

TIMING OF ASPHYXIAL INJURY AND BIRTH TRAUMA

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Obstetric Malpractice cases involving asphyxiated newborns present unique challenges to Plaintiffs' counsel. Standard of care experts will review the intrapartum clinical information to determine the time that delivery should have taken place in the absence of a breach of the standard of care. This analysis is often based on very limited intrapartum information available regarding fetal status. The primary and most common tool to evaluate intrapartum fetal status is through the fetal heart rate. The fetal heart rate may be measured through intermittent auscultation or, more commonly, electronic fetal monitoring (EFM). Other clinical information regarding fetal status may be available through the use of scalp sampling.¹

The timing of asphyxial injury is the crucial causation issue. Determining when an irreversible brain injury occurred in relation to the time at which the standard of care expert said the baby ought to have been delivered is a difficult challenge scientifically and legally. Precision in timing is often required for litigation. Precision from a scientific point of view is controversial. Despite the difficulty faced in establishing timing of asphyxial injury, the burden rests with the Plaintiff to establish that the injury occurred after the time at which the standard of care expert said the baby ought to have been delivered.

Asphyxial injury occurs when oxygen delivery to fetal organs, and particularly the heart and brain, is impaired or interrupted. Ischemia or a reduced blood supply, results in impaired oxygenation. A certain volume of oxygenated blood must be supplied to organs to avoid injury. Reduced perfusion results in inadequate oxygenation and can lead to injury. A decreased heart rate means less perfusion of blood, impaired oxygenation and the risk of harm.

This paper will look at some crucial issues in the timing of asphyxial injury. Those will include the following:

1. Fetal Heart Rate Changes
2. Cord Blood Gases
3. The Nature of the Asphyxial Insult, Brain Anatomy and Neuroimaging
4. Neonatal Haematology
5. Antepartum and Other Causes

¹ Scalp sampling involves obtaining a blood sample from the fetal scalp for analysis of fetal pH.

FETAL HEART RATE CHANGES

Fetal well being in utero is often best assessed through observations of the fetal heart rate. There are generally two ways of monitoring the fetal heart rate in utero. One method is through intermittent auscultation, where the observer will use a Doppler ultrasound to listen to the fetal heart rate periodically. The second, and more common way of monitoring the fetal heart rate, is through the use of electronic fetal monitor. The electronic monitor provides a continuous paper tracing of the fetal heart rate which can be used to assess fetal well being. EFM is generally done with the placement of an external transducer on the mother's abdomen that detects the fetal heart rate. A more direct and accurate way to measure the fetal heart rate would be through the use of a scalp clip or scalp electrode, which is an invasive technique requiring the device to be applied directly to the fetal head. This can only be done when the membranes have ruptured and there is at least two to three centimetres of cervical dilatation.

For the purposes of this paper, the discussion will be focused on electronic fetal monitoring and the paper printout that is generated. While this method of surveillance of fetal well being is available for both labouring and non-labouring situations, the discussion will be primarily focused on EFM during labour.²

The fetal heart rate pattern generated through EFM is assessed and interpreted based on certain standard parameters. Certain patterns are considered "normal" and are suggestive of fetal well-being. In fact, a normal fetal heart tracing can be taken as a reliable indicator that the fetus is coping well in the intrauterine environment.

A fetal heart rate tracing that is not normal will not allow the observer to be reassured about fetal well-being, but does not necessarily mean there is a problem. Commonly perfectly normal babies are born in the setting of an abnormal fetal heart rate tracing.

The electronic fetal monitoring will measure the fetal cardiovascular status. The tracing can provide important information on how the fetus is coping in the intrauterine environment. Certain well understood patterns exhibited on the tracing are known to indicate the potential for fetal distress and fetal compromise. The purpose of monitoring with EFM is for the obstetrical team members to be alerted to indications of potential problems in the intrauterine environment so that interventions can take place before fetal compromise.

Nurses and physicians assess the fetal heart rate tracing for certain characteristics. They include the baseline heart rate, baseline variability and periodic changes. Periodic changes can be broken down into accelerations and decelerations. Decelerations can be further broken down into early, late, variable and prolonged. Although the terminology used by obstetricians changes, for the

² EFM during labour will be referred to as the intrapartum period.

purposes of this discussion the fetal heart rate tracing pattern will be described as either reassuring or non-reassuring.³

The fetal heart rate baseline is said to be normal if it falls in the range of 120 to 160 beats per minute (bpm). The paper tracing has a vertical scale on the top half of the paper for the baseline heart rate. When the baseline falls below the normal range, the fetal heart is said to be bradycardic or the fetus is experiencing bradycardia. This simply means an abnormally low heart rate. Bradycardia is associated with poor oxygen perfusion to the heart. This causes a drop in the heart rate and a drop in the fetal blood pressure. Where the baseline is above the normal range, it is said to be tachycardic or the baby experiencing tachycardia.

The fact that the fetal heart rate is in the normal baseline range suggests fetal well-being. It cannot be taken, however, as an indication that all is well in the intrauterine environment. A fetus can be suffering from impaired oxygenation and still have a normal baseline heart rate. Generally the normal baseline heart rate will indicate that the fetus is coping in the intrauterine environment, but other patterns must be assessed in order to be satisfied of fetal well-being.

