Medical malpractice: Preparation and trial of birth injury cases

A look at continuing developments in the field, including history of fetal monitoring and causation issues

By James Bostwick

Over the past three decades the development and trial of birth injury cases due to intra-partum hypoxic ischemic encephalopathy has evolved dramatically. During the 1960s and early 1970s, the only method of monitoring a fetus during labor was by auditory auscultation (listening for changes in fetal heart rate during the contractions). In the late 1970s and early 1980s, electronic fetal monitoring was developed and it soon became a standard method for monitoring fetal well-being during labor (recordation of the fetal heart rate and the uterine contraction by either internal or external means on a continuous paper strip). It became widely used because it provided the only available window as to the well-being of the fetus. Over time, certain abnormalities suggesting "fetal distress" became recognized and it was recommended that intervention occur to prevent asphyxial injury to the fetus.

During the 1990s, the American College of Obstetrics and Gynecology (ACOG) began to realize that this essential monitoring tool was also providing recorded evidence concerning the fetus's condition for birth injury litigation. Considerable attention was devoted to developing strategies which expert witnesses and defense lawyers could use to nullify or lessen the impact of abnormal fetal heart tracings of children that later proved to have cerebral palsy. Major retrospective studies were undertaken which ostensibly provided evidence that over time the use of fetal monitoring made little or no difference in the percentage of children born with cerebral palsy. Extensive effort also went into the analysis of the pathology of the placenta, neonatal presentation and neuro-imaging findings; these were studied extensively looking for alternative explanations or potential markers which could suggest a given patient had not suffered an intra-partum insult. Attorneys specializing in this area over the decades have seen a variety of inventive hypotheses put forth as the *causation defense du jour*, including fetal nucleated red blood cells, infection, mediated cytokine cascade causing systemic inflammatory response (SIRS), as well as claimed placental abnormalities such as villitis, intravillus thrombi, low placental weight, umbilical cord abnormalities, eccentric cord insertions, nuchal cord, cord knots, etc.

By the early turn of the 21st century, this collaborative effort to create a systematic defense modality to counter successes in birth injury litigation culminated in the 2003 ACOG Task Force publication: Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. This "white paper" purported to outline criteria that were deemed essential in order to establish a causal link between claimed intra-partum hypoxic events and cerebral palsy. Ostensibly this was designed to provide the practitioner with definitions of intra-partum asphyxia and cerebral palsy. In reality it appeared to many knowledgeable observers the goal was to create exclusive criteria which experts testifying in birth injury cases would have to use to establish causation.

The publication listed certain "essential criterion" that had to be met before cerebral palsy could be attributed to an intra-partum event. They were as follows:
1. Evidence of an umbilical cord pH of less than 7 *and* a base excess (a component of the blood gas analysis intended to show the accumulation of lactic acid sufficient to cause brain damage) of 12 or greater.
2. Early onset of severe or moderate neonatal encephalopathy in infants at 34 or greater weeks in gestation.
3. Spastic or dyskinetic cerebral palsy.
4. *Exclusion* of other possible causes, such as infection, metabolic coagulation and genetic disorders. [The battles here were fought primarily concerning numbers 1 and 4.]

According to this study, there were also several "non-essential criteria" which, while not specific for an asphyxial injury, collectively would suggest that an injury had occurred during labor and delivery:
1. A sentinel hypoxic event occurring immediately before or during labor (such as a prolapsed cord, placental eruption or a maternal and/or fetal hemorrhage).
2. A sudden and sustained fetal bradycardia (reduced heart rate) or the *absence* of normal variability in the fetal heart rate with persistent, late or variable decelerations.

3. Apgar Scores (the assessment of a newborn's status on a scale of 0 to 10) of *0 to 3* when a baby is five minutes old or older.
4. Onset of multi-system organ damage within 72 hours of birth.
5. Early imaging studies showing an acute, non-focal cerebral injury. [The battles here were fought primarily on numbers 2, 3, 4 and 5.]

These essential criteria had been developed in earlier ACOG bulletins and other medical journal articles in order to bolster defense positions in birth trauma litigation. Both prior to the ACOG publications in January of 2003 and subsequent thereto, there were a multitude of criticisms concerning the various criteria. Many authors suggested that a cord blood pH cut-off of less than 7 was artificially low and failed to include many babies who clearly had suffered intra-partum asphyxia. Apgar scores are notoriously subjective and many children suffering clear intra-partum events and other signs of intra-partum hypoxic ischemic encephalopathy are often awarded Apgars higher than 3 at five minutes.

