SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK: PART 11

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M.W., an infant, by her mother and natural guardian, NICOLA WATKINS, INDEX NO. 80528814

Plaintiff,

-against-

NANCY CANDELARIO, DIANA ORTIZ, DAVID E. SEUBERT, LYDIA GARCIA, GARY MUCCIOLO, and NYU LANGONE MEDICAL CENTER,

Defendants.

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JOAN A. MADDEN, J.:

In this medical malpractice action based on injuries caused during labor and delivery at defendant hospital on November 20, 2007, defendants are moving for summary judgment and plaintiff opposes.

Plaintiff alleges defendants departed from the standard of care by not performing a timely caesarian section, and as a result her infant daughter sustained a prolonged period of hypoxia (loss of oxygen), resulting in brain damage and other neurologic injuries.

A defendant moving for summary judgment in a medical malpractice action must make a prima facie showing of entitlement to judgment as a matter of law by showing that "in treating the plaintiff, there was no departure from good and accepted medical practice or that any departure was not the proximate cause of the injuries alleged." Roques v. Nobel, 73 AD3d 204, 206 (1st Dept 2010). To satisfy the burden, defendant must present expert opinion testimony that is supported by the facts in the record, addresses the essential allegations in the complaint or the bill of particulars, and is detailed, specific and factual in nature. Id; see Joyner–Pack v. Sykes, 54

AD3d 727, 729 (2nd Dept 2008). Expert opinion must be based on facts in the record or those personally known to the expert, and the opinion of defendant's expert should specify "in what way" the patient's treatment was proper and "elucidate the standard of care." Ocasio-Gary v. Lawrence Hospital, 69 AD3d 403, 404 (1st Dept 2010). Defendant's expert opinion must "explain 'what defendant did and why.'" Id (quoting Wasserman v. Carella, 307 AD2d 225, 226 [1st Dept 2003]).

"[T]o avert summary judgment, plaintiff must demonstrate that the defendant did in fact commit malpractice and that the malpractice was the proximate cause of the plaintiff's injuries." Roques v. Nobel, supra at 207. To meet this burden, "plaintiff must submit an affidavit from a medical doctor attesting that the defendant departed from accepted medical practice and that the departure was the proximate cause of the injuries alleged." Id. Where the parties' conflicting expert opinions are adequately supported by the record, summary judgment must be denied. See Frye v. Montefiore Medical Center, 70 AD3d 15 (1st Dept 2009); Cruz v. St Barnabas Hospital, 50 AD3d 382 (1st Dept 2008).

In support of summary judgment, defendants submit affirmations/affidavits of three medical experts: Dr. William Ted Brown who is board certified in internal medical and genetics; Dr. Joseph Maytal a board certified neurologist; and Dr. Dwight J. Rouse who is board certified in obstetrics, gynecology and maternal fetal medicine. Based the opinions of these experts, defendants assert the infant plaintiff did not suffer hypoxia during labor and delivery, but rather suffers from a congenital syndrome known as Arthrogryposis Multiplex Congenita-Type C with peripheral contractures and central nervous system dysfunction and/or intellectual disability, and

that her physical, neurological, intellectual and developmental disabilities are related solely to this syndrome.

Dr. Brown opines that Arthrogryposis Multiplex Congentia – Type C was present in the infant plaintiff at conception and manifested while the infant was *in utero* as her nervous system developed. He states the term "arthrogryposis" describes approximately 400 identified congenital conditions involving decreased fetal movement *in utero* and multiple joint contractions at birth, which usually involve the limbs but can also include limitations of full range of motion of the jaw, neck or spine; here, the infant plaintiff suffers from Type C, which also involves impaired development of her central nervous system.

Dr. Brown opines that the diagnosis of Arthrogryposis Multiplex Congentia – Type C was "first suggested" when the mother noted decreased fetal movement approximately four weeks prior to premature birth at 34 weeks gestational age, and at birth the infant was small for gestational age (29-30 weeks).[1] He opines that these findings are "consistent with this congenital syndrome and are not consistent with injury due to a recent hypoxic event during labor and delivery."

Dr. Brown further opines that at delivery, the infant's cord blood gas results "confirm she was not deprived of oxygen in the preceding hours," and she needed resuscitation due to an "inability to breathe spontaneously, an apparent disconnect between the brain and the muscles of respiration, which is a sign of central nervous system dysfunction ("CNS") associated with

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[1]While Dr. Brown also opines that the infant plaintiff's head was "extremely microcephalic, meaning her head size was less than 1% of normal for her gestational age," the hospital records consistently note the "head" as "normal." Dr. Brown does not provide citations to portions of the record on which he relies. However, he does make this finding as a result of his examination on February 29, 2016 when the infant plaintiff was 8 years old.

