Neutral Citation Number: [2019] EWHC 2704 (QB)

Case No: HQ15P00008

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: Monday 14th October 2019

Before :

Her Honour Judge Deborah Taylor

- -

Between :

TRACEY BELL **(A protected party suing by her Stepfather and Litigation Friend DAVID URSELL)**	**Claimant**
- and - **BEDFORD HOSPITAL NHS TRUST**	**Defendant**

- -
- -

Miss Jane Tracy Forster (instructed by Price Slater Gawne) for Ms Bell
Miss Claire Toogood (instructed by Kennedys) for the Defendant

Hearing dates: 18 – 25 March 2019

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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.

.....

HHJ DEBORAH TAYLOR sitting as a Judge of the High Court.

1. On 26 March 2012 the Claimant Tracey Bell then aged 44 suffered a major stroke which has left her with significant permanent disabilities. It is now agreed that she had previously suffered transient ischaemic attacks (TIAs) in October 2009 and January 2010. She claims that there was a negligent diagnosis of a possible hypotensive episode in October 2009, and a failure to review her after the second episode in January 2010. Had a diagnosis of TIA been made on either of those occasions it is contended that the stroke would have been prevented.

The history prior to October 2009

2. Ms Bell was unable to give evidence herself. Her mother, Valerie Ursell was the main source of evidence about her life prior to 2009. Some of the evidence was obviously difficult for her to give, but I found Mrs Ursell to be a conscientious and credible witness, who did her best to assist the court.

3. Mrs Ursell said that there was a family history of high blood pressure on both sides of Ms Bell's family. Colin Bell, Ms Bell's father died of a stroke in January 2000 at the age of 54. He had been diagnosed with high blood pressure for which he had been taking medication. Until Ms Bell's stroke, the family were unaware that he too had a similar pontine stroke. All of Mrs Ursell's children had high blood pressure and were prescribed medication. Tracey was first diagnosed with hypertension when she was aged 15 and still living at home. In cross examination Mrs Ursell said that Ms Bell was not prescribed any medication at that stage, but advised to have her blood pressure checked.

4. By October 2009 Ms Bell was living away from her mother, bringing up three boys, then aged nearly 15, 14 and 5 on her own. She was working part time for her brother. She was eligible for free prescriptions, and therefore the cost of prescriptions ought not to have been an impediment to collecting medications.

5. Mrs Ursell said Ms Bell's life was very stressful. Her middle son was very challenging in his behaviour, and it was a struggle to get him to go to school, at a time when she had a much younger child. Ms Bell also had serious financial difficulties, of which Mrs Ursell only became fully aware after her stroke. Mrs Ursell and her second husband David, (who acts as Litigation Friend) helped Ms Bell both financially and with the boys as best they could, but nonetheless later learned that her problems had been so great she had been to a loan shark. She also had considerable difficulties with neighbours, including an occasion when a firework was put through her letterbox. This all put Ms Bell under greater stress.

6. Mrs Ursell said that she was against smoking, knew Ms Bell smoked, and was always trying to get her to stop. Ms Bell had tried to give up on a number of occasions. She managed it for a while, but due to her stressful home circumstances, always went back to it. She could not recall if Ms Bell gave up smoking during her pregnancies, but thought she may have done so.

7. Mrs Ursell was unaware of the details of Ms Bell's medication and whether she was taking it. She said in evidence that she thought Ms Bell would not tell her if she was not taking her medication, because she knew she would be cross. She and Ms Bell would always say what they thought, and often didn't agree. When Mrs Ursell went with Ms Bell to hospital in January 2010 and overheard that she had not been taking her medication, she said she had a go at her, and told her she ought to take it.

8. The GP records from October 2004 to the date of the first TIA event in October 2009 support Mrs Ursell's account of Ms Bell's periodic but unsuccessful attempts to give up smoking, and her medication and treatment. On 10 February 2005 smoking cessation was on her problem list. On 12 and 15 September 2006 she is recorded as being a heavy smoker. On 10 June 2007 her GP noted: "*moderate smoker. Managed to stop for 3d last month but succumbed under stress."*

9. The notes also record management of Ms Bell's hypertension. Medication was started on 25 October 2004. In March 2006, her blood pressure was up, and she was advised to come in for recheck the following week, but did not attend. On referral in October 2006 Mr Patil, an Obstetrics and Gynaecology consultant recorded her high blood pressure being *"well controlled with medication."* In January 2007 the dosage of Ramipril was increased as her blood pressure was not to target. Thereafter there are no records of attendance in 2008, but in 2009, the GP Notes record two letters sent to Ms Bell on 10 February and 15 September 2009 inviting attendance to monitor her blood pressure. There is no record of her responding to either.

October 2009 – the first TIA

10. There is now no dispute that on 22 October 2009 Ms Bell suffered a TIA. The accounts of the symptoms she reported vary, and are important in this case.

