Neutral Citation Number: [2015] EWHC 2363 (QB)

Case No: HQ13X01140

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 05/08/2015

Before :

MR JUSTICE STEWART

- -

Between :

(1) Timothy Owers as Administrator of the Estate of Karen Owers Deceased **(2) Timothy Owers (In His Own Right)**	**Claimants**
- and -	
(1) Medway NHS Foundation Trust **(2) Secretary of State for Health (formerly Medway Community Healthcare NHS Trust)**	**Defendants**

- -
- -

Gerwyn Samuel (instructed by **Withy King**) for the **Claimants**
Richard Booth QC (instructed by **Bevan Brittan**) for the **Defendant**

Hearing dates: 15 - 18, & 22 - 24 June 2015 & 7 & 9 July 2015

- -

Approved Judgment

I direct that pursuant to CPR PD 39A para 6.1 no official shorthand note shall be taken of this Judgment and that copies of this version as handed down may be treated as authentic.

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MR JUSTICE STEWART

Mr Justice Stewart :

Overview

1. Mrs Karen Owers was born on 1 October 1967. She died on 27 August 2014. Her estate brings a claim arising out of matters which occurred at Medway Maritime Hospital, Kent on 14 March 2010. The claim is brought for her pain, suffering and loss of amenity and consequential losses between that date and the date of her death. There is no claim that her death resulted from the alleged negligence of the Defendants. Although strictly the estate is the First Claimant I shall refer to Mrs Owers as "C1". C1's husband, Mr Timothy Owers, was born on 3 June 1967. He brings a claim in his own right arising out of psychological injuries said to have been sustained as a result of the Defendants' negligence on 14 March 2010. I shall refer to him as "C2".

2. Medway NHS Foundation Trust is vicariously liable for any negligence on behalf of its staff, except that, in the present case, two members of the stroke team (Caroline Bates – nee Hannon – and Chris Gedge) were employees of the Medway Community Healthcare NHS Trust and therefore the Second Defendants are vicariously liable for any negligence by them. For the purposes of this judgment the First and Second Defendants will be referred to as "Ds", there being generally no reason to distinguish between the two Defendants.

3. This trial follows the order of Master Cook who ordered "A preliminary issue shall be tried between the First Claimant and the Defendants as to whether or not the Defendants are liable to the First Claimant by reason of the matters alleged in the Particulars of Claim and, if so whether or not any of the injuries pleaded were caused thereby; if any such injuries were so caused, the extent of the same. The Second Claimant's secondary victim claim shall be tried at the same time as the preliminary issue."

4. It is sensible to consider C1's claim first since there is a considerable overlap between her claim and the claim of C2.

5. It is agreed that on 14 March 2010 C1 suffered a stroke. It was of a rare type, being an insult to one of the posterior circulatory arteries occurring as a result of Basilar Artery Occlusion (BAO). C1's stroke was as a result of Vertebral Artery Dissection (VAD). VAD is a flap-like tear of the inner lining of the vertebral artery, leading to the escape of blood and clot formation. C1's case is that Ds failed to diagnose and treat her stroke either by way of thrombolysis (clot busting drugs) or, if not a candidate for thrombolysis, by aspirin.

6. Some breaches of duty are admitted, some are denied. The task of the Court in relation to C1's claim is to determine relevant breaches of duty which are in dispute and issues of causation.

7. It may be helpful at this stage to set out the *dramatis personae* in this case apart from C1 and C2

   (i) Non expert witnesses:

Caroline Bates (nee Hannon), nurse member of the stroke team

Dr Ramphele, Senior House Officer in the Accident and Emergency Department

Christopher Gedge, physiotherapist and advanced stroke practitioner

Dr Mamun, Consultant Stroke Physician

(ii) Expert witnesses who gave oral evidence

Dr Paul Baines, Consultant in Accident and Emergency (for Cs)

Dr Steven Allder, Consultant Neurologist (for Cs)

Dr Gregor Campbell-Hewson, Consultant in Accident and Emergency (for Ds)

Professor Adrian Wills, Consultant Neurologist (for Ds)

(iii) Expert witnesses who did not give oral evidence

Professor Philip White (Cs) and Dr Marcus Likeman (Ds) Consultants in Neuroradiology).

Dr Mark Tattersall (C2) and Dr Niall Campbell (Ds) – Consultant Psychiatrists.

8. As part of setting the scene it should be noted that there are two main chemical treatments of acute stroke. These are:-

(1) Thrombolysis – this treatment chemically breaks up a blood clot so as to reconstitute normal blood flow back to brain tissues.

(2) Antiplatelets and Anticoagulants – the two referred to are heparin and aspirin. It is not C1's case that heparin should have been prescribed at Medway. It is her case that aspirin should have been prescribed there. This is conceded by the Defendants though there is a dispute as to the time by which it should have been prescribed. Aspirin does not break up a blood clot. Its effect (if it works) is to stop a blood clot getting bigger and it reduces the tendency of a clot to break up and send part of the clot further along the circulatory system, thereby potentially causing serious harm.

9. I attach to this judgment, as Appendix A, material sections of NICE clinical guidelines: Stroke, Diagnosis and initial management of acute stroke and transient ischaemic attack (TIA). Issued July 2008 (the NICE Guidance).

The Period up to C1's Arrival at Medway Hospital

10. This period, and in particular the period when C1 woke up at or just before 7am on Sunday, 14 March 2010 is of significance. This is because the onset of stroke symptoms is crucial for determining whether or not a patient can be prescribed thrombolysis. In 2010 the licence for the only drug in the market (Alteplase) specified that it should be given within 3 hours of onset of stroke symptoms. At the time there was also a trial at King's College Hospital London (KCH). KCH was the

regional centre and centre for excellence to which Ds referred difficult cases. The trial was for prescribing thrombolysis within an extended period of 6 hours of the onset of symptoms. Generally, Ds would have worked to the 3 hour time frame, on the basis that the knowledge at the time was that the increased risks of haemorrhage associated with thrombolysis after 3 hours outweighed the benefits. This is subject to the fact that Dr Mamun, the stroke physician at Medway, said that in 2010, in appropriate cases, he would prescribe thrombolysis in a young woman up to 4 - 4½ hours after onset of symptoms.

11. To return to the chronology, C1 and C2 (and their son Jake aged 9 at the time) visited C2's parents who live in Hoo, Kent. They drove from their home in Aston in Oxfordshire. C1's statement, dated 09.05.2014, admitted under the provisions of the Civil Evidence Act 1995, continues:

> "3… on the way there I had a bit of a headache. I do get migraines occasionally, usually about 3 or 4 times a year, if that. I have migraine tablets and when I feel the migraine coming on I take a tablet and this usually "nips it in the bud". I get a warning of flashing lights in the corner of my vision and a stabbing pain in my right temple. This headache was definitely not a migraine.
>
> 4. When we got to my parents in law I went upstairs for a sleep in the afternoon. I woke up later and although the headache was still there it was much better. I had a meal with the rest of the family in the evening, took some painkillers (panadol as I recall) and then went off to bed. At 5.50 I woke up. I do not recall what it was exactly that woke me up but when I woke I had a funny feeling in my eyes; they were itchy and painful. I had never had this before. I got up to the toilet. I have been asked if I had any problems getting up but my recollection is I got up normally. The only thing was the funny itchy eyes. I went back to bed and must have dozed off as I next woke at my usual time of waking at 07.00.
>
> 5. Tim was still asleep. I got out of bed and immediately felt a little bit "strange". The only way I can describe it is like being a little bit drunk as if I had a bit of a hangover. I managed to get dressed. I found I was doing things a bit slower than normal but otherwise I had no problems getting dressed. I picked up my book in my right hand and started to go down the stairs. About halfway down the stairs I suddenly started to feel dizzy and sat down on the stairs where I started to feel a little bit better so I got up again and carried on walking down the stairs. When I got to the bottom of the stairs I just did not feel right and I dropped my book on the floor. My father in law must have heard this because he came over and helped me to sit down. My father in law made me a cup of coffee and as I was starting to drink it I felt my right arm getting weaker and it just seemed to be becoming more and more of an effort to lift my arm. I would describe it as being heavy. I also remember it getting harder

> and harder to talk and get my words out and I became very frightened.
>
> 6. I knew that I was having a stroke. In any event my father in law, Harry, called NHS Direct and they told him to call an ambulance. He then told Jake, my son, to go and wake Tim up.
>
> 7. After a very short period of time a Rapid Response person arrived at the house and then the ambulance crew appeared shortly after. I have a very clear memory of these early events and the time spent in Medway Hospital, but I am not so clear about events later on from the time I went to Darent Valley Hospital and Kings College Hospital, London.
>
> 8. I went in the ambulance to Medway Hospital and Tim and his dad followed behind in his dad's car. I have seen the ambulance record obtained by my solicitors and know that the ambulance arrived at the hospital at 07.59. I was taken into a cubicle in Accident and Emergency and by the time we arrived my speech had got worse. Tim arrived shortly afterwards and stayed with me the whole time."

12. The two main issues arising out of this history are:

    (i) Has C1 proven on the balance of probabilities that she is accurate and reliable in relating what happened at 05:50 hours?

    (ii) Was C1 experiencing symptoms on waking up at about 7am or only after she got out of bed?

    It is common ground that the "funny feeling" in the eyes described at 05:50 hours is irrelevant to the issues in the case. C1 submits that, based on her witness statement, the relevant neurological symptoms first occurred shortly after waking up. The Ds cast doubt on the 05:50am incident and ask the Court to find on the balance of probabilities that C1 awoke suffering from stroke symptoms and, given that these were of uncertain time of onset, and may well have been present for more than 3 (or 4½) hours at or shortly after C1's arrival at hospital, she was excluded from receiving thrombolysis treatment. This factual evidence as to the onset of symptoms is therefore the first matter which the Court has to determine.

The Onset of Stroke Symptoms: The Evidence

13. C2 cannot really assist on this point since, on the Sunday morning, he was awakened at about 7am by Jake coming to the bedroom. He cannot therefore say what if anything happened at 05:50 hours or describe C1's state immediately upon waking, some minutes prior to him being awakened. However he does comment as to what happened subsequently at the hospital. These comments are relevant to the reliability of records.

14. Before turning to the oral evidence, it is important to set out a number of the medical records relevant to this point.

15. The ambulance crew arrived at 07:24. The presenting complaint was ? stroke – TIA as C1 had right-sided weakness. Her pupils were equal and reacting. On examination she had weakness on the right-side with slurred speech. The ambulance notes continue:

> "Pt woke 7am. ® sided weakness. ® sided facial droop. ?TIA. Speech problems.
>
> - had headache yesterday – worse than normal – did not clear with tablets
>
> * Hx – Pt collapsed 4 weeks ago while shopping after sudden episode of concussion. Investigated @ John Radcliffe Hospital (undiagnosed) admitted 3 days
>
> Pt sustained head injury from this previous episode …"

16. A care continuum document timed at 08:10 assesses C1 as "mobile, independent …headache/heavy on limbs…"

17. The next note which relates to the onset of symptoms is timed at 08:16 where at Triage, Nurse Farrall wrote "pt woke this morning feeling unwell and wobberly (*sic*). pt has slurring of speech and ® sided weakness".

18. At 08:20 hours Nurse Hannon, now Nurse Bates, the Designated Stroke Specialist, recorded:

> "Stroke Services
>
> HPC. Last night was feeling unwell. Felt sick with a headache. Took painkillers which helped. Patient went to bed and awoke with right-sided weakness* and ? slurred speech.
>
> O/E. Patient's speech very slow and deliberate with mild dysarthria. C/O headache like tight band around head. Patient able to push and pull and raise arms – when arm falls she is able to lift back up again with effort.
>
> Patient suffers with migraine – no previous episodes of aura. Also seizure 15 years ago – nil since. Also a fall and head injury 4 weeks ago – investigated in Oxford Hospital.
>
> Plan "Neurology non suggestive of CVA – requested to hand back to A+E for further Assessment at 08:30 hours."

(* See later)

19. C1 was then handed back to the A & E team by the Stroke Team and she saw Dr Ramphele, Locum SHO, who recorded in the notes at 08:36:

> "PC: Slurred speech, ® sided weakness. ? stroke.

> HPC: Woke up this morning with slurred speech and ® sided weakness.
>
> No hx of CVA. No hypertension.
>
> Or Cardiac disease
>
> Head injury – 4 weeks ago, after the patient collapsed.
>
> Investigated, no cause found…."

(Dr Ramphele examined but, for the present purposes, that is the relevant part of the note).

20. In terms of evidential value as to the onset of symptoms it is important to look to some other entries in hospitals to which C1 went on the Sunday afternoon.

21. Darent Valley Hospital

14.01 "Onset of Rt sided weakness and slurred speech at 7.00am, ?CVA, pt brought to Resus, seen immediately by A/E doctor"

14.25 "HPC patient presented with right sided weakness, started about 7.00am this morning and was seen at Medway Hospital and sent home after assessment by the stroke team…"

22. (i) KCH triage at 16.00 "…sudden onset ® sided weakness/slurred speech 0700 at home (found by husband)…"

(ii) Thrombolysis Pathway: summary. "Date and Time of onset 14/3/10 0700"

(iii) History taken by Neurologists, Dr Bercocal/Dr Haider (Specialist Registrar)

> "History
>
> Yesterday she had headache. This morning she woke up at 7.00 and since this moment she felt light weakness on her right arm, right leg and problems with her speech. No headache different from other days. Since this morning symptoms ~~are~~ have been worsening up to present situation…"

23. I shall turn in a moment to the Ds' evidence. Before that I will set out the context of how C1's case is put. It is this:

(i) The Court should accept on the balance of probabilities C1's evidence as to what happened at 05.50 hours and that her relevant symptoms began after she woke up and not upon waking up.

(ii) Members of the dedicated stroke team ought to have elicited this history on detailed questioning of C1. She should have been told why it was important to ascertain the last time she was free of neurological symptoms. C1 accepts that

the A & E team (Triage Nurses/SHO) would not have needed to go into so much detail when taking a history.

(iii) The criticism of the fact that C1 did not mention the 05.50 incident at any stage to the doctors in the hospital is misplaced because C1 would not have appreciated the importance of being symptom free at this stage. (It was in fact only upon questioning by the solicitor for the purpose of taking her witness statement that this matter came to light). It is further said that if C1 was to have invented the 05.50 incident then it would have been more likely she would have invented a later time of, say, 6.30am, thereby improving the prospects of success for her case by identifying a later time for the onset of symptoms.

