Thomas Benjamin Harding (A protected party by his Litigation Friend Tina Elizabeth Harding) v Buckinghamshire Healthcare NHS Trust

Case No: B90SB126 (TLQ/16/0744)

High Court of Justice Queen's Bench Division

13 September 2017

2017 WL 04618809

Before : Sir Alistair MacDuff Sitting as a Judge of the High Court

Date: Wednesday, 13th September 2017

Hearing dates: 15, 16, 18, 19, 22, and 25 May 2017

Representation

Susan Rodway QC (instructed by Novum Law) for the Claimant.

Bradley Martin (instructed by Capsticks) for the Defendant.

Approved Judgment

Sir Alistair MacDuff:

1 This is a claim for personal injury and consequential losses, allegedly caused by the negligence of the Defendant. The Claimant, Thomas Harding was born on 13th June 1990. The Defendant is the National Health Service Trust responsible for Stoke Mandeville Hospital.

2 The Claimant arrived at the hospital, by ambulance, on 25th July 2012 at 01.55 a.m. having been assaulted in Castle Street High Wycombe and having sustained a wound over the right side of the head. He was handed over to the Accident and Emergency (A&E) Department at 02.45 a.m. In very broad summary, the Claimant had suffered a skull fracture and, as a consequence, he was developing an extradural haematoma (EDH), which required emergency treatment. He was not sent for CT scanning until after 05.00 a.m. at which point the nature of the injury was first appreciated. As the hospital had no emergency neurosurgical facilities, he was later transferred to the John Radcliffe Hospital in Oxford where he received operative treatment, namely a decompression of the EDH. He has made a partial recovery. He claims that he was negligently treated; his true condition should have been diagnosed much earlier and, if that had been done, he would likely have made a full (or much fuller) recovery.

3 I am not concerned with the issue of quantum of damages. This is the trial of the issue of liability, or more accurately the trial of causation. The Defendant admits breach of duty but denies causation. It is admitted that the Claimant should have been sent for a CT scan shortly after arrival in the A&E Department. It is the Defendant's case that, with immediate and proper treatment, Mr Harding would have made no better recovery. He has suffered no loss by the admitted breach of duty.

4 We have spent a great deal of time in the course of this trial, on so-called "time lines". There are three time lines. First is the actual time line; that is to say the recorded times at which events happened. There are two hypothetical time lines: the Claimant, supported by expert evidence, suggests what a reasonable and proper time line should have been, absent any negligence. It is submitted that this is a timeline which should and could have been achieved. The Defendant puts forward a somewhat different hypothetical timeline. It is contended that the Claimant's timeline is (i) over-optimistic, (ii) would have been unachievable and (iii) would represent an unrealistic counsel of perfection. In the course of the evidence, it became clear that issues in respect of the two hypothetical time lines had narrowed since opening arguments had been filed ahead of the

trial. Now, in very broad terms, the Claimant asserts that the emergency decompression surgery (which actually commenced in the John Radcliffe at about 10.30 a.m.) should have commenced between 5.15 a.m. and 5.30 a.m. The Defendant accepts that the surgery should have taken place much earlier than in fact happened; but submits that, in reality, the operation would probably not have taken place until around 6.30 a.m.

The history (01.55 a.m. to 10.30 a.m.)

5 At 1.55 a.m. the police attended the Claimant and, at 2.02 a.m. the ambulance arrived at the scene. The journey to the hospital was between 2.14 and 2.39 and the Claimant was handed over to the A&E Department at 2.45. The Claimant's score on the Glasgow Coma Scale (GCS) was taken on three occasions before and during the journey and was recorded, on all three occasions, at 12/15 (*F6: 136-7*).

6 Although the Claimant had been admitted at 2.45, he was not formally registered until 3.27 a.m. The first relevant record discloses that by 3.30 a.m. the GCS had reduced to 11/15. It was similarly recorded as 11/15 at 3.55 a.m. *(F7: 2207)*

7 I pause to note that it is common ground that, where a patient presents with a GCS of 12 or less, and with an accompanying head wound, an urgent CT scan is mandated and it is for this reason that the Defendant admits that there was a breach of duty. The Claimant contends that the request for a CT scan should have been made immediately, that is to say by no later than 3.00 a.m. The Defendant contends that it should have been made by 3.30 a.m. I will need to explore this later. For the moment, I need to consider the history.

8 It appears that, in this instance, an assumption may have been made that the Claimant's lack of responses was a result of alcohol ingestion. In any event, nothing of note appears to have happened between 3.55 a.m. and about 4.45 a.m. at which time it was noted that there was no withdrawal to pain; the Claimant was put on intravenous fluids. At 4.50 a.m. he was moved to resuscitation. There was no response to painful stimuli, but pupils were still reactive. GCS was down to 4/15. This represented a very serious deterioration in the Claimant's condition over a period or about one hour. The pupils were becoming more dilated but were still reactive (albeit " *sluggish* ") at 5.00 a.m.

