[Cite as *Kuhbanani v. Ohio State Univ. Med. Ctr.*, 2017-Ohio-4457.]

NEWSHA KUHBANANI, et al. Plaintiffs v. THE OHIO STATE UNIVERSITY MEDICAL CENTER, et al. Defendants	Case No. 2011-08547 Magistrate Anderson M. Renick DECISION OF THE MAGISTRATE

{¶1} Plaintiffs, Shahram Gharibshahi, M.D. and his wife, Newsha Kuhbanani, who are the parents of plaintiff Sooshyance Gharibshahi, a minor, brought this action alleging negligence by the medical staff at defendant The Ohio State University Medical Center (OSUMC). The case proceeded to trial on the issues of liability and damages.

{¶2} Plaintiffs' claims arise from the delivery and care provided to Sooshyance who was born on May 17, 2008 at OSUMC. During the afternoon of May 16, 2008, Newsha was admitted to OSUMC with a full-term, normal weight baby. Newsha's obstetrician, Sarah Artman, M.D., decided to induce labor and initiate fetal heart monitoring, which provided readings that, by all accounts, were not concerning prior to 1:00 a.m. on May 17, 2008. Dr. Artman was assisted by several obstetrical nurses who, among a variety of duties, observed and interpreted the fetal heart monitor. The fetal heart monitoring strips initially showed normal, moderate variability, meaning fluctuations in amplitude and frequency from the heart rate "baseline," which is an indication that the baby was well-oxygenated. However, between 1:10 and 1:20 a.m., fetal heart rate "decelerations" were apparent, an indication of a transient interruption of the fetal oxygen supply. When meconium (fecal matter) was detected in the amniotic fluid a pediatric resuscitation team (peds team) was summoned to the delivery room. The peds team consisted of Christine Dugan (nka Adams), M.D., a third-year resident;

Edward Nehus, M.D., a second-year resident; and Sheria Spain (nka Wilson) M.D., an intern. The peds team prepared in the event that the baby required resuscitation related to either late decelerations or an effort to prevent development of meconium aspiration syndrome as a result of inhaling the fluid. By 1:30 a.m., delivery was in progress and Dr. Artman had become concerned by a change in heart rate variability to minimal or absent variability, followed by recovery and a rise in heart rate. Thereafter, the fetal heart rate continued to deteriorate and by 1:40 a.m. it was low with prolonged decelerations and little variability. At 1:43 a.m., Dr. Artman removed the scalp electrode that detected fetal heart tracings so that she could begin use of a vacuum system to extract the baby. After the scalp electrode was removed, an external ultrasound device was used periodically to detect a minimal fetal heart rate in the minutes before delivery at 1:52 a.m.

{¶3} When Sooshyance was born, he was not breathing and his heart rate was either dangerously low or non-existent. Sooshyance's condition was assessed using Apgar scores, a numerical rating system that reflects an assessment of a newborn's condition of breathing, tone, reflexes, movement, and color. The Apgar scores at 1, 5, and 10 minutes after birth were 0, 0, and 1, respectively. The members of the peds team followed an established protocol, the Neonatal Resuscitation Program (NRP), by first suctioning the airway to remove meconium and then administering oxygen with a "bag-mask" device to create positive pressure ventilation (PPV). The peds team continued use of the bag-mask and applied chest compressions for approximately three minutes. After Sooshyance did not respond, the residents performed an endotracheal intubation for ventilation. At 1:56 a.m., four minutes after delivery, Daniel Malleske, M.D., who was in his second year of a neonatology fellowship and had experience in resuscitating severely depressed neonates, arrived to assist with resuscitation. Dr. Malleske determined that Sooshyance was not adequately ventilated and he inserted a second endotracheal tube and then administered two doses of epinephrine (at 1:58 and

1:59 a.m.) to stimulate Sooshyance's heart. Approximately three minutes later, Sooshyance showed the first signs of a returning heart rate.

{¶4} Sooshyance has been diagnosed with a significant and permanent brain injury caused by hypoxic ischemic encephalopathy (HIE) which, according to plaintiffs' pediatric neurology expert, resulted in a diagnosis of spastic quadriparesis, a type of cerebral palsy which affects all four limbs and fine motor activity. As a result, Sooshyance is fed primarily through a gastric tube in his abdomen, although he can eat some food by mouth. Sooshyance also has a significant cognitive abnormality that impedes learning, seizure disorder which is partially controlled by medication, and a significant problem with speech and language.

{¶5} Plaintiffs contend that Sooshyance's resuscitation was delayed and that the delay was a proximate cause of his HIE. According to plaintiffs, the standard of care required an experienced resuscitator to be present prior to Sooshyance's delivery and the resuscitation team breached the standard of care by failing to timely notify Dr. Malleske of Sooshyance's grave situation. Specifically, plaintiffs maintain that either the nurses in the delivery room failed to notify the pediatric residents of Sooshyance's severely depressed heart rate, or if a nurse did communicate that there was a reasonable expectation that the baby would be born in a severely depressed state, that the peds team negligently failed to timely notify Dr. Malleske of the situation. According to plaintiffs, Sooshyance's injuries would not have occurred if Dr. Malleske had been present at the beginning of the delivery.

{¶6} "In order to establish medical malpractice, it must be shown by a preponderance of evidence that the injury complained of was caused by the doing of some particular thing or things that a physician or surgeon of ordinary skill, care and diligence would not have done under like or similar conditions or circumstances, or by the failure or omission to do some particular thing or things that such a physician or surgeon would have done under like or similar conditions and circumstances, and that

the injury complained of was the direct and proximate result of such doing or failing to do some one or more of such particular things." *Bruni v. Tatsumi,* 46 Ohio St.2d 127 (1976), paragraph one of the syllabus.

{¶7} The same standard applies equally to claims that a nurse negligently caused injury to a patient. *Ramage v. Central Ohio Emergency Serv., Inc.*, 64 Ohio St. 3d 97, (1992). "Because nurses are persons of superior knowledge and skill, nurses must employ that degree of care and skill that a nurse practitioner of ordinary care, skill and diligence should employ in like circumstances. Whether a nurse has satisfied or breached the duty of care owed to the patient is determined by the applicable standard of conduct, which is proved by expert testimony." *Berdyck v. Shinde*, 66 Ohio St.3d 573, syllabus (1993).