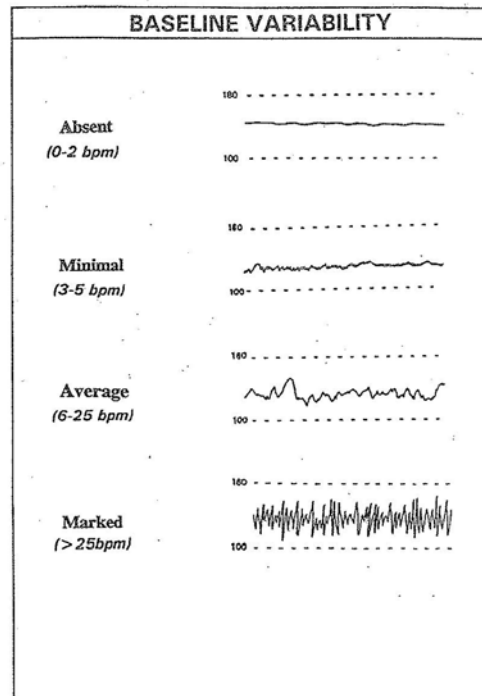
A drop in the fetal heart rate has two important consequences for the fetus. First, the drop in heart rate will result in a decrease in fetal blood pressure (hypotension). Second, the drop in the fetal heart rate will result in decreased perfusion of blood to the fetal organs. The second consequence really results from the first. That is, hypotension in the fetus will decrease perfusion of blood to fetal organs. Decreased perfusion of blood to fetal organs results in lower oxygen supply to the organs and impairs the ability of the organs to rid themselves of waste products from metabolism.

From a causation point of view, therefore, an indication of hypotension through an observed drop in the fetal heart rate may indicate impaired oxygenation. Impaired oxygenation, though it can cause harm to the fetus, does not necessarily mean that injury has occurred. It is important, therefore, to recognize the distinction between an insult and an injury. Where oxygenation to the fetus is impaired but the fetus is compensating for the decreased oxygenation, it can be said that there is an insult but no injury. At some point with impaired oxygenation the ability of the fetus to compensate is exhausted and the fetus will decompensate or suffer injury. Observations about changes to the baseline cannot pinpoint the time when insult becomes injury with sufficient accuracy without looking at other parameters.

Another characteristic of the fetal heart rate pattern important to an assessment of fetal well being is that of reactivity. Reactivity of the fetal heart rate requires the presence of “accelerations”, or increases in the baseline of 15 beats per

³ New guidelines from the SOGC (Fall 2007) have suggested abandoning the use of “reassuring” and “non-reassuring” to classify fetal heart rate patterns, in favour of “normal”, “atypical” and “abnormal”.

minute lasting 15 seconds. Another very important observation in the interpretation of fetal well being is variability of the fetal heart rate. Variability of the fetal heart rate refers to the fluctuations of the heart rate around the baseline. Average variability is normal and a reassuring pattern. If the fluctuation of the fetal heart rate around the baseline is less than five beats per minute, then variability is set to be reduced or minimal. Reduced or minimal variability, reflected in a relatively flat tracing pattern, is not a reassuring sign and maybe an indication of impaired oxygenation to the fetus.



Periodic changes refer to changes in the fetal heart patterns that are associated with uterine contractions. Periodic changes include accelerations and decelerations. For the purposes of analyzing asphyxial injury timing, the important periodic change is the deceleration. Decelerations are classified as early, late, variable or prolonged. Early and late decelerations are described in relation to uterine contractions. That is, a deceleration that occurs before a contraction is considered early. A deceleration in the fetal heart rate that occurs after the contraction is late. A variable deceleration is a deceleration that has a particular shape to it and is often associated with compression of the umbilical cord. Prolonged decelerations are decelerations that last for a period of time that may be of concern.

With respect to decelerations, there are two kinds of decelerations that are particularly important. Those are late decelerations and variable decelerations. Late decelerations are often associated with reduced blood flow during a contraction. This results in a lowering of fetal oxygenation which is reflected in the drop in heart rate. Variable decelerations are often associated with umbilical cord compression or occlusion. Variable decelerations can be particularly concerning depending on the frequency and the depth of the deceleration.

Prolonged decelerations are strong indicators of potential problems for the fetus in the intrauterine environment. Different standards describe prolonged decelerations in different ways, but a prolonged deceleration is probably a deceleration that lasts at least two minutes and has a sudden onset. They also should represent a drop in baseline of about 30 beats per minute or more. Prolonged decelerations are an indication of decreased placental blood flow and impaired oxygenation to the fetus which will result in hypoxia.

Deviations of the fetal heart rate from normal do not necessarily indicate injury to the fetus. In other words, there may be a fetal heart tracing that has potentially worrisome decelerations and other changes and yet a perfectly healthy fetus at delivery. This is referred to as a false positive. The importance of this is that fetal heart patterns are reliable indicators of fetal well being but are often unreliable indicators of fetal distress or compromise. The purpose of fetal surveillance, however, is to allow observations to be made about fetal response to the intrauterine environment so that the obstetrical team can intervene before insult becomes injury. From a causation standpoint, the presence of a reassuring tracing will indicate that injury has not yet occurred. The presence of a non-reassuring tracing may or may not suggest injury.

