Case No: TLQ/13/1324

Neutral Citation Number: [2015] EWHC 1311 (QB)
IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 13/05/2015

Before :

MRS JUSTICE COX DBE

- -

Between :

Mr SHAHINOOR CHOUDHURY
(by his Brother and Litigation Friend, Mr Saminoor Reza Choudhury)

Claimant

- and -

SOUTH CENTRAL AMBULANCE SERVICE NHS

First Defendant

PORTSMOUTH HOSPITALS NHS TRUST **Second Defendant**

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Michael Kent QC (instructed by **BL Claims, Solicitors**) for the **Claimant**
Sir Robert Francis QC and Katie Gollop (instructed by **DAC Beachcroft LLP, Solicitors**) for the **Defendants**

Hearing dates: 15-20 April 2015

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Judgment

Mrs Justice Cox:

Introduction

1. This is a desperately sad case, as counsel for the Defendants rightly observe. On 16 March 2010 the Claimant, Shahinoor Choudhury, then aged 42 suffered an ischaemic stroke. He developed "locked-in syndrome" due to infarction in the brainstem and cerebellum, secondary to basilar artery thrombosis. He is virtually totally paralysed, apart from limited vertical eye movements, tiny movements of his mouth and right index finger and automatic breathing. He is doubly incontinent. He has a tracheostomy, requires regular suctioning and is fed through a PEG tube. He cannot speak or swallow, his ability to communicate is extremely limited and he is utterly dependent on 24-hour nursing care. The nature and extent of his disability and of the difficulties in caring for him are clearly demonstrated in the DVD prepared by those representing him, which I saw before the trial started.

2. The Claimant's cognitive ability is impaired, such that he does not have the capacity to conduct this litigation, and his brother, Saminoor Choudhury, is acting as his Litigation Friend. The Claimant is, however, fully aware of his condition and the psychological consequences of this must be profound. There is no prospect of improvement and his life expectancy is markedly reduced. The experts have agreed that, on the balance of probabilities, he is likely to survive for only another seven years.

3. After being discharged from hospital in 2011 the Claimant was initially cared for by Saminoor Choudhury and his wife, Nikola, at their family home, with the help of carers. The evidence as to the challenges and difficulties they had to overcome and the constant, determined and devoted care provided, in particular from Nikola Choudhury, merits recognition and the Defendants rightly pay tribute to it, as do I. Since August 2013, however, the Claimant has lived at the registered nursing home "Cams Ridge" in Fareham, Hampshire where he is being provided with high quality, professional care.

4. The Claimant has suffered from locked-in syndrome since 17 March 2010. He brings this claim for damages for negligence against the First Defendant, as providers of emergency ambulance services, and against the Second Defendant, as the employers of medical staff at the Queen Alexandra Hospital in Portsmouth, where the Claimant was brought by ambulance on the previous day. It is alleged, in summary, that there was negligent delay in taking him to hospital on 16 March, in diagnosing his condition and in providing the necessary treatment for him.

5. Significant breaches of duty have been admitted by both Defendants, but there is a dispute as to causation of injury. The Claimant contends that on the balance of probabilities those breaches of duty, and in particular the failure promptly to start the Claimant on aspirin, caused his deterioration and the development of locked-in syndrome on 17 March. The Defendants submit that treatment with aspirin would not have resulted in a better outcome for this Claimant in the particular circumstances of this case; or rather that it cannot be shown to the requisite civil standard that he would have been left less disabled than he is now.

6. The hearing before me was originally listed to determine both causation and various quantum issues, which narrowed in the weeks leading up to trial. I heard oral evidence from Saminoor and Nikola Choudhury relevant to both issues and I read a witness statement from the Claimant's friend, Ben Farodoye. In light of the admitted breaches of duty it was unnecessary for the Defendants to call any factual evidence.

7. The causation issue turns on expert evidence, principally the evidence of the stroke experts, Professor Martin Brown, consultant neurologist called on behalf of the Claimant, and Dr Elio Giallombardo, consultant stroke physician for the Defendants, who both gave oral evidence. The parties also relied on the reports and joint statement of the consultant neuroradiologists, Dr Nelson (Claimant) and Dr Molyneux (Defendants); and, in relation to breach of duty, on the joint statement of the Accident and Emergency consultants Mr Cottingham (Claimant) and Mr Holburn (Defendants).

8. The causation issue is complex, being far easier to formulate than to resolve. In the event, having heard the causation evidence over three days, it was agreed that I should hear counsels' submissions and determine that issue first before proceeding to the quantum issues, if appropriate. My decision and the reasons for resolving the causation issue in the Defendants' favour are now set out in this judgment, which was reserved following the helpful submissions from both leading counsel.

The Relevant Facts

Events Before the Claimant Arrived at Hospital

9. In March 2010 the Claimant was living on his own in a flat on the top floor of a block of flats in St Simon's Road, Southsea. He was a generally fit, healthy and active man and for several years he had been working full time at the computer company IBM.

10. At around 1.00 am on 16 March he began to feel seriously unwell, because at 01:24 he called the ambulance service. He complained of feeling dizzy and short of breath, of being sweaty and having sharp pains in his head. The recording made of this call shows that he was breathing fast and deeply and was having difficulty talking because of his rapid respiratory rate.

11. An ambulance was sent and a paramedic attended him at his home at about 01:29. The ambulance report indicates that the Claimant referred to his partner having left him two weeks earlier, since when he had been working excessively and training, having little sleep. Observations made at 01:33 and 01:40 include observations that the Claimant's respiratory rate was raised, that his Glasgow Coma Scale (GCS) score was recorded as 15 (out of 15), and that his oxygen saturation was normal. No formal diagnosis was recorded. The paramedic advised rehydration, gave the Claimant a glass of water and assisted him to bed. The Claimant was advised to contact the Portsmouth Out of Hours GP service, if needed, and to see his GP in the morning. The paramedic left at about 02:15.

12. Allegations of negligence are made in relation to this first visit. It is alleged that the call taker failed to brief the paramedic fully; and that the paramedic failed to undertake an adequate neurological examination and to arrange the Claimant's admission to hospital for further investigation. These allegations are all denied by the First Defendant, but it is unnecessary to consider them further because it became

apparent, given the views of the experts, that they added nothing in terms of causation of damage to those allegations which are admitted. Thus, although not formally abandoned, these disputed allegations have not been pursued at this hearing.

13. At 03:11 the Claimant did telephone the GP Out of Hours service. A doctor called him back at 03:17 and the Claimant explained that he was having problems breathing and was feeling sick and dizzy; that he had vomited several times and passed out; and that he was unable to drink water or lift his head. The doctor advised him to call 999 with a view to his admission to hospital.

14. The Claimant called the ambulance service for the second time at 03:20, telling the call taker that he was having problems breathing and was being sick. He also described feeling pins and needles, and dizziness to the point that he felt he was spinning around. The recording of this call reveals that his speech was slurred and that the Claimant was still breathing fast and deeply. The call taker was unsure as to whether he was fully alert.

15. An ambulance was sent to the Claimant's home, arriving at 03:27. The paramedic had been told only that the Claimant was sick and conscious, and that his breathing was abnormal. The observations he recorded included a respiratory rate of 36, which he considered to be normal. The Claimant's oxygen saturation was 100 per cent, his pulse was 72 and his blood pressure was 130/80. His GCS score was recorded as 15. Before he left, the paramedic recorded the following on the report form:

> "*Presenting complaint – hyperventilating and vomiting.*
> *History of presenting complaint – similar episode 2 hours ago. Called ambulance - later called OOH → 999.*
> *Previous medical history – nil*
>
> *Observations A* [Airway] *B* [Breathing] – *hyperventilating – calms down when talking. Has vomited - strained to vomit. Has been to gym - worked out, had sauna, then ate – has vomited his food.*
> *Advised patient re rehydration. Not needed to go to hospital. Advised patient to sip fluids and go to bed."*

16. The First Defendant has always admitted that the failure to take the Claimant to hospital without delay, following this second 999 call, was a breach of duty. There is no dispute that, if he had been taken immediately to hospital, the Claimant would have arrived at about 04:00 and therefore before the first, significant deterioration in his condition, which I now relate.

17. At about 04:15 the Claimant rang his friend, Ben Farodoye, and left a voicemail message, as follows:

> "*Ben I can't come into work as I have been told by the paramedics that I have food poisoning. Please pass this message on to my colleagues at work.*"

Mr Farodoye said in his statement that the Claimant sounded unwell and very tired and that his speech sounded slurred and unclear.

18. Shortly after the Claimant left this message it is agreed that, at around 04:30, he suffered a serious collapse. At 04:23 the Claimant rang Nikola Choudhury's mobile phone. Aroused from sleep she called him back. The Claimant answered the phone, but she struggled to understand what he was saying because his speech was slurred and he sounded as if he had no control of his tongue. She asked questions, to which he was able to reply "no" or "yes". The Claimant was someone who regularly socialised and enjoyed having a drink with friends and, at the time, Mrs Choudhury thought that he may just have been drinking. She understood from his responses that he wanted them to go to him and Mr and Mrs Choudhury therefore drove over to his flat, which was about a mile away.