{¶8} Plaintiffs' expert, Joseph Ouzounian M.D., is board certified in obstetrics and gynecology (OBGYN) and maternal-fetal medicine, a subspecialty related to the care of high-risk pregnancies. Dr. Ouzounian testified that the fetal heart monitor tracings showed nothing remarkable prior to 1:00 a.m. Dr. Ouzounian estimated that Sooshyance's initial fetal heart rate baseline was approximately 145 beats per minute, which he characterized as normal, and he testified that moderate variability was a favorable finding. Dr. Ouzounian related that between 1:00 and 1:10 a.m. fetal heart rate decelerations became apparent, which could have been caused by either compression of the umbilical cord or contractions of the mother's uterus. Dr. Ouzounian testified that Soohyance's fetal reserves were "fine" at 1:20 a.m. as indicated by his recovery from the decelerations with moderate variability. At 1:30 a.m., the delivery was in progress and Dr. Ouzounian noted a change in variability to minimal, with at least a short period of absent variability with a deceleration, which he testified was very common in the minutes before delivery. However, Dr. Ouzounian opined that the deceleration at this point, down to the 60s, was "concerning" and that the baby would likely "need some help" at delivery. Dr. Ouzounian opined that by 1:40 a.m. a

pediatrician or nurse would have a reasonable expectation that the baby would have respiratory distress and require intubation. At 1:43 a.m., the scalp electrode was removed to effectuate delivery. Dr. Ouzounian explained that thereafter, the fetal heart rate was detected in the 60s, as recorded in nursing notes for the time just prior to delivery. Dr. Ouzounian opined that the fetal heart rate tracings indicate that "with proper resuscitation, the baby would be fine" and that "the majority of these kids do great." Dr. Ouzounian did not have any criticisms of Dr. Artman.

{¶9} Dr. Ouzounian related that HIE is brain damage due to a hypoxic event. Dr. Ouzounian explained that acidemia, a buildup of acid in the blood, usually precedes acidosis, which involves acid buildup in the tissue. Dr. Ouzounian testified that an interruption in oxygen flow causes respiratory acidosis in the blood, the first step in a continuum which is followed by respiratory acidemia, mixed respiratory/metabolic acidosis, then metabolic acidemia, ultimately resulting in brain injury. Dr. Ouzounian explained that if an interruption of oxygenation and the development of respiratory acidosis continues, acid will begin to build up at the tissue level, leading to respiratory acidemia. Eventually, a mixed respiratory and metabolic acidosis develops as the fetus begins to use its "buffer bases" (bicarbonate) to counter the acidemia. When the buffer bases begin to become depleted, acid will build up in the blood as metabolic acidemia and oxygen deprivation eventually cause brain damage. Dr. Ouzounian opined that metabolic acidemia is a prerequisite for brain damage due to intrapartum hypoxia. According to Dr. Ouzounian, respiratory acidosis is generally not harmful to the fetus.

{¶10} Soon after the delivery, umbilical cord gases for both the umbilical artery and umbilical vein were measured by OSUMC staff from blood that was drawn at the time of birth. Dr. Ouzounian related that the fetus gets oxygen from the mother and from the placenta. The umbilical artery contains blood coming from the baby to the placenta and the vein contains blood from the placenta to baby. Dr. Ouzounian testified that, conceptually, blood gases from the umbilical artery reflect the fetal status.

Dr. Ouzounian explained that the blood gases that were measured included pH, a measure of acidity; PCO2, or carbon dioxide, which reflects respiratory status inasmuch as carbon dioxide increases as breathing decreases; PO2 which reflects oxygenation; and HCO3, or bicarbonate, a measure of a buffer base that can offset the buildup of acid. Dr. Ouzounian testified that normal readings for umbilical artery blood are: pH between 7.25 and 7.35; PCO2, about 50; PO2, about 20; and HCO3 between 20 and 25. According to Dr. Ouzounian, the arterial blood gas readings, which reflect fetal status were within the normal range for PO2 and HCO3, while the PCO2 was very high and pH was very low. Dr. Ouzounian testified that a formula is used to correct the pH reading based upon the high PCO2, which in this case results in a corrected pH of approximately 7.35. According to Dr. Ouzounian, a pH of 7.35 is "basically normal" and indicates respiratory acidosis, or possibly a metabolic component, but does not suggest a brain injury because the lack of metabolic acidemia shows that the baby had fetal reserves. Dr. Ouzounian defined base excess, or base deficit, as a calculated value that demonstrates whether a component of metabolic acidemia exists. Dr. Ouzounian testified that base excess or base deficit builds up when bicarbonate has been depleted and is not available to neutralize acid buildup. Dr. Ouzounian calculated the base deficit as approximately 11. Dr. Ouzounian opined that prior to birth Sooshyance did not sustain any brain injury caused by intrapartum oxygen deprivation based upon Dr. Ouzounian's assessment of Sooshyance's fetal reserves, considering his oxygenation and buffer bases.

{¶11} Dr. Ouzounian testified that he would defer to the opinion of a pediatric neurologist regarding Sooshyance's condition after birth and his ability to respond to the supportive treatment that was provided thereafter. Nevertheless, Dr. Ouzounian was critical of the level of experience of the peds team, characterizing the team members as "rookies" who had never treated a baby in such a depressed condition. According to Dr. Ouzounian, every minute is critical when treating a baby who is born with no

respiration and no heart rate. Dr. Ouzounian testified that according to times noted in the medical record, there was approximately three and one half minutes between the time that Dr. Malleske testified that he would have intubated (an estimate of one and a half minutes after birth) and the time that the baby was effectively ventilated at 1:57 a.m. Dr. Ouzounian explained that there are five categories that are assessed in assigning an Apgar score and he opined that the one-minute Apgar was not significant in determining a prognosis, in part, because it is dependent on the effectiveness of resuscitation efforts.

{¶12} Dr. Ouzounian related that the American College of Obstetricians and Gynecologists (ACOG) is a professional organization that publishes clinical guidelines, including a guideline for determining the existence of HIE during labor due to intrapartum hypoxia. Dr. Ouzounian testified that the ACOG criteria included pH of less than 7 and a base excess greater than 12. Dr. Ouzounian stated that even if all the enumerated ACOG criteria were met, the guidelines also provide that all other possible causes for neonatal brain injury must be excluded and that Sooshyance's cardiac respiratory arrest after delivery was another explanation for HIE. During cross examination, Dr. Ouzounian testified that neonatal resuscitation is outside his area of expertise and that he has not performed that service since his residency. Dr. Ouzounian stated that he did not have any criticism of either the peds team, Dr. Malleske, or the nurses who assisted during and after delivery.