11. On 22 October 2010 she was seen by Lorna Rowe, a physician assistant, who recorded an "*uncertain diagnosis – possible posterior circulation TIA*" in the GP Notes. In her referral letter she says that Ms Bell described

> *"An episode of leg weakness, pins and needles in face, an inability to concentrate and the feeling of slurred speech. These symptoms were of sudden onset whilst driving but fully recovered within 10-15mins. She had a residual headache afterwards which has resolved.*
>
> *Today she is hypertensive at 176/126 after stopping her Ramipril several months ago. She is also a smoker and has a family history of CVA as her father died at 53 after a CVA. She has an ABCD2 score of 4/7. I have enclosed a copy of the ECG performed in clinic."*

12. The following day Ms Bell attended A&E at Bedford Hospital and was seen by Dr Snape. He recorded her account as

> *"Sudden onset heaviness in legs, sensory disturbance in face, dysphasia…*
>
> *Yesterday was driving in car, suddenly developed bilateral heaviness in both legs, numbness and tingling around her mouth, visual disturbance and her brother described her speech as incomprehensible.*
>
> *… After 2-3 minutes the symptoms resolved.*
>
> *Drove back to brother's had some food, developed slight dizziness and headaches. Dizziness resolved but still has headaches (not different from a normal headache for her)*
>
> *No further symptoms…"*

He made an entry in Past Medical History which may refer to occasional *"blurring of vision"* in last 2-3 months although that is unclear. Reference is made under "*TIA Risk - 3/4 out of 7",* to symptoms lasting 10 – 59 mins, and a family history of CVA and "*father died at 53".* Dr Snape made an initial differential diagnosis of *?TIA.*

13. Ms Bell was then referred on to Dr Joshi, a more senior doctor. He made notes that:

> *"driving car yesterday acute onset slurred speech (collateral history from brother). No receptive dysphasia.*
>
> *"Felt as though I was drunk +concentrating to get the words out right"*
>
> *Also concerned she would not be able to operate the car controls.*
>
> *No vertigo,*
>
> *No unilateral weakness.*
>
> *Episode lasted (less than) 10 minutes. Now feels fine. ..*
>
> *(off medications 2 years, home readings in 140/90)*
>
> *GP gave script for (aspirin) and Ramipril today*
>
> *..*
>
> *Probably a posterior circulation TIA. Risk factors +++ ABCD2 = 3.*

> *Most convincing part of the history is brother's view of her speech and her feeling drunk and uncoordinated…"*

Dr Joshi recorded a planned CT scan, but after he had a discussion with Dr Elmarimi, Ms Bell was sent home to attend the clinic in the morning for an MRI scan, not CT.

14. The following day 23 October, Ms Bell was seen by Dr Elmarimi. By then the TIA clinic had been running for about 3 months. It was set up by Dr Elmarimi who had become a Consultant in January 2010. He was not a neurologist, but a stroke specialist who had come from a background in geriatric medicine. There are no notes made by him in the file, but his evidence was that he would have made them on a separate sheet of paper (similar to Dr Joshi) which should then have been added into the file. The only record available is his proforma letter to Ms Bell's GPs, which contains the following:

> *"**Diagnosis**: Possible hypotensive episode.*
>
> ***Brief history of episode:** While driving she had generalised weakness with peri-oral numbness and dizziness, no focal neurological deficit. Symptoms lasted a few minutes and recurred once again later….*
>
> ***Management plan:***
>
> *Reassured about stroke diagnosis and risks*
>
> *Explained risk of vascular disease in view of family history, hypertension and high lipids. Please advise lifestyle changes.*
>
> *Please continue Simvastatin and keep BP below 145/85".*

15. The proforma also includes records of findings on examination, and subsequent results from blood tests. Dr Elmarimi said in his witness statement that he would have obtained the notes from A&E (which would have included Dr Snape's notes), and would have taken a further fuller history himself from Ms Bell on separate paper. In cross examination he accepted that he had not referred to the conversation recorded by Dr Joshi. He did not remember him, and was not sure if Dr Joshi's manuscript note was available to him. If the conversation took place, Dr Joshi would have told him the important parts of his assessment. He accepted that the description given to Dr Joshi entirely justified his presumptive diagnosis of TIA. Dr Elmarimi said he did not cast aside the views of the two more junior doctors who had seen Ms Bell, but arranged for her to be seen in clinic the next day.

16. Dr Elmarimi said that he decided on the basis of her presentation and his examinations that Ms Bell's symptoms were not indicative of a TIA. In his witness statement he said that he did not suspect a TIA. His reasons were that the generalised weakness she reported could not be attributed to one side of the body and therefore suggested a non-neurological cause. He accepted in cross examination that whilst he had said that bilateral leg heaviness is not a symptom of TIA, it could be. Slurred speech could be attributable to a large number of problems, but in any event Ms Bell

described her speech as incomprehensible, which was very different. In cross examination he said that it was extremely rare to have aphasia with posterior rather than anterior TIA. Dizziness also pointed away from a diagnosis of TIA, and there was no vertigo. Peri-oral numbness was not suggestive of TIA. He stood by his view that Ms Bell's symptoms were not diagnostic of a TIA. In his opinion at the time Ms Bell did not have a TIA, but the risk was still there. In cross examination he said that there was a low likelihood of a patient of Ms Bell's age and with her reported symptoms having a TIA. In the witness box he did a rough calculation that the likelihood was somewhere in the region of 2 in 100, 000. A posterior TIA was even less likely.

