

Review

Birth brain injury: etiology and prevention— Part I: Hypoxic-ischemic encephalopathy and cerebral palsy

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Abstract

Hypoxic-Ischemic Encephalopathy (HIE) is a birth brain injury that precedes the development of Cerebral Palsy (CP). Visualized on MRI scanning, the lesions of HIE are infarcts resulting from deficient generalized perfusion of brain tissue. Birth asphyxia/hypoxia is the widely assumed cause of this brain injury, however, HIE may occur without significant hypoxia. Hypovolemia/hypovolemic shock, is evident in the great majority of HIE newborns. Cord compression prior to birth pools fetal blood in the placenta and immediate cord clamping finalizes the hypovolemia. Heart failure (hypovolemic shock) follows.

Retraction respiration—pulses of negative intra-thoracic pressure—pulls venous blood into the heart and, with hypotension, pulls arterial blood into the thoracic aorta from peripheral arteries. Circulation in the heart and lungs is maintained at the expense of perfusion of all peripheral organs, including the brain. Prolonged deficient perfusion of the actively metabolizing areas of the brain (basal ganglia and cerebral cortex) results in infarction.

Thus the primary pathology in HIE/CP cases is massive blood loss into the placenta, not hypoxia. HIE/CP can be avoided by not clamping the cord and resuscitating all babies with the placental circulation intact.

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Keywords: birth brain injury, hypoxic-ischemic encephalopathy, cerebral palsy

1. Introduction

If a child is born alive in a modern hospital, it is very unlikely to die—neonatal mortality is a rare event; however, between 5% and 10% of newborns need intensive care, and the long-term morbidity of these children is considerable [1,2]. Perinatal and neonatal care is less successful in saving brains than saving lives. If resuscitation does not result in a five minute Apgar of 7 or more, permanent neurological impairment is likely [3]. Why do these neonates need intensive care, and what injures their brains?

Almost every child admitted to the NICU has been subjected to the same birth injury, immediate umbilical cord clamping (ICC); the placenta was amputated at the moment of birth. Neonatologists mandate this practice for neonatal rapid transfer to a resuscitation table [4], and the American College of Obstetricians and Gynecologists—ACOG [4-7] has promoted ICC to obtain cord blood samples for medico-legal evidence. The ICC procedure has been condemned in medical literature for over 200 years [8,9].

“Another thing very injurious to the child, is the tying and cutting of the navel string too soon; which should always be left till the child has not only repeatedly breathed but till all pulsation in the cord ceases. As otherwise the child is much weaker than it ought to be, a portion of the blood being left in the placenta, which ought to have been in the child.”

Erasmus Darwin, *Zoonomia*, 1801 3rd edition Vol. III. Page 302

During natural (physiological) birth, a large volume of blood is transferred from the placenta to the child—placental transfusion. (PT) The individual amounts vary greatly, but usually

range from 30% to 50% of the newborn's eventual normal blood volume [10]. The child clamps its own cord reflexively at the correct time when an optimal blood volume is achieved [11].

ICC interrupts and terminates PT. Thus almost every child admitted to the NICU is hypovolemic to some extent and they exhibit signs of blood loss [12] and deficient tissue perfusion: pallor, weakness, hypotension, hypovolemic shock, respiratory distress syndrome (RDS)[13] (shock lung), necrotizing enterocolitis (NEC), hypoxic-ischemic encephalopathy (HIE), oliguria, anuria, anemia, hypothermia and metabolic acidosis [14]. With deficient perfusion of the liver, hypoglycemia may result. *“Sick neonates are one of the most heavily transfused groups of patients in modern medicine [15].”*

The spectrum of injuries resulting from ICC and its incurred blood loss is very wide; the normal term child that cries when the head is delivered may receive a large PT and have a normal blood volume before the clamp can be applied [16]; it lies on the healthy end of a broad bell curve of injury. Many disorders are short term and may self-correct, but nervous system injuries that result in mental, behavioral and neurological deficiencies are life-long. The following discussion applies to severely injured term babies on the other end of that bell curve who have brain lesions that are identifiable radiologically.

