

# **Newborn Neurological Injury and Litigation: Medical Legal Challenges to Causation**

Richard C. Halpern  
Thomson Rogers  
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## **INTRODUCTION**

Newborn neurological injury due to trauma occurring in and around birth (peripartum) is often profoundly disabling. For affected infants and their families these injuries can be life-altering, resulting in unfathomable burdens. For health care professionals providing peripartum care these dreaded outcomes are immensely distressing.

Neurological injury in infants may result from idiopathic causes, antenatal causes, unpreventable causes and potentially avoidable causes. The objective in the medical legal context is to distinguish between potentially avoidable causes and all other causes.

Compensation for these profoundly injured children will be available through litigation only when it can be proved, on a balance of probabilities, that neurological injury was due to a potentially avoidable cause. This requires the plaintiff to prove that some form of intervention was required, by the applicable standard of care, and that intervention would have avoided some or all of the harm. This issue of causation is generally the most complex and controversial medical legal issue in birth trauma cases.

This paper will focus on the medical legal issue of causation in the context of newborn neurological injury caused by peripartum events or conditions. To appreciate the potential for contribution of peripartum conditions to poor neurological outcomes it is necessary to consider the potential impact of all other causes of neonatal neurological injury, and, on a balance of probabilities, rule those out. All of the available medical data must be evaluated together to arrive at the most likely diagnosis. It will be argued that the key to this exercise is the proper use of the differential diagnosis. It will be also argued that, generally speaking, obstetrics plays a relatively minor role in the diagnostic process.

In exploring the difficult causation issues in birth trauma cases, this paper will consider the current state of some of the important medical literature affecting causation as well as the recent development of medical views affecting this vital issue. The issues will be examined primarily in the setting of peripartum asphyxia of the term fetus.

## **The Legal Context**

Legal claims for damages resulting from neurological injury caused by peripartum obstetrical negligence result in some of the largest damages awards in all of personal injury litigation. Impaired fetal oxygenation during labour can cause brain injury resulting in profoundly disabling cerebral palsy (CP). Affected children may have physical and cognitive impairments that prevent them from ever working or functioning independently. Frequently these children are entirely dependent on others for all aspects of their lifetime care and supervision. Where affected children can be expected to live for many decades, the assessment of damages easily climbs into the millions of dollars. Though these claims are relatively infrequent, they represent a significant portion of the damages paid out in medical malpractice litigation.

From the perspective of the families of affected children, these are complex and expensive cases to pursue. They face difficult challenges proving both a breach of the standard of care and causation. Standard of care is the first hurdle. The plaintiff must prove, on a balance of probabilities, that the health care providers (usually physicians and nurses) failed to provide the appropriate level of care in the circumstances. Standard of care challenges are formidable in many of these cases, but a detailed review of standard of care is beyond the scope of this paper. Though standard of care will not be analyzed, it is important to note that the plaintiff must prove that any intervention required by the standard of care was required at a time before the injury occurred. Determining when *in utero* insults evolve into irreversible neurologic injury is another formidable challenge.

The sheer size of these claims has understandably provoked responses from the medical community concerned with avoiding liability for these devastating outcomes. While it is undoubtedly true that many cases of CP are unavoidable, it is equally true that some, though perhaps few, are indeed avoidable. It is an entirely legitimate exercise to ensure that liability is not imposed where it is unjustified based on the best medical knowledge available. The corollary is that when the application of the best medical knowledge available establishes liability, on a balance of probabilities, it would be unjust to deny compensation to injured infants and their families.

An unfortunate development arising out of birth trauma litigation is the response of the medical community through various professional associations, including the Society of Obstetricians and Gynecologists of Canada (SOGC) and the American College of Obstetricians and Gynecologists (ACOG). Consultation and

research spearheaded by these and other organizations has resulted in the publication of what have been loosely described as “guidelines” by both SOGC and ACOG which have been seen by many medical practitioners as the definitive statements on causation relating to neurological injury and peripartum Asphyxia. These organizations have purported to issue the definitive statement on whether neonatal neurologic injury can be linked to intrapartum asphyxia. The “guidelines” are in fact clinical medical criteria that these organizations allege must exist to make the causal link between neurologic injury and intrapartum asphyxia. They are not obstetrical guidelines at all as they do not instruct obstetricians on the management of labour and delivery. Indeed these criteria are largely outside the scope of obstetrical practice, and belong to the disciplines of neonatology, pediatrics, neurology and radiology.

More recently, many in the medical community have challenged the reliability of the ACOG and SOGC criteria and view them as providing an unduly high, and perhaps even medically inappropriate, threshold for proof. This paper argues that the criteria are not supportable on the available medical knowledge and, therefore, represent unjustifiable and unwarranted obstacles to equitable compensation for children with neurological injury caused by negligent medical care. As well, it will be argued that the criteria were not reliably based on a firm medical foundation supported by the science at the time they were created. Indeed, from the perspective of promoting better health care, the guidelines can be viewed as obstacles to improved care.

It is fair to say the both ACOG and SOGC are generally considered to be reputable and respected organizations. Guidelines from both organizations are published regularly and instruct physicians throughout the United States and Canada on the standards of obstetrical care. Consequently, standards of obstetrical practice, and in particular legal obstetrical standards of care, are likely to conform to ACOG and SOGC recommendations. Guidelines issued from these organizations are commonly accepted as authoritative and adopted by medical practitioners, directly affecting all the patients they treat. As such, it is submitted that ACOG and SOGC owe an obligation to more than just the members of these associations, but also to the patients they serve. The causation criteria do not guide obstetrical practice. Have ACOG and SOGC met their moral obligations in setting out the criteria for linking neurologic injury to peripartum asphyxia? Have they exceeded what would objectively be considered the appropriate scope of a mandate for an organization of obstetricians?

If there is any merit to the criticisms of the criteria contained in this paper and the medical literature that suggests deficiencies in the criteria, then any deference paid by courts to the criteria is unfortunate indeed for affected children. The final section of this paper will examine some of the ways the courts have approached the causation criteria.

## CAUSATION AND THE CRITERIA

### Medical and Legal Objectives Relating to Causation

Birth trauma cases resulting in neurologic injury typically involve allegations that intervention to deliver the baby ought to have occurred sooner based on the available clinical evidence, usually periodic changes in the fetal heart rate pattern and other available clinical evidence. Injuries resulting from the failure to respond appropriately are compensable only when the plaintiff can prove that earlier intervention would have made a difference to the outcome. Therefore, to the extent the neurologic injury was caused solely by events that occurred before intervention was warranted, the claim cannot succeed and no damages will be awarded.

As described below, the legal burden facing the plaintiff is a “balance of probabilities” or “more likely than not”. This must be contrasted with scientific standards that may demand a stricter threshold of proof. It is essential when medical experts opine on causation issues that they do so with the legal burden of proof in mind. This requirement alone may undermine the use of the causation criteria discussed below.

Hypoxia-ischemia (referred to as “asphyxia”) is an intrapartum condition that can lead to brain damage. Neonatal encephalopathy (NE)<sup>1</sup>, represented by depression or disturbed neurological function at and around the time of birth, can be the result of asphyxia or can be caused by other antenatal or perinatal conditions. Therefore NE may or may not be related to asphyxia.<sup>2</sup> The challenge in these cases is to distinguish between asphyxic and non-asphyxic causes for NE and, if asphyxic, whether the NE caused neurologic injury.

Much of the published medical literature on the subject, particularly as it relates to cerebral palsy, describe the rarity with which neurologic injury arises from asphyxia. Cerebral palsy arising out of intrapartum events is rare. Antenatal conditions can lead to neurologic injury as well, and perhaps more commonly. There are many conditions other than asphyxia that account for neonatal neurologic injury. Despite this, the fact is that some neonatal brain injury is in fact caused by asphyxia, and data regarding the relative infrequency should not be allowed to obfuscate this fact. This is the case for brain injury causing cerebral palsy, as well as for brain injury causing neurocognitive deficits without any motor involvement.

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<sup>1</sup> In 1976 Sarnat and Sarnat developed a method to score neonatal depression based on level of consciousness, tone, reflexes, autonomic function, seizures and EEG findings. See Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress: A clinical and electroencephalographic study. Arch Neurol 1976;33:696-705.

<sup>2</sup> The SOGC in the 2007 Consensus Statement, page S25, maintains that 70% of cases of NE occur prior to the onset of labour secondary to things like prenatal stroke, infection, cerebral malformation and genetic disorders.

Simply put, some cases of neonatal neurologic injury are due to substandard care provided during labour and are preventable. In recognition of this fact, the objective of the medical community should be to identify the circumstances that allow these occasional adverse outcomes; to take steps to prevent them from happening; and, to develop and promote treatments that might mitigate the harm caused by asphyxia. From the legal perspective, the objectives include: access to justice; accountability; deterring harmful behaviour; and the appropriate allocation of loss.

### **Obstacles to Proving Causation**

Unfortunately not all participants view the objectives in the same way. The sheer size of birth trauma lawsuits has provided considerable impetus to develop ways to avoid liability. The problem takes on far larger importance in the United States than it does in Canada. From time to time medical malpractice litigation in the U.S. has been described as being in "crisis". While it is beyond the scope of this paper to address this issue, suffice it to say that there is no medical malpractice crisis in Canada. In fact, the statistics published by the Canadian Medical Protective Association demonstrate a reduction in the number of claims against physicians over the last decade. The extent to which the perception of a crisis has influenced causation in birth trauma cases is certainly a matter for debate.