17. Mrs Ursell gave evidence in her statement that she attended the clinic on 23 October to be with Tracey, who phoned her to say that she had been to A&E and they had thought she may have had a TIA. When Mrs Ursell arrived, although she was on time, Tracey had already been seen by Dr Elmarimi and was upset, as she had been told that her MRI scan had been cancelled. She had been given no reason. When Mrs Ursell suggested going back into the clinic to push for an MRI, Tracey said she did not want to, and they would not do it. In cross examination she said that Tracey was upset and worried about her symptoms. Mrs Ursell did not think Tracey had felt reassured, but disillusioned and poorly.

18. In this respect, Dr Elmarimi said that the fact that Ms Bell's consultation was finished before it was due to start did not mean he spent little time with her. He sometimes started earlier. It would take more time to make a negative diagnosis, particularly where there had been a differential diagnosis of TIA the day before.

January 2010 – the second TIA

19. Following the incident in October 2009 Ms Bell was prescribed aspirin, and anti-hypertensive medication. On 9 November 2009 she was started on statins. The GP notes record that cholesterol and smoking was discussed with her.

20. On 19 January 2010 Ms Bell suffered a further episode, now agreed to be another TIA. Mrs Ursell said that after being phoned by her eldest grandson, and told that Tracey had had "a turn" whilst driving and had been taken to hospital by ambulance, she went to the hospital. After some tests, Tracey was discharged without any information about what was wrong with her. It was on this occasion that she overheard that Tracey had not been taking her medication.

21. The ambulance records of 19 January show that the initial symptoms were :

> *"BP 170/105 →166/104. 12h frontal headache. Sudden onset light-headedness. Aphasia lasting 15- 20 mins. O/E hypertensive, dysphasic. Symptoms improved on way to hospital."*

22. At hospital Ms Bell was seen by Dr Ramotar in A&E who recorded

> *"0700 today pt had episode of dysphasia (stuttering speech, difficulty producing words, no slurring)"*
>
> *Felt all limbs heavy – no specific neurological deficit*
>
> *peri-oral tingling, no visual changes, no facial asymmetry.*
>
> *Has had frontal headache for past few days. Similar to headaches she has had before*
>
> *.....*
>
> *Admitted with similar symptoms in '09 – CT head – no bleed, infarct or mass lesions*
>
> *Pt very non- compliant with medications."*

Dr Ramotar listed two possible diagnoses, TIA and an episode of anxiety and hyperventilation. He noted discussing with Mr Lloyd, another Consultant, the low risk score, raising the dose of Ramipril, lack of compliance with medication and referral to the TIA clinic.

23. Ms Bell was not seen in the TIA clinic. Her GP Dr Rashid was informed on 27 January that the appointment had been cancelled. Dr Rashid was concerned, and was advised to put in writing why Ms Bell needed to be seen, which she did. She wrote to Dr Elmarimi on 27 January 2010 with a summary of the A&E report and her history with a request for Ms Bell to be seen in the TIA clinic. The following day Dr Elmarimi replied that he had seen Ms Bell on 23 October and concluded that her symptoms were very unlikely to be caused by cerebrovascular disease. The recent episode was almost a repeat of the previous symptoms. He did not think the TIA clinic was the best place to investigate Ms Bell's symptoms. He advised *"ambulatory blood pressure and ECG monitoring if the symptoms recur, as well as maintaining tight control of her blood pressure."*

January 2010 until 9 March 2012

24. After the TIA in January 2010, the GP notes record attempts to stop smoking, and advice given on lifestyle. On 17 February, 24 March, and 1 November 2010 the notes record advice on giving up smoking and Ms Bell's unsuccessful attempts to do so. There is a further entry on 24 June 2011, and it is at about this time that it is subsequently recorded that Ms Bell picked up her last script for medication prior to her next visit in February 2012.

