STATEMENT OF WITNESS

STATEMENT OF JACK CRANE

DATED THIS 14th **DAY OF** January **2005.**

I, JACK CRANE, 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.

2. I am the pathologist who performed the post mortem examination of Robert Hamill on 9 May 1997. In terms of my qualifications, I hold a Batchelor of Medicine and a Batchelor of Surgery and I am a Fellow of the Royal College of Pathologists. I hold a diploma in Medical Jurisprudence in Clinical Forensic Medicine and a diploma in Medical Jurisprudence in Forensic Pathology. I am a Fellow of the Faculty of Pathology of the Royal College of Physicians of Ireland.

3. I conducted the post mortem examination of Robert Hamill on 9 May 1997 in the presence of a number of people including the investigating police officer Detective Inspector [redacted] a police photographer and a Scenes of Crime Officer (SOCO). A mortuary technician would have assisted me in conducting the post mortem. There may have been others present but I cannot recall.

4. I completed my report in the first week of November 1997. There is no time frame set down as to when the report should be submitted from the date of the autopsy. The completion of the final autopsy report is very much dependent on what other tests and investigations have to be done in respect of the case. The more complex the case then the longer it takes to have all of the additional

reports completed. In the intervening time we provide the police with some preliminary information regarding what we think the cause of death might be.

5. In this case the brain was submitted for neuropathology. As part of an autopsy I examine the scalp, the skull and the surface of the brain at the time of the post mortem but after that the brain is then taken out and is fixed by immersion in formaldehyde for a period of at least 6 weeks. Once fixed, the brain is given to a neuropathologist for examination. The neuropathologist cuts the brain to see whether there are any internal lesions and sections are taken for microscopic analysis. One has to then wait until the neuropathologist produces their report. Then I would have to discuss the findings with them so the process may take a considerable amount of time. It is my usual practice to be present when the brain is sectioned if possible but I cannot recall whether I was present on this occasion. The slides of the brain were considered by Dr Herron and me. Dr Herron's report is now produced and shown to me containing pages **31395 – 31396**. It is dated 29 October 1997. I am presuming that is the date Dr Herron signed his report but I am not sure on what date I received it.

6. The police photographer, Mr [redacted] took the photographs on my instructions. Those photographs are now produced and shown to me containing pages **1049-1068**. There has also been produced and shown to me the complete set of notes that I made at the time of the autopsy by dictation, these are produced and shown to me and contain page numbers **31514-31521**.The history was not completed until after autopsy when I gained access to the hospital records. The only hospital record which I had prior to the autopsy was a clinical summary produced by the hospital which is now produced and shown to me at page **72832**. I do not know when I received them. The only other information provided to me was from the investigating officer, DI [redacted] who provided an account of the circumstances of the event.

7. The clinical summary states:

> *"Closed head injury following assault. Transferred from Craigavon Hospital ....27 April 1997. Assaulted about 2.00am. GCS [Glasgow Coma Scale] 4 to 5 out of 15. Unconscious in CAH. Intubated, ventilated*

and then transferred to ICU. CT scan on arrival normal. CT spine X-rays normal. Ventilated until 27 April 1997 midday. Since extubation, extremely restless and agitated requiring regular sedation. GCS remained 6 to 8 throughout admission. Sedation has decreased since 7 May 1997. Morning of 8 May 1997, pxyrexic 37 degrees. Query central cause due to injury. Query infection. Blood cultures I2 taken. MSSU [midstream specimen urine]. Chest X-ray ordered. On return from X-Ray, temperature 40 degrees, PL unrecordable blood pressure. Registrar called. Periphery shut down. Poor respiratory effort. Anaesthetist and cardiac team called. Resuscitation commenced at about 3.50 pm. Nil response from IV fluids, adrenalin, sodium bicarbonate, calcium chloride, atropine. Defibrillation. Antibiotics given also. Resuscitation discontinued about 4.10pm. No output. No respiratory effort. Pupils fixed and dilated. Patient pronounced deceased. Clinical diagnosis is diffuse brain injury."

