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Case No: QB-2018-001617

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 29 January 2021

Before :

HIS HONOR JUDGE AUERBACH
(sitting as a Judge of the High Court)

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Between :

MR ROSS OWAIN DAVIES **(Executor of Estate of Mrs Gabrielle Jane Davies, deceased)**	**Claimant**
- and -	
FRIMLEY HEALTH NHS FOUNDATION TRUST	**Defendant**

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Henry Charles (instructed by **Thomson Snell and Passmore LLP**) for the **Claimant**
Matthew Barnes (instructed by **Weightmans LLP**) for the **Defendant**

Hearing dates: 9, 10,11 and 14 December 2020

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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.

HIS HONOUR JUDGE AUERBACH

Covid-19 Protocol: This judgment was handed down remotely by circulation to the parties' representatives by way of e-mail, by publishing on www.judiciary.uk and by release to Bailii. The date is deemed to be Friday, 29 January 2021.

HIS HONOUR JUDGE AUERBACH:

Introduction

1. This claim arises from the tragic death of Mrs Gabrielle Jane Davies from bacterial meningitis. On the morning of 25 February 2015 Mrs Davies was taken by ambulance from her home to Wexham Park Hospital in Slough. On the evening of 27 February she was declared dead. The hospital is run by the Defendant. The Claimant, Mr Ross Owain Davies, is the widower of the late Mrs Davies and executor of her estate. He brings the claim also on behalf of their two young children. It is his case that the death of Mrs Davies was caused by the negligence of the Defendant.

2. Administration of intravenous (IV) antibiotics to Mrs Davies began at 13.20 on the day of hospital admission, 25 February 2015. By the time that the matter came to trial it had been admitted and agreed that the Defendant was negligent by failing to begin administering antibiotics by 10.40 that day. In the event of liability being established quantum had also been agreed. In view of the interests of the children, I was asked to approve the agreements (as to the fact and time of negligence, and as to quantum), which I did at the start of the trial. The disputed issue that was tried before me is causation.

3. Causation is put by the Claimant in two alternative ways, being in summary (a) that, had intravenous antibiotics been administered by 10.40 on the day of admission, it is likely, on the balance of probabilities, that Mrs Davies would have survived; or (b) that the failure to do so made a material contribution to her death. Both are disputed. The Defendant specifically contends that the doctrine of material contribution is not, in law, applicable in this case.

4. The Defendant denies that earlier administration of intravenous antibiotics would have made any difference in this case. Its case, in summary, is that Mrs Davies experienced an unusually rapid decline from an exceptionally severe infection; and that the progress of the meningitis was already so advanced by 10.40 am, that, even had administration of IV antibiotics begun then, this would not have altered the course of the infection or saved her life.

5. I heard evidence in Court from three pairs of expert witnesses. The otolaryngologists (ENT specialists) were Mr Morrison, for the Claimant and Mr Hawthorne, for the Defendant. In matters of microbiology, I heard, respectively, from Professor Lever and Professor Masterton. The neurosurgeons were, respectively, Mr Norris and Mr Crocker.

6. Also before me was a witness statement from the Claimant. The contents were accepted by the Defendant as fact, so he was not required to give oral evidence. Also in my bundle were statements from Dr Mukundu, Dr Bansal, and Mr Litchfield, all of whom had some involvement in Mrs Davies' care at different points following her arrival at the hospital. None was called to give evidence in person. After some discussion it was agreed that I should read their statements. It was, properly, agreed that, where anything in the medical records, or what any of those three clinicians said in their statements, conflicted with the evidence of the Claimant, I should prefer his evidence.

Factual Overview

7. As to the general chronology of events, I make the following findings of fact.

8. Mrs Davies was born in 1978. She and the Claimant had two children, born in 2010 and 2013. Before the illness that led to her death she had no relevant prior illness or health condition.

9. On the morning of Tuesday 24 February 2015 Mrs Davies complained to her husband of tinnitus in her right ear. At 08.26, after he had left for work, she texted him complaining of a "stonking headache". At 16.06 she texted him that she was in "agony". A friend drove her to the GP where she was seen shortly after 5pm. The GP recorded the problem as "Otitis media" – that is, a middle ear infection. She was prescribed the oral antibiotic Azithromycin and Co-codamol. She returned home. (I should, in fairness, interpose that no criticism has been made of the GP.)

10. During the course of that night Mrs Davies vomited several times and became drenched in sweat. On the morning of the 25th she was, in the words of the Claimant, "beside herself" with pain. She told him that she needed to go to hospital. At 08.21 he dialled 999. He described her, to the call handler, as having stabbing pains in the head, which she had had all night, and which had come on quickly. The ambulance arrived at about 08.39. The crew recorded her temperature as 39.7˚C and her pulse rate as 99. She told the paramedics that she had looked on the internet and believed she had meningitis. She complained of a stabbing pain in her head and neck, and stiffness of the neck.

11. The ambulance took Mrs Davies to Wexham Park Hospital. The Claimant followed by car. Although there is a discrepancy between the ambulance and hospital records, taking account also of the Claimant's evidence, I find that the ambulance arrived at around 09.10. The ambulance crew appear to have recorded Mrs Davies' pulse rate on arrival at 101. The hospital recorded, shortly after admission, a temperature of 39.4˚C and a pulse rate of 95.

12. After triage, at around 09.41 Mrs Davies was seen by a Foundation Year 2 General Practice SHO, Dr Mukundu. She described her headache as a sharp stabbing pain. On a pain scale of 10, she put it at 11. Right-sided neck pain was noted. In light of the Claimant's evidence, and the ambulance crew record, I find that she also in fact by this time had some neck stiffness. Her Glasgow Coma Score (GCS) was assessed at 15. The Claimant at a certain point took over answering questions as his wife was, in his words, "becoming delirious." At 10.10 Mrs Davies was prescribed the anti-sickness medicine Cyclizine and morphine sulphate for the pain.

13. Dr Mukundu discussed her case with the Emergency Medicine Consultant, Dr Bansal. Blood test results received at 10.30 were reviewed at 11.20. There was an elevated white cell count of 13.1 and an elevated C-reactive protein (CRP) of 72. Intravenous paracetamol was given. A CT scan was requested.

14. At 12.04 the antibiotic Augmentin was prescribed; but first Mrs Davies was sent for the CT scan. After she returned from that, the intravenous administration of Augmentin began at 13.20. The CT scan results showed the presence of intercranial

air (pneumocephalus)[1]. Mrs Davies was then seen by another Emergency Medicine Consultant, Mr Litchfield, at about 13.30. At that point her GCS was again assessed at 15. She was observed to be drowsy, with head and neck stiffness. She was moved to the resuscitation area some time after 14.00. The Claimant described her as "pretty much incoherent" by this time and "her eyes were unable to focus on me."

15. At 14.30 that day, Mrs Davies' GCS had fallen to 9 and her right pupil was dilated. She was moved to the trauma bay. A GCS of 11 was recorded at 15.35; but an hour later it was recorded as 10, and soon after that as 8. Mrs Davies was intubated and ventilated at 17.15. Later there was some cardiovascular instability. She was moved to the intensive care unit that evening. The Claimant was told that her situation was very grave.

16. At around 01.20 on Thursday 26 February 2015 there was a cardiac event of peri (imminent) arrest. Thereafter at 04.00 Mrs Davies' pupils were recorded as dilated and fixed. At the start of the morning she was still being fully sedated; but during the course of the Thursday her pupils remained dilated and unreactive and her GCS was assessed as 3. By the end of that day all sedation had been removed with a view to facilitating testing for brain stem death. Following brain stem tests during the afternoon of the Friday, 27 February 2015, showing no response, the Claimant agreed to the withdrawal of support. Brain stem death was confirmed at 20.11 that evening. Cause of death was certified as acute pneumococcal meningitis and suppurative otitis.

Aetiology and Treatment of Bacterial Meningitis

17. The experts as a group gave me a clear and coherent picture of the general mechanisms of this disease.

18. Bacterial meningitis occurs when, through one route or another, bacteria enter the lining of the brain, and proceed to multiply there. A number of different kinds of bacteria may be involved. In this case it was pneumococci (more fully referred to as streptococcus pneumoniae).

19. The source of the original infection, and the pathway that the bacteria take to the lining of the brain, vary from case to case. In this case, there was no material dispute or doubt that, by the time she went to the GP on the early evening of Tuesday 24 February 2015, Mrs Davies had a severe or acute otitis media. So there were bacteria present in her middle ear at that point.

20. The onset of meningitis occurred when the bacteria then found their way into the intercranial lining surrounding the brain. What the particular mechanism of travel was, or probably was, in this case, was the particular province of the ENT specialists. Potentially, they might have passed by some direct route from the middle ear to the intercranial lining; and/or they might have travelled there borne on the blood stream, that is, bacteraemic spread. (Another possibility, involving direct transmission from the *inner* ear, was able to be discounted by the experts in this case.)

[1] The possibility was raised, in expert evidence, that this may have been gas generated by bacteria, rather than air; but air was thought more likely; and, as we shall see, the consensus ultimately was that it was not a relevant feature for the purposes of what I had to decide.

21. The former breaks down into three possible mechanisms. The first involves the bacteria travelling through certain veins. The second occurs where a bone called the tegmen is particularly fragile or damaged, enabling the bacteria to pass through it. The third occurs where the bacteria themselves cause damage to the tegmen, creating a means by which they can then pass through.

22. In this case the ENT experts agreed that the transmission was by one or other of the direct routes of travel, or at least predominantly so, though they did not discount the possible additional presence of some bacteraemic spread. In oral evidence Mr Morrison said that there was no clear evidence of the latter. He opined that it was probably a mixture of venous transmission and the bacteria causing some damage to the tegmen and passing through that way. Overall, I conclude that the substantial route was direct, and, if there was any contribution of bacteraemia, it was not significant to what I have to decide.

23. Returning from this particular case to the general aetiology of the disease, once the bacteria have entered the brain lining, if unchecked, a sequence of events will unfold. In their joint report the microbiologists put it like this:

> "Meningitis causes death in a number of ways but most commonly by meningeal inflammation leading to tissue oedema, brain swelling and decreased blood supply to the brain tissue leading to its ultimate death."

24. The critical mechanisms are the net increase in fluid and the brain swelling, leading to raised intercranial pressure (ICP), which eventually leads to the other catastrophic sequelae. Professor Lever described it as a universal feature of bacterial meningitis.

25. In a little more detail, the presence of the bacteria triggers a defensive reaction in the host which, through one or more processes, or a combination of them, lead to inflammation and a build-up of fluid in the brain lining (both by various forms of generation and influx of fluid, and by impaired venous drainage). It also leads to brain swelling. Because the skull is a rigid enclosed chamber, these phenomena lead to increased ICP. The ICP will, at a certain point, lead to ischaemia (restriction of blood supply) and to infarction, that is, damage to the brain tissue itself. This will cause a reaction in the brain that may in turn impact on blood pressure, and so affect the heart. The impact on the brain causing swelling is, itself, a further source of increased pressure, so that the process starts to self-reinforce in a cyclical fashion. In Mrs Davies' case, the damage to the brain led to brain stem death.[2]

26. It is not in dispute that *the* frontline method of treating bacterial meningitis is by the administration of IV antibiotics. Bacteria multiply by dividing. One cell splits into two, two become four, and so on. Unchecked, the growth is therefore properly described as exponential. Antibiotics check and arrest this part of the process by disrupting the ability of the bacteria to form new cell walls. They kill the bacteria.

[2] In the course of evidence, reference was frequently made to a table showing the multiple, often overlapping, pathiophysiological mechanisms and pathways by which meningeal infection may lead to increased ICP, appearing in Tariq et al, "The Role of IPC monitoring in meningitis", *Neurological Focus* (2017) 43, 1. Though reference to this table helped the experts to explain their evidence, the issues in this case do not require me to descend to the same level of fine-grained detail.

27. As Professor Lever explained in oral evidence, when the inflammatory response is triggered, other, healing, processes, which, through various measures, seek to control and counter-balance that response, are also triggered. But in untreated bacterial meningitis the inflammatory response outruns the healing response, and ICP continues to increase. However, if antibiotics are administered, and reach the brain, soon enough, then, as the antibiotics begin to take effect, so the balance will shift, and the healing response will slow down, and eventually halt, the inflammatory response.

28. This mechanism, and the effectiveness of antibiotic treatment as such, is regarded as well-established, and consistently observed, in both the literature and clinical experience, and is reflected in the treatment guidelines. Because of this, despite its seriousness and potential deadliness, overall mortality from pneumococcal meningitis is, as we shall see, low, although various factors may place the given individual in a higher, or lower, prior risk category.