Current causation criteria from SOGC and ACOG came about following the publication of the International Consensus Statement by MacLennan in 1999 (the "International Statement"). This was followed by a 2003 publication from ACOG (the "ACOG Criteria" or the "green book") and a 2002 SOGC publication (the "SOGC Criteria "). Each set of criteria will be considered and analyzed in detail. As well, the 3 separate guidelines will be compared and any differences discussed. Finally, the medical and logical foundation for each guideline will be critically explored.

### **The International Consensus Statement**

Alastair MacLennan is the chair of the International Cerebral Palsy Task Force funded by the Perinatal Society of Australia and New Zealand.<sup>3</sup> It was this task force that developed the International Criteria. Before examining the specific criteria, some of the background contained in the International Consensus Statement<sup>4</sup> will be reviewed.

At the outset it is absolutely crucial to note that the International Statement is concerned with CP and determining when the neuropathology supports damage

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<sup>3</sup> It should be noted that supporters of the International Criteria include ACOG and SOGC.

<sup>4</sup> MacLennan A, for the International Cerebral Palsy Task Force. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ* 1999;319:1054-9.

occurring at or around the time of labour and delivery. CP is a non-progressive neurological condition that includes motor dysfunction. The review is also primarily concerned with term gestation. Significantly, it fails to adequately consider the potential for neurological injury that does not involve motor impairment. Despite this, many experts have adopted this and other criteria to incorrectly suggest that in the absence of CP, neonatal neurologic injury can never be linked to intrapartum asphyxia.

The MacLennan publication is a “consensus statement” with consensus reached amongst the associations supporting the report, from a number of disciplines. Significantly, the supporters could not reach consensus on all issues, including the value of neuroimaging in determining the timing and cause of abnormalities seen on imaging.

An important, though half-hearted, concession in the MacLennan report is the fact that the task force acknowledged that “some cases of cerebral palsy probably originate in labour”. It seems almost absurd to modify this statement with the word “probably”. This might betray a bias and highlights the underlying medical-legal purpose of the document. It is difficult to imagine any physician credibly maintaining the possibility that cases of cerebral palsy never originate in labour. To say that neurologic injury can in fact originate in labour is trite.

The International Statement says that “intrapartum complications play an infrequent role in the causation of cerebral palsy”.<sup>5</sup> Acknowledging that intrapartum complications are contributors to CP, albeit infrequent ones, is to concede that they are, nevertheless, contributors on at least some occasions.

Limitations on the ability to assess fetal well-being *in utero* are, in part, the reason for many of the challenges to establishing the cause of neonatal neurologic injury. In the context of timing, the International Statement describes some of the difficulties related to timing. As the statement points out, peripartum hypoxia is a progressive process resulting in a gradually increasing hypoxemia and hypercapnia as well as a developing metabolic acidosis. This is an important consideration when examining the entire clinical setting and the potential for peripartum asphyxia to affect neonatal encephalopathy (NE). In considering timing it must be noted that NE does not establish causation and that hypoxic-ischemic encephalopathy (HIE), a subset of NE, must be established clinically to support the conclusion that peripartum Asphyxia is implicated in the brain injury.

According to the International Statement, there are three essential criteria necessary before acute intrapartum hypoxia can be considered as a possible cause of cerebral palsy:<sup>6</sup>

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<sup>5</sup> Page 1056.

<sup>6</sup> page 1056

1. Evidence of a metabolic acidosis in intrapartum fetal umbilical arterial cord or very early neonatal blood samples (pH <7.00 and base deficit  $\geq$  12 mmol/L);
2. Early onset of severe or moderate neonatal encephalopathy in infants of  $\geq$  34 weeks gestation;
3. Cerebral Palsy of the spastic quadriplegic or dyskinetic type;

Criteria that together suggest an intrapartum timing but by themselves are not specific:

4. A sentinel (signal) hypoxic event occurring immediately before or during labour;
5. a sudden, rapid and sustained deterioration of the fetal heart rate pattern usually after the hypoxic sentinel event where the pattern was previously normal;
6. Apgar scores of 0-6 for longer than 5 minutes;
7. Early evidence of multisystem involvement;
8. Early imaging evidence of acute cerebral abnormality.

The statement contends that if evidence for some of criteria 4 to 8 is missing or contradictory, the timing of the onset of the neuropathology becomes increasingly in doubt based on the assertion that these criteria are only weakly associated with an acute intrapartum damaging hypoxic event. Without any scientific evidence or justification, the statement also asserts that logically most of the final 5 criteria would have to be present for the balance of probabilities to suggest an acute timing of the hypoxic event.<sup>7</sup> With respect, this is careless and unscientific. There is nothing logical about that conclusion in the absence of reliable supporting scientific literature. Once again, this statement highlights the rather shaky medical foundation upon which the criteria are based.

The International Statement provides commentary on each of the criteria. In important ways, some of that commentary is of questionable scientific validity today, and may well have been at the time. A few remarks will be offered with regard to the commentary.

On the matter of the requirement for metabolic acidaemia, the statement contends that damaging intrapartum asphyxia cannot even be postulated unless metabolic acidaemia is present.<sup>8</sup> Without canvassing the medical literature that suggests some controversy on this point, the statement has failed to consider whether intrapartum insult can occur and be mitigated somewhat *in utero* through resuscitative measures before birth. In other words, if the cause of a developing metabolic acidosis is reversed (say by discontinuing oxytocin contributing to tachysystole) then there might be some improvement in the pH and base deficit.

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<sup>7</sup> See page 1056.

<sup>8</sup> Page 1056.

In the presence of a prolonged partial asphyxia, the fetus develops a progressive acidosis. There are steps that the health care provider can take during labour to resuscitate the fetus *in utero* when faced with potential of evidence of hypoxia on the fetal heart tracing. To the extent that such resuscitative efforts are successful, a developing acidosis would likely be mitigated to some degree. While there is no reliable scientific data to measure this phenomenon, many clinicians would support this conclusion. Cord blood gas analysis would reflect fetal status just prior to delivery and may not account for the effect of *in utero* resuscitation. Although in the vast majority of clinical settings one would anticipate delivery taking place around the peak of metabolic acidosis, there may be exceptions for prior intrauterine resuscitation. Strict application of the requirement for a severe metabolic acidosis, therefore, has its exceptions.

Interestingly, the International statement contends that if there is no umbilical arterial blood gas analysis to establish an offending level of base deficit then “it is not possible to say that hypoxia or asphyxia caused or contributed to the other clinical signs”.<sup>9</sup> This seems an absurd statement given the accepted way in which the differential diagnosis must be applied to the question of causation. In fact, it is astounding that the differential diagnosis is not part of the International Statement, as it is part of the ACOG criteria. Surely the failure of the nurse or doctor to obtain umbilical cord blood gas samples and perform the analysis cannot preclude a diagnosis. One must still look to and evaluate the best clinical evidence available to come to the most likely cause.

While the International Statement posits that a base deficit of 16 mmol/L is “a realistic cut off point for defining pathological fetal acidaemia that correlates with an increasing risk of neurological deficit”<sup>10</sup>, the criteria adopts 12 mmol/L as the cut off. Note should be made of the use of the words “increasing risk”, which suggest that there is nevertheless risk at a level under 16 mmol/L.

With respect to the need for NE, the statement underplays its importance in relation to asphyxia. MacLennan points out that moderate to severe NE is uncommon following a non-reassuring fetal heart tracing and that many cases of severe NE are not associated with intrapartum asphyxia.<sup>11</sup> The corollary to these remarks is that moderate to severe NE can follow a non-reassuring fetal heart tracing and that some cases of severe NE are associated with intrapartum asphyxia. Had the International statement wanted to maintain some degree of objectivity with regard to the first proposition they could have said: “Moderate to severe NE can be associated with non-reassuring fetal heart tracings, though this occurs relatively rarely”.

The fact that NE can be caused by many conditions other than intrapartum asphyxia does not diminish NE as one of the links in the chain to establishing

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<sup>9</sup> Page 1056.

<sup>10</sup> See page 1056.

<sup>11</sup> See page 1057.

causation. Despite the multiple causes for NE, it just so happens that intrapartum asphyxia is one of the causes of NE. A key to determining the likelihood that NE is caused by intrapartum asphyxia are periodic changes on the fetal heart tracing compatible with hypoxia. A fetus with a developing metabolic acidosis will always show changes on the tracing compatible with hypoxia. When the value of neuroimaging is added to the clinical evidence of NE and earlier abnormal tracings, a strong causal connection starts to emerge.

With regard to CP, the International Statement concludes that only quadriplegic and dyskinetic CP is associated with acute hypoxic intrapartum events.<sup>12</sup> The Statement goes on to contend that intellectual disability, autism and learning disorders in a child without spasticity are not associated with acute intrapartum asphyxia.<sup>13</sup> No evidence is offered in support of this assertion. Current evidence, discussed further below, suggests that this contention is unfounded. Further, it is indeed unfortunate that the Statement would go so far in the absence of reasonable justification. These unfounded assertions have very important adverse impacts on affected patients.

