CAUSE OF DEATH

1. There are two questions which the Panel may want to consider. The first is whether the assault was the cause of Mr Hamill's death, or whether there was some other intervening cause. The second is whether the medical findings reveal something about the intensity and duration of the assault.

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

We attach an expert opinion by Professor Peter Vanezis [**pasted below**], based solely on the Inquiry Team's synopsis of the evidence, which deals with most of the issues raised in this module.

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Ref:

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28th

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MEDICAL OPINION ON BEHALF OF BRITISH IRISH RIGHTS WATCH IN RELATION TO THE DEATH OF ROBERT HAMILL

INSTRUCTIONS

I was requested by British Irish Rights Watch to provide a report in relation to the above case.

WHAT IS REQUIRED TO BE ESTABLISHED?

I have been instructed to address the following two issues:

1. If the police officers had intervened earlier, could they have prevented the attack on Robert Hamill?

2. Did the position in which police officers place Robert Hamill, materially contribute to his eventual death?

IN ORDER TO ASSIST ME WITH THE PREPARATION OF THE REPORT, I WAS PROVIDED WITH THE FOLLOWING DOCUMENTATION:

1. E-mail from Jane Winter outlining the issues that required to be addressed
2. Executive Summary
3. Draft synopsis of evidence in the case

COMMENTS

1. I have read the documentation sent to me and am aware of the circumstances relating to Robert Hamill's death. My views below are based solely on the documentation available to me.

2. In relation to the question of whether his death could have been prevented, if the police officers had intervened earlier, it is not a matter for my expert opinion.

3. I am informed that Robert Hamill was knocked unconscious and almost immediately, possibly after being hit with a bottle, was then kicked by the crowd as he lay on the ground.

4. Clearly from examination of the draft synopsis of evidence given by various medical experts, Robert Hamill had suffered severe head injuries which led to his death. The hypoxia seen in the brain was a secondary phenomenon due to the head injury.

5. The position he may have been placed in by officers in my view would not have made any difference to the fatal outcome and furthermore did not lead to the hypoxia found in the brain.

6. Neuroleptic malignant syndrome which was commented on by the medical experts, in my view was a complication of the head injury and not due to any new event.

7. Robert Hamill, from the evidence I have read, had suffered multiple blows to his head which resulted in severe generalised brain trauma (diffuse axonal injury) from which he did not survive.

8. His treatment in hospital was appropriate and that the Chlorpromazine given to him was reasonable and necessary and furthermore did not contribute to his death.

9. The actions of police officers in their response after the attack would not have made any difference, in my view, to the eventual outcome.

Professor Peter Vanezis, OBE
MB, ChB, MD, PhD, FRCPath, FRCP(Glasg.), FFFLM, DMJ(Path)Director, Cameron Forensic Medical SciencesHome Office Accredited Forensic PathologistSenior Consultant in Forensic Medicine to the Armed Forces.

Submissions by Gus Campbell Solicitors (Marc Hobson)

If DAI seems not likely to be the immediate cause of death and the administration of the Chlorpromazine, as opposed to the actual dosage given, was not unreasonable and therefore did not break the chain of causation from injury to death, then whatever finding of fact the Inquiry arrives at as to whom was responsible for the injury no criticism can be made against any person convicted of or alleged to have been part of any assault as guilty beyond all reasonable doubt or on a balance of probabilities to have committed that murder and at most only potentially liable for inflicting S.18 GBH and the tribunal cannot as a finding of fact state who was responsible for RH death within their terms of reference.

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

With reference to "whether there was some other intervening cause", definition of the suggested intervening cause should also be given and expanded to outline (a) what other intervening cause and/or causes are suggested and a detailed definition of same be provided and (b) what are the suggested consequences of the intervening cause and/or causes.

The reference to the medical findings should be explored not just to what they reveal about the intensity and/or duration of the assault but should also include whether those findings reveal the nature of the assault; e.g. blow to person, bottle, kick, kicks, strike of person, hitting head on hard surface of ground. Further whether the medical findings give guidance as to the effect of the positions and/or position of Mr. Hamill's person once on the ground, e.g. the cradling of his head by Michelle Jameson and the actions taken by police officers (e.g. Silcock) prior to the ambulance arriving. was the omission of any action which reasonably could have been expected by those parties dealing with Mr. Hamill's person borne out by the medical evidence as a cause or contribution to Mr. Hamill's death.

Submissions by the Police Service of Northern Ireland

See below.

THE FIRST ISSUE: INTERVENING CAUSE?

Hypoxia

2. In respect of an intervening cause, there were suggestions prior to the hearings of oral evidence that Mr Hamill may have been starved of oxygen between the time of the assault and the time he was seen in the Craigavon Area Hospital.

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

In Professor Vanezis' opinion, the hypoxia was a secondary phenomenon due to the head injury.

Submissions by the Police Service of Northern Ireland

See 6 below

3. The materials available which deal with that are to this effect:

3.1 Res Con Cornett described the second man (Mr Hamill) as having shallow breathing. She said that his breathing was "husky" and she thought he had a punctured lung (9440).

3.2 Res Con Silcock said he thought Robert Hamill had difficulty breathing that and he was rasping (10478). He made a statement in which he says that he saw that Robert Hamill was unconscious and having difficulty breathing. Robert Hamill was being cradled by his cousin who was on her knees on the ground. His head was in an upright position. Res Con Silcock put his hand up Robert Hamill's back and noticed that he was now rasping for breath. Robert Hamill was placed front down and his head was turned to the side (702).

3.3 When Con Neill saw Robert Hamill on the ground he went over to him. His breathing was so rasping it sounded like a death rattle (9389).

3.4 Mr Hamill was put on oxygen in the ambulance (9186 & 9188).

3.5 Mr Fannin (consultant neurosurgeon) stated in the Discharge Summary that the primary diagnosis was of closed head injury; the subsidiary diagnosis was of cerebral hypoxia (26166).

3.6 Mr Fannin made a final comment that the cause of death was a relatively minor head injury; in all likelihood Robert Hamill was hypoxic at the scene (26101).

3.7 Following the DPP Direction of 13 May 1997 DCS Maynard McBurney and DI Michael Irwin visited Raymond Kitson of the DPP to brief him in relation to their visit to Professor Jack Crane. They reported that Professor Crane stood by his initial finding that the cause of death was a head injury, but that in itself should not have caused the death. Other contributory factors such as alcohol, the position in which the deceased lay and oxygen starvation were all likely to be present. They reported that further tests were to be carried out (19068).

3.8 Dr Boon Low made a statement to the effect that Mr Hamill's airway was partially obstructed and his jaw was in spasm. He was not maintaining sufficient oxygen saturation. He was immediately bagged with 100% oxygen through a mask (674).

3.9 Mr Fannin wrote to the Director of Risk and Litigation Management. He concluded that initially it had been thought that it was a relatively minor brain injury but the possibility of secondary insult such as hypoxia and the effects of high blood alcohol had also to be taken into account (38811).

3.10 Helen Reid (neuropathologist) made a report in which she said there was some evidence of hypoxaemic change but it was mild. It may have increased the white matter damage but by no more than an a third (72526).

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

It is alarming to hear that Robert Hamill was placed front down with his head turned to one side by a police officer (3.2). This is not the usual recovery position and is a position which might lead to positional asphyxia. However, Professor Vanezis is of the opinion that the position in which Robert Hamill was placed did not make any difference to the fatal outcome of the attack and did not lead to the hypoxia found in his brain.

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

The Panel will be referred to Mr. Fannin's evidence at 3.6 above, that the nature of the head injury was a minor injury and that Professor Crane (3.7 above) considered other contributory factors such as alcohol and the position in which the deceased lay to be present. Helen Reid at 3.10 notes that the hypoxaemic change was mild.

Submissions by the Police Service of Northern Ireland

See 6 below.

Submissions by the Public Prosecution Service

Correction

§3.7 refers to the *"DPP Direction of 13 May 1997"*. It is not correct to describe this as a *"DPP Direction"*. *"Direction"* is a term of art. As Mr Kitson explained in his evidence to the Inquiry, the use of the term by DCS McBurney is inappropriate (15.9.09, Day 63, pp35-6).

4 Witnesses were asked about the issue, and their written and oral evidence can be summarised as follows:

Boon Low

Statement

4.1 Para. 39: It is unlikely that first aid or different medical treatment would have had an effect on the injury.

4.2 Para. 40: There was no evidence of hypoxic damage over a significant period.

4.3 Para. 41: There was insufficient evidence to establish hypoxia as cause axonal injury or death.

4.4 Para. 42: There was obviously some hypoxic damage

Oral evidence

4.5 There were signs of upper airway obstruction (p.40).

4.6 Robert Hamill had an oxygen saturation of 75%, which is very low. 75% does not support consciousness. Dr Low believes it was associated with airway obstruction (p.40).

4.7 When trying to insert an airway, Robert Hamill's jaw was clenched shut. He cannot explain why the jaw was clenched but on the reading notes, believes Robert Hamill had cerebral irritation, which causes spasms.

