Bruce O. Berg, Ed. *Child Neurology: A Clinical Manual*, Second Edition. Copyright © 1994 J. B. Lippincott Company

# CHAPTER 11

# NEUROLOGIC ABNORMALITIES OF THE NEWBORN

*Joseph M. Scheller*
*Thomas K. Koch*

The examination of small infants in incubators connected to ventilators and central lines can be intimidating to those who are not accustomed to the neonatal intensive care unit. Nevertheless, the neurologic examination of these infants is possible and most important in determining the nature and extent of neurologic dysfunction of the ill neonate. Knowledge of the infant's gestational age is essential to understand what is the normal neurologic function at that stage of development, for the infant's responses to similar insults will vary according to the gestational age (see Table 11.1).

The head circumference, configuration, and rate of growth must be determined and carefully plotted on appropriate growth charts at birth and at regular intervals thereafter (see Chapter 1 and Appendices). Slower rates of head growth may indicate an earlier major brain insult, whereas accelerated head growth may suggest hydrocephalus, subdural fluid collections, or some metabolic diseases (see Chapters 2 and 4). Palpation of the suture lines and fontanels can also provide important information about abnormal head growth.

An infant's degree of alertness is an important function related to nervous system integrity and activity and is easily influenced by the gestational age of the infant, the time of last feeding, and manipulation. Before the age of 28 weeks' gestation, infants can be aroused only for brief periods of time after vigorous stimulation, but following this period, stimulation will arouse the infant who can then maintain this state of arousal for several minutes. By 32 weeks' gestation, the infant will spontaneously waken, and by 37 weeks, there is spontaneous crying during the wakened state. At term, the infant is attentive to visual and auditory stimuli.

Neonatal visual responsiveness is greatly dependent upon maturation. At 28 weeks' gestation, the infant will blink to light; at 32 weeks, light will result in lid closure, and visual fixation begins to appear. By 37 weeks' gestation, infants turn toward a light source, and at term, definite visual fixation is possible. The term newborn appears to have a visual acuity of at least 20/150 with a clear preference for colors.

The optic disk of the neonate lacks some pink color and is sometimes confused with optic atrophy. Retinal hemorrhages are observed in 20% to 40% of all vaginally delivered normal newborns, and they generally resolve within 1 to 2 weeks. The pupillary reaction to light is consistently present by 32 weeks' gestation but can be seen earlier. Extraocular motility, as demonstrated by the Doll's eye maneuver, can be observed as early as 25 weeks; premature infants tend to have more readily elicitable

**TABLE 11.1** External Characteristics Useful for Estimation of Gestational Age*

| EXTERNAL CHARACTERISTICS | GESTATIONAL AGE | | | |
|---|---|---|---|---|
| | **28 Weeks** | **32 Weeks** | **36 Weeks** | **40 Weeks** |
| Ear cartilage | Pinna soft, remains folded | Pinna slightly harder; remains folded | Pinna harder, springs back | Pinna firm, stands erect from head |
| Breast tissue | None | None | 1- to 2-mm nodule | 6- to 7-mm nodule |
| External genitalia (male) | Testes undescended, smooth scrotum | Testes in inguinal canal, few scrotal rugae | Testes high in scrotum, more scrotal rugae | Testes pendulous, scrotum covered with rugae |
| External genitalia (female) | Prominent clitoris, small, widely separated labia | Prominent clitoris, larger separated labia | Clitoris less prominent, labia majora cover labia minora | Clitoris covered by labia majora |
| Plantar surface | Smooth | 1–2 anterior creases | 2–3 anterior creases | Creases cover sole |

*Adapted with permission from Volpe JJ. *Neurology of the Newborn*, 2nd ed. Philadelphia: W.B. Saunders, 1987.

Doll's eye movements than older infants because of immature ocular fixation. The absence of ocular motility usually indicates brain stem dysfunction, which can sometimes be of metabolic origin.