8. I would have relied on the clinical summary for an indication of the drugs given to the patient prior to death. Robert Hamill was given Chloramphenical, which is an antibiotic, Cloforam, which is not an antibiotic, and Dexamethasone, which is a steroid often used to reduce swelling of the brain. Pathologists rely on the clinicians caring for the patient to indicate if there was any significant result to a test performed prior to death (such as a high potassium level or high blood sugar level). One of the things that I was told about Robert Hamill was that he had a raised temperature which is why I carried out tests from samples of spleen, cerebral spinal fluid and lung for microbiological examination to see if he had septicaemia or other infection, but there was no evidence of such an infection. Normally, blood would be taken. If a patient spikes a temperature but we cannot use blood after a person has died as the blood is contaminated. For this reason, therefore, I took a splenic swab.

9. The bacteriology report did reveal some organisms, staphylococcus and pseudomonas which we felt were probably as a result of post-mortem contamination. For example staphylococcus is present in the nasal passages, and I think that Dr Lawler agreed that they were probably were not significant. The summary did not mention malignant neuroleptic syndrome (NMS).

10. There has now been produced and shown to me a copy of my report dated 9 May 1997 containing page numbers **9559-9569**. As part of the external examination I noticed that there was a scar close to the hairline which is set out

at page 2 of my report. The clinical summary referred to a closed head injury but not to any external injury. I shaved the part of the head where the healing laceration was located and checked to see if it was reflected in the scalp but there was no bruising. A photograph of it was taken and is now produced and shown to me containing page number **1052**.

11. As part of the normal procedure for the external examination I turn the body over to check for injuries to the back. In this case no injuries on the back are recorded or photographed as there were no injuries. There may have been a healed laceration but no external injuries were visible at autopsy. There were no visible injuries to the back of the scalp when it was reflected. I did not fully shave the head but the scalp at the back was shaved as we were looking for evidence of whether he had fallen at the back and struck his head.

12. The lack of bruising may be a result of a healed injury or it may be because there was little actual bruising at the time of the injury. There was no internal bruising in relation to the bruise to Mr Hamill's upper eyelid. There was a hairline fracture on the front of the scalp. Overall, the external injuries were significantly less than we would expect to see in someone who had been the victim of a violent assault, even taking account of the time for healing prior to death. However, what has to be considered is that the external injuries may not necessarily reflect the possibility of more serious damage internally and particularly to the brain.

13. I think the significant injury in this case was the presence of diffuse axonal injury in the brain. That type of injury is not one that is necessarily associated with very severe external injuries because it is caused by the brain being shaken about inside the cranial cavity. It is possible to have a severe brain injury with very little external injury. Conversely, you can have quite severe external injury, but have very little damage to the brain inside. It depends on a number of factors, for example, the footwear that assailants are wearing or if they are struck blows with an implement. It is often the mechanism that is important. If a person is hit with a baseball bat, the scalp is lacerated and bruised but the

brain is often intact. Whereas if someone is kicked about the head and the brain is shaken they may suffer serious damage to the brain inside. I consider that there is no correlation between the severity of the external injuries and the severity of the diffuse axonal injury.

14. I have been referred to my evidence at trial which is produced and shown to me at page **8502** where I said "there is very clear evidence, documented work, to show that this type of injury occurs in assault." I go on to say, " It is however, recognised as occurring in assaults but curiously this lesion has not been described in any work which has been done on boxers." I think what I was explaining is that there has been some work done on the brains of boxers who have died and they don't show diffuse axonal injury. Whether this is because their heads are moving when they are struck, I do not think we quite know. What we do know is that people who are assaulted and particularly those who are lying on the ground when they are kicked, do show this injury.

15. It is a curious anomaly and we are not sure why this is the case. It may be that punching is not enough to cause diffuse axonal injury. It may require more force than that and presumably the amount of force that can be generated by kicking someone is more severe. It is fair to say that diffuse axonal injury is a relatively recent concept of brain injury which has only become more recognised over the past few years. Our understanding of it is increasing and in what circumstances it tends to occur. To some extent I think that clinicians are not perhaps as fully aware of it as pathologists are because there is nothing to see in the patient on scans.

