IN THE COURT OF APPEAL OF NEW ZEALAND

CA172/05
[2007] NZCA 304

	BETWEEN	ACCIDENT COMPENSATION CORPORATION Appellant
	AND	MIKAEL MATTI AMBROS Respondent

Hearing: 24 July 2006

Court: Glazebrook, Arnold and Ellen France JJ

Counsel: B A Corkill QC and N D Lawson for Appellant
Respondent in Person
B D Gray QC as Amicus Curiae

Judgment: 20 July 2007 at 3 pm

JUDGMENT OF THE COURT

A The appeal is allowed.

B The answer to the question in the case stated (set out at [6] below) is "no".

C The matter is referred back to the High Court to be dealt with in accordance with the principles set out in this judgment.

D There is no award of costs.

REASONS OF THE COURT

(Given by Glazebrook J)

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Introduction

[1] In the early hours of 14 July 2000, Mrs Susan Ambros was admitted to North Shore Hospital after suffering chest pains. Tragically, she died there on 20 July 2000. At the post mortem examination, it was discovered that she had suffered an acute myocardial infarction (heart attack) about one week prior to her death and a further one approximately 24 hours before her death. The heart attacks were secondary to a rare condition called spontaneous coronary artery dissection (SCAD). Mrs Ambros had given birth to her first child just under a week before her

admission into hospital and SCAD in women is commonly associated with pregnancy and childbirth.

[2] Mr Ambros sought cover under the then accident compensation legislation, the Accident Insurance Act 1998 (the 1998 Act), in relation to his wife's death. The claim was based on medical error or medical mishap. The Corporation declined cover. That decision was upheld on review and an appeal to the District Court was dismissed. On 8 February 2005 Mr Ambros' appeal to the High Court succeeded.

[3] A full Court of the High Court (Harrison and Heath JJ), in *Ambros v Accident Compensation Corporation* HC AK CIV 2004-404-3261 21 March 2005, held that Dr Hart who was North Shore Hospital's Clinical Director of Medical Services and Mrs Ambros' consultant, had failed to observe the standard of care and skill reasonably to be expected of him following Mrs Ambros' admission to hospital. The High Court held that he had failed to take a number of critical steps in the face of evidence that Mrs Ambros had suffered a significant myocardial infarction immediately before admission.

[4] The High Court accepted that there must be a causal nexus between the personal injury suffered and the alleged medical error before there is accident compensation cover. It held (at [46]), however, that Parliament must have intended proved failures to diagnose or treat to be regarded as causative of injury or death in a case where:

(a) injury or death resulted in close proximity to the failures; and

(b) there is no evidence that death or injury was inevitable; or

(c) there was no supervening cause producing the injury or death.

[5] In Mrs Ambros' case, there was no evidence to suggest that her death was inevitable. Nor was there any evidence to suggest a supervening cause of death. This meant, in accordance with the test postulated by the High Court, that Dr Hart's

failures had caused Mr Ambros' death and that accident compensation cover was available.

[6] On 3 June 2005, Harrison J stated a case for submission to this Court on the issue of causation – see *Ambros v Accident Compensation Corporation* HC AK CIV 2004-404-3261 3 June 2005. The question posed for determination (at [10(8)]) was as follows:

> [W]hether or not proof of a medical error involving a "failure" constituting a "medical error" within the meaning of s 38(2) of the 1998 Act, where injury or death resulted in close proximity, will be sufficient to establish the cause of death or injury in terms of s 39(2)(b) in the absence of evidence (including evidence that the injury or death was inevitable or that some intervening cause produced the injury or death) to the contrary.

[7] The question posed in the case stated is wider than the test actually used by the High Court in that it does not limit the options for displacing a causation finding to inevitability or intervening cause. In this sense, it is more akin to the test suggested by Mr Gray – see below at [9]. In this judgment, we assess the correctness both of the test actually used in this case by the High Court and of Mr Gray's test. If we decide that neither test is correct, we must decide what should follow from that finding.

Was the High Court's test of causation correct?

