Facts

• CP rate 1.5/1000
• No change in last 40 years
• No geographic/economic boundaries
Case Study

- 27 yo G2P1 28 wks/EGA
- PMH – IUFD at 31 wks
  - Severe preeclampsia
- Maternal transport – severe preeclampsia
Case Study

• Rx:
  - MGSO4
  - Oxytocin
  - Hydralazine PRN
Ten Hours Into Induction

- Repetitive late decelerations
  - Oxytocin stopped
  - Left lateral position
  - Mask oxygen

- Decelerations promptly resolved
Case Study

C-Section – Low Vertical
General Anesthesia
Case Study

970 gram infant

Apgar 1/0/0/0/0/0

Pronounced at 20 minutes
NATURE AND DURATION OF COMPLAINTS (Include circumstance of admission)

26-29 wk EGA Black Male infant
cSevere birth anoxia
- Prematurity
- Acute Cardio-respiratory failure with 7708 cm of inability to resuscitate at 29 weeks in delivery room.

Chronic intrauterine Birth asphyxia (per death cert) 7685
Did not have birth asphyxia. DM Threlfall

P-1
Autopsy Report

“...findings are consistent with chronic intrauterine anoxia, as well as possible acute anoxia secondary to prolonged labor or difficult delivery.”

Gross and microscopic examination normal.
## Cord Gas

<table>
<thead>
<tr>
<th></th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.273</td>
<td>7.30</td>
</tr>
<tr>
<td>pCO₂</td>
<td>57.6</td>
<td>50.0</td>
</tr>
<tr>
<td>pO₂</td>
<td>17.4</td>
<td>18.6</td>
</tr>
<tr>
<td>HCO₃</td>
<td>25.9</td>
<td>24.1</td>
</tr>
<tr>
<td>Base excess</td>
<td>-4.0</td>
<td>-1.6</td>
</tr>
</tbody>
</table>
Charge by Dr. Frank Miller

“To create a multidisciplinary task force to review and consider the current state of scientific knowledge about the mechanisms and timing of possible etiologic events which may result in neonatal encephalopathy and cerebral palsy.”
Task Force on Neonatal Encephalopathy and Cerebral Palsy Committee Members

Gary Hankins, MD, FACOG, Chair
Mary D’Alton, MD, FACOG
Mark Ira Evans, MD, FACOG
Larry Gilstrap, MD, FACOG
Richard Depp, III, MD, FACOG
Roger K. Freeman, MD, FACOG
Richard P. Green, MD, FACOG
Task Force on Neonatal Encephalopathy and Cerebral Palsy Committee Members

James A. McGregor, MD, FACOG
Karin B. Nelson, MD
Susan Ramin, MD, FACOG
Robert Resnik, MD, FACOG
Michael Speer, MD
Louis Weinstein, MD, FACOG
Stanley Zinberg, MD, MS
Neonatal Encephalopathy and Cerebral Palsy Task Force Consultants

James Barkovich, MD – Professor of Radiology, Neurology, Pediatrics, and Neurosurgery

Kurt Benirschke, MD – Professor Emeritus of Pathology and Reproductive Medicine

Robert R. Clancy, MD – Professor of Neurology and Pediatrics, Pediatric Regional Epilepsy Program

Gabrielle A. DeVeber, MD – Director, Canadian Pediatric Ischemic Stroke Registry

Ronald Gibbs, MD – Professor and Chairman, OB/Gyn

Gregory Locksmith, MD – Assistant Professor, OB/Gyn
Neonatal Encephalopathy and Cerebral Palsy Task Force Consultants

Jeffrey Perlman, MD, PhD – Professor of Pediatrics and OB/Gyn
Julian N. Robinson, MD – Assistant Professor, OB/Gyn
Dwight J. Rouse, MD – Associate Professor, OB/Gyn
George R. Saade, MD – Professor, OB/Gyn
Diana E. Schendel, PhD – Centers for Disease Control and Prevention
Rodney E. Willoughby, Jr., MD – Associate Professor, Pediatrics
Marjorie R. Grafe, MD – Professor, Placental Pathology
Methods

- Five meetings over 3 years
- Extensive consultation with clinicians and scientists
- Consensus development
- Draft and redraft
Methodology