25. On 28 February 2012GP notes record *"Telephone encounter. Severe headaches. BP high. Prev non compliant with meds but says been taking it for 2-3 wks. No script done since June 2011."* There are further entries on 28 February upon which Miss Tracy Forster on behalf of Ms Bell places great emphasis. Dr Mehta records

> *"long chat: discussed risk of not using the meds regularly, pt says there has been a prev scare of TIA, will use it daily. Red flag symptoms explained: if any pt to go straight to A&E Raised blood pressure reading headaches, no blurry vision.. sym ongoing for some weeks… she started Ramipril 2.5 mg and the statin and aspirin. Plan, 1. stop aspirin and BP very high 2. Ramipril incr to 5mg …rev after that."*

26. Mrs Ursell's evidence was that Tracey rang her and told her what Dr Mehta had said and that she was frightened about the risk of stroke, and was making sure she was taking her medication regularly. She said the talk had got home to Tracey, and that she had new prescriptions. Mrs Ursell agreed in cross examination Tracey had long known of the family history of risk. She knew that her father had died of stroke, that her own high blood pressure and cholesterol were a high risk, and that was why she had been prescribed anti- hypertensives, aspirin and statins. She continued to smoke and did not take her medication up to February 2012. Mrs Ursell had been unaware of this. However, after speaking to Dr Mehta, Tracey took her medication. Her eldest son made sure she was taking it when he brought her a cup of tea in the morning. Mrs Ursell was also checking on her.

9 March to 26 March 2012 - the major stroke

27. On 9 March 2012 Ms Bell had a further episode when driving after which she was admitted to hospital by ambulance. Mrs Ursell arrived at the scene to find Ms Bell's voice distorted and speech slurred, and she had lost the use of her legs. Mrs Ursell said that although Tracey improved at hospital she felt ill and in a cold sweat. She was later discharged for an appointment at the TIA clinic.

28. The A&E notes on 9 March record

> *"light-headedness + difficulty finding words since 18:10, lasting for 20mins, spontaneously resolved. Headaches. Tingling in the lips and hands. Has had TIA in the past. Seen by neurologist but according to patient was declared that it was not TIA".*

29. The appointment in the TIA Clinic again proved difficult to arrange, and Mrs Ursell recounts how Tracey attempted to phone the clinic when she did not receive an appointment. When she was then informed that others had greater need than she, she refused to have the appointment cancelled. On 14 March Ms Bell went to the clinic where she was seen by Dr Cox. Mrs Ursell was also present.

30. Dr Elmarimi gave evidence that after he received the request for Ms Bell to be seen again, he referred her to Dr Cox, a consultant Neurologist in the TIA unit, as he thought she would be able to review whether there was a neurological cause. On 14 March Dr Cox made a diagnosis of migraine. Her notes and letter to the GP,

dictated immediately after the appointment are available. She explained what is recorded in her notes :

> *"..a previous history 2 years ago. On this occasion, headache. Whilst driving vision goes, non- specific, arms and legs heavy, everything felt odd. Lasted 20 minutes. Found it difficult to talk to paramedic. Left facial weakness."*

The headaches were described as *"throbbing, constant for 6-8 weeks"*. There were no neurological abnormalities.

31. In her letter of 22 March (although she said she dictated it earlier) Dr Cox elaborated on the symptoms:

> *"she describes the onset of visual symptoms which she finds difficult to describe, although she can still see. Her vision seems shimmery, and then she feels both her arms and legs bilaterally feel heavy and she develops expressive dysphasia. This lasted 20 minutes then resolved, although she has felt nauseated after the episode.*
>
> *Two years ago she also developed a left facial droop..*
>
> *She has been suffering with a headache for 6-8 weeks which has been particularly severe, throbbing and constant in nature"*

32. Dr Cox diagnosed chronic migraine possibly triggered by hypertension. She recommended changes in medication and an MRI scan. She advised Ms Bell to go back to taking aspirin and to stop smoking.

33. On 26 March 2012 Ms Bell suffered a major stroke.

Expert Evidence

34. Experts were called in three disciplines: Professor Brown and Dr Bowler Consultant Neurologists on the issues of breach of duty and causation; Dr Butler and Dr Stoodley, Consultant Radiologists on the interpretation of imaging; and Dr Haworth and Dr Budd on compliance with medication. The experts also referred to a body of medical research papers in support of their views.

Breach of Duty

35. The test in *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 582 as modified in *Bolitho v City and Hackney HA* [1988] AC 232 is whether a doctor acted in accordance with a practice accepted as proper by a responsible body of the relevant

clinical opinion, and which is capable of being logically supported. As Lord Browne-Wilkinson said in *Bolitho*

> *"..It will seldom be right for a judge to reach the conclusion that views genuinely held by a competent medical expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which a judge would not normally be able to make without expert evidence."*

He referred to the judgment of Lord Scarman in *Maynard v West Midlands Regional Health Authority* [1984] 1 WLR 634, at 639 where he said:

> *"... a judge's 'preference' for one body of distinguished professional opinion to another also professionally distinguished is not sufficient to establish negligence in a practitioner whose actions have received the seal of approval of those whose opinions, truthfully expressed, honestly held, were not preferred."*

Was there a breach of duty on 23 October 2009?

36. It is agreed by Professor Brown and Dr Bowler that the diagnosis by Dr Elmirimi on 23 October 2009 of a possible hypotensive episode was not a reasonable diagnosis. Miss Tracy Forster therefore submits that is a breach of duty for which the Defendant is liable. Miss Toogood, whilst accepting the agreement of the expert witnesses, maintains that the real issue is whether Dr Elmirimi should have diagnosed a TIA, as without such a diagnosis Ms Bell cannot succeed in her claim, as the subsequent events would be no different.

