**SUPREME COURT OF THE STATE OF NEW YORK**
**COUNTY OF NEW YORK: PART 10**

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**JANET LASSALA, Administrator of the Estate of BASILIO MONTILLA deceased,**

**Plaintiff,**

Index No. 453086/2017
Motion Seq. 001

**-v-**

**DECISION & ORDER**

**STEPHEN RUSSELL, M.D., GARY MARSHALL, M.D., BELLEVUE HOSPITAL CENTER and NEW YORK CITY HEALTH AND HOSPITALS CORPORATION,**

**Defendants.**

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**GEORGE J. SILVER, J.S.C.:**

Defendants NEW YORK CITY HEALTH AND HOSPITALS CORPORATION, BELLEVUE HOSPITAL CENTER ("Bellevue"), STEPHEN RUSSELL, M.D. ("Dr. Russell"), and GARY MARSHALL, M.D. ("Dr. Marshall" collectively "defendants") move for summary judgment. Plaintiff JANET LASSALA ("plaintiff"), administrator of the estate of BASILIO MONTILLA ("decedent"), deceased, opposes the motion. For the reasons discussed below, the court grants the motion in part.

On October 18, 2015, decedent, then 24-years-old, was taken to Bellevue's emergency department ("ED") via ambulance for a traumatic brain injury ("TBI"). As a stunt performer, decedent was in a stunt course when he fell after jumping off a surface from approximately 14 feet and hit his head on the ground. Decedent lost consciousness at the scene, but was subsequently alert and oriented only to self. A physical exam revealed left periorbital ecchymosis[1] and edema,[2] and EMS recorded a Glasgow Coma Score ("GCS")[3] of 13.

[1] Ecchymosis describes a flat, blue or purple patch measuring one centimeter or more in diameter.
[2] Edema is swelling caused by excess fluid accumulation in the tissues.
[3] GCS is used to measure a person's level of consciousness, particularly when a person has a TBI.

In the ED, a physical exam showed occipital abrasion, a small scalp hematoma, and periorbital ecchymosis around the left eye. A chest x-ray ruled out pneumothorax and pleural effusions. At 12:36 p.m., a basic metabolic panel showed a sodium serum level of 139, and at 12:54 p.m., a non-contrast CT scan of the head, face, and cervical spine showed a left occipital fracture extending inferiorly to the skull base and across the petrous portion of the temporal bone, and contacted the left carotid canal. The CT scan also revealed a minimally displaced fracture of the left superior orbital rim, bifrontal parenchymal contusions, left retro-bulbar and optic nerve sheath complex hematomas, a left frontal and intra-hemispheric subdural hemorrhage, and a small left temporal subarachnoid hemorrhage. A follow-up CT scan within two hours showed no significant changes.

At 12:59 p.m., Dr. Peter Rozman ("Dr. Rozman"), a neurologist, examined decedent, and confirmed a GCS score of 13. Dr. Rozman reviewed decedent's CT scans, and noted bifrontal contusions, a left frontal interhemispheric subdural hematoma, and a small left temporal subdural hematoma. Decedent was arousable, but could not provide a coherent history. Dr. Rozman concluded that no invasive neurosurgical intervention was indicated at that time. The plan was to perform serial neurological exams, administer levetiracetam, an anti-seizure medication, and provide hypertonic saline[4] with a target sodium goal of 145-150. The plan also included a lab analysis, including sodium and serum osmolality every six hours.

Decedent received levetiracetam at 3:00 p.m., and hypertonic saline was initiated at approximately 10:00 p.m. at a rate of 50 cc/hr. At 6:27 p.m., a repeat CT scan showed that decedent's intracranial injury was stable with some overall improvement.

[4] Hypertonic saline is a crystalloid intravenous fluid composed of sodium chloride dissolved in water with a higher concentration of sodium compared to normal blood serum.

The following day, October 19, 2015, decedent was assessed with a GCS score of 15. Decedent's sodium levels remained at 140 throughout the day. Saline infusion was increased to 60 cc/hr at 1:00 a.m., and to 80 cc/hr at 6:00 a.m. to help decedent reach a sodium level of 145-155. At 10:00 p.m., decedent's sodium level was 142. On October 20, 2015, at approximately 12:07 p.m., hypertonic saline infusion was increased to 80 cc/hr.

On October 21, 2015, decedent was transferred to a "stepdown unit" where he continued to receive hypertonic saline infusion. As of 7:09 a.m., decedent's sodium level was 153, and it was ordered that decedent be weaned from hypertonic saline. Defendants reduced the infusion rate by 50% from 70 cc/hr to 35 cc/hr, and decedent's sodium levels continued to be stable.

On October 22, 2015, at 10:00 a.m., hypertonic saline was decreased to 18 cc/hr. At around noon, decedent complained of a headache. At 2:20 p.m., Dr. David Garber noted that decedent had no acute complaints. Later that evening, decedent complained of a headache, and was given Norco.

On the morning of October 23, 2015, decedent's sodium level was 136. At approximately 4:30 a.m., hypertonic saline infusion was increased from 18 cc/hr to 25 cc/hr. At 9:00 a.m., decedent's sodium level was 133. At 10:13 a.m., Anny Lee ("Ms. Lee"), a physical therapist, noted that decedent felt "ok," and that his headache had improved. Decedent tolerated physical therapy well, and stood for the first time during his admission with moderate assistance. Decedent was also oriented to self, but reported that the year was 2016. Ms. Lee assessed decedent with a Ranchos Level Scale[5] of 5, which equated to "confused-not agitated."

At approximately 10:33 a.m., decedent had no acute complaints, and his neurology exam remained the same. Decedent was somnolent but arousable, and was speaking in full sentences. At 1:44 p.m., decedent's sodium level was 128.

[5] Ranchos Level Scale is a clinical tool used to rate how patients with brain injury are recovering.