Submissions of the parties

[8] The Corporation's position is that the High Court was in error in considering that a modified approach to the traditional principles of causation was required by the 1998 Act. In the Corporation's submission, it was for Mr Ambros to prove actual causation on the balance of probabilities – see *Atkinson v Accident Rehabilitation Compensation and Insurance Corporation* [2002] 1 NZLR 374 (CA).

[9] Mr Ambros supports the High Court formulation of the test for causation. Mr Gray QC, as amicus curiae, submits that, if the High Court test is not accepted, it

should be modified to provide a presumption of causation able to be displaced by evidence. His suggested test is as follows:

> Where a personal injury is alleged to arise from a failure of medical diagnosis and/or treatment and:
>
> (a) is the very injury which the diagnosis and/or treatment was intended to prevent; and
>
> (b) is part of the medical event in respect of which the diagnosis and/or treatment was given;
>
> then, prima facie, the injury has been caused by the failure of diagnosis and/or treatment.

Issues

[10] We propose to deal with this part of the case under the following headings:

(a) Is this case governed by *Atkinson*?

(b) Are there grounds for reviewing *Atkinson*?

(c) What is the traditional test for causation?

(d) What common law developments have there been?

(e) How have the courts otherwise dealt with the difficulty of proof and uncertainty?

(f) Should we accept Mr Gray's suggested test?

Is this case governed by Atkinson?

[11] *Atkinson* concerned the medical misadventure provisions of the Accident Rehabilitation and Compensation Insurance Act 1992 (the 1992 Act), the predecessor to the 1998 Act. The appellant's argument in *Atkinson* was that no proof of causal connection was required for cover for medical misadventure or alternatively that, once medical misadventure was proved, the onus shifted to the Corporation to disprove causation. The appellant submitted in that case that this

approach should be accepted because factors relating to causation are especially within the knowledge of the defendant. It was also submitted that the no fault accident compensation regime requires the adoption of a broad approach to causation consistent with common law developments.

[12] The main common law development relied on by the appellant in *Atkinson* was a shift in focus from the cause of the injury itself to the risk of injury resulting from a wrongful act or omission. It was argued that, where this is the case, all the plaintiff has to establish is that the defendant's conduct was at least capable of causing or aggravating the damage and did in fact materially increase the risk of that damage.

[13] This Court in *Atkinson* said it was satisfied that the appeal must fail. It held that it was for the claimant to prove causation – see at [23]. The Court said, at [24], that the accident compensation legislation focuses on outcomes and not risk of injury or potential for injury. For cover to exist, the risk must be realised. All that had been proved in the particular case was that there was a possibility (or risk) that the medical misadventure (sub-optimal care during an operation at Wanganui Hospital) contributed to the infant's injury (brain damage). There may have been other factors also that could have caused the damage. Indeed, the evidential difficulty was more serious. The claimant had not even proved injury. Given his age, experts were not able to assess whether or not the infant's development had been normal before surgery and become slow after it or whether his development was always going to be slow.

[14] The Court went on to say that it is not sufficient to prove that the medical misadventure risked causing an injury. It must be proved to have done so and to the usual civil standard of balance of probabilities. The Court held that to accept a lesser standard of increased risk or to adopt a reverse onus approach does not accord with the statutory scheme. The public policy considerations, which had led to the common law developments relating to causation, were rejected as being inapplicable to a statutory no fault compensation scheme which is outcome and not risk focused - see at [25] of *Atkinson*.

[15] While the High Court in this case accepted that causation had to be proved by Mr Ambros, it required him to prove only a close temporal link between the injury and the medical misadventure and to exclude inevitability of injury and supervening cause. This effectively means that, under the High Court test, as long as the injury occurred in close proximity to the medical error, a claimant would have proved causation, absent supervening cause, if there was as little as a one per cent chance of survival had the medical error not occurred.

[16] This falls squarely within the proposition rejected by this Court in *Atkinson* that a risk or possibility that the medical misadventure caused the injury suffices to prove causation, absent evidence to the contrary. The High Court test does not, therefore, accord with that laid down by this Court in *Atkinson*. In order to uphold the High Court decision, we would need to depart from *Atkinson*. The next issue, therefore, is whether we should do so.

