

[2019] EWHC 1276 (QB)

IN THE HIGH COURT OF JUSTICE  
QUEEN'S BENCH DIVISION

Case No: HQ17C00256

Courtroom No. 14

The Royal Courts of Justice  
Strand  
London  
WC2A 2LL

Thursday, 21<sup>st</sup> March 2019

Before:

THE HONOURABLE MR JUSTICE MARTIN SPENCER

B E T W E E N:

MARGARET ARKSEY

Claimant

and

CAMBRIDGE UNIVERSITY HOSPITALS NHS FOUNDATION TRUST

Defendant

MR B THOMAS (instructed by Slater & Gordon (UK) LLP) appeared on behalf of the Claimant  
MS R VICKERS (instructed by Kennedys Law LLP) appeared on behalf of the Defendant

JUDGMENT  
(Approved)

MR JUSTICE MARTIN SPENCER:

1. This is judgment in the case of *Margaret Arksey v Cambridge University Hospitals NHS Trust*. This judgment will be in 10 parts. Firstly, an introduction, then I will deal with the facts, then I will describe the medical condition which this case gives rise to. I will then consider the ‘but for’ scenario. I will consider the lay evidence, the pleadings, the expert evidence, the issues which arise, legal considerations and, finally, my decision and discussion.

## **I Introduction**

2. By this action the claimant, Mrs Margaret Arksey, seeks damages for alleged negligence arising out of the failure of the defendant hospital, Addenbrooke’s in Cambridge, to admit her on 3 November 2012 after she had suffered a sentinel bleed from a cerebral aneurysm. It is alleged on behalf of the claimant that had she been admitted, she would have avoided a major re-bleed that occurred the following day, 4 November 2012, which has led to significant disability.
3. The defendant’s case is that, although they were admittedly negligent in failing to admit Mrs Arksey to hospital and carry out a CT scan, this in fact made no difference because nothing that would or could have been done would have avoided the re-bleed the following day.
4. The second major issue concerns the treatment. The claimant developed hydrocephalus and in May 2014 a ventricular peritoneal (VP) shunt was inserted to relieve intracranial pressure. After this was adjusted in September 2014, Mrs Arksey made considerable progress and improvement. It is admitted by the defendant that the VP shunt should have been inserted by the end of January 2014 and, therefore, there was a negligent delay from 31 January 2014 to 14 May 2014.
5. The claimant alleges that consideration of the insertion of a VP shunt should have been sooner and that the delay was closer to a period of one year, rather than the three and a half months admitted by the defendant.
6. I am only asked to consider liability, including causation. The quantification of the claim, should that arise, will be for another occasion.

## **II The Facts**

7. The claimant, Mrs Arksey, was born on 17 June 1953 and she was, therefore, aged 59 when she ruptured her cerebral aneurysm. The studies seem to show that females in their mid to late fifties are at particular risk of aneurysm and consequent subarachnoid haemorrhage and it would appear that Mrs Arksey fell within the prime risk cohort.
8. On 3 November 2012, an ambulance was called to the address of the Arksey family at 38 Cage Hill, Swaffham, Cambridgeshire, it being Mr and Mrs Arksey’s son who summoned the ambulance, as Mr Arksey, the husband, was at work. The patient record form, or PRF, for that attendance by the East of England Ambulance Service is at pages 276-277 of the medical records bundle and shows that the ambulance was dispatched at

9.11 hours on 3 November. The ambulance was mobile at 9.17 and arrived at the scene at 9.25. The comments of the ambulance personnel were that on arrival they found a 59-year-old alert [woman] standing at the bathroom sink trying to be sick. The history of the presenting condition was the she had been found by her son in the bathroom, 'staring through him', complaining of neck ache.

9. On examination, Mrs Arksey had slurred speech; she was confused as to her age and time, and appeared to be agitated, not her normal self. She was uncooperative, chewing on a pulse ox lead. Initial vital signs were observed at 10.00 hours, at which time the blood pressure was 216/131, this being a significantly elevated blood pressure. Further observations were carried out at one second before 10.16, by which time the blood pressure was 209/125 and the ambulance left the scene at 10.17, arriving at Addenbrooke's hospital at 10.33.
10. An emergency assessment was carried out at 10.40, when it was recorded that Mrs Arksey was hypertensive and confused. Her Glasgow Coma score was 14. The maximum that can be scored under the Glasgow Coma score is 15 and the loss of one point reflected the element of confusion. At 11.45 there was a review by a specialist registrar, who recorded, 'Patient not very cooperative, not confused just not engaging. When pushed will cooperate completely. Says symptoms for "couple of hours", cough few days, wants to sleep, no pain'.
11. If at 11.45 the symptoms had been present for a couple of hours, that implies that the ictus in retrospect was very soon before the ambulance was called at 9.11 and this is consistent with the fact that Mrs Arksey's son would have noticed, at that time of the morning, quite quickly, that she was acting strangely. I think it is fair to assume that the ictus on 3 November was at about 9am.
12. At 13.40 on 3 November, the claimant was reviewed by Dr Chesters, a Registrar, who recorded that she was unsure whether this presentation was pathological or simply a 'difficult personality'. In any event, she recorded that there was no clear answer but nothing to suggest any on-going pathology at that point, and she discharged the claimant with advice to return if the problem reoccurred. She recorded at 13.40 that the chest x-ray was normal and the bloods were normal, and that Mrs Arksey was awake, walking around the department and 'back to normal'.
13. Her husband, however, gave evidence that he came from work to the hospital and was alerted to his wife's location because she was being very vocal, and as far as he was concerned, she was not back to normal when discharged. He said that she was given two painkillers in the form of paracetamol, and that is confirmed by the records, and that she was still uncomfortable upon discharge and during the journey home, by which he meant she had a headache. However, as they approached their home and Mrs Arksey saw her two sons waiting for them outside, together with a recently born grandchild, she started to calm down and become herself again and Mr Arksey said that this probably coincided with the effects of the painkillers kicking in.
14. What happened then is described by Mr Arksey in his statement. He said:  

'9. Our sons gave me a hand and we managed to get Margaret into her favourite chair. I

recall her being sat there with her grandson, Oliver. She became very, very tired quite quickly and by the middle of the afternoon we'd managed to take Margaret to bed. In all honesty, we thought that things were going to be all right. Margaret slept solidly that night.'

That was in Mr Arksey's statement of 14 February 2017. He made a further statement on 1 March 2019, where he said:

'My wife went upstairs to bed in the late afternoon/early evening and was in bed with her head on two pillows and she was not laid flat. Between then and going to bed myself, in the same bed, I checked on her two to three times and she was still in bed. When I went to bed at about midnight, she was still in bed. I fell asleep and the next I knew my son had found her in the bathroom downstairs. I did not hear her going downstairs as I was asleep. I do not know if that was the only time she went downstairs to the toilet that night, as I was asleep throughout. In my earlier statement I assumed she had slept soundly because I was not aware of her movements at any stage during the night, but I was asleep. As to food and drink, she had paracetamol and a cup of tea that afternoon. I cannot recall her having anything else to drink that day. I am pretty sure she had no food.'

