I. BRAIN DAMAGE IN LABOR AND DELIVERY CASES

A. Trauma.

The first person to seriously examine the interaction between the fetal head and the maternal pelvis was the Dutch obstetrician Hendrik van Deventer (1651-1724). He was the first to emphasize that pelvic contractions are a factor in delayed or difficult labor. He also recognized that head molding could result in brain injury.[1] William Smellie recognized the dangers of trauma and excessive molding before the Revolutionary War. In 1752 he wrote:

> In lingering labor, when the head of the child has been in the pelvis so that the bones ride over one another and the shape is prenaturally lengthened, the brain is frequently so much compressed that violent convulsions ensue before or soon after delivery to the danger and oft times the destruction of the child.[2]

Approximately 150 years ago, William Little, M.D., published his classic work: *On the Influence of Abnormal Paturation, Difficult Labours, Premature Birth and Asphyxia Neonatorum, on the Mental and Physical Condition of the Child, Especially in Relation to Deformities*.[3] The work was so significant, that the resulting diagnosis of cerebral palsy became known as "Little's Disease" for years afterward. In 1862, Little stated:

> We are acquainted, for example, with abundant instances of deformities arising after birth from disorders of the nervous system-disorders of nutrition, affecting the muscular and osscous structures, disorders from malposition and violence.[4]

Dr. Little observed that in a previous publication he had showed that premature birth, difficult labours and mechanical injuries to the head and neck were apt to be succeeded by an effect on

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[1] Kriewall, T. et al, "Effects of Uterine Contractility on the Fetal Cranium," Chapter 7, *A Short History of Obstetrics and Gynecology* (1960).

[2] *Id.*

[3] Little, W., *On the Influence of Abnormal Paturation, Difficult Labors, Premature Birth and Neonatorium, on the Mental and Physical Condition of the child, Especially in Relation to Deformities*, Trans. Obstet. Soc. (London) 3:251 (1862), republished at Clin Orthop Relat Res. 46:7, (1966).

[4] *Id.* at p. 7

the limbs of the child, which he designated as spastic rigidity of the limbs.[5] It was obvious even then to Dr. Little that many of these injuries were traceable to the birthing process:

> It is obvious that the great majority of apparently stillborn infants, whose lives are saved by the attendant accoucheur, recover unharmed from that condition. I have, however, witnessed so many cases of deformity, mental and physical, traceable to causes operative at birth, that I consider the subject worthy the notice of the Obstetrical Society.
>
> . . .
>
> I believe I am now enabled to form an opinion of the nature of the anatomical lesions and the particular abnormal event at birth on which the symptoms depend.[6]

He observed that the forces of labor and delivery were potentially dangerous to the fetus. These forces included an increase to intrauterine pressure attendant to contractions. (And this was almost 100 years before Pitocin and other drugs began to be used to increase uterine contractions):

> This pressure is at first intermittent, the duration of the period of repose at first greatly exceeding the period of disturbance; as the final exit approaches, the pressure simply remits, until at length it is so considerable that prompt escape from the mother alone prevents mischievous results to the nascent organism.[7]

Little observed that trauma to the fetal head during labor and delivery could cause injury to the fetal brain. And he described some of the mechanisms known at that time:

> Doubtless in some of the instances I have recorded sufficient mechanical injury to head and neck was inflicted to account for whatever unfavorable consequences, whether these were fatal or not, may have ensued.
>
> . . .
>
> F. Weber ('Beiträ zur Pathologischen Anatomie der Neugebornen,' Kiel, 1851-54) found laceration of dura matter and effusion of blood between it and the bones, rupture of longitudinal and transverse sinuses of brain and considerable

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[5] *Id.* at p. 8.

[6] *Id.*

[7] *Id.* at p. 9.

> haemorrhage on the surface and base of brain, sometimes sufficient to envelop cerebellum and oblongation cases in which mechanical injury to bones of the head had occurred, whether or no instruments had been used to complete the delivery.[8]

Little studied a series of cases which supported his conclusions:

> A survey of the history of forty-seven cases, appended, shows that one fact is common to all the cases of persistent spastic rigidity of new-born children, namely, that some abnormal circumstance attended the act of parturition, or rather, the several processes concerned in separating the foetus from the parent and its establishment in the world as an independent being.
>
> . . .
>
> But general spastic rigidity I have, with one exception, found to have been preceded by some abnormal act connected with mode of birth.[9]

Even 150 years ago, it was obvious that injuries could result from trauma, pressure on the fetal head and compression of the fetal skull. Little continued:

> North ('Practical Observations on the Convulsions of Infants,' 1826, p. 52)—says "It cannot be doubted that convulsions occasionally arise from excessive and long-continued pressure of the head during protracted labour.
>
> . . .
>
> Smellie ("Midwifery". 1777, vol. I, p. 230) alludes to convulsions before or soon after delivery from compression of head, to the danger, and oft-times the destruction, of the child.
>
> . . .
>
> The severe lesions caused by mechanical compression and laceration, and extensive hemorrhages within the skull, when they do not destroy life, give rise to permanent deformity of cranium, to atrophy of injured portions of brain, and are the cause of many cases erroneously described as *congenital* idiocy. Dr. J. Crichton Browne ("Psychical Diseases of Early Life." 'Journal of Mental Science,' April, 1860) is one of the few observers who have traced idiocy to difficult labours (see also Dr. Howe, 'Causes of Idiocy,' Edinburg, 1858).[10]

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[8] *Id.*

[9] *Id.* at p. 13.

[10] *Id.* at p. 14-15.

Obvious advancements have been made in medical science and in clinical practice. However, there has been no doubt for hundreds of years that trauma, uterine hyperstimulation and compressive forces on the fetal skull have great possibility for injury to the baby during labor and delivery.

As Yates stated ~~almost~~ over fifty years ago, "[b]irth is a very traumatic event. This is particularly emphasized by the frequency of intra-cranial hemorrhage and cerebral damage in the newborn infant."[11] Modern obstetrical literature is filled with a variety of studies that examine the empirical relationship between the force and frequency of uterine contractions, the progress of labor and the effect on the fetal head. There are numerous statistical studies that look at the retrospective relationship between perinatal outcome and intrapartum factors such as the length and force of labor and the method of delivery.[12] For example, over 40 years ago (as the use of contraction enhancing drugs was becoming more commonplace), Clyne published his study. He first reviewed Courville's earlier study of 446 stillbirths or neonatal deaths in 10,000 consecutive autopsies done in Los Angeles. Courville had concluded:

> Distortion of the head is the main cause of physical injury, and gross subdural haemorrhage was its most characteristic manifestation. The second commonest traumatic lesion is a mixture of laceration and compression of brain tissue.[13]

Clyne then compared those results to his study of 2,122 stillbirths and 1,867 neonatal deaths which occurred in Scotland in 1962. In Scotland, Clyne summarized his results as follows:

> It will be seen that the stillbirths and neonatal deaths in Scotland fell into four main groups:

	Cases	Per cent total
1. Asphyxia	2,051	51.05
2. Foetal Defects Group	859	21.48
3. Difficult Labour and Birth Injury	473	12.27

[11] Yates, P., "Birth Trauma to the Vertebral Arteries, Archives of Disease in Childhood", 436 (1959).

[12] Kriewall at p. 295.

[13] Clyne, D., "Traumatic versus Anoxic Damage to the Foetal Brain," Develop. Med. Child Neurol. 6:455 (1964).

4. Other causes	606	15.20[14]

Clyne confirmed Courville's study, and confirmed that mechanical trauma as well as lack of oxygen to the fetal brain during labor and delivery carried serious consequences.[15] As neuroimaging techniques developed and refined, the analysis of traumatic and ischemic injury due to excessive forces in labor and delivery have become more refined. Govaert studied a series of such cases:

> Ischemia within the regions supplied by the cerebral and posterior cerebral arteries has been described as complication of birth injury, either by direct trauma or by compression from a herniated temporal uncus. Ischemia within the territory of the middle cerebral artery has been documented after a stretch injury of the vessels elastica eterna.[16]

Govaert described a series of seven personal observations of birth trauma related to cerebral stroke. He found three neonates with an uncal herniation type of occipital stroke and four with hypoperfusion of the middle cerebral artery or one of its major branches. In three of those four, he found a basal convexity subdural hemorrhage, probably induced by ischemia. In the other, it was associated with a hemorrhagic contusion of the parietal lobe.[17]

Govaert described mechanical birth injury from trauma, resulting in injury to intracranial arteries in four different patterns: (1) laceration of the middle meningeal artery after difficult instrumental delivery; (2) stretch injury of the vertebral artery and its branches; (3) occlusion of the posterior cerebral artery after uncal herniation due to increased intracranial pressure; and (4) stretch injury of the middle cerebral artery in the absence of supratentorial subdural bleeding.[18] Govaert described several ischemic patterns of injury as a result of physical trauma during the

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[14] *Id.* at p. 457.
[15] *Id.*
[16] Govaert, P., et al., "Traumatic Neonatal Intracranial Bleeding and Stroke", Arch. Dis. Child, 67:840 (1992).
[17] *Id.*
[18] *Id.*

birthing process, both as a result of disruption of intracranial vessels and also as a result of increased intracranial pressure.[19] It should be noted that the midcerebral artery is vulnerable to traumatic injury, resulting in neonatal stroke in operative vaginal deliveries, particularly when forceps or vacuum extraction is forcefully applied or misapplied. Four factors must be kept in mind in analyzing these injuries. First, the midcerebral artery originates off of the carotid immediately anterior to the back of the skull. This is the most stable part of the inner cranium. Second, the midcerebral artery is delicate and has a convoluted course after its origin. Third, the neonatal brain has a relatively high water content. Fourth, the delicate cerebral arteries provide much of the strictural support for the cerebrum. With these factors in mind, excessive force by misapplication of forceps or a vacuum extractor can traumatize and disrupt the intimal layer of midcerebral artery. If this occurs, it will trigger the clotting cascade, resulting in a stroke distal to the intimal disruption.

