

THE LITIGATION LITERATURE

By: Joel D. Cunningham

As the new chairperson of the WSTLA malpractice section, I have been asked to write a column about malpractice. I have decided to discuss a growing problem in malpractice cases: the corruption of the medical literature with articles designed to protect doctors in court. I call these publications the *litigation literature*. The majority of my law practice for the last 20 years has been devoted to obstetrical malpractice. The problem of *litigation literature* is particularly pronounced in this field of medicine.

Corruption of the medical literature is not new. It is now well-known that for years drug companies have influenced researchers to publish biased studies in the medical literature¹. Less well known is that American College of Obstetrics and Gynecology (ACOG) and some of its members have been doing the same thing. For the last several decades, *litigation literature* has been published in the obstetrical and pediatric literature disguised as scientific literature, but designed to defend obstetricians in court². For years, the defense bar complained about Plaintiffs using “junk science” to prove their cases, resulting in *Daubert* and its progeny. Following *Daubert*, it became important that medical literature be “peer reviewed” if it was going to be used in court. The purpose to the *litigation literature* is to publish “junk science” in the guise of peer-reviewed medical literature so that the articles can be used in court by defense counsel .

In the area of birth-injury cases, the purpose of most of the *litigation literature* is to try to convince judges and juries of two things: (1) Depriving babies of oxygen during the birth process almost never causes brain injury and (2) nothing an OB does or does not do can cause or prevent a baby from suffering a brain injury³. These articles are dangerous not only because they can and do mislead judges and juries, but because they can and do mislead health care providers⁴. This infection of the legitimate peer-reviewed medical literature with *litigation literature* has been spearheaded by ACOG through ACOG Technical Bulletins⁵ and, now, a monograph⁶. ACOG, in turn, relies heavily on its ally at the National

Institute of Health, epidemiologist Karin Nelson, M.D.⁷ (As Mark Twain said, "There are lies, damn lies, and then there are statistics.")

ACOG's latest - and perhaps its most powerful - *litigation literature* is the 2003 monograph, *Neonatal Encephalopathy in Cerebral Palsy: Defining the Pathogenesis and Pathophysiology*. The ACOG monograph sets forth criteria that must be met before an acute hypoxic intrapartum event (lack of oxygen during the birth process) can be said to cause cerebral palsy in a baby. Here is a summary of the "essential criteria":

1. Cord blood Ph of less than 7.0 and base excess of 12 mmol/L or more
2. Early onset of severe or moderate neonatal encephalopathy in babies 34 or more weeks gestation
3. Cerebral palsy of a quadriplegic or dyskinetic type.
4. Exclusion of other causes

If each of these criteria have not been met, says ACOG, then the baby was not damaged from a lack of oxygen during the birth process⁸. End of story. Look like a good way to defend OB cases in court? You bet it is. And it was intended to do just that.

These new criteria so narrowly define birth asphyxia (in the newspeak of the monograph, "acute intrapartum hypoxic events") that as defined it virtually never occurs. Criteria #4 goes so far as to make birth asphyxia a diagnosis of exclusion. So using these strict criteria, it will almost never be diagnosed. Then when epidemiologic studies are done using this narrow definition, birth asphyxia will be found to be a minuscule cause of CP. It is a circle jerk. Using similar artificially narrow definitions of birth asphyxia, medical epidemiologist have previously been able to conclude that only 8-15% of cerebral palsy is caused by hypoxic events during labor and delivery⁹. Let me give an example. If you say that arson only occurs when an elephant is found in the vicinity of the fire, then if an epidemiologic study is done using that definition, it will conclude that arson rarely if ever occurs.

I give ACOG credit. It is not a stupid organization. The OB's were able to recruit a number of other organizations, in particular, the American Academy of Pediatrics (AAP), to lend their names to this monograph giving it additional weight in the court room. Rarely have such prestigious bodies published such biased literature in the name of science. And rarely has it been done so deceptively well.

The monograph claims that its purpose is to accurately define and diagnose acute hypoxic intrapartum events. And to help clinicians in practice. And if you believe that, I have a bridge I would like to sell you. I suggest that the true purpose of this monograph was not simply to help doctors accurately "define" birth asphyxia but rather to *rule it out* for *professional liability* purposes. Consider how ACOG itself advertises the monograph to its members:

This report contains a set of criteria that may be used to define (**or rule out**) an acute intrapartum hypoxic event sufficient to cause or suggest cerebral palsy. These criteria and the body of th evidence contained in the report may prove to be useful reference tools for addressing clinical care **and professional liability**.