19. On their arrival Nikola Choudhury phoned the Claimant again and he answered her call. She was still unable to understand him and she also heard him being sick. By now it was about 04:35 or 04.40. She asked him to press the entry release button to let them in, but she got no clear response. On pressing the entry button she heard what sounded like loud snoring noises at the other end. After trying unsuccessfully to get in or to get any response from the Claimant, and hearing continued snoring, they both concluded that he had probably fallen asleep after drinking heavily and left. They knew the Claimant to be a snorer because he had previously lived with them for a while.

20. Nikola Choudhury went back to the block of flats at about 07:00 am, before she went to work, but the Claimant was not answering his phone. When she pressed the entry button she could still hear him snoring. She then had to go to work but, getting no response from him during the morning and feeling worried, both Mr and Mrs Choudhury decided to go back to the flat during their lunch breaks.

21. After pressing the entry button and still hearing the sound of snoring, Nikola Choudhury gained entry to the building by alerting neighbours. The front door to the Claimant's flat was ajar and it was difficult to push open, because of an obstruction. Pushing harder, she found the Claimant lying on his side on the floor, blocking the way and with his feet facing the door. He was not moving and, although his eyes were open, she assumed that he was unconscious because she could not rouse him or get any response from him. He was still making the same snoring sounds and she could see vomit on the floor next to him. Mr Choudhury joined her and both of them were obviously extremely distressed. She called 999 and the Claimant remained as he was until the ambulance came, making no voluntary movements of any kind and making what she described as "light breaths".

22. The paramedic arrived at 13:00 and, as he was dealing with the Claimant, Nikola Choudhury noticed that vomit was spread around different parts of the flat. On examination the paramedic recorded that the Claimant's airway was partially compromised, that his breathing was laboured and deep, and that his GCS was down to 8 out of 15 (Eyes 3 out of 4, Verbal Response 1 out of 5, and Motor Response 4 out of 6). The ambulance arrived at 13:13 and the Claimant was taken to hospital, where his arrival in Accident and Emergency was recorded at 13:48.

Events at the Hospital

23. In A&E the Claimant was assessed initially by a nurse at about 14:00, who noted "*Collapse? Cause*". At 14:50 the recorded observations included a temperature of

37.5, a respiratory rate of 28, a pulse of 85 and a GCS score of 10/15. His oxygen saturation was 100 per cent and remained at that level throughout the afternoon.

24. The Claimant was next seen in A&E by Dr Bradbury, whose note is untimed. Neurological examination was limited to ascertaining the size of the Claimant's pupils and noting the absence of meningism. It was decided to intubate him so that he could have a CT brain scan. There was no diagnosis or differential diagnosis at this time.

25. The CT scan was performed at 15:30 and was reported as showing no abnormality. The report reads:

> "*No haemorrhage, infarct or space-occupying lesion seen. The ventricular system is within normal limits. Conclusion: No cause for collapse seen.* "

26. It is accepted that the radiologist's report was incorrect. The scan in fact showed abnormalities, including an abnormal high density in the upper part of the basilar artery, consistent with acute thrombosis, and patchy low densities, consistent with non-haemorrhagic infarctions in the cerebellar hemispheres, subsequently confirmed on the MRI performed on 19 March.

27. Following the scan the Claimant was extubated, at which point he was able to open his eyes and was noted to have an "*Excellent cough*" and to be able to lift his head off the pillow for three seconds. He was then referred back to Accident and Emergency and reviewed at 17:20, when the doctor noted that the CT scan was normal. His observations remained stable and his GCS score was recorded as 11/15. No diagnosis was noted and a medical opinion was sought.

28. Dr Brown, Medical Specialist Registrar, attended at 20:00 and noted that the Claimant was snoring, with a GCS of 9/15. He noted possible left-sided weakness, but no detailed neurological examination was recorded. The Registrar made a differential diagnosis of encephalitis, toxin or ischaemic CVA not seen on the initial CT scan. A range of blood tests and a lumbar puncture were requested.

29. The lumbar puncture was performed at 22:30 and was recorded as revealing clear, colourless cerebrospinal fluid, with an opening pressure of 18.5cm. The Claimant was then noted to be "*lying flat – no complications*" and was referred to the ITU Registrar.

30. Overnight, however, the Claimant's condition suddenly deteriorated further. At 02:30 on 17 March his GCS was noted to have dropped to 6/15 and it had then dropped to 3/15 by 04:00. He was admitted to the ITU at 06:57, where he was first seen by Dr Knighton at 10:25 on 17 March. That doctor was also unable to make a diagnosis and ordered further tests. The Claimant was reviewed by Dr Knighton at 14:54, when his eye movements were first noted to be abnormal. At 17:00 Dr Knighton noted that there was still no clear diagnosis and that the Claimant appeared unable to move his limbs at all, even to painful stimuli.

31. On 18 March Dr Knighton saw the Claimant at 08:20 and considered that he might be suffering from a "*pontine locked-in type vascular event.*" He requested a neurological opinion and at 15:12 the Claimant was examined by consultant neurologist, Dr

Halfpenny, who also reviewed the CT scan and correctly regarded it as abnormal. He concluded that the Claimant had suffered an infarct in the left upper cerebellum. He also established that the Claimant was conscious and he considered, correctly, that he was suffering from locked-in syndrome. The Claimant was first given aspirin on 18 March. The MRI scan carried out on 19 March showed "*Basilar occlusion with brainstem and cerebellar infarcts*."

32. On 20 March Dr Knighton explained to the Claimant that he had suffered a stroke, caused by the blockage of an artery at the base of his brain. He was transferred to the Stroke Ward on 9 April and underwent rehabilitation, without improvement, until he was transferred to the Phoenix Rehabilitation Centre on 27 July 2010 for multi-disciplinary input. He was eventually discharged home to live with Mr and Mrs Choudhury on 5 April 2011.

33. The Second Defendant has admitted a number of allegations of breach of duty at the hospital, namely: delay in performing the CT brain scan; incorrectly interpreting the scan as normal, when in fact, in the clinical context, it showed a basilar artery occlusion; consequent delay in providing treatment with aspirin, which should have been given after the CT scan had excluded haemorrhage; and consequent performance of a lumbar puncture to investigate meningitis, which would have been avoided if the basilar artery occlusion had been diagnosed.

The Causation Issue

34. Helpfully, the parties have agreed a chronology of the events that would have happened in this case but for the negligence of both Defendants, as follows:

(i) The paramedic who arrived at the Claimant's home at 03:27 on 16 March would have taken him to hospital.

(ii) The Claimant would have arrived there at about 04:00 but in any event before 04:30.

(iii) On arrival the Claimant would have been triaged and would have had some basic observations taken.

(iv) At around 04:15 the Claimant's speech would have become slurred, as in fact it did.

(v) He would have had vomiting, as in fact he did.

(vi) At around 04:30 he would have suffered a major collapse and been unresponsive, as in fact he did and was. He would have become incapable of movement and of speech and his eyes would have been fixed and unreactive.

(vii) That major collapse would have prompted the doctors to suspect an intracranial event.

(viii) An out of hours CT scan would have been requested soon after the collapse.

(ix) The Claimant would have needed to be intubated, in order to protect his airways before the CT scan could have been performed.

(x) The CT scan would have been performed by 06:00.

(xi) Radiologically, the signs of a basilar artery occlusion would have been difficult to assess, but in combination with the clinical picture, a diagnosis of suspected basilar artery occlusion would have been made.

(xii) The Claimant would have been treated with aspirin. That treatment would have been started shortly after 06:00.

35. Originally the Claimant's case was that, if he had been taken to hospital by either the first or the second paramedic, he would have been treated with intravenous thrombolysis. In the course of preparation for trial the evidence established that thrombolysis was not available out of hours at Queen Alexandra Hospital and so would not have been given. That allegation is therefore no longer pursued. By the time of this hearing the Claimant's case was clearly advanced on the basis that anti-thrombotic therapy in the form of aspirin would have been administered, as part of the good quality stroke care that the Claimant would have received. As is now well understood, the role of aspirin in stroke prevention is to inhibit platelet stickiness and thereby to inhibit clot formation or propagation.

36. The Claimant's pleaded case on causation, based on the opinion of Professor Brown, is as follows:

> "29. Had the Claimant been taken to hospital on either of the occasions when a paramedic attended him at home, he would or should have received an adequate neurological examination promptly on arrival in hospital followed by a CT scan within 60 minutes of arrival and then treatment would or should have been commenced. In the case of basilar artery thrombosis, the Basilar Artery International Cooperation Study found that the outcomes in the case of basilar artery thrombosis were similar whichever treatment was given such that even had he only been given aspirin or anticoagulation [heparin], the probability is that he would have avoided reaching the point of suffering from locked-in syndrome and would have been left with no more than some residual disability such as being slightly unsteady on his feet, some residual impairment of fine motor coordination and possibly some slurring of the speech. On the balance of probabilities, he would have been able to walk, care for himself and work.
>
> 30. By the time the Claimant actually arrived at hospital, it is unlikely that he would have made a good recovery. However, the deterioration in his condition which occurred while he was in hospital would have been avoided. Further or alternatively, had he not been extubated and had he not been exposed to the lumbar puncture, but still suffered from locked-in syndrome, the probability is that he would have been left with some movement of the fingers or head sufficient for him to be able to operate a communication device. As it is, he is effectively virtually totally paralysed."

