



# HOW TO HANDLE CAUSATION IN BIRTH ASPHYXIA CASES

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*Published in Trial News, the monthly newspaper of the Washington State Trial Lawyers' Association May, 2002*

In evaluating possible birth asphyxia cases, one must not overly focus on the events during the perinatal period and assume that if there was a delay in reasonably responding to signs of hypoxia and if the baby was later found to have cerebral palsy, then the two are likely related.

On the other hand, one should not dismiss a possible claim too readily for want of causation just because the newborn's clinical presentation does not mirror the published hallmarks of hypoxic encephalopathy.

The purpose of this article is to explore, in a general way, the basic questions pertaining to causation in birth asphyxia cases: (1) when did the injury likely occur; and (2) if it occurred at about the time of birth, was there a sufficient hypoxic event during labor and delivery to cause that injury? To address these questions, I will generally discuss the relationship between birth asphyxia and cerebral palsy, and the frequently cited criteria for evaluating whether there was sufficient birth asphyxia to cause brain injury at birth.

## TIMING OF DAMAGE

It is estimated that the incidence of cerebral palsy is somewhere between 1 and 2 per 1000 live births(American

College of Obstetricians and Gynecologists, 1992). The damage may range from slight to severe. Although intrapartum asphyxia is a significant cause of cerebral palsy, the vast majority of infants with cerebral palsy have no evidence of intrapartum asphyxia, and most infants who suffer intrapartum asphyxia develop no signs of permanent brain damage. More common risk factors are genetic abnormalities, congenital malformations, birth weight less than 2000 grams, gestational age less than 32 weeks, and infection.

It is estimated that somewhere between 5 and 15% of cerebral palsy occurs near the time of birth; however, a substantial portion of these cases occur because of prematurity. In fact, largely because of the increase in survival of low-birthweight babies, the rate of cerebral palsy has not materially declined over the past 50 years despite the advent of fetal heart monitoring, the heightened concern over perinatal asphyxia, and the delivery of approximately one out of four babies by Cesarean section.

Different etiologies result in differing forms of cerebral palsy. In addition, the same etiology at different gestational periods can result in differing forms of cerebral palsy because of changes which occur during the development of the fetal brain, and the relative susceptibility of the different structures to asphyxia. For example, asphyxia

during the late second and early third trimester may well result in injury to the transitional germinal matrix which lies along the lining of the cerebral ventricles and is composed of primitive cells. These cells ultimately contribute to the formation of the basal ganglia and other neuronal structures of the brain. Depending upon the developmental stage at the time of the prenatal asphyxial event, the fetus may also suffer direct injury to the basal ganglia, and to the periventricular white matter. These structures are particularly susceptible because of their high metabolic demand for oxygen.

An asphyxial injury during this stage of development may well result in spastic diplegia. Children with that affliction present with high tone in their lower extremities, with or without other significant disabilities. Depending upon the degree of hypoxia, the brain damage can extend outward through the cerebrum to the cortex, and therefore manifest itself as well in cortical disabilities such as mental retardation, epilepsy and/or blindness.

Although children who have suffered severe hypoxia during the prenatal period may present with smaller than normal heads at birth, that is not always the case. In fact, the child's head can be normal size. An MRI, however, would likely show increased ventricle size accompanied by loss of periventricular white matter or areas of necrosis. Such

child's head size can be expected to grow at a slower than normal rate, and may ultimately result in microcephaly.

Indications that the fetus may have been subjected to prenatal asphyxia can often be found through placental pathology. The placenta should be evaluated for signs of degeneration, maternal infection, and inadequate functional capacity. Also of particular interest is the location of cord insertion, number of vessels, and length of cord.

When the incident of severe hypoxia does not occur until after 35 weeks a different pattern of injury occurs to the brain. This is because the germinal matrix is no longer present, and the basal ganglia and periventricular white matter have become more resistant to hypoxic injury by that time. Consequently, severe hypoxia at term is more likely to result in diffuse cortical damage. Such asphyxia can cause disproportionate damage to those areas of the cortex more remote from the main blood supply. This is commonly referred to as a watershed distribution and is analogized to the affect of turning down the spigot on one's sprinkling system. On the other hand, a complete loss of blood supply to the fetal brain at term, whether from severe bradycardia or heart stoppage, can cause discrete injury to the deep brain including the basal ganglia, thalami and putamen. Such injury can result from as little as 10 minutes of anoxia such as might occur from a cord prolapse or ruptured placenta.

An asphyxial injury at term may well result in spastic quadriplegia, truncal hypotonia and athetosis. Generally, the more severe the asphyxial event, the more serious the child's

physical disabilities, and the more likely the child is to have serious mental retardation. There is some dispute, however, as to whether asphyxia can cause mental retardation or learning disabilities in the absence of physical disability. Nevertheless, there is a wide range of outcomes from similar degrees of apparent asphyxia at birth, which suggests that certain children are considerably better able to withstand the affects of asphyxia than are others.

### *How Serious Was the Birth Asphyxia? Why the Usual Criteria May Not Apply*

Generally, the infant who suffered acute brain injury at birth will be severely depressed and difficult to resuscitate while those whose injuries are subacute tend to recover more quickly. Such severe depression will usually be in stark contrast to a fetus which was active prior to birth by report of the mother, and as evidenced by a normally twisted cord. The difficulty comes when one attempts to define objective criteria to judge whether or not the degree of asphyxia at birth was sufficient to cause the child's brain damage.