4.8 If it was definitely established there was no hypoxic brain injury then whatever first aid was given, it would not have prevented Robert Hamill's death.

4.9 There was some evidence of oxygen deprivation in the brain but not enough to suggest hypoxia was the main cause of brain damage (pp.20-21).

Dr Herron

Statement

4.10 Para.33: If he were writing up his findings again he would discuss hypoxia/ischaemia more as was some acute reaction that occurred shortly before death but this would not have explained all his injuries or Robert Hamill's prolonged unconsciousness.

4.11 Para.34: Per Dr Reid's statistical analysis (72533), which suggested hypoxia contributed to the worsening of the initial brain injury by less than one third. Dr Herron knows of no literature or mechanism that would allow him to make a statement like that.

4.12 Para 35: Per Dr Todd 72644, need to be aware that uncommonly hypoxia in the absence of trauma can be associated with axonal injury. He states that looking at the case overall, the distribution of axonal injury is absolutely consistent with trauma, not hypoxia.

Oral evidence

4.13 Mr Hamill had no hypoxic/ischaemic damage at the time of admission. It was not the cause of his unconsciousness and it was not the cause of his axonal damage. Evidence of hypoxia is due to an 11 day reaction in the brain (p.65).

Dr Lawler

Oral evidence

4.14 He has no doubts that TDAI (Traumatic Diffuse axonal injury) was caused during the assault (p.16). The hypoxic/ischaemic damage sustained is no more or less than would be expected in association with head injury causing Grade II TDAI. It follows Robert Hamill did not sustain significant hypoxic/ischaemic damage when he was in the roadway.

4.15 The lack of First aid did not cause or significantly exacerbate any injury sustained during the assault (p.21). He found nothing to suggest that the hospital treatment caused, contributed to, or exacerbated Robert Hamill's injuries.

Maureen Millar

Statement

4.16 Para. 9: Robert Hamill was taken into the Resuscitation room. Given that his breathing was "loud & snorty," oxygen would have been applied straightaway.

Oral evidence

4.17 Robert Hamill's face was very, very ruddy on arrival (p.64).

4.18 That could be due to poor respiration. (p.74)

Glen Stewart

Statement

4.19 Para.10: His priority was to maintain Robert Hamill's airway and enable oxygen flow.

4.20 Para 14: He tried to insert a plastic airway but Robert Hamill's teeth were still clenched. He was given oxygen therapy with a mask and bag.

David Morrow

Oral Evidence

4.21 He gave him oxygen in the ambulance (p.11).

4.22 He does not recall anything about raspy breathing. He checked Robert Hamill's head and found liquid but thought it was from bottle as they checked for blood (p.12). He recalls a bottle nearby.

Submissions by Edwards & Co Solicitors (Serving and Retired Police Officers)

It is clear from the evidence of Reserve Constable Silcock and others that the police administered first aid to Robert Hamill at the scene prior to the arrival of the ambulance. This allegation, that the Police did not do so, is a matter which caused understandable anguish to the Hamill family as a result of lies told mainly by Hull and McNiece.

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

We would refer the Panel to the evidence of Dr Boon Low at 4.1 above which refers to paragraph 39 of his statement stating "It is unlikely that first aid or different medical treatment would have had an effect on the injury."

Submissions by the Police Service of Northern Ireland

See 6 below.

Comment

5. The Panel may wish to consider whether, while there is evidence that Mr Hamill was hypoxic at the scene and on admission to the hospital, it was not a contributory factor in his death. Even if it were the Panel may need to determine whether it was a direct result of the assault.

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

Please see comment at 2 above

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

We take no issue with the conclusion from the evidence that Mr. Hamill was hypoxic at the scene and his admission to hospital and that it was not a contributory factor in his death. We would suggest it is appropriate to determine whether or not Mr Hamill being hypoxic at the scene was a direct result of the assault. Further the evidence of Doctor Lawler suggests that Robert Hamill did not sustain significant hypoxic/ischaemic damage when he was on the roadway (4.14 above) and further at 4.15 the evidence is that the lack of first aid did not significantly exacerbate any injury sustained in the assault.

Submissions by the Police Service of Northern Ireland

The evidence from Res. Con. Silcock establishes that Mr. Hamill appeared to be experiencing difficulties with breathing at the scene. He intervened to examine Mr. Hamill and by directing that he be placed in the recovery position and by assisting with this procedure.

The PSNI rely upon the findings of Dr. Boon Low. He has stated that there was no evidence of hypoxic damage over a significant period (para 40). He has opined that any absence of first aid did not effect the outcome (para 39). Dr. Lawler has reached similar views (p.16 and 21).

Medication

6. The experts disagree about whether Mr Hamill died because he had DAI ("Diffuse Axonal Injury") resulting from the assault or whether he contracted neuroleptic malignant syndrome ("MNS" or "NMS"). Nonetheless, they appear to agree that whatever was the immediate cause of death, it appears to have resulted directly from the assault.

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

Professor Vanezis is of the view that diffuse axonal injury was the cause of death and that neuroleptic malignant syndrome was a complication of the head injury and was not due to any new event.

Submissions by Edwards & Co Solicitors (Serving and Retired Police Officers)

We agree with this statement

Submissions by the Police Service of Northern Ireland

See section 9 below.

7. The materials dealing with the extent of agreement and disagreement show this:

7.1 Mr Fannin stated that there was a sudden, dramatic deterioration in Robert Hamill's condition on the afternoon of 8 May 1997 and he ultimately died later that afternoon. Several possibilities were considered, including a pulmonary embolus from a deep venous thrombosis, a septicaemic episode or even a malignant neuroleptic syndrome, which can occur secondary to the use of neuroleptic drugs of various types (38811).

7.2 Dr Brian Herron produced report and commented that the dominant finding was a severe diffuse axonal damage of a pattern consistent with being caused by trauma indicative of a head injury (31395).

7.3 Dr Patel made a statement about Robert Hamill's death. He said that Robert Hamill was seen at 15.30 on 8 May 1997. He was pale with no recordable blood pressure and had a temperature of 42C (9209).

7.4 Professor Jack Crane made a statement and a post-mortem report. He concluded the cause of death to be diffuse brain injury associated with fracture of the skull due to blows to head (9211). See also the report of the autopsy p954.

7.5 Professor Crane was cross-examined at the trial of Mark Hobson and he discounted malignant neuroleptic syndrome (MNS) because of the absence of relevant findings in the kidneys, liver and brain. He discounted a number of other possible causes, which left diffuse axonal injury. He stated that there was no bruising to the back of the scalp and he gave the example that a man who was running and who had slipped hitting the back of his head could suffer the same injury. He also stated that there was no correlation between the dosage of chlorpromazine and the onset of MNS. In addition, he stated that axons are a fixed part of the brain and there is no brain function without them (p.8487 – 8541).

7.6 Helen Reid's report concluded that there was Grade 2 traumatic DAI but not severe. In her opinion Robert Hamill had sustained blows to his head and during those injuries he had acceleration/deceleration damage to his brain. She agreed that his death was probably due to NMS. (72526: Dr Reid Report).

7.7 Dr William Lawler made a report in which he found the immediate cause of death to be NMS. There was an "unequivocal and incontrovertible" direct causal relationship between the assault and the death. The prescribing of chlorpromazine seems to have been perfectly reasonable. There was nothing in the treatment or omission of treatment which contributed in any way to the death of Robert Hamill (72226).

7.8 Mr Todd made a report. He commented on whether there was diffuse axonal injury (72617). The report was followed up with a letter dated 18 July 2006. He commented on Dr Lawler's findings of 23 November 2005 on NMS and stated that his general understanding is that Dr Lawler was correct about the use of chlorpromazine for injured head patients in an agitated state (72811);

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

The dispute regarding the medical evidence rests with the Panel

Submissions by the Police Service of Northern Ireland

See section 9 below

8. Again, witnesses were asked to deal with this issue in writing and orally:

Boon Low

Statement

8.1 Para 7: He recorded a history of "Assaulted? Hit of left side head by bottle. Attacked by nine people" from someone. He did not know who.

Professor Crane

Statement

8.2 Para. 29: Ruled out NMS due to absence of damage to kidneys. There was no rigidity in muscles or necrosis in brain.

8.3 Para.30: Injury caused damage to hypothalamus, which controls body temperature. This may explain Robert Hamill's raised temperature, although raised temperature is a common symptom of head injury.

8.4 Para. 31: He would have expected adverse reaction to chlorpromazine on 1st or 2nd May as the drug breaks down quickly and does not build up in body. NMS is an idiosyncratic reaction so he does not know when someone will get NMS.

8.5 Para. 32: Did not include NMS in autopsy report as he tended to put his opinion in his report. NMS is not included in the notes as they are contemporaneous and do not cover later tests.

8.6 Para. 34: He did not know how Dr Lawler based his diagnosis on elevated CK ("Creatinine Kinase") and temperature, both of which can be explained (see above). Dr Lawler failed to consider the absence of changes in liver, brain & kidneys.