2. Definition of the Injury and Causal Agents

The most common cause of (all) brain injury is loss of circulation—a stroke (arterial occlusion) or a period of cardiac arrest—loss of blood pressure, loss of perfusion. Integrity of brain tissue is maintained by constant, copious perfusion with blood;

the lesion produced by deficient perfusion is an infarct—dead tissue. Hypoxic-ischemic encephalopathy, (HIE) the precursor of cerebral palsy (CP), is visualized in the neonate using MRI scanning. Necrosis of brain tissue (infarction) is evident mainly in areas of high metabolic activity—the basal ganglia, the cerebral cortex and, in the preemie, the germinal matrix. The ischemia is generalized, not confined to an arterial distribution area [17]; these infarcts occur and progress after birth regardless of the oxygen content of the blood.

3. Hypoxia

Hypoxia/asphyxia is generally assumed to be the “cause” of brain damage, and brain damage (identical to HIE/CP) is readily produced in newborn primates by asphyxiation [18,19]. Babies that develop HIE are usually born “asphyxiated” [20], and ACOG [7] “strongly supports the concept that a neonate who had hypoxia **prior to delivery** severe enough to result in HIE will show other signs of hypoxic damage including all the following:

1. Cord arterial blood pH <7
2. Apgar 0-3 at 5 minutes
3. Neurological sequelae and [multi organ dysfunction]”

However, in a very comprehensive study on the timing of HIE, Cowan [17] contradicts ACOG, stating: “there is no evidence that brain damage occurs **before birth**.” However, Cowan’s criteria for the clinical definition of HIE:

1. Abnormal tone
2. Feeding difficulties
3. Altered alertness

Plus three or more of the following: (a) late decelerations, (b) delayed respiration, (c) cord pH < 7.1, (d) Apgar scores < 7 at five minutes, and (e) multi-organ dysfunction—implicate some degree of hypoxia or an “insult” near the time of birth. Cowan’s first three (mandatory) criteria for HIE eerily echo old descriptions of the immediately clamped child [21].

HIE demonstrated on MRI may develop without evidence of significant hypoxia [17]. Neonates with HIE are routinely oxygenated after birth. For the past 30 years, asphyxiated (fetal distress) babies have increasingly been rapidly diagnosed, rapidly delivered and rapidly oxygenated and intensively cared for. The incidence of CP has remained constant at about 1-2 per 1000 births for 30 years; correction and prevention of hypoxia does not appear to be very beneficial in preventing CP.

On closer examination of the asphyxiated experimental animal [19] (Figure 1), the primary lethal effect of asphyxia is cardiac arrest—the heart needs oxygen to beat and to generate blood pressure—after loss of blood pressure, brain infarction occurs and generalized infarction follows. If an “asphyxiated” child is born with a pulsating cord, the heart is receiving enough oxygen to perfuse itself and the placenta; the heart should also be perfusing and oxygenating the brain. Why then, after birth, with the heart beating and the lungs supplying oxygen, does HIE begin and progress? The fetal brain grows and develops while copiously perfused with blood that is not fully saturated with oxygen. Despite ACOG’s concept [7], hypoxia cannot be the major factor in neuron necrosis; what, then, is the origin of the ischemia in HIE?

4. Ischemia

Figure 1 illustrates the progress of an asphyxiated newborn monkey [19], cord clamped, trachea occluded. The heart fails from lack of oxygen and the BP falls eventually to zero—cardiac arrest—and brain damage ensues. During heart failure, gasping occurs and has a significant effect on cardiac function. With “gasps” there are spikes of increased pulse rate (increased cardiac output.) At the same time, there are downward spikes of diastolic blood pressure to the zero line (decreased cardiac output!) After resuscitation (ventilation with oxygen) and adrenaline injection there is rapid recovery; then the BP again falls and gasping returns persistently. Hypoxia does not cause this gasping.