{¶13} Plaintiff's nursing expert, Laura Mahlmeister, RN, PhD., had worked as a floor nurse for over 40 years before retiring to teach as a clinical professor of nursing at the University of California San Francisco School of Nursing. Dr. Mahlmeister also writes, lectures and serves on committees that formulate polices and procedures regarding labor and delivery. Dr. Mahlmeister testified that obstetrical nurses are "the eyes and ears of the doctor." Her review of the medical record showed that there were four experienced obstetrical nurses in the labor and delivery room, Nurses Cox,

Jenkins, Elliot, and Cowles, and one nurse in training. Dr. Mahlmeister testified that she had no criticisms in this case if Nurse Cox notified the residents of the decreased fetal heart tracings as Cox testified in her deposition. Dr. Mahlmeister opined that the standard of care required the nurses to notify the residents of that change in condition. Dr. Mahlmeister explained that ensuring the proper personnel and equipment was ready for resuscitation was a crucial part of the nurses' duty. According to Dr. Mahlmeister, in the last 10 to 15 minutes before delivery, a reasonable obstetrical nurse under the same conditions would have had an expectation of severe birth depression based upon the very low fetal heart rate and rapidly diminishing variability. Dr. Mahlmeister testified that by about 1:40 a.m., a reasonable and prudent nurse would have communicated concerns about the fetal heart rate tracings. According to Dr. Mahlmeister, the peds team would have had to be notified of the depressed fetal heart tones by no later than 1:48 a.m. to allow them to notify Dr. Malleske in time for him to arrive prior to delivery.

{¶14} During cross examination, Dr. Mahlmeister testified that a pattern of decelerations and minimal variability is a characteristic fetal pattern of metabolic acidosis and that the pattern was cause for the nurses to notify the peds team. Dr. Mahlmeister stated that metabolic acidosis is associated with a baby being born with an Apgar score of 0. Dr. Mahlmeister admitted that there is some controversy regarding the predictive value of electronic fetal monitoring. Dr. Mahlmeister acknowledged that lactic acid causes metabolic acidosis which then causes the decrease in heart rate variability. Dr. Mahlmeister testified that the residents knew there were decelerations when they were called, but she was not aware whether the heart rate monitor was audible in the delivery room. Dr. Mahlmeister opined that the only difference between Sooshyance's condition at birth and that of a stillborn baby is the fact that Sooshyance was able to be resuscitated.

{¶15} Edward Karotkin, M.D., plaintiffs' neonatology expert, testified that he has over 40 years of experience and that he is board certified in pediatrics and neonatal-

perinatal medicine. Dr. Karotkin is a coeditor (with defendant's expert, Dr. Goldsmith) of the textbook, *Assisted Ventilation of the Neonate*. Dr. Karotkin testified that for most of his career, he has practiced in a teaching hospital, instructing medical, resident, and fellowship students. Dr. Karotkin related that, the neonatal period refers to birth to 30 days of age; however, premature babies are often treated in the hospital for more than 30 days.

{¶16} Dr. Karotkin discussed the relationship between metabolic acidosis and bicarbonate (HCO3); the level of bicarbonate drops as it is used as a buffer to correct the baby's pH. Dr. Karotkin testified that the cord gases detected by OSUMC showed a bicarb in the normal range, which indicates that the metabolic acidosis was relatively minor based on the PCO2 and pH. According to Dr. Karotkin, there was a mixed metabolic and respiratory acidosis and the metabolic component was "mild to moderate." Dr. Karotkin opined that Sooshyance's cord gases show that he did not have a brain injury prior to delivery and that the asphyxia event was "very recent." Dr. Karotkin testified that the formula for bicarb calculation which defendant relied upon was not relevant because it was taken from an article that was based upon an adult population. According to Dr. Karotkin, adult bicarb physiology is significantly different from that of an infant. Dr. Karotkin stated that he is familiar with the standards published by both ACOG and the American Academy of Pediatrics (AAP). Dr. Karotkin related that according to AAP standards, the base excess calculation that was referenced by counsel for defendant at the time of his deposition (-11.3) falls below the number that would indicate the baby had a brain injury prior to birth (-12). Dr. Karotkin opined that neither the base excess nor any of the blood gases reported by OSUMC support a hypoxic-ischemic injury during labor and that the baby would have responded "very nicely" to appropriate resuscitation. Although Dr. Karotkin acknowledged that the pH and PCO2 values were "not good," he opined that the PO2 and HCO3 numbers were normal and, collectively, the gases were not "even close" to showing a brain injury

during labor and delivery. When asked to assume that the gases had shown a bicarb of 13 and a base excess of -17 to -18 and that brain injury occurred prior to delivery, Dr. Karotkin opined that the baby would have been born "close to dead" and would not have responded to resuscitation.

{¶17} Dr. Karotkin described the Neonatal Resuscitation Program (NRP) as a decision tree that provides guidance on intervention based upon the baby's clinical presentation upon birth. He testified that OSUMC's policy requiring at least one NRP-trained staff member to be present in the delivery room was consistent with NRP guidelines. Dr. Karotkin stated that performing an intubation can be difficult, that there are risks associated with improperly placing the tube, and that intubation provides more effective ventilation than PPV using a bag-mask. Dr. Karotkin opined that a physician with significant experience intubating infants would take less time in deciding to perform an intubation than would a physician with less experience. While he demonstrated the proper technique for both PPV using a bag-mask and intubation, Dr. Karotkin related that epinephrine can be administered through the endotracheal tube.

{¶18} Dr. Karotkin testified that the longer a baby is deprived of oxygen, blood circulation, and cardiac output due to a delay in resuscitation, the longer it will take for the baby to establish a normal respiration pattern, resulting in a greater risk of brain damage. Dr. Karotkin opined that Sooshyance was not effectively ventilated until approximately five minutes after birth and that had Dr. Malleske performed an intubation within one and a half minutes, as he indicated in his deposition, the baby would have responded within an additional one and a half minutes, without the need for epinephrine. Dr. Karotkin further opined that Sooshyance did not have a brain injury prior to delivery based upon the nursing notes which state that he had a heart rate in the 60s both just prior to birth and within the first minute of life. According to Dr. Karotkin, the standard of care required the peds team and the nurses, collectively, to summon Dr. Malleske prior to delivery once they became aware of the depressed fetal heart

tones. Dr. Malleske stated that there is a direct cause-and-effect relationship between the deviation from the standard of care and Sooshyance's brain injury.

{¶19} During cross examination, Dr. Karotkin acknowledged that a failure to resuscitate is not by itself evidence that there has been a violation of the standard of care. Dr. Karotkin opined that there was some degree of hypoxia at birth and that the baby had a period of depressed heart rate before his birth. Dr. Karotkin admitted that under certain circumstances, only several minutes with a heart rate of 60 or below can cause brain injury. Dr. Karotkin acknowledged that Dr. Malleske testified that he would have allowed a resident perform the intubation and Dr. Karotkin was not critical of that decision. Dr. Karotkin agreed that the failure to successfully place the endotracheal tube on the first attempt was not a violation of the standard of care. Dr. Karotkin testified that the sole basis for his opinion that the resuscitation efforts before the intubation were unsuccessful was that no heart rate was detected. Dr. Karotkin related that the heart rate monitor "beeps" in the delivery room and that once the peds team arrived, the nurses and residents would be able to tell that the heart rate was depressed.