(iv) It is said that the gist of the entries at Darent Valley Hospital and KCH is indicative of sudden onset of symptoms after waking up and not upon waking up.

(v) Had she woken up with symptoms of any significance she would have awakened her husband who was sleeping with her.

24. A preliminary matter needs to be determined based on the Defendants' evidence. That is in relation to the asterisked word in the above extract from Nurse Bates' entry at 8:20 hours. This had been interpreted as "weakness" until the outset of the trial when Mr Samuel suggested it may be "heaviness". At first Nurse Bates insisted it was weakness, partly because of reading her own writing and partly because she said that the word heaviness was not terminology she would use. It was pointed out that (i) she would be recording a word the patient used; (ii) the care continuum note at 08:10 records "heavy on limbs"; (iii) there appears to be an "i" in the word because of the dot on the line above. Further similarities in other letters of Nurse Bates' handwriting were pointed out to her and she accepted that quite possibly it did say "heaviness". I find on the balance of probabilities that, because of the reasons relied upon, the probability is that the word is "heaviness". That said, the expert evidence was that in terms of whether there was a focal neurological deficit, any distinction between heaviness and weakness was not material.

25. Nurse Bates initially denied that her history taking could have been better, though she later accepted that her note taking should have reflected the fact that she elicited information which was not out of the ordinary (which she did not note) and only noted information which was out of the ordinary[1]. As regards C1's evidence that she got up at 05:50, she said that she would have asked the question whether C1 got up in the night and the response was no. Had C1 told her that she got up in the night and felt okay, then she would have recorded this. Similarly, on or after awakening, she would have probed. She would have asked to whom C1 was speaking when she noted slurred speech, but, even if C1 had not spoken to anybody until some time after she got up then the onset of slurred speech would have been unknown. She said she could only record what she was being told. C1 was not, not co-operating. Nurse Bates did not tell C1 why she needed an accurate time of onset.

[1] [Professor Wills said in chief that he thought Nurse Bates' history taking was very reasonable. In cross-examination he also said that it was not below standard not to record positive and negatives. However he later conceded that on an initial history taking in a suspected stroke the position was different]

26. I appreciate that Nurse Bates said she recorded only that which was abnormal. On the balance of probabilities I do not accept that she specifically asked C1 whether she had got up in the night. The first time this became apparent was in cross-examination. I consider that she knows that she should have asked that question and in her own mind believes she did do. However she is mistaken in my judgment.

27. What about the history on/after C1 awakened? Again the note taking of Nurse Bates is unfortunately below standard. She says that she would have asked to whom C1 was speaking when she noticed slurred speech but there is no recording of this. The reality must be on the balance of probabilities that this was some minutes after waking when she spoke to her father-in-law. Of course Nurse Bates is right that such a history does not tell us whether, had she spoken to somebody earlier, her speech would have been slurred. Nevertheless on the question of fact I find that on the balance of probabilities Nurse Bates did not elicit the history in this detail. Given that C1 was co-operating I am forced to the conclusion that Nurse Bates did not "unpick" the history. It is likely that C1 "telescoped" the history to some extent by saying that she woke up with right-sided heaviness and slurred speech but, as Nurse Bates accepted, time of onset was very important and such a statement needed careful analysis. There is no evidence of this in the notes and, as I have said before, I believe that Nurse Bates has persuaded herself that she did do it. In my judgment she did not.

28. As to some of the other notes:

    (i) The ambulance notes do not purport to take a detailed history. They say that the patient woke at 7am. After that there is a full stop then "® sided weakness. ® sided facial droop. ?TIA etc."

    Mr Booth says one would expect a degree of precision and the notes suggest she woke with ® sided weakness. C1 says the notes are consistent with her case that she awoke at 7am and then developed ® sided weakness. In my judgment these notes are consistent with either account and do not assist on this issue.

    (ii) The Triage nurse notes describe the patient as having awakened feeling unwell and wobberley (*sic*). That is some, but limited, confirmation of Ds' case.

    (iii) Dr Ramphele under "HPC" recorded "Woke up this morning with slurred speech and ® sided weakness." He essentially accepted that he did not ascertain (in any detail) what happened in the ½ hour before the ambulance came. He said he thought that C1 had told him she awoke with right-sided weakness, but accepted that if it is assumed that C1 got up and dressed, picked up a book and went downstairs then it looked in hindsight that he failed to elicit this. [On agreed evidence, he is not to be criticised for this, given that he was an SHO in A & E]. Neither party submitted that his note assists me in deciding this issue. I agree.

29. I have set out above the notes from Darent Valley Hospital and KCH. They do not elicit the incident at 05:50, nor any detail of what happened when C1 awoke at or around 7am. The authors of those notes have not given evidence. Dr Allder said that anybody taking a history in circumstances similar to C1's would want to know what the person was like when they went to bed and during the night, when they woke up and in the couple of minutes following. He was asked in cross-examination about the

KCH history and he said it was a reasonable history. It was pointed out that there was no mention of a 05:50 incident. His explanation was that they were going to get tests and they did not think they would find anything on the history. He said he would have expected KCH to ask the question as to whether C1 got up in the night, and to have noted the answer. He did interpret the KCH note as the stroke starting at 7am. He said that is what they thought and by that point there was no detail because the extent of the deterioration was driving the decision making. He interpreted the KCH note: "This morning she woke up at 7:00 and since this moment she felt light weakness on her right arm, right leg and problems with her speech" – as not being a stroke in the middle of the night, but accepted that on that history one could not determine the time of onset.

30. In Dr Allder's report at 11 March 2015, he referred to the history at 05:50 and said "This information was obtained by Mrs Owers Solicitor so it is difficult to understand why this could not have been discerned by the medical staff questioning her." However he accepted that when he had produced his draft liability report in 2011 he did not elicit the 05:50 history, and therefore conceded that what he said in his 2015 report was unfair criticism (adding that 05:50 was not a big thing and that the onset on the stairs was more likely to be the onset – see later).

31. In relation to the 05:50 incident:

(i) I have already found that Nurse Bates did not ask whether C1 awoke during the night. What if she had so asked? In other words is the witness statement of C1 reliable on this point?

(ii) On the balance of probabilities I do not find that C1 is accurate about the 05:50 incident.

(iii) The Defendants have not had the chance to cross-examine C1 and I must take that into account when weighing her Civil Evidence Act statement in the evidential balance.

(iv) It is not clear precisely when that evidence first emerged but, so far as the Court is aware, the first record is in C1's statement, dated 9 May 2014.

(v) There is unfortunately no corroboration whatsoever of the 05:50 incident.

(vi) I should emphasise that this does not amount by any means to a finding that C1 has tried deliberately to mislead the Court. Of course I appreciate that what happened on this day was much more likely to remain in her memory than most other days since the event was so highly significant and tragic. Nevertheless, there is the real potential for error especially in/after very distressing circumstances.

(vii) I therefore find on the balance of probabilities that the 05:50 incident did not occur.

32. That leaves the question as to what happened on the balance of probabilities when C awakened. This is a difficult issue given (a) C1 cannot be cross-examined, (b) the authors of the notes at Darent Valley and KCH have not been cross-examined (c) I am

not persuaded for the reasons I have already given that Nurse Bates "unpicked" C's condensed history of waking up with right sided heaviness and slurred speech. [I shall deal later with C2's evidence as to Nurse Bates' examination]. I accept Mr Owers' evidence that Jake came into the bedroom and woke him up at 7 o'clock and he went downstairs and C1 was sitting on the settee and that he, C2, thought that she was having a stroke. If this is right, it is corroboration of C1's evidence that she had got up without speaking to C2 and got herself downstairs. It is also very likely that she had dressed herself before going downstairs. This is some corroboration of C1's own statement.

33. I now consider the other notes of 14 March 2010:

(i) The Darent Valley records, if anything, slightly support C1's case generally. However, there is no detail to suggest that anyone elicited more from C1 when, as I shall find, there was more to be elicited.

(ii) The same goes for the note of Dr Bercocal/Dr Haider which is more favourable to Ds. Again there is no detail. One possible explanation is that they considered that if she woke up with these problems, then they were focal neurological deficits at time of waking. (I do not believe that the later typed note of Dr Haider can assist more than the written note).

(iii) In respect of the Darent Valley and KCH notes re onset, it must also be recalled that they were being taken long after the normally accepted window for thrombolysis had passed, even on a 7am onset.

34. So far I have mentioned only briefly C2's evidence on the onset of symptoms issue. In relation to Nurse Bates, he says that her note of what happened bears no resemblance to his recollection of what happened. His recollection was that Nurse Bates came in, very briefly spoke to him and his wife, and then said "I am going out to find the notes". She just disappeared and they did not see her again until she came back much later with Mr Gedge. He does not recall her examining C1 or asking her about symptoms or when they started. Despite this evidence I find that Nurse Bates did take the history and examine as recorded in her notes. She started this at about 8am and finished at about 8:20 when she wrote up the note. Other things were going on in this time, such as neurological observations and blood being taken. Nevertheless, I do not accept that Nurse Bates transcribed notes from other sources and/or fabricated an examination which did not take place. One possibility which she mentioned, and which may well have been the case, is that C2 was not there when she first saw C1. In any event, my general findings as to C2 are that he is an honest witness but not accurate in certain regards. This is not a criticism of him. As I said about C1, in these circumstances there is always the potential for error because of the distress. For example, C2 accepted that paragraph 9 of his witness statement was incorrect when he said "It is certainly not my recollection that Dr Ramphele saw Karen at that time. I thought he saw her much later." He also said that C1 did not suffer with migraine whereas her own statement (paragraph 3) says she gets occasional migraines, usually about 3 or 4 times a year, if that. She had migraine tablets and if she felt it coming on she took a tablet which usually nipped it in the bud. She had a warning of flashing lights in the corner of her vision and a stabbing pain in her right temple.

35. Cs originally cast doubt on Nurse Bates' note because she refers to C1 having had a head injury 4 weeks before and notes "Investigated in Oxford hospital". The ambulance record also says "Investigated at John Radcliffe Hospital". The suggestion was that Nurse Bates may have merely transcribed this from the ambulance record, and not taken this history. This is because the evidence of C2 was that his wife was taken to Cheltenham and not Oxford hospital and she would not have made this error. In fact the error was seemingly that of C2 himself since records show that C1 did go to the John Radcliffe hospital in February 2010 and went to Cheltenham in 2008.

36. Considering the evidence as a whole, I accept as probably correct paragraph 5 of C1's statement subject to two qualifications. The first is this: I accept that she awoke with right sided heaviness as Nurse Bates recorded. She would not have been aware of the slurred speech until some minutes later when she spoke to her father-in-law. This also fits reasonably well with the neurologists' KCH note that she awoke feeling "light weakness on her right arm, right leg and problems with her speech." (There again the problem with her speech has been "telescoped"). Therefore I find that she awoke with some right sided heaviness but it was mild or "light"[2].

37. To this must be added an important point which arose during Professor Wills' evidence. He said that the history of being a little bit drunk would have required clarification as to whether C1 meant that she was unsteady on her feet. If she had felt unsteady on her feet then that would have been ataxia, a neurological symptom noticed within moments of waking up. Again there was no opportunity to cross-examine C1 on this. However the balance of probabilities shifting in favour of ataxia as the main evidential indicator is to be found in paragraph 7 of the original Particulars of Claim.[3] This states:-

> "When she got up she felt a little bit drunk. As she was dressing she felt that she could not coordinate her arms and felt unbalanced. She felt weird but after she had finished dressing she went downstairs…"

As it is necessary, the Particulars of Claim is signed with a statement of truth by C1's solicitor. Therefore I can assume that these were C1's instructions. On the basis of that statement, Professor Wills said, and I accept, that this indicates ataxia which probably (though not necessarily) occurred in the night.

38. In short my findings therefore are:-

(i) C1 is not accurate about the 05:50 incident

(ii) Otherwise C1's history in paragraph 5 of her statement is broadly correct save on the balance of probabilities:-

(a) she awoke with right sided heaviness/weakness, there being no material difference between the two;

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[2] There is the possibility that the word "light" in the KCH record reads "Right". However this makes less sense because the right arm and right leg are then mentioned and, to be "right" there would have to be "R" in the middle of a sentence, which seems unlikely.

[3] I say "main" because there is some weak corroboration of this account in the Triage note "pt woke this morning feeling unwell and wobberly…" (my underlining)

(b) she had ataxia immediately she got out of bed. I accept Professor Wills' evidence that this means that the neurological deficit so manifested probably existed prior to waking.

Consequences of the Findings as to the Onset of Symptoms

39. It is common ground that the result of my findings is that they put an end to the allegation that C1 should have been thrombolysed. I therefore do not need to deal with the disputed evidence as to whether the head injury in February 2010 contra-indicated thrombolysis.[4] The case then proceeds as to when C1 should have been prescribed aspirin and the causative effect of the failure to prescribe her aspirin.

Events at Medway Subsequent to Nurse Bates' Examination

40. Dr Ramphele examined at about 08:36am. By this time C1 had already been seen by the ambulance crew, triage, the author of the care continuum and Nurse Bates. An ECG had also been done. In addition there was a neurological chart entry at 08:10 which showed blood pressure 122/82, mild weakness in right arm and leg and a pain score of 2. Dr Ramphele accepted he would have seen all the records except the care continuum which may not have been available to him.

41. I have already set out part of Dr Ramphele's note. The note continues, so far as material:

> "CNS: GCS 15 PEARL
>
> Sensation: intact upper and lower limbs.
>
> ?slurred speech
>
> No facial weakness
>
> Tone: normal, upper and lower
>
> Motor: power: 5/5
>
> Reflexes: normal
>
> Δ? Psycho-somatic/? TIA
>
> P – bloods √ CT: NAD
>
> - ECG: NAD
>
> - D/W Dr Tolat
>
> - Advised: - get Rapid Response assessment
>
> - Stroke team to review again

[4] In an attempt to focus submissions and streamline the case it was agreed by all that I should hear submissions first on time of onset of symptoms and announce my findings of fact on this. Having done this, the parties did not therefore address me on whether the head injury was relevant.

D/W: Medical reg: Advi (H)"[5]

42. It was clear to Dr Ramphele that some parts of his note were added after the 08:36 examination. On balance those parts which were added were ?TIA, CT:NAD and everything after the words "advised: - get Rapid Response assessment…" He said at the end of his examination at 08:36 he would have put a presumptive diagnosis and at that stage it was ? Psycho-somatic.