Facial mobility can be difficult to assess in the quiet infant, and careful attention should be directed to the width of the palpebral fissure, the nasolabial folds, and the position of the angles of the mouth when crying. An acoustic blink can be present as early as 28 weeks' gestation, but with increasing maturity, the infant has more subtle responses to sound, including changes in respiratory pattern or the cessation of motor activity; sucking and swallowing are more complex functions requiring the integrity of the brain stem and brain stem reflexes. Reasonably efficient oral feeding can be maintained between 32 and 34 weeks, but earlier brief sucking efforts can be present as early as 28 weeks.

The development of muscle tone progresses with increasing flexor tone in a caudal-rostral manner. Before 28 weeks' gestation, there is little flexor tone, and the quiet infant lies with minimal limb flexion. At 32 weeks, however, there is definite flexor tone in the lower limbs, with flexion of the knees and hips at rest. At 36 weeks, there is flexor tone present in both the upper and lower limbs, and at rest there is early flexion of the elbows with well developed flexion of the lower limbs. Term infants assume a posture of flexion of all limbs (see Figure 16.4). Motor movements are generally flexor in nature and in unison by 32 weeks' gestation. By the age of 36 weeks, however,

**Table 11.2** Neonatal Examination

- Estimation of gestational age
- Head circumference, configuration, and growth
- Level of arousal
- Cranial nerve function
  - Visual acuity
  - Pupillary responses
  - Extraocular motility
  - Facial mobility
  - Auditory responsiveness
  - Sucking, swallowing, lingual function
- Motor function
  - Muscle tone
  - Muscle power
  - Reflexes
    - Tendon reflexes
    - Moro reflex
    - Grasp reflex
    - Tonic neck reflex

movements are more forceful and can be alternating, especially notable in the lower limbs. At term, the wake neonate tends to be quite active, especially if stimulated, and the movements are commonly alternating. Neck control is also improved, with good extension and fair flexion.

The stretch reflexes that are generally readily elicited include the biceps, the patellar, and the ankle jerks. The pectoralis is often the first stretch reflex present in the very premature infant. In the normal newborn, 5 to 10 beats of ankle clonus are commonly elicited and can persist for several months. A crossed adductor reflex is also commonly elicited.

Primitive neonatal reflexes include the Moro reflex, the palmar grasp, and the tonic neck reflex. The fully developed Moro reflex is characterized by hand opening, arm extension and abduction, followed by forward flexion of the arms, and is often associated with a momentary cry. By 28 weeks' gestation, hand opening is present, and by 32 weeks, arm extension, abduction, and a cry can be present. Forward flexion with a fully developed Moro reflex is present by 37 weeks' gestation. In normal infants, the Moro reflex will disappear by age 6 months. A palmar grasp can be observed as early in 28 weeks and is quite vigorous by 32 weeks' gestation. This involuntary reflex is replaced by a voluntary grasp beginning at age 2 months. The tonic neck reflex, or "fencing posture," appears by 35 weeks' gestation and disappears by age 7 months. This response should not be obligatory, and the infant should be able to overcome the posture (see Table 11.2).

## Disorders of the Neonate

Infant morbidity and mortality are a function of birth weight, and notable neurologic complications in normal newborns weighing more than 2500 grams are rare. A 5-minute Apgar score of 0 to 3, or an umbilical artery pH <7.00 occur in approximately 2 per 1000 live births, and sepsis, meningitis, seizures, and brachial plexus palsy occur in fewer

than 1 per 1000. Head trauma that occurs during delivery and resulting in skull fracture or intracranial bleeding has become a rare occurrence at this time. In contrast, infants with birth weights less than 2500 grams are at higher risk for neurologic complications, and those infants whose birth weight is less than 1500 grams are at the highest risk for developing neurologic complications.

Most infants hospitalized in neonatal intensive care units are premature and of low birth weight and have an increased risk of developing sepsis and meningitis; it is not uncommon that many of these infants also have been exposed to alcohol, cocaine, amphetamines, or other toxic substances *in utero* (see Chapter 21).

