

February 29, 2000

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Re: Maggie XXXX
ADDENDUM REPORT

Dear Mr. _____:

Thank you for allowing Medically Speaking to assist you with the medical record review concerning Maggie XXXX. The birth records are incomplete.

According to the medical records provided, On July 1, 1998, Maggie was born at the University of Alabama by elective cesarian section for prenatally diagnosed myelomeningocele. Her Apgars were 8 and 9. She received oxygen XXXX with nasopharyngeal and oropharyngeal suctioning (suctioning of the nose and mouth) and was taken to the Neonatal Intensive Care Unit for stabilization. She was subsequently transported to Children's Hospital.

On admission, the records describe spina bifida with a "giant" lumbar myelomeningocele (a portion of the spinal cord and covering of the spinal cord is outside the body due to failure of the bones in the spinal column to fuse/close properly). She underwent surgery on July 2, 1998 for closure of the lumbar myelomeningocele.

Diagnostic tests included, video EEG monitoring, which was found to be abnormal due to the presence of moderate to severe generalized slowing, greater over the right hemisphere than the left. Echocardiogram revealed a normal heart with persistence of neonatal high TA pressure and resistance. Ultrasound revealed an apical muscle ventriculoseptal defect (hole in the heart). She was noted to have a grade 2/6 systolic murmur. Renal ultrasound revealed a possible ureteropelvic junction obstruction. Voiding cistourethrogram on July 6, 1998 showed no reflux, however Amoxicillin was prescribed for urinary tract infection prophylaxis.

Maggie was discharged on the July 8, 1998 (p. 31-33) with the following diagnoses:

1. Myelomeningocele.

2. Term birth, living child.
3. Rule out sepsis.
4. Ventriculoseptal defect.
5. Rule out neurogenic bladder.
6. Bilateral calcaneal (heel) valgus deformity.

Maggie was readmitted to Children's Hospital on July 14, 1998 with a preoperative diagnosis of hydrocephalus (increasing head size due obstruction of the flow of cerebral spinal fluid). At the time of admission, she was noted to have lower extremity movement with the exception of plantar flexion. She underwent insertion of a ventriculoperitoneal shunt by Dr. Paul XXXX. Her postoperative course was noted to be benign and she was discharged home on July 15, 1998 in stable condition.

Maggie was readmitted to Children's Hospital on July 24, 1998 with diagnoses of:

1. Rule out shunt malformation and infection.
2. Ventriculoseptal defect.
3. Status post ventriculoperitoneal shunt.
4. Status post myelomeningocele repair.
5. Diaper dermatitis.

She was admitted with complaints of fever and irritability and placed on antibiotics. Cerebrospinal fluid cultures were positive, however Neurosurgery felt this was a contaminant. Repeat cultures were negative with the exception of blood cultures, however, the organism was also thought to be a contaminant.

Maggie was seen in follow-up by Dr. Michael XXXXX on January 13, 1999 (p. 8). She was now six months of age. Her mother's only complaint was that Maggie's "feet are small" and the left foot turned in more than the right. Maggie was sitting independently when placed there, but could not get herself into a sitting position as yet. She was able to scoot on her bottom and roll both prone to supine and supine to prone. Her motor exam was grossly intact; however, it was felt there might be a slight degree of weakness of the foot plantar flexors.

On March 2, 1999, Maggie underwent revision of the ventriculoperitoneal shunt for proximal shunt obstruction. She was noted to have presented with progressive macrocephaly (enlargement of the head) and an enlarging ventricular system with a ventricular catheter in place. It was felt the catheter had become too short and had migrated out of the ventricular space. Upon removal of the Rickham hub from the ventricular catheter, there was very poor flow of cerebrospinal fluid. A new catheter was advanced down with brisk return of clear cerebrospinal fluid. No further information is provided concerning this admission.

A CT of the head was performed on April 7, 1999 showing persistent moderate dilation of the third and lateral ventricles. The radiologist felt these findings could represent shunt malfunction and he discussed the findings with Dr. Paul XXXX and advised clinical correlation.

On this same date, Dr. XXXX (p. 10) documents Maggie returned after falling and striking her head, with some swelling around the Rickham reservoir. On examination, she was happy, smiling and playful. There was some prominence of the veins and the fontanelle was full. Her occipital frontal circumference was 47 cm, which was greater than it was postoperatively a month previously. A CT revealed the ventricular size “has really not come down.” His suspicion was the ventricular catheter was probably caught in some scar tissue and she would benefit from placing the catheter elsewhere in the ventricular system. The plan was for admission for endoscopic shunt revision.

