Oxytocin is a dangerous drug. Its misuse in the United States injures thousands of babies. As stated by Dr. Steven Clark:

> Oxytocin is the drug most commonly associated with preventable adverse perinatal outcomes and was recently added by the Institute for Safe Medication Practices to a small list of medications "bearing a heightened risk of harm," which may "require special safeguards to reduce the risk of error." (Clark, et al. 2009)

That oxytocin is potentially dangerous is no secret. The package insert clearly lists permanent CNS or brain damage and fetal death as adverse reactions to a baby whose mom receives Oxytocin during labor. Moreover, it lists as a general precaution:

> All patients receiving intravenous oxytocin must be under continuous observation by trained personnel who have a thorough knowledge of the drug and are qualified to identify complications. A physician qualified to manage any complications should be immediately available. Electronic fetal monitoring provides the best means for early detection of overdosage.

In the overdosage section, the package insert provides:

> Overdosage with oxytocin depends essentially on uterine hyperactivity, whether or not due to hypersensitivity to this agent. Hyperstimulation with strong (hypertonic) or prolonged (tetanic) contractions, or a resting tone of 15 to 20 mm H2O or more between contractions can lead to tumultuous labor, uterine rupture…utero-placental hypoperfusion and variable deceleration of fetal heart, fetal hypoxia, hypercapnea….or death. (Physician's Desk Reference; 1998.

In 2008, the National Institute of Child Health and Human Development (NICHHD) along with the Society for Maternal-Fetal Medicine and ACOG convened a workshop to update definitions for fetal heart rate tracings. The controversy surrounding the new 3 tier system aside, it is necessary for labor and delivery nurses to understand and to keep in mind the physiology of labor and its effect on the mother and the fetus (Coletta, et al., 2012). The workshop effectively

removed the word "hyperstimulation" from the lexicon of those ordering and administering oxytocin and tending to moms and babies during labor and delivery (Macones, et al 2008). This is nothing more than semantics and does not change the well-known science with respect to injuries caused by overstimulation of the uterus, especially associated with the misuse of oxytocin. Whether the term "hyperstimulation" is used, or "over stimulation of the uterus", "excessive uterine activity", "abnormal forces of labor", or "uterine tachysystole", the basic science is the same and has been well-known and well-described in the obstetrical literature for decades.

Irrespective of the term used, excessive uterine activity can manifest in many ways:

(1) Contractions that are too long,

(2) Contractions that are too strong,

(3) Insufficient resting tone (i.e. resting tone above 20 mmHg), and

(4) Insufficient resting time between contractions (i.e., less than one minute).

Excessive uterine activity from the use of oxytocin can cause permanent neurologic injury to a child by three separate mechanisms, or as in some cases, a combination of the three. First, if there is insufficient resting tone, that is if the uterus does not relax enough for a long enough time between contractions, there will be a decrease in placental perfusion. If this persists over time, there will be a decrease in fetal reserve and a resulting hypoxic ischemic insult to the baby.

Second, excessive uterine activity from the use of oxytocin can cause permanent neurologic injury to a child by trauma and excessive molding.

Third, excessive uterine activity results in an increase in intra-uterine pressure. This causes a corresponding increase in intra-cranial pressure. It is well-known that an increase in

intra-cranial pressure, especially approaching and above the baby's mean arterial pressure, results in a decrease in cerebral perfusion, resulting in ischemic injury to the baby's brain. It is especially important to keep in mind that the set up for these injuries can occur before there are changes to the fetal heart rate pattern. If nurses wait until there are changes in the fetal heart rate pattern to turn off the oxytocin, it might be too late to avoid injury.

Hypoxic Ischemic Injury

It is well known that a baby gets its oxygen from the placenta. "Placental oxygen is blood flow limited. Using estimated utero-placental blood flow, Longo (1991) calculated oxygen delivery to be about 8mL 02/min/kg of fetal weight. And because fetal blood oxygen stores are sufficient for only one to two minutes, this supply must be continuous." (Cunningham, et al; 2010). That is to say, if there is not sufficient resting tone between contractions, the baby will be deprived of oxygen.