Oral Evidence

8.7 Robert Hamill's external injuries do not provide any indication to extent or severity of attack (pp.4 and5) (p.959)

8.8 If the attackers had worn trainers, there may not have been as many external injuries although, even if trainers were worn, he would have expected more bruising to the under surface of the skull if there were concerted kicks (p.39).

8.9 The diffuse axonal injury suffered by Robert Hamill is compatible with people jumping on his head (p.9). A severe amount of force is needed to cause DAI and cause loss of consciousness. (p.10).

8.10 Being struck by a thrown bottle would not generate sufficient force to cause DAI (p.11).

8.11 Punches on their own would probably not generate sufficient force to cause DAI (p.40).

8.12 The severity of DAI is not necessarily an indication of the length of the attack (pp.11-12). It is caused by the severity and not the duration of assault (p36).

8.13 There is no causal link between the degree of violence and place of injury with diffuse axonal injury (pp.12-13).

8.14 The fact that Robert Hamill was unconscious and remained so indicated severe DAI. Damage to the brainstem leads to an extremely poor prognosis (p12/13). He believes that Robert Hamill would have died from DAI (p.25).

8.15 DAI caused by "accelerated fall" is a possibility but is considerably less likely than blows, particularly with evidence of more than one injury to the head (pp.18/19). Furthermore, the most likely cause of DAI was the blows inflicted whilst Robert Hamill was on the ground (p.35).

8.16 CK levels were not high enough to indicate NMS. Would expect CK in the thousands.

Dr Herron

Statement

8.17 Para. 12: Robert Hamill's brain was abnormally heavy. There was no subarachnoid haemorrhage and there was no meningitis. The main findings were haemorrhages in the deep white matter in the brain on either side of the midline (parasagittal location). There was also haemorrhage in the thalamus and internal capsule. The white matter was congested. He did not see any focal haemorrhage in the corpus callosum, but Dr Herron did see what he described as diffuse punctuate haemorrhage in the brain stem. The cerebellum showed no specific abnormality.

8.18 Para. 14: Findings suggested Robert Hamill had diffuse brain injury that involved deep structures of the brain, which was important because he had been unconscious. Macroscopic findings suggested DAI was a cause of unconsciousness.

8.19 Para. 15: In 1997, DAI implied the cause of axonal damage was trauma to the brain. The terminology has now become more specific and DAI is now used to describe anything that affects the brain causing damage to the axons. That is the reason why Dr Reid in 2005 refers (72526) to "TDAI" as the "T" more accurately represents traumatic.

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

8.21 Para. 19: Several things need to be remembered when reading Dr Herron's report. Dr Reid's report was written in 2005, when more was known about DAI. In addition, recommendations relating to examination of the brain have changed. At 72533 Dr Reid suggested that too few blocks were taken, but examination guidelines were not published in 1997. Since his report more tests have become available, including the Beta APP test used by Dr Reid described at 72530. He would definitely have used this test if it were available, although it is more useful in the first few days after the injury. Even without this test, he had found enough damage to brainstem using routine stains to support his original findings.

8.22 Para. 20: The grade of injury reflects a difference in clinical & pathological grading systems. The impression he wanted to give was that this was a severe injury and there was severe brainstem involvement, which would have explained the clinical features.

8.23 Paras. 22 & 23: He disagrees with Dr Reid about the technicality of the grading.

8.24 Para. 24: It was the severity of the injury in the structures that matter. The bulk of the worst injury in this case was in the lower brain structures, which are more critical for survival, particularly for respiration & cardiovascular function. It may be misleading to use grading.

8.25 Para. 25: The cause of DAI would have been a severe rapid movement of the head.

8.26 Para. 27: The Glasgow Coma Scale reading of below eight indicates this was a serious brain injury.

8.27 Para. 28: The Distinction between the macroscopic finding of diffuse punctuate haemorrhage at 31395 and the microscopic finding of secondary brain stem haemorrhage in midbrain at 31396 is that secondary brain stem haemorrhage is a completely different pathological finding from diffuse punctuate haemorrhage as they occur at different times in the brain injury.

8.28 Para. 29: The question as to what caused Robert Hamill's demise is more difficult and open to speculation. Diffuse brain injury may have led to his death or permanent unconsciousness. He could never have returned to his pre-injury state. Dr Herron was not entirely surprised at Robert Hamill's death, unlike the clinical team, given the amount of brainstem injury from DAI.

8.29 Para. 30: He believes Dr Todd's conclusion (72646) that Robert Hamill died as a consequence of a grade III severe DAI in the absence of any other lesions is very reasonable. He refers to "Geddes" paper as describing various mechanisms that can cause death from DAI.

8.30 Para. 36: He disagrees with Dr Reid's conclusion at 72533 that NMS caused Robert Hamill's death.

8.31 Para. 39: Other reasons why it was unlikely to be NMS is because there was no myoglobinuria and there was no liver damage. In Dr Herron's previous cases, the liver was almost completely necrotic.

8.32 Para.40: Upon examination of the brain, he did not find neuropathological changes that can be attributable to NMS. The Purkinje cells were preserved. These cells are very sensitive to damage caused by hypoxic/ischaemic effect and have been specifically damaged in previous NMS cases.

8.33 Para. 42: He had asked neurosurgical and neurology colleagues about Robert Hamill's CK levels. The levels would not be of concern in the event of someone being in intensive care following an assault where there were bruises and muscle damage. It is normal to have a raised CK level, purely due to muscle damage.

Oral Evidence

8.34 It is debated as to whether DAI can result from a kick or kicks to the head (p.55).

8.35 One theory is that it could be caused by an accelerated fall. (p.54).

8.36 It would be very surprising to get DAI from the impact of a thrown bottle if the victim remained standing although if the person fell, it is possible (pp. 55/56).

8.37 Whether it is more probable to get DAI from a blow and accelerated fall than by a kick or kicks cannot be answered (p.76).

8.38 If a person is on the ground and unconscious then kicks may render DAI more likely as the neck does not protect from acceleration/deceleration. However, this is not from Dr Herron's neuropathological knowledge, only simple physics. (p.77).

8.39 It is not easy to draw an equation between amount of axonal damage and what caused it (p.79).

8.40 He had lot of experience at that time of NMS (p.59). He felt cause of death was not attributable to NMS as Robert Hamill lacked many features of NMS e.g. no inability to bend the limbs.

8.41 Temperature can be explained by damage to the hypothalamus (p.60).

8.42 Creatinine Kinase (CTK) result (suggested by Dr Lawler) was 900, 970 in Robert Hamill. In other cases CTK level was 50,000 to 60,000 (p.61). Numerous papers say elevated CTK levels can be caused by assault or injections in hospital (p.61).

8.43 He said there may be an unknown spectrum and for that reason he did not want to rule out NMS (p.61).The sudden deterioration of Robert Hamill and the actual direct cause of death are unknown. (p61/62)

Dr Lawler

Statement

8.44 (p.16): He has no doubts that TDAI was caused during assault. The hypoxic/ischaemic damage sustained is no more or less than would be expected in association with head injury causing Grade II TDAI. It follows Robert Hamill did not sustain significant hypoxic/ischaemic damage when he was in the roadway. The bruising on the left side of his abdomen represents areas of blunt force trauma.

8.45 (p.18): The reasons for NMS diagnosis: Marked fever; autonomic problems e.g. hypertension, sweating; significantly elevated CK level; elevated AST ("Aspartate aminotransferase") concentration; possible mild/early renal function impairment. There is no other explanation for these findings.

8.46 (p.19): He disagrees with Dr Crane that the lack of structural changes in brain, liver and kidneys exclude NMS. He disagrees with Prof Crane that NMS cannot develop in someone being treated for a week. Delay is not uncommon and once started, NMS can develop very quickly.

Oral Evidence

8.47 DAI was most likely caused by fall combined with a kick or kicks (p.25).

8.48 It was extremely unlikely that DAI was caused by an accelerated fall (p.9). If it had been an accelerated fall that caused the DAI then there would have been more external injuries (pp.26/36). An accelerated fall occurs when a fall involves considerable force, which contributes significantly to the force transferred to head on impact (p.27).

8.49 He believes NMS was the cause of death (p.11). He refers to the reasons at 72233 to show evidence for NMS (p.15).

8.50 NMS is an abnormal reaction to a drug that is neither an allergic response nor cumulative (p.40).

8.51 He believes, contrary to Dr Herron, that CTK levels were sufficiently high as to not provide a reasonable alternative explanation except NMS (p.32).

8.52 The high temperature due to hypothalamus damage is discounted as hyperpyrexia would have occurred earlier (p.17)

Dr Todd

Report

8.53 Para. 4. Histological examination of the brain showed no evidence of hypoxic ischaemic damage in neuronal structures that would be subject to such damage.

8.54 Para. 5. Histological examination of the brain demonstrated a DAI (a shearing injury of the white matter tracts) widely distributed throughout brain; in some areas that damage was thought by Professor Crane to be extensive.

8.55 Para 6. Such a DAI, widespread and in parts extensive, would typically be associated with high velocity road traffic accidents where there are severe and abrupt acceleration/deceleration forces.

