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Objectives

After completing this article, readers should be able to:

1. Describe the delivery conditions that increase the risk for birth trauma.
2. Explain why subgaleal hemorrhage can be a medical emergency.
3. Delineate the common presenting findings of traumatic intracranial bleeding.
4. Describe the risk factors for brachial plexus palsy.
5. Differentiate facial nerve palsy from congenital palsies and hypoplasia of the depressor anguli oris muscle.

Introduction

Birth injuries are sustained during the labor and delivery process. They can be divided into those due to physical trauma during the birth process (traumatic birth injury) and those due to lack of oxygen (hypoxic-ischemic injury). These types of injuries can occur separately or in combination. This review focuses on the diagnosis and management of traumatic birth injuries.

Traumatic injuries often are the result of a discrepancy between the size or position of the fetus in relation to the birth canal or an unusually rigid pelvis that has not adapted to the size of the fetal head. The reported rate of birth trauma from a 7-year review published in 1990 was 3.2%. Some injuries are avoidable, and with improvements in obstetric care, the frequency of birth injury as a cause for perinatal mortality has decreased over the past 25 years. Predisposing factors for traumatic birth injury are listed in Table 1. Macrosomic fetuses, including those from poorly controlled diabetic pregnancies, represent a particularly high-risk group for birth injury. However, predicting which macrosomic infants will be injured during the birth process is difficult. Injuries also are more frequent with instrumented vaginal deliveries. Use of forceps has been associated with facial nerve and brachial plexus injuries, skull and facial fractures, and intracranial hemorrhages. Ocular injuries, including fracture of the base of the orbit, intraorbital hemorrhage, corneal laceration, and breaks in Descemet's membrane with corneal opacification, as well as dislocated nasal septum and fractured nasal bones have been reported. Vacuum extractions are associated with cephalohematoma, skull fracture, scalp lacerations, intracranial hemorrhage, subgaleal hemorrhage, and retinal hemorrhage. The frequency of injury is increased by a longer duration of application, multiple applications, and a paramedian cup placement.

Cesarean section does not eliminate the possibility of birth trauma, especially when prior attempts have been made at delivery with vacuum extraction or forceps. A comprehensive listing of birth injuries is presented in Table 2. Reported frequencies of injuries (excluding bruising and minor scalp trauma) are greatest for clavicular fractures, followed by facial nerve injuries, brachial plexus palsy, intracranial hemorrhage, humerus fracture, diaphragmatic paralysis, spinal cord injury, and depressed skull fracture. Many injuries, such as soft-tissue trauma, are minor, but others, such as liver laceration, subgaleal hemorrhage, or large subdural blood collections, can be life-threatening and require prompt recognition and intervention. Traumatic birth injury can result in both physical and neurodevelopmental handicap.

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Table 1. Risk Factors for Traumatic Birth Injury

Rigid birth canal
• Primiparous • Older multipara • Small malformed pelvis
Failure of adequate birth canal adaptation
• Breech position • Precipitous delivery
Large baby relative to size of birth canal
• Macrosomia • Cephalopelvic disproportion • Shoulder dystocia
Abnormal presentations (face, brow, transverse)
Use of vacuum or forceps; difficult rotations
Prematurity

Head Trauma

Cephalohematomas

The clinical spectrum of traumatic injury to the head varies from minor to potentially life-threatening. Minor trauma evident in many vaginal vertex deliveries includes injuries from intrauterine fetal scalp electrodes, lacerations, bruising over the presenting part, and caput succedaneum (Fig. 1). The caput is localized scalp edema due to local venous congestion from the pressure of the head applied to the dilating cervix. The edema is soft and superficial, crosses suture lines, and resolves over the first few postnatal days. Cephalohematomas are localized subperiosteal collections of blood that occur in 0.2% to 2.5% of live births. The incidence is substantially increased in forceps and vacuum deliveries. These are caused by rupture of blood vessels that traverse from the skull to the periosteum. Because the bleeding is subperiosteal, it is limited by suture lines in the skull (Fig. 1). The most common site for these collections is over the parietal bones, and they are most often unilateral.