16. Diffuse axonal injury shows up in pathologist's findings as the level of detail is greater on a microscopy than on a CT scan. This level of detail can only be achieved post mortem when the brain has been sectioned. A further problem is that our ability to detect this type of injury depends to some extent on the period of time that the patient survives as well. For example if someone dies a couple of hours after assault the brain can be examined grossly and microscopically but it is only with the passage of time that the changes in the brain develop that the techniques we have are now able to detect.

17. There was a significant injury to the brain but I disagree with the report of Dr Lawler which is now produced and shown to me containing pages **72226-72250** which suggests that the injury could have resulted from a forceful impact on the roadway. Typically when a person falls and strikes the back of their head there is a 'contra coup' brain injury. There would be heavy bruising under the surface of the scalp at the back, a fracture of the underlying skull and severe damage to the front of the skull. Mr Hamill did not have that pattern of injuries. Diffuse axonal injury is not usually associated with a contra coup injury. It is typically associated with situations where the brain is accelerated and decelerated within the cranial cavity. That is not usually what happens with a simple fall onto the back of the head. Furthermore, I was not given any information which indicated that this was the mechanism of injury.

18. As indicated, there was a hairline fracture on the front of the scalp but this could not have resulted from an injury to the back of the scalp. From such a backward fall you would expect to see a fracture at the back of the skull not at the front. This skull fracture was not noted by the clinicians as they do not show up well on scans and simple X-rays are not widely used now. Whether there was a skull fracture or not would not have been of clinical importance. The main concern for the clinicians is the state of the brain and whether any surgical intervention is needed. A CT scan will be much better at showing whether the brain is swollen or has bleeding over it as these features do not appear on X-rays.

19. In my view, this injury to the brain was caused by Mr Hamill's head being struck whilst he was on the ground so that his head was moving backwards and forwards with the brain inside being shaken about. This is how the diffuse axonal injury is caused. It is a very well recognised pattern of injury in people who are assaulted where the head is shaken. There was bruising on the sides of the outside of the skull which is consistent with the head being shaken from side to side but no bruising on the back of the head. The whole scalp was reflected as shown in the photograph which is now produced and shown to me containing page **1064**.

20. I have been asked to comment on the external injuries on page 9 of Dr Lawler's report which is at page **72234** where he states:

> *"There was a puncture lesion on the left naso-labial fold. In his autopsy report, Professor Crane refers to this as an abrasion, in evidence at trial it is referred to as 'bruising'. Photographs 5 and 6 suggest an abrasion, rather than bruising, but I strongly suspect that it was fairly recent, i.e. that it occurred in hospital and after 29 April 1997."*

21. I called it an abrasion because it was an abrasion. He was looking at the photograph. Now it may have been caused in hospital but all I did was say that a mark was there and I recorded it as being present.

22. I am also asked to comment on the section headed "The Trunk" where the report at page **72234** states:

> *"There was a bruise, probably associated with a surgical suture, on the front of the right shoulder. This is not clearly seen on any of the photographs. I note that in evidence, Professor Crane suggested that it was the result of surgical treatment and this despite including it as an injury in his autopsy report!"*

23. There was a bruise with a suture. Unless the clinician is there and says to me that wasn't there when Robert Hamill came into hospital all I can infer is that that there is an injury there.

24. I have also been asked to comment on the section headed "*Left Upper Limb*" where it is stated, "*Professor Crane described a "number of" bruises on the back of the forearm, wrist and hand.*" My only comment is that a number of these injuries were probably related to the fact that Robert Hamill was being given drugs intravenously and they were taking blood from him every day in hospital. So the fact that there were four, five or ten is completely immaterial.

25. Whilst I am careful not to 'over-interpret' injuries (as there may be a number of possible causes for an injury), if I had found 'defence' injuries on the forearm I would have recorded them as such. There were no defence injuries in this case. The marks which I saw on the arms appeared to be associated with hospital treatment.

26. At page 5 of my report which is contained at page **9563** I noted that there was dark congested oedematous tissue which is normal in patients with head injuries as the lungs become water logged. This is simply a secondary effect of a head injury and is not linked to the fatal outcome. There was no evidence of pulmonary embolus from deep venous thrombosis. If vomit or some other substance had been inhaled at the time of injury there may have been some indication at the autopsy but none was seen. I note that there was a little bruising in the muscles of the anterior abdominal wall on the right side of the front. This was not linked to any external bruising but the abdomen is quite soft and so external bruising may not result from a blow in this area.