29. But the timing is critical. If, by the time antibiotics reach the brain lining, ischaemic damage has already occurred, it will plainly be too late. But, more than this, the sequence of causes and effects that I have described will not cease instantaneously when the source of infection is removed. If the overall process is too far advanced, the healing process may be unable to gain ascendance, and the progressive, and cyclically reinforcing, process generating increased ICP, and hence the ischaemic damage and infarction, will continue to unfold, leading to death. As the neurologists put it in their joint report:

> "The experts agree that early raised pressure is not a barrier to survival but that as it advances it becomes a positive feedback cycle best studied in traumatic brain injury in which under-perfusion results in ischaemia, swelling, cell death and further raised pressure."

30. It is therefore critical that the antibiotics begin to do their work before that process has reached the self-reinforcing point of no return.

31. In his second report, of December 2019, Professor Lever observed:

> "Meningitis caused by bacteria is not self-limiting. It is a medical emergency and since one cannot distinguish from the clinical picture alone whether a person presenting with symptoms and signs consistent with meningitis has a bacterial or non-bacterial cause the universally agreed approach is to treat as soon as possible on the basis that the cause is bacterial while trying to make a firm diagnosis through various laboratory tests. Hence all guidelines emphasise that treatment should be started on 'suspicion' of meningitis and that it is not necessary to have concrete diagnostic proof of a bacterial cause before starting antibiotics."

32. A little further on, he said, "All guidelines acknowledge that delay in treatment is associated with a poorer outlook."

33. Finally, I should note that there are various forms of neurosurgical intervention that may, potentially, provide a possible means of easing the pressure on the brain in some cases. But, for a combination of reasons, that proved not to be an option in this case; once again, the details of this aspect are not relevant to what I have to decide.

The Expert Evidence in Relation to This Case

34. The foregoing broad propositions about the disease and its aetiology in general were not, as such, controversial among the experts as a group in this case. As I have described, there was broad agreement about the original source, and route of travel to the brain, taken by the bacteria, and as to the mechanisms that led to death, in this particular case.

35. But the experts did not speak with one voice when it came to the questions of how far the process was advanced, in this particular case, as of 10.40 on the day of admission, and what, if any, effect IV antibiotics might have been expected to have had, had they been administered at that time. In short, there was disagreement as to whether Mrs Davies had already passed the point of no return by that time. Behind the disagreement on those broad questions lay areas of disagreement in relation to other questions, including whether Mrs Davies' case was, in some sense, unusual or exceptional.

36. These questions fell primarily within the domains of the microbiologists and the neurosurgeons. But although, on these matters, the ENT surgeons broadly deferred to the others, Mr Morrison, in particular, felt in a position to contribute a view.

37. So much in this case turns on the expert evidence, and the multi-faceted and at times fine-grained analysis to which it was subjected, that I need to give a fairly detailed account of it. I will set out what seem to me to have been the main features of the evidence of the six experts pertaining to causation in this case.

38. It should be borne in mind, when considering what the experts wrote, that until a late stage in the litigation both negligence and causation were in dispute, as was the possible time of negligence. Specifically, the amended Particulars of Claim postulated that it was negligent not to treat the case, on admission, as one of suspected meningitis, and to start IV antibiotics by 10.25. The amended Defence maintained that the case was properly treated on admission as one of uncomplicated sepsis, but admitted that, even so, administration of IV antibiotics should not have been delayed on account of the CT scan, and should have begun by 12.00. These two times were therefore sometimes used as reference points in the discussion of causation as well as of liability.

Microbiologists

39. Professor Lever, in his first report of 15 August 2016, opined that antibiotic administration should have begun, at the latest, at 10.10. He wrote:

> "In the context of meningitis the delay of three hours and 10 minutes to 13.20 is extremely significant. There are a number of publications documenting the issue of delay in antibiotic administration if meningitis is suspected. One of the most

> relevant is from Køster-Rasmussen (2008, Journal of Infection). This confirms, but more accurately quantitates, data from other publications showing that delay in antibiotic therapy correlates independently to unfavourable outcome. The odds for an unfavourable outcome may increase by up to 30% per hour of treatment delay. In this publication where outcome was determined in 184 cases and adult mortality was 33%, mortality was 10% for a delay in administration of antibiotics by up to 2 hours, but rose to 30% with a delay of 2 – 4 hours. Thus a more than doubling of mortality rate occurred. Although this cannot be extrapolated exactly for individual cases it suggests that the delay experienced by GD may have more than doubled her risk of death"

40. Further on, Professor Lever wrote that, had antibiotics been administered within the first hour "on the balance of probabilities … GD would have survived. Other than the delay in administration of antibiotics I do not think that actions subsequent to this materially affected her prognosis."

41. In a letter of 24 August 2016 Professor Lever responded to various questions. He was asked to address a scenario in which antibiotics had been administered at 10.10, *and* pneumocephalus had developed. Would the meningitis still have been treatable? He replied: "It was still potentially treatable even with pneumocephalus." He was asked whether the mortality rates he had quoted applied to meningitis arising from otitis media as opposed to other sources. He replied: "These are global figures but since the ear is a common site for infection that leads to meningitis they would likely apply to this situation."

42. Professor Lever wrote a further letter of 22 September 2016, specifically commenting on a report prepared by a microbiologist, Dr Gant, for the coroner, which raised the possible significance of pneumocephalus. In his response he cited literature indicating that there was no particular association of pneumocephalus with a worse outcome. He reiterated his opinion that in Mrs Davies' case treatment was unnecessarily delayed. "As mentioned previously the mortality rate rises significantly with every hour of delay."

43. Professor Lever concluded that letter as follows:

> "The rate of clinical deterioration of GD was rapid, however, we have no definitive way of knowing the exact time, and whether it was before or during her admission, at which bacteria first invaded her meninges, or the size of the invasive inoculum, or the virulence of the organism however I do not consider that the rate of progression of the illness was truly exceptional for an organism such as *Streptococcus pneumonia* in which fatality rates of 22% among adults in the Western world are quoted.
>
> Appropriate antibiotics given within an hour of admission to hospital would, I believe, on the balance of probabilities, have given GD a reasonable chance of survival."

44. In his second report, of December 2019, Professor Lever referred again to Køster-Rasmussen et al describing a 30% worsening of prognosis for every hour of delay. He wrote that a "similar trend is noted" in two other papers: Bodilsen et al (2016), and Proulx et al (2005).[3] Those presenting with less advanced illness "as judged by a higher Glasgow Coma Score" have a better outlook. He cited Schutte et al (1998) as indicating that 88% of those with a GCS greater than 12 have a good outcome and 88% of those with a GCS less than 8, a poor outcome.[4] He observed that poorer prognosis in bacterial meningitis was associated with those in the over 60s age group and who are obtunded (exhibiting depressed consciousness) on admission, citing Durand et al (1991).[5] He observed that intravenous antibiotics "will kill the bacteria … very quickly and reverse the pathology and it is not uncommon to see improvement, or at the very least, arrest of decline, within 30 – 60 minutes of administration."

45. Professor Lever concluded that report as follows:

> "44. The rapidity of her decline relates directly to the delay in giving her antibiotics. Delay in antibiotics is a greater risk in cases of meningitis than is the presence of a higher disease severity. In other words, antibiotic delay has a more profound negative effect on outcome than does the degree of severity of the presentation. I would emphasise again that we have no evidence to suggest that this particular organism was in itself particularly virulent; it may or may not have been, but whether it was is not germane to the argument. Whatever the level of virulence of an organism, it does not influence how susceptible it is to antibiotics. Antibiotics given at an appropriate time … as soon as possible in the case of meningitis … are more important than the completely speculative level of virulence of the organism involved.
>
> 45. GD on arrival at hospital had a GCS which put her in a group of predicted favourable neurological outcome, had she been treated. She was not obtunded or in an elderly age group to put her at increased risk of poor outcome. There is no reason to suppose that her case was atypical and she does not have any prior medical history suggestive that she would be expected to have a worse outcome than average.
>
> 46. My belief is that had IV antibiotics been given at 10.25 instead of 13.20 GD would have survived. I also believe that

[3] Køster-Rasmussen et al "Antibiotic treatment delay and outcome in acute bacterial meningitis", *Journal of Infection* (2008) 57, 449; Bodilsen et al, "Time to antibiotic therapy and outcome in bacterial meningitis: a Danish population-based cohort study", *BMC Infectious Diseases* (2016) 16:392; Proulx et al, "Delays in the administration of antibiotics are associated with mortality from adult acute bacterial meningitis", *Q J Med* (2005) 98, 291.

[4] Schutte et al, "A Prospective Study of Glasgow Coma Scale (GCS), Age, CSF-Neutrophil Count, and CSF-Protein and Glucose Levels as Prognostic Indicators in 100 Adult Patients with Meningitis", *Journal of Infection* (1998) 37, 112.

[5] Durand et al, "Acute Bacterial Meningitis in Adults", *The New England Journal of Medicine* (1993) 328, 21.

> had they been administered at any time up to 12.00 then on the balance of probabilities GD would have survived."

46. In a letter of 4 June 2020 Professor Lever was asked to comment on his use, in the letter of 22 September 2016, of the phrase "reasonable chance of survival". He acknowledged that this "appears much less emphatic" than his opinion of December 2019, but said that the use of this term was "careless", and that by "reasonable" he meant a "good chance"; and he referred to his reference to "balance of probabilities" earlier in the same sentence. He noted that this letter was by way of response to Dr Gant, and that, at the time, the focus was on the failure to administer antibiotics within an hour, in accordance with the Defendant's own protocol. He was not asked at that time to comment on the latest possible time for survival. Had he been asked to do so, he would have given the opinion subsequently given in his report of December 2019.

47. In his report of November 2019 Professor Masterton opined that on admission it was acceptable for Mrs Davies to be managed as an uncomplicated sepsis case, but nevertheless a delay of 4 hours and 10 minutes was unacceptable. Augmentin was prescribed at 12.04 but not administered until 13.20, because she went first to imaging. That was unacceptable. However, it was not a breach of duty to fail to administer antibiotics before 12.00; particularly as the blood test results did not indicate the presence of a very severe infection. In his opinion it was probable that the intercranial infection was present at the time of admission "though I accept that she was not showing a suite of typical signs and symptoms of same." This was indicative not of primary pneumococcal meningitis but of secondary spread from primary ear infection.

48. As to whether the outcome would have been different had antibiotics been administered by around 10.30 or by around 12.00, Professor Masterton wrote:

> "In my opinion, this case was very unusual with Mrs Davies showing a very rapid progression for which there is no obvious explanation.
>
> In these circumstances it is my opinion, on a balance of probabilities basis, that an earlier administration of antibiotics, against either of the above timelines, would not have altered the outcome in this case.
>
> I reach this conclusion because the delay in providing effective antibiotics rested, as demonstrated above, between 3 to 1.5 hours and it is unlikely that such a relatively short earlier administration would have been able to have altered the course of the infection such that the outcome would have been Mrs Davies' survival."

49. Professor Masterton disagreed with Professor Lever's reliance on medical literature, which, he said, referred to adult primary meningitis of all causes, and were not applicable to Mrs Davies' case, where the primary source was an ear infection and the pathogen was a pneumococcus "which is known to have a higher mortality rate than other types of meningitis."

50. In their joint statement of March 2020, after outlining, in the passage I have already cited, the process by which meningitis typically causes death, these two experts continued that: "The earliest stages of this are reversible with appropriate treatment but become progressively less reversible as treatment is delayed." However, they disagreed on whether it was reversible in this case.

51. Professor Lever stated that in his clinical experience of treating meningitis, and the literature, Mrs Davies "was in a low risk group of being young and otherwise fit without predisposing co-morbidities; she presented in a conscious state and was not obtunded". He referred to an overall mortality rate for pneumococcal meningitis of 20% and to Østergaard et al[6] reporting mortality secondary to otitis media at 9%. I interpose that in oral evidence Professor Lever corrected this, as the figure actually given in the paper is 7%. His contribution to the joint report continued that, as many of the patients in the latter group had additional predisposing conditions, Mrs Davies' predicted mortality was likely lower. His belief was that "with prompt treatment the deceased would have survived."

52. Professor Masterton agreed with Professor Lever's conclusion, and reasoning, as to Mrs Davies being in a low-risk group at admission. But he disagreed with the reliance on Østergaard et al, as the 9% outcome [*sic*] was measured at 100 days after admission. For those who died within two days of admission "no such benefit was observed." Nor did that paper include a multivariate analysis of the significance of treatment delay in otogenic disease. Mrs Davies was "a very unusual case who showed an extremely rapid progression for which there was no obvious explanation". In his clinical experience such instances did occur. No evidence in the literature "permitted it to be argued on a balance of probabilities that earlier intervention by around up to a maximum of 3 hours" would have altered the outcome.