#### 4 November

15. On Sunday 4 November, Mr Arksey recalls that their son, Joseph, found the claimant collapsed on the floor in the bathroom and had rung the emergency services, this being at about 7.30am. In fact, the records show that the ambulance was called back to 38 Cage Hill at 09.51 and so it is clear that Mr Arksey's recollection when he wrote his statement in February 2017 was significantly inaccurate so far as the timing was concerned.

16. Again, accurate facts are established by the ambulance PRF, which is at page 279 of the records bundle. The ambulance was on the scene at 10.04 and they found Mrs Arksey sat in her chair, complaining of a strange sensation in her left side. They immediately carried out observations and recorded a blood pressure of 194/135. In addition, the Glasgow Coma score was recorded as 15, indicating that Mrs Arksey was wholly conscious, alert and able to communicate. However, within a short period of time there was a significant deterioration in the claimant's condition. This is described in the PRF as follows:

'Shortly after arrival patient began to fit, increasing hypertension, flushed, tachycardic, GCS = 3, decorticate, decerebrate, respiratory arrest for one minute then spontaneous respirations returned following assisted ventilation'.

A Glasgow Coma score of 3 is the lowest score that it is possible to record and means that Mrs Arksey was effectively unconscious. Her blood pressure was 234/177.

17. It appears to be agreed that this deterioration represented a significant re-bleed from the cerebral aneurysm in the form a major subarachnoid haemorrhage. Given that the ambulance had been called when Joseph Arksey had found his mother on the floor of the bathroom, it would appear that the re-bleed started in a minor way at about 9.30 or thereabouts and then became a cascade bleed in the presence of the ambulance personnel, causing her to collapse, lose consciousness and stop breathing. It was probably fortuitous for Mrs Arksey that this occurred in the presence of the ambulance personnel because they were able to give immediate and life-preserving treatment, and but for their presence she

may well have died. With a re-bleed there is a significant mortality rate for ruptured cerebral aneurysm, in the region of 50%, as I understand it.

18. A third set of observations were carried out at 10.48 when the Glasgow Coma score was still 3, indicating that Mrs Arksey was still unconscious, and at 10.55 the ambulance departed the scene, with Mrs Arksey arriving at Addenbrooke's hospital at 11.08. There she was again seen by Dr Chesters, who was the emergency department registrar on call, who recorded:

'Seen by me yesterday, complaining of left-sided sensation today, then collapse and two-minute seizure'.

She recorded that airway management had been required, that the Glasgow Coma score was reduced and that Mrs Arksey had a decorticate posture, and there was assisted ventilation.

19. A CT scan of the brain was arranged. This was carried out at 12.04 and showed extensive, acute subarachnoid haemorrhage with early hydrocephalus: see the report in volume three at page 743. It was established that the cause of the acute subarachnoid haemorrhage was a ruptured anterior communicating artery aneurysm. Mrs Arksey was kept sedated and ventilated in intensive care until, on 5 November 2012 at 13.26, the aneurysm was embolised using a coil, as I shall describe later, effectively thereby treating the aneurysm and preventing any further bleed.

20. On 8 November 2012, a further CT scan was carried out at 15.03 and this was reported as follows:

'Coiling of the anterior communicating taking artery noted, there has been decrease in the amount of subarachnoid blood within the basal cisterns but there remains considerable subarachnoid haemorrhage within the sulci of both cerebral convexities, increased amount of intraventricular blood in both lateral ventricles...the ventricles are only slightly more prominent than before, the basal cisterns are not effaced.'

21. A further CT scan was carried out on 9 November at 15.46 and this was reported as showing no change in the volume of intraventricular and subarachnoid blood. There was no new hydrocephalus, mass effect or midline shift.

22. The next investigation was again by CT scan on 19 November 2012 and this was reported as follows:

'Unenhanced imaging shows mildly prominent lateral and third ventricles but these have not increased in size significantly when compared to 9 November 2012'.

23. The following day, on 20 November 2012, a lumbar puncture was carried out, described by Mr Trivedi, the Consultant Neurosurgeon, under whose care Mrs Arksey had been admitted, in his statement as follows:

'The opening pressure was 21cm, which is slightly raised, indicating a degree of hydrocephalus. 30ml was drained. Crucially, Mrs Arksey's symptoms did not alter following the procedure and, therefore, we were able to exclude acute hydrocephalus as a

cause of her clinical state at that time.’

24. Finally, so far as imaging is concerned, Mrs Arksey was given a MRI scan at 14.32 on 28 November 2012, which was somewhat marred by movement artifact but was reported as follows:

‘There is oedema in the cortex and subcortical white matter of the anterior part of the left superior frontal gyrus, which shows some increased signal on diffusion weighted imaging but no low signal on the ADC map. These appearances are in-keeping with subacute infarction. There are also small areas of high signal on DWI in the inferomedial aspects of both frontal lobes, larger on the right than the left, which are also likely ischaemic. There is mild to moderate hydrocephalus, small amount of haemorrhage remain in the occipital horns of both lateral ventricles.’

25. Mrs Arksey remained in hospital until she was transferred to a care home in March 2013, there being no further treatment or imaging after 28 November. She still continued to progress, as shown by physiotherapy assessments carried out at the care home from June 2013 and I refer to the records at page 549 onwards in volume three of the records.

26. On 10 June 2013, the claimant was seen by the Specialist Registrar in Neurosurgery, Dr Choudhary, and in a letter arising out of that clinic appointment he wrote:

‘From the neurosurgical point of view, we will endeavour to get an MRA [magnetic resonance angiogram] to assess the aneurysm and coiling approximately June 2014 and will see her in clinic thereafter’.

27. Mrs Arksey was seen in clinic by Mr Trivedi on 22 July 2013, and he wrote to the GP as follows:

‘I saw this lady for a review. She came for a check angiogram following her coiled aneurysm that was done in November of last year but unfortunately, she was unable to have this done because she could not provide consent. She is a resident at a care home in Newmarket and has been there since her discharge earlier this year. She is able to stand with the aid of a frame and requires help with dressing and washing, but can feed herself. She has some cognitive problems which are in evidence today in that her mini-mental test score was poor, which I suspect are lingering effects of her subarachnoid haemorrhage. However, her sleep is not disturbed. I could have a brief exchange of words with her, but it was very clear that she could not retain information sufficiently long enough to be able to provide informed consent. On the basis that it is still important, given that she is still in the early recovery phase, to ensure that her aneurysm remains well-secured, I think an MRA would be the way forwards and this has been recommended by our neurovascular MDT [multi-disciplinary team] service. I leave it in the hands of my Nurse Practitioner to organise accordingly.’

28. On 13 September 2013, the MR angiogram was carried out and this was reported as follows:

‘The images are quite degraded by motion artifacts. Allowing for the rather poor quality of the MRA, there has been no gross change in the size of the small residual neck of the coiled

anterior communicating artery aneurysm. Mild to moderate hydrocephalus has not changed since the last MRI. There is encephalomalacia in the left superior front gyrus, at the site of a previous infarct. Extensive areas of patchy, confluent white matter high signal on T2 weighted imaging are consistent with the effects of small vessel disease.'