There can be no doubt that excessive forces of labor caused by excessive contractions can indeed lead to traumatic and ischemic brain injury in the newborn.

1. Fetal Head Molding

One of the mechanisms by which abnormal forces of labor impart traumatic physical damage to the neonatal brain is through excessive head molding. Head molding during labor and delivery refers to changes in the cranial bone relationships that occur in response to the compressive forces of uterine contractions.

> It primarily refers to bony adjustments rather than soft tissue swelling. The change in shape is possible because of the pliability of the bones and the loose connection they have with one another at the sutures. The individual response of the normal fetal cranial bones to force is variable, and depends on a number of factors, including head position, labor character, and gestational age. In general,

[19] *Id.* at 843-845; Steinbrok, P., et al., "Acute Subtotal Hematora Associated with Cerebral Infarction in the Full Term Neonate," Pediatr. Neurosurg., 23:206 (1995).

> however, the typical molded newborn head is elongated and cylindrical, reflecting misalignment among the bones of the cranial vault (parietal, frontal, and occipital bones).[20]

It is well-known that excessive head molding from excessive forces of labor may result in cranial birth injuries from both physical trauma and also from a decrease in cerebral perfusion. In an article entitled "Adverse Perinatal Effects of Early Amniotomy During Labor," the authors observed that cranial molding may produce lesions on the fetal brain and they also cited research that concludes that displacement of cranial bones is an obvious cause of subdural hemorrhage, frequently located near the sutures.[21] Moreover, when the protrusion of the parietal bones becomes very marked, it may tear the cerebral flax and cerebellar tentorium with consequent hemorrhage.[22] According to Barkovich in *Pediatric Neuroimaging*, "the causes of both falx and tentorial tears seems to be excessive vertical molding of the head with frontal-occipital elongation."[23] In *Tentorial Hemorrhage Associated with Vaccum Extraction*, the authors observed that tentorial hemorrhage is commonly associated with mechanical injury to the fetal cranium and that its occurrence has been related to shearing forces on the tentorium.[24] In two of the patients studied, the tentorial hemorrhage was associated with ischemic complications.[25]

The decrease in perfusion results from an increase in intracranial pressure or decrease in venous return, or both in combination. Molding and excessive molding and their effects have

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[20] Carlan, S., et al., "Fetal Head Molding, Diagnosis by Ultrasound and a Review of the Literature," Journal of Perinatology, XI (2):105 (1991).

[21] Caldeyro-Barcia R., "Adverse Perinatal Effects of Early Amniotomy During Labor," Chapter 32, *Modern Perinatal Medicine*, (1974).

[22] *Id.* at 438.

[23] Barkovich, A. James, M.D., "Brain and Spine Injuries in Infancy and Childhood" *Pediatric Neuroimaging*, 3rd ed. (2000).

[24] Hanigan, W., et al, "Tentorial Hemorrhage Associated with Vacuum Extraction," 85 Pediatrics 534-539 (1990).

[25] *Id.* at p. 536.

been well-studied in infants, in autopsies, in biomechanical engineering models and in animals.[26] All demonstrate the same medical facts. Molding increases intracranial pressure. This decreases blood flow to the brain. Molding also depresses the venous sinus return and, likewise, decreases blood flow to the brain. Excessive head molding from excessive forces of labor causes a decrease in blood flow to the fetal brain and also traumatic injury. For example, over 30 years ago, Newton studied neonates, cadavers and dogs.[27] He found:

> Compression of the superior sagittal sinus may result from overlapping of the parietal and occipital bones in the newborn infant.
>
> . . .
>
> Overlapping of the parietal and occipital bones at the lambdoid suture is common in the newborn skull. Although previously considered to be benign, this molding may have serious consequences. Parietal-occipital overlap may compress the superior sagittal sinus (SSS) and slow cerebral circulation. Such compression may increase cerebral venous pressure and precipitate intracerebral hemorrhage, a common complication in neonatal respiratory distress syndrome.
>
> . . .
>
> The effect of parietal-occipital overlapping in one infant was shown by carotid arteriography and by venography of the sagittal sinus. Parietal-occipital overlap and its effect on the superior sagittal sinus were also studied by venography in 6 infant cadavers.[28]
>
> Changes in the heart rate and blood flow in the superior sagittal sinus associates with various degrees on anteroposterior compression of the skull were documented by electrocardiography and Doppler ultrasound in normal newborn infants.
>
> . . .
>
> The pressure in the SSS to varying degrees of obstruction was measured in six dogs.[29]

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[26] Towbin, A., *Brain Damage in the Newborn and its Neurological Sequels* (1998). Please see Figures 206 and Figure 207.

[27] Newton, T., et al., "Compression of Superior Sagittal Sinus by Neonatal Calvarial Molding," Radiology 115:635 (1975).

[28] *Id.* at p. 635.

[29] *Id.* at p. 636.

Newton's results demonstrate a decrease in blood flow to the brain in a live neonate:

> Carotid angiography in an 8-day old infant, with moderate parietal-occipital overlapping, demonstrated extremely slow cerebral circulation... The superior sagittal sinus did not fill and the veins drained toward the deep cerebral venous system. Direct injection into the superior sagittal sinus confirmed its patency and showed marked compression of the sinus at the point of parietal-occipital overlap.[30]

In cadavers, Newton found:

> Superior sagittal sinus in the 6 cadavers showed the degree of compression of the superior sagittal sinus to be directly related to the amount of anteroposterior compression applied to the head. With forceful pressure, parietal-occipital overlap was produced to a severe degree; this significantly compressed the superior sagittal sinus in all infants.[31]

And in dogs, Newton found:

> Intermittent pressure applied through the posterior burr hole to the SSS in dogs caused an abrupt rise in its pressure to a level two to three times normal baseline.
>
> . . .
>
> When obstruction was released, the SSS pressure returned to normal. The degree of SSS pressure was directly related to the degree of obstruction. No changes in heart rate were observed.[32]

In the late 70's, Lindgren likewise studied the traumatic effects of fetal head molding.[33]

Lindgren observed:

> In contracted pelvis, moulding of the skull bones is caused by the force of the amniotic fluid pressure and the resistance of the pelvis. In normal labour and abnormal uterine action the moulding of the skull bones is caused by the head to cervix pressure, as has been demonstrated by intra-uterine tokometry.[34]

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[30] *Id.*

[31] *Id.*

[32] *Id.*

[33] Lindgren, L., "The Influence of Pressure Upon the Fetal Head During Labor," Acta Obstet. Gynecol. Scand., 56:303 (1977).

[34] *Id.* at p. 303.

Lindgren had studied molding previously and found:

> The fetal head in particular is affected by various pressures which cause moulding of the skull bones, and fractures are sometimes seen. The mouldings may cause rupture of the tentorium, and cerebral hemorrhage. In a conservatively treated material from Sabbatsbergs Hospital, 1949-1959, comprising 23,836 infants, 647 died perinatally. Of the infants, 17.3% showed rupture of tentorium (Lindgren et al., 1962). By active obstetric management the risk has been reduced, but we do not know how many children survived who had cerebral palsy or mental retardation caused by these pressures.[35]

In the later study, Lindgren used strain gauges to study the biomechanics of molding in labor.

He found:

> By using the method of intra-uterine tokometry we found that during contractions in vertex presentation the head to cervix pressure is on average three to four times higher than the corresponding amniotic fluid pressure. The simultaneous recorded head to cervix pressure decreases towards the lower pool of the fetal head. During the first stage of labour the head to cervix pressure at the equator of the fetal head is on average the same at the same amniotic fluid pressure throughout the first stage, except after rupture of the membranes when the head to cervix pressure at the equator increases and the pressures at lower levels decrease. As the amniotic fluid pressure increases during the progress of the first stage of labour the head to cervix pressure at the equator of the fetal head also increases...
>
> The high head to cervix pressure at the equator causes a moulding of the skull bones. The parietal bones are elevated in relation to the frontal and occipital bones, giving a level difference in the coronal and lambdoid sutures. This moulding increases during the progress of labour...[36]
>
> . . .
>
> In some cases of hypertonic inertia, spastic contractions occur in the annular musculature of the lower part of the corpus. The head to cervix pressure increases and the moulding likewise. The moulding is otherwise of the same type as in normal labour. The dislocation of the skull bones can be large –up to 25 mm in this type of hypertonic inertia. In a material of 56 such patients all monitored and selected among women with violent contractions, 16 or (29%) of the infants died, all due to rupture of the tentorium.[37]

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[35] *Id.*

[36] *Id.* at pp. 303, 305.

[37] *Id.* at p. 307.

Five years later, Sorbe and others studied 319 vaginal deliveries.[38] They used a photographic method to measure the size and shape of the fetal head at delivery and three days later. They calculated a molding index and compared it with the conventional occipitofrontal circumference. They found:

> The mechanical forces of labor subject the infant's head to considerable compression, shearing, and molding. Intrapartum and neonatal death can occur from mechanical trauma to the brain during birth.[39]
>
> . . .
>
> The region of the brain in greatest jeopardy is determined by the spatial orientations of the head as it descends through the maternal pelvis.[40]

Importantly, they looked at the effect of Pitocin (Oxytocin) on molding. Not surprisingly, they found:

> Oxytocin stimulation was used for hypotonic inertia. Infants born after such labor had significantly higher molding indices (1.74) than those born after normal labor (1.68) ($P < 0.01$). Three days postpartum there was still a significant ($P < 0.05$) difference between the molding indices of the two groups.[41]
>
> . . .
>
> The rate of cervical dilation is of importance in producing the greater deformation of hypertonic inertia but it is of no great importance in hypotonic inertia.
>
> Cerebral hemorrhage (rupture of the tentorium cerebelli) is 15 times more common as a cause of infant death in primary inertia than in normal labor. The explanation seems to be the high pressures to which the fetal head is subjected in hypertonic inertia.[42]

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[38] Sorbe, B., et al., "Some Important Factors in the Molding of the Fetal Head During Vaginal Delivery-A Photographic Study," Int. J. Gynaecol. Obstet., 21:205 (1983).