37. The Claimant now accepts that the serious collapse he suffered at 04:30 on 16 March could not have been prevented by either Defendant. Further, he accepts that the basilar artery occlusion that occurred at that time caused irreversible brain damage, resulting in permanent disability. The injury in respect of which he claims damages is therefore the difference between that level of disability and the locked-in syndrome from which he now suffers and which, as is now agreed, developed after his further deterioration on 17 March.

38. The issue to be determined is essentially this. On the balance of probabilities, if aspirin and good stroke care had been given to this Claimant in hospital between 06:00 on 16 March and 02:30 on 17 March, would he have avoided the further deterioration and locked-in syndrome that occurred? Professor Brown's opinion is that he would. Dr Giallombardo considers it more likely that the outcome would have been the same.

The Cause of the Stroke

39. Professor Brown originally considered, in his causation report date 7 May 2014, that the cause of the Claimant's basilar artery thrombosis would probably never be known. However, he has since concluded, as he stated in evidence, that the most likely cause of the Claimant's basilar artery symptoms was embolism of thrombus from the vertebral artery.

40. He considers the evidence to indicate that the course of the Claimant's symptoms and signs was one of step-wise deterioration. The onset of symptoms at around 01:00 am on 16 March was followed by the Claimant's sudden, serious deterioration and collapse by his front door at about 04:30. While he sadly remained in that position, unattended, for just over eight hours the Professor's view, given the descriptions of the Claimant's condition on arrival at hospital, is that his stroke symptoms had not deteriorated over that eight hour period. Further, while in hospital his neurological condition remained stable from about 14:00 until his second, serious deterioration in the early hours of 17 March. At that point, starting at about 02:30 there was a sudden drop in his GCS score from 9 to 3 over a short period, so that by 04:00 he had suffered the catastrophic deterioration which led to his locked-in syndrome.

41. Professor Brown considers the Claimant's step-wise deterioration, marked by sudden episodes of this kind, to be typical of embolism. He points out that the commonest cause of basilar artery symptoms is embolism of thrombus from the vertebral artery (see *Ferbert A, Bruckmann H, Drummen R. Clinical features of proven basilar artery occlusions. Stroke 1990; 21: 1135-1142*). In this case, the nature of the step-wise deterioration indicates that the sudden deterioration on 17 March was probably the result of recurrent embolism. In his view, that is the most likely mechanism in the Claimant's case.

42. The scientific process involved mirrors the Claimant's symptoms. Blockage in the basilar artery reduces the supply of the blood to the cerebellum and brainstem, resulting in infarction. The cerebellum is responsible for the coordination of motor actions, including walking and speaking. The brainstem contains important nerve cells, including those that control breathing, heart rate and conscious level, and centres that control eye movements and are responsible for initiation of vomiting. The reduction in blood supply from occlusion of the basilar artery therefore results in

infarction in the territory of the blood vessels supplied by branches of the basilar artery.

43. The extent of that infarction will depend on the position and extent of the thrombus in the basilar artery, and the extent of collateral blood supply reaching that artery from the other blood vessels. The initial symptoms of basilar artery thrombosis often include dizziness, visual disturbance, slurred speech and vertigo caused by ischaemia in the brainstem. Dilation of the collateral blood vessels trying to get blood to the brain via other pathways often results in headache.

44. When the Claimant first called 999 at 01:24 he was clearly suffering from symptoms recognised to indicate the onset of the thrombosis. His symptoms when he called 999 for the second time were similar in nature, indicating that there had been no significant change at that stage.

45. It is also clear on the evidence that a serious and dramatic deterioration in the thrombosis occurred at around 04:30, with resulting ischaemia in the cerebellum and brainstem. The description by Mr and Mrs Choudhury of the Claimant's symptoms, as at that time, indicates that the Claimant had a reduced level of consciousness and was unable to produce any speech. The fact that, through the entry button system, they could hear him producing a snoring sound indicates that he was probably lying in the position where he was subsequently found, suffering weakness in some or all of his limbs and unable to move. The Claimant accepts that this deterioration was sufficient to cause permanent and irreversible brain damage affecting his mobility, motor control and speech.

46. The experts are agreed that, after this collapse at 04:30, the Claimant's neurological condition then remained stable until the next, serious deterioration in the early hours of 17 March. The Claimant's case, which I accept, is that the abnormal high density seen on the CT scan in the upper part of the basilar artery, at the level of the dorsum sellae, suggests that the basilar artery thrombosis was then at a high level and not blocking all the branches of that artery. This, in my view, would be consistent with the "*Excellent cough*" noted after the scan, together with the Claimant's recorded ability to lift his head off the pillow for three seconds and his consistent GCS scores.

47. Tragically, it is likely that the Claimant had a recurrent stroke when a further embolism from the vertebral artery suddenly broke away in the early hours of 17 March and added to the thrombus in the basilar artery, "back-filling" the artery completely and blocking the remaining lower branches. The resulting, extensive infarction transacted the brainstem at the level of the pons, causing the Claimant to develop locked-in syndrome.

48. Dr Giallombardo initially saw embolism as one of two equally possible mechanisms of injury, the other being partial occlusion of the artery initially, with the thrombus enlarging gradually to full occlusion on 17 March. However, he accepted in the witness box that embolism of clot followed by further embolism on 17 March was consistent with the clinical picture overall and he did not disagree with Professor Brown's analysis. I find on the balance of probabilities that this is, in fact, what occurred. It did not however affect Dr Giallombardo's conclusion that the Claimant's thrombosis would have followed its natural course in any event, and that aspirin would not have prevented the outcome. To that issue I now turn.

Prevention of the Claimant's deterioration and the outcome

49. Stroke is the third most common cause of death and the most common cause of disability in the UK adult population. The experts agree, however, that basilar artery thrombosis is relatively uncommon, accounting for only 1 per cent of all strokes. Further, as Professor Brown explained, basilar artery thrombosis has a different pathological substrate compared with other types of stroke, as evidenced by its tendency to show step-wise deterioration of the kind that occurred in this Claimant's case. It is a serious condition because of its connection to the brainstem and the serious and sometimes fatal damage that can result. He recognised, at paragraph 7.7 of his causation report, the "high risk of progression" in cases involving thrombosis of the basilar artery.

50. Given this tendency for step-wise deterioration, the normal approach towards a mild stroke, namely that it will stay a mild stroke and that the patient will not get worse, simply does not apply. The prognosis for patients with basilar artery thrombosis is worse than for those afflicted by other strokes. The authors of the Basilar Artery International Cooperation Study (BASICS) in 2009, upon which Professor Brown places considerable reliance in this case, noted that "*Despite recent advances in the treatment of acute stroke, the rate of death or disability associated with BAO [*basilar artery occlusion] *is almost 80 per cent.*"

51. The consensus of the literature considered by both experts in this case is that recurrent stroke is relatively rare. Professor Brown said in cross-examination that, in general, the rate of recurrent stroke within one year is 4 per cent. No literature addresses specifically the incidence of recurrent stroke in cases of basilar artery thrombosis, but it would seem likely, given its characteristics, that it is higher than the incidence in other types of stroke. Both experts are agreed that the further, sudden deterioration suffered by the Claimant in the early hours of 17 March was entirely consistent with what is known of the natural history of this relatively rare condition.

52. The Claimant's case is therefore that, but for the negligent failure to provide treatment with aspirin as part of good stroke care, from 06:00 onwards on 16 March, the recurrent basilar artery thrombosis starting at about 02:30 on 17 March would have been prevented. The Defendants, although accepting that in general it has been established that the beneficial effects of aspirin outweigh the risks of providing it, submit that there is simply no evidence that, in the circumstances of this Claimant's case, giving him aspirin as part of good stroke care after 06.00 on the 16th would have prevented his deterioration in the early hours of the following morning. The suggestion that it would amounts only to speculation.

53. Both the experts who gave evidence in this case are highly experienced in the treatment of stroke patients. Professor Brown is rightly acknowledged by the Defendants as an eminent authority on stroke and he is also a highly experienced expert witness. He is Professor of Stroke Medicine at the Institute of Neurology, University College London, and Consultant Neurologist at the National Hospital for Neurology and Neurosurgery (NHNN) at Queen's Square and University College Hospital (UCH). His CV is extensive and impressive. As he explained in evidence, together with his colleagues he has been closely involved in developing a comprehensive service for patients with stroke at these institutions. This work has involved several innovations in service delivery, including the Hyperacute Stroke

Clinic at UCH and other Acute Stoke Units at the NHNN. He was a founding member and then President of the British Association of Stroke Physicians and was the first Chairman of the National Stroke Medicine Subspeciality Advisory Committee (2002-2008). As the Royal College of Physicians' representative on the Intercollegiate Working Party for Stroke he was involved in the development of the National Clinical Guidelines for Stroke, in that the College fed into those Guidelines, which were issued in 2008 and remain in place.

