Perinatal strokes, which can result in devastating injuries to newborns, are sometimes caused by medical negligence during labor and delivery. To prove causation, lawyers need to understand how to identify and investigate the injury.
Strokes

Strokes that occur at or near the time of birth, which carry a substantial risk for abnormal neurodevelopment, are often overlooked as a significant contributing factor to cerebral palsy. As child advocates, plaintiff attorneys handling these cases must examine the facts closely: Was the perinatal stroke the preventable result of medical negligence that resulted in the child’s permanent brain damage?

Perinatal arterial ischemic stroke (PAIS)—a known cause of long-term neurologic impairment—is caused by a disruption in blood flow to a major cerebral artery, resulting in an area of damaged cerebral tissue. It can be the result of thrombosis, embolism, trauma, compression, hypoxic–ischemic encephalopathy (blood and oxygen deprivation to the brain), vasospasm, or obliteration by an inflammatory (infectious) process. While PAIS is occasionally detected in the posterior, anterior, or multiple cerebral arteries, the middle cerebral artery is the most common blood vessel involved.

Plaintiff attorneys must have a general understanding of brain perfusion (how blood passes through the brain) to ascertain the potential origin of the injury—and its possible causes and effects—to better evaluate, investigate, and prove these cases.

Arterial Blood Supply to the Brain

The brain is one of the most highly perfused organs in the body, meaning that—like the liver, heart, and kidneys—it depends on adequate oxygen and nutrients being supplied through a dense network of blood vessels. Cerebral blood flow begins at the heart, and it permeates through the carotid arterial system (two blood vessels in the neck divided into internal and external carotid arteries) and the vertebral arterial system, which consists of two arteries that create the basilar artery in a complex called the “vertebrobasilar system.” This system supplies blood to the brain when the carotid arteries cannot. The basilar artery and the internal carotid arteries also join with other arteries to form an arterial ring at the base of the brain called the “Circle of Willis.” (See Fig. 1, p. 48).
The internal carotid arteries branch to form the anterior cerebral artery and the middle cerebral artery (MCA), which is the largest cerebral artery. The MCA supplies blood to the parts of the brain that control the primary motor and sensory areas of the face, throat, hand, and arm—as well as the areas that control speech. An MCA stroke results in a sudden onset of neurological deficits related to these areas—such as seizures—due to cerebral infarction (an area of necrotic tissue) or ischemia (an area with inadequate blood supply) in the parts of the brain supplied by this artery. Nearly all unilateral lesions—those occurring in only one hemisphere of the brain—in the perinatal period involve the MCA.

**Mechanisms of Injury**

Brain imaging shows evidence of brain lesions in 80 percent of newborns with “neonatal encephalopathy”: full-term infants who show neurological impairments within the first few days of birth. Early and serial MRI examinations are extremely important—they detect not only the existence of lesions but also when they formed. Several factors during childbirth can cause ischemia in a newborn.

**Direct trauma to an intracranial vessel.** Brain ischemia within the regions supplied by the internal carotid arteries may be caused by direct trauma to the newborn’s head. Direct trauma can occur during a complicated delivery—for example, excessive use of force during a Zavanelli maneuver or from a vaginal, operative, or instrumental delivery. In one case, the obstetrician fractured the baby’s skull while removing the baby’s head from the mother’s pelvis by hand, causing an intracranial bleed in the newborn. The newborn suffered a seizure disorder, feeding difficulty, and respiratory distress. Today, the child has cerebral palsy as a direct result of delivery trauma.

**Practice tip.** It is not uncommon for the delivery record to be vague—or completely silent—as to the placement of the forceps, the number of times the forceps were used, and the number of pulls on the baby’s head. When handling these cases, it is crucial to question the delivering physician at both deposition and trial to discover that information. Also ask the client for other sources of this information, such as a birth video or photographs. When explaining forceps placement and dissection or compression of the artery to the jury, be sure to use diagrams and animations.

**Vacuum.** Other medical issues that may lead to mechanical vaginal deliveries are prolonged rupture of membranes, a prolonged second stage of labor, cephalopelvic disproportion (when the baby’s head is too big to fit through the birth canal), and otherwise difficult deliveries. Similar to forceps, vacuum-extracted deliveries can result in major fetal trauma, including subdural, subgaleal, or cerebral hemorrhage; shoulder dystocia with a risk for brachial plexus injury; convulsions; central nervous system depression; mechanical ventilation; and cerebral infarction. In one study, for
example, trauma from a vacuum-assisted delivery caused stretching of the middle and posterior cerebral arteries, leading to skull fracture, ischemia, and cerebral infarction. In another study, 80 percent of perinatal strokes occurred in complicated deliveries, two-thirds of which occurred in failed vacuum-assisted deliveries.