{¶20} Plaintiff's pediatric neurology expert, Yitzchak Frank, M.D. testified via deposition that he serves as a clinical professor of pediatrics, neurology, and psychiatry at Mount Sinai Medical Center.[1] He is board certified in both neurodevelopmental disabilities and neurology with a special competence in child neurology. Dr. Frank has been practicing primarily as a pediatric neurologist for 40 years. Dr. Frank related that, up until 2013, he often practiced in the NICU treating children with neonatal issues including HIE.

[1]The video depositions of Drs. Yitzchak Frank, Christine Adams, and Daniel Malleske were played during trial and counsel agreed that the depositions would become part of the record. Accordingly, the depositions have been marked as Court's Exhibits 1, 2, and 3, respectively.

{¶21} Dr. Frank opined that Sooshyance did not have a permanent brain injury at the time of his delivery based upon cord blood gas results which did not show significant metabolic acidosis that is associated with brain damage. Dr. Frank testified that the presence of a heart rate both prior to birth and at the time of birth shows that Sooshyance had metabolic reserves when he was born, another indication that he did not have brain damage. Dr. Frank explained the process that causes metabolic acidosis, the components of cord blood gas test results, the concept of fetal reserves, and the direct connection between the presence of excessive lactic acid and brain damage. According to Dr. Frank, respiratory acidosis is not necessarily indicative of brain damage.

{¶22} Dr. Frank opined that in this case, the measures for metabolic acidosis were not excessively high, in that the pH was low, PCO2 was very high, and bicarbonate was normal, all of which show that the nature of the acidosis was respiratory and that there was no significant brain damage at the time of birth. Dr. Frank described the continuum from respiratory to metabolic acidosis and he opined that brain damage occurred after birth when Sooshyance's heart stopped, resulting in a lack of perfusion and cerebral blood flow, causing HIE. Dr. Frank further opined that if Sooshyance already had brain damage at birth, he could not have survived an additional nine to ten minute period without perfusion due to cardiorespiratory arrest. Dr. Frank estimated that Sooshyance's permanent brain injury occurred "somewhere in the range of five minutes into his life" and he testified that Sooshyance was not effectively ventilated in the first four or five minutes which resulted in a longer period of time before his heart rate resumed.

{¶23} During cross examination, Dr. Frank conceded that the abnormal cord blood gas readings could have been caused by cord compression; however, he testified that he had no opinion regarding cord compression and that it would not change his opinion about the significance of the blood gas results. Dr. Frank testified that acidosis

has a deleterious effect on the time required for resuscitation and restoring heart rate. Dr. Frank admitted that the blood gases may show a component of metabolic acidosis, but that the overwhelming picture is that of respiratory acidosis, which did not cause brain damage. Dr. Frank dismissed as speculation the suggestion that an increase in the base deficit was caused by intrauterine cord compression followed by "acid wash out" after perfusion was restored.

{¶24} Defendant's OBGYN expert, Michael Belfort, M.D., serves as chairman of the OBGYN department at the Baylor College of Medicine. Dr. Belfort teaches OBGYN residents and practices maternal-fetal medicine, with a sub-specialty in fetal medicine and fetal surgery. He is board certified in both general OBGYN and maternal-fetal medicine.

{¶25} Dr. Belfort testified that Sooshyance's injuries resulted from complete umbilical cord occlusion which deprived his brain of oxygenation before and during his birth. Dr. Belfort provided a detailed review of the fetal heart monitor tracings, during which he related that the lower portion of the monitor depicted uterine activity, showing periods of contraction and the mother's "pushing" efforts. Dr. Belfort explained the difference between a variable deceleration, an early deceleration, and a late deceleration, the latter term being related to uteroplacental perfusion where the baby was responding to a hypoxic episode. Dr. Belfort testified that when the fetal reserve is diminished, a late deceleration pattern indicates the baby has decreased ability to withstand the intermittent deprivation of oxygen. According to Dr. Belfort, a variable deceleration begins when blood pressure increases, usually because of a cord compression, and a relatively thin-walled vein that is bringing blood to the fetal heart shuts down while blood is still flowing through the two thicker-walled arteries, causing tachycardia. As the contraction continues the arteries also become compressed and blood flowing from the heart is also obstructed, causing the heart rate to slow in

response to hypertension. When pressure releases, the arteries open up first and the heart rate rebounds quickly.

{¶26} Dr. Belfort opined the tracings showed that, after 1:00 a.m., the fetal heart rate was decreasing in a manner that indicated decelerations related to fetal-placental insufficiency, with moderate variability showing the baby had fetal reserve. According to Dr. Belfort, by 1:10 a.m. it appears that the baby was under some stress but still tolerating the contractions well. Between 1:15 and 1:20 a.m. there was at first moderate variability that became "a concerning late type deceleration" and then minimal variability between 1:20 and 1:21 a.m. Dr. Belfort testified that a prolonged deceleration (heart rate less than 110) lasts between two and 10 minutes and that a deceleration longer than 10 minutes would be classified as bradycardia. Dr. Belfort opined that the combination of late decelerations and variable decelerations shows that the decelerations were likely related to cord compression, rather than uteroplacental insufficiency. The change from much diminished variability at 1:21 a.m. back to more regular variability two minutes later shows the baby still had reserves at that time.

{¶27} According to Dr. Belfort, between 1:28 and 1:33 a.m., there was almost no variability remaining, which is highly suggestive that the baby was beginning to become acidemic. Dr. Belfort opined that between 1:30 and 1:31 a.m. the cord became at least partially if not completely compressed or occluded. At about that time, Dr. Artman decided to expedite delivery. Dr. Belfort related that the hospital report stated that there was minimal twisting of the umbilical cord, which increased the risk of cord compression. Dr. Belfort opined that eventually, both the vein and arteries were completely compressed between the baby's body and the bone of the symphysis pubis, effectively stopping the flow of blood and oxygen from the placenta. Between 1:34 and 1:39 a.m., variability was absent, reserves were depleted, and acid was being produced at a high rate. Dr. Belfort testified that from 1:40 to 1:43 a.m., the baby had lost its ability to control its heart rate. At approximately 1:43 a.m., the tracings end because the

scalp electrode was removed to effectuate delivery, making it difficult to accurately detect fetal heart function.