Are there grounds for reviewing Atkinson?

[17] Where it is a question of statutory construction, this Court will adopt an extremely cautious approach to departing from previous authority – see *R v Chilton* [2006] 2 NZLR 341 at [108] (CA). The 1998 Act was passed against a background of the causation principles set out in *Atkinson.* Indeed, it is even clearer under the 1998 Act than it was under the 1992 Act that causation must be proved. The 1992 Act talked about injury "resulting from" medical misadventure. Section 39(2)(b) of the 1998 Act provides cover for personal injury (including death) "caused" by medical misadventure (medical error or medical mishap) suffered by the insured. Given this, the 1998 Act must be seen as a legislative acceptance of the principles in *Atkinson*. Any change to those principles would, in our view, need to have been made by Parliament by amendment to the 1998 Act.

[18] In any event, we consider *Atkinson* to have been correctly decided. In our view, the High Court test does not accord with the statutory wording. As we said at [15], that test could mean that causation is proved even when there was a very low chance of avoiding injury. In ordinary usage, one would not normally say that an

injury was caused by medical error when that injury was highly likely to have occurred without the error.

[19] *Atkinson* also remains consistent with English authority, despite the subsequent developments, discussed below at [26] - [52]. The closest case is *Wilsher v Essex Area Health Authority* [1988] AC 1074 (HL). In *Wilsher*, the claimant was an infant who had negligently received excess oxygen. He developed retrolental fibroplasia (RLF) which made him almost blind. Excess oxygen can cause RFL but it can also occur in premature babies who have not received oxygen. A possible causal link exists between RFL and at least four other conditions common in very premature babies. The House of Lords held that it had not been proved that the excess oxygen, rather than some different agent, caused the RFL. Reversing the onus, which found favour with Lord Wilberforce (but not the other Law Lords) in *McGhee v National Coal Board* [1973] 1 WLR 1 at 6 (HL), was rejected in *Wilsher* at 1087.

[20] *Wilsher* has survived all the later developments in England as set out in more detail below at [26] – [52]. It was affirmed by four of the five Law Lords in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 (HL) - see at [22] per Lord Bingham of Cornhill, at [70] per Lord Hoffmann, at [118] per Lord Hutton and at [149] and [170] per Lord Rodger of Earlsferry. It was not mentioned by Lord Nicholls of Birkenhead but the tenor of his judgment does not suggest that he would have been inclined to review it. It was also affirmed in *Barker v Corus UK Ltd* [2006] 2 AC 572 (HL) – see at [24] per Lord Hoffmann, at [64] per Lord Scott of Foscote and at [114] per Lord Walker of Gestingthorpe.

[21] As the High Court test does not accord with *Atkinson* and there are no grounds for reviewing the decision in that case, the appeal must be allowed. The next task is to assess whether we should accept Mr Gray's suggested test which creates a presumption of causation able to be displaced by evidence from the Corporation. In order to do this, we need to examine further the current test of causation, the developments in other jurisdictions and the manner in which the courts have traditionally dealt with difficulties of proof and uncertainty, with a view to seeing if Mr Gray's suggested test fits within that context.

What is the traditional test for causation?

[22] *Atkinson* states that causation must be proved on the balance of probabilities and that a risk of injury does not suffice. The judgment was, however, relatively brief and did not discuss how the courts should assess causation. We discuss the causation issue in more depth below so as to give a proper framework for any decision on causation in this case. In doing this we are also attempting in some measure to answer the plea for more guidance on causation principles by Associate Professor Joanna Manning in Skegg and Paterson (eds) *Medical Law in New Zealand* (2006) at [24.8.1], although the framework in this case can only be indicative for future cases as it may not assist in different factual circumstances.

