

## **INTRAPARTUM PATHWAYS TO NEONATAL NEUROLOGIC INJURY - A LAWYER'S VIEW OF THE MEDICINE**

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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.

Lawyers representing children with suspected neonatal injury from peripartum events must have a thorough understanding of the pathways leading to these injuries in order to properly advance the interests of their clients. Only with knowledge of what causes newborn neurologic injury can one know what to look for, what questions to ask and who to consult. Identifying the likely causal pathway to neonatal neurologic injury is challenging and at times controversial.

The first step is to determine whether peripartum events are the likely cause of injury. Conventional medical thought focuses on hypoxia-ischemia (asphyxia) as the main cause of neonatal neurologic injury that may result in a compensable injury. Certainly these are the cases that tend to be pursued in our courts. Though less common, there may be other causes of neurologic injury giving rise to a cause of action. One such possibility is "head compression".

The second step is to show how peripartum events led to the injury. The pathophysiology of peripartum neurologic injury is a matter of some controversy. In obstetrics, neonatology and neuroradiology there is disagreement regarding many of the medical issues that surround causation in the context of neonatal neurologic injury. Unfortunately, and unjustifiably, some of the thinking and science regarding causation has been influenced and distracted by concerns about legal liability.

This paper will explore the possible intrapartum pathways to neonatal neurologic injury and how some of the science and literature impact on the representation of affected children. In doing so, this paper will review neonatal neurologic injury in the context of a fetal systemic metabolic acidosis and in the alternate setting where neonatal neurologic injury is not accompanied by a fetal systemic metabolic acidosis.

These represent two distinct pathways to injury. It is widely accepted that substandard intrapartum care can result in a systemic metabolic acidosis leading to brain injury (the conventional intrapartum asphyxia model). It is not widely accepted that substandard intrapartum care can result in brain injury in the absence of systemic metabolic acidosis and localized impaired cerebral perfusion.

Generally speaking, the importance of peripartum events in causing neonatal brain injury has been understated and underappreciated in the scientific literature.<sup>1</sup> The failure to acknowledge the significant contribution of peripartum events to neonatal brain damage is most prominent in the obstetrical community. This is due, in part, to a number of propositions promoted by some in the medical community, including: the inappropriate adoption of “essential” causation criteria that are not essential<sup>2</sup>; the focus on cerebral palsy as the only neurological outcome to flow from asphyxia, ignoring other possible neurological injury not associated with motor dysfunction<sup>3</sup>; the suggestion that intrapartum events rarely cause neonatal encephalopathy<sup>4</sup>, when the fact is that intrapartum events are responsible for a large source of preventable neonatal brain injury<sup>5</sup>; and, the failure to appreciate the contribution of cerebral ischemia without a systemic fetal metabolic acidosis.

It is well recognized in medical literature that neonatal encephalopathy (NE) can result from intrapartum events. The subset of NE attributed to intrapartum events is generally referred to as hypoxic-ischemic encephalopathy (HIE). Importantly, while HIE refers to both “hypoxia” and “ischemia”, it is ischemia that causes

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<sup>1</sup> See Volpe Neurology of the Newborn (5<sup>th</sup> Edition) p. 331: “Among the many adverse consequences of the explosion in obstetrical litigation has been the tendency in some quarters of the medical profession to deny the importance or even the existence of intrapartum brain injury...this tendency is particularly unfortunate.”

<sup>2</sup> See the Green book page 74. See Volpe p. 401 where he indicates that the link between intrapartum insult and neonatal brain damage is “likely” when there is (1) evidence of fetal distress; (2) depression at birth, and (3) an overt neonatal neurological syndrome in the first hours and days of life.

<sup>3</sup> Green book p. xvii says: “...absent cerebral palsy, neither epilepsy, mental retardation, nor attention-deficit hyperactivity disorder are caused by birth asphyxia”.

<sup>4</sup> Green book p. 8.

<sup>5</sup> See Volpe p. 331. Looking at cerebral palsy alone, ignoring other adverse neurological outcomes, Volpe, on page 332 states that 17% to 24% of cases of cerebral palsy are related to intrapartum asphyxia. See also Cowan F, Origin and timing of brain lesions in term infants with neonatal encephalopathy. Then Lancet, Vol 361, March 1, 2003, pp 736-742, at p. 739 which says that more than 90% of term infants with NE had evidence of perinatal acquired insults.

neuronal damage and consequent disability. Given that ischemia is a more important consequence of oxygen deprivation than hypoxemia<sup>6</sup>, it is vital to appreciate the potential for intrapartum cerebral ischemia (with or without hypoxemia). Traditionally, much of the medical literature identifies the pathway to brain ischemia as one that inevitably passes through hypoxia leading to a metabolic acidosis. The developing metabolic acidosis may be followed by cardiac dysfunction that results in decreased cerebral perfusion (ischemia) which can lead to neurologic injury. Hypoxia-induced ischemia, with some exceptions, can be expected to be accompanied by a metabolic acidosis. Intrapartum threats to fetal well-being, however, are not confined to hypoxia. Mechanical forces associated with labour, and the effect of these forces on the fetal head and cerebral blood flow (CBF), expose the fetus to risk of morbidity and mortality.

The main objective of monitoring of the fetal heart rate during labour is to assess the adequacy of fetal oxygenation through the identification of certain characteristics affected by hypoxic conditions.<sup>7</sup> Where the intrauterine environment is hypoxic, there exists a risk that hypoxemia will lead to metabolic acidosis that ultimately causes ischemia and possible neurologic injury. Likewise, the fetal heart rate will respond to an intrauterine environment causing excessive pressure on the fetal head. Responding to fetal heart rate patterns suggestive of hypoxia or head compression allows for intervention to avoid neurologic injury.

The conventional hypoxic pathway to intrapartum neurologic injury is focused on impaired gas exchange between the mother and the fetus. That impaired gas exchange can come in the form of factors that diminish utero-placental perfusion or factors that affect umbilical blood flow. This approach, however, will not account for the more direct impact of mechanical forces on the fetus that affect cerebral blood flow.

## **TRADITIONAL APPROACH TO BIRTH ASPHYXIA CASES AND CAUSATION**

### **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 that neurologic injury was caused solely by events that occurred before intervention was warranted, the claim cannot succeed and no damages will be awarded.

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<sup>6</sup> See Volpe p.247

<sup>7</sup> See Freeman R.K. Fetal Heart Rate Monitoring (4<sup>th</sup> Edition), p.8.

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 (also referred to as “asphyxia”) is an intrapartum condition that can lead to brain damage. Neonatal encephalopathy (NE)<sup>8</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>9</sup> The challenge in these cases is to distinguish between asphyxic and non-asphyxic causes for NE and, if asphyxic, to ascertain whether the NE caused neurologic injury.