The International Statement diminishes the importance of fetal heart rate monitoring. The discussion regarding fetal heart rate seems contradictory. On the one hand, it is suggested that non-reassuring fetal heart rate patterns are not useful in predicting CP given a 99.8% false positive rate, yet the Statement supports early delivery where fetal heart rate patterns suggest potential severe fetal compromise.<sup>14</sup> It is widely recognized that certain fetal heart rate patterns suggest possible hypoxia. Suggesting that intervention not take place until there is potential “severe” fetal compromise seems patently unreasonable and unsafe. The objective of fetal surveillance must be the recognition of potential compromise and need for intervention before irreversible harm of any nature. As well, the fetal heart rate changes really need to be assessed in the entire clinical context and not in isolation. The treatment of fetal heart rate monitoring by this task force, ACOG and SOGC is an entirely different subject matter for a different paper.

With regard to neuroimaging, the fact that cerebral edema early in the neonatal period suggests recent insult is acknowledged by the International Statement.<sup>15</sup> The Statement also notes the difficulty in using imaging to time the insult. The statement, however, fails to recognize the importance of neuroimaging as a tool in the diagnostic process. Admittedly advances in neuroimaging since 1999 have enhanced its value, but imaging was still a vital piece of the diagnostic puzzle when the Statement was published.

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<sup>12</sup> Page 1057.

<sup>13</sup> Page 1057.

<sup>14</sup> Page 1057.

<sup>15</sup> Page 1058.

The Statement sets out conditions, other than asphyxia, that might cause cerebral palsy.<sup>16</sup> These possible antenatal causes of neurological impairment, reduce but do not necessarily eliminate the likelihood that acute intrapartum hypoxia caused or contributed to the impairment. They include:

1. Umbilical artery base deficit less than 12 mmol/L or pH higher than 7.00
2. Congenital or metabolic abnormalities
3. Systemic infections
4. Longstanding neurological abnormalities evident on early imaging
5. IUGR
6. Reduced FHR variability from the onset of labour
7. Microcephaly
8. Antenatal placental abruption
9. Congenital coagulation disorders
10. Other antenatal risk factors (preterm birth, multiple gestation, autoimmune disease)
11. Presence of major postnatal risk factors for cerebral palsy
12. A sibling with the same type of CP

Again this is really the process of differential diagnosis. What the Statement fails to say is that the balance of probabilities likely favours intrapartum asphyxia as the cause for neurological injury in the absence of these other causative conditions. Further, the contribution of antenatal causes to later neurologic injury may be less common than the Statement suggests. In his text, *Neurology of the Newborn*, Volpe states:

Although hypoxic-ischemic injury certainly can occur in the antepartum period, this injury cumulatively accounts for only a small proportion of neonatal hypoxic-ischemic encephalopathy. However, antepartum factors may *predispose* to intrapartum hypoxia-ischemia during the stresses of labour and delivery, especially through threats to placental flow.<sup>17</sup>

A publication like the International Consensus Statement needs to be considered in terms of its value to medicine and its impact on legal proceedings. The statement is problematic on both accounts. Admittedly, the comments in this paper are offered without a shred of medical training or experience. On the other hand it seems that the Statement makes bold assertions about the seemingly insurmountable chore of linking neurologic injury to asphyxia with little in the way of medical evidence in support of its conclusions. At the same time, I am not aware of any attempt by the Statement's authors to modify the Statement in view of medical developments since its publication.

From the legal perspective, the Statement simply has it wrong. For example, the Statement asserts:

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<sup>16</sup> page 1058

<sup>17</sup> See Volpe page 401.

It is not possible to ascertain retrospectively whether earlier obstetric intervention could have prevented cerebral damage in any individual case where no detectable sentinel hypoxic event occurred.<sup>18</sup>

First, the occurrence of a sentinel event is not one of the 3 “essential” criteria, but is listed as a non-specific criteria. Second, in describing what constitutes a sentinel event the Statement lists ruptured uterus; placental abruption; cord prolapse; amniotic fluid embolism; and, fetal exsanguination from vasa previa or fetal-maternal haemorrhage. It is important to note that the described list is not exhaustive of all intrauterine conditions that cause asphyxia. Cord compression and other conditions leading to placental insufficiency will not manifest clinically in the same way.

There are generally two types of brain injury patterns arising out of intrapartum asphyxia.<sup>19</sup> The basal ganglia-thalamus pattern (BGT) is the one most often seen following an acute sentinel event. It is also the pattern more likely to follow an acute near-total asphyxia. The watershed predominant pattern (WS) is the pattern that commonly follows prolonged partial asphyxia. The criteria fail to point out this distinction when indicating the requirement for a sentinel event.

Incredibly the Statement goes on to claim that an “intrapartum hypoxic event can be silent”. There is no medical evidence cited in support of this proposition and there is no indication of what “silent” means. Despite this comment, intrapartum hypoxia leading to a developing metabolic acidosis and subsequent neurological injury is never “silent”. The unfriendly intrauterine environment that leads to these developments is one where the fetus will be sure to express some signs of discomfort through periodic changes in the fetal heart rate. It is only the antepartum conditions that cause neurologic injury that might be silent in the intrapartum period (and often they are not silent). Indeed, fetal displeasure with potential asphyxia will inevitably be communicated through periodic changes in the fetal heart rate and the likely timing of irreversible insult is often most reliably revealed by analyzing the tracing, in conjunction with other intrapartum and neonatal clinical data. The role of the obstetrical team is to appreciate when the pattern suggests possible exhaustion of fetal reserves and when the amazing capacity of the fetus to compensate for hypoxia turns into decompensation.

If the International Consensus Statement was intended to set out the state of the medical knowledge on the link between intrapartum asphyxia and CP then one wonders why it was necessary to conclude with recommendations about who should act as expert witnesses in cerebral palsy cases. I suggest that this document was motivated in large part to address medical-legal issues and set out the best defence available to those named as defendants in these cases.

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<sup>18</sup> page 1058.

<sup>19</sup> See de Vries LS. Patterns of neonatal hypoxic-ischaemic brain injury. *Neuroradiology* (2010) 52:555-566.

The International Consensus statement is lacking in objectivity and firm medical foundation.

### **The ACOG Criteria**

The American College of Obstetricians and Gynecologists set out its criteria on the link between intrapartum asphyxia and cerebral palsy in a 2003 publication called Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. This publication will occasionally be referred to as the “green book”.

ACOG offers 9 criteria for making a connection between CP<sup>20</sup> and intrapartum causes, 4 of which are “essential” and 5 “non-essential”. By essential, ACOG is saying that all of the 4 essential criteria must be present. If any one is absent, the connection between CP and intrapartum events cannot be established. This section looks at the data used by ACOG to support the putative essential criteria.

The ACOG criteria to define an acute intrapartum event sufficient to cause cerebral palsy, as modified by this Task Force from the template provided by the International Cerebral Palsy Task Force, are listed as follows:

#### Essential criteria (must meet all four)

1. Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit  $\geq$  12 mmol/L);
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks gestation;
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type;<sup>21</sup>
4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders.

Criteria that collectively suggest intrapartum timing (within close proximity to labor and delivery, eg, 0-48 hours) but are nonspecific to asphyxial insults:

5. A sentinel (signal) hypoxic event occurring immediately before or during labour;
6. 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 a hypoxic sentinel event when the pattern was previously normal;
7. Apgar scores of 0-3 beyond 5 minutes;
8. Onset of multisystem involvement within 72 hours of birth; and

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<sup>20</sup> Do they mean CP only or any neurological injury?

<sup>21</sup> They rely on Nelson, KB, Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. Am J Obstet Gynecol 1998; 179; 507-13.

9. Early imaging study showing evidence of acute nonfocal abnormality.

The ACOG criteria and International criteria differ in some important ways. First, ACOG has recognized the importance of the differential diagnosis in criterion 4. It is submitted that the differential diagnosis is in fact the only “essential” criteria that should be applied to the causation analysis, the other criteria being instructive, or even persuasive, but never determinative. It is not really, however, a criterion at all but rather an essential diagnostic process. Second, the International criteria set out a threshold for an Apgar score of 6, while ACOG has seen fit to lower it to 3, making for a more stringent diagnostic criterion.

Before looking at the foundational support for the ACOG criteria it is necessary to raise a matter that is unclear from the publication. It seems that ACOG's essential criteria might apply only to cerebral palsy as an outcome arising from intrapartum asphyxia rather than all forms of neurological injury, including those that do not result in motor deficits. Having said that, there would appear to be a number of inconsistencies in the document that raise doubts about whether the criteria should be interpreted that narrowly.

In its forward<sup>22</sup> the green book makes reference to the relationship between severe metabolic acidosis and “a type of cerebral palsy that could have been caused by hypoxia”. Later the document describes the types of CP<sup>23</sup> that can be caused by hypoxia, but is this intended to suggest that other neurological injury cannot be related to intrapartum asphyxia? The green book states:

...absent cerebral palsy, neither epilepsy, mental retardation, nor attention-deficit hyperactivity disorder are caused by birth asphyxia.<sup>24</sup>

It is clear, however, that neurologic damage, such as isolated mental retardation, attention deficit disorder, or seizure disorder, cannot be attributed to birth asphyxia in the absence of newborn encephalopathy.<sup>25</sup>

The green book acknowledges indirectly that there are infants with mild to moderate neonatal encephalopathy who do not develop normally, and is explicit

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<sup>22</sup> P. xii

<sup>23</sup> The task force maintains that spastic quadriplegia, especially associated with movement disorder, is the only type of CP associated with an acute interruption of blood supply. Purely dyskinetic or ataxic CP is not caused by peripartum asphyxia and usually has a genetic origin according to the report. See p. xvii.

<sup>24</sup> P. xvii

<sup>25</sup> p. 2

in saying that infants with severe NE are more likely to sustain long-term neurological morbidity,<sup>26</sup> a vague statement that may or may not incorporate CP.