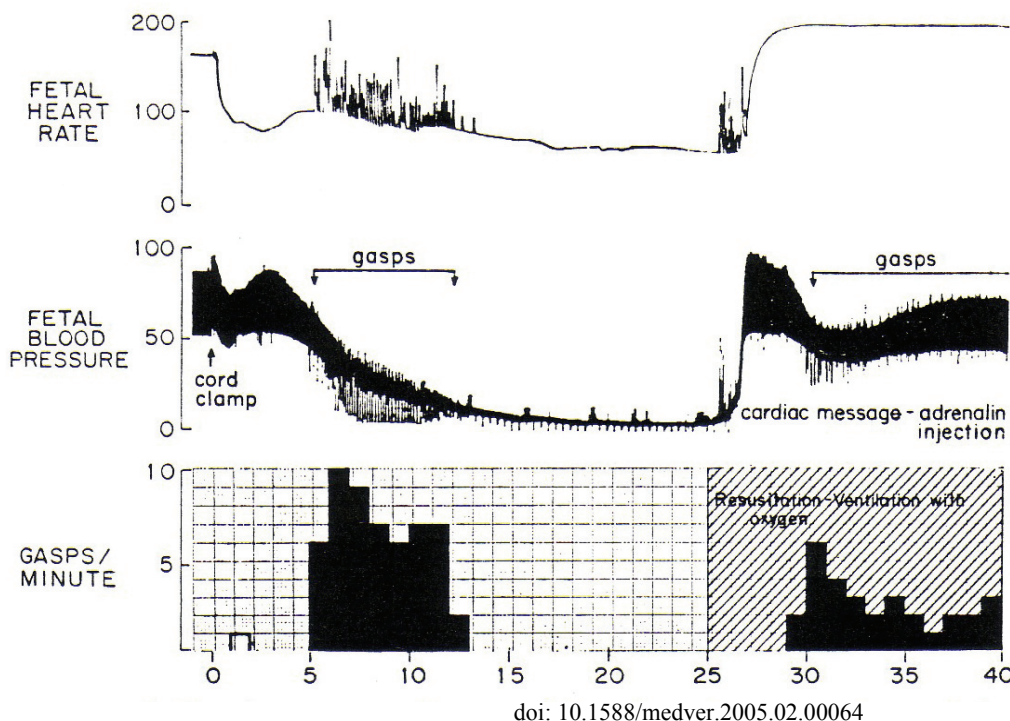


Figure 1. Heart rate and blood pressure changes (top) and gasp pattern (bottom) associated with 25 minutes of total asphyxia in Term Fetus 1735. Fifteen minutes of resuscitation are also represented. The gasp pattern is identifiable in the cardiometer and blood pressure recordings as well as in the bar graph. Efforts at cardiac massage during the 26th and 27th minutes also appear. Epinephrine, 0.1 mg, was injected intra-arterially at the beginning of resuscitation during the 26th minute. Gasping reappeared during the 5th minute of resuscitation. Reprinted with permission. Myers RE. Two patterns of perinatal brain damage and their conditions of occurrence. Am J of Obstet and Gynecol, 1972 Jan 15; [Figure 1] 112(2):247.

Note that the cord was clamped immediately at birth; the heart rate (and cardiac output) fell immediately by 50% in response to abrupt, total loss of oxygen supply and decreased venous return. A large amount of blood volume was clamped in the placenta.

Gasping is retraction respiration (RR) and is a reflexive response to cardiac failure/low blood pressure/low central venous pressure. It causes pulses of high negative pressure in the thorax that draw venous blood into the right heart and the lungs, and into the left heart if the foramen ovale is open – note pulse-rate spikes. With hypotension, it also pulls arterial blood back into the thoracic aorta from peripheral arteries—note collapses of peripheral diastolic blood pressure to zero or below zero. During gasps, blood may be drained from the brain arteries, and cerebral circulation may become tidal—it may virtually cease. The net effect of RR is to maintain some circulation and blood volume in the heart and lungs at the expense of all peripheral organs – multi-organ dysfunction [22].

5. Heart Failure: Hypoxic and Hypovolemic

The term “heart failure” means that the heart is not generating an adequate blood pressure. In Figure 1, gasping is first caused by hypoxic heart failure. After resuscitation, the animal (revived from “death”) is well oxygenated, but when the pulmonary circulation fills with blood, (ventilation dilates pulmonary arterioles) the BP collapses and heart failure with gasping recurs—a major portion of the blood volume was clamped in the placenta at birth. This hypotension/heart failure is hypovolemic in origin, not hypoxic. In this particular monkey, the hypovolemic heart failure was not progressive; the animal adapted to moderate hypovolemia with generalized vasoconstriction. RR in an oxygenated human neonate is a sign of hypovolemic heart failure requiring immediate blood volume replacement; persistent RR with progressive hypotension will result in cerebral ischemia and possible brain damage.

