STATEMENT OF WITNESS

STATEMENT OF BRIAN HERRON

DATED THIS 4th DAY OF December 2008

I, BRIAN HERRON, declare that this statement is true to the best of my knowledge and belief and I make it knowing that if it is tendered in evidence at the Inquiry I will be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

1. The Inquiry has disclosed a number of documents to me. Where I make specific reference to a document in my statement I have given the number of the relevant page. It is important to note that this statement made to the Robert Hamill Inquiry relates to events ten years previously and is the text from a verbal interview and not a prepared report to specific written questions.

2. I am a Neuropathologist and am presently a Consultant at the Royal Victoria Hospital (RVH), Belfast. I am initially a medical doctor with the qualifications MB BCh BAO. Subsequently, I obtained the DRCPath, which is a Diploma of the Royal College of Pathologists. I then became a Member and subsequently a Fellow (FRPath) of the same College. My specialty is neuropathology, so I am a Consultant Neuropathologist. I am also a general pathologist, in that I run the adult autopsy service for the majority of the hospitals in Belfast. In addition, I am an autopsy examiner for the Royal College of Pathologists final FRCPath examination.

3. Some important points need to be made in order for the inquiry to understand my job and my involvement in the case of Robert Hamill:

    - I am a full time employee of the NHS based at the Royal Victoria Hospital. I work in a team with one other Neuropathologist and a number of scientific and secretarial staff.

- I am not an employee of The State Pathologist or the State Pathologist's Office.
- I am not an employee of the Northern Ireland Office.
- I am not an employee of the DPP and have no contract with the DPP.
- I am not employed by the police, nor have I ever been.
- In each case I report I have no knowledge of arrests or charges of any specific individuals. In the case of Robert Hamill, I do not remember having any contact with police. This point is also made at the end of my report
- Neuropathology is a highly specialised job. There are only a handful of Neuropathologists in the UK or Ireland with the expertise to give opinions in complex forensic neuropathology trauma cases. As far as I know most of them work for the NHS.
- My job in the NHS involves the diagnosis and management of neurological and neurosurgical disease for all patients in Northern Ireland. This includes the diagnosis and management of all patients who have a tissue diagnosis of a brain tumour. This is the job for which I am paid.
- A Forensic Pathologist investigates deaths and especially suspicious deaths. In most cases they do not need the expert opinion of a Neuropathologist. In some instances there are issues that the Forensic Pathologist feels is beyond their expertise and they seek advice.
- When there is a head injury or complex neurological disease they may seek advice from a Neuropathologist.
- In this current year I will be asked for advice on approximately 70 forensic cases from Northern Ireland. The majority of these cases involve complex neurological disorders, suspicious deaths or murders.
- Most cases take at least 10 hours to finish. One recent case from outside Northern Ireland took me 55 hours excluding court time to complete. <u>This is on top of my NHS job</u>.
- When asked for an expert opinion by The State Pathologist or any other doctor in Northern Ireland, for instance in the case of Robert Hamill, I do not charge for this work and do not receive any fee whatsoever. I am

under no obligation by means of any contract to do this work. I do it as well as, and on top of, my work for the NHS.