9 At around this time, an on-duty doctor (Dr Lal) was called and a request was made for a CT scan. This showed a right-sided extra dural haematoma with an overlying fracture of the skull and active bleeding. This was diagnosed at approximately 5.30 a.m. A call was made to the John Radcliffe Hospital at around 6.10 a.m. to initiate a possible transfer process. Dr Lal spoke with Mr Andrew Manning, a neurosurgical registrar at the Radcliffe. Mr Manning wrote in the medical records the note to be found at *F8/ 151* . This note is transcribed in his witness statement at *B7/ 109-110* .

10 A summary: Dr Lal had informed Mr Manning that the Claimant had been put in the observation bay. She, Dr Lal, had been called at 5.30 by the nursing staff and noted that at this time there were bilateral fixed dilated pupils; the Claimant was unresponsive with a GCS of 3/15. He had not been intubated. Dr Manning advised (i) immediate intubation and ventilation (ii) the administration of drugs (mannitol and phenytoin) and (iii) that Dr Lal should call back *("stressed within 20 – 30 minutes* ") with information as to any improvement. He thought it highly likely that, by this time, the Claimant was " *coning* " (that is to say suffering a prolapse of the brain) and was unlikely to survive. In these circumstances, emergency decompression (especially after the time loss taken up by the transfer) would be unlikely to be of any benefit. Stoke Mandeville did not phone back as requested and Mr Manning himself made further calls at 6.25, 7.00 and 7.30. On the first two of those three calls, he re-emphasised the need for intubation. (In fact, almost certainly, Dr Lal had seen the Claimant somewhat earlier than 5.30; she wrote up the note at F7/ F2211 at 5.30 a.m. and had requested the scan at 5.01 a.m.)

11 In fact the Claimant was commenced on mannitol at 6.39 and intubated at some time between about 7.05 and 7.20.

12 In the meantime Mr Manning had spoken to the on-call consultant neurosurgeon, Mr Cudlip. When, by about 7.30 it was clear that there had been no reported improvement in condition, and that there had been fixed dilated pupils since at least 5.30, a decision was made not to transfer.

There is a record in the Stoke Mandeville notes at 8.25 that Mr Manning had " *advised that Thomas is already coned and there is no chance of … getting any better by surgical intervention* " and a decision had been made not to transfer. On this evidence it was assumed that death was imminent.

13 The decision not to transfer was later rescinded when, at around 8.30 a.m. the Claimant was observed to be coughing on his endotracheal tube. This was a positive sign. The transfer took place and the Claimant arrived at the John Radcliffe Hospital at 10.25 a.m. The decompression operation commenced immediately at 10.30 a.m.

14 I have been able to skip through the history at some speed because the precise events between Dr Lal's first involvement and 10.30 a.m. (when the operation commenced) are not of any great significance. For the purposes of this judgment, the important matters are these: (i) that the Claimant should have been sent for a CT scan shortly after his admission and at a time when his GCS was still at 11/15 (ii) that scan would have shown a skull fracture and developing extra dural haematoma which would have required surgical decompression; (iii) there would have been no question but that the Claimant should then have been started on drugs, intubated, transferred to the John Radcliffe and the operation performed at a very much earlier time.

15 Thus, I have these questions to answer. (i) What is the correct hypothetical time line? That is to say, at what time would the decompression operation have taken place, absent any negligence? This involves an assessment of what would reasonably have been achieved during the night of 25th July 2012. (ii) If the neurosurgical treatment had been given at that earlier time, would the final outcome have been the same (as the Defendant contends) or would the decompression have resulted in a better outcome?

16 In respect of both those questions, I make the following observation. We can never know how soon the operation may have taken place, nor what the outcome would have been. That, of course, is a consequence of the Defendant's negligence. In those circumstances, I make it clear that I adopt the approach explained by Longmore LJ in *Keefe v Isle of Man Steam Packet Co Ltd. [2010] EWCA Civ 683* . Where a lack of important evidence is a consequence of the Defendants' breach or breaches of duty the court should judge the Claimant's case benevolently and the Defendants' case critically.

What is the correct hypothetical time-line?

17 As set out above, the Claimant did not in fact have the operative treatment until around 10.30 a.m. Quite apart from the Defendant's negligence, there was significant delay caused by the initial decision not to transfer; a decision which was revisited later when the Claimant was seen to cough.