Thus hypovolemia/hypovolemic shock (rather than *hypoxia*) is a much more probable and more plausible cause of the metabolic acidosis, low Apgar scores, multi-organ dysfunction and HIE that ACOG assumes to be hypoxic damage [7]. ACOG specifically mentions *hypoxic* gastro-intestinal injury and renal dysfunction [7]. However, in the fetus, the gut and kidneys are perfused with the same hypoxic blood that is going to the placenta to be oxygenated! With adequate perfusion, the gut and kidneys grow and thrive on this hypoxic blood supply; with inadequate perfusion/hypovolemic shock, regardless of oxygenation—infarction occurs—renal cortical necrosis and NEC. In this hypovolemic situation, especially if the child is “retracting,” the brain is also very susceptible to infarction. In the adult during terminal hypovolemic shock or heart failure, gasping or RR is termed “air hunger.”

6. Intra-partum Hypoxia/Intra-partum Hypovolemia

Despite some pallor, most normal, term babies seem to adjust to blood loss caused by ICC [10]. However, in the “high risk” “asphyxiated” depressed, term baby, the sequence of ICC at birth, delayed breathing, with hasty resuscitation and ventilation often results in hypotension, generalized weakness, respira-

tory distress syndrome (RDS, RR) and a low 5-minute Apgar score. Cord compression during birth—a tight nuchal cord [23,24], a firm, true knot, or a prolapsed cord – is the most common cause of birth asphyxia / fetal distress. Cord compression does much more than asphyxiate the fetus.

Intra-partum cord compression acts as a venous tourniquet with umbilical arteries engorging the placenta while the compressed vein allows a minimal amount of oxygenated blood to flow back into the child—producing prolonged or late decelerations on the FHR monitor. (See Fig. 2 and Cowan’s criteria.) Cord compression thus produces fetal hypoxia and **fetal hypovolemia—the fetal blood volume shifts to the placenta.** [23, 14] Linderkamp [24] terms this “Intra-partum Asphyxia.” Eventually, placental engorgement increases umbilical venous pressure that overcomes compression; oxygen return to the fetus improves and the “fetal distress” appears to be relieved. However, the placenta remains engorged, the child remains very hypovolemic and it may be in shock [12,16,23]. In the extreme, the child is born ashen pale, “dish-rag” limp with pea-soup meconium; most of its blood volume is in the placenta. The routine neonatal management of this child is ICC followed by immediate ventilation.

7. Hypovolemia in Extremis

Unlike the monkey fetus in Figure 1, the cord-compressed human fetus is hypovolemic before and at birth, [23] and ICC removes a massive portion of feto-placental blood volume. What little blood that is left in the child may flow into the lungs, but the peripheral circulation then collapses. Five minutes of intensive resuscitation (intubation, oxygenation, central venous lines, arterial lines, dopamine) may produce a five-minute Apgar below 7, and the child will enter the NICU oxygenated, but with marginal vital signs. If intravenous fluids maintain tissue perfusion, the child may stabilize; not infrequently, the child stops breathing, and frantic resuscitation occurs with mechanical ventilation. Then, hours after birth, the child convulses, announcing the onset of HIE, and the brain is treated with phenobarbital. Two or three weeks after birth, the hemoglobin is 10 gm% confirming that the child lost 50+% of its blood volume at birth, and red cell transfusion [15] is given. “Nelson and colleagues have noted that a tight nuchal cord was seen at delivery significantly more often in infants with neonatal encephalopathy than in controls [17].”

Hypoxia caused by cord compression entails, even guarantees hypovolemia; the fetal oxygen supply is dissolved in blood. ACOG’s concept of fetal “hypoxia severe enough to result in HIE” thus implies the tandem concept of fetal “hypovolemia severe enough to result in HIE.” To avoid hypovolemic injury, immediate blood volume replacement is needed, not oxygen.