The two statements above are quite different. The first suggests that in the absence of CP, the noted conditions cannot be attributed to birth asphyxia. The second suggests that they can be attributed to birth asphyxia provided NE is present. These statements are potentially contradictory. Requiring NE and CP to connect neurological injury to intrapartum asphyxia is quite different than requiring NE to connect either CP or the other conditions to intrapartum asphyxia.

The task force also states:

“The full range of impairment following an unbiased assessment of neonatal encephalopathy and its subset HIE has not been well-established in a recent and large population-based study. Long-term follow-up studies of children enrolled in large, population-based studies...are needed.”<sup>27</sup>

Based on this statement, the task force should not be seen to suggest that impairments without CP cannot be caused by intrapartum asphyxia. Having said that, experience has shown that many medical experts have interpreted the criteria to suggest that, absent CP, there can be no link between neurologic injury and intrapartum asphyxia. There is no reliable data to support this proposition.

It should also be observed that the Task Force’s objective was really to “consider the current state of scientific knowledge about the mechanisms and timing of possible etiologic events which may result in neonatal encephalopathy”.<sup>28</sup> This objective was not confined to CP, as the ACOG report seems to be. ACOG seems to have neglected to adequately canvas the mechanisms that give rise to NE that result in non-motor brain dysfunction.

The task force maintains “with certainty” that intrapartum hypoxia-ischemia leading to CP “must” progress through NE.<sup>29</sup> They neglect to say whether neurological injury leading to non-motor brain injury must likewise do so, though presumably they would draw that conclusion. In other words, the absence of NE indicates a cause remote to the intrapartum period as an explanation for subsequent neurologic injury. The presence of NE is supportive of an intrapartum cause for later neurological injury, but other causes can also give rise to NE. The task is then to determine whether the NE is caused by hypoxic-ischemic encephalopathy related to intrapartum events or by other, perhaps more common, causes.

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<sup>26</sup> p. 6

<sup>27</sup> p. 7

<sup>28</sup> p. xiii

<sup>29</sup> p. xvii

Clearly the medicine around intrapartum asphyxia and neurological injury has evolved over time, particularly due to advances in neuro-imaging. In examining the validity, relevance and reliability of the ACOG criteria, one must do so in this context. If, as argued in this paper, the ACOG criteria should not be relied upon for their stated purpose, the question becomes whether the ACOG criteria were based on the best medicine at the time and are no longer valid or whether the criteria were flawed from the outset. It is submitted that the ACOG criteria were in many ways seriously flawed when published and subsequent medical research and knowledge has further undermined many of ACOG's conclusions.

The 3<sup>rd</sup> essential criterion from ACOG is “cerebral palsy of the spastic quadriplegic or dyskinetic type”. The task force states:

Spastic quadriplegia and, less commonly, dyskinetic cerebral palsy are the only types of cerebral palsy associated with acute hypoxic intrapartum events. Spastic quadriplegia is not specific to intrapartum hypoxia. Hemiparetic cerebral palsy, hemiplegic cerebral palsy, spastic diplegia, and ataxia are unlikely to result from acute intrapartum hypoxia.

In support of this statement the task force relied on a medical journal article from Nelson published in 1998 (the “1998 Nelson article”).<sup>30</sup> The International Consensus statement has done likewise. In connection with the statement quoted above, the following questions need to be explored:

- 1) Is spastic quadriplegia the only type of CP associated with intrapartum asphyxia?
- 2) Are there neurological injuries without motor dysfunction (not CP) that can be caused by intrapartum asphyxia?
- 3) Does the 1998 Nelson article support the task force conclusion that epilepsy, mental retardation and attention deficit hyperactivity disorder cannot be related to birth asphyxia?
- 4) If the 1998 Nelson article does not support the task force conclusion, what is the evidence in support of that conclusion?
- 5) Did the task force intend to confine this criterion to term babies?<sup>31</sup>
- 6) Is there any scientific support for this criterion apart from the 1998 Nelson article?
- 7) Have subsequent developments in neuroimaging and other studies brought this requirement into question?
- 8) If there is new data, when did it become available and what has ACOG done in response?

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<sup>30</sup> See page xviii. The article is Nelson KB, Grether JK. Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. *Am J Obstet Gynecol* 1998; 179:507-13.

<sup>31</sup> The report does say at page 1 that the document is focused on term (greater than 37 weeks gestation) and near-term (greater than 34 weeks gestation) infants.

- 9) Was the data used sufficient to draw conclusions regarding causation that justified carving out strict, mandatory criteria?

The green book also refers to the fact that most cases of cerebral palsy are related to antepartum factors and not to isolated intrapartum events.<sup>32</sup> The report lists antepartum conditions associated with CP, including preterm birth; intrauterine infections; intrauterine growth restriction; multiple pregnancies; coagulation disorders; antepartum bleeding; congenital or genetic anomalies; and infertility treatment. It is revealing that the green book pays little attention to how the diagnostic challenge is to be dealt with when the best clinical evidence shows a complete absence of these other conditions that might lead to CP. Obviously this is a clinical scenario encountered by medical practitioners from time to time. The green book would have better served the medical community and patients if it had gone on then to look at the best clinical evidence available to reveal a diagnosis: likely the fetal heart tracing, neuroimaging, HIE, etc. However, to do so would be to explicitly acknowledge that in the absence of antenatal causes, intrapartum asphyxia should be very high on the index of suspicion.

Ultimately the issue concerns the differential diagnosis. In the absence of evidence of antenatal conditions that might cause or contribute to neonatal brain damage, clinical intrapartum evidence of possible hypoxia together with evidence in the neonatal period of neurological compromise (neonatal encephalopathy) will strongly suggest an intrapartum cause. Neuroimaging will most reliably confirm the diagnosis. Timing will be best determined by evaluating the intrapartum clinical evidence (fetal heart tracings, uterine contraction patterns, etc) in the context of the imaging.

The importance of neuroimaging cannot be overstated in making the connection between neonatal brain injury and intrapartum events. The process begins with compromised cerebral perfusion leading to the shunting of blood from the cerebral cortex to the deeper structures of the brain. Crucially, evidence of cerebral edema developing in the first days of neonatal life in a background of an uneventful pregnancy should be seen as compelling evidence for a brain insult occurring at or near the time of birth.

The green book includes 4 essential criteria. The 4<sup>th</sup> is not really a criterion at all, but rather the process that one needs to apply to make a diagnosis. The 3<sup>rd</sup> essential criterion is CP of the spastic or dyskinetic type. The green book cites an article by Nelson in support of the CP criterion.<sup>33</sup> The Nelson article does not support this “essential” criterion as is suggested in the green book.

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<sup>32</sup> Page 25

<sup>33</sup> page 74. Nelson, Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. *Am J Obstet Gynecol* 1998;179:507-13.

The green book also relies on an article by Rosenbloom 1994 and an article by Stanley, to be reviewed below.

The objective of the Nelson article “was to examine the association of cerebral palsy with conditions that can interrupt oxygen supply to the fetus as a primary pathogenic event”. It is clear that the cohort studied consisted of children with spastic quadriplegia. Crucially, children exposed to potentially asphyxiating events that went on to suffer non-motor neurological deficits were not part of the study. It follows that no conclusions can be drawn with respect to the connection, if any, between intrapartum asphyxia and later non-motor neurological deficits.

All the children in the Nelson study had a birth weight greater than or equal to 2500g, survived to age 3 and had moderate or severe cerebral palsy.<sup>34</sup> The control group did not have cerebral palsy. Therefore, the only comparison made was between children with or without CP that qualified by birth weight and age.

The study defined “Potentially Birth-Asphyxiating Events” to include abruptio placentae, placenta previa, large placental infarctions, prolapsed cord, cord compression, maternal shock, true cord knot, or tight nuchal cord. Out of a total population of 155,636 children, there were 46 children suffering from unexplained cerebral palsy. Unexplained cerebral palsy is CP that did not result from brain malformation, prenatal infarction or congenital nonbacterial infection.<sup>35</sup> There were 378 controls.

The study found that 39% of children with unexplained CP had tight nuchal cord, while 19% of controls did as well. Eight CP kids had tight nuchal cord, while 15 controls had tight nuchal cords. For some reason, that data is not expressed in percentage terms in the study. It is a rather significant finding which appears to point to intrapartum asphyxia associated with tight nuchal cord in a number of children with CP. The fact that some controls experience tight nuchal cord without CP does not tell us anything about any later neurologic compromise. Data comparing metabolic acidosis amongst all the children with tight nuchal cord would probably reveal some interesting findings.

With respect to intrapartum factors, the study notes that 61% of children with unexplained CP and 29% of controls had periodic changes in the fetal heart rate during labour associated with hypoxia.<sup>36</sup> The comments in the report about fetal heart rate monitoring abnormalities are not particularly helpful. There is no qualitative assessment of the abnormalities in the cohort and no way to assess the impact on fetal well-being to allow comparison. One interesting finding from the study is that neonatal seizures were only seen in children with CP.<sup>37</sup> Another

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<sup>34</sup> Nelson page 508

<sup>35</sup> Nelson page 509.

<sup>36</sup> Nelson page 509.