43. Dr Ramphele said that he was surprised to read that Nurse Bates had noted "neurology non suggestive of CVA". He had had a chat with her. He did not remember it precisely but he said that she had said she had assessed for stroke and could not find objective evidence for it. He did not question her but went and assessed C1 himself. He said he would have been influenced to some extent by the fact that Nurse Bates had more experience of stroke because she was dealing with strokes at that time. That would have set the bar for diagnosis slightly higher. However he would not have carried out his examination in a less diligent way. Even if it was not a stroke there were neurological problems and therefore needed a full examination.

44. He had decided at the end of his examination to discuss with Dr Tolat and he told her face to face that he was not sure what was happening. There is no note of his discussion with Dr Tolat or subsequent discussions which he said he had with her. Nevertheless his evidence was that he did speak to her. He asked her to see C1 but she did not see her. Essentially what he had communicated to Dr Tolat was that he had a patient referred with a possible stroke. The stroke team was saying she was not having a stroke and he was not sure what was happening.

45. At 09:40 the care continuum document says

> "obs recorded. Pt now cannot feel arm – is upset"

(The neurological chart at 09:40 shows mild weakness in right arm and leg and pain score of 2).

46. C1's statement (paragraph 11) says:

> "The nurse came to take my observations a second time and I see from the records that this is timed at 09.40hrs. The only reason this was done is because Tim went out to find someone because I had deteriorated. From arriving at the hospital I could feel myself getting gradually worse and when the nurse was there I told the nurse I couldn't feel my right arm and I also knew my speech was getting worse. I was very upset and crying. Tim kept asking them shouldn't they be doing a scan

[5] (There are two ticks under the words upper and lower. There is also a large bracket which covers the spaces after the words, vertically read "normal, 5/5, normal, /bloods". Therefore the words upper and lower and the ticks beneath them and ?TIA and CT:NAD appear after the bracket though the ? before the TIA straddles the bracket.)

> because all they seemed to be discussing was getting me home. After I got worse Tim made a big fuss because they still wanted to send me home and it was after this that they sent me for a CT Scan. I now know from the notes that this was not until about 11am, about 1 hour and 20 minutes after I had told the nurse I could no longer feel my right arm and 3 hours after I arrived at the hospital."

There are echoes of this paragraph in paragraph 10 of C2's statement.

47. Dr Ramphele said that the nurse told him that C1 had deteriorated. He did not re-examine and accepted that this was a significant failure in his care. At this point he said he would also have tested for gait and co-ordination and diagnosed a stroke. Had he done this, he would have got in touch with the stroke team again because they were primarily responsible. He would have said to them that her signs were more consistent with a stroke.

48. The next entry in the notes is timed at 11am and it was written by Mr Gedge. As to timing, Mr Gedge said he may have started the examination a little before, perhaps at 10:50, but it was possible that he started at 11am. He said the stroke team were bleeped to re-review as C1 was deteriorating. He said both he and Nurse Bates would be bleeped. C1 was to be reassessed. Mr Gedge's entry reads:

> "Stroke team
>
> Asked to R/V pt again by A & E as pt presenting with worsening weakness. Husband present who reports symptoms have worsened.
>
> Pt c/o headache and photophobia.
>
> Vision – NAD
>
> Face – some R facial droop present but inconsistent and appears athetoid.
>
> Speech – no aphasia
>
> Dysarthria present"

For the upper limbs he recorded normal tone 0/5 power and "tingling" sensation on the right and normal tone power and sensation on the left. For the lower limbs the record is the same as for the upper limbs. The note then continues:

> "Symptoms appear to have changed although pt inconsistent during multiple assessment.
>
> No risk factors present.
>
> P/ CT head"

49. Two issues arise with which I shall deal with briefly:

(i) C2 says that Mr Gedge's examination was "perfunctory" and that the detailed examination in the note "simply did not happen". For reasons similar to my rejection of the like suggestion in respect of Nurse Bates at 08:20, I do not accept C2's recollection as reliable on this.

(ii) C2 says that Mr Gedge arrived with Nurse Bates in the cubicle and they both left the cubicle together. This was not accepted by Mr Gedge or Nurse Bates. Mr Gedge said that if they were bleeped, as the two members of the on call stroke team, they would go to the A & E department together so that the other would be there in case a discussion was needed. Mr Gedge's note records in the margin "C Gedge", then underneath "C Hannon". Mr Gedge said he documented Nurse Bates' presence as meaning that she came up to A & E with him, not that she was in the cubicle. My finding on this is that it is unlikely she was there all the time, but also unlikely that C2 is totally mistaken. On balance I find that Nurse Bates was in the cubicle for some, but not necessarily all, of the duration of this examination.

50. Mr Gedge decided at the end of his examination to order a CT scan. At that moment his provisional diagnosis was a stroke. That was his clinical impression. It took about two minutes to get C1 to the CT scanner and she arrived there at 11:15. The CT scan had been done by 11:25. It was negative. Mr Gedge said that he was aware that this did not exclude a stroke diagnosis. What he then noted at 11:30am was:

> "CT head – normal
>
> NB* in CT patient was able to lift bottom up to get onto sliding board and sustain in crook position despite apparent "dense weakness".
>
> Impression: - unlikely stroke
>
> ? functional overlay/conversion disorder
>
> ?? hemiplegic migraine
>
> P – Pass back to A & E – 11:36."

51. In paragraphs 21 – 22 of his witness statement Mr Gedge said this:

> "21. It was at this point that I requested an urgent CT scan, based on her current clinical condition, as I was concerned with the sudden deterioration in her symptoms, her apparent lack of stroke risk factors and also in light of her reported recent head injury. I liaised with the CT imaging department who agreed to do the scan urgently. Along with a member of the A & E team I assisted to take her round to the CT scanning department.
>
> 22. On assisting her to transfer from the A & E trolley to the CT scanning table I observed that she was able to lift her right leg up into the crook position and then lift her bottom up to "bridge" and held this position whilst a sliding board was

> placed underneath her to transfer her safely. I again found this very odd seeing as a few minutes earlier she was completely unable to lift her right leg up during an assessment.
>
> …….
>
> 24. From my own objective assessment and review of previous assessments by Caroline and Dr Ramphele I did not feel that the presentation was consistent with that of a stroke and actually her presentation was more that of a possible stroke mimic. I did question whether this was a case of functional overlay or conversion disorder or whether she was suffering with a hemiplegic migraine. However I did not make any particular diagnosis as our protocol is that if we believe the patient symptoms are not suggestive of stroke we pass the patient back to A & E to review and they make their own formal diagnosis."

52. Mr Gedge said that that he had never seen stuttering/fluctuating symptoms which are consistent with a BAO stroke. His mindset was that a patient can have dysarthria with lots of other conditions. Therefore he thought it was more likely to be a stroke mimic. The facial droop presentation was inconsistent, he said. His note of "athetoid" meant that she could smile and raise her eyebrows. He did not expect that. C1 was complaining of headache and photophobia. A person does not always have a headache with a stroke and could have had migraine. He did not write history of migraine but he had seen a history from the ambulance, Nurse Bates' and Dr Ramphele's notes.

53. Shortly after 11:36 C1 was referred back to A & E. The only notes relevant to this are the last few lines, already dealt with above, from Dr Ramphele's record. He said he did not make proper notes at this time and it looks as though he was trying to squeeze everything on the same page. He said he did not see C1 at this stage save at a distance in the department and he only examined her once. He said he was trying to get her admitted and he spoke to the medical registrar. There is no note that he was trying to get C1 admitted but he was insistent that he did. He said if she was not admitted by the stroke team then she could only be admitted under the medical team. Dr Tolat advised to get a Rapid Response assessment. Dr Ramphele did not know why she said that rather than coming to see C1. He explained to Dr Tolat that it seemed like a stroke. However Dr Tolat, the Registrar, said send her home even though she had not seen C1. Getting the Rapid Response assessment was to ensure that C1 was fit to discharge. The entry "stroke team to review again" was a note which was added afterwards.

54. Dr Ramphele was an honest witness. I accept his evidence as probably correct in relation to what happened after 11:36, so far as he was concerned. [I should add that Dr Tolat was not a witness and any implied criticism of her is based on the evidence put before the Court. It would be wholly wrong for anybody to consider anything in this judgment to be a definitive criticism of Dr Tolat when she has not had the opportunity to be heard].

55. I also accept as a probability the recollection of C1 and C2 that Dr Ramphele said that they were to go and find a wheelchair and take C1 home so she could sleep it off. C1 had to be helped into the wheelchair by her husband and she recalls deteriorating in the 5 or 10 minutes from getting into the wheelchair to getting into the car.

56. That brings to an end the history up to the moment of discharge at Medway Hospital.

The Time When Aspirin Should Have Been Prescribed

57. Ds accept that aspirin should have been prescribed at some point between about 10:40am and 11am. This is on the basis that after the deterioration in C1's condition at 09:40am, she should have been seen by a doctor. She was not. After a re-examination of some 10 minutes or so, the diagnosis of probable stroke should have been made, an urgent CT scan undertaken within about 30 minutes and the aspirin prescribed in about a further 10 minutes. Allowing for a few minutes to get a doctor and some variability, the window was therefore about 10:30am – 11am. Precise timings at this stage do not matter. This is because there was no further significant deterioration after 09:40 and before aspirin should have been prescribed. Indeed C1's stroke was still "fluctuating" at the time she was in fact having the CT scan, when Mr Gedge saw her able to lift her bottom up to get onto the sliding board and sustain herself in the crook position. This, on the evidence, was between about 11:15am and 11:25am.

58. C1's case is that aspirin should have been prescribed earlier. It is submitted that it was substandard practice in 2010 that C1 was not diagnosed as having a stroke by about 08:20am, which was approximately the time Nurse Bates finished her examination. The time window for this submission is very tight. This is because of the significant deterioration in C1's condition at 09:40am. Therefore, working back from 09:40am, C1 would have had to have been seen by a doctor capable of diagnosing a stroke by about 08:30[6]am so as to allow 10 minutes for the examination, 20 – 30 minutes for the urgent CT scan, 10 minutes to prescribe aspirin and 30 minutes for the aspirin to potentially take effect. It takes about 30 minutes for aspirin to work.

59. There is no doubt that there were clear failings in the Medway Stroke Pathway. Most particularly, it was wholly unclear whose responsibility it was to make a diagnosis and to whom a patient should be referred in a difficult case where a stroke could not be excluded. The pathway at the relevant time said:

> "4.1 A patient suitable for thrombolysis should be referred for urgent CT scanning and the on-call/consultant bleeped"

Albeit under the heading "Patient's (*sic*) Presenting as a TIA", the advice for a patient not a stroke was:

> "3.2 If the patient is not a stroke and the suitability for admission or the cause of the patient's condition is unclear or

[6] At the very latest 08:40

> uncertain then the patient is to be handed back to the ED[7] doctor for further assessment and planning of care."

60. From this it is apparent, as Dr Baines said, that there was a lot of confusion as to the role of the stroke team and it was not clear as to what to do with ambiguous patients. In similar vein Professor Wills was critical of the pathway in difficult cases. There was nobody to take ownership of a difficult case.

61. Nurse Bates said she was not there to diagnose but to triage to a pathway. Mr Gedge's evidence was similar on this point. There was, on the evidence[8] no criticism of Nurse Bates or Mr Gedge (as stroke nurse and stroke physiotherapist) if they acted in a screening role. The real criticism is that they should not diagnose in difficult cases (which they say they did not) and of the Medway Stroke Pathway provision for what should happen in a difficult case. Doctor Mamun[9] expressly said that he did not expect Nurse Bates or Mr Gedge to diagnose in difficult cases.

62. What happened in Medway in a difficult case in 2010? Unsurprisingly, given the content of the Medway Stroke Pathway at the time, Nurse Bates said that if she thought a patient was suffering from a clear stroke or there was an unclear diagnosis, she would call the stroke physician if the patient was in time for thrombolysis. This she took to be 3 hours from time of onset of relevant symptoms. If she did not think somebody was having a stroke or was uncertain, she passed the patient back to Accident and Emergency to investigate further. She would return the patient to the SHO who had access to more senior doctors. Mr Gedge's evidence was similar. If a patient was outside the 3 hour window from clear onset of symptoms he would not contact the stroke consultant. If there was a probable stroke and a patient was within the 3 hours he would refer to the stroke physician. If he thought there was probably a stroke and more than 3 hours had passed from the clear onset of symptoms, he would liaise with A & E for aspirin prescription and contact the acute stroke unit for a bed and contact the on-call medical team. If he thought there was a real possibility of a stroke, and the patient was within 3 hours of clear onset of symptoms, he would hand the patient back to A & E. If the pathway at the time had said that in cases of doubt, refer to the stroke physician, he would have referred C1 to Doctor Mamun. That is what the Medway Stroke Pathway says now. However, it was practice and training to hand back to A & E in difficult cases in 2010.

63. Taking on board these deficiencies in the 2010 Medway Stroke Pathway, Ds concede, for the reasons I have given, the failure to make a stroke diagnosis following the 09:40 deterioration. The question is as to what should have happened in a competent stroke unit in 2010 at about 08:20am when Nurse Bates finished her examination.

64. As a preliminary matter, if Nurse Bates had telephoned Doctor Mamun at home at 08:20 and he had come in, as he said he could, within 30 minutes of a call, then even that would have been too late on the timings to have made a difference. He would not have arrived at the hospital until about 08:50am. Mr Samuel initially said that Dr Mamun could have ordered a CT scan over the telephone, but accepted that there was no evidence to support that he should or would have done so. Therefore C1's case

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[7] Emergency Department
[8] Including Dr Baines and Doctor Allder
[9] Consultant Physician and Stroke Physician at Medway from 2010 onwards

has to depend, as said above, on her needing to be seen and diagnosed by a consultant physician or neurologist or registrar with special knowledge of strokes, by 08:30 – 08:40am at the latest.

65. C1 relies first on Dr Baines. His opinion was that C1 presented with atypical features but from the A & E perspective there were sufficient features that a stroke should have been suspected. He said that in 2010 there were no stroke teams without the support of a physician or neurologist in the hospital, or out of hours from a medical registrar. He said that a senior medical person from the stroke team could have attended after Nurse Bates' examination and examined C1 and ordered an urgent CT scan. The process would have been to contact the senior medical support. There would not have been a confirmed diagnosis at this point, but a more urgent CT scan and then prescription of aspirin. He said that at 08:20am a stroke should not have been ruled out but the matter escalated to a neurologist or stroke physician; he had earlier said that if Nurse Bates had not been sure and C1 had been admitted to the acute stroke unit that would be satisfactory.