8. Physiological Resuscitation with Placental Transfusion

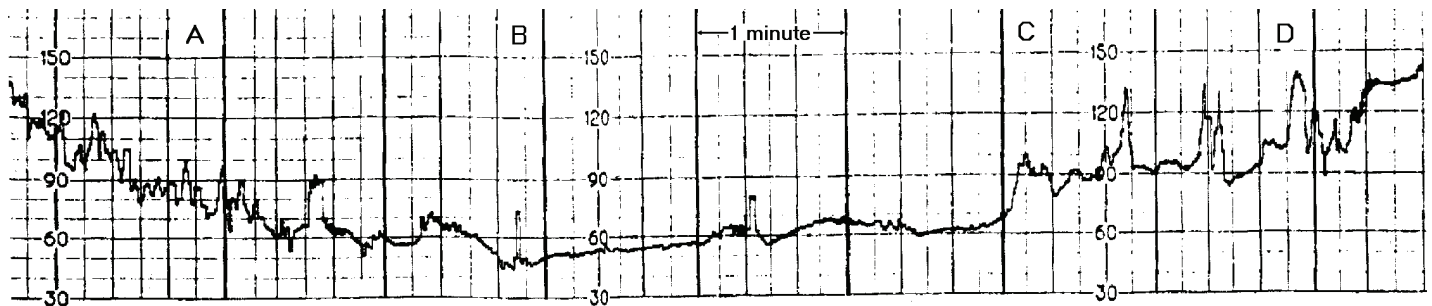
The only way to avoid the above scenario is to resuscitate with the placental circulation intact; **do not clamp the cord** [10, 14]. A nuchal cord should never be clamped and cut. If it cannot be reduced over the head or the shoulder, allow delivery to proceed while manipulating the head and neck towards the vulva and unwind the cord after delivery. Position the child

below the level of the placenta for gravity placental transfusion. With cord compression relieved, (release the nuchal cord, loosen the firm knot) massive placental transfusion replaces the child's blood volume, establishes copious pulmonary blood flow and perfuses/oxygenates all vital organs. Within a minute or two, neonatal cardiovascular hemodynamics are restored to a normal, healthy status.

The tracing of Figure 2 was discovered twenty minutes after it occurred. It continued with good FHR variability but with

minor late decelerations, never below 100 BPM. Occult cord prolapse was found at C-section and the child (no meconium) was pale and somewhat limp at birth; cord pulsation was vigorous, the lips and mucous membranes were purple. Intravenous oxytocin was given to the mother to contract the uterus to hasten placental transfusion. The child was breathing at one minute and was crying, squirming and ruddy pink (no pallor) at four minutes when cord pulsations ceased. The cord was then clamped and cut. Five minute Apgar score ten.

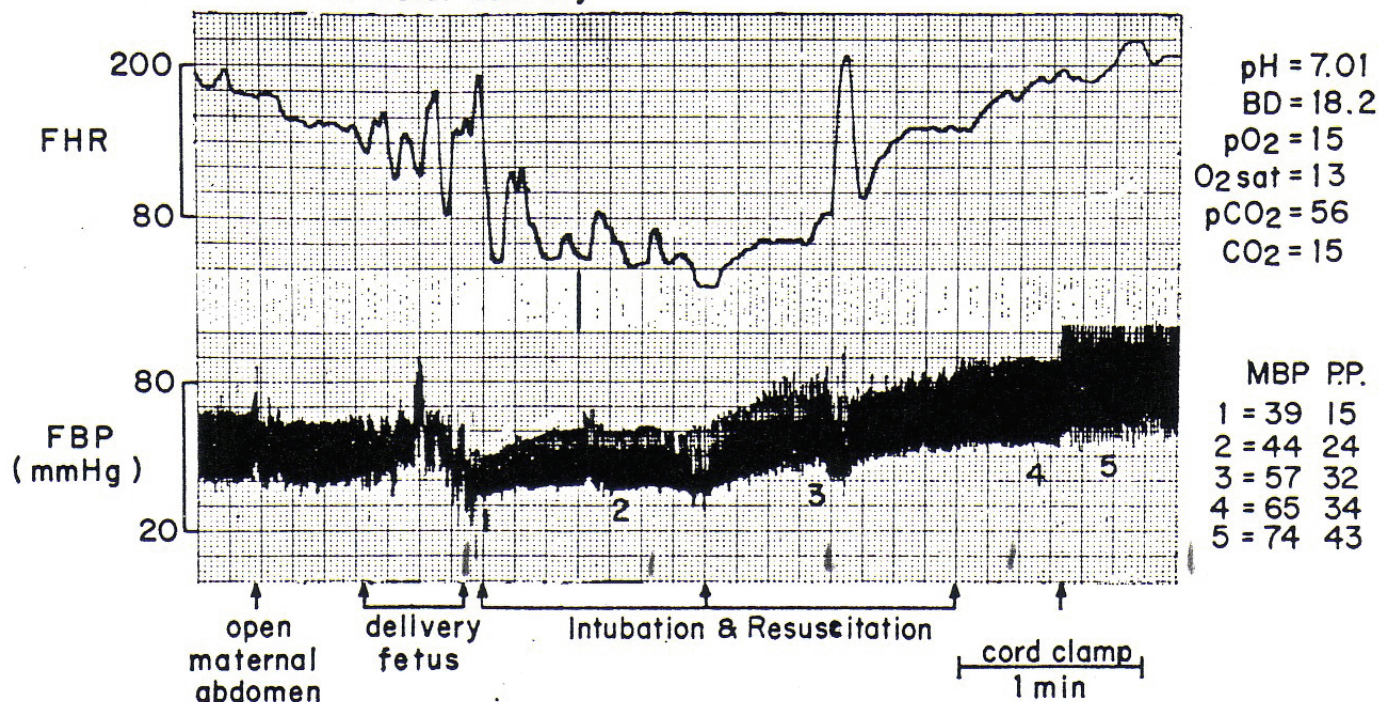
Figure 2. A prolonged fetal heart rate deceleration typical of the initial incident of cord compression showing progressive bradycardia as hypoxia increases. (A) severe hypoxia with FHR below 60 bpm. (B) then gradual relief of hypoxia as placental venous pressure increases to overcome the compression after the arteries engorge the placenta. (C & D)