18 As to the time line there were seven matters upon which the Claimant and the Defendant did not agree:

i) Given that the GCS was known to be 12/15 on arrival, should the request for a CT scan have been made at 3.00 a.m. (Claimant's case) or at 3.30 a.m. (Defendant)?

ii) What would have been the likely and proper lapse of time between the CT request and the analysis of the results? 30 minutes (Claimant) or 60 minutes (Defendant)?

iii) How quickly, in any event, would the Claimant have been placed in the CT scanner, given that there were two other patients (AB and JP) who were scanned around that time? Would the Claimant have been prioritised?

iv) Might the identification of the extra dural haematoma have been delayed, given that the radiographer was inexperienced and may not herself have been able to identify the extra dural haematoma?

v) How quickly, following the identification of the extra dural haematoma, would the

Claimant have been ready at the ambulance departure point?

vi) What was the likely journey time?

vii) How long would it have taken at the Radcliffe for the decompression to have been effected?

19 I heard evidence on the first five of these issues from Mr Skinner and Mr Campbell-Hewson, the A&E experts as well as factual evidence from Jane Dickinson the General Manager of Imaging at the Defendant Trust. I also had evidence from Dr Halpin, a consultant neuroradiologist.

20 The A&E experts were referred to the NICE guidelines and the ATLS Manual (Advanced Trauma Life Support). These gave guidance as to procedures and best practice. I do not propose to burden this Judgment with those documents; suffice it to say that I heard the evidence and considered the documents.

21 The GCS had been taken by the ambulance crew and was known to the Defendant at the time of admission. It was the Claimant's case that an urgent CT scan was mandated and the request for a CT scan could and should have been made by 3.00 a.m. immediately after admission. A doctor would have to request the scan; but that need take no more than a short moment. The doctor would confirm the GCS and, without more, would request the scan. It was the Defendant's case that a fuller clinical examination was called for which would take some additional time. I accept the evidence of Mr Campbell- Hewson that a fuller examination was called for. There might have been other serious injury – for example an abdominal injury. It would be proper and normal practice for a full examination to be made before the patient was sent for scans and (if necessary) other investigation. This opinion was supported by the ATLS and NICE guidelines. Moreover, at this stage the GCS was not massively depressed and this would not have presented itself as an immediate emergency. The purpose of the scan would have been to exclude serious injury; and in many cases no serious injury would have been disclosed. The further question is this: what time should be added to the hypothetical time line to take account of this? The Defendant suggests that it would add about 30 minutes. Undoubtedly, in my judgment, there would have been some delay beyond 3.00 a.m.

22 The second issue is informed in part by the third and fourth issues, which are subsidiary to it. I will deal with those sub-issues first. On these issues the Defendant relied upon the evidence of Ms Dickinson. In fact, in the early hours of 29th July 2012 other patients were imaged in the radiology department. Only two are of real relevance. At approximately 3:15 a.m. a patient, referred to as AB, had a CT head scan within the department and was followed by JP, whose scan was at around 3:40 a.m. There was only one radiographer available and the claimant would have to compete with those other patients for scanning priority. Clinical notes for both those patients were available and I had evidence about possible prioritisation from Mrs Dickinson as well as from Dr Halpin.

23 Ms Dickinson also gave relevant evidence upon the fourth issue. As noted above, it was the claimant's case that the haematoma would have been diagnosed at or around the time of the imaging. However, there was no radiologist on the site, and the radiographer, Nicola Hayling, was a relatively inexperienced band 5 radiographer. It was suggested by the Defendant that there would have been no expectation for her to interpret the imaging. Interpretation of images was being outsourced, with the images being sent electronically. Unless a clinician had reviewed the image in real time, or had seen it on the system a short time afterwards, diagnosis would have had to await the report from the remote radiologist. Almost certainly, that would have taken one hour. I note that Ms Hayling (who could have been expected to tell us whether she could have recognised the extra dural haematoma) was not called to give evidence.

24 Thus, it is suggested on behalf of the Defendant that it would be generous to allow (as the Defendant is prepared to do) a full hour between CT scan request and diagnosis of the extra dural haematoma. The NICE guidelines allow a time of one hour and it was highly likely that the diagnosis would not have been made until the radiologist's report was received. When one adds

in the possible delay in gaining access to the scanner and the competing demands of other patients, an hour (submits the Defendant) was as fast as it was reasonably likely to be.

25 We will never know. I am not prepared to hold that there would have been delay as other patients were scanned; also, in all the circumstances, I am prepared to hold that there was every prospect that either the radiographer would have made the diagnosis, and / or that the clinician would have seen the picture in real time or within a couple or so minutes. It follows that I find that there were good prospects that the diagnosis would have been made in less than the hour for which the Defendant contends.