At 1:50 p.m., Dr. Yevgenia Shekhtman, a chief resident in neurosurgery, was called to decedent's bedside after the surgical intensive care unit team noticed that decedent exhibited seizure-like activity and bilateral fixed and dilated pupils. Decedent was tachypneic, and had difficulty clearing his secretions. Decedent's upper extremities were spasmodic, and decedent was intubated for airway protection. Dr. Paul Huang ("Dr. Huang"), a neurosurgery physician, observed that decedent was unresponsive with bilaterally blown pupils. A head CT scan showed worsening edema with brain compression and loss of basal cisterns. An intracranial pressure monitor showed an initial reading of 50 mmHg. Defendants administered mannitol intravenously, and increased the hypertonic saline infusion to 50 cc/hr. Defendants also recommended a left hemicraniectomy to release intracranial pressure. Dr. Huang subsequently performed a left hemicraniectomy and lobectomy, however, decedent remained unresponsive.

On October 24, 2015, apnea testing showed that decedent was brain dead. Decedent's family chose to withdraw care, and decedent died the following day.

Plaintiff alleges that defendants' failure to monitor and/or prevent decedent's intracranial swelling following a TBI resulted in brain swelling, intracranial pressure, and decedent's death.

## ARGUMENTS

Based on the record before the court, defendants argue that summary judgment must be granted, because plaintiff cannot establish that defendants' medical treatment of decedent deviated from accepted standards of care or proximately caused decedent's alleged injuries and/or death.

In support of their motion, defendants annex the affirmation of Dr. Martin Zonenshayn ("Dr. Zonenshayn"), a physician board-certified in neurological surgery. Dr. Zonenshayn opines that defendants' treatment of decedent comported with the standard of care in light of decedent's clinical presentation and clinical course. Dr. Zonenshayn contends that upon decedent's arrival at

the ED, decedent had a GCS of 13, indicative of a mild TBI, and that defendants timely performed a CT scan of decedent's head, face, and cervical spine within 30 minutes of decedent's arrival. Dr. Zonenshayn also avers that defendants properly initiated hypertonic saline infusion based on decedent's GCS score of 13 and CT scan finding of bifrontal cerebral contusions.

In Dr. Zonenshayn's opinion, given decedent's stable neurological status, defendants appropriately weaned the hypertonic saline infusion by reducing the rate by 50% on October 21, 2015, approximately 72 hours post-injury. Dr. Zonenshayn explains that hyponatremia, or low sodium in the blood, can lead to brain swelling, which can potentially result in brain damage or death, especially in a post-TBI patient. As such, Dr. Zonenshayn asserts that the standard of care is to treat hyponatremia with hypertonic saline infusion in TBI patients, especially patients with known brain contusions. According to Dr. Zonenshayn, hypertonic saline infusion requires careful control and monitoring of the patient's sodium and serum osmolality levels to attain the appropriate serum sodium target and to avoid rapid overcorrection. Dr. Zonenshayn elaborates that in a TBI patient, such as decedent, the target sodium level is raised to 145-150, or even 155, in order to reduce the risk of brain swelling. Dr. Zonenshayn further remarks that once a patient reaches the target rate, the timing of the weaning of hypertonic saline is a "judgment call" based on the patient's clinical condition. In that regard, Dr. Zonenshayn highlights that decedent reached the target rate by October 21, 2015, as decedent's sodium level had shown stability, and reached the higher end of the serum sodium target.

Dr. Zonenshayn also opines that defendants timely and properly monitored decedent's sodium levels at six-hour intervals throughout his admission at Bellevue. According to Dr. Zonenshayn, based on decedent's sodium level readings, defendants were able to adjust the

hypertonic saline infusion rate to appropriate levels. Specifically, Dr. Zonenshayn notes that defendants properly reduced the hypertonic saline infusion rate on October 21, 2015.

In addition, Dr. Zonenshayn opines that based on decedent's clinical condition and radiographic findings, surgical intervention was not indicated prior to October 23, 2015. Dr. Zonenshayn highlights that there was no basis to initiate surgical intervention as decedent remained neurologically stable from October 18, 2015 to October 22, 2015. Specifically, Dr. Zonenshayn notes that decedent's GCS was 13, and that a ventriculostomy[6] is generally not recommended for a patient with a GCS of greater than eight. According to Dr. Zonenshayn, surgical intervention is only performed when it is "clearly indicated" as surgeries carry significant risks and complications, including bleeding, infection, stroke, and death. Moreover, Dr. Zonenshayn avers that decedent's complaint of a headache on October 22, 2015 was not indicative of any change in his neurological status, and was not cause for alarm as headache is the most common symptom in all TBI patients, and can often last for weeks or months after the trauma. Dr. Zonenshayn also points out that decedent did not exhibit any other changes, or signs or symptoms indicative of an acute neurological issue. As such, Dr. Zonenshayn concludes that the timing of defendants' surgical intervention was within the standard of care.[7]

Similarly, Dr. Zonenshayn opines that there was no indication to place decedent on an intracranial pressure monitor prior to his acute neurological deterioration on October 23, 2015. Dr. Zonenshayn contends that while there was a decrease in decedent's sodium level, decedent's lowest recorded sodium level of 139 on October 22, 2015 was within normal limits. Dr.

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[6] A ventriculostomy involves the placement of a catheter connecting the ventricles of the brain to an external collecting device.

[7] Dr. Zonenshayn notes that decedent underwent emergent placement of an intracranial pressure monitoring device, and when his neurological status changed, and a repeat CT scan showed worsening edema, brain compression, and loss of basal cisterns, defendants properly took decedent to the operating room for surgery.

Zonenshayn also notes that when decedent's sodium level declined to 136 the following morning, defendants properly increased the hypertonic saline flowrate by approximately 50% to 25 cc/hr. Dr. Zonenshayn further points out that when decedent experienced mild hyponatremia, decedent was still receiving hypertonic saline, which defendants appropriately adjusted upwards. In that regard, Dr. Zonenshayn explains that rebound hyponatremia can rarely develop after abrupt discontinuance of hypertonic saline, and can result in worsening brain swelling in a small subset of head trauma patients. However, Dr. Zonenshayn notes that decedent's lowest recorded sodium level was 128, which is only "rarely concerning," as this sodium level alone would not cause such severe and acute brain edema more than 120 hours after decedent's injury so as to cause brain herniation and death.