[23] *Atkinson* also held that the developments in the common law (including loss of chance) are not applicable to the accident compensation regime. Again, there was no detailed explanation of why that is the case. It is helpful to discuss the reasons that the earlier developments are not relevant under an accident compensation regime in more depth in this judgment as that puts the later developments since *Atkinson* in context. We have held, at [19], that those later developments do not give cause to review *Atkinson*. The discussion of those developments set out below provides a fuller explanation for that decision.

[24] Causation in tort law is usually split into two separate inquiries: causation in fact and causation in law – see Todd (ed) *The Law of Torts in New Zealand* (4ed 2005) at [21.2] (causation in fact) and [21.3] (causation in law) and Hart and Honoré *Causation in the Law* (2ed 1985) at 109 - 110. Causation in fact relates to whether the tortious conduct has an historical connection with the injury. This is usually assessed on the basis of a "but for" test although the courts have relaxed this test in some circumstances (see below at [26]). The "but for" test poses the question whether the plaintiff would have suffered the injury without (in this case) the medical error. If it is more likely than not that, absent the error, he or she would have avoided the injury, then there is causation in fact.

[25] The second stage of the inquiry, causation in law, requires an assessment of the appropriate scope of liability for the conduct. There is then a third level of

inquiry into proximity (remoteness) between the cause and the damage, although this will often merge into the second stage of the causation inquiry. Under a no fault accident compensation regime, the third stage is likely to be more important than the second stage. This is because the emphasis at the second stage is on the extent to which it is appropriate to assign responsibility to particular persons. It is not the aim of the accident compensation regime to assign blame. At the broadest level of generality, its aim is to promote distributive rather than corrective justice by spreading the economic consequences of negligent conduct over the whole community and to provide compensation for injury (regardless of fault) – see Royal Commission of Inquiry *Compensation for Personal Injury in New Zealand* (Report December 1967) at [88] ("*The Woodhouse Report*"). This is often described as a social contract: in return for the loss of the right to sue for personal injury, the community shares the costs of the injury – see *Queenstown Lakes District Council v Palmer* [1999] 1 NZLR 549 at 555 (CA). Any inquiry at the second stage would, in New Zealand, be designed to identify and take into account policy issues arising in relation to the accident compensation scheme generally and the particular statutory manifestation of that regime at issue in the case.

What common law developments have there been?

[26] There have been a number of situations where dissatisfaction with the result of the traditional test of causation has led to calls for a modification to the "but for" stage of the inquiry. The challenges to the traditional test can be placed into three categories: industrial diseases, loss of chance and informed consent. In this section we describe these challenges, their success (or otherwise) and discuss whether they are applicable to the New Zealand accident compensation regime.

INDUSTRIAL DISEASES

[27] One response to the difficulty of proving causation has been an approach that does not require proof that the tortious conduct was the sole, or even the main, cause of injury. All that must be proved is that it made a material (in the sense of more than trivial) contribution to the damage –

see *Bonnington Castings Ltd v Wardlaw* [1956] AC 613 (HL). That was a case where the claimant contracted pneumoconiosis from inhaling air containing silica at his workplace. The air came both from a "guilty" source (where the employer was negligent) and an "innocent" one (where there was no negligence). The "but for" test could not be met as in all likelihood the claimant would, had there been no guilty dust, have contracted the disease from the innocent dust. It was also not possible to apportion part of the injury to the guilty dust. The House of Lords was prepared to depart from the "but for" test in such circumstances and merely require proof that the guilty dust made a material contribution to the disease.

[28] The decision in *Bonnington* was part of the legal background against which the accident compensation regime was first enacted. It is thus possible that the test for causation was intended to include the concept of "material contribution" in circumstances like those in *Bonnington*. See Armstrong and others *Personal Injury in New Zealand* (looseleaf ed) at [IP32.17] and, in the criminal context, *R v Martin* (1832) 5 Car & P 128; 172 ER 907 (CA), *R v Burdee* (1916) 12 Cr App R 153, *Mamote-Kulang v R* (1964) 111 CLR 62 and *R v Renata* [1992] 2 NZLR 346 (CA).