54. Since 2006 Dr Giallombardo has been Consultant Stroke and General Physician at North Hampshire Hospital in Basingstoke. He is also the Principal Investigator in a number of multicentre trials supported by the UK Research Network. He has been a consultant physician in the UK since 1995 and now has special responsibility for stroke, caring almost exclusively for patients referred with stroke or suspected stroke. Unlike Professor Brown, Dr Giallombardo is, on his own admission, an inexperienced expert witness. He frankly admitted that he found the balance of probabilities approach to causation puzzling from the medical perspective and his reports, in part, reflected that. His work has, however, involved him spending considerable time in the Emergency Department assessing and treating stroke patients in the hyper-acute period, and there is no doubt that he has extensive clinical experience.

55. Both experts agree that there has in general been a vast improvement in stroke management and treatment since the 1990s. Since 2008, as Professor Brown explained, the accepted view has been that stroke patients admitted to Accident and Emergency should be sent immediately to the stroke unit/department at the relevant hospital, to be managed appropriately. There was such a specialist stroke service at Queen Alexandra Hospital in March 2010. Those patients who have received this specialist treatment have been observed to have improved outcomes, when compared with patients for whom there is a delay in assessment by the specialist stroke team and management by general medical services instead. Quite apart from the prescription of appropriate medication in the form of thrombolysis, aspirin or anticoagulant (heparin), much of the benefit has come, in Professor Brown's view, from early recognition of the diagnosis of stroke, appropriate monitoring, the prevention of complications and early interventions to manage deterioration through the correction of dehydration, with early intubation and ventilation of the unconscious patient.

56. In general terms none of this is controversial. It is Professor Brown's transposing of these generalities to the specifics of this case, and the way in which that has been done to which the Defendants object. It is submitted that, while his opinions have been confidently expressed, they are, on a correct analysis, unsupported by the underlying material in this case; and that he has been prone to overstate the conclusions that can properly be drawn, in particular as to the effects of aspirin. A number of criticisms have been raised of his approach and of the way his views in this case have changed, which I address at this stage. Save for some minor changes made in the witness box, irrelevant to these criticisms, Professor Brown, in his evidence-in-chief, adopted all his reports and letters as representing his analysis and conclusions in this case.

57. His first report for the Court, dated 25 February 2013, addressed the Claimant's condition and prognosis. It is clear, from paragraphs 7.1 and 8.1, that at that stage he considered the CT scan of 16 March to show not only that there was abnormal density in the basilar artery, but also that the brainstem in the region of the pons was swollen

and that the adjacent pontine cisterns were effaced. This led him to conclude that the infarction in the pons, secondary to basilar artery thrombosis and which then resulted in locked-in syndrome, had in fact occurred on 16 March and not in the early hours of the following day.

58. He maintained this position in his causation report of 7 May 2014, expressing the same opinion (at paragraph 5.1) as to the abnormalities he considered were shown on the scan. It is clear from paragraphs 6.5 and 6.6. that he considered the pontine infarction to have occurred after the second 999 call, at about the time the Claimant collapsed.

59. At paragraph 8.5, assuming that the Claimant had been taken to hospital after 03:20, as he should have been, his view was that the Claimant's deterioration and collapse would have occurred in hospital at around 04:30. This would have mandated an emergency CT scan which, in the clinical context, would have been diagnostic of basilar artery thrombosis. This, he said, would have led:

> "… to the immediate prescription of medication to prevent thrombosis. The usual antithrombotic treatment recommended by neurologists for basilar artery thrombosis, if thrombolysis and thrombus extraction are not available, is anticoagulation with heparin. This is preferred over aspirin in patients with progressive symptoms because heparin has a very rapid action within minutes after administration. ... the heparin would have had an immediate effect on the process of thrombosis halting any progression."

60. In relation to the Claimant's subsequent deterioration, Professor Brown expressed the following view in addition at paragraph 8.6:

> "It is notable that he deteriorated after the lumbar puncture and it is likely, on the balance of probabilities, that at least part of his deterioration was the result of the lumbar puncture lowering the pressure of cerebrospinal fluid. It is therefore likely that he sustained additional, unnecessary brain damage as a result of not being cared for by a specialist stroke service, as a result of not being ventilated or monitored closely while he had impairment of his Glasgow Coma Score and as a result of the unnecessary lumbar puncture. Thus it is likely that Mr Choudhury's outcome would have been better in the event if he had been admitted and diagnosed earlier after the first or second 999 call, irrespective of specific treatments for basilar artery thrombosis. "

61. He concluded, at paragraph 10, that by arriving at hospital by 04.00 the Claimant would have had the benefit of the diagnosis of stroke, heparin treatment and correction of dehydration. He would have avoided respiratory compromise, the adverse effects of lumbar puncture and being 'locked-in' by his stroke. He considered it likely, on the balance of probabilities that the Claimant would have been far less disabled, requiring some help but able to walk unassisted, with the probability of further improvement with rehabilitation.

62. It is clear from paragraph 8.11 that his view at this time was that, by the time the Claimant was admitted to hospital at 13:48 on 16 March, it was already too late to avoid locked-in syndrome. He arrived at the following conclusions:

> "If he had been treated properly when he eventually arrived in hospital at 13:48 after becoming unresponsive, it is unlikely on the balance of probabilities that he would have made a good recovery. By the time he was eventually taken to hospital, it was too late to prevent the devastating pontine infarction which led to his locked-in syndrome. On the other hand, his outcome would, on the balance of probabilities, have been better if the diagnosis of basilar artery thrombosis had been made, and he had been referred to a specialised stroke service with avoidance of respiratory compromise and had not had the lumbar puncture. Most patients with locked-in syndrome who have survived as long as Mr Choudhury recover some slight movement of the fingers or head, sufficient for them to operate a communication device and I therefore consider it likely, on the balance of probabilities, that he would have recovered at least to this level if he had been properly managed when he eventually arrived in hospital."

63. A number of criticisms were made of the opinions expressed in these passages, quite apart from Professor Brown's use of the BASICS findings which I will consider later on in this judgment.

64. First, the Defendants submit that his opinions were expressed on the basis of an interpretation of the CT scan which was incorrect, and which was not supported by the consultant neuroradiologists instructed in this case. Relying on his own interpretation of the scan, Professor Brown proceeded on the basis that the window of opportunity to avoid locked-in syndrome had already closed by the time of the Claimant's actual admission to hospital at 13.48.

65. They point out however that, by the time of this causation report, Dr Nelson had already reported (December 2013), and had found no evidence of pontine changes on the CT scan. In his evidence Professor Brown made it clear that he would defer to neuroradiological opinion as to the imaging, but he had not at any stage revisited the views expressed in his report to reflect it, or to set out his conclusions having regard to it. Nor did he do other than adopt this report in his evidence-in-chief.

66. There is some force in this criticism. What the scan showed was very relevant to the Claimant's condition at the time, to the step-wise deterioration described by Professor Brown, and therefore to the issue of causation. I make allowance for the complexity of the case and the usual evolution of expert opinion as a fuller picture of events is obtained, but this is an important factor in the case. It would have been helpful to the court if, with the benefit of further analysis, Professor Brown had considered whether any amendments were required to what were on any view robustly expressed conclusions, in particular as to the probability of avoiding locked-in syndrome by the provision of good stroke care and heparin, even after the serious brain injury he then believed had occurred by 04.30.

67. When giving evidence, far from contending that the scan showed a "devastating pontine infarction", as he had reported, he sought in cross-examination to rely on the

fact that the scan showed that there had been no extensive damage to the brain at 15.30, stating that "…. the infarcts we could see developing were not extensive."

68. He relied upon this in seeking to resist the Defendants' suggestion that the deterioration suffered at 04.30 and the Claimant's resulting injury were severe, when the conclusions in his report were clearly based on his view that the deterioration at this time had been serious and had led to locked-in syndrome by the time of his admission to hospital.

69. There was an obvious inconsistency between his evidence at the hearing, that the Claimant was in a stable, neurological condition from 04.30 until his sudden, step-wise deterioration in the early hours of the 17 March, and the description in his report, that just two hours after the Claimant's admission to hospital, the scan showed devastating damage. I accept that, at the time he wrote this report and indeed some of his subsequent reports or letters, he may not have had a clear picture of the Claimant's progress, and that his views had evolved over time, as he explained. It was, however, unfortunate that he did not revisit his report and clarify the position before the hearing, in particular in a complex case where he would be aware that his opinion as to causation was the subject of challenge, and that clarity and consistency would be of particular importance in evaluating his evidence.

70. The Defendants make further criticisms in this respect, in relation to the views Professor Brown now advances as to the cause of the Claimant's stroke and the imperative of immediate treatment with aspirin, neither of which featured in his causation report. That they are absent from this report is correct, but his views on these matters became perfectly clear during the subsequent joint meetings and reports. These criticisms are, in my view, unfounded.

71. As to the cause of the stroke, I reject without hesitation the suggestion that embolism has been advanced as the cause, late in the day, to lend support to his reliance on recent research by Professor Rothwell into the effects of aspirin (to which I shall refer later on). Before he set out his detailed conclusions as to the cause, answering the Defendants' questions (23 February 2015), the tendency of basilar artery thrombosis to show step-wise deterioration had already been referred to by Professor Brown at the first joint experts' meeting in October 2014; and the recurrence of stroke was addressed in his supplementary causation report dated 28 January 2015. For the reasons I have already set out above, I accept his analysis as to the probable cause of the Claimant's stroke on the totality of the evidence. Ultimately, Dr Giallombardo did not disagree with his analysis.

