Neutral Citation Number: [2014] EWHC 4190 (QB)

Case No: HQ12X03121

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 11/12/2014

Before:

MR JUSTICE JAY

- -

Between:

PROFESSOR CARL HENEGHAN
(Son and Administrator of the Estate of
JAMES LEO HENEGHAN, Deceased)

Claimant

- and -

(1) MANCHESTER DRY DOCKS LTD
(2) 00722056 LTD
(3) CARILLION CONSTRUCTION (CONTRACTS) LTD
(4) R. BLACKETT CHARLTON LTD
(5) S.C. CHEADLE HULME LIMITED
(6) KELLOGG BROWN & ROOT LTD

Defendants

- -

David Allan QC and Simon Kilvington (instructed by **Pannone Part of Slater & Gordon**) for the **Claimant**
David Platt QC (instructed by **Berrymans Lace Mawer LLP**) for the **Defendants**

Hearing dates: **25th and 26th November 2014**

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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.

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MR JUSTICE JAY

MR JUSTICE JAY:

Introduction

1. Mr James Heneghan was born on 8th March 1938. During the course of his working life, he was exposed to respirable asbestos fibres and dust. In November 2011 Mr Heneghan began to develop symptoms of adenocarcinoma of the lung, and a diagnosis to that effect was made in early 2012. He died from the disease on 3rd January 2013.

2. The deceased was employed by the six Defendants on a sequential basis between 1961 and 1974. There were earlier employers who have not been sued. Owing to the lack of available records, the precise dates of his various employments are unclear, but the parties are agreed that the deceased's exposure to asbestos over the course of his working life can be quantified, and that the total exposed "share" of those Defendants who have been sued is 35.2%. As between the six Defendants, the distribution of their respective exposures has also been agreed, and ranges from 2.5% to 10.1%.

3. Consequent upon the deceased's death, claims have been brought in the usual way under the Law Reform (Miscellaneous Provisions) Act 1934 and the Fatal Accidents Act 1976. The Claimant, Carl Heneghan, is the son of the deceased and, I note, Professor of Evidence-Based Medicine at the Department of Primary Care Health Sciences at the University of Oxford. Unfortunately, the deceased's widow, Mrs Doreen Heneghan, is not in good health.

4. Liability has been admitted by all six Defendants, and Judgment was therefore entered against them on 21st December 2012 (Order not sealed until 14th January 2013). On that occasion, Master McCloud ordered the trial of three issues. Given that the issues of contributory negligence through smoking, and quantum, have now fallen away, the sole live issue for this Court to determine is "whether each Defendant is liable for damages in full or for only a portion of the damages". If the Claimant's case is right, it is common ground that he should receive the sum of £175,000, being the damages in full figure contemplated in the Master's Order; but if the Defendants' arguments prevail, it is equally not in dispute between the parties that the Claimant should receive £61,600, based on the total "exposed" share of 35.2% of the gross sum.

5. One might be forgiven for thinking that the answer to this issue ought to be found in previous authority, because it must have arisen in the past. As it happens, this issue has not been previously determined. In these circumstances, the present case gives rise to problems of some difficulty and importance.

6. The nature of the agreement of the parties means that lay witness evidence is not required. The expert engineering evidence is agreed, but needs to be summarised in

order that the issue in contest may be properly understood. The medical evidence is largely agreed, save for two points which I will need to examine.

The Engineering Evidence

7. In his detailed and helpful report dated 16th May 2012, Mr John Raper has considered the deceased's levels of exposure to asbestos fibres referable to his employment with ten employers over many years. Mr Raper has examined the deceased's work history and the various tasks undertaken over time, from which data he has been able to draw reasonably solid inferences of the likely levels of asbestos exposure. The deceased's aggregate asbestos dose was of the order of 133 fibres/ml years. The six Defendants' cumulative exposure was 46.9 f/ml years, and the exposure attributable to the employers who have not been sued was 86.2 f/ml years, yielding an apportionment division of 35.2%.

8. Mr Raper has also been able to analyse the deceased's exposure on a Defendant-specific basis. This analysis has produced doses or exposures ranging between 13.4 f/ml years (for the Third Defendant), in other words 10.1% of the total exposure, and 3.3 f/ml years (for the Fourth Defendant), in other words 2.5% of the total exposure.

9. The significance of these figures may be properly discerned when consideration is given to the Helsinki criteria, formulated by an international panel of experts in 1997. According to a strict application of these criteria, cumulative exposure of 25 f/ml years is sufficient to enable an inference properly to be drawn that lung cancer in any individual case is attributable to asbestos, provided that there is a minimum ten year interval from first exposure to onset of the cancer.

10. These criteria have been refined over the years. The 25 f/ml year criterion remains appropriate for exposure to mixed fibre types with a preponderance of amphiboles. If exposure involved equal quantities of amphiboles and chrysotile, then cumulative exposure of 40 f/ml is probably necessary to double the risk of lung cancer. And the exposure threshold is much higher in a case of exposure to commercial chrysotile only.

11. Given that the deceased's exposure of 133.1 f/ml years included 114 f/ml years of amphibole, the relevant threshold applicable to his case is likely to be below the figure of 40 f/ml years mentioned above. The parties have been content to proceed on the agreed basis of a more than fivefold increase of the risk of lung cancer in the instant case – taking the cumulative exposure to asbestos fibres over the whole of the deceased's employments.

The Medical Evidence

12. Dr Robin Rudd (for the Claimant) and Dr John Moore-Gillon (for the Defendants) gave oral evidence in line with their reports. Each is pre-eminent in his field, and intellectually formidable. Both amplified slightly on their reports, and following their

respective cross examinations it became quite clear where the slight differences between them lay.

13. I begin with the common ground.

14. The deceased's lifetime risk of developing lung cancer had he (a) not smoked and (b) not been exposed to asbestos fibres, would have been in the order of 0.5%. His smoking increased the risk to 2% or thereabouts: in other words, it increased the baseline risk by a multiple of four. Dr Rudd explained in his first report that the effects of smoking and asbestos exposure were not merely synergistic but multiplicative. In short, given the fourfold and fivefold increases in risk which I have mentioned, the risk of this deceased developing lung cancer was more than twenty times the baseline risk. Put another way, given that the deceased was a smoker, the risk of his developing lung cancer at these levels of asbestos exposure were more than five times greater than they would have been had he just been a smoker. Dr Moore-Gillon did not disagree with the multiplicative approach or these figures.

15. On 12th November 2013 the medical experts produced a Joint Statement to the Court, points 2, 3 and 5 of which are relevant for present purposes:

> "2. His risk of lung cancer was increased by smoking and by asbestos exposure.
>
> 3. On the balance of probabilities he would not have developed the lung cancer if he had not been exposed to asbestos.
>
> …
>
> 5. The risk that lung cancer will develop increases with the amount smoked and with the dose of asbestos received. If a lung cancer does develop, then the duration and severity of symptoms and the eventual outcome are not affected by the amounts of smoking or asbestos exposure which led to that increase in risk."

16. The medical experts are also agreed about the multiple mechanisms involved in the carcinogenesis of asbestos-related lung cancer. This involves a consideration of the microbiological processes occurring in cells, in particular cellular DNA, of the inhibitive effect asbestos has on programmed cell death, and of the reduction in the effectiveness of the body's natural defences against cancer caused by the presence of asbestos fibres in lung tissue. The precise detail does not matter for present purposes; it is sufficient to record that the experts are in general agreement that anything between four and seven stages in cellular mutation is required before the emergence of a cancer cell which may potentially progress to become a clinically apparent carcinoma of the lung.

17. The differences between the experts, such as they are, emerge from comparing the Appendix to Dr Moore-Gillon's report dated 22nd January 2013 with Dr Rudd's report dated 22nd May 2014. Mr David Platt QC made the forensic point that Dr Rudd should have drawn attention to these differences in the Joint Statement because the obligation under the CPR is to identify both the areas of agreement and disagreement. Although

I agree with Mr Platt that it would have been more helpful had Dr Rudd raised the issue at that juncture, I have to say that in the end nothing really turns on this.

18. These conclusions in Dr Moore-Gillon's Appendix are not shared by Dr Rudd, and so I set them out in full:

> "My view though is that whilst it is not possible to infer a causal connection between all inhaled (and retained) asbestos and the development of lung cancer, it is perfectly reasonable to consider its role in probabilistic terms. Thus, I do not think that any significant exposure to asbestos can or should be excluded in terms of its contribution to the *risk* of the individual developing lung cancer.
>
> …
>
> [I]t is not known and cannot be known whether asbestos fibres inhaled from a specific source *did in fact* contribute to the specific lung cancer which an individual develops. Quite clearly, the distribution of fibres, retained in the lung, which originated from one source is very likely to be similar to those originating from another source. Thus, there can be no reason to suspect that fibres close to the site at which a tumour arises were more or less likely to have come from one source or another. Nevertheless, even when considering the influence that many fibres may have had upon causation … the contribution made by any period of exposure to asbestos can only be discussed in terms of probabilities – albeit often extremely strong ones."

19. Dr Rudd joins issue with Dr Moore-Gillon in the following terms:

> "My comment is that Dr Moore-Gillon confuses different models of causality. If establishing causation depends upon scientific knowledge of the precise molecular and cellular events which resulted in the emergence of a cancer the conclusion would have to be that the cause of a particular cancer can never be ascertained. Any cancer can occur spontaneously as a result of spontaneous mutation and it could never be said with certainty that any particular cancer had not arisen in this way. In recognition of the lack of utility of such an approach to causation medical science adopts an approach based on epidemiological observations …
>
> …
>
> Dr Moore-Gillon therefore acknowledges that the probabilistic attribution of causation of cancer based upon epidemiological evidence accords with common understanding of the meaning of causation; in common parlance and understanding there is no meaningful difference between the statement that 'on the

> balance of probabilities cancer would not have occurred but for smoking' and the statement that 'smoking caused lung cancer'."

20. Under cross examination, Dr Rudd explained that the real distinction here is between a mechanistic, deterministic model on the one hand, and a probabilistic one on the other. In his view, the second model applies at all stages of the analysis, including the stage which holds that in a multi-party case where the exposure for which each employer is responsible is above *de minimis* levels (as here), it may be inferred that all asbestos sources have materially contributed to the causation of the lung cancer. The fact that the incidence of lung cancer is stochastic makes no difference to this analysis. On my understanding of Dr Rudd's evidence, further support for this inferential conclusion may be derived from (a) the overall quantities of asbestos fibres in the lung being extremely large, measurable in millions, (b) large numbers of such fibres (on my understanding, not all of them) being involved in the carcinogenic process, (c) the strong biological plausibility of the carcinogenic processes, and (d) the multiplicative effects previously mentioned. As Dr Rudd put it in re-examination, it was highly more likely that some of the fibres involved in these processes came from each source of exposure. On the other hand, Dr Rudd accepted in cross examination that had the deceased not been employed by the Fourth Defendant (which contributed 2.5% of the total exposure), then he would still probably have developed lung cancer. Indeed, it was possible to go further: the same conclusion flows if any of the six Defendants were notionally removed from the frame. Dr Rudd was not asked what his position would be if all six Defendants were excluded from account, and my note of his answer does not correspond with the Defendant's solicitor's, but in the end nothing turns on this: it seems to me that the logic of his previous answers must yield the same riposte.

21. During the course of his cross-examination, Dr Rudd was taken to the passages in Jones and others v Secretary of State for Energy and Climate Change and another [2012] EWHC 2936 (QB) where Swift J reviewed his evidence in relation to lung cancer and the probabilistic model. To my mind, Dr Rudd has maintained a consistent position. At paragraph 8.21 of the Judgment, Dr Rudd is recorded as having given the following evidence:

> "Dr Rudd was asked why, if this thesis [sc. every exposure to a carcinogen will play a part in the process] was correct, there had been any need for the *Fairchild* exception. His response was that, in *Fairchild*, he and the other experts had been instructed to consider from what source the asbestos fibre(s) that had caused the final step in the production of the malignant cell had come. They were unable to do so; hence the impossibility of establishing causation and the necessity for the creation of the *Fairchild* exception. He said that the expert evidence in *Fairchild* was given in the light of carcinogenesis at the time [February 2001] … and since then understanding of the molecular basis of carcinogenesis has improved considerably. Dr Rudd said that, if he were asked the same questions now as he had been asked in *Fairchild*, he would say

> that it was probable that the asbestos fibres from each source had contributed to the carcinogenic process."

22. In his oral evidence Dr Moore-Gillon explained that the carcinogenic processes were effectively the same in lung cancer cases as they were in mesothelioma, although in the latter instance the slope of increasing risk (envisaged as an escalating curve on a graph) is much steeper. Lung cancer may be caused at much lower doses than those contemplated in the Helsinki criteria, and may not be the outcome at much higher doses. The differences between lung cancer and mesothelioma should be seen as quantitative, not qualitative. Addressing head-on Dr Rudd's criticism that he had confused different models of causality, Dr Moore-Gillon said that although generalised judgments may be made on the basis of epidemiological evidence, this probabilistic approach cannot avail the fact-finder in relation to the individual case, still less enable that person to conclude that causation, as opposed to enhancement of the risk, has been made out. Thus, in concrete terms apposite to the circumstances of this particular case, one simply cannot say which of the deceased's six employers has caused his lung cancer. Dr Moore-Gillon said that there was a conceptual jump between risk and causation; or, put another way, it did not follow from the proposition that everything contributed to the risk that everything contributed to the causation.

23. Under cross examination, Dr Moore-Gillon accepted that paragraph 3 of the Joint Statement (see paragraph 15 above) was predicated on paragraph 2, namely that the deceased's asbestos exposure contributed to the risk of his developing lung cancer. Dr Moore-Gillon agreed that what he was saying, in effect, was that on the balance of probabilities the deceased's asbestos exposure caused his lung cancer, and that this was a conclusion safely to be deduced from the epidemiological evidence. Put in those terms, it may be seen that the concepts, in Dr Moore-Gillon's terminology, of "increasing the risk" and "causation on the balance of probabilities, having regard to the epidemiological evidence" are capable of being envisaged as very closely related. However, Mr David Allan QC representing the Claimant never managed to persuade Dr Moore-Gillon to accept that these concepts were exactly synonymous. This issue became the more acute when the question was posed: "on the balance of probabilities, have at least some fibres from each source contributed to [i.e. were causally implicated in] the carcinogenic process?" Dr Moore-Gillon did not accept that this was so, because the current state of medical science simply cannot permit one to say how many fibres were involved and from where they were derived. All that may be said is that each employment contributed to the risk.

24. Having heard and reflected on the medical evidence, it is clear to me that the conclusions to be drawn from it may be expressed in this way. In principle, the probabilistic model may be applied to this sort of case, provided that care is taken to discern its practical limitations. My understanding of a probabilistic model in the context of this case is one which applies statistical methods to large numbers of events occurring randomly in nature - and which are therefore normally distributed - in order to enable inferences of causality to be drawn in the face of empirical or theoretical uncertainty. These inferences are inherently probabilistic in nature. I hope that my understanding matches Dr Rudd's. "Empirical uncertainty", in this context, means the uncertainty which arises from the absence of evidence relating to the cellular changes occurring in the lung tissue of an individual claimant. I have included "theoretical uncertainty" in my working definition to reflect Dr Rudd's oblique reference in his

oral evidence to Heisenberg, although that to my mind introduced an unnecessary complication.

25. In order to examine how this model operates in practice, it is appropriate to consider two different types of outcome.

26. First, in the event that the epidemiological evidence clearly establishes that the relative risk is greater than 2:1, the concepts of "increasing the risk" and "causing the damage" are, in effect, synonymous. This is exactly the situation which obtains in the present case in the context of the asbestos versus the smoking risk. The relative risk was greater than 2:1 (and by a significant margin, once the multiplicative effects are brought to bear), the risk was therefore increased, and the damage was therefore caused, as a matter of probability. At that point, of course, the common law adopts the fiction – in relation to a past event – of that having been found more probable than not being certain. In purely scientific terms one may not "know" that to be so, but anyone using that verb in such a context is implicitly applying a higher standard of proof. It is true that many scientific judgments even in the medical sphere do not entail the application of a standard of proof as such (see Professor Sir Michael Rawlins' De Testimonio: On the Evidence for Decisions about the Use of Therapeutic Interventions [The Harveian Oration, 2008]), but in using the verb "know" I am merely highlighting an aspect of Dr Moore-Gillon's approach which Mr Allan succeeded in drawing out.

27. Secondly, in a case where the relative risk is *below* 2:1, is seems to be that the concepts of "increasing the risk" and "causing the damage" diverge. This is exactly the sort of situation which has vexed the common law for decades. This state of affairs does not apply to the proper consideration of the first hypothesis under scrutiny (viz. whether the deceased's lung cancer was caused by occupational exposure), but it is precisely in play when one examines the logically subsequent hypothesis that Defendant X's exposure caused the deceased's lung cancer. (Strictly speaking, at both stages the Court is examining the null hypothesis of *absence* of a relevant association or causation, but that is a detail). The hard data demonstrate that the odds or chances of that having been the case are significantly lower than 50%.

28. Recognising this difficulty, Mr Allan sought to build a case on the evidence that it is more likely than not that the deceased's exposure when employed at any of the Defendants contributed to the causation of his lung cancer to the extent that some of the fibres from each source of exposure must have contributed to the carcinogenesis. Dr Rudd came to that conclusion by applying his probabilistic model, fortifying it with reference to the microbiological factors which were re-emphasised during re-examination. Dr Moore-Gillon accepted that the risk was increased but did not agree that causation may be inferred.

29. I cannot accept Dr Rudd's attempt to resolve this conundrum by invoking the microbiological factors as additional support for his position. On analysis, the points which he listed in re-examination serve to strengthen the epidemiological or statistical evidence rather than to bear on the underlying microbiological processes. Those processes remain inscrutable, save to the extent that their occurrence may be inferred from the epidemiology. Those inferences remain the same however the factors are examined.

30. Whether material contribution may be inferred in relation to any of the Defendants raises difficult questions. At one stage during the course of the trial, I was inclined to think that Dr Moore-Gillon – in refusing to accept that such an inference may be drawn – was applying a higher standard of proof than the probabilistic standard that the common law recognises, and on that account I considered that I could properly embrace Dr Rudd's thesis into its furthest reaches. Upon further reflection, and mindful that no other court has been so bold as to infer material contribution to the damage in a lung cancer or similar case where there has been no doubling of the risk, I have concluded that even if Dr Moore-Gillon did err in that respect, he was right to baulk at drawing such an inference. Although the causation of lung cancer is dose-related, it does not follow from the proposition that because 2.5% of the dose may be attributed to the Fourth Defendant, that Defendant has materially contributed to the carcinoma. Asbestos burden cannot be equated with the silica dust which causes pneumoconiosis. The greater the accumulation of such dust in the lungs; the greater the damage that is being caused to the lung tissue of an individual patient with that disease. The dust is directly contributing to the disease process. The greater the exposure to asbestos fibres, on the other hand, the greater the risk that lung cancer may result. Although I would agree that 2.5% represents millions of asbestos fibres with the potential to cause a fatal disease process, whether these fibres actually did so remains an open question. Recourse to epidemiology or statistics does not resolve that agnosticism, nor does recourse to the microbiology. Indeed, an accurate application of the epidemiological and statistical data leads to the conclusion that, in relation to the causative potency of the Fourth Defendant's involvement, it cannot be said on the balance of probabilities that this exposure contributed to the deceased's lung cancer. It increased the risk of that cancer occurring, but the quantum of that increase was by a very considerable margin below 51%.

31. The final issue which arises for determination in relation to the expert evidence is whether lung cancer cases should be treated in the same way as mesothelioma cases. Self-evidently, they are different conditions and involve different cells: lung cancer typically involves epithelial cells, and mesothelioma cells of the mesothelium, typically those located in the pleura. The incidence of lung cancer is dose related whereas mesothelioma is not, as Lord Bingham explained at paragraph 7 of his opinion in Fairchild v Glenhaven Funeral Services Ltd [2003] 1 AC 32:

> "It is accepted that the risk of developing a mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled: the greater the quantity of dust and fibre inhaled, the greater the risk. But the condition may be caused by a single fibre, or a few fibres, or many fibres: medical opinion holds none of these possibilities to be more probable than any other, and the condition once caused is not aggravated by further exposure."

32. It is correct that the "single fibre" theory has fallen into desuetude, but on my understanding the remaining parts of Lord Bingham's summary of the state of medical opinion on the issue remain good. No one sought to argue in Fairchild that proof on the balance of probabilities could be established on inferential grounds from the first sentence of this citation. In any event, the second sentence from this citation meant that epidemiological or statistical evidence could not be deployed to close the

gap between increasing the risk on the one hand, and causing the damage on the other (see Lord Phillips' explanation of this conundrum in paragraph 97 of his judgment in Sienkiewicz). Dr Moore-Gillon's point that the differences between these two cancers are quantitative rather than qualitative is not, I believe, well-founded. In any event, I am not prepared to revisit the factual premises of Fairchild (save for the single fibre point) in response to the somewhat insubstantial evidence I heard on this issue.

33. However, whether the common law should now treat lung cancer and mesothelioma as equivalent, or without legally materially distinctions, raises a separate issue to which I will need to turn in due course.

The Rival Submissions

34. I am grateful to Counsel for the clarity and excellence of their submissions. Their oral arguments also served to narrow the issues which I am called upon to decide.

35. It was common ground between Counsel that lung cancer is an indivisible injury because its severity does not depend on the asbestos dose. It was also common ground that, if the Claimant is able – on what may be termed entirely conventional principles - to prove *some* causation of damage against any one of the six Defendants before the Court (and for these purposes it makes no difference which Defendant it is), then that Defendant is liable to compensate the Claimant for the entirety of the injury suffered (c.f. paragraph 90 of Lord Phillips' judgment in Sienkiwicz). Finally, it was accepted by Mr Allan that if the instant case should fall to be accommodated within the exceptional rule laid down by the House of Lords in Fairchild, because causation cannot be proved otherwise, then the apportionment rules laid down by the House of Lords in Barker v Corus UK Ltd [2006] 2 AC 572 must apply. Mr Allan reserves his position in this Court as to whether Barker is still good law in the light of subsequent authority.

36. The essence of Mr Allan's submission is that lung cancer and mesothelioma are materially different conditions, both in fact and in law. In a lung cancer case, the "but for" test must always be satisfied in connection with medical causation, and recourse may not be had to the more Claimant-friendly concept of "material contribution" derived from Bonnington Castings Ltd v Wardlaw [1956] AC 613. The "but for" test was amply satisfied in the instant case because the experts are agreed that it is more likely than not that the deceased's lung cancer was caused by occupational exposure. However, a different question arises at the second stage of the analysis, namely the attribution of causation to any of the Defendants individually sued. At that stage, submits Mr Allan, the Court is entitled to conclude by inference that any Defendant who has made a material contribution to the exposure has made a material contribution to the injury.

37. It is important to recognise that at this second stage Mr Allan was not seeking to rely on any epidemiological evidence. This is because that evidence cannot avail him in proving his case on the balance of probabilities against any individual Defendant, or indeed all the Defendants taken together: 35.2% is always less than 51%. Mr Allan did submit that Dr Rudd's re-examination fortifies his approach, but for the reasons I have already given I do not consider that it does. Ultimately, Mr Allan's crucial

argument was that the present case is governed by the principles expressed by or derivable from Bonnington Castings. On my understanding of his submissions, either that authority applied precisely, because material contribution to the injury may be inferred, or – in the event that I should hold that it could not be inferred – it applied with minor modifications.

38. Mr Allan submitted that those modifications were identified by Lord Rodger in a series of key passages in Fairchild, paragraphs 130-133. There, Lord Rodger reviewed two post-1956 decisions of the House of Lords, namely Quinn v Cameron & Roberton Ltd [1958] AC 9 and Nicholson v Atlas Steel Foundry and Engineering Co Ltd [1957] 1 WLR 613. In both those cases, it is clear that liability was established on the premise that the tortfeasors had materially contributed to the risk of injury rather than to the injury itself. As Lord Rodger put it, at paragraph 133:

> "Questions of the risk or increased risk of causing harm are more frequently considered in relation to issues of foreseeability and fault. But these passages are important precisely because they show that the House was proceeding on the basis that, in considering whether the pursuers in *Nicholson* had proved that the defenders had materially contributed to the deceased's condition, it was relevant to consider whether the defenders' wrongful act had exposed him to an increased risk of disease. In other words, in that case proof of increased risk of harm to the deceased was relevant to proof of causation of that harm."

39. On my understanding of his submissions, Mr Allan was contending that the common law had already permitted a modest extension of the Bonnington Castings material contribution to the harm principle before the Fairchild exception was created to deal with the special vices of mesothelioma cases. It followed that Mr Allan believed that he could find a comfortable middle ground which did not find itself sinking like quicksand directly into the mire of Fairchild – and, I would add, its unappealing companion, Barker.

40. Furthermore, submitted Mr Allan, there were no policy reasons militating against such an approach. The policy considerations which exercised Swift J in Jones and others v Secretary of State for Climate Change and another (*loc. cit.*) are inapplicable here, because medical causation has been established. Because of the unfairness which clearly arises if a strict application of the burden of proof were applied at this second stage, it is entirely apposite for the Court to countenance the modest, pre-Fairchild adjustments which serve to achieve sound and practical justice in a multi-party case within the flexibility of the common law.

41. Additionally, Mr Allan submitted that this Court is precluded by *stare decisis* from extending Fairchild to non-mesothelioma cases.

42. Mr Allan made further detailed submissions on the authorities. I will deal with those, to the extent necessary, when I come to review them more fully.

43. The essence of Mr Platt's submissions is that two different questions are apt to arise in a multi-party case. Taking his lead from Professor Stapleton's article, Factual

Causation and Asbestos Cancers ([2010] LQR, 126 (Jul), 351-356), the first question is the "what" question of medical causation: in other words, which of competing potential causes was in fact responsible for the deceased's cancer. In the instant case, the sole candidates are asbestos exposure and smoking. The "what" question is answered by applying the conventional test of the balance of probabilities, and epidemiological evidence is relevant to the exercise. The second question is the "who" question, which is self-explanatory. Mr Platt's core submission was that the "who" question is not a logical derivative of the "what" question, and cannot be answered with reference to epidemiological evidence alone.

44. As a matter of principle, submitted Mr Platt, in the circumstances of the instant case medical scientists could not design an epidemiological study which could adequately differentiate between tortfeasors. Furthermore, there are dicta of the highest authority which indicate that recourse to epidemiology alone is inapt, or certainly highly problematic, in these circumstances: see Sienkiewicz v Greif (UK) Ltd [2011] 2 AC 229 (Lord Rodger, at paragraph 163; Lord Mance, at paragraphs 190-192; Lord Kerr, at paragraph 206; Lord Dyson, at paragraphs 218-221).

45. Mr Platt submitted that either the Fairchild exception applied, and apportionment ensued, or the claim altogether failed for want of proof. The middle ground which Mr Allan was seeking to occupy was illusory and contrary to authority.

46. Mr Platt drew attention to paragraph 24 of Lord Hoffmann's opinion in Barker v Corus UK Ltd [2006] 2 AC 572, at 587C-E which, at least on one reading, supported the Claimant's contention that lung cancer cases fall outside the Fairchild exception. There, Lord Hoffmann said this:

> "… In my opinion it is an essential condition for the operation of the exception that the impossibility of proving that the defendant caused the damage arises out of the existence of another potential causative agent which operated in the same way. It may have been different in some causally irrelevant respect, as in Lord Rodger's example of the different kinds of dust, but the mechanism by which it caused the damage, whatever it was, must have been the same. So, for example, I do not think that the exception applies when the claimant suffers lung cancer which may have been caused by exposure to asbestos or some other carcinogenic matter but may also have been caused by smoking and it cannot be proved which is more likely to have been the causative agent."

47. I agree with Mr Platt to the extent that in this passage Lord Hoffmann was drawing attention to the error he made in Fairchild where he suggested that in a case involving competing potential causes the offending cause does not have to be proved on the balance of probabilities. The correct approach, as Lord Hoffmann recognised in Barker, is that it depends. If the competing causative agents operate in different ways, then proof on the balance of probabilities *is* required. If, on the other hand, the competing causative agents operate in the same way, then (subject to the fulfilment of other conditions) the Fairchild exception applies. Given that asbestos and smoking are causative agents which operate in different ways, it follows – see the last sentence of this citation – that the conventional rules are applicable. In my view, the last sentence

of paragraph 24 does not illuminate a case where a claimant suffers exposure to the same causative agent at the hands of several defendants. On the other hand, this sentence does tend to support the proposition that where medical causation is issue, a claimant may succeed only if he proves causation on a conventional basis, and that any different approach cannot be espoused.

48. Mr Platt also drew my attention to the decision of the High Court of Australia in Amaca Pty Ltd v Ellis [2010] HCA 5 where the claim failed on the epidemiological or statistical evidence. On the basis of that decision, and more generally, Mr Platt contended that the correct approach in a lung cancer case with multiple defendants was that envisaged by Professor Stapleton in her LQR paper, namely the application of flexible, Fairchild-type principles and apportionment.

49. Mr Platt made detailed submissions on the authorities which I will address, to the extent necessary, when I come to review them.

Analysis and Conclusions

50. Had the common law adhered to strict logic and principle, all the difficulties which bedevil cases such as these would not have arisen, and would not continue to arise. The disadvantage of strict adherence to logic and principle is that frank injustice may arise in certain types of case, and therefore the common law constantly strains at the leash of the intellectually pure approach. Policy considerations often serve to buttress the logic and principle of the reasoned outcome in any individual case, but there are circumstances where the application of such considerations cuts across the very principles in play.

51. In a case where medical causation is in issue, strict adherence to logic and principle would demand proof on the balance of probabilities either of the whole of the damage suffered or of a material part of it. For these purposes, there is no distinction between the whole and the part, provided that the part is significant. The clearest exposition of this principled approach is to be found in the opinion of Lord Reid in Bonnington Castings at pages 620/1. I will call this the conventional common law approach.

52. I note that this was not the interpretation placed on Lord Reid's opinion by Toulson LJ in IEGL v Zurich Insurance plc UK Branch [2013] EWCA Civ 39, paragraph 24. According to Toulson LJ, Lord Reid in Bonnington Castings applied a broader test of causation based on material contribution to the risk. I would prefer to put the matter slightly differently. In McGhee v NCB [1973] 1 WLR 1, Lord Reid purported to apply Bonnington Castings, but in reality he was extending it. He recognised that in the earlier case it was proved that every particle of dust played its part in the onset of the disease. Given the current state of expert knowledge, that could not be said in McGhee. However, a "broader view of causation" [page 4G/H], and the equation of material contribution to the damage with material contribution to the risk, led to the same conclusion. So, it was only in McGhee that we see the equation in Lord Reid's mind between these two concepts.

53. The conventional common law approach began to be whittled away, and policy considerations perhaps inadvertently to intrude, as early as 1957. The precise, windy

path the common law took need not be examined in detail. At this stage, I need only identify the end-point, by which I mean the point which the common law has reached which is furthest away from the conventional approach. That end-point has been described in these proceedings, and elsewhere, as the "Fairchild-extension". This extended principle undoubtedly arises in mesothelioma cases, but an issue falls for determination in the instant case as to whether it also arises in a multi-party lung cancer case. Unusually, because the tables are normally turned the other way, the Defendants contend that it does and the Claimant contends that it does not.

54. But there is an issue which is logically anterior to this, and that issue seems to lie at the heart of this case. Mr Allan submits that the common law has recognised a category of case which falls neatly between the conventional approach and the Fairchild-extension. If a case falls within this intermediate category, it is sufficient for a claimant's purposes to prove on the balance of probabilities that the *risk* of injury or damage was materially increased. Mr Allan defines this principle as an extension of the common law which is less far-reaching than Fairchild. He limits its contours further by conceding that the principle may only apply when medical causation is proved applying the conventional test, and the issue for resolution is whether any defendant under scrutiny has caused the claimant's loss.

55. In my judgment, there is no intermediate category of the kind Mr Allan is seeking to identify. There are only two categories. The first category embodies the conventional approach. The second category entails the application of the principles laid down in Fairchild and subsequent cases decided at the highest level. The bounds of that second category need to be defined for the purposes of this case, but once a case properly falls within it, the claim succeeds if proof of an enhancement of the risk is made out on the balance of probabilities. Thus, for the purposes of the first category, proof of damage, in whole or in part, is required; whereas for the purposes of the second category, proof of enhancement of the risk (of damage) is required. Mr Allan's intermediate category does not exist: it is, in fact, the same as the second category.

56. In order to explain why this is so, I need to examine how the common law has developed since 1956. A chronological review of the authorities is not required. Instead, I will attempt a thematic approach.

57. The burden is on the Claimant to prove causation of damage and consequential loss. In this case, this means the deceased's personal injury, his death, and the consequential losses which flow from both of these. Although causation is a unitary concept, it is often convenient and helpful in a multi-party case to state that there are two stages to the inquiry. At the first stage, the Court considers whether medical causation has been made out. If it has not, one need go no further. In the present case, medical causation involves a consideration of whether the Claimant has proved that the deceased's lung cancer was caused by asbestos fibres and dust rather than smoking. At the second stage, the Court considers whether causation is proved against each of the Defendants.

58. Given paragraph 3 of the experts' Joint Statement, the Claimant must succeed at the first stage. Medical causation was not, therefore, an issue at this trial. The reason why the experts have attained common ground on this issue is because analysing the epidemiological evidence and the deceased's personal circumstances clearly establishes that it is more likely than not that his asbestos exposure was causative

rather than his smoking. The epidemiological evidence relating both to asbestos exposure and cigarette smoking is wide-ranging, and based on a number of studies of varying quality. That evidence is sufficient to enable medical scientists to quantify the risks in populations of lung cancer patients who have been exposed to either or both of these carcinogens. However, that evidence has also been applied to the deceased's personal situation - inasmuch as detailed consideration has been given to the levels of exposure involved (see the engineering evidence) and the deceased's smoking habit – and modified to reflect those circumstances. Thus, a holistic view of the evidence has led to the robust conclusion that the relative risk of asbestos being the culprit in the deceased's case is more than 2:1; or, as the common law would express the same point, the case has been proved on the balance of probabilities.

59. Proof of medical causation has not required the application of any extended principle of the common law. Indeed, it has not even required the application of what might be called the second limb of the conventional approach, namely proof of material contribution to the injury or harm. At this stage I note that there are *dicta* to the effect that the concept of material contribution to the harm cannot be deployed in a claimant's favour in a cancer case without recourse to the Fairchild-exception. I will need to return to that point in due course.

60. The second stage in a multi-party case involves a consideration of whether the claimant can prove to the requisite standard that the defendant whose case is under consideration caused the deceased's lung cancer. Mr Allan does not seek to discharge the burden of proof by relying on evidence of an epidemiological nature. I would prefer to say – "evidence of a mathematical or statistical nature" - because no epidemiology is involved. Mr Platt rightly points out that it would be close to impossible to devise an epidemiological study which is capable of discriminating between different sequential exposures, and no one has suggested that such a study has been undertaken. On the facts of the instant case, it is simply a question of doing the basic arithmetic and reaching the conclusion that the contributions of each of the defendants, whether viewed individually or collectively, amount to less than 51%. The relative risk is less than 2 and/or the chances are less than 51%. For the avoidance of doubt, I agree with Mr Platt that the respective contributions of each of the Defendants cannot be aggregated for this purpose, but on the facts of the present case this matters not.

61. Thus, there is no need in the present case to examine the limits of the deployment of epidemiological or statistical evidence indicated in Sienkiewicz and Amaca. That issue would only have arisen had the Claimant sued W. Blackwell, which was responsible for 56% of the deceased's total asbestos exposure. I would have seen no difficulty in principle in concluding that a 56% contribution to the deceased's overall exposure should be regarded as sufficient to prove this hypothetical claim on the balance of probabilities. As I have sought to make clear, this should not be envisaged as a conclusion primarily based on any epidemiology; it is basic arithmetic. I do not understand the objections expressed in Sienkiewicz, as well as in Professor Stapleton's article, to the deployment of epidemiology to prove individual cases as applying to this sort of situation; but in the event that those objections were applicable I would find myself parting company with these authoritative, albeit non-binding, opinions. The deceased's exposure to W. Blackwell's asbestos was more likely than not – by some margin - to have been the cause of his lung cancer. To the extent

necessary, one may safely infer from that level of exposure that W. Blackwell's asbestos is directly inculpated in the disease process which the deceased sustained.

62. However, for the reasons I have already given (see paragraph 30 above), this probabilistic inference cannot be drawn in relation to the Defendants who have been sued. Because these exposures were each less than 51% by a considerable margin, it cannot be inferred that any individual Defendant's asbestos is inculpated in the disease process. All that may be inferred is the *possibility* of that having occurred, which is insufficient for the Claimant's purposes.

63. In my judgment, it makes no sense in this sort of case to say that each Defendant's exposures materially contributed to the disease process, invoking the second limb of Lord Reid's formulation in Bonnington Castings. Adopting Lord Reid's approach in McGhee, the evidence does not establish that every asbestos fibre, or exposure, was or must have been implicated in the disease process. The aetiology of lung cancer is different from silicosis or pneumoconiosis. In those cases, which depend on the gradual accumulation of lung dust, the disease process is continuous and the concept of material contribution has an intelligible role. In lung cancer cases, there is no analogue to the gradual accumulation in the lungs of asbestos or cigarette smoke. The risk of the disease eventuating is proportionate to the quantum of exposure, but that is a statistical judgment, not an assessment which may be linked to the physical presence of deposits of dust in the lung.

64. The High Court of Australia made this point extremely clearly in Amaca, at paragraph 68:

> "This description of the issue of causation in *Bonnington Castings* shows how different it is from the issue of causation in this case. The issue in *Bonnington Castings* was whether one source of an injurious substance contributed to a gradual accumulation of dust that resulted in disease. The issue here is whether one substance that *can* cause injury *did* cause injury. … Questions of material contribution arise only if a connection between Mr Cotton's inhaling asbestos and his developing cancer was established. Knowing that inhaling asbestos *can* cause cancer does not entail that in this case it probably *did*. For the reasons given earlier, that inference was not drawn in this case. Questions of material contribution do not arise."

The issue in Amaca was whether Mr Cotton could prove that his lung cancer was caused by asbestos exposure rather than smoking. The inference of the former could not be drawn in his case because none of the four expert witnesses assigned a probability greater than 23% to the chance that his cancer was so caused. That was the end of the matter, but for the avoidance of any doubt the inference of the former also could not be drawn by invoking the material contribution principle, for the reasons explained.

65. In my view, the Court of Appeal came to the same conclusion, albeit for different reasons, in paragraph 150 of its Judgment in Ministry of Defence v AB and others [2010] EWCA Civ 1317. Mr Allan criticised that part of the reasoning of the Court of Appeal which predicated the application of the material contribution principle on

divisible conditions. I tend to agree with his criticism, but the fact remains that the Court of Appeal refused to apply the material contribution limb of Lord Reid's test (as originally formulated in 1956) to cancer cases resulting from radiation exposure.

66. In his masterly *tour d'horizon* of the issue of medical causation in Sienkiewicz, Lord Phillips expressly considered whether the material contribution test could apply to a lung cancer case. During the course of reviewing the decision of Mackay J in Shortell v BICAL Construction Ltd (unreported, 16/5/08), at paragraph 75 of his Judgment Lord Phillips said this:

> "The expert evidence, given by both medical and epidemiological experts, but based in the case of each, I suspect, on epidemiological data, was that asbestos and cigarette smoke not merely combined cumulatively to cause lung cancer, but that they had a synergistic effect in doing so. This evidence was enough, as I see it, to satisfy the *Bonnington* test on causation, as the victim had been exposed both to significant quantities of asbestos fibres and to significant cigarette smoke."

Lord Phillips was puzzled by Mackay J's approach which was to apply the "doubling of the risk" test. But, in my respectful view Mackay J's approach was correct in the circumstances of the case he was deciding. The starting point was to consider whether the relative risk of contracting lung cancer through asbestos exposure was higher than the risk of doing so through smoking. If it was not, the material contribution test could not assist, because lung cancer differs from pneumoconiosis. If it was, then (but only then) could it properly be inferred that asbestos exposure, rather than cigarette smoke, was implicated in the disease process which caused Mr Shortell's lung cancer. Overall, there was no basis for invoking the second limb of Lord Reid's test in Bonnington Castings.

67. Mr Allan did not rely on Lord Phillips' approach in circumstances where it might have availed him to do so. In disclaiming such reliance, Mr Allan was exhibiting the same intellectual rigour and candour as his expert, Dr Rudd – the latter, sometimes somewhat disarmingly. Strictly speaking, that approach was *obiter* (see, for example, Lady Hale, at paragraph 169 of her Judgment), and on my reading of the Law Report Lord Phillips' reasoning is not shared elsewhere. It is also probably inconsistent with the final sentence of paragraph 24 of Lord Hoffmann's opinion in Barker. Further, in Jones Swift J highlighted the difficulties and followed a different path. In particular, at paragraphs 8.53-8.60 she explained why Bonnington Castings does not apply to lung cancer cases, and I respectfully agree with her. Instead, she applied a "doubling of the risk" test, and at paragraph 8.61 said this:

> "All these considerations lead me to the conclusion that it cannot be right to approach the cases of lung cancer – nor indeed those of bladder cancer – by applying the *Bonnington* principle. Moreover, to adopt the claimants' arguments would, as the defendants have pointed out, have potentially far-reaching effects. It would mean that, in any case of cancer where a claimant could establish tortious exposure to a carcinogen that was 'material' … the claimant would succeed

> in establishing causation and would be entitled to 100% damages. Whilst I have some sympathy with the predicament of claimants who may have difficulty in establishing a link between occupational exposure to carcinogens and the development of their cancers, I cannot accept that such a result would be fair to potential defendants who would be required to pay full damages in many cases in which occupational exposure had played a small part or, perhaps, no part at all."

68. Reflecting where principle and authority was leading, the final resting place of Mr Allan's submissions was that the deceased's exposure to each Defendant's asbestos fibres materially increased the risk of his developing lung cancer, and that the common law recognises an intermediate position lying between conventional principles and the fully-fledged Fairchild-exception. Mr Allan developed that submission by taking me through Lord Rodger's review of the post-1956 jurisprudence which he undertook in Fairchild (paragraph 130ff). It is clear from these later cases (cited in paragraph 36 above) that the House of Lords moved very quickly from the articulation of a conventional test – material contribution to the damage – to a more flexible one - material contribution to the risk. Mr Allan ended his submission at paragraph 133 of Lord Rodger's opinion, the text of which I have already set out.

69. Mr Allan also urged me to hold that the policy considerations alluded to by Swift J in Jones only applied to the issue of medical causation, and not to the issue of attribution of causation to one or more defendants.

70. I cannot accept Mr Allan's submissions on this issue. I agree with him to the extent that, as early as 1957, the House of Lords had shown a willingness to equate increase in the risk with causation on a conventional basis, but – at least for Mr Allan's purposes – that proves too much.

71. The answer to Mr Allan's submission is that as soon as one begins to depart from Lord Reid's conventional formulation, and contend that proof of material contribution to the damage is not required, one either arrives in the enclave of Fairchild or at zero recovery. One cannot stop, as Mr Allan did, at paragraph 133 of Lord Rodger's opinion. At paragraph 134 Lord Rodger commenced his analysis of McGhee and concluded that it was an example of a material increase in the risk case (*pace* subsequent attempts of the House of Lords – as mentioned in paragraphs 149 and 150 of Lord Rodger's judgment in Sienkiewicz - to explain it otherwise). I do not pause to mention other landmark decisions which Lord Rodger also reviewed (these were different causative agent cases), but in my view it is clear that the decision in Fairchild was based on McGhee – albeit clarifying that its application was confined to cases with very specific attributes. Those are the characteristics listed under paragraph 170 of Lord Rodger's opinion, which I need not set out.

72. Thus, in my judgment, there is no difference of any substance between (a) the brace of House of Lords cases decided in the wake of Bonnington Castings, (b) McGhee, and (c) Fairchild. They are all based on the concept of material increase in the risk, which is the very concept Mr Allan seeks to pray in aid. It follows that there is no intermediate ground for Mr Allan to occupy, only the metaphorical quicksand I mentioned earlier.

73. Even if, contrary to the above, Bonnington Castings should now somehow be regarded as a material increase in the risk case, that would not help Mr Allan. His argument would fail for the very same reason that it cannot be distinguished from Fairchild.

74. Accordingly, at Mr Allan's second stage, that of causal attribution as between Defendants, Bonnington Castings cannot help him in either of its guises. It cannot avail him in its application *stricto sensu* because, as previously explained, each Defendant has not materially contributed to the damage. In that regard, there is no difference between the first and second stages, which is the point which I understood Mr Platt to be making in his brief Reply. Nor can it help him in its modified form, because in that guise, whatever the nomenclature, the principle being relied on is the same as the Fairchild-extension.

75. There is an additional reason for rejecting Mr Allan's submission that, because medical causation may be proved here without reference to anything other than conventional common law principles, the present case may be treated in a different way from those cases where the Fairchild-extension must apply at the medical causation or "what" stage for the claim to get past first base. In my judgment, there is no category distinction between the two stages under discussion: rather, they are merely convenient ways of approaching the unitary concept of causation in cases of this type. Moving away from the circumstances of the instant case, causation in cases of this type must be proved against each defendant who has been sued; and, if the law chooses to relax the standard of proof because the case cannot be proved against a defendant on conventional grounds, then it does so on the looser basis that it is sufficient to prove material contribution to the risk. That looser principle may be required only at the defendant attribution stage; or it may be required at both stages. It matters not, because causation is an indivisible concept.

76. In any event, it is over-simplifying matters to state that medical causation has been proved in the instant case on the balance of probabilities. It depends on how the question is framed. The Claimant has demonstrated on the balance of probabilities that the deceased's lung cancer was not caused by non-occupational factors. However, if the question is re-framed in these terms – *has it been proved that any of the Defendants who have been sued caused the deceased's lung cancer?* – the answer must be in the negative.

77. I appreciate that the present case is not a mesothelioma case, but it is clear from an examination of the trilogy of cases - Fairchild, Barker and Sienkiewicz – that Professor Stapleton's two stages are extremely difficult to be kept apart.

78. One may test the point in this way. Take a hypothetical mesothelioma or lung cancer case where the entirety of the non-smoking claimant's occupational exposure was in three separate employments. In such a case, medical causation may be proved on conventional principles, without Fairchild. But, without the more benevolent rule the claim against the three separate defendants must fail. Or take another hypothetical mesothelioma case involving a claimant who smoked and was employed by a number of negligent employers not all of whom have been sued. Why should it make any difference to the outcome, I ask rhetorically, that such a claimant cannot prove medical causation without Fairchild? Or, take a hypothetical lung cancer case where the entirety of the non-smoking claimant's occupational exposure was in three

separate employments. Unless any individual defendant conferred the majority of the asbestos dose, the claim against each employer may only succeed by invoking the benevolent rule. Or, finally, take the facts of the instant case but remove from the equation the deceased's smoking habit. Again, the claim against each defendant may only succeed by relaxing the standard of proof.

79. On my understanding of his closing oral argument, Mr Allan adhered to the submission that mesothelioma and lung cancer cases should be treated in different ways. In my view, this submission may be addressed on three levels. First, only Lord Brown in Sienkiewicz held that the extended principle was limited to mesothelioma cases (see paragraph 186 of his Judgment). It follows that *stare decisis* does not preclude the application of the extended principle to lung cancer cases, provided of course that such cases satisfy the preconditions for the application of that principle. Secondly, in my judgment those preconditions are satisfied in lung cancer cases. These are the preconditions most clearly and helpfully set out at paragraph 170 of Lord Rodger's opinion in Fairchild. There may be slight differences in the formulations of Lord Bingham (paragraphs 2 and 34) and Lord Hoffmann (paragraph 61), but their preconditions are also satisfied. Lord Bingham's reference to mesothelioma related to the facts of the case before him; he was not intending to limit his formulation to that particular cancer.

80. If I am wrong about this very last point, it would not follow that Mr Allan is right and that the Claimant is entitled to full recovery. It would follow that the correct outcome would be zero recovery.

81. Thirdly, it should be made clear that this result does not depend on any conclusion that mesothelioma and lung cancer are indistinguishable in aetiological terms, which was the subsidiary case Mr Platt was seeking to run on the back of Dr Moore-Gillon's evidence. I have already explained why I consider that they are distinguishable. The result is arrived at for a different reason, namely that lung cancer and mesothelioma are legally indistinguishable.

82. Mr Allan conceded for the purposes of these proceedings but not any appeal, that should I conclude that the Fairchild-extension applies, the Claimant cannot avoid apportionment in view of Barker. In paragraph 48 of his opinion in Barker, Lord Hoffmann left open the mechanics of apportionment for subsequent judicial determination. Those mechanics have been agreed between the parties in the present case.

83. In my judgment, the policy arguments that Mr Allan urges me to reflect in my decision may carry very little weight. In any event, the very arguments which Swift J identified in Jones apply, it seems to me, with exactly the same force in the present context of defendant attribution as they did in the context of Jones, viz. medical causation. Indeed, this is the point Lord Phillips made in paragraph 105 of his judgment in Sienkiewicz. The upshot is identical: if Mr Allan were right, then the Fourth Defendant would have to pay damages on a 100% basis even if it were the only Defendant which could usefully have been sued (the remaining Defendants being uninsured, for example). That, to my mind, would not be a fair outcome. A proportionate recovery may not be a particularly principled one, in the sense that in an indivisible injury case such as the present principle would require full recovery; but it seems to me that over-reliance on principle may scarcely avail Mr Allan. After all,

adherence to the conventional common law approach, which is entirely principled, would lead to no recovery at all.

84. It follows that apportionment is the appropriate outcome in the present case, and the Claimant's recovery is limited to the sum of £61,600. There must be Judgment for the Claimant in that amount.