[29] An extension of the material contribution approach occurred in *McGhee*. That case concerned a claimant who contracted dermatitis from the presence of brick dust on sweaty skin. The exposure to dust at the work site was inevitable and there was no negligence on the part of the employer in that regard. The employer, however, negligently failed to provide washing facilities on site and the claimant had to cycle home with brick dust on his skin. It was impossible to prove whether or to what extent the dust contributed to the dermatitis. At best, it could be proved that the failure to provide washing facilities increased the risk of dermatitis. A majority of the House of Lords were prepared to treat this material increase in the risk of injury as equivalent to a material contribution to the injury.

[30] In *Wilsher*, Lord Bridge of Harwich treated *McGhee* as a case where the Court had been prepared to draw a robust and pragmatic inference of causation. Other jurisdictions have regarded it in a similar fashion – see, for example, the comments of Spigelman CJ in *Seltsam Pty Ltd v McGuiness* (2000) 49 NSWLR 262 at 280 (CA). See also Khoury *Uncertain Causation in Medical Liability* (2006)

at 151 – 152 and 213. In *Fairchild*, their Lordships (apart from Lord Hutton at [94] – [108]) departed from this position. They held that *McGhee* did not concern inferences but a rule of law that, in the circumstances pertaining in *McGhee* where there was one noxious agent rather than multiple agents, a material contribution to risk was equivalent to a material contribution to the disease – at [22] per Lord Bingham, at [44] per Lord Nicholls, at [67] – [69] per Lord Hoffmann and at [144], [147] and [150] per Lord Rodger. See also the discussion in *Barker v Corus* at [13] per Lord Hoffmann, and Lord Scott (at [50] and [64]) and Lord Walker (at [103] – [104]) who both agreed with the reasoning of Lord Hoffmann.

[31] The question is when the principle in *McGhee* stops and the rule in *Wilsher* (see at [19] above) begins. The answer appears to be that, where the defendant's breach of duty increases an existing risk factor, such as in *McGhee*, the court may, taking if necessary a robust and pragmatic view of the evidence, infer (presumably after *Fairchild* as a rule of law) that there must have been some material contribution to the injury. Where, however, the defendant's breach of duty merely adds a new discrete risk factor to the existing risk factors (as in *Wilsher*) it is not legitimate to infer that it was the guilty factor which probably caused the damage – see *Barker v Corus* at [24] per Lord Hoffmann, at [64] per Lord Scott and [114] per Lord Walker and Dugdale and Jones (eds) *Clerk & Lindsell on Torts* (19ed 2006) at [2-33].

[32] To the extent that *McGhee* treats the increase in risk as equivalent to a material contribution to injury as a rule of law, it must be seen as contrary to *Atkinson* and thus not applicable in New Zealand insofar as the assessment of causation under the accident compensation regime is concerned. However, it may still have relevance if it is seen as a case where the Court was prepared to draw a robust inference of causation. As Khoury says at 214, material increase of risk does in fact fit squarely within inferential reasoning, which typically infers from a known fact (e.g. the increase in risk) the existence of an unknown fact (e.g. material contribution). Whether the inference will be drawn would, however, depend on the totality of the circumstances. Professor Stapleton makes a similar point. She posits the situation where a particular surgical error more than doubles the risk of post-operative cardiac arrest. Where the error is made and post-operative arrest

occurs, then, in her view, a court would be entitled to infer causation because the error has added more than the existing risk. See Stapleton "Scientific and Legal Approaches to Causation" in Freckelton and Mendelson (ed) *Causation in Law and Medicine* (2002) 14 at 22 – see at [65] – [70] below.

[33] The next relevant development is *Fairchild*. *Fairchild* concerned three appeals where workers had contracted mesothelioma following exposure to asbestos at work. The difficulty was that there had been multiple employers and no way of proving which employer was responsible for the disease. While the risk that mesothelioma will occur increases in relation to the total dose of asbestos received, the severity of the condition and the resulting disability do not vary with the dose. The House of Lords held that, in the circumstances pertaining in the case (see for example at [21] per Lord Bingham, at [65] and [67] per Lord Hoffmann, at [109] and [116] per Lord Hutton and [170] per Lord Rodger) proof of contribution to risk sufficed to prove a material contribution to the disease.