Arthrogryposis Multiplex Congentia – Type C." He opines that the "features" of this condition were "so apparent" at birth, e.g. contractures of her extremities, clenched fists, hypertonia) the diagnosis was immediately made on admission to the nursery, and confirmed by a "genetic consultation" while at a patient in the NICU. He additionally opines that since Arthrogryposis Multiplex Congentia – Type C and CNS dysfunction are the "sole cause" of the infant's condition, delivery at any time prior to the caesarean section performed at 11:36 p.m. on November 20, 2007, "would not have affected the infant-plaintiff's condition," as Arthrogryposis Multiplex Congentia – Type C and CNS dysfunction develop during the "early stages of gestation and the brain's development" and are not caused by hypoxia. Dr. Brown opines hat based on the infant's presentation at birth and post-birth laboratory work and diagnostic films, there is no evidence the infant was deprived of oxygen and sustained "any type of injury as a result of oxygen deprivation" that could have been avoided with "earlier delivery."

Dr. Brown opines this diagnosis and his opinions are confirmed by his examination of the infant plaintiff on February 29, 2016. He states that on examination, he noted that "microcephaly" was "apparent" and her head was "long, narrow and cone-shaped with mild occipital prominence and a flat occiput," which are "congenital anomalies known as Brachycephaly and Turricephaly." He also noted the following conditions: "strabismus with exotropia with her right eye looking far to the right (a sign of abnormal neural connections)"; "an extremely marked Pectus Carnatum or 'pigeon chest deformity' also a congenital abnormality"; "a marked cervical kyphosis"; fists tending to be clenched with bilateral ulnar deviation; fingers tending to be tightened; left hand transverse palmar crease was nearly

completely transverse; elbows could only be extended to 120 degrees (145 degrees is normal); flat footed with clenched toes; and "profoundly mentally retarded." He opines that these are "all signs" of Arthrogryposis Multiplex Congentia – Type C, with Peripheral Contractures and CNS (central nervous system) dysfunction. In conclusion, Dr. Brown opines that the "undeniable presence" of Arthrogryposis Multiplex Congentia – Type C, and the "uncontroverted proof" that the infant plaintiff did not have hypoxia at birth or suffer a "hypoxia insult at any time," establish within a reasonable degree of medical certainty that none of the alleged injuries are "related to the treatment rendered in this matter," and there is no causal connection between the treatment and the injuries alleged, which are "due to a genetic disorder present from the time of conception."

Defendants' neurologist, Dr. Maytal, confirms of the opinions of Dr. Brown. Dr. Maytal opines that there is no objective evidence of hypoxic ischemic encephalopathy ("HIE") during Ms. Watkins' labor and delivery, as brain imaging studies did not show any evidence of HIE, and the infant's arterial cord blood gas was within "safe range." He opines that since the infant was diagnosed with arthrogryposis, a pre-natal condition that occurred during gestation and not during labor and delivery, that condition "is more likely than not responsible for the infant's neurological deficits." He opines that the November 29, 2007 MRI did not show "typical signs of HIE such as diffuse bilateral cerebral edema or evidence of hyper intense lesions over the basis ganglia and the thalami," and the "subependymal cysts noted . . . were considered insignificant or incidental."

Dr. Maytal opines, that "[m]ost significantly, the diagnosis of arthrogryposis at birth is inconsistent with HIE," as arthrogryposis multiplex congenita is a neurogenic condition which

results in joint contracture with weak muscle tone," and "occurs as a result of the abnormal development of cells in the spinal cord and brainstem and cortex and leads to central nervous system deficits." He opines that the "diagnosis of arthrogryposis at birth involved the central nervous system and this condition occurred prenatally."

Dr. Maytal states that it is "accepted within the medical and scientific communities" that when an infant suffers HIE during labor and delivery, it is "reflected in the arterial cord blood gas, which typically become acidotic as a result of neonatal asphyxia." He opines that here the infant's arterial cord blood gas of a pH of 7.11 and base excess of -8.9 are "values . . .within the accepted range outlined by the American College of Obstetrics and Gynecology (ACOG); according to ACOG guidelines, arterial cord blood of 7.0 or less and base excess larger than -12 indicate the "possibility the baby was at risk for HIE during labor and delivery." Dr. Maytal opines that based on the "data provided," the infant plaintiff "did not suffer HIE during labor and delivery," and her "low Apgar scores at birth were more likely than not the result of her congenital defects," as evidenced by the infant's "small for gestational age and intrauterine growth restriction." He concludes by opining that the infant plaintiff's "normal cord-blood gas and normal brain MRI" and diagnosis of arthrogryposis are "irrefutable evidence that the infant did not experience an acute hypoxic event prior to delivery sufficient to cause HIE."