72. In relation to heparin, I accept Professor Brown's evidence that he referred only to heparin, and not aspirin, in his original report because in 2010 neurologists tended to prefer anti-thrombotic medication in the form of heparin for patients with basilar artery thrombosis. However, at the first joint meeting in October 2014, he agreed with Dr Giallombardo that, once haemorrhage had been excluded by the CT scan, either aspirin or heparin could have been given; that either were acceptable alternatives; and that the conclusions in his report were based on treatment with either medication. His position was clear at that stage.

73. In his witness statement Dr Jarrett, consultant physician at Queen Alexandra Hospital, states that aspirin would have been the main treatment in the Claimant's case. And

the 2008 NICE Guidelines recommended aspirin for people suffering acute ischaemic stroke. Professor Brown readily accepted that, for arterial disease, heparin and aspirin are now thought to have similar effects and with similar speeds. There is therefore no difficulty or inconsistency caused by his failure to refer to aspirin specifically in his original report, or with the arguments he now advances as to the importance of aspirin and its likely benefits in this case.

The relevance of the lumbar puncture

74. As shown by the extracts from his report set out above, it was Professor Brown's clear view at that stage that the lumbar puncture performed at 22.30 was causative, at least in part, of the Claimant's subsequent deterioration and of additional, unnecessary brain damage. He stated, at paragraph 7.8, that lumbar puncture is contraindicated in patients with basilar artery thrombosis "…because of the risk that the reduction in cerebrospinal fluid pressure resulting from the lumbar puncture will cause coning in the presence of stroke."

75. Dr Giallombardo considers there is no evidence that the lumbar puncture contributed in any way to the final outcome. In cross-examination (it was not addressed in his evidence-in-chief) Professor Brown did not resile from his view as to the relevance of the lumbar puncture, but it is fair to say that he advanced it with less enthusiasm. He described it as "conceivable" that fluid had continued to leak out of the theca after the procedure, lowering the pressure and possibly "sucking down" the already swollen brain, causing a shift in the intracranial contents and compressing the brain stem.

76. Dr Giallombardo was unfamiliar with this theory and Professor Brown accepted that there was no evidence of this in fact having occurred on any of the imaging. The neuroradiologists agreed that the MRI scan on 19 March showed no evidence of significant posterior fossa swelling or coning. While Professor Brown suggested that this could be explained by the theca having sealed up again by the time of the MRI, he accepted that any contribution to the Claimant's deterioration on 17 March was a possibility rather than a probability. There is, however, no evidence at all in this case of hydrocephalus, or of significant swelling of the cerebellum, or that the basilar artery was compressed by any swelling of the brain stem.

77. At the joint experts' meeting in October 2014 Professor Brown accepted that the Claimant's clinical progression could simply have been caused by the thrombosis, unrelated to the lumbar puncture. He accepted in his evidence that it was more likely that the Claimant's deterioration was caused by another blood clot and that overall, on the balance of probabilities, there was more evidence that the cause of the deterioration could just as well have been a thrombosis. I find on all the evidence that the lumbar puncture had no effect on the Claimant's condition, or on his deterioration in the early hours of 17 March.

Causation: the competing expert opinions

78. Essentially, Professor Brown's analysis and opinion is as follows. In assessing its benefits, the giving of aspirin cannot be seen in isolation. It forms an important part of the appropriate and necessary treatment for a patient who has suffered an ischaemic stroke, including basilar artery occlusion. Aspirin will work only in an environment where the patient is stabilised and well-positioned, and where he is properly hydrated

and there is no hypoxia. These are all cumulative benefits. For aspirin to work the patient should therefore be in optimum condition, as the Claimant should and would have been if he had been admitted to hospital by 04.00 on 16 March and treated appropriately.

79. The taking of all these steps in specialist stroke units have, in general terms, resulted in a fall in the stroke mortality rate and in improved outcomes, which are now well understood. Recent research, in particular the findings in BASICS, provides good evidence that, for patients in optimum condition, aspirin is more effective than was previously thought.

80. In this case, based both on his experience and the BASICS findings and other research he referred to in evidence, it is his opinion that, if the Claimant had been in optimum condition and had been given aspirin from about 06.00 on 16 March, he would not have deteriorated in the early hours of the following day. On the balance of probabilities he would not have suffered a recurrent stroke and would not have developed locked-in syndrome. He would have been left with moderate or moderately severe disability, but his level of disability would have improved with continued rehabilitation and on balance he would have been able to return to work with appropriate aids or support.

81. Dr Giallombardo agrees that there is now compelling evidence that organised stroke care can, in general, reduce mortality and severe disability. He also accepts that early use of aspirin can, in general, increase a patient's chances of surviving a stroke with better functional outcome. He agrees that this Claimant should have had both aspirin and appropriate stroke care. However, he disagrees that BASICS and the other research referred to in evidence can assist the Claimant in this case. His view, based in particular on two randomised controlled trials in 1997 and his own clinical experience, is that it cannot be said to be more likely than not that, in the particular circumstances of this case, the Claimant would have avoided the deterioration that occurred. There is, in his view, a greater than fifty per cent chance that good stroke care and the early use of aspirin would not have altered the actual outcome in this case.

The Literature

82. The way in which the various studies referred to emerged in this case is relevant in evaluating these competing expert opinions. None of them directly addresses the specific causation issue arising in this case. As in all cases where statistical and epidemiological evidence is referred to, its significance and its assistance will depend on the nature of that evidence and of the particular facts of the case (**Sienkiewicz v Greif [2011] UKSC 10**).

83. In his causation report of May 2014, in stating his opinion as to whether earlier treatment would have been beneficial for the Claimant, Professor Brown referred only to BASICS, a large study published in the Lancet and entitled: "Treatment and outcomes of acute basilar artery occlusion in the Basilar Artery International Cooperation Study (BASICS): a prospective registry study", *Wouter J Schonewille and others on behalf of the BASICS study group* (The Lancet, neurology, Vol 8 August 2009.) Professor Brown relied heavily upon this study in advancing the contentions in his report, upon which the pleaded case on causation was based, and he has continued to

rely upon it in his subsequent reports and joint statements, and in evidence at this hearing.

84. As its title indicates, BASICS was a "*prospective, observational international registry of consecutive patients aged 18 years or older who presented with an acute symptomatic and radiologically confirmed BAO* [basilar artery occlusion]" between 1 November 1 2002, and 1 October 2007. The authors referred to the almost 80 per cent rate of death or disability associated with BAO, despite the recent advances in the treatment of acute stroke, as I have noted earlier in this judgment. They also stated that BAO had not been studied in isolation in randomised clinical trials because of its low incidence, noting that "*….only about 5 per cent of all patients given thrombolysis for stroke have BAO*."

85. The primary aim of the study was expressed as being "*....to obtain a better understanding of outcomes after acute BAO and to study potential differences in treatment response in anticipation of a definitive randomised controlled trial of acute treatment in these patients.*" This was not therefore a randomised, controlled trial and the authors recognised that, as such, it had "*....all the limitations of a non-randomised study"* and that "*....data collection in a registry is generally not as accurate as it is in a randomised treatment trial.*" The study compared the efficacy of three different types of treatment, intra-venous thrombolysis (IVT), intra-arterial thrombolysis (IAT) and antithrombotic treatment (heparin or, in most cases, aspirin) relative to each other. It did not compare or purport to compare the efficacy of one or more of these treatments with no treatment at all. There was no "placebo" group.

86. The results suggested that "*… there is a difference in the efficacy of treatment strategies in patients with an acute BAO, depending on the severity of the stroke.*" Most patients in the study received IAT but, perhaps surprisingly for those involved in the research, the results did not support unequivocal superiority of IAT over IVT. The conclusion was that "*….the efficacy of IAT versus IVT in patients with an acute BAO needs to be assessed in a randomised controlled trial.*"

87. For the purposes of the study, stroke severity was categorised as "severe", which was defined as "*coma, locked-in state, or tetraplegia*" or "mild-to-moderate", namely "*any deficit that was less than severe."* Clinical outcomes were determined in accordance with the modified Rankin Scale (mRS), the scores on that scale being commonly used in the weeks that follow a stroke to measure the degree of disability or dependence of stroke patients. Each score reflects a different outcome, as follows:

0 – No symptoms at all

1 – No significant disability: able to carry out all usual duties and activities despite some symptoms

2 – Slight disability: unable to carry out all previous activities but able to look after own affairs without assistance

3 – Moderate disability: requiring some help, but able to walk without assistance

4 – Moderately severe disability: unable to walk without assistance, and unable to attend to own bodily needs without assistance

5 – Severe disability: bedridden, incontinent and requiring constant nursing care and attention

6 – Patients who do not survive.

