CRITERIA FOUND IN NEONATAL ENCEPHALOPATHY AND CEREBRAL PALSY: DEFINING THE PATHOGENESIS AND PATHOPHYSIOLOGY (NEACP) IS UNRELIABLE

The Criteria to Define an Acute Intrapartum Event as Sufficient to Cause Cerebral Palsy, found in a publication published by the American College of Obstetrician and Gynecologists (ACOG) entitled *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology* ("NEACP")[1] is not reliable. It was written by obstetricians. Obstetricians do not diagnose or treat hypoxic ischemic encephalopathy. The purported criteria are not widely accepted in the pediatric neurology or neonatology communities, i.e. by those who do diagnose and treat such conditions.

ACOG Monograph/The Green Book/ NEACP

Many defense experts rely upon the NEACP, arguing that an injured child did not meet the ACOG criteria to establish a hypoxic-ischemic event during labor sufficient to cause neonatal brain injury and cerebral palsy. Defense experts imply that the ACOG criteria are definitive and necessarily support their position with respect to the mechanism and timing of injury. They are incorrect.

First, the diagnosis and treatment of hypoxic ischemic encephalopathy and cerebral palsy are within the fields of child neurology and neonatology, not obstetrics. The Obstetrical Monograph, NEACP, is not widely accepted by the neurological or neonatology communities that actually make the diagnosis. Indeed, the physicians who actually make the diagnosis and treat these children use different criteria. Second, the

[1] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology, (2003) Chapter 8. Please see Appendix. The appendix contains all references to medical literature and depositions.

ACOG criteria was created for litigation and is not reliable. Third, the ACOG criteria is scientifically unreliable. Fourth, the use of the criteria has been rejected by other courts.

A. The NEACP criteria are not widely accepted.

Obstetricians neither diagnoses nor treat either hypoxic ischemic encephalopathy or cerebral palsy in their own practice.[2] Indeed, they will often decline to give true *causation* opinions, admitting it was essentially out of the area of obstetrics[3]. Nevertheless they often claim reliance on the NEACP for portions of their standard-of-care opinions, deferring to the neurologists and neonatologists for causation.[4] Of course, NEACP does not purport to address standard of care. Still, defense obstetricians think it's relevant in that it shows that:

> "there are a lot of doctors who do very good things, treat patients appropriately, and wind up with bad outcomes. It helps physicians understand that it may not be their fault, so to speak, or that they did something wrong.").[5]

Defendants often claim that an injured child does not meet criteria for a classic hypoxic injury. This is disingenuous. Joseph J. Volpe, Branson Crothers of Neurology, Harvard Medical School and Neurologist in Chief at Boston's Children's Hospital, is one of the preeminent neurologists in the country, and has published extensively in the field. In his widely-used textbook[6] Neurology of the Newborn (4th Edition, 2001), Dr. Volpe addressed the criteria for intrapartum asphyxia, as follows:

> The Neurological syndrome that accompanies serious intrauterine asphyxia is the prototype for neonatal hypoxic-ischemic encephalopathy. *The occurrence of a neonatal neurological syndrome, indeed, is a sina*

[2] For this reason alone, his opinions on the NEACP ought to be stricken.
[3] Deposition of Boehm p. 24-25.
[4] Deposition of Boehm p. 36-37. Not only are NEACP criteria insufficient bases for expert opinion on causation, they aren't intended to establish practice guidelines or standards of care.
[5] Deposition of Boehm p. 49-50.
[6] Deposition of Rohrbaugh.

> *qua non for attributing subsequent brain injury to intrapartum insult(s).* Indeed, I consider that three features are important in considering that intrapartum insult is the likely cause of neonatal brain injury: (1) evidence for fetal distress (e.g., fetal heart rate abnormalities, meconium-stained amniotic fluid), (2) depression at birth, and (3) an overt neonatal neurological syndrome in the first hours and days of life.[7]

As mentioned, the NEACP monograph was published in 2003. Five years later, Dr. Volpe readdressed intrapartum asphyxia in the 5th Edition of his text. He stated:

> The neurological syndrome that accompanies serious intrauterine asphyxia is the prototype for neonatal hypoxic-ischemic encephalopathy. *The occurrence of a neonatal neurological syndrome, indeed, is a sine qua non for attributing subsequent brain injury to intrapartum insult.* Indeed, I consider three features important in considering that intrapartum insult is the likely cause of neonatal brain injury: (2) evidence of fetal distress (e.g., fetal heart rate abnormalities, meconium-stained amniotic fluid), (2) depression at birth, and (3) an overt neonatal neurological syndrome in the first hours and days of life.[8]

It is important that the neurological community has not adopted the ACOG criteria for intervention. For example, a recent experimental therapy for term infants suffering an acute brain injury is whole-body hypothermia.[9] The idea is that if the brain temperature is reduced 2°C to 5°C, it would provide neuroprotection to baby's suffering from ischemic insult to the brain. This was recently studied in fifteen participating centers of the National Institute of Child Health and Human Development Neonatal Research Network. The criteria for inclusion in the study were different from ACOG's "essential criteria".[10]

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[7] Volpe, Joseph J, *Neurology of the Newborn*, 4th Edition, p. 332.
[8] Vole, Joseph J. *Neurology of the Newborn*, 5th Edition, p. 401.
[9] Shankaran, S., et al., *Whole-Body Hypothermia for Neonates with Hypoxic-Ischemic Encephalopathy*, N.Eng. J. Med. 353:1575 (2005).
[10] *Id*. It is anticipated that Defendants' expert, Dr. Hall, will admit this criteria was used at his own institution when he is deposed.

