Dr. William Lawler, M.D., F.R.C.Path.

[redacted]

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Re: Robert HAMILL (deceased)

I have been asked, by Ms. Patricia Fitzmaurice, Deputy Solicitor to the Robert Hamill Inquiry, to provide an expert pathology report to the Inquiry, to address seven specific questions, and to indicate any other factors which I consider to be significant into the cause of Robert Hamill's death.

DOCUMENTS

I have read all the documents provided to me by Ms. Fitzmaurice:-

- The deceased's medical records
  - Craigavon Area Hospital notes
  - Royal Victoria Hospital, Belfast, notes
  - Neurosurgical Unit notes
  - Coroner's investigation file
- Professor Crane's autopsy report
- The Cory Collusion Inquiry Report
- Transcripts of the evidence given at trial by Professor Crane.

I have examined the copies of the 18 photographs taken during the post mortem examination (reference number H1278/97) and copies of the non-neuropathological microscope slides prepared for Professor Crane (reference 48933).

BACKGROUND

Before offering my own opinions and attempting to address the specific questions which I consider to be within my field of expertise, it would, I think, be helpful if I were to try to summarise, briefly, what I consider to be all the relevant points which arise from the documentation which I have read, and I would do so under three main headings.

1) THE INCIDENT

I realise that the remit of the Robert Hamill Inquiry is to inquire into the circumstances surrounding the incident which took place shortly after 01.20 or so on April 27th, 1997, and I have absolutely no intention of wishing, in any way, to pre-empt the conclusions of this process.

However, from my reading of the Cory Collusion Inquiry Report, I believe that there are three aspects which are relevant to me and to my pathological considerations.

It is, I think, important for me to emphasise that I have read none of the statements made by any of the individuals involved in the incident. My comments below are, therefore, derived entirely from the Cory Report: the quotations and the page numbers to which I refer relate to that Report.

The three aspects which I consider to be relevant, and some hopefully helpful comments, are as follows:-

a) ASSAULT

F _____ "saw Robert lying on the ground and 20 or 30 people were kicking at him in the head and shouting 'die you bastard'" (page 14). She also "testified that .... Robert and D [were] struck to the ground very shortly after they arrived at the junction" (page 40).

Mr. H "said that Robert was knocked to the ground and was being kicked while the crowd shouted 'kill the Fenian bastard'" (page 15).

Witness A "observed a number of individuals kicking a man who had been lying in the centre of the road. She said they were kicking this man about the head and body and jumping on him" (page 33). In the light of paragraph 2.100 on page 34, this man must have been Robert Hamill.

Witness B "saw Mr. O kick and punch the person lying on the ground. He said he was kicking him in the chest area. It can be inferred from the location of the victim described by this witness that the man on the ground was Robert Hamill. Witness B also said that he saw Mr. S kick Robert Hamill in the face. He said that during these assaults the injured man did not move" (page 34).

COMMENTS. These witnesses suggest that the deceased was knocked to the ground, although it is not clear whether his head struck the roadway or not; once on the ground, he was repeatedly kicked and, possibly, punched. It is, I think, worth emphasising that the deceased appears to have sustained his injuries very shortly after he arrived at the scene.

b) UNCONSCIOUSNESS

E refers to him "unconscious and lying across the road" (page 13), and the clear implication is that he remained unconscious until the ambulance arrived.

Ambulance Driver A "noticed a male lying on the right hand side of the road near Eastwoods and this person he later identified as Robert Hamill" (page 16). He "did not remember noting that Robert Hamill had trouble breathing. His records did indicate that Robert Hamill had a strong pulse and appeared to be breathing normally although he was unconscious. He noticed an abrasion on the side of the head but apart from that there were no other signs of injury" (page 27).

Officer A said that the deceased "was lying on his back and there was liquid around his head which he subsequently came to believe was alcohol. Robert Hamill's breathing was laboured and he was making a rasping sound" (page 19-20).

Constable E referred to "two male persons who were lying motionless in the road" (page 26).

Officer G saw the deceased "on the ground and noted that Robert Hamill was having difficulty breathing. He moved glass away from his head" (page 37).

COMMENTS. Whatever the exact context of the assault may have been, the deceased appears to have been rendered unconscious during it and to have remained unconscious until the ambulance arrived.

c) DELAY

The delays in this case are clearly fundamental to the Inquiry, and I find it impossible, from reading the Cory Report, to ascertain any real idea of the time the deceased was lying, unconscious, in the roadway before he received any sort of medical or even first aid assistance. I do, however, get the impression that a possible or even probable delay of four or five minutes would be neither unfair nor unreasonable.

2) HOSPITAL COURSE

Following the incident, the deceased was taken to Craigavon Area Hospital, where his condition was assessed and stabilised. Within 4.5 hours, he had been transferred to the Royal Victoria Hospital, Belfast.