Since non-reassuring fetal heart rate patterns do not necessarily mean the fetus is distressed, timing of asphyxial injury may be difficult to pinpoint from heart rate patterns alone. Some more dramatic or worrisome patterns can be highly suggestive of harm, but fetuses have a remarkable ability to compensate in a stressful intrauterine environment. Consequently, for the purposes of timing asphyxia injury all relevant clinical and diagnostic information must be evaluated along with the fetal heart rate pattern.

THE NATURE OF ASPHYXIAL INSULT, BRAIN ANATOMY AND NEURO-IMAGING

Impaired oxygenation to the fetus can arise from many different causes. Cord compression, uterine rupture and placental abruption are but a few. The degree to which oxygenation becomes impaired can vary as well. The extent of the damage suffered by a fetus will depend on the degree of impaired oxygenation and the duration of impaired oxygenation. The more severe the degree of impaired oxygenation, the greater risk of permanent neurological injury. The longer the duration of the impaired oxygenation, the greater the risk of neurological injury. The challenge in these cases is to find out when the degree and duration of impaired oxygenation has resulted in irreversible neurological harm.

Where oxygen delivery to the fetus is totally cut off, the fetus is said to suffer from a sudden profound asphyxia often called an acute total asphyxia. Almost as severe would be a near-total asphyxia. This event represents a complete interruption in the supply of oxygen. This total lack of oxygen will, as one would

assume, be tolerated for only a relatively brief period of time before there is permanent neurological damage and, if unabated, will cause fetal death. Where the degree of impaired oxygen delivery is less dramatic, the fetus is said to suffer from a prolonged partial asphyxia. The fetus is equipped with a number of compensatory strategies that allow it to withstand this impaired oxygen gas exchange for hours, if not days. The ultimate impact of impaired gas exchange on the fetus will depend on the duration and magnitude of the insult.

A fetus may suffer from a prolonged partial asphyxia which is then followed by a profound total asphyxia. In these circumstances, the fetus is said to have suffered from a "mixed pattern" of asphyxia.

The placenta, in part, operates as a means of gas exchange between mother and fetus. Oxygen (O₂) is delivered to the fetus and carbon dioxide (CO₂) and other metabolites are expelled. When this gas exchange is impaired there is less oxygen in the fetal blood, known as hypoxemia and a build up of CO₂ in the fetal blood. Asphyxia is simply the impaired blood gas exchange which leads to progressive decrease in O₂ and increase in CO₂ leading to a condition within the fetal blood called "metabolic acidosis". Increasing levels of metabolic acidosis increases the risk of neurological injury, among other things.

A gradual and incomplete impairment of gas exchange, as stated earlier, is prolonged partial asphyxia and can last for hours, and perhaps days, without causing permanent neurological harm to the fetus. Fetal response to this impaired gas exchange can be revealed through changes in the fetal heart rate pattern as monitored on the EFM tracing. Therefore, clinical indications of a fetus that might be decompensating due to prolonged partial asphyxia will be revealed on the tracing.

As stated earlier prolonged partial asphyxia causes a gradual increase in the build up of acids in the blood leading to metabolic acidosis. The fetus has three main compensatory responses to increasing acidosis. These compensatory measures help the fetus avoid any permanent injury from arising acidosis.

The initial response to a build up of acids is called the "buffer stage". The fetal blood contains natural alkaline (base) chemicals which are said to buffer or neutralize the acids when they build up. The buffering capacity of the blood can hold off the acidification of the blood for some time. The stages that occur to this point with prolonged partial asphyxia are as follows:

1. An event causes some impairment of gas exchange.
2. There is a build up of acid so that there is more acid than base in the fetal blood.
3. A chemical reaction occurs which neutralizes the build up of acid (called the buffer).

During the buffer stage there is no risk of neurological injury to the fetus. When the body detects that more than just the natural chemical buffers are needed to

neutralize the building acidity the fetus initiates a cardiovascular compensatory measure.

The cardiovascular stage of fetal compensation for increasing acidosis involves a change in blood flow. There are two significant aspects to the cardiovascular response. First, blood is directed from non-essential organs to essential organs. The essential organs that receive preferred blood flow are the heart, brain and adrenal glands. The second aspect of the cardiovascular response is to cause blood vessels to be dilated and fetal blood pressure to increase, allowing a greater volume of blood to be circulated. In this way the essential organs continue to be adequately perfused with blood.

The increased blood supply to the heart and the dilated blood vessels allow the heart to pump enough blood to the brain to avoid any neurological injury. As long as this process continues, the increased perfusion of blood to the brain compensates for the reduced oxygen in the blood.