Cephalohematomas present as a firm, tense mass that enlarges after birth. Rarely is the degree of blood loss sufficient to cause either hemodynamic instability or anemia. Linear skull fractures beneath the hematoma have been reported in up to 5% of cases, but are of no major consequence except in the unlikely event of formation of a leptomeningeal cyst. This is indicated by a widening fracture accompanied by an extracranial enlarging fluid-filled mass. Most cephalohematomas resorb in 2 weeks to 3 months, leaving some residual circular calcification at the base.

Skull Fractures

Depressed skull fractures can be seen in neonates, but they rarely require surgical elevation because of the resilient nature of neonatal bone that results in a "ping-pong" deformity without discontinuity. The fracture can be confirmed with plain skull films, but depressed fractures can be associated with intracranial bleeding, so computed tomography (CT) of the head also is indicated, especially if neurologic symptoms are present in the nursery.

Subgaleal Hemorrhage

Subgaleal hemorrhage (SGH) occurs beneath the scalp in the subaponeurotic space (Fig. 1). The galea aponeurotica extends from the occiput to the eyebrows in the anterior-posterior direction and laterally to the insertion of the temporalis fascia. The injury results from traction on the scalp that shears the emissary veins between the scalp and intracranial venous sinuses. The most common risk factor is a difficult vacuum or forceps extraction. The likelihood of this injury is increased with multiple applications of the vacuum cup and reported "pop offs." The overall incidence of SGH is 1 in 2,000 births, but may be as high as 1 in 200 vacuum-assisted deliveries. These hemorrhages are characterized by boggy fluid collections with a ballotable fluid wave beneath the scalp and bleed-

Table 2. Traumatic Birth Injuries

Type of Injury	Examples
Soft-tissue injuries	Abrasions, bruising, fat necrosis, lacerations
Extracranial bleeding	Cephalohematoma, subgaleal
Intracranial bleeding	Subarachnoid, epidural, subdural, cerebral, cerebellar
Nerve injuries	Facial, cervical nerve roots (brachial plexus palsies, phrenic), Horner syndrome, recurrent laryngeal
Spinal cord injuries	Epidural hemorrhage of the cervical cord
Fractures	Clavicle, humerus, femur, skull
Dislocations	
Torticollis*	
Eye injuries	Subconjunctival and retinal hemorrhage
Solid organ injury	Liver, spleen

*Secondary to bleeding in the sternocleidomastoid muscle.

Table 3. Management of Subgaleal Hemorrhages

1. Immediate placement of central venous access with an umbilical venous line
2. Placement of an umbilical arterial line for blood draws and blood pressure monitoring
3. Aggressive replacement of volume losses in 10- to 20-mL/kg infusions
   - Normal saline
   - Whole blood or packed red blood cells
   - Replacement of coagulation factors and platelets
4. Anticipation of 40-mL blood loss for each 1-cm increase in occipital frontal circumference
5. Correction of metabolic acidemia with 1 to 2 mEq/kg of sodium bicarbonate
6. Careful attention to oxygenation with supplemental oxygen and mechanical ventilation
7. Monitoring of serial hematocrits and coagulation studies (disseminated intravascular coagulation screens)
8. Accurate monitoring of volume infused and urine output

ing extending to above the eyes and back to the insertion of the trapezius muscles. The ears are pushed forward if there is a large subgaleal fluid collection. In contrast to caput succedaneum, this fluid collection increases in size after birth. The infant's occipital frontal circumference will increase 1 cm with each 40 mL of blood deposited in the subaponeurotic space. The space is large, which allows extensive blood loss, shock, and anemia. Thus, these infants can present early in life with pallor, tachycardia, tachypnea, mottling, delayed capillary refill, hypotension, and hypotonia. During the first few hours after birth, the hematocrit drops because of ongoing blood loss and hemodilution. Bleeding can be exacerbated further by dilution and consumption of clotting factors, resulting in a coagulopathy. Multiorgan damage due to hypoperfusion, shock, and asphyxia also can complicate the course of the infant who has SGH. Associated intracranial bleeding also has been reported.