Instead, Dr. Zonenshayn opines that seizures were the most likely etiology of decedent's brain swelling and sudden neurological deterioration. According to Dr. Zonenshayn, seizures are a known complication of brain trauma, and may first occur at any time following the traumatic event, even months or years later. Dr. Zonenshayn also notes that seizures can occur in TBI patients regardless of sodium levels[8] or the administration of hypertonic saline. Dr. Zonenshayn also underscores that anti-seizure medication lowers the risk of seizures in the acute period (first seven days following a head trauma), but does not fully eliminate the risk. As such, Dr. Zonenshayn proffers that defendants comported with the standard of care by ordering levetiracetam for seven days.

Finally, Dr. Zonenshayn opines that defendants properly monitored and controlled decedent's blood pressure with hypertensive medication. Dr. Zonenshayn highlights that decedent received labetalol to prevent an elevated systolic pressure, and that decedent's systolic blood

[8] Dr. Zonenshayn emphasizes that a decrease in decedent's sodium level to 128 in isolation would not be likely to cause seizures.

pressure remained in the low 130-range throughout his admission. Dr. Zonenshayn also notes that defendants first noted an increase in decedent's blood pressure when decedent exhibited signs of a seizure at approximately 12:00 p.m. on October 23, 2015. As such, Dr. Zonenshayn concludes that decedent's systolic pressure did not cause or contribute to his alleged seizures and/or injuries.

In opposition, plaintiff annexes the affirmation of Michael DiLuna, M.D. ("Dr. DiLuna"), a physician board-certified in neurosurgery. Dr. DiLuna opines that defendants deviated from the standard of care in their evaluation, management, and treatment of decedent, which likely caused decedent's death. Dr. DiLuna avers that had defendants followed accepted standards of practice, decedent would have likely survived and recovered from his injuries.

In Dr. DiLuna's opinion, due to the nature and severity of decedent's brain injuries, decedent needed to be placed on "strict brain injury protocols" during his admission, including careful monitoring in intensive care and control of his serum sodium levels. Dr. DiLuna contends that close monitoring and control of serum sodium levels are critically important to the management of brain injuries such as those sustained by decedent. According to Dr. DiLuna, one of the principal dangers of brain injury is the development of high intracranial pressure (abnormally high levels of pressure within the skull).[9] Dr. DiLuna expounds that because the skull is a confined space, a rise in intracranial pressure following a brain injury poses an inherent danger in causing brain damage and death. Therefore, Dr. DiLuna submits that a physician treating a brain-injured patient is responsible for having knowledge about the mechanisms affecting intracranial pressure, and for monitoring signs and laboratory measures that may indicate or "precipitate the development" of increased intracranial pressure.

[9] Dr. DiLuna explains that high intracranial pressure can develop from and following brain injury by a number of mechanisms, including direct effects of intracranial bleeding within or around the brain such as a large subdural hematoma, neurovascular trauma or blunt injury leading to inflammation and swelling of the brain days after an injury, and hormonal effects of brain injury that alter the body's electrolytes.

In addition, Dr. DiLuna opines that defendants failed to appreciate and "react" to decedent's decrease in sodium levels on October 22, 2015, which continued without correction in the early morning of October 23, 2015, and continued to decrease until decedent became hyponatremic at approximately 1:44 p.m. on October 23, 2015.[10] According to Dr. DiLuna, a "mainstay" of controlling intracranial pressure in patients with brain injury, such as decedent, is to maintain blood sodium concentrations above 145 mmol/L with a target range of 145-150. In that regard, Dr. DiLuna notes that while the normal serum sodium level for individuals without head injury is generally between 137-147, physicians want to keep patients with brain injury hypernatremic with sodium levels between 145-150, and sometimes higher, as this helps the brain shed excess fluid, the accumulation of which causes higher intracranial pressure. Conversely, Dr. DiLuna emphasizes that hyponatremia is "extremely dangerous," as this causes additional fluid to become absorbed in brain tissue, which can quickly lead to damaging or fatal high intracranial pressure for patients with brain bleeds and brain contusions, such as decedent.

According to Dr. DiLuna, the continuous and uncorrected decrease in decedent's sodium levels to below the acceptable range most likely caused decedent's "seizure-like activity" and high intracranial pressure in the afternoon of October 23, 2015. Dr. DiLuna notes that "1:44 p.m." corresponds to shortly after the time that decedent was observed to exhibit "seizure-like" activity and "posturing." Dr. DiLuna also points out that decedent's "seizure-like" activity at 1:50 p.m. was likely seen some time shortly before 1:30 p.m., which was when defendants noted "ticket to ride"[11] in decedent's chart. Dr. DiLuna further highlights that defendants first measured decedent's

[10] Dr. DiLuna highlights that decedent's sodium levels were below the acceptable range at 9:24 a.m. (143), 2:46 p.m. (142), and 7:26 p.m. (139) on October 22, 2015, and at 3:13 a.m. (136), 8:59 a.m. (133), and 1:44 p.m. (128) on October 23, 2015.

[11] Dr. DiLuna notes that a "ticket to ride" indicates that defendants sent decedent for a CT scan in response to a change in his condition.

intracranial pressure at approximately 4:45 p.m., which showed an initial reading of "at or above 50," although another note revealed a reading of "42-50," which is "extremely elevated."[12]

In Dr. DiLuna's opinion, because defendants were to sample decedent's sodium levels approximately every six hours, there was ample time and opportunity to appreciate the steady and ominous drop in decedent's sodium level from the evening of October 22, 2015 through the morning of October 23, 2015, and to correct the same prior to 1:30 p.m. on October 23, 2015. Dr. DiLuna notes that by 1:30 p.m. on October 23, 2015, decedent's low sodium level resulted in the accumulation of fluid in his brain, which caused "seizure like" activity. As such, Dr. DiLuna maintains that contrary to Dr. Zonenshayn's opinion,[13] while it is generally appropriate to wean patients off hypertonic saline, it is a departure to do so when the patient's sodium levels are inadequate and decreasing.