- Regarding the technical aspects of reporting a case, each case must go through a number of stages.
- The brain is retained and suspended in a chemical called formalin in order that it hardens and can be examined more accurately. This takes at least one month and sometimes longer. At a time convenient to the Forensic Pathologist and the Neuropathologist (as both should be present) the brain is dissected. A description is made of the abnormalities visible to the naked eye. Small pieces of brain tissue are then taken and examined under the microscope. It may take several days or weeks to prepare these samples. These are reviewed by the Neuropathologist. Depending on what they show, more investigations are performed on the tissues. This is what happened in the case of Robert Hamill.
- The small pieces of tissue began processing in my laboratory on 25/7/97 and were initially available for me on 6/8/97. Further work was requested on these pieces of tissue. While all of this work is going on there may be clinical discussions about a case. In the case of Robert Hamill, the issues that needed to be resolved clinically and pathologically were some of the most complex of any case in which I have been involved in 16 years as a Neuropathologist. These complexities are reflected in the discussions of other experts instructed in this case. One of the issues was the possibility of Neuroleptic Malignant Syndrome being a potential diagnosis and the other was the significance of diffuse axonal damage-these are discussed later.
- When asked as an expert for a report, the expert must be allowed to access the relevant medical literature; otherwise the report may be deficient.
- At the time of reporting this case there was a dearth of literature on Neuroleptic Malignant Syndrome and a burgeoning literature on diffuse axonal injury. It must be also noted that in 1997 there was not the availability of the internet to obtain medical papers. The papers often

had to be ordered from the British Library or other sources and they could take weeks to arrive. It was absolutely necessary to have and review the literature in this complex case. Some degree of the complexity is reflected in the expert opinions obtained from other sources, and the research papers alluded to in the remainder of this report.

- Despite all of these factors my report was ready about three months after the brain dissection which is in very good time considering the points discussed above. My report was typed on 29/10/97 and I can think of no mechanism by which a proper report could have been safely prepared any quicker.

4. This is the first statement that I have given in relation to the death of Mr Hamill.

5. Once I had examined the brain I would normally have recorded my thoughts on the brain pathology. I can confirm that a copy of these is at page 31395, although the report I would have sent to Professor Crane would have contained 2 additional pages containing a clinical summary. Professor Crane would then assimilate my opinions into his Autopsy Report (pages 09559 to 09569). At any stage that there may have been discussion between us. I would have had the opportunity to discuss the findings and clarify any issues in the understanding of my report before he finished his report.

6. Before my examination Professor Crane would have provided me with a clinical summary of the case. The information in that clinical summary appears in Professor Crane's manuscript notes of the examination under the 'history' section on page 31515 with a continuation on page 31519. I would normally have dictated my findings on a tape and not written any notes. Therefore I have no written notes on this case and the dictation tapes are not routinely retained. I do not specifically recall whether I saw Mr Hamill's hospital notes, containing pages 38549 to 38724, 38950 to 38951 and 38972 to 38973, but I may have done. Professor Crane may have had them and I could have seen them if I had wanted. Anything in the notes detailing the tests and the results of tests carried

out on Mr Hamill before he died would have been available to me. Professor Crane would have had the book of post-mortem photographs and shown them to me to give me an overall picture of what was going on. However, I would not have become too involved with the extent of the external injuries as that is a forensic aspect of the case.

7. I may have been provided with further information in addition to that in the clinical summary by the doctors managing the care of Mr Hamill. This was not unusual because I would have known some of the RVH doctors involved in the care of Mr Hamill quite well. There were also issues at the time about suggested avenues to pursue from a diagnostic point of view. One of the major issues in this case was the possibility of diagnosing Mr Hamill with Neuroleptic Malignant Syndrome (NMS), which I shall discuss below. I do have memories of speaking to people about whether NMS formed part of the diagnosis, but I made no written records of these conversations.

8. There are a number of stages to a brain examination. The first stage is to look at it macroscopically (with the naked eye), then small pieces of tissue are taken and they are examined under the microscope. The dates of 7/8/97 and 29/10/97 referred to in my report at page 31396 are secretarial typing dates. The first date may refer to the first part of the procedure when the macroscopic brain description is typed, and the second date may refer to the date when the second stage of microscopic examination is typed.

9. The usual procedure would be that Professor Crane would attend a 'brain cut' where cases referred to us are dissected and blocks of tissue taken to enable microscopic examination. The blocks are a few centimetres in size and are put into wax to make it easier to put a blade through them to take small slivers of tissue. At this stage, ten years later, I cannot remember specifically, whether the brain cut took place in the Forensic Department or in Neuropathology. Since the tissue blocks have a forensic number on them it is likely the brain cut took place in the Forensic Department. The Neuropathology paperwork supports this conclusion because it suggests that we only received blocks from the Forensic Department, rather than the whole brain.