53. These experts did agree that the infection had likely entered Mrs Davies' brain cavity prior to admission. Professor Lever considered that raised ICP was already present when Mrs Davies was admitted. Professor Masterton considered that the answer to this depended on the Court's findings, in particular, as to the presence, or not, of neck stiffness at that time.

54. Professor Lever opined that, had antibiotics been administered at any time up to 12.00, Mrs Davies would, on the balance of probabilities, have survived, on the basis of the literature, her low-risk group, and his clinical experience. Antibiotics given later "might" also have led to survival. He had seen "a significant number of patients in severe risk groups who are obtunded and with other bad prognostic futures rescued from a severe state of the disease."

55. Professor Masterton noted that "in his clinical experience of advising on the management of patients with fulminant pneumococcal septicaemia and meningitis, which was a relatively small number of cases as this condition is very unusual, then the outcome of death tended to be inevitable and not apparently responsive to short timeline interventions."

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[6] Østergaard et al, "Clinical presentation and prognostic factors of Streptococcus pneumoniae meningitis according the focus of infection", *BMC Infectious Diseases* (2005) 5:93.

56. These experts were asked, in the event that they were unable to give a view on the balance of probabilities, whether they considered that provision of antibiotics by any given time would have made a "more than minimal contribution" to survival. They observed that, as they had both expressed balance of probabilities views, "this does not apply."

57. In oral evidence Professor Lever speculated that otitis media cases have a lower mortality rate because there is already an inflammatory process triggering a local reaction before meningitis ensues. He considered Mrs Davies' symptoms to be "textbook", and the rate of advance in her case to be not unusually different to a number of cases he had seen. In cross-examination he agreed that the overnight vomiting and headache on the 24th/25th were likely indicators that the infection had begun to spread to the brain, and of raised ICP. But it was difficult to judge the stage reached on arrival at hospital. The clinical criteria were very crude and we did not have an exact measure of raised ICP. As to speed of development, he accepted that she was towards the rapid end, but not exceptionally rapid. He considered that she appeared stable, but then went downhill rapidly from about 14.00, probably because a tipping point of catastrophic changes in the brain was reached around that time.

58. It was put to Professor Lever that, contrary to his June 2020 letter, there was a marked change in his opinion from the language of his 2016 report to that of the subsequent letters of 2016, which was not down to mere carelessness. He agreed that it might seem that way to the reader, but maintained that his opinion, and the meaning that he had intended, had not changed.

59. It was also put to him, in so many words, that his depiction of the literature was skewed. Køster-Rasmussen et al was in fact more favourable to the Claimant's case than the other two papers that he mentioned; and the account in his December 2019 report, lacked the qualifier that delay "may" contribute to increased risk, that he had used in his 2016 report. Further, the type of bacteria, origin of the infection, and age, were all significant variables.

60. Professor Lever's broad response was that he accepted the various limitations of the literature that Mr Barnes raised. He had written in his report that it was hard to extrapolate from the literature. He could find flaws in all the papers that he had cited. There were no randomised double-blind controlled trials in which antibiotics were administered to one group but not the other, because it would be unethical to conduct them. But all the patient data studies pointed to the importance of early treatment. He did not accept that he had not been even-handed. He had highlighted Køster-Rasmussen, because it was the one that gave an hour-by-hour analysis. He repeated that, unusually, treatment is recommended on suspicion of meningitis, even before a confirmed diagnosis. It remained his belief that Mrs Davies had a perfectly treatable infection.

61. Challenged as to his assertion that delay was more significant than the severity of the particular disease, Professor Lever pointed to the multivariate analysis in Køster-Rasmussen et al, showing delay as statistically significant, with an odds ratio of 1.3 for those treated within 12 hours. He accepted that it was 1.09 for the whole cohort, but not the criticism that he should have cited both. He said he had also had in mind another source, when observing that delay was more significant than severity, but could not recall what it was.

62. It was put to him that Mrs Davies' pre-disposition and GCS indicators at 13.20 were the same as at admission, yet she did not survive – which showed that there was something else exceptional about her case. He replied that the indicators were the same, taken in isolation, but this did not take account of the clinical context. The disease did not stand still. Things had moved on. You had to consider the overall clinical picture and trajectory. We know that she declined rapidly after 13.20, so her condition must have been worse at that time. He would have agreed that Mrs Davies' case was exceptional, had she been treated with IV antibiotics *at 10.40* and yet not survived. He had seen another case of delay of 2 ½ hours where, after antibiotics were administered, the patient went downhill.

63. In further cross-examination Professor Lever said that where bacteria were present in the cerebral spinal fluid (CSF), antibiotic treatment could show an improvement in consciousness level within half an hour to an hour. Further, once the antibiotic was established in the brain, it would neutralise any bacteria continuing to come in from the ear.

64. In oral evidence Professor Masterton agreed that, once present in the CSF, the antibiotics would neutralise any bacteria continuing to enter from the ear. He maintained that the rapidity in Mrs Davies' case was exceptional: first because of the pneumocephalus, which he had never before seen; secondly, because she effectively died within 24 hours. This was a fulminant disease – an overwhelming infection that kept going. It was rare, but he had seen it.

65. Professor Masterton accepted that the figure of 4 hours he had given to "sterilise" bacteria in CSF was a timescale to reach complete elimination. He agreed that it was possible to see an impact within 30 minutes to one hour. The various specific points that the Claimant's witnesses took from the various academic literature were put to him, and he accepted them, as such. He agreed with Professor Lever's overall description of the general aetiology of the disease; and as to the universality of the guidelines. Questioned about the five cases he had seen of "fulminant pneumococcal meningitis" Professor Masterton could remember that they were adults, and not HIV cases, but acknowledged that he did not have direct management of any of them and that, beyond this, his recollection of them was sketchy.

66. Professor Masterton agreed with Professor Lever's description of the process reaching a point of sudden decline towards the end. He said this was well-recognised. You fall off a cliff edge; you can no longer cope, and then deteriorate very rapidly and severely. He accepted that pneumocephalus was of no significance for prognosis or causation.

67. Professor Masterton agreed with Professor Lever that the way in which Mrs Davies' case developed up to the point of admission was not unusually rapid, and was within the range encountered; but he referred to a period of 24 hours to brain death as being unusual. In further evidence, he explained that, by this he meant that it was the rapidity of the process from the start of Mrs Davies' noted deterioration at 2.30 on the afternoon of admission, through to what he took to be effective brain death by some time the next day, that was unusual. What happened up to midday on the day of admission was to be expected. It was after that, that the deterioration became evident and the body went beyond its ability to compensate. However, it was likely that the brain injury that led to that conclusion had occurred several hours before.

ENT experts

68. Mr Morrison, in his report of 16 January 2020, commenting on the suggestion that the speed of decline in this case was exceptional, said: "I do not think there was anything exceptional about this patient or this organism." He stated that he had to defer to the opinion of the microbiologists regarding whether immediate IV antibiotics would have prevented death.

> "But I believe high doses of the appropriate IV antibiotic given early will have a beneficial and important effect within an hour. I believe that the delay in giving intravenous antibiotic therapy by 4 hours must have contributed to the irreversible meningitis and death. I defer to Professor Lever in relation to the latest time when IV antibiotics would have avoided Mrs Davies' death. I am aware that I must not be swayed by any other experts' opinions and I do believe my view is my own and independent. I do not consider that Mrs Davies' death was inevitable and any significant delay in giving intravenous antibiotics would certainly have contributed to her deterioration and death."

69. Mr Morrison cited Bodilsen et al (above) to the effect that increased mortality was found when treatment delays exceeded six hours compared with delays within only two hours of admission, though the number of variables meant it was "difficult to extrapolate" to Mrs Davies' case. Nevertheless, it showed that every delay above two and up to six hours added 10% to mortality. Albers[7] reviewed 23 patients with intracranial and complicated otitis media. High morbidity and mortality were correlated between the early symptomology and the start of treatment, with doctors' delay the most significant factor. Auburtin et al (2006)[8] identified variables independently associated with 3-month mortality, one of which was an interval of more than three hours from admission to administration of antibiotics. Delay was a predictor of mortality in patients with pneumococcal meningitis.

70. Mr Morrison concluded: "Based on personal experience, as well as the literature and considering the opinions of the other experts, I believe on a balance of probability that this failure of duty of care led to the death of Mrs Gabrielle Davies."

71. Mr Hawthorne, in his November 2019 report, said: "This lady had a rapidly progressive fulminating infection which rapidly spread from the right middle ear to the meninges." As to the time that antibiotics should be given, and the effects of giving or not giving them at different times, he deferred to the microbiology, emergency medicine and/or neurosurgery experts.

72. In their joint report, as to whether there was a rapid decline, and if so, when, they jointly deferred to other experts, but, with that proviso, expressed a joint opinion that, if there was vomiting, severe right hemisphere headache and raised temperature overnight on the 24th/25th "this indicated a decline of the situation from a normal

[7] Frans Albers, "Complications of Otitis Media", *The American Journal of Otology* (1999) 20: 9.
[8] Auburtin et al "Detrimental role of delayed antibiotic administration and penicillin-nonsusceptible strains in adult intensive care unit patients with pneumococcal meningitis: The PNEUMOREA prospective multicenter study", *Critical Care Medicine* (2006) 34, 2758.

acute otitis media"; and that "the next point at which there was an evidence of a rapid decline was at 1430 hours". Mr Morrison felt that, by that time "the condition was extremely severe." Though they deferred to the neurosurgeons, raised ICP had probably developed by that point.

73. In oral evidence Mr Morrison referred to having seen 4 – 5 cases a year of ear infections spreading to intercranial meningitis. He referred to Auburtin et al's analysis of parameters associated with 3-month survival of this strain of bacterial meningitis. Their multivariate analysis identified an interval to treatment of greater than 3 hours as having an odds ratio of more than 14, with a 95% confidence interval of just under 4 to just over 50 (and low p-value). He had seen cases of comparable severity to Mrs Davies' not lead to death.

74. Cross-examined as to the disparity between his observation that the delay "must have contributed" to death and that "on the balance of probability" it led to death he said he had a problem with the idea of material contribution: either you die or you don't. He said his opinion was that if she had been given antibiotics within two hours she would have survived, based on his 30 years' experience and the literature. Of the 100 or so cases of spread he had seen, 15 or 20 had full meningitis. He accepted that only six of these were both adults and secondary to otitis media, but maintained that he saw a lot of cases of how quickly antibiotics work. He did not claim to know more than a neurosurgeon, but he regarded it as part of his expertise. He examined students on it.

75. Mr Morrison said that it would take about an hour for the antibiotics to start to clear bacteria from the meninges. It would take as much as a day or two to completely resolve the infection in the ear; but the primary and driving source of infection had moved to the brain. He considered that you would expect to see an improvement in vital signs within a couple of hours. He maintained that he had sufficient experience and understanding to give an opinion.

76. Cross-examined about the literature, Mr Morrison accepted the limitations of some of the papers, for example in terms of the low numbers involved, overall or in sub-groups directly comparable to Mrs Davies. But he maintained in particular that the multivariate analysis in Auburtin et al, showing a high odds ratio in relation to a delay of three hours or more, is significant.

77. When it was put to him that Mrs Davies' clinical markers were the same at 10.40 and at 13.20, and yet she died, and therefore was exceptional, Mr Morrison responded that no-one would say she was the same three hours later; the position only became apparent at 14.30. He did not accept that she was "moribund" when she came in. He accepted that the rapidity of her disease was shorter than most of the literature, probably twice as quick as most.

78. Mr Hawthorne confirmed in oral evidence that he deferred to the microbiologists and neurosurgeons on causation. He could not recall having seen a case of someone at the GP of an evening and 24 hours later "virtually brain dead." On the balance of probabilities the meningitis was under way overnight on the 24th/25th.

Neurosurgeons

79. Mr Norris, in his report of 27 December 2019, opined that Mrs Davies "died from a treatable middle ear and brain infection/meningitis" and agreed with Professor Lever that she was "salvageable" had treatment been expedited on arrival. Further on, he wrote that the GCS at 2.30 indicated that she was "nearly comatose." The eye observations at that time also represented "a very serious decline in her neurological condition." Further on he wrote: "The onset of coma in the presence of a poorly/partially treated meningitis dramatically increases the morbidity and mortality of the cranial sepsis."