## Neonatal seizures

While seizures are not an uncommon problem of the neonate, one must consider that some "unusual" motor activity may not be epileptic in origin. Video-electroencephalographic (EEG) monitoring of neonates thought to have seizures has demonstrated that about two thirds of infants recorded had clinical events that were not associated with electrographic seizure activity. The earlier classification of neonatal seizures has now been reconsidered in an electroclinical frame of reference (see Table 11.3). It is now clear that certain clinical events such as focal clonic (unifocal or multifocal) and focal tonic events are usually "epileptic events" or seizures, associated with a consistent electrocortical discharge. Focal clonic activity has been sometimes confused with tremor, but unlike tremor, focal clonic activity has a slower frequency (1–4 Hz) and does not stop when light physical restraint is applied to the moving body part. Focal tonic activity can be observed in the limbs, trunk, or eyes. Clinical events without consistent

**Table 11.3** Neonatal Seizures

| CLINICAL SEIZURES | ELECTROGRAPHIC SEIZURES | | ETIOLOGY |
|---|---|---|---|
| | Common | Uncommon | |
| Focal clonic | + | | Hemorrhage<br>Parenchymal<br>Subarachnoid |
| Focal tonic<br>Infarction<br>Infection | + | | |
| Subtle<br>"Bicycling" movements<br>Chewing<br>Facial movements | | + | |
| Generalized tonic<br>Infection | | + | |
| Nystagmus | | + | |
| Myoclonic | | + | |
| Apnea (see text) | | + | |

electrocortical activity include myoclonic jerks, generalized tonic fits, and motor automatisms, which can consist of sucking, chewing, or "bicycling" movements, as well as nystagmus. These events may be more likely related to a brain stem release phenomena than to epileptic seizure activity. Recurrent apneic spells as the sole manifestation of seizure activity are most unusual, and the vast majority of apneic spells in the preterm infant are not epileptic in nature. While apnea can be associated with abnormal electrocortical activity in the term infant, it is not associated with bradycardia occurring within the first 20 seconds of onset. Electrocortical seizures can also occur without associated physical manifestations.

Neonatal seizures are the most significant and frequent clinical sign of brain dysfunction in the neonate. The current approach concerning electrocortical association does not alter the significance of these events. Certain seizure types are often associated with specific causes, and the recognition of this relationship is important in order to expedite the care and management of the ill neonate. Seizures with electrographic abnormalities (focal clonic and focal tonic seizures) are often associated with focal structural abnormalities (parenchymal hemorrhage, infarction), subarachnoid hemorrhage, or metabolic abnormalities. Clinical seizures without a consistent electrographic abnormality (tonic posturing and motor automatisms) are most often seen with hypoxic-ischemic encephalopathy (HIE). Both electrographic and nonelectrographic seizures can occur with infection (see Table 11.3).

The time of onset of the seizure activity can also provide information regarding possible etiology. Neonatal seizures can be divided into early onset, occurring within the first 3 days of life, and late onset, occurring after 3 days of life (see Table 11.4). The most common etiology for early onset seizures is HIE, accounting for as much as 60% to 70% of all neonatal seizures occurring during the first 2 days of life. Other causes include intracranial hemorrhage—either subarachnoid, periventricular/intraventricular (especially in preterm infants), or subdural—hypoglycemia, hypocalcemia, drug withdrawal, developmental anomalies, pyridoxine dependency, and familial benign convulsions. Late onset seizures can occur following hypoxia-ischemia but have no typical clinical characteristics. Other more common causes of seizures include hypocalcemia,

**Table 11.4** Neonatal Seizures and the Age of Onset

| |
|---|
| **Early Onset (day 0–3)** |
| Hypoxia-ischemia |
| Intracranial hemorrhage (subarachnoid, periventricular, subdural) |
| Hypoglycemia |
| Hypocalcemia |
| Drug withdrawal |
| Developmental anomalies |
| Pyridoxine dependency |
| Familial benign convulsions |
| **Late Onset (day 3–30)** |
| Hypocalcemia |
| Infection (meningitis, encephalitis) |
| Inborn errors of metabolism |
| Developmental anomalies |
| Drug withdrawal |
| Hypoxia-ischemia |
| "Fifth-day fits" |

infection, inborn errors of metabolism, developmental anomalies, drug withdrawal and "fifth-day fits," an unusual syndrome characterized by onset of seizures during the latter part of the first week of life in presumably normal full-term infants. The "fifth-day fits" are usually multifocal clonic in nature and last less than 24 hours in most cases and completely disappear within 2 weeks. The etiology of this category of seizures remains unknown, although a deficiency of zinc has been suggested as the cause by some authors.