27. After the autopsy, I was telephoned by one of the senior clinicians from the hospital, I cannot now recall who, to enquire about the results of the autopsy. It is not unusual for clinicians to take an interest in the cause of death for a patient who had been under their care. Here, I understand that there was some surprise amongst the clinicians at the suddenness of the decline in Mr Hamill's health and they raised with me the possibility that there could be a cause other than the brain injury.

28. Specifically, we considered the possibility of malignant neuroleptic syndrome and I was in discussion and correspondence with Dr Herron as to whether this was the cause of death or the head injury alone. Factors which pointed to NMS were the fact that the patient had a head injury, he had a spiked temperature and he had been given chlorpromazine. At the time of the autopsy, I was not aware of the results for the CK tests showing 924 per litre but that level of CK is not especially elevated. A raised CK level is not unusual where a patient has undergone physical trauma. I would have expected a far higher level of CK, in the thousands, in a case involving NMS.

29. The reasons I ruled out NMS include the absence of damage to the kidneys, where NMS would have caused casts of myoglobin but none were present. I did special stains on the kidney to look for muscle damage but there was none. There were no casts identified within the tubules. There was an absence of

damage to the liver, with no necrosis evident. There was no rigidity in the muscles which is a notable symptom of NMS. Neither were there the changes to the brain which would normally be associated with NMS such as necrosis, particularly in the cerebellum. We specifically looked for all of the changes that you get in this syndrome. It was in my view a possibility and one that we needed to consider. All of the typical markers that are associated with the syndrome were not found and that is why I excluded it as being a cause of death.

30. The injury did cause damage to the hypothalamus which is the part of the brain which controls the body temperature and this may explain the raised temperature in this case, albeit that raised temperature is a common symptom generally associated with head injuries in any event. The damage to the hypothalamus is recorded in the histology section of Dr Herron's report which is contained at page **31395** where it states, "*Examination shows no evidence of haemorrhagic necrosis. There is however axonal damage in the hypothalamic region.*"

31. As the chlorpromazine was given on the 1 and 2 May 1997, a week before the patient's death, I would have expected any adverse reaction at that stage. There is not a cumulative effect to the drug, the half life of the drug is such that it breaks down and that is why it is given fairly regularly. It is not as though the level of the drug is building up throughout the week. It tends to be an idiosyncratic reaction, in other words, you don't know someone is going to get NMS. Further, I note that no mechanism for death through NMS has been provided, and the usual mechanisms such as acute degeneration of the liver, kidney failure or necrosis in the cerebellum were not present.

32. I did not include consideration of NMS in my autopsy report as what I tend to do is put my opinion in the autopsy report. The possibility of NMS was not included in my notes either because the notes at the post mortem are contemporaneous and do not include tests which are carried out subsequently, including looking at the microscopy.

33. I have been asked to comment on Dr Lawler's report at page 18 and 19 of his report which is contained at pages **72243-72244**.

> *"Professor Crane excluded it on the basis of there being no histological findings in the brain, liver or kidneys to support this diagnosis... I disagree that the lack of appropriate structural changes in the brain, liver and kidneys automatically excludes a diagnosis of NMS particularly when other clinical and laboratory findings are present."*

34. I cannot understand how it is possible to make a diagnosis if Dr Lawler is basing a diagnosis on an elevated CK and an elevated temperature, both of which can be explained by (a) the finding of hypothalamic diffuse axonal injury and secondly the fact that this man was assaulted. What he has failed to explain is if this syndrome is present why the other changes are not present in the liver, kidneys and brain. A diagnosis has to be based on what is found.

35. Dr Herron and I came to the conclusion together that the findings were not consistent with NMS but they were consistent entirely with diffuse axonal injury. I disagree with the comments of Dr Lawler at page 12 of his report contained at page **72237** where he states that, "*The neuropathological findings, per se, are insufficient to cause relatively sudden and relatively unexpected death.*" With respect all Dr Lawler is doing is simply looking at what Dr Reid has said. The injury suffered by Robert Hamill would accord with an account of a young man lying on the ground and his head being struck a number of times.