80. He concluded:

> "Patients who 'walk and talk' on arrival at hospital have a good chance of survival if 'triaged and treated' urgently with IV antibiotics. Had IV antibiotics been given three hours sooner at 10.25 instead of 13.20 then on a strong balance of probabilities the Deceased would have survived. A delay in iv antibiotics any time up to 12:00 on a balance of probability she would have survived."[9]

81. Mr Crocker, in his report of November 2019, described Mrs Davies' deterioration as "unusually rapid" and her infection as "rampant." He wrote:

> "Antibiotics alone will not treat the raised intracranial pressure of infection and only if given before the start of the vicious cycle will they prevent progression to infarction. It is my opinion that by the time the Deceased started to develop a severe headache and vomiting the meningitic process was becoming well established and her death was, on the balance of probability, inevitable."

82. Further on, he wrote that Mrs Davies was suffering "fulminant pneumococcal meningitis" on admission. Had she been given antibiotics earlier "the inflammatory reaction that resulted in brain swelling and death would not have been arrested in time to prevent progression to death." While deferring to the microbiologists he highlighted the distinction between administration of antibiotics to prevent systemic sepsis and their impact in relation to severe brain infections, which requires them first to cross the blood brain barrier:

> "… and I would therefore suggest that the application of 'antibiotics within the hour' principles to severe brain infections is sadly less time critical than would be hoped: if there is (as in this case) a fulminant brain infection and a deteriorating patient I do not think the immediacy of antibiotic management will likely resolve the problem whereas this is more likely to be the case with systemic sepsis."

[9] Underlining in the original.

83. In their joint report of April 2020 these experts noted that neurosurgeons rarely treat meningitis, but observed:

> "They agree that overall patients with pneumococcal meningitis that present alert to hospital are not likely to die on balance of probability. The prognosis will be influenced by comorbidities of the patient (the deceased was normally well), rate of progression of the neurological sequelae with rapidly progressive disease having a worse outlook, and any undue delay in instituting treatment."

84. They were asked what factors on presentation in this case were suggestive of likelihood of (a) survival; and (b) death. They replied:

> "Survival would have been more likely as the deceased was orientated, rather than comatose, and premorbidly healthy.
>
> Death would have been more likely due to the rapid deterioration during the course of the day 25.02.2015 which is indicative of a fulminate version of meningitis."

85. They observed that Mrs Davies was apparently orientated during the morning "but once she started to deteriorate within a short period (approx. 2.5 hours) she was GCS 9 at 1430 which is borderline comatose." She went from alert to comatose between assessments. "This is a rapid decline. Mr Litchfield saw her at 1330 and recorded GCS 15/15 but drowsy (sic). The experts agree that the decline in the deceased's condition had started by this time."

86. Mr Norris considered that consciousness level was the best indicator of raised ICP, and, as GCS was 15 at 13.30, ICP was at that time normal. Mr Crocker considered that ICP can be elevated "without alteration of conscious level which follows as the condition progresses" and ICP was probably elevated even on arrival. However, they agreed that intercranial infection was, on balance, of itself, survivable, and (in a passage I cited earlier) that early raised pressure was not a barrier to survival, but as it advances it becomes a positive feedback cycle. Pneumocephalus by itself carried minimal prognostic weight.

87. Asked at what point in time at the latest IV antibiotics would have had to begin for Mrs Davies to have survived, Mr Norris "feels it should have been as soon as possible or at least within 1 hour of arrival at hospital." Mr Crocker said that "antibiotics would have to have been given substantially earlier than midday to have any prospect of preventing the rapid deterioration seen which was inevitably fatal. He states that antibiotics given any time after 10am would not have prevented death."

88. Asked whether, if they could *not* give a balance of probabilities assessment at a particular point in time, they could say whether antibiotics would at that time "have made a more than a minimal contribution" to survival, they referred back to the earlier answers, but added that Mr Crocker "feels that antibiotics after 10am would not have enabled survival." Mr Norris was of the view that antibiotics given within one hour "is probably survivable but if delayed by 3 or more hours death/stroke is probably inevitable and unsurvivable."

89. In a letter of 12 June 2020 Mr Norris referred to the joint statement and wrote:

> "I consider that had intravenous antibiotics been given within one hour (about 10:10) then, on the balance of probabilities, Mrs Davies would have survived.
>
> Had they not been given for three hours (about 12:00) then I believe that, on the balance of probabilities, she would have died.
>
> Between those times, I consider that the outcome is uncertain and I am not therefore able to express a view on the balance of probabilities. However, I can say with confidence that during the time Mrs Davies deteriorated I consider that any delay between about 10:10 and 12:00 made a material contribution to her decline and death."

90. In a letter of 14 October 2020 Mr Crocker clarified what he had written in the joint statement. It was not to be inferred from his reference to 10:00 am that he considered that antibiotics before that time would have prevented death. It was impossible to identify the tipping point. That time was illustrative. "In my opinion the Deceased arrived at hospital with a fulminant infection. Antibiotics, at any time after admission, whilst appropriate, would only have treated one aspect of her deteriorating condition and for this reason antibiotics could not have been given at a reasonable time to prevent her death."

91. Mr Norris, in cross-examination, agreed that the fixed and dilated pupils following the cardiac event early on the 26th were indicators of very substantial brain-stem injury, and that the process over that night was rapid. He was asked about the change from his original report: "strong" balance of probabilities at 10.25 – to his letter of 9 June 2020: balance of probabilities views at 10.10 and 12.00, but not in between. He said that the records had not changed, but as the process proceeded he became more cautious and "felt I had to moderate. I've modified my opinion." This was the product of ruminating and mulling over: "I was not so sure."

92. Mr Norris maintained that the level of GCS was the most reliable indicator of when the ICP became raised in this case. The views of others, that it had begun by the time of admission, were supposition, in the absence of a lumbar puncture or other ICP monitor reading. He agreed, however, that earlier raised ICP would have a negative prognostic implication. You could have a "reasonable guess" at it from other clinical observations. His opinion on the contribution of the delay to increased risk was based on clinical experience.

93. In oral evidence Mr Crocker noted that brain-stem death testing is a very strict and specific process, but there was no evidence of brain stem function in this case after 02.00 on 26 February. In cross-examination he agreed with Professor Lever that, while the overnight vomiting and headache on the 24th/25th signified the onset of meningitis, people could recover from this. However, he used the word "fulminant" to convey the specific meaning that the case was one in which the disease has struck so rapidly that, by the time the clinicians realise that it is striking, they are on the back foot and the disease has the upper hand. Raised ICP for any brain condition was not a good thing, but its relationship with GCS score in meningitis was "unknown".

Clinical observation was not as reliable as we would like it to be. For example (at 13.30 on the day of admission) Mrs Davies had a GCS of 15 when she was also "drowsy". Patients reached a point of falling off a cliff.

94. Mr Crocker said he had gone for a time of 10.00 because he felt confident and comfortable about it. He agreed with Mr Norris' construct – a time before which the balance of probability favoured survival, a later time after which the balance pointed to death, and a period of uncertainty in between – but not his actual timings. He accepted that Mr Norris was much more clinically experienced, but they had a different view of the contribution of the antibiotics. In his view, after 10.00 was far too late in the "natural history" of the process, because, even after they were given, she continued to deteriorate in a "fulminant" manner. The inflammatory cascade was a major feature and a reason why her deterioration was "fulminant".

Causation on the Balance of Probabilities – Argument

95. I had the benefit of clear opening skeletons, and a full day of oral closing argument, drawing on very detailed final written submissions on both sides. I thank both counsel for it. I have reflected on it all.

96. In this section I consider the arguments about the expert evidence and as to what conclusions I should draw from it all, about whether, had she been given IV antibiotics from 10.40 am on the day of admission, Mrs Davies would, on the balance of probabilities, have survived. I shall first summarise what struck me as the main, and most significant features, of the argument on each side.

Claimant

97. Mr Charles submitted that Professor Lever has extensive clinical experience. Over 30 years as a consultant in infectious diseases with a microbiology qualification, he has dealt with an average of ten pneumococcal meningitis cases a year. He has published on meningitis. Professor Masterton, he said, had relinquished direct responsibility for patient care in 1991, and his clinical work from 2001 was limited.

98. Mr Morrison had experience in meningitis cases (including spread from otitis media and this particular bacterium). Over a 36-year career he had significant experience in the day-to-day efficacy of antibiotics. Mr Hawthorne's opinion on causation was very limited.

99. Mr Norris specialises in brain surgery. Mr Crocker, while he already has an impressive CV, himself acknowledged Mr Norris' much greater experience. He largely deferred to the microbiologists whilst asserting that neurosurgeons are pre-eminent as to the final stages of the process.

100. Professor Lever was clear that on the balance of probabilities Mrs Davies would have survived had she been given antibiotics as late as 12 noon. Mr Morrison's view was also strongly held, and maintained under cross-examination. These views were underpinned by the (eventual) consensus among Professors Lever and Masterton, and Mr Morrison, that improvement in symptoms can be seen in a range of half an hour to one to two hours.

101. Mrs Davies' age group and lack of prior co-morbidities were in her favour, as was her overall state on arrival at hospital. The overall picture from the literature, of the benefits of early administration of antibiotics, was clear. Professor Lever and Mr Morrison agreed that her indicators at the time of admission pointed to a good outcome. Once antibiotics reached the brain, they would also take care of any bacteria continuing to enter from the middle ear.

102. It was common ground that raised inter-cranial pressure will be a feature of meningitis, but, even if it was raised in Mrs Davies' case by the time of admission, there was no real evidence that it reached a dangerous or prognostically significant level until 14.30 on the day of admission, when the collapse in GCS was recorded. There was a rapid decline from that time, leading to major damage by the early hours of 26th February. The consensus was that the pneumocephalus had no causative significance.

103. There was consensus among the experts that at a certain point in the aetiology of the disease, when what was described as the vicious cycle or self-feeding fire has begun, antibiotic treatment will come too late. Professor Masterton agreed with Professor Lever's view that decline did not follow a linear pattern. At a certain point there was a cliff edge and the body was unable to compensate. He put that point in this case as 14.30 on the day of admission.

104. It was contended for the Defendant that Mrs Davies' decline was exceptionally rapid and that she had an unusually "fulminant" infection; but the Defendant's experts did not point to any literature in support of this. Mr Crocker had very limited clinical experience. The five cases to which Professor Masterton referred were raised by him for the first time in oral evidence, and his recollection of them was poor and lacking in detail.

105. By contrast Professor Lever and Mr Morrison were both well able to draw on clinical experience. Mr Norris had not fundamentally changed his position, but, on reflection, had been more circumspect, as was appropriate, given that he is a neurosurgeon. While approaching the literature with due caution, the multivariate analysis in Auburtin et al, showing a 14-fold increase in mortality for treatment of pneumococcal meningitis after three hours, with high significance, was important. So was the Durand et al[10] study of 493 episodes of acute bacterial meningitis. Their univariate analysis associated three factors with higher mortality in single-episode community-acquired cases: age over 60, obtunded mental state on admission and seizure within 24 hours of admission. Of those who died, 98% had at least one. Mrs Davies had none.

106. This, said Mr Charles, was a case like *Schembri v Marshall* [2020] EWCA Civ 358. The statistics emerging from the literature could properly be drawn upon to support the conclusion, on the balance of probabilities, that, had she been treated promptly, Mrs Davies would have been in the majority who survive.

[10] Note 5 above.

Defendant

107. Mr Barnes submitted that none of Professor Masterton or Messrs Crocker or Hawthorne supported the Claimant's case. Mr Norris' considered view, after further reflection, did not support the Claimant's case. While Mr Morrison gave evidence in support of the Claimant, he also deferred to other experts on causation. Further, on examination, it was apparent that he had very little, and only indirect, experience of cases of meningitis secondary to otitis media.

108. The Claimant was, submitted Mr Barnes, "left relying solely on Professor Lever." He suggested that Professor Lever had four main points: (a) it is established that delay in antibiotic treatment is associated with poorer outlook, hour by hour; (b) once administered, antibiotics will quickly slow and reverse the pathology. Improvement or at least arrest of decline can be seen within 30 minutes to one hour; (c) delay in administering antibiotics is a greater risk than the presence of a higher severity of disease; (d) at 10.40 Mrs Davies was in a group with a good prognosis and her case was not unusual or atypical. But, on analysis, each of these was a misleading simplification.