- Throughout the process, primary source documents were cited to the fullest extent possible.
- Comments solicited from a number of professional organizations:
  - American Academy of Pediatrics
  - The Canadian Paediatric Society
  - The Child Neurology Society
  - The Society for Maternal-Fetal Medicine
  - The March of Dimes Birth Defects Foundation
Methodology

- The Centers for Disease Control and Prevention, Department of Health and Human Services
- The National Institute of Child Health and Human Development
- The Royal Australian and New Zealand College of Obstetricians and Gynaecologists
- The Society of Obstetricians and Gynaecologists of Canada.
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Most Peer-Reviewed Document on the Subject
Neonatal Encephalopathy – “A clinically defined syndrome of disturbed neurological function in the infant at or near term during the first week after birth, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, altered level of consciousness, and often seizures.”
Differential Dx: Neonatal Encephalopathy

- Developmental abnormalities
- Metabolic abnormalities
- Autoimmune disorders
- Coagulation disorders
- Infections
- Trauma
- Hypoxia

- IUGR
- Multiple gestations
- Antepartum hemorrhage
- Chromosomal abnormalities
- Persistent breech/transverse lie
On the Diagnosis of Birth Asphyxia

- Relies on:
  - Clinical markers of fetal distress (MSAF/Abn FHR)
  - Laboratory markers (Cord pH or base excess)
  - Newborn status (Apgars, time to respirations)

- No data to support:
  - Equivalence of criteria
  - Specificity to intrapartum asphyxia

- Potential for misclassification enormous
Relationship of Intrapartum Asphyxia to Neonatal Encephalopathy and Cerebral Palsy
“Epidemiological studies suggest that in about 90% of cases of cerebral palsy intrapartum hypoxia could not be the cause of cerebral palsy and in the remaining 10% intrapartum signs compatible with damaging hypoxia may have had antenatal or intrapartum origins.”
Key Publications - ACOG

- Committee Opinion #49, Nov 1986/1989
  - *Use and Misuse of the Apgar Score*
  - Committee on Obstetrics: MFM (ACOG)
  - Committee on Fetus and Newborn (AAP)
- ACOG Technical Bulletin #163, January 1992
  - *Fetal and Neonatal Neurologic Injury*
- Committee on Obstetric Practice #137, April 1994
  - *Fetal Distress and Birth Asphyxia*
- Committee on Obstetric Practice #197, Feb 1998
  - *Inappropriate Use of the Terms Fetal Distress and Birth Asphyxia*
Antepartum Risk Factors for Newborn Encephalopathy: The Western Australian Case-Control Study

- Metropolitan Western Australia June 93-Sept 95
- All 164 term infants with moderate/severe encephalopathy
- Controls – 400 randomly selected
- Stats
  - Birth prevalence of moderate/severe newborn encephalopathy 3.8/1000 term live births
  - Neonatal Fatality 9.1%
- Conclusions
  - Causes of newborn encephalopathy are heterogeneous and many of the causal pathways start before birth
Risk Factors for Newborn Encephalopathy

- Abnormal Placental Appearance: 2.07
- Emergency C/S: 2.17
- Instrumental Delivery: 2.23
- Family Hx Seizure: 2.55
- Family Hx Neurological Disorder: 2.73
- Viral Illness: 2.97
- Mod/Severe Antepartum Bleeding: 3.57
- Intrapartum Fever: 3.82

Badawi, BMJ 317:1549
“A New York woman infected with the West Nile virus gave birth to a brain-damaged infant in November who was also infected with the virus, according to a report from officials with the Centers for Disease Control and Prevention.”
Risk Factors for Newborn Encephalopathy

- OP Presentation: 4.29
- IUGR 3-9%tile: 4.37
- Infertility Treatment: 4.43
- Acute Intrapartum Event: 4.44
- Severe Preeclampsia: 6.3
- Maternal Thyroid Disease: 9.7

Badawi, BMJ 317:1549
Risk Factors for Newborn Encephalopathy

- Abnormal Placental Appearance: 2.07
- IUGR <3%tile: 38.23
Risk Factors for Newborn Encephalopathy

- No Labor: 0.17
- Elective C/S: 0.17
Distribution of Risk Factors for Newborn Encephalopathy