[39] *Id.* at p. 205.

[40] *Id.* at p. 206.

[41] *Id.* at p. 211.

[42] *Id.*

Lapeer and others studied fetal head molding from a biomechanical engineering perspective.[43] They presented a non-linear model of the deformation of a complete fetal skull during the first stage of labor. The authors noted that:

> Fetal head moulding is a phenomenon which may contribute to satisfactory progress during delivery as it allows the fetal head to accommodate to the geometry of the passage. In contrast, excessive head moulding may result in cranial birth injuries and thus affect the infant shortly or even long after birth.[44]

They also noted that:

> Excessive moulding occurs when labour is prolonged or when contractions are too forceful or when there is a malposition of the fetal head or inept instrumental interference. Excessive displacements of the skull bones may cause bony lesions, dural membrane injury, intracranial hypertension, congestion of the Galenic venous system and direct injury of major intracranial vessels.[45]

Importantly, but not surprisingly, they found that the pressure on the fetal skull increased at higher cervical dilatations:

> The experiment described in this section involves the evaluation of the entire skull when subjected to the IUP [intrauterine pressure] and HCP [head cervix pressure]... We saw that higher dilatations result in significantly higher HCPs. Experiments at different dilatations as reported in Lapeer (1999) showed that higher HCPs corresponded to higher degrees of moulding.[46]

That is to say, as the mother dilates, the pressure on the fetal head increases dramatically, as does the potential for excessive molding and injury. Lapeer's findings, while from a different perspective, were consistent with those who had otherwise studied head molding:

> Despite the use of a relatively small number of parameters compared to the many involved in the birth process, and the potentially large variation within this small set of parameters, the model shows good agreement with clinical experiments, both in terms of *shape after deformation* and the *degree of deformation.* Moreover, the lifting of the parietal bones, is a commonly known phenomenon in

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[43] Lapeer, R. et al., "Fetal Head Molding: Finite Element Analysis of Fetal Skull Subjected to Uterine Pressures During the First Stage of Labor," J. of Biomechanics, 34:1125 (2001).

[44] *Id.* at p. 1125.

[45] *Id.*

[46] *Id.* at p. 1129.

> the obstetric and paediatric communities and has previously been reported in Govaert (1993), Lapeer (1999) and McPherson and Kreiwall (1980b).[47]

As a final note, Lapeer pointed out that:

> The actual IUP [intrauterine pressure] changes during time with a frequency of about 18-30 uterine contractions per hour (Lindgren, 1977).[48]

This is the same as three to five contractions in a 10-minute period. Five or more contractions in a 10-minute period is hyperstimulation by definition.[49]

In an article published in the *American Journal of Obstetrics & Gynecology* in 1963, John V. Kelly, M.D., noted that compression of the fetal skull may produce brain damage by one of three mechanisms:

> 1. The increased pressure is transmitted inside the calvarium where it may overcome the intravascular blood pressure resulting in the arrest of the cerebral circulation. The ensuing development of anoxia and asphyxia may damage not only the brain cells, but also the blood vessel walls, making them liable to rupture when exposed to hypertension.
>
> 2. It will be recalled that the brain is covered by two protective envelopes, the dura (with fibrous tentorium cerebelli and falx cerebri) and the calvaria. The dura represents a framework which fixes the brain to the skull bones and supports the cerebral blood vessels. The anterior margins of the tentorium cerebelli and the inferior portions of the falx cerebri are characterized by thickened hands of connective tissue, "stress bands," which represent protection against stretching. Such stretching occurs whenever the mobile and separated skull bones are distorted due to cerebral compression. The resultant pull and stretch of the tentorium of the falx may cause them to tear. If the laceration extends into the venous sinuses, bleeding into the confined subdural space occurs and the pressure of the accumulating blood may damage vital centers.
>
> 3. Severe compression on the fetal head may cause a fracture of a skull bone with laceration or direct injury of the underlying brain tissue.[50]

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[47] *Id.* at p. 1132.

[48] *Id.* at p. 1133.

[49] Lite.

[50] Kelly, John V., M.D., "Compression of the Fetal Brain," Am J Obstet Gynecol 85:687 (1963).

Lennart Lindgren studied the effects of pressure gradient on the fetal cranium. He observed that during labor the fetus is influenced mainly by two stress factors -- decreased oxygenation and pressure of the uterine contractions.[51] He concluded that the fetal cranium is subjected to pressures of various kinds and magnitudes that result in various types of deformation of the fetal skull bones during the labor process.[52] Another result of this pressure is various kinds of cerebral lesions.[53]

2. Common Cases Where Traumatic Brain Injury Occurs in Labor & Delivery

A. Forceps – what to look for
   1. Informal consent
   2. Misapplication
      a. High forceps delivery
      b. Midforceps delivery
      c. Length of time pulled
      d. Number of pulls
      e. Force used
      f. Rotation
      g. Delay in delivery
      h. Forceps marks on baby's head
      i. Correlation to neuroimaging

B. Vaccum Extraction – what to look for
   1. Informed consent
   2. Misapplication
      a. Not properly placed on baby's head
      b. Too many pulls
      c. Too much pressure
      d. Number of pulls
      e. Force used
      f. Pop offs
      g. Failed vacuum and forceps – don't go to one after other failed
      h. Repeated attempts cause injury

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[51] Lindgren at p. 357.

[52] *Id.* at 423.

[53] *Id.*

3. Pitocin - Hyperstimulation and Decrease in Perfusion

Abnormal strength and/or frequency of contractions may cause devastating injury to a baby's brain even absent excessive molding and apart from the effect upon the placental. It is well-known and undisputed that an increase in intracranial pressure above the mean-arterial pressure results in a decrease in perfusion to the brain.[54] It is likewise well-known that the forces of uterine contraction during labor and delivery cause an increase in intracranial pressure and a decrease in cerebral perfusion. This has been well-studied in animals and humans. It is well-known to the medical community throughout the world.

Over 30 years ago, Mann and others prospectively studied the decrease in cerebral blood flow as a result of increased extracranial pressure on sheep.[55] Obviously there are ethical proscriptions from doing such experiments on humans. In their study, they first surveyed the literature regarding the effect of contractions of fetal cerebral blood flow in humans:

> Schwarcz *et al* used flat pressure receptors introduced between the uterine wall and fetal head to evaluate the pressure exerted by uterine contractions on the head. Receptors placed at the level of the equator (plane of largest diameter) of the fetal head recorded pressures with a uterine contraction that were up to 2.5 times higher than amniotic pressure. As the distance from the equator increased, pressure decreased and equaled amniotic pressure. Recorded at the equatorial zone, pressure increased with rupture of the membranes and descent of the fetal head. The difference between amniotic fluid pressure and the higher pressures recorded at the equatorial zone represent pressure due to the resistance offered by muscular and bony pelvic structures. If the area upon which the pressure is exerted is unchanged, then the force (Force = Pressure x Area; kilograms or pounds) is similarly increased.
>
> Fleming and associates modified a forceps, by adding strain gauges, to study traction and compressive forces exerted on the fetal head during forceps delivery. Ullery *et al*, Kelly and Pearse reported an average instrumental traction of approximately 30 to 50 pounds and an average head compression of 5 to 6 pounds (2 to 3 kg) during forceps delivery. *The force exerted on the fetal head with*

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[54] Volpe at p. 307.

[55] Mann, L., et al., "The Effect of Head Compression of an FHR, Brain Metabolism and Function," Obstet. Gynecol., 39(5):721 (1972).

forceps application and delivery is quite similar to that exerted by a contraction with an amniotic fluid pressure of greater than 40 to 50 mm Hg.[56]

Otherwise stated, the pressures exerted in the fetal head in an operative vaginal delivery are the same as in vaginal delivery. So are the decreases in blood flow to the fetal brain. As to the actual experiment, Mann demonstrated a dramatic decrease in blood flow to the brain.[57]

Thirty experiments were conducted on 15 fetuses of mixed breed ewes.[58]

	Heart Rate Beats/min	pO_2 mm Hg	Ph	Perfusion Pressure Mm Hg	Resistance mm/L/min	Blood Flow ml/100 g/min	Q O_2 ml/100g/min
Prior to Head Compression	130		18	7.258	39	539	237
3.32							
Head Compression – 1 min	128		17	7.255	48	5574	28
.76[59]							

Mann concluded that:

> [c]ompression 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 intracerebral 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. As soon as the compressive force was released, blood flow returned rapidly and once again the brain was well oxygenated.[60]

Subsequently, O'Brien and others did similar experiments on near term fetal lambs.[61]

O'Brien found that:

> Tissue blood flows for heart, cortex, subcortex, brainstem, and cerebellum prior to and during the early period to cuff inflation are demonstrated... Highly significant

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[56] *Id.* at p. 721 (emphasis added).

[57] *Id.* at p. 721.

[58] *Id.* at p. 722.

[59] *Id.* at p. 724.

[60] *Id.* at p. 725.

[61] O'Brien, W. et al., "Effect of Cephalic Pressure on the Fetal Cerebral Flow," Amer. J. of Perinatology, 2:223 (1984).

