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In Brief

Brachial Plexus Injury

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Author Disclosure

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Injury to the brachial plexus during birth has been documented in the medical literature for more than 200 years. Although the ability to predict brachial plexus injury antenatally remains elusive, knowledge of its epidemiology, risk factors, variations in presentation, prognosis, and management can guide therapeutic decisions and potentially avoid lifelong disability.

Intrapartum trauma to the brachial plexus encompasses a spectrum of injuries involving the lower cervical and upper thoracic nerves (C5 through T1), which supply the plexus. These five spinal nerve roots combine to form the upper (C5 through C6), middle (C7), and lower (C8 through T1) trunks of the plexus, and the peripheral nerves originating from the plexus innervate the muscle groups of the shoulder, upper

arm, forearm, wrist, and hand. The phrenic nerve, comprised of fibers from C3 through C5, and the sympathetic fibers of T1 are affected commonly in brachial plexus injuries, resulting, respectively, in ipsilateral diaphragmatic paralysis and Horner syndrome (miosis, partial ptosis, slight enophthalmos and anhidrosis of the affected side).

The incidence of brachial plexus injury is approximately 1 in 1,000 live births. Erb palsy, injury to the C5 through C7 spinal nerves, accounts for approximately 90% of cases. Klumpke palsy, injury to the lower trunk (C8 through T1), is rare and accounts for 1% of cases. Total plexus injury accounts for 10%. Bilateral injury is found in 10% to 20% of cases, occurring almost exclusively in the setting of breech presentation.

Maternal, fetal, and parturitional factors can affect an infant's risk for brachial plexus injury. Maternal risk factors include uterine abnormalities, such as fibroids or a bicornuate uterus, and maternal diabetes. Fetal factors that may predispose an infant to injury include macrosomia, transverse lie, poor tone, and neonatal depression. Among the parturitional factors are abnormal presentations, dysfunctional labor, and the mechanical forces of labor. Although the etiology of brachial plexus may be multifactorial, the pathogenesis of injury is believed to be from traction or stretch injury to the plexus. The classic example is injury from extreme lateral flexion and traction of the head in the attempt to deliver the shoulder during cephalic deliveries that involve shoulder dystocia. Conversely, traction can be applied to the plexus via the shoulder in the process of delivering the head during

breech deliveries. Abdominal wall and intrauterine forces acting on a posterior shoulder pressed against the sacral promontory also have been implicated in brachial plexus injuries.

There are four types of neuronal injury, and the severity of injury influences the likelihood of spontaneous recovery. The most severe form of injury, associated with poor spontaneous recovery, involves avulsion of the nerve root from the spinal cord, often with injury to the cord itself. Equally poor in prognosis is neurotmesis, axonal rupture with disruption of the nerve sheath. Outcome is somewhat improved with axonotmesis, which involves axonal rupture with the nerve sheath left intact. Fortunately, the most common form of injury, neuropraxia, involves damage to the nerve sheath alone, resulting in a temporary conduction block. This type of injury is associated with complete spontaneous recovery.

The clinical manifestations of brachial plexus injury often are recognized in the labor suite. Infants who have Erb palsy have been described classically as having a "waiter's tip" appearance. Weakness at the shoulder of abduction and external rotation, at the elbow of flexion and supination, and at the wrist and fingers of extension results in adduction and internal rotation of the shoulder, extension of the elbow, pronation of the forearm, and flexion of the wrists and fingers. The biceps reflex is not present, and the Moro and tonic neck reflexes are asymmetric. The grasp reflex usually is intact. Klumpke palsy presents with weakness of the long flexors of the wrist and the intrinsic muscles of the hands, resulting in an absent grasp reflex. The biceps reflex is intact. A flaccid arm that has absent

reflexes throughout indicates total plexus injury. An asymmetric Moro reflex suggests a brachial plexus injury.

Management of an infant who has a brachial plexus injury requires gentle handling of the affected limb to avoid additional trauma. Many affected infants have a torticollis, putting them at risk of developing plagiocephaly. Radiographs of the clavicle and humerus should be obtained to exclude bony injury. The infant should be observed for respiratory distress (potential diaphragmatic injury) and examined for signs of Horner syndrome. Magnetic resonance imaging and electromyography have been used as adjunctive examinations, more in planning surgical exploration than in routine diagnosis and management. No consensus has been reached about the effectiveness of short-term immobilization, but prolonged immobilization is not recommended. Generally, infants should be referred to therapists for passive range of motion exercises beginning 7 to 10 days after birth. Therapy should include passive range of motion of the neck and proximal arm.

The prognosis of brachial plexus injuries depends on the severity and extent of the lesion. Upper injuries, limited to C5 through C6, have the best prognosis; lower plexus and total plexus injuries have poor prognoses. Because Erb palsy, involving only the upper plexus, is by far the most common injury, spontaneous recovery occurs in approximately 90% of cases. Onset of recovery within 2 to 4 weeks is a favorable prognostic sign; flaccid paralysis of the entire limb, especially with coexisting Horner syndrome or diaphragmatic paralysis, is an adverse prognostic factor.

Follow-up care involves serial neurologic examinations. If antigravity movement of the affected muscle groups is present by the end of the third month of age, the prognosis is excellent. Satisfactory, but imperfect recovery can be expected if perceptible contractions are present by the end of the third month and antigravity movement is present by the end of the fifth month. If no progress toward recovery is made within the first 2 to 3 months after birth, referral to a specialized center for

evaluation is warranted. If no improvement is detected between 3 and 6 months, the likelihood of spontaneous recovery is grim, and surgical exploration may be considered. Overall, more than 90% of patients are destined to attain complete recovery by 4 months of age. Although recovery of function is most rapid in the first few postnatal months, improvement may continue for up to 1 year of age.

Comment: Credit due should be given. In the literature, Erb palsy also is referred to as Erb-Duchenne paralysis. Wilhelm Erb (1840 to 1921) was a German neurologist whose name also is attached to Erb-Charcot disease (spastic diplegia) and to Erb spinal paralysis (chronic myelitis from syphilis). Guillaume Duchenne (1806 to 1875), a French neurologist, is best known, of course, to pediatricians for his description of muscular dystrophy.

*Henry M. Adam, MD
Editor, In Brief*

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