Much of the published medical literature on the subject, particularly as it relates to cerebral palsy, describes the rarity with which neurologic injury arises from asphyxia. Cerebral palsy (or other neurologic injury without a motor component) arising out of intrapartum events is considered to be relatively rare, but a better understanding of how some intrapartum events impact the fetus might suggest it is more common than thought. 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.

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 compensation for loss.

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<sup>8</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>9</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.

## **OBSTACLES TO PROVING CAUSATION**

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. Finally, the medical and logical foundation for the guideline will be critically explored.

### **The International Consensus Statement**

Alastair MacLennan was the chair of the International Cerebral Palsy Task Force funded by the Perinatal Society of Australia and New Zealand.<sup>10</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>11</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 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

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<sup>10</sup> It should be noted that supporters of the International Criteria include ACOG and SOGC.

<sup>11</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.

credibly maintaining the position that cases of cerebral palsy never originate in labour. To say that neurologic injury can in fact originate in labour is obvious.

The International Statement says that “intrapartum complications play an infrequent role in the causation of cerebral palsy”.<sup>12</sup> Acknowledging that intrapartum complications are contributors to CP, even if 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) must be established clinically to support the conclusion that peripartum asphyxia is implicated in the brain injury.

### **Criteria For CP**

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>13</sup>

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;

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<sup>12</sup> Ibid Page 1056.

<sup>13</sup> Ibid page 1056

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>14</sup> There is nothing logical about that conclusion in the absence of reliable supporting scientific literature. This statement highlights the rather shaky medical foundation upon which the criteria are based.

On the matter of the requirement for metabolic acidemia, the statement contends that damaging intrapartum asphyxia cannot even be postulated unless metabolic acidemia is present.<sup>15</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 resolution of acidosis. A metabolic acidosis is also not expected in the context of a more sudden and profound total or near-total asphyxia. In that scenario, ischemic injury (tissue hypoxia or poor delivery of oxygen to vital organs) most definitely can occur without a systemic metabolic acidosis.

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>16</sup> Given the accepted way in which the differential diagnosis must be applied to the question of causation, I suggest that this is inaccurate. In fact, it is surprising that the differential diagnosis approach 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. Further, blood samples can become contaminated in some circumstances, making result inaccurate.

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<sup>14</sup> Ibid.

<sup>15</sup> Ibid.

<sup>16</sup> Page 1056.

While the International Statement posits that a base deficit of 16 mmol/L is “a realistic cut off point for defining pathological fetal acidemia that correlates with an increasing risk of neurological deficit”<sup>17</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 understates 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>18</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 authors of 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 causation. Despite the multiple causes for NE, it just so happens that intrapartum fetal asphyxia is one of the causes of NE. A key to determining the likelihood that NE is caused by intrapartum asphyxia relates to periodic changes in the fetal heart tracing compatible with hypoxia. A fetus with a developing metabolic acidosis will always show changes on the tracing compatible with tissue (or vital organ) 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>19</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>20</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, both in terms of the care they receive and their access to justice.

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<sup>17</sup> See page 1056.

<sup>18</sup> Ibid See page 1057.

<sup>19</sup> Ibid.

<sup>20</sup> Ibid.

## **FETAL HEART RATE MONITORING**

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>21</sup> Importantly, fetal heart rate monitoring is not designed to “predict CP”. It is, however, widely recognized that certain fetal heart rate patterns suggest tissue hypoxia. Suggesting that intervention not take place until there is potential “severe” fetal compromise is 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.

## **NEUROIMAGING**

With regard to neuroimaging, the fact that cerebral edema early in the neonatal period suggests recent insult is acknowledged by the International Statement.<sup>22</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. Neuroimaging as a diagnostic aid to determining the etiology of NE was important at the time the International Statement was published. With advances in technology and interpretation, neuroimaging is even more crucial to identifying the cause and timing of NE.

## **ANTENATAL CAUSES**

The Statement sets out conditions, other than asphyxia, that might cause cerebral palsy.<sup>23</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:

- Umbilical artery base deficit less than 12 mmol/L or pH higher than 7.00
- Congenital or metabolic abnormalities
- Systemic infections
- Longstanding neurological abnormalities evident on early imaging
- IUGR
- Reduced FHR variability from the onset of labour
- Microcephaly

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<sup>21</sup> Ibid.

<sup>22</sup> Ibid 1058.

<sup>23</sup> page 1058

- Antenatal placental abruption
- Congenital coagulation disorders
- Other antenatal risk factors (preterm birth, multiple gestation, autoimmune disease)
- Presence of major postnatal risk factors for cerebral palsy
- A sibling with the same type of CP

Again, this is really the process of differential diagnosis. Significantly, the Statement fails to acknowledge 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>24</sup>

There are generally two types of brain injury patterns arising out of intrapartum asphyxia.<sup>25</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.

## **SO CALLED “SILENT” HYPOXIA**

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, I would suggest that intrapartum hypoxia leading to a developing metabolic acidosis and subsequent neurological injury is never “silent”. The unfriendly intrauterine environment that leads to these outcomes is one where the fetus will be sure to express some signs of discomfort or distress through periodic changes in the fetal heart rate. Antepartum injury, occurring while the fetus is not monitored, may go undetected (silent). Indeed, fetal distress with potential intrapartum asphyxia will inevitably be manifested through periodic changes in the fetal heart rate. Often the likely timing of irreversible insult is most reliably revealed by analyzing the tracing, in conjunction with other intrapartum and neonatal clinical data. The responsibility of the obstetrical team is to appreciate when the pattern

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<sup>24</sup> See Note 1, Volpe page 401.

<sup>25</sup> See de Vries LS. Patterns of neonatal hypoxic-ischaemic brain injury. *Neuroradiology* (2010) 52:555-566.

suggests a potential for exhaustion of fetal reserves sufficient enough to warrant expedited delivery.

## 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>26</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>27</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;

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<sup>26</sup> Do they mean CP only or any neurological injury?

<sup>27</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.

8. Onset of multisystem involvement within 72 hours of birth; and
9. Early imaging study showing evidence of acute non-focal abnormality.

The ACOG criteria and International criteria differ in some important ways. First, ACOG has recognized the importance of the differential diagnosis by including it 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, however, in fact 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.

## **TYPE OF CP**

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>28</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>29</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>30</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>31</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>28</sup> See Green book at page xii

<sup>29</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 Green book at page xvii.

<sup>30</sup> Ibid P. xvii

<sup>31</sup> Ibid p. 2

in saying that infants with severe NE are more likely to sustain long-term neurological morbidity,<sup>32</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>33</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. In fact, the literature establishes that intrapartum asphyxia can cause non-CP neurologic injury.

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>34</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 neurologic dysfunction.

The task force maintains “with certainty” that intrapartum hypoxia-ischemia leading to CP “must” progress through NE.<sup>35</sup> They neglect to say whether neurological injury leading to non-motor sequelae 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

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<sup>32</sup> Ibid p. 6

<sup>33</sup> Ibid p. 7

<sup>34</sup> Ibid p. xiii

<sup>35</sup> Ibid p. xvii

to NE. The task is then to determine whether the NE is caused by hypoxic-ischemic encephalopathy related to intrapartum events or by other causes.