Similarly, Dr. DiLuna opines that defendants should have been on notice by the morning of October 23, 2015 that decedent had low levels of sodium for almost 24 hours,[14] during which time decedent had likely been accumulating excessive amounts of water in his brain tissue. In that regard, Dr. DiLuna also underscores that defendants' increase in the rate of infusion from 18 cc/hr to 25 cc/hr at approximately 4:30 a.m. on October 23, 2015 was "too late." According to Dr. DiLuna, this increase in drip rate was in response to decedent's sodium level of 136 at 3:13 a.m. on October 23, 2015, however, if defendants' response was to increase the rate of infusion (which was an "insufficient correction" even at 4:30 a.m.), defendants should have been vigilant for decedent's next sodium reading (obtained at approximately 8:59 a.m.) to ensure that the levels

[12] Dr. DiLuna notes that this is "by definition brain damaging and from that point likely to prove fatal."
[13] Dr. DiLuna notes that Dr. Zonenshayn asserts that hypertonic saline was "titrated down by 50% to 18ccs per hour" at approximately 10:00 a.m. on October 22, 2015.
[14] Dr. DiLuna notes that decedent's sodium level began dropping below 145 at 9:24 a.m. on October 22, 2015.

were being corrected to an acceptable range. Dr. DiLuna notes that the level and duration of decedent's low sodium, as measured at 3:13 a.m. and 8:59 a.m., should have alerted defendants to correct what had likely evolved to a dangerous condition—elevated intracranial pressure that was likely to worsen to a critical point if not rapidly corrected. However, Dr. DiLuna highlights that defendants did not appreciate or respond appropriately to decedent's sodium level of 133 at 8:59 a.m., which was worse than decedent's hyponatremic reading of 136 at 3:13 a.m. According to Dr. DiLuna, a sodium level of 133 is marginally lower than the normal range for a person without head injuries,[15] and is dangerous for a person with recent and severe brain injury, including a brain bleed and brain contusions. Dr. DiLuna also submits that a sodium level of 133 is significantly below the target range for hypernatremia (145-150).

Dr. DiLuna further opines that defendants failed to take effective measures to increase decedent's serum sodium levels after the 3:13 a.m. and 8:59 a.m. readings on October 23, 2015. According to Dr. DiLuna, the increased in the rate of hypertonic saline infusion from 18 cc/hr to 25 cc/hr at approximately 4:30 a.m. did not correct decedent's low sodium as decedent experienced a further drop in sodium level from 136 at 3:13 a.m. to 133 at 8:59 a.m.[16] Dr. DiLuna also underscores that decedent's sodium level was continuing to decrease, the amount of excess fluid accumulation in decedent's brain was increasing, and decedent's intracranial pressure was likely increasing during this time. As such, Dr. DiLuna concludes that it was unreasonable for defendants to persist with the same course of treatment (infusion rate of 3% at 25 cc/hr) from the time decedent's lab results were available 8:59 a.m. Notably, Dr. DiLuna observes that there was no

[15] Dr. DiLuna notes that the normal reference range is 137-147.
[16] Dr. DiLuna elaborates that increasing the drip rate from 18 cc/hr to 25 cc/hr was "simplistic" and "inadequate" since decedent had a serious and potentially life-threatening inability to maintain adequate serum sodium by 3:13 a.m. on October 23, 2015. Dr. DiLuna notes that defendants overlooked the failure of increasing the rate from 18 cc/hr to 15 cc/hr to correct the situation.

further correction (increase) in hypertonic saline infusion rate or sodium concentration, or any other measures to increase decedent's sodium serum level between 4:30 a.m. to approximately 1:30 p.m. when defendants observed seizure-like activity. Instead, Dr. DiLuna maintains that defendants should have closely monitored decedent for fluid input and output, increased decedent's sodium infusion more aggressively,[17] and limited the amount of free water or isotonic fluids given.

Additionally, Dr. DiLuna disagrees with Dr. Zonenshayn's assertion that Ms. Lee's clinical observations at approximately 10:13 a.m. on October 23, 2015 and "Critical Care Consult" at 10:33 a.m. were reassuring to the extent of obviating active measures to increase decedent's serum sodium. Rather, Dr. DiLuna notes that decedent was "far from neurologically normal" during this time, as decedent continued to be confused, and was noted to be "somnolent but arousable." Dr. DiLuna also avers that "gross evaluations" of the nature that Ms. Lee recorded are not sensitive, and are not to be relied upon to rule out this type of "insidious drop in sodium" with increased fluid retention and increased intracranial pressure. As such, Dr. DiLuna reiterates that the standard of care required defendants to closely follow and "react" to the objective lab values of decedent's sodium levels, which defendants did not do.

Likewise, Dr. DiLuna disagrees with Dr. Zonenshayn that decedent's "lowest recorded sodium level" of 139 on October 22, 2015 was "well within normal limits." Rather, Dr. DiLuna proffers that a sodium level of 139 is within normal limits for a person without brain injury, but is below the suitable range (145-150) for a patient with the type of brain injury that decedent sustained.

[17] Dr. DiLuna asserts that it was mandatory to increase the drip rate or sodium concentration and/or saline especially after decedent's 8:59 a.m. labs confirmed that the increase in drip rate at approximately 4:30 a.m. was inadequate to correct decedent's low sodium levels.

Moreover, Dr. DiLuna opines that defendants should have made decedent's 8:59 a.m. sodium level results available for review prior to 10:37 a.m. on October 23, 2015. Dr. DiLuna notes that Dr. Jacob Lassalle ("Dr. Lassalle"), the anesthesia resident who saw decedent at about 10:37 a.m. on October 23, 2015, described that the drip rate was increased in response to "last Na of 136." Dr. DiLuna elaborates that "136" refers to the sodium value measured at 3:13 a.m., however, by the time Dr. Lassalle entered this note, another reading had been obtained at 8:59 a.m. According to Dr. DiLuna, Dr. Lassalle did not review the 8:59 a.m. reading, or this reading was not available to Dr. Lassalle.

Dr. DiLuna also posits that while Dr. Marshall was decedent's attending physician, Dr. Marshall was not aware of, or did not review decedent's sodium level of 133 obtained at 8:59 a.m. Dr. DiLuna avers that despite countersigning Dr. Lassalle's note at 11:19 a.m., which documented a sodium level of 136 at 3:13 a.m., there no evidence that Dr. Marshall was aware that the last increase in sodium rate (18 cc/hr to 25 cc/hr at approximately 4:30 a.m.) was not correcting the situation as decedent's sodium level continued to decrease. As such, Dr. DiLuna concludes that Dr. Marshall departed from the standard of care by failing to apprise himself of decedent's lab values obtained at 8:59 a.m., and react "more aggressively" to the same by increasing the concentration and/or drip rate of the saline solution when he reviewed and countersigned Dr. Lassalle's 11:19 a.m. note.