Further, the FDA has in fact defined intrapartum asphyxia differently from ACOG's NEACP, and in doing so, specifically pointed out it was rejecting the ACOG criteria:

> FDA approved protocol for the definition of moderate to severe HIE
> . . .
> Recently the FDA has approved the use of the Olympic Cool-Cap to treat term infants with moderate to severe HIE, within the first six hours of life.
>
> In doing so, the FDA gave approval to the proposed definition of moderate to severe HIE, which is considerably different from that found in The American College of Obstetricians and Gynecologists publication Neonatal Encephalopathy and Cerebral Palsy (2003). This definition of moderate to severe HIE is similar to the one used in a nation-wide study of body cooling organized by the HIE, the results of which were published in the New England Journal of Medicine, 2005, 353; 1574-84 (October 13, 2005 issue).[11]
> . . .

B. The ACOG Criteria Are Unreliable and Are Designed for Litigation, Not Medicine

The ACOG criteria are a political tort reform effort by ACOG. The reality of the 2003 ACOG monograph is that it is a political agenda masquerading as medical science. Dr. Karin Nelson, a pediatric neurologist, credited with work to try to prove that physicians only rarely if ever cause cerebral palsy,[12] conceded on the release of the NEACP criteria that she "is not a fan of the report's nine-point checklist – '*It's intended for litigation,*[13]' she said - she believes juries don't understand the limits of current technologies."[14]

[11]Redefinition of Hypoxic Ischemic Encephalopathy (HIE) by the U.S. Food and Drug Administration (DFA) and by the National Institutes of Health (NIH).
[12] Deposition of Boehm p. 35-36
[13] i.e., not for use in treatment or as a diagnostic tool
[14] McCullough, *A Dispute On Doctors' Cerebral Palsy Role*, Features Health & Science Section, 2/10/03 PHILA. INQUIRER (Philadelphia Newspapers Inc. 2003)

1. The ACOG criteria are a political tort reform effort by ACOG.

The Court should not be misled by the Defendants' use of the word "essential" because the reality of the 2003 ACOG monograph is that it is a political agenda masquerading as medical science. If there is any doubt that ACOG is not a lobbyist for so-called "Medical Liability Reform", one has only to visit its website and view numerous ACOG News Releases promoting the passage of tort reform litigation. Plaintiffs are attaching a copy of ACOG's February 1, 2006, press release, which is directly on point.[15]

ACOG admits that one of its main purposes is to defend against lawsuits. In July 2004, ACOG's president, Dr. Franklin Miller, admitted under oath that the 2003 monograph was developed in order to defend against lawsuits.

> Q. But your – your point was – the reason you were developing it, or stated reason, was to defendant lawsuits?
>
> A. Well, I thought that – that we were in an area where the – where were being sued for reasons that we – over which we had no control. And – in a high percentage of the time.
>
> And so, yes, that's – that was one of the area – one of the things I wanted to address. And I – and I – and I will stand by that.[16]

Dr. Boehm, another defense obstetrician, was questioned in this case about Dr. Miller's statement. Dr. Boehm stated:

> 49
> 8 Q. Yes. Do you know who Frank Miller is?
> 9 A. I do.
> 10 Q. Was he president of ACOG?
> 11 A. He was.
> 12 Q. Was he president of ACOG in 2000?
> 13 A. That sounds about right. I don't remember

[15] ACOG News Release, http://www.acog.com/from_home/publications/press_release/nr02-01-06.cfm
[16] *Deposition of Dr. Franklin Carl Miller,* In the United States District Court of the Western District of Tennessee at Memphis, *Miller v. Dacus*, Case No. 03-2701 M1 V, July 22, 2004 at p. 46, lls. 14-25,.

the exact year, but he was the one that initiated the -- the Neonatal Encephalopathy Group.
Q. Which ultimately generated the neonatal encephalopathy and cerebral palsy publication that we have talked about a moment ago?
A. Correct.
Q. And in fact, he was the one that picked who was going to be on the group, didn't he, sir?
A. I don't know if he personally picked everybody, but he was certainly the one who was the stimulus to put the group together.[17]

One of Dr. Miller's stated goals for ACOG during this decade was to "reduce medico legal risks for obstetrician-gynecologists" by developing evidence that could be "used to defend against unwarranted claims and challenge false testimony by expert witnesses and others".[18] Or, as Dr. Boehm stated:

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Are you aware that he's testified that the purpose of that undertaking was to help defend lawsuits?
MS. SMITH: Object to form.
THE WITNESS: No.
BY MR. FREEMAN:
Q. If he testified that the purpose was to undertake to defend lawsuits, would you take him at his word?
MS. SMITH: Object to form.
THE WITNESS: Sure. I don't take that as a pejorative statement. The -- 80 percent of obstetricians are sued at least once, and 25 percent, at least the latest figures I had, are sued four times or more.
So from my review of cases over the last 20 -- almost 29 years, I think there are many cases where our literature was in bad need of -- of terminology that was clear and consistent with facts that were evidence-based so that we could defend ourselves against experts who clearly spoke outside the system of using evidence-based medicine and

[17] Deposition of Boehm, p.49
[18] Miller, F., "Ten Goals for ACOG for the First Decade of the Next Millennium," OB&GYN, 94(1):1 (2000) at pg. 4.