The cord-compressed, “asphyxiated” neonate has an enormous resuscitation potential stored in the placenta that will nearly always guarantee full recovery if the cord is not clamped; the

abruption placenta neonate and Linderkamp’s “intra-uterine asphyxia” fetus [24] do not possess this advantage.

Figure 3. Rapid Cesarean section delivery of an asphyxiated monkey fetus. During delivery and intubation, severe bradycardia and hypotension developed. However, with 100% oxygen ventilation, the heart rate and blood pressure again increased toward normal values. Umbilical cord clamping after resuscitation led to a sudden jump in the mean blood pressure (from 65 to 74 mm Hg). Printed with permission. Myers RE. Two patterns of perinatal brain damage and their conditions of occurrence. *Am J Obstet Gynecol*, 1972 Jan 15; [Figure 16] 112(2):262



9. Abruptio Placenta: Pure Asphyxia

Placental abruption removes placental respiration; this is pure hypoxia/asphyxia without blood volume shifting. Myers' Figure 16 (3) [19] shows the effects of pure asphyxia. The mother was breathing nitrogen and the falling fetal heart rate indicates the increasing hypoxia while the cord remains unclamped. Despite the increasing hypoxia, the BP never fell to the extent shown in Figure 1, but it would have done so if delivery and resuscitation had not intervened. The second half of the tracing confirms the value of resuscitation with the placental circulation intact [10,14]. The pulse rate and the blood pressure recover, and, in marked contrast to Figure 1, the pulse rate and blood pressure *increase* when the cord is eventually clamped – after the lung vessels filled with placental blood. This monkey (+cord +placenta) always had a normal, ample blood volume with continuous tissue perfusion despite hypoxia; it also had no brain damage.

If delivery had not occurred, hypoxic heart failure would have ensued together with gasping, and the general pattern of Figure 1 would have been repeated. Severe brain ischemia (five minutes or more of zero BP will suffice) would produce brain infarction in utero—similar to the monkey in Figure 1. This situation comes close to concurring with ACOG's concept of hypoxia causing HIE, but heart failure and loss of perfusion actually produce the brain infarcts, hypoxia (extreme) causes the heart failure.

If abruption babies can be delivered and resuscitated with the placenta still attached before they are markedly “depressed,” they should recover well. If they are born limp and a-reflexic, they do not have a large reservoir of oxygenated blood in the placenta to rescue their brains—that may be already damaged. Anecdotally, several neonatologists have mentioned that the depressed abruption neonate is the least likely to respond well to resuscitation, implying that ante-partum brain damage has already occurred.

If cord compression is strong enough to occlude all the cord vessels, the child is in the same situation as the complete abruption baby—rapid anoxic heart failure, brain death and demise follow soon. This may occur in cord prolapse when the cord is outside the vulva. Once the cord reaches the atmosphere, water evaporates from Wharton's jelly and the cord cools rapidly. All the cord vessels respond to cold with constriction and closure. Most cord prolapse cases that present with an exteriorized cord produce a stillborn. If the cord is still pulsating, replacement in the warm, moist vagina and prompt c-section may save the child's life.