A careful clinical history, including a complete family history, physical examination, and laboratory studies will usually define the cause of the seizures in the majority of cases. Appropriate laboratory studies include an EEG, computerized tomographic (CT) or magnetic resonance imaging (MRI) brain scans, lumbar puncture (LP), serum chemistries, urine for toxicology, and other metabolic studies (see Chapter 10). Although head ultrasonography is noninvasive, readily available, and appropriate for examining the lateral ventricles, it is not sufficiently sensitive to adequately delineate brain structural malformations, vascular events, and lesions in the posterior fossa.

Neonatal seizures with an electrographic concomitant require treatment with antiepileptic drugs (AEDs), primarily the administration of phenobarbital (20 mg/kg IV). This will usually produce a blood level of the drug in the therapeutic range; however, if complete seizure control is not achieved, another 5 to 10 mg/kg can be administered, raising the blood level to 40 to 50 μg/ml. Since some patients can develop apnea and hypotension at this serum level, they must be carefully monitored. Once the seizures have been controlled, maintenance can be accomplished by the administration of 2 to 3 mg/kg/day. Phenobarbital has a very long half-life in the newborn (up to 120 hours), so children who require large doses will remain lethargic for days. In the 10% to 20% of infants who do not respond to the administration of phenobarbital, phenytoin (15–20 mg/kg IV) or lorazepam (0.05 mg/kg IV) can be administered. Neither drug is effective orally. Paraldehyde is occasionally useful when administered IV at 0.1 ml/kg/hr or per rectum at 0.3 ml/kg/day. There is little experience with carbamazepine and valproic acid in the neonate. While AED therapy may suppress nonepileptic neonatal seizures, the requirement for their administration has been questioned. However, since these paroxysmal events have been traditionally treated with AED, withholding these drugs is controversial.

There is no consensus regarding the duration of auticonvulsant treatment for neonates who have had seizures. Commonly the medication will be continued for the first several months of life and then slowly be tapered and discontinued, particularly if the child is neurologically normal and free of further seizures. A longer course of treatment should be used in children with congenital brain anomalies, vascular accidents, and those who had a severe neonatal encephalopathy.

The neurologic outcome for patients with neonatal seizures requires careful attention to the etiology of the seizures. Since epileptic seizures are often seen in infants with structural lesions, hemorrhage, metabolic abnormalities, or infection, their short-term prognosis is generally favorable. In contrast, nonepileptic seizures present in infants with hypoxia-ischemia and infection can have a less favorable prognosis (see Table 11.5).

## Hypoxic-Ischemic Encephalopathy

Perinatal hypoxic-ischemic injury is a major cause of neonatal morbidity and mortality. The insult commonly appears to have occurred prenatally. Maternal factors, such as

**Table 11.5** Prognosis of Neonatal Seizures

| ETIOLOGY | OUTCOME (% FAVORABLE) |
|---|---|
| Hypocalcemia | |
| Late onset | 100 |
| Early onset | 50 |
| "Fifth day fits" | 100 |
| Hemorrhage | |
| Subarachnoid | 90 |
| Intraventricular | 10 |
| CNS infection | 30–50 |
| Hypoxia-ischemia | 40–50 |
| Hypoglycemia | 50 |
| CNS malformation | 0 |
| Unknown | 60–70 |

placental integrity, infection, intrauterine growth, and diabetes mellitus, are all important in this regard.