25 giving opinions based on their own personal

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1 experience.
2 So I mean, I think that material has aided
3 plaintiff attorneys frequently in being able to
4 settle lawsuits or to win trial cases. It works both
5 ways. This is -- this is the best evidence we have,
6 and when we violate the tenets that result in -- in
7 the outcomes that -- that the group wrote about, then
8 we are at fault, and we belong to the -- to
9 a -- we -- we are required, then, to -- to make
10 restitution.
11 So I -- I think that that document both
12 protects physicians and nurses and holds them
13 accountable so that when there is departure from the
14 standard where these tenets are violated or there's
15 a -- or there's evidence to support that, then I
16 believe the physician and nurse should be held
17 liable.
18 So I think it is not a pejorative or negative
19 thing to say, "I did this to defend lawsuits." It
20 was an intellectual exercise to help lawyers and
21 doctors understand the complexities of these issues
22 and that they're not just associated with one expert
23 giving an opinion on something that has nothing to do
24 with evidence-based medicine.
25 So the answer to your question is, if he said

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1 it, I agree with it, and I don't have a problem that
2 it sounds something negative to patients or -- or
3 plaintiff attorneys.[19]

2. History of NEACP

Neither the "essential criteria" nor the purpose for them are new. ACOG's monograph derives from two earlier sources: ACOG Technical Bulletin 163 (1992) and a British medical journal article published in 1999 and referred to as the The Template.[20]

[19] Deposition of Boehm, pp. 51 – 52.
[20] MacLennan, A., "A Template for Defining Causal Relation Between Acute Intrapartum Events and Cerebral Palsy: International Consensus Statements, " Brit.Med.J. 319:1054 (1999).

According to the author of The Template, Dr. Alistair MacLennan, the International Cerebral Task Force was convened to bring together the modern literature on the cause of cerebral palsy to benefit research into cause and prevention and **to help those who offer expert testimony in court**.[21] Dr. MacLennan even provided "recommendations for expert witnesses giving evidence on cerebral palsy causation." The recommendations included criteria for the qualifications of expert witnesses on cerebral palsy causation and suggestions concerning how the expert witness should conduct himself or herself.[22] This motive was carried forward from Technical Bulletin #163. It is underscored by the following comments by Dr. Richard Perkins to Dr. Goodlin's article criticizing previous "essential criteria" in ACOG Technical Bulletin #163.

> The request was to reach a generalization that vastly exceeded the legal concept of "reasonable medical probability" (that is, somewhat >50% of the time) and to approach the medically tolerable concept of "reasonable medical probability" (that is, with only rare exception). It was an honest effort to stop the bleeding. It cured most ills fairly and staunched an evil tide of wild speculation and unsound litigation while awaiting refinement in perspective.[23]

Notably, Dr. Boehm is of a similar mind. In his view, the NEACP criteria raised the statistical confidence level of intrapartum asphyxia to 95%:

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>
> If you meet these criteria set forth in Chapter 8, does that give you a 95 percent confidence level that you've had an intrapartum asphyxia?
>
> MS. SMITH: Object to form.
>
> THE WITNESS: Well, it does for me. I don't know whether it does for all scientists involved. What I can tell -- well, it does for me. I don't know what the literature says, what

[21] *Id*.
[22] *Id.* at 1059.
[23] Perkins, R., "Letters," Am.J.Obstet.Gynecol. 174:2 (1996) at pp. 798-799.

25 confidence level, but it certainly does for me.

Dr. Goodlin's response back to Dr. Perkins is instructive:[24]

> If the framers of the ACOG bulletin had spoken in terms of Perkins' "reasonable medical probability" instead of their "must be" criteria, the impact of their concepts on obstetric through would have been different. **The "must be" criteria go far beyond the legal issues, for they now appear as definitions of fetal asphyxia and neonatal hypoxic-ischemic encephalopathy. These criteria intrinsically eliminate intrapartum insults as the cause in most cases of cerebral palsy and lead to the view that obstetricians can do nothing to prevent cerebral palsy.**
>
> **In their apparent desire to stem Perkins' figurative "bleeding with ligature," the framers of ACOG criteria ignored scientific principles and created hard dogmatic rules from soft data (as described in my paper**)."[25]

Clearly ACOG's purpose in drafting ACOG 163 was to create a virtually impossible causation hurdle for malpractice cases. It is not a great leap of faith to conclude that ACOG has the same motivation concerning the current "essential criteria". The ACOG criteria do not represent the knowledge and beliefs of the relevant medical community for Daubert purposes. Further, there are no well-done laboratory or clinical studies that supported ACOG's four stringent criteria, much less that all four must be present.[26]

The 2003 ACOG monograph, The Template, and the ACOG Technical Bulletin 163, are powerful examples of the inherent conflict of interest these publications represent. All embody what has been labeled as "inherently unacceptable behavior that