I would summarise what I consider to be the relevant findings from the clinical notes under nine separate headings:-

a) ADMISSION TO CRAIGAVON

The deceased was seen by the triage nurse (Staff Nurse Maureen HAGAN) at 02.05 on 27.04.97. She noted that he was "unconscious" and "breathing noisily". She also documented "wound to back of head".

The "Nursing Care Plan" (at page 116 of the RVH notes) refers to a "laceration" at the "left side head"; no further details are given. There is also reference to a "large graze left side of head".

The deceased was seen by the Casualty Officer (presumably Dr. Boon LOW) at, I think, 02.15. He was described as "unresponsive", with a Glasgow Coma Scale (GCS) of 5/15. The Casualty Officer noted "upper airway obstruction. Will not open jaw to introduce ... airway"; the oxygen saturation was 75%. He also wrote "no open injury".

The deceased was intubated by an anaesthetist, and a urinary catheter was inserted. One of the doctors noted "ecchymosis left temple" (RVH notes, page 120).

I understand that a blood specimen taken shortly after arrival at hospital was analysed for alcohol and that a reading of 221 mg per 100 ml was obtained.

It is, I think, important to point out that no other injuries were mentioned in the Craigavon Area Hospital notes available to me.

b) TRANSFER TO ROYAL VICTORIA HOSPITAL

I believe that the deceased was transferred to the ITU of the RVH for a CT scan (the machine in Craigavon was not working) and for further management.

He arrived on the ITU at approximately 06.30. The only reference to any injury made by the admitting doctor was "Limbs – no obvious sign of injury" (page 51).

One of the Nursing Notes (page 84) refers to a "large abrasion left temple" and a "small wound approximately 1 cm long on occiput"; another entry (page 103) refers to a "large grazed area on left hand side of head".

c) REST OF 27.04.97

The CT brain scan (report at page 150) showed "soft tissue swellings in the scalp over the left temporo-parietal region and also over the right temporo-occipital region"; within the cranium, there was what was thought to be a mild degree of frontal lobe atrophy, but no injuries were seen. X-rays of the cervical spine were normal.

By 10.40, sedation and relaxants had been stopped, and the intention was to allow the deceased to wake up (page 54).

By noon, he had been extubated (page 54).

By 18.00, he was making some purposeful movements, but he was very agitated (page 54).

A Nursing Note (page 105: time unclear) refers to "left parietal/temporal area red and grazed. Redness also on temporal region right side. Small wound approx 1 cm long occiput".

d) 28.04.97

The deceased was rather restless, moving all four limbs and trying to get out of bed. He was not obeying commands. Sedation was started (page 55).

e) 29.04.97

The deceased was less agitated, and so sedation was stopped. He was apyrexial (page 55).

At approximately 12.15, he was transferred from the ITU to Ward 39 under the care of the neurosurgeons (page 56). On admission, the Nursing Notes refer to "dry grazed area left temple. Small sore on occiput", and his GCS was reported as being "4-7" (page 7). They also say "severe weakness in arms, mild weakness in legs", and there is reference to agitation and restlessness. The medical notes refer to his being very agitated and being on sedation (page 56); the drug charts show that he was prescribed chlorpromazine (pages 47 & 49).

Nasogastric feeding was started (page 57).

f) 30.04.97

A further brain CT scan was performed; no significant changes were seen from the previous scan (page 57: report page 152).

g) 01.05.97

The medical notes refer again to distress, agitation and the need for increasing sedation; decerebrate movements are also noted. There was some weakness of both arms and both legs (pages 57-59).

h) 02.05.97 to 06.05.97

The only relevant entry in the medical notes during this period is a very brief assessment by a Consultant in Rehabilitation Medicine on 05.05.97 (page 60).

The Nursing Notes imply some fluctuations in the deceased's general condition, but his overall status showed no significant changes. The GCS remained at about 5-6 (pages 22-29), and variable weakness of arms and legs was documented. Sweating was documented on 04.05.97, and on 06.05.97, it was noted that he "continues to sweat profusely".

During this period, the deceased's body temperature also fluctuated, but it was invariably either normal or only slightly raised. He was receiving chlorpromazine during this time, although I find interpretation of the drug charts (pages 44-49) very difficult.

i) 07.05.97

Under this date, the medical notes say "Pyrexia 40°C. Blood culture taken. Chest X-ray ordered" (page 60).

These events are not supported by the Nursing Notes for 07.05.97, but all are described on 08.05.97 (pages 29 & 40), and I therefore strongly suspect that the incorrect date was used in the medical notes.