29. The claimant's case was discussed in an MDT meeting on 26 September 2013 and, on 11 November 2013, the claimant was seen by Mr Patel, a specialist registrar in neurosurgery in clinic, on behalf of Mr Trivedi. Mr Patel wrote as follows:

'I reviewed Margaret Arksey on behalf of Mr Trivedi in clinic today. She is currently staying at a residential home. She was accompanied by her husband. Her husband tells me she does have on-going symptoms of urinary incontinence, short-term memory problems and difficulty in walking. Her MRA scan, done on 13 September 2013, shows ventriculomegaly. She was discussed at the MDT and the plan was to review her in clinic to see whether she would benefit from a shunt. Margaret is suffering from symptoms of chronic hydrocephalus. Following discussions with Mr Trivedi, I have offered for her to have a VP shunt. We will place her on the waiting list and will be in touch with a date.'

30. Although the insertion of the VP shunt was scheduled for February 2014, that was cancelled by the hospital and Mrs Arksey was readmitted to the hospital on 13 May 2014, when, at 17.44, she had a further CT scan for the purpose of assessing the ventricular size prior to insertion of the VP shunt. The report of that scan says:

'There is symmetrical prominence of the third and lateral ventricles compared to the previous MR examination of September 2013. There has also been no change of significance in the bilateral, patchy, low-density white matter lesions found in both hemispheres and the small, established focal damage in the left superior frontal gyrus.'

Accordingly, the shunt was inserted the following day on 14 May 2014.

31. Mrs Arksey was discharged back to the care home but readmitted to hospital in September 2014 and a further scan was carried out on 29 September 2014 at 9.03 hours and was reported as follows:

'Right parietal approach ventricular shunt terminating in the posterior lateral ventricle. The ventricles appear similar in appearance to previous CT study. There are no new intra or extra axial collections, no mass effect or midline shift, unchanged appearances of the bilateral periventricular patchy low densities, anterior communicating artery aneurysm clip noted.'

This report is difficult to understand, given that the aneurysm had not been clipped but had been coiled. In any event, the shunt was reprogrammed in September 2014 and on 6 October 2014, Mr Garnett, another consultant neurosurgeon at Addenbrooke's hospital who was responsible for the VP shunt, wrote to the GP as follows:

'On examination she is still pleasantly confused but able to gradually follow three-stage commands. Her strata valve is set at 2.5 and we have reduced this to 2.0 today. Overall we are very pleased with her progress since the operation and are discharging her back to your care.'

32. Thereafter, Mrs Arksey made good progress and eventually she was able to return home in September 2015. In a physiotherapy report on 28 July 2015, it had been reported that Mrs Arksey was independently mobile with a two-wheel walker, still incontinent of urine but had shown signs of improvement, had made significant improvement both physically and cognitively so that they could plan for her return home. On 16 September 2015 she was seen by her general practitioner who recorded that she had made a great functional recovery, was now walking with a frame, she had reasonable communication but some memory problems and confidence issues. There were no medical concerns and she had a care package whereby a carer would attend twice daily, and she was back living with her husband and son.
33. In his statement of 2017, Mr Arksey describes his wife as being wheelchair-bound and using a frame to walk about. He says she gets tired easily, her memory has been affected by the events, she would be unable to return to work and her cognition was not the same as it had been before.

### **III The Medical Conditions**

34. It is relevant for the purposes of this judgment to describe the medical conditions concerned.

#### **Aneurysm**

35. As I understand it, in human anatomy the anterior communicating artery is a blood vessel of the brain that connects the left and right anterior cerebral arteries. A brain or cerebral aneurysm is where the wall of an artery or blood vessel in the brain is weakened, causing it to swell into a blister-like shape. This is known as a saccular aneurysm. As aneurysms grow they put pressure on the surrounding tissue and this can cause symptoms, even before there is any rupture. Where this weakened wall of the artery does rupture, that causes a subarachnoid haemorrhage with bleeding from the artery into the subarachnoid space.
36. Diagnosis of an aneurysm may be coincidental if a patient has had a scan for some other reason and the scan shows an aneurysm or it could be because there has been a sentinel bleed, giving a hospital the opportunity to treat the aneurysm before a major re-bleed, with subarachnoid haemorrhage causing brain damage. Of course, some aneurysms may immediately bleed into subarachnoid haemorrhage without any opportunity for treatment or prevention.
37. Treatment for aneurysm offers two alternative options. Traditionally and in the old days, this involved surgical clipping whereby a section of the skull was removed and a clip was placed over the neck of the aneurysm to stop blood flowing into it. However, since the earlier 2000s, clipping has generally been replaced by a procedure known as coiling, carried out by an interventional radiologist, rather than a neurosurgeon, whereby a series of platinum coils are threaded from the patient's lower body up into the brain aneurysm and the aneurysm is then filled with the platinum, thereby stopping blood flow in a form of surgical 'Polyfilla'.
38. A ruptured aneurysm is, of course, an extremely serious condition, requiring emergency



medical attention. The symptoms include headache, nausea, neck pain, sensitivity to light, blurred or double vision, sudden confusion and, as in Mrs Arksey's case when the ambulance personnel attended at 10 o'clock on 4 November 2012, loss of consciousness and seizures. Another recognised symptom is weakness on one side of the body, and that accords again with the finding by the ambulance personnel that Mrs Arksey was initially complaining of a strange sensation in the left-side of her body before her collapse.

### Hydrocephalus

39. I should also mention the condition described as hydrocephalus, which literally translated means water on the brain, but more accurately is a clinical condition associated with enlarged ventricles.
40. Mr Battersby, the consultant neurosurgeon instructed on behalf of the defendant, stated in his report of September 2018 as follows:

‘If there has been a significant cerebral subarachnoid haemorrhage bleed, particularly if the ventricles are involved and contain blood, this will interfere with drainage of cerebrospinal fluid from the ventricle. The ventricles will subsequently enlarge, a condition termed post-haemorrhagic hydrocephalus. Following cerebral subarachnoid haemorrhage where there has been a reasonable blood load into the subarachnoid space after the initial haemorrhage, post-haemorrhagic hydrocephalus invariably occurs. This can be treated acutely, if necessary, by lumbar puncture, which has the advantage of not only improving symptomology by reducing headache but also provides clearance of some of the blood from the subarachnoid space. Persistence of blood products within the subarachnoid space may lead to the development of a persisting post-haemorrhagic hydrocephalus that does not settle spontaneously. However, most patients do not require shunt treatment after subarachnoid haemorrhage as their condition will usually improve naturally, assisted by lumbar punctures as required.’