66. Mr Baines considered that in 2010 stroke teams had the unilateral support of the stroke physician or neurologist with immediate access to physicians for diagnostic support (or out of hours the medical registrar).

67. Doctor Allder said that in 2010 the diagnostic phase always involved a senior doctor and he did not think a nurse or physiotherapist could be trained to do this diagnosis. It needed a doctor au fait with strokes, at least a registrar who could telephone the consultant if need be. He said that by that date it was clear that there needed to be a physician on-call and regular liaison with A & E, to ensure that people did not fall through the gaps in the system. If there was any difficult case and any room for doubt then a stroke doctor should be involved.

68. Professor Wills did not criticise Nurse Bates handing back to a junior doctor, but said that if the system had worked Doctor Ramphele would have engaged a registrar in A & E or general medicine. He did not accept that such a registrar would have diagnosed a stroke. He said he thought that the medical registrar would have thought it was a migraine. His view was that the average/poor registrar would have sent C1 home. A good registrar may have realised the potential significance of the previous head injury and admitted C1 to a medical bed. On balance at this stage the diagnosis would have been migraine. This takes into account that C1 did not have typical dysarthria. Nurse Bates' evidence was that C1, at the time of her examination, had a very unusual speech pattern. Some words at the end did appear slurred but this was not consistent.

69. The totality of the evidence leads to a confusing picture. Apart from the difference in evidence, there are the following factors for me to take into account:

    (i) A number of features can be consistent with a stroke but have differential diagnosis such as migraine or transient ischaemic attack[10]. As Doctor Campbell-Hewson said, there was no lateralised hemiplegia. There was a history of right-sided weakness not seen by Doctor Ramphele. That account of

[10] It is only safe to make a diagnosis of TIA if symptoms have fully resolved. However if symptoms are or appear to be in the process of resolving then TIA has to be considered as a possibility

improving from an equivocal diagnosis is more likely to be a resolving TIA. Similarly Professor Wills said that on balance migraine was the probable diagnosis at the pre 09:40 deterioration time.

(ii) Despite Dr Baines' oral evidence, he had said in the joint statement (paragraph 7):

> "…If Nurse Hannon or Mr Gedge did not feel that a patient met the typical criteria for them to make an independent diagnosis of stroke, the patient could be referred back to the emergency department. However under such circumstances it should be clarified that a diagnosis of stroke had not been excluded."

(iii) Commenting on Dr Ramphele's examination Dr Baines said that after his examination he would have expected a 10 minute conversation with Dr Tolat and then a CT scan and referral to the general medical team. He said that the scan at that stage would have been requested as non urgent but at 09:40 would trigger urgency[11].

(iv) On the other hand Professor Wills had to explain as an "error" this statement he made in his 3 March 2015 report:

> "I am surprised that neither Ms Bates nor Mr Gedge sought advice from a senior medical member of the stroke team (either consultant/physician or consultant neurologist) in these circumstances."

He said that this was his view at 2015 rather than in 2010. He added that a registrar in an ideal world would contact the consultant physician who would probably have admitted C1 at this early stage and see how things went.

(v) Doctor Mamun accepted that at 08:30am a stroke was at least a realistic possibility. He said that it was not sufficient then for CC to be examined just by an SHO. She should have been seen by the A & E registrar or the medical registrar or she could have telephoned him[12]. He added that there were several consultant physicians in the hospital.

70. My findings on the balance of probabilities are as follows:

(i) There were consultant physicians on call in the hospital and Doctor Mamun, a stroke physician, was on call at home and could have been in the hospital within 30 minutes of a call.

(ii) The stroke pathway and the training of Nurse Bates should have been such that she did not refer back to an SHO on the findings on her examination.

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[11] It was only if the history was as C1 gave in terms of onset of symptoms and had been elicited for example by Nurse Bates, that the CT scan should have been ordered urgently. I have not found that the history was as C1 gave it. Therefore this does not apply.

[12] As before stated I do not accept that there is criticism of Nurse Bates in this regard. Nevertheless the evidence is of relevance as to Doctor Mamun's view of what should have happened (had Nurse Bates had a proper pathway or been properly trained in this regard).

(iii) The least that the pathway in 2010 should have required was that C1 be seen urgently after 08:20 by a doctor of the level of A & E registrar or medical registrar.

(iv) An A & E or medical registrar would probably not at that stage have diagnosed a stroke. Differential diagnoses of resolving TIA or migraine would have been entirely possible.

(v) However, because stroke could not have been excluded, a CT scan would probably have been ordered by an A & E/medical registrar but on a non urgent basis.

(vi) The alternative and perfectly feasible system at the time would have been for Nurse Bates to have been required by the pathway to telephone Doctor Mamun. Even if he had come in by about 08:50 and examined her, then the relevant time window had passed for the prescription of aspirin after a CT scan and for the aspirin to take effect prior to the 09:40 deterioration in C1's condition[13].

71. C1 relied in submissions on this statement from Doctor Allder's March 2015 report, page 34:

> "At the time of the assessment by Nurse Hannon, with a clinical syndromic diagnosis of acute stroke, the first line diagnostic would have been a CT scan to exclude the possibility of a haemorrhagic stroke and enable the consideration of treatment through thrombolysis or antiplatelet therapy. Depending on the conclusion reached by the clinical team, either thrombolysis or antiplatelet therapy would have been offered at this point."

For reasons I have given above I do not accept that a diagnosis of acute stroke would or should have been made at the time of assessment by Nurse Bates or shortly afterwards. Dr Allder's statement, which does not go into the detail of the various possible findings, does not influence my overall decision.

72. In summary the remainder of this judgment will proceed on the basis that Ds were not negligent in failing to prescribe aspirin prior to the 09:40 deterioration, but were negligent shortly thereafter. I do not need to make findings as to precisely when thereafter since the admission of 10:40am – 11am was well within the next relevant therapeutic window on any view of the case.

73. I therefore turn to the issue of causation.

The Consultant Neurologists – Overview

[13] Doctor Mamun said that sometimes he would give aspirin before checking there was no bleed on a CT scan. However he said that usually one would want to make sure there was no bleed though sometimes one has to take a clinical decision. I do not believe that on the balance of probabilities in this situation (i.e. examining C1 at about 08:50am and from what we know of her presentation) he would have taken this chance. Other experts said that they would not prescribe aspirin without a CT scan. See also the NICE guidance paragraph 1.4.2.1.

74. Dr Allder is a Consultant Neurologist at Hampshire Hospitals NHS Foundation Trust. From January 2004 to March 2015 he was a consultant neurologist at Plymouth Hospitals NHS Trust. From 2008 he was the clinical director for the whole of the Plymouth area, including the outlying hospitals. He had had a particular interest in strokes since 1997 and had been involved in substantial stroke research. He opened the stroke unit in Plymouth in 2005.

75. Professor Wills has been a Consultant Neurologist at Nottingham University Hospitals and Honorary Consultant Neurologist at Queen Square Hospital since December 1998. He has published a number of neurological articles, though has not been involved in stroke research, apart from participating in a stroke trial. Since 2008 he has spent every Tuesday on the acute stroke unit. He has a TIA clinic and is on the on call rota for strokes – this included weekends until last year. In his hospital he is the neurologist responsible for stroke.

76. Dr Allder was asked how many basilar artery strokes he had seen since these are very rare. At first he guessed 200. He then estimated that he had seen more than 10,000 patients and that 1% of them would approximately be basilar artery strokes, thereby concluding with a figure of about 100. Professor Wills said that he had seen very few basilar artery strokes. When one comes in to his hospital the doctors discuss it. He said he worked in a very busy teaching hospital and he found it extraordinary if Dr Allder had actually seen 100 basilar artery strokes. He wondered whether Dr Allder had estimated how many posterior circulatory strokes, rather than basilar artery strokes, he had seen – given that basilar artery strokes are only one category of posterior circulatory strokes. He said that he saw some 200 – 300 strokes a year. Statistics would suggest that he would see some 2 – 3 basilar artery strokes but said that he did not see that many, probably because they die in A & E. He estimated he had diagnosed about 10 to 20 basilar artery strokes in the acute phase over the last 10 years. He said that his experience was also gained from talking to other stroke doctors when a basilar artery stroke came in. Some of the basilar artery strokes have had mild symptoms and he and his colleagues used heparin/thrombolysis not aspirin.

77. As is not unusual in adversarial litigation, each party sought to attack the reliability of the other party's expert, not only on the basis of the accuracy and coherence of their argument, but also in ways which undermined their professionalism. I shall take very briefly a few examples.

78. C1 said that Professor Wills:

- Produced literature during the trial and said he had arrived late at a conclusion and wanted to express his doubts as to the efficacy of thrombolysis yet during cross-examination accepted that treatment within 3 hours would have helped C1. This, Mr Samuel described as "a complete volte face".

- Failed to set out his case on why aspirin would not have made a difference in his main report, and in the joint statement evaded the question relating to aspirin by digressing to heparin and thrombolysis.

- Failed to consider the evidence impartially by failing to take into account the evidence of C1 and C2 that Nurse Bates did not carry out a thorough examination.[14]

79. Ds said that Dr Allder:

- Was very vague in his report so that for example he said (page 41) "Aspirin would have afforded the opportunity to avoid further deterioration" and in his conclusion (report page 42)

- Was inconsistent for example in his report at page 34 he said that C1 had a moderate severe clinical syndrome when he conceded in evidence that was not the case.

- Put an absurdly positive spin on the external reports of the poor stroke service in Plymouth in 2008 and 2011 over which he had presided since 2005.

80. I have to say that, listening carefully to the evidence over many days and taking on board the force with which the rival submissions were made, I give little if any weight to these criticisms of the doctors. All parties agreed that this was an extremely difficult case. Both experts made some errors, both made concessions in evidence from their reports and it will be clear from this judgment that on particular matters there are occasions when I have rejected the accuracy of both experts. Nevertheless, in this complex case, both were professional and assisted as much as possible. I do not find that either was partisan. My assessment of the evidence is based on my independent and objective analysis, this not being tainted by any suggestion on either side of lack of professionalism by the experts.[15]

81. C1 submitted that I should prefer Dr Allder's evidence because of his more extensive stroke experience. Ds submitted that Professor Wills was the expert who produced the most up to date and relevant literature on the respective effects of antithrombotics and thrombolysis in BAO and that Dr Allder did not produce or rely upon Schonewille 2009 before Professor Wills produced it, it then becoming a main plank in Dr Allder's causation argument. I did not find any of these points particularly helpful in coming to my conclusions. That is not a criticism of the fact that they were made by Counsel. Nevertheless in my role as judge, my evaluation of the expert evidence is based on what is set out below in detail rather than these matters. The personal experience of both doctors does not help me in arriving at a conclusion on the difficult issue of causation. For that the central evidence is general aetiological

[14] I specifically mention that Professor Wills, in cross-examination, gave evidence that C1 had complained that he had not undertaken a detailed examination. He said that he had asked his solicitors to ensure that C1 came with a chaperone and she came un-chaperoned so he could not fully examine. He said he was upset by the allegation that he had not done his job properly and conceded he may have used that as part of his judgment as to what Nurse Bates did or did not do in terms of examination. I have not relied upon Professor Wills' opinion on that particular point about Nurse Bates' examination. Although this exchange was unfortunate, I do not accept that it affected the professionalism of Professor Wills generally or his reliability as an expert witness.

exposition, the facts surrounding C1's case, the medical literature and a critical analysis of the reasoning of the experts.

Aspirin Prescription

82. The NICE Guidance suggests that all people presenting with an acute stroke and a normal CT scan should be given aspirin "as soon as possible but certainly within 24 hours" (paragraph 1.4.2.1).

83. At paragraph 4.2 The Guidance says "Aspirin administered within 48 hours of acute ischaemic stroke improves outcome compared with no treatment or early anticoagulation".

84. I shall examine later the detailed medical literature. It is important to do so. It is also important to realise that it does not answer the precise questions which fall to me to decide.

85. A simple diagrammatic representation of the basilar artery and surrounding arteries was produced by Cs. It was agreed to be helpful and I attach it to this judgment as Appendix B. On the diagram can be seen two vertebral arteries which lead into the basilar artery. We know that C1 had suffered a vertebral artery dissection (VAD), a tear in the lining of one of the vertebral arteries. This can cause emboli to form.

86. The NIH Stroke Scale (NIHSS) is an assessment tool that provides a quantative measure of stroke related neurological deficit. The Modified Rankin Scale[16] (mRS) is a scale for measuring the degree of disability/dependence of stroke patients. The scale runs from 0 – 6. mRS 1 is no significant disability, mRS 2 slight disability, mRS 3 moderate disability, mRS 4 moderately severe disability, mRS 5 severe disability and mRS 6 dead.

87. Aspirin is an antiplatelet medicine. Heparin is an anticoagulant.

88. C1's NIHSS score prior to 09:40am was in the region of 2 – 4. By 11am it was 11 – 12. Her case on causation as put forward by Dr Allder is, in broad terms, that if she had been prescribed aspirin so as to take effect before 09:40am then she had a 58% chance of mRS 0 – 1. After 09:40am Dr Allder says that her probable outcome was mRS 3.

89. In the joint statement in answer to question 36 "What is the prognosis for patients who suffer a basilar artery occlusion?" Dr Allder replied:

> "SA's opinion is that this is a complicated issue. The first issue is the distinction between total basilar artery occlusion and basilar artery stenosis. The digital subtraction angiogram that Mrs Owers finally underwent at KCH showed a distal basilar artery occlusion with collateral circulation. However, given the fact that Mrs Owers clinical state fluctuated considerably – certainly for 8 hours after her deterioration at 7:00 – and her MRI showed basilar artery stenosis as opposed to occlusion, this suggests that during the period of her assessment at

[16] C1's eventual outcome was mRS 4

> Medway and at Darent Valley, Mrs Owers did not necessarily have total basilar artery occlusion. Therefore, application of the evidence associated with basilar artery occlusion needs to be considered carefully. The second issue is that as diagnostic modalities and treatment of stroke have matured, so the complexity of issues determining prognosis following basilar artery occlusion has emerged. For an individual patient, the prognosis will be determined by the precise position of the occlusion, the critical extent of the occlusion and the presence, or not, of collateral circulation. Therefore, in any given patient, the prognosis can vary from a very mild clinical deficit through to catastrophic brain stem infarction.
>
> Mrs Owers did not present with a devastating clinical brain stem syndrome. Her imaging appears to reveal a varying degree of occlusion and the presence of a certain amount of collateral circulation. Therefore her prognosis cannot be considered inevitably poor, either with or without treatment."