36. The degree of severity attributed to the diffuse axonal injury is primarily a matter for the neuropathologist but I am happy to agree with Dr Herron's view that this was a severe diffuse axonal injury. Further, I am satisfied that the diffuse axonal injury was sufficiently severe to be the cause of death. I do not concur with Dr Lawler's view that the neuropathological findings *per se* are insufficient to cause this relatively sudden and unexpected death.

37. It is understandable that the clinicians found this death sudden and difficult to explain. It is often very difficult for clinicians to identify a diffuse axonal injury as it does not show up on the scans. There has been a considerable increase in

the knowledge about this type of injury since 1997. Techniques have been developed for identifying such a brain injury within about 6 hours of the injury. It was easier to identify in this case as Mr Hamill survived for 11 days.

38. There is no specific treatment for a diffuse axonal injury. The prospects of recovery depend on where the injury is and its severity. Some people may recover completely, others may be left in a vegetative state or they may be left with some residual brain damage. An injury in the brainstem is the most serious because that is where the vital centres of the brain are located which control essential functions such as breathing. There is usually a poor outcome when the brainstem is affected.

39. In all the circumstances, it is very unlikely that any first aid or different medical treatment could have had an effect on this injury.

40. I did consider whether there was evidence of hypoxic damage but I excluded this as a cause of death. Hypoxic brain injury is often associated with a very swollen or oedematous brain but this did not appear to be the finding from the CT scans taken. There was no neuropathological evidence of the nature which one would expect to find if the brain had been deprived of oxygen for a significant period such as laminar necrosis of the cortex. If there had been significant starvation of oxygen at the time of the injury one would expect to see clear changes in the brain develop after a week.

41. It is possible for axonal injury to be caused by hypoxia where there is no physical trauma but there was insufficient evidence of hypoxia in this case to establish it as the cause of this axonal injury or death. In terms of the hypoxic brain damage, I have been referred to Dr Reid's report at page 7 which is contained at page **72532**, that there are some changes. It states:

> *"There was some evidence of hypoxaemic/ischaemic change with pink neurones in some areas of the cortex, also mild loss of neurones was present as well. There was no evidence in the slides or the macroscopic description of the brain from the post-mortem report of infarction so that the hypoxaemic/ischaemic change was mild. This change could well be related to the initial assault but some may have been due to the*

hypothermia he had. Also, the amount of white matter damage may have been increased by the hypoxaemic/ischaemic, but I do not think that this has increased the damage by more than one-third and it is probably much less."

42. There was obviously some kind of hypoxic damage to a limited extent, but not significant hypoxic damage. I do not think there was any evidence of that either as a result of hypoxia at the time or as a result of neuroleptic malignant syndrome where you do get areas of acute degeneration in the brain which is well recognised, particularly in the cerebellum Purkinje cells. That was not present.

43. I have considered the police file note which has been produced and shown to me at page **14803** which states that when I was interviewed further by the investigating officer I said that Mr Hamill should not have died from his head injury but this was the diagnosis of the cause of death with possible contributing factors of (a) intoxication (b) oxygen starvation to the brain whilst being cradled at the scene and (c) septicaemia. I consider that reflects a misinterpretation of my words. It is more likely that I simply said at autopsy that I was surprised at the death given the lack of evidence of external damage to the brain and I was considering these possible contributory factors. The blood alcohol was 220 milligrams. Although that is not a level that is going to kill someone, clearly a level like that may have an effect on an individual being able to withstand an assault.

44. There is some evidence, although not terribly well written up in the scientific literature, that alcohol can potentiate the effects of a head injury. In general terms people who are intoxicated are more susceptible to the effects of head injury than people who are sober.

45. I would not have committed myself to any conclusion until I had the results of Dr Herron's work.

46. I have no recollection of speaking to the police at any other stage but it would not be unusual to be contacted while an investigation was ongoing. I was aware

of the allegations made against the RUC in connection with this case at the time of the autopsy but this was not a matter of interest to me, my concern was to establish the cause of death.

SIGNED: Jack Crane .....

JACK CRANE

DATED: 14 January 2009 .....