8.56 Para 7. Such a pattern of injury does not usually follow a simple assault where a punch leads to the patient being knocked out briefly.

8.57 Para 8. In his opinion, Mr Hamill's head/brain was subjected to such significant forces that it caused the sort of severe axonal injury that is normally associated with high velocity road traffic accidents.[72643] In 1992, Graham et al published 15 cases of fatal head injury caused by an assault where there was neuropathological evidence of DAI. In 10 of the 15 patients the DAI was severe (Grade 3). In some cases, the assault was a punch or punches, in other cases, there were further injuries to the head, either an assault with a heavy object or kicking, in some cases, the victim fell striking his head on the ground. Graham et al make the point that in most of their cases full details of the nature of the assault were not available.

8.58 There are a number of reports of axonal injury following assault. We have already considered the 15 patients reviewed by Graham et al. Grade 2 DAI has been identified in a man who died thirteen days after an assault. The paper contains the witness evidence that 'the victim was attacked while lying on the ground. The perpetrator stomped and kicked the victim and also struck the victim's head with a piece of cement'. This suggests that severe injury is required to cause a grade 2 DAI following assaults.

Dr Reid

Report

8.59 In this case there is no description of a macroscopic infarct of any type and no infarcts are present in the sections. There is some hypoxaemic/ischaemic neuronal damage, that is pink neurones are seen and if this is graded as in the reference from Graham et al it is diffuse and mild. This change can occur within an hour of hypoxic/ischaemic damage. It can also be due to other insults than the original episode when he was assaulted. Hypothermia itself can also cause hypoxic/ischaemic neuronal damage.

8.60 [72533] from the information and slides available: (a). I do not agree that there is severe traumatic DAI in this case. The grade of DAI is II with scattered white matter damage. (b). The lesions seen histologically showing a macrophage response with little in the way of ongoing damage are not, in my opinion, enough to cause his sudden death. The probability of his death through Neuroleptic Malignant Syndrome is one which I would agree with. (c). The white matter damage may have been made worse by hypoxaemia/ischaemia, but these lesions in the white matter are not enlarging with more recent changes in the surrounding white matter.(d). There is hypoxaemic/ischaemic neuronal change, which is diffuse but mild as it involves several gyri in several slides, but there are neither cerebral infarcts nor evidence of laminar necrosis in the cortex.

Mr Todd

Report 2 [72811]

8.61 I do not consider myself an expert in the diagnosis of the neuroleptic malignant syndrome. My general understanding is that Dr Lawler's list of major findings in the neuroleptic malignant syndrome are correct.

8.62 Dr Lawler also comments that he would be unable to distinguish the neurological features that were a consequence of NMS and those that were attributable to the primary brain injury. I agree with that. We know that Mr Hamill had a grade 3 DAI that involved the brain stem. Such brain stem damage could be associated with marked fever (probable hypothalamic damage) with autonomic problems (brain stem injury) and rigidity (for example, the arching of the back noted on 01.05.97).

8.63 However, what I can say with complete confidence is that the use of chlorpromazine in a head-injured patient who is agitated and restless is entirely reasonable. The neurosurgeons cannot be faulted for using chlorpromazine under these circumstances, and of course it will be recognized that the development of the neuroleptic malignant syndrome is rare.

8.64 In my report I accepted Professor Crane's view that Mr Hamill suffered a severe grade 3 DAI. Dr Reid believes that the DAI was less severe, grade 2. This weakens my suggestion that Mr Hamill suffered a prolonged and violent assault rather than a single blow to the head and it supports my view that it is not beyond reasonable doubt that he suffered a less severe primary injury causing grade 2 axonal injury.

8.65 I remain of the view that Mr Hamill's head injury was at a level of violence considerably greater than the average 'Saturday night punch-up'. As you will have noted from my supplementary report, it is simply not possible to be certain as to what level of head injury Mr Hamill suffered even if there was a grade 3 DAI, and still less when the DAI is not considered to be grade 2.

Dr Fannin

Statement

8.66 He regards DAI as a perfectly acceptable diagnosis (80251)

Dr Gray

Statement

8.67 He agrees with Professor Crane's autopsy report

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

The Panel are referred in particular to the oral evidence of Professor Crane at 8.12 above stating that the severity of DAI is not necessarily an indication of the length of the attack. It is caused by the severity and not the duration of the assault.

Submissions by the Police Service of Northern Ireland

See section 9 below

Comment

9. It is clear that Mr Hamill suffered axonal injury. Further, the positioning of it, deep in the brainstem, appears to show that it was a serious injury, which would have led to permanent incapacity at best. However, the Panel may wish to consider whether it seems likely that the DAI was not the immediate cause of his death, and that the only other explanation for it is the reaction to chlorpromazine. It has not been suggested that the prescription of that drug was unreasonable, and it follows that it did not break the chain of causation between the assault and Mr Hamill's death.

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

Please see comments at 6 above

Submissions by P J McGrory Solicitors (Family of Robert Hamill)

CAUSE OF DEATH

1. Overview

2. Primary Injury

3. Hypoxia

(a) Observations

(b) Pathology

4. Medical Treatment

5. Causation

Overview

1. The Inquiry have identified in their closing submissions and indeed in their opening two questions which may have to be addressed in the context of the issue of the cause of Robert Hamill's death.

2. The first is whether the assault was the cause of Robert Hamill's death or whether there was some other intervening cause.

3. The second is whether the medical findings reveal something about the intensity and duration of the assault.

4. It is the family's submission that it is inappropriate to deal with the second of these two matters in the context of the Inquiry's document on the cause of death. It is our submission that the evidence as to intensity and duration of assault must be viewed as a whole. That is to say the medical findings cannot be considered in isolation from the eyewitness evidence regarding the blows struck. The evidence of the latter may, for example, help to reconcile conflicts in the former and vice versa.

5. In respect of the first question it is assumed that the use of the phrase "intervening cause" means an intervening act sufficient to break the chain of causation between the primary injury as the result of the assault and the death of Robert Hamill.

6. It is the family's submission that this question, (which does relate directly to the cause of death) as a matter of evidence may be divided into two parts:

7. Firstly whether there was in fact an intervening event or process subsequent to the initial injury from the assault which can be considered a material cause of death.

8. Secondly, if the panel do decide that there was in fact an intervening cause and that it materially contributed to the death, then the question becomes one of whether it was sufficiently material and independent of the original injury so as to break the chain of causation.

PRIMARY INJURY

9. Of course it is implicit in the foregoing analysis that there must be a finding of fact as to the nature of the major or primary injury sustained as a direct result of the assault. All the medical witnesses are agreed that the primary injury to Robert Hamill was Diffuse Axonal Injury. Their findings are as follows.

(i) Professor Jack Crane (State Pathologist)

Autopsy Report (9567)

"Detailed examination of the brain however and in particular its microscopic examination revealed widespread damage within its substance of a type known as diffuse axonal injury. This condition, most frequently encountered in acceleration/deceleration injury as a result of road traffic accidents is also well recognised as occurring as the result of repeated blows to the head such as by punching or kicking and this would seem the most likely mechanism of injury in this case."

Inquiry Statement (80218)

Para. 13-16
"Significant injury in this case was diffuse axonal injury (DAI). This injury may occur without there necessarily being severe external injury. Occurs as a result of the brain being shaken inside the skull. It is recognised as occurring in assaults particularly those lying on the ground when kicked. DAI shows up in pathology examinations rather than CT scan as the former are microscopic."

During both his oral evidence at trial and to the Inquiry Prof Crane's finding that there was DAI was not challenged. His evidence rather concentrated on what degree of force and what mechanism might produce DAI.

(ii) Dr Herron (Neuropathologist)

Prof Crane's findings were informed by the neuropathology report of this witness

Report 29/10/97 (31396)

Comment:
"In summary, the predominant finding in this case is the severe diffuse axonal damage… This pattern of injury is consistent with having been caused by trauma. It is indicative of a severe head injury"

Inquiry Statement (80441)

Para14
"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."

(iii) Dr Lawler (Independent Consultant Pathologist)

Report 23/11/05 (72236)

"As I believe that the neuropathology is fundamental to the pathological interpretations in this case, I have advised that this aspect be reviewed by a specialist neuropathologist"

Dr Lawler then goes on, later in his report, to adopt the findings of Dr Reid.

Oral evidence (16-01-09)

"25 A. I think that we have evidence externally and internally

8
1 of significant blunt force injury to the deceased's
2 head. I have seen cases where there has been far more
3 than there is here. Of course I have, but,
4 nevertheless, there is evidence that there has been
5 significant blunt force trauma.
6 I think that we have to look very closely at the
7 neuropathological findings in this context also, because
8 I think that the extent of the diffuse axonal injury
9 described by all those who have examined the microscope
10 sections from the deceased's brain I think also reflects
11 the fact that there have been multiple impacts to the
12 deceased's head."