The Nursing Notes do refer to pyrexia (38.4°C at 07.30) and to "sweating profusely", and overnight (07-08.05.97), a catheter specimen of urine was obtained, - presumably for microbiological culture (page 41). I note that his urine output was falling, and that the ward testing of the urine showed marked proteinuria and a "moderate amount of blood".

j) 08.05.97

According to the Nursing Notes, the deceased's temperature continued to rise (it was 38.5°C at midnight; 38.6°C at 06.00; 39.5°C at 10.30; and 39.9°C at midday). Overnight, he was "sweating profusely", with hypertension (160/100 and 190/105), tachycardia (150 and 160 b.p.m.), and tachypnoea (42 per min). Blood cultures were taken shortly after midday, and he was taken for a chest X-ray at approximately 14.00 (pages 41-42).

By 14.30, his temperature was above 40°C (at page 61, a Dr. Patel refers to 42°C), and he had become hypotensive (B.P. 60/?). His condition seems then to have deteriorated rapidly into cardio-respiratory arrest. All resuscitation attempts were unsuccessful; they were abandoned at 16.10, although death was not formally certified until 17.03 (page 60-62).

Several relevant investigations were performed on 08.05.97; these results are as follows:-

- If I read the photocopies correctly, the blood cultures showed no growth after seven days incubation (pages 146A & B, and 147A & B);
- The catheter specimen of urine grew a coagulase negative staphylococcus (NSU notes pages 1 and 1A);
- The chest X-ray was reported as "the cardiac contour and pulmonary vascularity are normal. No significant lung lesion is present" (NSU notes, page 2);
- The full blood count showed some haemoconcentration, with a mild leucocytosis (NSU notes, page 3), although the white cell count was normal in a later specimen (NSU notes, page 6);
- The electrolytes also showed some haemoconcentration, with mild elevations in sodium, potassium, urea and creatinine concentrations, although the last three could well reflect some acute renal function impairment (NSU notes, page 7);
- The serum creatinine kinase enzyme (CK) concentration was significantly elevated at 924 U/L, the normal adult range being 30-140 U/L; the CK MB (cardiac) fraction was normal (NSU notes, pages 5 and 8);
- The serum aspartate aminotransferase enzyme (AST) concentration was elevated at 154 U/L, the normal adult range being 10-40 U/L; the serum total bilirubin was at the upper limit of the normal range (NSU notes, page 7).

3) AUTOPSY FINDINGS

The autopsy was carried out by Professor Jack Crane on 09.05.97, and he has provided a detailed report. Unfortunately, this report does not include a summary of the ante mortem clinical findings, and it is therefore not possible to see whether he appreciated all the relevant

clinical history when carrying out his examination and when formulating his "Commentary".

It would, I think, be easiest if I were to try to emphasise what I consider to be the relevant findings, and to offer some hopefully helpful comments; I would do so under four headings:-

a) EXTERNAL APPEARANCES

Under the heading "Injuries", Professor Crane lists several according to anatomical locations. Almost all are seen on the photographs provided, although I note that none of the photographs includes a scale.

1) Head.

- The left forehead showed an abrasion which incorporated what appears on the photographs to be an almost horizontally orientated, healing, linear scar. The latter must have pre-dated the incident on 29.04.97, and I would venture to suggest that the former may have occurred more recently than 29.04.97.
- There was bruising into the left upper eyelid. The photographs are not clear, but I wonder whether there is also some vague, fading and diffusing bruising involving both upper and lower eyelids and extending towards the bridge of the nose.
- There was a punctate lesion on the left naso-labial fold. In his autopsy report, Professor Crane refers to this as an abrasion: in evidence at trial (transcript, page 229) it is referred to as "bruising". Photographs 5 and 6 suggest an abrasion rather than bruising, but I strongly suspect that it was fairly recent, - i.e. that it occurred in hospital and after 29.04.97.
- Professor Crane makes no mention of any other scalp injuries, although I note that the deceased had what appears on the photographs to be thick, dark hair, and that there is no evidence to indicate that Professor Crane shaved the scalp.

2) Trunk.

- There was a bruise, "probably associated with a surgical suture" on the front of the right shoulder. This is not clearly seen on any of the photographs. I note that in evidence, Professor Crane suggested that it was the "result of surgical treatment" (transcript, page 229), - and this despite including it as an injury in his autopsy report! Its location and the presence of a suture strongly suggest to me that it was related to therapeutic intravenous access whilst in hospital.

- There was a bruise in the upper left abdominal quadrant. I wonder whether photograph 8 also shows at least three other, smaller bruises on the left side of the anterior abdominal wall; I can see nothing on the right side, despite there being deep bruising seen internally (*v.i.*).

3) Left Upper Limb.

- There were two bruises on the outer upper arm.
- Professor Crane described "a number of" bruises on the back of the forearm, wrist and hand. How many is "a number of"? I think that I can see eight on photograph 10, but I am sure that several (perhaps as many as five) were related to therapeutic intravenous access whilst in hospital and not to trauma.

4) Right Upper Limb.