109. As to (a) the literature had only limited value, given the small numbers in the studies relied upon, and that only a subset of these were adults with pneumococcal meningitis secondary to acute otitis media. Professor Lever's use of the literature was also skewed: Køster-Rasmussen et al found two odds ratios for late treatment, 1.3 under 12 hours, but 1.09 overall. Both should have been considered. Bodilsen et al and Proulx et al did *not* find similar results. They were less favourable to the Claimant's case. The former found mortality rates increased from 14% in the first two hours, to 16% from two to four hours, to 20% from 4 – 6 hours, and 30% above 6 hours; but the results before six hours were not statistically significant. In the latter, the figures for the same first three time intervals were 5%, 5%, and 6%. Køster-Rasmussen et al referred to other papers correlating delay to worse outcome where the disease was at a *late stage*. Professor Lever was obliged to concede in cross-examination that all that could be said was that there may be an association been delay in administering antibiotics and prognosis.

110. This was a case where Lord Nicholls of Birkenhead's words of caution as to the use of statistics in *Gregg v Scott* [2005] 2 AC 176 at [28] were in point.

111. As to (b), Professor Lever failed to take account of the fact that antibiotics will take longer to sterilise bacteria in the brain than in the blood. He failed also to explain that, even after the bacteria in the brain have been sterilised, the cyclical process of the disease, if sufficiently advanced, will persist. These were points that he was obliged to accept in cross-examination.

112. As to (c) Professor Lever was unable, when cross-examined, to cite a basis for this proposition; and therefore no weight should be placed upon it.

113. As to (d) Mr Barnes accepted that, on the basis of the literature, in particular, Østergaard et al, Mrs Davies would have been in a low-risk group at 10.40 am on the day of admission. But the difficulty with this was that, on the basis of the same literature, the same could have been said of her at 13.20. Yet administration of antibiotics at that time did not save her. The assumption derived from the literature

therefore could not be relied upon in her case. Professor Lever's reliance on his own clinical experience of patients with good prognostic indicators, was similarly flawed, because it failed to engage with this feature of Mrs Davies' case.

114. Mr Barnes submitted that there were further reasons to doubt Professor Lever's objectivity. At one point, when asked about the statistical significance of the low numbers in a study, he referred to the death of one as one too many, and referred to Mrs Davies' husband and children. His opinion on causation in his 2016 letters was not as strong as it was in his 2019 opinion. His view of the impact of the delay in this case was more guarded in his 2016 opinion than in his 2019 opinion. His explanations for these shifts were unconvincing.

115. There was, however, an explanation for why Mrs Davies, despite the literature and Professor Lever's experience suggesting that her prospects were good, did not survive. This was that her deterioration was unusually rapid. The ENT surgeons agreed about that. Professor Lever accepted that it was at the rapid end of the spectrum. Professor Masterton described her disease as "fulminant". The neurosurgeons agreed that there was a rapid deterioration, indicative of a fulminant version of meningitis. In short, Mrs Davies died because she had a fulminant and severe bacterial meningitis; and, because she had a fulminant and severe disease, it would also probably have led to her death, even had she received antibiotics at 10.40 am.

Causation on the balance of probabilities – Discussion and Conclusion

The Law

116. If asked what *caused* Mrs Davies' death, the lay person might simply say that she died of bacterial meningitis consequent on a middle ear infection. That is, indeed, what the death certificate, in more clinical language, says. It is what actually happened.

117. But because the Defendant was, it has been admitted and agreed, negligent, by failing to begin the administration of intravenous antibiotics by 10.40 am on the day of admission, the question that I had to determine, to which the law gives the label of "causation", is what would have happened if antibiotics *had* been administered at that time. Because this did not actually happen, I cannot be sure. I have to decide on the balance of probabilities. That is to say, I have to consider, *if* that had happened, is it more likely that Mrs Davies would have survived than died; or more likely that she would have died than survived?

118. I have to do that by drawing, appropriately, on the picture painted by *all* of the evidence available to me. The first source is evidence about Mrs Davies and what actually did happen in her case. The second is the evidence of the experts, which in turn draws on their respective experience and judgment, and on the literature to which they referred, and which was in my bundle.

119. In deciding *causation* the Court can, and should gain whatever insight it properly can from hindsight. This can include, where appropriate, drawing proper inferences from later events where they may cast retrospective inferential light back, on what was happening earlier on. What no-one can do is make good gaps in the evidence. The Court has to do the best it can with what it has got. In this case, for example, as a

matter of fact, Mrs Davies never had a lumbar puncture or other ICP monitoring. So I have no evidence of a direct measured read of her CSF pressure level at any point in time.

120. In *Gregg v Scott* [2005] 2 AC 186 Lord Nicholls of Birkenhead, at [28], observed that "[s]tatistical evidence, however, is not strictly a guide to what would have happened in one particular case" and expanded on what he meant. He also further expanded on this at [32]. I do not need to set out these passages. They are wise words from a source of high eminence, about the care to be applied when invited to extrapolate from the literature to the particular case; but they do not prohibit doing so, nor are they a statement of a rule of law. The Court must, of course, read the literature with a critical eye, and apply sound methods of reasoning when drawing upon it. But what it may contribute to the picture in a given case, and how much probative weight can or should be attached to it, is also highly fact-sensitive to the issue at hand.

121. *Schembri v Marshall* [2020] EWCA Civ 358 provides an example of a case where breach of duty was admitted (in that case by a GP failing to make a hospital referral of a patient who subsequently died of a pulmonary embolism) but causation disputed. In that case the Judge properly had regard to his assessment of the picture he formed, from the literature, and the experts' evidence as to their experience, that it was very unusual for those promptly admitted to hospital, and then properly assessed and treated, to die. This meant that, though the claimant in that case had not shown the precise mechanism by which, had the deceased been admitted to hospital, she probably would have been rescued, the bigger picture pointed to the conclusion that, on a strong balance of probabilities, she would have survived.

122. The evidence as to what actually happened in the present case, as to the clinical experience of the experts, and as to the literature, all have something potentially to contribute. Nor do they sit in silos, as each may inform, support or undermine the other sources. I have to form an overall picture. In *Schembri* McCombe LJ (Holroyde and Phillips LJJ concurring) cited with approval at [44] the following pithy encapsulation from the learned editors of *Clerk & Lindsell on Torts* (22nd edition, 2018), particularly the last sentence:

> "On the other hand, care should be taken not to take the logic of this reasoning too far in the opposite direction. If the evidence is that, say, 80 per cent of patients survive with prompt treatment, but 20 per cent die even with prompt treatment, the fact that the patient died following delayed treatment does not establish that he probably fell into the 20 per cent category at the outset and therefore the delay did not contribute to the death. The assessment of causation would turn upon the detailed medical evidence, both as to the overall statistical chances of survival and the particular condition and circumstances of the patient."

Analysis

123. Every disease is different. Some, if untreated, are liable to be fatal. Others are not so serious. Different diseases typically take their course at different rates – over hours, days, weeks, months or years. They are more or less predictable in their aetiology.

They are more or less amenable to different kinds of treatment. It therefore helps at the start, to get one's general bearings by reflecting on what the experts, and the literature, tell me about the broad characteristics of *this* particular disease. Four broad propositions stand out.

124. The first is that untreated bacterial meningitis is deadly. The second is that, whilst rates of advance of course vary, this is a disease that, unchecked, advances fast. Its progress is measured in hours and days. The third is that, provided that they are administered soon enough, IV antibiotics are highly effective at treating the disease by a well-understood and proven mechanism. The fourth is that the aetiology and mechanisms of the disease mean that at a certain point in any given case it will be too late for the introduction of antibiotics to help.

125. It is this combination of features which explains why, though the disease is deadly, and fast, nevertheless survival rates are high. It also explains why the universal guidance is to begin IV antibiotics upon *suspicion* of bacterial meningitis on presentation at hospital, and without awaiting a definitive diagnosis to be confirmed by tests. This striking feature seems to me to be reflective of the recognition that the combination of the nature of the disease and the nature of the treatment does mean that hours can often matter.

126. Of course, clinical guidance as to how quickly antibiotics should begin may well err on the side of caution and generalisation. It does not by itself show that the first hour or few hours are critical in every case. How far does the literature assist? Retrospective cohort studies do not have the particular advantages of randomised large-scale double-blind controlled trials. But they do have the advantage of being based on actual patient data; and the critical reader can pay attention to how the cohort was selected and winnowed out, the quality of the data, what univariate and multivariate analysis has been conducted, the size of the odds ratios, and the confidence intervals and p-numbers attaching to them, and so on.

127. One of the themes of Mr Barnes' cross-examination, and submissions, about the literature, is that attention needs to be given to how closely the cohort studied (or the sub-group) resembles the case with which we are concerned, by reference to variables which have been shown to be significant, such as age, the primary source of infection, and the particular type of bacterium. That is a fair general proposition. But low numbers are not necessarily a problem. The paper should tell us how statistically significant the disaggregated results are (and whether they derive from univariate or multivariate analysis); and, provided they are recognised for what they are, the broader level figures may also still have utility for their contribution to the overall contextual picture.

128. Comfortably the largest group in the Køster-Rasmussen study were adults with pneumococcal meningitis. Antibiotic delay was at the top of the multi-variate analysis table. The odds ratios for delay overall and delay of up to 12 hours were both statistically significant and the latter (higher) figure is, I think, more pertinent to this case. As the authors explain, in the discussion section, their results both confirm *and build upon* earlier studies that found significant effects of delay at a late stage of the disease. They write, fairly, it seems to me, of their results:

> "In conclusion, this retrospective study adds to the known data of bacterial meningitis by specifying the incremental effect of treatment delay on clinical outcome in general, and not only after the severity of the disease has progressed."

129. Mr Barnes is right that the Bodilsen and Proulx studies do not provide *as strong* support for the Claimant's case as Køster-Rasmussen. But nor do they undermine or contradict it. The data are simply, for our purposes, not as rich or fine-grained with respect to the window of time with which I am particularly concerned. I think that Professor Lever was right to say that Køster-Rasmussen is, in that regard, the most useful for our purposes; and the other studies go with the grain of it.

130. Østergaard et al's results are also striking. They sought to identify clinical features and prognostic factors in patients with streptococcus meningitis according to the focus of infection. Of 187 cases in the cohort, in about 30% (57 patients) the focus was the ear (otogenic). Overall, 7% with an otogenic focus died during hospitalisation. The very high (and strongly significant) odds ratio for treatment post 3-hours reported by Auburtin et al also helps to fill out the canvas of the bigger general risk picture, relevantly to the present case, in which treatment began some 4 hours after admission. It was also not controversial among the experts that Mrs Davies' age, general health and lack of co-morbidities, put her in a low prior risk category.

131. It was the overall consensus view of the experts, and I find, that in this case the meningitic process began during the course of the evening or night of 24th/25th February, as signified in particular by the onset of the persistent vomiting and the intensification of the right-hand-sided headache. I also find that, because of the persistent vomiting, the oral antibiotic was not able to have any, or any appreciable effect.

132. There were mixed views from the experts as to whether there was raised ICP by the time of admission, something about which they were only able to give impressionistic or inferential views, because of the lack of any diagnostic measurement in this case. Looking at the overall picture I find that it probably was somewhat elevated by this time, but not yet critically. It was recognised, in particular by the microbiologists, that the *onset* of the meningitic process, and, in due course, the *onset* of the raised ICP associated with it, do not, in and of themselves, mark the passing of the point of no return.

133. While the experts, and I, did not have the benefit of any direct reading of ICP, I did have other relevant evidence to draw on, as to Mrs Davies' general clinical condition when she arrived at the hospital and when seen shortly after. The GCS score on admission is not, in my view, an irrelevant part of the picture. It is, as such, a well-regarded and established assessment tool. Plainly, though its very design aims to reduce the element of subjectivity, it has its limitations in terms of the nature of the test and the degree of precision of insight that it can give. But it forms a part of the overall picture of an individual's general clinical condition, that is of particular relevance in relation to this particular disease. Further, the correlation which Schutte et al found between GCS *at admission* and outcome was strong.

134. As to other indicators, Mrs Davies' temperature and pulse were both raised on arrival, but they did not point to the process being fatally advanced. Professor Masterton

observed that her blood count and CRP did not indicate that the infection was particularly severe on presentation. Professor Masterton also, in accord with Professor Lever to *this* extent, did not regard the way in which her disease had developed overnight following the GP visit, up to the point of admission, or even up until midday that day, to be particularly unusual. To the extent that Mr Crocker was of a different view about the significance of developments over the night before admission, he was an outlier among the experts; and, as I will describe, I was not persuaded by his opinion.

135. Mrs Davies was clearly already, by the time she arrived at hospital, in appalling pain which she literally put off the scale. She was not, at all times, clear and coherent. But she was still able to communicate with the ambulance staff, hospital staff and Dr Mukundu, though I have noted that the Claimant stepped in to help at a certain point in the latter consultation. Nevertheless, it seems clear, and not really in dispute, that by around 14.00 her condition in this regard was plainly much worse. The Claimant remarked on her having become pretty much incoherent, and her eyes having lost focus, around that time. The experts agreed that the observations at 14.30 reflected a profound decline, and, though giving somewhat different estimates, they broadly agreed that this had occurred at some point within the preceding hour.