26 I assess the evidence in the following way. The request for a CT scan would have been made between 3.15 and 3.30 a.m. The diagnosis of extra dural haematoma could have been made between 30 minutes and one hour after the request. I have to assess the prospects using a broad brush but adopting the *Keefe* approach. I do not distinguish between these two periods. My finding is that the diagnosis would have been made by 4.00 p.m.

27 Although I heard a great deal of evidence on issues (v) and (vi) it is now realistically conceded by the Defendant that the Claimant would have been ready for transfer one hour thereafter and that the transfer itself would have taken half an hour. I find that the Claimant would have arrived at the Radcliffe Hospital at 5.30 a.m.

28 The final piece of the jigsaw is the time taken between arrival at Oxford and the decompression. Again, I heard much evidence about this. These were still live issues during the trial. However, I note from final submissions that there is effectively agreement that decompression (opening of the bone flap) would have been about 30 minutes after arrival. I hold that decompression would have been complete by 6.00 a.m.

Was the delay causative of the Claimant's injury?

29 The question posed may be put in a number of different ways: If the decompression had been complete (as it should) at 6.00 a.m. would there have been a more favourable outcome? Did the delay cause or make a material contribution to the Claimant's serious injuries?

30 Before answering that question, I should make three observations. The first observation is this. It is a matter of general surprise that the Claimant made a remarkable recovery (albeit that he remains seriously injured). As we have seen, his GCS was 3/15 at 4.50 a.m. and within a relatively short time after that his pupils were not reacting to light. It is common ground that by 4.50 a.m. Mr Harding was "coning". At around 7.30 a.m. Mr Manning, after discussion with Mr Cudlip decided that he had "coned" and that surgery would be of no benefit. Every indication was that his life could not be saved. It is also common ground that for reasons described in paragraph 37 below, the downward spiral suddenly stopped. This is a feature of some importance to which I will need to return.

31 I should also record that it is common ground that, if there had been no negligence, the Claimant would have been intubated and ventilated at a much earlier stage before the GCS had tumbled, and at a time when the extra dural haematoma would have been much smaller. It is a matter of dispute as to how beneficial earlier intubation would have been and the extent to which, if at-all it would have arrested the deterioration in the Claimant's condition.

32 My third observation is this. I have heard a great deal of expert evidence touching on the time-line dispute as well as on the causation issue. In view of the concessions made in final submissions, it has not been necessary for me to consider large parts of that evidence in this Judgment. On the issue of causation, the crucial expert witnesses are the two consultant neurosurgeons, Mr Nannapaneni for the Claimant and Mr Mannion, called on behalf of the Defendant. I was, however, assisted to some extent on issues of causation by other expert witnesses. I should note that I heard evidence from the following expert witnesses:

a) As already mentioned, Mr Skinner and Mr Campbell-Hewson the A&E experts. Their evidence was relevant to the time-line issue.

b) Professor Schapira, a consultant neurologist, called on behalf of the Claimant. His report

dealt principally with condition and prognosis. On behalf of the Defendant Mr Martin had said that he was content for the report to be read and the Defendant did not call an expert neurologist. Professor Schapira's report did, however, provide his assessment of the mechanics of the injury, referring to " *cerebral herniation"* or *"coning",* meaning displacement of the brain as a consequence of pressure. Different things may cause coning and the brain may be displaced in different directions depending upon the position and direction of the pressure. Here, the cause was the unilateral compression from the extra dural haematoma causing downward and lateral displacement. Professor Schapira had assumed that coning was complete ("he had coned") by 5.00 a.m. but this was based upon his belief that the eyes were not reacting to light at 5.00 a.m. This belief had been caused by a misreading of the medical notes at F7-2207 (a mistake that was easy to make). At 5.00 a.m. the eyes were still recorded as reacting albeit that this was "sluggish'. In fact, Professor Schapira should have recorded that they were no longer responding to light by 5.45 a.m. He explained that coning was not something that happened instantaneously but evolved over a short time. The fact that there was some sluggish reaction to light at 5.00 a.m. may mean that coning was still taking place having begun at or about 4.50. (I note, incidentally, that there is a different single entry in the notes suggesting that the eyes were not reactive at 5.00; F7-2197) However, I consider that the more comprehensive records at 2207 are likely to be the more accurate. The conclusion from this is that coning was not complete, as he had first thought, by 5.00 a.m.

c) Dr Cockings (Claimant) a Consultant Intensive Care Physician and Professor Hardman (Defendant) a Consultant Anaesthetist. Dr Cocking's evidence, to which I will need to return, has a bearing on the ameliorative effects of intubation and ventilation.

d) Dr Halpin, a consultant neuroradiologist called on behalf of the Claimant. As already mentioned, his evidence was concerned with the prioritising of patients for CT scanning. He also assisted with a reading of the image.

e) I was also given a short document " *Resolution of Neuropsychiatric Issues* ". A word of explanation. The parties had been given leave to call neuropsychiatric experts. Reports were prepared by Doctors Fleminger (Claimant) and Agrawal (Defendant). In the event, they were not called and produced the agreed document in which it was accepted that (i) if the frontal lesions could not have been avoided or materially reduced in severity (by earlier surgery) the neuropsychiatric injuries could not have been avoided; and (ii) the frontal lesions have in fact caused or materially contributed to the neuropsychiatric injuries. It was also agreed that whether or not the frontal lesions could have been avoided was not within the expertise or remit of the neuropsychiatrists.