Acute blood loss and hypovolemia from subgaleal bleeding is a life-threatening emergency that should prompt immediate intervention. Even concern for a possible subgaleal fluid collection should mandate transfer to the neonatal intensive care unit (NICU) for close monitoring and observation. These infants can deteriorate rapidly and if circulating volume is not preserved, the result can be multiorgan dysfunction due to ischemia. Principles of stabilization are outlined in Table 3. Acute volume resuscitation should be undertaken initially with normal saline as 10-mL/kg bolus infusions followed by emergent whole blood or packed red blood cell transfusion. O-negative blood available for emergency use should be accessed and fresh frozen plasma used initially for a developing coagulopathy. Serial hematocrits and blood pressure should be monitored closely. It is critically important to maintain stable hemodynamics and assure adequate organ perfusion. Pressors should be added (dopamine 5 to 20 mcg/kg per minute) if volume alone is not sufficient or there is coexisting cardiac decompensation.

Intracranial Hemorrhages

Traumatic intracranial hemorrhages include epidural, subdural, subarachnoid, and less commonly, intraventricular, intracerebral, or intracerebellar. Epidural hemorrhages (Fig. 1) are rare. Linear skull fractures in the parietotemporal region are present in most cases. Irritability, lethargy, and seizures progress to signs of increased intracranial pressure (full fontanelle, hypertension, and bradycardia) and ultimately to unilateral pupil dilatation indicating uncal herniation. Diagnosis is confirmed by CT showing a characteristic convex, lenslike appearance of the epidural blood collection. Three major varieties of subdural hemorrhages have been described: 1) posterior fossa hematomas due to tentorial laceration with rupture of the straight sinus, vein of Galen, or transverse sinuses (Fig. 2) or due to occipital osteodiastasis (a separation between the squamous and lateral portions of the occipital bone); 2) falx laceration, with rupture of the inferior sagittal sinus (Fig. 2); and 3) rupture of the superficial bridging cerebral veins.

Clinical symptoms appear within 24 hours of birth and include focal or generalized seizure activity, altered level of consciousness, irritability, and focal neurologic signs dictated by the location of the bleeding. Posterior fossa collections lead to signs of brainstem compression or signs of increased intracranial pressure due to obstruction of spinal fluid flow. The initial signs are those of an altered level of consciousness: lateral deviation of the eyes not altered by a doll's eyes maneuver and unequal pupils. This can progress to lower brainstem signs, including apnea and bradycardia. Falx tears can cause bilateral cerebral signs (eg, seizures and focal weakness) or can be asymptomatic. If the bleeding extends infratentorially, signs of brainstem compression evolve. Hemorrhage over the cerebral convexities is the most common site of subdural bleeding and presents with focal or multifocal seizures and focal cerebral signs. In rare circumstances, the accumulation of blood can be large enough to in-

crease intracranial pressure and lead to uncal herniation. Subarachnoid bleeding, if limited, may be asymptomatic; when the bleeding is more extensive, irritability and seizures alternate with normal interictal periods. Other less common sites of traumatic bleeding are intracerebellar (associated with occipital osteodiastasis), which presents with signs due to brainstem compression, and intraventricular, which usually presents with seizures.

Helpful diagnostic tests for evaluating infants who have suspected intracranial bleeding include repetitive clinical examinations, serial hematocrits to assess blood loss, and CT imaging of the brain looking for epidural, subdural, or subarachnoid blood. A bedside ultrasonographic examination, although easy to perform, can miss significant blood collections that are located in close proximity to the skull. A lumbar puncture can be performed in infants who do not have signs of increased intracranial pressure. The presence of xanthochromia, red blood cells, and an increased protein level in the cerebrospinal fluid also support a diagnosis of intracranial bleeding. It is also helpful to obtain a coagulation screen with a prothrombin time, partial thromboplastin time, fibrinogen, and platelet count to consider congenital coagulation disorders, diffuse intravascular coagulation due to shock, and isoimmune thrombocytopenia as causes for the bleeding, especially among infants who have no overt history of birth trauma.