10. For the microscopic examination, slides are taken by cutting small slivers from the blocks of tissue and dyeing them with specific dyes to reveal the different brain cells present. There were 12 blocks of tissue taken in this case. Each case is individual, and we would select those pieces of tissue that might be relevant to the particular pathology. The brain is quite a large organ, so perhaps only 1% to 2% of the brain was examined microscopically. This is normal procedure.

11. The normal procedure is that, once the examination is complete, a second report is prepared combining the macroscopic findings, that is, what is seen at the brain cut, and the histological findings from viewing the slides under a microscope. A conclusion would be prepared and the report would then be sent back to the referring pathologist. There may have been some further discussion between us in order to clarify any issues that needed further explanation

12. At page 31395 of my report, my reference to an "external examination" relates to the macroscopic findings. I noted that Mr Hamill's brain weighed 1588 grams, which is abnormally heavy. There was no subarachnoid haemorrhage and there was no meningitis. My main findings were that there were haemorrhages in the deep white matter in the brain in a parasagittal location, which means on either side of the midline. There was also haemorrhage in the thalamus, which is one of the deep structures in the brain, and also haemorrhage in the internal capsule. The white matter was congested. I did not see any focal haemorrhage in the corpus callosum, but I did see what I described as diffuse punctuate haemorrhage in the brainstem. The cerebellum showed no specific abnormality.

13. Professor Crane refers to a hairline fracture of the skull in his report at pages 72234 and 72240. I did not find any specific brain injuries relating to the fracture. There were no contusions (bruises) on the surface of the brain that may be caused by an impact, where the brain slides over the base of the skull. This would be a common injury where there is an assault with a collapse to the ground, but no such injury was present in this case.

14. The findings suggested to me that Mr Hamill had a diffuse brain injury that involved the deep structures of the brain, which was important, because he had been unconscious. The macroscopic findings suggested to me that the particular diagnosis of diffuse axonal injury was the cause of the unconsciousness.

15. Diffuse axonal injury is now a non-specific term that means the brain has been damaged and the axons are the main structures of the brain that are damaged. A nerve cell in the brain is like an electricity switch and the axons are like the wires. In general terms diffuse axonal injury means that these wires in the brain have been damaged. In 1997 the term "diffuse axonal injury" implied the cause of axonal damage was trauma to the brain. The terminology has changed and become more specific. The term "diffuse axonal injury" is sometimes now used to describe anything that affects the brain causing damage to the axons. This is also why, in 2005, Dr Reid referred to it as TDAI in her report (pages 72526 to 72534), which is a more accurate term because it refers to "traumatic" diffuse axonal injury.

16. In diffuse axonal injury it is not always possible to determine the severity of the injury with a macroscopic examination; diffuse axonal injury is predominantly a microscopic (histological) diagnosis.

17. One of the ways to grade axonal injury is to look for focal lesions in the white matter, corpus callosum and in the brainstem. In general and subject to certain caveats, the more focal lesions there are in the deeper part of the brain, the higher the grade of diffuse axonal injury. If there are haemorrhages in the brainstem, the injury is more severe.

18. I do not have it in front of me during this interview but as far as I remember, in his evidence at the trial of Marc Hobson, containing pages 08487 to 08541, Professor Crane said that in general terms, traumatic diffuse axonal injury of a severe degree, leads to unconsciousness, and unconsciousness is present from the time of injury. The structures in the cerebral hemispheres were not as badly affected as those in the brainstem including the pons. The corpus callosum, which is a higher structure in the brain, was not as severely affected. Thus, I

thought there was severe damage to the brainstem. Diffuse axonal injury would be a pathological substrate to explain his unconsciousness, and possibly to explain his death.