- There was a bruise on the outer upper arm.
- There was a bruise close to the front of the elbow. This is seen on photograph 12, and I strongly suspect that it was related to therapeutic intravenous access whilst in hospital and not to trauma.
- There was a bruise on the back of the hand. In his report, Professor Crane suggests that it was "possibly related to two needle punctures"; in evidence (transcript, page 231), he seemed to be more certain, - "I think that's related to treatment as opposed to an actual injury". I am sure that it was.

5) Right Lower Limb.

- There was bruising over the outer pelvis and buttock.

b) INTERNAL FINDINGS

Again, Professor Crane has listed his findings according to anatomical locations, and some, but by no means all, are seen on the photographs.

1) Head.

- The deep scalp and temporalis muscles showed two separate areas of bruising, one on each side. These surely correspond with the bruising seen on the CT scan on 29.04.97.
- There was a complex linear skull fracture involving the anterior part of the wing of the left sphenoid bone and the roof of the left orbit, and this was associated with a very small amount of overlying extradural haemorrhage.

- The brain (which, unfortunately, was not photographed) was examined after fixation. Professor Crane told me that this was done in conjunction with a neuropathologist, although this is not stated in his report. 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.
- There is no reference to any facial dissection in Professor Crane's report, but, in evidence at trial (transcript, page 249), he said that there was no facial bone damage.

2) Neck and Chest.
- The only significant abnormality here is the identification of heavy, dark, congested, oedematous lungs.

3) Abdomen.
- There was "a little bruising" into the anterior abdominal wall on the right side. As there is no photograph of this area, I find it difficult to grasp the concept of "a little bruising". Clearly, it was unrelated to any bruising seen externally.
- The spleen was enlarged, and it had a "soft, mushy texture". This enlargement and texture suggest systemic sepsis, but I note that none was identified either clinically or pathologically (discussed elsewhere).
- The bladder mucosa was haemorrhagic. This would have been the result of the indwelling urinary catheter.

c) HISTOLOGY

The brain sections have been examined and reported upon by a neuropathologist (*v.i.*).

Professor Crane sampled four other organs, and he has kindly provided me with duplicates of the sections. I agree with his observations and conclusions, - *viz*:-

1) Left ventricle. Normal.

2) Lungs. There is extensive acute congestion and oedema, with variable and, in places, extensive intra-alveolar haemorrhage. There are also several aggregates of intra-alveolar pigment containing macrophages. There is no associated inflammation.

3) Liver. Normal.

4) Kidneys. No obvious abnormalities are seen, although I wonder whether, in view of the relatively short time interval between death and autopsy, some of the cortical tubule changes are ischaemic rather than autolytic. There are no significant intratubular casts.

d) NEUROPATHOLOGY

I have now received and read Dr. Helen Reid's detailed neuropathology report dated 21.11.05. I, of course, accept her observations, interpretations and opinions.

Specifically, I note her comments in relation to fractured skull, contusions, traumatic diffuse axonal injury (TDAI), and hypoxic/ischaemic changes, and, in particular, her conclusions, -

- The major finding is one of traumatic diffuse axonal injury (TDAI);
- The TDAI is not considered to be "severe", but to equate to Grade II, with "scattered white matter damage";
- There are some hypoxic/ischaemic neuronal changes, but these are mild, and "I therefore consider the hypoxaemic/ischaemic changes not to have been a significant effect in this case, but they did have an effect in worsening his initial brain insult of the order of less than one third".
- The neuropathological findings, *per se*, are insufficient to cause relatively sudden and relatively unexpected death.

e) MICROBIOLOGY

Professor Crane submitted a lung swab and a sample of cerebrospinal fluid for microbiological culture: the former grew staphylococcus and a few coliforms; the latter grew pseudomonas and Enterococcus.

I agree with Professor Crane, when he said in evidence, that these results were "not thought to be significant" (transcript, page 244); I am sure that they represent post mortem contamination, and that they do not reflect ante mortem infection.

COMMENTS AND OPINIONS

Before attempting to address the specific questions asked of me by Ms. Fitzmaurice in her letter dated May 5th, 2005, I would like to address three specific issues which I consider to be particularly important.

1) INJURIES SUSTAINED ON 27.04.97

I must confess that I find determination of the injuries sustained by the deceased on 27.04.97 much more difficult than I had anticipated. This is, I think, partly due to the inadequacy of the clinical and the nursing notes made on 27.04.97, and partly due to the duration of survival of eleven days before death, with the consequent modifications, by healing, of the injuries.

Perhaps I could address each body area in turn, and try to draw some reasonable conclusions:-

a) SIDES OF HEAD

Clearly, there was a bruise on each side. The CT scan report from 27.04.97 refers to the left temporo-parietal region and the right temporo-occipital region: Professor Crane refers to the left temporalis muscle and the right parietal region. Despite these differences, and allowing for healing and the possible effects of gravity, I am sure that these are the same areas of bruising.