The clinical severity of HIE can be divided into three groups that correlate with eventual prognosis (see Table 11.6). A *mild encephalopathy* is characterized by irritability, jitteriness, and a hyperalert state that persists less than 24 hours and generally has an excellent prognosis. A *moderate encephalopathy* is characterized by lethargy, hypotonia, decreased movements, and seizures that usually develop within the first 24 hours. About 20% to 40% of these infants will have permanent neurologic sequelae, especially if the results of neonatal neurologic examination continue to be abnormal longer than 1 week. Patients with *severe encephalopathy* have seizures, flaccidity, brain

**Table 11.6** Hypoxic-Ischemic Encephalopathy

| CLINICAL MANIFESTATIONS | PROGNOSIS |
|---|---|
| Mild Encephalopathy | Excellent |
| Irritability | |
| Jitteriness | |
| Hyperalert state | |
| Moderate Encephalopathy | Fair |
| Lethargy | |
| Hypotonia | |
| Decreased movements | |
| Seizures | |
| Severe Encephalopathy | Poor |
| Coma | |
| Flaccidity | |
| Seizures | |
| Brain stem dysfunction | |
| Other organ injury | |

**Table 11.7** Pathologic Patterns of Hypoxic-Ischemic Encephalopathy

| TYPE | INFANT |
|---|---|
| Selective neuronal necrosis | Term |
| Status marmoratus | Term |
| Parasagittal cerebral injury | Term |
| Focal and multifocal infarcts | Term |
| Periventricular leukomalacia | Preterm |

stem dysfunction, and are in coma. There is often later evidence of injury such as microcephaly, mental retardation, spasticity, and seizures.

At neuropathologic examination there are several patterns of cerebral injury that can occur with hypoxic-ischemia (see Table 11.7). The first is selective neuronal necrosis, which is more common in term infants and is characterized by neuronal injury to the hippocampus, diencephalon, brain stem, cerebellum, and spinal cord. These infants can have seizures, respiratory dysfunction, hypotonia, disorders of ocular motility, and sucking. The long-term manifestations include seizures and intellectual and motor impairment.

Injury to the basal ganglia and thalamus presumably result in *status marmoratus*, an irregular pattern of hypermyelination in the lateral part of the corpus striatum and less often in the caudate nucleus and thalamus, which is more commonly seen in term infants. There is no clearly identifiable clinical neonatal syndrome, but the long-term sequelae can be that of a static extrapyramidal syndrome. At the present, this concept of clinical-neuropathologic changes has been questioned.

Another type of cerebral insult results in parasagittal injury. This syndrome is exclusively seen in term infants and represents an ischemic lesion. The injury is bilateral and characterized by watershed infarcts. On examination, the infant demonstrates proximal limb weakness or "man in the barrel" syndrome. Long-term sequelae probably include spastic quadriplegia and visual perceptive dysfunction.

Focal and multifocal ischemic infarcts comprise another neuropathologic pattern that is manifested in term infants. The middle cerebral artery is usually involved, and after dissolution and cavitation of the involved area of brain, there is a single porencephalic cyst or multicystic encephalomalacia. In those cases in which there is a severe loss of brain substance, the condition has been referred to as hydranencephaly. The clinical neonatal manifestations include seizures or focal motor weakness, which persist as long-term sequelae.

The final pattern of brain injury is periventricular leukomalacia, which is most often seen in preterm infants and is the major ischemic lesion of the premature. At neuropathologic examination, there is necrosis of the periventricular white matter at the border zones of the major vascular distributions. Not infrequently, there is hemorrhage into the lesion. The clinical manifestations in the newborn are limited to hypotonia and limb weakness, especially in the lower limbs. The long-term result is spastic diplegia. With severe injury there can be a resultant spastic quadriplegia and visual deficits. The most appropriate management of HIE is in its prevention. Careful attention to the prevention of intrauterine asphyxia and avoidance of systemic hypotension in the premature infant will have significant impact.

## Intraventricular Hemorrhage

Intraventricular hemorrhage (IVH) is the most common form of intracranial hemorrhage in the preterm infant with an incidence of 29% to 49% of infants with birth weights less than 2000 grams. Immaturity of the cerebrovascular system, especially in the region of the subependymal germinal matrix, an area adjacent to the caudate nucleus, appears to be an important factor explaining the vulnerability of the premature infant. The tenuous capillary integrity of this region is inherently sensitive to changes in a majority of intravascular and extravascular factors occurring in premature infants. Of particular importance are the wide variations of cerebral blood flow and vulnerability to hypoxic ischemia. Attention must be directed to the premature infant's respiratory and cardiovascular status and underscores the vulnerability of the critically ill preterm infant with severe respiratory distress syndrome.

