 months, which can improve the functional prognosis in most cases.

Shoulder stiffness in internal rotation is a frequent sequela when there is partial recovery with or without nerve surgery. It can appear very early, beginning in the 5th month. It should be screened and treated rapidly before osteoarticular deformities set in. Cases of total paralysis, however, have a poor prognosis. They are managed surgically and this should be done as early as 3 months. However, the extent and severity of the lesions is often an obstacle to optimal nerve repair, explaining the frequency of poor results.

Disclosure of interest

The author declares that he has no competing interest.

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