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>36</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>37</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?
9. Was the data used sufficient to draw conclusions regarding causation that justified carving out strict, mandatory criteria?

## **ANTEPARTUM VS. INTRAPARTUM EVENTS**

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>38</sup> The report

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<sup>36</sup> Ibid 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>37</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.

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 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 neuroimaging.

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 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>39</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.

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<sup>38</sup> Ibid Page 25

<sup>39</sup> Ibid page 508

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>40</sup> There were 378 control subjects.

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 tissue hypoxia.<sup>41</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>42</sup> Another interesting finding is the apparent association between tight nuchal cord and unexplained CP.<sup>43</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>44</sup> More fundamentally, a study of this nature should not be relied on as establishing definitive guidelines

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<sup>40</sup> Ibid page 509.

<sup>41</sup> Ibid page 509.

<sup>42</sup> Nelson page 510.

<sup>43</sup> Ibid page 511.

<sup>44</sup> Ibid page 512.

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>45</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.

## **MOTOR VS. NON-MOTOR NEUROLOGIC INJURY**

An article by Rosenbloom<sup>46</sup>, cited in the green book, 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>47</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.

A 1993 article by Stanley and others<sup>48</sup>, also cited in the green book, 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 so that all the children had motor dysfunction. As a result, no conclusions can be drawn about the potential for intrapartum asphyxia to cause neurologic injury without motor dysfunction. The study looked for, among other things, the "common antecedents which might give clues to causation and possible prevention".<sup>49</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

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<sup>45</sup> Ibid, page. 512.

<sup>46</sup> Rosenbloom L. Dyskinetic Cerebral Palsy and Birth Asphyxia. Development Medicine and Child Neurology, 1994, 285-289.

<sup>47</sup> Ibid page 3

<sup>48</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>49</sup> Ibid page 191.

- b) That if such encephalopathy had been present, it was more likely to have occurred in an already vulnerable infant.

The sample studied by Stanley was confined to children with quadriplegia. The study notes<sup>50</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>51</sup> Work done by Miller<sup>52</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 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>53</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. 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

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<sup>50</sup> Ibid page 198.

<sup>51</sup> See Miller SP. Patterns of brain injury in term neonatal encephalopathy. *Journal of Pediatrics*, April 2005, page 453.

<sup>52</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.

<sup>53</sup> Gonzalez FF, Miller SP, Does perinatal asphyxia impair cognitive function without cerebral palsy, *Arch Dis Child Fetal Neonatal Ed* 2006;91:F454-F459.

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.

Importantly, the focus of this discussion is on the term fetus (after 36 weeks gestation). There are different considerations for preterm fetuses. A discussion of NE and prematurity is beyond the scope of this paper. Another consideration that must be entertained, no matter the gestational age, is the possibility of co-morbid causes. Again, a subject that will not be addressed here.

## **DOUBTING THE ACOG AND SOGC GUIDELINES**

Maintaining that intrapartum events cannot result in neurological injury that does not include motor dysfunction is contrary to recent literature. 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>54</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 perirhinal cortex.<sup>55</sup>

Neuroimaging has allowed reasonable accuracy in timing brain lesions.<sup>56</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):

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<sup>54</sup> See Miller 2009 The Spectrum of Abnormal Neurologic Outcomes Subsequent to Term Intrapartum Asphyxia, volume 41, No. 6, *Ped Neurol* (2009)

<sup>55</sup> Miller SP from selective vulnerability to connectivity: insights from newborn brain imaging (2009) *Trends in Neuroscience* Volume 32, No. 9, p. 500.

<sup>56</sup> See Miller note 54 2006 page F457

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-ischemia. Recent imaging studies support this assumption in showing acute changes with patterns of injury that are most consistent with hypoxia-ischemia 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>57</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>58</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>59</sup>

Metabolic acidosis follows from significant tissue hypoxia. The level of metabolic acidosis that predicts fetal morbidity was the subject of a study done by Low and others in 1997.<sup>60</sup> This study concluded that the threshold of metabolic acidosis associated with moderate to severe newborn complications is an umbilical artery base deficit greater 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>61</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

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<sup>57</sup> See note 54, page F458.

<sup>58</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>59</sup> *Ibid* page 291.

<sup>60</sup> Low JA, Lindsay BG, Derrick EJ, *Am J Obstet Gynecol* 1997;177:1391-4.

<sup>61</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,

neurologic outcomes other than cerebral palsy can occur following intrapartum asphyxia. If correct, this clearly contradicts the ACOG criteria.

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>62</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, 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>63</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>64</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>65</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>66</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-deficit/hyperactivity disorder (ADHD) occurs more often in children with NE than in controls.<sup>67</sup>

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<sup>62</sup> See note 1, Volpe page 339.

<sup>63</sup> Armstrong-Wells J, Bernard TJ, Boada R, Manco-Johnson M, Neurocognitive outcomes following neonatal encephalopathy, *NeuroRehabilitation* 26 (2010) 27-33.

<sup>64</sup> *Ibid* Page 29.

<sup>65</sup> de Haan, Brain and cognitive-behavioural development after asphyxia at term birth. *Dev Sci* 9:4:350-358.

<sup>66</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, pp 103-108.

<sup>67</sup> *Ibid* at page 107.

A study by Steinman in 2009<sup>68</sup> looked at MRI changes in 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 intravascular boundary-zone white matter, plus cortical gray matter when severe; and, a basal ganglia-distribution (BG) pattern involving deep gray nuclei, hippocampi and periolanadic cortex, with additional cortical involvement when severe. The study is important for recognizing an association between the degree of WS injury and 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>69</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 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>70</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 NE and severe NE are associated with increased morbidity.<sup>71</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.

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<sup>68</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>69</sup> See de Vries LS. Patterns of neonatal hypoxic-ischaemic brain injury. *Neuroradiology* (2010) 52: 555-566.

<sup>70</sup> See Note 1 Volpe page 339.

<sup>71</sup> *Ibid* page 74.

Comments in the green book regarding the fetal heart tracing patterns and their role in predicting acidemia are vague. The authors acknowledged, however, that there are patterns which suggest current or impending damaging acidemia. They go on to suggest that the patterns acknowledged to be associated with cerebral palsy have a very high false positive rate.<sup>72</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 timing the onset of fetal decompensation and irreversible neurologic injury. Fetal heart rate patterns are not to be used to identify when a fetus has already suffered irreversible brain damage! 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>73</sup>

## **THE PROPOSED APPROACH TO CAUSATION – DIFFERENTIAL DIAGNOSIS**

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 (International, ACOG and SOGC) 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 these unreliable principles.

While the starting point for diagnosis varies from cause to cause 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

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<sup>72</sup> Ibid page 76.

