September 29, 2014

Michael J. Cronkright, Esq.
Kronzek & Cronkight, PLLC
420 S. Waverly Road, Suite 100
Lansing, MI 48917

Re: Preliminary Report on Naomi Burns

Dear Mr. Cronkright,

I have reviewed all of the materials forwarded to me including the depositions on Dr. Mohr and Mr. and Mrs. Burns, and the reports by Dr. Barnes, Dr. Guertin, Dr. Tawansy and Dr. Scheller. I have also reviewed all the radiological studies and any pertinent clinical information which you forwarded to me.

The clinical scenario in this patient has been well reviewed by expert witnesses.

In summary Naomi Burns was born by C-section after a 3 1/2 to 4 hour labor in which 4 attempts were made using vacuum assistance to extract the baby. According to Brenda Burns, the baby was born with bruising around the right ear. Subsequently the child did well except for a bout of gastroenteritis. Both Mr. and Mrs. Burns were at home during the child's first 3 months of life. It should be noted that the head circumference went from the 75th to the 90th percentile during early development. Several days prior to March 18, 2014, Naomi had episodes of emesis and the mother noted that she was also pale. The father indicated while holding Naomi and after getting off the phone with his wife Naomi slipped forward and, using his hands, he grasped her quickly whereby he scratched her face.

Persistent emesis and paleness and suspected dehydration led the parents to bring the baby to the hospital on several occasions where hydration was administered and the patient was discharged. Ultimately, however, on 3/18/2014 the patient was admitted and required intubation secondary to respiratory distress. An MRI scan was obtained and while initially was read as showing no significant abnormality with respect to acute hemorrhage, although widening of the extracerebral spaces bifrontally and bitemporally was present, a second reading indicated extracerebellar hemorrhage posteriorly and bilaterally. An initial chest x-ray showed increased density in the left lung and the endotracheal tube was noted to be abnormally positioned in the upper portion of the right mainstem bronchus and most likely atelectasis was present in the left lung. Subsequently the patient was extubated and the chest x-ray returned to normal. Whether or not any episodes of hypoxia occurred is difficult to determine since no documentation was obtained of a significant decrease in oxygenation. The patient was discharged on 3/24/2014.

Recurrent emesis and possibly seizures occurred and the patient was reportedly readmitted 6 to 8 hours later. A second MRI scan was obtained and showed new subdural blood around the left and right cerebral hemispheres bilaterally. There was no change in the appearance of increased density posterior to the left and right cerebellar hemispheres (e.g. suspected blood posterior to the cerebellum). An ophthalmologist consult was requested and obtained. This examination revealed retinal hemorrhages bilaterally. A skeletal survey was also obtained which was entirely normal and there was no evidence of any fractures.

Noted during multiple examinations was bulging of the anterior fontanelle suggesting possible increased intracranial pressure which could have been episodic. On two separate occasions lumbar punctures were obtained without any mention of opening pressures. Laboratory tests revealed thrombocytosis and elevated platelet counts in the 700,000 to 900,000 range. A screen for Von Willebrand's disease suggested that this may be present. A hypercoaguable state may have been present and significant (infra vide).

Independent imaging review: MRI scan 03/18/2014

The axial FLAIR images demonstrate widening of the extracerebral space around the frontal and temporal lobes. There is a very thin area of low signal intensity adjacent to the sulci and gyri similar in signal intensity to cerebrospinal fluid. Surrounding this is a slightly larger collection which is only slightly more hyperintense. Posterior to the cerebellum there is a thin 1 to 2-mm in thickness high signal abnormality which follows the contour of the cerebellum from the torcula Herophili to the sigmoid sinuses bilaterally. On the gradient echo (T2*) images there is a very subtle difference between what appear to be two separate fluid collections surrounding the frontal and anterior temporal lobes. The more external collection is slightly hyperintense compared to the more internal collection around the brain substance. There appears to possibly be a thin membrane separating these two collections. Posterior to the cerebellum the signal abnormality is both mixed high and low. On the conventional fast spin echo T2 weighted images the two hyperintense fluid collections around the cerebral hemispheres appear essentially iso-signal with each other with a membrane separating these two collections. The rim around the posterior cerebellar hemispheres is low in signal intensity. On T1 imaging there is widening of the extracerebral space and this extracrebral fluid is hyperintense compared to CSF. There is small linear area of hyperintensity along the membrane separating the two fluid collections on the left and to a lesser degree on the right which may represent thickening of the dural membrane itself. This is not convincing for recurrent subdural bleeding. The extracerebellar “collections” are also hyperintense on FLAIR and T1 imaging and slightly greater so than the extracerebral fluid. There is a small extracerebral collection T1 hyperintense immediately to the right of midline as best seen on the sagittal views adjacent to the superior sagittal sinus along the medial parietal lobe which is also hyperintense on FLAIR imaging and isointense to brain on T2 and slightly ihyperintense to CSF on T2 imaging. This may represent a small collection of

