

Supreme Court of the State of New York
Appellate Division: Second Judicial Department

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H/ct/prt

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Argued - June 24, 2011

REINALDO E. RIVERA, J.P.
JOSEPH COVELLO
ANITA R. FLORIO
PLUMMER E. LOTT, JJ.

2010-00962
2010-02506

OPINION & ORDER

Jacob Lugo, etc., et al., appellants, v New York City
Health and Hospitals Corporation, etc., respondent.

(Index No. 37871/04)

APPEALS by the plaintiffs in an action, inter alia, to recover damages for medical malpractice, etc., (1) from an order of the Supreme Court (Allen Hurkin-Torres, J.), entered December 15, 2009, in Kings County, which, after a hearing, granted that branch of the defendant's motion which was for summary judgment dismissing the complaint, and (2) from a judgment of the same court entered February 1, 2010, which, upon the order, is in favor of the defendant and against them dismissing the complaint.

Fitzgerald & Fitzgerald, P.C., Yonkers, N.Y. (John E. Fitzgerald, John M. Daly, Eugene S. R. Pagano, Mitchell L. Gittin, and John R. Langdell of counsel), for appellants.

Michael A. Cardozo, Corporation Counsel, New York, N.Y. (Edward F.X. Hart and Jane L. Gordon of counsel), for respondent.

COVELLO, J.

Introduction

New York courts apply the rule of *Frye v United States* (293 F 1013) that expert testimony based on scientific principles or procedures is admissible, but only after a principle or

procedure has gained general acceptance in its specified field. In this medical malpractice action, the principal question presented on this appeal is whether the Supreme Court, in applying the *Frye* test, properly determined that the opinion testimony of the plaintiffs' experts that the infant plaintiff's brain injuries were caused by an episode of severe neonatal hypoglycemia lasting 81 minutes was inadmissible. For the reasons set forth below, we answer this question in the negative.

Factual and Procedural Background

Factual Background

In 2001, the plaintiff Brenda Almodovar (hereinafter the mother), who was pregnant with the infant plaintiff, Jacob Lugo, began receiving prenatal care at Woodhull Hospital (hereinafter Woodhull), a facility owned and operated by the defendant. On August 11, 2001, at 31 weeks of gestation, the mother was admitted to Woodhull for signs of preterm labor. During that admission, her blood glucose level was measured at 26 mg/dL, an abnormally low level, but was subsequently measured at a normal level. The mother was discharged on August 13, 2001.

On September 2, 2001, at 34 weeks of gestation, the mother, who had a history of seizures dating back to childhood, was brought to Woodhull by emergency medical services (hereinafter EMS) personnel after experiencing a grand mal seizure. On that date, she was evaluated but not admitted.

On October 5, 2001, the mother gave birth to Lugo at Woodhull by normal spontaneous vaginal delivery at 11:39 A.M. Lugo's Apgar scores, 9 at one minute, and 9 at five minutes, were "excellent," and he initially appeared normal. However, by the time Lugo was 40 minutes old, he was experiencing tremors and, at 12:25 P.M., he was admitted to the neonatal intensive care unit.

According to the deposition testimony of Dr. Frantz Brea, the director of neonatology at Woodhull, tremors are a sign of hypoglycemia¹ in a newborn. At 12:25 P.M., when Lugo was admitted to the neonatal intensive care unit, his blood glucose level was measured, through a "heel stick" test, at less than 20 mg/dL, and laboratory testing of blood drawn from Lugo at that time later measured a glucose level of 3 mg/dL. According to Dr. Brea, a normal glucose level for an infant approximately 40 minutes old is about 40 mg/dL. Lugo was given a "glucose IV push" and a glucose infusion, and at 1:00 P.M., his blood glucose level was measured at 71 mg/dL, within normal limits.

¹Hypoglycemia means low blood sugar.

Thereafter, Lugo's blood glucose level remained within normal limits until he was discharged from Woodhull on October 7, 2001.

In 2002, Lugo was referred to Woodhull for evaluation due to his delays in reaching certain developmental milestones. On April 29, 2003, Lugo underwent a brain magnetic resonance imaging (hereinafter MRI) examination at Brookdale Hospital, and the resulting MRI report set forth a finding of "non-specific white matter loss in parietal and occipital lobes with dilation of the occipital horn . . . which suggests periventricular leukomalacia, as can be seen with perinatal ischemia."² Ultimately, Lugo was diagnosed with cerebral palsy (spastic diplegia type).

Commencement of this Action

Lugo, by his mother, and the mother, suing derivatively, commenced this action, inter alia, to recover damages for medical malpractice. In their verified bill of particulars, the plaintiffs alleged that the defendant had departed from good and accepted medical practice by, among other things, failing to timely diagnose and treat the hypoglycemia of both the mother and Lugo. They alleged that Lugo's hypoglycemia had caused, among other things, his brain damage and cerebral palsy.

The Defendant's Motion for Summary Judgment or a *Frye* Hearing

By notice of motion dated May 15, 2007, the defendant moved for summary judgment dismissing the complaint or, in the alternative, for a *Frye* hearing in the event that the plaintiffs, in opposition to the motion, proffered a sworn statement from an expert opining that Lugo's injuries were caused by the "possible transient episode" of maternal hypoglycemia on August 11, 2001, or the "transient episode" of hypoglycemia on October 5, 2001. As relevant here, the defendant supported its motion with the expert affirmation of Dr. Armando Grassi, who opined that Lugo's episode of neonatal hypoglycemia did not cause his alleged injuries. According to Dr. Grassi, the white matter loss shown on Lugo's April 2003 MRI was in the periventricular area and was a typical lesion resulting from a decrease in oxygenation or perfusion to the brain. In contrast, he affirmed, lesions typical of hypoglycemia are "diffuse lesions" in the brain and are not found in the periventricular area. Dr. Grassi opined that Lugo's brain injury, as depicted on his MRI, was a result

²According to expert testimony presented in this matter, perinatal ischemia—in the context of the instant action—is a decrease in the flow of blood and/or oxygen to the brain of a fetus.

of decreased oxygenation to his brain at 32-34 weeks gestation, and was not caused by the “transient hypoglycemic episode” at his birth. Dr. Grassi asserted that it was not accepted in the medical profession that “a short and promptly treated” episode of hypoglycemia in a newborn could cause brain damage in the periventricular area, as seen on Lugo’s MRI film, and that Dr. Grassi had “never heard or read of a single case of periventricular leukomalacia caused by hypoglycemia.”

