Dust Diseases Tribunal of New South Wales

CITATION :	**John William Booth v Amaca Pty Limited and Amaba Pty Limited [2010] NSWDDT 8**
PARTIES :	John William Booth (plaintiff) Amaca Pty Limited (first defendant) Amaba Pty Limited (second defendant)
MATTER NUMBER(S) :	8212 of 2008
JUDGMENT OF:	Curtis J at 1
CATCHWORDS:	DUST DISEASES TRIBUNAL - Negligence :- brake mechanics - foreseeability - causation - deduction of Dust Diseases Board payments from damages
LEGISLATION CITED:	s25B Dust Diseases Tribunal Act 1989 Workers Compensation (Dust Diseases) Act 1942 Workers Compensation Act 1987
CASES CITED:	HG v The Queen (1999) 197 CLR 414 Jones v Dunkel (1958 -1959) 101 CLR 298 EMI (Australia) Ltd v Bes [1970] 2 NSWLR 238 E.M. Baldwin & Son Pty Ltd v Plane (1998) 17 NSWCCR 434 Rose v Abbey Orchard Property Investments Pty Limited (1987) Aust.Torts Report 80-121 James Hardie and Coy Pty Limited v Roberts [1999] NSWCA 314 Bonnington Castings Ltd v Wardlaw [1956] AC 613 Amaca Pty Ltd v Ellis [2010] HCA 5 Workers Compensation (Dust Diseases) Board of New South Wales v Smith, Monro and Seymour [2010] NSWCA 19 Seltsam Pty Ltd v McNeill [2006] NSWCA 158 Lo Presti v Ford Motor Company of Australia Ltd [No 2] [2008] WASC 12 NSW Sugar Milling Cooperative Limited v Manning (1998) 44 NSWLR 442 Julia Farr Services Inc v Hayes [2003] NSWCA 37 Bendix Mintex v Barnes (1997) 42 NSWLR 307
DATES OF HEARING:	22, 23, 25 and 26 February 2010; 1, 2 3 4 and 5 March 2010

and 6 May 2010

DATE OF JUDGMENT: 10 May 2010

LEGAL REPRESENTATIVES: Mr P C B Semmler QC with Mr S Tzouganatos instructed by Turner Freeman appeared for the plaintiff

Mr G M Watson SC with Mr J C Sheller instructed by Holman Webb Brisbane appeared for Amaca Pty Limited
Mr G M Watson SC instructed by DLA Phillips Fox appeared for Amaba Pty Limited

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JUDGMENT:

Dust Diseases Tribunal of New South Wales

Matter Number 8212 of 2008

John William Booth

v

Amaca Pty Limited

and

Amaba Pty Limited

10 May 2010

CURTIS J
JUDGMENT

1. The plaintiff, Mr Booth, has contracted malignant pleural mesothelioma as a result of asbestos inhalation. With the exception of three years between 1969 and 1971, Mr Booth worked between 1953 and 1983 as a motor mechanic.

2. In the course of this work Mr Booth regularly liberated asbestos dust from Hardie-Bestos and Hardie-Ferodo brake linings manufactured by Amaca Pty Limited (Amaca) between 1953 and 1962, and thereafter by Amaba Pty Limited (Amaba).

3. Mr Booth asserts that the asbestos fibres released from these products materially contributed to the contraction of his disease and claims damages from each of them.

4. The defendants each contend that Mr Booth's mesothelioma did not result from asbestos fibres emanating from its product, and that if it did, because the injury was not reasonably foreseeable, they owed no duty to Mr Booth.

Mr Booth's Exposure to Asbestos

Domestic

5. In 1943, when Mr Booth was about eight years old, his father built a two-bedroom home and a boat shed at Lugarno. The sides of the house were clad with a combination of weatherboard to a height of 4 feet, and a further 4 feet with asbestos cement. The eaves were also asbestos cement. The boat shed was clad with asbestos cement.

6. Mr Booth recalls that about 20 sheets in total were used, but only about six sheets had to be cut. He said that the work with fibro took five or six hours, although the cutting took no more than about half an hour, and that: *"as an eight-year-old you would be sent off to play and get out of the road"*. He said that "*sometimes I held the sheeting down with my hands while he used fibro cutters"*.

7. In about 1953, when Mr Booth was about 16 years old, he helped his father build a fibro garage at Peakhurst. The work occupied two days, with fibro sheeting work performed on one day. About 10 flat sheets were used, of which only three sheets were cut for the door and windows. The cutting, with fibro cutters, was done by Mr Booth's father while he held the sheets in place. Mr Booth then held the sheets against the timber frame while his father nailed them in place.

8. Mr Booth can't recall seeing any dust being generated from the fibro cutters, which, he said *"simply punched the sheet"*.

Occupational as a Driver

9. For some months in 1959 Mr Booth worked as a truck driver. On one day he spent 20 minutes helping load hessian bags of asbestos on to his truck at the Sydney wharves. He then drove to a factory at Camellia, where the truck was unloaded by others.

Occupational as a Mechanic

10. Between February 1953 and April 1954 Mr Booth worked as an apprentice mechanic with Banksia Modern Motors. He replaced two sets of brake linings a month, on average, under the supervision of a mechanic. He was present within the workshop when the mechanic worked on other brake linings.

11. Between April 1954 and November 1957 Mr Booth was employed by Saville Brothers at Penshurst. He replaced the brake linings on at least two cars each week, sometimes more. Again he worked in close proximity to other mechanics performing brake relining. Brake work took place within the workshop every day.

12. From November 1957 until the end of 1958 Mr Booth was employed by the Paragon service station at Bexley. He replaced one set of brake linings each week on average, as did four other mechanics within the workshop.

13. For a period of about six to eight months from January 1959 Mr Booth worked in the Postmaster General's workshop at St Peter's. He worked on trucks and averaged two sets of brake relinings each week. Within the workshop brake linings were replaced by other mechanics every day.

14. From early 1960 until 1964 Mr Booth worked at the Mascot service station, Rosebery. He

performed brake relining on one car each month, and in addition on one or two wheels of damaged vehicles approximately once each fortnight.

15. Between 1964 and 1968 Mr Booth was employed by N. B. Love at Enfield. On average he replaced the brake linings on one or two trucks and two to three cars each week. Other mechanics worked more regularly than he on brake linings, and work was performed on brake linings within the workshop every day.

16. Mr Booth did not work as a mechanic between November 1969 and August 1972.

17. Between 1972 and August 1978 he worked for N.B. Love in Western Australia. He estimates that he worked on about two to three cars and one or two trucks each week replacing at least two to three sets of brake linings each week. Other mechanics in the workshop replaced brake linings every second day.

18. Between August 1978 and September 1983 Mr Booth was self-employed as a mechanic. On average he replaced about two sets of brake linings each week. His employees also did this work within the workshop, more frequently than Mr Booth. Approximately 3 times each year Mr Booth offered free brake inspections and these offers generated hundreds of brake lining jobs.

Fibre Burden

19. Asbestos fibre was liberated from new brake linings in three ways. First, by Mr Booth using a hammer to punch a rivet through the brake lining so as to fix it to the metal shoe. The holes provided in the brake lining for the rivets did not line up exactly with the holes in the shoe. Secondly, by Mr Booth using an electric drill to ream out the holes for the rivets, when the misalignment was too great to allow the rivet to be forced through with a punch. Thirdly, by grinding the leading edge of the brake lining on a bench grinder to ensure smooth operation of the brakes; this was a very dusty process.

20. The liberated asbestos collected upon Mr Booth's clothes, the work-bench, and floor of the workshop. It was re-agitated into the atmosphere by brooms, passing feet, and the use of compressed air to clean the workbench.

21. Mr Booth said that it took four hours to replace the linings on a passenger vehicle, and up to three hours per wheel to replace the linings on commercial trucks.

Causation

The Plaintiff's Case

22. The following matters are not in dispute:

(1) The plaintiff's mesothelioma was caused by the inhalation of asbestos fibre.

(2) Chrysotile asbestos has the capacity to cause mesothelioma.

(3) The brake linings manufactured by the defendants contained chrysotile asbestos.

(4) Mr Booth inhaled chrysotile asbestos fibres liberated from the defendants' products.

23. The plaintiff relies upon the expert medical opinions of Professor Douglas Henderson, Dr James Leigh, Dr Morris Heiner, and Professor William Musk, that all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma in a particular individual, and that Mr Booth's exposure to chrysotile in the course of his work as a mechanic materially contributed to the cause of his disease.

24. Professor Henderson, Dr Heiner and Professor Musk have each experienced cases of mesothelioma in their respective practices where the only identified exposure derived from working with brake linings.

Professor Henderson

25. Professor Henderson explained the mechanical and chemical steps by which, in his opinion, the accumulation of asbestos fibres cause mesothelioma in these terms:

> *When there are multiple episodes of asbestos exposures, and the individual concerned inhales increasing number of fibres on different occasions, that contributes to the total burden of asbestos fibres deposited in the lung, and translocated to the pleura and is thought that mesothelioma develops because of an inter-action between the asbestos fibres and the mesothelial cells by way of secondary chemical messages.*
>
> *And to simplify the answer, the point is that the more fibres there are, the greater number of fibres there will be interacting with the mesothelial cells which themselves undergo proliferation. And so the progress goes on, with increasing numbers of mesothelial cells interacting with increasing number of fibres, so that the ultimate development of mesothelioma, and its probability of development, will be influenced by the number of fibres interacting with mesothelial cells over multiple periods of time and probably over multiple different generations of mesothelial cells.*
>
> *And I think this is a fairly well accepted model now and it flies in the face of what used to be called the one fibre hypothesis, that mesothelioma came about from a single fibre interacting with a single mesothelial cell which in biological terms is a ridiculous proposition.*

26. Professor Henderson said that while the science was incomplete, and some uncertainty remained as to the causative links at a cellular level, *"It is, I think, almost universally accepted that all asbestos exposure, both recalled and unrecalled, will contribute causally towards the ultimate development of a mesothelioma".*

Professor Musk

27. Although at times Professor Musk spoke in terms of cumulative exposure to asbestos increasing the *risk* of contracting mesothelioma, he did not in cross-examination resile from his evidence that, where a mesothelioma has occurred, all exposure has materially contributed to the development of that mesothelioma, and that this was so in the case of Mr Booth.