90. Dr Allder had not mentioned this theory in his report. Nevertheless it was subject to detailed evidence. In any event it is to be noted that Dr Allder's opinion in the joint statement as set out above was guarded. As to the specific question relating to the prescription of aspirin or other anticoagulation or antiplatelet therapy on the long term outcome, Dr Allder replied:

> "SA refers to his original report in respect of his view concerning aspirin and antiplatelet therapy:
>
> > "Aspirin would have afforded the opportunity to avoid further deterioration. Antiplatelet therapy reduces micro emboli within 30 minutes of oral administration by 33% - 66%, therefore on the balance of probabilities I conclude that timely administration would have made a contribution to avoiding her stroke progression. Without this further deterioration, based on the severity of her clinical state prior to the deterioration, I would predict she would have achieved an independent, not dependent, final clinical outcome.""[17]

91. I have already found that C1's stroke was a fluctuating stroke while she was at Medway. This means[18] that she had fluctuating symptoms with alternating periods of neurological worsening and improvement.

92. It was common ground that the way aspirin operates is to stop emboli forming and breaking away from the lining of the artery. Professor Wills explained that the

[17] The sole basis for this opinion by Dr Allder was a paper Goertler et al "Acetylsalicylic Acid and Micro Embolic Events Detected by Transcranial Doppler in Symptomatic Arterial Stenosis". Cerebro Vascular Disease (2001)
[18] Schonewille (2005)

mechanism for natural improvement during a fluctuating stroke is that small parts of the clot blocking the artery break off and the brain recovers to some extent.

93. Cs' final submissions were that if that is the mechanism and aspirin shuts down the formation and propagation of emboli, there is no mechanism to explain why she would have deteriorated after periods of improvement had aspirin been given. In a fluctuating stroke when no aspirin (or thrombolysis) is given one can understand Professor Wills' explanation as to how the fluctuating symptoms are affected by small clumps of thrombus breaking off from the main clot, thereby allowing blood to flow more freely and thus permitting an improvement in functions. However, if there were no further emboli emanating from the VAD to assist in reforming the thrombos to add to the occlusion there is no rational explanation for the subsequent deterioration. Therefore had aspirin been given at Medway the micro emboli would not have been capable of forming or propagating or sticking to the main clot and so she would not have got naturally worse with time. If there is a complete occlusion then aspirin would make little if any difference but in a partial occlusion, if the mechanism for the propagation of emboli is stopped, the advancement of the thrombus will also stop and there will be no deterioration.

94. This, I find, was based on a misunderstanding of Professor Wills' evidence. While he accepted that the mechanism by which aspirin would work, if it did, he did not accept that it does in fact stop emboli in the vast majority of cases. He accepted that if aspirin does in fact work then it shuts off the source of emboli and if it does do so, that there is no other way of doing damage. However he said that in reality it has only a minor effect in actually achieving this outcome. He said there was a difference between the theory and the in vitro results of aspirin and the reality of the chances of it actually working in that way in the human body.

95. Nor did I understand Dr Allder to say that aspirin will always work in practice as it does in theory. Hence the guarded terms of his joint statement answer (set out above) and the fact that he only referred to probabilities. If, as C1 submitted, aspirin did work like that in cases of partial occlusion, then it would stop any deterioration.

96. Thus I find that if aspirin does work then that is <u>how</u> it works. The question is <u>how likely</u> is it to work in a partially occluded basilar artery stroke? My review of the literature will show that there is little room for any optimism here. Further, if Dr Allder's theory is correct, then one wonders why the specialist team at KCH did not prescribe aspirin. Prior to day 2 the basilar artery was not completely occluded. C1's case is that the literature does not separate between heparin and aspirin in terms of effect. Professor Wills' opinion was that heparin is more effective, particularly in basilar artery stroke. Yet C1 was prescribed heparin constantly at KCH from 00:07 on 15 March 2010 throughout that day[19]. If the effects of heparin and aspirin are

[19] C1 sought to establish that probably heparin was stopped at about 08:30am on 15 March 2010 at KCH, relying on the "record of intravenous infusions…" and that deterioration during that day may have been due to the withdrawal of heparin. Professor Wills accepted that had heparin been stopped and C1 had deteriorated then that was probably correct. However on the balance of probabilities that is a misreading of the documents. The syringe still had 26 mls left at 08:30. There is no record on that document of anything further. Nor is there a record on that document or elsewhere that the infusion was stopped. The "patient results" document shows that there was no period on 15 March when the blood test (the APTR) used to monitor heparin in C1's blood was below the therapeutic range. (Though the sample at 12:14 may have been affected). The result above the

equivalent in outcome (though different in mechanism) as is C1's primary case, then what happened at KCH does not assist and in my judgment undermines Dr Allder's theory.

97. Also, as already mentioned, Dr Allder in his original report relied on the Goertler et al paper only as to aspirin causation. This paper, which was a pharmacological paper, specifically said:

> "Whether the effects of an anti thrombolytic therapy can be monitored by TCD (transcranial Doppler) detected micro embolic events and corresponds to the clinical efficacy of a drug therapy cannot be judged on the basis of the cross sectional observation presented here."[20]

Literature and Overall Evaluation of Effects of Aspirin

98. In order to consider the issue of causation further it is necessary to analyse the medical literature on the subject. Since that literature does not provide crystal clear answers, I have had to hear differing opinions from Dr Allder and Professor Wills as to its effect. I am very aware, as I said during the hearing, that in the medical world there would potentially be teams of doctors pooling views and working together over a period of time to answer the question I have to answer. Then, if they produced a report, it would be peer reviewed to look for errors etc. I have only the bare literature and the two neurologists on which to form my judgment on the balance of probabilities.

99. The literature is complex and inconclusive. It does not, perhaps cannot, deal clearly with what the prognosis is likely to have been if aspirin had been prescribed at 8:35am (C1's case) or 11am (Ds' case) for C1 as an individual. Such literature as there is has to be considered with caution as:-

(a) many papers do not deal with prognostic outcome in the rare BAO strokes;

(b) the papers which do deal with BAO strokes;

(i) contain strong caveats[21]

(ii) do not distinguish in the results between heparin and/or aspirin when assessing the effects of antithrombotic (AT) therapy;

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therapeutic range of 5.42 may well be that copied into the document headed "Over Anticoagulation with Warfarin" which is untimed but has the 5.4 figure and says "stop for 60 minutes then restart at 1.6 ml/hour."

[20] Further the paper dealt with patients with symptomatic carotid artery stenosis, not basilar artery strokes. Professor Wills said that the paper showed that aspirin can have quick effects but not that it is necessarily effective.

[21] "The results of our study should be interpreted with caution", (Schonewille 2005)
"Our study is observational and has all the limitations of a non-randomised study. The interpretation of our results is hampered by the absence of a treatment protocol for all patients who entered the study" (Schonewille 2009).

(c) There are no trials comparing the effects of AT therapy (or just aspirin) on the one hand and a placebo on the other – though Professor Wills said that this undermines confidence in the literature a little, but should not materially affect the position as far as the Court is concerned.

100. The starting point is that aspirin affords a small absolute benefit (around 1%) in patients with acute ischemic strokes and causes fewer haemorrhages than does heparin. This statement comes from the review paper by Mattle and others (2011)[22]. The footnote in Mattle refers to the International Stroke Trial (IST) paper (1997)[23] for this information. The IST paper does not specifically mention a 1% figure. Further the IST paper deals with acute ischemic strokes overall and not BAO strokes. In this context it does make the following statements:

> "Aspirin v Avoid Aspirin
>
> ….Among aspirin – allocated patients there were non – significantly fewer deaths within 14 days (9.0% vs 9.4%), corresponding to an absolute reduction of 4 (SD4) per 1000 patients…
>
> Outcome at 6 months…At 6 months, there were fewer deaths among aspirin allocated patients but the absolute decrease of 10 (SD6) per 1000 was not significant…
>
> Sub Group Analyses…Analyses of the effect of aspirin sub-divided by the prognostic score indicated greater benefit among good prognosis patients but the trend was not significant…..
>
> Aspirin
>
> The effects of immediate aspirin use in acute ischaemic stroke on the unadjusted primary outcomes in the IST were not significant…since most patients with acute ischaemic stroke are likely to benefit from long term antiplatelet therapy, the IST and CAST results for the safety (and slight additional benefit) of giving aspirin immediately in acute ischaemic stroke are reassuring…
>
> Clinical Implications
>
> Because the evidence on aspirin is based on 40,000 randomised patients (IST and CAST),[24] it is more reliable than that for heparin. The benefit from the IST and CAST, of about 10 deaths or recurrent strokes avoided per 1,000 patients treated with aspirin in the first few weeks is about the same size as the benefit per year from long term aspirin treatment in stroke survivors…"

[22] Basilar Artery Occlusion; Lancet Neurology
[23] The Lancet; May 31 1997. The International Stroke Trial (IST): a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19,435 patients with acute ischemic stroke.
[24] Chinese Acute Stroke Trial

The IST paper examined a large number of acute ischaemic stroke patients, but of the cohort only a small percentage received treatment within 3 hours (4%) or 4 – 6 hours (12%)[25], only 5% were aged under 50 and only 12% were posterior circulation strokes (it is unknown what percentage of this 12% were BAO). Because of these factors the IST paper, referred to in Mattle, is no more than a starting point. It tells us little about the effect of aspirin on BAO strokes.

101. The first paper specific to BAO strokes to consider is that of Schonewille and others[26]. This paper considered 82 patients who had had BAO and had been treated conventionally i.e. had not been thrombolysed, but treated with antiplatelets, anticoagulation or both. The patients had been treated in three academic hospitals with a special interest in stroke in an 11 year period ending in 2002. In common with other papers, to evaluate outcome Schonewille used mRS. The authors stated:

> "Most studies …have used a modified Rankin Score of 0-1 or 0-2 to define good outcomes. Because of the poor natural history of Basilar Artery Occlusion we used a modified Rankin Score of 0-3 as a measure of independence and good outcome."

Therefore the outcome results were in three groups:

mRS 0-3, mRS 4-5 and death.

102. The overall outcome figures were in relation to the total patients numbering 82:

0-3: 17 (21%)

4-5: 32 (39%)

Death 33 (40%)

On these bald figures, the chances of even a 0-3 mRS outcome is only 21%.

However there are a number of variables to which C1 points. The difficulty with these variables is that it is not possible to evaluate them by their combined effect. Looking at table 2 "Predictors of Good Outcome":[27]

(i) 35 of the 82 patients were under 60. Of those 11 (31%) achieved a "good outcome" (mRS 0-3). This was still well below 50% but more likely than for patients over 60.

(ii) Exactly the same numbers applied to patients presenting with a minor stroke. "Minor" in the context of this paper was "non comatose and not tetraplegic, intubated or locked-in at time of presentation."

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[25] Of course it is not known when during the night prior to 7am C1's symptoms first began

[26] Journal of Neurological and Neurosurgery & Psychiatry 2005

[27] Patients were admitted to the study if they fulfilled at least one of 3 diagnostic criteria (A) – (C). These all suggested a worse initial presentation than C1 and the only one possibly relevant to her was Group (A) "acute neurological deficit attributable to the posterior circulation and basilar artery occlusion". If, as may well be the case, "occlusion" means full occlusion, then C1 at Medway had only partial occlusion.

(iii) Fluctuating strokes were present in 13 patients. They did marginally better than those who did not have fluctuating strokes (23% good outcome compared with 20% for those with non fluctuating strokes).

103. It is to be noted that 95% confidence intervals (CI) were very wide on all these sub groups, which is not surprising given the low number. Nevertheless:

(a) Those under 60 and those presenting with minor strokes were assessed to have an odds ratio (OR) of 3.1 compared to the patients in those categories.

(b) Yet in each group their chance of a 0-3 mRS outcome was still only 31%.[28]

104. The second paper is also by Schonewille and others and dates from 2009[29]. This paper considered 592 patients who had BAO. Of these 183 had been treated with AT. In this paper that was antiplatelets or systemic anticoagulants. The remainder were treated with IVT (thrombolysis) or IAT (thrombolysis, mechanical thrombectomy, stenting or a combination). This paper is based on what is known as the BASICS study and the patients' registry in Utrecht ran from 2002 to 2007. Patients were categorised as "mild to moderate" at time of treatment if they were not in a coma, or tetraplegic or in a locked-in state. If they were any of these, they were "severe". The mild to moderate deficit patients had a mean NIHSS score of 10.7, though this was for all patients, not just those treated with AT and table 1 shows that the NIHSS score of AT treated patients was lower than that of the patients on IVT or IAT.

105. The main purpose of the 2009 paper was to try to compare outcomes of the three different types of treatments. The paper is heavily qualified in the last two pages, with references to the fact that it is an observational study with all the limits of a non randomised study, together with numerous other possible biases/variables which could not be eliminated or catered for.

106. C1 relies on the 2009 paper for the information in the Figure. This shows that there were 104 patients treated with AT who were in the mild to moderate category at time of treatment. Their mRS outcome was:

mRS 0	10	(9.6%)
mRS 1	19	(18.3%)
mRS 2	9	(8.7%)
mRS 3	22	(21%)
mRS 4/5	31	(29.8%)
Death	13	(12.5%)

[28] C1 submitted that Dr Allder had said that because the OR was 3.1, that, being more than 2, gave a greater than 50% chance of a good outcome. Nobody else had this note of his evidence. If he did say it, I reject it as inconsistent with only 11/35 in each subgroup achieving a good outcome. It may be that this submission is based on a confusion of relative risk and odds ratio. The two concepts are statistically distinct.

[29] Lancet Neurology August 2009. This was a prospective study, not a retrospective study which Schonewille 2005 was; also the cohort was larger. However, these were not suggested by the experts to be significant in evaluating the evidence.

Total	124	(99.9%)

Therefore 36.6 achieved an mRS outcome of 0-2 and 22% an mRS outcome of 3 making a total of 58.6% who achieved an mRS outcome of 0-3.

107. The comparison of those presenting with a severe deficit is quite stark. Those figures are:

mRS 0-3	(6/79)	7.6%
mRS 4-5	(30/79)	38%
Death	(43/79)	54.4%

108. To the extent that direct comparison between Schonewille 2005 and Schonewille 2009 is possible, the statistics suggest a massive improvement since, as we have seen, minor strokes achieved a good (mRS 0-3) outcome in only 31% in the 2005 paper as opposed to 58.6% in the 2009 paper.