- Antepartum risk factors only (69%)
- Antepartum risk factors and intrapartum hypoxia (25%)
- Intrapartum hypoxia only (4%)
- Unknown (2%)

Badawi, BMJ 317:1557
Intrapartum Asphyxia: A Rare Cause of Cerebral Palsy

“It was estimated that in only 8% (15/183) of all the children with spastic cerebral palsy was intrapartum asphyxia the possible cause of their brain damage.”
### Theoretical Scenarios for Timing of Neurological Insult in Newborn Encephalopathy

<table>
<thead>
<tr>
<th>Antepartum period</th>
<th>Intrapartum period</th>
<th>Neonatal period</th>
<th>Newborn outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Insult</td>
<td></td>
<td>Encephalopathy</td>
</tr>
<tr>
<td>2</td>
<td>Insult</td>
<td>Further Insult</td>
<td>Encephalopathy</td>
</tr>
<tr>
<td>3</td>
<td>Insult</td>
<td></td>
<td>Encephalopathy</td>
</tr>
<tr>
<td>4</td>
<td>Insult</td>
<td>Insult</td>
<td>Encephalopathy</td>
</tr>
</tbody>
</table>

Badawi, BMJ 317:1557
Uncertain Value of Electronic Fetal Monitoring in Predicting Cerebral Palsy

• Population
  – Four California counties 1983-1985
  – Singleton – Birthweight ≥ 2500 gm
  – Survival to age 3 and EFM (N=78)
  – Moderate/severe cerebral palsy

• Abnormal FHR
  – Multiple late decelerations
  – Decreased beat to beat

• Controls
  – Matched appropriately (N=300)

Nelson, NEJM 334:613
### Uncertain Value - Continued

<table>
<thead>
<tr>
<th>Pattern</th>
<th>CP N=78</th>
<th>Control N=300</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tachycardia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 160</td>
<td>22 (28.2)</td>
<td>85 (28.3)</td>
<td>1.0 (0.6-1.7)</td>
</tr>
<tr>
<td>&gt; 180</td>
<td>5 (6.4)</td>
<td>16 (5.3)</td>
<td>1.3 (0.4-3.4)</td>
</tr>
<tr>
<td><strong>Bradycardia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 100</td>
<td>27 (34.6)</td>
<td>75 (25.0)</td>
<td>1.5 (0.9-2.5)</td>
</tr>
<tr>
<td>&lt; 80</td>
<td>13 (16.7)</td>
<td>35 (11.7)</td>
<td>1.5 (0.8-3.0)</td>
</tr>
<tr>
<td><strong>Multiple Lates</strong></td>
<td>11 (14.1)</td>
<td>12 (4.0)</td>
<td>3.9 (1.7-9.3)</td>
</tr>
<tr>
<td><strong>Mult. Lates or ↓ BTB</strong></td>
<td>21 (26.9)</td>
<td>28 (9.3)</td>
<td>3.6 (1.9-6.7)</td>
</tr>
</tbody>
</table>

Nelson, NEJM 334:613
“The 21 children with cerebral palsy who had multiple late decelerations or decreased variability in heart rate monitoring represented only 0.19 percent of singleton infants with birth weights of 2500 g or more who had these fetal-monitoring findings, for a false positive rate of 99.8%.
Extrapolation from Nelson by Hankins

1. Based on multiple late or persistent decreased beat to beat variability, 500 cesarean sections are required to prevent 1 case of cerebral palsy!

2. Future uterine rupture rate 0.5% = 2.5 ruptures
   40% risk of neonatal death or CP = 1.0

3. Future risk of previa and acreta 10-20 x RR

4. Excess maternal risk of cesarean
   - EBL at least doubled
   - Infection 5-10 x RR
   - DVT/PE 5 x RR
“It is not possible at the current time to prospectively recognize during labor the point in time when a reduction in cerebral perfusion results in irreversible brain injury.”

Jeffrey M. Perlman, M.D.
“Any claims to the contrary are not supported by any scientific merit.”