37. These are in fact two different issues. Despite his own somewhat combative defence of his original diagnosis, there is no real dispute between the experts that Dr Elmarimi was in breach of duty in making the diagnosis he did. It is to be noted at this stage that the diagnosis was "possible" hypotensive episode.

38. The second issue, without conflating causation, is whether he was also in breach in not diagnosing TIA, or even keeping open the possible diagnosis of TIA. The neurologists disagreed on this issue. In their Joint Statement they agreed that the symptoms relied upon by Dr Elmirimi in coming to his diagnosis (generalised weakness, bilateral leg heaviness, incomprehensible speech, dizziness and tingling round the mouth) could be typical of various neurological conditions, but they could also be caused by non-neurological causes. The symptoms should be viewed as a whole in context, including additional relevant features of the history. Professor Brown maintained in the Joint Statement that the symptoms which were reported to Dr Elmarimi should have led him to "suspect a TIA as a possible explanation of the symptoms". Dr Bowler considered it was reasonable for him not to consider TIA as a serious differential diagnosis in view of Ms Bell's age, and complex array of symptoms. Migraine was a more likely diagnosis. Dr Elmarimi's evidence that

"When making a TIA diagnosis you have to think about how likely the patient is to have the illness and how likely the symptoms are to fit with the illness" was supported by Dr Bowler. He and Dr Bowler both gave evidence that it was very unlikely that a 42 year old woman had suffered a TIA. Less than 10% of strokes occur in patients under the age of 45. Both considered that the symptoms were not typical of TIA.

39. The experts' views were explored further in evidence. Professor Brown had concluded in his report that Ms Bell's symptoms were specific and typical of posterior circulation TIAs. In evidence he said that on 23 October 2009 Dr.Elmirimi did not have to come to a definite diagnosis of TIA, but it should have been top of the list of differential diagnoses, and an MRI scan should have been ordered. If the imaging was normal, the diagnosis should still have been a possible TIA, but if abnormal, a probable TIA. A short duration TIA was unlikely to cause ischaemic changes visible on an MRI scan and therefore lack of changes would not confirm or rule out a TIA. Whilst migraine was an alternative explanation, TIA had the most serious outcome of all possible diagnoses, it fitted better with the symptoms as a whole, and should still have been kept in mind.

40. Professor Brown accepted in cross examination that statistically Ms Bell's age was a factor making it less likely she had suffered a TIA. However, she had other risk factors including hypertension since a teenager, high cholesterol and smoking, and the array of symptoms, all of which should be considered. He agreed that numbness and tingling in the mouth may be an unusual symptom of a TIA. Dizziness, rather than vertigo, was a vague symptom, and on its own unlikely to be diagnostic. Headaches had many causes, including migraine, and headache was present in only 30% rather than the majority of patients with posterior circulation TIAs. However, a doctor in a specialist clinic had a duty to recognise less common conditions. The more junior staff at the clinic had raised a diagnosis of TIA, and in Professor Brown's opinion, there was no logical basis for Dr Elmarimi to dismiss their diagnosis.

41. Whilst Dr Bowler considered that the most plausible diagnosis at the time would have been migraine, a common occurrence, he accepted, with hindsight, that the symptoms were also consistent with posterior circulation TIA, a relatively rare occurrence, and that the episodes probably were TIAs. Miss Toogood submits that in such circumstances it would still have been reasonable to make a diagnosis of migraine as the more likely option.

42. In considering breach of duty at this stage there are three important factors in this case which bear on the diagnosis made by Dr Elmarimi in October 2009. Firstly, this was a specialist TIA clinic. Professor Brown argued, with some logic, that such clinics and clinicians at them should be aware of rare occurrences as well as the more common. Secondly, and importantly in this instance, at this stage two more junior doctors (and a physician assistant) did raise the possibility of what might be a rare occurrence as their primary differential diagnosis. Dr Bowler suggested in evidence that one doctor may have been influenced by the views of the other. Nonetheless, at

this initial stage they kept open the possibility of a TIA, and that is in fact what is now agreed as most likely. Therefore, this is not merely, as Miss Toogood suggested, a diagnosis only available with hindsight. The diagnosis was made by doctors at the same clinic as Dr Elmarimi, and whose notes are available, on the accounts given to them by Ms Bell. Thirdly, this was on any view an unusual range of symptoms. There was a family history of stroke and early death. Dr Elmarimi's own diagnosis of hypotensive episode was provisional. He did not make a diagnosis of migraine. His proforma letter does not specifically mention the possibility of a TIA, although there is mention of stroke.