28. Professor Musk said that: *"The relationship between the inhalation of asbestos and the development of mesothelioma is so consistent that it is accepted as the causative relationship".* And *"It's a proven risk - and most people, as far as I know, are prepared to say that it's a causative association."*

29. In cross-examination Professor Musk agreed that if asbestos were a "complete carcinogen", a single incident of inhaling asbestos may explain a mesothelioma diagnosed 30 or 40 years later in the absence of further exposure. He did however add that while it may not require any further exposure, the risk increases as the dose of asbestos inhaled increases.

30. This evidence, relating to a theoretical scenario, is not inconsistent with acceptance of Professor Musk's opinion that if there *were* further exposure then that exposure would have materially contributed to the development of the mesothelioma.

Dr Heiner

31. Dr Heiner says that causation in cases of mesothelioma is best explained by total cumulative asbestos exposure because there is no threshold dose below which mesothelioma will not occur, and the incidence of mesothelioma increases with cumulative dose.

32. He said that the mesothelioma resulted from migration of inhaled asbestos fibres to the pleural surface where they activated various chemicals to carcinogenic effect.

33. Dr Heiner also agreed in cross-examination that a single incident of inhaling asbestos may explain a mesothelioma diagnosed 30 or 40 years later in the absence of further exposure. He did however add that: *The majority of people who inhale asbestos fibres do not develop mesothelioma, so I think it's drawing a long bow to say that if he had only had that experience when he was eight years old, he was going to develop mesothelioma, I think it's being adventurous to say that.*

Dr Leigh

34. Dr Leigh explained the biological mechanisms by which, in his opinion, the accumulation of asbestos fibres causes mesothelioma. Echoing the opinion of Professor Henderson, Dr Leigh said that: "*the current consensus view is that asbestos is involved in both the initiation phase and the promotion/proliferation phase of mesothelioma tumour development"*.

35. It is because of this capacity of asbestos fibres to be involved at several stages of tumour development that Dr Leigh considers that, in an individual case, all cumulative exposure to asbestos fibre must play some part in causation.

36. Although Dr Leigh at times used the word *"risk"* interchangeably with *"cause"* in his evidence, he explained that once the disease had occurred, the accumulating risk had come home, and that it was the accumulation of fibres that caused the disease in the particular case.

37. In cross-examination Dr Leigh agreed that, *if* there had been no other exposure, the childhood exposure, or the exposure as a truck driver, either separately or in combination was sufficient to cause Mr Booth's mesothelioma. He further agreed that he could not say that, because of this earlier exposure, Mr Booth would not have contracted mesothelioma in the absence of the work on asbestos brake linings.

38. These answers do not detract from Dr Leigh's opinion that the work on the brake linings materially contributed to Mr Booth's mesothelioma. The questions and the answers were directed at a purely hypothetical scenario which postulated that there was no further occupational exposure.

The Defendant's Case

39. Mr Watson SC for the defendants argues that:

> (1) Because the biological process whereby the inhaling of asbestos causes mesothelioma is incompletely understood, medical science cannot support the proposition advanced by the plaintiff's expert witnesses that all asbestos inhaled materially contributes to the causes of mesothelioma. Such expressions of opinion are inadmissible because they are not based upon that which is "*recognised to be accepted as a reliable body of knowledge"* (*HG v The Queen* (1999) 197 CLR 414 per Gaudron J at [58]).
>
> (2) It is highly unlikely that Mr Booth's work on brake linings caused his mesothelioma because: (a) chrysotile is unlikely to produce mesothelioma, (b) brake linings contain resins which adhere to the chrysotile fibres rendering them non-respirable, (c) the handling of brake parts produces asbestos fibres so short that they are harmless, and (d) the number of chrysotile fibres produced by the handling processes are so few that the effect is harmless.
>
> (3) The only epidemiological studies admitted into evidence demonstrate that brake work does not increase the risk of mesothelioma.
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> (4) When quantified, Mr Booth's cumulative exposure to the products of either Amaca or Amaba is insignificant in comparison to his background and other exposures, and made no material contribution to the causes of his mesothelioma.

The Limitations of Medical Science

40. I reject the submission that medical science cannot support the proposition that all asbestos inhaled materially contributes to the causes of mesothelioma.

41. Professor Henderson is an eminent pathologist. Pathology, he said, is "*the study of disease processes in the broadest sense, including their causes, mechanisms of development, characteristics once they have developed, and their natural history."*

42. Professor Henderson's professional and academic interests over the last 35 years have concentrated upon asbestos related disorders. He is the senior editor and a major contributor to the textbook "Malignant Mesothelioma" published in New York in 1992.

43. Dr James Leigh is an occupational physician who holds a PhD in that discipline. He has been a researcher in the field of asbestos related diseases for almost 30 years.

44. Both Dr Heiner and Professor Musk are Respiratory Physicians with extensive clinical experience with mesothelioma.

45. In support of his first submission Mr Watson relies upon the following passage from the judgment of Dixon CJ in *Jones v Dunkel* (1958-1959) 101 CLR 298 at 305:

> *But in any case we are not concerned with a choice among rival conjectures. In an action of negligence for death or personal injuries the plaintiff must fail unless he offers evidence supporting some positive inference implying negligence and it must be an inference which arises as an affirmative conclusion from the circumstances proved in evidence and one which they establish to the reasonable satisfaction of a judicial mind. It is true that "you need only circumstances raising a more probable inference in favour of what is alleged". But "they must do more than give rise to*

> *conflicting inferences of equal degree of probability so that the choice between them is mere matter of conjecture". These phrases are taken from an unreported judgment of this Court in Bradshaw v. McEwans Pty. Ltd. (Unreported, delivered 27th April 1951). which is referred to in Holloway v. McFeeters* [1956] HCA 25; (1956) 94 CLR 470*, by Williams, Webb and Taylor JJ. The passage continues: "All that is necessary is that according to the course of common experience the more probable inference from the circumstances that sufficiently appear by evidence or admission, left unexplained, should be that the injury arose from the defendant's negligence. By more probable is meant no more than that upon a balance of probabilities such an inference might reasonably be considered to have some greater degree of likelihood."* (at pp 480, 481) *But the law which this passage attempts to explain does not authorise a court to choose between guesses, where the possibilities are not unlimited, on the ground that one guess seems more likely than another or the others. The facts proved must form a reasonable basis for a definite conclusion affirmatively drawn of the truth of which the tribunal of fact may reasonably be satisfied. (at p305)*

46. In his paper *Science and Judicial Proceedings: Seventy-Six Years on* Published in 2010 84 ALJ 244, Chief Justice Robert French, discussing an address by Sir Owen Dixon on *Science and Judicial Proceedings* delivered to the Medico-Legal Society of Victoria on 13 September 1933, observed that:

> *Today medical science is informed in both theory and its practical application by many other disciplines and sub-disciplines including physics, chemistry and molecular biology. Its diagnostic tools use concepts and techniques undreamed of in 1933. There are, however, many elements of medical judgments which are still probabalistic in their character. Biological systems are so complex and affected by so many factors that predictions about their behaviour or inferences about their status often have to be expressed in terms of probabilities.*

47. Mr Watson contends that, because Professor Henderson, Dr Leigh, Dr Heiner and Professor Musk each concede that the biological process of causation is incompletely understood, and because several alternative and competing theories of causation have been canvassed in the evidence, the proposition that all fibres contribute to the development of the mesothelioma is merely a guess, and not a legitimate inference.

48. He suggests that several competing theories of causation emerge from the evidence. These are said to be the single fibre theory; the mechanical theory based on the physical properties of asbestos; the chemical theory; and the complete carcinogen theory.

49. The evidence does not support this submission.

50. No witness accepted the single fibre theory. Professor Henderson rejected it as *ridiculous,* and Dr Leigh as *silly.* Professor Musk conceded that, depending upon fibre type, a single incident of inhaling asbestos of more than a minimal dose may explain the development of a mesothelioma 30 to 40 years later. He did add that: "*it may not* ***require*** *any further exposure, but the risk increases as the dose of asbestos that is inhaled increases".* (Emphasis added).

51. Because the defendants call no medical evidence, it is unnecessary to review in further detail the evidence from the plaintiff's medical witnesses. I think it sufficient to observe that the mechanical theory and the chemical theory of causation are not competing but complementary, and that the complete carcinogen theory is not incompatible with the proposition that all exposure materially contributes to the development of the mesothelioma.

52. The plaintiff's experts, conceding that some of the steps necessary to form the opinion on purely deductive and scientific grounds are yet to be discovered, are each of the opinion that all asbestos fibres contribute to the development of a mesothelioma. They adopt that theory as most probably according with the actual aetiology of the disease. Their conclusions are not guesses, but reasonable inferences drawn from the current state of medical knowledge.

53. Ultimately it is for the Tribunal to draw the necessary inference from the established facts with the assistance of medical evidence.