2003 ACOG - Mid-year Savings Catalog (bold print mine)

I do not have a problem with ACOG publishing a monograph to help its doctor-members defend themselves in court. But I do have a problem with ACOG pretending that the monograph is something other than that. Articles and ACOG Technical Bulletins published not for their scientific truth, but to assist obstetricians in defending themselves in court, should be labeled "*litigation literature*" and not referred to as "*medical literature*." To pretend such publications are peer-reviewed scientific articles is a deception¹⁰. To create them to manipulate courts and juries is nothing less than a fraud on the civil justice system.

Let me give some evidence here that may give some credence to what I am saying. The ACOG task force chairman for this monograph is a perinatologist named Gary Hankins, M.D. He is a well-known litigation expert, testifying most frequently for the defense, particularly in OB cases.

He is as litigation-savvy as any member of ACOG. This is the person ACOG chose to lead its search for the scientific “truth.” In addition, the monograph states it is modeled after Consensus Statement of the International Task Force on Cerebral Palsy published in the British Medical Journal¹¹ in 1999. This “medical” article not only published similar “necessary criteria” required to prove that a lack of oxygen during the birth process caused brain injury, it actually went on to say what types of experts could testify to these criteria in court! And it made clear it was intended for use in the courts¹²! For this, the authors were soundly criticized by their peers¹³. But at least those authors were straightforward. When these criteria were copied into the ACOG monograph, ACOG was more deceptive. All references to litigation, litigation experts, or use of the monograph in court were omitted. It was disguised as a purely scientific publication.

Litigation literature is not a new phenomenon in the obstetrical literature. *Litigation literature* has been created by ACOG and its members for use by defense counsel in court to deny justice to children suffering from cerebral palsy for the past several decades. A perfect example of this is ACOG Technical Bulletin #163 (1992). Bulletin #163 averred that fetal or neonatal brain injury could not be said to be caused by birth events like lack of oxygen unless all four of the following criteria were met:

1. Ph of less than 7.0
2. Apgars of 0-3 for five or more minutes
3. Neonatal difficulties like seizures and coma
4. Multi-organ dysfunction following birth

(Please carefully note that criteria #2 and #4 are no longer “essential” under ACOG’s latest criteria.)

Statistics from the medical literature were manipulated to create this false set of “essential criteria.”¹⁴ Obstetricians and hospitals quickly discovered that they could defeat causation in court under Bulletin #163 simply by having the nurses assign an Apgar of 4 (on a scale of 10) at five minutes of age, thereby denying the plaintiff one of the four criteria. Higher Apgar scores became more “liberally” assigned to very sick babies.

Unfortunately for ACOG, some scientists¹⁵ began attacking the criteria of Technical Bulletin #163. For example, Phelan, et al.¹⁶, looked at 47 children with cerebral palsy that were clearly asphyxiated at birth. These infants had suffered clear hypoxic events during labor from such complications as a prolapsed cord, or uterine rupture, which resulted in brain injury. Only 21% of these clearly asphyxiated, brain-injured children satisfied all four ACOG criteria. *That means that 79% of these babies could have been denied justice in court if Bulletin #163 was believed by the jury.* Despite such articles, as long as Bulletin #163 existed, defense attorneys were able to continue to use it to successfully defeat legitimate birth injury cases in court.

Articles like Phelan's did start causing ACOG trouble, however. It was becoming increasingly obvious that Bulletin #163 was a scientific fraud. Trial lawyers were beginning to crucify it in court as more and more experts agreed it was in error. It got so bad that even ubiquitous defense stalwarts like Dr. Larry Naeye had to admit it was just plain wrong. Imminent members of the organization urged ACOG to change the criteria to make them science-based. For that and other reasons the ACOG Task Force on Neonatal Encephalopathy and Cerebral Palsy was created. As the task force evolved, however, it unfortunately became an effort to make a better Bulletin #163 for use in court. You will not see the scientific errors in Bulletin #163 discussed in the 2003 monograph that replaces it. Instead, it is written as if Bulletin #163 never existed. To admit the mistakes in Bulletin #163 would cast a pall over the new "essential criteria."