109. Further the overall (i.e. mild to moderate together with severe) figures suggest a marked improvement from the 2005 to the 2009 papers as follows:

Year	mRS	%	mRS	%	Deaths
2005	0-3	17%	4-5	32%	33%
2009	0-3	36% (66/183)	4-5	33% (61/183)	31% (56/183)

(Other figures are contained in the paper of Mattle and others. This review paper, amongst other things, compares the two Schonewille papers in table 4. However it compares only the overall 2005 Schonewille figures with the 2009 mild to moderate figures and thus shows an even starker contrast).

110. Professor Wills addressed the change in outcome statistics between the two Schonewille papers. Professor Wills said the apparent improvement in outcome in the two studies[30] is probably explained by the fact that the period of patient recruitment was much later in the 2009 studies. The therapeutic effect of the AT drugs cannot have changed. Professor Wills's best estimation[31] was that it would be the evolution of stroke units over the two periods which has made the difference. In other words, the general overall specialist treatment which was being received by patients in the middle of the first decade of the millennium compared to those in the 12 year period finishing in 2002. One might assume that as C1 was admitted in 2010 therefore she should be entitled to be classed amongst the 2009 figures in terms of the effect of AT therapy. To this Professor Wills gave a convincing response. He said that the 2005 paper assessed outcome at time of discharge or in-hospital death. The 2009 paper assessed outcome at 1 month. C1 had the benefit of the more modern stroke unit

[30] Assuming the studies are reliable and I note again the caveats of the authors
[31] Dr Allder was not asked about this point

treatment.[32] The only benefit she did not have is that she was not prescribed AT treatment (particularly aspirin) at Medway. Therefore she cannot pray in aid the advantage bestowed by the improvement of the 2009 figures. Unfortunately her outcome was not a good outcome despite her more modern stroke unit treatment at KCH. For this reason I accept that on balance the 2005 Schonewille outcome figures in relation to the effect of aspirin are more likely to reflect the likely consequences of AT had it been prescribed to C1 at Medway.

111. Looking therefore at the 2005 Schonewille paper, although age and "minor" stroke (and to a much lesser extent fluctuating stroke) were predictors of good outcome, individually they were all well below 50% in producing an outcome of mRS 0-3. There are no statistics to deal with the situation of a person with the three favourable predictors. Nor do we know the number of patients, if any, who were common to more than one of the sub groups. Although it is possible that the combined effect would be to produce a more than 50% chance of a mRS 0-3 outcome, this in my judgment is impressionistic only and lacks any proper scientific/statistical basis. It may or may not be correct. Nor does the fact that C1 was aged 42 years, and therefore well under 60, or that her symptoms were at the very mild end of the spectrum at 8:35am and only moderate at 11am, provide any proper sound warrant for finding that the chances of a 0-3 mRS outcome exceeded 50%.[33]

112. In the Mattle paper (Table 5) there are 7 predictors of favourable outcome and 7 predictors of unfavourable outcome. Professor Wills accepted that at 8.35am C1 had 5 favourable predictors according to that table[34]. The other two favourable predictors[35] were unknowns. There was only 1 out of 7 unfavourable predictor namely dysarthria. It must be remembered that Mattle is a review paper. Apart from the two Schonewille papers, there are footnote references to papers which have not been examined in court. Mattle provides, in my judgment, no proper basis for a quantative assessment of C1's probable outcome had she been prescribed aspirin at Medway. As Professor Wills said, the paper is not subtle enough to give a percentage outcome. Further, the text in Mattle in relation to anticoagulation agents is not encouraging in relation to a submission that C1 probably would have had a favourable outcome. I shall refer to this text later in the judgment.

113. Dr Allder estimated that because of C1's favourable predictors, if C1 had been given aspirin at Medway at 8.35am, she would have had a 58% chance of an outcome of mRS 0-1. If aspirin had been given at 11am then he estimated that C1 would not have had a 0-1 or 0-2 mRS outcome but probably a mRS 3 outcome. Given that there is no clear literature to support these figures, the assessment appears to be based first on what I have referred to as an "impressionistic" evaluation of the Schonewille 2009 figure statistics and the fact that at 8.35am C1 was at the very mild end of the symptom spectrum, whereas at 11am her symptoms were moderate. I have already rejected the 2009 paper as a sound basis for considering C1's case; nor do I accept

[32] Wills said that AT is only one aspect of the holistic package on a specialist stroke unit. Patents can improve even after total BAO stroke e.g. because other parts of the brain take over the functions of the damaged part; or other therapies, e.g. speech therapy, can be significant.

[33] Though Professor Wills accepted that logic might suggest an increased chance of a better outcome in these circumstances.

[34] Younger age, minor stroke, mild to moderate deficit, acute onset and single – sector stroke.

[35] Feeding arteries to the posterior circulation on mR angiography and reversed Basilar flow

that I can extrapolate an even more favourable outcome from C1's symptomatology or other favourable predictors to give me any confidence that Dr Allder is correct. He may be, but I do not find it to be convincing on the balance of probabilities.

114. Of course, Dr Allder also relied on his theory as to aetiology. I add the following comments as to why this theory does not persuade me:

(i) There is no literature on stenosis/partial occlusion of the basilar artery and its effects so as to support Dr Allder's theory.

(ii) Dr Allder accepted that he could not quantify his theory that stenosis/partial occlusion, treated by aspirin, would produce a better outcome.

(iii) Therefore even if correct, the theory only really explains how aspirin might produce a better outcome in a case where it is administered at a time where there is only stenosis/partial occlusion of the basilar artery. It does not assist in the assessment of the extent to which it does have that effect. Schonewille 2005 tells us that an mRS 0-3 outcome is achieved after administration of AT only in 31% of patients presenting with a "minor" stroke and 23% of those presenting with a fluctuating stroke. Therefore the substantial majority of each sub group does not achieve an mRS 0-3 outcome despite the administration of AT.

115. Dr Allder's theory, and it is no more than a theory, is not given any support from the information we have in the literature as to the potential outcome for C1 had aspirin been administered at Medway.

116. There is the further feature that even if the literature showed that AT administration would probably have led to a better than 50% chance of an mRS 0-3 outcome in C1, the literature does not distinguish between aspirin and heparin as the two main forms of AT. There is no evidence to show the effect of aspirin only in BAO strokes. Professor Wills said that prior to thrombolytic treatment, he was trained to use heparin, as KCH did. He said that in the vast amount of strokes similar results are produced by heparin and aspirin but the vast amount of strokes are not embolytic BAOs. Dr Allder did not accept that it is heparin, not aspirin, that is more responsible for producing such positive effects as the Schonewille papers show.

117. Had I been persuaded of C1's case taking into account the expert evidence, the literature and in particular the Schonewille papers, I would not have found that there was sufficient evidence to distinguish between the effects of heparin and aspirin in relation to favourable outcome. Again Professor Wills may be correct but this point alone would not have swung the causation balance in favour of Ds.

118. I will make brief reference to a paper by Shahpouri and others (2012)[36]. This review article comes from Iran. It deals with ischaemic stroke as a whole and not specifically BAO strokes. In the context of acute ischaemic strokes the difference between patients treated with heparin versus placebo showed no improvement in overall outcome at 3 months.[37] Sub group analysis suggested a higher rate of favourable

[36] Journal of Research in Medical Sciences: anticoagulant therapy for ischemic stroke: a review of literature.
[37] In progressive strokes generally therefore this paper suggests heparin doesn't halt neurological worsening (though note that it was the drug of choice for C1 at KCH). Also in cervical artery dissection there were no data to support the therapeutic superiority of anticoagulants over antiplatelets.

outcomes in patients treated with heparin who had a large artery atherosclerotic stroke. Professor Wills said, and I accept, that although the basilar artery is a large artery, one cannot use these figures for BAO strokes. As the Mattle and Schonewille statistics show, the overall outcome of BAO strokes even after AT therapy is not good.[38]

119. Professor Wills was asked about a paper by Redekop[39] (2008). Again this is a review article. In relation to the point Professor Wills made about heparin, he was asked about the statement that "the Canadian Stroke Consortium reported a non randomised pilot study comparing anticoagulation and aspirin in 116 patients with carotid or vertebral dissection, and found no significant difference in stroke rate." He accepted that aspirin and heparin in this context are of equal efficacy but said that the paper did not show they were of equal efficacy when the vertebral artery dissection had led to BAO. I accept this, but this does not affect my view that I am not persuaded that there are probably different effects of aspirin and heparin in respect of BAOs.

120. I began this section with a general point made about the IST study which dealt with the whole range of strokes and was pessimistic about the effect of AT. I have then dealt in some detail with the literature and expert evidence and concluded that C1 has not proven on the balance of probabilities that her outcome would have been mRS 0-3 had aspirin been prescribed either at 8.35 or 11am at Medway. Reverting solely to the literature, although I am fully aware, as I hope I have made clear throughout this section of the judgment, that there are numerous variables and, most particularly, a number of the papers do not refer to BAOs, nevertheless the overall tenor of the efficacy of AT in stroke treatment is not encouraging. In this regard there are the following additional extracts from the literature:

(i) The Mattle paper

> "Aspirin affords a small absolute benefit (around 1%) in patients with acute stroke and causes fewer haemorrhages than does heparin. However comparative studies of antithrombotic verses placebo in people with BAO are not available. In hospital based series of patients with BAO treated with antithrombotics outcomes were good in 20% to 59% of patients; however, in a large series, case fatality was still 40%. Among survivors, 65% remained dependent (mRS 4-5). Good outcome (mRS 0-3) was reached by only 20%. Table 5 lists predictors of outcome from these series."

(ii) Lohse and others (2011)[40]

> "Recent research has suggested that for patients with unambiguous basilar artery thrombosis, the only promising treatment option is early thrombolytic treatment…

[38] In any event it appears to me that an increase in large artery strokes of 68% favourable outcome with heparin compared to 55% with placebo suggests that only 13% of those patients do better with heparin AT than if they were not treated at all.
[39] Canadian Journal on Neurological Science: extract cranial carotid and vertebral artery dissection: a review
[40] The Open Critical Care Medicine Journal: Clinical Diagnostics, Therapy and Outcome after Basilar artery thrombosis

> Nowadays the prognosis of basilar artery thrombosis is still poor. Thus, it is common sense that nearly any promising intervention appears justified, because of the otherwise disastrous outcome..…
>
> The natural course of basilar artery (thrombosis) is often lethal and even survivors bear a high risk for mortality and morbidity…"

(iii) Redekop (2008)

> "Both anticoagulation and antiplatelet agents have been advocated as treatment methods, but there is limited evidence on which to base these recommendations."

(iv) Schonewille (2009)

The conclusions of this paper refer to the efficacy of IVT (primary intravenous thrombolysis) and IAT (intra-arterial therapy). There is no consideration, despite the statistics in the paper, of recommending AT therapy for BAO stroke. The conclusion is:

> "Our observations underscore the continued absence of a proven treatment modality for patients with an acute BAO and that current clinical practice varies widely. Furthermore, the often held assumption that IAT is superior to IVT in patients with an acute symptomatic BAO is challenged by our observations. Therefore, we believe that a randomised controlled trial to compare IVT with IAT in patients with acute symptomatic BAO is a high priority.
>
> In the meantime our results should encourage clinicians to treat patients who have acute symptomatic BAO and a mild to moderate deficit with IVT. In case of subsequent acute worsening, additional IAT can be considered…"

121. In summary, basing myself upon an analysis of the evidence from Dr Allder and Professor Wills and the medical literature in this area, I cannot be persuaded on the balance of probabilities that had C1 been prescribed aspirin at 11am (or even prior to 9am) at Medway she probably would have achieved an mRS of 0-3. Her chances would have been somewhat improved but, on the best evidence available, I find that on the balance of probabilities she unfortunately would not have had any better outcome than in fact was the case.

122. Both Counsel addressed me on the balance of probabilities and sought a ruling on that basis. As an alternative C1 submitted that potentially she could rely upon the case of Bailey v Ministry of Defence[41]. At paragraph 46 Lord Justice Waller said:

> "In my view one cannot draw a distinction between medical negligence cases and others. I would summarise the position in

[41] [2008] EWCA Civ 883

> relation to cumulative cause cases as follows. If the evidence demonstrates on a balance of probabilities that the injury would have occurred as a result of the non-tortious cause or causes in any event, the claimant will have failed to establish that the tortious cause contributed. *Hotson* exemplifies such a situation. If the evidence demonstrates that 'but for' the contribution of the tortious cause the injury would probably not have occurred, the claimant will (obviously) have discharged the burden. In a case where medical science cannot establish the probability that 'but for' an act of negligence the injury would not have happened but can establish that the contribution of the negligent cause was more than negligible, the 'but for' test is modified, and the claimant will succeed."

123. The present case can be decided on the balance of probabilities. In my judgment the evidence demonstrates that on a balance of probabilities, even if C1 had been prescribed aspirin at any time at Medway, her outcome would have been the same. Therefore C1 has not proven on the balance of probabilities that the Defendants' breach of the duty of care in failing to treat her promptly with aspirin caused or materially contributed to her eventual outcome.

C2's Claim: His witness evidence

124. C2 was with C1 throughout the day from the moment when he followed the ambulance to the Medway Maritime Hospital (arriving a matter of minutes after her) until late that night, he estimated around midnight, when he left KCH. There was a period at KCH on arrival when he did not see her for a couple of hours. When he left KCH he went to his parents' house, stayed overnight and was intending to take his son back to Oxford. He was then going to return to KCH. He had just got through the Dartford Tunnel when he received a telephone call from a doctor at KCH telling him that his wife had deteriorated and to come back straight away which he did, arriving at about 1pm – 2pm.

125. C2 gave evidence as to how his experience on the Sunday and Monday affected him. In broad terms I find his evidence to be accurate and reliable. There are certain aspects where, in comparison with the notes, I believe that he is mistaken as a result of the pressures of those days and the passage of time.

126. Whilst at Medway Hospital C2 said he noticed his wife deteriorating around 09:40am. He saw it himself. After that his levels of frustration increased rapidly. He said his wife was deteriorating in front of him and it was if they were being ignored. He felt that something should be happening and his fear was that if she deteriorated further and faster it would be too late. Later, when C1 was handed back to Doctor Ramphele and he discharged her, C2 was incredulous. He did not know how Doctor Ramphele could say she wasn't having a stroke. By 11:30 – 12 noon C1 could make noises but not speak properly. He said he did not think that later her speech improved but he understood it more. C2 said that when they left Medway C1 could not get into the wheel chair and he could not do it without the assistance of his father. I accept the evidence about the wheelchair but Dr Baines effectively said that because of the Darent Valley records, C1 was not in such a poor state that, when she left Medway,

she could not move either arm or leg and could only move her eyes and could not speak reasonably clearly.