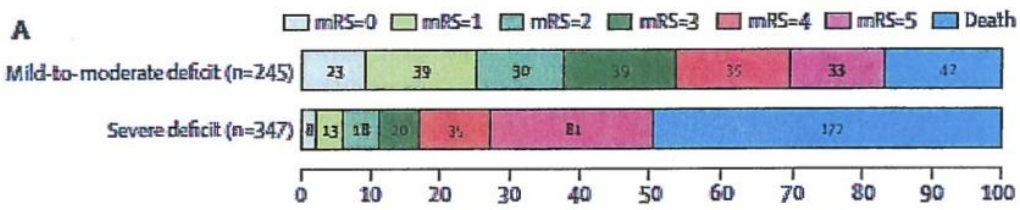
88. There is no dispute that the Claimant is at point 5 on that scale and has remained at that point since March 2010. The debate in this case has been whether, on the balance of probabilities, he would have been at a lower point if treated appropriately and given aspirin from 06.00 on 16 March and, if so, at which point.

89. It is common ground that the findings of BASICS indicated that the outcomes for patients were similar, whichever treatment was given. Professor Brown expressed the view in his report that, if admitted after the second 999 call (ie as now agreed, at 04.00), the Claimant would at that stage have been in the "mild-to-moderate" category. Dr Giallombardo has agreed with that view since that category, by definition, includes any patient who was not in a coma, locked-in or tetraplegic.

90. Professor Brown's opinion, in these circumstances was expressed as follows:

> "Overall, the outcome of the condition was poor. However, if the patients were treated when they only had mild-to-moderate stroke, then 114 out of 245 patients (47%) were left dead or very disabled (score 4 or greater on the modified Rankin score when measured at one month after admission…The remaining 53% had less degrees of disability, although few made a full recovery….On the other hand, of patients not treated until they had severe deficit, 288/347 (83%) were left dead or very disabled with a Rankin of 4 or more."

The percentage figures referred to were obtained from Figure A in the article, representing outcomes at one month according to the severity of the deficit at the time of treatment, which was reproduced in his report and is as set out below:



His evidence is that the finding shown by this bar chart was that more than half the patients who were started on treatment at a time when they had mild to moderate deficit, as in the case of this Claimant, had a modified Rankin score of 3 or better. Thus he states that 70 per cent of patients did better than mRS 5, whatever treatment was given.

91. Professor Brown regards these findings as consistent with his views as to the benefits of early assessment and treatment, including treatment with antithrombotic agents halting the spread (or recurrence) of the thrombosis. He considers it likely that the Claimant would have been dehydrated or volume depleted when he arrived in hospital due to vomiting repeatedly, which increases the viscosity of the blood, promoting

thrombus formation. Intravenous fluid to correct this, if he had been admitted at 04.00, would also have reduced the risks and improved his outcome.

92. He acknowledged in his letter of 23 February 2015 (paragraph 3.1.5.2) that the best chance of benefit from aspirin would have been if the Claimant had been started on aspirin before his collapse at 04.30, but he nevertheless considers it likely that the Claimant would have benefitted from aspirin if it had been started after the scan, at about 06.00 as now agreed. On the balance of probabilities he considers that, if the Claimant had been admitted after the second 999 call, promptly diagnosed and treated appropriately, he would have avoided being locked-in and would have been left with an mRS score of at least 3 at one month after onset, 3 being taken by him as the midpoint of the scores of the BASICS patients started on treatment when they had mild-to-moderate stroke.

93. Since the majority of patients improve with rehabilitation beyond one month after admission, he considers that the Claimant's final condition would have improved further, although he would have remained disabled. Professor Brown then described what, in his view, was the likely level of that disability and what the Claimant would have been able to achieve, which it is unnecessary for me to refer to here, save to emphasise that his views are very much in dispute in this case.

94. In his report of 27 May 2014, and in subsequent discussions and reports Dr Giallombardo considered that BASICS, as an observational study, did not assist the Claimant. That remains his view. While it showed, as might be expected, that outcomes tended to be better in patients with less severe symptoms at the time of presentation, whatever the treatment, BASICS was not designed to study the efficacy of different forms of treatment as against no treatment at all, or as against delayed treatment. Further, as the authors stated, "*The time to treatment in patients treated only with AT* [ie aspirin or heparin] *was not recorded accurately."* There was therefore no reliable information on the timing to treatment for patients who were given aspirin. The findings suggested to him that the various treatments had a relatively minor impact on the natural course of BAO; and there was insufficient evidence to demonstrate the superiority of one treatment over another.

95. Dr Giallombardo relied, in his report and in evidence, upon the findings of two major, randomised controlled stroke trials carried out in 1997 which, enrolling around 40,000 patients in total, tested the effectiveness of early use of treatment with aspirin versus no aspirin treatment (the placebo group) in cases of acute ischaemic stroke. Designed to provide reliable evidence, for the first time, about the effects of early aspirin treatment in such cases, these large trials were the Chinese Stroke Trial (CAST), a "*randomised placebo-controlled trial of early aspirin use in 20,000 patients with acute ischaemic stroke"* (Lancet 1997; 349: 1641-49*);* and the International Stroke Trial (IST), a "*randomised trial of aspirin, subcutaneous heparin, both, or neither among 19,435 patients with acute ischaemic stroke"* (Lancet 1997;349:1569-81).

96. Neither of these trials was referred to by Professor Brown in his causation report, and the Defendants criticise his failure even to refer to trials which were clearly relevant and which, it is said, still remain the best available evidence as to the efficacy of aspirin in preventing deterioration after major ischaemic stroke, as measured against no aspirin therapy. Dr Giallombardo considers that they remain the most

representative studies of the effect of aspirin and points out that they have been recently endorsed by the Cochrane Collaboration review published in 2014.

97. It is common ground that the results of the CAST and IST trials showed that, overall, the beneficial effects of aspirin were modest and increased the chances of surviving with a better functional outcome by only a limited extent. In his report Dr Giallombardo, proceeding on the basis that one hundred patients needed to be treated for one of them to benefit (there was shown to be a real reduction of about 10 deaths or recurrent strokes per 1000 patients), considered both trials to show that early use of aspirin prevented death or stroke recurrence by only 1 per cent, when tested against the placebo group.

98. He increased this Claimant's chances of a better outcome to 10 per cent in his report, on the basis only that aspirin intervention in patients with evolving symptoms and optimal management "…could conceivably be more efficacious." He frankly admitted in the witness box that the 10 per cent figure referred to had no scientific or statistical basis and was effectively no more than "a hunch". However, whatever the relevant percentage chance of a better outcome, he remains of the view that, even if given aspirin soon after the CT scan, the outcome for this Claimant would not have changed.

99. Both experts have agreed, as recorded in their last joint statement, that the combination of aspirin therapy and good stroke treatment started after 04.30 would have "…improved the chances of the claimant avoiding the deterioration on 17 March." The question for me to determine is whether the Claimant has demonstrated on all the evidence that it is more likely than not that he would have avoided that deterioration. If that were to be my conclusion, they are agreed that, on the balance of probabilities, the Claimant would have had a modified Rankin score of 3 or 4 at one month after onset; and that with continued rehabilitation for up to 12 months his condition would have improved further by at least one point on the scale.

100. On all the evidence I find that, if the Claimant had not suddenly deteriorated on the 17 March, the stroke that he had already suffered, involving an undoubtedly serious collapse at 04.30, would have left him with a modified Rankin score of 3, even after continued rehabilitation. That, in my judgment, would have been the most likely outcome for this Claimant on the totality of the evidence. There remains a dispute between the experts as to the nature and extent of that improvement, in terms of his employability, but it is not necessary for me to address that here in view of my conclusion on the main causation dispute.

Discussion and Conclusion

101. Deciding what, if any conclusions can properly be drawn from the studies referred to above in the circumstances of this Claimant's case is no easy task. There is no doubt that basilar artery thrombosis is a rare condition, and that there are therefore only relatively low numbers of patients with that particular condition within the category of ischaemic strokes. In the CAST and IST studies, posterior artery stroke (of which basilar artery thrombosis would form part) accounted for only 1 per cent and 12 per cent, respectively, of patients treated with aspirin.

102. Since these randomised studies demonstrated that aspirin was of some benefit to stroke patients generally, and the findings led to aspirin being given as routine, studies of aspirin therapy versus placebo could not now be repeated. There has not therefore been, nor can there be any randomised controlled trial showing how patients with basilar artery thrombosis would react. BASICS was an observational study and it is the only observational study undertaken so far for such patients. It was not however concerned with the efficacy of aspirin versus no aspirin.

103. Further, it is agreed that, in general terms, recurrent stroke is rare. The incidence of recurrent stroke in CAST and IST was about 4 per cent and 2-3 per cent respectively. The particular features of basilar artery thrombosis, in particular the tendency for step-wise deterioration, would suggest that the incidence of recurrent stroke is likely to be higher in such cases and, as I have stated earlier, it is accepted to have a worse prognosis than for other types of stroke.

104. However, as Sir Robert Francis QC points out, BASICS does not relate the outcomes for basilar artery thrombosis to their causes. Of the 183 patients given aspirin, the thrombosis was caused by embolism in 55 cases and by other factors (eg atherosclerosis or dissection) or by cause unknown in the other 128. We have only the outcomes for patients studied after one month of treatment, with no information as to the cause of the thrombosis, or as to how many or which patients suffered a recurrence. There is therefore no information as to how many patients suffered a single stroke which had a poor outcome, or had step-wise deterioration, or clot propagation with gradual deterioration, or as to the effects of the various treatments broken down in relation to these different circumstances, or even in relation to age, gender or the other factors referred to in the baseline characteristics listed in Table 1 in the study. Of the 55 patients with an embolic cause there is no information as to whether they had had milder symptoms to start with.