41. Mr Battersby goes on to describe how recovery from subarachnoid haemorrhage takes time and he says that most of the recovery will have occurred within six to 12 months but continuing improvement can be expected, depending on the age and the initial clinical condition of the patient, for several years thereafter. He says that if there is clear evidence of clinical neurological deterioration associated with progressive ventricular enlargement, then it is an easy matter to decide that treatment with ventriculo-peritoneal shunting is required. On the other hand, if there is clinical improvement and the ventricular morphology improves, then no surgical intervention with a shunt is necessary. He also says that if there is clinical improvement, but the ventricular morphology is unchanged, a shunt remains probably unnecessary. He says that it is those patients who fall into the category of persisting ventriculomegaly and an unchanging neurological state that are more problematic. He describes all shunt surgery as carrying significant risks of both morbidity and occasionally mortality, particularly where there are inappropriate indications to do so. He says:

‘Shunting a patient who would otherwise make a spontaneous recovery from a post haemorrhagic hydrocephalus will usually engender shunt dependency for the remainder of their life, with all the associated complications and risks that an indwelling shunt confers, such as infection, blockage, etc. Therefore, a very careful analysis of the clinical condition

of the patient and the state of their ventricular system is required. If a patient fails to improve after a reasonable interval, namely six to 12 months, and there is evidence of persisting ventriculomegaly, then it is reasonable to consider whether a shunt would be appropriate, even in the absence of other radiological evidence which can suggest raised intracranial pressure, for example, flattening of the cortical sulci, persisting enlargement of the third ventricle and transventricular exudation of CSF, although the latter may also indicate a stable state. The decision to offer a patient a shunt in the recovery phase after significant subarachnoid haemorrhage requires very careful weighing of the risks and benefits likely to accrue. Hasty, inappropriate shunting is more likely to harm than assist the patient.’

#### Ventriculomegaly

42. This means enlargement of the ventricles. It is described and discussed in paragraph 58 below in the context of Mr Trivedi’s evidence.

#### **IV The situation ‘but for’ the admitted negligence**

43. As stated, the defendant has admitted that, on 3 November 2012, the claimant should not have been discharged from the hospital but the symptoms were such that a CT scan should have been carried out on that day. Had it been, it would probably have revealed some bleeding into the subarachnoid space but, in particular, the aneurysm.
44. What then would have happened is taken up in the witness statements of Dr Antoun, who was the interventional radiologist on duty at the time, and Mr Santarius, a consultant neurosurgeon at the hospital. Dr Antoun confirms that a CT angiogram on 3 November would have led to the diagnosis of the anterior communicating artery aneurysm and Mrs Arksey would have been referred to neurosurgery by late afternoon on that date, Saturday 3 November. The neurosurgical team would have approached him that evening and he says that if her condition had been stable and a grade one or two subarachnoid haemorrhage confirmed, he would have considered her for coiling. He says that he would have agreed to coil Mrs Arksey’s aneurysm and would have set that up for the following morning, 4 November 2012, that is the Sunday morning. He says that the coiling procedure requires a large team, with at least seven personnel involved: himself and an anaesthetist, an assistant anaesthetist, a radiographer, a radiology nurse, a running nurse and the porter.
45. Dr Antoun then describes the procedure for preparation and anaesthetisation of the patient. Mrs Arksey would probably have been fetched from the neurosurgery department at 9am, taken to the anaesthetic side-room, they would have gone through the consenting process with her again and then the team members would have introduced themselves and their role. He says that, on average, it takes between 45 minutes and an hour to have the patient ready on the table and he estimates that Mrs Arksey would have been ready to start the procedure at around 10am. He says that on average it takes about an hour from the start of the procedure to get to the point where he can start to place the coils, although there is patient-to-patient variation. Using a working basis of one hour, he estimates it would have been about 11am before he would have reached that point. Even then, it takes a further hour to secure the aneurysm. He then says this:

‘In Mrs Arksey’s case, we know that she had a re-bleed at 10am on Sunday 4 November 2012. I would not have realistically been able to secure the aneurysm before this time. If the re-bleed had happened whilst she was on the table or under anaesthetic, then the anaesthetist may have noticed something that happened through a change of blood pressure or pulse, which would have suggested the onset of the bleed. If I had already started the procedure, then I would have had no choice but to reverse the anti-coagulants very quickly, as they would have made clotting difficult and potentially increased the amount of haemorrhage. If I was nowhere near the aneurysm at that point, I would have done an angiogram to confirm the re-bleed and I would have faced a difficult decision to either stop the procedure or to carry on and both could have had rather serious consequences. On balance, if I was nowhere near the aneurysm then I would have stopped the procedure.’

46. Thus, it appears that in the ‘but for’ world of diagnosis of aneurysm on 3 November and Mrs Arksey’s admission to hospital, if one assumes that the bleed and the major subarachnoid haemorrhage which occurred at home shortly after 10am on 3 November would have occurred in hospital at the same time, then that would have caused significant difficulties for Dr Antoun.
47. The major issue in this case is whether Mrs Arksey’s admission to hospital on 3 November and anything which the hospital could have done whilst waiting for the coiling procedure to take place the following morning, would have prevented the re-bleed at 10am. If not and it would have occurred anyway, then it would appear that the hospital’s negligence was not causative of the consequences of the subarachnoid haemorrhage and, indeed, the consequences of the haemorrhage could have been worse because anti-coagulants would have been administered as part of the anaesthetising process and would have been in Mrs Arksey’s bloodstream at the time that the re-bleed occurred, thereby hindering coagulation.
48. If, on the other hand, treatment at the hospital pending the coiling would have affected the onset of the subarachnoid haemorrhage at all, then the claimant will succeed because she was only some two hours from safety in the hypothetical world of her admission to hospital. In other words, it would only have needed to have been delayed for a couple of hours for the coiling procedure to have avoided the re-bleed and the subarachnoid haemorrhage.
49. Dr Santarius, in his agreed evidence, sets out for the Court what would have happened had Mrs Arksey been admitted under his care on 3 November 2012, as she would have been because he was the on-call neurosurgeon. He says:

‘Had Mrs Arksey been referred to neurosurgery she would have been given a bed on the ward some time that evening. She would have been treated according to the subarachnoid haemorrhage protocol. I would have reviewed her at the end of the day. I would have explained to her the diagnosis and treatment options to prevent re-rupture. Nimodipine would have been prescribed as part of the protocol. The purpose of Nimodipine is to reduce the probability of occurrence and severity of vasospasm, which can result in an ischaemic stroke. It does not reduce the risk of aneurysm re-rupture. As per the SAH treatment protocol, she would have been put on bed rest. When patients are told that they have a potentially life-threatening condition and need brain surgery, it is a very upsetting and unsettling experience for them and I would have tried my best to reassure Mrs Arksey as

much as possible.’

50. He then describes how he would have referred Mrs Arksey to Dr Antoun and how she would have stayed on the neurosurgery ward overnight until transfer the next morning. He says, ‘Based on my own experience, I am 90% certain that the aneurysm would not have been secured by 10.00 hours on Sunday 4 November 2012’.
51. Dr Santarius produces, as an exhibit to his statement, the protocol which sets out the steps taken pre-operation, post-operation and then in the ensuing days after operation until discharge. The relevant section is day one, that is the day of admission, pre-operation. This provides for the following; 1) two-hourly neuro obs., vital signs and MEWS, 2) maintained on bed-rest and head up 30 degrees, 3) can sit out commode only, 4) input up to three litres/24 hours daily, 5) strict fluid balance, 6) patient information given to patient and family. The protocol also confirms the prescription of Nimodipine. Furthermore, the protocol provides for the relatives to be met and for the patient’s condition to be discussed and documented.
52. One of the issues that has arisen in this case is whether the claimant would have been compliant with this protocol and I have no reason to believe that she would not have been. Although she is recorded as being a smoker, she could not have smoked on the hospital premises and I regard it as being highly unlikely that she would have disobeyed medical advice and taken herself off the hospital premises in order to smoke.
53. In any event, given her husband’s description of her condition at home, namely feeling very, very tired and wanting to go to bed and then sleeping for most of the rest of day and the night, I have no reason to believe that she would not have felt the same way in hospital and, therefore, would have complied with the advice to be maintained on bed-rest, only getting out of bed to sit on a commode.
54. As Mr Battersby described, this pre-operative protocol is, apart from the prescription of Nimodipine which is not relevant for present purposes, essentially a nursing protocol and does not provide any medical treatment, as such, which would be expected to reduce the risk of a re-bleed.