(iv) Dr Reid (Independent Neuropathologist)

Report 21-11-05 (72532)

"THE EXACT NATURE OF THE BRAIN DAMAGE
In my opinion he had sustained blows to his head which resulted in the fractured left sphenoidal wing and the haematoma over the left supra-orbital plate…

During these injuries he had acceleration/deceleration damage to his brain resulting in traumatic diffuse axonal injury of Grade II microscopically."

Whilst, in our submission it can be clearly established as a fact that DAI was the primary injury the question then arises whether it can be established as a fact on the available evidence that there was an intervening act which materially contributed to Robert Hamill's death. The Inquiry submissions in this regard focus on two possibilities, firstly that of hypoxic brain damage as a result of first aid or the lack thereof; and, secondly the possibility of Neuroleptic Malignant Syndrome as a result of the administration of

Chlorpromazine by the medical personnel treating Robert Hamill's. (These two matters are referred to at various points in the Inquiry bundle as potential factors in Robert Hamill's death.)

HYPOXIA

10. The medical evidence on this issue relates to the observations of eyewitnesses at the scene and the medical personnel treating Robert Hamill's and the scientific findings of both sets of Pathologists.

(a) Observations

(i) R/Con Cornett
Police Interview re Neglect 2-10-97 (9464)

"…then I went to (Robert Hamill), now he had hustly breathing, it was very hustly and I actually thought that he could have had a punctured lung and I was, I sort of worried, and that's why I kept screaming for ambulances"

(ii) R/Con Silcock

Police statement 27-04-97 (0700)

"…having difficulty breathing…"

Disciplinary interview 20-09-01 (10480)

"…rasping from his breathing…"

Inquiry Statement (81161)

Para 9
"…I could hear that he was struggling with his breathing. I am trained in first aid and put my hand on his back to turn him over into the recovery position. It was clear to me from the vibration I felt in his back as I moved him that he was definitely having trouble breathing."

(iii) Con Neill

Police statement (00680)

"The male outside Eastwoods was breathing, rasping…"

Police Interview re Neglect (09417)

NEILL "…I had already been over to
Hamill and he was, his breathing was really rasping

Well what do you mean by rasping now ?

NEILL It was going, it was like, it was nearly the death rattle you know the last (intake of. breath) it was.

As if he couldn't breath?

NEILL As if he, as if, actually he wasn't breathing through his mouth as if it was somewhere else.

Inquiry Statement (81035)

Para 29
"…he was breathing but it was raspy."

Oral evidence 19-05-09

20
9 Q. Was he breathing in a laboured way throughout?
10 A. It was just sort of raspy, you know, whether it was --
11 just a raspy sort of breath, but he was still breathing,

(iv) David Morrow (Paramedic)

Inquiry Statement (80978)

Para. 15
"He did not have any difficulty breathing but an unconscious patient will breathe more slowly and so it is standard procedure to give them oxygen. Glen (Stewart) and I agreed at the time that he had a good strong pulse and a good colour which shows he was receiving adequate oxygen."

(v) Glen Stewart (Paramedic)

Inquiry statement (81204)

Para. 10
"he was breathing on his own but I would say it was laboured."

Para. 14
"I tried to insert a plastic airway into his throat to assist his breathing but his teeth were clenched. Mr Hamill's breathing was still laboured in the ambulance…"

Oral evidence 25-02-09 para 8.

"8 Can I get you to expand, please, on this and tell us
9 what you mean by "laboured breathing", and how that
10 would have appeared at the time?
11 A. Well, laboured breathing is, when we breathe normally,

you wouldn't hear us unless somebody would get down with their ear to listen to your breathing, but you could hear the noise when he was breathing, laboured breathing when -- the breathing was slow. It wasn't -- it was out of the normal sync for anybody breathing. So it was like slow breathing, but there was a noise, noisy breathing. It is classed as laboured breathing when it is slow and noisy.

Q. Would "raspy" describe it?

A. "Raspy" could be fair enough to describe it, yes.

(vi) Maureen Millar (Nurse in Charge CAH A&E)

Inquiry Statement (80966)

Para. 5
"He was brought in by ambulance and was being given oxygen en route to the hospital."

Para. 9
"Given that his breathing was very loud and snorty, oxygen would have been applied straightaway."

Oral Evidence

67

Q. Then you have, "Respiration:" is "inadequate". Can you help us with any recollection you have about that?

A. His breathing, I recall, was quite snorty, loud and inadequate. He wasn't breathing properly, which meant that his oxygen levels wouldn't be very good. So he would have oxygen applied immediately.

(vii) Maureen Hagan (Staff Nurse CAH A&E)

Inquiry Statement (80339)

Para. 20
"I have also written in the top right hand corner of the (triage) form: "unconscious, breathing noisily, wound to back of head". I obtained this information from my own observations of the patient."

(viii) Dr Boon Low (SHO CAH A&E)

Inquiry Statement (80691)

Para. 17

"I noted that Mr Hamill was breathing but there was evidence of upper airway obstruction and he would not open his jaw. I was trying to insert an airway into his mouth to lift the tongue out of the way to help with the breathing as this is the common cause of something obstructing the airway, but his jaw was clenched tight."

Para. 19
"Mr Hamill's oxygen saturation was 75% which is a very life threatening level of oxygenation. It was therefore necessary to "bag him". By this I mean putting a mask over his face and squeezing a bag which delivers oxygen to help him breathe. In effect, blowing air into his lungs. I was able to tell there was upper airway obstruction because Mr Hamill was breathing noisily which is usually a tell-tale sign of upper airway obstruction. His circulation appeared to be stable…"
Oral evidence (14-01-09)

38
19 Q. Where you say:
20 "Upper airway obstruction. Will not open jaw."
21 Is that the same thing? Has he got an obstruction
22 which is, in fact, that he wouldn't open his jaw, or
23 were they two separate things?
24 A. What happens is that, to assess upper airway
25 obstruction, the first sign of it is obviously you can
39
1 hear sounds, a bit like snoring. So there are signs of
2 upper airway obstruction. I felt in this patient there
3 were signs of upper airway obstruction. The next thing
4 I did was try to open his airway -- open his mouth to
5 put what we call a Guedel airway to try to lift out the
6 tongue, because quite often it is the tongue that falls
7 down and obstructs the upper airway.
8 Q. The oxygenation of 75 --
9 A. Uh-huh.
10 Q. -- can you tell us the significance of that?
11 A. Well, I mean, anyone with oxygen saturation of 75%,
12 that's very low. Normal person's oxygen saturation
13 is -- well, it is 95% to 100%.
14 For example, patients with COPDI, people who have
15 chronic lung disease and are very used to a low level of
16 oxygen, you know, about 88%, 89%, 90%. 75%, if anybody
17 has oxygen of 75%, they would be very, very -- well,
18 they wouldn't be conscious, would they? So something is
19 very wrong there.
20 Q. Is that associated with airway obstruction?
21 A. I believe so, yes.
22 Q. By "bagged", I think you explain in your witness
23 statement at a later point that this is a bag over to
24 introduce oxygen?
25 A. Yes. We put a mask over the face and then this mask is

1 attached to a bag which is filled with an oxygen
2 reservoir. Then we squeeze the bag to blow the oxygen
3 into the lungs to try to bypass the airway.

(b) Pathology

(i) Dr Herron (neuropathologist)

Inquiry Statement (80441)

Para 31
If the patient has lost a lot of blood and his heart has stopped or his airway has been obstructed, or 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.

Para 32
I excluded hypoxic/ischaemic injury as a cause of the unconsciousness as it would have had to have been there from the time of the 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... There was no evidence of an 11day reaction to suggest that there was a hypoxic/ ischaemic brain injury that caused his initial unconsciousness.

Para 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 caused the initial unconsciousness. I suspect that at the time (of writing his report) I was trying to emphasise that major pathology which was the axonal injury."

Para 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 around the time of death and this did not have an effect earlier on. However Dr Reid gives a statistical statement at page 72533 that hypoxia contributed to worsening the original 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…To be clear, for the reasons I have already stated, my view is that the hypoxia/ischaemia was not related to Mr Hamill's original condition.

Oral evidence p 62

"We have been told that
24 the oxygen saturation of Mr Hamill when he arrived at
25 Craigavon Hospital was 70%. Can you comment on what
63
1 that tells us?
2 A. I can't comment on that statistic because it is not
3 something I deal with on a day-to-day basis, but I will
4 try to clear this issue to the understanding of
5 everybody.
6 Hypoxia means there is not enough oxygen in your
7 blood. If you were allowed to do experiments where you
8 could put a patient on a ventilator and keep their heart
9 going and remove their oxygen, they wouldn't come to any
10 significant damage. There has to be a loss of blood
11 flow as well. The hypoxia per se does not do long-term
12 damage. That's when I have put "the hypoxic/ischaemic
13 change". Ischaemic change is lack of blood flow. So it
14 is the combination of those things you would typically
15 get after a cardiac arrest.
16 The brain reacts in a stereotypic way to
17 hypoxic/ischaemic damage. It causes a particular change
18 that we see under the microscope to the nerve cells.
19 There was a change to the nerve cells that probably
20 happened in the day or so before his death that could
21 have a number of causes, but what we needed to address
22 was the cause of this man, Mr Hamill's, initial
23 presentation.
24 The pattern of injury to the brain showed that there
25 was no hypoxic/ischaemic damage that would have occurred
64
1 at the time of admission to hospital and that was not
2 the cause of his unconsciousness and that was not the
3 cause of his axonal damage. If it had been, then the
4 brain would have an 11-day reaction to that, and it
5 didn't.
6 Q. That's quite clear, is it?
7 A. I think it is accepted by all the authors, yes."