These presumably represent at least one blunt force impact to each side, and more than one impact may well have been involved; I would have thought that punches, kicks and/or stamps are likely to have been responsible.

I note references in the Nursing Notes to an abrasion on the left side. This suggests possible contact with a rough surface (e.g. the roadway), and I therefore wonder whether the left side of the deceased's head was in contact with the roadway when the injury to the right side of his head was inflicted.

I also note what I think is the only entry referring to an external I injury on the right side, - "redness also on temporal region right side"; I

suspect that this may refer to bruising rather than to an abrasion, but I can offer no further comments.

b) BACK OF HEAD

At one point, this was described as a "small wound approx 1 cm long occiput".

Whilst I acknowledge that this description is limited, it does suggest to me contact with the roadway, although I accept that it could have been caused by kicking or stamping.

I am aware that no external or internal injury to this region was described by Professor Crane in his autopsy report, although, as noted previously, I do not think that he shaved the scalp. The Nursing Notes from the deceased's stay in hospital clearly indicate that this injury was healing, and perhaps it had healed by the time that he died.

The Cory Report refers to "blood on the collar and back of Robert Hamill's jacket" (page 75), and "the forensic scientist that worked on the case noted that the back and collar of Robert Hamill's jacket was 'extensively blood stained'" (page 29). The presence of an open wound in the area at the back of the deceased's head could, I am sure, explain the source of some, if not all, of the blood responsible for this blood staining.

This injury is considered again under heading d) below.

c) LEFT EYE

The autopsy photographs clearly show bruising into the left upper eyelid, and there may also be some more extensive, local, fading and diffusing bruising.

I am very surprised that there appears to be absolutely no reference, in either the medical or the nursing notes, to any bruising to this region, and the only explanation which I can offer is that it was not present immediately, but that it "evolved" gradually over several hours. If so, then this suggests a delay whilst deep bruising spread to involve (and hence to become apparent at) the surface. This leads me on to my next consideration:-

d) IMPACT WITH ROADWAY?

There is a constellation of four findings in this case, all of which, in my opinion, could have resulted from a forceful impact with the roadway, - as in, for example, a fall over backwards following a punch or a push. Two of these have been considered above, but there are two others; these four are:-

i) Occipital Scalp Injury. There is no doubt that the injury to the back of the head, considered under b) above, could have resulted from an impact with the roadway.

ii) Left Orbital Roof Fracture. It is very well recognised that fractures of the orbital roof, where the bone is very thin, can be caused indirectly by forces transmitted from an occipital impact (i.e. a contre-coup phenomenon). This possibility in this case would certainly explain the location of the fracture and also why, as pointed out in evidence by Professor Crane at trial, "it didn't relate to the bruising of the scalp" which he described (transcript, page 233). Indeed, this explanation would have dealt much more easily with the cross examination about the fracture (transcript, pages 254-258).

iii) Left Eyelid Bruising. It is also very well described that eyelid bruising may not result from a direct blow into the eye socket but that it may follow percolation of blood into the orbit from a fracture of the orbital roof. Under these circumstances, the blood would accumulate over a period of time, - thus, perhaps, explaining the possible delay in the development of this bruising in this case - as discussed under c) above. It would, of course, also explain why the eyelid bruising was confined to the left side.

iv) Bilateral Frontal Lobe Contusions. Although no contusions were seen on either of the CT scans taken during life or macroscopically at autopsy, and although no contusions were identified histologically by Dr. Reid, Professor Crane's report clearly states "examination of the sections from the right and left frontal lobes showed the presence of white matter contusions". If present, these must surely represent a contre-coup phenomenon, and thus provide support for a significant fall backwards.

I am aware that no deep scalp bruising was found in the occipital region, at autopsy, by Professor Crane. This, I realise, does not assist my hypothesis, but I still believe that a fall to the roadway which resulted in the deceased's striking the back of his head is the most likely explanation for this combination of four apparently disparate findings. I do not, of

course, know how far backwards and downwards the scalp was reflected when the deceased's brain was removed at autopsy, and I do not know whether the injury noted (and documented) by the nursing staff was in the lower occipital region.

e) BRAIN

It is Dr. Reid's assessment that the major neuropathological finding was one of traumatic diffuse axonal injury (TDAI), although she adds that, in her opinion, it was not severe.

TDAI represents direct traumatic damage to the brain as a consequence of shearing or twisting forces. These forces are often complex, and involve acceleration, deceleration, rotation and, sometimes, shaking. Consequently, whilst TDAI often results from road traffic incidents, it is well recognised to occur during assaults, particularly when the head is subjected to repeated kicking, punching or stamping.

In this case, therefore, I have no doubts that the deceased's TDAI must have been caused during the assault shortly after 01.20 or so on 27.04.97. Furthermore, I think that the identification of significant, even if not severe, TDAI must reflect the severity of that assault.