On April 9, 1999, Maggie was admitted to Childrens Hospital of Alabama. Her development was noted to be normal according to the nursing admission assessment, with no physical limitations.

On the above date, Maggie underwent surgery for endoscopic revision of her ventriculoperitoneal shunt. According to the operative report, on disconnection of the Rickham hub, there was no flow of cerebrospinal fluid from the ventricular catheter. The ventricular catheter was replaced, with brisk, clear cerebrospinal fluid. She left the operating room in stable condition.

The nurse documents at 0950 hours, she received Maggie from the PACU (Post-Anesthesia Care Unit). Her vital signs were stable. She was noted to have a dressing to the right scalp which was intact with no visible drainage noted. The pupils were equal and reactive and she documents “parents informed of plan of care.” Maggie was offered Pedialyte at 1000 hours. However, at 1130 hours, she had an emesis episode. At 1355 hours, she was given 100 mg of Tylenol orally for discomfort. At 1400 hours, Nurse Bryant (p. 199) documents Maggie was awake, but slightly irritable, but there was no change from the initial assessment. At 1500 hours, Maggie was asleep in her mother’s arms. The dressing to the right scalp was dry and intact without drainage noted. There was no change at the 1800 hour assessment. At 2015 hours, Nurse Bishop (p. 199) documents the parents were informed of Maggie’s discharge the next day. Her I.V. was converted to a heparin lock. There were no obvious complaints of headache or pain and she was documented as being intact neurologically. At 2200 hours, Maggie was asleep. Oxygen saturations were 97% on six liters of oxygen.

At 2325 hours, Maggie “woke up shaking...having a chill.” Axillary temperature was 101 . However, rectal temperature was noted to be 103 . Maggie was given Tylenol orally for the fever and a light blanket was applied.

At 2335 hours, a tepid sponge bath was given.

At 2340 hours, Maggie’s oxygen saturation was noted to be decreased to 86%. Her heart rate was elevated to 230 and the nurse reported she “looks like she might start seizing.” She appropriately paged Dr. XXXXXXXXX and applied oxygen at 6 liters by facial mask.

According to the Cardiorespiratory Resuscitation Record, at 2342 hours, Maggie’s heart rate was elevated to 223. Oxygen saturation was 99%.

Cardiorespiratory Resuscitation Record (p. 158), documents the seizure began at 2335 hours.

While the nurses notes document the seizure did not begin until 2345 hours. The notes reads, Maggie was noted to have started seizing on the left side with eyes deviated to the left. There is no documentation by the nurse she again tried to contact Dr. XXXXXXXX to notify him Maggie had started seizing. There is no documentation in the nurse's notes that Dr. XXXXXXXX had given orders for anti-seizure medication at the time of the prior conversation. However, the physician's orders for this period of time are missing. It would appear, since no medication was administered by the nurse when Maggie started seizing, that no orders were given for medication to control the seizure.

At 2350 hours, the Cardiorespiratory Resuscitation Record (p. 158) reflects the "team arrived." Oxygen saturation was 99%.

According to this form, the nurse present was Jason Peterson, R.N. The recorder (the person completing the cardiopulmonary resuscitation form) was Penny Bishop, R.N. The physicians present were Dr. XXXXXXXX, Dr. Hartig, Dr. Siegel and Dr. XXXXXXXX. It is not clear exactly who of the above arrived at 2350 hours, however, it does not appear the physicians arrived at this time. From the records provided, it appears nothing was done for Maggie in attempts to control the seizure, even after "the team" arrived at 2350 hours. Therefore, it needs to be determined exactly who was present and exactly when they arrived.. The seizure had been continuing for 5-15 minutes by this point in time without definitive action being initiated.

There is no documentation labs were drawn, nor an acu-check to check blood glucose level.

At 2355 hours, 10-20 minutes after the seizure began (depending on which note is accurate), there is no documentation that any medication was given to control the seizure activity. The nurse does document that a sponge bath was given and that Maggie was "still seizing."

At 0010 hours, temp was 101.3 rectal, 100.3 axillary.

At 0013 hours, Nurse Bishop documents she paged Dr. XXXXXXXX again and Maggie was still seizing. The seizure was noted to be "harder." Oxygen saturation was down to 87%. Heart rate was 250. The nurse documents, "Can't get blood pressure." The nurse should have paged the physician immediately, when Maggie began to seize. The fact she did not page immediately when there was a detrimental change in Maggie's condition is below the standard of care, as is the further delay of the additional 28-38 minutes after the seizure onset. The rapid heart rate and low oxygen saturation, in combination with the inability to obtain a blood pressure raises (hypotension) concerns of poor perfusion to the body tissues, and more importantly the brain.