(ii) Prof Jack Crane

Oral evidence 15-01-09 para. 19

"5 Q. Can you help us with how somebody with 75% saturation at
6 that stage would then not, a week or so later, show the
7 signs of a hypoxic injury?
8 A. Yes. Maybe I have oversimplified it in my statement.
9 There was -- and I think it is reasonable to consider
10 this -- some evidence of some hypoxic damage in the
11 brain. I think Dr Reid felt there was and Dr Herron
12 felt there was. I was not saying there was no evidence

of hypoxic, but if the brain is deprived of oxygen for a significant period of time, and I mean deprived of oxygen so no oxygen is getting to the brain for a significant period of time, then irreversible changes will occur in the brain. If that individual is kept alive for a period of time, for example, on a ventilator, then there are very significant changes, very clear changes that will be seen whenever the brain is examined.

Now those significant, well-developed changes from hypoxia were not present. Obviously there were some changes that were present, but if hypoxia was the main cause of Mr Hamill's brain injury, if we put it -- or

20

brain damage, then I think we would have seen much more evidence of that than was present.

Q. Thank you. Is it possible to conclude from what you have just told us that the 75% saturation was temporary or not?

A. Yes. I mean, I think that's reasonable. He may have been breathing better initially. There may have been some degree of obstruction to his breathing. Clearly, when he got to hospital, I am sure the first thing they would have done was to ensure that his airway was properly secured, that he was given oxygen. So that would be, therefore, to improve the oxygenation of the brain, which is crucial in these cases.

THE CHAIRMAN: If there is an improvement and not too long a period of starvation, then there is no permanent damage?

A. That's correct, yes.

(ii) Dr Lawler

Report (72226)

"Another observation by Dr Reid, which I consider to be equally important in the context of this particular case, is that although there was some hypoxic/ischaemic neuronal damage, it was only mild and not significant. This is really little or no more than I would expect to be found in association with a traumatic head injury of sufficient severity to cause grade II TDAI. It therefore follows that the deceased did not sustain significant hypoxic/ischaemic damage whilst he was lying unconscious in the roadway after the assault and before receiving medical assistance from the paramedics."

(iii) Mr Todd (consultant neurosurgeon)

Report (72617)

Para. 120

"If there had been a significant hypoxic injury to the brain one would expect to see histological evidence of damage to cortical neurons in excess of any damage seen in white matter tracts. The cerebellar Purkinje cells are metabolically very active and one would expect to see evidence of damage to the Purkinje cells. In Mr Hamill's case there was no evidence of damage to these metabolically active structures."

From the observations made by those at the scene and at the Hospital it appears that Robert Hamill was having some difficulty with his breathing. This tends to be supported by the measurement of his oxygen saturation level on admission to CAH of 75% when the normal range should be 95%-100%. Dr Boon Low says that he was concerned that there was an obstruction to his airway and that the most likely reason for this was Robert Hamill's tongue falling back. He also describes him as having clenched teeth so he could not introduce a plastic airway to maintain his airway. Clenched teeth are also described by Glenn Stewart the paramedic. He says that he tried to introduce an airway. He does not say whether he was successful in doing so although it is probable that he wasn't given that Dr Low had to attempt the same procedure. It seems that Robert Hamill was administered oxygen by way of face-mask in the ambulance and by bag in A&E. It can be inferred that it is likely that Robert Hamill whilst on the ground had some problem with his airway before receiving oxygen in the ambulance and in hospital. However, from the pathology it appears that this period of obstruction of his airway was insufficient to cause any injury to the brain and therefore could not have been a contributory factor to Robert Hamill's brain injury as the result of the assault. If anything the pathology tends to suggest that the initial treatment received by Robert Hamill was successful in preventing hypoxic brain injury.
In the family's submission hypoxia does not fall to be considered as an intervening cause and no issue of causation therefore arises in relation to it.

MEDICAL TREATMENT

11. The next factor to be considered as a possible intervening cause is the treatment Robert Hamill received in Hospital from the time of his admission until his death on the 8th May 1997. The focus here is on the administration of Chlorpromazine, a neuroleptic drug, used in the treatment of various psychiatric illnesses including schizophrenia. The literature referred to in the Inquiry bundle tells us that neuroleptic drugs can cause a potentially fatal adverse reaction in a small minority of those to whom it is administered. This is called Neuroleptic Malignant Syndrome (NMS)

12. As all the authors of the pathology reports prepared in the aftermath of Robert Hamill's death and for the Inquiry are agreed that the primary injury as the result of the assault is Diffuse Axonal Injury the question is whether there is sufficient evidence to support a finding that NMS was present and materially contributed to the cause of death. If that is established then one can consider whether the medical treatment viz the administration of chlorpromazine, was an intervening act in the sense that it was sufficient to break the chain of causation.

13. In our submission, having made a finding that the primary injury was DAI, the panel should first weigh the scientific evidence and consider what are said to point toward NMS and what the various medical experts have to say on the subject.

14. It is accepted that Robert Hamill was administered chlorpromazine. (The appropriateness or otherwise of this can be examined later).

15. Prof Crane gives his findings on the issue of NMS in his Inquiry Statement at para 27 et seq:

"I understand there was some surprise amongst the clinicians at the suddenness in decline of 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 discussed 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 that he had a head injury, he had a spiked temperature and he had been given chlorpromazine. At the time of autopsy I was not aware of the results of 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 kidneys 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."

16. Prof Crane says that he was alerted to the possibility of NMS by clinical features which had been present prior to death but that none of the pathology he would expect to find was present and therefore concluded that it was not a factor in the death.

17. This was also the gist of his evidence on the issue of NMS at the trial of R v Hobson where he had this to say:

Q You then said that you had obviously read medical notes and records in relation to it, and on the day of his death he received an injection of a drug to calm his irritation there were questions as to whether that may have contributed or caused the death?

A. The drug that he was given is Chlorpromazine, and he was given Haloperidol . There is a condition associated with both these drugs. It is a rare condition. It is an unusual allergic reaction to, these drugs which is occasionally seen in some individuals known as the malignant neuroleptic syndrome. It is characterised by a high temperature and various changes in both the brain, the liver and the kidney. Mr Hamill did have a raised temperature, and I think that the doctors, looking after him, were concerned that-there was the possibility that this could have been a factor in the death. Whilst the condition of malignant neuroleptic syndrome is not associated with specific changes at autopsy there are some changes that occurred and are recognized both in the brain, and the liver, where you can see acute degeneration, and in the kidneys, where you can see the breakdown of red blood cells. We were keen to ascertain whether that was the case or not. There were no findings either in the brain, the liver or kidneys to indicate that the malignant neuroleptic syndrome associated with the drugs administered contributed to his death."

18. Dr Herron agrees with Prof Crane on this issue, in his Inquiry statement he said at Para 37:

"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 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 it was appropriate to address it as Mr Hamill had been given neuroleptic drugs that may cause the syndrome; he also had a raised temperature and he was sweating excessively. Professor Crane asked for my thoughts on NMS and I wrote back explaining the 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 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 in 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 to say that it could not. I suspect that it could cause similar changes to hypoxic/ischaemic damage bit 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 caused his unconsciousness.

42
Even if I had thought NMS was the diagnosis, it would not explain why Nr 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 death that recorded a creatinine kinase level of about 924. This level does not support a diagnosis of NMS because with NMS, the creatinine 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 creatinine 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 creatinine kinase, purely due to the muscle damage."

19. This view was reiterated by him in his oral evidence and he was not challenged on the point.

Para 57

"12 Can I ask you about Neuroleptic Malignant Syndrome?
13 Would it be fair to summarise what you have said in your

statement that you don't rule anything out definitively in terms of NMS, but you think it is highly unlikely here to be a mechanism?

A. Neuroleptic Malignant Syndrome is a rare condition that has only been recognised to any great extent in the last 20 or 30 years. It is a condition that occurs as an idiosyncratic and unexpected reaction to certain types of medication.

Now, in 1997, there were very few cases reported in the literature as to the pathology of this. Since 1997, there have been loads of papers written about it. It is a condition that is manifested by the patient developing

58

a very high temperature and a thing called autonomic instability, where your heart rate will change and you may sweat a lot. Those were two of the features present in Mr Hamill that were part of that syndrome. So we considered whether or not we thought Neuroleptic Malignant Syndrome was present and Professor Crane and I debated this at length.

I think to put it into context -- and it has possibly been suggested in the subsequent reports that maybe we did not think about it -- there was another patient in the Royal in the same year, and I don't want to give a name for confidentiality reasons, whom I did diagnose Neuroleptic Malignant Syndrome in. So it is something I am very aware of.