The most commonly used yardstick for determining whether the extent of birth asphyxia was sufficient to cause significant brain damage is that from the American College of Obstetrics and Gynecology (ACOG) Technical Bulletin 163. That document states:

*"In assessing a possible relationship between perinatal asphyxia and neurologic deficit in an individual patient, all of the following criteria must be present before a plausible link can be made:*

*\* profound umbilical artery metabolic or mixed acidemia (pH < 7.00). (Normal pH for a newborn is 7.25 to 7.35. Acidemia can be due to a buildup in the blood stream of carbonic acid formed by oxidative metabolism of carbon dioxide which is called respiratory acidemia, or by a build up organic acid from anaerobic metabolism which is called metabolic acidemia. Where both are present, it is called mixed respiratory-metabolic acidemia.)*

*\* Persistence of an Apgar score of 0-3 for longer than 5 minutes. (The Apgar score is named after Dr. Virginia Apgar who developed the scoring system in 1952. There are five signs which are evaluated with either 0, 1, or 2 points assigned to each one depending upon the findings at certain intervals, commonly 1, 5 and 10 minutes after birth. The five signs are heart rate, respiratory effort, muscle tone, reflex irritability, and color. A perfect score would be 10, but that is rarely assigned.)*

*\* Neonatal neurologic sequelae, eg. seizures, coma, hypotonia*

*\* Multi-organ system dysfunction, eg. Cardiovascular, gastrointestinal, hematologic, pulmonary, or renal"*

Since these guidelines were published, however, numerous studies have found that asphyxial injury can occur without the presence of all four of these criteria. In one study reported in 2 Prenatal Neonatal Medicine 286-93 (1997), the medical records of 16 severely neurologically impaired term infants were reviewed. All had suffered acute intrapartum asphyxia from a documented event such as a cord prolapse or uterine rupture, and no other explanation for their brain

injuries. Only one met all four criteria, and only four met three of the criteria. The study also found that: five had a pH <7.00; five had a 5-minute Apgar of 5 or less; 14 had seizures within the first 24 hours; and half had no multi-organ dysfunction.

In a separate study, the same group of investigators reported in 9 *Journal of Maternal-Fetal Medicine* 101-06 (1999) that of 47 neonates with a documented acute asphyxial injury resulting in permanent brain injury, 10 met all 4 criteria, 14 met 3, 14 met 2, 8 met 1, and 1 met none of the criteria.

The same study also tended to dispel one of the previously commonly held assumptions relating to the timing of seizures; namely that an early onset of seizures after birth was an indication of an earlier asphyxial event. The assumption was that seizures from birth asphyxia resulted from the build up of brain edema, which generally takes a minimum of 6 to 8 hours to fully develop. If that were always the case, then seizure activity within a couple of hours of birth would indicate that the prenatal asphyxial event must have taken place hours before the onset of the second stage of labor when most cord accidents occur and when fetal heart rate tracings are more apt to show signs of fetal hypoxia.

While it is true that the level of edema necessary to effect seizure activity, by itself, can take that long to develop, the more recent view is that the most serious asphyxial injuries result in seizures within the first couple of hours. In such cases, it is thought that the hypoxic injury is sufficient to directly cause disruption of brain function rather than just leading to the build up of edema which in turn

precipitates seizure activity. In fact, the study showed that the median time for seizures following documentable severe asphyxial events was 3.5 hours. Some infants demonstrated seizure activity within 2 hours of the onset of the asphyxial event. This general conclusion regarding the timing of seizures was also reported in 37 *Clinical Pediatrics* 673-76 (1998). In the study of 25 infants who suffered seizures following acute asphyxia, the mean time between the event and the onset of seizures was 3.1 hours, after excluding one infant that did not seize for 90 hours.

In addition, these studies demonstrated that many infants can develop brain injury from asphyxia without coincident acidemia reflected in a low pH value. Such conclusion is also supported by a study reported in 93 *British Journal of Obstetrics and Gynecology*. It concluded that the highest frequency of neurologically damaged infants was found in the group with low Apgar scores but with less abnormal pH values. Similar conclusions were reported in 161 *American Journal of Obstetrics and Gynecology* 213-20 (1989). In this study, infants with acidemia but with very low Apgar scores fared better than those with a more normal blood pH and very low Apgar scores. More normal pH under those circumstances might indicate some inability on the part of the fetus to continue to exchange gasses between the blood and the cells of the body, including those of the brain. This may be indicative of a lack of effective circulation. In addition, it is postulated that the presence of acidosis with effective circulation may increase relative blood flow to the brain, and decrease brain oxygen demands.

It is of course one thing to generally understand the medicine relating to causation and quite another matter to effectively present that evidence at trial. Not only is it important to find experts who are well credentialed, but it is at least as important to find ones who will also take the time to thoroughly prepare and assist you to communicate a consistent, simpler explanation of the medicine to the jury. Such expert witnesses may well include a neuro-radiologist, a placental pathologist, a neonatologist, and a pediatric neurologist. The more experts one calls, the more chance there is for inconsistent testimony. To avoid or to at least minimize those pitfalls, it is necessary for the attorney to first understand the complex nature of the medicine and of the medical facts of the case before preparing the expert witnesses to present their testimony in a manner which will be understood by the jury.