Additionally, Dr. DiLuna opines that there was "inadequate consideration" of syndrome of inappropriate antidiuretic hormone ("SIADH").[18] Dr. DiLuna contends that typically, three-to-five days after a head injury, patients can experience an abnormal increase in antidiuretic hormone, and that in the presence of increased levels of antidiuretic hormone, patients will excrete relatively

[18] SIADH occurs when an excessive amount of antidiuretic hormone (a hormone that concentrates urine) is released, resulting in water retention and low sodium level.

concentrated urine while retaining water, which causes sodium levels in the blood to drop. In that regard, Dr. DiLuna asserts that defendants departed from the standard of care by failing to recognize cardinal signs of SIADH in the "classic window of time" as there was "a pattern of continuing decrease in sodium levels in the face of increasing rate of hypertonic saline." Instead, Dr. DiLuna underscores that the standard treatment for SIADH includes administering appropriate urine labs, initiating strict fluid restriction, and escalating aggressive sodium supplementation.

Furthermore, Dr. DiLuna disagrees that the mechanism of decedent's death was an unheralded seizure that led to high intracranial pressure as opposed to the other way around. According to Dr. DiLuna, once frank seizure activity occurs, a patient's intracranial pressure will increase to dangerous levels; however, here, there was a pattern of uncorrected low sodium levels persisting over a sufficient period of time (and likely in the presence of SIADH), which was the most likely cause of increased intracranial pressure. Dr. DiLuna elaborates that defendants should have prevented this dangerous increase in intracranial pressure by adequately controlling decedent's sodium level, as decedent's sodium level likely triggered a seizure-like response due to prolonged excessive accumulation of fluid in the brain.

In that regard, Dr. DiLuna notes that the testimony of Graciela Montilla ("Ms. Montilla"), decedent's sister, challenges defendants' theory that a sudden unheralded seizure placed decedent into generalized seizure activity, as opposed to a cumulative increase in intracranial pressure that reached a tipping point at around 1:30 p.m. Dr. DiLuna highlights that Ms. Montilla observed that decedent was less verbal with her on the morning of October 23, 2015 than on prior days, and that over the course of several minutes, decedent began "stiffening," and seemed to become unaware. Dr. DiLuna posits that while clinical observations of this type are not adequate to reassure a physician in the face of ominous lab values, the process of "stiffening" over several minutes is

consistent with changes in decedent's neurological status and the culmination of increased intracranial pressure.

Similarly, Dr. DiLuna notes that the testimony of Nurse Lourdes Juliano ("Nurse Juliano") shows that the steady rise in intracranial pressure due to uncontrolled serum sodium caused decedent's death as opposed to an unheralded seizure. Dr. DiLuna highlights that Nurse Juliano testified that when she first responded to calls from decedent's family, decedent was "posturing" (hands drawn to center of the body with palms facing outwards), and his pupils were "blown" (abnormally dilated). According to Dr. DiLuna, this type of posturing is most consistent with brain damage due to high intracranial pressure as compared to the type of movements more characteristic of a primary seizure event.

Dr. DiLuna further opines that decedent was deprived of "any significant chance for survival" once defendants noted seizure-like activity at approximately 1:50 p.m. on October 23, 2015. Dr. DiLuna contends that while attempting to diagnose and correct the underlying mechanism of decedent's sudden clinical decline at about 1:30 p.m. on October 23, 2015, defendants should have decreased decedent's intracranial pressure as soon as possible. However, Dr. DiLuna points out that defendants allowed decedent's elevated intracranial pressure to cause additional fatal brain damage over the next hours.

Dr. DiLuna also opines that based on decedent's history and CT scan findings,[19] defendants deviated from the standard of care by not administering mannitol "stat" (between about 1:30 p.m. and 1:45 p.m.). Dr. DiLuna posits that the first line of medication to lower intracranial pressure is mannitol, but instead of administering mannitol within a short time after decedent's sudden

[19] Dr. DiLuna notes that the CT scan showed effects of fluid accumulation in the brain, increase in pressure, edema, and new "midline shift" and "effacement" of sulci (normal spaces in the brain) that are highly suggestive of high intracranial pressure.

deterioration, defendants ordered mannitol at 4:24 p.m., and administered the same at approximately 4:54 p.m. [20] According to Dr. DiLuna, the failure to timely administer mannitol caused additional brain damage, and deprived decedent of any meaningful chance of recovery from that point.

Similarly, Dr. DiLuna opines that defendants delayed in administering any effective measures to lower decedent's intracranial pressure. Dr. DiLuna highlights that Dr. Huang first performed intracranial pressure monitoring at around 4:00 p.m., approximately two-and-a-half-hours after defendants should have undertaken "stat" measures, and placed the monitor at around 4:45 p.m., at which time decedent's initial pressures were grossly elevated and damaging.

Likewise, Dr. DiLuna points out defendants' performance of the decompressive surgery at 5:17 p.m. was grossly delayed and unlikely to "achieve a benefit" as decedent had already likely suffered fatal brain damage. As such, Dr. DiLuna concludes that defendants' gross delay in administering medication to lower decedent's intracranial pressure, and delay in performing a "stat" decompressive surgery exacerbated decedent's brain damage, and removed what remained of an opportunity for survival.

Finally, Dr. DiLuna opines that decedent's intraoperative findings confirm that the mechanism of low sodium with increased fluid retention in the brain caused increased intracranial pressure, which likely caused decedent's brain damage and subsequent death. According to Dr. DiLuna, the rise in intracranial pressure was a direct result of defendants' failure to control decedent's sodium levels, which caused an increase in intracranial pressure that culminated in the "seizure like activity" observed at around 1:30 p.m. on October 23, 2015.