I treated a patient previous to this case, as a junior doctor, who developed Neuroleptic Malignant Syndrome and died. I had written a series -- and presented a series of cases in a paper to the British Neuropathological Society describing the pathology of Neuroleptic Malignant Syndrome. At that stage, that was the largest series I think in world literature. So we were very aware of this diagnosis and we considered it.

The reasons that I didn't think this syndrome was present -- the main reason was I felt that the brain pathology in itself was to explain everything that had

59

happened.

There is no controversy, I think, between all the expert witnesses that this patient had a traumatic brain injury. Everything is accepted. The grading of it is debated by one of the authors, and two of them suggest the possibility of Neuroleptic Malignant Syndrome as being what has caused his death at the end.

I felt that the -- medics always like to look for one diagnosis rather than two. I felt there was enough pathology in the brain to explain everything that happened to Mr Hamill.

12 The other reasons why I didn't think there was
13 enough to make the diagnosis of Neuroleptic Malignant
14 Syndrome was he lacked a lot of features I had seen in
15 my case series.
16 One of the particular features of Neuroleptic
17 Malignant Syndrome clinically is an almost inability to
18 bend the patient's arms and legs. You know what the
19 signs of meningitis are besides the rash; you get
20 a stiffening of the arms. The patient I had looked
21 after, as a junior doctor, was so stiff you could not
22 actually -- they call it lead pipe rigidity. I am not
23 aware that that was present in Mr Hamill's case. The
24 temperature can be explained by damage to the
25 hypothalamus.
60
1 Dr Lawler suggests we did not know about the
2 creatinine kinase result until after the post mortem.
3 That would not have swayed me either way. Creatinine
4 kinase is an enzyme that is present in muscle.
5 In the cases I had written up of NMS, the enzyme
6 level in the blood was in the order of 50,000 to 60,000.
7 In the case of Mr Hamill, it was about 900, 970,
8 something like that.
9 There are numerous papers that describe the causes
10 of creatinine kinase and one of those is assault or
11 injections in the hospital. So everything could be
12 explained by his brain injury and his management in the
13 hospital environment to explain all the symptoms that
14 others think may be Neuroleptic Malignant Syndrome.
15 But this is a rare disease. There may be a spectrum
16 we don't know about. For that reason, I don't like to
17 absolutely exclude it being part of the diagnosis.

20. Professor Crane and Dr Lawler both say that NMS was specifically considered as a possible cause of death. Dr Lawler in particular claims experience of this syndrome and was not challenged about this when giving his medical evidence. He discusses the findings of Dr Reid and Dr Lawler both of whom focus on NMS because some of the clinical symptoms were present prior to death. However, and crucially in the family's opinion none of the pathology that both Dr Herron and Professor Crane would expect to find in a case of NMS was present.

21. Dr Reid qualifies her report (72526 at72533) by saying:

"The neuropathology in this case has been hampered by no photographs of the brain…The number of slides is also small and if they are representative of the lesions, miss out some of the areas of the brain which are important to take in cases of diffuse axonal injury, for example sections of the posterior part of the corpus callosum and further sections of the upper brain stem"

Indeed Dr Reid's conclusions are set out under the heading:

"FROM THE INFORMATION AND SLIDES AVAILABLE"

She comes to the view that:

"b. The lesions seen histologically showing a macrophage response with little in the way of ongoing damage are not, in my opinion, enough to cause his sudden death. The probability of his death through Neuroleptic Malignant Syndrome is one which I would agree with."

22. As Dr Reid was not called to give evidence the panel simply do not know whether her view might have been altered by her having seen further evidence of DAI throughout the brain.

23. Dr Herron, who had the benefit of examining the brain itself says the following in his Inquiry statement having noted haemorrhaging in the brain stem

Para 24
"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 there was severe damage in this region and I would still say that this was a case of severe traumatic diffuse axonal injury."

24. As the pathologists found no evidence of NMS post mortem and Dr Lawler in his report (72226 at 72243) identifies only some of the clinical features of NMS which, in the view of Professor Crane and Dr Herron can be explained by the brain injury.

25. It is the family's submission that there is simply insufficient scientific evidence available for there to be finding that NMS was a factor in Robert Hamill's death. It is clear that this was a matter that was given careful consideration as a cause of death and expressly ruled out by the pathologists at the time both of whom had experience of the syndrome in particular Dr Herron who had made a study of it. If the panel accept, as the family say they should, that the issue of his treatment and in particular the administering of neuroleptic drugs do not play any part, on the evidence, in Robert Hamill's death then it does not fall to be considered as an intervening act and so no issue as to causation arises.

CAUSATION

26. In the alternative if the panel do find that the administering of the neuroleptic medication was an intervening act the family submit that it is insufficient to break the chain of causation for the following reasons of legal principle.

27. There can be no dispute that there is a factual connection between the assault on Robert Hamill and his death. The assault was the sine qua non of his

admission and subsequent treatment in hospital. The question then arises whether the assault was the legal cause of death or whether the NMS was a novus actus interveniens sufficient to break the chain of causation.

28. The legal cause of death must be an operative and substantial cause but it need not be direct. In McKechnie(1992) 94 Cr App Rep 51, head injuries inflicted by D which prevented doctors operating on a duodenal ulcer which burst, killing V, were held to be a legal cause of death. Each case will be a matter of fact and degree as to whether the event in question is sufficiently proximate to be the legal cause.

29. That case involved the prevention of medical treatment for an existing condition. In Robert Hamill's case we are dealing with his admission to hospital for treatment as a result of an assault. One aspect of which may have caused his death as a result of his sensitivity to a drug. This could be argued to fall within the concept of the "eggshell skull rule". It is well recognised that D must take his victim as he finds him. This usually relates to particular vulnerability to injury but there seems to be no reason in principle why it should not extend to a situation where V is abnormally pathologically susceptible to a treatment which ordinarily might reasonably be expected to have a therapeutic benefit. The concept has been extended to a situation where life-saving treatment has been refused by V on the grounds of religious belief. Blaue [1975] 1WLR 1411 Jehovah's Witness refusing blood transfusion.

30. While it is obviously foreseeable that the victim of an assault may require medical treatment but it is also foreseeable that such treatment may be negligently performed or injuries misdiagnosed. Failure to provide proper treatment will either aggravate the original injury or simply allow it to take its course.

31. In Jordan (1956) 40 Cr App R 152, D stabbed V and he died. On appeal new evidence showed that the wound was almost healed but that as a result of a mistake he was given antibiotics to which he had, earlier in his treatment, proved to be highly allergic. The CA held that if the jury had been aware of this they would have concluded that the death was due to the medical treatment.

32. The significant distinction between that case and Robert Hamill's is that the treatment in Jordan was negligent. There is no evidence to suggest that the administration of chlorpromazine Robert Hamill was inappropriate. There is no evidence of any prior knowledge on anyone's part of his sensitivity to the drug. Dr Lawler takes the view in his report that it was a perfectly proper treatment in the circumstances. The authors of Blackstone's 18th Ed. assert that the Courts hardly ever categorise incorrect medical treatment as a novus actus interveniens. A fortiori then, cases in which medical treatment is properly administered.

33. In Chesire [1991] 1 WLR 844, V died as a result of complications arising from a tracheotomy necessary as the result of a gunshot wound

inflicted by D. The gunshot wounds had healed at the time of death. D's conviction was upheld by the CA on the basis that the complications were a natural consequence of his act. Beldam LJ having reviewed the authorities said:

" …when the victim of a criminal act is treated for wounds or injuries by a doctor or other medical staff attempting to repair the harm done, it will only be in the most extraordinary and unusual case that such treatment can be said to be so independent of the acts of the defendant that it could be regarded in law as a cause of the victims death to the exclusion of the defendant's acts…"

Chesire has subsequently been followed by the English CA in Mellor [1996] 2 Cr App R 245 and Gowans [2003] EWCA Crim 3935.

34. Applying these criminal law principles the panel even if it finds as a fact that the medical cause of death was NMS, in the family's submission it should not find other than Robert Hamill's death was caused, in law, by the assault.

Submissions by the Police Service of Northern Ireland

No comment

THE SECOND ISSUE: DOES THE MEDICAL EVIDENCE SHED LIGHT ON THE ASSAULT?

10. In addition to the materials set out above, there is the following evidence:

10.1 When Robert Hamill was seen in A&E the notes recorded him as urgent, that he may have been involved in a fracas in Portadown and he may have been hit on the head by a bottle (38972).

10.2 Mr Hamill was unconscious and had an abrasion on left hand side of his head (9186: Statement of David Morrow).

10.3 Dr Boon Low gave evidence at the trial of Marc Hobson. He noticed that when Robert Hamill was admitted, there was no open injury P.8406.

Boon Low

Oral Evidence

10.4 Mr Hamill was noted by Dr Low as having "no open injury" (p.42). Dr Low interpreted an open injury as being "blood and a cut" (p.43).