At 0020 hours, temperature was now 101.3 axillary. Saturation was down to 79%. Finally, 35-45 minutes after the onset of the seizure, Ativan was administered to control the seizure. Five minutes later, Dr. XXXXXXXX was noted to be in the room and additional Ativan was administered.

There are two physicians orders for this period of time. The first is a telephone order from Dr. XXXXXXXXX at 0020 hours, to place “a pulse oximeter on patient. Use oxygen as needed. Give Ativan 0.2mg IV now.” The second is an order at 0025 to have “Ativan 2mg at bedside for MD.”

At 0030 hours, Ativan 0.25 mg was given for seizure. Oxygen saturation was now up 95%. However, Maggie was still tachycardic with a heart rate of 230. Oxygen was in place at 6 liters.

At 0034 hours, additional Ativan was given as Maggie was “still seizing.”

At 0035 hours, additional Ativan was given.

At 0040 hours, more Ativan was given. However, the dose was increased from 0.25 mg I.V. to 0.5 mg I.V. Oxygen saturation was noted to be 100%. Maggie was tachycardic with a heart rate of 234.

At 0042 hours, oxygen saturation was 99%. Maggie’s heart rate was 223. Additional Ativan was given and Maggie’s mouth was suctioned. Dr. XXXXXXXXX “attempted to intubate patient.” However, the nurse documents that intubation was unsuccessful.

At 0050 hours, the seizure finally stopped - 1 hour and 5 minutes or 1 hour and 15 minutes (depending on which record is correct) after the seizure began. Of particular interest, Nurse XXXXX note (p. 200) documents in the nurses notes the Code Team arrived at 0050 hours, which is in contradiction to her note at 2350 hours on the cardiopulmonary resuscitation records which says, “Team arrived.”

However, of particular importance no Dilantin or phenobarbitol were administered, only Lorazepam. Just because the seizure stops with the Ativan, does not mean the seizure activity in the brain has stopped, Dr. XXXXXXXXX failed to give an anti-seizure medication, such as Dilantin early on. The importance of this will be discussed later in this report.

At 0100 hours, Maggie’s heart rate had decreased to 201 and the Nursing Supervisor had arrived to be with the parents.

At 0105 hours, Norcuron (medication given to paralyze) and Versed were administered for sedation so Maggie could be intubated. Heart rate was 202. Blood pressure was 67/21 and the oximeter was reading 92%.

At 0110 hours, the anesthesiologist arrived to intubate Maggie (23 minutes after Dr. XXXXXXXXX’s failed attempt). He stated he intubated with a #4 endotracheal tube with “one attempt.” This is of critical importance, since Dr XXXXXXXXX’s failed attempts to intubated essentially wasted 28 minutes. The nurse’s notes document after intubation, Maggie’s heart rate was 195 with saturation at 94%.

At 0115 hours, an oral gastric tube was placed. At 0120 hours, a shunt tap was attempted. However, there is no further information in the note regarding the results of this.

At 0132 hours, Maggie was transported to the Pediatric Intensive Care Unit in stable condition. CT scan of the head was performed at approximately 0150 hours, showing postoperative changes with intracranial catheter appearing continuous with the cranial extracavillarial component, interval change in the size of the lateral ventricles, and question of mild edematous change throughout the inferior left temporal parietal lobe (p. 275-276).

At 0215 hours, the nurses document Maggie's temp was 103.3 rectally. Dr. A XXXXXXXX and Dr. XXXXXXXX were at the bedside and aware. Tylenol 100 mg was ordered and given. Ice packs were applied to the axillary and groin areas. The progress note of Dr. XXXXXXXX (p. 170-171) documents "while on the floor tonight, Maggie had a fever of 103 and soon after, started seizing. The seizure lasted about 30 minutes." However, this is contradicted by the nurse's notes. Dr. XXXXXXXX indicates Maggie received Ativan on the floor, resulting in respiratory depression and low oxygen saturation. The nurses notes reflect, Maggie's saturation was low prior to the administration of the Ativan (lorazepam), although it is obvious the large amounts of Ativan did cause severe respiratory depression. On Dr. XXXXXXXX's arrival to the room, Maggie was not seizing, but had increased work of breathing and some "perioral cyanosis." She was "bagged to a pink color." It appears clearly Maggie was not being oxygenated adequately at the time of Dr. Sorrentio's arrival. Status epilepticus in and of itself can lead to decreased cerebral perfusion at the peak of each seizure. The fact she was bagged to a pink color and was cyanotic on the arrival of Dr. XXXXXXXX, leads one to consider Maggie was not being adequately ventilated by the bag and mask prior to the arrival of Dr. XXXXXXXX and during the failed attempts to intubate by Dr. XXXXXXXX over a prolonged period of time lead. All of the above lead to decreased perfusion and cerebral hypoxia.