In opposition, the plaintiffs argued, inter alia, that summary judgment was improper because there were triable issues of fact concerning, among other things, the nature and cause of Lugo’s periventricular leukomalacia (hereinafter PVL) and cerebral palsy. As relevant here, they submitted the expert affirmation of Dr. Rosario Trifiletti. Dr. Trifiletti opined that Lugo had been born with “profound hypoglycemia,” and that the delay in diagnosis and treatment from 11:39 A.M. to 1:00 P.M. was a substantial factor in causing his brain damage. Dr. Trifiletti disagreed with Dr. Grassi’s conclusion that the mother’s seizure had caused Lugo’s brain injuries. According to Dr. Trifiletti, Lugo’s normal appearance and good Apgar scores at birth, and the delay of the onset of his tremors until approximately 40 minutes after birth, were consistent with depletion of glucose stores after birth rather than a primary hypoxic injury. Dr. Trifiletti characterized Lugo’s post-birth tremors as “subtle seizures” as defined in Volpe’s *Neurology of the Newborn* (hereinafter the Volpe textbook), and he opined that Lugo’s “tremors” or “subtle seizures” had been caused by his profound hypoglycemia at birth.

In Dr. Trifiletti’s opinion, Lugo’s MRI report was “essentially accurate” in its finding of PVL about the posterior (occipital) horns of the lateral ventricles, and he disagreed with Dr. Grassi’s assertion that the pattern of injury it depicted was not characteristic of lesions caused by hypoglycemia. Dr. Trifiletti affirmed that there is “substantial overlap” in the lesions resulting from hypoxia and from hypoglycemic injury. Citing Arie L. Alkalay, *et al.*, *Brain Imaging Findings in Neonatal Hypoglycemia: Case Report and Review of 23 Cases*, 44 *Clin Pediatr* 783-790 (2005), an article published in the November/December 2005 edition of the journal *Clinical Pediatrics*, Dr. Trifiletti asserted that there was a tendency towards occipital injury (as was seen in Lugo’s case) with hypoglycemia. He saw nothing on Lugo’s MRI film that excluded hypoglycemia as the etiology of the “obvious white matter loss and occipital horn dilation” and, in his experience of reviewing brain MRIs as part of his clinical practice over the years, he had seen “similar patterns of brain injury in comparable instances of perinatal hypoglycemia.”

In its reply papers, the defendant proffered the expert affirmation of Dr. Steven Pavlakis. Dr. Pavlakis affirmed, among other things, that after performing a search on “Pub Med,” September 13, 2011

he found no evidence that the white matter damage seen on Lugo's MRI film could be caused by "short lived transient hypoglycemia," and that it was not generally accepted that a period of transient neonatal hypoglycemia such as that suffered by Lugo could cause his clinical outcome. Dr. Pavlakis disagreed with Dr. Trifiletti's opinion that Lugo had suffered from "subtle seizures" as defined in the Volpe textbook, and he asserted that the Alkalay article cited by Dr. Trifiletti did not discuss any patients who had experienced an episode of hypoglycemia similar to that experienced by Lugo.

In an order dated November 5, 2007, the Supreme Court granted that branch of the defendant's motion which was for a *Frye* hearing and held in abeyance that branch of the defendant's motion which was for summary judgment dismissing the complaint. The Supreme Court determined that the plaintiffs' experts had provided "scant reference" to medical or scientific literature to support their opinions, and that a *Frye* hearing should be held to determine whether their deductions were based on principles which were sufficiently established to have gained general acceptance.

The *Frye* Hearing

After additional motion practice not at issue on this appeal, the Supreme Court conducted a *Frye* hearing in April and May 2009. The first expert to testify for the plaintiffs was Dr. Michael Katz, a private practitioner who was board-certified in pediatric neurology and neurodevelopmental disabilities. As background, Dr. Katz testified that the normal blood glucose range for newborns is between 40 and 60 mg/dL, that a level below 40 mg/dL is considered hypoglycemia, that Lugo's measured blood glucose level of 3 mg/dL was "[p]rofoundly low," and that hypoglycemia is a medical emergency which must be treated immediately because it is a toxic state which causes brain damage. Dr. Katz's working hypothesis was that Lugo's blood glucose level was 3 mg/dL from 11:39 A.M., when he was born, until 1:00 P.M., when his blood sugar was normalized. In Dr. Katz's opinion, Lugo's brain injury was caused by this episode of hypoglycemia.

Dr. Katz testified that his opinion that an episode of hypoglycemia at a level of 3 mg/dL lasting 1 hour and 21 minutes could cause neurologic damage of the type sustained by Lugo was based on the following generally accepted scientific principles: (1) hypoglycemia causes brain injury; (2) certain infants are more susceptible than others to neurologic injury secondary to hypoglycemia; (3) hypoglycemia is a toxic and dangerous state; and (4) there is no safe level of hypoglycemia. Dr. Katz testified that his opinion that hypoglycemia caused Lugo's brain injury was based on the fact that Lugo's MRI film showed a brain injury, that Lugo had suffered from a period of proven and profound hypoglycemia, and that there appeared to be nothing else in the record or

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around the time of Lugo's birth suggesting that anything besides hypoglycemia caused Lugo's injury. Dr. Katz did not believe that the mother's seizure at 34 weeks of gestation had injured Lugo in the nature of a hypoxic ischemic event resulting in brain MRI abnormalities because Dr. Katz had difficulty visualizing a mechanism by which a seizure during pregnancy could cause a decrease in blood flow in the infant's brain.