54. In *EMI (Australia) Ltd v Bes* [1970] 2 NSWLR 238 at 242, Heron CJ said:

> *Medical science may say in an individual case that there is no possible connection between the events and the death, in which case, of course, if the facts stand outside an area in which common experience can be the touchstone, then the judge cannot act as if there were a connection. But if medical science is prepared to say that it is a possible view, then, in my opinion, the judge, after examining the lay evidence, may decide that it is probable. It is only when medical science denies that there is any such connection that the judge is not entitled in such a case to act on his own intuitive reasoning. It may be, and probably is, the case that medical science will find a possibility not good enough on which to base a scientific deduction, but courts are always concerned to reach a decision on probability and it is no answer, it seems to me, that no medical witness states with certainty the very issue which the judge himself has to try.*

55. Uninstructed by medical evidence, I would not regard Mr Watson's suggestion that mesothelioma may be contracted by exposure to a threshold dose, with further exposure irrelevant, as consistent with the epidemiological evidence that the incidence of mesothelioma increases in proportion to increased dose.

56. Because most people exposed to asbestos fibres do not contract mesothelioma, it is thought that persons contracting the disease suffer from some underlying genetic susceptibility.

57. If the threshold dose were sufficient to explain the contraction of the disease in those persons, then increasing doses should yield few further cases of mesothelioma. The fact that the incidence rises with increasing dose suggests that the further exposure plays a causative part in the aetiology of the disease.

58. In *E.M. Baldwin & Son Pty Ltd v Plane* (1998) 17 NSWCCR 434 the Court of Appeal considered the evidence of Professor Henderson concerning the cumulative process by which asbestos fibres liberated from locomotive brake blocks caused mesothelioma. At [89], Fitzgerald AJA, (with whom Meagher JA and Beazley JA agreed) said this:

> *…Jsekarb's submission that the Tribunal had no basis for acceptance of Professor Henderson's "cumulative effect" was wrong. There was no contrary view proffered by an appropriate expert with Professor Henderson's qualifications and expertise, and his opinion was based on his experience, related to published statistics, information (e.g. the Australian Mesothelioma Register) and opinions, logically defensible, and at least to some extent, supported by other experts.*[94]
>
> *…*
>
> *Professor Henderson was undoubtedly the pre-eminent expert witness on the aetiology or pathogenesis of mesothelioma*

> *... the Tribunal was entitled, and, in my opinion, correct, to accept Professor Henderson's "cumulative effect" theory, and to reject Jsekarb's fundamental proposition that inhalation of more than one amphibole form of asbestos fibre raises a number of separate, independent, possible causes of mesothelioma.*[95]

59. At issue between the parties in this case is the proposition that all exposure to chrysotile asbestos, other than trivial or *de minimis* exposure, that occurred in a latency period of between 26 and 56 years, materially contributed to the cause of Mr Booth's mesothelioma. I resolve that issue in favour of the plaintiff.

60. Over 10 years have elapsed since the decision in *E.M. Baldwin & Son Pty Ltd v Plane*, yet the defendants in this case call no medical evidence to suggest that the "*cumulative effect"* theory may not be accepted because of further advances in medical knowledge.

61. His Honour the President in *(Re Jones-Mashman) Amaca Pty Ltd v CSR Ltd No 2* [2009] NSWDDT 24 (unreported) determined, for the purpose of **s25B** of the *Dust Diseases Tribunal Act 1989,* that all asbestos exposure within an acceptable latency period makes a material contribution to the cause of mesothelioma.

62. Upon the facts in this case I specifically determine for the purpose of **s25B** that all exposures to chrysotile asbestos, other than trivial or *de minimis* exposure, occurring in a latency period of between 25 and 56 years, materially contributes to the cause of mesothelioma.

Improbability of the Causal Mechanism

63. I reject each of these submissions. It has been demonstrated by epidemiological studies, and it is admitted by each defendant in this case that chrysotile can cause mesothelioma. The mere fact that chrysotile fibres are less potent than amphibole fibres does not assist the resolution of causation in this case where exposure to amphibole fibres is taken into account by the medical and industrial experts.

64. Although Professor Berry, the defendants' expert epidemiologist, said that a 1983 study of 13,500 workers in a manufacturing plant producing friction materials with chrysotile asbestos detected no increased incidence of mesotheliomas, he did not argue that this study proved that chrysotile did not cause mesothelioma. Epidemiology draws its conclusions from studies of large populations.

65. The evidence is entirely to the contrary of the proposition that, because some chrysotile fibres liberated from brake linings remained bound in resin, the balance of fibres are non respirable.

66. There is accumulating evidence, to which I will later refer, that short fibres may produce lung disease.

Epidemiological Evidence

67. Epidemiological studies have demonstrated that exposure to chrysotile asbestos increases the likelihood of contracting mesothelioma. A 1998 World Health Organisation monograph on chrysotile, entitled *Environmental Health Criteria 203: Chrysotile Asbestos* states expressly that chrysotile asbestos poses increased risks for mesothelioma, and that no threshold of exposure had been established. A paper published by the International Agency for Research on Cancer published in 1987 is to the same effect.

68. In the course of the trial counsel for the defendants conceded that the question of the capacity of chrysotile to cause mesothelioma was not in issue. (Transcript 210.7)

69. The authoritative paper by Hodgson and Darnton, *The Qualitative Risks of Mesothelioma and Lung Cancer In Relation to Asbestos Exposure,* published in the *Annals of Occupational Hygiene 2000; 44:565-601* reported that a cumulative exposure of 1 fibre/ml/years to chrysotile asbestos yields a lifetime "best estimate" risk of about 5/100,000 mesothelioma deaths.

70. The defendants rely upon 19 epidemiological studies published in the international peer reviewed literature upon the incidence of mesothelioma in automotive mechanics. Three meta-analyses, by Wong, Goodman *et al*, and Laden *et al* have combined the results from several studies to produce what is said to be a more precise estimate of the risk.

71. Wong concluded that: "*... clearly there is no evidence to support or even to suggest an association between an increased risk of mesothelioma and exposure to brake linings On the contrary garage mechanics mesothelioma risk is similar to that in the general population. None of the six studies reported any increased mesothelioma risk among auto mechanics".*

72. Goodman *et al* stated that the epidemiological data showed that: *"Employment as a motor mechanic does not increase the risk of developing mesothelioma".*

73. Laden *et al* stated that "*... the consistent pattern that emerges from this research indicates no increase of risk of... mesothelioma*, and that *"automobile mechanics are not at an elevated risk of... mesothelioma from their occupation."*

74. Professor Berry, commenting on the analyses of Wong and Goodman, said that the studies were sufficient to demonstrate that there was no large increase in the risk of automotive mechanics contracting mesothelioma from exposure to asbestos fibres liberated from brake linings, but that they could not demonstrate that there was no small increase in risk. He did not refer to the paper by Laden *et al.*

75. One obvious criticism of the studies combined into these meta-analyses is that the cohort studied were variously described as "motor mechanics", "garage workers", and "vehicle mechanics". In the same manner as persons described as "carpenters" may have been heavily exposed to asbestos fibres released from asbestos cement, or not at all exposed, the average exposures of "motor mechanics" may have little in common with the particular exposure of Mr Booth.

76. Dr Leigh, who is trained in epidemiology, gave cogent evidence criticising many of these studies upon the basis of weak case reference design, and low statistical power. He said that the standard text by Breslow and Day, *Statistical Methods in Cancer Research* Volume 2, table 7.7 required a power of 80 per cent before a study may be declared negative, and that none of the studies upon which the defendants rely achieved that power.

77. Specifically the power of the study by Welsh to detect a relative risk of 1.5 was 9 per cent. The power of the study by McDonald to detect a relative risk of 1.5 was 18 per cent. The power of the study by Woitowitz to detect a relative risk of 1.5 was in the order of 37 per cent; the power of that study to detect a relative risk of 1.2 was only 8 per cent.

78. At a relative risk of 1.2, for every 10 persons who may contract mesothelioma from background risks, an additional 2 will contract the disease because of occupational exposure as an automotive mechanic. The statement by Wong that: *there is no evidence to support or even to suggest an association between an increased risk of mesothelioma and exposure to brake linings* is correct but misleading.

79. Professor Berry said of the Wong paper that; *"... one can say with 95 per cent confidence from this study that a relative risk bigger than 1.23 is excluded but the relative risk between one and 1.23 is a possibility".*

80. Professor Berry also said that the study by Goodman was not capable of detecting small increases in risk. The unqualified statement by the authors that the epidemiological data showed that: *"Employment as a motor mechanic does not increase the risk of developing mesothelioma"* is not justified by the data.

81. Dr Leigh argues that much of the data does support a strong correlation between exposure to asbestos as an auto mechanic and the contraction of mesothelioma. I do not propose to canvass his evidence on this matter, nor do I think it necessary to repeat the persuasive detail contained in the thorough written submissions prepared by counsel for Mr Booth.

82. I am not persuaded that the epidemiological evidence specific to automotive mechanics is adverse to the submission that causation has been proved in this particular case.

Quantification of Exposure

83. Professor Berry agreed that there was no threshold of exposure to asbestos below which mesothelioma could not be contracted, and with the proposition that; *"there are some very small risks but given enough people some of those risks will come home".*

84. Without reliance upon the controversial epidemiological studies canvassed above, Professor Berry chose to express his opinion upon the basis of accepting the quantification by the defendants' occupational hygienists, Mr Pickford and Mr Rogers, of Mr Booth's reported exposure, and comparing that fibre burden with conclusions drawn from general epidemiological studies.