Dr. SXXXXXXX's note continues: Anesthesia was called to the bedside and made the decision to intubate. Dr. XXXXXXXX performed a tap of the shunt which did not reveal any increased intracranial pressure. A stat CT showed a small intraventricular hemorrhage and left temporal edema. His impression was nine-month-old with spina bifida status post shunt revision and status post status epilepticus with respiratory distress and fever. The plan was to maintain on a ventilator until the sedatives/paralytics wore off, then extubate. Pan cultures would be obtained to rule out an infection. It was felt there was a possible combination of febrile seizure and irritation status post manipulation. He stated any further seizures would be treated symptomatically per Neurosurgery.

Dr. XXXXXXXX's Neurosurgery progress note states he was called to evaluate Maggie after she had a seizure of acute onset with a temp of 103 F. He reports he "tried to cool her down with compresses and Tylenol. Seizure continued for more than ten minutes." By documenting the seizure continued for "ten minutes", it appears he is trying to refer to the guidelines of the National Epilepsy Foundation, which states that medication should be administered for any seizure that continues for ten minutes. However, this the nurses notes tell the true story. He states Ativan was given, with oxygen saturations in the 90's to 100's with the head of her bed up and bag mask ventilation being performed. He documents she was intubated, with a latent drop

in oxygen saturation to the 60's! Further supporting the inadequate oxygenation and perfusion during the failed attempts at intubation over 28 minutes. With Anesthesia Services' help, she was paralyzed and sedated. A shunt intraventricular tap was performed without complications, showing right, then left ventricular spontaneous flow, with a small decrease in pressure. He reports "CT of the head positive for left temporal edema and left lateral ventricle intraventricular hemorrhage with the ventricular catheter through the third ventricle. Question tip in the cistern." His plan was to wean and extubate when stable, check for seizure activity and keep her temp below 100 F.

The nurse's notes document a chest x-ray was performed at 0220 hours to check for endotracheal tube placement. At 0225 hours, an in-and-out cath was performed. At 0300 hours, her temp was down to 100.6 rectal. CBC, blood cultures and cath urine cultures were sent to the Lab. At 0400 hours, a cooling blanket was applied. At 0410 hours, Maggie was noted to be more awake. She was gagging on the nasogastric tube and endotracheal tube, with large amounts of formula-like emesis noted. Dr. XXXXXXXX was aware. At 0450 hours, Maggie was extubated to room air with oxygen saturations of 96-97%. Bilateral breath sounds were equal, but coarse, and she was moving air well.

At 0500 hours, pupils were equal, left greater than right. Both were briskly reactive. The mother indicated that one pupil was greater than the other at times. Maggie was placed on oxygen at two liters per nasal cannula. At 0510 hours, oxygen saturations were 88%, then increased to 100%.

According to the physician's progress note, Maggie was obtunded with arousal to deep pain and purposeful withdrawal. The PICU Flow Sheet documents her Glasgow Coma score was 11 out of a possible 15. A consultation was requested by Neurology with regard to Maggie's seizures. The records document Maggie was seen by Dr. XXXXXXXX, who felt the source of her fever was pulmonary. Her extremities were noted to be flaccid with no definite movement of the legs to foot stimulation. However, she was moving all the extremities spontaneously and appeared to be asleep. EEG was noted to show generalized bursts of slow wave activity with background suppression.

There is a physician's progress note at 1440 hours which documents a conversation was held with Dr. XXXXXXXX in Neurosurgery because of concerns secondary to fever and focal seizure lasting about one hour. The note reports there was a shunt revision, but no cerebrospinal fluid was collected for culture. The physician was obviously concerned by Dr. XXXXXXXX's response documenting, "Dr. XXXXXXXX's explained it was too early since the revision was only the day prior, but he would encourage Neurosurgery to tap the shunt for cultures, especially if there were any more seizures or she continued to have a febrile spike." The Glasgow Coma score at 2000 hours was noted to be down to a 9-10/15. CT of the head was repeated at 2144 hours showing a slight increase in size of the ventricles and a decrease in size of the intraventricular clot. There had been no significant interval change since the study of 0150 hours.