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 <u>not</u> sustain significant hypoxic/ischaemic damage to his brain whilst he was lying unconscious in the roadway after the assault and before receiving medical assistance from the paramedics.

f) ABDOMINAL WALL

I think that there are several bruises on the left side externally, and Professor Crane documented one internally on the right side. I can not imagine that these were caused during the deceased's stay in hospital; I therefore think that they represent areas of blunt force impact, and I would suggest that they are likely to have been caused by punching and kicking. Clearly, the amount of force involved must have been sufficient

to produce the bruising, but it was obviously insufficient to cause damage to any of the intraperitoneal contents.

g) ARMS AND HANDS

Although several bruises were documented at autopsy, I find it difficult to try to decide how many of these were related to the incident on 27.04.97, and this is for two reasons. Firstly, I have no doubts that at least some of them have been caused by needle punctures during treatment, but I can not determine how many. Secondly, I agree with the concession made by Professor Crane (at pages 250-251 of the transcript) that some could have been caused in hospital during restraint, - for whatever reason. Consequently, whilst I could not disagree with Professor Crane's autopsy report comment that some of the bruises on the deceased's left forearm and hand "could have been sustained if the arm was struck whilst raised in a defensive gesture", I think that the evidence for this is rather weak.

h) RIGHT BUTTOCK AND THIGH

I can not imagine that this bruising was related to treatment, and I therefore think that it reflects trauma. Its appearance on photograph 14 suggests that it resulted from the partial coalescence of several smaller bruises, and these could have been caused by punching, kicking and/or stamping.

i) OTHER POSSIBLE INJURIES ELSEWHERE

It is, I think, very important to realise that eleven days elapsed between incident and death in this case. In this time period, abrasions and bruises (particularly small ones) would have been subjected to the normal healing processes, and they could well have healed completely (i.e. disappeared). Consequently, it is at least possible that there were other injuries sustained on 27.04.97 which were not apparent at autopsy because they had healed; in other words, absence of evidence is not necessarily evidence of absence!

2) CAUSE OF DEATH

Given all the information available to me in this case, both pathological and clinical, and both positive and negative, I believe that

the immediate cause for the deceased's death was the Neuroleptic Malignant Syndrome (NMS).

This is a rare, and probably the most serious, complication of neuroleptic medication, and it has been reported following treatment with chlorpromazine.

Criteria for making the diagnosis of NMS seem to vary somewhat between papers[1-5], but the major findings seem to include:-

- Fever, particularly over 38.5°C;
- Neurological features – e.g. rigidity, dysphagia, tremor, dystonia, chorea, opisthotonous, seizures, trismus, and fluctuating conscious levels;
- Autonomic instability – e.g. hypertension, hypotension, tachycardia, tachypnoea, and sweating;
- Laboratory abnormalities – e.g. elevated CK levels, leucocytosis, elevated transaminase levels, myoglobinuria, and renal function impairment;
- Absence of other identifiable physical illnesses.

In this particular case, I would point to the following findings documented over the last 24 hours or so of the deceased's life:-

- Marked fever – "over 40°C" and "42°C" were mentioned;
- Autonomic problems – hypertension, hypotension, tachycardia, tachypnoea, and sweating;
- Significantly elevated serum creatinine kinase enzyme (CK) concentration;
- Elevated serum aspartate aminotransferase enzyme (AST) concentration;
- Possible mild/early renal function impairment;
- No other explanations for these findings, - in particular, the blood cultures were negative, the chest X-ray was normal, and no evidence of local or systemic infection was found at autopsy.

Several neurological features were present, but I can not distinguish any which may have specifically resulted from NMS from those directly attributable to the deceased's brain damage.

I am aware that the subject of NMS was raised at trial, and that Professor Crane excluded it on the basis of there being no histological findings in the brain, liver or kidneys to support this diagnosis (transcript,

pages 247-248 and 272-276). I disagree that the lack of "appropriate" structural changes in the brain, liver and kidneys automatically excludes the diagnosis of NMS, particularly when other clinical and laboratory findings are present. I also disagree with Professor Crane when he implies (at page 273 of the transcript) that NMS can not develop in an individual who has been receiving chlorpromazine for a week; such a delay is not uncommon[3], and, furthermore, once started, NMS can develop very quickly[3].

Consequently, if it is accepted that NMS is the immediate cause for this man's death, then it would, in my opinion, explain his fairly rapid deterioration to death; in fact, I can think of no realistic alternative explanation.

I note that there is no reference in the transcript of Professor Crane's evidence to the considerably elevated serum CK concentration in a blood specimen taken on the day of the deceased's death. To be fair on Professor Crane, I am aware that the report of this result was not issued by the biochemistry laboratory until 09.05.97 (NSU notes, page 5), and it is therefore likely that he was not aware of it at the time he performed the autopsy. It would, however, have been available at the time of the trial, but it does not appear to have been mentioned, and I therefore wonder whether Professor Crane (or, indeed, anyone else) knew about it. I believe that it is a very important laboratory finding, as it provides an extremely strong pointer towards NMS.