[20] Dr. DiLuna highlights that while other notes suggest that defendants may have administered mannitol at 4:45 p.m., this was nonetheless "grossly delayed," and a departure from accepted practice.

In reply, defendants argue that plaintiff has improperly raised a new theory of liability in opposition to their motion for summary judgment that defendants failed to recognize and treat signs of SIADH.[21] Defendants contend that even if this argument was proper, it not supported by the medical records. According to defendants, while Dr. DiLuna claims that decedent could have experienced an increase in antidiuretic hormone, which caused him to retain fluid, decedent's input/output of fluids was always net-negative (the amount of total fluid outtake exceeded the intake). Defendants also note that there is no indication that decedent received iatrogenic fluid, but rather, decedent only received hypertonic saline, which was less than decedent's fluid outtake.

Similarly, defendants argue that plaintiff alleges for the first time in opposition that defendants should have administered mannitol earlier. Defendants also maintain that even if this argument was proper, the medical literature does not support a theory that the failure to administer mannitol decreases the likelihood of recovery for a TBI patient. Rather, defendants submit that there is no confirmed benefit on overall clinical outcome with the use of mannitol.

In addition, defendants argue that contrary to Dr. DiLuna's opinion that defendants failed to appreciate and react to a decrease in decedent's sodium level from the evening of October 22, 2015 to the morning of October 23, 2015, decedent's target sodium level was between 145 and 150. In that regard, defendants reiterate that once the target sodium rate is reached, the timing of the weaning of hypertonic saline is a "judgment call" based on the patient's clinical presentation.[22]

Defendants also argue that decedent reached and remained at the target sodium level within 36-to-72 hours after his traumatic injury, the timeframe in which brain swelling peaks. Defendants note that because decedent's accident occurred at approximately 11:45 a.m. on October 18, 2015,

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[21] Defendants submit a supplemental affirmation of Dr. Zonenshayn to address plaintiff's new theories of liability.

[22] Defendants reemphasize that defendants properly started to wean the hypertonic saline at 7:09 a.m. on October 21, 2015 (70 cc/hr to 35 cc/hr) after decedent had minimally surpassed his target sodium level.

peak swelling occurred between 11:45 a.m. on October 19, 2015 and 11:45 a.m. on October 21, 2015. Defendants also highlight that they initiated hypertonic saline infusion on October 18, 2015, the date of decedent's hospital admission, and that decedent's target sodium level was reached by 7:16 p.m. on October 20, 2015. Defendants further point out that decedent's sodium level was 153 at approximately 7:09 a.m. the following day.

In that regard, defendants contend that while plaintiff claims that decedent's sodium level decreased on October 22, 2015 through October 23, 2015 without a proper response, decedent's sodium level remained within the normal range (135 to 145) throughout October 22, 2015. To be sure, defendants underscore that decedent's sodium level first approached the lower end of the normal range at 3:13 a.m. on October 23, 2015 (more than 100 hours post-trauma) with a reading of 136, a value that was still within the normal range.[23] Defendants also proffer that when they received decedent's sodium level readings, they increased the hypertonic saline infusion from 18 cc/hr to 25 cc/hr at approximately 4:00 a.m., which was proper to prevent rapid overcorrection.

Defendants further argue that while Dr. DiLuna opines that decedent reached a dangerous sodium level as he had a recent and severe brain injury, decedent's TBI was mild based on his GCS score of 13 and 15. Similarly, defendants aver that despite assuming that decedent's decrease in sodium level automatically means an increase in intracranial pressure, Dr. DiLuna does not show that decedent experienced any clinical signs or symptoms that would result from an increase in intracranial pressure at any point until after decedent exhibited seizure-like activity at 1:30 p.m. on October 23, 2015. Rather, defendants emphasize that decedent's neurological status remained unchanged. Specifically, defendants note that Dr. Lassalle found that decedent was alert and oriented, following simple commands, and moving all extremities with normal strength.

[23] Defendants note that Dr. DiLuna does not contest this fact, and fails to account for the fact that decedent had passed the peak time for expected swelling.

Moreover, defendants argue that Dr. DiLuna offers no basis in which to challenge the accuracy of Dr. Lassalle's neurological assessment. Notably, defendants highlight that decedent's headache, which was expected following a TBI, had improved, which is not consistent with increasing intracranial pressure. Rather, defendants reiterate that decedent remained neurologically unchanged, and that there were no clinical signs of brain swelling. Likewise, defendants note that Dr. DiLuna concedes that decedent had a "sudden deterioration" likely between 1:30 p.m. and 1:50 p.m., which contradicts his earlier statement that decedent's intracranial pressure had "likely" been increasing for almost 24 hours.

Additionally, defendants argue that plaintiff failed to show that defendants delayed in performing surgery. Defendants note that decedent first began to exhibit seizure-like activity at approximately 1:30 p.m. on October 23, 2013, and that decedent underwent a CT scan at approximately 2:00 p.m. that day. Defendants also highlight that within minutes thereafter, they placed decedent on a respirator and inserted an intracranial pressure monitor. Defendants further point out that they informed decedent's family of decedent's poor prognosis and the option to perform a hemicraniectomy. According to defendants, the performance of a hemicraniectomy at approximately 4:00 p.m., two-and-a-half hours after decedent first presented symptoms, was "extremely timely" and within the standard of care. Defendants further argue that performing surgery after decedent was found to have dilated pupils was a "long shot" as decedent's prognosis was poor.

Finally, defendants assert that Dr. Russell is entitled to summary judgment as plaintiff does not assert any claims of malpractice prior to the early morning of October 23, 2015. Defendants

also note that Dr. Russell was neither at the hospital nor oversaw decedent's care as of the end of his shift on October 22, 2015.[24]

**DISCUSSION**

To prevail on summary judgment in a medical malpractice case, a physician must demonstrate that he did not depart from accepted standards of practice or that, even if he did, he did not proximately cause the patient's injury (*Roques v. Noble*, 73 A.D.3d 204, 206 [1st Dept. 2010]). In claiming treatment did not depart from accepted standards, the movant must provide an expert opinion that is detailed, specific and factual in nature (*see e.g.*, *Joyner-Pack v. Sykes*, 54 A.D.3d 727, 729 [2d Dept. 2008]). The opinion must be based on facts in the record or personally known to the expert (*Roques*, 73 A.D.3d at 207). The expert cannot make conclusions by assuming material facts which lack evidentiary support (*id.*). The defense expert's opinion should state "in what way" a patient's treatment was proper and explain the standard of care (*Ocasio-Gary v. Lawrence Hosp.*, 69 A.D.3d 403, 404 [1st Dept. 2010]). Further, it must "explain 'what defendant did and why'" (*id*. *quoting Wasserman v. Carella*, 307 A.D.2d 225, 226 [1st Dept. 2003]).