Dr. Katz addressed, at length, the medical literature upon which his theory of causation was based. He noted that the Volpe textbook indicated that hypoglycemia causes brain injury and brain damage. In addition, the Volpe textbook discussed neuropathic studies indicating that hypoglycemia is a precedent of PVL and that both perinatal ischemia and hypoglycemia could cause an identical brain injury: namely, PVL. Dr. Katz explained that PVL is an injury to the white brain matter in the distribution around the ventricles.

Next, Dr. Katz discussed Arie L. Alkalay, *et al.*, *Plasma Glucose Concentrations in Profound Neonatal Hypoglycemia*, 45 Clin Pediatr 550 (2006), an article published in the July 2006 edition of the journal Clinical Pediatrics (hereinafter the Alkalay article). He explained that the authors had compiled 16 different studies in an attempt to define low thresholds of plasma glucose concentrations constituting treatable or profound hypoglycemia, and they had concluded that plasma glucose levels of less than 25 mg/dL of several hours' duration may increase the relative risk for adverse neurologic outcome. Dr. Katz testified that a plasma glucose level is essentially the same as a whole blood glucose level, and that a plasma glucose level of 25 mg/dL is "much higher" than a whole blood glucose level of 3 mg/dL.

Dr. Katz acknowledged that one of the studies reviewed in the Alkalay article, Anne Kinnala, *et al.*, *Cerebral Magnetic Resonance Imaging and Ultrasonography Findings After Neonatal Hypoglycemia*, 103 Pediatrics 724-729 (1999) (hereinafter the Kinnala article), published in the April 1999 edition of the journal Pediatrics, had excluded infants who had experienced only one episode of hypoglycemia before six hours of age. However, he did not believe that this fact affected the overall conclusion of the Alkalay article, which had examined 15 other studies besides the Kinnala article. Dr. Katz noted that the Kinnala article included a patient who had shown evidence of neurologic injury on an MRI after experiencing a hypoglycemic episode lasting two hours where the lowest glucose level was 32 mg/dL, a level "dramatically" higher than Lugo's glucose level of 3 mg/dL.

Finally, Dr. Katz discussed Burns, *et al.*, *Patterns of Cerebral Injury and Neurodevelopmental Outcomes After Symptomatic Neonatal Hypoglycemia*, 122 Pediatrics 65 (September 13, 2011)

(2008) (hereinafter the Burns article), an article published in the journal Pediatrics in 2008. He explained that the authors had studied 35 term infants and had attempted to limit their study to symptomatic neonatal hypoglycemic patients, meaning those who had suffered from tremors, and to exclude brain injuries from other causes such as hypoxic ischemic encephalopathy. Sixty-three percent of the patients studied in the Burns article had experienced only one episode of hypoglycemia which had resolved promptly with treatment, and 94% of all of the patients studied had shown evidence of MRI abnormalities. The article also examined neurodevelopmental outcomes and determined that six of the subjects had developed cerebral palsy and three had developed mild motor delays.

Dr. Katz acknowledged that it was “unclear” exactly what duration and level of hypoglycemia causes neurologic injury in humans, and that there was no specific article, report, or study stating, in unambiguous terms, that an episode of hypoglycemia lasting 1 hour and 21 minutes at a level of 3 mg/dL had caused, or could cause, neonatal brain injury. However, he testified that there was not a “whole lot” of medical literature on hypoglycemia because “it is really an impossible task to prospectively look at hypoglycemia in children.” Dr. Katz also acknowledged that there are a number of potential causes of PVL in addition to hypoglycemia, including hypoxic ischemia, and that it was possible that Lugo had sustained his injury during the mother’s seizure and been asymptomatic at the time of birth. Dr. Katz stressed, however, that Lugo had been symptomatic for hypoglycemia, that Lugo’s MRI results were consistent with hypoglycemia, that the medical literature indicates that low blood sugar causes brain damage, and that his opinion was based on the “confluence” of the medical information he had discussed.

Dr. Robert Peyster, the chief of neuroradiology at Stony Brook University Medical Center, also testified for the plaintiffs. Dr. Peyster explained that PVL is not a specific term, but, rather, refers to damage to the deep white brain matter next to the ventricles that appears as an abnormality on a CT scan or an MRI, and that PVL can be caused by both hypoglycemia and perinatal asphyxia. At the hearing, Dr. Peyster reviewed Lugo’s MRI films in detail and testified that they depicted PVL. Based on Lugo’s measured profound hypoglycemia and high Apgar scores, Dr. Peyster opined that the cause of Lugo’s PVL was his episode of hypoglycemia and not perinatal asphyxia. Although he acknowledged that a seizure during pregnancy could potentially be severe enough to damage the brain of a fetus by reducing blood flow across the placenta, he was unaware of any reported cases where a child who had experienced such an event had received normal Apgar scores at birth.

Like Dr. Katz, Dr. Peyster addressed relevant medical literature at length. He agreed with Dr. Katz that the Volpe textbook supported the position that hypoglycemia leads to PVL. Dr. Peyster testified that the Burns article was significant because it was the largest series to date addressing MRI findings and other issues in neonatal hypoglycemia, because it had excluded patients who might have had hypoxic ischemia, and because 94% of the patients had shown white matter abnormalities on their MRI brain scans. He considered the Burns article to be a “good paper” and the best available article addressing generalized principles regarding hypoglycemia and injuries to infants. However, Dr. Peyster conceded that the Burns article had not been designed to test the relationship between the severity or duration of hypoglycemia and neurodevelopmental outcomes and had not found any such relationship, and that the subjects studied in the Burns article had received MRI brain scans at a much earlier age than Lugo had.

Dr. Peyster acknowledged that he had not located any articles or reports specifically addressing a patient who had experienced an episode of hypoglycemia of the same level and duration as Lugo’s episode, but he testified that this fact did not change his opinion that Lugo’s injuries were caused by hypoglycemia because the literature he had reviewed had studied cases representing a wide range of duration times, Lugo had PVL, and Lugo’s glucose level had been measured at close to zero. Dr. Peyster testified that there was no threshold of duration and severity, generally accepted by most physicians, below which hypoglycemia could *not* cause abnormalities like those seen on Lugo’s MRI.