136. Further, the overall picture I have from the experts is that IV antibiotics will probably arrive at the brain, and start to do their work, within a range of about half an hour, to an hour and a half after they are administered. Professor Lever was clearly of that view; and, on cross-examination, I do not think Professor Masterton materially disagreed. Mr Morrison's opinion did, in my view, lend weight here, drawing on his long and extensive clinical experience of antibiotics in action, and notwithstanding that he has not seen many cases matching the precise profile of that of Mrs Davies.

137. Further, even though, in a case of otitis media, the bacteria in the ear may take days, rather than hours, to fully sterilise, the consensus of those experts who opined on it, was that, once at the brain, the antibiotics present there will quickly neutralise any bacteria that continue to come in from the ear.

138. Pausing there, the *overall* picture that I have, of events as they unfolded, up to 10.40 and indeed later into that first morning, of Mrs Davies' background risk, and of the presentational and diagnostic indicators that we do have from that time, would, *by itself*, therefore, point to the overall conclusion that, had IV antibiotics been administered at 10.40, it would be more likely that she would then have survived than died.

139. But I have to decide causation on the basis of the *whole* of the evidence. As I have described, the Defendant's case is that later events show this case to be different from how it appeared at the point of admission. On the basis of the literature, her prior risk group, and GCS readings, Mrs Davies would have had the same good prospects at 13.20, as she had had at 10.40. Yet administration of intravenous antibiotics at that time did not save her. They say that that, and the rapidity of her decline in the next 24 hours, showed that it could not, after all, be assumed that antibiotics at 10.40 would probably have saved her.

140. I turn then, to those later events, and what I can take from the differing views that were given to me, of what we can infer from them about the overall nature of this

case, and how they add to, and colour or alter, our understanding of how matters probably stood by 10.40 am on the day of admission.

141. Professor Lever's answer to the conundrum posed by Mr Barnes – that the prior risk factors and GCS were the same at 1.20 as on admission – to the effect that this failed to take account of the fact that the disease would still have been progressing, and the wider clinical picture – seemed to me to be entirely coherent, and persuasive. My reasons for so saying are as follows.

142. First, the absence of prior risk factors (age, co-morbidities) was not going to change. Secondly, I accept the logic of Professor Lever's evidence, that the GCS assessment at different times needs to be viewed, not in isolation, but in the context of the overall clinical picture of how the situation was unfolding. Importantly, Professor Lever's take on this also fitted with his evidence that, as the meningitic process advances, deterioration is not linear. Rather, it reaches a crisis point at which there will be a sudden or marked decline. Also striking to me, was Professor Masterton's ready agreement with that depiction as characteristic, and his own use of the language of falling off a cliff. Mr Crocker also seemed to accept the image as apposite. It would follow (and I find) that the "good" GCS assessment at 13.30 probably masked the fact that at that point Mrs Davies was by then close to, or on the very edge of, the cliff. This, in my judgment is, essentially, the answer to Mr Barnes' critique of what he identified as the fourth plank of Professor Lever's case.

143. For reasons I have described, I also do not accept that Professor Lever's account of the literature was skewed or badly misleading. I have also been able to assess the literature myself. Different papers give more or less detailed or direct support to the Claimant's case, but none of them belie or undermine Professor Lever's conclusion; and it was not suggested that any other research out there does.

144. As I have described, Professor Lever's account of how quickly the bacteria may be expected to have an impact in the brain cavity, and of the significance (or not) of the continuing presence of bacteria in the middle ear, held up in cross-examination; and it was, ultimately, and as such, materially agreed by Professor Masterton. There was also consensus about the fact that at a certain point the impact of the ICP will feed itself as the vicious cycle takes hold. This was not something about which Professor Lever demurred. I see (and he himself accepted) the point that he could have given a more detailed account of the process in his report, but the same could be said of Professor Masterton; and the detailed account which Professor Lever did give in oral evidence, under close questioning from Mr Barnes, including his nuanced critique of the finer points of the diagram in Tariq et al, was impressive.[11]

145. Professor Lever, as I have noted, accepted in cross-examination that Mrs Davies' decline, after the collapse recorded at 14.30 on day one, was at the rapid end of the range, but not, in his clinical experience, exceptionally rapid. Regarding his assertion that rapidity of treatment is more important than the severity of the particular infection, certainly, had he identified the other source in the literature that he said he had in mind, that would have added weight to his evidence on this point.

[11] See note 1 above.

146. But I do not agree with Mr Barnes that I should, on that account, discount this evidence. It was not any part of the Defendant's case that Mrs Davies' meningitis was of a strain, or virulence, that was in some way intrinsically different, so that it was not amenable to the mechanisms of antibiotic treatment at all. Professor Masterton observed that pneumococcal infections are known to be more severe than other strains; but it was not suggested that the literature shows that antibiotics will not kill and inhibit the growth of these particular bacteria in the same way, provided always that they are caught soon enough. Nor, therefore, can I see that Professor Lever's observation, which also clearly drew on his clinical experience, is inherently illogical or implausible. There may be less time to catch a particularly aggressive infection, before the process has passed the point of no return, but as long as it *is* caught before that moment, the treatment should still be expected to work.

147. The personal remark which Mr Barnes highlighted in submissions was indeed out of place in the context of expert evidence. But I do not regard this brief expression of natural empathy, during the course of a long cross-examination, in the course of which this witness' responses were consistently measured and without any obvious side, as enough to make a real dent in the foundations of the overall credibility of Professor Lever's evidence.

148. Turning to the 2016 letters, Mr Barnes is of course right about the importance of the language in which experts express their assessments of chances; and any professional expert should be fully alive to this. But I note that Professor Lever was not at the time a particularly experienced expert witness, and even seasoned experts can occasionally stumble on the nuanced disparities between legal terminology, clinical terminology, and ordinary English language. More importantly, perhaps, the sense of this passage needs to be understood in the context of a letter specifically written as a response to Dr Gant and the pneumocephalus issue. The overall sense of the letter is that, presented with this particular point of challenge, Professor Lever disarms it by reference to the pertinent literature, and maintains his prior view; not that consideration of this feature has caused him to change that view.

149. Pausing there, and standing back, I am not, so far, persuaded that I should regard Professor Lever's evidence as unreliable, or lacking proper foundations. It appears to me to have been fairly rooted in his appraisal of the facts of this case, in light of both the literature and his own clinical experience. His reasoning as to why the way that events unfolded later on the day of admission, and into the next day, did not lead him to form a different view of Mrs Davies' prospects at 10.40 on day one, was not obviously flawed, or at odds with the literature; nor was his credibility seriously damaged in cross-examination or by Mr Barnes' critique of his opinions in submissions.

150. I turn, however, to review the other experts' views; and to consider whether a more compelling, and different, picture emerged, once these are added in to the overall picture on this aspect.

151. Professor Masterton, as I have described, initially considered that the pneumocephalus, which he had not seen before, also put this case into an unusual category. But he accepted in cross-examination that this feature was not causatively significant. His observation about Østergaard et al does not take away from the broad and striking contextual significance of their conclusions; and, as I have noted, he in

fact agreed with Professor Lever about Mrs Davies' general prior risk category on admission, and that the picture of how events unfolded prior to admission, and initially following it, was not particularly remarkable.

152. Professor Masterton relied on his clinical experience to support his conclusion that how this case unfolded thereafter was very unusual. But, simply as a factual observation, I do not think that the breadth of his relevant clinical experience matched that of Professor Lever. His recollection of the five cases he mentioned (only) in oral evidence was very limited. Further, his view on causation in his report was framed by his view on liability, leading him to use a reference frame of 10.30 to 12.00. He also referred to a timeframe of up to 4 hours for antibiotics to *fully* build up in the brain, but in cross-examination he agreed that they could be expected to show effects within a much shorter time frame. These features all served to limit the persuasive force of his opinion that the rapidity and severity of the later decline pointed to the conclusion that, by 10.40 on day one, it was too late.

153. Mr Morrison properly accepted that he could not match the microbiologists' expertise on causation; and that he had seen only a handful of cases specifically of adults with meningitis secondary to otitis media. Nevertheless, his wider clinical experience of the impact of intravenous antibiotics was plainly extensive, he drew appropriately and persuasively on the literature, and his defence of his opinion in cross-examination was tenacious and impressive. Mr Morrison's evidence provided support to that of Professor Lever, and a further counter-weight to the suggestion that the latter's lacked robustness.

154. Mr Hawthorne, as I have noted, more squarely deferred to other experts on causation; but he agreed with Mr Morrison that, following the onset of meningitis overnight on the 24th/25th February, the next significant decline was at 14.30 on the 25th.

155. I turn to the neurosurgeons. I will take first, Mr Norris.

156. In his opening skeleton, albeit in the context of the "material contribution" issue (of which, more later) Mr Barnes submitted that Mr Norris' evidence was flawed, given the journey that he had taken from certainty to uncertainty, without explanation. In closing submissions, however, Mr Barnes submitted that Mr Norris was to be credited for having reflected on, and revised, his view during the course of the process. Further, he submitted that Mr Norris' considered view did not support the Claimant. Mr Charles submitted that Mr Norris had modified his view, not fundamentally changed his opinion.

157. In my judgment, the significance of Mr Norris' change of view, and the import of his final, considered, view, need to be examined with some care. As Mr Norris himself observed in cross-examination, the underlying evidence had not altered. Rather, he had moderated his opinion, as, on further reflection, he was not so sure as before. I observe that, when he gave his initial view, he had not yet had the benefit of discussion with his fellow expert, Mr Crocker. He did not tell me that he specifically moderated his view in light of that discussion, but, if he did, that would be an example of the process operating as it should.

158. Be that as it may, Mr Norris' final view reflects a more cautious, and broadly-expressed, conclusion, that the balance tipped earlier than he at first thought, but also

that the best he could do, on consideration, was identify a period during which it tipped, rather than a precise moment when it probably did. True it is that he does not say that, on the balance of probabilities Mrs Davies would have survived if treated at 10.40. He only feels able to say that of the scenario of treatment before 10.10. In that sense, Mr Barnes was right to submit that his evidence does not, in the end, *support* the Claimant's case. But nor, I think, can it be said that Mr Norris' considered view goes *against* the Claimant's case in the same way as does that of Mr Crocker. Ultimately, on the question of whether treatment at 10.40 would probably have saved Mrs Davies, Mr Norris feels unable to express a view either way at all.

159. I turn to Mr Crocker. In his original report, he went so far as to say that, by the time that Mrs Davies started to develop a severe headache and vomiting, her death was probably inevitable. In the joint report, he uses a reference time of 10.00am, but his 14 October 2020 letter states that she arrived at hospital with a "fulminant infection" that it was already too late to treat. Further, as he explained in that letter, and oral evidence, 10.00am was not, in his view, the tipping point, which he could not precisely place; but it was a time by which, in effect, he felt comfortable saying that the tipping point had been passed.

160. Like Mr Norris, Mr Crocker has therefore opted for a window of uncertainty. But, unlike that of Mr Norris, his window closes clearly before 10.40, rather than straddling it. His view therefore not only does not support the Claimant, but, unlike that of Mr Norris, positively does support the Defendant. However, Mr Crocker fairly acknowledged that Mr Norris had far greater clinical experience than he. Further, Mr Crocker's explanation of what he meant by "fulminant" did not, with respect to him, as such, add to my understanding of the basis of his opinion as to *why* he put Mrs Davies' disease in that category. It was merely another way of stating his conclusion.

161. Having reviewed Mr Crocker's written and oral evidence it seems to me that his answer to that rested on three strands. These are: his view (in particular in his initial report) of the significance of the overnight pain and vomiting the night before; his judgment that the fulminant nature of the disease in this case could be inferred back from the rapidity and severity of the decline from around 13.30 – 14.30 and then over the next 24 hours; and his view (again in his initial report) that, in view of this being a case not of systemic sepsis, but of brain infection, other experts had overstated the rapidity with which IV antibiotics could be expected to start to have some impact.

162. However, as to the first of these, as I have noted, Mr Crocker was, it seems to me, alone among all the experts, in taking that view. Professor Masterton in particular shared Professor Lever's view that the way in which Mrs Davies' disease developed over the night prior to admission was not particularly out of the ordinary. Further, this phase of events was more squarely in the territory of the microbiologists than the neurosurgeons; and while Mr Crocker inferred (probably correctly, in my view) that Mrs Davies' ICP was elevated on arrival, he also agreed that early raised pressure was not, as such, a barrier to survival.