> decreases in flow to all cerebral tissues were noted. The overall decrease in flow approached 95%. There was no significant change in cardiac blood flow. Vascular resistance was calculated by division of the mean arterial pressure by the measured blood flow. Cerebral vascular resistance increased from 0.37 to 11.9 units (1 unit = 1 mm HG/mL/min/100 g). Coronary vascular resistance was unchanged (0.28 vs 0.34 units).[62]

Over ~~Almost~~ 20 years ago, Amiel-Tison and others described the effects on uterine contractions and blood flow to the fetal brain – and the potential for cerebral injury as a result.[63] She first discussed various potential mechanisms for injury:

> CNS [Central nervous system] injury related to labour must represent a response to several factors: (1) duration and severity of asphyxia, e.g. umbilical cord compression or abruption placentae; (2) the ability of a given fetus to tolerate stress, e.g. the well-known limited tolerance to stress observed in post-term or intrauterine growth-retarded (UGR) fetuses; and (3) the circumstances under which the fetus is being stressed, e.g. when excessive mechanical forces are applied to the fetal head.[64]

She then discussed what was, and is, well-known about the forces of labor and delivery. That is, tremendous pressures are generated and visited upon the fetal head during labor:

> The main consequence of uterine contractile activity is the transmission of forces to the presenting part by means of *either* the so-called "axial pressure", i.e. local pressure exerted by the fundus on the fetal buttocks and transmitted by the spine to the head, or by a uniform increase of pressure in the uterine cavity. This debate has been a matter of long-standing controversy (see in particular the book of Rydberg (1954) on this matter). No matter how these pressure are transmitted, their main consequences are cervical dilatation and descent of the fetal head. Both are associated with strong counter-pressure exerted on the fetal head by the cervix and by pelvic structures, i.e. bones and soft tissues.
>
> The studies of Lindgren (1960, 1968, 1981; Lindgren and Sienner, 1966) have clearly demonstrated two points concerning the cervix: (1) cervix-to-head pressure is high, much higher than the intrauterine pressure, in fact up to three to four times the intrauterine pressure, depending on the clinical situation and the

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[62] *Id.* at p. 224.

[63] Amiel-Tison, C., et al., "Chapter 10 Cerebral Handicap in Full Term Neonates Related to the Mechanical Forces of Labor," Bailliere's Clinical Obstetrics and Gynecology 2(1):145 (1988).

[64] *Id.* at p. 145.

> state of the fetal membranes: and (2) the cervix-to-head pressure remains high, even *between* contractions, a point which must be emphasized.[65]

And these forces decrease the blood flow to the baby's brain:

> The mechanical events during the first stage of labour are reviewed, showing how uterine contractions result in cervical dilatation and descent and rotation of the fetal head. The consequences of these forces on the fetal intracranial pressure and blood flow are discussed: FHR remains normal up to a certain pressure threshold, above which decelerations occur. In other words, excessive pressure applied to the fetal head, either spontaneously (e.g. uterine tetany) or iatrogenically (e.g. traumatic forceps delivery or excessive fundal pressure) can increase fetal intracranial pressure to such a degree as to result in significant decreases in cerebral blood flow that are associated with fetal heart rate decelerations. Even when decelerations are simultaneous to contractions, decelerations cannot be considered as reflex and innocuous, as they are indeed associated with a decreasing cerebral blood flow.[66]

Pressure does not uniformly cause early decelerations. Lindgren found that:

> Early decelerations may be due to stimulation of the vagus by the high pressures exerted at the equator of the fetal head during labor The deformation of the fetal skull bones support this interpretation. However, early deceleration has not been especially prominent in the uterine hypercontractility associated with large deformations of the fetal skull bones or in contracted pelvis. Another objection to the interpretation that high pressures cause early deceleration is that early deceleration is also observed in breech presentation.[67]

The dilatation effect varies greatly among different women. Mann observes that the mean value for fetal heart rate does not change significantly as a result of fetal head compression. However, the heart rate decreased in eleven experiments increased in twelve and remained unchanged in seven.[68]

Given that it is well-known that uterine contractions provide a great force on the fetal head during the first stage of labor and more so during the second; and given that this increase in pressure is well-known to decrease blood flow to the baby's brain, it is absolutely no surprise

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[65] *Id.* at p. 147.

[66] *Id.* at p. 163.

[67] Lindgren at pp. 420-421.

[68] Mann at p. 724.

that there is a decrease in oxygen to the fetal brain. This is especially true during the second stage of labor when pushing occurs. Aldrich and others measured that effect.[69] They actually measured the fetal cerebral concentrations of oxyhaemoglobin and deoxyhaemoglobin and cerebral blood volume, before and during pushing. Their results:

> [F]ollowing the onset of maternal pushing, mean cerebral deoxyhaemoglobin concentration increased by a mean of 0·79 (SD 0·59) μmol.100 g^{-1} , ($P < 0·01$) without any consistent change in the oxyhaemoglobin concentration. These changes were associated with a significant decrease in the calculated mean cerebral oxygen saturation from a mean of 46·8% (SD 8·6) to 38·1% (SD 5·2) ($P < 0·01$). Pushing was also associated with a significant increase in the mean cerebral blood volume, which rose by a mean of 0·33 ml.100 g^{-1} (SD 0·37) ($P < 0·05$).[70]

Aldrich concluded:

> We have shown that coordinated and sustained maternal pushing during the second stage of labour is associated with a significant decrease in fetal cerebral oxygenation, judged by decreases in cerebral [Hb_{diff}] and $SmcO_2$.
>
> . . .
>
> Our study indicates that a reduction in fetal brain oxygenation with altered haemodynamics occurs as a result of maternal effort during the second stage of labour. However, these findings are based on a small samples size and further studies are required to confirm these observations and determine the mechanisms involved with the changes. Whilst the described effects on cerebral oxygenation and blood volume may not be clinically significant if the fetus is healthy, such alterations may have important consequences if fetal oxygenation is already reduced prior to pushing, or if maternal effort is prolonged.[71]

It is within this historical, medical and physiological context that the concept of resting time between contractions is absolutely critical to keep a baby out of harm's way during paturation. Keeling reported that a fetus could withstand marked head compression for short

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[69] Aldrich, C., et al., "The Effect of Maternal Pushing on Fetal Cerebral Oxygenation and Blood Volume During the Second Stage of Labor," Brit. J. of Obstet. and Gynecology, 102:448 (1995).

[70] *Id.*

[71] *Id.* at pp. 451-452.

periods of time with no ill effect.[72] However, it is common sense and medical fact that if there is not sufficient resting time between contractions, disastrous effects over time will result. As stated by Keeling:

> Svenningsen et al. (1988) measured fetal head compression during spontaneous labour. They recorded large differences in maximum compression pressure during maternal bearing down. There was no relationship between maximum pressure and presence of retinal haemorrhage and concluded that the fetus could withstand marked head compression for short periods with no ill effect.
>
> The normal fetus may be subject to abnormal stresses during labour in several circumstances. It may be stressed because labour is prolonged. Excessive uterine contraction, either naturally occurring or because of the use of oxytoxic drugs (Schwarcz et al. 1974), may adversely affect the fetus. Increased pressure to the fetal head may occur because the abnormal shape or resistance of the birth canal impedes fetal passage. This problem may be aggravated by maternal bearing down efforts, which can impair uterine circulation by interference with both arterial perfusion and venous drainage, by compression of the aorta and vena cava respectively by the gravid uterus (Bassell et al. 1980).[73]

The actual mechanism of injury is well documented and accepted by the scientific community. It has been described by Joseph Volpe, M.D., in one of the most widely recognized and utilized textbooks in pediatric neurology:

> Determination of intracranial pressure is of particular importance in neonatal neurologic disorders, since marked alterations of this pressure have major implications for diagnosis and management. **Intracranial pressure alterations per se may lead to deleterious consequences via two basic mechanisms, disturbances of CBF [Cerebral Blood Flow]** and shifts of neural structures within the cranium. **With the former consequence, cerebral perfusion pressure is related to the mean arterial pressure minus the intracranial pressure. Therefore when intracranial pressure increases, cerebral perfusion pressure decreases; if intracranial pressure increases markedly, cerebral perfusion pressure declines below the low limit of autoregulation and CBF [cerebral blood flow] may be impaired severely.** Indeed, recent evidence suggests that because normal arterial blood pressure in the newborn, especially

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[72] Keeling J. (ed.), Intrapartum Asphyxia and Birth Trauma, Chapter 10, *Fetal and Neonatal Pathology*, 2nd ed., p 240 (1993).

[73] *Id* at p. 241.

> the premature newborn, is relatively low, cerebral perfusion pressure already may be dangerously close to the downslope of the autoregulation curve.[74]

Otherwise stated, if there are too many contractions, there will not be enough time for the baby to catch upon the oxygen necessary for the brain to survive. This simple fact has likewise been studied and measured. Peebles and others found that:

> Changes in cerebral oxyhaemoglobin concentration were positively, and in deoxyhaemoglobin negatively, correlated with the time interval between contractions ($P<0.001$). A mean contraction interval of 2.3 min. was found below which the concentration of oxyhaemoglobin usually fell and that of deoxyhaemoglobin rose, indicating a fall in cerebral haemoglobin saturation. Conversely, longer contraction intervals were associated with finding indicative of a rise in cerebral haemoglobin saturation. CONCLUSION: Short contraction intervals (2.3 min) were associated with a decrease, and longer contraction intervals with an increase in fetal cerebral oxygen saturation. Contractions occurring repeatedly at intervals less than 2.3 min are likely to result in progressive cerebral desaturation.[75]

Another way of simply stating this: If the uterus is hyperstimulated with five or more contractions over a 10-minute period, over time, the baby's brain will run out of the oxygen the baby's brain needs to survive. In an article reviewing the role of intracranial pressure in chronic cerebral arterial spasm, J. Keith Farrar observed the following:

> A review of the literature suggests that human cerebral arteries normally exhibit only mild constrictions in response to subarachnoid blood during the chronic phase of spasm. In the present study, a mild constriction in the absence of increased ICP or a moderate increase in ICP (45 mm Hg) in the absence of constriction produced minor reductions in arterial diameter and an average flow reduction of only 5% to 10%. However, when ICP was increased to 45 mm Hg in the presence of a mild constriction, severe arterial narrowing resulted and flow was reduced by 50%.[76]

It is with this background that obstetricians are currently using Cytotec and Pitocin to induce and augment labor in patients in order to move labor along. Importantly, Cytotec has

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[74] Volpe, at pp. 153-154.