85. Professor Berry's assumptions are set out in the following table.

	Asbestos Type	**Pickford**	**Rogers**
1) Home Renovations	Mixed	0.0017 f/ml/yrs	0.001 f/ml/yrs
2) Brake Repair	Chrysotile	0.26 f/ml/yrs	0.18 f/ml/yrs
3) Loading Trucks	Chrysotile and/or amosite and/or crocidolite	0.23 f/ml/yrs	0.02 f/ml/yrs

86. The lifetime risks posed by inhalation of asbestos fibre depend upon the relative causal potency of the asbestos fibres chrysotile, amosite and crocidolite, and also upon the time lapse since the relevant exposure. Professor Berry calculated the relative risks of Mr Booth's several exposures in accordance with the data collected by Hodgson and Darnton.

87. The composition of the asbestos cement to which Mr Booth was exposed as a child is unknown. Dr Berry prepared his risk analysis upon the alternatives of 50 per cent chrysotile admixed with 50 per cent amosite, and 50 per cent chrysotile admixed with 50 per cent crocidolite.

88. Similarly the composition of the asbestos fibre that Mr Booth loaded onto trucks is unknown, and Professor Berry has factored in the three possibilities of chrysotile, amosite and crocidolite.

89. Professor Berry summarised his results in the following table:

Lifetime Risks of Mesothelioma per Million

	Brake Repairs	Home Renovations		Loading Trucks	
Pickford's Estimates	18	50% amosite	15	chrysotile	29
		50% crocidolite	93	amosite	550
				crocidolite	3,850
Rogers' Estimates	13	50% amosite	10	chrysotile	5
		50% crocidolite	62	amosite	90
				crocidolite	610

90. This table takes into account the greater potency of the earlier exposures.

91. Upon assumptions that the asbestos in the home renovations was 50 per cent amosite and 50 per cent chrysotile, and that the asbestos loaded onto trucks was chrysotile; the lifetime risks created by the accumulation of brake exposure, home renovations, and loading trucks were 28 per million based on Mr Rogers' estimate of exposure, and 62 per million based on Mr Pickford's estimate. Of these risks the brake repair work contributes 46 per cent in accordance with Mr Rogers' estimate, and 29 per cent in accordance with Mr Pickford's assessment.

92. Upon the assumptions that the asbestos in the home renovations was 50 per cent chrysotile and 50 percent crocidolite; and the asbestos loaded onto the trucks was crocidolite, the brake work contributed 2 per cent on Mr Rogers' estimate, and 0.5 per cent on Mr Pickford's estimate, to Mr Booth's lifetime risk of mesothelioma.

93. Professor Berry expresses his conclusions in terms of risk. Because asbestos fibres act cumulatively to cause mesothelioma, those calculations, based on the observed incidence of mesothelioma in populations exposed to increasing fibre/ml/years of exposure, may be taken as estimates of causal contribution.

94. As Professor Berry says in his report, his conclusions depend upon the assumptions which he is asked to make.

The Estimates of Cumulative Exposure

95. Mr Gordon Stewart, the plaintiff's occupational hygienist, readily admitted that retrospective assessments of cumulative exposure were "*not really a valid way of getting at what the exposure was"*. Professor Henderson said that it was almost impossible to reconstruct with any degree of numerical accuracy a quantitative assessment of cumulative exposure in a workplace environment where there was no practice of systematic measurement. Professor Berry noted that he had, in other cases, seen widely differing estimates of cumulative exposure from occupational hygienists, and that "*sometimes there are differences of say tenfold"*.

96. In the present case Mr Pickford's estimate of home renovation exposure was nearly double that of Mr Rogers. His initial estimate of the exposure loading trucks, on similar assumptions, was 11 times greater than that of Mr Rogers. These differences do not engender confidence in the accuracy or utility of the exercise.

Mr Pickford's evidence

97. I accept Mr Pickford's expertise in the estimation of fibre concentration, however an examination of his report reveals some unfairness in the application of that expertise.

98. After cross-examination, Mr Pickford prepared revised estimates of Mr Booth's cumulative exposure to accord more closely with the evidence of Mr Booth in respect to his experiences both in the automotive workshops and loading raw asbestos onto trucks. Mr Pickford's initial estimates had made no allowance in respect of exposure to background asbestos fibres in the workshop generated by the work of others, and by cleaning, and he had been misinformed as to the duration of the work loading asbestos at the wharves.

99. The revised estimates were as follows:

Home Renovations	0.0017 f/ml/years
Loading Trucks	0.016 f/ml/years
Brake Work	0.442 f/ml/years
Total	**0.4597 f/ml/years**

100. These estimates reduce by 93 per cent the previous estimate of exposure to asbestos when loading trucks, and increase by 70 per cent the previous estimate relating to brake work.

101. Mr Pickford's estimates should be further adjusted.

Exposure loading trucks

102. The evidence of Mr Booth, which was unchallenged, was this:

> *The company carried general goods but one of the jobs I did for him was carting hessian bags full of asbestos from the Sydney waterfront to a factory at Camellia. I did that on one occasion. Each of the bags came off a loader and I took each bag over my shoulder and stacked it on pallets on the back of the truck. Some of the hessian bags had holes in them and dust came out. Dust got onto my clothes as I loaded the bags. There were six pallets on the back of my truck and I loaded six bags on each pallet. It took me about 20 minutes to load the bags with the assistance of another driver who was waiting for his loads. I then drove to a factory yard in Camellia. I got out, untied the ropes and then workers at the factory used a forklift to remove the pallets from the back of my truck. This took about 10 minutes. I was not involved in the unloading process."*

103. In his initial report Mr Pickford said that he estimated airborne asbestos concentrations when loading trucks at between 70 fibres/ml and 100 fibres/ml for the purpose of applying the Monte Carlo mathematical simulation.

104. The Monte Carlo simulation, Mr Pickford explains, is a mathematical construct pursuant to which a computer model is programmed with alternative assumptions described as Scenarios A and B, Scenario A being "worst-case" and Scenario B being "typical or more likely". The various assumptions are randomly multiplied together many thousands of times so as to produce an estimated cumulative exposure at a certain level of confidence.

105. The application of this model is not consistent with legal reasoning. It proceeds upon the basis that probable conclusions as to past exposure are reduced by other possibilities. Upon the

authority of *Malec v J C Hutton*, that is a process which is permissible in relation to future contingencies. It is not permissible in relation to the past, where a fact found to be probable is a certain fact for the purpose of attaching legal significance.

106. It is apparent from Mr Pickford's report that in the application of the Monte Carlo simulation his "*more likely"* estimates of fibre burden have been tempered by assumptions acknowledged to be improbable.

107. By simple calculation, Mr Pickford's revised estimate of an accumulated 0.016 fibre/ml/years accrued over 20 minutes accords with an estimated airborne concentration of 94 fibres/ml. (For the purpose of expressing accumulated fibre doses in terms of fibre/ml years, there are 1920 hours in the standard year. 0.33 hours x 94 fibres/ml 1920 hours = 0.016 fibre/ml/years.)

108. Mr Pickford could refer to only one direct measurement of the fibre concentration when carrying hessian bags of asbestos across the shoulders. This was in a report prepared at an asbestos cement factory in 1971 by a Mr T J Armstrong who reported that the asbestos concentration when carrying hessian bags of chrysotile asbestos across the shoulder was 5-7 fibres/ml.

109. Because measurement conducted at a James Hardie asbestos cement factory on identical mixing processes showed that the generated airborne asbestos concentrations of amosite were generally 5 to 10 times greater than chrysotile, Mr Pickford multiplied the estimate from the Armstrong report by 5, so as to estimate that the concentration of fibres if the bags carried amosite was 25-35 fibres/ml. He then, notwithstanding that Mr Booth said that only *some* of the hessian bags had holes in them, multiplied those figures by a further factor of 5 to reflect what he regarded as a probability that *"every bag or up to 1 in every 3 bags was broken or badly pierced",* giving a concentration of between 125 fibre/ml and 175 fibre/ml.

110. This reasoning is specious. An extensive survey was conducted into the handling of imported raw asbestos fibre by the Commonwealth Department of Health in 1971. This included exposure monitoring by the New South Wales Division of Industrial Hygiene.

111. Dust levels between 1 and 18 fibres/ml, with mean values around 3 to 10 fibres/ml, were recorded when stacking unlined hessian bags onto pallets. Mr Rogers, who referred to this data in his report, adopted a mean exposure for truck loading of 5 fibres/ml.

112. By some calculation, the workings of which are not revealed in his report, Mr Pickford has factored into his Monte Carlo simulation assumptions that Mr Booth may have variously carried bags of chrysotile, amosite and crocidolite in some unstated proportions so as to generate a probability that his average exposure was to a concentration of 94 fibre/ml.

113. In the same report Mr Pickford states that around 95 per cent of asbestos used in Australia was chrysotile asbestos. Mr Pickford calculated that in accordance with the 95 per cent probability that the asbestos was chrysotile and 2.5 per cent each amosite and crocidolite, the airborne concentrations of fibre would have been 95% 7/fibresml (chrysotile) + 2.5% 17 fibres/ml (crocidolite) + 2.5% 35 fibres/ml (amosite) = 8.0 fibres/ml. Even if that figure were to be multiplied by 5, to account for the bags being broken, the resulting 40 fibres/ml is less than half the figure adopted by Mr Pickford to calculate the accumulated dose.