Seizures are a common sign of intra-partum asphyxial injury and were not even mentioned as one of the criteria.

Experienced observers noted that in clear cases of intra-partum asphyxia there often was not a sudden and sustained fetal bradycardia; further, that the complete absence of variability in the fetal heart rate, when there are persistent late or variable decelerations is rarely seen in clearly asphyxiated children. Investigators have frequently argued that a base excess greater than 12 is highly suggestive of ongoing intra-uterine asphyxia, whether or not the blood pH was less than 7. (There are a variety of circumstances that can artificially elevate the arterial cord gas pH in an asphyxial event including variations in maternal blood pressure and an occult or a frank cord prolapse.) There also was huge controversy about the significance of early imaging findings.

National Fetal Monitoring Standards

All but one of the criteria described above deal with the retrospective analysis of causation after the occurrence of a potential intra-partum event. Only one of the criteria, that which relates to fetal monitoring, provides a prospective as well as a retrospective tool for the analysis of fetal well-being. Only fetal monitoring can potentially determine on a real time basis whether a fetus may be suffering intra-partum asphyxia and therefore needs immediate conservative and/or emergent intervention. In the litigation context, defense experts continued to cite the old studies, claiming there is no decrease in the number of cerebral palsy children since the advent of the use of fetal monitoring. On the other hand, monitoring is widely used by all practitioners; indeed, not even the staunchest defense experts are willing to forego fetal monitoring in the care of their own patients. Why? It is clearly the only helpful tool that exists to provide evidence concerning fetal status. Studies make it clear that suboptimal EFM practices contribute to the occurrence of fetal asphyxia and consequent birth injuries.

In Sweden, there was a study of 177 children with severe labor-related asphyxia; they found that approximately 50 percent of the cases were related to physician and nursing malpractice. It was the conclusion of the authors that the failure to identify ominous fetal heart rate patterns associated with fetal hypoxia and acidosis and to initiate appropriate interventions had the clear potential to lead to severe asphyxia, death and/or cerebral palsy.[1] Another Swedish study concluded that intra-partum events are very important potential causes of post-natal neurologic symptoms because they are preventable; they note that misinterpretation of electronic fetal monitoring patterns is often a contributing factor in cases of asphyxia.[2]

As far back as 1997, a Taskforce met and started the process of developing standardized definitions for interpretations of fetal heart rate tracings and for defining the presence of abnormal electronic fetal monitoring patterns as it related to the prediction of the potential for fetal compromise. These very basic definitions establishing a normal baseline (110-160 beats per minute), the (reassuring) importance of accelerations of more than 15 beats per minute for at least 15 seconds and the absence of prolonged variable or late decelerations (non-reassuring), and of course, the importance of normal fetal heart rate variability (short and long term variations of heart rate). They stated that guidelines needed to be developed for clinical management of fetuses with tracings between the extreme of normal and the obvious ominous pattern, so that clinicians could be alerted to the development of intra-partum asphyxia and recognize it early enough to prevent injury or death.

In 2005, despite the fact that fetal monitoring is utilized in about 85 percent of live births in the United States, ACOG published a New Practice Bulletin which essentially stated that it was unrealistic to expect that a non-reassuring fetal heart rate tracing could predict cerebral palsy. The practice bulletin went on to assert that there was a wide variation in the way obstetricians interpret and respond to fetal monitoring tracings. If true, this certainly would seem to cry out for more definitive standards to prospectively help the practitioner deal with potentially concerning fetal heart rate patterns.

Finally, in 2008, ACOG updated the definitions for fetal monitoring from the 1997 guidelines and proposed that clinicians adopt a new three-tiered system for categorizing fetal heart rate patterns. Those categories are:
Category 1 is essentially normal fetal heart tones with a normal baseline rate and moderate fetal heart rate variability. There may be decelerations and either present or absent accelerations.
Category 2 is (the large, gray area) defined as all patterns not classified as Category 1 or Category 3. These patterns are

described as "indeterminate and don't predict abnormal fetal acid-base status and require 'evaluation and continued surveillance.'" These situations would include bradycardia, provided that there's not *totally absent* variability, tachycardia, decreased or minimal variability, absent variability with no current decelerations, marked variability, absence of accelerations after scalp or other stimulation, recurrent variable decelerations with decreasing or minimal variability, prolonged decelerations (less than 10 minutes), recurrent late decelerations with decreasing or minimal variability, and variable decelerations with a slow return to baseline.