<sup>73</sup> Ibid 336-7.

also helps with timing. Timing itself can help rule out some diagnoses. There can also be multiple causes for neonatal neurologic injury.

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.

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 DIFFERENTIAL DIAGNOSIS APPROACH TO CAUSATION**

There is a professional 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.

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>74</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>75</sup> It

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<sup>74</sup> See Volpe, Note 1

<sup>75</sup> Ibid page 332.

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 than Volpe has said that such a conclusion is incorrect.<sup>76</sup>

## THE LAW

### Standard of Care

Where and how criteria set out by professional medical associations fit within the context of medical malpractice litigation is important. Although this paper is primarily concerned with causation, some law on standard of care may be instructive and will be reviewed.

The case of *Crits v. Sylvester*<sup>77</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>78</sup> In *ter Neuzen v. Korn*<sup>79</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.

### Causation

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

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<sup>76</sup> Ibid page 332.

<sup>77</sup> *Crits v. Sylvester* (1956), 1 D.L.R. (2d) 502 (Ont.C.A.), affirmed at [1956] S.C.R. 991, page 508.

<sup>78</sup> See *MacGregor v. Potts* (2009)

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

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>80</sup>

In *Snell v. Farrell*<sup>81</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>82</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>83</sup>

The reliability of scientific theories is subjected to scrutiny by our courts. Generally speaking the courts must consider whether the scientific theory has been subjected 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>84</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

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<sup>80</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.

<sup>81</sup> *Snell v. Farrell*, [1990] 2 S.C.R. 311, at page 328.

<sup>82</sup> *Allen v. Mueller*, (2002) ABCA 195, E

<sup>83</sup> *Ibid*, paragraph 19.

<sup>84</sup> See *Allen v. Mueller* 2002 Alberta Court of Appeal and *R. v. Mohan*, [1994] 2 S.C.R. 9.

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 (obstetricians), might be perceived by those on the outside as somewhat less than objective.

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>85</sup>

The appropriate use of the SOGC guidelines were in issue in the 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 artery metabolic acidosis.<sup>86</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>87</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.

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<sup>85</sup> See Note 1 page 401.

<sup>86</sup> See Note 82 paragraph 33.

<sup>87</sup> See Note 82, paragraph 35.

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>88</sup>

## **CONCLUSIONS REGARDING ASPHYXIA**

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 ACOG and SOGC 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 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.

## **MECHANICAL FORCES, HEAD COMPRESSION AND CEREBRAL ISCHEMIA**

There is substantial resistance in the obstetrical community to the notion that cerebral tissue ischemia from the stress of labour, in the absence of a global metabolic acidosis, can cause neurologic injury. Some leading authors in obstetrics have suggested that this notion is no more than a “popular legal theory” without a scientific basis.<sup>89</sup> At the same time these authors have failed to offer a reasonable scientific foundation for the rejection of this “legal theory”. The idea that labour causes direct mechanical stress on the fetal head is beyond dispute. Whether that mechanical stress can result in impaired cerebral blood flow (CBF) leading to neurologic injury without a global metabolic acidosis is the subject of much debate and of this section.

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<sup>88</sup> See McKenzie v. Fife Acute Hospitals NHS Trust, [2006] CSOH 63, at paragraph 34.

<sup>89</sup> See Freeman Note 7 page 25. He says:

“Although it has become a popular legal theory, there remains no scientific basis for the notion that cerebral ischemia caused by the pressures of labor and in the absence of fetal hypoxia with metabolic acidosis is a cause of CP.”

In exploring the notion of ischemic injury from mechanical stress, there are two secondary issues of importance that need to be considered from the medical-legal perspective. First, it is vital to determine whether there are clinical clues during labour that might suggest the risk of mechanically-induced ischemic injury<sup>90</sup>. This would include fetal heart rate patterns<sup>91</sup>, abnormal uterine contraction patterns<sup>92</sup>, and poor or arrested labour progress<sup>93</sup>. Second, there must be standards of practice for management of intrapartum care when undue mechanical stress is suggested by the clinical circumstances. In other words, we must determine whether, at least in some cases, mechanically-induced ischemic injury is preventable.

Opponents of the theory of mechanical ischemic injury often concede that neurologic injury can be caused by hypoxia or trauma. They also offer that other mechanisms (i.e., infection, hypercoagulable states, etc.) can result in injury. Sometimes, they assert, the cause is idiopathic. But mechanical ischemic injury is trauma. Ultimately the issue is concerned with the reason for a neonatal encephalopathy being encountered at birth. Determining the cause requires, as stated earlier, employing the differential diagnosis. The absence of evidence of a metabolic acidosis in arterial cord blood must be seen as only one variable in the search for a cause of NE.

Cerebral blood flow (CBF) is of obvious importance since brain ischemia is the cause of neonatal brain injury. It follows that any event that impairs CBF has the potential to cause ischemia. If this interruption in CBF is sufficient, neurologic injury results. As stated by Volpe:

...alterations in CBF are of prime importance for understanding the neuropathological and neurological consequences of all varieties of perinatal asphyxia and hypoxic-ischemic insults, as well as the pathogenesis, prevention and treatment of these consequences.<sup>94</sup>

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<sup>90</sup> For convenience the term "mechanical ischemic injury" will be used in this paper to describe intrapartum neurologic injury caused by the mechanical stress of labour resulting in a disruption to cerebral blood flow causing ischemia in the absence of a global metabolic acidosis. It also refers to injury occurring to the blood vessels and sinuses of the fetal brain leading to, among other things, subdural hemorrhage in the term fetus.

<sup>91</sup> Early decelerations are thought to be associated with head compression. While considered benign for the most part, repetitive early decelerations may not be. As well, variable decelerations in the second stage of labour are also associated with head compression.

<sup>92</sup> Tachysystole, often in the setting of the injudicious use of oxytocin, clearly increases the mechanical stresses to which the fetus is exposed.

<sup>93</sup> An arrest of labour, or perhaps even atypical progress, is likely to add to the mechanical stress. The longer it goes on the higher the risk. Poor progress must be considered as an obstacle to normal vaginal delivery.

<sup>94</sup> See Note 1. p. 291.

While a detailed analysis of the science is beyond the scope of this paper<sup>95</sup>, it is necessary to touch on some of the medicine in order to appreciate the logic of the propositions put forward. CBF is subject to *Autoregulation*, which involves, in simple terms, the maintenance of adequate oxygenation through sustaining blood flow independently from blood pressure within a certain physiologic range.<sup>96</sup> It is accepted that hypoxia can disrupt CBF<sup>97</sup>, which has an indirect effect on cerebral function.