subdural blood. There is no scalp soft tissue swelling.

MRI scan O3/27/2014

Since the prior MRI there is now a very high signal abnormality occupying what previously appeared to represent an external and separate fluid collection surrounding the frontal and anterior temporal lobes. The appearance is that of a relatively recent occurrence of bleeding into the subdural space (e.g. acute or subacute subdural hematoma). There is a change in the signal abnormality around the cerebellar hemispheres which is now higher in signal intensity on T1 imaging suggesting some acute hemorrhage into the subdural space right greater than left. The appearance of suspected small subdural hemorrhage along the medial right parietal lobe is unchanged. There is no scalp soft tissue swelling.

Skeletal survey 3/27/2014

There are no fractures identified. There is no indication of a skeletal anomaly such as osteogenesis imperfecta.

Assessment:

Based on the initial MRI scan of 03/18/2014 there is an underlying abnormality which appears to represent chronic bilateral extracerebral fluid based on its signal characteristics. These fluid collections most likely consist of a chronic bilateral subdural hematoma on the left and right around the frontal and temporal lobes. There also appears to be smaller CSF-like fluid collections around the temporal and frontal lobes, as well, located in the subarachnoid space. There is **no** significant effacement of the contiguous sulci or gyri. In this regard, there does not appear to be any significant increased intracranial pressure at the time that the study was done. In view of the patient's history of a traumatic delivery, the findings most likely represent bilateral chronic subdural hematomas. The amount of cerebro-spinal fluid around the frontal and temporal lobes is not excessive and may be within normal limits. Typically subdural hematomas resolve over time but on occasion they may not resorb and actually may continue to increase as bilateral subdural hygromas. This would clearly explain the patient's continued enlargement of the head. This appearance is indicative of cephalocranial disproportion. As a result of previous subdural hematoma a vascular membrane may develop in the subdural space containing vessels which are particularly fragile and susceptible to rehemorrhage. In addition, the normal cortical veins which drain the brain of venous blood become stretched across the subdural space (as they course from the brain substance to the diploic space in the calvarium containing diploic veins and also to the dural sinuses) as the subdural space becomes abnormally widened. This too makes the veins in this area particularly susceptible to rehemorrhage either with no significant insult or only very minor trauma. The presence of increased signal on the FLAIR images posterior to the cerebellar hemisphere has been interpreted to represent bilateral

cerebellar subdural hematomas. This has been explained by Dr. Mohr as being caused by violent shaking of the baby. This is being interpreted in conjunction with Dr. Mohr's feeling that the retinal hemorrhages are pathognomonic for shaken baby syndrome. It appears generally agreed that the episode of the baby falling from the father's arms would not appear to be substantial enough to induce this kind of injury (i.e. retinal hemorrhage and bilateral extracerebellar subdural hematoma.