Clinically, IVH occurs within the first few days of life, with 90% of hemorrhages having occurred by age 3 days. The hemorrhages can present as a catastrophic event with the sudden presentation of coma, insufficient respiratory drive, posturing, nonreactive pupils, and associated decreased hematocrit, bulging anterior fontanel, autonomic instability, and metabolic acidosis. The prognosis is often poor, and for the surviving infants, hydrocephalus is a common occurrence.

A more subtle presentation can occur in which there is a depression of consciousness, hypotonia, decreased spontaneous movements, and abnormalities of ocular motility. These findings can evolve over days and may have a stuttering course. The prognosis of these patients is generally favorable and related to the degree of the intraventricular hemorrhage. Lastly, IVH can have a silent presentation and can only be detected by neuroimaging with head ultrasonography, CT and/or MRI head scans.

The diagnosis of IVH is readily made by neuroimaging studies and/or lumbar puncture. Head ultrasound is the imaging study of choice due to adequate sensitivity and the ease with which it can be obtained in critically ill infants. Serial ultrasonography can define the timing and severity of the hemorrhage. Most IVH occurs within the first 1 to 3 days of life. A grading scale has been devised based upon the presence or absence of blood in the germinal matrix and lateral ventricles. A Grade I hemorrhage involves bleeding into the germinal matrix without or with minimal intraventricular bleeding. Grade II hemorrhage involves less than 50% intraventricular extension without ventricular enlargement. Grade III hemorrhage implies extensive bleeding into the ventricles with subsequent enlargement. A special emphasis must be noted regarding hemorrhagic involvement of the parenchyma, previously called Grade IV.

The risk of IVH is directly related to the degree of infant prematurity, and efforts to prevent IVH are limited by the inability to prevent prematurity. Various medications including phenobarbital, indomethacin, vitamin E, and ethamsylate have been used in clinical trials; however, none have been accepted as effective on the basis of these studies. If hydrocephalus develops, treatment can be initiated with repeated LP and/or diuretics, such as acetazolamide. When conservative measures fail, neurosurgical shunting is necessary. It is of great importance that high-risk infants be closely followed with serial head ultrasonography to identify those infants who may present silently with IVH, to avoid late presentation complicated by hydrocephalus.

The short-term outcome of these patients is related to the extent of the intraventricular hemorrhage. Mild bleeds have a low mortality, whereas Grade III hemorrhage with intracerebral involvement can have a mortality of 60%. The long-term prognosis is less clear, but the greater the extent of the hemorrhage the greater is the incidence of neurologic morbidity and mortality. With intracerebral extension there is

usually considerable morbidity, seen as spastic motor deficits and intellectual impairment.

## Birth Trauma

The major forms of birth-related injury include extracranial hemorrhage, skull fracture, intracranial hemorrhage (epidural, subdural, subarachnoid, and intraparenchymal hemorrhages), spinal cord injury, and injury to the peripheral nerves. Extracranial hemorrhage can result in caput succedaneum, subgaleal hemorrhage, or a cephalohematoma, each of which is relatively benign, resulting from a difficult or precipitous delivery. No specific therapy is warranted, and each of these hemorrhages will resolve spontaneously. Skull fractures are usually parietal and can be associated with an epidural hematoma or cephalohematoma. Small uncomplicated fractures require no intervention.

Epidural or subdural hematomas and subarachnoid hemorrhage require surgical intervention only when there are clear signs of brain or brain stem compression. Subarachnoid hemorrhage is often silent or occasionally can result in recurrent seizures. These types of intracranial hemorrhage are more commonly seen in large, full-term infants who had difficult or precipitous deliveries.