43. Dr Elmarimi in his evidence was out of step with the views of both neurological experts in standing by his original opinion. In the absence of his notes, there is no contemporaneous record other than the proforma letter, and no record of his considering Ms Bell's symptoms in detail. I bear in mind the evidence of Mrs Ursell, which I accept, that the consultation was over before its allotted time. Overall, I found Dr Elmarimi showed an element of arrogance about his own views. Insofar as it is necessary to do so, I reject his evidence that he took sufficient time to carefully consider Ms Bell's presentation before positively excluding the possibility of a TIA.

44. The Defendant relies on the fact that on 9 March 2012, over two years later, Dr Cox concluded that the symptoms Ms Bell recounted were attributable to migraine. She considered that her symptoms would be unusual for TIAs and the recurrence of symptoms made TIAs more unlikely. It was argued that Dr Cox acted in accordance with a responsible body of stroke physicians and that her diagnosis supported the argument that it was not mandatory for Dr Elmarimi to diagnose TIAs in October 2009 and January 2010. In my judgment this does not take account of the differences in recorded symptoms, in particular the "shimmery "nature of the visual disturbance, which it was agreed was consistent with migraine, and not present on the two previous occasions.

45. Taking the evidence as a whole, in the circumstances of this case, I conclude that it was a breach of duty not to keep a possible diagnosis of TIA in mind, even if not a probable diagnosis at this stage, and even if an alternative diagnosis of migraine was also possible.

Was there a breach of duty on 28 January 2010?

46. Professor Brown accepted that it was unusual to have a further TIA or stroke 3 months after the first. It was not disputed that most recurrences are within a month, with a crescendo within the first week. Nonetheless, it is now accepted that Ms Bell did have a second TIA after 3 months. Professor Brown's opinion was that having made a tentative diagnosis of possible hypotensive episode on the first occasion, even though it was unusual to have a second TIA after 3 months, Dr Elmarimi should have had the possibility of a TIA in mind and seen Ms Bell again. Dr Ramotar, a fourth practitioner, had again raised the possibility of a TIA. Further,

Dr Elmarimi was subsequently requested to review his decision not to see Ms Bell by her GP, Dr Rashid, who was clearly concerned.

47. Dr Bowler's evidence was that if Dr Elmarimi had made a firm diagnosis that excluded TIA in October, it was logical, as the symptoms were similar, not to see her in January 2010. Dr Elmarimi was entitled to take into account his previous diagnosis, as the reported symptoms were the same.

48. In her closing submissions, Miss Toogood conceded that if Dr Elmarimi should have considered Ms Bell's symptoms to be indicative of a possible TIA in October 2009, it follows that he should have reached the same conclusion in January 2010. She made that concession accepting that the recurrence of symptoms three months later would be unusual for TIA and made the diagnosis less, not more, likely.

49. I have found that Dr Elmarimi should have considered the symptoms to be indicative of a possible TIA in October 2009, and therefore conclude that Dr Elmarimi should have reviewed Ms Bell when she had a further episode in January 2010, and come to a similar conclusion. His own previous diagnosis of hypotensive episode in October 2009 was tentative, not firm as Dr Bowler postulated. Two more junior doctors had raised the diagnosis of possible TIA in October 2009, and in this instance Dr Ramotar, an A&E doctor who saw Ms Bell nearer the time of the episode in January 2010 had independently come to a similar diagnosis on the reported symptoms and examination. The episode should have been investigated more thoroughly, including for the possibility of its being a TIA, not least because the consequences could be very serious. Dr Elmarimi's failure to even review Ms Bell, and at least keep open the possibility of a TIA was a breach of duty.

Had Ms Bell been diagnosed with a TIA, or kept under review as having a possible TIA what treatment and advice would she have received?

50. It is Ms Bell's case that had she been diagnosed with a TIA or even a possible TIA a more intensive approach would have been taken, by her family and by medical practitioners. She would have been coded for TIA at her GP practice, and dosage and compliance would have been regularly checked and increased as appropriate to ensure that hypotension, cholesterol levels and blood pressure were kept at acceptable limits.

51. Dr Bowler's opinion was that the treatment and advice would have been the same. Ms Bell would have, as she had been, advised to make changes in her lifestyle, to stop smoking and to take her medication. She would have been, as she had been, prescribed statins for her cholesterol, aspirin and blood pressure medication.

52. An important aspect, in my judgment, is that it is agreed Ms Bell had already been prescribed the same type of medication, and given the same advice on stopping smoking and losing weight as she would have been given had a possible TIA been diagnosed in either October 2009 or January 2010. Even if a definite diagnosis of

TIA had been made (and Professor Brown did not go this far in evidence), again, the range of medication and advice would have been the same.

53. The evidence of Dr Haworth, supported by Professor Brown was that if a positive diagnosis of TIA had been made, Ms Bell's notes would have been marked accordingly, and engagement with her GPs would have been more targeted and better monitored. It was as a result more likely that compliance with medication, and cessation of smoking would have been achieved. Dr Haworth said in evidence that he believed that Ms Bell would have been asked to come back for review more frequently if she had been coded as having suffered a TIA on the GP's computer. If she had attended for review, she may have been given higher doses of statins or anti-hypertensive medication to control her cholesterol level and blood pressure.