114. I find, because it is more probable than not, that the asbestos fibre which Mr Booth loaded onto trucks was chrysotile. (See *Rose v Abbey Orchard Property Investments Pty Limited* (1987) Aust. Torts Reports 80-121, *James Hardie and Coy Pty Limited v Roberts* [1999] NSWCA 314 (13 September 1999)). For present purposes I accept Mr Armstrong's highest

figure of 7 fibres/ml as the probable exposure.

115. Such a concentration reduces Mr Pickford's cumulative estimate of the fibre dose from 0.016 fibre/ml/years to 7 94 0.016 = 0.0012 fibre/ml/years. This is 0.5 per cent of Mr Pickford's original estimate of 0.235 fibre/ml/years.

Exposure in Home Renovations

116. Mr Pickford made the following assumptions for his Scenario A:

> (1) That Mr Booth, as an eight-year-old child, for a period of five hours, held asbestos cement sheets while his father cut them with fibro cutters, and that during this time he was exposed to an asbestos concentration of 0.4 fibres/ml.
>
> (2) That Mr Booth was additionally exposed for a period of 0.6 hours during the actual work with fibro cutters, and during this time he was exposed to an asbestos concentration of 0.05 fibres/ml.
>
> (3) That Mr Booth when he was 16 years old, for a period of two days, each of eight hours, held asbestos cement sheets against the timber frame of a garage while his father nailed them into place .

117. Although Mr Pickford does not set out his workings, the accumulated fibre/ml/years generated for Scenario A may be derived by multiplying the times appearing on page 1 of the chart in his report entitled "*Estimated Exp Durations",* by his assumed concentrations of fibre. This exercise demonstrates a total cumulative exposure on scenario A of 0.0343 fibre/ml/years.

118. For the purpose of Scenario B Mr Pickford assumed the following:

> (1) That in the first period Mr Booth held the fibro sheets for five hours and was exposed to asbestos concentration of 0.2 fibres/ml.
>
> (2) That in that same period he held the sheets while his father cut them for a period of 0.3 hours and was exposed to a fibre concentration of 0.05 fibres/ml.
>
> (3) That in the second period he held the sheets for a period of 8 hours and again was exposed to asbestos concentration of 0.1 fibres/ml.

119. By the same method as used for Scenario A, these assumptions demonstrate a cumulative exposure on Scenario B of 0.000938 fibres/ml/years.

120. The average of Scenario A and Scenario B is 0.0176 fibres/ml/years. Mr Pickford derived his estimate of 0.017 by application of the Monte Carlo simulation.

121. Mr Pickford says that he derived his estimates of 0.4 fibres/ml for Scenario A and 0.2 fibres/ml for scenario B from his experience in the field with construction workers installing asbestos cement sheets. He reports that such workers on average spent 20 per cent of the time carrying and handling asbestos cement exposed to 0.6 fibres/ml, 30 per cent of the time using hand cutting methods exposed to 0.4 fibres/ml, 24 per cent of the time nailing or screwing asbestos cement exposed to 0.1 fibres/ml and 26 per cent of the time in measuring and planning activities exposed to 0.05 fibres/ml.

122. Mr Pickford asserts that, because the time weighted average of such construction workers

using hand tools in the course of an eight-hour day calculates out at approximately 0.3 fibres/ml, that, in respect of the activity of an eight-year-old child occasionally holding asbestos cement sheets for his father to cut with fibro cutters *"it is reasonable to assume that the above estimate* [0.3 fibres/ml] *may be between 0.4 and 0.2 fibres/ml for Scenarios A and B respectively".*

123. Pursuant to this further assertion, Mr Pickford has assumed that the intermittent exposure of this eight-year-old child may be greater than that of a construction worker using hand tools over a period of five or six hours.

124. Mr Pickford's assumptions are not at all reasonable.

125. Mr Booth's evidence concerning his childhood exposure is as follows:

> *I remember holding the sheets for dad while he cut them with fibro cutters. I cannot recall any dust being generated from the fibro cutters. The fibro cutters simply punched the sheet. There were about 20 sheets in total, but only about six sheets had to be cut. The work involving the fibro took place over five or six hours with the cutting taking no more than about half an hour.*

126. Mr Pickford says that the concentration of asbestos fibre to which Mr Booth was exposed holding the asbestos cement sheets for his father to cut was 0.05 fibres/ml. I propose to adopt that measure in respect of Mr Booth's exposure as a child for the one half hour during which the cutting took place. I believe that otherwise he was, as he said, *sent off to play and get out of the road*.

127. The accumulated fibre burden of Mr Booth's childhood exposure is calculated as: 0.5 hours x 0.05 fibres/ml 1920 hours = 0.000013 fibre/ml/years.

128. Mr Pickford assigned an average concentration of 0.1 fibres/ml during eight hours in respect of the work holding sheets for his father to nail to the garage frame when he was 16 years old. This assumed concentration of 0.1 fibres/ml is in the breathing zone of the person doing the nailing. Common sense would tell us that when assisting his father Mr Booth did not grasp the sheet close to where his father held one end in place for nailing.

129. Further, an allowance of eight hours does not seem consistent with Mr Pickford's stated experience that construction workers spend only 20 per cent of their time nailing or screwing asbestos cement.

130. More reasonable assumptions are that the exposure when holding the sheet was in the order of half the concentration of 0.1 fibres/ml, that is, 0.05 fibres/ml for a period of two hours. Mr Pickford says 0.05 fibres/ml is the concentration associated with measuring, and being near to, asbestos sheeting. It is the same concentration as Mr Pickford allowed for Mr Booth holding an asbestos sheet while his father applied fibro cutters.

131. The accumulated fibre burden from this work may be calculated as 2 hours x 0.05 fibres/ml 1920 = 0.000052 fibre/ml/years.

132. The accumulated fibre burden from home renovations is then 0.000013 + 0.000052 fibre/ml/years = 0.000065 fibre/ml/years. This is 4 per cent of Mr Pickford's calculation of 0.0017 fibre/ml/years.

133. Taking into account these adjustments to Mr Pickford's estimates Professor Berry's table

of lifetime risks may be recast as follows:

Lifetime Risks of Mesothelioma per Million

Brake Repairs	Home Renovations		Loading Trucks	
18 x 170% = 30.6	50% amosite 50% crocidolite	15 x 4% = 0.6 93 x 4% = 4	Chrysotile	29 x 0.5% = 0.15

134. Upon the assumption that the asbestos in the home renovations was amosite, the combined lifetime risk created by the accumulation of brake exposure, home renovations, and loading trucks with chrysotile was 31.4 per million. Of this risk, the brake repair work contributed 97 per cent.

135. Upon the assumption that the asbestos in the home renovations was crocidolite, the combined lifetime risk created by this accumulation of exposures was 35 per million. Of this risk the brake repair work contributed 87 per cent.

136. Professor Berry says that it may be appropriate to assume that the background exposure of Mr Booth to asbestos fibre as a consequence of general low-level concentrations of asbestos in urban air corresponds to a lifetime risk of 70 per million.

137. The brake repair work increased the background causal component of 70 per million lifetime risks by a further 30.6 per million lifetime risks. Expressed in terms of cause, the brake work increased by approximately 44 per cent that fibre burden which comprised the background risk.

138. I regard such a contribution as material.

139. Even if the background risk was 170 per million lifetime risks, Professor Berry's upper limit, the contribution from the brake work remains material.

Mr Rogers' Evidence

Loading trucks

140. Mr Rogers assumed that Mr Booth was exposed for a total of eight hours to an average dust concentration of 5 fibres/ml, which included loading, driving, unloading and cleaning the vehicle. It is difficult to accept that while sitting in his cab driving Mr Booth was exposed to the same concentration of fibre as when loading the bags. There is no evidence that Mr Booth unloaded the truck or cleaned the vehicle. I accept that when removing his clothes that evening Mr Booth would have been exposed to additional fibre but in comparison to the work exposure it is probably insignificant.

141. Allowing, consistently with the evidence, for 20 minutes exposure while loading the truck with the assistance of another man, the cumulative dose is as follows: 0.33 hours x 5 fibres/ml 1920 = 0.0009 fibre/ml/years. This is 4.5 per cent of the estimate of 0.02 fibre/ml/years upon which Professor Berry made his calculations.

Home renovations

142. Mr Rogers calculated the cumulative exposure upon the assumption that Mr Booth over a period of 5 to 6 hours in total was exposed to an average concentration of 0.4 fibres/ml while he held fibro sheets for his father to cut them with fibro cutters. He concluded that the cumulative exposure was 0.001 fibre/ml/years.

143. Mr Rogers adopted 0.4 because this was the fibre concentration generated by the use of fibro cutters in the *James Hardie, Asbestos Cement Cutting Tool Study* of February 1980. Mr Pickford was present during this study, and says that the study was based on unrealistically high amounts of work, and a more appropriate figure for fibro cutting would be 0.2 fibres/ml.

144. Accepting for the moment that Mr Booth was exposed to the same concentration of fibre as his father who was actually doing the cutting, the appropriate calculation for this exposure for 20 minutes is: 0.33 hours x 0.2 fibres/ml 1920 = 0.00003 fibre/ml/years.

145. Because Mr Rogers made no allowance for the time spent by Mr Booth helping his father nail asbestos cement sheeting to the garage, I propose to add to Mr Rogers' estimates 0.000052 fibre/ml/years in respect of the work on the garage, (my previous calculation), that is a total of 0.00008 fibre/ml/years.

146. This is 8 per cent of the 0.001 fibre/ml/years upon which Professor Berry made his calculations.

Brake Work

147. Mr Rogers, upon a review of the literature, concluded that the historical daily time weighted average exposure of workers involved in routine brake and clutch repairs on automotive passenger vehicles and light commercial vehicles is no more than 0.2 fibres/ml (and most likely 0.05 fibres/ml or less).