33 I can now move to consider the causation issue. In stark terms: would decompression at 6.00 a.m. have made no difference to the outcome (as the Defendant contends)? Or would there have been a significantly better outcome?

34 The mechanism of the injury is uncontroversial. There had been a fracture of the skull with bleeding into the extradural space The extradural haematoma had rapidly developed causing increasing intracranial pressure and causing a 10 to 15 mm midline shift. The brain was being compressed downwards and it is agreed that, by the time the pupils were fixed (bilateral fixed dilated pupils) the Claimant had coned. Although the record at F7/ 2207 recorded bilateral fixed dilated pupils for the first time at 5.45 a.m. Dr Lal told Mr Manning (see paragraph 10 above) that the eyes were fixed by 5.30 a.m. This is supported by Dr Lal's record made at 5.30 at F7/ F2211. The record was made at 5.30 a.m. but may well record fixed pupils at an earlier time.

35 Coning, as the experts agreed is considered a pre-terminal sign. The brainstem had by this time been compromised. The coning mechanism (see D32 questions 18 & 19) in this case was " *the process whereby an increase in intracranial pressure for whatever reason (in this case expanding haematoma) results in herniation of the brain structures outside the normal anatomical compartments. This is a typically pre-terminal event from which very few patients survive and those that do will of often have devastating neurological injuries* ." This process caused

widespread multiple infarcts.

36 It was because the coning process had completed that the John Radcliffe Hospital initially did not agree to accept Mr Harding for transfer. Coning is seen as a " *pre-terminal event* " and it was thought that he had missed the window of opportunity for successful operative intervention.

37 In parentheses, I should mention the agreed mechanics of the unexpected and remarkable degree of recovery. I quote from the joint report of Mr Nannapaneni and Mr Mannion at D35: " *The experts agreed that, given the circumstances of the patient deteriorating from GCS 11 to 3, developing fixed dilated pupils and having a large extradural haematoma, the usual outcome will be that predicted from Oxford. They therefore agreed that the fact that the claimant had made a much better than predicted outcome must have been that the haematoma stopped expanding shortly after the patient coned, and therefore the ICP did not continue to rise and he subsequently went on to make the recovery that he did.* "

38 The causation issue divides into two parts and two disputes. It was the Defendant's case that, even if there had been an early diagnosis (by 4.00 a.m. as I have found) the development of the extra dural haematoma would have continued on the same path; that the GCS would have fallen to 3/15 by 5.00 a.m. or thereabouts and the coning would have taken place just as it did. Intubation and ventilation was the only available ameliorative procedure and – although it would have been done (and would have been beneficial) – it would not have arrested the deteriorating condition in any material way. When this claim was first formulated, it had been alleged on behalf of the Claimant that the earlier administration of mannitol with intubation and ventilation would have delayed the " *catastrophic deterioration* " by 1½ hours. If that could be shown to be true, the window for successful decompressive surgery would have been widened / extended by that amount.

39 The second part: It was the Claimant's case that, even after the bilateral fixed dilated pupils (that is to say after coning) there was a further window of opportunity for decompression to make a difference. That period could be as long as a further 1½ hours. The Defendant had ever said that the damage, irreversible, was done during the coning process.

40 These are the two principal areas of dispute; whether the coning process could be significantly delayed by mannitol and intubation; and whether the damage to the brain is a continuing process, such that part of the damage would be caused during an unspecified (but significant) period after bilateral fixed dilated pupils. In either event, the likely outcome would have been better; in those circumstances, the negligence would have made a material contribution to the injury.

41 I should now consider the first of those. Could the deterioration have been slowed down by mannitol and / or intubation and ventilation?

42 I can deal speedily with issues surrounding mannitol. In their joint discussions, Mr Nannapaneni and Mr Mannion agreed that the administration of mannitol was not mandated, at least prior to the start of coning. Their full discussion is to be found at pages D25 – 26. They agreed that it would not have been until around 4.50, at a time when the claimant had deteriorated into coma, that mannitol was mandated. After long discussions about the possible benefits and other effects of mannitol they summarised their agreement as follows: " *The experts therefore agreed that while mannitol would be prescribed to a patient in coma from extradural, this was in the knowledge that surgery was being planned as quickly as possible, and what benefits this would have to the claimant are not known* ."