<sup>37</sup> Nelson page 510.

interesting finding is the apparent association between tight nuchal cord and unexplained CP.<sup>38</sup>

The study did not reveal any association between potentially asphyxiating events and spastic hemiplegia or diplegia. From that it was concluded that with respect to children with CP, it is CP of the spastic quadriplegic type that occurs with the potentially asphyxiating events. The study specifically states that “there was no observed association of potentially asphyxiating conditions with spastic hemiplegia or diplegia”. Can the absence of such an association in this one study of 48 infants reliably stand for the proposition that there is no such link? In this same study there were no cases of CP due to uterine rupture, but presumably the authors would concede that uterine rupture could result in CP. It is conceded that the study has certain limitations, particularly given the small numbers of children with these low prevalence outcomes.<sup>39</sup> More fundamentally, a study of this nature should not be relied on as establishing definitive guidelines relating to causation in birth trauma cases insofar as neurologic injury and asphyxia are concerned.

The Nelson study also notes that neonatal markers of illness (which presumably means neonatal encephalopathy) were present in most kids with unexplained CP but were scarce in controls.<sup>40</sup> Having found that, the study also observed that of children with quadriplegia, neonatal encephalopathy was no more common in those quadriplegic children who had potentially asphyxiating conditions than in those without potentially asphyxiating conditions. This is likely a short-coming of the study rather than an important finding relating to the relationship between quadriplegia and neonatal encephalopathy. Undoubtedly, careful analysis of blood gas base deficits and neuroimaging would shed more light on this discussion. The study acknowledges that neonatal encephalopathy may be associated with non-asphyxiating disorders. The study also did not analyze how co-morbidities interact.

The Rosenbloom<sup>41</sup> article does not support the contention that CP must result from brain injury induced by intrapartum asphyxia. That article examined the connection between birth asphyxia and dyskinetic CP.<sup>42</sup> In other words, all the patients studied in fact had CP. Thus, one cannot conclude from this study that the absence of CP disposes of any connection between intrapartum asphyxia and non-motor neurological deficits.

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<sup>38</sup> Nelson page 511.

<sup>39</sup> Nelson page 512.

<sup>40</sup> Nelson, p. 512.

<sup>41</sup> Rosenbloom L. Dyskinetic Cerebral Palsy and Birth Asphyxia. *Development Medicine and Child Neurology*, 1994, 285-289.

<sup>42</sup> Rosenbloom page 3

The 1993 article by Stanley and others<sup>43</sup> reviews a case-control study that investigated the genetic and epidemiological patterns of a group of spastic quadriplegic children. Like the Nelson study and the Rosenbloom study, the cohort examined by Stanley only included children with CP. In other words, all three studies had pre-selected a cohort of children with CP and so that all the children had motor dysfunction. As a result, no conclusions can be drawn about the potential for neurologic injury caused by asphyxia without motor dysfunction. The study looked for, among other things, the “common antecedents which might give clues to causation and possible prevention”.<sup>44</sup> Among the hypotheses to be tested by the Stanley study were:

- a) That few individuals with moderate or severe spastic quadriplegia had neonatal encephalopathy compatible with birth asphyxia; and
- b) That if such encephalopathy had been present, it was more likely to have occurred in an already vulnerable infant.

One wonders if posing the hypotheses in this way betrays a bias of the study. There certainly was a way to express the same notions more objectively, letting the conclusions speak for themselves. The sample studied by Stanley was confined to children with quadriplegia. The study notes<sup>45</sup> that the proportion of cases of cerebral palsy due to intrapartum causes increased over time. It is speculated that this increase may be due to developments in neonatal intensive care that have allowed more neonates to survive, so that some who would have previously died are now surviving with cerebral palsy. Parenthetically, the fact that more babies are surviving, together with the relatively rare incidence of CP caused by intrapartum asphyxia, might explain why the wide use of electronic fetal heart rate monitoring has not measurably decreased the CP rate. In other words, it is not the efficacy of electronic fetal heart rate monitoring that is in issue, but rather extraneous factors that impact the statistical analysis. Again, the treatment of electronic fetal monitoring by ACOG and other associations merits further analysis.

MRI has been instrumental in identifying the heterogeneity of brain injury in the setting of NE, dependent on the duration and severity of the ischemia.<sup>46</sup> Work done by Miller<sup>47</sup> confirms that neonatal encephalopathy is not homogeneous and may result in cognitive deficits in the absence of CP. The evidence demonstrates that abnormal neurological outcome is not limited to CP. This

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<sup>43</sup> Stanley FJ, Blair E, Hockey A, Petterson B, Watson. Spastic Quadriplegia in Western Australia: A Genetic Epidemiological Study. I: Case Population and Perinatal Risk Factors. *Development Medicine and Child Neurology*, 1993; 35: 191-201.

<sup>44</sup> See page 191.

<sup>45</sup> See page 198.

<sup>46</sup> See Miller SP. Patterns of brain injury in term neonatal encephalopathy. *Journal of Pediatrics*, April 2005, page 453.

<sup>47</sup> See above. See also Miller SP. MRS Predictors of 30-month outcome following perinatal depression: role of socio-economic factors. *Pediatr Res* 2002;52:71-7.

should come as no surprise given the fact that there has been no reliable medical evidence to suggest the contrary.

In 2006 an article by Gonzalez and Miller<sup>48</sup> the authors concluded that there was increasing evidence to show that children surviving neonatal encephalopathy may have cognitive impairments without functional motor deficits. The review points out that the risk of cognitive deficits is related to the severity of the neonatal encephalopathy and the pattern of brain injury demonstrated on neuroimaging.

Admittedly the non-specific indicators of intrapartum asphyxia have not been helpful in predicting adverse neurological outcome attributing a specific cause to adverse outcomes. These non-specific indicators have included Apgar scores, umbilical cord gases, periodic changes on fetal heart rate tracings and the presence of meconium. Likewise, the presence of neonatal encephalopathy is not specific for hypoxia-ischemia due to intrapartum asphyxia. These indicators must be examined in the context of all the available clinical information if the cause of neurological injury is to be attributed appropriately. How each of the indicators interact or intersect is complex as they all fall within a wide spectrum. Moreover, antenatal conditions may increase the vulnerability of a particular fetus to adverse effects from intrapartum hypoxia. With regard to neonatal encephalopathy, the fact that there are stages in the development of this condition suggests that both the severity and duration of the insult varies along with the capacity of a particular fetus to withstand an insult. As stated earlier, these factors highlight the importance of the only real “essential” criteria, which is the process of prudently applying the differential diagnosis.

Maintaining that intrapartum events cannot result in neurological injury that does not include motor dysfunction does a disservice to the medical profession and, more crucially, to the affected patients. Fortunately more recent medical literature has come to recognize that the functional impact of brain injury can affect different domains, including motor, cognition and behaviour as well as vision and hearing.<sup>49</sup> It is time that ACOG and SOGC formally recognized that cognitive and learning disabilities related to intrapartum events can occur in children who do not suffer CP and associated motor deficits.

Miller has found that hypoxia-ischemia in term newborns can result in a watershed predominant pattern of white matter injury that can extend to grey matter when severe, resulting primarily in cognitive disabilities. On the other hand, severe motor disabilities are associated more often with a pattern of brain damage that results in basal nuclei predominant injury involving the deep grey nuclei and perirolandic cortex.<sup>50</sup>

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<sup>48</sup> Gonzalez FF, Miller SP, Does perinatal asphyxia impair cognitive function without cerebral palsy, Arch Dis Child Fetal Neonatal Ed 2006;91:F454-F459.

<sup>49</sup> See Miller 2009 From Selective vulnerability...

<sup>50</sup> See Miller 2009 page 500

Currently, hypothermia is used to treat neonatal encephalopathy due to intrapartum hypoxia-ischemia. Obviously spastic quadriplegia is not a criteria for administration of hypothermia. If the ACOG and SOGC criteria were correct (that CP with spastic quadriplegia must be present for hypoxia-ischemia to be the cause of neonatal encephalopathy) then this important treatment to avoid or mitigate neurological damage in the neonate would not be available. Clearly there are babies born with NE attributed to asphyxia who are not quadriplegic and who, with timely treatment, will avoid the extensive brain injury that may have resulted without treatment. Yet, these neonates clearly have NE induced by asphyxia.

In a 2005 study by Gluckman<sup>51</sup> the objective was to investigate whether 72 hours of selective head cooling with mild systemic hypothermia within 6 hours of birth improves neurodevelopmental outcome at 18 months with infants suffering from moderate or severe NE. The inclusion criteria included an Apgar score of 5 or less at 10 minutes after birth; the need for resuscitation; and, a severe acidosis, defined as a pH less than 7.0 and a based deficit of 16 mmol/L or more. In the discussion, reference is made to the fact that NE is a progressive syndrome and that many infants immediately after birth show initial transient recovery of cerebral oxidative metabolism, followed by secondary deterioration between 6 and 15 hours after birth. This is the window of opportunity to avoid or mitigate neuronal damage. A study by Shankaran in 2005<sup>52</sup> concluded that whole-body hypothermia reduces the risk of death or disability in infants with moderate or severe hypoxic-ischemic encephalopathy.

Neuroimaging has allowed reasonable accuracy in timing brain lesions.<sup>53</sup> Resolving brain edema captured on early imaging suggests brain injury occurring at or near the time of birth, and can rule out antenatal causes in many situations. This evolution of brain injury tracked on neuroimaging must be considered with the non-specific indicators and ought to substantially influence the differential diagnosis. This is true despite the absence of motor disability. In relation to where current medical thinking should be with respect to the differential diagnosis, consider the following from Gonzalez and Miller (2006):

The assumption in many of these studies that once other causes of encephalopathy are excluded, such as genetic syndromes or congenital infections, the remaining cases are primarily related to hypoxia-ischaemia. Recent imaging studies support this assumption in showing acute changes with patterns of injury that are most consistent with hypoxia-

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<sup>51</sup> See Gluckman PD, Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomized trial. *Lancet* 2005;365:663-70.