{¶28} Dr. Belfort testified that the presence of thick meconium shows the baby was emptying its bowels as a reflex to significant stress. Between 1:43 and 1:52 a.m., three attempts with the vacuum were made to deliver the baby, and Dr. Belfort opined that the cord was completely compressed and occluded during that nine-minute period of time. Dr. Belfort testified that based upon the fetal heart rate monitor tracings, some degree of resuscitation was expected, but there was nothing in the tracings that would predict a 0 Apgar score. Dr. Belfort stated that the fetal heart tracings can be summarized as a series of prolonged decelerations and that the peds team was in the delivery room when the pattern of decelerations changed to a significantly prolonged deceleration in the last minutes before birth. According to Dr. Belfort, the peds team was advised of decelerations when they were called and nurses in the delivery room did not have any new information from the monitor to relate to the residents.

{¶29} Dr. Belfort opined that the blood gas from the cord sample did not represent the status of the baby because the cord occlusion prevented both circulation of oxygenated blood and the removal of rapidly accumulating acid, such that the status of the baby was worse than the blood gas detected in the cord sample. With regard to heart rate, Dr. Belfort testified that when a fetal heart rate approaches 60 there is minimal perfusion and it was highly improbable that the “thready” pulse of 60 measured soon after birth was perfusing. Dr. Belfort opined that total cord occlusion prior to delivery resulted in significant acidosis that caused brain damage. Dr. Belfort testified that acidosis had a direct depressant effect on the heart and increased the resistance in the pulmonary artery, delaying the transition to the lungs becoming the source of oxygen after birth. Considering the blood gas values and the effects of acidosis on the heart, Dr. Belfort concluded that even with the most effective ventilation, it was not surprising that it took a considerable amount of time to resuscitate Sooshyance. Dr.

Belfort testified that an MRI showed a deep brain matter pattern that is consistent with total and acute deprivation of profusion, such as complete cord occlusion, and is not consistent with injury from a difficult resuscitation. Dr. Belfort opined that the period of time when the umbilical cord became compressed prior to birth was sufficient to cause the acidotic, hypoxic event which stopped the heart, and was necessarily severe enough to cause brain damage.

{¶30} During cross examination, Dr. Belfort admitted that initially, he was not aware that nursing notes referenced a heart rate of 60 just prior to delivery, but he stated that the additional information did not change his opinion because such a heartbeat would not be sufficient to perfuse organs. Dr. Belfort conceded that the blood gases reported by OSUMC show a mixed metabolic acidosis with primarily respiratory acidosis, but he reiterated that the cord gases did not reflect the child's acid-based status at delivery. Dr. Belfort admitted that he had not seen the bicarbonate (HCO3) calculation that Dr. Goldsmith used before he testified in this case.

{¶31} Defendant's expert Jay Goldsmith, M.D., is a professor at Tulane University School of Medicine and the chairman of the neonatology department. Dr. Goldsmith has performed extensive research and published in the areas of resuscitation and ventilation of newborns, including a chapter in his textbook, *Assisted Ventilation of a Neonate*. Dr. Goldsmith testified that he served for ten years on the committee that developed the NRP and that for a period of time he also served as co-chair for the committee. Dr. Goldsmith testified that the NRP is "universally accepted" and used by virtually every hospital in the United States and taught in about 100 other countries. Dr. Goldsmith related that the NRP is updated every five years and that the 2005 version of the NRP was in use in 2008. Dr. Goldsmith explained the NRP protocol and its use in resuscitation.

{¶32} Dr. Goldsmith testified that, by definition, the fetal heart monitor did not show bradycardia because the fetal heart rate did not drop below the baseline for ten

minutes; however, he noted that the colloquial use of the term, bradycardia, generally refers to any lowering of heart rate. Dr. Goldsmith stated that the obstetrical nurses provided the relevant information to the peds team by informing them of decelerations and meconium and that the resuscitation team was appropriately trained. Dr. Goldsmith opined that the fetal monitor tracings did not indicate a need to call a more experienced physician to attend, such as a fellow or a neonatologist. According to Dr. Goldsmith, based upon the tracings and the knowledge that Dr. Malleske was approximately two minutes away, the reasonable approach for the peds team was to assess the baby upon delivery and call for assistance if needed. Although the tracings showed prolonged decelerations, there was no new information that required the nurses to update the peds team. Dr. Goldsmith testified that there was nothing in the tracings that would have predicted that the baby would be born in such a depressed condition.

{¶33} With regard to cord blood gases, Dr. Goldsmith testified that the low arterial pH of 6.85, compared to a relatively normal reading for vein blood gas, was very worrisome and "almost diagnostic of a cord compression problem." Dr. Goldsmith related that with cord compression, the actual profile of the baby is often much worse than the cord blood gases indicate. According to Dr. Goldsmith, when cord blood gas readings are troubling, the condition of the child is often worse, but never better, than the cord blood gas values. Furthermore, Dr. Goldsmith testified that the PCO2 in the artery was "over the top" and one of the highest readings he had ever seen, suggesting complete cardiac standstill and no gas exchange. The umbilical cord compression shut off effective blood flow, such that the low heart rate of about 60 provided no effective cardiac output. Dr. Goldsmith explained that a normal umbilical cord is twisted to provide a protective mechanism, and the pathological findings show that the cord was more vulnerable than most to being obstructed. Dr. Goldsmith opined that the blood gases indicated a very acidotic bicarbonate reading.

{¶34} Dr. Goldsmith reviewed the two components of acidosis, respiratory and metabolic, and he discussed how the components of acidosis were reflected in blood gases. Dr. Goldsmith testified that the base deficit calculation for the blood gas values was minus 18, showing there was considerable metabolic acidosis. Dr. Goldsmith related that acidosis is a marker of the inability to respire and it creates depression of the myocardium, reducing the ability of the heart muscle to generate cardiac output. Dr. Goldsmith testified that acidosis also creates increased pulmonary artery pressure, inhibiting blood flow through the lungs, even during ventilation. Furthermore, it can cause decreased function of the cellular metabolic functions.

{¶35} Dr. Goldsmith opined that bag-mask ventilation can be as effective as endotracheal tube ventilation when there is no airway obstruction. Regarding the resuscitation timeline in this case, Dr. Goldsmith testified that, including the time needed in suctioning for meconium, bag-mask ventilation, chest compressions, and performing an intubation by approximately 1:55 a.m., the resuscitation was performed in accordance with the NRP and within the accepted standard of care. According to Dr. Goldsmith, Dr. Malleske's testimony showed that if he had been present at the time of delivery, at most, he would have performed the intubation one or two minutes earlier because Dr. Malleske testified that he would have been in an instructional role and allowed the residents one or two attempts at intubation before he would have performed the intubation himself. Dr. Goldsmith opined that successfully intubating one or two minutes earlier would not have made a difference in the outcome of this case inasmuch as "the injury was in place" at the time of birth. Dr. Goldsmith further opined that the slow response to resuscitation and delay in spontaneous circulation was not the result of any breach of the standard of care by the peds team; as evidenced by the fetal heart monitor tracings, the 0 Apgar score, the cord blood readings that show severe metabolic acidosis, and the lack of response to adequate ventilation. Dr. Goldsmith testified that the MRI findings were consistent with an acute total pattern, or almost total lack of

oxygen. Dr. Goldsmith opined that Sooshyance was born with brain damage and that the most skilled resuscitation would not have prevented the injury. Dr. Goldsmith concluded that there was no causal connection between Sooshyance's current condition and any action the peds team either did or did not take in resuscitating.