The new "essential criteria" are already under attack as unduly narrow. "Essential criteria" #3 is certainly open to question. There is no compelling scientific evidence that the only type of cerebral palsy caused by hypoxia at birth is spastic quadriplegia or dyskinetic¹⁷. This "scientific truth" is based on a retrospective study of data collected in the 1960's and statistically "interpreted" by Karin Nelson¹⁸. The ACOG monograph cites her study and nothing else to support this sweeping statement.

Dr. Hagberg, one of the world's leading authorities on the causes of cerebral Palsy, has published an article¹⁹ proving that that Nelson's conclusion, and that of the task force, is simply wrong. Dr. Hagberg

looked at 32 children with proven birth asphyxia. He documented both on a clinical basis and via neuroimaging that these 32 children suffered brain injury during labor and delivery (the “intrapartum period”). Then he discussed the whether the Task Force’s “essential criteria” #3 holds water:

“We also feel that the requirement that only dyskinetic and quadriplegic types of CP should be considered to have been caused by intrapartum hypoxic events is too conservative. Dyskinetic CP and spastic tetraplegic/severe diplegic subtypes occurred in 59% of the children, *but mild diplegic and hemiplegic subtypes comprised the remaining 41%.*” (emphasis mine)

Furthermore, the monograph itself is internally inconsistent regarding what type of “cerebral palsy” can be caused by hypoxia. To reiterate, essential criteria #3 requires that the cerebral palsy be of the spastic quadriplegia or dyskinetic type. “Spastic quadriplegia” means tightness in all four extremities. “Dyskinetic” refers to uncontrolled movement. In Chapter 3, the authors of the monograph describe intrapartum events that can cause hypoxic injury (always with the caveat that injury occurs only once in a blue moon, of course.) One clear case of an acute hypoxic intrapartum event is where the mother suffers a cardiac arrest during labor. This, of course, cuts off the oxygen supply to the baby as well. In Chapter 3, page 34, such a clear case of hypoxia at birth is described as follows:

Survival of an infant delivered 22 minutes after medically documented maternal cardiac arrest has been reported. At age 18 months, the child was clinically normal *except for persistent mild hypotonia* and her Denver developmental screen test scores were normal. (italics mine)

This child, who was given as an example of a baby who suffered an acute hypoxic event during labor, did not develop spastic or dyskinetic cerebral palsy, and instead developed only “persistent mild hypotonia” (mild muscle weakness, the opposite of spasticity). Yet, if ACOG’s “essential criteria” are applied, this child’s brain injury would be classified as unrelated to her mother’s 22 minute cardiac arrest! Nonsense begets nonsense.

But the biggest “scientific” bone I have to pick with the ACOG monograph is that it relegates neuroimaging studies to “supportive evidence.” A picture does speak a thousand words, and serial MRI’s and CT’s “brain pictures” not only can **PROVE** the diagnosis of hypoxic brain injury, they can often time the injury. As Dr. Jim Barkovich (ironically one of the task force “consultants”) has written²⁰:

MR appears to be a powerful tool in assessing brain damage from perinatal asphyxia.

This was written in 1990. The ability of MRI’s to prove or disprove perinatal asphyxia as the cause of brain injury is much greater today²¹.

I close with the words of Forrest Bennett, M.D., a local physician who has spent a good part of his lifetime sorting out the causes of brain injury in disabled children. He wrote these words in 1989 as the forward to the text, Fetal and Neonatal Brain Injury (B.C. Decker, Inc. 1989). They were prophetic of the wave of *litigation literature* that would swamp the obstetrical medical literature during the following decade:

The current medicolegal climate surrounding “birth injuries” has produced a somewhat concerning type of scientific response.

...

This contemporary scientific response to a serious medicolegal dilemma...often appears to be forcing prenatal (or idiopathic) explanations from inadequate data and minimizing the role of intrapartum asphyxia except in the most extreme cases. This approach strikes me as unfounded and hazardous as the narrow perinatal viewpoint and threatens to underestimate in physicians’ minds the diverse neurodevelopmental consequences of asphyxia²².

Shame on ACOG for its latest deception. It has forgotten that the ends do not justify the means. ACOG and its members should stop publishing *litigation literature*.