There is no doubt that uterine contractions are work for mother. It causes an increase in maternal oxygen demand and in consumption, resulting in a decrease in maternal oxygenated blood to the placenta. In addition, during a contraction, the uterus squeezes spiral arteries, decreasing maternal blood flow to the placenta, as well as venous outflow from it. If there is not sufficient resting tone between contractions to allow for re-oxygenation of the baby, the cumulative effect is hypoxia to the baby, and ultimately acidosis (Murray, et al; 2009). As stated by Michelle Murray:

> If contractions are more frequent than every two minutes, or the interval between them is less one and a half minutes, fetal SaO2 decreases to as low as 18%. Less than 30% is abnormal. When there are fewer contractions, fetal Sa02 is 54% or higher. Normal fetal Sa02 is between 30 and 70%. Maternal supplemental oxygen increases fetal Sa02 7 to 11% within three minutes (McNamara and Johnson, 1995; Johnson, Johnson, McNamara, Montague, Jongsma and Aumeerally, 1994; Johnson, Van Oudgaarden,

Montague and McNamara, 1994). Therefore, it is best for fetal health if at least one and a half minutes elapses between contractions (Murray, et al; 2009)

If there is not sufficient resting between contractions, the ultimate result is the risk for hypoxic-ischemic brain injury or death.

Trauma and Excessive Head Molding

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. (Kriewall, et al; 1960). 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. (Kriwall, et al; 1960)

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*. (Little; 1966) 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. (Little; 1966)

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 the limbs of the child, which he designated as spastic rigidity of the limbs (Little; 1966). 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. (Little; 1966)

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 oxytocin 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. (Little; 1966)

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 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. (Little; 1966)

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. (Little; 1966)

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). (Little; 1966)

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 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." (Yates; 1959) 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 (Kriewall;1960). 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. (Clyne; 1964)

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
4. Other causes	606	15.20

(Clyne; 1964)

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 (Clyne; 1964). 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. (Govaert, et al; 1992)

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.

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, however, the typical molded newborn head is elongated and cylindrical, reflecting misalignment among the bones of the cranial vault (parietal, frontal, and occipital bones). (Carlan, et al 1991)

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 (Caldeyro-Barcia 1974). Moreover, when the protrusion of the parietal bones becomes very marked, it may tear the cerebral falx and cerebellar tentorium with consequent hemorrhage (Celdeyro-Barcia 1974). According to Barkovich in *Pediatric Neuroimaging*, "the causes of both falx and tentorial tears seem to be excessive vertical molding of the head with frontal-occipital elongation." (Hanigan 1990) In *Tentorial Hemorrhage Associated with Vacuum 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. In two of the patients studied, the tentorial hemorrhage was associated with ischemic complications (Hanigan 1990).

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 been well-studied in infants, in autopsies, in biomechanical engineering models and in animals (Towbin 1998). 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 (Newton 1975). 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.

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.

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.

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.

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.

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

(Lindgren 1977). 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. (Lindgren 1977)

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.

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...

. . .

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.

Five years later, Sorbe and others studied 319 vaginal deliveries. 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. (Sorbe 1983)

. . .

The region of the brain in greatest jeopardy is determined by the spatial orientations of the head as it descends through the maternal pelvis.

Importantly, they looked at the effect of 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.

. . .

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.

Lapeer and others studied fetal head molding from a biomechanical engineering perspective (Lapeer, et al 2001). 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. (Lapeer, et al 2001)

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. (Lapeer, et al 2001).

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.

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

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).

This is the same as three to five contractions in a 10-minute period. More than five contractions in a 10-minute period are hyperstimulation by definition (Murray, et al 2011).

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 (Kelly 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. 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. Another result of this pressure is various kinds of cerebral lesions (Lindgren 1977).

Hyperstimulation and Decrease in Cerebral Perfusion – Ischemia

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 placenta. 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 (Volpe). 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.

Forty years ago, Mann and others prospectively studied the decrease in cerebral blood flow as a result of increased extracranial pressure on sheep (Mann, et al 1972). 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 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.* (Mann, et al 1972).

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 (Mann, et al 1972).

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

	Heart Rate Beats/min	pO₂ mm Hg	Ph	Perfusion Pressure Mm Hg	Resistance mm/L/min	Blood Flow ml/100 g/min	Q O₂ 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 (Mann, et al 1972).							

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.

Subsequently, O'Brien and others did similar experiments on near term fetal lambs (O'Brien et al 1984). 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 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).

Over 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 (Amiel-Tison et al 1988). 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.

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

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.

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. (Lindgren 1977)

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 (Mann, et al 1972)

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 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 (Aldrich, et al 1995). 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) $\mu mol.100\ g^{-1}$, ($P < 0.01$) 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) ($P < 0.05$).