## **V The lay evidence**

55. I have already described the evidence of Mr Arksey, Dr Antoun and Mr Santarius. In addition, I heard from Mr Trivedi, who, in a witness statement dated July 2014 after the amendment to the claim to which I shall refer in due course, made a statement to deal with the treatment of Mrs Arksey after the coiling procedure. He describes how, on 4 December 2012, Mrs Arksey had been stable and awaiting rehabilitation to Lewin Ward for more intensive neuro-rehabilitation. He said he was not concerned that pressure, by which he means intracranial pressure, was confounding the clinical picture and he would have expected a clinical deterioration if pressure had been an issue but he says the trajectory was upwards in terms of neurological progression, and Mrs Arksey had a fluctuating Glasgow Coma score of 14-15.
56. He further says in his statement that Mrs Arksey was confused and cognitively poor, and the symptoms were all in-keeping with frontal lobe infarcts, and the effects of subarachnoid haemorrhage. He says her recovery was slow; she was not transferred to Lewin Ward but

underwent rehabilitation on the ward as part of normal care until she was discharged on 13 March 2013. Her appointment with Dr Choudhary on 10 June 2013 was part of the routine follow-up and he describes how, on 22 July 2013, he had reviewed Mrs Arksey and attributed her ongoing problems to the subarachnoid haemorrhage. He says that she was significantly better compared to the initial days following the haemorrhage. He did not take an interrogative history, looking for features of normal pressure hydrocephalus, but would have had that in mind as part of his assessment, and the need to consider normal pressure hydrocephalus once the MR angiogram had been carried out. That was done on 13 September and the patient was then discussed at the neurovascular-angio MDT meeting on 26 September, when it was felt that there may have been subtle enlargement of the size of the ventricles, so they decided to review her in clinic to assess whether she should be treated for hydrocephalus.

57. Mr Trivedi describes Mrs Arksey being seen by his registrar, Mr Patel, and once they had discussed the situation, including enlarged ventricles evidence on imaging, they decided to offer Mrs Arksey the VP shunt. He says:

‘There was no clear indication for intervention until Mrs Arksey was seen in clinic in November 2013 as it was only after the MRA in September 2013 and the review in November 2013 that there was both a change in size, albeit subtle, of the ventricles and the presence of suggestive symptoms that prompted consideration of a normal pressure hydrocephalus syndrome, for which an elective placement of shunt would have been considered.’

58. Mr Trivedi confirmed his evidence orally and he described how there is a difference in terminology between radiologists and neurosurgeons because radiologists use the words, ‘ventriculomegaly’ and ‘hydrocephalus’, interchangeably but neurosurgeons are more nuanced and distinguish between ventriculomegaly, which he said is detectable radiologically, and hydrocephalus, which is a diagnosis based on both radiological signs of ventriculomegaly and also clinical signs. He said that an increasing ventriculomegaly may not be a problem in these cases, if it is not associated with high pressure. Whilst persisting hydrocephalus will cause problems, persisting ventriculomegaly may not. He said that no responsible neurosurgeon would consider a VP shunt only 23 days after subarachnoid haemorrhage and that if there was uncertainty as to whether there was high pressure hydrocephalus, a lumbar puncture would be performed, as was done on 20 November. He said that there was no dramatic deterioration in Mrs Arksey’s condition between 20-28 November to indicate any further lumbar puncture or indeed any invasive procedure to reduce intracranial pressure. He said that the reason why a VP shunt was considered in September/November 2013 was because, by that time, her symptoms had plateaued and they wanted to see if there was anything further they could do to improve her situation. He said that the decision did not come from anything said by the clinical radiologist but had come from the review, including at the MDT meeting, and the fact that the claimant had clinically plateaued.
59. Mr Trivedi said that normal pressure hydrocephalus syndrome is a hydrocephalus which may be due to reabsorption problems and that where that occurs, a VP shunt can help even where the intracranial pressure is essentially normal. He said that he first accepted that there had been a change in the ventricle size in September 2013.

## **VI The statements of case**

60. Proceedings were issued in February 2016 and particulars of claim dated 15 June 2016 were confined to allegations of negligence arising from the 3 November 2012.
61. In pre-action protocol correspondence, there had been an admission by the defendant on 6 August 2015 that the failure by Dr Chesters on 3 November 2012 to consider the possibility of a subarachnoid haemorrhage had been a breach of duty and that there should have been a CT scan. Thus, the particulars of claim went no further than the admission that had already been made and it was apparent that the issue was one of causation.

## **VII The Expert Evidence**

62. The particulars of claim were based upon a medical report by the claimant's expert consultant neurosurgeon, Mr David Sandeman, who had reported on 13 April 2016 and it was this report which proffered the opinion that had Mrs Arksey's condition been managed according to the protocol, she would have been less likely to have suffered the catastrophic haemorrhage that she did on 4 November 2012, but he acknowledges it is still possible she could have had the haemorrhage when she did. He says re-haemorrhage whilst waiting for coiling in hospital is uncommon, comprising 2-3% of neurosurgical cases and, 'on the balance of probabilities, therefore, she would not have had a re-bleed before the aneurysm was coiled'. He expressed the opinion that in his view it would have been highly unlikely that she would have had the second haemorrhage if the first had been diagnosed and she was still being managed in hospital. Mr Sandeman referred to the literature suggesting that an initial misdiagnosis is associated with a fourfold increase in the chances of poorer outcome, referring to study by Kowalski and others from 2004.
63. It has to be said that it is surprising to find disclosed as part of the liability evidence a report on liability from an expert which pre-dates the exchange of pleadings to. I say that because, in general, an expert would not submit a final report until certain procedures have been followed through, including, not least, exchange of witness statements, and no reasonable expert reporting for a claimant would want to finalise his report until he had had an opportunity to see any witness statements submitted on the part of the defendant.
64. The importance of that in this case is obvious. Significant and highly material information was contained in the statements of Dr Antoun and Mr Santarius, which would have informed Mr Sandeman's report on causation and the consequence of Mr Sandeman's report not being revised and being served in its original form is that those statements were not referred to at all and the contents of them were not taken into account.
65. The report of Mr Sandeman made no allegations in relation to the treatment of Mrs Arksey after the coiling procedure and, in particular, did not suggest that the hospital had been in breach of duty in delaying the insertion of the VP shunt.
66. The claimant has disclosed an addendum medical report from Mr Sandeman dated 4 June 2017 in which he states that, in response to a neuroradiology report addressing breach of duty from Dr Andrew Molyneux dated May 2017, he had been asked to review the notes again to consider the treatment of hydrocephalus in Mrs Arksey's case and whether there was a delay in instigating treatment for this, and what impact that delay might

have had on her eventual outcome. This report then considered the treatment and concluded that the deterioration in Mrs Arksey's mobility had been due to the development of hydrocephalus and suggested that there had been a negligent delay in the treatment of the hydrocephalus by the insertion of the VP shunt. As a result, amended particulars of claim were produced enlarging the allegations of breach of duty to include delay in treating the claimant's hydrocephalus between November 2012 and May 2014, it being alleged that this resulted her suffering a longstanding, chronic hydrocephalus causing increased pressure and secondary brain damage. It was pleaded that the delay was a material contributing factor to her pain and suffering during the periods of delay, and her overall ongoing disability.