ACOG Task Force on Neonatal Encephalopathy and Cerebral Palsy
A Template for Defining a Causal Relationship Between Acute Intrapartum Events and Cerebral Palsy

Method:
- Multidisciplinary review of pertinent medical literature
- Open to any and all who might add

Goal:
1. Benefit research into causation and prevention of CP
2. Help those who offer expert opinion when counseling (parents or would be parents) or giving opinions in court.

Participation Invited:
- All members Perinatal Society ANZ
- International scientific contributors

Paper redrafted 8 times until consensus reached
Need for timely updates acknowledged
<table>
<thead>
<tr>
<th>Panel Members</th>
<th>Discipline</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alastair MacLennan (Chair)</td>
<td>Obstetrics and Gynecology</td>
<td>University of Adelaide</td>
</tr>
<tr>
<td>Fiona Stanley</td>
<td>Epidemiology</td>
<td>Institute for Child Health Research, Perth</td>
</tr>
<tr>
<td>Eve Blair</td>
<td>Epidemiology</td>
<td>Institute for Child Health Research, Perth</td>
</tr>
<tr>
<td>Greg Rice</td>
<td>Fetal Physiology</td>
<td>Royal Women’s Hospital, Melbourne</td>
</tr>
<tr>
<td>Peter Stone</td>
<td>Obstetrics and Gynecology</td>
<td>University of Otago</td>
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<tr>
<td>Jeffrey Robinson</td>
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<tr>
<td>David Henderson-Smart</td>
<td>Perinatal Medicine</td>
<td>University of Sydney</td>
</tr>
<tr>
<td>Victor Yu</td>
<td>Neonatal Intensive Care</td>
<td>Monash Medical Centre</td>
</tr>
<tr>
<td>Michael Harbord</td>
<td>Pediatric Neurologist</td>
<td>Flinders Medical Centre</td>
</tr>
<tr>
<td>Panel Members</td>
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</tr>
<tr>
<td>Leon Stern</td>
<td>Pediatric Rehabilitation</td>
<td>Crippled Children’s Association of South Australia</td>
</tr>
<tr>
<td>Helen Chambers</td>
<td>Perinatal Pathology</td>
<td>Women’s and Children’s Hospital, Adelaide</td>
</tr>
<tr>
<td>Margaret Furness</td>
<td>Radiology</td>
<td>Women’s and Children’s Hospital, Adelaide</td>
</tr>
<tr>
<td>Tina Hayward</td>
<td>Radiology</td>
<td>Women’s and Children’s Hospital, Adelaide</td>
</tr>
<tr>
<td>Kerena Eckert</td>
<td>Midwifery Research</td>
<td>Women’s and Children’s Hospital, Adelaide</td>
</tr>
<tr>
<td>Christopher Boundy</td>
<td>Barrister and Solicitor</td>
<td>Adelaide</td>
</tr>
<tr>
<td>Susan Merrett</td>
<td>Medical Administrator</td>
<td>SA Health Commission</td>
</tr>
<tr>
<td>Mark Kenny</td>
<td>Medico Legal Services</td>
<td>Women’s and Children’s Hospital, Adelaide</td>
</tr>
</tbody>
</table>
Template Criteria to Define an Acute Intrapartum Hypoxic Event

• Essential criteria
  1. Evidence of a metabolic acidosis in intrapartum fetal, umbilical arterial cord or very early neonatal blood samples. pH < 7.00 and base deficit ≥ 12 mMol/L and;
  2. Early onset of severe or moderate neonatal encephalopathy in infants ≥ 34 weeks gestation and;
  3. Cerebral palsy is of the spastic quadriplegic or dyskinetic type.
Criteria Required to Define an Acute Intrapartum Hypoxic Event As Sufficient to Cause Cerebral Palsy

Essential criteria (must meet all four)

1. Evidence of a metabolic acidosis in fetal, umbilical cord arterial blood obtained at delivery (pH 7.00 and base deficit $\geq 12$ mmol/L).