Defendants' expert in obstetrics, gynecology, and maternal fetal medicine, Dr. Rouse, opines that infant plaintiff's neurological deficits are "completely unrelated to labor and delivery," there was "no acute hypoxic insult intrapartum," and "it is clear the infant plaintiff's development *in utero* was impaired from the earliest stages due to a genetic defect." He details the prenatal care provided by defendant Dr. Mucciolo and the care provided at defendant

hospital. He opines that on October 23, 2007, when plaintiff complained of decreased fetal movement, Dr. Mucciolo "appropriately evaluated" her by "non-stress testing and a biophysical profile," and when the results of the testing were "within normal limits for gestational age," she was "appropriately discharged home."

Dr. Rouse states that when Ms. Watkins saw Dr. Mucciolo on November 19, 2007, she explained that she had gone to another hospital for "questionable pre-term labor" and was discharged when it was determined she was not in labor. Dr. Rouse states that at the November 19, 2007 visit, Dr. Mucciolo performed a vaginal exam, and noted the "cervix was long, closed and posterior," indicating she had not experienced labor; he also performed a "transvaginal sonogram to determine cervical length, which was within normal limits at 3.08 centimeters." Dr. Rouse opines that a "short cervix is a risk factor for preterm labor and the length of the cervix indicated that she was not in preterm labor." He opines that his review of the chart and Dr. Mucciolo's testimony "confirm that all other parameters were normal during this time period: blood pressure; weight and urine; the fetal position was vertex (head first); fundal height (distance from the pubic bone to the top of the uterus) was 34 cm, and the fetal heart rate was 148 beat per minute." He opines that given the entire examination and "most importantly the cervical exam, there were no signs of pre-term labor present on November 19, 2007," and "no indications for any additional testing or workup."

Dr. Rouse opines that on Ms. Watkins' admission to the hospital on November 20, 2007, the "care plan and management" provided by the hospital was "well within good and accepted practice and it continued to be throughout delivery." He states that when Ms. Watkins called Dr. Mucciolo at approximately 8:30 p.m. on November 20, 2007, and told him that her membranes

had ruptured, he told her to go defendant hospital. He states that according to the labor flow sheets, Ms. Watkins was admitted at 9:01 p.m., placed on an external fetal heart monitor and made comfortable; at 9:14 p.m., Dr. Lydia Garcia, a second year ob/gyn resident, conducted a "thorough evaluation," by taking a complete history and physical exam, including a pelvic exam. Dr. Rouse states that an ultrasound was performed to confirm that the fetus was in a "vertex presentation"; blood work was taken; antibiotics were started since Ms. Watkins' "Group B Strep carrier status was not known at the time"; and on "vaginal exam the cervix was fingertip and she was only 50% effaced and fetal station was -2 indicating Ms. Watkins was not in active labor." He states that Dr. Garcia's "assessment" is noted in the chart, along with her telephone call with Dr. Mucciolo, who "agreed with the plan" to continue monitoring Ms. Watkins. Dr. Rouse states that the "fetal heart rate baseline was 140 to 150 bpm and contractions were "irregular" and "palpated by Nurse Candelario and Nurse Ortiz since they were not strong enough to be picked up by the monitor."

Dr. Rouse opines that the labor flow sheets show that Ms. Watkins "continued to be carefully monitored . . .every few minutes; the "fetal heart rate tracing baseline and reactivity were well within normal limits for the gestational age of 34 weeks"; and at 9:52 p.m., "some variable decelerations started to occur which were immediately recognized by nursing staff." He states that "variable decelerations are an abrupt decrease in fetal heart rate below the baseline which may or may not be associated with uterine contractions," and that "appropriate management, as done here, includes maternal repositioning, fluid resuscitation, ensuring adequate maternal oxygenation and frequent evaluation." He opines the "most likely" cause of

variable decelerations is "intermittent cord compression and thus, such decelerations most often do not require any treatment and are associated with normal prenatal outcomes." He opines that at 9:52 p.m., there was a variable deceleration to 70 bpm lasting 40 seconds; at 9:55 p.m., a variable deceleration to 70 bpm lasting 40 seconds; and that the fetal heart rate "returned to baseline with appropriate interventions such as changing Ms. Watkins' position, increasing IV fluids and administering oxygen via face mask."