Practice tip. Newborns with infarcts generally have abnormal neurodevelopmental and lower cognitive test scores compared to newborns with similar patterns of brain injury without infarcts. As part of your damages argument, have a pediatric neuropsychology expert explain to the jury the resulting cognitive deficits that are not apparent to the naked eye—such as impaired concentration, hyperactivity, or poor communication skills. You can turn the results of the expert’s diagnostic tests, such as the Behavior Assessment System for Children, into demonstrative exhibits to use at trial in conjunction with the testimony. For example, use day-in-the-life videos to demonstrate to the jury the impact of the injuries on the child’s everyday life.

It is crucial to explain to the jury that health care professionals may violate the standard of care when they incorrectly use an instrument, use the wrong type of instrument, or fail to consider safer alternative approaches. For example, to properly apply forceps, the user must know the fetal position and the degree of asynclitism (how far a baby’s head is tilted to one side). And certain conditions—including incomplete cervical dilation, inadequate maternal pelvic size, and a previously unsuccessful vacuum extraction attempt—should alert medical professionals that vacuums or forceps should not be used.

Carefully review the medical records to see if any of these circumstances existed: They are essential to support your argument that the health care professional’s or medical facility’s negligence caused the injury.

Practice tip. Oxytocin is a naturally occurring substance in the human body associated with childbirth, but Pitocin

**Was the perinatal stroke the preventable result of medical negligence that resulted in the child’s permanent brain damage?**

Fetal station” is determined by the degree of descent of the baby’s head through the birth canal in relation to the mother’s pelvis. As the baby moves down, contraction forces on the fetal umbilical cord and cerebral vasculature impede blood flow. These alterations, called “decelerations,” are evaluated by monitoring the fetal heart rate. Early decelerations—a sign of head compression—are apparent when the lowest point of the baby’s heart rate coincides with a contraction’s peak.

Generally, natural labor forces do not cause permanent injury to the baby despite compression of the fetal head. But fetal injury due to prolonged and excessive uterine activity during labor has long been documented: It can result in oxygen deprivation to the fetus or fetal cardiac impairment, among other complications, all of which may ultimately lead to cerebral ischemia.

Regional cerebral ischemic injury, however, also can occur from direct trauma caused by excessive labor forces. This is known as cranioencephalocerebral compression ischemic injury, which can lead to craniocephalocerebral compression ischemic encephalopathy (CCIE). This injury occurs when the interval between the mother’s contractions is less than one minute—a baby requires at least that amount of time to reperfuse his or her brain with oxygenated blood.

Drugs commonly used to induce labor are associated with excessive uterine activity that may result in these perinatal injuries. For example, oxytocin or Pitocin, which prepare the cervix for labor induction, may lead to excessive uterine activity during labor, as well as abnormal fetal heart rate patterns. The mechanisms of injury from excessive uterine activity as the fetal head passes through the mother’s pelvis are the same as for other types of trauma injuries. Severe molding, bruising, or fracturing that manifests externally as bleeding may manifest internally as an intracranial hemorrhage that may cause an embolism. Additionally, CCIE caused by decreased blood flow resulting from external compression and increased intracranial pressure may also lead to ischemic stroke.

**Practice tip.**
is a synthetic oxytocin that affects mothers in a different way. The best way to determine if Pitocin has caused excessive uterine activity is by reviewing the fetal heart monitoring strips. Count how many contractions the mother is experiencing every 30 minutes: If she is experiencing more than 15 contractions in that time period, then she is experiencing excessive contractions.

Keep in mind that Pitocin is not a dose-dependent drug. Each individual responds differently to the dosage, so a dose of Pitocin that may be appropriate for one patient may be excessive for another. In 2007, the FDA issued a “black-box” warning—the strongest warning—about Pitocin, recommending that it only be used when induction of labor is medically necessary or in select cases of stalled labor.55

Trauma from head compression caused by cephalopelvic disproportion. A disparity between the size of the fetus and the maternal pelvis can also result in increased uterine activity, exacerbating trauma. A mother who is post-40 weeks gestation, for example, or one who has gestational diabetes, a small maternal pelvis, or a large baby,56 may cause a mismatch between the fetal head and the ischial spines, the narrowest inlet of the maternal pelvis.57 This is known as cephalopelvic disproportion (CPD).

Factors that increase the relative maternal-fetal size mismatch, such as an occiput-posterior position (the back of the baby’s head is against the mother’s spine),58 can lead to prolonged labor and traumatic birth.59 CPD also alters the progress of labor, which may be complicated by the baby’s failure to descend or the cervix’s failure to dilate—all increasing the risk of neonatal arterial ischemic stroke.60

Practice tip. Attorneys should examine the medical record for descriptions of the doctor’s vaginal examinations during the active phase of labor, which can demonstrate whether these issues were present during delivery.