19. When reading my report, I think we have to remember several things. I am aware that in some aspects of the case Dr Reid reached a different view in 2005, but it is important to point out that the brain of Mr Hamill was examined in 1997 when less was known about diffuse axonal injury. Not only has the terminology changed, but also the recommendations as to examining a brain with axonal injury. At page 72533 Dr Reid suggested that too few blocks were taken, but examination guidelines had not been published in 1997. At page 31396 I indicate that I used the test "immunohistochemistry for neurofilament protein and stains for myelin". Since then, further tests have become available to detect such an injury. One of these is a stain that we now use to detect amyloid precursor protein (APP), and Dr Reid refers to this as the Beta APP test at page 72530 of her report. Were I to have a case now, I would definitely test for APP, but it is more useful in the first few days after an injury, when the injury is more acute. I felt that even without APP, I had found enough damage to the brainstem using routine stains to support my original thinking. I still hold to that opinion.

20. Dr Reid also talks about the grade of the diffuse axonal injury at page 72531, but in my report I wanted to create an impression that, no matter what the grade, this was a severe injury because of the pattern of injury affecting the brainstem. The difference between calling the injury severe and grading it as I, II or III reflects a difference in a clinical and pathological grading system. Dr Reid cross-references Professor Graham et al, Greenfield's Neuropathology 2002, Volume 1, Chapter 14. If we use the method in this textbook, Mr Hamill's diffuse axonal injury could be grade II or III, but that was not necessarily what I wanted to emphasise in my report. While there is a correlation between clinical severity and grade, the impression that I wanted to give was that this was a severe injury and there was severe brainstem involvement that would have explained the clinical features.

21. I would like to refer to 2 publications on this point. I note that it has been found in a study in 2000 that "only the severest acceleration-deceleration forces with or without deceleration caused diffuse axonal damage extending down into the brainstem" (Geddes, Whitwell and Graham in Neuropathology and Applied Neurobiology 2000, issue 26, pp 105 – 116 (110)). Although I do not have the reference to hand during this interview, there is also a 1989 paper by Adams and Graham in the journal Histopathology that I remember says that it is unfortunate that focal lesions are the mainstay of grading diffuse axonal injury. Accordingly I consider that while it is possible to grade the injury in the manner which Dr Reid has done in her report, I would still say that this was a severe injury.

22. I do not agree with Dr Reid when she states at page 72531 that:

> "As I do not think there are haemorrhages with axon bulb formation in the corpus callosum and around the aqueduct of the mid brain I would not classify this as severe TDAI"; and
>
> "Therefore, if they were as in traumatic diffuse axonal injury they would be Grade II as defined by Adams et al (reference 1), but it is not severe";

23. and at page 72533

> "I do not agree that there is severe traumatic diffuse axonal injury in this case. The grade of DAI is II with scattered white matter damage"

24. As indicated in the literature, the fact that there is not a focal lesion in the corpus callosum does not mean that this should not be thought of as a severe brain injury and severe axonal injury. Whilst it could be grade II if you go by the criteria, it was severe in the structures that matter. The bulk of the worst pathology in this case was in the lower brain structures, which are more critical for survival, particularly for respiration and cardiovascular function. I thought that there was severe damage in this region, and I would still say that this was a case of severe traumatic axonal injury. It may be misleading to use grading here, which could be why I did not use the grade in my report. If I had used grade II rather than grade III that may have misled Professor Crane to thinking that this was not a severe brain injury.

25. The cause of the diffuse axonal injury would have been a severe rapid movement of the head – a rotational movement in any way, left to right, up or down, which moves the brain within the skull. The movement needs to be fairly rapid, and there is a lot of literature about what types of injuries can cause this. The normal type of incident that causes diffuse axonal injury is a road traffic collision, a motorcycle accident or fall from a height. It has been described in assaults, but not usually from a single punch for instance, and it may be that it does not result from a single kick to the head on the ground, although that is still slightly speculative. In most of the assaults described in Professor Graham's work, it seems that an accelerated fall may have been implicated. However, because of the sometimes poor clinical histories, the mechanism is not entirely known. In experimental studies and in general it is thought to be a rapid movement of the head with acceleration/deceleration of the brain inside the skull.