Epilogue

The ACOG monograph speculates based on interpretation of statistical data that in most cases even term babies who suffer severe perinatal hypoxic events are not damaged by such an event. For example, it speculates that severe heart rate decelerations during labor are usually a manifestation of an earlier insult to the baby and are not the cause of brain injury. To test this hypothesis in “real life,” a relatively large study would have to be done using neuroimaging and autopsy findings. The monograph cites no such study. If the monograph is correct, such a study would show evidence of antecedent brain damage(brain damage present before labor delivery) on 85-90% of the neuroimaging studies and autopsies.

What if I were to tell you that such a study has recently been done? And that the results proved what Lincoln said is true: you can fool some of the people all the time, and all the people some of the time, you can't fool all the people all the time? I will discuss this important peer -reviewed “real” scientific study in an upcoming issue.

1. McCrary SV, et al, A national survey of policies on disclosure of conflicts of interest in biomedical research. *N Engl J Med.* 2000 Nov 30;343(22):1621-6; Bekelman, JE,et al, Scope and impact of financial conflicts of interest in biomedical research: a systematic review. *JAMA.* 2003 Jan 22-29;289(4):454-65.

2. Stevenson, et al., *Fetal and Neonatal Brain Injury* (B.C. Decker, Inc. 1989), forward by Forrest Bennett, M.D., commenting on how far the pendulum has swung from blaming all cerebral palsy on birth events to blaming everything under the sun except birth events: “Unfortunately, partially motivated and fueled by the 1980's medical malpractice crisis involving so-called ‘bad baby’ cases, an increasing volume of reports utilizing the National Institutes of Health Collaborative Perinatal Project data from 25 years ago, consensus conference publications, and editorials seems, at times, to be attempting to move the etiologic pendulum too far in the opposite direction.” Bennett cites Karin Nelson’s use of the Collaborative Project data as an effort to diminish the importance of asphyxia as a cause of cerebral palsy.

3. See, E.G., Nelson KB, et al. *How much of encephalopathy is due to birth asphyxia? *AJDC* 1991;145:1325-31.*; Nelson, et al, Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. *Obstet Gyn* 1998; 179:507-13(She concludes from her statistical analysis of old data that such devastating events as uterine rupture and abruption don’t cause cerebral palsy!! These conclusions have been dis-proven. See, E.G., Changing Panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year Period 1991-1994, *B Hagberg, et al. *Acta Paediatr* 90:271-277(2001).* Furthermore, a confidential study was done in England that demonstrated that a whopping 64% of birth encephalopathies and 75% of perinatal deaths were associated with sub-optimal care. E.Draper, et al, *A confidential inquiry into cases of neonatal encephalopathy, *Arch Dis Child Fetal Neonatal* ED2002;87:F176-F180.*

4. ACOG Technical Bulletin #207, *Fetal Heart Rate Patterns* (1995) can be read as saying that electronic monitoring is of no benefit to the fetus and listening intermittently with a hand held Doppler is just as effective. I had a case where several midwives and a perinatologist apparently didn’t understand this was just *litigation literature* designed to defend OB’s in court. So they didn’t use electronic monitoring while attempting the labor and vaginal delivery of a breech baby. The result was a disaster for the baby – and the health care provider’s insurers who paid \$13,500,000 in settlement. Worse yet, diminishing the role of birth asphyxia diverts research away from ways to treat asphyxiated babies to prevent or diminish brain injury.

5. ACOG Technical Bulletins, now called Practice Bulletins, have been published for a number of decades, and are provided free of charge to ACOG members. They come out periodically, and are updated periodically. They are now published in yearly compendiums for convenience. They are vetted by lawyers to make them difficult for plaintiff’s attorneys to use in court. See, E.G., the language at the beginning of the 2001 Compendium: “ The information in these documents should not be viewed as establishing or dictating standards or rigid rules.... . [and so on]”

6. Neonatal Encephalopathy and Cerebral Palsy, *Defining the Pathogenesis and Pathophysiology* (ACOG, Jan. 2003). Hereafter cited “ ACOG Monograph.”

7. Karin Nelson is the authority cited for essential criteria #3, stating that only cerebral palsy of the spastic quadriplegia or dyskinetic type is caused by intrapartum asphyxia. Her conclusion was based on what Dr. Bennett and others believe is out-dated data. See note 1. *ACOG Monograph, Executive Summary, xviii*. In a quick review, I counted 42 times her work was cited in the ACOG Monograph.