Professor Crane

Statement

10.5 Para. 25: There were no defence injuries.

Oral Evidence

10.6 External injuries indicate 2 or 3 blows to the head (pp.36/37). One could have been caused by fall (p.37).

10.7 Other injuries to body could have been caused by a number of blows (p.37).

10.8 He rules out NMS as cause of death as its indicators did not show mechanism of death (pp23/24) and believes injury occurred by Robert Hamill's head being struck whilst he was on ground.

Dr Herron
Oral Evidence

10.9 Professor Crane refers to hairline fracture of the skull (72234 & 72240). Dr Herron did not find any specific brain injuries relating to the fracture. There were no bruises on the brain's surface, a common injury where there is an assault with a collapse to the ground.

Dr Lawler

Statement

10.10 (p.13): Clearly there was a bruise on each side of the head. He is sure that the CT scan report and Professor Crane refer to same area of bruising. Presumably, they represent at least one blunt force trauma to each side. He points to references in nursing notes, which refer to an abrasion on the left side. This suggests contact with rough surface.

10.11 (p.14): "small wound approx 1 cm long occiput" to back of head. This suggests contact with a roadway but it could have been caused by kicking or stamping. He is aware Professor Crane does not note any injury in this area but Professor Crane did not shave the back of Robert Hamill's head. The nursing notes "clearly indicate" that the injury was healing and perhaps it had healed by the time of death. This injury could explain some, if not all, the blood on Robert Hamill's jacket. Autopsy photographs show bruising "into the left upper eyelid". He was very surprised there was no reference anywhere to bruising in this region. The only explanation that he can offer for this is it "evolved" over several hours, which suggests delay whilst deep bruising spread.

10.12 (p15): He believed four findings could have resulted from forceful impact on the roadway: (1) Occipital Scalp Injury; (2) Left Orbital roof fracture. It is very well recognised orbital fractures can be caused by occipital impact. He explained the location of the fracture and why Professor Crane said "it didn't relate to the bruising of the scalp"; (3) Left Eyelid Bruising: Very well described eyelid bruising can occur from percolation of blood following

orbital fracture; 4) Bilateral frontal lobe contusions: Although contusions on CT scans and none identified by Dr Reid, Professor Crane stated there were contusions in the white matter. If present, they surely represented a "contrecoup" phenomenon. He is aware that there was no deep scalp bruise in the occipital region but he still considers there was a backwards fall onto the roadway.

10.13 (p17): The marks on Robert Hamill's arms and hands were too weak to suggest defensive wounds. They may have occurred in hospital. The bruising on the right buttock and thigh was not related to the treatment but to blunt force trauma and was a coalescence of several smaller bruises. It is possible there were other injuries sustained that had healed.

Oral Evidence

10.14 It appeared that Robert Hamill's head was moving when he was on the ground. This suggests that there was an assault on the head (p.6).

10.15 It impossible to say how many blows there might have been. (p.24).

10.16 Mr Hamill could have been struck by a bottle (p.35).

10.17 He believes that a fall onto the roadway is the most likely explanation for injuries, but only barely (not DAI) (pp.7/8, 23) [p.72240]. It is less likely than if bruising in scalp at back of head had been found (p.8).

<u>Maureen Hagan</u>

Statement

10.18 Para. 29: 38665 under 'appearance of skin' says "bloody ++head + face". That is not her writing and she did not know who wrote it. She cannot explain why Maureen Millar would have written it, as she does not believe that Maureen Millar was involved in Robert Hamill's care.

10.19 Para 30: Symbol ++ indicates there was quite a lot of blood but she did not record anywhere in her notes that Robert Hamill had any bleeding. Did not write it on 38972, 38973 or 38666.

Oral Evidence

10.20 Robert Hamill's face and head was not covered in blood at any stage. He had a graze on his head (p.52) When the document was filled out there would have been no blood as he would have been cleaned before intubation and X-Ray.

10.21 (p.52) She wrote "Large graze left side of head", and believes it was about ¾ inches in diameter (p.56)

<u>Maureen Millar</u>

Statement

10.22 Para. 13: She completed some entries on 38665/6: Appearance of skin & "bloody ++ head + face".

Oral Evidence

10.23 There was much bleeding around his head and face "Bloody ++head+face" (p.68). Blood can flow from wound on side of head onto face (p.75).

10.24 As to the entries, "?involved in fracas in Portadown. ?hit on the head by a bottle", she did not recall where this information came from, but question marks denote that it was not a confirmed fact. As to the entry "wound to back of head", she does not know who observed that but she does not recall examining the back of Robert Hamill's head and she cannot recall anything about such a wound. It could have been a visible observation. Any lacerations or injury to the scalp will bleed profusely. Sometimes it is possible to think that a wound is somewhere it is not; due to way that blood travels. Robert Hamill was lying flat so a laceration to side of head would look like it had come from the back.

Dr Gormley

Statement

10.25 He can recall blood on Robert Hamill's face but not whether he was actually bleeding or how much blood there was (80328).

Dr Lavery

Statement

10.26 Robert Hamill had very obvious bruising in two separate areas of his head outside the skull (80635).

Glen Stewart

Oral Evidence

10.27 He saw a haematoma on Robert Hamill's face. He did not see open wounds and there was no bleeding on the face or body but he was not 100% sure, whether there was blood on the back of Robert Hamill's head (p.13).

10.28 He thinks he would have noticed blood if there was any present. He travelled in the back of the ambulance. There were 3 casualties and 2 girls (p14).

David Morrow

Oral Evidence

10.29 There were bruises and abrasions around his head (p.11).

10.30 He had a good pulse, no great blood loss, no wound requiring dressing, had good colour, nothing looked over-serious (p23/4)

Submissions by British Irish Rights Watch and Committee on the Administration of Justice

Professor Vanezis is of the opinion that Robert Hamill suffered multiple blows to the head, leading to diffuse axonal injury, from which he did not survive

Submissions by the Police Service of Northern Ireland

See section 11 below

Submissions by Richard Monteith Solicitors (Civilian Witnesses)

We agree with this synopsis and note 10.10 - contact with rough surface may indicate a fall which may support the view at 8.47. We agree that it appears that one cannot say how many blows there may have been

Comment

11. The Panel may wish to consider whether the injuries, apart from the DAI, show that Robert Hamill was the subject of significant blows, probably while he was on the ground. However, the injuries do not suggest that the beating was prolonged. The Panel may need to determine whether the DAI must have been the result of severe acceleration/deceleration, which appears to be most consistent with kicking. The Panel may wish to determine whether the injuries show that there were many kicks.

Submissions by Edwards & Co Solicitors (Serving and Retired Police Officers)

Professor Crane agreed at p 34/35 that it could have been one or two severe kicks to the head that caused the injuries he found on the head. He also agreed that one severe kick could have caused the axonal injury. He stated that "one can't say with any certainty that there were more than two or three kicks to the head". There were also a number of minor injuries which could have been caused by a number of minor blows.

The lack of other injuries to the head and the relatively minor nature of the injuries to the body are not suggestive of a prolonged attack.

The totality of the medical evidence strongly suggests that the attack was over in seconds. This corresponds with the evidence of Maureen McCoy?

Submissions by John P Hagan Solicitors (Robert and Eleanor Atkinson)

We refer to previous comments regarding the evidence relating to the severity of the assault. The evidence suggests that the beating was not prolonged.

Submissions by the Police Service of Northern Ireland

The medical evidence suggests relatively few external injuries. There were some external injuries to the area of the head, consistent with two or three blows/kicks one of which could have been occasioned by a fall (per Professor Crane at page 36/37). Professor Crane accepted that there was no evidence of sustained blows to the head. The life threatening injuries could have been sustained in seconds (per Professor Crane at page 36).

There were no open wounds such as to lead to cuts and blood detected by the doctor at accident and emergency (per Dr. Low p. 43). However, Dr. Lawler has referred to a small (1cm) wound to the occiput (p.14).

There were some soft tissue injuries to the body, probably caused by a number of blows (per Professor Crane at p. 37).

Most significantly there was the DAI which was severe (per Professor Crane at p.12/13). The evidence establishes that it is the severity of the assault rather than the duration of the assault which is the significant issue (per Professor Crane at p.36). Dr. Lawler has opined that DAI was most likely caused by a fall combined with a kick or kicks to the head (per Dr. Lawler at page 25). The literature refers to examples of victims of DAI having suffered kicks and stamps to the head (per. Dr. Todd).

The PSNI agree with the comment (above) that the injuries do not suggest that the assault was prolonged. Rather it is submitted that the attack in all likelihood was of short duration with a relatively small number of powerful blows being inflicted.

This is persuasively established by reference to the relatively small number of external injuries. It may be impossible to be absolutely precise about how many blows/kicks were struck (per Dr. Lawler at p. 24) but Professor Crane's evidence appears to be a sound estimate.

The medical evidence that the attack on Mr. Hamill may have concluded in a matter of seconds is also supported by the evidence from those who were closest to Mr. Hamill: see for example per E in her evidence to the Marc Hobson trial at 8276.

Submissions by Richard Monteith Solicitors (Civilian Witnesses)

We agree that the evidence as a whole supports the view that the attack was over quickly