Proving HIE in a Baby Case - Using Evolving ACOG Literature as Support for a Change

For years, the American College of Obstetricians and Gynecologists (ACOG) has published guidelines. Over the past several decades, these guidelines have become largely self-serving documents, arguably written exclusively for the purpose of assisting the defense of its members in lawsuits.[1]

Perhaps the most vivid example is in the area of the cause of neonatal brachial plexus injuries (NBPP). In 1998, ACOG produced a guideline which candidly stated "Brachial plexus injuries result from excessive lateral traction on the fetal head in attempting to dislodge the anterior shoulder.... *The key to preventing fetal injury is avoidance of excess traction on the fetal head.*"[2] Undoubtedly inspired by a desire to defend its members in lawsuits, the most recent ACOG position now takes the completely contrary position, stating "neither high-quality nor consistent data exists to suggest that NBPP can be caused only by a specific amount of applied force beyond that typically used by healthcare providers during any delivery. Instead, available data suggests that the occurrence of NBPP is a complex event, dependent not only on the forces applied at the moment of delivery, but also on the constellation of forces that have been acting on the fetus during the labor and delivery process, as well as individual fetal tissue characteristics."[3] Given that the nerve structure of babies has obviously not changed in the past twenty years, nor the way in which babies are delivered, it is not hard to understand the motivation behind this radical change in ACOG's position.

However, on the topic of neonatal hypoxic-ischemic encephalopathy (HIE), ACOG's most recent monograph actually takes a somewhat softer approach than its predecessor. The criteria established by ACOG in 2003 for defining an acute intrapartum event sufficient to cause cerebral palsy was very rigid. Per the guideline, there were four "essential criteria" which must be met to satisfy causation, including (1) evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH $<7$ and a base excess $\geq -12$); (2) an early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks gestation; (3) cerebral palsy of the dyskinetic type; and (4) exclusion of other identifiable etiologies such as trauma, coagulation disorders, infection conditions, or genetic disorders.[4] The guideline then listed five additional criteria which "collectively suggest an intrapartum timing" (within about 48 hours of labor and delivery) including a sentinel hypoxic event occurring immediately before or during labor, a sudden and sustained fetal bradycardia after a hypoxic sentinel event when the pattern was previously normal, APGAR scores of 0-3 beyond 5 minutes, onset of multisystem involvement within 72 hours of birth, and early imaging studies consistent with acute non-focal cerebral abnormality.[5]

By contrast, the second edition of ACOG's neonatal encephalopathy monograph states in its preface that "for the current edition, the task force concluded that a broader perspective might be more fruitful." Rather than specifying four essential criteria which must be met, the 2014 guideline sets forth numerous factors which are "consistent with an acute peripartum or intrapartum event," rather than a rigid checklist of "essential criteria" as was the case in 2003.[6]

Moreover, ACOG's 2014 guideline softens the specific criteria for establishing an acute

event. First, whereas the 2003 guideline used an APGAR threshold score of 0-3 beyond 5 minutes, the 2014 guideline uses an APGAR of less than 5 at 5 and 10 minutes.[7] With regard to cord gases, the 2003 guideline used a pH < 7 **and** a base excess ≥ -12, whereas the 2014 guideline utilizes a pH < 7, **or** a base excess ≥ -12.[8]

In addition, the 2014 ACOG guideline contains additional language which can often be helpful to the victim. Extremely helpful language is found at page xxiv of the executive summary which states "the patient who presents with a category I heart rate pattern that converts to category III...is suggested of a hypoxic/ischemic event." The next paragraph is also quite helpful, stating "additional fetal heart rate patterns which develop after a category I fetal heart rate pattern on presentation, which may suggest intrapartum timing of a hypoxic/ischemic event, include tachycardia with recurrent deceleration, and persistent minimal variability with recurrent deceleration." Therefore, if the fetal tracings in question are consistent with these criteria, the 2014 ACOG guideline can provide valuable support.

I handled a recent case in Georgia which is illustrative of how the ACOG neonatal encephalopathy guidelines can be now used as support. The case involved a mom who was 33 weeks pregnant when she was involved in a serious motor vehicle accident which included direct abdominal trauma. She was immediately taken to the hospital and admitted to labor and delivery fo observation. The baby was born the following day with severe cerebral palsy, which we contended resulted from hypoxia-ischemia in the hours before birth, as a consequence of failing to perform a c-section in the face of clear indications. The defense was focused largely on causation, contending that the injury was traumatic in nature from the motor vehicle accident. The baby's APGAR scores were 2 at 5 minutes and 4 at 10 minutes, which satisfied the criteria of the 2014 guideline, but would not have satisfied the criteria of the 2003 guideline. Further, while the pH was over 7, the base excess was -12.4, which also satisfied the 2014 guideline, but not the 2003 guideline. There was also no dispute that the fetal monitoring strips were category I strips at admission, which subsequently converted to category III strips just prior to delivery following persistent minimal variability with recurrent late decelerations. Lastly, I was able to utilize very useful language in the maternal trauma section of the monograph which states "direct attribution of fetal trauma during pregnancy as a causative event of childhood neurologic sequelae is difficult."[9] There is no doubt that using the 2014 ACOG guideline as support certainly added significant credibility to the case.

In sum, the practice of ACOG of writing self-serving guidelines, and then having its authors serve as frequent defense experts as they bask in the glow of the masterpieces they have created has not changed. Though the 2014 ACOG neonatal encephalopathy guideline was obviously not written to assist victims and their families, and much of the criteria is still overly strict and must be vigorously challenged, the guideline can often be used in support of victims and their families in these extremely important cases.

1. ACOG freely acknowledges that it has a political purpose. In 2010, ACOG formed a related entity under the same acronym called the American Congress of Obstetricians & Gynecologists, which, per the ACOG website, is focused on "socioeconomic, political and grievance activities for its members."

2. See ACOG's Precis, An Update in Obstetrics and Gynecology, 1998, at 95.

3. See ACOG's Neonatal Brachial Plexus Palsy, 2014, at xvi.

4. See ACOG and The American Academy of Pediatrics' Neonatal Encephalopathy and Cerebral Palsy – Defining the Pathogenesis and Pathophysiology,, 2003, at xvii.

5. See id.

6. See ACOG and The American Academy of Pediatrics' Neonatal Encephalopathy and Neurologic Outcome,, 2nd Edition, 2014 at xx. It is worth noting that while the American *College* wing of ACOG released these two "educational" publications in 2014, the topics covered are the two areas in which its members are sued the most by far, such that these monographs serve primarily the role of the American *Congress*'s efforts to aid its members.

7. See Neonatal Encephalopathy and Cerebral Palsy – Defining the Pathogenesis and Pathophysiology, 2003, at xvii; Neonatal Encephalopathy and Neurologic Outcome, 2014, 2nd Edition, at xxii.

8. See id.

9. See Neonatal Encephalopathy and Neurologic Outcome, 2nd Edition 2014, at 38.