Dr. Rouse opines that "at all times," the labor and delivery nursing staff "appropriately monitored and evaluated" Ms. Watkins and her fetus, and nurses Candelario and Ortiz followed the guidelines of the American College of Obstetrics and Gynecology (ACOG) as to "management of a patient exhibiting this type of pattern on EFM tracing." He opines that "following a prolonged variable deceleration" at 10:54 p.m., the nursing staff showed "good judgment" by paging Dr. Garcia for a "further evaluation of the patient." He states that since Dr. Garcia was in a cesarean section delivery and could not respond, Dr. Seubert, the on-call attending and the Chief of Obstetrics and Maternal Fetal Medicine at NYU Medical Center, "was at the patient's bedside within two minutes, evaluating the fetal heart monitoring strips and performing a vaginal exam," which showed that the "cervix was only 1 cm dilated and still only 50% effaced, with the fetal station at -1, indicating that Ms. Watkins was not yet in active labor." Dr. Rouse opines that since Dr. Seubert "could not feel the presenting part on exam, he performed an ultrasound which confirmed a vertix presentation but showed an 'occult' cord, which meant the umbilical cord was lying next to/in front of the fetal head." He opines that the "greatest risk in such a situation is that the umbilical cord will deliver before the presenting part

of the baby, causing compromised blood flow to the fetus with possible risk of fetal damage or death"; therefore, as recognized by Dr. Seubert and the labor and delivery team, an "urgent cesarean section was the management choice to prevent cord prolapse into the vagina and fetal compromise." He opines that following Dr. Seubert's ultrasound at 11:01 p.m., Dr. Mucciolo was called and advised that he would be on his way to the hospital but to proceed without him.

Dr. Rouse states that the consent for the cesarean section was signed at11:09 p.m.; Ms. Watkins was in the operating room and "positioned" for epidural spinal anesthesia at 11:17 p.m.; the first incision was made at11: 32 p.m.; and the baby was delivered at 11:36 p.m., "35 minutes after the ultrasound was performed and 27 minutes after the consent was obtained for the cesarean section." He opines that "even though the fetal heart tracings always returned to baseline and were always reassuring, the infant plaintiff was delivered within the accepted time frame for an 'emergency' cesarean section delivery." He opines that in any event, the ACOG guidelines state that the "use of '30 miniutes to delivery' is a rather arbitrary number and refers to the desired capability of a hospital that provides obstetric services to be able to respond to an emergency," and that it is "not intended to be a standard of practice in which a particular cesarean section delivery is to be carried out." Citing a 2006 journal article, Dr. Rouse opines that "it has been noted that in cases where the decision to perform an emergency cesarean section was for non-reassuring fetal heart tracings, the adverse neonatal outcomes were not increased where the cesarean section was not commenced within 30 minutes after the decision to operate."

Disagreeing with the claims in plaintiff's expert disclosure, Dr. Rouse opines that "it is not the standard of care for the labor and delivery room team to contact a patient's attending

ob/gyn because of Category II variable decelerations when those decelerations respond to appropriate interventions," and in those situations, "acceptable management" is positioning the mother, fluid resuscitation and oxygen administration, which the staff administered to Ms. Watkins. He further opines that when Dr. Seubert examined Ms. Watkins at approximately 11:00 p.m. and "discovered an occult cord on ultrasound, Dr. Mucciolo was called and knowing this patient was in good hands, asked that they proceed while he was on his way to the hospital," and Dr. Seubert, as Chief of Obstetrics and Gynecology and Maternal Fetal Medicine, "was more than capable of delivering this patient."

Dr. Rouse also disagrees with plaintiff's claim that Ms. Watkins should have been given general anesthesia instead of an epidural. He opines that an epidural is "safer" for both mother and fetus where, as here, there is no acute fetal distress; it can be accomplished "quickly as it was here"; and the risks of general anesthesia include "difficulties in airway management and maternal death, and decreased uterine blood flow and neonatal depression." He opines it is "very doubtful that induction with general anesthesia would have been accomplished more rapidly and the risks outweighed any benefits particularly since Ms. Watkins had not been NPO [nothing by mouth], which increases the risk of aspiration of stomach contents into the lungs."

Dr. Rouse further disagrees with plaintiff's claim that a cesarean section was not timely performed. He opines that following each of the variable decelerations, "there was always a return to baseline, indicating fetal well being," and that none of the "events" during labor and delivery, including the fetal heart rate tracing in their entirety, are "consistent with a hypoxic ischemic insult or hypoxic ischemic encephalopathy during labor and delivery." He opines that

the infant's cord blood gases following delivery "were within normal range and inconsistent with hypoxic ischemic encephalopathy," and that the infant's "low APGAR scores and difficulty breathing at the time of delivery and thereafter were the result of her congenital syndrome and prematurity." He states that he reviewed the placental pathology report, and notes the "presence of diffusely increased syncytial knots and diffusely increased perivillous fibrin with micro infarcts," and opines such findings are "indicative of chronic malperfusion of the placenta in the months prior to labor and delivery, and are not indicative of an acute hypoxic event." Dr. Rouse opines that the fact that the infant plaintiff was "born small for gestational age and microcephalic . . . is evidence of abnormal development *in utero* well before labor and delivery," and states that he is "aware that the infant-plaintiff has been diagnosed with Arthrogryposis Type C shortly after birth, a congenital syndrome which is consistent with her clinical picture."