8. Please note that MRI studies of the brain which can actually document with pictures of the brain the cause and timing of the brain injury were relegated to only being “supportive” of an intrapartum event. MRI and CT’s in the neonatal period can provide conclusive proof of intrapartum hypoxic injury. If the above criteria, aren’t met, the MRI pictures of the brain are simply overruled. This is the huge hole in ACOG’s attempt to define away intrapartum hypoxic events as a cause of CP. A picture does indeed speak a 1000 words.

9. See note 2, *infra*. Blair, et al. Intrapartum asphyxia: a rare cause of cerebral palsy. *J Pediatr* 1988;112:515-19.

10. L.Rosenbloom, et al. There are problems with the consensus statement. *BMJ* 2000;320:1076, “Furthermore, although this document undoubtedly represents the consensus after extensive discussion among those selected to participate, no way does it contain a meta analysis of the kind usually required before an authoritative guideline can be produced.”

11. McClennon A. A template for defining a causal relationship between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ* 199;319:1054-9. At page 1059, there is a subsection entitled: “Who should be and expert witness in cases of cerebral palsy?” Then “criteria” for such an expert are set forth in Box 5.

12. *id*, McClennon concludes: “This international consensus statement has been prepared to help the public, health care workers , those researching in this area, and **courts of law** to better understand the probability of whether, in any particular case, there is convincing evidence to suggest the pathology causing cerebral palsy occur red during labor and delivery and whether it was reasonably preventable.” (Bold print mine). Dr. Nadia Badawi was a member of the International Consensus Statement. Consider Dr. Badawi’s comments in an editorial in the *Medical Journal of Australia*;172:199-200(2000): “ The consensus statement may not prevent litigation, but it may provide courts with more definitive guidelines for interpreting the arguments put forward by both plaintiffs and defendants. It will no longer be feasible for a plaintiff to argue that an abnormal cardiocograph and depressed appgar scores without evidence of newborn encephalopathy, constitute evidence of damaging asphyxia in a child who later develops Cerebral Palsy.” So was the Consensus Statement developed for science or litigation?

13. See note 9, *supra*.

14. Litigating Catastrophically Injured Infant Cases, ATLA Birth Trauma Group, Feb., 2000

15. Korst, et al., Can Persistent Brain injury Resulting from Intrapartum Asphyxia be predicted by Current Criteria? *2 Prenatal Neonatal Med.* 286-93(1997).

16. Korst, Phelan JP, et al, Acute Fetal Asphyxia and Permanent Brain Injury: A Retrospective Analysis of Current Indicators, J Matern Fetal Med. 1999 May-Jun;8(3):101-6.

17. This criteria is based on the study by Karin Nelson, M.D.: Potentially Asphyxiating Conditions and Spastic Cerebral Palsy in Infants of Normal Birth Weight, Am J Obstet Gynecol, 1998;179:507-13. Please keep in mind that Dr. Nelson's mission in all of her literature is to prove that lack of oxygen really doesn't cause cerebral palsy at all – it is always something else. See Stevenson, et al., Fetal and Neonatal Brain Injury, pg. 831-32 (Cambridge Press, 3rd Ed 2003).

18. P. Ellison, Neurological Implications of Perinatal Complications, Neuropsychology of Perinatal Complications 59 (Gray and Deans, eds, 1991); C.M.T. Robinson, Follow-up of Term Infants with Perinatal Asphyxia, in Stevenson, et al, Fetal and Neonatal Brain Injury, 3rd ED., Chpt. 41(Cambridge Univ. Press, 2003)

19. Changing Panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year Period 1991-1994, B Hagberg, et al. Acta Paediatr 90:271-277(2001.)

20. Barkovich J, Brain Damage from Perinatal Asphyxia: Correlation of MR findings with Gestational Age, ANJR 11, Nov./Dec. 1990.

21. Roland E, et al, Perinatal Hypoxic-Ischemic Thalamic Injury: Clinical Features and Neuroimaging, Ann Neurol 1998;44:161-166; Rutherford M, et al, Hypoxic-ischemic encephalopathy: early and late magnetic resonance imaging findings and their evolution, Neuropediatrics 1995;26:183-91.

22. See note 1, supra.