



EEG (1981) I am a member of the Child Neurology Society, American Academy of Neurology, American Epilepsy Society, and National Association of Disability Examiners.

4. I was asked to review the death of Phillip Buell, age 33 mos. from an allegedly intentional blunt force trauma/fatal fall that resulted in severe cerebral edema and the prosecution of Ken Marsh in 1983. I do not know the petitioner, petitioner's counsel or anyone in the Buell family. I have received no compensation whatsoever to review the records and have done so on a *pro bono* basis.

5. I have reviewed numerous records including but not limited to paramedic reports, the autopsy photographs, the coroner's investigative report, the autopsy report, Alvarado, Sharp/Children's Hospital patient records.

6. Upon reviewing the above records, I find to a reasonable medical certainty that Phillip's Buell's head injuries were accidentally inflicted. I base my finding on the following:

A. A pre-existing weakness in the clotting system can convert a minor trauma, normally well tolerated, to a potentially life threatening event. I have reviewed the Declaration of Dr. Innis and his findings that Phillip had an abnormal clotting problem caused by Infectious Mononucleosis and Hepatitis diseases preceding his fall. This condition made Phillip "at risk" for bleeding after minor trauma.

B. On 4/27/83 at 11:00 a.m. E.M.T./paramedics were called. The history given was that Phillip fell off a couch and hit his head on a brick fireplace hearth, breaking an ashtray in his fall. (1773) The child was not responsive. He had a pulse and his pupils were reactive. His head felt "mushy." (1773) A mushy feeling of the scalp indicates Phillip's blood had not clotted.

C. Mouth-to-mouth resuscitation was begun. At 11:18 a.m., Medvac transported the child to Alvarado Hospital arriving at 11:20 a.m. (1773) At 11:41 a.m. Phillip had ventilator assisted spontaneous respirations. He was hyperventilated with an ambubag. (2280) No skull fractures or battle signs (an indication of a basal skull fracture) were observed at Alvarado Hospital. (1737)

D. Paramedics were advised of a "blood abnormality" and "mononucleosis." (1738) Phillip was given 10 mg of Decadron at Alvarado Hospital. (1827). Decadron is helpful in cerebral edema secondary to a known tumor and has not been of help in this clinical situation. In my opinion, the patient was not stable but he had not yet deteriorated.

E. The Children's Hospital transport unit arrived at Alvarado Hospital at 12:00 p.m. (1828). Dr. David Johnston, a Children's Hospital resident physician, examined Phillip. Phillip had a heart rate of 80 and a blood pressure of 100. (2231) (2225) (2231)

F. According to the pediatric record, at 12:20 p.m. Phillip was administered 8 gm of Mannitol by intravenous push (as rapidly as possible) into his jugular vein. (1737) (2225) (2231) Mannitol should not be given without an understanding of the potential risks. Mannitol is given as a last resort when impending brain death is a concern as when a pupil becomes fixed and dilated suggesting the onset of uncal herniation. If retinal hemorrhages were present this would not be an indication for Mannitol only that increased intracranial pressure was present. Since neither of these conditions were noted at Alvarado Hospital there was no reason to give Mannitol.

G. At 12:30 p.m., ten minutes after the Mannitol transfusion was initiated, Phillip's blood pressure dropped to 90. He was deteriorating due to fluid loss. 1 unit of blood was

hung and the transport left for Children's Hospital. (2225) There were no blood transfusions records provided with the Pediatric Transport Record or the Children's Hospital's medical record to determine the exact time the 500 cc's of whole blood was transfused.

H. Phillip's blood pressure dropped to 80 systolic over the next 20 minutes. The intravenous line was reported to be infiltrated and thus an ineffective pathway to deliver fluids. (2225) (2231) Mannitol injected into the jugular vein goes directly into the heart and promptly into the cerebral circulation. Increased fluid volume will initially expand the brain volume and increase intracranial pressure. In the presence of ineffective clotting in the brain this will increase bleeding into the subdural space and also lead to increased secondary cerebral edema further compromising brain tissue perfusion.