3) CAUSATION

In my opinion, there is an unequivocal and incontrovertible, direct cause and effect relationship between the assault on 27.04.97 and the deceased's death on 08.05.97.

I believe that the sequence of events can be summarised as follows:-

- Assault, causing significant and permanent traumatic diffuse axonal injury and temporary unconsciousness; this necessitated
- Admission to Hospital, where treatment included
- Administration of Chlorpromazine. Although I am not a clinician, the prescribing of chlorpromazine in this case seems to be perfectly reasonable, given the agitation, the restlessness and the apparent lack of co-operation documented in the notes. This caused

- Development of the Neuroleptic Malignant Syndrome. This is a well recognised, but very rare, complication of treatment with chlorpromazine and other similar drugs. This then caused
- Death.

SPECIFIC QUESTIONS

I have been asked seven specific questions in the letter which I received from Ms. Patricia Fitzmaurice, Deputy Solicitor to the Robert Hamill Inquiry, dated May 5th, 2005.

I believe that I have addressed all the relevant issues above, but a few further comments would, I think, be helpful. The questions asked, together with these comments, are as follows:-

1. What injuries did Robert Hamill receive at the scene of the assault on 27th April 1997?

and

2. How were those injuries caused?

I have tried to discuss each and every one of the deceased's injuries as documented clinically and/or pathologically, and I have also tried to deal with possible causation. In addition, I tried to emphasise the point that, at least in theory, there could have been injuries not documented clinically which had healed completely by the time of the autopsy.

I do not think that I can add anything further here.

3. What first aid did those injuries call for?

This is, of course, outwith the remit of a forensic pathologist like me.

4. What first aid was administered?

From my reading of the documents which were sent to me, it is my understanding that none was administered until the paramedics arrived.

5. Was any injury sustained or exacerbated by any omission or first aid at the scene or treatment in transit to hospital or at the two hospitals?

Given the neuropathological findings and interpretations provided by Dr. Reid, I do not think that the lack of first aid provision at the scene (i.e. after the assault and before the arrival of the paramedics) caused or significantly exacerbated any injury which had already been sustained during the assault itself.

I found nothing in any of the case notes provided to suggest that any treatment given, whether in transit to hospital or at either of the two hospitals, caused, contributed to, or in any way exacerbated the deceased's injuries.

6. What was the cause of death – including the role, if any, of the following factors:

a) Original injuries

It is my opinion that there is an unequivocal and incontrovertible, direct cause and effect relationship between the assault on 27.04.97 and the deceased's death on 08.05.97. My interpretation of the sequence of events which I think applies is documented above.

b) Consequences of the first aid provided or not provided

Again, as stated previously, I do not think that the lack of first aid contributed significantly to the deceased's brain damage, and, hence, to his ultimate death.

c) Consequences of medical treatment provided in transit to hospital

I found nothing to suggest that the medical treatment provided in transit to hospital contributed in any way to the deceased's death.

d) Consequences of the medical care provided at Craigavon Area Hospital and Royal Victoria Hospitals

As noted above, I believe that the chlorpromazine treatment given in the Royal Victoria Hospital was a factor in the deceased's death (albeit an unintentional one) by giving rise to the neuroleptic malignant syndrome.

I found nothing to suggest that any other aspects of the medical care provided at either of these hospitals contributed in any way to the deceased's death.

e) The blood alcohol level of the deceased at the relevant time.

I understand that a blood specimen taken shortly after arrival at Craigavon Area Hospital was analysed for alcohol and that a reading of 221 mg per 100 ml was obtained.

Although I do not know the exact time when this specimen was taken, it would, I think, be reasonable to presume that the deceased's blood alcohol concentration at the time of the incident would probably have been between 230 and 240 mg per 100 ml.

There is considerable variation between individuals in the way in which they react and respond to different blood alcohol concentrations, and this relates largely to the frequency with which they consume large amounts of alcohol (i.e. their tolerance). I would not, therefore, have been as dogmatic as Professor Crane, who wrote "Such a level would leave no doubt that he was moderately intoxicated at the time of the incident", as I do not know the deceased's tolerance to alcohol; consequently, the degree of intoxication could have been anything from mild to marked. Nevertheless, I am sure that such a level would have caused at least some degree of unsteadiness and, therefore, increased the deceased's chances of falling during an assault.

Professor Crane also says "it is well recognised that alcohol intoxication exacerbates the effects of head injuries and may well have played a part in the fatal outcome in this case". This is something which is often quoted, but it is very difficult, if not impossible, to quantitate or even to prove. Given the overall findings in this case and my interpretations of them, I doubt whether alcohol intoxication played a significant role in exacerbating the deceased's head injury, but I know of no way of proving or disproving any possible contribution.