### **Mechanical Forces of Labour on the Fetal Head and Dysfunctional Labour**

Obviously the fetal head is exposed to stresses during labour from the pressure of uterine contractions. Contractions achieve cervical dilatation and descent of the fetal head. Contact of the fetal head with the cervix and pressures associated with uterine contractions and contact with the maternal pelvis clearly result in resistance and consequent counter-pressure.<sup>98</sup> The impact that these stresses have on cerebral blood flow are crucial to any understanding of the potential for morbidity. An animal study has shown that increased intracranial pressure triggers a response to maintain cerebral perfusion.<sup>99</sup> This same study found a redistribution of systemic blood flow in response to increased intracranial pressure, much like that seen in response to hypoxia and associated metabolic acidosis.

The fetal head must pass through the lower segment of the uterus, the cervix and the vagina. The relative size of the fetal head to the passage is important and is affected by the orientation of the fetal head. Malposition of the fetal head can increase the pressures associated with contractions.<sup>100</sup>

There must be appropriate adaptation of the fetal head to pass through the birth canal. It begins with the orientation of the fetal head. Other ways in which adaptation occur include<sup>101</sup>:

- Molding<sup>102</sup>
- Caput succedaneum. This occurs as a result of differences of pressure above and below the largest circumference of the

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<sup>95</sup> An beyond the scope of the expertise of the author.

<sup>96</sup> See Note 1, p. 291.

<sup>97</sup> Volpe chapter 6

<sup>98</sup> Amiel, Tilson C. Cerebral handicap in Full Term Neonates Related to Mechanical Forces of Labour, 1988 *Obstet Gynceol* Mar 2(1):145-65

<sup>99</sup> Harris AP, Cerebral and Perpheral Circulatory Responses to Intracranial Hypertension in Fetal Sheep (1964) *Cir Res* May 5:991-1000

<sup>100</sup> Amiel, Tilson C. Cerebral handicap in Full Term Neonates Related to Mechanical Forces of Labour, 1988 *Obstet Gynceol* Mar 2(1):145-65

<sup>101</sup> Taken from Amiel-Tison, Note 48, p. 148.

<sup>102</sup> Molding is Caused by Bone Displacement and Bone Bending According to p. 157 of Amiel-Tison 1988 (Note 98)

presenting part of the fetal skull. Caput must be considered a warning for mechanical difficulties. Caput reflects excessive uterine pressure.

- Cephalohematoma. More often occurs during the second stage of labour as a consequence of subperiosteal bleeding.

Further evidence that mechanical forces are important stresses in labour includes the data showing that fetal head presentation at birth affects the degree of head molding – with occiput posterior subjected to significantly less molding than occiput anterior.<sup>103</sup>

Every labour is associated with mechanical forces. There are circumstances where mechanical forces on the fetal head are excessive as a consequence of abnormal or dysfunctional labour. Mechanical forces that are avoidable, and that ought to be avoided by observing applicable standards of care, can give rise to legal liability where the causal connection can be made between those excessive forces, a breach of the standard of care and injury. It is necessary to understand if and how a dysfunctional labour might impact on neonatal neurological status. This understanding requires a review of two important issues: first, one must appreciate what constitutes dysfunctional labour; second, the impact of dysfunctional labour on neonatal neurologic status, must be appreciated. When considering the impact of dysfunctional labour on neonatal neurologic status, it is crucial to consider neurologic injury in the setting of systemic fetal metabolic acidosis and in the setting where metabolic acidosis is absent.

Uterine contractions impose forces on the fetal head resulting in increased intracranial pressure and consequent effects on blood flow.<sup>104</sup> There is a threshold of intracranial pressure above which the impact of head compression is reflected in changes in the fetal heart rate, shown clinically by a deceleration. Decreases in CBF resulting from intracranial pressure above this threshold cause decreases in the fetal heart rate.<sup>105</sup> Thus, decelerations characterized as early (those typically associated with head compression) ought not to be considered as entirely benign. Rather, these periodic changes must be considered in the context of the entire clinical situation<sup>106</sup>, with particular regard to the factors tending to exacerbate labour stress on the fetus.

Molding and caput are clinical signs of head compression. They may be correlated with tracing abnormalities and poor neonatal outcomes. Pressure on the fetal head may impair venous return making the heart work harder to supply oxygenated blood. This phenomenon may be mechanically-induced and it

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<sup>103</sup> See p. 21 Sorbe B. Some Important Factors in the Molding of the Fetal Head During Vaginal Delivery – A Philosophic Study June 21, 1983, *Gynecol Obstet* (3);205-12.

<sup>104</sup> Amiel, Tilson C. Cerebral Handicap in Full Term Neonates Related to Mechanical Forces of Labour, 1988 *Obstet Gynceol* Mar 2(1):145-65p. 163

<sup>105</sup> Ibid

<sup>106</sup> Ibid

seems to follow logically that it can occur with or without impaired oxygenation to other systems. The issue to be address in this scenario is the effect of caput and molding on cerebral perfusion.

A 1994 study (Peebles)<sup>107</sup> found a clear relationship between uterine contractions and cerebral blood flow, particularly contractions occurring close together. The study measured cerebral blood flow by application of near infrared spectroscopy to the fetal scalp in order to measure cerebral concentration changes in oxyhaemoglobin and deoxyhaemoglobin. While this study was measuring cerebral blood flow in the context of uterine contractions, the authors presumed that the fall in intracerebral saturation was due to a decrease in oxygen supply to the placenta during contractions. Though the change in cerebral blood flow correlated with contractions, the study did not examine whether diminished placental perfusion was the only cause. The study is important for the conclusion that frequent uterine contractions are associated with changes in fetal cerebral oxygen saturation. The cause of reduced cerebral oxygenation was not studied.

### **Dysfunctional Labour**

The concept of “normal” labour is probably subject to debate. Certainly there are variations of normal, some of which depend on clinical circumstances, including maternal size, fetal size, fetal position, parity, etc. Despite any controversy regarding what constitutes a normal labour, clinicians ought to be sensitive to variations from normal, or even within normal, that might suggest a degree of dysfunction in the particular clinical circumstances. Protracted or arrested labours can reasonably be expected to increase the degree of fetal stress and thus morbidity. Ultimately the objective is a labour that results in both cervical dilatation and fetal descent within a normal time frame that avoids harm.<sup>108</sup>

Quite apart from problems associated with cervical dilatation, descent and fetal positioning, excessive uterine activity is an undesired clinical complication that increases morbidity. Excessive uterine activity may be problematic for two reasons: first, it can impair utero-placental blood flow, thereby impairing fetal oxygenation; second, it increases the frequency with which the fetal head is compressed during contractions. It, therefore, might be expected to contribute to hypoxia, hypoxia and ischemia, or brain ischemia without systemic hypoxia. Unfortunately concerns about slow cervical dilatation and descent may result in a tendency to increase uterine contraction frequency and intensity with oxytocin,

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<sup>107</sup> Peebles DM, Relation Between Frequency of Uterine Contractions and Human Fetal Cerebral Oxygen Saturation Studied During Labour by Near Infrared Specroscopy, Jan 1994, Br J Obstet Gynecol 101 (1):44-8.

