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Neonatal brachial plexus palsy—Management and prognostic factors

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ABSTRACT

Successful treatment of patients with neonatal brachial plexus palsy (NBPP) begins with a thorough understanding of the anatomy of the brachial plexus and of the pathophysiology of nerve injury via which the brachial plexus nerves stretched in the perinatal period manifest as a weak or paralyzed upper extremity in the newborn. NBPP can be classified by systems that can guide the prognosis and the management as these systems are based on the extent and severity of nerve injury, anatomy of nerve injury, and clinical presentation. Serial physical examinations, supplemented by a thorough maternal and perinatal history, are critical to the formulation of the treatment plan that relies upon occupational/physical therapy and rehabilitation management but may include nerve reconstruction and secondary musculoskeletal surgeries. Adjunctive imaging and electrodiagnostic studies provide additional information to guide prognosis and treatment. As research improves not only the technical aspects of NBPP treatment but also the ability to assess the activity and participation as well as body structure and function of NBPP patients, the functional outcomes for affected infants have an overall optimistic prognosis, with the majority recovering adequate functional use of the affected arm. Of importance are (i) early referral to interdisciplinary specialty clinics that can provide up-to-date advances in clinical care and (ii) increasing research/awareness of the psychosocial and patient-reported quality-of-life issues that surround the chronic disablement of NBPP.

Introduction

The management of patients with neonatal brachial plexus palsy (NBPP) begins with the understanding that stretching the nerves of the brachial plexus in the perinatal period manifests as a weak or paralyzed upper extremity, with the passive range of motion greater than the active, in a newborn.

Classification

The most useful classification scheme for the management and the prognosis of NBPP was proposed by Gilbert and Tassin,[1] refined by Narakas[2,3] (Table 1), and supported by Birch et al.[4] Group I represents the clinical findings resulting from injury to the nerve roots C5 and C6—characterized by paresis/paralysis of the deltoid and biceps but active function in the limb extensors, wrist, and hand. The clinical findings in Group II are related to injury of the nerve roots C5, C6, and C7; therefore, in addition to paresis/paralysis of the deltoid and the biceps, paresis/paralysis of the triceps and the wrist extensors is also present, but the long flexors and the intrinsic muscles of the hand are relatively unaffected. Group III represents paresis/paralysis of the muscles of the entire arm (flail arm), consistent with injury of all the nerve roots of the brachial plexus (the C5,

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Table 1 – The Gilbert and Tassin/Narakas classification scheme used for grading the severity of NBPP and for prognosis.

Group	Affected nerve roots	Rate of full spontaneous recovery (%)
I	C5 and C6	~90
II	C5, C6, and C7	~65
III	C5, C6, C7, C8, and T1	<50
IV	C5, C6, C7, C8, and T1 with Horner's syndrome	~0

C6, C7, C8, and T1). Group IV manifests as a flail arm with the additional presence of Horner's syndrome (ptosis, meiosis, and anhydrosis) of the ipsilateral eye and face, presuming injury to all the nerve roots of the brachial plexus with a very proximal injury to the lower nerve roots. When this classification system is used between 2 and 4 weeks after birth, it facilitates determination of the extent of injury to guide prognosis and subsequent management.

Other classification schemes that guide the prognosis and the management rely upon the anatomy and physiology of the nerve injury. Sunderland reported a physiologic scheme comprising five types of pathology in increasing severity: (1) neurapraxia (transient nerve injury that may result from a brief ischemic episode or from any form of compression, demyelination, or axonal constriction or stretch); (2) axonotmesis (transient or permanent nerve injury in which the majority of the supporting structures of the nerve, endoneurium, perineurium, and epineurium are preserved, but disruption of the axonal nerve fibers is present); (3) lesion of the axon and the endoneurium (likely resulting in permanent nerve injury); (4) lesion of the axon, endoneurium, and perineurium (likely resulting in permanent nerve injury); and (5) complete transection of the entire nerve (permanent nerve injury).[5] For example, most nerve reconstruction surgeons manage patients conservatively before proposing surgical intervention to ensure that they do not operate on neurapraxic lesions.

Fig. 1 – Waiter's-tip posture of the right arm.

There is also an anatomical scheme comprising four categories based on the anatomical location: upper, lower, and total plexus palsy.[6,7] The concept of an "upper" plexus palsy involving the C5, C6, and sometimes C7 was initially defined anatomically by Erb[8] in 1874 after Duchenne[9] described four cases of complete paralysis of shoulder movement and elbow flexion in 1872. The upper palsy, also commonly referred to as Erb's palsy, is the most common type of NBPP.[4,10] Erb's palsy is visually recognized by the stereotyped "waiter's-tip posture" with the arm adducted, shoulder internally rotated, wrist flexed, and fingers extended (Fig. 1). Similarly, Klumpke's palsy is visually recognized by a flaccid hand in an otherwise active arm, characterizing "lower" plexus palsy,[11,12] this type of NBPP is extremely rare[13] (e.g., 1 in 350 patients in the author's practice). "Pan"plexopathy is characterized by total plexus palsy, as described by Narakas Groups III and IV, with total loss of function of the arm (flail arm).

Assessment of the neonate with NBPP

Physical examination

Obstetric providers may suspect NBPP on the basis of initial observations of the infant in the perinatal period. However, physical examination and ultimate diagnosis are best achieved by the combined efforts of neonatologists, neurologists, pediatricians, physiatrists, and occupational/physical therapists.

The basic premise of the brachial plexus examination relies on an understanding of the complex anatomy of the nerves of the brachial plexus (the complete description of the brachial plexus is outside the scope of this article but can be found in the published literature[14]). Many of the maneuvers in the physical examination are best evaluated by seeking a patient's voluntary cooperation, which neonates are unable to provide. Therefore, different strategies must be used to assess NBPP in neonates compared with older individuals, although the basic principles remain constant. These strategies will also vary substantially based on the normal development of the infant during the first 2 years of life as motor and sensory function mature.

To provide the appropriate context for the physical examination, a thorough family, maternal, and perinatal history must be obtained. Soon after birth, the treating physician should assess the infant for skeletal injuries or bony fractures by clinical and/or radiographic examination because some musculoskeletal injuries preclude early occupational/physical therapy for NBPP. Note that no substantial evidence exists to support further injury of the nervous elements with gentle handling of the neck and the affected limb (during exercises to ensure passive range of motion), and immobilization is not recommended except when associated with skeletal injuries. Surveillance of spontaneous movements and normal reflexes should be performed as part of the observational examination as global neurologic deficits may indicate other neurologic disorders that can occur concurrently with NBPP.[15] Similarly, keen observation of ptosis and meiosis, consistent