Category 3 tracings are considered to be abnormal and "may" predict abnormal fetal acid-base status at the time of observation. These include sinusoidal patterns or *absent* variability with recurrent late decelerations, recurrent variable decelerations or bradycardia. (Note here that they have tightened the definition of bradycardia indicating abnormal status only includes low heart rates with *absent* variability.)

For quality clinical care, it would have been best to provide clear and comprehensive guidelines especially in the gray areas of Category 2. The Category 3 situations are already potential disasters. Many of the scenarios in Category 2 may be developing situations that need intervention before it is too late to alter the outcome. In addition, even though the bulletin clearly established that Category 3 patients are *abnormal* and that they may *predict* abnormal fetal basis acidosis status, all they required the clinicians to do is to make an "effort to expeditiously resolve the abnormal pattern." In describing the appropriate interventions, they mention all of the standard, conservative attempts to change fetal status, such as administering oxygen, change in position, discontinuing stimulation and treating maternal hypotension, but stunningly, they never bothered to mention *surgical intervention or other expedited delivery*. It was evident that these "guidelines" were purposely worded in a vague and ambiguous fashion, with little concern for the clinician or their patient and an overriding goal of providing no definitive statements that might be utilized in birth injury litigation.

In the years after the 2008 Taskforce paper was published, ACOG continued to emphasize the limitations of electronic fetal monitoring, its uncertain efficacy and its high false-positive statistics in their annual practice bulletins. Yet, in practice, most of the live births in America continued to be assessed with electronic fetal monitoring because clinicians were aware this remained the only method to determine the well-being of the fetus. The practicing obstetricians intuitively recognized the obvious explanation for these misleading statistics. The overall prevalence of cerebral palsy failed to diminish with the use of fetal monitoring simply because the greatest percentage of cerebral palsy cases are caused by events before the onset of labor, only a relatively modest number can be in whole or in part attributed to intra-partum events. The other obvious reason the incidence of cerebral palsy has not decreased is the remarkable advances made in the neonatal care of the premature infant. Many premature children that would have died in the past now survive; unfortunately, they often have the complications common to survivors of early gestation, such as cerebral palsy. So, while fetal monitoring may have improved the chances the term infant that gets into difficulty during labor will avoid cerebral palsy, the increasing number of premature children with cerebral palsy has kept the overall statistics from showing an improvement. In the years after 2008 it continued to be clear that clinicians needed more definitive guidelines to help them make intervention decisions, but ACOG continued to rely on its vague litigation-oriented definitions.

2014, a breath of fresh air

In March of 2014, the American Academy of Obstetrics and Gynecology and the American Academy of Pediatrics published a Second Edition entitled *Neonatal Encephalopathy and Neurologic Outcome*. In this document, there are finally some major changes in the evaluation of neonatal encephalopathy causation and the eventual neurologic outcome, particularly as it relates to the role of asphyxia occurring during intrapartum events.

In the First Edition, as discussed above, the Taskforce outlined essential criteria necessary to establish a causal link between intra-partum hypoxic events and the subsequent development of cerebral palsy. This Second Edition clearly reflects a broader perspective put forth by the current Taskforce. It proposes that this process should involve a comprehensive multi-dimensional assessment of neonatal status to determine the likelihood that an acute hypoxic event occurred in and around the labor and delivery timeframe and contributed to a neonatal encephalopathy resulting in a long-term neurologic injury. They have redefined portions of the essential and non-essential criteria in some very specific ways that are more inclusive and more reflective of the scientific evidence that has been available for the last two decades. They admit they do not have a definitive test or set of markers that reliably establish that neonatal encephalopathy is or is not attributable to an acute intra-partum event. They recognize the importance of being able to assess that issue as a matter of probability and, in this publication, provide significantly more definitive and more inclusive tools to aid in making that determination.