Glasgow Coma scores at 0200, 0500 and 0600 on April 11, 1999 were 10, 12 and 13 respectively out of a possible 15. A Neurology note documents Maggie had a left-sided seizure Friday evening. She was given Ativan, which “stopped the seizure, although the seizure apparently went on for at least 30 minutes prior to giving Ativan and continued after the Ativan, with patient subsequently paralyzed and sedated, then given additional Ativan.” His impression was “Nine-month-old with myelomeningocele status post focal seizure associated with fever to 103 and reported intraventricular hemorrhage. Patient’s current clinical picture most likely secondary to sedative effects of medication and prolonged seizure. He recommended close observation and determine the source of the fever.

Dr. XXXXXXX’s Neurology note of April 13, 1999 reports Maggie was doing well. She was afebrile with no recurrence of seizures. However, she did have posturing of her left hand and leg with mild clonic jerking.

Maggie was seen by Dr. Alan XXXX on April 14, 1999, who reported she was still having a series of generalized focal partial seizures despite being loaded on phenobarbital. She was also continuing not to move the left upper extremity; however, there had been some movement that day. The plan was to burst the phenobarbital to the effective level and observe.

On April 15, 1999, Dr. XXXXXXX’s Neurology note documents Maggie had two seizures the day prior and received an extra dose of phenobarbital. She continued afebrile.

On April 16, 1999, Dr. XXXXXXX reported Maggie had been seizure-free and the left-sided weakness was improving. An Ophthalmology consult was recommended. Maggie was seen by Dr. Ed XXXXX, who documented her eyes did not fix and follow. She had good brisk pupillary reaction to light, but did not cry with indirect light in her eyes. He felt she probably had decreased visual acuity in both eyes with possible paralysis of the visual cortex, which he felt would resolve.

CT of the brain was repeated on April 17, 1999 (p. 279-280), showing:

1. Slight decrease in size of the lateral and fourth ventricles, with the third ventricle being unchanged in size.
2. Resolving hemorrhage within the occipital horn of the left lateral ventricle with no evidence of acute hemorrhage.
3. Patchy areas of low attenuation within the left frontal and right occipital region, suggestive of edema.

A Neurology note documents no further seizures overnight with a Phenobarbital level at 40. Maggie was more alert. However, she had minimal evidence of fixing and following, and had clear evidence of a left hemiparesis (left sided weakness/paralysis). Her left palpebral fissure had widened and she had only slight movement of the left upper extremity. He recommended physical therapy and an ophthalmologic consult regarding her vision. The physician’s progress

note at 1000 hours reports no seizures overnight. She was moving her left upper extremity slightly more and appeared to be more aware of her surroundings. She was able to turn her eyes laterally to approaching fingers. It was felt she was improving gradually. A Neurology On-Call Note at 1140 hours states, "called to bedside for decreased level of consciousness. Eye deviation to the left and tonic posture on the left. Ativan 0.5 mg I.V. given. Child somnolent, but arousable. Responds to noxious stimuli. Eye deviation slight to the left, but extraocular movement intact. Decreased tone, left upper and lower extremity...probable breakthrough seizure."

On April 19, 1999, the physician's progress note states "no seizures since Saturday afternoon. Phenobarbital level 53 yesterday. Clinically, still has left hemiparesis with increased tone, but without tonic movement. No visual tracking yet, per mom. Waking up some yesterday." An ophthalmologic consultation was performed by Dr. Elin (p. 161). He reported that Maggie had no vision and no response to light or threat in either eye, with left hemiparesis (left-sided weakness). The impression was cortical blindness after status epilepticus. The plan was for visual evoked response if no improvement at age one year.

On April 19, 1999, there is an occupational therapy note which states Maggie was exhibiting decreased tone in the left upper extremity. She was not responding to stimulation on the left side of her face consistently, nor was she blinking to threat. She did exhibit some rooting and was able to hold a pacifier in her mouth.

On April 22, 1999, it was Dr. XXXXX's opinion Maggie had suffered a probable CVA. MRI of the brain was performed, showing persistent increased T2 white matter signal, which was felt to represent evolution of white matter edema.

Maggie was discharged on April 23, 1999.

Head CT was repeated on April 29, 1999 (p. 27-28). The impression was "low density abnormality in the right temporal parietal occipital region compatible with a right middle cerebral artery ischemic lesion. I feel this was likely present on 04/17/99. Stable ventricular size status post shunt."

On May 5, 1999, a Neurosurgery note (p. 11-12) documents Maggie had lost motor and verbal skills. Her vision was improving. Her tone had increased in the upper extremities. She was still having recurrent seizures despite being on phenobarbital, with cognitive decline. She was to continue physical therapy and phenobarbital. When the seizures were stabilized, the plan was to switch her to Tegretol.