43 But there is a further feature. It is common ground that (as was in fact done later than it should have been) a telephone call would have been put through to the John Radcliffe and this would have been taken by Mr Manning. Mr Manning took the later call, of course. His evidence was that, at that earlier stage, he would not have advised mannitol; he would not have considered it appropriate at that early stage. Thus I am satisfied that mannitol (i) would not have been given and (ii) would have conferred no benefit in any event. That appears to be consistent with the agreement in the joint report, although Mr Nannapaneni, when he gave evidence, persisted in suggesting that mannitol would or may have helped (prior to the start of coning) to keep down the intracranial pressure.

44 Of more importance was the evidence about intubation and ventilation. This issue did not rest solely with the two neurosurgical experts. Dr Cockings also gave relevant evidence, as did his

opposite number Professor Hardman, although Professor Hardman was more reluctant to do so, saying that this was on the edge of his expertise. I will return to Dr Cockings in a moment.

45 In his report Mr Nannapaneni had said this at C63: " *it is likely that the deterioration in the patient's condition would have been delayed or prevented by earlier intubation and ventilation, such that any deterioration would have occurred much later than 4.50 in any event (if at all)."* This, of course, was one of the questions on the agenda for the joint report. It may be noted that, in the answer, Mr Nannapaneni did not repeat that same opinion. This is question 12 at pages D30-1. " *The experts agreed that the reduction in ICP from intubation and ventilation are not on the expanding haematoma. They are via a reduction in brain volume. The claimant's brain was not injured or swollen, it was being compressed and pushed out of its normal anatomical space by the expanding haematoma* ." This was part of the agreed answer.

46 Mr Mannion then expressed his own opinion (at D 31) that where there was diffuse brain injury with a swollen brain, then the benefits (of intubation and ventilation) could be understood. However, in the circumstances of an expanding haematoma, Mr Mannion could not see what the benefits of intubation and ventilation might be.

47 Mr Nannapaneni then added his opinion: " *These were the critical management steps that would have been advised as per the ATLS and other guidelines regarding management of head injury patients* ". Mr Mannion did not disagree with this. The important points from all of this, as Mr Martin pointed out in cross-examination, was that Mr Nannapaneni had failed to reassert his opinion that intubation and ventilation would have delayed or prevented the deterioration. Furthermore, his original opinion was a bald assertion, unsupported by any empirical evidence or any line of reasoned argument.

48 I need to mention Dr Cockings. I should begin by looking at the joint discussions between Dr Cockings and Prof Hardman. Question 13 on the agenda is at page D 22. It asks the question: Do you agree that earlier intubation and ventilation would have delayed or postponed the deterioration in the Claimant's condition? There was agreement that earlier intubation and ventilation would likely have postponed the deterioration if associated with sedation and blood pressure control. Prof Hardman expanded upon this. Earlier intubation and ventilation **might** (his emphasis) have delayed the collapse apparent at 4:50 if brain swelling, airway obstruction, hypoxaemia, hypercarbia or haemodynamic instability materially contributed to that collapse. He went on to say that there was no evidence of any of these prior to the deterioration: " *We cannot ascertain the cause of the deterioration at 4:50 and an earlier intubation might not have affected outcome (for example if the deterioration was caused by the rapid expansion of the extra dural haematoma). The cause of that deterioration is better addressed by neurosurgeons and, in the absence of knowing the cause, we cannot speculate on the likelihood that earlier intervention would have affected outcome* ". It is of real interest that this mirrored almost exactly, the opinions of Mr Mannion. Dr Cockings did not dissent from any of this either at the time of the joint discussion, or later, when he gave evidence. In his opinion, there was not sufficient evidence within the medical notes to be able to **exclude** (my emphasis) these conditions prior to 4.50.

49 When Dr Cockings gave evidence, he continued to assert that earlier intubation and ventilation would likely have delayed the collapse. He was taken to the opinion of Mr Nannapaneni concerning respiratory rates, haemodynamic stability oxygen saturations and so on. He maintained his position. I am not sure that this position was logical. It could only be based upon the hypothesis that, more likely than not the brain was in fact swollen and that more complete investigation prior to 4.50 a.m. would probably have so demonstrated.

50 In my judgment, this issue is principally one for the neurosurgeons and, for reasons which I will explain below, I much prefer the evidence of Mr Mannion on this issue (and indeed other issues). It is clear to me that (i) reduction in intracranial pressure from intubation and ventilation does not affect the expansion of the haematoma but acts to reduce the volume of the brain; (ii) there was no evidence here that the Claimant had any swelling of the brain and there was no evidence for any of the conditions (those mentioned in para 48 above). All the records (incomplete as they were) and other evidence pointed to an absence of these conditions; (iii) on a substantial balance of probabilities, the cause of the deterioration was a rapidly expanding extra dural haematoma which intubation and ventilation could not have affected; and (iv) insofar as there would have been any effect at-all (and I note that this is not ruled out by Mr Mannion) it would have been marginal, making but a minimal difference.