54. Insofar as different dosage would have been given, there is no evidence that this would have involved more than subtle increases or decreases. At one stage Ms Bell was taken off aspirin, which was reinstated by Dr Cox. Dr Haworth agreed that there was a balance between the gastric effects of continued use of aspirin, and its accepted efficacy in preventing stroke.

55. In any event, any monitoring or changes in drug regime were dependent on Ms Bell attending and complying.

Would Ms Bell have been compliant in taking medication and/or in following the advice given?

56. There was general agreement between Dr Haworth and Dr Budd that research evidence and their own experience showed better adherence to medication as secondary prevention as opposed to primary prevention. Dr Budd's opinion was that there was no clear reason to suppose that Ms Bell would have followed medication advice any more assiduously.

57. Dr Haworth said in cross examination that even if Ms Bell had no firm diagnosis, but believed she had a TIA, whether she was as influenced by that would depend on her level of understanding and the full facts being available to her. The degree of compliance was dependent on the GP - those with more skills of persuasion and able to form a rapport were more likely to achieve better results. Whilst he believed that Ms Bell's compliance would have been improved by a diagnosis of TIA, he could not say that it was more likely than not that she would have taken her medication every day as prescribed, but that she was likely to have taken more medication than before.

58. Dr Haworth was referred to the paper *Medical Adherence-where are we today?* and the range of factors affecting compliance. There are a number of factors in the section headed Patient- Related Dimension which may apply to Ms Bell in this case, in particular, confidence in ability to follow treatment, motivation and psychosocial stress.

59. Statistical research data needs to be considered against the factual evidence in individual cases. It is clear from the letters and notes from Ms Bell's GP practice, that irrespective of diagnosis, considerable effort was put in to supporting her and providing advice, encouragement and support in stopping smoking. Requests and reminders to attend for monitoring of blood pressure were sent and ignored. Dr Rashid insisted that Ms. Bell have an appointment with Dr Elmarimi despite his reluctance. Dr Mehta gave strong advice about compliance. The notes show that Ms Bell believed she had a TIA, and that there was a continuing belief at the practice that she may have had a TIA. Nonetheless, Ms Bell's attendance at appointments and compliance with medication was poor. According to Mrs Ursell there had been some improvement after Dr Mehta's advice. The incident on 9 March and the subsequent stroke were during this period of reported compliance.

60. I conclude that even if Ms Bell had been given a diagnosis of possible, probable or definite TIA, and advised of any necessary changes in her lifestyle in October 2009, on a balance of probabilities she would have been no more compliant with medication, stopping smoking, or losing weight for any more time than previously, nor in a way which would have made a difference. Similarly, had she been diagnosed with a possible, probable or definite TIA in January 2010, it is unlikely that her ability to make changes in her lifestyle and compliance with taking medication would have improved. The history apparent from her notes shows numerous attempts to give up smoking, failures in compliance with advice given and failure to take medication prescribed. The stress of aspects of her life is understandably given as a reason for her inability to effect change. This is one of the factors identified in the research paper referred to above as affecting adherence. Those stress factors remained.

61. In addition, the evidence from the notes and from Mrs Ursell is that, even though this was not Dr Elmarimi's view, Ms Bell believed that she had had a TIA, knew of the family history of stroke, and was aware of her own medical risk factors. Against that background she had been advised very strongly by doctors at her own surgery as well as on visits to hospital of the need for compliance, but had not managed to achieve it on a concerted continuing basis.

Would compliance have prevented the stroke?

62. Despite finding that Ms Bell would probably not have complied with prescribed medication, I turn to whether, had she complied, the stroke would have been prevented.

63. If Ms Bell's accounts to medical practitioners, and to Mrs Ursell are accepted, she had been taking medication from about the second week of February, a period of about 7 weeks prior to the stroke. This is in contrast to long periods when she had not been taking medication.

64. The cause of the stroke is in dispute and relevant to whether on a balance of probabilities, stroke would have been avoided. It is agreed that if the stroke was caused by atherosclerosis, compliance with medication from October 2009 or January 2010 would on balance have avoided the stroke. Whilst Professor Brown suggested that compliance with medication would have had a significant effect on strokes of a non-atherosclerotic cause, the research materials show that medication would have had a significantly lesser reduction in risk on causes of stroke other than atherosclerosis, and that overall it is unlikely that a stroke would have been avoided.

65. There are no contemporary MRI/MRA scans available. Angiograms were carried out in August 2012, after the stroke, and the radiologists Dr Butler and Dr Stoodley agree that there is no evidence of major atherosclerotic disease on them. Dr Stoodley's evidence was that basilar atherosclerotic disease tends to occur late in patients with known generalised atherosclerotic disease. Dr Butler agreed in oral evidence that the usual place for atherosclerosis to develop is in the cervical carotid circulation.