On May 19, 1999 a SPECT scan was performed of the brain (p. 28), showing:

1. No abnormal focus of increased nor decreased activity to suggest specific seizure focus.
2. Diffuse symmetric uptake throughout the brain. In particular, no focal decreased activity is seen to suggest a complete infarction.

A cranial MR was performed on this same date (p. 29), reporting that since the prior exam, there had been a slight interval increase in size of the lateral ventricles, now moderately enlarged, with a third ventricle now moderately prominent in overall volume. The pericerebral fluid spaces bilaterally appeared moderately prominent and moderately more conspicuous than on the prior exam. The lateral ventricles were slightly asymmetric in size, left larger than right, with bilateral white and grey matter thinning.

At the time the records end, in May 1999, Maggie had not returned to her pre-incident status.

DISCUSSION:

The medical records provided for review indicate Maggie had a prolonged seizure episode lasting 1 hour and 5 minutes or 1 hour and 15 minutes (depending on which entry is correct).

Status epilepticus is a seizure which lasts greater than 30 minutes of:

1. Continuous seizure activity or
2. Two or more sequential seizures without full recovery of consciousness between seizures.

It appears Maggie had generalized convulsive status epilepticus, which is the most common and dangerous type. Status epilepticus is considered a medical emergency. Research has demonstrated that failing to treat status aggressively early on, increases the likelihood that the patient will not respond to one or even two medications.

According to the Epilepsy Foundation,

...convulsive status epilepticus is an emergency that is associated with high morbidity and mortality. The outcome depends on etiology, but prompt and appropriate pharmacological therapy can reduce, morbidity and mortality. The goal of therapy is rapid termination of clinical and electrical seizure activity; the XXXXer the seizure continues the greater the likelihood of an adverse outcome.

According to *Cecil's Textbook of Medicine*,

...status epilepticus can take either convulsive or non-convulsive forms. Convulsive status epilepticus is a medical emergency that requires timely and appropriate treatment in order to minimize serious systemic and neurologic morbidity.

Like self-limited seizures, convulsive status epilepticus may either be idiopathic and of generalized onset or secondary to bilateral spread from focal epileptogenic brain area. Convulsive status epilepticus is the first manifestation of epilepsy in about 10% of cases. More than 50% of patients with status epilepticus do not have a history of epilepsy. An

acute precipitating factor or specific cause such as metabolic abnormality, hypoxia, infection, stroke or tumor can be identified in 50-65% of patients with status epilepticus. The mortality rate approaches 30% in adults, but death usually relates to the underlying condition. Status epilepticus itself accounts for death in 10% of cases.

Treatment protocols are designed to eliminate seizure activity, and to identify and treat any underlying medical or neurologic disorder. Initial management focuses on ensuring adequate oxygenation and maintaining blood pressure. There must be unimpeded access to circulation, and cardiac function must be monitored continuously.

*Diagnostic studies should be initiated concurrently with blood obtained for anti-epileptic drug levels, blood count or routine chemistries. Brain imaging is necessary, but control of seizures **must be the first priority**. Lumbar puncture must be performed in meningitis if strongly suspected.*

The records indicate the nurse contacted the physician to inform him that Maggie looked as if she “might start seizing.” However, not only was there a failure by the nurses to re-notify the physician after the seizure began, but a failure to act for 28-38 minutes. The first physician’s order was not documented until 0020 hours. The *Standard of Nursing Care* requires that the collection of data about the health status of a patient be systematic and continuous, and that these *data be communicated to the appropriate persons*, and recorded and stored in a retrievable and accessible format. Priority of data collection is determined by the immediate physical condition of the patient. I would recommend obtaining all policy and procedures on seizure and status epilepticus; code blues, and physician notification. There are concerns about discrepancies in the records as to when the Code Team actually arrived. There is documentation the “team arrived” at 2350 hours, at which time Maggie had been seizing for 5-15 minutes. It appears no action was taken by the “team” until 0013 hours, when Dr. XXXXXXXXX was paged “again.” The first medication given for the seizure was at 0020 hours 35-45 minutes after the seizure began, depending on which record is accurate. Dr. XXXXXXXXX arrived at 0025 hours. The seizure activity continued until 0050 hours.

The Epilepsy Foundation recommends that an antiepileptic drug should be initiated whenever a seizure has ***lasted ten minutes!*** No medication of any kind was administered to Maggie for 35-45 minutes!