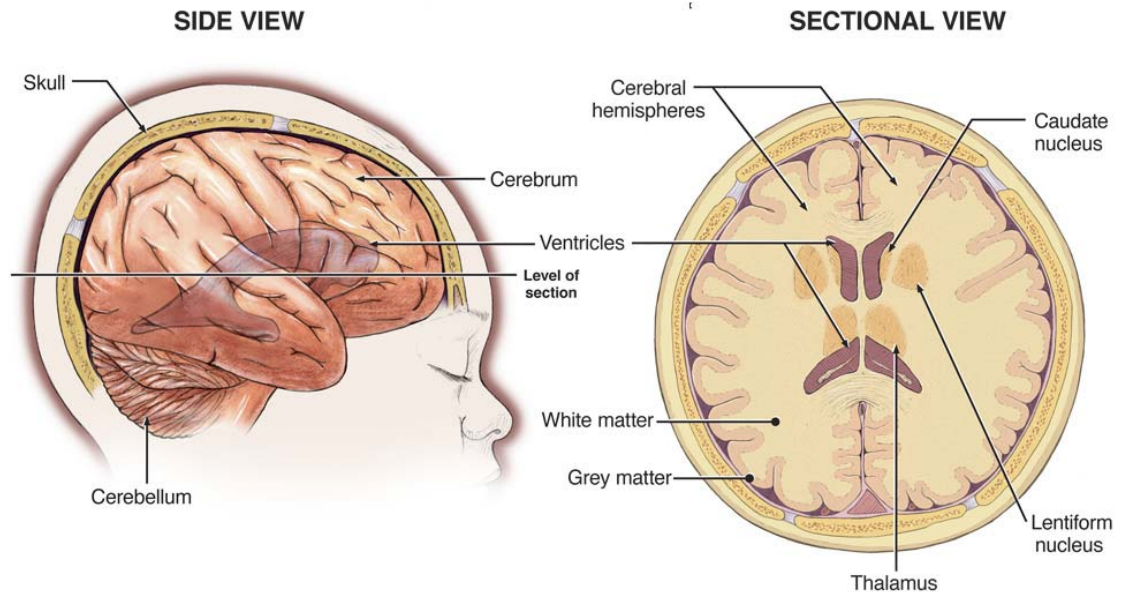
The adrenal glands, which sit on the kidneys, are essential organs and are protected because they are important in stressful situations. The adrenal glands release important hormones that help the fetus cope with the stress of impaired oxygenation. Some hormones released from adrenal glands increase the pumping action of the heart, constrict some vessels to non-essential organs and dilate blood vessels to essential organs.

During the cardiovascular stage of fetal compensation, the brain is protected from injury. The increased blood flow delivers oxygen and allows the brain to get rid of acid. Like the buffer stage, the cardiovascular stage provides a level of protection to the fetal brain during an increasing acidosis.

As the acidosis worsens, the brain may require more protection to avoid injury.

Following the cardiovascular stage, there is another compensatory measure that provides added protection to the brain. This is the brain blood flow stage. Increasing acidosis triggers a response within the brain to direct more blood to the areas of the brain with higher metabolic needs and less blood to the areas of the brain with lower metabolic demands. The areas of the brain with the higher metabolic demand are the deep structures, including basal ganglia and thalami. The areas of the brain with less need for oxygen are the outer areas of the brain, referred to as the cerebral hemispheres or the cerebral cortex.

EARLY NORMAL FETAL BRAIN ANATOMY



Conceptual illustrations constructed from medical literature

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In the brain blood flow stage, the cerebral cortex receives less blood, while blood vessels within the brain direct a preferential amount of the blood to the deep structures. When this occurs, the cerebral cortex, even though receiving less blood, can remain uninjured because its metabolic demands drop so that its energy demands are very low. In order to continue to avoid neurological injury, maintaining blood pressure is essential. The clinical evidence of a maintained blood pressure is the fetal heart rate. Maintaining a base line heart rate within the normal range of 120 to 160 beats per minute indicates that blood pressure is being maintained. When the heart rate drops outside the normal range, it means that blood pressure also drops, resulting in less perfusion to the brain and, if allowed to continue, the risk of permanent neurological injury. A drop in the heart rate would be caused by ischemia to the heart. The heart is first affected by ischemia, followed by a drop in heart rate and blood pressure which then leads to brain ischemia. There is an important relationship between the functioning of the heart and the perfusion of blood to the brain, and therefore an important connection between the pumping of the heart and the avoidance of brain injury. If the heart is pumping properly, there is no ischemia to the brain. A normally pumping heart will provide the same volume of blood with each beat. The volume of blood is obviously important for the delivery of oxygen to the brain. There can be, however, a decreased oxygenation within the blood in spite of a normal rate of perfusion of volume.

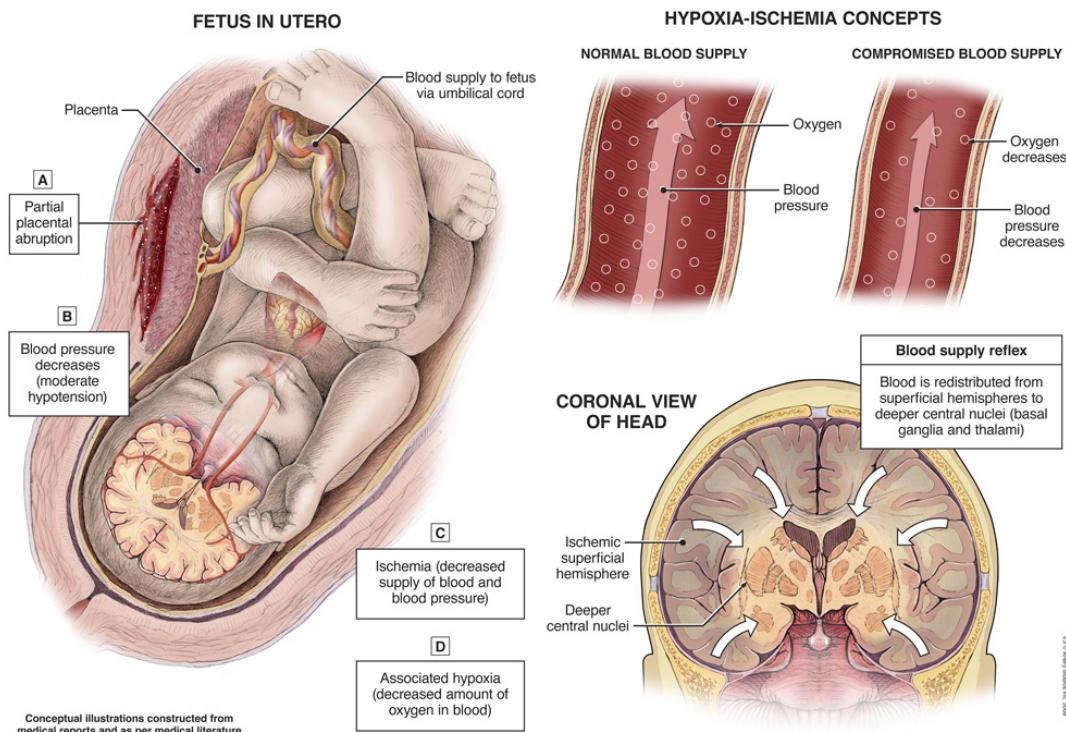
With respect to the relationship between the functioning of the fetal heart and the brain, it is important to recognize that when the fetal heart drops, there is a drop in blood pressure and a corresponding decrease in blood perfusion to the brain. In a case of prolonged partial asphyxia, the area of the brain that will be first to