51 Before moving on to consider the next issue (whether the damage continued to develop beyond the end of coning or whether it was substantially caused by and during the coning process) this may be a good moment to explain why I prefer Mr Mannion's evidence over that of Mr Nannapaneni. I begin by saying that I found Mr Mannion to be an impressive witness. His evidence was consistent throughout, from his first report, the joint report, and his evidence from the witness box. His opinions were well argued and analysed. Unlike Mr Nannapaneni, he did not make bald unsupported assertions of opinion. Each of his opinions was based upon careful analysis, and was fully explained. Thus it was, for example, that Mr Mannion was the source of the information that intubation and ventilation acted on the size of the brain, with no independent effect on the haematoma. It was he who first postulated why Mr Harding had achieved a relatively good outcome, when the prospects had originally seemed bleak. This was not something with which Mr Nannapaneni dealt in his initial report.

52 In contrast, Mr Nannapaneni's original report contained a number of assertions and opinions which were unsupported by any reasoning or supportive evidence. It has been submitted on behalf of the Defendant that Mr Nannapaneni's opinions "unravelled" at the expert joint meeting. That is a submission with which I can agree. There are numerous examples. He completely backtracked over the administration of mannitol. At best, many of his opinions were inconsistent. On a number of occasions, when giving evidence, he resiled from the opinions which he had agreed at the joint meeting. I regret to say that, as I found Mr Mannion an impressive witness, I was wholly unimpressed by Mr Nannapaneni.

53 I should give examples. When dealing with the notional timeline, he had said in his report that the time, from arrival at Oxford to decompression, would be but 15 minutes. In the joint statement, he agreed to an overall time of 30 to 35 minutes. He changed his mind again, when he gave evidence. On this issue, I am afraid to say that I found his answers to cross examination disingenuous. There were two distinct periods. First, there was the time between arrival and the beginning of the operation (knife to skin). There was a second period before the surgical opening of the skull would be complete. These were agreed respectively at 15 or 20 minutes, and 15 minutes making an overall time of 30 to 35 minutes. I do not need to rehearse the lengthy cross-examination on this point when Mr Nannapaneni decided to revert to an overall time of 15 minutes. It is sufficient for me to say that I found his response to be evasive and unacceptable.

54 The final issue for determination: if the decompression had been completed (as it should have been) by 6.00 a.m. would Mr Harding have achieved a materially better outcome? This is an issue which I also have to determine in favour of the Defendant. I am entirely persuaded by Mr Mannion's evidence that the material damage had occurred during the coning process. I have already said that I prefer Mr Mannion's evidence and have given my reasons. But, once again, it is apparent that Mr Nannapaneni has shifted his ground.

55 I start with Mr Mannion. His initial report was clear. It contained this: " *Coning is typically a pre-terminal event. When coning occurs, some of the arteries that supply the brain hemispheres are also compressed, because they lie adjacent to fixed structures (the falx cerebri and tentorium cerebelli) which leads to a stroke (infarction)… The vast majority of patients who cone do not survive. This is because whatever the cause of brain herniation (in this case expanding extradural haematoma) leads to an intracranial pressure so high that eventually no arterial blood is able to enter the intracranial compartments… That the Claimant survived informs us that the pressure in his head never reached these heights. Instead the pressure was high enough for him to cone but then must have stabilised shortly after, presumably because the arterial bleeding leading to the extradural haematoma stopped. This means that once coning has occurred, there is no window of opportunity to reverse the damage caused by coning - which is why the majority of neurosurgeons consider surgical intervention futile in these circumstances."* (C 277-8)

56 It is readily apparent that this is a carefully considered analysis of the process of coning and the way in which the injury occurred. There is no comparable section in Mr Nannapaneni's report. On the contrary, he again made bald assertions without more.

57 His report contained this: " *Surgical evacuation carried out with it within two hours after the onset of coma can still result in a very good clinical outcome and in complete or near complete recovery.* " And a few lines further on: " *I note that it is contended that by 4.50 it was inevitable that Mr Harding's outcome would have been the same or materially the same as it had been in any event. I strongly disagree with this suggestion. As explained above surgical evacuation carried out within two hours after the onset of coma can still result in a very good clinical outcome*

and incomplete or near complete recovery ." In fact there was no "explanation", only assertion; and no description of the process. He did refer to literature (Haselsberger et al 1988) and his own clinical experience. I will have words to say about the literature below. He went on to say that, if surgical evacuation of the extradural haematoma had occurred at any time up to 6.50, " *I would have expected him to make a complete recovery".* No analysis, no explanation, and no attempt to justify the statement.