105. The BASICS findings are nevertheless relevant in this case. While it was not a controlled study, I accept that the court should look at all the available evidence which bears on the question to be determined. However, the CAST and IST trials are clearly relevant on this basis in addition. Professor Brown's explanation for failing to refer to those studies at all in his causation report is that he did not consider them to be relevant to the Claimant's case. This was because at the time they were carried out stroke management was generally poor; the number of cases of basilar artery thrombosis included would have been very low; and the incidence of deterioration or recurrent stroke was low.

106. While I accept that these would constitute reasons for suggesting that these trials provide little or no assistance in this case, they cannot reasonably be regarded as having no relevance. They are the only randomised controlled trials comparing aspirin therapy with a placebo group and they provide information as to the percentage contribution made by aspirin in preventing recurrent stroke in some circumstances over a period of time. As an investigator himself in the IST trial, Professor Brown's failure to refer to these studies, even if only to discount their validity, is even more inexplicable.

107. Further, the studies were reviewed as recently as 2014 by the Cochrane Collaboration (*Oral antiplatelet therapy for acute ischaemic stroke (Review))* and were at that stage, in my view, still being regarded as methodologically sound and as having continuing

relevance, the bulk of the data referred to coming from those two studies. As Professor Brown accepted, the Cochrane reviews are widely regarded as the gold standard in the assessment of randomised controlled trials and there is nothing in the review to suggest that these studies are regarded as no longer relevant. Notwithstanding the advances in stroke treatment since these studies were done, Dr Giallombardo considers that, were they to be repeated today, similar results would probably be obtained, so far as the effectiveness of aspirin is concerned.

108. In circumstances where these studies do not assist the arguments that Professor Brown advances, their omission and his explanation for that omission is unsatisfactory. I do not accept the Defendants' submission that Professor Brown has gone beyond giving an objective expert opinion in this case and has become an advocate for the claim. But I do consider that their omission and his reference only to the BASICS findings as "the best evidence available concerning the effectiveness of aspirin therapy in basilar artery thrombosis" was influenced by the Professor's own, strongly held personal views as to the general potency of aspirin for patients with ischaemic stroke.

109. That would not, in itself, undermine the validity of his views if they were supported by the underlying data and by other evidence in the case, as Mr Kent QC contends. And Dr Giallombardo was also the subject of criticism in this case.

110. In opening the case for the Claimant, Mr Kent was critical of the approach adopted by Dr Giallombardo to the interpretation of evidence from the CAST and IST randomised controlled trials, having regard to the necessary civil standard of proof. It is correct that, in his report and in the last joint statement signed on 28 March 2015, Dr Giallombardo's view was that the appropriate statistic to apply for that interpretation was the absolute risk reduction of adverse outcome. That, as he had stated, was about 1 per cent for aspirin. He disagreed with Professor Brown's view that the appropriate statistic to use in this case was the relative risk reduction in the rate of recurrence or worsening of stroke during the Claimant's time in hospital.

111. This criticism, indicating his unfamiliarity and discomfort with the correct legal test, was justified on the basis of his written opinion, maintained until the start of this trial. However, at the very start of his evidence in chief, Dr Giallombardo frankly and fairly acknowledged what he described as an 'error of judgment' in this respect. Having heard counsel's opening submissions and having considered the matter further, he now accepted that the relative risk reduction was the appropriate statistic to apply in this case.

112. That concession was properly made and it was consistent with what I found overall to be a careful approach to this case by this expert. As Professor Brown points out, the Claimant's condition did in fact deteriorate in hospital. Referring to the absolute number of stroke patients that needed to be treated with aspirin for one patient to avoid deterioration does not therefore provide the information required to answer the question whether this Claimant would have avoided his current condition if aspirin had been given to him as from 06.00 on 16 March, because most stroke patients do not deteriorate.

113. Both experts agreed during their evidence that, in the CAST and IST trials, the percentage contribution made by aspirin in preventing recurrent stroke, having regard to the Cochrane review (at page 40), was therefore approximately 23 per cent. There

is no evidence as to what that percentage contribution would be in a case where aspirin was given for some twenty hours following earlier basilar artery thrombosis, which had already caused moderate brain damage. However, even if we were to add a small additional percentage, as Dr Giallombardo did, to reflect the benefits of today's more advanced stroke management in such cases, that extra percentage would not be anywhere near 28 per cent.

114. Dr Giallombardo's view remains that, on the basis of these studies, it could not be said that aspirin would have made a difference. It could not therefore be said, on the balance of probabilities, that this Claimant would have avoided the outcome that in fact occurred. Professor Brown fairly accepted that, on the basis of the results obtained from the CAST and IST studies alone, it could not be said that giving aspirin to patients with basilar artery thrombosis would, on the balance of probabilities, prevent their further deterioration.

115. In paragraph 6 of his supplemental causation report Professor Brown referred, by way of further support for his views, to an analysis, or re-analysis of randomised trials, recently carried out by Professor Peter Rothwell. These were said to be trials which studied the effect of aspirin versus placebo in patients who had suffered a transient ischaemic attack (TIA) or minor stroke (that is, a stroke with symptoms lasting more than 24 hours but from which a good recovery is expected).

116. In a subsequently amended passage in Professor Brown's report, this analysis was said to show that although on average, in all stroke patients combined, aspirin resulted in a reduction of recurrent stroke of about 25 per cent, those patients treated in trials excluding major stroke had more than 50 per cent benefit in the reduction of early recurrence of disabling or fatal stroke compared with those treated with placebo. The Professor relies upon this research as providing "good evidence" that, in the modern context of patients managed hyper-acutely in specialist units, with treatment commenced within a few hours of onset of symptoms, aspirin is likely to be far more beneficial than the average benefit reported in the older CAST and IST trials.

117. In subsequent correspondence Professor Brown also referred to an earlier study in which Professor Rothwell was involved, namely the "Effect of urgent treatment of transient ischaemic attack and minor stroke on early recurrent stroke (EXPRESS study): a prospective population-based sequential comparison" *Peter M Rothwell and others* (Lancet 2007; 370: 1432-42). This was said to show a much lower rate of recurrent stroke within 90 days for patients with TIA or minor stroke, who were not admitted directly to hospital and who had been started on treatment with aspirin immediately, when compared with those for whom treatment was delayed.

118. There are, however, a number of difficulties with both the EXPRESS study (which was not a randomised study and contained very few, if any patients with basilar artery thrombosis) and the recent Rothwell research and Professor Brown's reliance upon it.

119. First, neither the study nor the recent research seems to me to provide assistance in this Claimant's case. It is agreed that the Claimant suffered a major collapse at 04.30, which could not have been prevented, and Professor Brown himself accepted that the best chance of benefit from aspirin would have been if treatment had been started before that time. After 04.30 it is clear that the Claimant could not be said to be suffering symptoms of TIA or minor stroke. None of the patients given aspirin in the

study had any major stroke symptoms at the time their treatment was started. I do not therefore see how this research can help in answering the specific causation issue arising on the facts of this case.

120. Secondly, the recent research relied upon was said to be "data presented at UK Stroke Forum 2014 and personal communication from Peter Rothwell." Only when asked further questions by the Defendants about this data did Professor Brown disclose two slides provided to him by Professor Rothwell, in personal email communications between them following a lecture given by Professor Rothwell at the Forum in December 2014.

121. These slides are said to show, in graph form, the early effects of aspirin in cases of TIA or minor stroke. However, this research is as yet unpublished. It has not been peer reviewed and it was provided to Professor Brown privately. It is doubtful whether Professor Rothwell understood that it was going to be referred to in court and subjected to forensic examination, or relied upon as support for this Claimant's case on causation. I do not for one moment doubt Professor Rothwell's considerable expertise in the area of stroke prevention treatment and trials, but the Defendants and the court are entitled to examine the underlying data before deciding what weight can be placed on the findings and the expert opinions expressed.

122. As it is, there is no underlying data available concerning the numbers of patients who were given aspirin or who were in the placebo group. Nor is there any data as to how many patients in the two groups had recurrence of stroke within 12, 24 or 48 hours. Further, as Professor Brown concedes, the proportion of patients in the early stages of basilar artery thrombosis in this trial would be likely to be around 1 per cent or less. It is also unclear which randomised controlled trials Professor Rothwell had analysed. In the circumstances I accept the Defendants' submission that this is not research upon which the court can properly rely, but in any event its particular focus does not seem to me to assist in determining causation in this case.

123. There is one final, randomised study to which Professor Brown referred, in the course of the expert meetings and in evidence before me, which is said to provide further, recent evidence as to the effectiveness of aspirin in recurrent stroke prevention. This is the CADISS study, "Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial", *The CADISS trial investigators* (Lancet, neurology Vol 14 April 2015).