{¶36} During cross examination, Dr. Goldsmith admitted that the blood gas readings alone did not establish that Sooshyance had a brain injury in labor. However, Dr. Goldsmith opined that when those readings are combined with the 0 Apgar score, the fetal heart tracing, and the slow response to resuscitation, it becomes more likely than not that the brain injury occurred prior to birth. Dr. Goldsmith agreed that even a one-minute delay in resuscitation can be critical in the life of an asphyxiated neonate. Dr. Goldsmith testified that he, personally, would intubate a pulseless and apneic baby without using bag-mask PPV, but that Sooshyance first needed to be intubated to suction meconium. Dr. Goldsmith testified that he read the report of defendant's pathology expert, Dr. Salafia, but he did not believe the fetal-placental ratio was relevant to this case because cord compression was the cause of the brain injury.

{¶37} Defendant's expert, Richard Martin, M.D., is a professor of neonatology at Case Western Reserve University and a practicing neonatologist who has conducted extensive research on breathing problems in babies. Dr. Martin is the author of a leading textbook of neonatology, among other book chapters and publications. Dr. Martin has served as chair of the neonatology organization which board-certifies neonatologists.

{¶38} Dr. Martin testified that the qualifications of the peds team members conformed to the accepted standards of practice. Dr. Martin opined that the peds team was appropriately informed of the clinical situation when they arrived in the delivery room and that the pattern of late decelerations which occurred thereafter did not require the nurses to provide any updated information to the team. Dr. Martin testified that the arterial gases from the cord sample were "terrible" and showed profound acidosis, while

the venous gases, from the placenta to the baby were "pretty good." Dr. Martin opined that it was reasonable to conclude that the difference between arterial and venous blood gases was caused by a cord occlusion. Dr. Martin related that the high PCO2 showed there was no perfusion and no gas exchange from the baby to the placenta and that the baby was asphyxiating from lack of oxygen. Dr. Martin explained that as CO2 increases, bicarbonate will be elevated and that in this case, the bicarbonate calculation does not support a conclusion that there was no metabolic acidosis. Dr. Martin admitted that he made a mistake during his deposition regarding the Siggard-Anderson curve and his bicarbonate calculation. According to Dr. Martin, the correct base deficit calculation is approximately minus 18, which "is way off the charts." Dr. Martin related that the ACOG criteria for considering HIE, by either the 2002 or 2014 criteria, had been met inasmuch as both pH below 7 and base deficit greater than minus 12 were detected. Dr. Martin opined that acidosis resulted in both respiratory and myocardial depression, and increased pulmonary artery pressure, making resuscitation more difficult.

{¶39} Dr. Martin reviewed the resuscitation efforts performed by the peds team and he opined that the team followed the NRP and acted within the standard of care. Dr. Martin testified that if Dr. Malleske had been present at the time of birth, he may have been able to successfully intubate Sooshyance more quickly, but not in a significantly shorter period of time such that the outcome would have been different. Dr. Martin opined that based upon the significant acidosis, 0 Apgar, and slow response to resuscitation, Sooshyance's brain injury was the result of intrauterine events. Dr. Martin further opined that it was not likely that Sooshyance's brain damage was caused by improper resuscitation.

{¶40} Carolyn Salafia, M.D., defendant's pathology expert, is board certified in each of the sub-specialties of anatomic, clinical, and pediatric pathology. Dr. Salafia performs approximately 95 percent of her work as a placental pathologist related

directly to examining placentas. Dr. Salafia also teaches residents as the director of research in obstetrics at New York Methodist Hospital. For this case, Dr. Salafia reviewed the surgical pathology report and slides that were made from tissues taken from the placenta. From her examination, Dr. Salafia opined that the placenta was relatively small due to issues with maternal blood flow that caused some damage to the placenta. Dr. Salafia testified that the umbilical cord was larger than average, which is typical of cases where an obstruction caused increased pressure in the cord. Dr. Salafia related that the cord had "minimal twisting," a condition that is associated with a greater risk of obstruction. Dr. Salafia observed evidence of bacteria in the amniotic fluid, inflammation of the blood vessels of the umbilical cord, and bruising of the placenta. Dr. Salafia testified that she agreed with the findings in OSUMC's pathology report, but it omitted the presence of an intra-amniotic infection. Dr. Salafia opined that her findings in this case are consistent with the opinion that there was a cord occlusion. Specifically, Dr. Salafia's opinion was based upon her observation of ruptured capillaries and tissue samples that were unusual for an otherwise uncomplicated infectious process. During cross-examination, Dr. Salafia admitted that she was not aware of any clinical signs of amniotic fluid infection. However, she testified that it is common for the mother not to show signs or symptoms of such infections.

{¶41} Susan Drummond, R.N., M.S.N., defendant's nursing expert, testified that she is currently employed at Vanderbilt University Medical Center in the OBGYN department where she serves as the perinatal regionalization coordinator. Nurse Drummond's duties include teaching clinical skills such as fetal monitoring classes and reading tracings to nurses, residents, and medical students. Nurse Drummond also works as a staff nurse in labor and delivery.

{¶42} Nurse Drummond testified that the obstetrical nurses would expect the team members to execute the steps of the NRP but would not inquire about the level of experience of the peds team. Nurse Drummond discussed the timing of repetitive

decelerations that were depicted on the fetal monitoring strips, defining a period of prolonged deceleration as occurring from two to ten minutes. Nurse Drummond explained that prolonged decelerations had occurred before the peds team arrived and that pattern persisted until the time of delivery. Nurse Drummond opined that the standard of care did not require the nurses in the delivery room to inform the peds team of any additional information because the team was already aware of the presence of decelerations and meconium. Although Nurse Drummond testified that the monitoring strips were concerning, she related that there was nothing on the strips to indicate the baby would be born with a 0 Apgar score. According to Nurse Drummond, the monitoring strips did not show bradycardia by any definition of the term. Based upon her review of the monitoring strips, Nurse Drummond stated that she would expect the baby to be somewhat depressed at birth, but not to require resuscitation involving chest compressions, ventilation, or intubation for ventilation and epinephrine.