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[24] Deposition of Boehm, p. 64.
[25] *Id.* (emphasis added)
[26] Goodlin, R., "Do concepts and prevention of cerebral palsy require revision?," Am.J. Obstetrics and Gynecology 172:1830 (1995. *See also*, Lisa M. Korst, et al., *Acute Fetal Asphyxia and Permanent Brain Injury: A Retrospective Analysis of Current Indicators*, J. Maternal-Fetal Med. 8:101 (1999) *See* Phelan, J., et al., "Intrapartum Fetal Asphyxial Brain Injury with Absent Multi-Organ System Dysfunction." J. Maternal-Fetal Med. 7:1 (1998) .

directly threatens the integrity of research."[27] In a "National Survey of Policies on Disclosure of Conflicts of Interest in Biomedical Research" published in the *New England Journal of Medicine*, the authors conclude that these conflicts of interest may appropriately influence the design, conduct, or reporting of research, thus threatening its scientific value.[28]

The implications for these litigation-driven publications have even more dire consequences. According to the ACOG Task Force, one goal of "overriding importance" is to "develop recommendations for evaluation of the newborn with encephalopathy *to assist the clinician* in defining both the cause and the timing of the encephalopathy."[29] ACOG, in this publication, is mixing its political agenda with the medical/scientific advice it offers its members concerning the diagnosis and treatment of the fetus and the newborn. This is a conflict of interest that has far-reaching repercussions, not just for the obstetricians who deliver babies, but also for the babies themselves. The ACOG 2003 monograph, NEACP, is precisely the kind of "junk science" that the United States Supreme Court decried and which was the rationale for the far-reaching changes in evidentiary standards promulgated by the Court in *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 113 S.Ct. 2786, (1993).

In *Neurology of the Newborn*, Dr. Volpe addressed the dangers of the medical profession's attempt to deny the importance or even the existence of intrapartum brain injury in the face of an explosion of obstetrical litigation. His words deserve serious

[27] McCrary, V., et al., "A National Survey of Policies on Disclosure of Conflicts of Interest in Biomedical Research," N. Eng. J. Med. 343:1621-1626 (2000).
[28] *Id*.
[29] "The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysicology*, (2003) at xiv.

consideration, particularly in light of the Defendant's attempt to impose "mandatory" criteria for the assessment and treatment of intrapartum hypoxic injury to the exclusion of all other factors:

> The occurrence of injury to brain during the birth process has been the focus of clinical research for over a century. In my view that work has shown that brain injury in the intrapartum period does occur, affects a large absolute number of infants worldwide, is obscure in most cases in terms of exact timing and precise mechanisms, awaits more sophisticated means of detection in utero, and **represents a large source of potentially preventable neurological morbidity.** (emphasis added).
>
> Among the many adverse consequences of the explosion in obstetrical litigation **has been a tendency in the medical profession to deny the importance or even the existence of intrapartum brain injury**. (emphasis added). Although it is unequivocally clear that true obstetrical malpractice is a *rare* occurrence and that the obstetrician is called upon to deal with perhaps the most dangerous period in an individual's life with *inadequate methods*, this tendency is particularly unfortunate. With the recognition from experimental studies that much of hypoxic-ischemic brain injury evolves after cessation of the insult and can be interrupted to a considerable extent by several approaches (see Chapters 6 and 8), the ultimate possibility of intervention both in utero and in the early post natal period is strongly suggested. **Denial that intrapartum injury occurs may impair development and application of such brain-saving intervention**. (emphasis added). [30]

It is unthinkable that the development and application of brain-saving interventions would be sacrificed to the self-interests of ACOG and its membership, [31] To adopt the argument that the four essential criteria are "mandatory" and must be met before diagnosis, and therefore treatment, is unconscionable. It is a position based upon nothing but literature clearly generated for purposes of litigation and is wholly unreliable. See, *Daubert v. Merrell Dow, Pharms., Inc.* 43 F.3d 1311, 1317 (9th Cir. 1995) (research

[30] Volpe 4th Edition at p. 283.

[31] These physicians are called upon in the Hippocratic Oath to "prescribe regiments for the good of my patients according to my ability and my judgment and never do harm to anyone."

conducted independently of litigation provides important, objective proof that the research comports with the dictates of good science).

C. The ACOG criteria and its predecessors are and were scientifically unreliable.

1. Technical Bulletin 163.

ACOG Technical Bulletin 163 previously claimed that four criteria must be present before cerebral palsy could "plausibly" be linked to birth asphyxia. Criticism of these criteria came quickly.[32] In one study, researchers evaluated 47 newborns diagnosed with cerebral palsy.[33] No cause was found for the infants' neurologic injuries other than intrapartum asphyxia. Yet only 10 infants satisfied all of the ACOG Technical Bulletin 163 criteria and only 24 satisfied at least three of those four criteria. Fourteen of the babies had no other organ damage. Many of the babies had a blood pH level greater than 7.0. Only slightly more than half had APGAR scores of less than three at five minutes. Two of the babies did not have documented seizures in the early neonatal period. The authors concluded that the ACOG Technical Bulletin 163 criteria were not valid.[34] Another study of 292 infants with permanent neurologic injury caused by fetal asphyxia revealed that 36% did not have multi-system organ damage.[35] Another study reported that about 20% of infants with apparent fetal asphyxia have no evidence of end organ injury. Yet this was an essential criteria under Technical Bulletin 163.[36] Clearly, Technical Bulletin 163 was not valid.