67. In its amended defence, the defendant has addressed the additional allegations and it was pleaded that it was not mandatory for the defendant to have offered surgery for the insertion of a VP shunt prior to November 2013. However, the defendant admitted that, having made the decision in November 2013, the procedure should have been undertaken by the end of January 2014, and that there was, therefore, delay between end of January 2014 and 13 May 2014, when the shunt was in fact inserted. This amended defence was served on 1 December 2017 and Mr Trivedi provided his statement, to which I have already referred, in July 2018, to deal with the allegations of negligence effectively aimed at him or his team alleging a wider or longer delay than that which had been admitted by the defendant.
68. Clearly, Mr Trivedi's statement and the information contained therein setting out his reasoning for his treatment was highly important information and evidence for the experts to consider. However, and astonishingly, again Mr Sandeman's addendum report was served as his evidence on liability in relation to the additional allegations of breach of duty in its original form and without any consideration of the evidence of Mr Trivedi. In particular, had Mr Trivedi's statement been taken into account, Mr Sandeman would have seen the reference to the lumbar puncture on 20 November 2012, which formed an important part in informing Mr Trivedi's management decisions.
69. The lumbar puncture was a procedure which Mr Sandeman had missed altogether in both of his reports. In his evidence he explained this to me on the basis that he had not been given a full set of the medical records. However, this did not wash. Mr Sandeman met his opposite number, Mr Battersby, for the purpose of a joint expert report in December 2018, by which time Mr Sandeman had seen Mr Battersby's reports of May 2017 and September 2018. Those reports not only referred appropriately to the witness statements of the relevant witnesses, which Mr Sandeman's reports did not, but also set out the full medical history, including reference to the lumbar puncture of 20 November 2012.
70. It must, therefore, have been obvious to Mr Sandeman well before December 2018 that Mr Battersby had access to medical records which he, Mr Sandeman, said he had not had access to and which, therefore, meant that he had reported on a false, or at least incomplete basis. However, again, yet more astonishingly, Mr Sandeman appears not to have drawn this to the attention of his instructing solicitor and he told me that it was only in the week before trial, when he had access to the trial bundle which contained the full medical records, that he had access for the first time to the full medical records. He nevertheless went into the witness box and gave evidence affirming the accuracy and correctness of two medical reports which simply did not stand up to a moment's scrutiny, given that they had been prepared on a false and wholly incomplete basis.

71. I regret to say that, in my judgment, Mr Sandeman's evidence fell far below the standard to be expected of a reasonable, competent expert witness, both in relation to the preparation of his reports and in relation to his preparing to give evidence. I could see no excuse for this whatever.
72. I should say, though, that I do not altogether exonerate the lawyers who have represented the claimant because they allowed Mr Sandeman to go into the witness box despite these clear and obvious deficiencies in Mr Sandeman's written evidence, and this was something which should have been addressed by the lawyers long before the trial.
73. In the end, the continual apologies from Mr Sandeman in the course of his evidence, as the magnitude of the deficiencies became apparent, were embarrassing.
74. Whilst I am considering Mr Sandeman's evidence I can also indicate that his oral evidence was unimpressive. It was intemperate, at one stage he even used an expletive, and there was a failure on his part to address the questions that he was being asked: recognising the difficulties of some of the questions, not just from Ms Vickers for the defendant but also from the bench, he would stray into other areas and different areas so as to avoid answering the questions. I had no doubt, listening to Mr Sandeman's evidence, that this was a deliberate ploy on his part to avoid answering the questions, rather than any kind of misunderstanding on his part as to what he was being asked, and the technique was adopted by him because of the difficulty he found himself in, in addressing the questions.
75. By contrast, I found Mr Battersby to be a wholly straightforward and reliable witness, who had prepared reports which fully complied with Part 35 of the Civil Procedure Rules and the Practice Direction to Part 35, which set out the full documentation to which he had had access, and which addressed the lay evidence, making appropriate concessions to those questions which were for the Court to decide. I had no difficulty in preferring Mr Battersby's evidence to that of Mr Sandeman on every point of dispute between them.
76. I also heard from two expert interventional neuroradiologists, Dr Andrew Molyneux and Professor White. In the end there was very little difference between them in their evidence and, in my judgment, any residual dispute between them does not need to be resolved in this judgment for the purpose of resolving the issues between the parties. They only address the amended allegations arising out of the case concerning delay in treatment, although to some extent they also have produced medical authorities which address the issue of causation, which is the principal issue for me to decide.

## **VIII The Issues**

77. Counsel for the parties, in advance of my giving judgment, helpfully provided me with a list of issues to be determined and I shall start by dealing with the factual issues.
78. The first issue is: 'approximately when did the ictus occur on 3 November 2012' and, as I have already stated, I find that that was at or about 9am on 3 November.
79. The second issue is: 'how did Mrs Arksey present when she suffered her initial bleed from the aneurysm prior to the ambulance being called'. It is not wholly clear to me what significance this has but I find that her presentation was as described and set out earlier in



this judgment by the ambulance personnel and then reflected in the examinations at the hospital.

80. The third issue asks, ‘what activity was undertaken by Mrs Arksey when she returned home following her discharge from hospital at around 13.40 on 4 November 2012’; that should of course be 3 November 2012. This may have been important insofar as, if Mrs Arksey had been active and behaving in a way which was not compliant with the protocol I have described, then it may have been that that would have had an effect upon her blood pressure with a consequently enhanced risk of repeat bleeding. On the claimant’s case a systolic blood pressure at or above 160 millimeters of mercury is a significant risk factor for re-bleeding. My finding is as per the witness statement of Mr Arksey, which I have quoted above, namely that when she got home, Mrs Arksey first sat in her usual chair but soon became very tired, went to bed and effectively stayed in bed for the rest of the day and most of the night. We know that she must have got up at some stage because she was found downstairs in the bathroom but there is no evidence that she got up at any other stage and I am not going to speculate and assume that she did so. It seems to me that her level of activity was probably very little different to the level of activity as it would have been had she been admitted to the hospital and been compliant with the protocol.
81. The next issue is; ‘how did Mrs Arksey present on the morning of 4 November, prior to the call to the emergency services?’ In my judgment she would have presented in a way which was consistent with the start of a re-bleed but before the full haemorrhage which led to her losing consciousness. She had some of the symptoms of a re-bleed, that is a strange sensation down the left side of her body, but she remained fully conscious, alert and able to communicate with the ambulance personnel. However, I also find that she was actively bleeding and that as the blood entered the subarachnoid space she became comatose and fitted, and exhibited the panoply of symptoms which result from a major subarachnoid haemorrhage. This was one bleed, not two, but little turns on that. It was effectively something that started as a minor bleed and then became a fully-fledged haemorrhage.
82. The next issue is: ‘when did the re-bleed occur on 4 November?’ It seems to me highly unlikely that this would have been very long before the ambulance was called at 9.50. It does not seem plausible that, once Mrs Arksey started to bleed, it would have taken long before this turned into the full subarachnoid haemorrhage and the only advantage of the sentinel symptoms was that they enabled the ambulance personnel to attend in time to look after Mrs Arksey and save her life when she collapsed and suffered her respiratory arrest.
83. The next issue is: ‘what was the purpose of the subarachnoid haemorrhage protocol?’ It seems to me that the purpose of the protocol was to preserve the *status quo* as best could be, pending surgical procedure by clipping or coiling. I use the words, ‘as best could be’, advisedly because there is an issue as to how efficacious the protocol is, in particular in preventing a re-bleed but, insofar as the hospital is able to do anything at all to assist the patient, it appears that the protocol is the best that can be devised.
84. The next question is at the heart of the case and asks: ‘does compliance with the protocol, specifically bed rest, monitoring and advice, reduce the risk of re-bleed and delay the timing of the onset of a re-bleed?’ For reasons which I shall explain in due course my answer to both of those questions is no.