suffer damage is the cerebral cortex, due to the fact that blood flow to that area of the brain is already reduced. The chance of any brain damage in the cerebral cortex before a fall in heart rate and blood pressure is very unlikely, and if it does occur it would tend to be subtle. The more profound injury to the brain must result from ischemia which occurs with a drop in heart rate and blood pressure.

Both hypoxia (reduced oxygenation) and ischemia (poor perfusion of blood to the organs) is needed to cause damage to the brain. The process starts with hypoxia, for which the fetus will compensate as indicated above. When the point is reached where the acidity in the blood become unbearable, and circulating acids cannot be removed, ischemia will develop and begin the process of increasing neurological harm.

In the absence of profound total asphyxia, discussed below, the areas of the brain most vulnerable to injury from prolonged partial asphyxia are the cerebral cortex and white matter. Sudden profound asphyxia can occur with or without prolonged partial asphyxia. When they occur together there is said to be a mixed pattern of injury, which will have different consequences for the areas of the brain that become damaged.

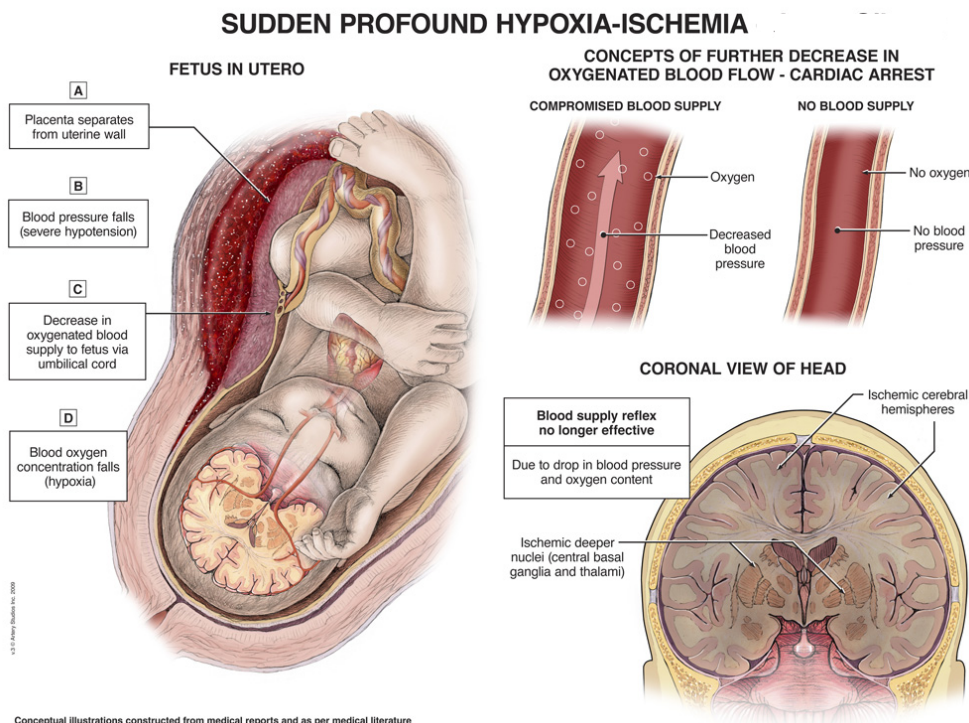
PROLONGED PARTIAL HYPOXIA-ISCHEMIA



Acute total asphyxia or near total asphyxia is a complete or near complete interruption in the supply of oxygen. This total lack of oxygen will, as one would assume, be tolerated for a relatively brief period of time before there is

permanent neurological damage and, if unabated, death. With total profound asphyxia the heart rate drops dramatically within a minute or two of the onset of the profound interruption in blood supply and oxygen. The fetal heart rate, in the presence of a total profound asphyxia will drop within minutes to a level below normal, called a bradycardia. The bradycardia associated with a profound total asphyxia or near total asphyxia is severe.