[75] Peebles, D., et al., "Relation Between Frequency of Uterine Contractions and Human Fetal Cerebral Oxygen Saturation Studied During Labour by Near Infrared Spectroscopy," Brit. J. of Obstet. and Gynecology, 101(1):44 (1994).

[76] Farrar, K., "Chronic cerebral arterial spasm," J. Neurosurg. 43:408 (1975).

never been approved by the FDA for such use. Indeed, it is contraindicated in pregnant patients. Moreover in August of 2001 Searle sent a Dear Doctor letter to healthcare providers warning against its use. Nonetheless, many obstetricians insist on using it, often without explaining to the patient (or even understanding) the risk of its off label use. Pitocin is then often added to the mix. Either of these drugs or both in combination can cause hyperstimulation of the uterus. This drug-induced hyperstimulation can manifest in three ways: 1) contractions that are too hard or too long, 2) contractions that are too frequent or 3) insufficient resting tone and/or time between contractions. According to the Pitocin package insert, when it is administered, someone must monitor the patient who is familiar with the complications and someone must be available to respond. Our experience is that this is often not the case. We have found many nurses cannot define hyperstimluation, let alone identify it. The results can be a neonatal encephalopathy from one of four mechanisms: 1) trauma, (2) decrease in cerebral perfusion, 3) decrease in placental perfusion or 4) a combination. These are dangerous drugs and must be treated with respect.

B. Fetal Distress

The concept of fetal distress does not at first seem complicated. It is when the fetus is not reacting well to the stresses of labor and is at risk for neurologic injury or death during pregnancy or during labor and delivery. Obstetricians and nurses use electronic fetal heart monitors to detect fetal hypoxia at its earliest stage in order to intervene to attempt to prevent potentially result in encephalopathy or death. Defense-minded obstetricians and nurses have attempted to change the lexicon and also to downplay the importance of fetal monitoring in modern obstetrics. Obstetricians and nurses are often taught not to use the term "fetal distress". Many claim they not only do not use the term, they do not know what it means. Additionally, the often claim that the importance of monitoring is overplayed. After 40 years of electronic

fetal monitoring, the incidents of cerebral palsy has not decreased. This argument is disingenuous. The statistics can easily be explained by the fact that proper use of fetal monitoring has indeed decreased the incidence of neonatal encephalopathy in term infants. Over the same time period, we have experienced a tremendous increase in capabilities in neonatal intensive care units. Thus we have a substantial increase in the number of very premature infants who survive. Unfortunately, we have a corresponding increase in the number of periventricular leukomalasia encephalopathies of prematurity.

In any event, fetal monitoring is here to stay and, if used properly by skilled practitioners, should assist them in timely intervention in order to improve neonatal outcome.

The electronic fetal monitor has two components: the fetal heart tracing and the contraction tracing. Both are important. Important concepts to keep in mind in interpreting the electronic fetal monitor strip are baseline, variability, periodic and non-periodic changes. The baseline is the heart rate that persists over a time period. Typically one look at at least a 10 minute interval to determine baseline. A normal range is generally felt to be between 120 and 160. Above 160 beats per minute is considered tachycardia. Below 120 beats per minute is typically considered bradycardia. The baseline is typically identified as a range. This is because the fetal heart varies from one beat to the next. The sympathetic nervous system in a healthy fetus is constantly trying to increase the fetal heart rate. The parasympathetic nervous system is correspondingly constantly trying to slow the fetal heart rate. The result is fetal heart rate variability. Variability in reasonable ranges is a good thing.

Periodic changes include accelerations and decelerations that occur in relation to contractions. Decelerations are typically described as early, variable or late. An early

deceleration is typically a mirror image of the contraction. It is typically the results of compression or parasympathetic stimulation. Intervention is not required.

Variable decelerations can occur at any time. It can be before, during or after a contraction. They can be when no contraction is present. Typically, a variable deceleration is a sudden decrease in fetal heart rate with a sudden increase back to baseline. Variable decelerations can vary in size, timing, depth, duration and in shape. Typically, variable decelerations are associated with cord compression. Accordingly, if they persist and other non-reassuring signs develop such as loss of variability or significant change in baseline, the obstetrician or nurse should suspect that the fetus is not tolerating well the cord compression and intervention is warranted.

Late decelerations begin after the apex of the contraction and return to baseline after the contraction is over. Typically late decelerations are as a result of placental insufficiency. As the contraction increases, blood flow through the placenta is diminished. The fetal reserves have been diminished as well. The late deceleration begins. It returns to baseline after placental flow is restored. Late decelerations do not have to be deep decelerations to be significant. Even subtle late decelerations can be ominous. As indicated, there are two components to the electronic fetal monitor: the fetal heart rate and contractions. The monitor measures and records contractions. It is important to keep in mind that with an external monitor, contractions will be recorded. However, the amplitude of the recording tells nothing about the intensity of the contraction. It only reports its existence. The actual intensity of the contraction can only be measured with an intrauterine pressure catheter.

Thus, it is important for the healthcare provider to carefully monitor the fetal heart rate, the fetal heart rate periodic changes in relation to contractions and the contractions themselves.

Each can, in themselves and in combination, indicate circumstances in which intervention is mandatory in order to keep mother and fetus out of harm's way.

C. Failure to Timely Perform C-Section

Many birth injuries are as a results of the healthcare team's failure to timely perform a Cesarean Section. Typically there is an argument about interpretation of the electronic fetal monitoring strip. The defendants will try to move the time based on the strip that a C-section was necessary towards delivery and then claim they moved as quickly as they could in performing the delivery. They will claim the times are approximations based on different clocks and try to recreate reality to defend their conduct. The electronic fetal monitor strip is important because it shows important information with respect to how the fetus is tolerating labor. It is also important because it shows a constant clock. Therefore, it is important to look at the electronic fetal monitoring strip in conjunction with all other clinical information recorded in the chart. In addition, it is common practice for practitioners to note data from examinations and interventions on the strip.

As indicated, in many cases the issue will be at what point did fetal distress or fetal non-reassurance reflected on the strip mandate a Cesarean section. Nonetheless, that should not be the only focus. In the first place, the purposes of fetal monitoring should not be to be able to monitor fetal distress or non-reassurance to a point where an emergent C-section is necessary to hopefully salvage a good outcome. Instead, throughout labor, the healthcare providers should monitor all aspects of the progress of labor, including dilatation, effacement, station, as well as how the fetus is reacting to labor. The focus of the monitoring should not be whether the situation has been allowed to progress to an emergency. Instead, the question should be asked throughout labor, what is the reasonable likelihood of going forward with a safe vaginal delivery.

Once that question is answered negatively, either by failure to progress, failure to descend, fetal intolerance of labor or otherwise, then a different plan should be chosen. Once the focus of monitoring changes from triggering Hail Mary attempts at salvage to keeping baby and mother out of harm's way, outcomes will improve.

D. Group B Streptococcus

Perinatal infection typically refers to a host of bacterial, viral or other infections that can occur during pregnancy.[77] Neonatal sepsis is the term used to describe infection in the newborn.[78] Neonatal infection and sepsis often become the central focus in birth injury litigation. The defense will typically argue that the poor outcome was outside of the control of the healthcare providers and not preventable. Such, however is not always the case.

Perinatal infection is the result of the offending microorganism reaching the fetus through use of three routes. The organism can ascend through the cervix, through the dicidua, through the fetal membranes into the amniotic fluid. The organism can pass from the maternal blood stream crossing over to the fetal circulation. The organism can reach the fetus by direct contact during vaginal delivery through the birth canal.[79] Infection can cause injury to the fetal brain in one of two ways. Either the meninges or brain tissue can become infected or the fetus can become systemically infected and septic, resulting in septic shock. Obviously, antibiotics treatment is necessary after birth whenever there is the possibility of neonatal infection. The problem is that this may be too late to prevent injury to the baby's brain. Healthcare providers should identify the fetus at risk for perinatal infection prior to delivery and begin antibiotics before the fetus becomes septic. Timely intervention is critical in the prevention of neonatal group B

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[77] *See generally* American College of Obstetrics & Gynceologists, American academy of Pediatricians. Guidelines for Perinatal Cure 117-40 (3d ed. 1992); Perinatal Viral and Parasitic Infections, ACOG Technical Bulletin 4, 17, Feb. 1993).

[78] Yancey, M., et al, Risk Factors for Neonatal Sepsis, 87 Obstet. Gynecol. 188(1996).

[79] *See* Guidelines for Perinatal Care, supra at 117-40.

streptococcus. The critical question is to determine the event or condition that should have triggered intervention.[80]

The most common type of infection countered in birth injury litigation if the ascending infection through the cervix into the placental membrane and amniotic fluid. Significant complication of maternal infection include premature rupture of membranes, preterm labor and prematurity.[81] Prompt diagnosis and early treatment are critical components of good obstetrical care.

E. Neuroradiologist's Role in Timing Brain Damage

Neuroimaging is important in determining the timing, nature and pattern of injury in birth injury cases. It is also important in ruling out congenital malformations and other pre-existing causes of encephalopathy.

Serial neuroimaging can be critical in determining the timing of neonatal injury. Hypoxic ischemic insults causing encephalopathy produce a predictable evolution of pathophysiologic changes that can be detected radiographically. Imaging techniques include sonogram, CT and MRI. Early manifestations of hypoxic ischemic injury include cytotoxic edema. Edema usually begins as a result of insult within 24 hours. Neuroimaging techniques can detect it somewhat earlier. These changes evolve over a period of a week, then begin to abate. By comparison of serial imaging during the first week to 10 days of life, the neuroradiologist can narrow the timing of the insult to within the perinatal period. Clinical correlation is required to narrow the neurologist's window for precise timing.