26. I have been asked about the severity and mechanism of the assault which caused the damage. This is a forensic question, and I am not a Forensic Pathologist. This issue is well discussed at pages 111 – 112 of Geddes' paper (2000) to which I have already referred, where she describes and interprets different scenarios. Similarly, I am not in a position to comment on the correlation between Mr Hamill's external injuries and the severity of the diffuse axonal injury.

27. My conclusion was that Mr Hamill had suffered a severe head injury. Mr Hamill was unconscious from the time he was admitted, and never regained consciousness. His coma scale was always 8 or below as far as I remember, and usually a lot lower than 8. A paper called 'Current Concepts, Diffuse Axonal Injury Associated with Brain Injury' published in Archives of Physical and Medical Rehabilitation on 28 October 2001 states, "A Glasgow coma scale of less than 8 is indicative of a severe injury" again supporting my view that this was a severe brain injury.

28. I have been asked to clarify the distinction between my macroscopic finding of diffuse punctuate haemorrhage at page 31395 of my report, and my microscopic finding of no secondary brain stem haemorrhage in the midbrain at page 31396. Secondary brainstem haemorrhage is a completely different pathological finding from diffuse punctuate haemorrhage. The latter is primary injury and describes lesions that are associated with diffuse axonal injury or traumatic axonal injury, where the brain is shaken so much that the blood vessels rupture at the time of injury. Again, this would support my case that there was a severe brain injury, because the lesions were found so low down in the brainstem. Secondary brainstem haemorrhage is a completely different entity that occurs later in the process of head injury, as a result of brain swelling causing blood vessels to become obstructed and leak into the brainstem.

29. Although my view is that the diffuse axonal injury was the main pathology here, the question of what actually caused Mr Hamill's rapid demise at the end is more difficult, and I still think it is open to speculation. The severe diffuse brain injury may have led to his death or permanent unconsciousness. He could never have returned to his pre-injury state. Whilst I am aware that there was surprise from the clinical team at the death of the patient, I was not entirely surprised given the amount of brainstem damage from the diffuse axonal injury. The brainstem affects respiratory and cardiac function and therefore severe damage to it can lead to death. Another common mechanism by which diffuse axonal injury can lead to death would be from an infection in an unconscious patient, but I cannot really comment with any expertise on this possibility as I only examined the brain.

30. In Dr Todd's report, containing pages 72617 to 72647, he concludes at page 72646 that Mr Hamill died as a consequence of a grade III severe diffuse axonal injury in the absence of any other lesions. In my view, this statement is very reasonable. He also refers to what he describes as the very helpful paper by Geddes to describe the various mechanisms than can occur to cause death from diffuse axonal injury.

31. It is necessary to bear in mind the context of the case when drawing my conclusions. I was aware that the patient was unconscious from the time of admission and I would need to account for that pathologically. A clinician would think of 2 main possible causes of Mr Hamill being unconscious from the day he was admitted, but dying 11 days later: diffuse traumatic brain damage or hypoxic/ischaemic brain injury. If the patient has lost a lot of blood and his heart has stopped or his airway has been obstructed, or if his brain is so swollen then blood and oxygen cannot get to the vital areas of the brain. The cells that are responsible for consciousness may die, but a person can be unconscious and still survive for a prolonged period of time in a vegetative state. This is a form of hypoxic/ ischaemic injury

32. I excluded hypoxic/ischaemic brain injury as a cause of the unconsciousness as it would have had to be there from the time of injury. Since Mr Hamill died 11 days later, the brain cells would have had this period of time to react and that reaction was not there. The body reacts to injury in a particular way. For example, if you break your leg there is a system of repair that takes place. There is an acute inflammatory reaction, then a chronic one, and then a repair reaction; a bruise from a bang on the arm will change from one colour to another over a number of days because of the chemical reaction in the bruise. The brain reacts to injury in a very particular way, so, if there is damage to part of the brain then the nerve cells react to that. There was no evidence of an 11 day reaction to suggest there was a hypoxic/ ischaemic brain injury that caused his initial unconsciousness.