124. This trial, as is clear from its title, was not concerned with basilar artery thrombosis. In addition, of the 250 patients studied, those with severe symptoms who could not consent were excluded. The presenting signs and symptoms were stroke, TIA and local symptoms (eg headache or neck pain) and the table of baseline characteristics suggests that the patients involved in this study were in a far less serious condition at presentation than this Claimant. In addition, some patients were apparently treated with a combination of antiplatelets, rather than just aspirin. Once again I do not find this study to be of assistance in this case.

125. In the circumstances I do not consider it necessary or helpful to consider these other studies in any further detail in this judgment. In cross-examination Professor Brown accepted that the study he was really relying upon in this case was BASICS, because it described data which he believed was most relevant to the Claimant's condition.

He fairly accepted that, if he were wrong as to the conclusions which could be drawn from BASICS, his arguments in this case were less strong, and that the other studies and statistical evidence did not, on their own, meet the necessary balance of probabilities threshold in answering the causation issue in this case.

126. His approach to BASICS is straightforward, as set out above (paragraphs 89-93). Looking at the bar chart it appears, at first glance, to be correct to say that the majority of patients in the mild to moderate category had a better outcome than an mRS score of 5 and that, statistically, the Claimant's chances of a better outcome were therefore 70 per cent.

127. The problem is that, without a control 'no treatment' group, it is simply not possible to assess what proportion of patients in that category might have had a similar outcome to the Claimant in any event, in particular given the particular features that Professor Brown identified as distinguishing basilar artery stroke from other kinds of stroke. It is a rare condition, and it carries a high risk of mortality and morbidity. The incidence of recurrent stroke generally is low, though it may well be higher in the case of basilar artery strokes. What we do know is that in the Claimant's case his further deterioration occurred rapidly, just less than 24 hours after his earlier collapse at 04.30. This was therefore a rare event in a stroke which accounts for less than 1 per cent of strokes overall.

128. In my judgment BASICS tells us nothing about the extent to which aspirin would be likely to prevent a recurrence of basilar artery stroke after some 20 hours of treatment, in someone who has already suffered a stroke which caused moderate and permanent brain damage. It tells us nothing about whether aspirin would result, for someone in the Claimant's condition after 04.30, in a better outcome than if no treatment were provided. There are too many unknown variables which could affect the end result.

129. It is important to keep in mind the aim of this observational study, namely to compare the efficacy of different types of treatment, relative to each other. In my view the figures reported do not provide a proper basis for proving, to the requisite civil standard, and comparing like with like, that starting this Claimant on aspirin at 06.00 on 16 March would have prevented his further deterioration from mRS level 3 disability in the early hours of the following day. I accept the Defendants' submissions in this respect.

130. We know in this case, as I have found, that after his serious deterioration at 04.30 on 16 March, the best and most likely outcome for this Claimant on the evidence was a modified Rankin score of 3, even after rehabilitation. I agree with Sir Robert Francis that, in those circumstances, it is illogical to include those patients who achieved an mRS score of 0, 1 or 2 in the calculation of the percentage chance of a better outcome for this Claimant. The error in doing so is that you are not then comparing like with like, having regard to what the evidence shows to be established in this case, either as undisputed fact or on the balance of probabilities.

131. As the Defendants point out, if the relevant group for the purposes of the calculation is adjusted to as to include only the percentage number of patients who achieved mRS scores of 3, 4 and 5, (given that the Claimant survived), then 39 out of 107 patients achieved an mRS score of 3 which, expressed in percentage terms, is 36.44 per cent. This demonstrates, in my view, the flawed approach adopted by Professor Brown

towards these BASICS figures and the caution that has to be applied in determining what can properly be drawn from them.

132. There is a further point as to the validity of including those patients who achieved an mRS score of 0 – 2 in calculating the percentage chance of a better outcome, even on the approach adopted by Professor Brown. There was some dispute between the experts in evidence as to where precisely this Claimant would have been placed on the National Institutes of Health Stroke Scale (NIHSS) after his collapse at 04.30. The Scale is a systematic assessment tool that provides a quantitative measure of stroke-related neurologic deficit, now widely used as a clinical assessment tool to evaluate the acuity of stroke patients.

133. There is insufficient evidence, in my view, to ascribe accurately the relevant numerical score to the Claimant, under each head, and assess the total figure. I do not consider it necessary to do so however. Professor Brown fairly accepted that the category of "mild to moderate" patients in BASICS covered an extremely broad range of disability, given that it effectively covered everyone other than those with coma, locked-in syndrome or tetraplegia. Within that range he accepted that after 04.30 the Claimant would have been at the moderate end of the range.

134. On the evidence there is the Claimant plainly had a reduced level of consciousness and was unable to produce speech. Although the extent of the weakness in his limbs cannot be assessed accurately, we know that he was lying on the floor unable to move and unable to communicate. In my judgment the Claimant would properly fall to be placed at the upper, more serious end of that mild to moderate range. For that reason in addition, it was illogical to include those patients who achieved an mRS score of 0 -2 in the percentage calculation for this Claimant.

135. Mr Kent rightly points out that Professor Brown's opinion on causation is not based solely on the BASICS findings and his calculation of the likely effect of aspirin in the Claimant's case. He relies, rather, on his experience and upon the giving of aspirin as part of good stroke management, which this Claimant would have had from the moment he arrived in hospital, and on the cumulative benefits those measures would have provided.

136. The factors emphasised by Professor Brown in this respect were hydration, the avoidance of hypoxia and good positioning, all of which would have been properly monitored and maintained if he had arrived at hospital by 04.00 on 16 March. There is no evidence that poor positioning on its own played any part in this case and in evidence the experts concentrated on dehydration and hypoxia.

137. In relation to hydration I accept Professor Brown's opinion that the Claimant was likely to have been dehydrated to some extent by the time he was admitted to hospital at lunchtime. The evidence shows that he had been vomiting repeatedly from the early hours and, after his collapse at 04.30, he had been lying on the floor and not drinking any more water. Dr Giallombardo accepts that dehydration could increase the chances of a negative outcome.

138. However, there is no evidence that, on his arrival at A&E, the Claimant was significantly dehydrated. Further, the records show that intravenous fluids were prescribed soon after his admission at about 14.00 or 14.50 and, although the time

they were actually given after prescription is unclear, there is no evidence that the Claimant was dehydrated either at or after that time.

139. Dr Giallombardo drew attention to the record of urine output of 700 ml at 14.00, probably on catheterisation, which he regarded as inconsistent with prior dehydration. A further 270 ml of urine was obtained between 14.00 and 17.00. Professor Brown contends that, in assessing the level of the Claimant's dehydration, no reliance can be placed on a single urine output reading of 700 ml; and that the absence of clinical signs of dehydration can be explained by the fact that external signs, such as skin changes, do not show until later on.

140. There is, however, no evidence of any abnormal blood test results and therefore no evidence that his blood viscosity had been affected, such as could increase the risk of clots. And as Dr Giallombardo observed, many people sleep through the night for a long period without any fluid intake and do not become dehydrated, or at any rate not dangerously so. In my view therefore there is no evidence in this case that the level of dehydration that existed before the Claimant's arrival at hospital was sufficient to cause any adverse effect, or that it did in fact have any such effect.

141. Nor is there any evidence that the Claimant was hypoxic on admission. The records show that oxygen saturation was and remained at 100 per cent from 14.50 to 17.30 and, notwithstanding the note made by the paramedic that the Claimant's airway was partially compromised, he took measurements of SPO2 at 99 per cent at 13.30 and 13.40 after oxygen was given at 13.00. There was no evidence of a rise in his CO2 levels when the arterial blood gases were measured on admission, which Dr Giallombardo said you would expect to see if there had been any significant drop in oxygen levels. Nor was there any evidence of respiratory failure due to weakness in the respiratory muscles.

142. Professor Brown points out that the Claimant was heard to be snoring earlier on and I accept that snoring can indicate a compromised airway, in particular in someone with a depressed level of consciousness. However, the experts are agreed in this case that the Claimant remained in a stable neurological condition from the time of his collapse at 04.30 until his deterioration in the early hours of 17 March, which indicates in my view that he suffered no significant hypoxia or hypoxic brain damage during the period he lay unattended at home.

143. For all these reasons, having considered all the evidence and counsels' submissions with care, I have concluded that the Claimant has not demonstrated, on the balance of probabilities, that if aspirin and good stroke care had been provided to him in hospital, between 06:00 on 16 March and 02:30 on 17 March, he would have avoided the further catastrophic deterioration and locked-in syndrome that occurred.

144. This is, as I recognised at the outset, a tragic case. The Claimant suffered a rare form of stroke, which is acknowledged to be associated with a high rate of death or serious disability. He sustained significant brain damage in the early hours of 16 March. In the context of admitted breaches of duty by both Defendants he then suffered a recurrence in the early hours of the following day while he was in hospital. Locked-in syndrome was, however, the outcome for a large number of those patients in the BASICS mild to moderate category, even though they had the benefit of antithrombotic therapy and good stroke management. On all the evidence I found the

opinion of Dr Giallombardo in this case to be the more persuasive, based as it was on his extensive clinical experience of the management of acute stroke patients. I therefore find in favour of the Defendants on the issue of causation.

145. Accordingly I now invite further submissions from the parties as to the appropriate order and directions required in this case.