Management

Traumatic head injuries are managed by recognition, monitoring, and treatment of serious consequences. Infants at risk for intracranial pathology should receive continuous cardiorespiratory monitoring for apnea, periodic breathing, hypotension, and heart rate irregularities. Serial neurologic examinations should focus on level of consciousness, evidence of focal muscle weakness, brainstem or cranial nerve signs, and evidence of seizure activity. Seizures should be treated with a 20-mg/kg intravenous loading dose of phenobarbital. A second 20-mg/kg dose can be administered for further seizures, with the addition of phenytoin-fosphenytoin or benzodiazepine for more difficult-to-control seizures. Hypovolemia, if present, can be treated with a 10-mL/kg infusion of normal saline over 30 minutes repeated twice more (a total of 30 mL/kg) if blood pressure and perfusion do not improve. Whole blood or packed red blood cell transfusions usually are not necessary for most traumatic intracranial hemorrhages. Continued hypotension after infusion of 30 mL/kg normal saline should trigger an evaluation for coagulopathy and myocardial performance. Hematocrit should be followed serially, and the infant should be monitored later for the development of jaundice. Infants who develop signs of increased intracranial pressure from convexity subdural hemorrhages or epidural collections require acute surgical drainage of the blood. Posterior fossa hemorrhages with evidence of progressive brainstem compression also require emergency drainage. Depressed skull fractures rarely require intervention. Occasionally external elevation may be needed and, less commonly, surgical elevation.

Traumatic Nerve and Spinal Cord Injuries

Peripheral Nerve

Trauma to peripheral nerves produces another major group of birth injuries. Brachial plexus injuries occur in 0.5 to 2.5 per 1,000 live births and are caused by stretching of the cervical nerve roots from traction on the neck during delivery. Risk factors for this type of injury include macrosomia (birthweight, >4,500 g), difficult delivery, shoulder dystocia, breech position, multiparity, and assisted deliveries. Like other forms of birth injury, knowledge of risk factors cannot predict the occurrence of injury in individual cases. Upper arm palsy (Erb-Duchenne) is the most common injury and is caused by damage to the 5th and 6th cervical nerve roots. Isolated lower arm palsies (Klumpke) that result from damage to the 8th cervical and first thoracic nerves are rare. Damage to all the nerve roots results in total arm paralysis. The injury is usually stretch rather than avulsion, which allows for recovery of function over the first several weeks to months after birth.

The diagnosis is based on the presence of unilateral arm weakness. In an Erb paralysis, the arm is limp at the side and internally rotated with flexion of the fingers (waiter's tip position). Loss of finger extension can be confirmed by stroking the back of the hand. The presence of a grasp reflex differentiates Erb palsy from total arm paralysis. Brachial plexus injuries also can be associated with damage to the nerve roots that form the phrenic nerve and control diaphragmatic function. The presence of tachypnea and an oxygen requirement suggest this possibility. Other clinical clues are asymmetric chest motion during respiration and diminished breath sounds on the side of the paralysis. The diagnosis can be supported by a chest radiograph showing an elevated hemidiaphragm and confirmed by ultrasonography demonstrating a lack of or paradoxic diaphragmatic movement. In addition, damage to the sympathetic outflow via nerve root T1 can result in an associated Horner's syndrome with miosis, ptosis, and enophthalmos. Finally, fractures of the clavicle and humerus and shoulder dislocations may be associated.

Facial Nerve

Facial nerve injury is the most common neonatal traumatic nerve injury (up to 1% of live births), caused either by pressure on the facial nerve from the sacral promontory or fetal shoulder as the infant passes through the birth canal or by forceps application. Although forceps application is overrepresented in these infants, the injury is related more commonly to intrauterine position and prolonged labor. Clinically there is loss of motion on the affected side of the face, with an open eye, drooping mouth, lack of expression, and inability to wrinkle the brow. Acquired traumatic facial nerve palsy must be distinguished from congenital nerve palsy, which more frequently involves other craniofacial dysmorphism or multisystem findings (eg, Goldenhar and Moebius syndromes). Traumatic facial nerve palsy also must be distinguished from congenital hypoplasia of the depressor anguli oris muscle causing a localized movement abnormality of the corner of the mouth.

Spinal Cord

Spinal cord injuries at birth are rare, but catastrophic. They are associated with mid-forceps rotations and difficult breech extractions causing excessive longitudinal or lateral traction or rotation of the spinal cord. Upper cervical injuries are associated with rotations from a cephalic position; thoracolumbar injuries occur with breech rotations. The type of lesion varies from localized hemorrhage in the anterior cornua to complete destruction of the cord at one or more levels. Epidural bleeding of the cervical cord has been described as well. Clinical presentations include: 1) stillbirth or rapid neonatal death, 2) respiratory failure, and 3) generalized weakness. Among the clues to this diagnosis are paralysis and areflexia of the lower extremities with variable upper extremity involvement, a distended bladder, and patulous anus. These injuries should be considered in the differential diagnosis of infants who have generalized hypotonia. In nearly two thirds of cases, the diagnosis is confounded by the presence of associated hypoxic-ischemic encephalopathy. Radiographs of the neck should be obtained to rule out vertebral fracture or dislocation that possibly may be amenable to neurosurgical decompression. Bedside ultrasonography can identify swelling and hemorrhage in the cord. MRI allows a definitive diagnosis and can rule out a potentially treatable lesion such as occult dysraphism.