2. The Template

[32] Goodlin, *supra*.
[33] Korst, supra.
[34] Id.
[35] Phelan, supra
[36] *See*, ACOG Technical Bulletin 163.

The 1999 report of the International Cerebral Palsy Task Force ("The Template") included four essential and five non-essential criteria "suggesting" that acute intrapartum hypoxia was the cause of cerebral palsy. Note: these were not "mandatory" criteria as the hospital would have us adopt in this case. The findings of this task force were targeted for criticism soon after publication. Several British obstetricians observed, among other things, that the cord blood pH cut-off of less than 7.0 was too low and excluded a significant number of babies who may have suffered intrapartum asphyxia.[37] These critics pointed out that "none of the evidence on which the …document is based meets the criteria for grade-A [medical] evidence" and that "the level of evidence is at best grade C."[38] The Template was no more valid than Technical Bulletin 163.

3. ACOG 2003 Monograph

a. A lack of input from neuro-scientists.

The purposed objective of Task Force on Neonatal Encephalopathy and Cerebral Palsy was to "create a multidisciplinary task force to review and consider the current state of scientific knowledge about the mechanism and timing of possible etiologic events which may results in neonatal encephalopathy.[39] However, in the end analysis, one of the problems with the findings of the task force was the lack of input from neuroscientists – a critical discipline to any multidisciplinary task force studying brain neurologic injury. Of the 29 people listed as either members of the task force, staff or consultants, there was

[37] Dear, P., et al. "Response to the Proposal of a Template of Defining and Causal Relation Between Intrapartum Events and Cerebral Palsy," Clinical Risk 6:137-142 (2000).
[38] *Id*. At 138.
[39] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogeneis and Pathophysicology*, (2003) at xiii.

not a single practicing pediatric neurologist listed.[40] Indeed, the only child neurologist included is Karin Nelson, M.D., who has not treated patients in decades and whose writings have been criticized in the peer-reviewed literature.[41] It is not surprising that actual neuroscientists' views differ from those of the monograph and ACOG. The "essential criteria" were developed from studies that lacked proper statistical analyses and were conducted before data from more reliable contemporary imaging studies were available. Again, for example, ACOG's President Franklin Miller admitted in wrong testimony last year:

> Q. Do they, in your mind and understanding of them, take into account the imaging studies one would expect to see after a sudden near-total occlusion of the cord – and acute near-subtle – sudden occlusion of the cord?
> A. Imaging of?
> Q. The brain.
> A. I don't believe that they do that, no.[42]

In March 2002, the American Academy of Neurology published a practice parameter for the diagnostic assessment of children with cerebral palsy.[43] According to the actual neuroscientists, not only should the neuro-imaging studies of the brain be taken into account, they are the starting point of the analysis.[44]

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[40] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogeneis and Pathophysicology*, (2003) at v-viii

[41] In fact her epidemiology based conclusions about the lack of correlation between increased cesarean section and rate of cerebral palsy have been criticized as using "fallacious logic", Shier and Tilson, *Scientific Contribution: The Temporal Stage Fallacy: A novel statistical fallacy in the medical literature*, MEDICINE, HEALTH CARE AND PHILOSOPHY (2006) 9:243–247.

[42] Deposition of Franklin Carl Miller at p. 37, lls, 12-21.

[43] "Practice Parameter: Diagnostic Assessment of the Child with Cerebral Palsy (Report of the Quality of Standard Subcommittee of the American Academy of Neurology in the Practice Committee of the Child Neurology Society),", *Neurology* 62:851 (2004)

[44] *Id*. At 861.

Barkovich and his associates also observe that MR spectroscopy performed in the first 24 hours after birth is sensitive to the severity of hypoxic-ischemic brain injury and have had considerable effect on the diagnosis of brain injury.[45]

b. ACOG Statistics are not reliable:

One of the principal studies upon which the monograph relies for support is authored by Nadia Badawi and concludes that the "causes of newborn encephalopathy are heterogeneous and many of the causal pathways start before birth."[46] A recent study testing this hypothesis unequivocally confirmed that events during labor and delivery, not earlier ones, are responsible for most cases of neonatal encephalopathy and early seizures.[47] The investigators' studied 351 term infants who exhibited either neonatal encephalopathy, early seizures or both, within 72 hours of birth. Neonatal encephalopathy was indicated by abnormal tone, feeding difficulties, altered alertness, and at least three of the following criteria: late decelerations or meconium staining, delayed onset of respiration, arterial cord blood pH less than 7.1, APGAR scores of less than 7 at five minutes, and multi-organ failure. The babies were all evaluated neuroradiologically and results compared with signs recognized as suggesting an antepartum event or developmental abnormality, as opposed to injury during the labor and delivery process. The scans showed that 261 of the babies met the criteria for neonatal encephalopathy. The scans further revealed that 197 of those babies showed evidence of acutely evolving lesions compatible with a hypoxic-ischemic insult. Of the 351 babies scanned, 306 showed evidence of an acute intrapartum injury.