Once defendant makes a *prima facie* showing, the burden shifts to plaintiff "to produce evidentiary proof in admissible form sufficient to establish the existence of material issues of fact which require a trial of the action" (*Alvarez v. Prospect Hosp.*, 68 NY2d 320, 324 [1986]). To meet that burden, plaintiff must submit an expert affidavit attesting that defendant departed from accepted medical practice and that the departure proximately caused the injuries (*see, Roques*, 73 AD3d at 207). "Summary judgment is not appropriate in a medical malpractice action where the

[24] Defendants highlight that Dr. Russell testified that Dr. Wong, the director of neurosurgery, was the attending physician on October 23, 2015, and that he would not have been informed of decedent's deterioration until he was on-call on Saturday morning. Defendants also note that decedent's medical chart confirmed that Dr. Russell did not see decedent on October 23, 2015, as there is no note by Dr. Russell, or any other reference that Dr. Russell was aware of decedent's condition at any time on October 23, 2015.

parties adduce conflicting medical expert opinions" (*Elmes v. Yelon*, 140 A.D.3d 1009 [2nd Dept 2016] [citations and internal quotation marks omitted]). Instead, the conflicts must be resolved by the factfinder (*id.*).

Here, defendants set forth a *prima facie* showings in favor of dismissal, as evidenced by the submission of defendants' medical records, and defendants' expert affidavit, all of which attest to the fact that defendants' treatment of decedent was in accordance with accepted standards of care and did not proximately cause decedent's alleged injuries and/or death. To be sure, defendants' expert affirmation is detailed and predicated upon ample evidence within the record. As defendants have made a *prima facie* showing, the burden shifts to plaintiff.

**I. New Theories of Liability**

"It is axiomatic that a plaintiff cannot defeat a summary judgment motion that made out a prima facie case by merely asserting, without more, a new theory of liability for the first time in the opposition papers" (*Biondi v. Behrman*, 149 A.D.3d 562, 563–64 [1st Dept. 2017]; *Abalola v. Flower Hosp.*, 44 A.D.3d 522, 522, 843 N.Y.S.2d 615, 616 [1st Dept. 2007]). Here, plaintiff has impermissibly raised a new theory of liability in opposition to defendants' motion for summary judgment. Notably, plaintiff's allegation that defendants failed to recognize and treat signs of SIADH was not pleaded in plaintiff's complaint or bills of particulars (*see, Marti v. Rana*, 173 A.D.3d 576, 577 [1st Dept. 2019]). Accordingly, this claim is dismissed.

However, plaintiff's bills of particulars set forth a generalized allegation that defendants "fail[ed] to diagnose and treat increased intracranial pressure." Such broad allegation is sufficient to encompass plaintiff's claim that defendants failed to timely administered mannitol, the "first

line of medication to lower intracranial pressure." As such, defendants' application to dismiss this claim is denied.[25]

## II. Triable Issues of Fact

Substantively, plaintiff has raised triable issues of fact sufficient to preclude summary judgment. For instance, the parties disagree as to whether defendants' care and treatment of decedent comported with the standard of care. Notably, defendants argue that in light of decedent's clinical presentation and clinical course—which included a GCS of 13 upon decedent's arrival at the ED, and a CT scan finding of bifrontal cerebral contusions—defendants timely performed a CT scan of decedent's head, face, and cervical spine, and properly initiated hypertonic saline infusion. By contrast, plaintiff avers that defendants deviated from the standard of care in their evaluation, management, and treatment of decedent, which likely caused decedent's death. Specifically, plaintiff underscores that due to the nature and severity of decedent's brain injuries, defendants needed to place decedent on "strict brain injury protocols" during his admission, which include careful monitoring in intensive care and control of decedent's serum sodium levels. Defendants, however, assert that they timely and properly monitored decedent's sodium levels at six-hour intervals throughout his admission, and based on decedent's sodium levels, defendants properly adjusted the hypertonic saline infusion rate to appropriate levels. Because these issues cannot be resolved by the facts before the court, summary judgment is denied.

Significantly, there is a triable issue of fact as to whether decedent's sodium levels were within the appropriate range during his admission, and whether defendants departed from the

[25] In that regard, the court will consider Dr. Zonenshayn's supplemental affirmation that addresses the administration of mannitol. To summarize, Dr. Zonenshayn avers that the medical literature does not support a theory that the failure to administer mannitol decreases the likelihood of recovery in a TBI patient. Dr. Zonenshayn also notes that there is no confirmed benefit on overall clinical outcome with the use of mannitol in the presence of increased intracranial pressure.

standard of care by weaning decedent off hypertonic saline. While plaintiff submits that defendants failed to appreciate and react to the fact that decedent had persistently low levels of sodium for almost 24 hours, defendants maintain that decedent reached the target sodium level (145-150) by October 21, 2015, and remained within the normal range (135-145) throughout October 22, 2015. Defendants also contend that based on decedent's stable neurological status, and the fact that decedent had reached the target sodium rate by October 21, 2015, it was appropriate to wean the hypertonic saline infusion on October 21, 2015 by reducing the rate by 50%. Plaintiff, however, disagrees, and argues that while it is generally appropriate to wean hypertonic saline, it is a departure to do so when a patient's sodium levels are inadequate and decreasing. To be sure, plaintiff underscores that defendants should have appreciated the steady and ominous decrease in decedent's sodium levels from the evening of October 22, 2015 through the morning of October 23, 2015, and corrected the same prior to 1:30 p.m. on October 23, 2015 when decedent's low sodium levels resulted in the accumulation of fluid in decedent's brain. Still, defendants aver that once a patient reaches the target sodium rate, the timing of the weaning of hypertonic saline is a judgment call based on the patient's clinical condition, and that it is within the standard of care to wean the patient and monitor him/her clinically. Accordingly, there are triable issues of fact here sufficient to preclude summary judgment.

