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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.

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 Métaizeau 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

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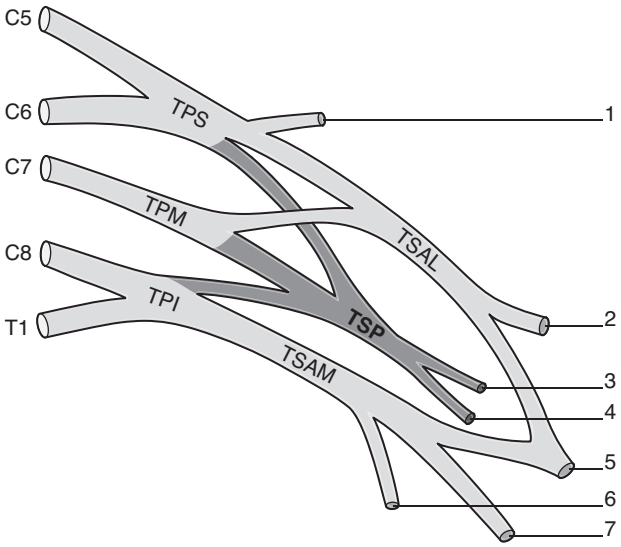

Fig. 1. Anatomy of the brachial plexus. 1: suprascapular nerve, 2: musculocutaneous nerve, 3: axillary nerve, 4: radial nerve, 5: median nerve, 6: medial cutaneous nerve of the forearm, 7: ulnar nerve.

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 sympathic 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, C5C6 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 C8T1 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 (breech 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 (breech 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 intrauterine 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 C7C8T1 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.

Table 1
Summary of functional anatomy of the brachial plexus.

Fonction	Racines
Shoulder	
Abduction–external rotation	C5C6
Adduction–internal rotation	C5–T1
Elbow	
Flexion	C5C6
Extension	C6C7C8
Wrist	
Supination	C5C6
Extension	C5C6C7
Radial inclination	C5C6C7
Flexion	C6C7C8
Pronation	C6T1
Ulnar inclination	C7C8
Hand	
Extrinsic muscles	C7C8T1
Intrinsic muscles	C8T1

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]: