Hilary Term

[2016] UKPC 4

Privy Council Appeal No 0110 of 2014

JUDGMENT

Williams (Respondent) vThe Bermuda Hospitals Board (Appellant) (Bermuda)

From the Court of Appeal of Bermuda

before

Lady Hale

Lord Clarke

Lord Hughes

Lord Toulson

Lord Hodge

JUDGMENT GIVEN ON

25 January 2016

Heard on 18 and 19 November 2015

Appellant	Respondent
Caroline Harrison QC	Benjamin Browne QC
Andrew Bershadski	Luka Krsljanin
(Instructed by Bevan Brittan LLP)	(Instructed by Wakefield Quin Limited)

Intervener (
Litigation Aut
(Writte
submissions
Philip Have
Jeremy Hy
(Instructe
Bevan Britta

LORD TOULSON:

1.

On 30 May 2011 the respondent, Kamal Williams, went to the emergency department of the King Edward VII Memorial Hospital in Bermuda, complaining of abdominal pain. He was suffering from acute appendicitis. Later that day he had an appendectomy, but there were complications. He was seriously unwell for a period of weeks, but he finally made a full recovery. He sued the appellant hospital board, which is responsible for the management of the hospital, for damages for his pain and suffering, medical expenses and loss of earnings. He alleged that the complications were the result of negligent delay in his treatment.

2.
The trial judge, Hellman J, found that there had been negligence but that Mr Williams had not proved that the culpable delay caused the complications. He awarded Mr Williams $2,000 in damages for his extra suffering during the period of culpable delay prior to the operation. The Court of Appeal (Evans and Ward JJA and Bell AJA) reversed the judge's decision on causation and remitted the case to the trial judge for a fresh assessment of damages. Hellman J increased the award to $60,000 excluding interest.

3.
The hospital board asks that the original judgment be restored. It submits that the trial judge decided the case on orthodox legal principles and that the Court of Appeal was wrong to interfere with his judgment. It has paid the amount of the increased award to Mr Williams (who has donated it to charity), and has stated through counsel that it will not seek repayment if it succeeds in the present appeal, but it is pursuing the appeal because of its concern about the effect of the Court of Appeal's judgment as a precedent.

Facts

4.
At the trial, the evidence of all but one of the factual witnesses was contained in statements and exhibits which were admitted without cross-examination. The exception was Dr Christine Di Lullo (one of the doctors involved in Mr Williams' treatment), who was called at the suggestion of the judge to give oral evidence to clarify a point in her witness statement.

5.
The judge heard oral expert evidence from Dr Michael Leitman and Dr Alasdair Conn. Dr Leitman was head of the general surgery division at a New York hospital, and was called on behalf of Mr Williams. Dr Conn was head of emergency services at a hospital in Boston, Massachusetts, and was called on behalf of the hospital board. There was also a written report tendered on behalf of the hospital board from Dr Randall Zusman, a cardiologist at the same hospital as Dr Conn and an associate professor at Harvard Medical School.

6.
Mr Williams' time of arrival at the hospital emergency department was 11.17 am. At 11.40 he was examined by Dr Chikezie Dean Okereke, a physician specialising in emergency medicine. Dr Okereke was limited in the examination which he was able to make because of the pain which Mr Williams was suffering and Dr Okereke prescribed an analgesic. After about 30 minutes Dr Okereke was able to conduct a fuller examination. He decided to order a CT scan of the abdomen to help to determine whether Mr Williams was suffering from appendicitis or some other condition. It was common ground that this was an appropriate decision. Dr Okereke said in his witness statement that he ordered the scan approximately one hour after Mr Williams' arrival in the emergency room, ie at 12.15 or thereabouts, and that would coincide with the time of his second examination. That evidence was not

challenged, and Dr Leitman said that an hour from admission was a reasonable time frame for ordering the scan.

7.
The judge found that Dr Okereke probably ordered the scan at 13.10 (which was the time that the imaging department received a faxed request, according to a later internal investigation by the hospital board), and his finding has not been disputed on appeal. The scan was performed at 17.27. There was therefore a delay of over five hours from the time when Dr Okereke on his evidence decided to request a scan until it was performed, and over four hours from the time when the judge found that the request for a scan was sent to the imaging department until it was performed. In the interim Dr Okereke's shift ended and he handed over care of Mr Williams to Dr Di Lullo.

8.
Dr Di Lullo did not receive a report on the scan until around 19.30, ie two hours after it had been performed. Part of the reason for this delay was that the hospital's imaging department closed at 18.00. Because Mr Williams' scan (performed 30 minutes earlier) had not by then been interpreted, it was sent in accordance with the hospital's usual practice to an overseas reporting agency for interpretation and report.

9.
Dr Di Lullo, rightly, discussed the interpretation of the scan and the appropriate treatment with a surgeon, Dr Council Miller. They re-examined Mr Williams and advised him that there was a strong likelihood that he had acute appendicitis requiring surgery. Mr Williams gave the necessary consent and he was taken to the operating theatre at around 21.30.

10.
The findings at operation included ruptured appendix and widespread pus throughout the pelvic region, with phlegmon (a purulent inflammation) around the liver and cecum (a pouch from which the appendix hangs). Dr Miller said that from his observation of the phlegmon he formed the opinion that the pus had been there for some time. The accumulation of pus resulting from the ruptured appendix led in turn to myocardial ischaemia. (Dr Zusman explained in his report that sepsis is known to increase cardiac oxygen requirements and potentially to precipitate myocardial ischaemia.) During surgery Mr Williams' blood pressure became precariously low. He suffered what the experts agreed was some form of myocardial ischaemic event and lung complications, requiring life support in the intensive care unit. It is unnecessary for present purposes to go into further details of the complications. The judge found in summary that "Sepsis from the ruptured appendix caused injury to his heart and lungs".

The trial judge's findings on negligence and causation

11.
At the relevant time the hospital did not have a policy about when a request for a CT scan should be for a "STAT" CT scan (derived from the Latin word "statim" meaning immediate). The judge found a CT scan requested in a case of suspected appendicitis should as a matter of normal practice be obtained on a STAT basis, and that in this regard the management of Mr Williams' case fell below the proper standard. If Mr Williams' scan had been obtained on a STAT basis, it was probable that his condition would have been diagnosed and he would have been treated more rapidly.

12.

Dr Leitman and Dr Conn disagreed about what would have been an acceptable time frame and what, if any, difference in outcome would have been likely. One difference was over when the process of rupture began. It is right to emphasise the word “process” because the judge accepted that the rupture of the appendix would not have been an instantaneous event but a gradual process. The judge accepted Dr Leitman’s opinion that the earliest evidence of the process beginning was at 15.19, when Mr Williams developed an abnormally rapid pulse. (This did not mean that perforation had occurred at that moment.) The judge rejected Dr Conn’s opinion that the presence of pus and phlegmon in the abdominal cavity at the time of the operation was indicative that the appendix had begun to rupture prior to 15.19. (Dr Conn had suggested that the rupture had begun before Mr Williams arrived at the hospital.)

13.
The judge also accepted Dr Leitman’s evidence that he had seen patients who had presented in a similar way to Mr Williams and had developed a substantial amount of pus in a matter of hours. Dr Leitman’s evidence was that 3.5 hours’ delay before the operation was performed was sufficient time for the pus to form, but that it would generally take four to five hours, or longer, to have the amount of infection reported by Dr Miller. Dr Leitman also said that the extent of infection was eliminated or greatly reduced the sooner surgery takes place.

14.
Dr Leitman gave a time frame for proper treatment, taking as his starting point Dr Okereke’s evidence that he ordered a CT scan around 12.15. From that time Dr Leitman would have allowed an hour to an hour and 15 minutes for Mr Williams to be given an oral contrast fluid prior to performance of the scan, and 15 to 30 minutes for interpretation and communication of the result. He would have allowed another hour for consultation with the surgeon and preparation for surgery. On this time frame, the operation would have begun sometime around 15.00 to 15.15, ie about four hours after Mr Williams’ arrival at the emergency department. There was no evidence about the level of staffing on the day in question.

15.
Dr Conn did not put forward an itemised rival time frame, but he was of the opinion that the overall time table suggested by Dr Leitman was much too short.

16.
The judge considered that Dr Leitman’s four-hour time frame (which he mistakenly took as four hours from the ordering of the scan) may have been achievable but was optimistic. Allowing for “known unknowns”, such as whether there might have been a longer wait for the operating theatre if the scan had been carried out and interpreted earlier, he found that had the scan been ordered on a STAT basis Mr Williams would probably have been operated on within the next five to six hours. This meant that the operation would probably have begun sometime between 17.15 (if there was a five-hour interval and the scan was ordered at 12.15) and 19.10 (if there was a six-hour interval and, as he considered more likely, the scan was ordered at 13.10). The failure to order the scan on a STAT basis therefore led to a delay of between four hours 15 minutes and two hours 20 minutes in the start of the operation.

17.
The judge concluded:

“In the circumstances I find that the plaintiff has failed to prove that the complications that Mr Williams developed during and after surgery were probably caused by the [hospital board’s] failure to

diagnose and treat him expeditiously. Had the CT scan been obtained and interpreted promptly these complications might have been avoided, but I am not satisfied that they probably would have been avoided."

The Court of Appeal's judgment

18.
The judgment of the Court of Appeal was given by Ward JA. After summarising the history, he posed the question whether the hospital board omitted to do anything which it ought to have done and as a result of which the patient suffered damage. He stated the answer to be "with resounding clarity that it did", and that the "numerous delays" were contributing factors to the damage ultimately suffered. He referred in general terms to "delays between arrival, admission, examination, the ordering, taking and reading of the CT scan and the surgery", but did not itemise the period or periods of culpable delay. He was critical of the judge's reference to other possible delaying factors, such as a longer wait for an operating theatre if the scan result had been obtained promptly, describing this as "unwarranted speculation".

19.
He held that the trial judge was in error "by raising the bar unattainably high" in his finding that Mr Williams had failed to prove his case. The proper test of causation, he said, was "not whether the negligent delay and inadequate system caused the injury to [Mr Williams] but rather whether the breaches of duty by [the hospital board] contributed materially to the injury". That the breaches contributed materially to the injury was in his view beyond argument. He stated that the "but for" test is sometimes relaxed to enable a claimant to overcome the causation hurdle when it might otherwise seem unjust to require the claimant to prove the impossible, and he referred to Bailey v Ministry of Defence[2009] 1 WLR 1052 as a case in which the "but for" rule was modified.

20.
Ward JA described as instructive statements made by Lord Hoffmann in Gregg v Scott[2015] 2 AC 176 that for loss to be recoverable it must be shown that the damage in question was attributable to the defendant's wrongful act and that there must be a "sufficient" causal link between the defendant's conduct and the claimant's injury. He added that the question of causation is "no longer a question of all or nothing but one of sufficiency". He concluded that in the present case "causal or causative links between the inordinate delays coupled with the defective system which together contributed to [Mr Williams'] injury were clearly established".

Submissions

21.
The Board has had the advantage of well presented arguments on both sides. On behalf of the hospital board, Ms Caroline Harrison QC submitted that the trial judge applied orthodox rules of causation and made findings of fact which were properly open to him. He was right, in her submission, to conclude that on the balance of probabilities Mr Williams could prove only that the delay in surgery caused him two hours of avoidable pain. She argued that the Court of Appeal was led into error by a misinterpretation of "material contribution" as sufficient for the purposes of causation, perhaps induced by the decision of the Court of Appeal of England and Wales in Bailey.

22.
Ms Harrison submitted that the well known decision of the House of Lords in Bonnington Castings Ltd v Wardlaw[1956] AC 613 on material contribution as sufficient for causation did not assist Mr

Williams. At its broadest, Bonnington was authority that a claimant may recover damages for personal injury where he can show that there was a single causative agent; the defendant contributed to the pathological process in a way that was material (ie could not be disregarded as insignificant); the defendant's contribution to the pathological process was concurrent with any non-negligent cause; and as a matter of probability the defendant's contribution increased the magnitude (and not merely the risk) of the harm which the claimant suffered. In the present case sepsis had developed by 19.10 (and indeed by 17.15), and Mr Williams could not prove as a matter of probability that the complications during and after surgery would not have occurred but for the ensuing delay in the performance of the operation.

23.
Ms Harrison also criticised the Court of Appeal for apparently substituting factual findings of its own for the facts found by the judge and for a lack of clarity and proper analysis in doing so. Ward JA referred to delays between arrival and admission, and between admission and examination, but no such criticism had been made by Mr Williams or his expert, Dr Leitman. Similarly, Ward JA referred to delay in ordering the scan, but Dr Leitman had said that it was ordered within a reasonable time. (It is right, however, to note that Dr Leitman gave that evidence on the basis that it was ordered when Dr Okereke said that he ordered it, which was an hour before the time when the judge found that it was ordered.) Since the Court of Appeal did not identify the time at which they considered that surgery should have begun, Ms Harrison submitted that its conclusions about causation were fatally flawed.

24.
On behalf of Mr Williams, Mr Ben Browne QC argued the case on two lines. As to the facts, he submitted that although the Court of Appeal did not specify a time at which surgery ought to have been started, it appeared to have concluded that the overall time allowed by the judge was too long and it was entitled to do so. In particular, no explanation was advanced for the lapse of almost an hour between the time when Dr Okereke said that he ordered a scan (ie around the time of his second examination of Mr Williams at about 12.15) and the time when it was ordered. Ward JA therefore had legitimate cause to speak of delay in ordering the scan. He was also entitled to criticise the judge for apparently making some allowance for the possibility that there may have been difficulties, of which there was no evidence, in obtaining a theatre if the scan had been carried out sooner. Removing speculative factors, Mr Browne submitted that surgery ought to have commenced by 17.15 or at the latest by 18.10. By 17.15 sepsis would have been developing for about two hours, according to the judge's finding that the process of rupture began at 15.19. By 18.10 it would have been developing for nearly three hours. By the time that surgery began at 21.30, sepsis had been developing for over six hours. On those facts, Mr Browne submitted that the Court of Appeal were entitled to take the robust approach to causation which they did. They were entitled to infer that the greatly extended period for the development of sepsis materially contributed to the outcome.

25.
As to the law, Mr Browne did not accept that the application of the Bonnington principle is confined in the way suggested by Ms Harrison. He submitted that the principle, that it is enough that the defendant's negligence has contributed to the claimant's injury, applies where the evidence points to the probability that there were cumulative causes. It does not apply where there are merely several possible causes, any of which may have been entirely responsible for the injury. The question whether it is probable that the defendant's negligence was a contributory factor may depend on inference. In the present case, he submitted, the Court of Appeal had strong ground to conclude that the

complications were the product of a steadily worsening accumulation of sepsis over several hours, which was caused in part by the negligence of the hospital board.

Analysis

26.
The Board was referred to a large volume of authorities and academic literature, but the central argument was about Bonnington. So much has been written on the subject that it is right to begin by reminding oneself what the issues were and what was actually decided.

27.
In Bonnington the claimant contracted pneumoconiosis from the inhalation of dust, containing minute particles of silica, in the course of his employment at a foundry. Most of the dust originated from the operation of pneumatic hammers, but some of it escaped from swing grinders. The former involved no fault on the part of the employers, but the latter resulted from a breach of statutory duty in failing to intercept and remove that part of the dust. The Lord Ordinary and a majority of the Inner House held that the burden was on the employers to disprove that the dust from the swing grinders helped to produce the disease. Lord President Clyde dissented. He held, first, that the burden was on the claimant to prove that his inhalation of dust from the swing grinders made at least a material contribution to his contraction of the disease and, secondly, that all the evidence pointed to the pneumatic hammer (the innocent cause) as the most probable one: 1955 SC 320, 339.

28.
The House of Lords agreed with the Lord President on the first point. The leading opinion was given by Lord Reid, who said at [1956] AC 613, 620:

"… the employee must in all cases prove his case by the ordinary standard of proof in civil actions: he must make it appear at least that on a balance of probabilities the breach of duty caused or materially contributed to his injury."

29.
But in disagreement with the Lord President, the House of Lords held that causation was sufficiently established on the medical evidence. Lord Reid summarised the effect of the medical evidence as being that the claimant's disease was caused by a gradual accumulation in his lungs of minute particles of silica. He continued at p 621:

"That means, I think, that the disease is caused by thewhole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to one source or the other. I am in agreement with much of the Lord President's opinion in this case, but I cannot agree that the question is: which was the most probable source of the respondent's disease, the dust from the pneumatic hammers or the dust from the swing grinders? It appears to me that the source of his disease was thedust from both sources and the real question is whether the dust from the swing grinders materially contributed to the disease. What is a material contribution must be a question of degree." (Emphasis added.)

30.
Lord Reid concluded, at p 623, that it was proved not only that the swing grinders may well have contributed, but that they did in fact contribute, a quota of silica dust which was not negligible to the claimant's lungs and therefore helped to produce the disease. That was sufficient to establish liability against the employers.

31.
As Professor Sarah Green has succinctly observed (Causation in Negligence, Hart Publishing, 2015, Chapter 5, p 97):

“It is trite negligence law that, where possible, defendants should only be held liable for that part of the claimant’s ultimate damage to which they can be causally linked … It is equally trite that, where a defendant has been found to have caused or contributed to an indivisible injury, she will be held fully liable for it, even though there may well have been other contributing causes ...”

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. The Lord Ordinary recorded in his opinion that it was conceded by the employers’ counsel that the claimant had contracted pneumoconiosis arising out of and in the course of his employment, although “there was reserved for argument the question of which part of the process was the probable source of infection”, and that the employers argued that “on the balance of probabilities the source of the infection was the silica dust which was discharged during the dressing process involving the use of the pneumatic tools, and nothing else”: 1955 SC 320, 321, 324. It was not argued by the employers that the dust from the swing grinders could be linked, at most, to only a small part of the severity of his disease and that any damages should reflect the limited injury thereby caused. [1]

33.
On Lord Reid’s interpretation of the medical evidence, the question posed by the Lord President was the wrong question because it involved a false “either or” premise. Since the disease was caused by the totality of the toxic material inhaled, the relevant question was whether the particles from the swing grinders made any material contribution to the whole.

34.
Lord Tucker and Lord Keith of Avonholm both saw it as a matter of inference that the dust from the swing grinders was a contributory cause of the disease. Lord Tucker said, at p 623, that the inference to be drawn from the known facts was that “the silica dust discharged from the swing grinders contributed to the harmful condition of the atmosphere, which admittedly resulted in the pursuer contracting pneumoconiosis, and was therefore a contributory cause of the disease”. Lord Keith said, at p 626, that the claimant had proved enough to support the inference that the employers’ fault had materially contributed to his illness, because prima facie the particles inhaled were acting cumulatively and that the natural inference was that, had it not been for the cumulative effect, he would not have developed pneumoconiosis when he did.

35.
The parallel with the present case is obvious. The Board is not persuaded by Ms Harrison’s argument that Bonnington is distinguishable because in that case the inhalation from two sources was simultaneous, whereas in the present case the sepsis attributable to the hospital’s negligence developed after sepsis had already begun to develop.

36.

In considering that argument, it is instructive to compare and contrast Hotson v East Berkshire Health Authority[1987] AC 750. The claimant fell from a tree and fractured his left femoral epiphysis. He was taken to hospital, where for several days his injury was not properly diagnosed or treated. He suffered avascular necrosis of the epiphysis, leaving him with a permanent disability. The House of Lords held that on proper analysis of the evidence the avascular necrosis must have been caused in one or other of two ways. Either it was caused by irreparable rupture of the blood vessels to the epiphysis at the moment of the fall, or it was caused by later pressure within the joint from bruising or internal bleeding. There was no room for finding that the avascular necrosis was caused by a combination of the two factors. The trial judge's findings were to the effect that on the balance of probabilities the cause was the original traumatic injury. The claim therefore failed.

37.
Lord Bridge said, at p 782, that unless the claimant proved on a balance of probabilities that the delay in treatment was at least a contributory cause of the avascular necrosis, he failed on causation, and that the judge's findings amounted to a finding of fact that the fall was the sole cause of the avascular necrosis. He added, at p 783:

"But if the plaintiff had proved on a balance of probabilities that the authority's negligent failure to diagnose and treat his injury promptly had materially contributed to the development of avascular necrosis, I know of no principle of English law which would have entitled the authority to a discount from the full measure of damage to reflect the chance that, even given prompt treatment, avascular necrosis might well still have developed."

That passage runs counter to Ms Harrison's submission that in principle the "material contribution" approach is confined to cases in which the timing of origin of the contributory causes is simultaneous.

38.
The distinction drawn by Ms Harrison is also inconsistent with the opinion of Lord Simon of Glaisdale in McGhee v National Coal Board[1973] 1 WLR 1, 8. Referring to Bonnington and to Nicholson v Atlas Steel Foundry andEngineering Co Ltd[1957] 1 WLR 613, Lord Simon said that where on the balance of probabilities an injury is caused by two (or more) factors operating cumulatively, one (or more) of which is a breach of duty, it is immaterial whether the cumulative factors operate concurrently or successively.

39.
The sequence of events may be highly relevant in considering as a matter of fact whether a later event has made a material contribution to the outcome (as Hotson illustrates), or conversely whether an earlier event has been so overtaken by later events as not to have made a material contribution to the outcome. But those are evidential considerations. As a matter of principle, successive events are capable of each making a material contribution to the subsequent outcome.

40.
A claim will fail if the most that can be said is that the claimant's injury is likely to have been caused by one or more of a number of disparate factors, one of which was attributable to a wrongful act or omission of the defendant: Wilsher v Essex Area Health Authority[1988] AC 1074. In such a case the claimant will not have shown as a matter of probability that the factor attributable to the defendant caused the injury, or was one of two or more factors which operated cumulatively to cause it. In Wilsher the injury was a condition known as retrolental fibroplasia or RLF, to which premature babies are vulnerable. The condition may be caused by various factors, one of which is an over supply of oxygen. The claimant was born prematurely and as a result of clinical negligence he was given too

much oxygen. He developed RLF, but it was held by the House of Lords that it was not enough to show that the defendant's negligence added to the list of risk factors to which he was exposed. The fact that the administration of excess oxygen was negligent did not warrant an inference that it was a more likely cause of the RLF than the various other known possible causes. The House of Lords distinguished the case from Bonnington in which the injury was caused by a single known process (the inhalation of dust).

41.
In the present case the judge found that injury to the heart and lungs was caused by a single known agent, sepsis from the ruptured appendix. The sepsis developed incrementally over a period of approximately six hours, progressively causing myocardial ischaemia. (The greater the accumulation of sepsis, the greater the oxygen requirement.) The sepsis was not divided into separate components causing separate damage to the heart and lungs. Its development and effect on the heart and lungs was a single continuous process, during which the sufficiency of the supply of oxygen to the heart steadily reduced.

42.
On the trial judge's findings, that process continued for a minimum period of two hours 20 minutes longer than it should have done. In the judgment of the Board, it is right to infer on the balance of probabilities that the hospital board's negligence materially contributed to the process, and therefore materially contributed to the injury to the heart and lungs.

43.
That conclusion means that it is unnecessary for the Board to address in further detail the rival arguments about the way in which the Court of Appeal dealt (or did not deal) with the judge's finding about the length of culpable delay. Although the Court of Appeal was critical in some respects about the judge's finding (notably about the time of ordering the CT scan), it made no clear finding about when the operation should have begun. However, no useful purpose would be served at this stage in going into that aspect of the matter further, since it makes no difference to the outcome of the appeal, the purpose of which has been to determine a question of principle about the proper approach to causation in the circumstances of this case.

44.
Although not strictly necessary, it may be helpful to comment by way of postscript on two matters which were raised in argument. First, Ms Harrison was critical of the decision, and more particularly the reasoning, of the Court of Appeal in Bailey. The starting point is Foskett J's findings of fact, which were set out in close detail in his judgment: [2007] EWHC 2913 (QB).

45.
The claimant was admitted to hospital suffering from a gall stone requiring surgical removal. There was a delay in diagnosis but that was not itself a significant matter. On 11 January 2001 she underwent an endoscopic procedure known as an ERCP. Her treatment in the aftermath of the ERCP was negligent. As a result, she had to undergo further major procedures over the following days which should not have been necessary and which led to her being in a weakened state. In addition, she developed pancreatitis, which was an unfortunate, but non-negligent, complication of the ERCP. For 12 days she was in the ITU until she was transferred to the renal unit on 26 January. There she vomited in her sleep and aspirated the vomit, causing her to suffer a cardiac arrest and hypoxic brain damage.

46.

The judge found on the strength of medical evidence that “the claimant’s generally weakened and debilitated condition on 26 January caused her not to be able to respond naturally and effectively to the emergence of vomit from her gut with the consequence that she inhaled it” (para 54). The question was whether this was too remote a consequence of her negligent treatment following the ERCP, having regard to the fact that her weakened state was partly due to the pancreatitis for which the hospital was not responsible. The judge’s critical finding was at para 60:

“I do not think it can be doubted that there were two components to the weakness of the claimant as at 26 January, both very closely interlinked and having their foundation in the ERCP carried out on 11 January. One component was the weakness engendered by the pancreatitis, the other was the weakness engendered by the consequence of the negligence on 11-12 January which led to a very stormy passage for the claimant ending (purely from a surgical point of view) on 19 January when the packing of the liver was removed. Even leaving out of account the independent effect of the pancreatitis, it defies all common sense to say that she had recovered from the effects of all that by 26 January. I am satisfied, on the balance of probabilities, that she had not and that she was weakened as a result. I cannot say whether the contribution made by this component was more or less than that made by the pancreatitis and it follows that I cannot say whether the contribution made by the pancreatitis was greater or smaller than the contribution of the other component. All I can say is that the natural inference is that each contributed materially to the overall weakness and it was the overall weakness that caused the aspiration.”

47.
In the view of the Board, on those findings of primary fact Foskett J was right to hold the hospital responsible in law for the consequences of the aspiration. As to the parallel weakness of the claimant due to her pancreatitis, the case may be seen as an example of the well known principle that a tortfeasor takes his victim as he finds her. 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.

48.
Finally, reference was made during the argument to the “doubling of risk” test which has sometimes been used or advocated as a tool used in deciding questions of causation. The Board would counsel caution in its use. As Baroness Hale of Richmond said in Sienkiewicz at para 170, evaluation of risk can be important in making choices about future action. This is particularly so in the medical field, where a practitioner will owe a duty to the patient to see that the patient is properly informed about the potential risks of different forms of treatment (or non-treatment). Use of such evidence, for example epidemiological evidence, to determine questions of past fact is rather different. That is not to deny that it may sometimes be very helpful. If it is a known fact that a particular type of act (or omission) is likely to have a particular effect, proof that the defendant was responsible for such an act (or omission) and that the claimant had what is the usual effect will be powerful evidence from which to infer causation, without necessarily requiring a detailed scientific explanation for the link. But inferring causation from proof of heightened risk is never an exercise to apply mechanistically. A doubled tiny risk will still be very small.

49.
The Board will humbly advise Her Majesty that the appeal should be dismissed with costs.

[1] In later cases it has been the accepted view that pneumoconiosis is a “divisible” disease, its severity being dependent on the quantity of dust inhaled; and, therefore, where there has been more than one source of toxic material, the extent of the liability of a defendant responsible for part of the exposure should reflect the degree of injury suffered by the claimant as a result of that exposure. See the judgment of Lord Phillips of Worth Matraversin Sienkiewicz v Greif (UK) Ltd[2011] 2 AC 229, para 90: “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.”