85. Given those answers, the next question, namely 'what management would have been in place and would it have been strictly implemented?' does not arise.
86. The next question is whether it has been established as likely that Mrs Arksey's blood pressure would not have been significantly different if she had been in hospital as compared to being at home, and again this is at the heart of the issues between the parties. I address this issue in this way: the medical research literature appears to establish that there are certain risk factors recognised for re-bleeding and those risk factors include a systolic blood pressure of over 160 millimeters of mercury. The claimant has sought to address the causation issue in this case by looking at the overall risk of re-bleeding whilst a patient is in hospital awaiting treatment and this issue has been addressed through certain data produced by Mr Sandeman, from his own sequence of patients, but also from other studies which have been produced, and then by comparing that to assumed statistics in relation to the risk of re-bleeding out of hospital.
87. The difficulty is that there is very little data on re-bleeding out of hospital for obvious reasons. Firstly, where a subarachnoid haemorrhage or aneurysm is missed, as happened originally in this case, and the patient is discharged, a significant proportion of those patients will die and never get back to hospital. In any event, there appears to be no mechanism whereby a proper study could be put together to measure the risk of re-bleeding out of hospital compared to the risk of re-bleeding in hospital. In particular, the only factor which, in the end Mr Sandeman relied upon as being significant for the purpose of the risk of re-bleeding, was systolic blood pressure. It would therefore need to be shown that because she had been discharged from hospital, Mrs Arksey behaved in some way that caused her systolic blood pressure to be in the risk area of 160 plus, when, had she been admitted to hospital and put on the SAH protocol, her systolic blood pressure would have been below 160. It would then need to be established by the claimant that the enhanced risk from this was causative so that, but for the increased blood pressure, Mrs Arksey would not have re-bled either at all or when she did.
88. It seems to me that the claimant fails in relation to all these steps. Firstly, I find that Mrs Arksey's blood pressure when at home would, if anything, have been lower than in hospital. As Mr Santarius has explained, to be told that you have a serious, life-threatening aneurysm in your head which could, at any time, re-bleed and kill you, would be incredibly upsetting and causative of anxiety in a patient, however much the hospital tried to reassure the patient, and raised anxiety is one of the accepted factors for raised blood pressure. Furthermore, in so far as the prescription - if I can call it that - is bed rest, Mr Battersby told me that, in his view, Mrs Arksey was probably getting more peace and quiet and rest at home than she would have got in hospital, where she would have been woken every two hours for neurological observations and where she would have been in a hospital environment, which is inevitably noisy to some extent.
89. I was convinced by Mr Battersby on those points and it seems to me that, if a doctor could be sure that his patient would follow instructions to stay in bed and to be as quiet and restful as possible, then such a doctor would probably prefer the patient to be at home than in hospital.
90. The irony is that the benefit to Mrs Arksey was in fact the misdiagnosis. As far as she was concerned, she had been discharged with reassurance from Dr Chesters, she had made a

substantial recovery from whatever had gone on and there is no evidence whatever that she was concerned or worried about her condition ongoing. On the contrary, her behaviour, to go to bed and to go to sleep, was consistent with somebody who was not being kept awake with any kind of anxiety. Thus, her anxiety levels would have been, in my view, significantly lower than they would have been had she been diagnosed and admitted to hospital, and, if anything, her blood pressure was probably lower at home than it would have been in hospital.

91. Even were it to be the case that I had found that the blood pressure was raised out of hospital compared to what it would have been in hospital, I would not have found that this was causative of the subarachnoid haemorrhage and re-bleed that occurred at about 10am on 4 November. To have drawn that conclusion would have involved making the leap from increase in risk to causation.

### **IX Legal Considerations**

92. It is the Claimant's case that the breach of duty on the part of the Defendant made a material contribution towards the sustaining by her of the SAH, and accordingly it is relevant and necessary for me to consider the doctrine of material contribution, and whether it applies in the present case. In particular, did the Defendant's breach of duty increase the risk of the Claimant suffering a sub-arachnoid haemorrhage and, if so, can the court draw an inference from this that it made a material contribution?

93. I start by reminding myself of what Lord Hoffman said in *Gregg v Scott* [2005] 2 AC 76, paragraph 79:

'What these cases show is that as Helen Reece points out in an illuminating article, 'Losses of chances in the law', [(1996) 59 MLR 188] the law regards the world as in principle bound by laws of causality. Everything has a determinate cause, even if we do not know what it is. The blood-starved hip joint in *Hotson's* case, the blindness in *Wilsher's* case, the mesothelioma in *Fairchild's* case, each has its cause and it was for the plaintiff to prove that it was an act or omission for which the defendant was responsible. The narrow terms of the exception to this principle in *Fairchild's* case only serves to emphasise the strength of the rule. The fact that proof is rendered difficult or impossible because no examination was made at the time, as in *Hotson's* case, or because medical science cannot provide the answer, as in *Wilsher's* case, makes no difference. There is no inherent uncertainty about what caused something to happen in the past or about whether something which happened in the past will cause something to happen in the future. Everything is determined by causality. What we lack is knowledge and the law deals with lack of knowledge by the concept of the burden of proof.'

94. Thus, what Lord Hoffman illuminates for us is that the burden of proving causation remains strongly, and fairly and squarely, upon the claimant, and it is not enough for a claimant to assert that because there are gaps in the evidence arising out of the defendant's negligence, therefore, the claimant is any way relieved of the obligation of the causation. Lord Hoffman says in terms that that is not the case. The Court can, of course, draw inferences and it may be that, in drawing inferences, the Court will be benignly favoured towards a claimant where a claimant is in evidential difficulties because of the negligence of the defendant but the inferences need to be ones which are consistent with the evidence that

does exist, and the Court cannot invent past facts or hypothetical facts simply because the evidential basis for the claim is lacking.