127. When they left Medway C2 decided to go back to his parents intending then to continue to Oxford and the John Radcliffe Hospital. Something changed his mind about going to Oxford. C1 told him that she remembered somebody saying it would take too long for them to go to Oxford and they should go to the nearest hospital. They therefore went to Darent Valley Hospital. They arrived some 2 hours after leaving Medway Hospital.

128. Later on the drive to Darent Valley Hospital she deteriorated further. He said he could hear her choking. She could not alert him to the fact that something was wrong because she could not move or speak. He said this was the worst moment. He thought she was dying. He had to remove a bit of biscuit from her mouth because she could not swallow it. She was slumped in the front seat, seemingly lifeless and choking, with her head on one side and her eyes wide open. She was panicking. [I find that this incident of choking did happen. She may well not have been able to swallow something as large and hard as a biscuit.].

129. C2's recollection was that at Darent Valley the staff recognised the stroke straight away and before he knew it she was in the ambulance to KCH. He accepted that the records show that she was there for something more than an hour, however. He said that at Darent Valley he thought that that was the last time he was going to see her. She was unable to move except her eyes. She wanted to communicate but could not. He was trying to talk to her and calm her. It was difficult for him to put into words how bad he felt and how useless he felt.

130. The Glasgow Coma Scale (GCS) score at Darent Valley was 15 out of 15. This suggests that at this stage C1 was apparently able to answer questions and was fully orientated. The examination in the clinical notes at Darent Valley does not suggest that C1 was as bad as C2 recalls. There is reference to right sided facial droop with pupils being equal and reacting (C2 referred to C1's eyes being all over the place). Right sided weakness was noted with muscle power in the right upper limb and right lower limb at 1/5 but in the left upper limb and left lower limb at 5/5. Therefore, at this stage, I find that C2 was in a poor state but that she had right sided weakness rather than bilateral weakness.

131. From Darent Valley C1 was taken to KCH in an ambulance. C2 did not see her there for a couple of hours after he arrived. He said it seemed like an eternity. He was expecting the doctor to come and tell him that his wife had passed away. The next day when he returned having received the telephone call once he had exited the Dartford Tunnel, C1 was ashen. C2 thought he was too late and was as if his whole world had fallen through the floor. He said he found it difficult to put into words. He was devastated.

132. Similar comments can be made about C1's condition on arrival and during the initial hours at KCH, as I have made above in relation to Darent Valley. Again C2's recollection was that C1 could not move. Also that she could not really communicate. The clinical notes at KCH from Dr Bercocal/Dr Haider (untimed) record slow speech that seemed to be more intense dysarthria and no dysphasia. It is recorded that C1 made no mistakes in the naming of colours/objects. The GCS score was 15 out of 15.

Again the neurological deficit was on the right side, being complete facial palsy, no movement at all in the right limbs and hypaesthesia on the right side with the plantar right up going. On examination C1 tended to sleep but awoke with minimum stimulation.

133. In summary, I broadly accept C2's recollection as to C1's state save that her condition was not as bad as he recalls. On the Sunday her neurological deficit was overwhelmingly right sided rather than bilateral, her speech was slurred and the GCS scores were normal. Nevertheless she was very poorly and deteriorated during the day.

C2's Claim in the Light of Ds' Breaches of Duty

134. It must be recorded that it is common ground that C2's claim can succeed despite the failure of C1's claim on the issues of causation. This is subject only to the issues as to whether or not C2 satisfied one of the "control mechanisms" applying to secondary victims, and to causation in his case. I shall first set out how C2's claim is put in the light of my findings in relation to C1.

135. In this judgment I have explained that Ds were negligent in failing to prescribe aspirin shortly after the 09:40 deterioration. It has always been admitted that Ds were negligent in discharging C1 and not admitting her to hospital. In addition I accept that from about 09:40 C2 kept on asking some staff in A and E why they were not sending C1 for a scan. In short it is said that C2 suffered psychiatric injury because C1 was not properly treated and was then discharged. Had she been properly treated she would have been admitted to Medway, C2 would not have found himself thinking about driving to Oxford, then changing his mind and also witnessing his wife's state on discharge and then in the car, including the choking incident. None of this, it is said, would have taken place, absent Ds' breach of duty towards C1.

136. What is the medical evidence relevant to these issues? Some extracts from the reports of Dr Tattersall and Dr Campbell should be cited:

(i) Dr Tattersall:

> "5. My instructions arise following Mr Owers' wife suffering a stroke on 14 March 2010, followed by a delay in this being diagnosed in the Accident and Emergency Department at Medway Hospital resulting in urgent treatment to limit the extent of the damage this caused then not being given. This event is referred to within this report as "the Index Event"…"

(In relation to the discharge)

> "13. Mr Owers said that with some difficulty he was able to transfer his wife into their car using a wheelchair and initially drove to his parents' home. However, his wife was continuing to deteriorate and he was increasingly anxious about the choking, so he drove her to another hospital…"

"20. As Mr Owers described these post traumatic stress type symptoms, I explored in further detail his experience of the broader range of symptoms of post traumatic stress disorder, as follows:

(a) Mr Owers experienced in the index incident an event that threatened his wife with death, and resulted in her developing an enduring severe and life changing disability.

(b) Mr Owers said that during the index incident[42] and its immediate aftermath, his emotional response was of shock, horror and helplessness. He said that an (*sic*) initially he had felt "very scared" when he realised that his wife was having a stroke, and then increasingly frustrated, angry and hopeless at the delay in any assessment or treatment. He said that the subsequent days when his wife became so ill that she might have died was a very frightening time for him, and he also was stressed by the uncertainty regarding her prognosis during the subsequent months of rehabilitation.

(c) Mr Owers described experiencing recurrent intrusive and distressing recollections of the index incident occurring in the form of "a video loop" of his experience (as if seen through his own eyes at the time) of waiting for his wife to have appropriate and effective assessment and treatment in the Accident and Emergency Department at Medway Hospital, associated with feelings of frustration he had felt at the time. He said that these reliving "flashbacks" occurred every day, usually in the evenings when he feels tired…"

(ii) Dr Campbell:

"2. Post Traumatic Stress Disorder (DSM V Criteria)

A) Witnessed his wife suffering from severe illness which could have been fatal. Mr Owers was additionally traumatised by the failure of Medway Maritime Hospital to correctly diagnose his wife and has he understands it to instigate proper treatment…"

" Causation…

Precipitating Factors

- The trauma of the events of the 14 March 2010, namely the rapid onset of a serious life threatening illness of his wife.

[42] As previously described in paragraph 5 of his report (see above)

- The knowledge that during her 4 hours in Medway Maritime Hospital they had failed to diagnose her condition.

- Failure to treat his wife's illness in Medway Maritime Hospital.

- Premature discharged (*sic*) her despite her physical deterioration.

It was the intensity of this distress which acted, on the balance of probabilities, to cause his post traumatic stress disorder and depressive illness.

137. The best indication as to causation, separating out the very serious illness of C1 from the failure by Ds properly to treat her and admit her to hospital is dealt with by Dr Campbell in this way:

> "I have been asked to comment on the following scenarios. The likely outcome to Mr Owers if:-
>
> Mrs Owers had been diagnosed at the Medway Maritime Hospital with a pontine infarct type stroke.
>
> - This was not treatable.
>
> - She had continued to develop the same physical complications.
>
> In this case Mr Owers would still have been very upset by his wife's sudden illness, but would not have suffered the trauma of a missed diagnosis, no discussion of any treatment options and a premature discharge in a deteriorating physical condition.
>
> In as much as it is possible to say, in retrospect in this scenario he is likely to have developed the lesser diagnosis of an Adjustment Disorder with depressive symptoms, and not PTSD.
>
> If she had gone on to develop the same physical disability he would still, on the balance of probabilities, have developed a major depressive episode from the initial adjustment disorder."

138. Therefore I find on the balance of probabilities that the Ds' breach of duty towards C1 caused C2 to suffer from the PTSD. He would in any event have suffered depression and an Adjustment Disorder.

C2's Claim: The Legal Background

139. Claims based on psychiatric injury following witnessing clinical negligence in respect of a loved one have recently been considered in two cases. The first was Shorter v Surrey & Sussex HC NHS Trust [2015] EWHC 614 QB; the second Liverpool

Women's Hospital NHS Foundation Trust v Ronayne [2015] EWCA Civ. 588. Both deal extensively with the authorities. I shall start with the Shorter case, a decision of Swift J.

140. In Shorter the deceased suffered a subarachnoid haemorrhage (SAH). The claim was brought by her elder sister as a secondary victim. The Claimant was aware that her sister had suffered a bleed to the brain on the morning of the 12 May 2009, having been telephoned by her sister's husband. She was panicky and knew that her sister was in the danger zone. She was present with her sister from late morning that day, following her sister's admission to East Surrey Hospital, until the evening when she left to make child care arrangements. During this period her sister was in some pain and the Claimant, who had specialist knowledge of neurological patients and SAH, became increasingly worried and frustrated about the failure to transfer her sister to a specialist unit and to give her nimodipine that had been prescribed hours earlier. At 22:00 hours she was "terrified" by a telephone call that her sister was still at East Surrey. At about midnight she was told of her sister's first seizure at St George's Hospital. This raised her level of anxiety. A subsequent call that there had been more seizures and, probably, a re-bleed made her "absolutely terrified". The Claimant went back to St George's Hospital, saw her sister in the ITU and had distressing discussions with doctors, until finally her sister was confirmed brain dead at 12:45pm on 13 May 2009. None of this should have happened as there had been a negligent interpretation of a CT scan at East Surrey on 5 May 2009.

141. Swift J set out the prerequisites for liability to secondary victims deriving from the decisions of the House of Lords in McLoughlin v O'Brian [1983] 1AC 40 and Alcock v Chief Constable of South Yorkshire Police [1982] 1AC 310. The essential element in the present case, as in Shorter and Ronayne, is the proximity to a shocking event. In Alcock Lord Oliver said this (416E – 417A):

> "The necessary element of proximity between plaintiff and defendant is furnished, at least in part, by both physical and temporal propinquity and also by the sudden and direct visual impression on the plaintiff's mind of actually witnessing the event or its immediate aftermath…"

At page 401F Lord Ackner said that the element of "shock" involved:

> "The sudden appreciation by sight or sound of a horrifying event, which violently agitates the mind. It has yet to include psychiatric illness caused by the accumulation over a period of time of more gradual assaults on the nervous system."

142. Having referred to other accident cases on this point, and in particular Galli-Atkinson v Seghal [2003] 1 Lloyd's Rep Med 285 at paras 25 – 27, Swift J turned to the trio of claims by secondary victims following negligent medical treatment, namely Taylor v Somerset Health Authority [1993] PIQR P262, Sion v Hampstead Health Authority [1994] 5 Med LR 170 and North Glamorgan NHS Trust v Walters [2002] EWCA Civ. 1792.

143. In Shorter the Claimants relied upon Walters where the Court of Appeal had found that once the "event" started, a period of 36 hours could be regarded as a "single

seamless event". The Defendant argued that Walters was distinguishable because the mother had woken up and witnessed her son rigid, bleeding and choking and therefore there was a "sudden appreciation of the horrifying events". In Shorter the Defendant said there was no sudden "horrifying event". The Claimant's realisation came on gradually as a result of telephone calls, her own concerns and the events at hospital. It was submitted that the Claimant did not have the required degree of proximity to a specific and shocking "event".

144. Swift J accepted that the Claimant "must also show that her psychiatric illness was caused by the sight or sound causing an assault to her senses. She must establish sufficient proximity to the event, a sudden and direct visual impression on her mind of witnessing the event or its aftermath." (Paragraph 202). She also said that cases of clinical negligence present particularly difficult problems. The factual background of cases can be very different and often quite complex. The nature and timing of the "event" which the breach of duty gives rise will vary from case to case (paragraph 209). The nub of her decision can, I believe, be found in the following extracts:

> "213…I do not consider that, at the time, the sight of Mrs Sharma had the visual effect on the Claimant which was later described. In the case of Walters, the trial judge and the Court of Appeal laid considerable emphasis on the start of the "event", when the mother awoke to find her baby rigid and choking after a convulsion, with blood pouring out of his mouth. Ward LJ likened that to the "assault upon her senses" the mother would have suffered if she had seen her child bleeding in a seat after a road traffic accident. That sort of "assault upon the senses" is, it seems to me, of a very different order to the scene in the A & E Department at ESH on 12 May. Indeed, even if Mrs Sharma had for a short time been in the state described by the Claimant, I do not consider that the sight would have come within the type of "event" described in *Walters* and the other relevant authorities. Mrs Sharma's condition was fluctuating; she did not have obvious injuries; she was not – or at least did not appear at that stage to be – in any obvious or immediate danger…"

(In paragraphs 216 and 217 Swift J pointed out that during a period of 9 hours overnight at St George's, the Claimant did not see her sister and was not proximate to the events that were unfolding and it was not until she saw her sister on the life support machine that the reality became clear. That must have been deeply upsetting but was not "a sudden or unexpected shock")

> "218… It does not seem to me that what happened in this case can properly be described as a "seamless single horrifying event". There was a series of events over a period of time. The Claimant was proximate to some of those events, during the periods spent in ESH and SGH. However, much of her fear, panic and anxiety were caused by information communicated to her by telephone, or face-to-face by Mr Sharma, when he told her that her sister had "gone". I do not consider that any of the individual events within the series actually witnessed by the

> Claimant gave rise to the sudden and direct appreciation of a "horrifying event". Even when she witnessed her sister on the life support machine, her perception was informed by the information she had been receiving over the previous 15 hours or so and by her own professional knowledge. Mrs Sharma did not have the type of injuries suffered by the deceased in Galli-Atkinson, was not in obvious pain and had not been pronounced dead at that time. In the circumstances, it does not appear to me that the sight of her can be regarded as a "horrifying event"; nor was it sudden or unexpected."