<sup>108</sup> See Simpson KR, Assessment and Optimization of Uterine Activity During Labor. 2011 Clin Obs Gyn, vol 54, number 1, pp40-49. p. 43

exacerbating any impaired oxygen delivery to the fetal vital organs associated with excessive uterine activity.

As stated above, while every labour is associated with a degree of fetal stress, dysfunctional labours may result in excessive stress. Fetal ability to compensate for excessive stress is not unlimited. The issue of concern is whether this excessive stress increases fetal morbidity. Dysfunctional labour includes:

1. Prolonged latent phase;
2. Slow slope active phase;
3. Active phase arrest; and
4. Arrest of descent or prolonged second stage.<sup>109</sup>

In the absence of a fetal blood acidosis, the issue is whether the mechanical forces associated with these types of dysfunctional labours can affect cerebral blood flow leading to localized brain ischemia. Where clinical data points to a peripartum timing of injury in the absence of fetal acidosis it may follow that mechanical factors associated with dysfunctional labour may account for neonatal neurologic injury. Many of the variables that lead to a diagnosis of HIE are also of value in diagnosing IE (ischemic encephalopathy) occurring peripartum.

This is really the crux of the issue. The clinical scenario is one where all of the antepartum, intrapartum and post-partum data point to an intrapartum timing of injury in the setting of a dysfunctional labour, but with cord blood gas results that fail to reveal a metabolic acidosis. Do normal cord blood gases (leaving aside a sudden and profound asphyxia where cord gases might be normal) rule out an intrapartum injury or can intrapartum injury from dysfunctional labour (prolonged labour or tachysystole, as examples) result in neurologic injury?

### **Effects of Head Pressure on the Fetus**

A 1984 study by O'Brien and colleagues looked at the fetal response of near-term lambs to cephalic pressure.<sup>110</sup> The study is important for its findings on the impact the pressure on the fetus has on both the fetal heart rate and cerebral blood flow. To the extent that cephalic pressure affects the fetal heart rate, it needs to be appreciated clinically to avoid harm. The relationship between cerebral blood flow and cephalic pressure is important when trying to understand the causal connection between cephalic pressure and morbidity.

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<sup>109</sup> Amiel, Tilson C. Cerebral handicap in Full Term Neonates Related to Mechanical Forces of Labour, 1988 *Obstet Gynceol* Mar 2(1):145-65 p. 151

<sup>110</sup> O'Brien WF, Effect of Cephalic Pressure on Fetal Cerebral Blood Flow, *Am J. Perinata* Apr 1984, 1(3):223-6.

A 1998 study (Harris) states that fetal intracranial pressure increases during uterine contractions, which can lead to decreased cerebral perfusion pressure and cerebral ischemia together with fetal distress. There are two ways the fetus defends against increased intracranial pressure. The first is through cerebrovascular autoregulation. The second is by increasing the mean arterial pressure (MAP).<sup>111</sup> Ischemia results when either are overwhelmed.

With distress during labor, emphasis customarily is placed on hypoxemia, hypercarbia, and acidosis arising from umbilical cord compression or uteroplacental insufficiency. Fetal head compression also can evoke cardiovascular changes indicative of fetal distress."<sup>112</sup>

In 1971 Mann and colleagues<sup>113</sup> studied the effect of head compression on fetal sheep. The study looked at heart rate, cerebral metabolism and cerebral function. The study found that as CBF was impaired by intracranial pressure cerebral oxygen consumption (ischemia) decreased markedly. The authors' state:

"Compression of the fetal head by an externally applied force caused severe cerebral ischemia due to a marked reduction in cerebral blood flow. The resistance to blood flow increased as intracranial pressure was increased by vascular narrowing and collapse. The obstruction to flow prevented well-oxygenated blood in the carotid artery from reaching the fetal brain."<sup>114</sup>

In the O'Brien study, an inflatable cuff was applied to the parietal region of the fetal skull on near term lambs. Cardiac function was measured using a catheter placed into the fetal aortic arch. There were a number of important findings in this animal study that might be revealing about the response of the human fetus to cephalic pressure. The following are some of the important findings (in italics):

1. *A dramatic fall in blood flow to all cerebral tissues occurred during the early period of cuff inflation during fetal bradycardia. The extent of this fall (approximately 95%) was considerably greater than the previously reported 50% decrease as estimated by changes in carotid blood flow.*<sup>115</sup>

The importance of this finding is that head compression dramatically reduced cerebral blood flow, which is due to ischemia. The significant degree of ischemia would logically result in neuronal damage if sufficiently prolonged.

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<sup>111</sup> Harris AP 1998 p. 50.

<sup>112</sup> Ibid

<sup>113</sup> Mann LI, The Effect of Head Compression on FHR, Brain Metabolism and Function, Obstet Gynecol 1971, Vol. 39, No. 5, 721-726

<sup>114</sup> Ibid, at page 726

<sup>115</sup> O'Brien WF, Effect of Cephalic Pressure on Fetal Cerebral Blood Flow, Am J. Perinata Apr 1984, 1(3):223-6. p. 226.

2. *The decrease in cerebral blood flow appears to be due to an increased vascular resistance as demonstrated by the approximately 30-fold increase in calculated vascular resistance.*<sup>116</sup>

Presumably the increase in vascular resistance inhibits the flow of oxygenated blood to the brain due to significant ischemia caused by external pressures.

3. *Cardiac blood flow was used as a measure of flow to noncerebral tissues, as previous studies have demonstrated that flow to the brain and heart respond in a similar fashion during alterations in fetal status such as in hypoxemia. The lack of change in cardiac blood flow noted in this study demonstrates that the fall in cerebral blood flow represents a localized change rather than a fall in total cardiac output or a generalized increase in vascular resistance.*<sup>117</sup>

This is a very significant finding. It supports the notion that a reduction in cerebral blood flow induced by cephalic pressure can cause brain ischemia in the absence of a global metabolic acidosis. In other words, ischemia and neurological injury can occur without systemic hypoxemia, or outside the traditional asphyxia model. In the setting where hypoxia is the pathway to neurologic injury, one expects a building global metabolic acidosis to trigger compensatory responses (including a redistribution of systemic blood flow to the heart, adrenal glands and brain) followed by cardiac dysfunction, diminished cardiac output and ensuing brain damage. The maintenance of adequate cardiac output in the presence of cephalic pressure allows for the opportunity for ischemic brain injury with adequate cardiac function and no systemic metabolic acidosis. The injurious process is one that affects the brain in isolation, without the involvement of other systems.

4. *Although the decrease in flow was dramatic in all areas, this effect was most prominent in the cortex (95%) and least profound in the brainstem (88%).*<sup>118</sup>

This finding demonstrates a pattern of brain injury associated with cephalic pressure that mimics that associated with a prolonged partial asphyxia and associated metabolic acidosis. The presence of a pattern of brain injury that is normally associated with HIE can, based on these data, be associated with a purely mechanical ischemic injury.