[45] Barkovich, A.J., et al. "Proton Spectroscopy and Diffusion Imaging on the First Day of Life after Perinatal Asphyxia: Preliminary Report," *Am.J. Neuroradio* 22: 1786-1794 (2001) attached to this Motion as and incorporated fully by reference.
[46] Badawi, N. et al., "Intrapartum Risk Factors for Newborn Encephalopathy: The Western Australian Case-Control Study," Brit. Med. J. 317:1549-1554 (1998).
[47] Cowan, F., et al, "Origin and Timing of Brain Lesions in Term Infants with Neonatal Encephalopathy," Lancet 361:736 (2003).

Only 45 suggested some evidence developmental, metabolic or other disorders that preceded the labor and delivery process. The authors delivered a clear message:

> Some investigators have reported that only 8% to 15% of terms infants with neonatal encephalopathy, and far fewer with neonatal seizures, have evidence of asphyxia immediately before birth. Furthermore, others have suggested that many neurological signs once thought to be caused by intrapartum asphyxia are a manifestation of a process begun during the antenatal period. **However, we have found little evidence for the two proposals that acute perinatal injury is uncommon in such infants and that injurious processes have been taking place antenatally**. (emphasis added).[48]

The ACOG 2003 monograph cites the incidence of neonatal encephalopathy attributable to intrapartum hypoxia at an estimated 1.6 per 10,000 births.[49] This figure is cited no less than four times in the monograph. However, the reported incidence of conditions labeled birth asphyxia, HIE, or post-asphyxial encephalopathy in term or near-term infants ranges from 1 to 8 per 1,000 births.[50] Even the Badawi article, otherwise relied on by the monograph, cites the incidence of newborn encephalopathy as 3.8 per 1,000 term live births.[51] Even if one were to accept that only approximately 30% of neonatal encephalopathy is attributable to intrapartum hypoxia as found by Badawi, but contrary to the some 90% found by Cowan, then 30% of 3.8 equals 1.14 per 1,000 births.[52] The ACOG 2003 monograph's figure understates the supported figures by ten-fold.

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[48] *Id*.
[49] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysicology* (2003) at p. 3
[50] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysicology* (2003) at p. 3
[51] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysicology* (2003)
[52] Cowan, *supra*

c. The ACOG pH criteria are invalid.

One of the monograph's "essential criteria" for determining that a baby's brain injury was caused by intrapartum asphyxia is a cord blood pH of less than 7.0 and a base excess of 12 or greater. This criteria is not supported by valid data. The medical literature has documented cases in which babies suffering from a hypoxic-ischemic encephalopathy indeed had a blood pH value greater than 7. In fact, the pH value is not even considered in one of the major studies in which the monograph relies for its hypothesis that most cases of cerebral palsy occurred in the antepartum period.[53] In Badawi's study, the criteria used to determine whether a baby was exposed to intrapartum hypoxia were abnormal fetal heart rate, meconium staining, or both, along with a one-minute APGAR score of less than 3 and a five-minute score of less than 7. Several of the babies included in that study had a cord pH greater than 7.[54] This is not surprising. In point of fact, Hermansen recently studied the relationship between acute birth asphyxia, acidosis and brain injury. He challenged the "traditional assumptions" regarding pH. Dr. Hermansen noted that:

> Ruth and Raivio reported a series of infants who had an adverse outcome with perinatal asphyxia as a possible or the most credible cause from an initial sample of about 1000 consecutive births. Nearly all (93%) had a pH of >7.15 and 79% had pH >7.18.[55]

Clearly the monograph's criteria are not valid. Dr. Hermansen, a well known neonatologist, has testified unequivocally that the criteria is not valid.

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[53] Badawi, *supra*
[54] Id. At 1555
[55] Hermansen, M., "The acidosis paradox: asphyxial brain injury without coincident acidemia," Developmental Medicine & Child Neurology, 45:353-356, (2003); see also, Casey, B., et al., "Outcomes Among Term Infants When Two-Hour Post Natal pH is Compared with pH at Delivery," Am. J. Obstete. Gyncol., 184: 44 (2001).

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Q. Have you testified before that if the criteria is met, you can state that birth asphyxia did occur during the intrapartum period sufficient to cause cerebral palsy?

A. I don't know my exact words and the case it was relevant to. But I'll tell you, the criteria are worth looking at as you assess a case. And the more criteria you have, the stronger the case is going to be as an asphyxia/cerebral palsy connection. If you have all the criteria, it's probably a very strong asphyxia case. You don't need them all. There are, in truth, no essential criteria. But they are worth looking at. And if you meet the criteria, I suspect you're going to call it asphyxia.[56]

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Q. And you agree that Apgar scores don't need to be part of the four essential criteria? You agree with that?

MR. FREEMAN: Objection. Form.

THE WITNESS: They don't include those in their essential criteria. See, my problem with agreeing with it is it suggests that I even agree

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that there are essential criteria. I think the whole concept is a fraud.