148. Mr Rogers reconstructed Mr Booth's asbestos exposure by assuming that on each day that he performed brake work, although that work may have taken only one hour, his bystander exposure increased the total exposure up to the equivalent of a single eight hour time weighted exposure to all fibres of 0.2 fibres/ml.

149. I do not think this fairly reflects Mr Booth's exposure because on those days when he was not himself engaged upon brake work he was still exposed as a bystander to the work of others.

150. Nevertheless, Mr Rogers' method generated a total cumulative exposure from brake repair work of 1.8 fibre/ml/years. Asserting that due to thermal breakdown, the asbestos content of the fibres released when using compressed air on the brake drums was around 1 per cent, Mr Rogers said that the proportion of chrysotile fibres in the total dust exposure from new and old friction parts was less than 10 per cent and that Mr Booth's cumulative exposure was therefore less than 0.18 fibre/ml/years.

151. The blowing out of brake drums was only one source of dust in Mr Booth's atmosphere. The far greater source of asbestos fibre was the material thrown off by grinding the new brake linings.

152. Mr Rogers does not further explain his assertion that 90 per cent of Mr Booth's cumulative exposure was to fibres other than chrysotile asbestos and I do not accept it. The assertion was ignored by Professor Berry, who was provided with a copy of Mr Rogers report, yet applied Mr Rogers' unmodulated cumulative assessment of 1.8 fibre/ml/years.

153. With the exception of his estimate of the fibre concentration to which Mr Booth was exposed when loading hessian bags of asbestos onto his truck, I reject the evidence of Mr Rogers. I am unpersuaded that his assumptions and methods fairly reflect the circumstances in which Mr Booth was exposed to the inhalation of asbestos fibre.

Additional reservations concerning the evidence of Mr Pickford and Mr Rogers

154. Upon the basis of reported studies Mr Pickford assumed concentrations of airborne asbestos dust during brake work as varying between 0.05 fibres/ml and 0.4 fibres/ml, and Mr Rogers, a concentration of 0.2 fibres/ml.

155. Most, if not all, of the studies tendered in evidence, detected fibres by optical microscopy and did not count fibres shorter than 5 m in length. The defendants have relied upon studies by the American National Institute of Safety and Health (NIOSH) which tend to discount the exposure of brake mechanics to asbestos fibres.

156. In a paper entitled "*Asbestos Exposure during Brake Lining Maintenance and Repair"* Rohl *et al* compared the results of examination by optical microscopy, x-ray diffraction, transmission electron microscopy, and scanning electron microscopy. They reported that: *optical microscopy, employing polarised light, was generally not useful for detecting asbestos in brake drum dust. A number of factors are considered responsible for this phenomenon including the low relief and birefringence of chrysotile and the nature of the matrix, consisting largely of road dust, resin binder, and pyrolysed residue, which in optical microscopic preparations, readily obscures the smaller asbestos fibres.*

157. The authors state that the standard NIOSH optical fibre counting method, which only counts fibres longer than 5 m may be only a fractional indicator of total asbestos exposure, and that their findings indicate that the total exposure of brake workers, including fibres shorter than 5m, is much higher than the technique records.

158. In a later paper entitled *Asbestos Content of Dust Encountered in Brake Maintenance and Repair* published in *Environmental Research* in 1976, Rohl cites accumulating evidence which suggests that such small fibres may produce disease. (Holt et al 1964, Davies 1965, Pott et al 1972, Wagner et al 1973, Hilscher et al 1970, Bouhuys 1975).

159. Richard Lemen in his paper "*Asbestos in Brakes: Exposure and Risk of Disease"* published in the *American Journal of Industrial Medicine* 45:229-237 (2004) refers to a meeting of government agencies, University scientists and industry representatives convened by the American National Institute of Occupational Safety and Health (NIOSH) in 1975. The meeting reported that the average peak asbestos air concentration for specific brakes servicing operations, including blowout, grinding, and levelling of new truck brake linings resulted in average peak asbestos air concentrations within 10 feet of the operator of 10.5, 3.75 and 37.3 fibres/ml greater than 5 m in length. Significantly, further analysis found that, overall, almost all of the fibres actually released were shorter than 4 m in length.

160. Although it is unnecessary to decide, it seems likely that the estimates of fibre concentration applied by Mr Pickford and Mr Rogers in this case do not accurately reflect the accumulating risk of exposure to asbestos fibres in brake repair work.

Conclusions on Causation

Generally

161. The expert opinions as to fibre concentration expressed by Mr Pickford, and Mr Rogers in relation to the work on the wharves, when applied to the facts of this case, prove to my satisfaction that the plaintiff's exposure to the asbestos contained within brake linings materially contributed to the causes of his mesothelioma.

162. In any event, proof of causation in this case does not turn upon the epidemiological evidence, or upon questionable estimations of total fibre burden. An overwhelming inference of causation may be drawn from the following facts:

> (1) Mr Booth's mesothelioma was caused by the inhalation of asbestos fibre;
>
> (2) Mesothelioma very rarely occurs in persons who have not been exposed to asbestos fibres beyond the background level that pervades urban environments;
>
> (3) For a total of 27 years, week in and week out, Mr Booth was additionally exposed to asbestos fibres liberated from asbestos brake shoes by his own work, and by the work of others in his vicinity,
>
> (4) The previous exposure, in the course of home renovations and truck loading was, in comparison, trivial.

Specifically

163. Hardie-Bestos and Hardie-Ferodo brake linings were manufactured by Amaca between 1953 and 1962, a period of nine years. Between 1962 and 1982 they were manufactured by Amaba. Mr Booth did not work as a brake mechanic between 1969 and 1971, so that period of his exposure to Amaba's products is 17 years.

164. Over the course of his career Mr Booth, and others around him, worked with a number of different brands of brake linings, including Don, Safety Circle, Girlock, Bendix Mintex, Better Brakes and Hardie-Ferodo or Mintex. He estimated that about 70 to 75 per cent of the brake linings to which he was exposed were Hardie-Bestos or Hardie-Ferodo.

165. Because, on the evidence, the proportion of asbestos fibres in the brake dust released from brake drums is insignificant, I find that 70 per cent of the asbestos fibres to which Mr Booth was exposed over the period 1953 and 1962 were released from brake shoes manufactured by Amaca, and 70 per cent of the asbestos fibres to which Mr Booth was exposed for the periods 1962 to 1969 and 1971 to 1983 were manufactured by Amaba.

166. Although I do not think that the mathematics are necessarily compelling, if it were necessary to assign mathematical weight to the exercise, the products of Amaca were responsible for 70 per cent (the proportion of Amaca products) of 33 per cent (the proportion of the 27 years of brake work exposure) of 44 per cent (the excess accumulation of fibre burden beyond background exposure) which equals 10 per cent of the additional fibre burden beyond background which caused Mr Booth's mesothelioma.

167. Upon the same basis, the products of Amaba were responsible for 70 per cent of 66 per cent of 44 per cent, which equals 20 per cent of the additional fibre burden.

168. I appreciate that the causal contribution of Amaba's products is probably somewhat less than indicated by this simple apportionment, because of the greater potency of the earlier period of exposure to Amaca's products. The adjustment would be relatively minor.

169. In *Bonnington Castings Ltd v Wardlaw* [1956] AC 613 Lord Reid at 621 said that:

> *What is a material contribution must be a question of degree. A contribution which comes within the exception de minimus non-curat lex is not material, but I think that any contribution which does not fall within that exception must be material.*

170. The issue in *Bonnington Castings* was whether one fractional source of an injurious substance, silica, contributed to a gradual accumulation of that dust that resulted in disease. The High Court in *Amaca Pty Ltd v Ellis* [2010] HCA 5 (3 March 2010), held that this passage was not apt for application to a circumstance where a dispute exists as to whether the substance is causally implicated in the disease, but did not disapprove of his Lordship's statement of law as applied to the facts in *Bonnington.*

171. In *Workers Compensation (Dust Diseases) Board of New South Wales v Smith, Monro and Seymour* [2010] NSWCA 19 the New South Wales Court of Appeal, Basten JA (Allsop P and Handley AJA agreeing) held that one factor can materially contribute to an outcome even though, relative to another factor, it has a minor effect.

172. I find that asbestos dust liberated from brake linings manufactured by each of the defendants Amaca and Amaba materially contributed to Mr Booth's contraction of mesothelioma.