163. As to the second aspect, all of the experts agreed that Mrs Davies' decline from the point of collapse on the first day was (at least) at the rapid end of the scale, and that, she developed (at least) very significant signs of brain stem death over the next 24 hours. In that sense there was a consensus that her particular infection was particularly aggressive. But, as I have discussed, it was not said that this infection

was different in kind, in terms of its inherent amenability to antibiotic treatment, provided always that it was started soon enough; and I am not persuaded, reviewing the evidence over all, that this feature supports an inference that Mrs Davies' disease had probably already passed the point of no return before 10am on the day of admission.

164. As to the third aspect, as I have described, Professors Lever and Masterton agreed that IV antibiotics will take longer to have an impact on an infection within the brain lining than in the bloodstream itself, and, ultimately, as to the broad likely timescale for that impact to start to be felt. They also agreed that, once they arrive at the brain, they will take care of any bacteria still coming in from the ear. Mr Morrison provided support. I do not think, therefore, that these other experts did mistakenly fail to take fair account of this factor.

Causation on the Balance of Probabilities – Conclusion

165. The experts were asked to give views about a counterfactual, on the balance of probabilities, in relation to a disease process which unfolded over time, and on the basis of the limited clinical evidence available. In such a case, whilst intellectually fully appreciating the meaning of "balance of probabilities", an expert may balk at being asked to pick a precise time at which the balance tipped, and feel more comfortable postulating a period of uncertainty, only before, or after, which they feel able to express a view as to the likely position.

166. However, the task of the Court, in such a case, *is* to come to a view on the balance of probabilities, doing the best it can, and notwithstanding the limitations of the evidence. My conclusions, having regard to all the foregoing, and on the balance of probabilities, are in summary as follows.

(1) Mrs Davies had a pneumococcal meningitis which was aggressive and towards the severe end of the spectrum.

(2) However, it was still, in principle, amenable to effective treatment by IV antibiotics, if started early enough.

(3) The meningitic process had begun overnight on the night before she was admitted.

(4) The virulence of this infection meant that there was less time to start IV antibiotics, and hope to make a difference, in this case, than had it been less virulent, or had she arrived at hospital at an earlier stage.

(5) However, it had not, by 10.40 that day, reached what I have called the tipping point. It reached the tipping point at some time between 13.30 and 14.30 that day.

(6) By the time antibiotics were in fact administered, Mrs Davies was almost at the tipping point, which she reached some time in the next hour or so. That was not enough time for them to reach the brain and make a difference to the unfolding and self-reinforcing process. However, there would still have been enough time for antibiotics to reach the brain, ahead of the tipping point being reached, had they been administered at 10.40.

167. I therefore conclude that but-for causation is made out on the balance of probabilities; and this claim, for that reason, succeeds.

Material Contribution

168. Mr Charles submitted that, should I not conclude that administration of antibiotics at 10.40 would, on the balance of probabilities, have saved Mrs Davies' life, I should still find for the Claimant on the basis that the failure to administer timely antibiotics made a material contribution to her death. As I have found for the Claimant on ordinary principles, I am not obliged to consider this alternative contention; but as it was fully argued before me, and was a matter of significant doctrinal contention, I will address it.

169. The main *evidential* basis for such a finding is said to be Mr Norris' statement, in his letter of 9 June 2020, that "[b]etween [10.10 and 12.00], I consider that the outcome is uncertain and I am not therefore able to express a view on the balance of probabilities. However, I can say with confidence that during that time Mrs Davies deteriorated I consider that any delay between about 10:10 and 12:00 made a material contribution to her decline and death." That was said to found liability on the distinct *legal* basis that the negligence made a material contribution to the disease process that led to death.

The Authorities

170. As to whether there is a distinct legal doctrine that was potentially applicable in this case, a large number of authorities were cited to me. I shall not refer to those of the High Court, some of which were *obiter*, and, because I am in any event bound by the higher authorities. I shall also focus on those authorities which appear to me to add to the jurisprudence, rather than merely reiterating, or applying, what other authorities have said.

171. The catalyst for the doctrinal debate is *Bonnington Castings Limited v Wardlaw* [1956] AC 613, a case about pneumoconiosis caused by inhalation of silica dust. The employee had, during his time working for the employer, inhaled dust generated by two different pieces of machinery. The employer was not in breach of duty in relation to the exposure from one (pneumatic hammers), but was in relation to the other (swing grinders). Lord Reid said:

> "The medical evidence was that pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years. That means, I think, that the disease is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other."

172. On the facts found, although the precise proportions of dust from each source could not be quantified, it was held that it *could* be said that the dust from the culpable source made a material contribution to the employee contracting the disease. Put the other way, it could *not* be said that the "innocent" dust was substantially the sole cause. On that basis the employer was held to be liable.

173. In *Fairchild v Glenhaven Funeral Services Limited* [2003] 1 AC 32 the House of Lords considered the scenario of a claimant who was exposed to asbestos dust at more than one workplace and later developed mesothelioma, assuredly from one or other such exposure, but where medical science could not establish which. In an avowedly policy-driven decision it was held that the fact that exposure at a given workplace had increased the *risk* of getting the disease was sufficient to establish liability against that employer. In *Barker v Corus (UK) Plc* [2006] 2 AC 572 it was, however, held that, in such case, a given tortfeasor would only bear liability proportionate to the contribution that its negligence had made to that risk; but that decision was then reversed by Parliament, in respect of mesothelioma, in the Compensation Act 2006.

174. Meantime, in *Gregg v Scott* [2005] 2 AC 176 a negligent failure to diagnose cancer led to a significant delay in the start of treatment. As a result the claimant's prospects of surviving 10 years were reckoned by the Judge to have fallen from 42% to 25%. By a majority, the House of Lords upheld the dismissal of his claim, declining to find that the reduction in chance of a favourable outcome was a recoverable head of damage. The majority declined to widen the application of the *Fairchild* principle to such a case.

175. In *Bailey v Ministry of Defence* [2009] 1 WLR 1052 the claimant, in a weakened state, aspirated vomit, causing her to suffer a cardiac arrest which, in turn, caused brain damage. The question was whether the weakened state had been caused by the first defendant's negligent care, earlier in the month, and/or by a condition of pancreatitis. The Judge was not able to say that, absent the negligent care, the injury would not have occurred; but he did consider that both the negligent care and the pancreatitis made a material contribution to the overall weakened state, which in turn caused the aspiration; and that was sufficient in law to establish causation.

176. Waller LJ (Sedley and Smith LJJ concurring) said (at [39]):

> "It is important to be clear precisely what *Wardlaw* decided. Did it decide that in a cumulative cause case where the inadequacies of medical science meant the relative potency could not be established all a claimant had to establish was a "material" contribution which in the words of Lord Reid meant something more than de minimis? Or did a claimant still have to establish that 'but for' the contribution of the negligent cause, the injury would not have occurred?"

177. After reviewing the speeches in *Wardlaw*, and *dicta* in other authorities, his answer was that Lord Reid's "ultimate conclusion" showed that he was applying the "anything greater than *de minimis* test." He continued, at [43]:

> "It seems to me thus respectfully that Lord Rodger in *Fairchild* accurately summarises the position when he says in paragraph 129 that in the cumulative cause case such as *Wardlaw* the 'but for' test is modified."

178. Then, at [46], Waller LJ said this:

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

179. In *AB v Ministry of Defence* [2010] EWCA Civ 1317 the claimants had been exposed to radiation in nuclear tests in the 1950s. They complained of conditions, of which such exposure was only one of the possible causes. At [134] the Court set out the following submission from the MoD.

> "The decision of the House of Lords in *Bonnington* amounted to a modification of the 'but for' rule of causation because the plaintiff recovered damages for the harm caused by all the dust, not just the tortious component. At no stage in that case was it suggested that the damages should be apportioned as between the effect of the tortious and non-tortious components. If that had been suggested, and if expert evidence had been called showing the effect of the different components (as we think it would be nowadays), the damages would probably have been apportioned. The plaintiff would have recovered damages for only the harm caused by the tort and there would have been no need for any modification of the 'but for' rule. This type of modification of the 'but for' rule is still available where the negligent and non-negligent causative components have both contributed to the disease (as opposed to the risk of the disease) and it is not possible to apportion the harm caused and therefore the damages. This method of proving causation (by showing that the tort made a material contribution to the condition or disease) is only available where the severity of the disease is related to the amount of exposure; further exposure to the noxious substance in question is capable of making the condition worse. Thus the MoD's submission is that, in the present cases, at least so far as the cancers were concerned, that could not be said. The cancers either developed or they did not. Their severity did not depend on the extent of the exposure. It could not be said that the exposure to radiation had made a material contribution to the disease, only to the risk that it might occur.

180. Further on, the Court concluded:

> "149. We accept the submissions of the MoD. First, unless there were to be an extension of the *Fairchild* exception, the claimants will have to show 'but for' causation: see *Wilsher*.
>
> 150. Second, we accept that, at least so far as cancers are concerned, the claimants cannot rely on proving that the radiation exposure has made a material contribution to the disease, as in *Bailey* and *Bonnington Castings.* This principle applies only where the disease or condition is 'divisible' so that an increased dose of the harmful agent worsens the disease. As is well known, in *Bonnington*, the claim succeeded because the tortious exposure to silica dust had materially aggravated (to an unknown degree) the pneumoconiosis which the claimant might well have developed in any event as the result of non-tortious exposure to the same type of dust. The tort did not increase the risk of harm; it increased the actual harm. Similarly in *Bailey*, the tort (a failure of medical care) increased the claimant's physical weakness. She would have been quite weak in any event as the result of a condition she had developed naturally. No one could say how great a contribution each had made to the overall weakness save that each was material. It was the overall weakness which led to the claimant's failure to protect her airway when she vomited with the result that she inhaled her vomit and suffered a cardiac arrest and brain damage. In those cases, the pneumoconiosis and the weakness were divisible conditions. Cancer is an indivisible condition; one either gets it or one does not. The condition is not worse because one has been exposed to a greater or smaller amount of the causative agent."

181. The Court also said:

> "152. Finally, these are not cases to which the *Fairchild* exception could foreseeably be made to apply. The House of Lords in that case and in *Barker* has made it plain that the scope of the exception will be very narrow. It is clear that the exception will only apply where the two or more potential causes act either through the same agent (eg asbestos dust in *Fairchild* or brick dust in *McGhee v National Coal Board* [1973] 1 WLR 10) or possibly through different agents which act on the body in the same way."

182. In *Heneghan v Manchester Dry Docks Limited* [2016] ICR 671 the deceased died of lung cancer caused by exposure to asbestos fibres. He had been exposed to such fibres when working for six different employers. The trial Judge held that *Fairchild* applied, and apportioned damages among the defendants[12], a decision upheld by the

[12] It appears that, as this was an application of *Fairchild* to a non-mesothelioma case, the Compensation Act 2006 did not apply, so the approach to compensation in *Barker v Corus (UK) plc* applied.

Court of Appeal. The Claimant argued that the Judge should have found that each Defendant materially contributed to the cancer, and that each was liable for the full amount of the loss. The Court of Appeal rejected that. At [27], the Master of the Rolls (with whose reasons Tomlinson and Sales LJJ agreed) observed of *Bonnington Castings*:

> "It was not necessary to rely on statistical evidence in that case to demonstrate that dust emanating from the swing grinders contributed to the disease. It contributed to the disease because its severity was proportionate to the amount of dust inhaled and the amount attributable to the swing grinders was material."

183. He also said of the test in that case, at [46]:

> "That test is to be applied where the court is satisfied on scientific evidence that the exposure for which the defendant is responsible has in fact contributed to the *injury*. This is readily demonstrated in the case of divisible injuries (such as silicosis and pneumoconiosis) whose severity is proportionate to the amount of exposure to the causative agent."

184. *Sinkiewicz v Grief (UK) Limited* [2011] 2 AC 229 is a decision of the Supreme Court chiefly concerned with issues peculiar to mesothelioma and the *Fairchild* principle, which are not, as such, relevant to the case before me. However, Lord Phillips, at [12] to [15], divided diseases into three types. The first has a single trigger, and is indivisible. The second is triggered by exposure to a noxious agent surpassing some minimum threshold; but, once it is surpassed, the disease caused is indivisible, and not affected by the degree of exposure. In the third, the degree of exposure affects both the onset of the disease *and* its severity. The harm is divisible.