10. Intra-Uterine Asphyxia/Oligohydramnios

Linderkamp described the “intrauterine asphyxia fetus” [24] as exhibiting a shift of blood volume from the placenta to the fetus. This situation happens in oligohydramnios; the cord is not “cushioned” by fluid and the umbilical vein becomes generally compressed. As in acute cord compression, the placenta becomes engorged and chronic placental capillary hypertension results. This forces fluid from the fetus into the maternal circulation and the whole feto-placental unit becomes progressively dehydrated.

In the extreme, there is generalized vasoconstriction of placenta, cord and fetus, loss of interstitial fluid, severe hemoconcentration to the point of hypovolemia, oliguria and/or anuria. At birth, the cord vessels may appear as three “pencil strings” in Wharton's jelly. There is no placental transfusion available. This is the perfect “hyperviscosity syndrome [16].”

Today this situation seldom occurs; ultrasound diagnoses and amnio-infusion corrects the pathology and the child is rarely born “in extremis.” If it is, the clinical assessment and management should be based on the condition of the cord. “Pencil strings” should be clamped and cut immediately and catheters placed for immediate replacement of fluid and correction of acidosis by the neonatologist. If there is palpable cord pulsation with the FHR above 100 bpm and the vein is filled, try to allow the placental circulation to restore hydration, maintain life support and supply what placental transfusion may be available during the third stage while pulmonary resuscitation continues. Bradycardia is an indication for clamping and cutting the cord to give rapid IV fluid replacement.

The only other situations that justify clamping a pulsating cord are spontaneous cord rupture at birth and incision of an anterior placenta previa at cesarean section. Plasma volume expanders should be used liberally in such cases, and preservation of placental blood for autologous transfusion should be attempted.

11. Discussion

Current neonatal resuscitation [4] – ICC and immediate ventilation—violates the most basic concept of rational medical care—restoration of normal form and function without harm. If a child is born depressed—obviously incapable of spontaneous breathing – the condition is not due to failure of its lungs, but to dysfunction of organs and systems that are needed to initiate spontaneous breathing—usually the cord and placenta. If the cord is pulsating, the dysfunction is not total—the cord and placenta are keeping the child alive; placental/cord function should be maintained and restored, not amputated, to maximize recovery of the child.

This principle is used to correct fetal distress in utero—changing the mother's position, or giving amnio-infusion to relieve cord compression, or elevating the presenting part from a prolapsed cord. After birth, during the third stage of labor, use of this principle is essential in successfully “reviving” the depressed newborn; it permits the normal, orderly process of transition from placental life support to activation of the neonate's own organs of life support. If an apneic neonate's cord is beating at 100+ bpm, the child has an adequate placental oxygen supply; it is not asphyxiated. The color of mucus membranes will be purple, not pink, due to fetal circulation. Cord clamping creates rapid, total asphyxia.

In the apneic newborn, ICC precipitates panicked ventilation to reverse the iatrogenic total asphyxia. If the child does not have sufficient blood volume to perfuse the ventilated lungs and convert the fetal circulation to the adult, the asphyxia will not be reversed. During physiological birth, placental blood volume is transfused to the lungs, and after ventilation and establishment of lung function, oxygenated blood flows through the umbilical arteries—they respond with constriction and closure.

Physiology thus maintains placental respiration until pulmonary respiration is well established; there is no period of asphyxia in physiological transition, and there is no hypovolemia.

ACOG's experimentation with ICC [5, 6] may be in demise; ACOG quietly withdrew bulletins 216 and 138 from publication in 2001 and 2003 following publication [25] and exposure [26] of ICC's injurious effects. However, ACOG Bulletin 303 still mentions cord arterial pH as an evaluation tool. ACOG has not issued any caution regarding ICC, yet ICC, required to procure cord arterial blood samples, correlates strongly with the development of HIE and CP in "asphyxiated" neonates [17,20, 27,28]. The Cochrane Review [4] advises that delayed clamping be employed to avoid brain damage in preemies. Parents have been advised not to use aspirin on children because of occasional correlation with the brain damage of Reyes' syndrome. The comparative risks of ICC surely need mention.