BY MS. SMITH:

Q. A fraud?

A. Yes. It's not honest. It's not science.

Q. Well, if --

A. There's no scientific basis for it. It was done for legal reasons.[57]

It is interesting to note that over the course of several years, ACOG has aggressively tried to lower the value of blood pH values that is significant in evaluating the condition of neonates. In 1976, ACOG stated in Technical Bulletin No. 42 that a pH of below 7.20

[56] Deposition of Hermansen, p. 63.
[57] Deposition of Hermansen, pp. 65, 66

determined fetal distress.[58] Only one year later in 1977, ACOG states that when a pH of 7.20 occurred, expeditious delivery was required.[59] In 1989, ACOG started backing off this position, stating that a pH of *less than* 7.20 was required for medical intervention.[60] In 1992, ACOG further restricted their position, stating that a cord pH as low as 7.00 was required to justify a causal link between intrapartum asphyxia and neurologic injury.[61] "The poor predictive value of newborn blood pH for subsequent cerebral palsy has been repeatedly noted.[62] The problem is that there is no pH value that clearly separates those babies who have experienced potentially damaging intrapartum asphyxia from those who have not.[63]

In clinical and medico-legal practice, no diagnoses can be made or refuted on the basis of a single laboratory measurement and the idea of a set cut-off is naïve. The recommended cut-off value of 7.0 is too low and will exclude a significant number of babies who have possible encountered damaging intrapartum asphyxia.[64]

> Unfortunately, there is no pH value that separates cleanly those babies who have experienced intrapartum injury from those who have not – no prognosis can be made or refuted on the basis of a single laboratory measurement.[65]

d. The monograph's literature does not support its conclusions

ACOG and the Defendants exaggerate the persuasive power of NEACP and its underlying studies. The much touted essential criteria are set forth in chapter eight of ACOG's 2003 monograph. The criteria are allegedly based on data from 72 articles. A

[58] *Technical Bulletin No. 42,* Am.C. Obstetricians & Gynecologists (1976)
[59] *Technical Bulletin No. 44,* Am. C. Obstetricians & Gynecologists (1977)
[60] *Technical Bulletin No. 127,* Am.C. Obstetricians and Gynecologists (1989)
[61] *Technical Bulletin No. 163,* Am. C. Obstetricians & Gynecologists (1992)
[62] Goodlin at p. 1831
[63] Dear at p. 139.
[64] *Id*.
[65] Schifrin at 439, Socol, M., et al, "Depressed Apgar scores, acid-base status, and neurologic outcome." Am. J. Obstet. Gynecol., 170-991 (1994 reference. (reporting that several newborns with complicated neonatal course had pH> 7.0 and base excess < 12).

careful analysis of these articles, however, reveals that the monograph criteria is a classic example of "junk science". Twenty-seven of the articles support none of the essential criteria. Another 16 of the articles represent nothing more than their authors' opinions. Of the 29 articles cited to support one or more criteria, not a single one is based on a properly designed, randomized, control trial.[66] .

Contrary to ACOG's pronouncements in the 2003 monograph, asphyxia sets into motion a series of biochemical and clinical events that vary from one patient to the next. As stated by Dr. Pasternak:

> Hypoxic ischemic encephalopathy is notoriously variable from infant to infant. Seemingly similar degrees of insult may completely spare one child and devastate another. To some extent, this extraordinary variability is evidence of inability to accurately quantify the magnitude of the impairment of gas exchange in utero. Thus, seeming similar degrees of insult may in fact not be similar at all. Furthermore, intrinsic fetal differences . . . may also affect the response to hypoxic insult and play a key role in determining outcome.[67]

III. The ACOG criteria has been rejected by Courts.

Use of the essential criteria set out in some form in all three publications is a common defense strategy and has been rejected by at least two courts. In *Koval v. Kincheloe*, 2001 WL 35748892 (W.D. Okla. 2001), the defendants moved for summary judgment alleging that the plaintiffs did not have a sufficient scientific basis for their opinion that the minor plaintiff's cerebral palsy was caused by hypoxia or ischemia. *Id*. at 1. Specifically, they contended that there were criteria which according to ACOG must be present in the newborn before cerebral palsy can be linked to the events of labor and delivery as set out in ACOG Technical Bulletin No., 163 and in the Template. *Id*. at 2-3.

[66] The 72 articles cited in Chapter 8 are in the Appendix.
[67] Pasternak, J.F., "Hypoxic-ischemic Brain Damage in the Term Infant," Pediatric Clinics N.Am. 40:1061, 1062 (1993).

In response to this contention, the district court observed that "while the ACOG criteria are significant, they are not necessarily the only determinative factors on which a physician may reasonably rely in considering the cause of cerebral palsy." *Koval v. Kincheloe*, 2001 W.L. 34748892 at 4. As to the factors laid out in The Template, this consensus statement was simply suggestive of a disagreement in the medical community regarding whether cerebral palsy can be shown to have been caused by intrapartum event of hypoxia. *Id*.