After the plaintiffs’ experts testified, the defendant presented the testimony of Dr. Caren Jahre, a private practitioner and an assistant professor of radiology at New York University School of Medicine. Dr. Jahre testified that Lugo’s MRI films depicted a “classic pattern” of PVL seen in the context of hypoxic encephalopathy or perinatal ischemia at 26 to 34 weeks of gestation, and that the literature she had reviewed did not associate this specific pattern with neonatal hypoglycemia. According to Dr. Jahre, medical literature indicated that the “hallmark” of brain damage resulting from hypoglycemia is cortical involvement, and some of that literature reported white matter damage caused by hypoglycemia either “out in the periphery” or against the ventricles, but limited to certain areas. In contrast, according to Dr. Jahre, the brain damage on Lugo’s MRI film had a diffuse pattern tracking along the ventricles and no cortical involvement. However, she acknowledged that she and Dr. Peyster disagreed on the precise appearance of the pattern depicted on Lugo’s MRI film.

In Dr. Jahre’s opinion, the Burns article was flawed because, based upon the medical records of the patients it had studied, the authors had failed to exclude patients who had suffered from

health issues other than neonatal hypoglycemia, including hypoxic ischemic encephalopathy. Additionally, according to Dr. Jahre, none of the MRI images in any of the literature discussed at the *Frye* hearing looked “anything close to what [Lugo’s] brain looks like.”

The defendant also presented the testimony of Dr. Steven Pavlakis, a professor of neurology and pediatrics at Mt. Sinai School of Medicine and the director of pediatric neurology at Maimonides Hospital. Dr. Pavlakis had performed a search and had found no literature on MRI changes resulting from hypoglycemia in newborns lasting less than two hours. He agreed that hypoglycemia can cause MRI abnormalities, that severe hypoglycemia can cause brain damage, and that Lugo’s measured glucose level of 3 mg/dL was very low. In addition, he acknowledged that the scientific community does not recognize any specific level or duration of hypoglycemia which would *not* cause brain damage and that it was a generally accepted medical principle that individual susceptibility to toxic states varies.

According to Dr. Pavlakis, it was “relatively common” for newborns to have hypoglycemia, low blood sugar was a common cause of tremors such as those experienced by Lugo, and such tremors were distinguishable from seizures and did not correlate to an underlying condition or particular outcome. Based on Lugo’s normal appearance at birth and recovery with sugar infusions, Dr. Pavlakis did not believe that his episode of hypoglycemia had caused his brain damage. Dr. Pavlakis also excluded hypoglycemia as a cause of Lugo’s injuries because “there’s no case like him” of which Dr. Pavlakis was aware in the literature or in his practice.

According to Dr. Pavlakis, decreased oxygen or blood flow to a fetus between the ages of 28 to 40 weeks is the cause of PVL in “99.99 percent” of cases. He testified that PVL could be caused by anything that decreases oxygen or blood supply to a fetus under 40 weeks of gestation, including, hypothetically, a seizure like the one experienced by the mother. However, like the plaintiffs’ expert Dr. Katz, Dr. Pavlakis was unaware of any instance in which such a seizure had actually resulted in PVL, and he could not opine, to a reasonable degree of medical certainty, that Lugo’s PVL had been caused by the mother’s seizure.

When asked whether the positions taken in the Burns article were “generally accepted in the scientific community,” Dr. Pavlakis responded by asserting that Lugo was not like the patients in the Burns article, who had “a lot of other issues going on,” and had not experienced a short episode of hypoglycemia lasting even 1½ hours. Like Dr. Jahre, Dr. Pavlakis testified that the Burns article had not been entirely successful in selecting a group of patients suffering purely from hypoglycemia, but he opined that the authors had done a good job of setting up their study and that he was not sure

if a better study was possible.

Dr. Pavlakis testified that the medical literature discussed at the hearing, when considered in the aggregate, did not demonstrate that a child like Lugo who had a glucose level of 3 mg/dL for 1 hour and 21 minutes would develop PVL as a result, since none of the patients discussed in the literature had experienced a relatively short period of hypoglycemia before being discharged from the hospital without further problems. Therefore, according to Dr. Pavlakis, the theory of causation offered by the plaintiffs' experts was not scientifically accepted.

A running theme throughout the *Frye* hearing was whether the experts considered the medical literature they had reviewed to be "authoritative." Although both Dr. Katz and Dr. Peyster testified that they did not consider any of the literature they had discussed to be "authoritative," Dr. Katz testified that the Volpe textbook and the articles he had addressed were the sources he would consult for the current science in the areas discussed at the hearing. Dr. Peyster testified that he did not consider *any* medical literature, including his own book, to be "authoritative" because that term implied that everything in the article or study was correct and was not subject to any further changes. Dr. Peyster's reluctance to apply this label to medical literature was echoed by the defendant's expert Dr. Jahre, who agreed that this term was not used frequently to describe medical literature and that doctors relied upon articles not considered to be "authoritative" to assess the state of the science.

The Order and the Judgment Dismissing the Complaint

In an order entered December 15, 2009, the Supreme Court granted that branch of the defendant's motion which was for summary judgment dismissing the complaint after concluding that the plaintiffs' expert testimony regarding causation was inadmissible. In the order, the Supreme Court framed the issues to be resolved as: (1) whether the scientific community generally accepts that a short episode of hypoglycemia can cause PVL such as that shown on Lugo's MRI; and (2) whether the plaintiffs' experts could reasonably opine that Lugo's episode of hypoglycemia actually caused his injury. With respect to the first issue, the Supreme Court concluded that the plaintiffs had failed to demonstrate that it is generally accepted that hypoglycemia can cause PVL "as suffered by [Lugo]." In arriving at this determination, the Court highlighted the testimony of the defendant's experts that the patients studied in the Burns article could have suffered from hypoxic ischemic encephalopathy, and noted that the Volpe textbook stated that the topography of injuries associated with PVL differed "somewhat" from that observed with hypoxic ischemic injury. In addition, the Supreme Court concluded that Dr. Peyster's inability to label any of the medical literature he had

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reviewed as authoritative ran “counter” to a conclusion that the findings set forth therein were generally accepted in the scientific community.