Similarly, the parties disagree as to whether defendants properly treated the decrease in decedent's sodium levels. While defendants argue that they properly increased the hypertonic saline infusion rate from 18 cc/hr to 25 cc/hr at approximately 4:00 a.m. when decedent's sodium level declined to 136, plaintiff emphasizes that defendants' adjustment was inadequate to correct

the situation.[26] Indeed, plaintiff highlights that despite the adjustment at 4:30 a.m., decedent's sodium level further declined to 133 at 8:59 a.m. In that regard, contrary to defendants' position that they appropriately adjusted the saline infusion rate to prevent rapid overcorrection, plaintiff asserts that defendants should have closely monitored decedent's fluid input and output, increased the saline infusion rate more aggressively,[27] and limited decedent's amount of free water or isotonic fluids. In rebutting plaintiff's argument, however, defendants maintain that the medical literature does not support a theory that an increase in hypertonic saline infusion rate after 4:00 a.m. on October 23, 2015 would have resulted in a rapid increase in decedent's sodium levels or prevented decedent's seizure-like activity noted at approximately 1:30 p.m.[28] Because these issues cannot be resolved by the facts before the court, summary judgment is denied.

Furthermore, plaintiff raises an issue of fact as to whether defendants departed from the standard of care by not timely administering mannitol. Notably, while plaintiff contends that based on decedent's history and CT scan findings, defendants should have administered mannitol between about 1:30 p.m. and 1:45 p.m., defendants argue that there is no confirmed benefit on overall clinical outcome with the use of mannitol in the presence of increased intracranial pressure. To be sure, contrary to plaintiff's assertion that the failure to timely administer mannitol caused additional brain damage, and deprived decedent of any meaningful chance of recovery, defendants emphasize that the medical literature does not support a theory that the failure to administer

[26] Plaintiff notes that the level and duration of decedent's low sodium, as measured 3:13 a.m. and 8:59 a.m., should have alerted defendants to correct what had likely evolved into a dangerous condition of elevated intracranial pressure that was likely to worsen to a critical point if not rapidly corrected.
[27] Plaintiff asserts that it was mandatory to increase the drip rate or concentration and/or saline especially after decedent's 8:59 a.m. labs confirmed that the increase in drip rate at approximately 4:30 a.m. was inadequate to correct decedent's low sodium levels.
[28] *See*, Dr. Zonenshayn's Supplemental Affirmation (p. 4).

mannitol decreases the likelihood of recovery in a TBI patient. Accordingly, there are triable issues of fact here sufficient to preclude summary judgment.

Likewise, plaintiff raises a triable issue of fact as to whether defendants delayed in performing surgery, and as to whether this alleged departure proximately caused decedent's alleged injuries and/or death. Notably, plaintiff maintains that defendants' performance of a decompressive surgery at 5:17 p.m. was grossly delayed and unlikely to "achieve a benefit" as decedent had already likely suffered fatal brain damage. Defendants, on the other hand, posit that there was no basis to initiate surgical intervention on October 18, 2015, or at any time before decedent's acute neurological deterioration on October 23, 2015 since decedent had remained neurologically stable[29] from October 18, 2015 to October 22, 2015. Instead, defendants contend that they timely performed a hemicraniectomy at approximately 4:00 p.m. on October 23, 2015, two-and-a-half hours after decedent first presented symptoms of seizure-like activity. In further refuting plaintiff's theory of causation, defendants underscore that the alleged delay in performing a hemicraniectomy did not cause decedent's ultimate outcome. According to defendants, since decedent had "blown" or dilated pupils by 1:30 p.m. on October 23, 2015, which is typically indicative of irreversible brain damage following a head trauma, decedent's likelihood of recovery at that time was minimal at best. Because these issues cannot be resolved by the facts before the court, summary judgment is denied.

Moreover, the parties' conflicting positions as to the "mechanism" or "etiology" of decedent's condition and subsequent death further support denial of summary judgment. Indeed, plaintiff refutes defendants' argument that seizures were the most likely etiology of decedent's intracranial pressure elevation, brain swelling, and sudden neurological deterioration. Rather,

[29] Defendants note that decedent's clinical status continued to be stable on October 22, 2015, and that decedent did not exhibit any other changes, or signs/symptoms indicative of an acute neurological issue.

plaintiff submits that the continuous and uncorrected decrease in decedent's sodium levels most likely caused decedent's seizure-like activity and high intracranial pressure in the afternoon of October 23, 2015.[30] By contrast, defendants assert that plaintiff does not show that decedent experienced any clinical signs or symptoms that would result from increased intracranial pressure at any time prior to decedent's display of seizure-like symptoms at approximately 1:30 p.m. on October 23, 2015. To highlight, defendants emphasize that decedent's neurological clinical status remained unchanged as demonstrated by Dr. Lassalle's findings (i.e. decedent was alert and oriented, following simple commands, and moving all extremities with normal strength). Accordingly, there are triable issues of fact here sufficient to preclude summary judgment.

Finally, as there are triable issues of fact as to whether defendants departed from the standard of care prior to October 23, 2015, *see supra*, defendants' application to grant summary judgment in Dr. Russell's favor is denied.

Based on the foregoing, it is hereby

ORDERED that defendants' motion for summary judgment is granted to the extent that plaintiff's claim that defendants failed to recognize and treat signs of SIADH is dismissed; and it is further

ORDERED that the clerk is directed to enter judgment as to this claim; and it is further

ORDERED that the remainder of defendants' motion for summary judgment is denied in its entirety; and it is further

ORDERED that the parties are directed to appear for a virtual conference before the court on September 11, 2020 at 11:00 AM.

This constitutes the decision and order of the court.

Dated: July 29, 2020

George J. Silver

**HON. GEORGE J. SILVER**

[30] Plaintiff notes that decedent's uncorrected low sodium level persisting over a sufficient period of time was the most likely cause of decedent's increased intracranial pressure by accumulation of excess fluid in the brain.