It is expected that only extreme intrauterine events (like a placental abruption, uterine rupture or a problem with the umbilical cord) will result in a sudden profound asphyxia. In these circumstances, the brain will stop receiving oxygen and ischemia will result. Commonly, the areas of the brain affected by an acute total asphyxia tend to be the deeper structures of the brain, the basal ganglia and the thalami. The cerebral cortex may be spared damage in the setting of an acute total asphyxia without a prolonged partial asphyxia. Having said that, an acute total asphyxia can result in damage to both the deep structures (the basal ganglia and thalami) and the cerebral cortex.



With a fetus exposed to a prior period of prolonged partial asphyxia, however, the capacity to withstand a subsequent profound total asphyxia is compromised. In other words, a fetus with a prior period of hypoxia due to prolonged partial asphyxia will have already become somewhat acidotic, with a low pH, which means that it will take less time during a profound total asphyxia to reach the critical level of acidosis where things start to fail catastrophically.

The magnitude of the insult is maximal with profound total asphyxia. When the fetus has already been subject to prolonged partial asphyxia, the ability to

withstand a period of total profound asphyxia is reduced. This is the “mixed pattern”.

Where the fetus is exposed to a period of prolonged partial asphyxia followed by a period of profound total asphyxia the outcome tends to be worse than those exposed to either a profound partial asphyxia or a profound total asphyxia.

The longer the prolonged partial asphyxia, the greater the risk of injury from a subsequent profound total asphyxia.

Neuroradiology involves imaging of the central nervous system, including the brain and its coverings, through, among other things, MRI, CT scan and ultrasound. Neuroradiology can be used to pinpoint the timing of an injury to the brain suffered in the uterus. Imaging, by itself, can pinpoint the timing to within days. When coupled with available clinical information, however, neuroimaging can be used to pinpoint the onset of neurological injury far more precisely.

Intrapartum asphyxia resulting in neurological damage will cause edema, or swelling of the brain. Generally the swelling of the brain will not occur until at least 24 hours into the neonatal period. The presence of edema at the appropriate time will suggest the occurrence of an injury within a number of hours immediately antepartum. Pinpointing the exact timing of the injury involves recognition of clinical evidence in the intrapartum period that is compatible with the nature of the radiological changes seen on a neuroimaging. Commonly the clinical evidence that corresponds to the changes on neuroimaging would be periodic changes in the fetal heart rate pattern that are known to correspond with impaired oxygenation. In particular, evidence of a drop in blood pressure (hypotension) is required to establish ischemia to the brain. Ischemia to the brain only occurs when there is a drop in the heart rate with the corresponding hypotension. Further, neuroimaging which demonstrates damage exclusively to the cerebral cortex will suggest a prolonged partial asphyxia. Neuroimaging which demonstrates damage exclusively to the deeper structures of the brain will suggest a profound total asphyxia or a near total asphyxia. Neuroimaging which demonstrates damage to the outer structures and the deeper structures of the brain may indicate a combined pattern, although a severe total or near total asphyxia can damage the inner and the outer areas of the brain.

CORD BLOOD GASES

The body needs oxygen to burn fuel, like fire. The less oxygen the less available fuel that is burned. If the supply of oxygen is only marginally reduced, the fire is still capable of burning most of the fuel. As the oxygen becomes increasingly reduced, gradually less fuel gets used up and less energy is generated. It is all a matter of degree. When the body has a shortage of oxygen, its ability to burn fuel and create energy becomes gradually impaired. The fuel that the body burns is generally glucose. When the ability to burn glucose is impaired there is a build up of acids in the body resulting in a metabolic acidosis. It is termed

“metabolic” because the ability of the body to process or metabolize fuel is reduced.

With impaired oxygenation acids build up in the body and can be measured using the pH. The opposite of acid is a base or alkali. The pH is a measure of acidity. When the acidity goes up, the pH goes down. A neutral pH level is 7.4, though there is a range of normal. As the fetus becomes increasingly acidotic, the pH level of its blood and tissues drops. When the body is unable to neutralize the acid, the metabolic acidosis develops. The degree of the metabolic acidosis (also referred to as an oxygen debt) is measured by the “based deficit”. The based deficit (BD) measures the oxygen debt of the fetus. As the BD rises, the oxygen debt rises. BD is measured in units called millimoles per litre (mmol/L). The BD of a fetus before labour is generally considered to be two mmol/L. A BD approaching 30 mmol/L is considered fatal.

The BD is measured from blood taken from the umbilical artery. The blood in that vessel best represents fetal status at the time of birth as blood flows from the fetus to the placenta through the umbilical artery. The blood is then measured for “gases”. The measurements include BD as well as partial pressure of oxygen and partial pressure of carbon dioxide.

With impaired oxygenation, the fetus will experience a rising base deficit. The fact that the BD is rising, however, does not mean that the fetus has suffered irreversible brain injury. In fact, the fetus has a number of mechanisms that allow it to cope with impaired oxygenation without suffering permanent neurological harm. To understand the risk of neurological injury with a rising BD, reference should be made to a 1997 article by Dr. Low, called “Threshold of Metabolic Acidosis Associated with Newborn Complications”.⁴

As an illustration of the levels of BD that are associated with brain injury, please see Table 1 below.⁵ In the table, the categories of central nervous system damage are described as none, meaning no neurological injury at all; minor, meaning only irritability and jitteriness; moderate, meaning profound lethargy or abnormal tone; and, severe, meaning coma or abnormal tone with seizures.