Foreseeability

The Case against Amaca

173. Asbestos has been a well-known hazard in industry for at least 70 years. The harmful affect has been known to be cumulative, with the risk of disease present at even comparatively low concentrations of asbestos dust. In their "*Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry"* (1930) Merewether and Price wrote that*: The appropriate methods for suppression of dust may only be fully determined when the harmful effects of comparatively low concentrations of asbestos dust are duly appreciated.*

174. The perceived dangers were not confined to persons working in the asbestos manufacturing industry. An article published in 1932 in the journal *Quarry and Road Making* referred to conditions liable to produce the disease of asbestosis as including *the sawing grinding and turning in the dry state of articles composed wholly or partly of asbestos* ***such as motor brakes*** *and clutch linings.* (Emphasis added)

175. In 1939 the UK Inspector of Factories reported that: *it is not many years ago when the dust of asbestos was regarded as innocuous, while today it is recognised as highly dangerous.*

176. Dr Smith, Director-General of Health in New South Wales, in his 1948 report confirmed the concern of industrial physicians and hygienists in relation to exposures to even small amounts of asbestos. At page 70 Dr Smith wrote in relation to a metal casting process:

> *The exposure to asbestos was intermittent and of short duration, about five minutes at each pouring, and, as at the time asbestos was being used two or three times a week the dust hazard was considered to be small. However, in view of the dangers associated with asbestos dust it was advised a substitute such as magnesia… should be used.*

177. In 1953, in an article entitled *"The Prevention of Dust Diseases"* published in the *Lancet*,

McLaughlin referred to the inappropriateness of maximum allowable concentration of dusts, writing:

> *... individuals too vary greatly in their capacity to deal with dust, and of two men who have been working at the same job for the same length of time one may get a disease of the lung and the other may be unaffected. This is why I am not impressed by the validity of what are known as the maximum allowable concentrations of dust of which lists have been drawn up in various countries.* [They] *seem to be based on the assumption that the man is a standardised machine, which he is not. The reasons for the differences in individual reaction to dusts are not accurately known, but it is likely that they depend on anatomical physiological and biochemical variations from one person to another.*

178. In 1956 an article entitled *"Dust Hazard in Industry"* was published in the magazine *Manufacturing and Management.* The author stated that workers prone to asbestosis included those who worked on brake linings.

179. In January 1957 the *Medical Journal of Australia* published an article entitled *"Pneumoconiosis in Victorian Industry"* written by Dr Thomas of the Industrial Hygiene Division of the Victorian Health Department. Dr Thomas noted occupations which potentially gave rise to a risk to health, and wrote:

> *The following occupations are involved: handling the substance in its raw form; grinding the substance prior to its use in some processes; mixing with diatomaceous earth or kaolin to form lagging materials;* ***sawing cutting and fitting any product containing asbestos-for example brake linings,*** *asbestos sheeting and various insulation materials; tearing down old lagging-this is a very dangerous process, even in the open-air; spraying asbestos on walls and ceilings as an insulator.* (Emphasis added)

180. On 18 March 1957, Mr N Gilbert of Amaca's Brooklyn office referred to this article by Dr Thomas and noted: ***"I not think there is anything in this which we do not already know."***

181. This material and other documents in evidence to which I have not referred, persuade me that by 1953—when the plaintiff began working as a motor mechanic—the inhalation of asbestos fibre by motor mechanics working on brake linings was generally recognised as dangerous, even at exposure levels below industrial standards, because of individual susceptibility to the cumulative effect of fibre inhalation, and the fact that no safe lower limit had been established.

182. In *Seltsam Pty Ltd v McNeill* [2006] NSWCA 158 (26 June 2006) Bryson JA said at [36]:

> *In my opinion questions of reasonable foreseeability and existence of a duty of care are not correctly decided by applying, as if they express a syllogism, Mason J's references to a risk which is remote, and to a risk which is not far-fetched or fanciful.*
>
> *The question of foreseeability must be looked at in the broad. The test of foreseeability is undemanding, but there is no basis for treating it as fictional or nominal, or for equating foreseeability with the limits of the imagination; it must be approached as a test which exists in reality and operates within the limits of the reasonable.*
>
> *It is not real, reasonable or just to adopt one broad class of end users to whom*

> *risks of exposure to asbestos were foreseeable. Persons whose exposure to asbestos was reasonably foreseeable include many whose exposure occurred in circumstances altogether different from those of the respondent.*
>
> *Workers who handled asbestos in mining, manufacturing or other industrial operations cannot on a reasonable or just basis be included in the same class as the respondent for purposes of deciding foreseeability. Their exposure was to undiluted asbestos and, as reasonably foreseen, extended to exposure for hours in the course of working days, and to continuous exposure over periods of months or years in a working career. Information which is known or ought to have been known to the appellant with respect to risks to them would be misapplied if it were treated as showing or tending to show some risk of injury relevant to the respondent's position.*

183. The class to which Mr Booth belonged was that of persons regularly and cumulatively exposed to the inhalation of asbestos fibres released from asbestos products upon which they worked in an industrial setting. The class described by Bryson JA as *Workers who handled asbestos in … industrial operations.*

184. Although, as the epidemiological evidence reveals, the risk faced by Mr Booth was not high, it was certainly a risk recognised before 1953, and it is a risk which has come home.

185. In *Julia Farr Services Inc v Hayes* [2003] NSWCA 37 (28 April 2003) Giles JA (with whom Spigelman CJ and Cripps AJA concurred), after referring to the judgments of Beazley JA and Stein JA in *Bendix Mintex v Barnes* (1997) 42 NSWLR 307, and Fitzgerald AJA in *E.M. Baldwin & Son Proprietary Limited v Plane* said:

> *Foreseeability … is not confined to knowledge of the dangers of exposure to high levels of asbestos, or levels of asbestos above any particular standard.*

186. I find that in 1953 it was reasonably foreseeable by Amaca that an automotive mechanic who was, in the course of every week over many years, exposed to the inhalation of asbestos fibre released from brake linings upon which he worked with grinding tools, may contract an asbestos related disease.

The Case against Amaba

187. Mesothelioma was referred to in the *British Medical Journal* in 1960. Discussing the complications of asbestosis the Journal noted that:

> *Another hazard is mesothelioma of the pleura. This rather rare tumour may draw attention to the fact that a patient has worked in asbestos dust…*
>
> *… nevertheless asbestos dust is most toxic and the amount needed to cause asbestosis is not known so constant vigilance and new preventative measures are needed if this disease is to be abolished.*

188. In July 1964 the same Journal published an article by Fowler et al, which reported on two cases of mesothelioma of the pleura. The authors wrote:

> *The association between mesothelioma of the pleura and exposure to asbestos is unlikely to be fortuitous: but to establish this point all cases of mesothelioma, whatever the occupational history, should be studied with this possibility in mind*

for evidence is now accumulating that this disease is an industrial hazard.

189. A copy of this article was, on 2 February 1965, forwarded by Professor Brian Gandevia to Mr E T Pysden, the personnel manager of James Hardie and Coy. On 16 February 1965 Mr Pysden wrote to Mr J B Reid, a director of James Hardie and Coy, saying of an article enclosed with the letter:

> ***The article is not new****-it is merely one of many reports in world studies which have been conducted since 1935 when the association between exposure to dust and carcinoma of the lung, mesothelioma of the pleura, tumour of the bladder and uterus and other fatal complications was first recognised.*
>
> *...*
>
> *Four types of asbestos-chrysotile, amosite, crocidolite and anthrophylite have similar effects on the body and it has not been possible to establish that one is more pathogenic than the others.* (Emphasis added)

190. These documents were discovered by Amaba.

191. In 1965 the British Journal of Medicine published the paper by Newhouse and Thomson, *Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area.* The authors refer to several instances of mesothelioma following slight exposure to asbestos, including one case of a worker who manufactured brake linings and another described as a garage hand and mechanic. They reported that: "*there seems little doubt that the risk of mesothelioma may arise from both occupational and domestic exposure to asbestos".*

192. Also in 1965, Dr Selikoff published his article *Relationship between Exposure to Asbestos and Mesothelioma,* in the *New England Journal of Medicine.* Dr Selikoff concluded that mesothelioma often resulted from relatively light and intermittent asbestos exposure.

193. By 1967 informed members of the general public were increasingly aware of the dangers of asbestos. On 15 May 1967, in an article appearing in *The Age* newspaper, reference was made to products incorporating asbestos used by the public. After reading the article, Mr Arthur Baker of Repco telephoned Mr R L Seers, the Manager Research and Development of James Hardie and Coy, inquiring as to the asbestos content in brake linings.

194. On 13 September 1967, under the heading, *"Death in Asbestos",* the Sydney Morning Herald published an article which stated that: *Workers grinding and cutting asbestos were particularly susceptible to the disease (mesothelioma),* and that the disease of mesothelioma was *"invariably fatal".*

195. In 1968 the Asbestos Research Council published a Code of Practice relating to asbestos in the motor industry. Amaba was a member of this Council. A section in the document entitled "Asbestos in the Motor Industry" appears in question and answer format. The following extracts are relevant:-

> *"Is there any risk in riveting or bonding brake linings?*
>
> *No there is no risk if the linings are predrilled, in the case of riveted linings. Exhaust ventilation should be provided at the drilling operation if this is a regular practice. No dust is generated in bonding brake linings, so there is no risk.*

What precautions ought to be taken when brake linings are being ground?

Exhaust ventilation should be provided. The dust extraction plant fitted to most commercial brake lining grinding machines is adequate.

Are these precautions necessary when grinding is only done occasionally, say for 8 hours a week?

Yes, in order to eliminate all possible risk, it is advisable to take these precautions.

Is the dust found in brakes and clutches when dismantling harmful?

The amount of asbestos fibre found is minute but the dust is very fine and it is good practice to remove it by vacuum, rather than by an air blast." (PX 48(2) (Tab 47))

196. By 1969 the dangers posed by exposure to chrysotile asbestos fibres released from brake linings were certainly recognised. Two British researchers, D E Hickish and K L Knight, were moved to conduct a study quantifying the asbestos exposure of a mechanic engaged in blowing the dust out of brake drums in the course of servicing motor cars and trucks. Their report was published in the *Annals of Occupational Hygiene* in 1970.

197. The authors concluded the report with this paragraph:

> *Our environmental studies have not included maintenance procedures which involve the filing or grinding of brake lining material, and we would envisage that these would give rise to considerably increased air contamination by chrysotile asbestos, with the attendant need for strict precautions to prevent the inhalation of fibres.*

198. In 1962 it was reasonably foreseeable by Amaba that an automotive mechanic who worked on brake linings over many years may contract an asbestos related disease.