66. In his report, Professor Brown argued that had a combination of MRI and MRA scans been carried out at the time of the first TIA, the scans would have shown stenosis or irregularity of the basilar artery. Atherosclerosis was by far the commonest cause of basilar artery thrombosis. In support of this opinion he relied upon a post mortem study from Castaigne and others (1973: *Pathology of VB occlusions*) which involved 44 patients, 11 of whom were women between the ages of 58 to 76 years. In evidence he relied further on the paper to argue that atherosclerosis can occur in one artery alone, as 6 patients with basilar artery occlusions had no involvement of the other major vessels.

67. Miss Toogood on behalf of the Defendant submitted that this was over simplistic. Analysis of the paper, which was not based upon MR angiograms, showed that 10 out of 16 patients (62.5%) with basilar artery occlusions due to atherosclerosis had other sites of occlusion and/or tight stenosis, Within the study occlusion was defined as a complete block of the arterial lumen and tight stenosis was defined as a stenosis which reduced the lumen by 75% or more., and therefore the study did not report on whether atherosclerosis was present where the stenosis reduced the lumen by less than 75%.

68. Further, she submitted that the Castaigne study has little relevance to Ms Bell, then aged 42, and therefore considerably outside the age range of women in the study. Dr Bowler had referred to larger statistical data in the 2009 Handbook of Clinical Neurology. *"Stroke among women, ethnic groups, young adults and children"* which led to Professor Brown accepting in the Joint Statement that atherosclerosis accounts for between 15% and 33% of ischemic stroke in adults aged less than 45 years.

69. In his evidence Dr Bowler disagreed with Professor Brown's view that the most likely cause of the stroke was atherosclerosis. His opinion, like Dr Stoodley, was that changes in the basilar artery were typically late and would not usually be seen

in the absence of significant and readily apparent atherosclerosis elsewhere. Therefore, in the absence of such apparent atherosclerosis on the 2012 angiograms, over 2 years on from the TIAs, there was no compelling evidence of atherosclerosis in the basilar being the cause of the stroke. Statistically, Ms Bell's age was also not typical for atherosclerosis being the cause of her stroke.

70. During the course of the trial on 19 March 2019 Professor Brown gave additional evidence about a possible stenosis on one of the images identified by Dr Stoodley in his original report. This stenosis had not originally been seen by Professor Brown or Dr Butler when reviewing the imaging. Professor Brown said that he could not say it was due to atherosclerosis, as it may be artefact. However, it appeared to him to be quite a considerable stenosis, although Ms Bell did not have symptoms of occlusion. Dr Bowler considered that if it were a stenosis, it was minor and not indicative of general atherosclerosis. Overall, it was difficult to see that this new evidence added support to Professor Brown's view. Ultimately, he agreed that it may or may not have been caused by atherosclerosis.

71. Professor Brown further asserted that the recurrent nature of the TIAs was an indicator that the cause was atherosclerosis rather than other potential causes. However, there was no support he could find in the literature for the proposition that atherosclerosis could cause recurrence of the pattern in this case: TIAs three months apart, followed by another TIA over two years later, followed by a stroke.

72. In cross examination as to the cause of the stroke, Dr Bowler said that it was unknown. Whilst there were other alternatives, such as dissection and cardio embolic causes, as with atherosclerosis, there was no evidence as to either being the cause. Dissection was more likely than cardio embolic causes, and dissection could recur in different arteries with healing between events. The fact that the MRA after the stroke did not show dissection did not mean there was none, as it may have healed. Professor Brown considered that dissection was an unlikely cause as dissection in the carotid was often fatal, and dissection overall was unlikely to recur in the same place at the intervals in this case.

73. Professor Brown's view that the cause of Ms Bell's stroke was atherosclerosis is based upon the overall picture of Ms Bell's history, and a reliance on the Castaigne paper. In my judgment, the paper does not provide sufficient support, having regard to the ages of the research group, where age is an important factor. Further, this is a small study, carried out post mortem. Even in this study atherosclerosis of only the basilar artery, upon which Professor Brown's views are based, is lacking on analysis. The literature as a whole is more supportive of the opinions of Dr Stoodley and Dr Bowler that basilar artery atherosclerosis comes late in development of atherosclerosis which more commonly involves the wider arterial system. That wider development is not supported by the 2012 MR angiograms. Similarly, the angiograms show no evidence to support dissection as a cause. Nor was any support for Dr Bowler's views to be found in the medical literature.

74. The burden is on the Claimant to prove causation. In this case, there are two potential causes, atherosclerosis and dissection which each have substantial factors on the evidence which render them equally unlikely. There is a third potential cause cardio embolism which is even more unlikely. I am therefore unable to conclude that the Claimant has succeeded in proving on a balance of probabilities that atherosclerosis was the cause of the stroke she suffered, and that as a result adherence to medication and advice given after a diagnosis of TIA would have prevented the stroke occurring.

Conclusions

75. Whilst the Claimant has succeeded in proving breach of duty in this case, she has failed to prove causation. The claim must be dismissed.