<sup>52</sup> See Shankaran S. Whole-Body Hypothermia for Neonates with Hypoxic-Ischemic Encephalopathy. *N Engl J Med* 353;

<sup>53</sup> See Miller 2006 page F457

ischaemia injury. These imaging changes correlate well with both acute and long-term neurological findings.

As the limitations of studies of neonatal encephalopathy are recognized, it is increasingly clear that childhood survivors of neonatal encephalopathy are at risk of cognitive deficits, even in the absence of functional motor deficits. With sophisticated and detailed measures of cognition, there seems to be an association between specific cognitive deficits, such as language and memory deficits, with the severity of neonatal encephalopathy and the pattern of brain injury, even in those without functional motor deficits. These differences are apparent in survivors of moderate and severe overt neonatal encephalopathy, particularly with the watershed predominant pattern of brain injury.<sup>54</sup>

Doubt about the ACOG criteria was raised long ago, yet the guidelines remain and continue to be relied upon by medical experts. In an article published in 1997 by Korst and others, the authors asked whether intrapartum brain injury could be predicted by the ACOG criteria.<sup>55</sup> In that review, of the 27 neonates who suffered an intrapartum asphyxial event, only 4 met all of ACOG's essential criteria. The review concluded that it could not identify a plausible link between the ACOG criteria and neurological injury caused by intrapartum events.<sup>56</sup>

Metabolic acidosis follows from significant hypoxia. The level of metabolic acidosis required to expose the fetus to morbidity was the subject of a study done by Low and others in 1997.<sup>57</sup> This study concluded that the threshold of metabolic acidosis associated with moderate to severe newborn complications is an umbilical artery base deficit of more than 12 mmol/L.

There is a "spectrum" of possible neurological outcomes associated with intrapartum asphyxia. In a 2009 article by Al-Macki and others<sup>58</sup> the possible outcomes were studied. In this study, children who had suffered intrapartum asphyxia were grouped into those with and those without cerebral palsy. Of 40 children meeting the study criteria, 17 developed abnormal neurologic outcomes that did not include cerebral palsy. The study concluded that abnormal neurologic outcomes other than cerebral palsy can occur following intrapartum asphyxia. If correct, this clearly contradicts the ACOG criteria.

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<sup>54</sup> page F458.

<sup>55</sup> Korst LM, Phelan JP, Ahn MO, Martin GI, Can persistent brain injury resulting from intrapartum asphyxia be predicted by current criteria?, (1997) *Prenat Neonat Med* 2 286-293.

<sup>56</sup> See page 291.

<sup>57</sup> Low JA, Lindsay BG, Derrick EJ, *Am J Obstet Gynecol* 1997;177:1391-4.

<sup>58</sup> Al-Macki N, Miller SP, Hall N, Shevell M, The spectrum of abnormal neurologic outcomes subsequent to term intrapartum asphyxia, 2009, *Pediatric Neurology* Vol. 41, No. 6, 399-405,

In *Neurology of the Newborn*, 5<sup>th</sup> edition, Volpe notes a study of children exposed to intrapartum asphyxia in which 2/3<sup>rd</sup> of the children exhibiting neurological deficits after 1 year did not have motor abnormalities.<sup>59</sup>

In the Al-Macki study the authors were careful to review only children with strong evidence of intrapartum asphyxia based on all the non-specific criteria recognized as possible indicators of hypoxia. As well, the authors were careful to ensure that there were no other etiologic or concurrent factors (such as congenital malformation, IUGR or SGA, for example) that would predispose to possible intrapartum asphyxia. That the study concludes that there is a spectrum of abnormal outcomes should not be a surprise. There has never been a reliable study to suggest otherwise.

Armstrong-Wells and others, in a paper published in 2010<sup>60</sup>, took issue with the ACOG requirement that motor dysfunction (CP) be present to establish a link between perinatal asphyxia and neurological injury. The study maintains that recent research has established a relationship between perinatal asphyxia and poor cognitive outcomes, regardless of motor impairments. Importantly, there tends to be delayed recognition of affected children without motor impairments flowing from intrapartum asphyxia. The authors found that neonates who suffer moderate encephalopathy have a range of difficulties with cognition and behaviour, even in the absence of motor impairment.<sup>61</sup> The study cited another paper which found that brain injury in the watershed pattern was associated with cognitive impairment alone without motor deficit.

There are other authors who have recognized the spectrum of disability caused by asphyxia. In a 2006 article by de Haan and others<sup>62</sup>, studies are cited that have found cognitive and behavioural deficits in children without motor dysfunction who suffered NE, both moderate and severe.

A study by Britt (2008)<sup>63</sup> studied corpus callosum size in school-age children with NE. The study found poorer motor skills in children with NE than in controls without any evidence of cerebral palsy. It was also suggested that attention-deficiti/hyperactivity disorder (ADHD) occurs more often in children with NE than in controls.

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<sup>59</sup> volpe page 339.

<sup>60</sup> Armstrong-Wells J, Bernard TJ, Boada R, Manco-Johnson M, Neurocognitive outcomes following neonatal encephalopathy, *NeuroRehabilitation* 26 (2010) 27-33.

<sup>61</sup> Page 29.

<sup>62</sup> de Haan, Brain and cognitive-behavioural development after asphyxia at term birth. *Dev Sci* 9:4:350-358.

<sup>63</sup> Britt JM. Corpus Callosum Size in Relation to Motor Performance in 9 to 10 Year Old Children with Neonatal Encephalopathy. *Pediatric Research* Vol. 63, no. 1, 2008.

A study by Steinman in 2009<sup>64</sup> looked at MRI changes on children with NE likely secondary to hypoxia-ischemia and found that watershed injury resulted in impairment with and without motor dysfunction. The study looked at the 2 characteristic patterns of brain injury following HIE, which includes: watershed (WS) distribution pattern involving the intervascular boundary-zone white matter, plus cortical gray matter when severe; and, a basal ganglia-distribution (BG) pattern involving deep gray nuclei, hippocampi and perirolandic cortex, with additional cortical involvement when severe. The study is important for recognizing an association between the degree of WS injury and the future verbal disabilities, suggesting a spectrum of disability. This study found that the pattern of brain injury, not just the severity, is important in determining future impairments whether motor or cognitive.

A study in 2010 by de Vries looked at the use of MRI in full-term infants following hypoxic-ischemic brain injury to evaluate the relevance of the findings in predicting neurodevelopmental outcome.<sup>65</sup> This study discussed the importance of neuroimaging in identifying antenatal brain injury. Given the ability to identify antenatal injury, it would be reasonable to assume that early cranial ultrasound would also help rule out antenatal injury. If followed within hours or days by brain edema, this should lend strong support to an intrapartum cause for NE. In reference to the WS type of brain injury, de Vries indicated that neurological manifestations at birth in the presence of these injuries may be mild with the onset of neurological symptoms delayed. The author went on to say that severe motor impairments are uncommon in this group of infants who tend not to manifest cognitive problems until early childhood.

The comments with respect to the International criteria apply equally to the ACOG criteria. Some additional remarks relating to the green book's justification for the criteria will be made.

The requirement for a pH of less than 7.0 as an essential criterion is inappropriate. While there have been many studies on this issue subsequent to the green book, this paper will examine just one issue that casts doubt on this criteria. It is not uncommon for cases of acute total asphyxia to involve umbilical arterial cord blood pH levels above 7.0. In *Neurology of the Newborn* by Volpe, 5<sup>th</sup> edition, one study is cited in which 60% of infants who later exhibited major neurological deficits had an umbilical cord pH higher than 7.0.<sup>66</sup>

With regard to NE, the green book recognizes that intrapartum insults severe enough to cause ischemic cerebral injury will manifest with NE and that moderate

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<sup>64</sup> Steinman KJ. Neonatal Watershed Brain Injury on Magnetic Resonance Imaging Correlates with Verbal IQ at 4 Years. *Pediatrics* vol. 123, no. 3, March 2009.

<sup>65</sup> See de Vries LS. Patterns of neonatal hypoxic-ischaemic brain injury. *Neuroradiology* (2010) 52: 555-566.

<sup>66</sup> See Volpe page 339.

NE and severe NE are associated with increased morbidity.<sup>67</sup> The presence of NE raises the possibility of an intrapartum cause. Asphyxia ought to be in the differential diagnosis in the presence of NE. The green book spins the issue the other way, indicating that the incidence of NE attributed to intrapartum asphyxia is very rare, 1.6 per 10,000 infants.

The green book uses the same examples of a sentinel event as the International criteria. It is unclear why the green book maintains that an intact fetus must undergo a sentinel event, like those described, to sustain neurological injury intrapartum. There is no medical evidence cited in support and, as with the International criteria, this is a non-specific indicator and fails to consider other conditions giving rise to hypoxia.

Comments in the green book regarding the fetal heart tracing patterns and their role in predicting acidemia are vague. It is acknowledged, however, that there are patterns which suggest current or impending damaging acidemia. They go on to point out that the patterns acknowledged to be associated with cerebral palsy have a very high false positive rate.<sup>68</sup> That does not, however, diminish the clinical importance of fetal heart rate tracings in determining when intervention is warranted for fetal well-being. Nor does it diminish the value of fetal heart tracings in prospectively timing the onset of fetal decompensation and irreversible neurologic injury. In his text *Neurology of the Newborn* 5<sup>th</sup> edition, Dr. Joseph Volpe states:

A distinct relationship has been demonstrated between intrapartum abnormalities of fetal heart rate, sometimes with documented fetal acidosis, and neurological morbidity in the neonatal period and after 1 year follow-up...