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<sup>116</sup> Ibid p. 226.

<sup>117</sup> Ibid p. 226.

<sup>118</sup> O'Brien WF, Effect of Cephalic Pressure on Fetal Cerebral Blood Flow, Am J. Perinata Apr 1984, 1(3):223-6. p. 226

5. *Our results demonstrate that such pressure accompanying a dramatic fall in the cerebral blood flow is associated with alterations in pulse rate, blood pressure, and pulse pressure. This fall in blood flow appeared to be most prominent in the cortex with relative sparing of the brainstem.*<sup>119</sup>

Based on these conclusions, one would expect that cephalic pressure sufficient to impact on cerebral blood flow would cause alterations of the fetal heart rate.

Increased pressure exerted on the fetal skull is accompanied by increased intracranial pressure and in normal labour situations is tolerated by the fetus.<sup>120</sup> The ability of the fetus to adapt to these stresses results in little or no impact on the fetal heart rate. Where, however, the intracranial pressure is beyond a certain threshold, one can expect to see the impact of these stresses reflected in the response of the fetal heart rate. Decelerations associated with head compression (early decelerations) is a reflex that occurs as a result of disruption to the intracranial vascular equilibrium and therefore cannot be seen as normal.<sup>121</sup> Intracranial pressure that is too high diminishes blood flow, which is not optimal.

The impact of head compression on clinical signs of impaired cerebral perfusion through changes in the fetal heart rate has not received enough attention in the medical literature. It has been mistakenly assumed that a shallow deceleration that is synchronous with a uterine contraction is benign<sup>122</sup>, though this not necessarily so. Westgate and colleagues (2007) observed that older data establishes that bradycardia can result from pressure on the fetal head resulting from reduced cerebral blood flow associated with increased intracranial pressure.<sup>123</sup>

## **Oxytocin and Mechanical Forces of Labour**

Oxytocin is a drug used to induce or augment uterine contractions. The use of this drug is, in my view, a very important contributor to neonatal morbidity. The objective is to achieve uterine activity that is most effective in expelling the fetus without undue stress on either mother or baby. Ideally, contractions should be occurring every 2 to 3 minutes, lasting 40 to 60 seconds in duration and be moderate to strong in intensity. Normal uterine contraction frequency is generally

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<sup>119</sup> Ibid p. 226

<sup>120</sup> Amiel, Tilson C. Cerebral handicap in Full Term Neonates Related to Mechanical Forces of Labour, 1988 *Obstet Gynceol* Mar 2(1):145-65 p. 148

<sup>121</sup> Ibid p. 149

<sup>122</sup> Westgate JA, The Intrapartum Deceleration in Center Stage: A Physiologic Approach to the Interpretation of RFetal Heart Rate Changes in Labor. *Am J Obstet Gynecol* 2007 Sept, 236 e1-236 e.11

<sup>123</sup> Ibid at page 236.e2

no more than 5 contractions in a 10 minute period.<sup>124</sup> Uterine contractions are stressful to the fetus. Between contractions the uterus needs to rest and allow, among other things, the fetus to recover from any decreased perfusion resulting from a contraction.

Oxytocin is intended to increase the frequency and intensity of uterine contractions. The dose is titrated up to the level needed to produce the desired level of uterine activity. Deviations from the desired level of uterine activity can increase the risk of fetal morbidity and mortality. Uterine contractions that are too frequent, for example, may cause undue stress on the fetus and cause harm. “Tachysystole” is the term<sup>125</sup> used to describe overly frequent uterine contractions. There are other abnormal uterine contraction patterns that demand attention which will not be covered in this paper.<sup>126</sup>

Where abnormal uterine contraction patterns are associated with oxytocin use, the failure to decrease or stop oxytocin infusion can increase the risk of fetal morbidity. Medical literature establishes that oxytocin stimulation is associated with a higher incidence of head molding.<sup>127</sup> Orientation of the fetal head is another factor in head molding, with occiput posterior presentations showing less molding that occiput anterior.<sup>128</sup>

Tachysystole is known to be associated with increased risk of fetal asphyxia. Neurologic injury in this context is recognized to occur from decreased utero-placental perfusion and/or umbilical cord compression that can lead to a developing metabolic acidosis – the conventional asphyxia model. There is less appreciation for the effects of tachysystole on head compression and its impact on CBF with or without a corresponding decrease in gas exchange between mother and fetus.

The mechanical forces of labour on the fetal head will obviously be affected by any forces on the fetal head in opposition to the expulsive forces of uterine contractions. It follows that any extra resistance to expulsive forces would increase the pressure on the fetal head. Ultimately the fetal head must pass through the maternal pelvis and mechanical problems can arise that make that process more difficult. If the fetal head is not ideally oriented, the labour may not progress as desired. Dystocia is the term used for a labour that progresses at an abnormally slow rate. This can be caused by inadequate uterine activity; issues with the size of the presenting part of the fetus; or, issues with the orientation of the presenting part.

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<sup>124</sup> See Fetal Heart Rate Monitoring: Principles and Practices (4<sup>th</sup> edition), AWHONN, at page 121

<sup>125</sup> Some also called it “uterine hyperstimulation”.

<sup>126</sup> Including coupling of tripling of contractions where there is very little interval between contractions. As well, the absence of adequate resting tone between contractions can lead to fetal morbidity.

<sup>127</sup> See Sorbe note 103, at page 211.

<sup>128</sup> *ibid*

Progress of labour is measured based on the rate of cervical dilatation and the rate of descent of the presenting part (usually and ideally the head). Where dilatation and descent do not proceed at desired rates, the labour may be said to be stalled or there is an “arrest of labour”.<sup>129</sup> Where the labour has arrested in the setting of the fetal head having trouble passing through the maternal pelvis for mechanical reasons, one can expect that each uterine contraction is adding to pressure on the fetal head that has essentially encountered an immovable object. This may lead to impaired cerebral blood flow.

Medical literature has recognized molding of the head as a mechanism for injury. To the extent that abnormal uterine activity increases head molding and to the extent that dystocia is associated with orientation or size of the fetal head, one can appreciate how these two circumstances might result in neonatal brain damage. An article by Takenouchi (2012) noted several mechanisms for perinatal brain injury:

The risk for brain injury during labor may be mediated via several mechanisms. The most frequently recognized cause is regarded as secondary to an interruption of placental blood flow, a state characterized biochemically by progressive hypoxia, hypercarbia, and acidosis, and often referred to as asphyxia...

A second mechanism may result from tears in the falx and tentorium or bridging cortical veins, secondary to stretching. This tearing occurs when the anterior-posterior compression of the head is associated with excessive vertical molding and frontal-occipital elongation of the cranium, resulting in hemorrhage, e.g., into the subdural space.<sup>130</sup>

While this article does not address the notion of decreased cerebral perfusion leading to localized brain ischemia without systemic metabolic acidosis, it does acknowledge that cerebral hemorrhage can result from molding of the fetal head. An issue to be considered in the litigation context is whether there was mismanagement of intrapartum care and whether this type of injury would have been avoided had the standard of care been met.