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[80] Ohlsson, A., et al An Analysis of Antenatal Tests to Detect Infection in Preterm Premature Rupture of Membranes, 162 American Journal Obstet. Gynecol. 809 (1990).
[81] Newton, E., et al, Bacterial Veginosis and Intraamniotic Infection, 176 American Journal Obstet. Gynecol, 672 (1997).

With clinical correlation, the pattern of injury can also narrow timing of insult. Two distinct patterns of injury emerge: acute profound (also known as total or near total) and partial prolonged. The acute profound insult occurs when the oxygenated blood supply to the fetal brain is totally or almost totally interrupted, for example with a complete abruption. With these types of injuries, the deep grey matter structures of the brain, the basal ganglia and the thalamus, are first affected. Some studies indicate that with an acute profound insult, the fetus can survive neurologically intact if oxygenation is restored to the baby's brain in 18 minutes or less. If not, the injury progresses rapidly.

Partial prolonged insults, as the name implies, involve a partial, but not total, interruption of oxygenation to the fetal brain over a longer period of time. These injuries typically affect the watershed vascular distribution of the cerebral cortex. Thus with clinical correlation, the timing of injury can be more precisely identified. *See generally*, Barkovich, J., *Pediatric Neuroimaging*, 3d ed., Lippincott, Williams & Wilkins (2000).

F. Mild-Moderate-Severe Injuries

Depending on the degree of the insult during labor and delivery and the portions of the brain affected, different patterns of injury emerge. Clinical presentation varies, depending on the severity of the injury. Patients with a severe or profound injury pattern have different neurologic deficits than those with a moderate or mild injury. Patients with a severe injury tend to have quadriparesis, severe seizure disorders and mental retardation. Additionally, the tend of present with athetosis. Patient with a less severe injury may initially appear to develop normally. It is important to note that many children who develop choreoathetosis may not show extrapyramidal signs until after the first year of life. Most of them develop it between ages 1 and 4. Some patients, however, do not develop abnormal movement until as late as 17 to 14 years of life.

Nearly half of these patients have a history of normal neurologic development until the extrapyramidal signs and symptoms develop.[82]

It is important to note that even mild injuries will pose significant problems for the patient as they mature. In that regard, three things should be kept in mind. First, even the most sophisticated neuroimaging techniques will not pick up nerve damage on a cellular level. Accordingly, it is possible for structures of the brain to be injured and not show up on neuroimaging.

Second, even if injury does show up on neuroimaging, clinical manifestations may well not become apparent until the child matures, even into adulthood. For example, significant injury to the frontal lobes may well not produce a behavioral deficit until the child develops into adolescence and fails to develop normal frontal executive behaviors. Likewise, focal lesions in regions not fully matured at the time of the hypoxic-ischemic insult may not produce deficits which are clinically apparent until the structures involved become necessary to reaching some particular neuropsychological function. As the child develops, particularly with mild white matter injury, there often emerges a pattern of behavioral deficits, inability to control emotions, developmental delay, learning disability and lack of executive abilities which govern a variety of behaviors, including sustained anticipation, planning, problem-solving, organization, self-monitoring, error detection and correction and control functions. It is of little solace to children with mild injuries that their motor function is relatively intact if they will be unable to succeed in school, control behavior, interact socially with their peers and get and keep employment as an adult. For the mild to moderate birth injury patients, neuropsychological testing can be very important. Such testing is typically thought to be of little benefit for patients under the age of 3.

[82] Barkovich, James, Pediatric Neuroimaging 190-1, Lippincott Williams and Wilkins (2000).

Moreover, the predictive ability of such testing increases the older the child is at the time of testing. Neuropsychological testing often includes:

1. Bayley Scales of Infant and Toddler Development – 3rd Edition;
2. Behavioral Assessment System for Children – Parent Report Form;
3. Behavior Rating Inventory of Executive Function – Preschool Version;
4. Clinical Evaluation of Language Fundamentals Preschool – 2nd Edition;
5. Developmental Neuropsychological Assessment (NEPSY);
6. Developmental Test of Visual Motor Integration (VMI);
7. Greenspan Social Emotional Growth;
8. Leiter International Performance Scale - Revised;
9. Wecshler Preschool and Primary Scale of Intelligence – 3rd Edition; and
10. Wide Range Assessment of Visual Motor Abilities (WRAVMA).

G. Defenses to Labor and Delivery Cases.

ACOG has aggressively attempted to rewrite science in recent years, not to improve patient care on outcomes, but specifically to defendant lawsuits. And it has done so in areas in which it is particularly ill-equipped. In 2003, it published a monograph, Neonatal Encephalopathy, and Cerebral Palsy (NECAP). Notably, not a single practicing pediatric neurologist was on its task force. Undeterred, it attempted to create science to avoid responsibility for harm caused by obstetricians to newborns.

Typically, Defendants, using NECAP, claim that in an ACOG task force "comprehensively" reviewed and analyzed the available body of literature on the subject of neonatal brain damage and published a report compiling its findings into one document. They then claim that because this "one document" does not address the mechanism of injury involved

in a given case, the concept is not worthy of consideration. They typically ten claim that the Plaintiff did not meet the four "essential criteria" to establish a hypoxic-ischemic event during labor sufficient to cause neonatal brain injury and cerebral palsy. Defendants then suggest that the ACOG criteria are somehow mandatory and imply, therefore, that the conclusions of the plaintiffs' experts with respect to the mechanism of injury cannot be valid. These assertions are incorrect. First, the ACOG monograph does not address all types of injury caused by healthcare providers. Second, the ACOG monograph does not set forth mandatory criteria. Third, the ACOG monograph is not reliable. Fourth, courts have rejected the offensive use of this document.

1. The ACOG Criteria Is Not Mandatory.

The ACOG monograph criteria was not intended to be rigid criteria, even by ACOG. In point of fact, the publication states that the information in *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology* "**should not be viewed as a body of rigid rules**" and will be well-served "if they provide a firm basis on which local norms may be built."[83] And even most defendants' experts agree that analysis of the causation issue should not be dictated by dogmatic adherence to rigid criteria.

2. The ACOG Criteria Are Designed to Defend Lawsuits.

a. No one should be misled by any defendants of the word "essential" because the reality of the 2003 ACOG monograph is that it is a political agenda masquerading as medical science. If there is any doubt that ACOG is not a lobbyist for so-called "Medical Liability Reform," one has only to visit its website and view numerous ACOG News Releases promoting

[83] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pedatircs, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiciology*, (2003).

the passage of tort reform litigation. See ACOG's February 1, 2006 press release, which is directly on point.[84]

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

> G. But your -- your point was -- the reason you were developing it, or stated reason, was to defendant lawsuits?
>
> A. Well, I thought that -- that we were in an area where the -- where were being used for reasons that we --over which we had no control. And – in a high percentages of the time.
>
> And so, yes, that's – that was one of the area – one of the things I wanted to address. And I – and I – and I will stand by that.[85]

One of Dr. Miller's stated goals for ACOG during this decade was to "reduce medicolegal risks for obstetrician-gynecologists" by developing evidence that could be "used to defendant against unwarranted claims and challenge false testimony by expert witnesses and others.[86] Apparently, to Dr. Miller and ACOG, any claim against an obstetrician-gynecologist is unwarranted and all plaintiff expert testimony is false. The so-called "essential criteria" are designed to protect ACOG's membership and not to assist the jury in finding the real truth, and certainly not to improve patient healthcare.

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[84] ACOG News Release, http://www.acog.com/from_home/publications/pressreleases/nr02-01-06.cfm.
[85] *Deposition of Dr. Franklin Carl Miller*, In the United States District Court of the Western District of Tennessee at Memphis, *Miller v. Dacus*, Case No. 03-2701 M1 V., July 22, 2004 at p. 46, lls, 14-25.
[86] Miller, F., "Ten Goals for ACOG for the First Decade of the Next Millennium", OB &GYN, 95(1):1 (2000) at p. 4.

Neither the "essential criteria" nor the purpose for them are new. ACOG's monograph derives from two earlier sources: ACOG Technical Bulletin 163 (1992) and a British medical journal article published in 1999 and referred to as The Template.[87]

According to the author of The Template, Dr. Alistair MacLennan, the International Cerebral Task Force was convened to bring together the modern literature on the cause of cerebral palsy to benefit research into cause and prevention and to help those who offer expert testimony in court.[88] Dr. MacLennan even provided "recommendations for expert witnesses giving evidence on cerebral palsy causation." The recommendations included criteria for the qualifications of expert witnesses on cerebral palsy causation and suggestions concerning how the expert witness should conduct himself or herself.[89] This motive was carried forward from Technical Bulletin #163. It is underscored by the following comments by Dr. Richard Perkins to Dr. Goodlin's article criticizing previous "essential criteria" in ACOG Technical Bulletin #163:

> The request was to reach a generalization that vastly exceeded the legal concept of "reasonable medical probability" (that is, somewhat >50% of the time) and to approach the medically tolerable concept of "reasonable medical probability" (that is, with only rare exception). It was an honest effort to stop the bleeding. It cured most ills fairly and staunched an evil tide of will speculation and unsound litigation while awaiting refinement in perspective.[90]

Dr. Goodlin's response to Dr. Perkins is instructive:

> If the framers of the ACOG bulletin had spoken in terms of Perkins' "Reasonable medical probability" instead of their "must be" criteria, the impact of their concepts on obstetric through would have been different. **The "must" be criteria go far beyond the legal issues, for they now appear as definitions of fetal asphyxia and neonatal hypoxic-ischemic encephalopathy.**

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[87] MacLennan a., "A Template for Defining Causal Relation Between Acute Intrapartum Events and Cerebral Palsy; International Consensus Statement," Brit. Med. J. 319: 1054 (1999).
[88] *Id.*
[89] *Id.* at 1059.
[90] Perkins, R, "Leters", Am. J. Obstet. Gynecol. 174:2 (1996) at pp. 598-799.