These data demonstrate that certain abnormal intrapartum fetal heart rate patterns alone can be valuable indicators of intrauterine insults, presumably hypoxic-ischemic, that result in neurological injury.

...certain fetal heart rate patterns are indicative of (or ultimately productive of) fetal hypoxia and the biochemical correlate of tissue oxygen debt, fetal acidosis.<sup>69</sup>

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<sup>67</sup> See page 74.

<sup>68</sup> See page 76.

<sup>69</sup> See Volpe 5<sup>th</sup> edition pages 336-7.

Before the 2003 ACOG Guidelines a previous publication by the same organization suggested that birth Asphyxia and neonatal encephalopathy could be connected in the presence of meconium staining, non-reassuring fetal heart rate patterns, low Apgar scores and NE. After 2003, from this list only NE was considered one of the "essential" criteria for connecting birth Asphyxia to injury. The other criteria were relegated to non-essential, but suggestive.

## **SOGC Guidelines**

The March 2002 Guideline no. 112 from the SOGC sets out the “essential criteria of the newborn response to asphyxia of such a degree as to be likely to cause harm” as follows:

- i. Apgar score 0-3 for 5 minutes or more;
- ii. Neonatal neurologic sequelae (e.g., hypotonia, seizures, coma);
- iii. Evidence of multi-organ system dysfunction in the immediate neonatal period;
- iv. Umbilical cord arterial pH < 7.0; and
- v. Umbilical cord arterial base deficit > 16 mmol/L.<sup>70</sup>

These “essential criteria” are the same as those set out in the 1995 report of the SOGC Task Force on Cerebral Palsy and Fetal Asphyxia.<sup>71</sup> Guideline 112 required that “all” of the criteria must be present.<sup>72</sup> In the absence of these conditions “one cannot conclude that hypoxic acidemia existed or had the potential to cause neurologic deficits”. As will be seen, the views on the essential criteria have changed. One observation that is important relates to the requirement for a base deficit greater than 16 mmol/L from the umbilical arterial blood. The leading study on the threshold of metabolic acidosis needed to expose the fetus to morbidity and mortality was done in 1997 by Low and established the threshold to be > 12 mmol/L.<sup>73</sup>

The SOGC criteria sets out a higher threshold for metabolic acidosis than the ACOG criteria (using a base deficit of 16 mmol/L rather than 12 mmol/L). Presumably fetuses south of the border do not begin to suffer neurologic injury in the presence of acidosis sooner than Canadian fetuses. The fact that the SOGC uses a higher threshold for metabolic acidosis than Low, ACOG and the International Statement undermines the SOGC position and demonstrates the folly of this as an “essential” criteria.

The September 2007 Journal of Obstetrics and Gynecology Canada, published by the SOGC sets out the Canadian guidelines as established by that organization.<sup>74</sup> The SOGC statement refers to the International Guidelines of

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<sup>70</sup> SOGC Clinical Practice Guideline, No. 112, March 2002, Fetal Health Surveillance in Labour.

McLennan in setting out the four essential criteria before a link can be made between “CP and intrapartum asphyxia”<sup>75</sup>:

1. Evidence of metabolic acidosis in umbilical cord arterial blood obtained at delivery: pH <7 and base deficit  $\geq$  12 mmol/L;<sup>76</sup>
2. Early onset of severe or moderate neonatal encephalopathy in infants born at or beyond 34 weeks’ gestation;
3. cerebral palsy of the spastic quadriplegic or dyskinetic type; and
4. exclusion of other identifiable etiologies, such as trauma, coagulopathy; infectious conditions or genetic disorders.

The SOGC statement also cites the ACOG non-essential criteria.<sup>77</sup> These include:

5. a sentinel (signal) hypoxic event occurring immediately before or during labour;
6. 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 a hypoxic sentinel event when the pattern was previously normal;
7. Apgar scores of 0-3 beyond 5 minutes;
8. onset of multisystem involvement within 72 hours of birth;
9. early imaging study showing evidence of acute nonfocal cerebral abnormality.

Without expressly saying so in the 2007 publication, it would appear that the SOGC has tacitly adopted the International Guidelines.

The SOGC makes reference to the fact that the “watershed” areas between the end branches of the major cerebral vessels are at highest risk with asphyxia.<sup>78</sup> The report says that “often” this injury involves the motor cortex and “the most frequent consequence of the injury is spastic quadriplegia”. One wonders whether there is some concession here to the fact that non-motor injury can

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<sup>71</sup> See SOGC Practice Guidelines for Obstetrics, Policy Statement, Task Force on Cerebral Palsy and Fetal Asphyxia, No. 19, released December 1995. See also SOGC Policy Statement No. 43 (1995).

<sup>72</sup> Page 2

<sup>73</sup> See Low 1997.

<sup>74</sup> Journal of Obstetrics and Gynecology Canada, September 2007, Volume 29, Number 9, SOGC.

<sup>75</sup> See page S26. Note once again that the link is with “CP” and does not say anything about whether other non-motor neurologic deficits might be linked to intrapartum asphyxia.

<sup>76</sup> Has the position of SOGC changed from Guideline 112 published in 2002 which set out the base deficit threshold at 16 mmol/L?

<sup>77</sup> See page S27.

<sup>78</sup> Page S25

result from asphyxia. It is difficult to reconcile these apparent “qualifications” with the statement of the SOGC “essential” criteria.

The SOGC also recognizes the efficacy of cooling in improving outcomes for moderate and severe NE.<sup>79</sup> Clearly hypothermia is offered to neonates suspected of suffering intrapartum asphyxia. None of these neonates have been diagnosed with spastic quadriplegia. If spastic quadriplegia is an “essential criteria” for linking NE with asphyxia, then this important treatment protocol would not be offered to any neonate.

In demanding CP of the spastic quadriplegic variety to make the link to asphyxia, the SOGC points out that this type is the “only type of CP” linked to asphyxia.<sup>80</sup> Taking that statement in isolation, it does not follow that CP is the only type of neurologic injury that can result from asphyxia, though the essential criteria would suggest that.

### **The Proposed Approach to Causation**

The differential diagnosis should be acknowledged to be the most reliable approach for the purpose of proving or disproving a connection between newborn neurological injury and peripartum asphyxia. While the clinical criteria set out in the three sets of guidelines are individually and collectively important in making the correct diagnosis, attempts to make those statements the last word are misguided. There is no reliable medical evidence to support the essential criteria described by MacLennan, ACOG or SOGC, yet it is fair to say that many medical practitioners have blindly relied on them with the possibility that some legitimate claims have been defeated through the application of the unreliable principles.

While the starting point for diagnosis varies for different cases, given the role that neuroimaging now plays in the field, it can often be the starting point. Neuroimaging helps to rule in or out many potential diagnoses and also helps with timing. Timing itself can help rule out some diagnoses.

All of the described “essential criteria” and all the non-specific criteria have a part to play in leading medical practitioners to the most likely diagnosis in accordance with the legal standard of proof – the balance of probabilities. Perhaps some of the criteria (for example, a base deficit of greater than 12 mmol/L) are more influential in properly attributing neurologic injury to a particular cause, but care should be taken to avoid hard and fast rules that are not firmly supported by the best medical evidence. To do otherwise is to do a disservice to patients and has the potential to deny fair and reasonable access to justice.

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<sup>79</sup> See page S26.

<sup>80</sup> Page S26.

In establishing criteria or providing guidance on making the correct diagnosis, the agenda should not be the avoidance of liability and accountability, two factors that arguably contribute to improved and safer medical care. Rather, complete objectivity should instruct the methods used to establish or refute the potential link between neurologic injury and intrapartum asphyxia.

### **Justifying the Proposed Approach to Causation**

There is a moral and ethical obligation on the part of physicians to acknowledge medical error. Upholding this obligation is essential for keeping patients adequately informed and the advancement of medicine through the reduction of medical errors. Physicians are accountable for the medical care they provide. Medical groups representing the interests of these physicians should take great care not to undermine these obligations.

The ACOG criteria are extremely difficult to satisfy. After a thorough reading of the green book, one is left with the distinct impression that the incidence of intrapartum asphyxia is so rare as to be virtually non-existent. Whether this impression is intended or not, the impression does not reflect medical reality. On the matter of intrapartum asphyxia one of the leading texts, *Neurology of the Newborn* by Volpe, states:

...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. Among the many adverse consequences of the explosion in obstetrical litigation has been a tendency in some quarters of the medical profession to deny the importance or even the existence of intrapartum brain injury... Denial that intrapartum injury occurs may impair development and application of...brain-saving intervention.<sup>81</sup>

Volpe also observes that “the data are remarkably consistent in showing that 17% to 24% of cases of cerebral palsy are related to intrapartum asphyxia”.<sup>82</sup> It is clear that despite the attempt to persuade readers that intrapartum asphyxia is rare and unimportant, this is simply not the case. No less an authority that Volpe has said that such a conclusion is incorrect.<sup>83</sup>

### **The Law**

Where and how criteria set out by professional medical associations fit within the context of medical malpractice litigation is important. Although this paper is

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<sup>81</sup> See Volpe JJ, *Neurology of the Newborn*, 5<sup>th</sup> ed, page 331.