Arguably, there are two mechanical factors that may figure in the cause of neonatal brain injury that is not of the conventional asphyxial type. One is intracranial hemorrhage associated with head compression. The other would be localized brain ischemia, also associated with head compression. Claims arising out of these causes of traumatic neonatal brain injury have seldom been pursued. The causes of these injuries need to be appreciated in order to know whether a claim might be meritorious.

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<sup>129</sup> See ACOG Practice Bulletin no. 43, December 2003.

<sup>130</sup> Takenouchi T. Changing Pattern of Perinatal Brain Injury in Term Infants in Recent Years. *Ped Neurol* 46 (2012) 106-110.

A term infant will present with a subdural hemorrhage where the injury is due to head compression with or without brain ischemia. That is, the trauma associated with head compression may cause a subdural hemorrhage with more focal brain damage than head compression that causes brain ischemia manifesting in bilateral watershed injury. Forces occurring during labour can disrupt cerebral veins and sinuses leading to hemorrhage.<sup>131</sup> The injuries with which lawyers might be concerned will arise from intrapartum issues concerned with the duration and progress of labour.<sup>132</sup> In particular, the possibility that substandard intrapartum care might be to blame for neonatal subdural hemorrhage could be present when: the baby is large relative to the birth canal; the duration of labour is unusually long, subjecting the head to prolonged compression and molding; and, when there is a difficult operative vaginal delivery (challenging forceps or vacuum delivery).<sup>133</sup>

Head compression that results in brain ischemia, rather than hemorrhage, is less well understood and not widely accepted as a cause of neonatal brain damage in the absence of systemic metabolic acidosis. This is because the pathway to injury is not the conventional asphyxia model starting with intermittent interruptions in oxygenation through impaired utero-placental perfusion or umbilical cord compression.

One must keep in mind the fact that molding of the fetal head is natural and the fetus adapts to the pressure of passing through the birth canal by shifting of the bones of the skull to allow it to “squeeze” through. We are concerned with excessive molding and scenarios where the fetus was unnecessarily exposed to head compression where it should have been apparent that safe vaginal delivery was unlikely. Further, in some scenarios operative vacuum delivery merely serves to increase the risk of brain lesions.<sup>134</sup>

There are animal studies that have looked at the effects of cephalic compression. The study by O'Brien (1984)<sup>135</sup> looked at the effect of cephalic pressure on fetal lambs. An inflatable pressure cuff was placed on the lambs head. Significant to the issue of brain ischemia, the study found that with pressure to the head there were significant decreases in flow to all cerebral tissues, but no significant change in cardiac blood flow. Further, there was no significant change in pO<sub>2</sub> or pH measurements (meaning no systemic metabolic acidosis).<sup>136</sup> Finally, while blood flow to cerebral tissues was generally reduced, the effect was most prominent in the cortex. As well, a drop in cerebral blood flow was associated with alterations in pulse rate, blood pressure and pulse pressure.<sup>137</sup>

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<sup>131</sup> See note 1, Volpe, at page 486.

<sup>132</sup> Ibid, at page 488.

<sup>133</sup> Ibid.

<sup>134</sup> See Lindgren L. The Influence of Pressure Upon the Fetal Head During Labour. *Acta Obstet Gynecol Scand* 56:303-309, 1997, at page 309.

<sup>135</sup> See note 110.

<sup>136</sup> Ibid, page 224.

<sup>137</sup> Ibid, page 226.

The article by Amiel-Tison (1988)<sup>138</sup> contains some important observations regarding the potential for head compression to cause localized brain ischemia, including:

1. An increase in pressure on the fetal skull is followed by a parallel increase inside the fetal cranium;<sup>139</sup>
2. Normal intracranial vascular pressure would not be expected to cause significant changes in the fetal heart rate;<sup>140</sup>
3. Intracranial pressures that prompt changes in the FHR are due to an imbalance between the sympathetic and parasympathetic vagal nerve systems;<sup>141</sup>
4. Early decelerations associated with head compression should not be considered benign. These occur only when the intracranial pressure is too high, causing diminished blood flow;<sup>142</sup>
5. Excessive pressure on the fetal head can jeopardize intracranial blood flow and provoke slowing of the FHR;<sup>143</sup>
6. The level of tolerance to pressure of the fetal head is revealed by the FHR;<sup>144</sup>
7. One must be concerned with caput succedaneum which reflects excessive pressure on the head and prevents spontaneous rotation.<sup>145</sup>

Despite these observations, Amiel-Tison cautions that a precise correlation between dysfunctional labour and neonatal outcome is not well-established.<sup>146</sup> Often there is a component of the asphyxial model of injury occurring at the same time as head compression. Separating the effects of mechanical factors from other causes of asphyxia is challenging. This is, indeed, the challenge lawyers face in investigating birth trauma cases that do not match up with the conventional asphyxia model.

## **STANDARD OF CARE AND MECHANICAL ISCHEMIC BRAIN INJURY**

If one accepts that mechanical factors can cause neonatal neurologic injury, then one must determine whether there was a breach of the standard of intrapartum care that led to the injury. To state again, the mechanical factors are either head compression resulting in subdural hemorrhage (in the term baby) or head

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<sup>138</sup> See note 98.

<sup>139</sup> Ibid, page 148.

<sup>140</sup> Ibid.

<sup>141</sup> Ibid, page 149.

<sup>142</sup> Ibid.

<sup>143</sup> Ibid.

<sup>144</sup> Ibid, at page 150.

<sup>145</sup> Ibid, at page 151.

<sup>146</sup> Ibid.

compression resulting in brain ischemia. As in the asphyxia model, the clinical developments that might suggest a problem include:

- a) Periodic changes in the fetal heart tracing, particularly early decelerations;
- b) Tachysystole or uterine hyperstimulation;
- c) Abnormally slow progress of cervical dilatation and descent of the presenting part, or an arrest of labour;
- d) The presence of caput or molding.

In the context of these clinical developments, the defendant obstetrician needs to be asked about what intrapartum clinical factors led her to conclude that there was a reasonable prospect of a safe vaginal delivery. As well, informed consent becomes prominent in these clinical settings as the mother is entitled to know the risk of a continued trial of labour.

The real challenge faced in litigating these cases is proving when intervention was required by the applicable standard of care and how intervention at that time would have, on a balance of probabilities, prevented the adverse outcome. This poses a considerable challenge for lawyers representing these clients and for their expert witnesses. It requires a thorough understanding of the literature, the pathophysiology of injury and the challenges to proof. Despite the obvious difficulties, it is my view that cases of injury due to head compression can be won by the plaintiff – just not easily.