2. Early onset of severe or moderate neonatal encephalopathy in infants of 34 or more weeks of gestation.
Criteria Required to Define an Acute Intrapartum Hypoxic Event As Sufficient to Cause Cerebral Palsy

3. Cerebral palsy of the spastic quadriplegic or dyskinetic type*.

4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders.
Criteria That Together Suggest an Intrapartum Timing but by Themselves Are Non-specific

1. A sentinel (signal) hypoxic event occurring immediately before or during labor.
2. A sudden, rapid and sustained deterioration of the fetal heart rate pattern usually following the hypoxic sentinel event where the pattern was previously normal.
3. Apgar scores of 0-6 for longer than 5 minutes.
4. Early evidence of multi-system involvement.
5. Early imaging evidence of acute cerebral abnormality.
Criteria That Collectively Suggest an Intrapartum Timing but Are Nonspecific to Asphyxial Insults

1. A sentinel (signal) hypoxic event occurring immediately before or during labor.

2. A sudden and sustained fetal bradycardia or the absence of fetal heart rate variability in the presence of persistent, late or variable decelerations, usually after an hypoxic sentinel event when the pattern was previously normal.
Criteria That Collectively Suggest an Intrapartum Timing but Are Nonspecific to Asphyxial Insults

3. Apgar scores of 0-3 beyond 5 minutes.

4. Evidence of multi-system involvement up to 72 hours.

5. Early imaging study showing evidence of acute nonfocal cerebral abnormality.
Case Study

- Temp 100.3 degrees F – Clinical Chorio
- Cx 9/C/0
- FHT’s 160 – Good variability
Case Study

- 2\textsuperscript{nd} Stage C/C/+1
- Bradycardia 70-90 x 3 min
- To delivery room
Case Study

- Bradycardia to 50 x 2 min
- Midforceps LOT → LOA
- Apgar 0/Intubated
- Weight 8 lb 1 oz
Umbilical Artery

- pH: 6.9
- pCO2: 130
- pO2: 10.9
- HCO3: 23.0
- BE: -9.7
Complete Asphyxia

- pCO2: ↑ 8 mm Hg/min
- Non-volatile acids: ↑ mEq/L/min
Relationship of Intrapartum Asphyxia to Neonatal Encephalopathy and Cerebral Palsy

- Intrapartum Asphyxia
- Neonatal Encephalopathy
- Cerebral Palsy
IMPORTANT

All other things being equal, neonatal resuscitation can potentially prevent or further the injury.
"This is your side of the family, you realize."
Delivering Justice

Curing Health Care / By Walter Olson

As the malpractice wars rage on, the press is finally noticing a new study with major implications for our medical liability system.

On Jan. 31, the American College of Obstetricians and Gynecologists, along with the American Academy of Pediatrics, released the results of a comprehensive two-year study surveying what is known about the causes of cerebral palsy and brain injury in full-term, live-born infants. According to the study, "the vast majority" of brain damage and cerebral palsy among these infants originates in factors largely or completely outside the control of delivery-room personnel -- factors that include prematurity, infection, genetic fetal abnormalities, disorders of blood clotting, maternal thyroid problems and diabetes. Contrary to what has long been assumed, an interruption of oxygen during labor is "not a significant cause in most cases of the condition."

Who is this news? In part because lawsuits blaming OB-GYNs for cerebral palsy and other infant brain damage can constitute the single biggest branch of medical malpractice litigation, yielding lawyers the highest settlements and the richest contingency fees, rivaled only by failure to diagnose cancer, if ACCOG's report is to be credited, much of this litigation looks to be scientifically unfounded.

Around 3,000 babies are diagnosed each year with cerebral palsy, an incurable disorder that in severe cases may require a lifetime of care. The distraught parent who turns to the Internet for information about a child's diagnosis will be bombarded with lawsuits as "delays in diagnosis cause cerebral palsy," blames one, another boasts of $29 million, $31 million and $335 million awards won by "our affiliated attorneys" who are the very same attorneys that could be assigned to sue that child. At CNN.com, until recently a picture of a wheelchair-flushed in among a roster of a stock of dollar signs "Your child's cerebral palsy may be the result of a medical mistake. Don't get mad. Get Enraged!"