Investigating the Injury

Keep in mind that newborns with PAIS usually seem like healthy babies after birth, with normal Apgar scores and cord pH values, and they often remain with their mothers in the postnatal ward. But their conditions deteriorate within three days of delivery: Newborns may experience seizures (generally presenting as apnea—episodes of not breathing), hypotonia (low muscle tone), jitteriness or lethargy, irritability, or poor feeding. They may also experience “dusky episodes,” occasionally turning blue around the mouth. It is crucial for health care providers to understand and recognize abnormal signs and symptoms associated with PAIS in the hours after birth and closely watch at-risk infants.

When handling these cases, it’s critical to vet for potential risk factors. Focus on clinically and statistically significant risk factors—such as abnormal placenta pathology—to overcome anticipated defense testimony as to speculative risk factors. Pay careful attention to the prenatal and neonatal records to rule out less likely causes for arterial cerebral infarction, including congenital heart disease, bacterial meningitis, and blood clotting disorders.

Other factors that increase the risk of PAIS are oligohydramnios (low amniotic fluid); preeclampsia; prolonged rupture of membranes; cord abnormality, such as when it has a single artery; chorioamnionitis (bacterial infection that moves up the vagina to the uterus after a mother’s waters have broken); and primiparity (when a mother is giving birth to her first child).

When you suspect arterial cerebral infarction, ask your client about the types of diagnostic imaging taken within the first three to seven days after birth. Then, request original formats of those scans so you can send them to your causation experts—such as a pediatric neuroradiologist—to support your theories of causation and timing. You should also retain all subsequent therapy records. Through expert testimony, you can demonstrate the various cognitive and physical disabilities your client suffered—and will continue to suffer—throughout his or her life.

Newborn stroke is a complicated medical issue—and one that is often overlooked as a cause of neurological deficits. Understanding the biology behind them will help you hold medical professionals accountable for the devastating consequences of this injury.

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Understanding Newborn Strokes

Notes
1. Perinatal strokes occur in up to 15 percent of term newborns. Mamdouha Abdab Barmada et al., Cerebral Infarcts with Arterial Occlusion in Neanotes, 6 Annals of Neurology 495, 495–96 (1979).
3. This article will not focus on neonatal cerebral sinovenous thrombosis or hemorrhagic strokes.
6. See Neuroscience, supra note 4.
10. A Zavanelli maneuver involves pushing the fetal head back into the birth canal in anticipation of a Caesarean section.
11. The baby also suffered from hypoxia as a result of negligence during the intrapartum period.
14. Id.
15. See, e.g., Govaert et al., supra note 9.
16. See Mary A. Rutherford et al., Neonatal Stroke, 97 Archives of Disease in Childhood F377 (2012); see also Manej Kumar et al. Contralateral Cerebral Infarction Following Vacuum Extraction, 21 Am. J. Perinatology 15, 17 (2004) (“Mechanical birth trauma has been recognized as a direct cause of intracranial arterial injury leading to ischemic or hemorrhagic stroke in the newborn. The trauma could result from the process of natural birth or from assisted instrumentation with forceps or ventouse [vacuum]”).
17. Con Sreenan et al., Cerebral Infarction in the Term Newborn: Clinical Presentation and Long-Term Outcome, 137 J. Pediatrics 351, 353 (2000).
18. Uros Roessmann et al., Thrombosis of the Middle Cerebral Artery Associated with Birth Trauma, 30 Neurology 889 (1980).
19. See, e.g., Govaert et al., supra note 9, at 843.
20. The second stage of labor starts when the cervix is fully dilated and ends when the baby is born. Danforth’s Obstetrics and Gynecology 23 (Ronald S. Gibbs et al. eds., 10th ed. 2008).
22. See, e.g., Dena Towner et al., Effect of Mode of Delivery in Nulliparous Women on...
24. 83.3 percent of the newborns had arterial infarcts. See Ramaswamy et al., supra note 21, at 2089.
25. Ramaswamy et al., supra note 21.
27. Id.; see also Dena R. Towner & Mary C. Ciotti, Operative Vaginal Delivery: A Cause of Birth Injury or Is It? 50 Clinical Obstetrics & Gynecology 563 (2007).
29. See id.
32. Govaert et al., supra note 9, at 844–45.
33. See Andrew P. Harris et al., Efficacy of the Cushing Response in Maintaining Cerebral Blood Flow in Premature and Near-Term Fetal Sheep, 43 Pediatric Research 50 (1998).
34. Leon I. Mann et al., The Effect of Head Compression on FHR, Brain Metabolism and Function, 39 Obstetrics & Gynecology 721, 726 (1972) (suggesting further research is warranted).
37. See Danforth’s Obstetrics and Gynecology, supra note 20, at 28 fig.28.
40. See Janyne E. Althaus et al., Cephalopelvic Disproportion Is Associated with an Altered Uterine Contraction Shape in the Active Phase of Labor, 195 Am. J. Obstetrics & Gynecology 739 (2006); see also Towner et al., supra note 22.