33. There was some acute reaction to hypoxia/ischaemia that occurred shortly before his death, but this would not have explained all of Mr Hamill's injuries or his prolonged unconsciousness. I think if I was writing up this case again, I would perhaps discuss the hypoxia/ischaemia more. I would add another paragraph to say that there was no established hypoxic/ischaemic damage that had caused the initial unconsciousness. I suspect that at the time I was trying to emphasise the major pathology, which was the axonal injury.

34. I essentially agree with Dr Reid's conclusions about hypoxia in her report at pages 72531 to 72533. I agree that there was perhaps hypoxic/ ischaemic change on or around the time of death and that this did not have an effect earlier on. However, Dr Reid gives a statistical statement at page 72533 that hypoxia contributed to worsening the initial brain injury by less than one third. I do not know of any literature or any mechanism that would allow me to make a statement like that. There was some terminal neuronal damage that could have multi-factorial causes: it could have been seizure related or hypoxic/ischaemic related. To be clear, for the reasons I have already stated, my view is that the hypoxia/ ischaemia was not related to Mr Hamill's initial condition.

35. I have been asked to comment on an aspect of Dr Todd's report at page 72644, where he writes, "We also need to be aware that uncommonly hypoxia in the absence of trauma can be associated with axonal injury". The medical papers he has reviewed refer to the change in terminology between diffuse axonal injury and traumatic axonal injury. Because many conditions can cause damage to axons, including hypoxia, a stroke or Multiple Sclerosis, I assume he means that there is a need to label the condition precisely as traumatic axonal injury or trauma. Looking at the case overall, the distribution of the axonal injury is absolutely consistent with trauma, and not with hypoxia.

36. I disagree with Dr Reid's conclusion at page 72533 that NMS caused Mr Hamill's death. I am not sure if I was aware of the CK serum levels at the time of my examination. However, the clinical pattern and the CK levels were not typical of NMS, and the autopsy findings did not suggest a mechanism as to how it could have caused death.

37. I did consider the possibility that Mr Hamill's death had been caused by NMS at the time of my examination. NMS is a very rare condition in which I have had a particular interest since I was a junior doctor in 1988. I saw a patient who was admitted to the medical ward from a psychiatric hospital, who was diagnosed with NMS and then died. I wrote a paper on this condition which I presented at the British Neuropathological Society meeting. I think it is still the case that this was the largest pathological series on fatal NMS published or presented.

Also in 1997 a patient was admitted with a head injury to intensive care at RVH and died. I diagnosed NMS in that patient at about the same time as I considered Mr Hamill's case.

38. Accordingly, at the time NMS was something very current both with the neurosurgeons and me, and we were very aware of this condition. NMS was a reasonable suggestion to make and I thought that it was appropriate to address it because Mr Hamill had been given neuroleptic drugs that may cause the syndrome; he also had had a raised temperature and he was sweating excessively. Professor Crane asked for my thoughts on NMS and I wrote back explaining reasons why it might not be NMS. I compared the findings in relation to Mr Hamill with those of the other 4/5 cases of which I was aware, but it did not really fit with them. You could not absolutely reject NMS as being a factor, but I do not think that there were enough symptoms or signs to include it. I did speak to Professor Crane extensively about this issue at the time.

