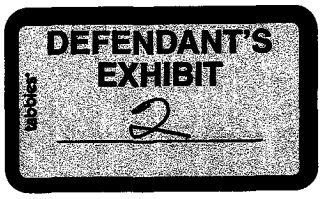

JAN E. LEESTMA, MD, MM
NEUROPATHOLOGY

January 9, 2011

Victor Abreu, Esq.
Public Defender
Curtis Center, Suite 545, West
Philadelphia, PA 19106

Dear Mr. Abreu:

At your request I now offer an appended report to the original report of 12/15/2010, because I have received information that was not available to me at the time of the original report.

This report deals with the death of a minor child, Jakarenn Gunter. I was provided the following materials that form the basis of this report:

1. Direct appeal opinion (Bourgeois).
2. Trial testimony transcripts [opening statement, Booth; EMT Burgess; Dr. Benton; McLaughlin, Dr. Kuffel, Dr. Rouse, Dr. Oliver; Dr. Senn; Dr. Chrz; Dr. Akhtar, Beckett]
3. Transcript of April 20, 2010 hearing before Judge Jack.
4. Forensic odontology reports of Dr. Senn, Dr. Chrz.
5. Autopsy report of Dr. Rouse.
6. Neuropathology report of Dr. Kagan-Hallet.
7. Affidavit of Dr. Spitz.
8. Visceral autopsy slides, A-02042, Wilford Hall Medical Center, TX.
9. Medical records and reports re: Jakarenn Gunter.
10. CT scan of Jakarenn Gunter.
11. Brain autopsy slides and photographs.
12. Autopsy photographs.
13. Copy of hand written notes of US Attorney re: Dr. Elizabeth Rouse.

To reiterate, very briefly, my understanding of the case is that the child, Jakarenn Gunter was a 2 year old female in the care of Mr. Bourgeois. The family of Mr. Bourgeois was frequently traveling with him in his commercial truck as he conducted his business of hauling freight. The Government has alleged that the child had apparently been maltreated and beaten by Bourgeois for some time before the child's death largely over matters concerned with toilet training. On the morning of July 27, 2002, Bourgeois arrived with goods on his truck to be delivered at the Corpus Christi Naval Air Station. During the course of positioning his truck at a loading dock at this facility an incident was reported to have occurred between Bourgeois and the deceased child during which Bourgeois was reported to have slammed the child's head against a window in the truck and the dashboard. After this occurrence the child was said to have become limp and unconscious. The child was brought to a medical facility where she never regained consciousness and died on July 28, 2002.

An autopsy was conducted by Dr. Rouse of the Armed Forces Medical Examiner's office. Dr. Rouse found multiple subgaleal hemorrhages (estimated 10 impacts), a 2ml. subdural hematoma with "minimal" organization, subarachnoid hemorrhage and cerebral edema, parenchymal brain damage including axonal pathology, bilateral retinal hemorrhages and optic nerve sheath hemorrhages, many areas of skin injuries of multiple ages. A recent bruise of the left shoulder was described also. She concluded that the child had died from closed head trauma with subdural hematoma with the manner of death homicide.

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A neuropathological examination of the brain was performed by Dr. Kagan-Hallett who reported the brain to be swollen, with a subarachnoid hemorrhage and a subdural hematoma that in its oldest portions was estimated to be a few weeks or approximately 10 days in duration. Dr. Kagan-Hallett also found axonal injury in the white matter with macrophage responses in the tissue. She also reported organizing contusion infarcts in the brain 7+ days in duration. The term "organizing" refers to a healing process ongoing in the hematoma that takes time to evolve indicating that the oldest part is 10 days or more old from time of death.

From the report of Dr. Kagan-Hallett, it is obvious that the child had a subdural hematoma, though it doesn't appear to be very large, and appears to have an acute component in addition to the component that was healing as discussed above. The older and larger component's age is derived from the microscopic appearance of the subdural hemorrhage and the healing process which occurs over time after the inception of the hematoma. This would clearly indicate that the oldest part of this lesion predated her final hospitalization by many days or longer. It is common to have relatively fresh red blood cells in a chronic subdural hematoma. Such cells could have appeared immediately before the child died or up to 2-3 days previously. Such "recent" bleeding cannot be aged any more precisely. It cannot be known when the most recent bleeding into the hematoma occurred within a window of two days or how it occurred. It also cannot be known if this acute component contributed or caused death.

My examination of the gross autopsy photographs relevant to the head and brain revealed a primarily right sided apparently recent subdural hematoma of the vertex dura from its external appearance. It appears relatively thin with a probable volume of a few ml. Photographs of the reflected scalp reveal subgaleal hemorrhage a few cm diameter above the temporalis muscle approximately over the coronal suture on the right side a few cm from the midline. The hemorrhage is dark red-black in color. The anterior reflected scalp has a number of apparent impact sites with the largest about 1 cm. These do not appear to be full thickness scalp hemorrhages and their age is not determinable from the photographs. The posterior scalp in the occipital region behind the right ear shows another subgaleal hemorrhage about 2 cm with indistinct margins and possibly some color changes tending toward brown rather than dark red-black.