185. *Bonnington Castings*, said Lord Phillips at [17], involved an important exception to the but-for test. It was a case of pneumoconiosis, which is a divisible disease. The employer had been held liable for 100% of the harm. But later cases had recognised that, where a number of exposures cumulatively caused a divisible disease, responsibility should be apportioned so that a given defendant was liable for "no more than his share".

186. At [90] Lord Phillips described "the rule in *Bonnington*" as follows:

> "Where the disease is indivisible, such as lung cancer, a defendant who has tortiously contributed to the cause of the disease will be liable in full. Where the disease is divisible, such as asbestosis, the tortfeasor will be liable in respect of the share of the disease for which he is responsible."

187. Lord Brown, after commenting on the radically different approach taken by the law to mesothelioma, continued, at [176]:

> "All other cases require that the claimant satisfies the "but for" test of causation. True, in the case of cumulative injuries, the law holds a negligent employer liable even if his negligence is

> responsible for part only of the victim's condition (provided only that it made a material, ie more than de minimis, contribution to the development of the condition). I have difficulty, however, in seeing this as a true exception to the "but for" test: although the claimant in *Bonnington Castings Ltd v Wardlaw* [1956] AC 613, the case which first established the principle, recovered full damages for his condition (pneumoconiosis from the inhalation of silica), that appears to have been because the defendants took no point on apportionment; in a series of subsequent such cases damages have been apportioned, however broadly..."

188. In *Williams v The Bermuda Hospitals Board* [2016] AC 888 there was a delay in treating appendicitis followed by sepsis and other complications. The Privy Council agreed with the Court of Appeal that, on the facts found, the trial Judge should have held that causation was established.

189. Reviewing the authorities, the Board, speaking through Lord Toulson, analysed the reasoning in *Bonnington Castings*, highlighting that it was treated at the time, as a case of indivisible injury, and stating, at [32]:

> "In *Bonnington* there was no suggestion that the pneumoconiosis was "divisible", meaning that the severity of the disease depended on the quantity of dust inhaled. Lord Reid interpreted the medical evidence as meaning that the particles from the swing grinders were a cause of the entire disease. True, they were only part of the cause, but they were a partial cause of the entire injury, as distinct from being a cause of only part of the injury. Lord Reid's approach was understandable in view of the way in which the case was argued."

190. In a footnote the Board noted that, in later cases it had been accepted that pneumoconiosis is a divisible disease, as its severity is affected by the amount of dust inhaled, so that, where there is more than one source, each defendant's liability should reflect the degree of injury caused by the exposure for which it was responsible. It cited from Lord Phillips in *Sinkiewicz* at [90].

Argument

191. It was – rightly – not suggested that *Fairchild* could have any application to the present case. However, Mr Charles submitted that there is a distinct doctrine of material contribution which *can* apply to a case of indivisible injury, arising from a disease process. Properly understood, *Bailey* was such a case. This possibility was confirmed, he said, by *Sinkiewicz*, and, in particular, by *Williams*. Even if the discussion in *Sinkiewicz* was narrower, and/or *obiter*, this was the import of *Williams*, by which I was bound.

192. Mr Charles also cited a number of High Court decisions which he said were instances of the application of the doctrine to indivisible injuries (although in some the discussion was *obiter*), as well as another Court of Appeal decision, *Popple v Birmingham Women's NHS Foundation Trust* [2012] EWCA Civ 1628. He also cited

a Court of Session decision, *Andrews v Greater Glasgow Health Board* [2019] CSOH 31, which followed, and applied, *Williams*.

193. Mr Charles also referred me to *Dickens v O2* [2008] EWCA Civ 1144 and *BAe Systems (Operations) Limited v Konczak* [2018] ICR 1. *Dickens* concerned a claim for psychiatric injury said to have been negligently caused by stress at work. Smith LJ (who was also one of the panel in *Bailey*) analysed the instant case as being, like *Bailey*, a case of indivisible injury with more than one cause, and inclined to the view that, in such a case, it was not appropriate to apportion damages. Sedley LJ, who also sat in *Bailey*, concurred.

194. *Konczak* concerned a claim for psychiatric injury said to have been caused by discrimination. The discussion of the general legal principles includes the following propositions: (1) that where the harm has more than one cause an employer should only be liable for the harm attributable to his own wrongdoing unless the harm is truly indivisible; (2) that an injury is to be regarded as indivisible where there is simply no rational basis for an objective apportionment of causal responsibility for it; and (3) that where two wrongdoers cause a single indivisible injury, then they are jointly and severally wholly liable for it. Mr Charles submitted that *Konczak* provided "a very useful summary of material contribution and indivisible injury."

195. Cases such as *AB* and *Heneghan* were industrial disease claims, where much of the argument was irrelevant to present concerns; and, in relation to material contribution, they failed on their facts, rather than being decisions of principle. The doctrine could therefore properly be applied in those cases involving a disease process causing indivisible harm, such as meningitis, in which it was not possible medically to determine whether, at the moment when the negligence occurred, the process had passed a critical phase. Were I to conclude, in this case, that medical science could not resolve whether there was but-for causation on the balance of probabilities, it would be open to me to find that the negligence had at least made a material contribution.

196. Mr Barnes submitted that the line of *Bonnington Castings*, *Bailey*, *AB* and *Heneghan* clearly establishes that the "but for" test may be modified by the application of a "material contribution" test, but only in cases where (a) but for causation on the balance of probabilities cannot be determined, and (b) the injury is divisible. He submitted that any suggestion, in *Sinkiewicz* and *Williams,* that it might apply also to indivisible injuries was, in both cases, *obiter*. *Sinkiewicz* was about mesothelioma. Lord Phillips' analysis was not binding, and, respectfully, wrong. Lord Brown's analysis was consistent with the line of Court of Appeal authority. *Williams* was concerned with the narrower issue of whether the reasoning in *Bonnington Castings* was limited to cases where the process attributable to negligence developed, in point of time, after the process that was not attributable to negligence. He also submitted that, on examination, *Williams* was in fact a case of divisible injury.

197. A number of the High Court cases relied upon by Mr Charles, as well as *Popple*, were also actually cases of divisible injury. The discussion of material contribution in *Andrews* was *obiter*, and, respectfully, wrong, and inconsistent with the Court of Appeal's reasoning in *AB*. The discussion in both *Dickins* and *Konczak* was *obiter*. In any event, the discussion in the latter was by way of a corrective to the former's conclusion that psychiatric injury was indivisible, preferring the view, drawing from

the well of *Sutherland v Hatton* [2002] ICR 263, that psychiatric injury may well be divisible.

198. The present case, submitted Mr Barnes, was one where the Court could (and should) determine causation only on ordinary but-for principles; and in which the injury – death caused by meningitis – was indivisible. For both reasons, the material contribution test could have no application. Alternatively, and in any event, submitted Mr Barnes, even if material contribution could apply, in law, to a case of indivisible injury, that could not be established factually in this case. All that the Claimant's experts could say was that the impact of the delay was that it increased Mrs Davies' *risk* of death.

Analysis of the authorities

199. Skilfully though the matter was argued, I cannot fully subscribe to the whole analysis of the authorities advanced by either Mr Charles or Mr Barnes. I have found it most helpful to come at the task in a slightly different way.

200. I start with what is, I believe, clear. First, where the harm is divisible, a party will be liable if their culpable conduct made a contribution to the harm, to the extent of that contribution. Secondly, where the harm is indivisible, a party will be liable for the whole of it, if they caused it, applying "but for" principles. Thirdly, if two wrongdoers have both together caused an indivisible injury, in respect of which it is impossible to apportion liability between them, then each is co-liable for the whole of the injury suffered. This approach may, at least in some sub-permutations, be seen as a modification of the "but for" test, but it is doctrinally well-established and accepted: *Konczak* points to the fuller discussion in *Rahman v Arearose Limited* [2001] QB 351. I call these the orthodox routes to liability.

201. *Fairchild* provides a further distinct route to liability, in the limited types of case to which it applies, based on contribution to risk, but leading to liability for the actual harm. Where it applies to a mesothelioma case, the effect of the 2006 Act is that each contributor to the risk is co-liable to the claimant for the whole of the harm. Otherwise, as in *Heneghan*, it is apportioned.

202. It seems to me that what the authorities since *Bonnington Castings* have wrestled with, is whether that decision establishes the existence, outside of *Fairchild* cases, of an additional route to liability, for either the whole or part of the harm suffered, that may be available where none of the routes I have referred to at [200] above applies, and which is conceptually distinct from all of them. One of the hazards when seeking to interpret the discussion in the later authorities, is that labels like "material contribution" and "cumulative cause case" are not always used in the same sense. Their use does not necessarily signify that the Court is contemplating that some such additional route to liability may be, or does, apply. Sometimes they are used merely to signify that a case is, or may be, of the first or third orthodox types, or, potentially, one or the other. Sometimes what is meant is less than clear.

203. In *Bailey* the Court of Appeal concluded that *Bonnington Castings* did point to the existence of a novel route by which a party could be held liable for the *whole* of the harm caused; but *AB* held that both *Bonnington Castings* and *Bailey* were conventional cases of divisible harm, and established no new principle. In

Bonnington Castings, accordingly, damages *ought* to have been apportioned, as best the Court could. The Court in *AB* was using "material contribution", I think, simply to refer to a divisible harm type of case. It declined to extend *Fairchild* or otherwise to create a new form of liability.

204. *Heneghan* takes the same view of *Bonnington Castings*. The fact that the claimant in that case hoped, by relying on its *outcome,* to shelter under it to secure an award of the full amount of the loss, should not distract from that.

205. In *Sinkiewicz*, while Lord Phillips at [17] described *Bonnington Castings* as involving an exception to the but-for test, that appears to have been, indeed, because it imposed 100% liability in respect of a divisible-harm disease. He went on to say, in effect, that liability *should* have been apportioned, and his statement of the "rule" in that case, at [100] is an articulation of conventional principles. Lord Brown's approach to *Bonnington Castings* is that it does not create a true exception, but is a case in which the whole loss was awarded only because apportionment was not sought. While the discussion is, I think, indeed *obiter*, this approach is in line with that taken in *AB*.

206. In *Williams* Lord Toulson considered that the outcome in *Bonnington Castings* was explained by the fact that it was a case in which the harm was *viewed* (at the time) as indivisible – in my typology, the third type of case[13] – though today it would not be viewed that way. He did not see it as establishing any new legal principle, and endorsed, in the footnote, Lord Phillips' approach: that is, that today it would be viewed in what, in my typology, is a type one case.

207. I do not think the discussion of general principles in *Williams* is *obiter*. It underpinned its analysis of the particular question of whether the temporal order of causes is significant or not. This is, I think, clear from the discussion at [38] – [42]. I will not further prolong this long decision by setting it out; but I note that it effectively concluded that sepsis was a process, but one which caused indivisible harm. He went on, at [47], to say the following of *Bailey*:

> "The Board does not share the view of the Court of Appeal that the case involved a departure from the "but-for" test. The judge concluded that the totality of the claimant's weakened condition caused the harm. If so, "but-for" causation was established. The fact that her vulnerability was heightened by her pancreatitis no more assisted the hospital's case than if she had an egg-shell skull."

208. Although this was offered as a "postscript" which was "not strictly necessary", this way of analysing *Bailey* is, it seems to me, entirely consistent with the way in which the Board rationalised *Bonnington Castings*, and the conclusion that it does not stand for any novel legal principle.

209. I conclude that, while *Bonnington Castings* was viewed in *Bailey* as establishing a novel principle, later authorities of the Court of Appeal, House of Lords and Privy Council view it as having resulted in an anomalous outcome, for peculiar reasons, and not as standing for any novel legal principle, distinct from the general jurisprudence

[13] Though with one party being the source of both causes.

on co-contribution to divisible or indivisible harms. This conclusion appears to me to accord with deep principle, and with the prevailing view at the highest level, ever since *Fairchild*, that it stands alone as an exception to orthodox principles, in a tightly circumscribed type of case. In any event, I am bound to follow what I understand to be the principles emerging from those authorities.

210. In the present case Mrs Davies died from a disease which, whilst it involved a process that took its course over a period of time, led to the indivisible outcome of death. The sole task of the Court has been to determine on the balance of probabilities whether, in a but for sense, the failure to start IV antibiotics by 10.40 on the day of admission caused her death or not.

211. As I have said, while I fully appreciate that some of the experts felt ultimately unable, on the clinical evidence available in this very difficult case, to answer that counterfactual question in quite that way, the Court is obliged, on the evidence it has, including such assistance as the experts feel able to provide, to do so, as best it can. That I have done. For the reasons I have given, I do not think that any other legal doctrine could have been brought to bear in this case.

Outcome

212. For the foregoing reasons, I will give judgment for the Claimant, in appropriate terms.