In conclusion, Dr. Rouse opines that defendants' medical care and treatment was "at all times well within" appropriate standards of care; the infant-plaintiff's neurological deficits are "completely unrelated to labor and delivery"; there was "no acute hypoxic insult intrapartum"; and it is "clear that the infant-plaintiff's development *in utero* was impaired from the earliest stages due to a genetic defect."

Turning to the opposition, plaintiff relies on one expert who primarily addresses the departures, and another expert who primarily addresses causation. First, as to the departures, plaintiff submits the expert affirmation of Dr. Engelbert, who is board certified in obstetrics and gynecology. He opines that defendants departed from the standard of care by failing to timely recognize and respond to the signs of fetal distress, which resulted in her "continuing to sustain a period of hypoxia."

As indicated above, the record shows that Ms. Watkins arrived at defendant hospital at or about 9:01 p.m., when she was first seen by defendant Nurse Candelario, and at 9:14 p.m., she was examined and evaluated by defendant Dr. Garcia. Dr. Engelbert opines that the fetal heart monitoring tapes show that "beginning a 9:04 p.m, there were periods of time when Ms. Watkins was having contractions that could be seen on the tapes" and the "baseline fetal heart rate started at 140, went up to 170 during the variable decelerations, and then returned to the 140s to the 150s," which Nurse Candelario characterized as Category I going to Category II and returning to Category I. Dr. Englebert states that at 10:18 p.m., Nurse Ortiz noted "minimal fetal heart rate beat to beat variability, and variable periodic fetal heart rate decelerations with a gradual return to baseline," and that prior to this time the beat to beat variability had been "average"; Nurse Ortiz characterized this as a Category II tracing which required "interventions," i.e. increasing fluids, administering oxygen and changing the patient's position.

Dr. Engelbert states that at 10:34 p.m. and 10:45 p.m., Nurse Ortiz recorded minimal variability; at 10:46 p.m., she repeated the prior note as to the interventions and characterized this as a Category II tracing; and 10:54 p.m. she repeated the prior note as to the interventions, noted Ms. Watkins was in the "left lateral position," and noted she had reviewed the fetal heart monitoring tapes and paged Dr. Garcia to "come and evaluate the patient"; she testified that for Category II tracings, after interventions, "someone needs to come and review this tracing to see if there is a plan of care change." Dr. Engelbert states that Dr. Garcia was not available and Dr. Seubert responded; at 10:56 p.m. Nurse Ortiz noted that Dr. Seubert reviewed the fetal heart monitoring tapes and performed a vaginal exam, finding Ms. Watkins "1 cm dilated and 50% effaced, with the presenting part of the fetus at the -1 station"; and the labor flowsheet notes that

consent for the cesarian was obtained at 11:09 p.m. Dr. Engelbert states that when Nurse Ortiz was asked to review the heart monitoring tapes, she testified to the presence of "deep variable decelerations" at 9:53 p.m and 9:55 p.m., as well as "variable decelerations" at 10:50 p.m., 10:54 p.m., 11:05 p.m., 11:07 p.m., 11:09 p.m., 11:13 p.m., 11:14 p.m., 11:17 p.m., 11:20 p.m., and 11:22 p.m.

Dr. Engelbert opines that Dr. Seubert had a "slightly different interpretation" as to why he was contacted, as he noted at 11:05 p.m. that he was called to see Ms. Watkins in response to a "prolonged deceleration," which he described as "variable deceleration that had started at 10:54 p.m., lasted five (5) minutes, and went down to 90 bpm." Dr. Engelbert opines that from Dr. Seubert's review of the fetal monitoring tapes, there were "variable decelerations lasting up to 40 seconds and going down as far as 80 bpm at 9:15 p.m., 9:52 p.m, 9:55 p.m, 10:50 p.m. and 10:54 p.m.; Dr. Engelbert notes that the decelerations at 9:15 p.m and 10:50 p.m. are not documented in the labor flowsheet.

Dr. Engelbert states that Dr. Seubert performed an ultrasound to confirm the presentation of the fetus and the location of the cord, and to check the volume of amniotic fluid, and found the presence of a "occult cord" and no amniotic fluid. He points to Dr. Seubert's testimony that he wanted to perform an "urgent" cesarean, meaning "as soon as we can take her back and safely administer anesthesia," based on the decelerations and the location of the cord, as the cord was "in front of the head with broken water, the cord was ultimately going to come out. The impact of that is compromise for fetal well being." Dr. Engelbert states that at approximately 11:09 p.m, plaintiff was disconnected from the fetal heart monitor and reconnected at 11:14 p.m. after she

was moved to the operating room; Dr. Seubert noted variable decelerations prior to disconnection at 11:03 p.m, 11:05 p.m and 11:08 p.m. , and variable decelerations after re-connection at 11:16 p.m. and 11:21 p.m. Dr. Engelbert states that Ms. Watkins was "positioned for epidural" at 11:17 p.m., the first incision was made at 11:32 p.m., and the baby was delivered at 11:36 p.m.