⁴ As indicated in the Low article, fetal asphyxia must be of a particular degree and duration before it will cause brain damage. While fetal asphyxia may be represented by a rising base deficit, if the extent and duration of the asphyxia does not reach a certain threshold, the fetus will not suffer permanent brain injury. In other words, a progressing acidosis does not cause brain damage unless sufficiently severe.

⁵ Taken from Low article, “Threshold of Metabolic Acidosis Associated with Newborn Complications”, American Journal of Obstetrics and Gynecology, December 1997.

Table II. Newborn complications

	A. Umbilical artery base deficit			Significance	B. Umbilical artery base deficit >16 mmol/L (n = 59)
	4-8 mmol/L (n = 58)	8-12 mmol/L (n = 58)	12-16 mmol/L (n = 58)		
Central nervous system					
None	55	48	42		23
Minor	1	10	11		12
Moderate	2	0	4		17
Severe	0	0	1	<i>p</i> = 0.01	7
Respiratory					
None	56	44	28		32
Minor	2	13	21		19
Moderate	0	3	6		5
Severe	0	0	3	<i>p</i> = 0.001	3
Cardiovascular					
None	55	44	47		18
Minor	3	13	10		23
Moderate	0	0	0		9
Severe	0	1	1	NS	9
Renal					
None	58	57	55		48
Minor	0	0	1		3
Moderate	0	1	0		3
Severe	0	0	2	NS	5

A, Complications in neonates of three groups with umbilical artery base deficit of 4 to 8 mmol/L, 8 to 12 mmol/L, and 12 to 16 mmol/L. B, Complications in previously described newborns with umbilical artery base deficit >16 mmol/L for comparison (modified from Am J Obstet Gynecol 1994;170:1081-7). NS, Not significant.

The data in Table 1 indicates that newborns with a BD between 12 and 16 mmol/L had only a 1.7% chance of a severe neurological injury. It also means that those same newborns had a 98.3% chance of not suffering a severe neurological injury. Out of newborns with a base deficit of between 12 and 16 mmol/L, 91.4% had no neurological injury or only minor injury.

This table illustrates the fact that even with a rising BD, most newborns do well. It also illustrates that the longer the fetus is left in an intrauterine environment without adequate oxygenation, the higher the BD value goes and the greater the likelihood of permanent neurological injury. With respect to the essential criteria for proving intrapartum asphyxia as established by organizations like the American College of Obstetricians and Gynecologists and the Society of Obstetricians and Gynecologists of Canada, it is generally considered that a base deficit value of greater than 12 mmol/L in umbilical cord artery blood is necessary to prove intrapartum asphyxia.

It is important to note that the BD alone is not determinative of whether a newborn will suffer a neurological injury. One must also consider the clinical situation before the baby was born and any signs of fetal compromise. Babies have been born with BD values of 26 and still suffered no permanent neurological injury. It is necessary to look for evidence of decompensation from the fetal heart tracing to correlate a BD with a clinical circumstance that suggests an insult becoming an injury.

The BD is also of value in timing the insult converting to injury. While there are a number of approaches that can assist in timing, extrapolating backwards from the umbilical cord artery BD is one useful tool. Since the 1960's, scientists have

studied acidosis, particularly in studies involving rhesus monkeys and sheep. There are also a handful of human studies. The human data, however, is sparse given the obvious ethical considerations in studying the impact of longer periods of asphyxia on the human fetus.

It is important to recognize at the outset that different fetuses have different capacities to withstand an increasing acidosis and an increasing BD. Undoubtedly an increasing BD exposes the fetus to greater risk of neurological injury, but fetuses with high BD levels can avoid permanent neurological injury entirely. As a result, information about BD must be looked at in the entire clinical context when attempting to time an asphyxial injury.

The rate of change of the BD during a period of acute total asphyxia would be more rapid than the rate of change during a prolonged partial asphyxia. As well, the rate of change in the base deficit will vary depending on the circumstances within the intrauterine environment and the capacity of the fetus to compensate for impaired oxygenation. The more severe the condition affecting oxygenation the more rapid their rise in the rate of change of BD. The most severe condition that would result in the quickest rise in BD would be that of acute total asphyxia.

There is virtually no literature relating to human fetuses that establish a clear rate of rise of BD. A study which is not aimed at specifically reviewing the rate of rise of BD, but which can be instructive and the potential rate of rise of BD is a study done in 1997 by Low called "Intrapartum Fetal Asphyxia: Clinical Characteristics, Diagnosis and Significance in Relation to Pattern of Development".⁶

The 1977 article by Low studied groups of fetuses demonstrating acidosis over three periods of time. The three groups including fetuses who did not start to show acidosis until two hours or less before birth; fetuses that did not start to show acidosis until one hour or less before birth; and, fetuses in the "terminal asphyxia group, who demonstrated no acidosis more than 30 minutes before birth, but had a developing acidosis in the last 30 minutes. The terminal asphyxia group would be the group most closely associated with a total profound asphyxia, although there may well have been some fetuses exposed only to prolonged partial asphyxia within that group. The study demonstrated that the rate of change of BD for the terminal asphyxia was one mmol/L per minute per average. The fetuses exposed to a less severe asphyxial episode had significantly slower rates of change of BD.