95. Sometimes, of course, it is possible for the Court to draw causation inferences arising from an increase in risk, but the authorities show that that can only be done where the ‘but for’ test of causation is satisfied. This was the unanimous conclusion of the House of Lords in *Chester v Afshar* [2004] 3 WLR 297, albeit they made an exception to that generally applicable proposition in the particular circumstances of that case. The rule derives from a sound body of authority dating back to *Bonnington Castings v Wardlaw* [1956] AC 613 through *McGhee v National Coal Board* [1973] 1 WLR 1.

96. In *McGhee*, in breach of duty the defendant had failed to provide a worker with shower facilities before he cycled home covered in brick dust and the issue was whether that failure was a material contribution to his contraction of dermatitis. The pursuer’s counsel argued that it was sufficient for him to prove that the defendant’s breach of duty increased the risk of dermatitis and that was rejected by the Lord Ordinary at first instance, who said:

‘Dr Hannay’s evidence was that he could not say that the provision of showers would probably have prevented the disease. He said that it would have reduced the risk materially, but he would not go further than that. Dr Ferguson said that washing reduced the risk. Pursuer’s counsel maintained that a material increase in the risk of contracting the disease was the same as a material contribution to contracting the disease and that Dr Hannay established this by his evidence. I think that defender’s counsel was correct when he said that the distinction drawn by Dr Hannay was correct and that an increase in risk did not necessarily mean a material contribution to the contracting of the disease. The two concepts are entirely different.’

97. In my judgment, this distinction, which was also submitted to me by Ms Vickers on behalf of the defendant, namely the distinction between increase in risk and causation, is a wholly valid one. What the House of Lords held in *McGhee’s case* was that in certain cases it may be possible for the claimant to prove that the defendant’s breach of duty made a material contribution to his injury by showing that it increased the risk of injury, whereby the Court will draw an inference that there was a material contribution. That this is the correct interpretation was made clear by Lord Roger in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 when, referring to *McGhee*, he said:

‘What Lord Reid does rather is to accept that the pursuer must prove that the defender’s conduct materially contributed to the onset of his illness but also, like Viscount Simmons and Lord Cohen in *Nicholson*, he considers what it is that the pursuer must prove in order to establish that material contribution. Taking the “broader view of causation” he holds that in these particular circumstances there is no substantial difference between saying that what the defender’s did materially increased the risk of injury to the pursuer and saying that it made a material contribution to his injury.’

98. Thus, it seems to me that *McGhee* is not to be taken to suggest that a claimant is absolved from proving that there was a material contribution to the damage occurring where there are concurrent potential causes. It is rather looking at the question of evidence and how a claimant can go about proving material contribution. Rather, what the House of Lords is saying is that in certain circumstances, where a claimant can prove that the breach of duty

increased the risk of an adverse outcome, the Court will draw an inference that the breach of duty made a material contribution.

99. Thus, the doctrine of material contribution, as developed through *Bonnington Castings Ltd* and *McGhee*, can be seen to be an exception to the need for the claimant to prove that the ‘but for’ test is satisfied and is confined to the situation where there are contributory causes, whether concurrent or successive.

## **X Discussion and Decision**

100. In my judgment, this is not that kind of case. This is not a material contribution case at all, but is a case where it is necessary for the claimant to prove the ‘but for’ test in order to prove causation. Therefore, even if I had found that the increased risk existed because of increased blood pressure, which I have not, I would not have made the leap of faith and translated the increased risk into causation. Referring to the medical evidence which I heard, there was no medical basis upon which I could draw that inference or make that leap of faith.
101. In particular, as Ms Vickers cogently and persuasively submitted to me, what is needed is evidence as to the relative risk between those who are kept in hospital and those who are allowed out of hospital. Such evidence does not exist and therefore a case based on that comparison does not get off the ground.
102. But even if the evidence did exist, in order to compare like with like, it would still be necessary to compare the activities of those out of hospital with those in hospital. I have been able to do so in this case because of my findings as to what Mrs Arksey did do upon her release from hospital and I have been able to compare that with what she would have done had she followed the protocol; but any epidemiological or statistical analysis would have to look at the activities of all those who re-bled out of hospital and see whether those activities were such as to raise the risk of re-bleeding. If there was a study which looked at those out of hospital who re-bled despite, like Mrs Arksey, going to bed and sleeping and engaging in no heightened activity at all, then the statistics would be potentially useful but there is no conceivable way in which a study which condescended to the necessary detail would ever be capable of being put together. That is why there is a dearth of statistical information for those out of hospital and why any kind of comparative exercise is doomed to failure. In that regard, I considered Mr Sandeman’s attempts to do so to be misconceived and to be based upon false criteria, and I accept Mr Battersby’s evidence that no such comparison is possible.
103. In the end Mr Battersby’s evidence was that, in his view, one cannot conclude from any assumed increased blood pressure, even in the increased risk range, that there was, on any kind of valid statistical basis, an increased risk of re-bleeding to enable one to say that, but for the raised blood pressure, the re-bleed would not have occurred when it did. On that basis, and given my preference for that evidence, the case for the claimant in relation to the first part of her claim has to fail.
104. So far as the second part is concerned, as I have indicated, in my judgment the approach to the treatment of Mrs Arksey by Mr Trivedi was wholly appropriate and in accordance with the medical principles which I have quoted from Mr Battersby’s report. The timing of the

decision to carry out the VP shunt was wholly in accordance with standard medical practice in cases of normal pressure hydrocephalus, as opposed to raised pressure hydrocephalus, which, in my judgment, Mrs Arksey never had. She did not have significantly raised intracranial pressure when the lumbar puncture was carried out on 20 November 2018 and the radiological evidence does not, in my view, establish otherwise.

105. Once Mrs Arksey's recovery had plateaued - and it was reasonable to consider that to be the case after 12 months - and given that by that time Mr Trivedi considered that there was convincing evidence of ventriculomegaly, it was reasonable for him to offer a VP shunt in the hope, rather than in the expectation, that it would help to alleviate Mrs Arksey's ongoing symptoms. That distinction, namely between hope and expectation, was one to which Mr Battersby wholly subscribed in his evidence. In the end, it turned out to be an effective part of Mrs Arksey's treatment, but I agree with Ms Vickers that, on the back of that outcome, to say that it should have done significantly earlier is to use the 20/20 retrospectoscope, or as she put it, 'the benefit of hindsight', prospectively: at the time the decisions were made, in my view the decisions were wholly correct.
106. The consequence is that there will be judgment for the claimant only for the admitted breach of duty in relation to the failure to carry out the VP shunt for a period from 31 January 2014 to 14 May 2014, a period of three and a half months, but otherwise the claim fails. In relation to the admitted breach of duty, the longer-term outcome has been no different, in my view, to what it would have been had the VP shunt been carried out when it should have been. The only effect in causation terms is that this has put back the recovery process, which otherwise would have started on 31 January, to 14 May 2014, and it will be for the parties either to settle, or for the Court to conclude on another occasion, how that delay translates in terms of damages.

Transcript from a recording by Ubiquis  
291-299 Borough High Street, London SE1 1JG  
Tel: 020 7269 0370  
legal@ubiquis.com

This transcript has been approved by the judge.