58 I now move to the joint statement. Mr Nannapaneni accepted (i) that the cause of the damage was coning (ii) that intracranial pressure must have stopped rising for the Claimant to make the recovery but (iii) he disagreed with Mr Mannion that the damage was all caused by the coning itself. (These are questions 19, 21 and 24 at pages D32-5). Once again it was Mr Mannion who was making the analysis and explaining the process, whereas Mr Nannapaneni just disagreed. For example: " *Mr Mannion felt that the major disability that the claimant has experienced can be attributed to the infarcts seen on his scan. He felt that this was caused by the process of coning at or around 4.50 hours when the brain herniated, as demonstrated by the changing Glasgow Coma Score and pupillary size and light reactivity. Once there is arterial compression this will result in permanent brain damage within 3 to 4 minutes i.e. the process of a stroke. Mr Nannapaneni felt that the permanent aspects of the Claimant's brain damage occurred later."* (q21 p D33).

59 The point was made (and I agree) that nowhere did Mr Nannapaneni explain why he rejected Mr Mannion's analysis or why he reached the different conclusion. I note that he had also agreed (see paragraph 35 above) that coning is *"typically a pre-terminal event which very few patients survive and those that do will often have devastating neurological injuries".* This does not sit easily with an opinion that decompression some 90 minutes to 2 hours after the completion of coning would result in little or no disability.

60 I need to say something about the literature. A number of publications were provided as support for Mr Nannapaneni's opinions. He had only referred to one of them (the Haselsberger paper 1988) in his report. We spent some time at the trial looking at this and the other papers but it became entirely clear that not one of the papers had any bearing upon the crucial issue in this case; whether a patient who had coned (and with a GCS of 3/15 and bilateral fixed dilated pupils) would be likely to make a good recovery with surgery up to 2 hours later. The Haselsberger paper, for example had reviewed 60 patients with acute extra dural haematoma after **closed** head injury. 25% had died; and 58% had made good recovery. Where the **onset** of coma and decompression exceeded two hours the good recovery rate was reduced to 13% and the mortality rate rose to 65%. But, of crucial importance, these studies were not limited to patients who had coned with a GCS as low as 3/15. (Incidentally, 3/15 is the lowest possible score on the scale; there is no provision for a score of 0, 1 or 2.) Without knowing more about the pre operative condition of the recovered patients, the paper does not add anything to the debate. What we do know (and one would have thought this to be obvious) is that the " *outcome was found to be predominantly influenced by the preoperative state of consciousness, associated brain lesions, and the duration of the time interval between onset of coma and surgical decompression* ". Insofar as the literature had any relevance to the issue I have to determine, it appeared to me to demonstrate that Mr Mannion's analysis was correct. The paper at D116 for example showed a very poor prognosis for patients with fixed dilated pupils and a GCS of 3. True it was – as Miss Rodway pointed out – that this study appeared to be concerned with patients with primary brain injury. However, by extrapolation, by the time Mr Harding had coned, he was arguably in a similar position to those patients. I do not place any great reliance upon this and other studies (D126, D103 for example) because they were concerned with severe traumatic brain injury. But they do provide some thought provoking material showing, for example, that bilateral fixed dilated pupils are regarded as a sign that the brain has herniated with concomitant injury to the brainstem.

61 In my judgment, Mr Mannion's analysis makes complete sense. It accords with the known facts and it fully explains the events and the nature of the Claimant's injury. I have no hesitation in accepting his evidence. It also seems to me that it is more likely than not that the levelling off of the intracranial pressure would itself mean that no further injury was occurring.

62 Before I leave this issue, I need to refer to the joint answer to question 23 on page D34. Therein, Mr Mannion made the concession that it was possible that persistently elevated intracranial pressure had caused further damage but explained why he believed that " *the majority of the Claimant's brain damage occurred during coning around 4.50 hours* ". Elsewhere

(as well as in his evidence to me) he explained that any post coning damage would have been, in his opinion, de minimis.

63 In case it is not clear, I make the following findings. If there had been early intubation and ventilation, the coning process would not have been delayed to any material extent. Any marginal benefit (from intubation and ventilation) would or may have postponed the coning by, at the most, a minute or two. In so far as it was accepted that a small part of the Claimant's injury occurred after the end of coning, decompression at 6.00 a.m. would have made no material difference to the eventual outcome.

64 I think it also follows from all of this that the Defendant's admitted negligence made no material contribution to the injuries. At the very most, in my judgment, the Claimant lost the chance of a very small improvement in outcome. There must be judgment for the Defendant.