Based on the above, Dr. Engelbert opines that it is "not custom and practice" to perform an "urgent" or "emergent" cesarean section for "reassuring fetal heart rates," but here it is "clear" the fetal heart monitoring tapes were not reassuring and "had not been reassuring for at least an hour before the decision to perform the cesarean was finally made." He opines the "situation was emergent," the delivery needed to be accomplished as "quickly as possible"; and when the "fetal heart monitoring tapes are bad, you cannot afford to wait any longer than you have to, particularly with a fetus that is premature." He also opines that if general anesthesia had been administered instead of an epidural, "the first incision could have been made 12 to 15 minutes earlier."

Dr. Engelbert opines that defendants departed from the standard of care by allowing Ms. Watkins to "remain in labor once a pattern of repetitive variable decelerations appeared, followed by a prolonged variable decelerations." He opines that Dr. Seubert failed to "more timely and emergently conduct the delivery," as once he arrived the necessity for a cesarean was "clear" and needed to be done "emergently" to "prevent or minimize hypoxic injury," given the evidence of fetal distress and the fact that the fetus was premature. He opines that the failure to more-timely delivery the infant plaintiff, "resulted in her continuing to sustain a period of hypoxia."

Addressing the opinions of defendants' experts as to the diagnosis of Arthrogryposis Multiplex Congentia –Type C, Dr. Engelbert opines that at "some point during birth admission of

the infant, a diagnosis of mild arthrogryposis was considered, but never confirmed," and that subsequent neonatology attending progress notes "repeatedly stated that M.W. was suffering from chronic lung disease, arthrogryposis of unknown etiology, and neurologic compromise." He also points to notes in the record from 2008 referring to a "neurologic insult" and "brain injury," and records from other hospitals indicating a diagnosis of hypoxic ischemic encephalopathy (HIE).

In conclusion, Dr. Engelbert opines there was an "unwarranted delay in the performance of the delivery secondary to the failure to respond to evidence of fetal distress," and since the fetal heart tracings were "not reassuring," a cesarean section was needed to "prevent or minimize a hypoxic injury." He further opines that the delay in delivery "led to the fetus suffering a period of hypoxia as evidenced by the presence of repetitive variable decelerations and the low Apgar scores," and that had "M.W. been delivered emergently, the degree of hypoxia could and would have been minimized."

As to the issue of causation, plaintiff submits the expert affirmation of Dr. Adler, who is board certified in pediatrics and neurology. Dr. Adler reviewed the medical records, Dr. Seubert's deposition testimony and the affirmations of defendants' experts; he also examined the infant plaintiff on August 1, 2016 and prepared a written report. He sharply disagrees with the opinions of defendants' experts, and opines that hypoxic ischemic encephalopathy (HIE) is the "cause of all of the infant plaintiff's neurological and neurodevelopmental disabilities, and that no other cause is possible."

Dr. Adler opines that the infant plaintiff was "small for her gestational age, weighing only 1330 grams at birth," and "suffered a degree of intrauterine growth restriction." He opines that

the findings of the pathological evaluation of the placenta are "consistent with poor perfusion of the placenta . . . [which] reduces delivery of oxygen and nutrients to the fetus and can sometimes lead to intrauterine growth restriction." He states that growth restricted infants "often tolerate labor poorly because of reduced utero-placental function," and have an increased risk for "intrapartum hypoxia." He opines that "intrapartum hypoxia" occurred here as evidenced by "intrapartum fetal heart rate abnormalities, which are indicative of fetal distress." He opines that the presence of "minimal variability and repetitive variable decelerations, including a prolonged deceleration lasting five (5) minutes support the claim that M.W. sustained a period of fetal distress between 9:10 p.m. and the time of her delivery." Dr. Adler states that after delivery, M.W. was in "cardiac arrest," as she was not breathing, had a one-minute Apgar score of zero, required resuscitation and "adequate output was not restored for many minutes." After noting the findings of a head sonogram on November 21, 2007 and a CT scan of the brain on November 27, 2007, Dr. Adler points to the MRI of the brain performed on November 29, 2007 which found "multiple subependymal cysts and T1 abnormalities in the globus pallidus." He states that even though "TI abnormalities can be a sign of immature myelination as discussed in the records, it is my medical opinion that in this case, when one considers all of the imaging studies together, these T1 abnormalities were caused by hypoxia." He opines that the "brain imaging abnormalities in the deep grey structures of the brain (caudate and globus pallidi) are diagnostic of brain injury from hypoxia and ischemia," and are "seen in infants who suffer sustained hypotension as a result of severe bradycardia and/or cardiac arrest." He opines that these "imaging abnormalities" were caused by "events that occurred on or about the time of M.W.'s birth," and it "was during this time interval that her brain was "injured."