With respect to the second issue, the Supreme Court asserted that “even if it were generally accepted that a hypoglycemic episode could cause [PVL], [the] plaintiff[s’] evidence fails to demonstrate a factual issue as to whether the hypoglycemic episode suffered by [Lugo] caused his brain injury.” Addressing the factors Dr. Katz cited in support of his conclusion that Lugo’s episode of hypoglycemia caused his injury, the Supreme Court concluded that, based on the testimony of the plaintiffs’ experts, although Lugo’s MRI did not exclude hypoglycemia as the cause of his injury, it also did not rule out other possible causes, such as hypoxia or ischemia. In addition, the Supreme Court concluded that nothing in the plaintiffs’ evidence “address[ed]” Dr. Pavlakis’s testimony that hypoxia and/or ischemia are the predominant causes of PVL. The Supreme Court noted that none of the articles relied upon by the plaintiffs’ experts addressed an episode of hypoglycemia lasting 1 hour and 21 minutes, like that suffered by Lugo, and that Dr. Katz had conceded that the question of what duration and severity of blood glucose levels caused neurologic injury in humans is unclear. The Supreme Court acknowledged that, according to the Volpe textbook, the presence of seizures is a major indicator that an episode of hypoglycemia will result in neurological damage, but it rejected the assertion of the plaintiff’s expert Dr. Trifiletti, set forth in his affirmation, that Lugo’s post-birth tremors were consistent with subtle seizures as defined in the Volpe textbook, and that the seizures or tremors constituted evidence that the hypoglycemia caused neurological damage.

Addressing Dr. Katz’s testimony that it was generally accepted that susceptibility to brain injury at a certain blood sugar level varies from individual to individual, the Supreme Court determined that Dr. Katz had provided “no indication” that Lugo was particularly susceptible to suffering such an injury from hypoglycemia. Additionally, the Supreme Court reasoned that although Dr. Katz testified that hypoglycemia is a toxic state that requires treatment regardless of the duration or blood sugar level, that testimony was inadequate to demonstrate causation in this matter. Finally, in response to Dr. Katz’s testimony that there were no other possible causes of Lugo’s injury, the Supreme Court noted Dr. Katz’s concession that there were other possible causes of PVL, and that it was possible for Lugo to have been born with normal Apgar scores if the injury occurred in utero.

Based on the foregoing analysis, the Supreme Court concluded that the plaintiffs’ experts had failed to demonstrate a foundation for their opinion that Lugo’s episode of hypoglycemia caused his injury “in light of the evidence that perinatal ischemia or hypoxia is the overwhelming cause of [PVL].”

“At best, even if [the] plaintiff[s]’ experts have raised the possibility that hypoglycemia caused his injury, their testimony fails to sufficiently rule out other more likely possible causes, such as perinatal ischemia or hypoxia. It cannot be said, therefore, that [Lugo’s] injury was, more likely than not, caused by the episode of hypoglycemia.”

Thus, the Supreme Court reasoned that a jury verdict in favor of the plaintiffs would be “nothing more than speculation and guesswork,” and the defendant was entitled to summary judgment dismissing the complaint because the plaintiffs had failed to raise a triable issue of fact regarding causation.

In a judgment entered February 1, 2010, upon the foregoing order, the Supreme Court dismissed the complaint. For the reasons that follow, we reverse the judgment.

Discussion

The Frye Test

In determining the admissibility of expert testimony, New York follows the rule of *Frye v United States* (293 F 1013) “that expert testimony based on scientific principles or procedures is admissible but only after a principle or procedure has ‘gained general acceptance’ in its specified field” (*People v Wesley*, 83 NY2d 417, 422, quoting *Frye v United States*, 293 F at 1014; see *People v Wernick*, 89 NY2d 111, 115; *Lipschitz v Stein*, 65 AD3d 573, 575; *Nonnon v City of New York*, 32 AD3d 91, 101, *affd on other grounds* 9 NY3d 825; *Zito v Zabarsky*, 28 AD3d 42, 44; see also *Giordano v Market Am., Inc.*, 15 NY3d 590, 601). In *Frye*, the United States Court of Appeals for the District of Columbia Circuit concluded that expert testimony as to the results of a “systolic blood pressure deception test” was inadmissible because the test had not yet gained general acceptance and scientific recognition among physiological and psychological authorities (*Frye v United States*, 293 F at 1014). In so concluding, the *Frye* court articulated the following holding concerning expert opinion testimony based upon deductive reasoning:

“Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs” (*id.*).

opinion and does not examine whether the expert's conclusion is sound. "*Frye* is not concerned with the reliability of a certain expert's conclusions, but instead with 'whether the experts' deductions are based on principles that are sufficiently established to have gained general acceptance as reliable'" (*Nonnon v City of New York*, 32 AD3d at 103, quoting *Marsh v Smyth*, 12 AD3d 307, 308; see *Lipschitz v Stein*, 65 AD3d at 576; *Alston v Sunharbor Manor, LLC*, 48 AD3d 600, 602; *DieJoia v Gacioch*, 42 AD3d 977, 979; see also *Ellis v Eng*, 70 AD3d 887, 892). Put another way, "[t]he court's job is not to decide who is right and who is wrong, but rather to decide whether or not there is sufficient scientific support for the expert's theory" (*Gallegos v Elite Model Mgt. Corp.*, 195 Misc 2d 223, 225). "[G]eneral acceptance does not necessarily mean that a majority of the scientists involved subscribe to the conclusion. Rather it means that those espousing the theory or opinion have followed generally accepted scientific principles and methodology in evaluating clinical data to reach their conclusions" (*Zito v Zabarsky*, 28 AD3d at 44, quoting *Beck v Warner-Lambert Co.*, 2002 NY Slip Op 40431[U], *6-7).