Case definitions

They now define neonatal encephalopathy as a syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, now manifested by a sub-normal level of consciousness *or seizures* and *often* accompanied by difficulty with initiating and maintaining respiration and depression of tone and

reflexes. This is the first time that they have presented seizures as an alternative presentation of neonatal encephalopathy as opposed to just an altered level of consciousness. It is also the first time that they have used the word 'often' rather than demanding there be 'difficulty in initiating and maintaining respiration and depression of tone and reflexes.' They go on to note that the neonatal encephalopathy due to acute hypoxic ischemia (related to intra-partum events) will be accompanied by abnormal neonatal signs and be associated with contributing events that were and are in close proximity to labor and delivery. They are proposing that determining causation should be a process of compiling a constellation of potential markers and contributing events combined with the developmental outcome to see if the constellation is consistent with an acute hypoxic ischemic event and is not explained by other etiologies. The approach provides that the more the various elements from the pertinent categories are consistent, the more likely it becomes that an intra-partum event has played a role in the development of the neonatal encephalopathy and ensuing injury.

Neonatal signs considered to be 'consistent with' an acute intra-partum event

They now define as *'consistent'* with an acute intra-partum event an Apgar Score of *less than 5* at five minutes and at ten minutes. They go on to say that if the Apgar Score at five minutes is *greater than or equal to* 7, it is *'unlikely'* that an intra-partum hypoxic ischemic event played a major role. There are several major changes evident here when compared with the 2003 criteria, which required Apgar Scores of 0 to 3 at five minutes. Here, they have not only increased the Apgar Score from 3 to less than 5, but suggest a score of 5 or 6 may also be 'consistent' because they indicate a score of 7 is necessary to fall in the category of 'unlikely' to be related to an intra-partum hypoxic ischemic event playing a role in causing neonatal encephalopathy. This essentially has changed the Apgar Score limit from 3 to 6. Furthermore, apparently in recognition of the subjective nature of Apgar Scores in general, they have not even eliminated Apgar Scores of 7 or greater; rather, they have simply placed them in the category of being 'unlikely.'

There have been major changes in the analysis of fetal umbilical artery acidemia, as it related to intra-partum asphyxia. The 2014 Second Edition states that "a fetal umbilical artery *pH of less than 7.0 or* a base deficit greater than or equal to 12 mmol/L, or both, increases the probability that neonatal encephalopathy has an intra-partum hypoxic component." They go on to state that *lesser degrees of acidemia [only] decrease that likelihood*. Finally, they state that "if the cord arterial gas *pH levels are above 7.20, it is unlikely* that intra-partum hypoxia played a role in causing neonatal encephalopathy." This is a sea change in the analysis of the acid-base status of the fetus, as it relates to intra-partum HIE. The previous essential criteria, which *had to be present*, demanded a blood pH of less than 7.0 *and* a base excess of 12 or greater. This huge 2014 change recognizes that base excess is the most important component of the asphyxial analysis because if it is 12 or greater, there is obvious clear evidence of acute metabolic acidosis – whatever the pH may be. There are a variety of reasons why the pH may or may not show a level consistent with that degree of metabolic acidosis (for example, interference with perfusion from cord prolapse or severe maternal hypotension, etc.). The 2014 Edition also takes the pH level up to just under 7.2 by stating that arterial gas pH above 7.2 is only 'unlikely' to be related to intra-partum hypoxia.

I suspect there have been hundreds of cases in the last decade tried to verdict where the acid base status of the fetus was the subject of dramatically conflicting expert witness testimony. I suspect many of those cases resulted in defense verdicts at least in part based on expert testimony this new criterion demonstrates to have been inaccurate.

Another factor which is noted to be an important part of this global assessment is neural imaging, evidence of acute brain injury seen on MRI or MRS consistent with hypoxic ischemia. The new edition states flatly that *MRI is the best definition* of the nature and extent of cerebral injury in neonatal encephalopathy. It states that *cranial ultrasonography and CTs simply lack sensitivity* for evaluation of timing and the nature and extent of the brain injury. It notes that there are distinct patterns of neural imaging that are generally recognized in hypoxic ischemic cerebral injury (watershed type versus deep gray matter). It points out that early MRIs obtained between 24 hours and 96 hours of life *may* be more sensitive for the delineation of the time of a peri-natal cerebral injury. It also clearly states that the ability to precisely time the occurrence is really a matter of *days rather than a matter of hours or minutes* in a hypoxic ischemic event. This recognizes the advances that have been made in the use of diffusion imaging in MRI and clearly calls into question the efforts of many defense experts to use CT or ultrasonography to try and time a hypoxic ischemic event to indicate it could not have been related to an intra-partum occurrence. It is clear these modalities may still have some limited application in certain specified circumstances, but that a timely MRI will be the most helpful in determining if there is any reason to suspect that a given case of neonatal encephalopathy does or does not qualify as an intra-partum occurrence. This is a dramatic change from the 2003 criterion, which made no distinction between the various modalities of imaging available to the clinician.