Injury to the brachial plexus occurs in 0.5 to 2 per 1000 live births. Brachial plexus (Erb-Duchenne) palsies involve the muscles innervated by the upper plexus nerve roots (*i.e.*, C5 to C6) and are thought to be related to traction on these roots during delivery. In cases of breech birth, other obstetrical factors, very large infants, and fetal depression delivered per vagina there is greater likelihood to develop a brachial plexus palsy, whereas infants delivered by Caesarean section are less likely to sustain insults to the brachial plexus. On physical examination, the affected arm is hypotonic, remaining limp at the infant's side. Shoulder abduction and elbow flexion are decreased to absent, and the biceps and supinator reflexes are also decreased to absent. The weakness can be bilateral with asymmetric involvement (10%). Occasionally, the C7 root can be involved, resulting in weakness of the triceps and wrist flexors, and rarely, the weakness extends to the hand muscles supplied by C8 and T1 (Klumpke palsy). Injury at this spinal level can cause a Horner syndrome, and since higher nerve roots can also be affected, paralysis of the diaphragm (C3 to C5) can also occur. Most children improve dramatically over the first few months of life. In the series of Gordon, 88% of affected infants were normal by age 4 months, 92% were normal by age 12 months, and 7% were yet abnormal at age 48 months. In an earlier study of Eng, however, one third of patients had recovered by 6 months with some minimal deficit and about one half had recovered within 1 year's time, though they had some moderate degree of residuae. If no improvement is noted by age 3 months, exploratory surgery of the plexus with possible nerve anastomoses can be considered.

The most common result of perinatal trauma is facial weakness. Although unilateral lower motor facial weakness can follow forceps deliveries, it often occurs with no history of related birth trauma and, like brachial plexus palsy, tends to resolve in the first few months of life. It now appears that most facial nerve injuries arise from the position of the infant's face on the sacral promontory, resulting in compression of the facial nerve against the bony protuberance. This type of facial weakness should not be confused with congenital absence of the depressor anguli oris muscle that affects only an area near the lower angle of the mouth and is usually noted when the child is crying or yawning (asymmetric crying face syndrome, cardiofacial syndrome). This syndrome can be associated with congenital cardiac and/or urologic malformations.

Bilateral facial weakness, when combined with bilateral weakness of cranial nerve VI, is known as the Moebius sequence. This anomaly is thought to result from a dysplasia or aplasia of the brain stem nuclei and/or hypoplasia of the facial nerve. Some patients can have more extensive cranial nerve involvement, and approximately 15% of patients can have mental impairment. Feeding difficulties and aspiration lead to failure to thrive. The Moebius sequence is generally felt to be of sporadic occurrence.

## Reference

Calciolari G, Perlman JM, Volpe JJ. Seizures in the neonatal intensive care unit of the 1980's. *Clin Pediatr* 1988; 27:119–123.

Cooke RWI. Early and late cranial ultrasonographic appearances and outcome in very low birth weight infants. *Arch Dis Child* 1987; 62:931–937.

Eng GD. Brachial plexus palsy in newborn infants. *Pediatrics* 1971; 48:18–28.

Fenichel GM. *Neonatal Neurology*, 3rd ed. New York: Churchill Livingstone, 1990.

Gordon M, Rich H, Deutschberger J, et al. The immediate and long term outcome of obstetric birth trauma. I. Brachial plexus palsy. *Am J Obstet Gynecol* 1973; 117: 51–56.

Levene MI, Sands C, Grindulis H, et al. Comparison of two methods of predicting outcome in perinatal asphyxia. *Lancet* 1986; 1:67–69.

Levene MI, Trounce JQ. Causes of neonatal convulsions. *Arch Dis Child* 1986; 61:78–79.

Mizrahi EM, Kellaway P. Characterization and classification of neonatal seizures. *Neurology* 1987; 37:1837–1844.

Scher M, Aso K, Beggarly ME, et al. Electrographic seizures in preterm and full term neonates: clinical correlates, associated with brain lesions, and risk for neurologic sequelae. *Pediatrics* 1993; 91:128–134.

Volpe JJ. *Neurology of the Newborn*, 2nd ed. Philadelphia: W.B. Saunders, 1987.