My examination of the visceral autopsy slides revealed that several sections of the scalp had deep subcutaneous hemorrhages with inflammatory reactions of varying age and degree, ranging from bruises that had preserved red cells (2-3 days or less in age), early inflammatory cell changes (a few days or older), granulation tissue and early scar formation (a week plus), and essentially healed skin lesions (many weeks old). The lungs showed congestion, edema and an "early" pneumonia with pulmonary macrophage activation. The kidneys, adrenals, liver, spleen and gall bladder showed congestion. The pancreas was autolyzed. One section of what was probably the tongue showed old lesions in the process of healing. The heart showed an area of myocardial hemorrhage and necrosis with reactive cells present. I concluded that the child had an early pneumonia which is not uncommon on individuals maintained on a respirator but could have preceded hospitalization. The skin lesions clearly represent dermal injuries of multiple ages from within two days of death to many weeks before death. The tongue shows a healing injury. It seems inescapable that this child had been physically assaulted over a considerable period of time before death.

On 1/5/2011 I received 6 neuropathology brain slides from the University of Texas (Kagan-Hallett's slides). Examination of these materials revealed a slide of dura and superior sagittal sinus that contained a thin chronic subdural membrane attached to a recent blood clot. The thin membrane was composed of 3-5 layers of fibroblasts and other cells. This was not attached to the dura. The dura contained many layers of chronic inflammatory cells and fibroblasts attached to the arachnoid granulations. The thickness of these reactive cells were sufficient to place the age of the inception of the bleeding in that location to about 10 days from time of death in keeping with Dr. Kagan-Hallett's estimations in her report. In addition, I found a thrombus in the superior

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sagittal sinus that was not attached to its wall and another thrombus separate from this one lying free. This was clearly a premortem thrombus and showed fibrin, layering and peripheral cellular reactions indicating it was likely attached to the wall of the sinus at one time. The estimation of this process' age is many days, perhaps a week from death

In addition I noted several necrotizing lesions of the surface of the brain that spilled over into the arachnoid and subarachnoid space. These all contained chronic inflammatory cells, some with pigment in them including macrophages and early capillary and vascular reactions to these processes. There was a chronic subarachnoid hemorrhage with recent component including chronic inflammatory cells. In one section this same process appeared to involve the subependymal brain tissue and ependyma. These lesions may represent venous infarctions whose most likely cause was cerebral venous thrombosis. The presence of macrophages, capillary reactions and pigmented macrophages makes these lesions probably about 7 days or more from the time of death.

Given the many scalp bruises of various ages it is reasonable to conclude that the child had suffered many impacts to the head over time which could have led to the subdural hematoma. It is not possible to determine if the physical actions ascribed to Mr. Bourgeois on July 27, 2002, caused new bleeding into the subdural hematoma since it is not possible to histologically age acute blood in a bruise more precisely than indicating that the red cells could have arrived in the hematoma anytime within about two days of death. My examination of the CT scans reveals a relatively small recent subdural hematoma along the falx, mostly posteriorly along with some mixed density material that probably represents the older component of the subdural mentioned above.

The finding of retinal hemorrhages and optic nerve sheath hemorrhages in this case do not provide any information regarding the mode of injury of this child. They only indicate that the child increased intracranial pressure during the course of her hospitalization prior to death. The cause of the increased intracranial pressure was in part due to cerebral edema which in turn has several causes (hypoxia-ischemia, prior old brain injury, new concussive head trauma, and other processes). The subarachnoid hemorrhage's cause may be due to reperfusion injuries secondary to resuscitation and disturbed perfusion of the brain in the face of high intracranial pressure and/or venous thrombosis intracranially. The myocardial focal hemorrhage and necrosis could have been due to an intracardiac injection during resuscitation, though I find no record of this having been done.

Regarding a critical question, that of when the fatal head injuries to the child were caused, it is clear that she had sustained a number of head impacts allegedly due to having been beaten over period of time. The subdural hematoma's cause is likely physical trauma but it cannot be determined whether this was inflicted or accidental. The role of the subdural hematoma in the death is somewhat problematic because its volume is so small (a few ml.). This volume should have been easily compensated for by reabsorption of cerebrospinal fluid and thus would not cause a fatal collapse by itself. Further, chronic subdural hematomas can bleed on their own without any new trauma after the initial event or can be exacerbated by new incidents of head impact. Nevertheless, it appears that the child's condition deteriorated after the reported incident in the father's truck. It should be pointed out, however, that there is very little information derived from the autopsy that can accurately pin point what physical injury resulted from this alleged incident or that it directly caused the child's death. Much of the pathology in this child is not acute. The scalp injuries are of variable ages in which the hemorrhages show aging of red cells, cellular repair and inflammatory reactions and healing-scarring. The brain lesions range from possibly a few days in age to days to weeks old. The child may have had a coagulopathy which appears to have included a sagittal sinus thrombosis and possibly cortical venous infarctions. These processes could have resulted in the child's death at any time with or without any new episode of trauma.

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I hold these opinions to a reasonable degree of medical and scientific certainty.

Sincerely yours,

Jan E. Leestma, MD, MM

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