Most nerve palsies resolve spontaneously as swelling around the nerve root(s) resolves. For infants who have persistent weakness related to brachial plexus injuries, physical therapy to preserve joint mobility and avoid muscle tightness is indicated, beginning at about 7 to 10 days of age. Approximately 75% to 90% of patients eventually achieve complete recovery. Continued recovery can be seen out to 6 months after birth, but examination findings at 3 to 4.5 months are generally predictive of outcome. Microsurgical repair can be offered, but usually not before 6 months of age. Results of these procedures are mixed, but MRI examination of the nerve roots can be used as a helpful diagnostic adjunct to determine the best candidates for surgical repair. Infants who have diaphragmatic paralysis can be managed symptomatically with oxygen supplementation or mechanical ventilation, if needed, for respiratory failure. Surgical diaphragm plication should be considered for infants who remain ventilator-dependent for 6 to 8 weeks. The only indicated management for facial nerve palsy is preservation of moisture in the eye on the affected side with liquid tears or lid closure; 90% of infants recover within 4 weeks of birth. Infants in whom spinal cord injury is suspected should be stabilized and referred to a tertiary center for further evaluation and management. Initial stabilization includes maintenance of adequate oxygenation and perfusion using mechanical ventilation and volume infusions as needed. The bladder should be decompressed with an indwelling catheter, the spine stabilized, and the infant sedated.

Bone and Soft-tissue Injuries

Bone

The clavicle is the most common bone to fracture during delivery, with an incidence of 0.5% to 1.5% of live births. Most clavicular fractures occur during normal, spontaneous vaginal delivery, but the incidence is increased in the presence of shoulder dystocia and breech extractions that require vigorous manipulations. The most common long bone fracture is of the humerus. If nondisplaced, fractures can be asymptomatic. Clinical signs of a fracture include crepitus, pain, swelling, and decreased limb movement. It is not necessary to document clavicular fractures with a radiograph. Fractures of the humerus and clavicle can be associated with brachial plexus palsies. Limiting mobility of the affected arm frequently is the only needed treatment. Humeral fractures may require immobilization for 2 to 4 weeks. The femur is the most common bone fractured in the leg. Treatment of these fractures can require traction, suspension, and casting.

Soft Tissue

The most common form of traumatic birth injury is soft-tissue injuries, including petechiae, bruising, and subcutaneous fat necrosis. Petechiae from delivery usu-

ally are present over the head, neck, and upper thorax. These are present after birth, do not progress, and are not associated with other bleeding. If there is any doubt about any of these clinical points, a platelet count should be obtained. Severe vaginal or scrotal edema and bruising can be seen in breech deliveries. This usually resolves spontaneously, but severe scrotal swelling merits a urology opinion; drainage of a hematoma around the testes may be needed in rare cases. Subcutaneous fat necrosis is characterized by a localized area of induration due to local ischemia from trauma. The induration, with red or purple discoloration, usually presents late during the first postnatal week and resolves by 6 to 8 weeks.

Manual stretching of the neck can cause bleeding into the sternocleidomastoid muscle that can lead to torticollis. The mean age of presentation of torticollis is 24 days. The head typically tilts toward the side of the affected muscle and rotates toward the opposite side. Treatment with stretching exercises results in a 90% rate of recovery. Most superficial injuries are related to difficult extractions from the breech position, shoulder dystocia, and use of the vacuum and forceps. Although common, most of these injuries are minor and of importance primarily due to an increased risk for significant hyperbilirubinemia.

Intra-abdominal Trauma

Solid organ injury can be related to downward rib pressure and traction on supporting ligaments. Liver fracture and rupture of subcapsular hematomas have been reported. Clinical signs and symptoms include shock, pallor, anemia, abdominal distension, and bluish discoloration of the abdomen. Splenic rupture occurs less commonly than hepatic injuries. Treatment is supportive, with volume infusions, blood and clotting factor replacement, and surgical intervention as needed.