These criteria intrinsically eliminate intrapartum insults as the cause in most cases of cerebral palsy and lead to the view that obstetricians can do nothing to prevent cerebral palsy.

In their apparent desire to stem Perkins' figurative "bleeding with ligature", the framers of ACOG criteria ignored scientific principles and created bad dogmatic rules from soft data (as described in my paper).[91]

Clearly, ACOG's purpose in drafting ACOG 163 was to create a virtually impossible causation hurdle for malpractice cases. It is not a great leap of faith to conclude that ACOG has the same motivation concerning the current "essential criteria." The ACOG criteria do not represent the knowledge and beliefs of the relevant medical community for *Daubert* purposes. Further, there are no well-done laboratory or clinical studies that support ACOG's four stringent criteria, much less that all four must be present.[92]

The 2003 ACOG monography, The Template, and the ACOG Technical Bulletin 163, are powerful examples of the inherent conflict of interest these publications represent. All embody what has been labeled as "inherently unacceptable behavior that directly threatens the integrity of research."[93] In a "National Survey of Policies on Disclosure of Conflicts of Interest in Biomedical Research" published in the *New England Journal of Medicine*, the authors conclude that these conflicts of interest may appropriately influence the design, conduct, or reporting of research, thus threatening its scientific value.[94]

The implications for these litigation-driven publications have even more dire consequences. According to the ACOG Task Force, one goal of "overriding importance" is to

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[91] *Id.* (emphais added).
[92] Goodlin, R., "Do concepts and prevention of cerebral palsy require revision?", Am. J. Obstetrics and Gynecology 172:1830 (1995). *See also* Lisa M. Korst, et al, *Acute Fetal Asphyxia and Permanent Brain Jury: A Retrospective Analysis of Current Indicators*, J. Maternal-Fetal Med. 8:101 (1999). *See* Phelan, J., et al "Intrapartum Fetal Asphyxial Brain Injury with Absent Multi-Organ System Dysfunction, "J. Maternal-Fetal Med. 7:1 (1998).
[93] McCrary, V., et al, "A National Survey of Policies on Disclosure of Conflicts of Interest in Biomedical Research," N.Eng.J. Med. 343:1621-1626 (2000).
[94] *Id.*

"Develop recommendations for evaluation of the newborn with encephalopathy *to assist the clinician* in defining both the cause and the timing of the encephalopathy."[95] ACOG, in this publication, is mixing its political agenda with the medical/scientific advice it offers its members concerning the diagnosis and treatment of the fetus and the newborn. This is a conflict of interest that has far-reaching repercussions, not just for the obstetricians who deliver babies, but also for the babies themselves. ACOG 2003 monograph is precisely the kind of "junk science" that the United States Supreme Court decried and which was the rationale for the far-reaching changes in evidentiary standards promulgated by the Court in *Daubert v. Merrel Dow Pharms., Inc.* 509 U.S. 579, 113 S. Ct. 2786, (1993).

Joseph J. Volpe, M.D., Bronson Crothers Professor of Neurology, Harvard Medical School and Neurologist-in-Chief at Boston's Children's Hospital, is one of the preeminent neurologists in the country and has published extensively in the field. In his widely used textbook, *Neurology of the Newborn*, Dr. Volpe addressed the dangers of the medical professions' attempt to deny the importance or even the existence of intrapartum brain injury in the face of an explosion of obstetrical litigation. His words deserve serious consideration, particularly in light of the hospital's attempt to impose "mandatory" criteria for the assessment and treatment of intrapartum hypoxic injury to the exclusion of all other factors:

> The occurrence of injury to brain during the birth process has been the focus of clinical research for over a century. In my view that work has shown that brain injury in the intrapartum period does occur, affects a large absolute number of infants worldwide, is obscure in most cases in terms of exact timing and precise mechanisms, awaits more sophisticated means of detection in utero, and ***represents a large source of potentially preventable neurological morbidity.*** (emphasis added)
>
> Among the many adverse consequences of the explosion in obstetrical litigation ***has been a tendency in the medical profession to deny the importance or even***

[95] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiciology*, (2003) at xiv.

> ***the existence of the intrapartum brain injury***. (emphasis added). Although it is unequivocally clear that true obstetrical
> malpractice is a *rare* occurrence and that the obstetrician is called upon to deal with perhaps the most dangerous period in an individual's life with *inadequate methods*, this tendency is particularly unfortunate. With the recognition from experimental studies that much of hypoxic-ischemic brain injury evolves after cessation of the insult and can be interrupted to a considerable extent by several approaches (see Chapters 6 and 8), the ultimate possibility of intervention both in utero and in the early post natal period is strongly suggested. ***Denial that intrapartum injury occurs may impair development and application of such brain-saving intervention*** (emphasis added).[96]

It is unthinkable that the development and application of brain-saving interventions would be sacrificed to the self-interests of ACOG and its membership.[97] To adopt the argument that the four essential criteria are "mandatory" and must be met before diagnosis, and therefore treatment, is unconscionable. It is a position based upon nothing but literature clearly generated for purposes of litigation and is wholly unreliable. See, *Daubert v. Merrell Dow Pharms, Inc.* 43, F3d 1311, 1317 (9th cir. 1995)(research conducted independently of litigation provides important, objective proof that the research comports with the dictates of good science). Importantly, the neurological community has not adopted this criteria for intervention. For example, a recent experimental therapy for term infants suffering an acute brain injury is whole-body hypothermia.[98] The idea is that if the brain temperature is reduced 2°C to 5°C, it would provide neuroprotection to babies suffering from ischemic insult to the brain. This was recently studied in fifteen participating centers of the National Institute of Child Health and Human Development Neonatal Research Network. The criteria for inclusion in the study were different from ACOG's "essential criteria."[99]

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[96] Volpe at p. 283.
[97] These physicians are called upon in the Hippocratic Oath to "prescribe regimens for the good of my patients according to my ability and my judgment and never do harm to anyone."
[98] Shankaran, S., et al "Whole-Body Hypothermia for Neonates with Hypoxic-Ischemic Encephalopathy, N. Eng.J.Med. 353: 1575 (2005).
[99] *Id.*

3. The ACOG Publications.

(a) Technical Bulletin 163.

ACOG Technical Bulletin 163 previously claimed that four criteria must be present before cerebral palsy could "plausibly" be linked to birth asphyxia. Criticism of these criteria came quickly.[100] In one study, researchers evaluated 47 newborns diagnosed with cerebral palsy.[101] No cause was found for the infants' neurologic injuries other than intrapartum asphyxia. Yet only 10 infants satisfied all of the ACOG Technical Bulletin 163 criteria and only 24 satisfied at least there of those four criteria. Fourteen of the babies had no other organ damage. Many of the babies had a blood pH level greater than 7.0. Only slightly more than half had APGAR scores of less than three at five minutes. Two of the babies did not have documented seizures in the early neonatal period. The authors concluded that the ACOG Technical Bulletin 163 criteria were not valid.[102] Another study of 292 infants with permanent neurologic injury caused by fetal asphyxia revealed that 36% did not have multi-system organ damage.[103] Another study reported that about 20% of infants with apparent fetal asphyxia have no evidence of end organ injury. Yet this was an essential criteria under Technical Bulletin 163.[104]

b. The Template

The 199 report of the International Cerebral Palsy Task Force ("The Template") included four essential and five non-essential criteria "suggesting" that acute intrapartum hypoxia was the cause of cerebral palsy. Note: these were not "mandatory" criteria. The findings of this task force were targeted for criticism soon after publication. Several British obstetricians observed,

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[100] Goodlin, *supra.*
[101] Korst, *supra.*
[102] *Id.*
[103] Phelan, *supra.*
[104] *See,* ACOG Technical Bulletin 163.

among other things, that the cord blood pH cut-off of less than 7.0 was too low and excluded a significant number of babies who may have suffered intrapartum asphyxia.[105] These critics pointed out that "none of the evidence on which the...document is based meets the criteria for grade-A [medical] evidence" and that "the level of evidence is at best grade C."[106] The Template was no more valid than Technical Bulletin 163.

c. ACOG 2003 Monograph

(1) A lack of input from neuro-scientists

The purposed objective of Task Force on Neonatal Encephalopathy and Cerebral Palsy was to "create a multidisciplinary task force to review and consider the current state of scientific knowledge about the mechanism and timing of possible etiologic events which may results in neonatal encephalopathy."[107] However, in the end analysis, one of the problems with the findings of the task force was the lack of input from neuroscientists – a critical discipline to any multidisciplinary task force studying brain neurologic injury. Of the 29 people listed as either members of the task force, staff or consultants, there was not a single practicing pediatric neurologist listed.[108] Indeed, the only child neurologist included is Karin Nelson, M.D., who has not treated patients in decades and whose writings have been criticized in the peer-reviewed literature. It is not surprising that actual neuroscientists' view differ from those of the monograph and ACOG. The "essential criteria" were developed from studies that lacked proper statistical analyses and were conducted before data from more reliable contemporary imaging studies were available. Again, for example, ACOG's President Franklin Miller admitted in

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[105] Deat, P., et al, "Response to the Proposal of a Template of Defining a Causal Relation Between Intrapartum Events and Cerebral Palsy," Clinical Risk 6:137-142 (2000).
[106] *Id.* at 138.
[107] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiciolgy* (2003) at xiii.
[108] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiciolgy* (2003) at v-viii.

sworn testimony last year:

H. Imaging of ?

Q. The brain.

A. I don't believe that they do that, no.[109]

In March 2002, the American Academy of Neurology published a practice parameter for the diagnostic assessment of children with cerebral palsy.[110] According to the actual neuroscientists, not only should the neuro-imaging studies of the brain be taken into account, they are the starting point of the analysis.[111]

Barkovich and his associates also observe that MR spectroscopy performed in the first 24 hours after birth is sensitive to the severity of hypoxic-ischemic brain injury and have had considerable effect on the diagnosis of brain injury.[112]

(2) ACOG's statistics are not reliable.