Dr. Adler opines that the records of subsequent treatment document that the infant plaintiff has "significant neurological disabilities," and those physicians "repeatedly" list the "working diagnosis" as HIE. Dr. Adler points to hospital's progress notes which state that the infant was "cyanotic at birth" (November 21, 2007 at 1:17 a.m.); admitted to the NICU for "prematurity, respiratory distress, hypertonia and intrauterine growth restriction" (November 21 at 4:00 a.m.); the infant continues to present with "hypertonia of the extremities (upper greater than lower), elevated deep tendon reflexes and clonus" and there is a "concern for HIE" (November 23, 2007). Dr. Adler states that "at some point during the birth admission," a diagnosis of mild arthrogryposis was "considered, but never confirmed." He states that on January 13, 2008, the "issue of a brain injury was raised" in a conversation with the family; and on February 27, 2008 it was noted that the infant is suffering from "chronic lung disease and neurologic insult," but arthrogryposis was not listed as the diagnosis; and genetic testing was reported as "normal."

Dr. Adler states that on March 8, 20008, the infant plaintiff was transferred to Blythdale Children's Hospital, where the "admission diagnosis included chronic respiratory failure, arthrogryposis and hypoxic ischemic encephalopathy"; "arthrogryposis" is crossed out on a subsequent note in the chart dated May 2, 2008; the diagnoses on a July 24, 2009 "Rehabilitation Medicine Outpatient Visit" are listed as chronic respiratory failure and HIE; and a September 29, 2009 "Certificate of Medical Necessity"lists the diagnoses as chronic respiratory failure and HIE. He opines that starting in April 2009, chronic respiratory failure and HIE are "primary diagnoses" on the pediatric records from Children's Physician of Westchester; an October 2010

progress note from Westchester Medical Center lists a diagnosis of Cerebral Palsy; and 2015 records from Mount Kisco Medical Group, P.C. list a diagnosis of Cerebral Palsy and quadriplegic.

Addressing the opinion of defendants' expert Dr. Brown as to the diagnosis of Arthrogryposis Multiplex Congenital-Type C, Dr. Adler opines that the "Hall article" cited by Dr. Brown is not relevant, as it relies on fetal akinesia (decreased fetal movement), and the record shows that Ms. Watkins had only one complaint of decreased fetal movement in October 2007 which was ruled out after tests; he opines that a "single episode of decreased fetal movement is not fetal akinesia." Dr. Adler further opines there is no evidence Ms. Watkins suffers from a "disorder" related to development or occurrence of arthrogryposis, as the infant plaintiff has "never been found to have a muscle or connective tissue disorder"; she does not have "mechanical limitations of movement of a type related to fetal akinesia"; and "nothing about" the pregnancy or the delivery suggests that she was at "risk" for arthrogryposis. Dr. Adler opines that the infant plaintiff does not have "tissue around the joints, disuse muscle atrophy or abnormal joint surfaces" which are the "characteristic findings" associated with Arthrogryposis Multiplex Congenital-Type C; and Dr. Brown's physical examination does not note the presence of such "key characteristics." In conclusion, Dr. Adler opines that the infant plaintiff's "disabilities, limitations and special needs are a direct result" of HIE, and that her neurological disabilities are "permanent in nature and continuing."

In response, defendants submit a reply affirmation from Dr. Brown objecting that Dr. Adler's statements and opinions "misconstrue or ignore . . .critical portions" of the medical records, "misstate" his findings and conclusions, and demonstrate a "profound lack of

understanding and knowledge of congenital anomalies and Arthrogryposis Multiplex Congentia-Type C with peripheral contractures and CNS [central nervous system] contractures." Dr. Brown objects to Dr. Adler's opinion that one "episode" of decreased fetal movement is not fetal akinesia, referring to Ms. Watkins' complaints on October 23, 2007. Dr. Brown points to Ms. Watkins deposition testimony that from that point on in her pregnancy, fetal movement "remained really slow and restricted."

Dr. Brown also objects to Dr. Adler's opinion that the infant plaintiff does not have an "increase of connective tissue around the joints . . . . characteristic findings associated with Arthrogryposis Multiplex Congentia-Type C." Dr. Brown states that Dr. Adler has "chosen either to ignore the neonatal chart or he does know what the signs and symptoms of a connective tissue deformity are." Dr. Brown opines that with "akinesia and constriction of the connective tissues of the body, it causes contractures, which are a permanent shortening or stiffening of muscles and tendons, which results in decreased movement and range of motion." He opines that the infant plaintiff was "noted to have contractures of the extremities and clenched fits upon admission to the Neonatal ICU following her birth, and on every examination thereafter," and that the diagnosis of arthrogryposis was made "at or around the time of birth, and from my reading of the neonatal chart, remained the main diagnosis along with prematurity and intrauterine growth retardation."