Outcome and Follow-up of Traumatic Birth Injuries

The outcome of traumatic birth injury is related to the severity of the initial injury. Superficial soft-tissue injuries resolve with the only sequelae being localized infection (scalp electrode) and neonatal hyperbilirubinemia (bruising and cephalohematoma). Subcutaneous fat necrosis can cause symptomatic hypercalcemia at 3 to 4 weeks of age that is characterized by vomiting, weight loss, decreased feeding, and irritability. Serial calcium determinations should be obtained in affected infants over the first month after birth. Fractures of the clavicle and humerus rarely are displaced and most commonly resolve without fixed immobilization. In 75% to 90% of cases, brachial plexus nerve injuries are stretch injuries, and arm function resolves completely over several days to 4 weeks. In the remainder of cases, some sequelae can be anticipated, and close follow-up is important to institute ongoing physical therapy and possible surgical intervention for nerve root repair if there is no return of function by 3 to 4.5 months of age. Facial nerve injuries are generally compression injuries that resolve over the first week of life. Spinal cord injuries often result in death or paralysis with dependence on long-term care. The prognosis is better for lesions below T4. Higher lesions are associated with poor motor outcome and recurrent respiratory infections. Disability correlates with the timing of onset of respiratory effort, extubation, and rate of motor recovery over the first 3 months after birth. If there is no clinical progress by 3 months, the prognosis is universally poor.

Infants who experience subarachnoid bleeding and convexity subdural hemorrhages generally develop normally without future seizure problems. Occasionally hydrocephalus or persistent focal neurologic findings result. Posterior fossa subdural blood collections can result in death related to brainstem compression, hydrocephalus, and long-term neurodevelopmental sequelae among survivors. Intracerebellar and intraventricular bleeding lead to a substantial number of survivors who have neurodevelopmental sequelae. The prognosis for subgaleal bleeding correlates with the degree of brain ischemia following delayed or incomplete correction of blood loss and hypotension. When birth trauma and asphyxia occur together, the prognosis depends on the degree of associated hypoxic-ischemic insult suffered perinatally. All infants at risk for neurodevelopmental sequelae should be monitored closely for attainment of developmental milestones. Physical and occupational therapy evaluations and preliminary developmental screening at 1 and 2 years of age with the Bayley Scales of Infant Development are indicated if milestones are not being reached. For older survivors, a preschool evaluation of development and school readiness is indicated. Infants discharged from the hospital on anticonvulsants should be followed for further seizures. If they remain seizure-free for 3 to 6 months, medication can be discontinued. Some recommend discontinuation only after normal findings on electroencephalography.

Suggested Reading

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NeoReviews Quiz

5. Traumatic birth injuries are caused by physical trauma during the birth process and involve different superficial and deep tissues. Of the following, the *most* common traumatic birth injury, excluding bruising and minor scalp trauma, is:

   A. Brachial plexus palsy.
   B. Clavicular fracture.
   C. Facial nerve injury.
   D. Intracranial hemorrhage.
   E. Liver laceration.

6. The clinical spectrum of traumatic injury to the head during birth varies from minor to potentially life threatening. Of the following, the *most* accurate statement regarding traumatic head injury during birth is that:

   A. Caput succedaneum usually enlarges after birth.
   B. Cephalhematomas are most often bilateral.
   C. Depressed skull fracture warrants surgical elevation.
   D. The most common risk factor for subgaleal hemorrhage is vacuum extraction.
   E. Ultrasonography is the modality of choice for the diagnosis of epidural hemorrhage.

7. Subdural hemorrhage results from tentorial laceration, falx laceration, or rupture of the superficial bridging cerebral veins. Of the following, the falx laceration *most* commonly involves rupture of the:

   A. Cavernous sinus.
   B. Inferior sagittal sinus.
   C. Straight sinus.
   D. Superior sagittal sinus.
   E. Transverse sinus.

8. Horner syndrome may be associated with brachial plexus injury and is characterized by miosis, ptosis, and enophthalmos on the affected side. Of the following, the *most* likely nerve root injured in Horner syndrome is the:

   A. Cervical 6.
   B. Cervical 7.
   C. Cervical 8 .
   D. Thoracic 1.
   E. Thoracic 2.

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