The perinatal professions have never performed a random, double blind, controlled, scientific study that compares the results of feto-placental physiology during the third stage of labor (no cord clamp used) with the results of immediate iatrogenic destruction of the umbilical circulation with a cord clamp. Yet amputation of the functioning placenta remains routine, standard care, with major Neonatology (Educational) publications stating, "For most term or near term infants, the time of cord clamping may not matter [4]." Meanwhile, the American Academy of Pediatrics (AAP) advises that delayed cord clamping be used to avert the need for blood transfusion, and NeoReviews [4] advises that the obstetrician "wait a minute" before amputating the preemie's placenta to avoid brain damage. The procedure of ICC with rapid ventilation is very difficult to justify; clamping a pulsating cord is also very difficult to justify.

The above, proposed random study would never occur; it requires the legal informed consent of the parents. Once legally informed (full disclosure) [29] about ICC, parents will opt for non-random alternative care. The ethical physician/midwife should be readily available.

Many, if not most midwives, especially with out-of-hospital deliveries, routinely delay cord clamping and cutting until the placenta is delivered. The apocryphal complications of physiological cord closure—hyperviscosity, [16] polycythemia, plethora [16] and jaundice [4] do not fill NICUs with home birth babies. In every other aspect of medical practice, physiology is maintained and restored. Third stage neonatal/cord/placental physiology is misunderstood, ignored, disrupted and destroyed; the consequences of that ignorance fill NICUs. An informed consent document, [29] signed by doctor/midwife/parents, that defines why, when and whether the cord should be clamped, should restore perinatology to objectivity and reason.

12. Conclusion

The large majority of HIE/CP babies result from ICC performed on an already hypovolemic infant that has suffered cord compression during birth. Brain damage is ischemic in origin and occurs after birth as a result of hypotension/hypovolemic heart failure usually compounded by retraction respiration. HIE/CP in these neonates is preventable by not clamping the pulsating cord and by resuscitation with the placental circulation intact. Placental transfusion restores normal perfusion of

the infant's life-support organs. In a minority of cases, such as total abruption of the placenta, HIE/CP may occur prior to birth caused by anoxic heart failure—and may be unavoidable. Routine care at every delivery should include not clamping the umbilical cord until pulsations have ceased and until the child is breathing and pink.

Conclusion by email

Hi Dr. Morley, I just did a birth yesterday that was a perfect example of your theory, (one which I have followed for 25 years). I had a 40 year old primip with a long but normal labor and with good FHR throughout until a sudden drop at crowning, down to 60 with no return to baseline, after a quick episiotomy and quick birth the baby had already entered 2nd apnea, I saw it "gasp" before the head was born. The baby had a nuchal cord as well as a true knot! She came out in a flood of thick meconium that she had inhaled during her birth. No pulse was palpable at birth but with the cord intact the pulse quickly came up to normal (Apgars 2, 7, 9) and with a lot of suctioning and her parents touching her and talking to her and her cord pulsating with a little bag and mask she is fine and now fully up to "normal" newborn status. When I think back to how this situation would have been handled in places where I have worked (I work in an out-of-hospital birth center now) I know this baby would have ended up in the NICU with mec pneumonia, the cord would have been cut, the neonatologists would have "saved" the baby, and who knows where the child would end up years later. Tracey A., CNM

Addendum—Retraction Respiration: A Simulation.

Retraction respiration is widely regarded as the significant sign of neonatal respiratory distress. To have retraction respiration redefined as the significant sign of hypovolemic shock and heart failure, without any pulmonary patho-physiology, will, no doubt, give rise to some controversy.

The causality of ischemic lesions that result from sudden, severe, neonatal episodes of negative intra-thoracic pressure will be equally controversial. The following adult simulation of retraction respiration should clarify resolution of these disputes.

While recording the radial pulse rate, breathe out fairly completely, then close the mouth and one nostril; inspire vigorously, maintaining the effort until the lungs are filled with air.

The radial pulse disappears during forceful inspiration.

Two mutual factors combine to produce this temporary "cardiac arrest:"

1. High negative intra-thoracic pressure counteracts thoracic aortic blood pressure.
2. High negative intra-thoracic pressure pulls blood into the pulmonary veins from the left atrium, preventing ventricular filling and briefly stopping cardiac output.

In a person with a normal blood volume and normal blood pressure, recovery is almost instantaneous, and the long "gasp" is harmless; but in a neonate with a systolic blood pressure of 50 mm Hg, with a very deficient blood volume and with a marginal cardiac output, repeated "gasping" every ten to twenty seconds will remove a significant portion of blood volume from systemic organ perfusion; retraction respiration thus becomes a very plausible cause of ischemic injury.

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