Objecting to Adler's opinion that the " developing fetus evidenced intrauterine growth retardation possibly related to poor placental function," Dr. Brown states that Dr. Adler "fails to appreciate" that the "placental is derived largely from the originally fertilized egg and therefore has the same genetic make-up as the fetus"; and the placental "therefore may have been defective

due to having the same genetic abnormality as the fetus had, causing her Arthrogryposis Multiplex Congenita-Type C."

Dr. Brown disputes Dr. Adler's statement that he/Dr. Brown failed to note any key characteristics of arthrogryposis. Dr. Brown points to the statement in his affirmation that his February 29, 2016 examination of the infant plaintiff "confirms" the diagnosis of Arthrogryposis Multiplex Congenita-Type C, and "reiterates" his "pertinent findings" as follows: the infant plaintiff's head is "long, narrow and cone shaped with mild occipital prominence and a flat occiput," which are "congenital anomalies known as Brachycephaly and Turricephaly," and are "clearly" seen on the photographs taken during his examination; she has "strabismus and exotropia (a sign of abnormal neural connections)" also seen on the photographs; she has a "pigeon chest deformity, a congenital abnormality which is quite marked"; her "fists were clenched and her elbows could only be extended to 120 degrees and she had clenched toes," which are "peripheral contractures" that are "key characteristics" of arthrogryposis; and she is "profoundly mentally retarded," which is "uniquely consistent with Arthrogryposis Multiplex Congenita-Type C with peripheral contractures and central nervous system dysfunction."

Dr. Brown further objects to Dr. Adler's report of his examination of the infant plaintiff. Dr. Brown states that Dr. Adler "acknowledges her microcephaly but makes no mention whatsoever of any of her congenital anomalies, not even her extremely marked Pectus Carnatum 'pigeon chest deformity,' a striking omission for a pediatric neurologists who claims to have examined her chest," and that his "only finding" as to her "extremities on examination are that they are 'non-edematous with normal pulses,' and yet he forms a clinical impression of 'spastic

quadriparesis.'" Dr. Brown opines that Dr. Adler's report "is not within acceptable standards of the medical community."

Finally, Dr. Brown notes that "arthrogryposis is a descriptive term and presents in over 400 specific conditions," and that Arthrogryposis Multiplex Congenita-Type C with peripheral contractures and central nervous system dysfunction involving profound mental retardation is "found in approximately 100 of these conditions, which individually are quite rare." He states that "each can be due to a number of different gene mutations, as reviewed in the Hall article," and it is "not surprising therefore that subsequent treating physicians such as those at Blythdale Children's Hospital attending to M.W.'s respiratory needs and other supportive care may be unaware of these conditions causing Arthrogryposis, which includes neurological damage and mental retardation."

Based on the foregoing, summary judgment is not warranted. Given the experts' separate and distinct opinions as to the diagnoses of Arthrogryposis Multiplex Congenital-Type C and hypoxic ischemic encephalopathy (HIE), each of which is adequately supported by the record, it is not possible to rule out one condition over the other so as to determine the issue as a matter of law. Plaintiff's and defendants' experts opine that each condition alone is solely responsible for the infant plaintiff injuries. The experts also sharply disagree as to whether defendants timely and appropriately recognized and responded to the sign of fetal distress; whether the cesarean section was timely performed; whether Ms. Watkins should have had general anesthesia as opposed to an epidural; and whether the infant plaintiff's physical, neurological, intellectual and developmental disabilities are related to the genetic disorder Arthrogryposis Multiplex Congenital-Type C that allegedly developed *in utero*, or were caused by HIE that allegedly

occurred during delivery and birth. In view of these conflicting expert opinions that are adequately supported by the record, triable issue of material fact are raised as to the alleged departures and causation, and defendants are not entitled to summary judgment. See Frye v. Montefiore Medical Center, 70 AD15 (1st Dept 2009); Cruz v. St. Barnabas Hospital, 50 AD3d 382 (1s Dept 2008).

Accordingly, it is

ORDERED that defendants' motion for summary judgment is denied; and it is further

ORDERED that the pre-trial conference previously scheduled for October 31, 2019 is changed to November 14, 2019 at 11:00 a.m. in Part 11, Room 351, 60 Centre Street.

DATED: October 16, 2019

ENTER:

J.S.C.

HON. JOAN A. MADDEN
J.S.C.