These sites offer an ostensively independent medical evaluation of a child's file, which, however, is not necessarily independent of the lawyers' incentive to find someone to blame. Among many in the litigation business, it is an article of faith that mistakes in labor and delivery causing hypoxia, or lack of oxygen, are a very frequent cause of what had neurologically normal infants. The next step is to break the horrible news to the parent: Your child would never have had to endure this preventable disability had the doctors only done their job properly. One father was recently quoted as saying that on hearing this news he was prepaced with a desire to kill the doctor, probably not an unusual reaction. The lawsuit that follows will claim that brain damage could have been avoided had doctors only given the mother a Caesarean section, or given her one or two shots, or administered medication in a different combination, or at a higher or lower dosage, or there's an ample supply both of causation theory and of experts for hire willing to testify to them. Hired experts for the doctor and hospital then often dispute the causal link between the alleged lapse in care and the child's plight.

Most jurors, it seems, decide such cases in favor of the defense. But those that find for the plaintiff return awards that are not infrequently top $10 million. Last year the National Law Journal's nationwide top-100 verdicts included 15 medical liability cases, of which 10 involved delivery and care of newborns and half or more alleged oxygen deprivation. (Causation was a major element in dispute in many but not all of the cases.) Of the 15, six came out of the New York courts, including a trio of Brooklyn cases at $95 million, $90 million and $62 million. The great majority of cases settle within trial, and even in a case where the causation element is commanding a handsome settlement if it is fixed in a liberal jurisdiction and if doctors handling the delivery can be depicted as callous, confused or chaotic. Yet defensive medicine, including the skyscraping of Caesareans, has failed to lower cerebral-palsy rates.

The report by no means relieves delivery rooms of responsibility. It estimates that between 6% and 10% of surviving brain injuries originate in events during labor and delivery, and that of these perhaps half, amounting to 3% to 5%, might be preventable (which does not mean that the failure to prevent them implies negligence in any given case); in a larger subset of cases, perhaps another 25%, the handling of labor and delivery may influence the extent of damage in cases where pre-existing risk factors already spell trouble for a child. The report does not dispute that some high-risk obstetric brain damage cases rest on valid science.

The report did not mention plaintiff's lawyers quoted in the Boston Globe, whose reactions ranged from dismissive to furious. One attorney called the report "dangerous, intellectually indefensible, and morally irresponsible." "This is not a peer-reviewed medical research paper," another said. On the contrary, said Dr. Gary Hatch, chair of the task force that produced the report, it was "one of the most highly peer-reviewed reports ever." ACGO and AAP also lined up an all-star list of institutions to support the report's findings: it's been endorsed by the federal government's National Institute of Child Health and Human Development and Centers for Disease Control and Prevention, by professional groups from Canada, Australia and New Zealand, and by the March of Dimes. The United Cerebral Palsy Research and Education Foundation likewise "recommends the findings of the report." Its medical director says: All of which suggests that the movement for medical liability reform needs to rethink not only the size of verdicts but also their legitimacy. Cuts on legal fees and limiting awards of any at best limited help if doctors lose all confidence that the legal system will get medical facts right in the first place. Lackluster surgeons where the silicone implant debacle, or negligence about the attack on pedal bone bones.

"Certificate of need" laws requiring plaintiffs to line up an expert before filing suit rather than afterward probably do no good, as do evidence rules empowering judges to exclude cases scientifically defensible. False and unequitably restrictive rules for keeping citizens with medical expertise from being systematically excluded from jury service.

More ambitiously, we could take a leaf from other nations' practices by moving toward the use of experts recommend to the courts, rather than outdening experts hired by the parties, and by adopting lower pay to discourage long-shot cases. One needlessly injured child, as will rightly be pointed out, is too many. Isn't one falsely accused defendant also too many?

Mr. Olson, a senior fellow at the Manhattan Institute, is the author of the newly published "The Rule of Lawyers." Mr. Felt's Press.

This is the fifth in an occasional series.
cases, of which 10 involved de-
newborns and half or more
privation. (Causation was
dispute in many
ases.) Of the
of the
sion and if doc-
attorney and depicted as
MATERNITY
All of the meri-
liability the
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ight
some
filed in
on and if doc-
Certificate
William Bramhall
Justice

The lawsuit that damage could have given the mother in her one earlier or variations in a different or lower dosage—of causation theo-

the report “dangerous, intellectually indefensi-

ble, and morally irresponsible.” “This is not a peer-reviewed medical research paper,” claimed another. On the contrary, said Dr. Gary Hankins, chair of the task force that produced the report, it was “one of the most highly peer-reviewed reports ever.” ACOG and AAP also lined up an