One of the principal studies upon which the monograph relies for support is authorized by Nadia Badawi and concludes that the "causes of newborn encephalopathy are heterogeneous and many of the causal pathways start before birth.[113] A recent study testing this hypothesis unequivocally confirmed that events during labor and delivery, not earlier ones, are responsible for most cases of neonatal encephalopathy and early seizures.[114] The investigators studied 351 term infants who exhibited either neonatal encephalopathy, early seizures or both, within 72

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[109] Deposition of Dr. Franklin Carl Miller at p. 37, lls. 12-21.
[110] "Practice Parameter: Diagnostic Assessment of the Child with Cerebral Palsy (Report of the Quality of Standard Subcommittee of the American Academy of Neurology in the Practice Committee of the Child Neurology Society)," *Neurology* 62:851 (2004).
[111] *Id.* at 861.
[112] Barkovich, A.J., et al, "Proton Spectroscopy and Diffusion Imaging on the First Day of Life after Perinatal Asphyxia: Preliminary Report," *Am. J. Neuroradio* 22: 1786-1794 (2001).
[113] Badawi, N., et al, "Intrapartum Risk Factors for Newborn Encephalopathy" The Western Australian Case-Control Study," *Brit Med. J.* 317:1549-1554 (1998).
[114] Cowan, F, et al, "Original and Timing of Brain Lesions in Term Infants with Neonatal Encephalopathy," *Lancet* 361:736 (2003).

hours of birth. Neonatal encephalopathy was indicated by abnormal tone, feeding difficulties, altered alertness, and at least three of the following criteria: late decelerations or meconium staining, delayed onset of respiration, arterial cord blood pH less than 7.1, APGAR scores of less than 7 at five minutes, and multi-organ failure. The babies were all evaluated neuroradiologically and results compared with signs recognized as suggesting an natepartum event or developmental abnormality, as opposed to injury during the labor and delivery process. The scans showed that 261 of the babies met the criteria for neonatal encephalopathy. The scans further revealed that 197 of those babies whose evidence of acutely evolving lesions compatible with hypoxic-ischemic insult. Of the 351 babies scanned, 306 showed evidence of an acute intrapartum injury. Only 45 suggested some evidence of developmental, metabolic or other disorders that preceded the labor and delivery process. The authors delivered a clear message:

> Some investigators have reported that only 8% to 15% of term infants with neonatal encephalopathy, and far fewer with neonatal seizures, have evidence of asphyxia immediately before birth. Furthermore, others have suggested that many neurological signs once thought to be caused by intrapartum asphyxia are a manifestation of a process begun during the antenatal period. **However, we have found little evidence for the two proposals that acute perinatal injury is uncommon in such infants and that injurious processes have been taking place antenatally**. (emphasis added)[115]

The ACOG 2003 monograph cites the incidence of neonatal encephalopathy attributable to intrapartum hypoxia at an estimated 1.6 per 10,000 births.[116] This figure is cited no less than four times in the monograph. However, the reported incidence of conditions labeled birth asphyxia, HIE, or post-asphyxial encephalopathy in term or near-term infants ranges from 1 to 8 per 1,000 births.[117] Even the Badawi article, otherwise relied on by the monograph, cites the

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[115] *Id.*
[116] The American College of Obstetrics and Gynecology (ACOG) and the American Academy of Pediatrics, *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogensis and Pathophysiciology*, (2003) at p. 3.
[117] *Id.*

incidence of newborn encephalopathy as 3.8 per 1,00 term live births.[118] Even if one were to accept that only approximately 30% of neonatal encephalopathy is attributable to intrapartum hypoxia as found by Badawi, but contrary to the some 90% found by Cowan, then 30% of 3.8 equals 1.14 per 1,000 births.[119] The ACOG 2003 monograph's figure understates the supported figures by ten-fold.

(3) The ACOG's pH criteria is invalid.

One of the monograph's "essential criteria" for determining that a baby's brain injury was caused by intrapartum asphyxia is a cord blood pH of less than 7.0 and a base excess of 12 or greater. This criteria is not supported by valid data. The medical literature has documented cases in which babies suffering from hypoxic-ischemic encephalopathy indeed has a blood pH value greater than 7. In fact, the pH value is not even considered in one of the major studies in which the monograph relies for its hypothesis that most cases of cerebral palsy occurred in the antepartum period.[120] In Badawi's study, the criteria used to determine whether a baby was exposed to intrapartum hypoxia were abnormal fetal heart rate, meconium staining, or both, along with a one-minute APGAR score of less than 3 and a five-minute score of less than 7. Several of the babies included in that study had a cord pH greater than 7.[121] This is not surprising. In point in fact, Hermansen recently studies the relationship between acute birth asphyxia, acidosis and brain injury. He challenged the "traditional assumptions" regarding pH. Dr. Hermansen noted that:

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

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[118] *Id.*
[119] Cowan, *supra.*
[120] *Badawi.*
[121] *Id.* at 1555.

79% had pH>7.18[122]

Clearly the monograph's criteria are not valid.

It is interesting to note that over the course of several years, ACOG has aggressively tried to minimize the importance of blood pH values in evaluating the condition of neonates. In 1976, ACOG stated in Technical Bulletin No. 42 that a pH of below 7.20 determined fetal distress.[123] Only one year later in 1977, ACOG states that when a pH of 7.20 occurred, expeditious delivery was required.[124] In 1989, ACOG started backing off this position, stating that a pH of *less than* 7.20 was required for medical intervention.[125] In 1992, ACOG further restricted their position, stating that a cord pH as low as 7.00 was required to justify a causal link between intrapartum asphyxia and neurologic injury.[126] "The poor predictive value of newborn blood pH for subsequent cerebral palsy has been repeatedly noted."[127] "The problem is that there is no pH value that clearly separates those babies who have experienced potentially damaging intrapartum asphyxia from those who have not."[128]

In clinical and medico-legal practice , no diagnosis can be made or refuted on the basis of a single laboratory measurement and the idea of a set cut-off is naïve. "The recommended cut-off value of 7.0 is too low and will exclude a significant number of babies who have possibly encountered damaging intrapartum asphyxia."[129]

> Unfortunately, there is no pH value that separates cleanly those babies who have experienced intrapartum injury from those who have not – no prognosis can be

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[122] Hermansen, M., "The acidosis paradox: asphyxial brain injury without coincident acidemia," Developmental Medicine & Child Neurology, 45:353-356 (2003). See also Casey, B., et al, "Outcomes Among Term Infants When Two-Hour Post Natal pH is Compared with pH at Delivery", Am. J. Obstet. Gynecol, 184:44 (2001).
[123] Technical Bulletin No. 42, Am. C. Obstetricians & Gynecologists (1976).
[124] Technical Bulletin No. 44, Am. C. Obstetricians & Gynecologist (1977).
[125] Technical Bulletin No. 127, Am. C. Obstetricians & Gynecologist (1989).
[126] Technical Bulletin No. 163, Am. C. Obstetricians & Gynecologists (1992).
[127] Goodlin at p. 1831.
[128] Dear at p. 139.
[129] *Id.*

made or refuted on the basis of a single laboratory measurement.[130]

(4) ACOG's literature does not support ACOG's conclusions.

ACOG exaggerates the persuasive power of the NECAP and its underlying studies. The much touted essential criteria are set forth in chapter eight of ACOG's 2003 monograph. The criteria are allegedly based on data from 72 articles. A careful analysis of these articles, however, reveals that the monograph criteria is a classic example of "junk science". Twenty-seven of the articles support none of the essential criteria. Another 16 of the articles represents nothing more than their authors' opinions. Of the 29 articles cited to support one or more criteria, not a single one is based on a properly designed, randomized, control trial.

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

> Hypoxic ischemic encephalopathy is notoriously variable from infant to infant. Seemingly similar degrees of insult may completely spare one child and devastate another. To some extent, this extraordinary variability is evidence of inability to accurately quantify the magnitude of the impairment of gas exchange in utero. Thus, seeming similar degrees of insult may in fact not be similar at all. Furthermore, intrinsic fetal differences...may also affect the responses to hypoxic insult and play a key role in determining outcome.[131]

(5) ACOG's criteria has been rejected by other Courts.

Use of the essential criteria set out in some form in all three publications is a common defense strategy and has been rejected by at least two courts. In *Koval v. Kincheloe*, 2001 WL 34748891 (W.D. Okla. 2001), the defendants moved for summary judgment alleging that the

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[130] Socol, M., et al., "Depressed Apgar scores, acid-base status, and neurologic outcome," Am. J. Obstet. Gyneco., 170:991 (1994).

[131] Pasternak, J.D., "Hypoxic-ischemic Brain Damage in the Term Infant," Pediatric Clinic N. Am. 40:1061, 1062 (1993).

plaintiffs did not have a sufficient scientific basis for their opinion that the minor plaintiff's cerebral palsy was caused by hypoxia or ischemia. *Id.* At *1. Specifically, they contended that there were criteria which according to ACOG must be present in the newborn before cerebral palsy can be linked to the events of labor and delivery as set out in ACOG Technical Bulletin No. 163 and in The Template. *Id.* at *2-3.

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

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

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

752 F. Supp. At 772. Nothing that other physicians likewise changed their positions subsequent to the filing of the lawsuit, the Court said it looked "with suspicion on the testimony of those witnesses who changed their position." *Id.*

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