{¶43} During cross-examination, Nurse Drummond testified that the standard of care requires a degree of cooperation, coordination, and communication between the obstetric nurses and the peds team. Nurse Drummond opined that the labor and delivery nurses met the standard of care in this case when they summoned the peds team.

{¶44} Upon review of the evidence presented at trial, the court finds that the treatment provided by OSU medical staff complied with the relevant standard of care at all times. Regarding the information that was communicated between the delivery room nurses and the peds team, there is no question that the peds team relied on the nurses to read and interpret the fetal heart monitoring strips. Plaintiffs argue that defendant's nursing staff committed a breach of the standard of care because the nurses failed to inform the peds team of an emergent situation that was apparent from the fetal heart monitoring strips. However, the court is not convinced that the information contained on the monitoring strips alone indicated a situation that required either the nurses to

provide an update or the peds team to summon Dr. Malleske. The court is persuaded by Nurse Drummond's opinion that the delivery room nurses met the standard of care in this case and that the variations shown on fetal monitoring strips did not require an update because the peds team members were already aware of the presence of decelerations and meconium. Dr. Adams confirmed that when the peds team was summoned, the members were advised of meconium-stained amniotic fluid and decelerations. The testimony showed that the nurses in the delivery room understood that the peds team was adequately trained and ready for any resuscitation situation, including intubation.

{¶45} Furthermore, Nurse Cox was adamant that she did communicate her concerns about the fetal monitoring strips. Nurse Cox testified that she arrived in the delivery room approximately ten minutes before delivery and that the monitoring strips in this case were "some of the worst" heart rate tracings she had ever seen in her four years as an OB nurse. Nurse Cox was aware that the peds team did not read or interpret fetal heart tones and that an update on that information would need to be made by either one of the OB nurses or Dr. Artman. Nurse Cox testified that she personally informed the peds team that the fetal heart tracings were very concerning and that they should "be prepared for the baby to not do well." Although the members of the peds team testified that they did not recall any update regarding changes or concerns with the fetal heart rate monitor, Dr. Adams testified the delivery room was a noisy and "hectic place" where the medical staff was working "in the zone" and that an update could have been voiced but not heard. The court finds that Nurse Cox's testimony that she communicated concern about the fetal heart tracings to the peds team was credible.

{¶46} Nurse Cox testified that, although the monitor tracings were concerning, they were not predictive and no one knew the baby was going to be born with a 0 Apgar score. Likewise, Nurse Drummond opined that the monitoring strips showed some

cause for concern, but no indication that the baby would be born unresponsive with a 0 Apgar score. Nurse Cox related that it was not uncommon to see very concerning tracings, particularly at the end of delivery. According to Nurse Cox, most babies with similar strips do well after delivery. Dr. Adams related that the peds team was surprised that the baby was born limp and unresponsive. Indeed, plaintiff's expert, Dr. Ouzounian testified that "bradycardias" or the type of prolonged decelerations shown in the tracings in this case are common and "the majority of these kids do great." Dr. Ouzounian admitted that the prolonged decelerations continued in the minutes prior to delivery and that the decelerations did not meet the definition of bradycardia. The court finds that, even if Nurse Cox had not informed the peds team of her concern, the nursing staff did not breach the standard of care regarding its duty to update the team with any critical changes in the information depicted in the monitoring strips.

{¶47} Plaintiffs' contention that Sooshyance could have been resuscitated quickly with effective ventilation is based, in part, upon the assertion that he had a palpable pulse greater than 60 soon after delivery. However, Nurse Cox recorded her observations during labor and delivery and she testified that she did not believe anyone detected a palpable pulse after delivery and that a 0 Apgar score was assigned the first time the baby was assessed at one minute after delivery. Nurse Cox related that it was difficult to trace a fetal heart rate prior to delivery and that, according to the medical record, fetal heart tones were heard in the last few seconds of pushing before delivery. The first time Nurse Cox became aware of a pulse being detected after birth was at 2:01 a.m., after approximately nine minutes of cardiorespiratory arrest.

{¶48} Although plaintiffs maintain that the peds team did not have the appropriate level of experience to handle the resuscitation of a severely depressed neonate, the three-member team included both a second year resident (Dr. Nehus) and a third year resident (Dr. Adams), who was only a few weeks from completing her residency and becoming an attending pediatrician. Each member of the peds team was NRP certified

and prepared for the worst case scenario. There is no dispute that the residents followed the NRP protocol and plaintiffs' experts acknowledged that they have no criticism of the treatment provided by peds team.

{¶49} Plaintiffs also contend that there was a critical delay of approximately three and one half minutes in resuscitating Sooshyance because Dr. Malleske was not summoned prior to delivery. Although plaintiffs argue that Dr. Malleske would have intubated the baby by one and one half minutes after birth, Dr. Malleske testified that even if he had been present at the time of delivery, he would have instructed the residents to perform the intubation in the same manner as they did in this case. Dr. Malleske stated that he would have been in an instructional role and allowed the residents to attempt intubation. Indeed, Dr. Nehus had attempted intubation once before Dr. Malleske performed the second attempt. Dr. Malleske testified that if he had been present when the baby was delivered he would have followed the NRP guidelines, suctioned for meconium, attempted PPV (bag-mask) and then if the baby had not been resuscitated after 30 seconds, he would have intubated the baby. Likewise, Dr. Nehus testified that, in accordance with the NRP, he performed PPV for about 30 seconds before he decided to start chest compressions. As chest compressions were being performed, Dr. Nehus began preparations for intubating for ventilation. Therefore, according to Dr. Malleske's testimony, if he had been present at the time of birth, he would have proceeded with resuscitation in the same manner as did Dr. Nehus. The court finds that it is more likely than not that Dr. Malleske would have also taken time to prepare for intubation while chest compressions were performed. The evidence does not support the conclusion that Dr. Malleske would have been able to perform an intubation in a significantly shorter period of time than that required by Dr. Nehus. Dr. Nehus intubated for ventilation at approximately 1:55 a.m. Dr. Malleske entered the delivery room at 1:56 a.m. and after assessing the baby, he re-intubated at approximately 1:57 a.m.

{¶50} Upon review of the evidence, the court finds that plaintiffs' contention that Dr. Malleske would have intubated the baby within one and one half minutes after birth is not supported by the evidence. Furthermore, for the reasons discussed below, plaintiffs' argument that ventilation provided by the peds team was inadequate or ineffective because Sooshyance did not quickly regain a heartbeat and respond to resuscitation is not persuasive. The court finds that there was no unreasonable delay in providing effective resuscitation. Moreover, plaintiffs' assertion that Dr. Malleske would have performed resuscitation in a different manner is not persuasive.