An alternative, and more realistic, explanation is that sinovenous thrombosis may have occurred as a result of the patient's state of dehydration associated with suspected acute episode of gastroenteritis (acute and chronic illness are associated with sinovenous thrombosis as is clearly indicated in the medical literature on this topic, see deVeber et al). It is difficult to conceive how a post traumatic (e.g. shaking injury) cerebellar subdural hematoma would occur preferentially in this baby to an acute or subacute subdural hematoma around the cerebral hemispheres when the veins in this latter region would be particularly susceptible to rupture because of their increased fragility as described above. It is not logical given the nature of the injury that is suggested as the cause of injury that a subdural hematoma acutely would **not** have occurred in the **extracerebral subdural space** but rather only in the **extracerebellar space.**

It is a well-established fact based on the experience of multiple pediatricians, pediatric neurologists and neurosurgeons that while sinovenous thrombosis is a relatively rare phenomenon occurring in only .67/200,000 patients based on the Canadian Stroke Registry established in 1992, it is associated with a number of intracranial injuries including venous infarction which is often hemorrhagic but also subdural hematoma. The information provided to me suggests that there was a 6 to 8 hour window since the patient had been discharged from the hospital. According to Brenda Burns, the patient, Naomi Burns, was in the custody and presence of both her parents during that time. There is no event established in that time frame that would account for the new onset of bilateral subdural hematomas in a pre-existing chronic subdural hematoma secondary to trauma. A more plausible and better explanation is the presence of sinus venous thrombosis with its subsequent complication of recurrent subdural hematoma.

It has been stated that the patient did not demonstrate any evidence of papilledema on physical examination that would indicate chronic increased intracranial pressure. However the presence of sinovenous thrombosis secondary to dehydration and thrombocytosis may result in transient increased intravenous pressure, transient increased intracranial pressure (intermittent bulging of the anterior fontanelle) and stagnant venous flow that could result in retinal hemorrhage.

The explanations provided above are a much more reasonable consideration when one analyzes all of the facts in their proper context.

It must be emphasized that the patient's cephalocranial disproportion put her at

risk for developing subdural hematoma in and of itself. There is no reason to believe that the chronic subdural hematoma following failed vacumn suction extraction of the fetus and caused by traumatic birth was related to any other event in Naomi's life up until 3/18/2014. Because of the fragility of the veins in the extracerebral subdural space it would be extremely unusual to develop bilateral extracerebellar hematomas with the violent shaking injury as is suggested without also disrupting the veins in the extracerebral space.

Summary: The evidence in this case can only be logically interpreted by a unifying theory that does not involve NAI.

1. Naomi's birth was traumatic and there is supporting evidence well accepted by the medical community, that the kind of trauma she sustained could easily be accompanied by subdural hematoma(s).

2. Over time, rather than resolving, these subdural hematomas increased in size leading to her head circumference increasing from the 75% to the 90%

3. As described above, widening of the subdural space and the presence of a vascular membrane increased Naomi's risk of rebleedding with either insignificant or minimal trauma.

4. Prior to her first admission, she had repeated episodes of vomiting and paleness, and a diagnosis of gastroenteritis was entertained. Dehydration requiring intravenous fluid replacement was required. Laboratory tests revealed thrombocytosis.

5. Reinterpretaion of the MRI suggested bilateral extracerebellar subdural hematomas

6. After discharge on 3/24 the patient was readmitted after a relatively short interval because of seizures. She was in the custody of both parents during the discharge interval.

7. The repeat MRI showed new bilateral extracerebral subdural hematomas and retinal hemorrhages.

8. Child protective services (Dr Mohr) indicate NAI

9. The chronology of events and the medical laboratory tests and imaging studies are much more consistent with a diagnosis of sinovenous thrombosis as cause of the recurrent extracerebral and extracerebellar subdural hematomas. It is not pathologically correct to expect extracerebellar SDH to occur without recurrent significant extracerebral SDH on the 3/18/2014 MRI (Note: the extracerebral small SDH adjacnt to

the medial parietal lobe is very small) when the latter is much more likely to be present given the purported nature of the injury inflicted on Naomi. Sinovenous thrombosis would also explain all of the findings.

I swear by the pains and penalties of perjury that the statements issued by me in this report represent to a greater degree of medical certainty that the abnormalities described on the MRI scans and opthalmological exam are due to sinovenous thombosis and not NAI. I reserve the right to add or delete any opinion should new information arise which may indicate doing so.

Sincerely yours,

Gregory M. Shoukimas, MD, PhD