Thus, the limited purpose of the *Frye* test is to ascertain whether the expert's conclusion is based upon accepted scientific principles, rather than simply the expert's own unsupported beliefs (see *DieJoia v Gacioch*, 42 AD3d at 980; *Zito v Zabarsky*, 28 AD3d at 46; see also *Rowe v Fisher*, 82 AD3d 490, 491). As Justice Catterson of the Appellate Division, First Department, stated in his concurrence in *Styles v General Motors Corp.* (20 AD3d 338), "[t]he *Frye* 'general acceptance' test is intended to protect[] juries from being misled by expert opinions that may be couched in formidable scientific terminology but that are based on fanciful theories" (*id.* at 342 [internal quotation marks omitted]). Similarly, as stated by Justice Saxe of the Appellate Division, First Department, in his concurrence in *Marsh v Smyth* (12 AD3d 307), "[t]he appropriate question for the court at . . . a [*Frye*] hearing is the somewhat limited question of whether the proffered expert opinion properly relates existing data, studies or literature to the plaintiff's situation, or whether, instead, it is 'connected to existing data only by the ipse dixit of the expert'" (*id.* at 312, quoting *General Elec. Co. v Joiner*, 522 US 136, 146).

Since 1923, when *Frye* was decided, New York courts have applied the *Frye* test to the results of scientific testing or measurement procedures (see e.g. *People v Angelo*, 88 NY2d 217 [polygraph test results]; *People v Wesley*, 83 NY2d 417 [DNA profiling evidence]; *People v Middleton*, 54 NY2d 42 [bite mark identification procedure]; *People v Magri*, 3 NY2d 562 [use of radar device to measure speed]; *Styles v General Motors Corp.*, 20 AD3d 338 [procedure combining two separate automobile roof-stress tests]). In addition, the *Frye* test has been applied to assess the

reliability of psychological or physiological theories or syndromes (*see e.g. People v LeGrand*, 8 NY3d 449 [expert testimony on the reliability of eyewitness identifications]; *People v Wernick*, 89 NY2d 111 [neonaticide syndrome]; *People v Taylor*, 75 NY2d 277 [rape trauma syndrome]; *Oppenheim v United Charities of N.Y.*, 266 AD2d 116 [multiple chemical sensitivity syndrome]).

New York courts have also applied the *Frye* test to assess the reliability of an expert's theory of causation in a particular case. For this category of expert opinion testimony, "it is not necessary 'that the underlying support for the theory of causation consist of cases or studies considering circumstances exactly parallel to those under consideration in the litigation. It is sufficient if a synthesis of various studies or cases reasonably permits the conclusion reached by the plaintiff's expert'" (*Zito v Zabarsky*, 28 AD3d at 44, quoting *Marsh v Smyth*, 12 AD3d at 312-313 [Saxe, J., concurring]; *see DieJoia v Gacioch*, 42 AD3d at 979). "The fact that there [is] no textual authority directly on point to support the [expert's] opinion is relevant only to the weight to be given the testimony, but does not preclude its admissibility" (*Zito v Zabarsky*, 28 AD3d at 46; *see DieJoia v Gacioch*, 42 AD3d at 979).

Accordingly, this Court has affirmed the preclusion of expert testimony as to causation in circumstances where there was a complete absence of any literature or studies supporting the particular causation theory espoused by the expert. For example, in *Cumberbatch v Blanchette* (35 AD3d 341), the plaintiff's expert could cite to no relevant scientific data or studies to support his causation theory that fetal distress resulting from the compression of the infant plaintiff's head due to labor contractions, augmented by Pitocin, resulted in ischemia, which, in turn, resulted in an infarction, and he could cite to no instance when this type of injury had previously occurred in that manner (*id.* at 342). Thus, this Court concluded that the opinion of the plaintiff's expert was scientifically unreliable (*id.* at 342-343). Similarly, in *Lewin v County of Suffolk* (18 AD3d 621), the plaintiffs' experts conceded that no scientific organization or national board has expressly recognized a causal relationship between in utero exposure to the pesticide Malathion and birth defects, and the peer-reviewed scientific articles and textbooks relied upon by the plaintiffs' experts did not establish the existence of such a relationship (*id.* at 622). Under those circumstances, this Court concluded that the methodology employed by the plaintiffs' experts in correlating such exposure to birth defects was "fundamentally speculative" and that the Supreme Court had properly precluded the plaintiffs' experts from testifying (*id.*). And in *Hooks v Court St. Med., P.C.* (15 AD3d 544), the plaintiff's expert could not cite to any relevant scientific data or studies showing a causal link between the misuse of an electric muscle-stimulating unit and glossopharyngeal neuralgia to support his theory

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that the improper placement of electrodes of an electrical muscle-stimulating unit on the anterior neck of a patient can cause permanent nerve damage, and he could cite to no instance when that type of injury had previously occurred in that manner (*id.* at 545). Accordingly, this Court determined that the expert's opinion was scientifically unreliable (*id.*).

Standing in sharp contrast are cases in which the expert's opinion satisfied the *Frye* test because it was deduced from generally accepted scientific principles and supported by existing data or literature, although the expert could not point to a case or study involving circumstances exactly parallel to those at issue in the litigation to support his or her theory of causation. For instance, in *DieJoia v Gacioch* (42 AD3d 977), the Appellate Division, Fourth Department, concluded that the Supreme Court had applied the *Frye* test too restrictively in precluding the plaintiff's experts from testifying that a cardiac catheterization in the plaintiff's groin was the cause of the plaintiff's aortic thrombosis, which led to an acute spinal cord infarct and paralysis (*id.* at 977-978). Although the experts did not produce medical literature documenting a prior case study in which cardiac catheterization through the groin was the cause of aortic thrombosis that led to an acute spinal cord infarct and paralysis or linking a cardiac catheterization in the groin to these injuries, the conclusions of the plaintiff's experts were nonetheless deemed admissible under *Frye* because they were based on accepted scientific principles involving medicine and the vascular system and were not based solely upon the experts' own unsupported beliefs (*id.* at 979-980). Similarly, in *Zito v Zabarsky* (28 AD3d 42), the opinion testimony of the plaintiff's expert that there was a causal connection between an allegedly excessive dose of Zocor, a cholesterol-lowering drug, and the onset of polymyositis, was precluded by the Supreme Court, which concluded that the *Frye* test could not be satisfied without medical literature expressly reporting a connection between an excessive dose of Zocor and the onset of the disease (*id.* at 44-45). This Court concluded that the Supreme Court's application of the *Frye* test was "overly restrictive" because the plaintiff's experts had supported their theory of a causal nexus between an excessive dose of Zocor and polymyositis with generally accepted scientific principles and existing data, including a case study documenting a patient who had been diagnosed with polymyositis after being prescribed a generic form of Zocor at a dosage different than that prescribed to the plaintiff (*id.* at 45). This Court held that the theory of causation of the plaintiff's experts "was based upon more than theoretical speculation, or a scientific 'hunch,'" and that the lack of textual authority directly on point pertained to the weight to be given to the experts' testimony, but did not preclude its admissibility (*id.* at 46).