<sup>82</sup> Volpe page 332.

<sup>83</sup> Volpe page 332.

primarily concerned with causation, some law on standard of care may be instructive and will be reviewed.

The case of *Crits v. Sylvester*<sup>84</sup> sets out the following comments regarding the medical standard of care:

Every medical practitioner must bring to his task a reasonable degree of skill and knowledge and must exercise a reasonable degree of care. He is bound to exercise that degree of care and skill which could reasonably be expected of a normal, prudent practitioner of the same experience and standing, and if he holds himself out as a specialist, a higher degree of skill is required of him than of one who does not profess to be so qualified by special training and ability.

Despite the fact that a physician has followed a recognized practice, the court may still find that practice to be negligent.<sup>85</sup> In *ter Neuzen v. Korn*<sup>86</sup> the court stated:

On the other hand, as an exception to the general rule, if a standard practice fails to adopt obvious and reasonable precautions which are readily apparent to the ordinary finder of fact, then it is no excuse for a practitioner to claim that he or she was merely conforming to such a negligent common practice.

That same rationale can be applied in the context of the discussion on causation. A court should not be expected to adopt conclusions about the link between CP and asphyxia promoted by a professional organization of physicians, no matter how widely accepted by practicing physicians, where it is readily apparent that there was and is little medical evidence to support those conclusions.

Ordinarily, the court will determine whether the standard of care has been met before considering causation. The plaintiff must prove on a balance of probabilities that the care provided did not meet applicable standards. The plaintiff must then go on to prove that on a balance of probabilities the breach of the standard of care caused harm.

Regarding causation, the plaintiff must prove that the injury would not have been suffered “but for” the breach of the standard of care.<sup>87</sup> Some case law has suggested that there may be times when the “but for” test is unworkable, in which case the court may apply the “material contribution” test. In *Resurface Corp v. Hanke*, the Supreme Court of Canada stated:

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<sup>84</sup> *Crits v. Sylvester* (1956), 1 D.L.R. (2d) 502 (Ont.C.A.), affirmed at [1956] S.C.R. 991, page 508.

<sup>85</sup> See *MacGregor v. Potts* (2009)

<sup>86</sup> *ter Neuzen v. Korn*, [1995] S.C.J. No. 79, paragraph 51.

<sup>87</sup> See *Horsely v. MacLaren*, [1972] S.C.R. 441; *Snell v. Farrell*, [1990] S.C.R. 311; and, *Athey v. Leonati* [1996] 3 S.C.R. 458.

The “but for” test recognizes that compensation for negligent conduct should only be made ‘where a substantial connection between the injury and defendant’s conduct’ is present. It ensures that a defendant will not be held liable for the plaintiff’s injuries where they ‘may very well be due to factors unconnected to the defendant and not the fault of anyone’...

However, in special circumstances, the law has recognized exceptions to the basic “but for” test, and applied the “material contribution” test. Broadly speaking, the cases in which the “material contribution” test is properly applied involve two requirements.

First, it must be impossible for the plaintiff to prove that the defendant’s negligence caused the plaintiff’s injury using the “but for” test. The impossibility must be due to factors that are outside of the plaintiff’s control; for example, current limits of scientific knowledge. Second, it must be clear that the defendant’s breached a duty of care owed to the plaintiff, thereby exposing the plaintiff to an unreasonable risk of injury, and the plaintiff must have suffered that form of injury...

The formulation of the rule may well have application in the context of birth trauma causation, though the material contribution test has yet to be applied in this, or any other, context.

In *Snell v. Farrell*<sup>88</sup> the Supreme Court of Canada promoted a more pragmatic approach to causation, stating “causation need not be determined by scientific precision”. That case was considered in *Allen v. Mueller*<sup>89</sup> where the Alberta Court of Appeal stated:

The plaintiff does not need to show causation to a level of medical certainty, but rather only on a balance of probabilities. Thus, the trial judge may draw an inference, where a medical expert would not, based on common sense and a consideration of all the circumstances. The plaintiff always bears the burden of adducing some evidence of causation, although how much is needed depends upon who holds the knowledge or how much knowledge exists.<sup>90</sup>

Publications from organizations like ACOG have been used and applied in Canadian courts. In one Ontario case, ACOG was referred to as an internationally recognized “leader in the development and publication of standards with respect to expected obstetrical practice...”.<sup>91</sup>

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<sup>88</sup> *Snell v. Farrell*, [1990] 2 S.C.R. 311, at page 328.

<sup>89</sup> *Allen v. Mueller*, (2002) ABCA 195.

<sup>90</sup> See *Allen*, paragraph 19.

<sup>91</sup> See *Vuong v. St. Joseph’s Hospital*, (2009) paragraph 24

The reliability of scientific theories is subjected to scrutiny by our courts. Generally speaking the courts must consider whether the scientific theory has been subject to empirical testing, peer review and publication. The court must look at the rate of error and, importantly, must consider whether the theory has attained acceptance within the relevant scientific community.<sup>92</sup> The issue as to whether the theory is accepted by the relevant scientific community is of particular relevance.

In deciding whether a particular theory has been adopted by a scientific community, one must first identify the community in issue. The theories at issue in this paper are propounded by organizations representing obstetricians and gynecologists, whereas the issue of linking intrapartum asphyxia to subsequent neurologic injury is a matter more within the expertise of the neonatology, neurology, pediatric and neuroradiology communities. Although participants in the ACOG task force included radiologists, pediatricians and others, there is no indication that the conclusions in the green book have been widely adopted by pediatricians, radiologists or others. Moreover, the asphyxia criteria, published by the specialty most concerned with avoiding liability for birth trauma, might be perceived by those on the outside as somewhat less than objective.

A problem with organizations endowed with the apparent credibility enjoyed by ACOG is that some in the medical community will tend to merely adopt ACOG conclusions without the requisite critical and or rigorous scientific analysis. This is a mistake made by many experts providing opinions in medical malpractice cases tend to fall in to. In this way, the deficiencies in publications like the green book become self-perpetuating.

From the perspective of the pediatric neurology community, it seems clear that the ACOG criteria are not generally accepted. To connect intrapartum insult to neonatal brain injury Volpe, says you need the following:

- a) evidence of fetal distress (fetal heart rate abnormalities, meconium stained fluid);
- b) depression at birth; and
- c) an overt neonatal neurological syndrome in the first hours and days of life.<sup>93</sup>

The appropriate use of the SOGC guidelines were in issue in Allen case, cited above. In that case the infant did not meet all of the diagnostic criteria set out by the SOGC. The Allen case, was decided in 2002 and appears to have referred to SOGC guidelines that preceded those referenced in this paper. The essential guidelines in the case cite: Apgar scores of 0-3 for longer than 5 minutes; neonatal neurologic sequelae; multi-system organ failure; and, profound umbilical

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<sup>92</sup> See Allen v. Mueller 2002 Alberta Court of Appeal and R. v. Mohan, [1994] 2 S.C.R. 9.

<sup>93</sup> Volpe page 401.

artery metabolic acidosis.<sup>94</sup> For the purpose of this analysis, the criteria do not matter. There was controversy amongst the experts who testified in that case as to the reliability of the guidelines. The court said:

The totality of the evidence supports the conclusion that controversy exists over the numerical parameters to be assigned to and the weight to be attributed to each criterion. Moreover, studies and conflicting evidence raised the question of the application of the criteria to this type of injury. Careful examination of the criteria as undertaken by the trial judge could fairly support a finding that the criteria were not determinative of the nature of Ashleigh's injuries or their cause.<sup>95</sup>

In Scotland a judge had the opportunity to consider the argument advanced by the defence that that a reduction in asphyxia in recent years has not seen a corresponding drop in the incidents of dyskinetic CP, from which the court was asked to conclude that CP is unlikely to be related to birth asphyxia. The court stated:

I am not persuaded that the decline in birth asphyxia and the absence of corresponding decline in dyskinetic cerebral palsy would allow me to infer that birth asphyxia is not an important cause of that type of cerebral palsy. As only a small proportion of cerebral palsies is caused by birth asphyxia, and as dyskinetic cerebral palsy is a relatively unusual form of cerebral palsy, I would not necessarily expect an overall decline in birth asphyxia to give rise to a detectable decline in the incidence of dyskinetic cerebral palsy.<sup>96</sup>

## CONCLUSIONS

There is a link between neurologic injury and intrapartum asphyxia. The neurologic injury that results from intrapartum asphyxia may or may not involve motor dysfunction. Where sub-standard medical care caused or contributed to the neurologic injury, there needs to be accountability. The criteria developed by the three entities reviewed in this paper have been used time and again in litigation as a shield to resist claims of profoundly injured children. The strict application of these criteria to these cases is, in my view, inappropriate, unjustified and has the potential to cause serious injustice. The criteria undermine the prosecution of some legitimate claims.

The scientific foundation for the criteria was entirely insufficient to support such a dogmatic approach to causation in birth trauma cases when the various guidelines were published. The medical literature published since has further

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<sup>94</sup> See Allen paragraph 33.

<sup>95</sup> Allen, paragraph 35.

<sup>96</sup> See McKenzie v. Fife Acute Hospitals NHS Trust, [2006] CSOH 63, at paragraph 34.

undermined the criteria. While research and study is currently underway to revise or amend the criteria, steps should be taken, based on the existing literature, to ensure that physicians and courts do not continue to apply the criteria as rigidly as the various guidelines call for, thereby avoiding any further injustice for children injured through birth trauma.