Finally, they again point out that the presence of multi-system organ failure is consistent with hypoxic ischemic encephalopathy. This recognizes that a fetus that has undergone a profound

hypoxic ischemic event, sufficient to cause a brain injury, will also probably have had a 'diving reflex' response causing a shunting of the blood to the brain. This tends to deprive other vital organs, such as the kidneys, liver, heart and the gut to be denied adequate perfusion and may result in evidence of dysfunction early in the neonatal clinical presentation. Importantly, the discussion in this area makes it clear that this is highly variable and often does not correlate with the degree of injury to the brain. This, again, is a significant departure from the 2003 criterion which simply stated that there should be an onset of multi-system organ damage within 72 hours of birth.

Type and timing of contributing factors consistent with acute intra-partum events

As before, one contributing factor that is consistent with an acute intra-partum event is a sentinel hypoxic or ischemic event, occurring just before or during labor, such as a ruptured uterus, an abruption of the placenta, cord prolapse, or other well-known catastrophic events.

The new edition contains dramatic changes in the description of fetal heart rate monitor patterns which they deem to be consistent with an acute intra-partum event:

They state that Category 1 or Category 2 fetal heart rate tracings associated with Apgar Scores of 7 *or higher* at five minutes, or *normal* umbilical cord arterial blood gas measurements, is "not consistent" with an acute hypoxic ischemic event. By inference, this leaves a number of situations that would be 'consistent' with acute hypoxia. This is a huge departure from the 2003 requirements that stated only a sudden and sustained fetal bradycardia or persistent late or variable decelerations with *absent variability* qualified. The 2003 requirement was that you had to have (what became in 2008) a "Category 3" presentation or this was not an intra-partum hypoxic ischemic event. Indeed, the 2008 definition also required bradycardia to have *absent* variability.

Now they define Category 1 or Category 2 fetal heart rate tracing as "not consistent" with an acute hypoxic event *only if there are good Apgars or normal umbilical cord arterial blood findings*. This critical change means that the large grey area of Category 2 patterns (which include patterns with minimal or decreased variability, tachycardia, bradycardia with decreased variability, marked variability, absence of acceleration after stimulation, recurrent variable or late decelerations with minimal or decreased variability, prolonged decelerations, decelerations with slow return to baseline, etc.) all can be defined as consistent with an acute hypoxic ischemic event if the child shows evidence of depression at birth, namely Apgar Scores below 7 or abnormal umbilical cord arterial blood gases (as redefined).

They point out that there are additional fetal heart rate patterns that develop after an original Category 1 presentation which may suggest intra-partum timing of a hypoxic ischemic event. These include tachycardia with recurrent decelerations and persistent minimal variability with recurrent decelerations. This again is a significant widening of the inclusion criteria for fetal heart patterns suggesting intra-partum asphyxia.

While these definitions only relate to causation, by inference this edition establishes new categories of "non-reassuring" patterns that by definition can result in asphyxial injury. Birth injury specialists representing brain injured children and their experts have been insisting for the last decade that *minimal or poor variability* with recurrent decelerations is an ominous pattern which should require aggressive intervention. Unfortunately, defense witnesses relied on the 2003 and 2008 definitions that variability had to be "absent" and testified that therefore based on these guidelines, standard of care did not require intervention. This resulted in many defense verdicts which were based on testimonial evidence that has now been revised to conform to reality.

Unfortunately, many practitioners out there in clinical situations also needed to have guidance on how to deal with the patient who presented with ongoing persistent decelerations and decreasing or minimal variability; many of them may have relied on the guidance of ACOG and chosen not to intervene because the variability had not become "absent." Indeed, many of those children might have avoided injury if ACOG had provided more definitive and inclusive guidelines for the doctors and nurses in the field.

