STATE OF WISCONSIN CIRCUIT COURT DANE COUNTY

STATE OF WISCONSIN,

Plaintiff,

Case No. 2007 CF 2381

--vs.--

Jennifer Hancock,

Defendant.

AFFIDAVIT OF DR. MICHAEL STIER

I, Michael Stier, M.D., hereby declare, under penalty of perjury under the laws of Wisconsin and the United States of America, that the following is true and correct.

1. I am a physician licensed to practice medicine in the state of Wisconsin. I am board certified in anatomic and clinical pathology and forensic pathology. I received my medical degree from the University of Wisconsin in 1994. Afterwards, I performed postgraduate studies in the areas of anatomic and clinical pathology, also at the University of Wisconsin. From 1998-2000, I undertook a postgraduate study of neuropathology at the University of Virginia. From 2000-2001, I undertook a postgraduate fellowship in forensic pathology at the Milwaukee County Medical Examiner's Office. Currently, I am an associate professor of forensic pathology at the University of Wisconsin School of Medicine and Public Health. I have been a practicing pathologist since July of 2001.
2. I conducted the autopsy of LW on September 12, 2007. My findings at that time included: a bilateral, thin subdural hemorrhage of varying ages; a skull bone irregularity;

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and a left femur fracture. With these findings, I made an initial conclusion that this was a non-accidental fatality.

3. On March 31, 2009, I testified for the prosecution in the trial of Jennifer Hancock, summarizing my findings of the autopsy of LW. I testified that LW had a thin, bilateral, subdural hematoma along with swelling in his brain. I also testified to several negative findings: an absence of impact focus to the scalp, or nothing that would imply a direct impact to the head, and no evidence of an impact to the skull. That is, I did not observe a skull fracture or any other signs of impact injury. In addition, I testified that LW had a very specific type of fracture to the distant end of the main bone of the thigh, in the growth plate of the left femur. At trial, I stated that I believed that these findings, absent any clear indication otherwise, are indicative of non-accidental head trauma. However, based on my clinical experience since trial and reference to more recent literature, I can no longer draw this conclusion to a reasonable degree of medical certainty.
4. Since the trial, I have observed that thin, bilateral subdural hematomas with no external evidence of trauma—precisely the findings in LW's autopsy that led me to infer a non-accidental cause—can actually occur from scenarios of hypoxic demise. These include opioid/analgesic deaths, drowning, and lung disease. I have witnessed this in autopsies I have performed.
5. Since the trial, I have conducted autopsies on people who had patterns of bleeding in the brain that were identical to those I observed in LW, but who had not experienced any identifiable head trauma. These autopsies included multiple people dying from drugs, one person who drowned, and one person who died of lung disease, all of whom had no identifiable head trauma, but exhibited this same type of bleeding. In light of the recent

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autopsies I have conducted, and I can no longer support an opinion that LW's intracranial findings could only have resulted from non-accidental causes. This is because I now believe that the acute subdural hematoma exhibited by LW could be explained by a process other than abuse, such as a lack of oxygen. This is plausible because LW had an identifiable virus in the heart tissue as well as an older or chronic hemorrhage in the subdural space that would put him at risk for a superimposed acute bleed precipitated by a hypoxic event.

6. At the time of trial, I believed the swelling in LW's brain was a result of his injuries, which I attributed to non-accidental injury. Since the time of trial, I no longer believe that the swelling in LW's brain could only be explained as a result of traumatic brain injury. I believe a process of hypoxia/ischemia and resuscitation may explain the swelling.

7. In the course of autopsy, a virus was identified in LW's heart tissue. I reported it as being of undetermined significance, and I did not list it as a potential cause of death. In light of my current understanding of alternate causes of hemorrhage, the virus may be of more significance to LW's demise. The heart virus provides and alternative, viable explanation of LW's demise.

8. Upon receiving LW's body for autopsy, I was told that LW had a radiographic skull fracture. I examined the area of the suspected fracture visually and microscopically, and identified none. Furthermore, there was no scalp injury as it relates to the supposed skull fracture or the genesis of the subdural hemorrhage.

9. At trial, I testified that I did not observe a skull fracture. However, when the prosecutor asked me to explain what I described as a skull-bone irregularity—specifically, whether I

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could say that the irregularity was a fracture—I responded "I'm not saying that it is, I'm not saying that it isn't." At that time, I felt pressured by prosecutors in the case against Ms. Hancock to leave the door open to the possibility of a skull fracture. However, if called to testify today, I would say definitively that there was no skull fracture.

10. In general, and as I testified at trial, pathology confirms radiology. This means that when radiologists observe potential irregularities on the scans of the patient, my role as a pathologist is to examine the body in question and reach an independent conclusion. Pathology is often authoritative when there is a conflict between radiology and pathology. In my opinion, while radiology reports may have indicated that there was an irregularity on LW's skull, the autopsy definitively established that there was no skull fracture.

11. At trial, defense counsel asked few questions with respect to the alleged skull fracture. Had Ms. Hancock's lawyer asked me more directly about this issue, I would have testified more clearly that there was no skull fracture. Had defense counsel explored the issue more thoroughly, I would have testified about the significant force required to cause such a fracture in an infant and that, given that force, the lack of scalp injury or other signs of head trauma were medically significant and supported my finding of no skull fracture.

12. During the autopsy, I also noted a bucket handle fracture on LW's left femur. At the time of trial, I considered this a feature of abuse. I would no longer regard a bucket handle fracture as a definitive indicator of abuse. That area of the femur is prone to fracture in children, and it is not known how much traction or torsion force is required to cause a fracture. Moreover, during initial emergency resuscitative and stabilizing efforts, at least

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eight attempts were made to insert an intraosseous line into LW's femur. The attempts at placement of the line under the conditions present during LW's admission could have caused the bucket fracture. Therefore, had I performed LW's autopsy in the present day, I would have reported the bucket fracture finding without indicating a conclusion that it was caused by abuse.

13. I am now unable to conclude to a reasonable degree of medical certainty that LW's injuries or death were the result of abuse. In my opinion, a death from natural causes may explain the findings at autopsy: a thin film subdural hemorrhage with both acute and chronic bleeding; a heart virus; very little swelling of the brain; no mass effect from the subdural hematoma; no retinal hemorrhages; and absolutely no evidence of any blunt force trauma to the head.

14. If I were to testify at trial today, I would not testify that LW's death was caused by non-accidental inflicted injury. Instead, I would testify that there is no definitive cause of death. In other words, the cause of death is undetermined.

Dated: 2/2, 2018.

Michael Stier, M.D.