NEONATAL HAEMATOLOGY

There has been some suggestion in literature that analysis of the neonate's blood in the immediate postpartum period can assist in the timing of asphyxia. It is important to recognize that the literature on haematology is perhaps helpful in

⁶ American Journal of Obstetrics and Gynecology, Volume 129, No. 8.

establishing the timing of insult, but not the timing of injury. Even with regard to insult, the use of haematology and timing would be very limited.

In terms of haematology, the primary measures thought to assist in the timing of asphyxia are the measures of lymphocyte and nucleated red blood cell (NRBC). Great care must be taken in using haematology to time asphyxia. Having regard to all of the tools available to time asphyxia, haematology is undoubtedly the weakest. The association between haematological results and adverse outcome is loose. The best that can be said about using lymphocyte count and NRBC measures is that they may well be consistent with other clinical evidence of timing. However, they are in and of themselves, conclusive of nothing. Moreover, there is good reason in most cases to believe that hemological results may not affect any conclusions about timing. Put another way, the haematology results are definite evidence of nothing in particular but may well be consistent with other conclusions drawn from other evidence.

ANTEPARTUM AND OTHER CAUSES

In determining the cause of neonatal neurological injury it is important to eliminate events outside the intrapartum period as contributing factors. Frequently the defence in these cases will argue that intrapartum asphyxia is an unlikely cause for the baby's injury because the vast majority of cases of cerebral palsy are caused by events other than intrapartum asphyxia. While the data may establish this, it is entirely irrelevant to whether or not a particular case is due to intrapartum asphyxia. It is a medical fact that intrapartum asphyxia can cause the type of brain damage (hypoxic ischemic encephalopathy) that causes cerebral palsy due to asphyxia. The issue in each individual case is whether there is clinical evidence that the injury was caused by intrapartum events as opposed to some other remote cause.

Care should also be taken with respect to preterm babies (born at less than 36 weeks gestation). Morbidity and mortality is higher for preterm babies than for term babies and preterm babies are subject to injury to the brain that may differ from brain injury suffered by term babies. Intraventricular haemorrhage, as an example, is a condition that can arise by virtue of prematurity but can also arise by virtue of asphyxia.

The neonatology expert must be careful to consider all possible antepartum causes for neonatal brain injury and the evidence that either supports or refutes those as a cause for the injury in the particular case. Good maternal and fetal health antenatally will be important. The presence of fetal movement antepartum as well as reaching appropriate growth parameters would also be important factors in ruling out antepartum causes for brain injury.

There are a variety of antenatal techniques to assess fetal well being that are routinely done during pregnancy. These will include ultrasound examinations and biophysical profiles. Experts retained for advice on these cases must have

access to this documentation to address the possibility of other causes for injury. Finally, placental pathology postnatally may also provide revealing evidence for the potential causes of neonatal neurological injury.

Other factors to consider in the diagnosis of intrapartum fetal asphyxia include genetic abnormalities, congenital abnormalities, infections, teratogenic (developmental) factors, metabolic disorders and endocrine disorders.

Factors that are thought to suggest neurological injury due to intrapartum asphyxia have been described by various medical associations including the Society of Obstetricians and Gynaecologists of Canada and the American College of Obstetricians and Gynaecologists. Some of the key factors that will affect the determination of causation include the following:

1. Apgar Scores
2. Neonatal Neurological Sequelae
3. Multi Organ System Dysfunction in the Early Neonatal Period
4. Umbilical Cord Artery pH Less Than 7.0
5. Umbilical Cord Artery Base Deficit Greater Than 12 mmol/L

Care must be taken to rely too heavily on these indicators. There are pitfalls associated with each.

It is generally thought that proving that intrapartum hypoxia was severe enough to cause cerebral palsy has occurred requires the proof of certain criteria. The criteria involves four essential components:

- (a) Evidence of metabolic acidosis supported by a cord arterial gas of greater than 12;
- (b) For infants of 34 weeks gestation or more, early onset moderate neonatal encephalopathy;
- (c) Cerebral palsy of the spastic quadriplegic type; and
- (d) Exclusion of other causes.

There are also criteria, while not essential, that suggest an intrapartum timing to the cerebral palsy, which are:

- (a) A sentinel hypoxic event occurring immediately before or during labour;
- (b) A sudden rapid and sustained deterioration of the fetal heart rate;
- (c) Apgar scores of 0-6 longer than 5 minutes;
- (d) Early evidence of multi-system involvement;
- (e) Early imaging evidence of acute cerebral abnormality.

CONCLUSION

Causation in compromised baby cases is usually very complex. There is plenty of room for disagreement amongst the experts. All of the antenatal information must be carefully assessed along with the intrapartum clinical picture and the clinical situation in the neonatal period. It is much like assembling the pieces of a puzzle. Inevitably, some pieces of the puzzle will not fit, but that will not necessarily mean that all is lost. Even essential criteria for proving intrapartum asphyxia, as set out by ACOG and SOGC, may not be essential on the particulars facts of a given case. Lawyers acting for the Plaintiff need a firm understanding of the science in order to ensure the causation experts have properly evaluated the case. Experts often get part of it wrong and a careful review of the experts' findings in a face to face meeting prior to the delivery of a report is **the** essential criterion for good lawyering in these cases.