199. From 1965 Amaba ought to have known, and certainly from 1968 did know, that automotive mechanics performing brake maintenance procedures involving the drilling and grinding of its brake lining products were at risk of contracting the fatal disease of mesothelioma.

Breach of duty

200. It follows from the above findings that the law imposed a duty upon Amaca and Amaba to take reasonable precautions to prevent Mr Booth suffering harm in consequence of using their products.

201. The scope of the duty depends upon the nature and extent of the risk. This calculus weighs the magnitude of the risk against the gravity of its consequence.

202. In matters of occupational health and safety there are no greater consequences than death.

203. It was known before 1953 that asbestosis was incurable. AC Haddow, in an article entitled *"Clinical Aspects of Pulmonary Asbestosis"*, published in the *British Medical Journal* 28 September 1929, wrote:

> *The… onset of anorexia signals to the worker that work is no longer possible.…*

> *The patient… becomes progressively weaker and more emaciated, more hopeless, sleepless and exhausted, until an attack of bronchial pneumonia or bronchitis brings death at last.*

204. In cases of mesothelioma death is most often accompanied by exquisite physical and mental suffering.

205. It is appropriate to impose upon a manufacturer of asbestos products a duty to take strong measures to obviate these consequences, notwithstanding any countervailing commercial interest.

206. Despite the fact that it knew of the risk, it was not until April 1978 that Amaba placed warning slips of paper in the packaging of its brake linings. The warning was in these terms:

> *Caution*
>
> *This product contains asbestos. Breathing asbestos dust can damage health. Keep dust down.*
>
> *Warning*
>
> *Keep dust down by following these simple safety rules:*
>
> *A tiny proportion of the dust found in used brake and clutch parts will be free asbestos fibre.* ***This is unlikely to be a health hazard*** *but you are advised to use damp cloth to remove it when servicing brake or clutch mechanisms and/or realigning your car breaks.*
>
> *Do NOT use an airline or brush to remove dust from brake drums.*
>
> ***Breathing asbestos dust can damage health***. (Emphasis added)

207. I do not regard this warning as adequate to discharge the duty imposed upon Amaba.

208. The consequences of ignoring the warning are expressed as *"Damage"* to health. The reader is not informed to the effect that the consequence of ignoring the warning may not be mere damage, but death.

209. Starkly, no warning is directed towards that activity responsible for almost all of the concentrated asbestos exposure generated when replacing brake linings, that is, the grinding of new brake shoes. The warning directs the reader only to the *tiny proportion* of the dust found in *used brake parts*.

210. Although the warning concludes with the words *breathing asbestos can damage your health,* the casual reader may read this in the light of the previous statement that *free asbestos fibre… is unlikely* to be a health hazard.

211. The warning does not even comply with the Code of Practice published by the Asbestos Research Council 10 years earlier, which advised that exhaust ventilation should be provided when grinding brake linings.

212. I regard the terms of this warning as entirely inadequate.

213. In written submissions, counsel for Amaba says that the alternative, a warning in graphic terms, or a warning of possible death *Is not a warning: in practical terms it would prevent the product being used.*

214. It is quite obvious, both from this submission and the historical evidence, that Amaba weighed the competing interests of its end users against its own commercial interests.

215. In a memorandum dated 14 May 1969 on the *"Biological Effects of Asbestos",* Mr Page, a director of James Hardie and Coy, referred to the existence of two papers on brake linings known to senior management at Better Brakes and Hardie-Ferodo as well as to James Hardie and Coy branch managers, factory managers and possible sales managers. Mr Page then wrote this: *I suggest that it would be worthwhile issuing a series of bulletins under the heading of say "The Asbestos Research Council"... **I don't intend that the bulletin should provide anything other than a completely partisan view of the problem** as seen from the industry's point of view.* (Emphasis added)

216. On 14 June 1972 James Hardie's Medical Director, Dr McCullagh, compiled the paper entitled *Medical and Hygiene Services-Progress Report* which he delivered in a presentation to the chairman and board of directors of James Hardie and Coy. Dr McCullagh wrote: *I foresee the time when the sales managers and I will be sitting down to draw up proposals that will protect both our end users **and our markets**.* (Emphasis added)

217. Mr Booth, who continued working with the brakes until 1983, does not recall seeing the warning slips included in the packaging after 1978. It may be that, because the warnings were not printed on the outside of the boxes containing the brake linings, the slips were not obvious, but mixed in with packaging material. It may be that Mr Booth read one such slip, dismissed the warning as unimportant, and has now forgotten that he ever read it. The necessary purpose of a warning being graphic is that it should arrest the attention of the reader.

218. Although Mr Booth had no memory of reading the warning slips included in the packaging of brake linings, in cross examination he conceded that had he seen a warning that breathing asbestos could damage his health he would have taken action to keep the levels of dust down.

219. I do not think that this concession assists Amaba's case. First because I find that the asbestos fibres liberated from Amaba's brake linings before 1978 materially contributed to Mr Booth's disease, and secondly because I am not persuaded that the warning given by Amaba was adequate to enter the consciousness of Mr Booth as a warning of sufficient threat to require precautions.

220. I accept the plaintiff's submission that the warning that should have been given was that formulated by Beech J in *Lo Presti v Ford Motor Company of Australia Ltd* [No 2] [2008] WASC 12 (19 February 2008) in these terms:

> *There is asbestos in the brake linings;*
>
> *Inhalation of asbestos dust and fibres can lead to life threatening disease;*
>
> *Steps should be taken to eliminate or reduce inhalation of dust from brake linings, including:*
>
> *(i) not using compressed air to blow out brake dust, instead using a vacuum cleaner or a wet method;*

(ii) using a respirator when filing, sanding or grinding brake linings, and

(iii) not dry sweeping areas which have brake dust."

Conclusions on liability

221. I find that both Amaca and Amaba failed to discharge their duty to warn Mr Booth of the dangers of asbestos, and that it is because of this failure that he has contracted mesothelioma.

Damages

General Damages

222. Mr Booth was born on 26 April 1937. He is now 73 years old. He contracted his disease in January 2008 and will probably die in the middle of 2011, after having suffered for three a half years. His is chronically short of breath, and every breath causes him pain.

223. Mesothelioma of the pleura has often been described by medical practitioners in this Tribunal as a *"most terrible disease"* and pleural pain as one of the most severe pains known to man, because of the exquisite sensitivity of the pleura, which carries a large number of pain receptors.

224. Mr Booth's pain is worsening. He already described it as "*nine out of 10. It is very severe, severe pain."* On exertion he has pain like "*someone is digging a knife into my chest".* Mr Booth says that despite the pain his greater suffering is that caused by the devastation of receiving his death sentence. He finds it "*extremely difficult to get any peace of mind".*

225. I assess general damages in the sum of $250,000. I allow interest on $180,000 at 2 per cent for 2.4 years. This amounts to $8,640.

Loss of Expectation of Life

226. According to the median life expectancy tables Mr Booth had a life expectancy of a further 13 years but for his mesothelioma. I award the conventional sum of $13,000.

Out-Of-Pocket Expenses

227. These will be met by the Workers Compensation (Dust Diseases) Board of New South Wales.

Griffiths v Kerkemeyer Damages

228. Although Mr Booth lives alone, he obviously needed care following hospitalisation and chemotherapy. He said that he got by *"with the assistance of friends".* An occupational therapist, Ms Lesley Stephenson, assessed the commercial cost of past care prior to 29 August 2008 as $4,085.95. It is reasonable to allow $5,000 in relation to past care.

229. Pursuant to s8(2)(d) of the *Workers' Compensation (Dust Diseases) Act 1942,* the Dust Diseases Board is required to provide to Mr Booth the benefits prescribed by Division 3 of Part 3 of the *Workers Compensation Act 1987.*

230. **S60** of the 1987 Act provides that a worker is entitled to be paid for the cost of medical or related treatment.

231. **S59** defines "medical or related treatment" to include

> *(f) care (other than nursing care) of a worker in the worker's home directed by a medical practitioner having regard to the nature of the worker's incapacity,*
>
> *(f1) domestic assistance services,*

232. **S60** is an indemnity provision. The rationale of *Griffiths v Kerkemeyer* plays no part in its construction. Services performed voluntarily or gratuitously are not covered by the section (*NSW Sugar Milling Cooperative Limited v Manning* (1998) 44 NSWLR 442).

233. Mr Booth intends that he should live with his sister and niece when he finds it too difficult to live on his own, and that his family will care for him. Ms Stephenson has, upon a life expectancy of 12 months, assessed the commercial cost of domestic support and personal care at $56,061.45. This includes allowances for lawn mowing and garden maintenance. In the circumstances those allowances are not appropriate.

234. The amount of care that will be provided is, to a large degree, uncertain and I allow the sum of $50,000.

Summary of Damages

235.

General Damages	$ 250,000
Interest on General Damages	$ 8,640
Loss of Expectation of Life	$ 13,000
Griffiths v Kerkemeyer Damages - Past	$ 5,000
Griffiths v Kerkemeyer Damages - Future	$ 50,000
TOTAL	**$ 326,640**

Orders

236. Judgment for the plaintiff against both defendants in the sum of $326,640.

> Defendants to pay the plaintiff's costs.

Mr PCB Semmler QC with Mr S Tzouganatos instructed by Turner Freeman appeared for the plaintiff
Mr G M Watson SC with Mr J C Sheller instructed by Holman Webb Brisbane appeared for the first defendant
Mr G M Watson SC instructed by DLA Phillips Fox appeared for the second defendant

Tribunal in which it was generated.