I. At 1:00 p.m., forty minutes after the Mannitol push, Phillip's pulse dropped to 46, no blood pressure was recorded, and he suffered Bradycardia (a slow heart rate and reflects a slow pulse). (2225) (2237) The doctors had concern about maintaining tissue perfusion. Phillip was then given Dopamine, Epinephrine and Atropine on his way to the CT Scan at Sharp's Hospital along with whole blood.

J. Although the child had an obvious head injury, he was not evaluated by a neurologist for almost 2 1/2 hours. At 1:40 p.m., an ICP (intracranial pressure) bolt monitor was placed in Phillip's head by Dr. Ott, a Children's Hospital staff neurologist. (2273).

7. There was no reason to transfuse Phillip with Mannitol during his transport from Alvarado Hospital to Children's Hospital. In cases of unrecognized increased intracranial pressure the improper use of Mannitol can be devastating. Mannitol increases blood flow and worsens intracranial hypertension. This coincides with the clinical deterioration in this child

after the intravenous administration of Mannitol. The exact mechanism of action of Mannitol is unknown. It is thought to produce cerebral vasoconstriction and thus reduce cerebral blood volume. This only works if the autoregulatory mechanisms of the brain are working. In head injuries, normal autoregulatory mechanisms of the cerebrovascular system are lost and Mannitol, being a potent volume expander, greatly increases cerebral blood flow while only minimally reducing intracranial pressure. If unreversed this leads to brain death and the demise of the patient.

8. Unfortunately, Phillip's blood pressure drop from the Mannitol was misinterpreted as shock and so more fluid (blood) was given further compromising the child. His increased intracranial pressure was provoked and complicated by the increased fluid volume from his coagulopathy ineffective clotting problem. This caused pressure on the vital control systems in the brain stem that regulated blood pressure. There was continued failure of the autoregulatory system of the brain to respond to hypoxia and hypercarbia.

9. In Phillip's case, there was no rationale for transfusing the pediatric patient without proper monitoring. The ICP monitor would have demonstrated whether he had adequate blood pressure in regard to his intracranial pressure. A subdural hematoma that initially is small and treatable may have secondary cerebral edema around it. Any further stress of hypoxia or hypotension would cause an abrupt deterioration in the child. This is why Mannitol should not be given without knowing the cerebral perfusion pressure which is calculated by knowing the blood pressure and the intracranial pressure in order to be certain the brain is adequately perfused or brain cells will die.

10. According to literature, Mannitol should not be given with active intercranial

bleeding and without monitoring intracranial pressure. The FDA (Federal Drug Administration) guidelines warn that Mannitol may increase the cerebral blood flow and worsen intracranial hypertension in children who develop a generalized cerebral hyperemia during the first 24 to 48 hours post injury. One of the FDA precautions is that the cardiovascular status of the patient should be carefully evaluated before rapidly administering Mannitol since sudden expansion of the extracellular fluid may lead to fulminating congestive heart failure.

11. Phillip suffered Bradycardia about forty minutes after the Mannitol infusion.(2225) Bradycardia may be the response to pressure on the brain stem from the expanding intracranial subdural hematoma and associated cerebral edema. Mannitol, by expanding cerebral fluid volume, will increase intracranial pressure. The increasing intracranial pressure will impede cerebral brain cell perfusion causing cellular injury with leakage of fluid into the extracellular space and adding to cerebral edema. The cerebral edema further adds to the increasing intracranial pressure.

12. In Phillip's case, I find the Mannitol exacerbated the magnitude of Phillip's brain swelling. I have reviewed Dr. Johnston's History and Physical Examination Report. (2231) His claim that "the transport was uneventful" and "no other medications were given" is inaccurate. The medical evidence indicates that Phillip had an expanding hematoma due to a consumptive coagulopathy, a complication of both head trauma and inadequate tissue perfusion. Blood loss with placement of the ICP monitor reflect the child's ineffective clotting. His poor brain perfusion led to excessive cerebral edema.

13. In short, it does not appear that anyone either recognized, reported or considered

Phillip's ineffective clotting problem, disease, or therapy intervention in diagnosing the massive cerebral edema as child abuse murder. These medical factors contributed to this child's deterioration and fatal outcome of massive cerebral edema.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Executed at \_\_\_\_\_, California on October \_\_\_\_\_, 2002.

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By: THOMAS A. SCHWELLER, M.D.