Neonatal Encephalopathy & Cerebral Palsy

Knowledge about the causes of neonatal encephalopathy and cerebral palsy is rapidly developing. A report published in 2003 by a joint task force of the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) shed new light on the cause of neonatal encephalopathy and cerebral palsy.

Previously, evidence of birth asphyxia and hypoxic-ischemic neonatal encephalopathy was based upon four nonspecific clinical signs: (1) meconium-stained amniotic fluid, (2) nonreassuring fetal heart rate patterns, (3) low Apgar scores, and (4) neonatal encephalopathy. The evidence-based findings of the ACOG Task Force demonstrate that these peripartum signs are most often the secondary results of pathologic processes established before labor. New data show that less than a quarter of infants with neonatal encephalopathy show evidence of hypoxia or ischemia at birth. In most cases the events leading to cerebral palsy are caused by numerous and unpreventable factors during fetal development or after delivery. A large proportion of cerebral palsy cases are associated with conditions such as pre-term birth, intrauterine growth restriction, intrauterine infection, coagulation disorders, multiple pregnancies, antepartum hemorrhage, breech presentation, and chromosomal or congenital abnormalities. Physicians should therefore be aware that these four nonspecific signs should no longer be used as the main evidence for a diagnosis of birth asphyxia.

ACOG has issued new criteria to define an acute intrapartum hypoxic event. The nine criteria help to assess the likelihood that the pathology causing the cerebral palsy happened during labor. They focus on the analysis of peripartum blood gases as essential to demonstrate that hypoxia was present around the time of birth. All four of the following essential criteria must be present to define an acute intrapartum hypoxic event sufficient enough to cause cerebral palsy:

1. Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit ≥12 mmol/L)
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type
4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders

If any one of the four essential criteria is not met, intrapartum hypoxia did not likely cause the cerebral palsy. If all four criteria are met, you must then establish whether the hypoxia is due to long-term hypoxia, or whether hypoxia occurred acutely during labor in a previously healthy fetus. Five criteria are reported to collectively suggest an intrapartum timing (within 48 hours of labor and delivery) but are nonspecific to asphyxial insults. Most or all of these five signs will be present as a group in severe cases of intrapartum hypoxia.

1. A sentinel hypoxic event (e.g., ruptured uterus) occurring immediately before or during labor
2. A sudden and sustained fetal bradycardia or the absence of fetal heart rate variability in the presence of persistent, late, or variable decelerations, after a hypoxic sentinel event when the pattern was previously normal
3. Apgar scores of 0–3 beyond 5 minutes
4. Onset of multisystem involvement within 72 hours of birth
5. Early imaging study showing evidence of acute nonfocal cerebral abnormality
Neonatal Evaluation

Assessing the depressed newborn, especially after an uncomplicated delivery, should include:

1. Consideration of maternal and family medical conditions, such as
   a. Thyroid or other immune disorders
   b. Deep vein and other thrombotic disorders
   c. Intolerance to oral contraceptives
   d. Early stroke or myocardial infarction
   e. History of prior pregnancy loss
      i. Death of twin fetus, even early in the pregnancy?
      ii. Maternal history of chorioamnionitis or sexually transmitted disease?
      iii. Evidence of intrapartum maternal fever?

2. Examination of the umbilical cord, membranes, and placenta

3. Immediate laboratory studies
   a. Examination of the placenta, cord, and membranes
   b. Cord gases
   c. Placental cultures
   d. Maternal and/or fetal blood cultures

4. Later laboratory studies
   a. Imaging studies
   b. Liver function tests and renal function tests
   c. In the event of signs of thrombosis or stroke, examine indicators of thrombophilia
   d. Consider submitting placenta for formal pathologic investigation

Conclusion

Much remains to be learned about the causes and prevention of cerebral palsy. The 2003 ACOG/AAP report reveals new information that might alleviate undue blame on physicians for cerebral palsy, especially when labor management was not ideal. Further research into the causes of neonatal encephalopathy and cerebral palsy will hopefully lead to interventions that will reduce the incidence of these conditions.

Key Points

Physicians are strongly advised to:

◆ Document whether or not the four criteria exist that define an acute intrapartum hypoxic event for all newborns who may have or develop neonatal encephalopathy or cerebral palsy. The documentation should be done as close as possible to the time of delivery.

◆ Document whether or not the five criteria exist which suggest an intrapartum timing but are nonspecific to asphyxial insults.

◆ Unless you have determined so from the above-mentioned criteria, refrain from using terms such as “newborn asphyxia,” “birth trauma,” or “hypoxic/ischemic encephalopathy from birth.”

◆ Refrain from mentioning a diagnosis such as “newborn asphyxia” to parents unless you have confirmed the information in the chart or with the delivering physician. Ensure that all members of the delivery team are cautioned on this point.

◆ Read the entire ACOG/AAP report, Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology.
Reference