39. Death in NMS cases may occur for a number of reasons, for example pneumonia, multiple organ failure, acute liver failure or due to the muscles in the body breaking down and blocking the normal fluid flow of the kidneys. There was no evidence of any of this happening with this patient; moreover, there was no myoglobinuria recorded in the notes. The main feature of my previous cases was that there was severe muscle rigidity, making it almost impossible to bend the patient's limbs, almost as if they had meningitis. This pointed to rhabdomyolysis, where the muscles break down. However, there was no evidence of rhabdomyolysis in Mr Hamill's case. Professor Crane looked at the kidneys for 'casts', which are bits of muscle that would break down and lodge the kidneys. In my previous cases, the liver was almost completely necrotic (dead). According to Professor Crane, there was no liver damage in Mr Hamill (page 09564).

40. My examination of the brain did not find neuropathological changes that can be attributable to NMS. The Purkinje cells were preserved. These cells are very sensitive to damage by hypoxic/ischaemic change, and have also shown to be

specifically damaged in previous cases of NMS I have seen. But there are so few papers describing the pathology of NMS it is difficult to absolutely exclude the possibility of its presence in this case.

41. I have been asked if NMS could have caused the axonal damage. It may be that NMS could be a cause of axonal injury, as not enough is known about it to say that it could not. I suspect that it could cause similar changes to hypoxic/ischaemic brain damage. But Mr Hamill only had the pyrexia the day or so before he died, and the changes in the axons were there longer than a day So it cannot be considered the condition that caused his unconsciousness

42. Even if I had thought NMS was the diagnosis, it would not explain why Mr Hamill died. The other people who have suggested NMS have not really given a reason why it could have caused the death. They have not suggested, for example, that he died in renal failure. The potassium levels were not high enough to cause death. Dr Lawler, in his report and glossary for this Inquiry containing pages 72226 to 72250 and pages 72270 to 72278, and Dr Reid have mentioned that a blood result came back after the death that recorded a creatine kinase level of about 924. This level does not support a diagnosis of NMS because, with NMS, the creatine kinase levels would be in the region of tens of thousands perhaps 50,000-60,000, and not below 1000. Further, without giving precise details of this case, I have asked my neurosurgical and neurology colleagues what they would think of this level of creatine kinase in a man who had been assaulted, had a lot of bruises and muscle damage and had been in intensive care and the wards for a period of time. They said they would not be particularly concerned because it would be fairly normal for a man who had been assaulted to have a raised creatine kinase, purely due to the muscle damage.

43. I have been asked if the brain examination could have determined whether the cause of death was septicaemia. One might observe some changes, but not enough to say categorically that what you see represents septicaemia. Sometimes you can see lots of white cells clogging the vessels, but I have never over-speculated on that. I think it is up to the pathologist to find the source of

infection. However, in my experience as a general pathologist, it is sometimes very difficult at autopsy to prove or disprove septicaemia. I absolutely agree with Professor Crane's evidence at the trial of Marc Hobson at page 08508 that it was not worth carrying out post-mortem blood cultures because they can be as misleading as useful.

44. I can confirm I considered the effects of alcohol and intoxication on head injury within my remit in examining the brain; it is something that is often referred to, but to my knowledge the literature to support it is poor. I was aware of the alcohol level in Mr Hamill's blood from the clinical history, but I do not think that I have ever said in any of my reports on trauma cases, that alcohol would make traumatic brain damage worse.

45. Once I had conducted my examination I wrote a report and forwarded it to Professor Crane to be incorporated into his Autopsy Report. He would have had to assimilate all the relevant reports and put them together for his final report.

46. I cannot really remember specifically ten years later, but I do not think that I had any contact with the police in relation to this case, nor did I give a statement. In those days, Professor Crane would normally take my reports and assimilate them into his final report; it was then he, rather than I, who would have contact with the DPP, the police, the prosecution and the defence etc. Nowadays, the contributors to an autopsy report may be asked separately to give comments.

SIGNED: ...Brian Herron.....

BRIAN HERRON

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DATED: .....4th December 2008.....