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.

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 <u>short</u> periods of time with no ill effect (Keeling 1993). 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).

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 the premature newborn, is relatively low, cerebral perfusion pressure already may be dangerously close to the downslope of the autoregulation curve. (Volpe)

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. (Peebles, et al 1994)

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%. (Farrar 1975)

CONCLUSION

It is with this background that healthcare providers today order and administer oxytocin. There is a reason that the Institute for Safe Medication Practices has listed oxytocin as a "high alert" medication. It is a dangerous drug with potentially serious consequences if misused.

REFERENCES

1. 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, 1995; 102:448.

2. 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, 1988; 2(1):145

3. Barkovich, A. James, M.D., Brain and Spine Injuries in Infancy and Childhood; Pediatric Neuroimaging, 2000; 3rd Ed.

4. Caldeyro-Barcia R., Adverse Perinatal Effects of Early Amniotomy During Labor, Modern Perinatal Medicine, 1974: Chapter 32.

5. Carlan, S., et al., Fetal Head Molding, Diagnosis by Ultrasound and a Review of the Literature, J Perinatol. 1991 Jun; 11(2):105.

6. Clark S, et al. Oxytocin: new perspectives on an old drug. Am J Obstet Gynecol 2009;200:35.e1-35.e6.

7. Clyne, D., Traumatic versus Anoxic Damage to the Foetal Brain, Develop. Med. Child Neurol. 2008: 6 (5):455.

8. Coletta, et al., The 5-tier system of assessing fetal heart rate tracings is superior to the 3-tier system in identifying fetal academia. www.AJOG.org 2012: 226.e1-e5.

9. Cunningham, et al., Williams Obstetrics, 2010, 23rd Edition: 86.

10. Farrar, K., "Chronic cerebral arterial spasm," J. Neurosurg. 1975; 43:408.

11. Govaert, P., et al., Traumatic Neonatal Intracranial Bleeding and Stroke, Arch Dis Child. 1992 July; 67(7 Spec No): 840–845.

12. [1]Hanigan, W., et al, "Tentorial Hemorrhage Associated with Vacuum Extraction," 85 Pediatrics 1990: 534-539

13. Keeling J. (ed.), Intrapartum Asphyxia and Birth Trauma, Chapter 10, Fetal and Neonatal Pathology, 1993; 2nd Ed.: 240.

14. Kelly, John V., M.D., "Compression of the Fetal Brain," Am J Obstet Gynecol; 1963; 85:687

15. Kriewall, T. et al, Effects of Uterine Contractility on the Fetal Cranium, Chapter 7, A Short History of Obstetrics and Gynecology; 1960.

16. 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 2001; 34:1125.

17. Lindgren, L. "The Influence of Pressure Upon the Fetal Head During Labor", Acta Obstet. Gynecol. Scand., 1977: 56:303.

18. 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 Clin Orthop Relat Res. 1966 May-Jun;46:7-22

19. Macones, George A., et al., The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring; Update on Definitions, Interpretation, and Research Guidelines. OB/GYN, September 2008; Vol. 112, No. 3: 661-666.

20. Mann, L., et al., "The Effect of Head Compression of an FHR, Brain Metabolism and Function," Obstet. Gynecol. 1972: 39(5): 721-725.

21. Murray, M., et al. Powers: Stimulation and Hyperstimulation, Labor and Delivery Nursing: A Guide to Evidence-Based Practice; 2009: 179.

22. Murray, M., et al., Essentials of Fetal Monitoring, 2001; 4th Ed.: 107.

23. Newton, T., et al., "Compression of Superior Sagittal Sinus by Neonatal Calvarial Molding," Radiology 1975: 115:635-636.

24. O'Brien, W. et al., "Effect of Cephalic Pressure on the Fetal Cerebral Flow," Amer. J. of Perinatology 1984; 2:223.

25. 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, 1994; 101(1):44.

26. Physicians' Desk Reference, 52nd edition 1998: 114-115.

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

28. Towbin, A., Brain Damage in the Newborn and its Neurological Sequels 1998; see Figures 206 and Figure 207.

29. Volpe, 307

30. Volpe, 4th Ed.: 153-154

31. Yates, P., Birth Trauma to the Vertebral Arteries, Arch Dis Child. 1959 October; 34(177): 436.