The guidelines recommend that after the seizure starts, the first priorities are the ABC’s of life support (airway, breathing, circulation). If feasible, and I do not see why it would not have been, an oral airway should be inserted. Oxygen was administered at 6 liters via nasal cannula. The protocol for treating status epilepticus according to the Epilepsy Foundation is as follows:

(The **bolded type** refers to actions documented by the staff treating Maggie. The remainder reflects their failure to follow the recommendations of the Epilepsy Foundation of America. The hospital protocol should be compared with the Epilepsy Foundation Recommendations.)

TIME IN MINUTES	ACTION
0-5	<ol style="list-style-type: none"> 1. Diagnose status epilepticus by observing continued seizure activity or one additional seizure. 2. Give oxygen by nasal cannula or mask. 3. Position patient's head for optimal airway patency. 4. Consider intubation if respiratory assistance is needed. 5. Obtain and record vital signs at onset and periodically thereafter. 6. Control any abnormalities as necessary. 7. Initiate ECG monitoring. 8. Establish an I.V. in one or both arms. 9. Draw venous blood samples for glucose level, serum chemistries, hematology study and toxicology screens, and to determine anti-epileptic drug levels, if applicable.
6-9	If hypoglycemia is established or a blood glucose determination is unavailable, administer glucose by direct push into an I.V.
10-20	Administer either 0.1 mg per kg of lorazepam at 2 mg per minute, or .0.2 mg per kg of diazepam at 5 mg per minute I.V. If diazepam is given, it can be repeated if seizures do not stop after five minutes. If diazepam is used to stop the status, phenytoin should be administered immediately to prevent recurrent status.
21-60	If status persists, administer 15-20 mg per kg of phenytoin by I.V., no faster than 50 mg per minute in adults and 1 mg per kg per minute in children. Monitor EKG and blood pressure during the infusion. Phenytoin is incompatible with glucose-containing solutions.
< 60	If status does not stop after 20 mg per kg of phenytoin, give additional doses of 5 mg per kg, to a maximum dose of 30 mg per kg. If status persists, give 20 mg per kg of phenobarbital by I.V. at 100 mg per minute. If phenobarbital is given after a benzodiazepine, the risk of apnea or hypopnea is great, and assistive ventilation is usually required. If status persists, give anesthetic doses of drugs such as phenobarbital. Ventilatory assistance with vasopressors is virtually always necessary.

There was a failure to spot check blood glucose and obtain labs. Ativan was not administered within ten minutes after the seizure began. Recommendations of the *Epilepsy Foundation of America* for treating status epilepticus:

*...are benzodiazepine 10 mg, repeated once, or **lorazepam (Ativan) should be given, followed immediately by intravenous phenytoin (Dilantin).*** The pediatric dosage recommendations are Lorazepam (Ativan) 0.05-0.5mg /kg for a total dose of 1.4mg, followed by Dilantin 20 mg/kg.

If seizures continue, an additional 5 mg per kg bolus of phenytoin should be given, but 80% of patients responded to this protocol. If status is refractory, the patient should be admitted to an Intensive Care Unit and anesthetized with intravenous phenobarbital, 5 mg per kg, followed by 25-50 mg every 25-50 minutes if necessary to produce a burst suppression pattern on continuously monitored EEG. Maintenance doses are 1-3 mg per kg per hour. Ventilatory assistance and vasopressors are invariably required.

In Maggie's case, Ativan continued to be given for multiple doses for a cumulative total dose of 1.7mg, but the recommendations call for Ativan *followed by Dilantin (phenytoin)*. No Dilantin or phenobarbital was administered until it appears April 13, three (3) days later, according to the physicians orders. The seizure was eventually stopped with large doses of Ativan, but since no Dilantin was administered, the amount of Ativan which lead to further respiratory compromise may not have been necessary. Dilantin terminates generalized convulsive status epilepticus in 40-91% of patients.

As noted above, the nurse's notes conflict as to when the Code Team arrived. The records reflect the team did not arrive until 0050 hours. Yet, Dr. XXXXXXXXX had been attempting to intubate Maggie since 0042 hours, without success. It appears Dr. XXXXXXXXX continued to try and intubate Maggie until the arrival of the anesthesiologist between 0105 and 0110 hours. If there was a "team" at Maggie's bedside, it is not clear why Dr. XXXXXXXXX, a Neurosurgeon, would be continuing to try and intubate when there were likely others more experienced at intubating pediatric patients. The anesthesiologist documents that despite the trouble experienced by Dr. XXXXXXXXX, **he** was able to intubate Maggie with one attempt. Maggie received a large amount of Ativan in an attempt to control the seizure *prior* to the attempt to intubate. The documentation of cyanosis on arrival of the anesthesiologist, the prolonged seizure, the hypotension, rapid pulse and low oxygen saturation, cause this delay in maintaining respiratory status to be of critical importance.