7). Was the investigation and report of Professor Jack Crane into the cause of Robert Hamill's death comprehensive and accurate?

I must begin my answer to this question by emphasising that it is very easy to apply 2005 standards to cases dealt with in 1997, and to be critical as a consequence.

As is, I think, apparent from what I have written above, there are four areas either where I would have done something which Professor Crane did not do, or where I would have drawn different conclusions. I wish to make it clear that these are not criticisms in the strict sense of that word – they are honest differences of opinion – and that it is inappropriate even to attempt to designate "right" and "wrong" in these circumstances. They are:-

a) Autopsy summary of ante mortem clinical findings. I am aware that Professor Crane subscribes to the view that such a summary will, inevitably, include hearsay. Whilst this may be true about descriptions of the initial incident (as provided, for example, by police officers), it does not, in my opinion, apply to clinical notes. As Professor Crane knows, the majority of Home Office Pathologists in England and Wales include such summaries in their statements, and I believe that this was the case in 1997. As noted previously, the absence of such a summary means that it is not possible to see whether Professor Crane appreciated all the relevant clinical history when carrying out his examination and when formulating his "Commentary"; specifically, in this particular case, I do not know whether he realised, firstly, that a significant occipital injury had been documented clinically and, secondly, that the serum creatinine kinase enzyme concentration was considerably elevated in blood taken from the deceased shortly before his death on 08.05.97.

b) Shaving of scalp hair at autopsy. Knowing that the assault on 27.04.97 involved kicking and, possibly, punching to the deceased's head, and knowing that "head injury", in the broadest sense of the term, was bound to be a major factor in this man's death, I am sure that I would, even in 1997, have cut very short, if not actually shaved, most, if not all, of the deceased's scalp, - certainly, I would have done sufficient to satisfy myself completely that I had identified all the healing scalp injuries. I am sure, therefore, that I would have found a healing occipital laceration (particularly knowing that such an injury had been described clinically), and I think that there may well have been other healing injuries also.

c) Impact with roadway. As noted above (at pages 15 and 16), I think that it is at least possible, and, perhaps, probable, that the occipital scalp injury, the left orbital roof fracture, the left eyelid bruising and the bilateral frontal lobe contusions described by Professor Crane could have resulted from a forceful impact with the roadway, - as in, for example, a fall over backwards following a punch or a push.

d) Cause of death. As described on pages 17-19 above, I believe that the immediate cause for the deceased's death was the Neuroleptic Malignant Syndrome (NMS). Indeed, I can think of no realistic alternative explanation for the deceased's fairly rapid deterioration to death.

REFERENCES

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3). Shalev A, and Munitz H. The neuroleptic malignant syndrome: agent and host interaction. Acta Psychiatr. Scand., 1986; **73**: 337-347.

4). Sachdev P, Mason C, and Hadzi-Pavlovic D. Case-control study of neuroleptic malignant syndrome. American Journal of Psychiatry, 1997; **154**: 1156-1158.

5). Chandran GJ, Mikler JR, and Keegan DL. Neuroleptic malignant syndrome: case report and discussion. Canadian Medical Association Journal, 2003; **169**: 439-442.

DECLARATION

The facts as stated in this report are true to the best of my knowledge and belief. I understand that this report and any oral testimony in relation to my findings and opinions may be used in court proceedings arising out of this case. I make it knowing that I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or that I do not believe to be true.

I understand that, as an expert witness, I have an overriding duty to assist the Court with matters within my expertise, and to advise independently of whoever has instructed me. I believe that I have complied with that duty.

PERSONAL BACKGROUND

I obtained my basic medical qualifications (M.B., Ch.B.) from Manchester University in 1971.

After House Officer posts and a rotating clinical pathology post at Manchester Royal Infirmary, I was appointed, in 1973, Temporary Lecturer in the Pathology Department of Manchester University. In 1975, I was promoted to Lecturer.

In 1980, I was awarded M.D. by thesis, and in 1981, I obtained Membership of the Royal College of Pathologists by examination.

Also in 1981, I was appointed Senior Lecturer in Pathology at Manchester University and honorary Consultant Histopathologist at Manchester Royal Infirmary.

In 1984, I was appointed a Home Office Pathologist, primarily to the Greater Manchester area, and, in 1992, I resigned from my university post in order to pursue a full time career as a self-employed forensic pathologist.

Since 1984, I have personally examined over 1,000 victims of homicide, and I regularly provide expert assistance to both the Crown and the Defence in cases such as this.

In 2002, I took semi-retirement from much of my police work, but I continue to provide opinions for both the Crown and the Defence.

WILLIAM LAWLER
Consultant Forensic Pathologist
Home Office Pathologist.
23.11.05.