Causation

{¶51} Plaintiffs must prove that Sooshyance's injury was the direct and proximate result of defendant's breach. *Bruni, supra*. While the court is sympathetic to the severe injury the child suffered, there is no presumption of malpractice merely because the patient has sustained an injury. *Ault v. Hall*, 119 Ohio St. 422, 428 (1928).

{¶52} There is no question that Sooshyance's HIE brain injury was the result of hypoxia; however, plaintiffs contend that HIE occurred after birth. Dr. Ouzounian testified that his central opinion in this case is that there could not have been hypoxic neurologic injury caused by intrapartum oxygen deprivation because there was a lack of evidence of metabolic acidemia. However, plaintiffs' experts acknowledged that there was at least some degree of metabolic acidosis at the time of birth. Both Dr. Ouzounian and Dr. Mahlmeister testified that the fetal monitor strips were consistent with metabolic acidosis. Both Dr. Karotkin and Dr. Frank acknowledged that there was a metabolic component, but Dr. Karotkin opined that there was only a mixed metabolic component based upon the bicarbonate and base excess. Dr. Karotkin agreed that when blood flow is isolated by sustained umbilical cord compression, a cord blood sample will not reflect the recent condition of the fetus, such as an artificially high pH reading. Dr. Karotkin acknowledged that there were "striking" differences between the venous and arterial blood samples, which could be diagnostic of cord compression.

{¶53} Dr. Ouzounian admitted that he did not know what caused the baby to stop breathing, the loss of heart rate, or the initial 0 Apgar score, and he testified that he would defer to the opinion of a pediatric neurologist on those issues. Dr. Ouzounian acknowledged that the ACOG guidelines for determining the existence of HIE during labor were revised in 2014 and that the enumerated criteria require only pH less than 7 *or* a base excess greater than 12, rather than both criteria. Dr. Ouzounian and Dr. Frank both testified that a difference between arterial and venous cord blood pH values can suggest that there was cord compression.

{¶54} In contrast to plaintiffs' experts, Dr. Goldsmith opined that there was considerable metabolic acidosis based upon the base deficit calculation; a value that was not initially considered by plaintiffs' experts. For instance, Dr. Frank did not calculate blood gases and he admitted that there was no base deficit calculation in the records he reviewed. Plaintiffs' experts did not provide any credible evidence to contradict Dr. Goldsmith's calculations, which objectively demonstrate there was significant metabolic acidosis present at the time of birth. Dr. Goldsmith also explained that acidosis causes depression of the myocardium and increased pulmonary artery pressure that inhibited Sooshyance's response to effective resuscitation. As discussed above, Dr. Goldsmith opined that the differential between the low arterial pH and the relatively normal vein blood gas was very worrisome and "almost diagnostic" of a cord compression. Dr. Martin reached the same conclusion. Dr. Salafia related that the condition of the umbilical cord was consistent with a greater risk of obstruction and that the findings in the pathology report supported the opinion that there was a cord occlusion. Dr. Belfort explained that the cord blood gases reported by OSUMC did not reflect the acid-based status at the time of delivery due to cord occlusion and the status of the baby was worse than the readings showed. Dr. Goldsmith opined that even if Dr. Malleske had been in the delivery room and effectively ventilated Sooshyance two

minutes after birth that "to a very high degree of probability," the baby was still going to require chest compressions and epinephrine due to severe acidemia.

{¶55} Based upon the totality of the evidence, the court finds that Dr. Goldsmith's testimony regarding the significance of the umbilical cord blood gases was credible and more persuasive than that of plaintiffs' experts. Furthermore, the court finds that there was significant metabolic acidosis present at the time of birth as a result of either a completely or severely occluded umbilical cord. The court finds that the evidence shows that the cord blood values in this case meet the applicable ACOG criteria for intrauterine causation. The court is persuaded by Dr. Goldsmith's opinion that it was not reasonably probable that Sooshyance would have regained circulation within two minutes if resuscitation had been handled differently.

Heart rate/circulation

{¶56} Moreover, Dr. Goldsmith explained that even if there was a heartbeat near the time of birth, when a severe cord occlusion exists there is essentially no blood flow or cardiac output from a non-perfusing rhythm. In addition to the physical obstruction, the depression of the myocardium reduced the ability of the heart muscle to generate cardiac output. Dr. Belfort opined that a heart rate of 60, or the "thready" pulse detected soon after birth, was insufficient to perfuse organs. According to Dr. Belfort, there was a sufficient period of time prior to birth where total or near total cord occlusion caused a lack of perfusion and circulation capable of producing significant brain damage. Dr. Belfort's opinion is supported by the MRI which both Dr. Belfort and Dr. Goldsmith agreed was consistent with a lack of perfusion caused by umbilical cord occlusion, but was not consistent with an injury caused by inadequate resuscitation. Plaintiffs' experts acknowledged that significant acidosis is consistent with a brain injury occurring prior to birth. Both Dr. Goldsmith and Dr. Martin opined that the medical record, including the blood gas readings combined with the fetal heart tracings, the condition of the child at

birth (0 Apgar scores), and the slow response to resuscitation, show that the brain injury occurred prior to birth.

{¶57} The court concludes that Sooshyance sustained prolonged hypoxia which caused HIE during an intrauterine cord compression event, as reflected by the differential in the cord blood gas values. The evidence shows that the peds team had the proper training and experience to handle an emergent resuscitation and acted within the standard of care by providing competent treatment for cardiac arrest in accordance with the NRP. The peds team employed PPV and endotracheal ventilation, chest compressions, and epinephrine, which ultimately resuscitated the child. However, their resuscitation efforts could not reverse the neurological damage sustained prior to birth.

{¶58} For the foregoing reasons, the court finds that plaintiffs failed to prove that Sooshyance's injury was the direct and proximate result of any breach by defendant. With regard to plaintiffs' claims for loss of consortium, "a claim for loss of consortium is derivative in that the claim is dependent upon the defendant's having committed a legally cognizable tort" upon the individual who suffers bodily injury. *Bowen v. Kil-Kare, Inc.*, 63 Ohio St.3d 84, 93 (1992). Since plaintiffs have failed to prove their claims of negligence, the loss of consortium claims must also fail. Accordingly, judgment is recommended in favor of defendant.

{¶59} *A party may file written objections to the magistrate's decision within 14 days of the filing of the decision, whether or not the court has adopted the decision during that 14-day period as permitted by Civ.R. 53(D)(4)(e)(i). If any party timely files objections, any other party may also file objections not later than ten days after the first objections are filed. A party shall not assign as error on appeal the court's adoption of any factual finding or legal conclusion, whether or not specifically designated as a finding of fact or conclusion of law under Civ.R. 53(D)(3)(a)(ii), unless the party timely and specifically objects to that factual finding or legal conclusion within 14 days of the filing of the decision, as required by Civ.R. 53(D)(3)(b).*

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