Here, too, the plaintiffs demonstrated that their experts' theory of causation was based

upon generally accepted scientific principles, as was their burden (*see Del Maestro v Grecco*, 16 AD3d 364), and in concluding that this opinion testimony was inadmissible, the Supreme Court applied the Frye test too restrictively. At the *Frye* hearing, the plaintiffs' expert Dr. Katz explained that his conclusion that an episode of hypoglycemia lasting 81 minutes at a level of 3 mg/dL could cause neurologic damage of the type sustained by Lugo, i.e., PVL, was based on several generally accepted scientific principles: namely, that hypoglycemia causes brain injury, that certain infants are more susceptible than others to neurologic injury, and that hypoglycemia is a toxic and dangerous state with no safe level. The defendant's experts did not dispute the general acceptance of the foregoing scientific principles. To the contrary, the defendant's expert Dr. Pavlakis confirmed that it was generally accepted that hypoglycemia can cause brain damage, that the scientific community does not recognize any level or duration of hypoglycemia considered safe and incapable of causing brain damage, and that individual susceptibility to toxic states varies among newborns.

In addition, the plaintiffs' expert Dr. Peyster explained that PVL was simply a term that refers to damage to the deep white brain matter next to the ventricles which appears as an abnormality on an MRI brain scan, and the evidence presented at the *Frye* hearing established general acceptance of the scientific principle that hypoglycemia can cause PVL. Both Drs. Katz and Peyster testified that their opinion that hypoglycemia can cause PVL was supported by the Volpe textbook, which discusses neuropathic studies indicating that hypoglycemia is a precedent of PVL. Dr. Katz characterized the Volpe textbook as a "well written outline" of certain neonatal neurologic principles, although he acknowledged that not everyone agreed with all of its conclusions, and Dr. Peyster characterized the Volpe textbook as the best text he knew of on the topic of pediatric neurology. These assessments of the Volpe textbook were not challenged by the defendant's experts. In addition, Dr. Jahre's testimony that hypoglycemia can cause brain damage in the form of white matter damage against the ventricles provided further evidence of the acceptance of the general principle that hypoglycemia can cause PVL. Although the defendant's expert Dr. Pavlakis opined that PVL is almost always caused by a decrease of blood flow or oxygen to a baby between 28 and 40 weeks of age, he cited to no medical literature or case studies to support this specific assertion, and even he acknowledged that hypoglycemia can cause brain abnormalities discernable on an MRI film.

Concededly, the plaintiffs' experts failed to produce a case or study reporting an occurrence of PVL in circumstances exactly parallel to those at issue here—i.e., after a single episode of neonatal hypoglycemia at a level of 3 mg/dL lasting 81 minutes, or any literature expressly supporting their theory that such an episode of hypoglycemia could result in PVL. Nevertheless, the

plaintiffs demonstrated that their theory of causation was reasonably permitted by a synthesis of the medical literature discussed at the hearing (*see DieJoia v Gacioch*, 42 AD3d at 979; *Zito v Zabarsky*, 28 AD3d at 44; *Marsh v Smyth*, 12 AD3d at 312-313). Although the Burns article was not designed to test the relationship between the severity or duration of hypoglycemia and neurodevelopmental outcomes, it limited its study to patients who had experienced neonatal hypoglycemia and excluded those who had suffered from other conditions, such as hypoxic ischemia, and it determined that 94% of the subjects studied, 63% of whom had only experienced one episode of hypoglycemia, had evidence of white matter abnormalities on their MRI brain scans. Although the Kinnala article had excluded infants who had experienced only one episode of hypoglycemia prior to six hours of age, it also documented a patient who had experienced an episode of hypoglycemia at seven hours of age which lasted two hours at a minimum glucose level of 32 mg/dL, a level “dramatically” higher than Lugo’s glucose level of 3 mg/dL during his episode of hypoglycemia. That patient had shown evidence of neurologic injury on an MRI, although that abnormality had subsequently resolved. Finally, the Alkalay article, which reviewed the Kinnala article and 15 others, concluded that plasma glucose levels of less than 25 mg/dL of several hours’ duration—again, a level far higher than that experienced by Lugo—may increase the relative risk for adverse neurologic outcome.

To be sure, none of the foregoing articles, read in isolation, provides conclusive support for the theory of causation espoused by the plaintiffs’ experts. However, when considered in the aggregate for the limited purpose of applying the *Frye* test, and against the backdrop of the undisputed generally accepted principles concerning hypoglycemia set forth at the hearing, those articles establish that this theory was properly based upon far more than theoretical speculation or a scientific “hunch” (*see Zito v Zabarsky*, 28 AD3d at 46). Synthesized, the materials produced by the plaintiffs’ experts at the *Frye* hearing provided an objective basis for their opinion that a period of severe hypoglycemia of relatively short duration can cause neurologic injury reflected as PVL on a MRI brain scan. The absence of medical literature directly on point with the circumstances at bar pertains to the weight to be given to this opinion testimony, but does not preclude its admissibility (*see DieJoia v Gacioch*, 42 AD3d at 979; *Zito v Zabarsky*, 28 AD3d at 46).