The 2014 factors also include timing and type of brain injury patterns based on imaging studies that are consistent with the occurrence of an acute intra-partum event. They note that echogenicity can be found on ultrasonography obtained approximately 48 hours or longer after an ischemic event. However, they emphasize that ultrasound lacks sensitivity for brain injury in the encephalopathic newborn. They also point out that computerized tomography imaging lacks sensitivity for brain injury in the newborn and is not helpful for timing because it will often not reveal abnormalities in the first 24 to 48 hours after an injury.

Most importantly, they note that diffusion abnormalities on MRI are most prominent between 24 hours and 96 hours of life if the injury relates to an acute peri-partum or intra-partum event. However this can only provide evidence as to a range of days from injury, not hours or minutes.

They also point out that there are several well-defined patterns that are relatively typical of hypoxic ischemic cerebral injury. These include: deep gray matter injuries (which are often associated with more acute or profound injuries and usually present with relatively dramatic motor compromise and often spared cognitive function); or watershed cortical injury (which is more typically associated with partial prolonged asphyxial

events and can produce global or more modest encephalopathy often presenting with both cognitive injuries and mild to severe motor involvement). They also note that neuro-imaging cannot distinguish the etiology of hypoxic ischemic events, such as whether it was a placental insufficiency or an interruption of umbilical cord blood flow.

Next they again discuss the importance of considering whether there are other potential causes of the neonatal encephalopathy. However, unlike the 2003 "essential criterion" which required the *exclusion* of other possible causes, such as infection, trauma, metabolic coagulation, and genetic disorders, the 2014 statement simply states that the coexistence of other significant risk factors, such as abnormal fetal growth, maternal infection, hemorrhage, sepsis, and/or chronic placental lesions, simply lessen the likelihood that an acute intra-partum event is the *sole underlying pathogenesis* of a neonatal encephalopathy. In other words, there is a clear recognition that two conditions may co-exist in a fetus. Indeed, abnormal fetal growth, maternal infection, chronic placental lesions, etc., may in fact be the underlying cause of intra-partum events which culminate in neonatal asphyxia. The Second Edition acknowledges that this does not require an either/or analysis.

Finally, while in 2003 it was stated flatly that the neurologic presentation had to be one of spastic or dyskinetic cerebral palsy to qualify for intra-partum causation, the 2014 Second Edition simply states that "other subtypes of cerebral palsy are *less likely* to be associated with acute intra-partum hypoxic ischemic events."

Summary

For attorneys who prepare and try intra-partum hypoxic ischemic encephalopathy cases, this 209-page tome is required reading. The Taskforce has finally recognized that identification of the cerebral palsied child injured by an intra-partum event cannot be autocratically limited based on a narrowly defined set of markers perhaps inspired less by science than the perceived need to defend lawsuits. At long last, ACOG has chosen to provide the clinician reasoned and thoughtful retrospective guidelines grounded on evidence-based medicine. This work makes it clear that neither side of the legal process has all the answers. Sensibly, the authors have noted that a multitude of elements may, or perhaps may not, combine to produce an intra-partum hypoxic ischemic event. The definition of causation in neonatal encephalopathy is clearly a work in progress. This edition, however, has taken a huge step forward in acknowledging that fact as well as in recognizing the huge variability of human response to asphyxial insult. Most importantly, based on these new definitions of the fetus at risk, today perhaps the doctors and nurses out there in the field watching that baby's heart rate during labor will have substantially more guidance in the decision making process.


Bostwick

James Bostwick is a member of the Bar in California and Hawaii and tries cases in many different states. He is a Fellow of The Inner Circle of Advocates, The International Academy of Trial Lawyers (President 2004), ABOTA, a Founding member of The American Board of Professional Liability Attorneys and was Best Lawyers's "Trial Lawyer of the Year" for 2012 and 2013. See profile in this issue.

Endnotes

[1] Sophie Berglund, et al., Severe Asphyxia due to Delivery Related Malpractice in Sweden, 1990-2005, 115 Brit. J. Obstetrics and Gynecology, 316 (2008).
[2] Hakan Noren, et al., Fetal Electrocardiography in Labor and Neonatal Outcomes: Data from the Swedish Randomized Control File on Inter-Partum Fetal Monitoring. 188AM.J.Obstretics and Gynecology. 183, 190 (2003).