Concerning blood pressure, cerebrovascular resistance falls and cerebral autoregulation is severely impaired during status epilepticus, making cerebral perfusion directly dependant on systemic blood pressure. Relative hypotension can lead to ineffective cerebral perfusion and potentiate excitotoxic brain damage. Systolic blood pressure should be maintained at normal or above normal levels during prolonged status epilepticus using vasopressors if necessary. It appears there was a failure to initiate timely intubation and maintain adequate oxygenation and perfusion.

*According to the **Lippencott Manual of Nursing Practice**, status epilepticus is considered a serious neurologic emergency. It has a high mortality and morbidity rate (permanent brain damage and severe neurologic deficits). Management:*

- A. *Ensuring adequate cardiorespiratory function and brain oxygenation:*
 1. *Airway is established and blood pressure maintained.*
 2. *Blood studies conducted for glucose, blood urea, nitrogen, electrolytes, anticonvulsant drug levels.*
 - *Determine metabolic abnormalities that serve as a guide for maintenance of biochemical hemostasis.*
 3. *Oxygen administered.*
 - *There is some respiratory arrest at the height of each seizure which may produce venous congestion and hypoxia of the brain.*
 4. *I.V. lines established and kept open for blood sampling, drug administration and infusion of fluids.*

- B. *Controlling seizures to prevent permanent brain damage and death:*
 - 1. *Intravenous anticonvulsants such as Valium and Dilantin given slowly to ensure effective brain tissue and serum concentrations.*
 - 2. *Additional anticonvulsants as directed.*
 - *The effects of diazepam (Valium) are of short duration.*
- C. *Anticonvulsant drug levels monitored regularly.*
- D. *Measures instituted to correct acidosis, electrolyte imbalance and dehydration.*
- E. *Mechanical ventilation is employed as needed.*
- F. *If initial treatment is unsuccessful, general anesthesia may be required.*

Nursing interventions include:

- 1. *Monitor the patient continuously. Depression in respiration and blood pressure induced by drug therapy may be delayed.*
- 2. *Assist with stabilization and metabolic balance.*
- 3. *Assist with search for precipitating factors.*
 - A. *Monitor vital and neurologic signs on a continuing basis.*
 - B. *Employ EEG monitoring to determine the nature and abolition (after Valium administration) of epileptic activity.*
 - C. *Determine from family members if there is a history of epilepsy or recent infection.*
- 4. *Support patient undergoing diagnostic work-up.*
- 5. *Continue to monitor patient after seizures have been controlled as patient may succumb from cardiac involvement or respiratory depression.*

Further information needs to be obtained regarding:

- 1. The “team” that arrived at 2350 hours. The “team” that arrived at 0050.
- 2. The failure by the nurse to contact the physician and what actions were taken after Maggie started seizing.
- 3. Policies and procedures concerning seizures/status epilepticus.
- 4. Policies and procedures concerning when a code can be called and who the nurses may contact in an emergency if the attending physician is not available.
- 5. Policies and procedures concerning the availability of physicians.
- 6. Policies and procedures concerning intubation of infants/pediatric patients.
- 7. Dr. XXXXXXXX’s experience with intubation.
- 8. Are there or were there standing orders for seizures, in neurosurgical patients

9. Why did the nurse not call respiratory therapy to assist with ventilation and are the respiratory therapists trained to intubate?
10. If Dilantin was given appropriately, or why it was not.
11. If Dr. XXXXXXXXX was aware of the treatment recommendations of the hospital and the Epilepsy Foundation.

In the absence of a co-existing acute brain insult, promptly and appropriately treated status epilepticus usually is associated with a favorable outcome, especially in children. However, when status epilepticus is prolonged, the risks of death, mental handicap, other neurological abnormalities, and epilepsy increase dramatically. This underscores the need for early and aggressive intervention. Seizure etiology and duration are key determinants of outcome. Studies suggest, morbidity is lowest among children with idiopathic status epilepticus who are treated promptly. Prolonged seizures increase the risk of residual neurological problems ranging from subtle to severe. The goal of therapy is to stop the seizure as quickly as possible and minimize the adverse physiological consequences, which may include hypoxia, hypoglycemia, hypotension, and hyperthermia. The most crucial aspect of therapy is having a predetermined plan, and adhering to it.

If you should have questions or concerns, please do not hesitate to call.

Sincerely,

Kerrie E. Bradshaw, R.N., B.S.N.
Executive Director of Consulting

KEB/dm

Enclosures