145. Finally, I turn to Ronayne. In that case, over a period of 24 hours, the Claimant observed a rapid deterioration in the condition of his wife, manifested most vividly in two distinct episodes:

(a) At about 5pm on 18 July the Claimant saw his wife connected to various machines

(b) Sometime on the following day he observed her in the post operative condition unconscious, connected to a ventilator and being given four types of antibiotic intravenously. Her arms, legs and face were very swollen. Pressure pads were in place. Later he described his wife's then appearance as resembling the "Michelin Man".

Mrs Ronayne's condition on 18 and 19 July 2008 were as a consequence of the Defendant's negligence.

(Paragraphs 3 and 4).

146. At paragraph 13 the Court of Appeal agreed with what Swift J had said at paragraph 214 of Shorter and in particular "That the question whether an event is for these purposes to be recognised as in the relevant sense "horrifying" must be judged by objective standards and by reference to persons of ordinary susceptibility." The Court of Appeal also endorsed observations of Judge Hawkesworth QC in Ward v Leeds Teaching Hospital NHS Trust [2004] EWHC 216 (QB) where at paragraph 21 he had said (amongst other things):

> "…An event outside the range of human experience, sadly, does not it seems to me encompass the death of a loved one in hospital unless also accompanied by circumstances which were wholly exceptional in some way so as to shock or horrify…To describe an event as shocking in common parlance is to use an epithet so devalued that it can embrace a very wide range of circumstances. But the sense in which it is used in the diagnostic criteria for PTSD must carry more than that colloquial meaning."

(The Court of Appeal pointed out that that was said in the context of a determination where the PTSD had been suffered as opposed to a severe and prolonged bereavement reaction, but the same principles apply to an assessment whether an event could be properly characterised as shocking as intended by Lord Ackner in Alcock).

147. The Court noted that the only case of which Counsel were aware where the Claimant had succeeded at trial, in a claim in consequence of observing in a hospital setting the consequences of clinical negligence, was Walters. At paragraph 17 Tomlinson LJ said:

> "That is in my view unsurprising. In hospital one must expect to see patients connected to machines and drips, and…expect to see things that one may not like to see. A visitor to a hospital is necessarily to a certain degree conditioned as to what to expect, and in the ordinary way it is also likely that due warning will be given by medical staff of an impending encounter likely to prove more than ordinarily distressing."

148. The Court of Appeal then dealt with Walters and the facts of the Ronayne case. The appeal was allowed in favour of the Defendant Hospital Trust on the basis that there was no "sudden appreciation of an event" and that what the Claimant saw was not "horrifying by objective standards." I find the following citations to be helpful:

> "35. …It was not, like Walters, "a seamless tale with an obvious beginning and an equally obvious end." In Walters the obvious beginning was the mother awakening to see her baby rigid and choking after a convulsion, with blood pouring out of his mouth. The obvious end was the tragic death of the baby in the mother's arms. The working out of the tragedy, with the raising of hopes, the journey up the motorway to London following in the wake of the ambulance, and the dashing of hopes and then their final destruction was almost Sophoclean in its seamlessness.
>
> 36. The present case is in my judgment not comparable, just as Swift J found the facts in Shorter not comparable. As there, so here, there was in my judgment a series of events over a period of time. There was no "inexorable progression" and the Claimant's perception of what he saw on the two critical occasions was in each case conditioned or informed by the information which he had received in advance and by way of preparation….
>
> 40. It follows that this was not in my judgment a case in which there was a sudden appreciation of an event. As Swift J found in Shorter, there was a series of events which gave rise to an accumulation during that period of gradual assaults on the Claimant's mind. Ward LJ in Walters contrasted what there occurred with a "gradual dawning of realisation that her child's life had been put in danger by the defendant's negligence," which would not have amounted to a sudden and unexpected assault on her mind. That in my judgment is an apt description of what here occurred – a gradual realisation by the Claimant that his wife's life was in danger in consequence of a mistake made in carrying out the initial operation. At each stage in this

> sequence of events the Claimant was conditioned for what he was about to perceive…
>
> 41. Furthermore what the Claimant saw on these two occasions was not in my judgment horrifying by objective standards. Both on the first occasion and on the second the appearance of the Claimant's wife was as would ordinarily be expected of a person in hospital in the circumstances in which she found herself. What is required in order to found liability is something which is exceptional in nature… I can readily accept that the appearance of Mrs Ronayne on this occasion must have been both alarming and distressing to the Claimant, but it was not in context exceptional and it was not I think horrifying in the sense in which that word has been used in the authorities. Certainly however it did not lead to a sudden violent agitation of the mind, because the Claimant was prepared to witness a person in a desperate condition and was moreover already extremely angry."

C2's Claim: discussion

149. In relation to the four control mechanisms applied to claims by secondary victims[43] Ds accept that C2 had a close tie of love and affection with C1, that he was close to the incident in time and space and that he directly perceived the incident. Apart from causation, upon which I have already ruled, the issue is whether C2's illness was induced by a sudden shocking event. As was said in paragraph 8 in Ronayne there are two interrelated points:

> "(a) Whether the events concerned were of a nature capable of founding a secondary victim case, i.e. were they in the necessary sense "horrifying"; and
>
> (b) Whether the sudden appreciation of that event or those events, i.e. shock, caused the Claimant's psychiatric illness."

150. I have set out in some detail the Court of Appeal's helpful exegesis of those points and also noted that the Court of Appeal laid emphasis on the fact that the only case of which Counsel were aware in that case of a Claimant succeeding in a hospital negligence case was Walters. The bar is set very high for secondary victim claimants in that context. The question is whether C2 has overcome that high bar.[44]

151. In my judgment, what was witnessed by C2 from shortly after 09:40am until C1 was discharged from Medway, and the incidents in the car before she eventually arrived at Darent Valley, were very distressing. However they were not "horrifying" as judged by objective standards and by reference to persons of ordinary susceptibility. They were not wholly exceptional. His wife was in the throes of a severe illness and C2 should have seen her admitted and looked after (irrespective of the eventual outcome).

---

[43] Set out at paragraph 10 of Ronayne,

[44] I take account of the fact that C2 suffered PTSD, which is an indicator (but not determinative) of a sudden shocking event. Also the psychiatric evidence shows that C2 was more vulnerable than the average person to developing PTSD.

After the deterioration of 09:40 he not only saw a failure properly to diagnose and treat, but also the negligent discharge of his wife who was by then, on any account, very seriously ill. In the aftermath, which should have been avoided had the Defendants acted non-negligently, he perfectly understandably gave her a biscuit to eat and witnessed her choking upon it. Also, to borrow the words from Ronayne (para 13) "this was not, like Walters, "a seamless tale with an obvious beginning and an equally obvious end." There was therefore no sudden appreciation of a "horrifying" event.

152. For those reasons C2's claim also fails.

Conclusion

153. This has been a long arduous journey for Mr and Mrs Owers – a journey which Mrs Owers sadly completed last year after suffering 4 years of serious disability. They have established that the Hospital acted in breach of its duty of care towards Mrs Owers, but not established causation in her case, or liability in Mr Owers' case. There have been difficult factual matters for the witnesses to address and for the court to determine on the evidence presented. While I understand that the failure of Mrs Owers' action in particular will no doubt be very difficult for Mr Owers to come to terms with, nevertheless I hope it will be of some consolation that highlighting the failures in the system in diagnosing timeously his wife's condition has seemingly led to a greater awareness and improvements. This should now benefit patients in a similar position.

Appendix A

National Institute for Health and Clinical Excellence (NICE)

Diagnosis and Initial Management of Acute Stroke and Transient Ischaemic Attack (TIA). Issue date: July 2008.

NICE Clinical Guideline 68

(Page 6)

Definitions
Symptoms of stroke include numbness, weakness or paralysis, slurred speech, blurred vision, confusion and severe headache. Stroke is defined by the World Health Organization as a clinical syndrome consisting of "rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 h or leading to death with no apparent cause other than that of vascular origin. A transient ischaemic attack (TIA) is defined as stroke symptoms and signs that resolve within 24 hours… symptoms of a TIA usually resolve within minutes or a few hours at most, and anyone with continuing neurological signs when first assessed should be assumed to have had a stroke…"

(Pages 8 – 9)

Specialist Care for People with Acute Stroke

- All people with suspected stroke should be admitted directly to a specialist acute stroke unit following initial assessment, either from the community or from the A&E department (1.3.1.1).
- Brain imaging should be performed immediately[8] for people with acute stroke if any of the following apply:

……

- unexplained progressive or fluctuating symptoms

…

- severe headache at onset of stroke symptoms (1.3.2.1).

("Immediately") is defined as "ideally the next slot and definitely within 1 hour, whichever is sooner"

(Pages 10 – 11)

1 Guidance

…….

1.1 Rapid recognition of symptoms and diagnosis

There is evidence that rapid treatment improves outcome after stroke or TIA. The recommendations in this section cover the rapid diagnosis of people who have had sudden onset of symptoms that are indicative of stroke and TIA. ….

1.1.1 Prompt recognition of symptoms of stroke and TIA

1.1.1.3 People who are admitted to accident and emergency (A&E) with a suspected stroke or TIA should have the diagnosis established rapidly using a validated tool, such as ROSIER (Recognition of Stroke in the Emergency Room).

1.3.2 Brain imaging for the early assessment of people with acute stroke

1.3.2.1 Brain imaging should be performed immediately for people with acute stroke if any of the following apply:

…….

- unexplained progressive or fluctuating symptoms

…….

- severe headache at onset of stroke symptoms.

1.4 Pharmacological treatments for people with acute stroke

Urgent treatment has been shown to improve outcome in stroke. This section contains recommendations about urgent pharmacological treatment in people with acute stroke.

1.4.1 Thrombolysis with alteplase

1.4.1.1 Alteplase is recommended for the treatment of acute ischaemic stroke when used by physicians trained and experienced in the management of acute stroke. It should only be administered in centres with facilities that enable it to be used in full accordance with its marketing authorisation.

……

1.4.2 Aspirin and anticoagulant treatment

People with acute ischaemic stroke

1.4.2.1 All people presenting with acute stroke who have had a diagnosis of primary intracerebral haemorrhage excluded by brain imaging should, as soon as possible but certainly within 24 hours, be given:

- aspirin 300 mg orally if they are not dysphagic

……..

People with stroke associated with arterial dissection

1.4.2.6 People with stroke secondary to acute arterial dissection should be treated with either anticoagulants or antiplatelet agents, preferably as part of a randomised controlled trial to compare the effects of the two treatments.

……

(Page 27)

4.2 Aspirin and anticoagulant treatment for acute ischaemic stroke

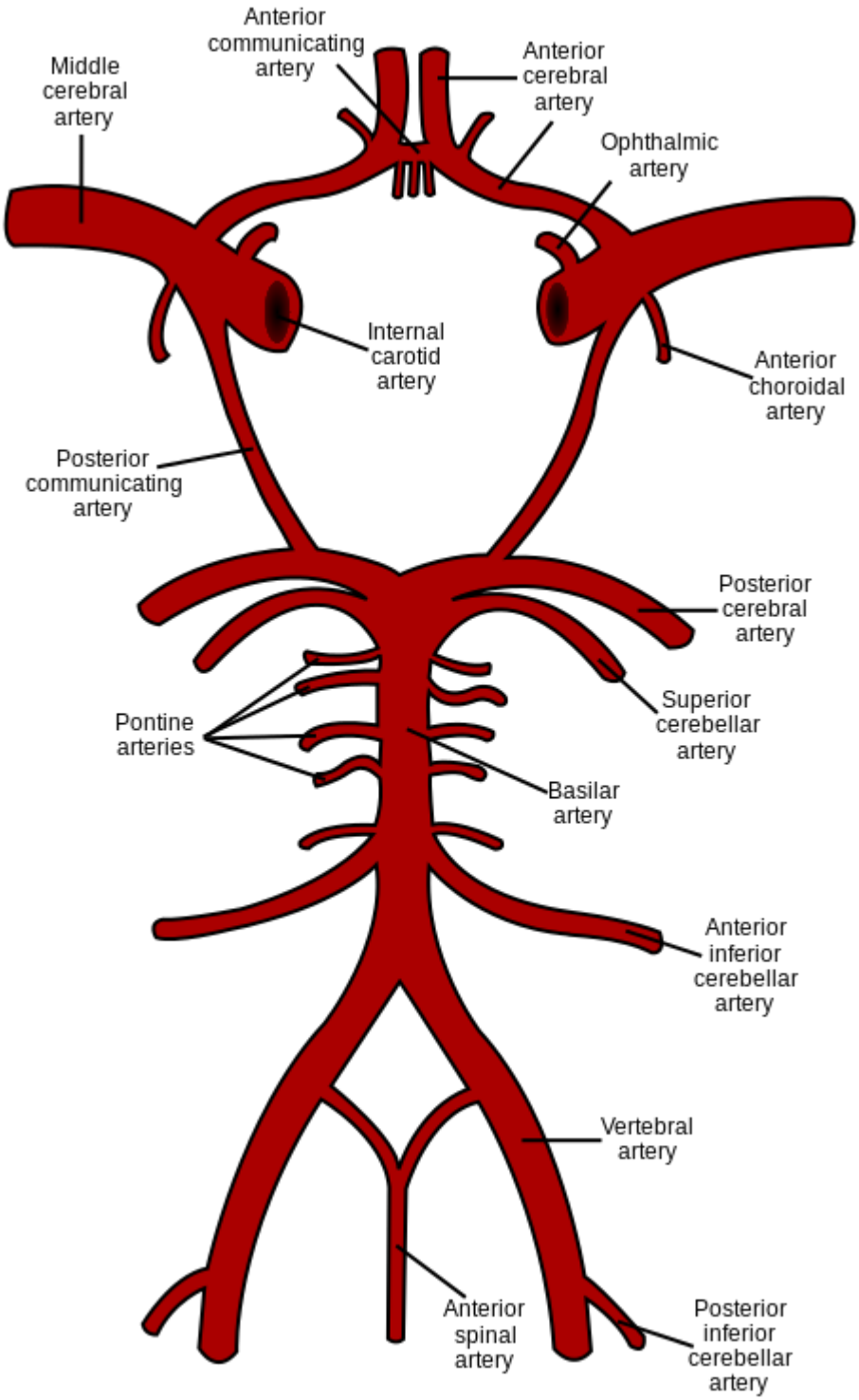
……

Why this is important

Aspirin administered within 48 hours of acute ischaemic stroke improves outcome compared with no treatment or early anticoagulation.

……..

Appendix B



The basilar artery (middle of figure) arises from the vertebral arteries and terminates when it bifurcates in the left and right posterior cerebral arteries