In concluding that the opinion testimony of the plaintiffs’ experts did not satisfy the *Frye* test, the Supreme Court emphasized the fact that those experts were unable to characterize the literature upon which they relied as “authoritative.” Seemingly, the Supreme Court ascribed significance to the experts’ willingness to apply this label while disregarding the hearing testimony that the term “authoritative” is not generally applied to medical literature and that the materials

discussed at the hearing represented the current science with regard to brain injuries resulting from neonatal hypoglycemia.

We agree with Justice Saxe that when the *Frye* test is applied to a theory of causation, “the court’s concern must be limited to making sure that within the scientific field in question, there is a substantive, demonstrable, objective basis for the expert’s conclusion,” and that “[t]he focus of the inquiry in such an instance should not be upon how widespread the theory’s acceptance is, but should instead consider whether a reasonable quantum of legitimate support exists in the literature for the expert’s views” (*Marsh v Smyth*, 12 AD3d at 312). In this case, the plaintiffs’ experts amply demonstrated the existence of such a basis for their theory of causation, and in precluding their opinion testimony, the Supreme Court applied the *Frye* test in an overly restrictive manner. Both the plaintiffs’ experts and the defendant’s experts agree that an episode of severe glucose deprivation in a newborn can cause neurologic damage; the principal dispute between them, which was emphasized by the testimony at the *Frye* hearing, is over how long such an episode must last before neurologic damage results. This factual disagreement should not have been resolved as a matter of law by the Supreme Court in the course of its *Frye* inquiry.

The purpose of the *Frye* test is not to preclude expert opinion testimony based upon reasonable extrapolations from conceded legitimate empirical data. It would be as unreasonable to preclude a 45-year smoker from seeking recovery if the only available empirical data addressed 50-year smokers as it was to preclude the instant plaintiffs’ experts from testifying, based on their reasonable extrapolations from existing legitimate empirical data, that Lugo’s severe episode of neonatal hypoglycemia caused his brain injuries.

Foundation

In addition, we disagree with the Supreme Court’s conclusion that the theory of causation espoused by the plaintiffs’ experts lacked an adequate foundation for admissibility. “The *Frye* inquiry is separate and distinct from the admissibility question applied to all evidence--whether there is a proper foundation--to determine whether the accepted methods were appropriately employed in a particular case” (*Parker v Mobil Oil Corp.*, 7 NY3d 434, 447; see *People v Wesley*, 83 NY2d at 428-429; *Jackson v Nutmeg Tech., Inc.*, 43 AD3d 599, 601). “The focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial” (*People v Wesley*, 83 NY2d at 429). “The foundation . . . should not include a

determination of the court that such evidence is true. That function should be left to the jury” (*id.* at 425).

Here, the level (3 mg/dL) and duration (81 minutes) of Lugo’s hypoglycemia episode were precisely quantified by the plaintiffs’ experts at the *Frye* hearing (*cf. Parker v Mobil Oil Corp.*, 7 NY3d at 449-450), and the Supreme Court did not conclude that these measurements were unreliable. In addition, the plaintiffs’ experts made specific reference to the contents of numerous articles documenting brain MRI abnormalities in patients who had experienced hypoglycemia to support their opinion that there was a causal connection between Lugo’s episode of hypoglycemia and the brain abnormalities later observed on his MRI film (*see Jackson v Nutmeg Tech., Inc.*, 43 AD3d at 602). Under these circumstances, we conclude that the Supreme Court improvidently exercised its discretion in concluding that the plaintiffs’ experts failed to proffer sufficient foundational evidence to support the admissibility of their testimony at trial.

The Supreme Court’s conclusion that the opinion of the plaintiffs’ experts lacked an adequate foundation rested largely on its findings that the evidence presented at the *Frye* hearing established that perinatal ischemia or hypoxia is the overwhelming cause of PVL and that the testimony of the plaintiffs’ experts did not eliminate other “more likely possible causes” of Lugo’s PVL. In relying upon such reasoning, the Supreme Court, in effect, rendered an assessment as to the ultimate merit of the opinion testimony of the plaintiffs’ experts (*see People v Wesley*, 83 NY2d at 425). Clearly, numerous factual disagreements between the parties’ experts were highlighted at the *Frye* hearing, including, but not limited to, the specific appearance of Lugo’s brain MRI abnormalities and their cause. However, these factual disagreements go to the weight to be accorded to the testimony of the plaintiffs’ experts by the trier of fact, and not the admissibility of such testimony (*see Jackson v Nutmeg Tech., Inc.*, 43 AD3d at 602).

Summary Judgment

Finally, in light of our determination that the theory of causation espoused by the plaintiffs’ experts is admissible at trial, we conclude that the Supreme Court improperly granted that branch of the defendant’s motion which was for summary judgment dismissing the complaint. Briefly, although the defendant’s expert submissions established, *prima facie*, that Lugo’s brain damage was not caused by his episode of neonatal hypoglycemia, the plaintiffs, in opposition, raised a triable issue of fact on this point through the submission of admissible expert opinion evidence (*see generally Alvarez v Prospect Hosp.*, 68 NY2d 320, 324; *Zuckerman v City of New York*, 49 NY2d 557, 562).

Thus, under the particular circumstances of this case, the Supreme Court should have denied that branch of the defendant's motion which was for summary judgment dismissing the complaint.

The appeal from the intermediate order must be dismissed because the right of direct appeal therefrom terminated with the entry of judgment in the action (*see Matter of Aho*, 39 NY2d 241, 248). The issues raised on the appeal from the order are brought up for review and have been considered on the appeal from the judgment (*see CPLR 5501[a][1]*).

Accordingly, the judgment is reversed, on the law, that branch of the defendant's motion which was for summary judgment dismissing the complaint is denied, and the order is modified accordingly.

RIVERA, J.P., FLORIO and LOTT, JJ., concur.

ORDERED that the appeal from the order is dismissed; and it is further,

ORDERED that the judgment is reversed, on the law, that branch of the defendant's motion which was for summary judgment dismissing the complaint is denied, and the order is modified accordingly; and it is further,

ORDERED that one bill of costs is awarded to the appellants.

ENTER:


Matthew G. Kiernan
Clerk of the Court