In *1st of America Bank, Mid-Michigan, N.A. v. U.S.*, 752, F. Supp. 764, 765 (E.D. Mich. 1990), the court determined that Air Force physicians were guilty of malpractice and that their malpractice was a proximate cause of the cerebral palsy suffered by the minor plaintiff, Michaela King. Mrs. King testified that after Michaela's birth, she was told by two physicians that the child's injuries were the result of anoxia at birth. *Id*. at 769. However, after the lawsuit involving Michaela's care was filed, they "stopped referring to anoxia, and only referred to cerebral palsy". *Id*. In its written opinion, the District Court found particularly "troublesome" the fact that the defendant's witnesses had not held a consistent position with regard to causation; most notably, Dr. Silverstein, who treated Michaela in 1985 and 1986 and clearly determined that perinatal asphyxia was the most likely cause of her condition. *Id*. at 772. At trial, she testified that perinatal asphyxia was not the cause of Michaela's condition, despite the fact that she did not see Michaela in between her change in position. The court noted:

> **In response to questions from the Court about why she had changed her position, Dr. Silverstein stated that in the profession generally, in response to litigation, there had been writing and discussion on diagnosing asphyxia in more limited circumstances, and that her thinking had evolved as a result of what she had read. The Court finds her testimony evasive, her attitude disdainful and arrogant, and the**

motivation for her change of opinion clearly suspect. The Court will disregard her changed opinion.

752 F. Supp. at 772. Noting that other physicians likewise changed their positions subsequent to the filing of the lawsuit, the Court said it looked "with suspicion on the testimony of those witnesses who changed their position." *Id*.

Dr. Silverstein testified that an article by Dr. Karin Nelson, provided to many of the doctors and upon which they relied in changing their opinions, was written in response to litigation. *Id*. at 773. Dr. Nelson set out certain criteria that had to be satisfied before a diagnosis of cerebral palsy could be made: (1) intrapartum difficultly; (2) other organ difficulties and a newborn course consistent with asphyxia; (3) other causes ruled out, and (4) all studies complete. *Id* at 774. The District Court rejected these criteria finding that they were of "questionable value" in litigation and observing that if a court held a plaintiff to these criteria before finding causation, the plaintiff would be required to do more than demonstrate causation by a preponderance of the evidence. The plaintiff would have to produce essentially conclusive proof. *Id*. Plaintiff asks this court to reject the Defendant's attempt to place a higher evidentiary burden than imposed by Arkansas law.

In *Tavares v. New York City Health and Hospitals Corp*., No. 45757/00, 2003 WL 22231534 (N.Y. Sup. Ct. June 23, 2003), the court was asked to conduct a *Frye*[68] hearing to determine if the plaintiffs' theory regarding the cause of an infant's injuries was admissible. The defendants claimed that ACOG Technical Bulletin No. 163, the International Template, and Chapter 8 of the NEACP precluded the plaintiffs' experts

[68] *Frye v. United States*, 293 F. 1013 (1023), still applied in name at least in New York (holding that innovative scientific evidence must be based on a principle or procedure which has gained general acceptance as reliable in the relevant scientific community). *Tavares* notes that the same result is reached under the more liberal *Daubert* standard. *Tavares* 2003 WL 22231534, at *18.

from testifying that the infant's exposure to intrapartum asphyxia caused Cerebral Palsy.[69] *Tavares* cites *Koval* with approval and reached the same conclusion: there is sufficient evidence in the medical literature criticizing the validity of the obstetrical community's litigation criteria, supporting that the notion that "a rigid criteria of three or four factors must be present to find that cerebral palsy was caused by asphyxia or by a hypoxic event, [is] not generally accepted by the medical community as necessary to a diagnosis of neurological defect. Rather, plaintiffs have demonstrated that many authorities have indicated that the Apgar score and pH level are poor indicators of neurological defects and that not all infants born suffering from cerebral palsy have multiorgan involvement. Further, the literature cited also demonstrates that Bulletin 163, the BMJ Template, and Chapter 8 have been criticized for lack of reliability." *Tavares* 2003 WL 22231534, 18.

The reasoning of the dissenting opinion of Justice Castille in *Blum v. Merrell Dow Pharmaceuticals*, 764 A.2d 1 (PA 2000), is relevant, by way of example, to ACOG's NEACP criteria too. *Blum* is a products liability case, applying *Frye*, in which the relevant scientific community appears to have rejected a causal link between the drug Bendectin and birth defects. Justice Castille was concerned that there must be a limited exception to *Frye* when there is evidence to show that the scientific orthodoxy that is invoked to exclude minority views was a result of proprietary research influenced by an interested party, similar to the case with ACOG and its criteria. In *Blum*, Justice Castille emphasized that Merrell Dow largely created the "generally accepted orthodoxy" that would freeze out viewpoints contrary to their litigation interests. Merrell Dow subsidized or otherwise influenced most of the studies that concluded that Bendectin does not cause birth defects. Merrell Dow's role in virtually creating, and then slanting, the "scientific

[69] *Tavares*, 2003 WL 2223 1534, at *9-11

community" should be a relevant factor in the analysis. Castille said, "[t]here is something not a little offensive about an entity, creating a biased, litigation-driven scientific 'orthodoxy,' and then being permitted to silence any qualified expert holding a dissenting view on grounds of 'unorthodoxy.' Where the would-be relevant scientific community is a community beholden to the defendants' litigation interests, then that biased community should not be permitted to squelch dissenting opposing opinions. The trial court here properly refused to allow that unjust result to occur." *Blum*, 764 A.2d at 16-17.

State and federal trial and appellate judges should follow Justice Castille's lead and prevent any biased organization or limited scientific community from attempting to squelch dissenting opinions from otherwise-qualified experts.