[34] The exact question *Fairchild* was dealing with would not, of course, arise under a no fault accident compensation regime. There is no requirement to assign responsibility to any particular person. Conceivably, however, there could be situations where the existence of cover may depend on the identity of the responsible agent or timing and *Fairchild* could have relevance in such situations. The principle in *Fairchild* appears, however, to be very limited. In *Barker v Corus* (discussed below at [43]), Lord Hoffmann made it clear (at [24]) that the *Fairchild* exception applies only where the impossibility of proving the defendant caused the damage arises out of the existence of another potential causative agent which operated in the same way. Thus in this case where there were two possible (and differing) causes of Mrs Ambros' death (medical error or the underlying condition), *Fairchild* can have no application.

[35] Any further consideration of whether *Fairchild* may be applicable in New Zealand to cases under the accident compensation regime must wait for a case where it arises. We do note, however, that the *McGhee*/*Fairchild* exceptions have not been without their critics. Murphy *Street on Torts* (11ed 2003) at 286 for example has described the decisions where the English courts have modified the

traditional test of causation to deal with evidential uncertainty as arguably creating as many problems as they solve. This is because of their uncertain scope and the absence of any overarching principle. Some articles which cover both sides of the debate are: McAdams "*Barker* and the King of Persia" (2006) 156 NLJ 1433; Plowden and Volpe "*Fairchild* and *Barker* in MRSA Cases: Do *Fairchild* and *Barker* Provide an Argument For a Relaxation of Causation Principles in Claims for Hospital Acquired MRSA" (2006) 3 JPI Law 259; Stapleton "Cause-in-Fact and the Scope of Liability for Consequences" (2003) 119 LQR 388; Green "The Risk Pricing Principle: A Pragmatic Approach to Causation and Apportionment of Damages" (2005) 4 Law, Probability & Risk 159; Arnell "Causation Reassessed" (2002) 32 SLT 265; and Miller "Judicial Approaches to Contested Causation: *Fairchild v Glenhaven Funeral Services* in Context" (2002) 1 Law, Probability & Risk 119.

[36] We also note that the view has been expressed that the *McGhee*/*Fairchild* line of cases should be limited to industrial diseases. Khoury argues (at 214) that it may be inadvisable to extend the *McGhee/Fairchild* line of cases outside of industrial diseases into the field of medical malpractice. She says that in products liability, industrial pollution and industrial diseases, the creator of the risk is also the beneficiary of the risk which has generally been created for commercial profit. In medical cases the pre-existing condition of the claimant is usually negative (through pre-existing sickness) and grounded in an occurrence other than the medical act. Further, uncertainty is an inherent part of medical practice and medicine is of high social utility. Lord Hoffmann in *Fairchild*, at [69], made similar comments as to the difference between medical negligence and industrial diseases and the greater social cost of liability if loss of chance principles are extended to medical negligence cases generally – see also *Gregg v Scott* [2005] 2 AC 176 at [90] (HL) per Lord Hoffman.

LOSS OF CHANCE

[37] The next challenge to traditional causation principles has been the call to extend the loss of chance principles to cover cases of medical negligence. For a recent discussion on loss of chance principles in New Zealand see *Benton v Miller & Poulgrain (a firm)* [2005] 1 NZLR 66 (CA) and Barker's commentary on

that case in Barker "Damages for Loss of Chance" [2007] NZLJ 151. See also Tobin: "Apportionment of Liability for the Loss of a Chance: *Barker v Corus* a Recent Development in Asbestos Litigation" (2006) 12 NZBLQ 216; Coote "Loss of a Chance, or the Chance of a Loss" (2007) 13 NZBLQ 15; Coote "Recovery for Loss of a Chance: Could it be for All or Nothing at All?" (2006) 12 NZBLQ 127; and Peel "Loss of Chance in Medical Negligence" (2005) 121 LQR 364.

[38] The loss of a chance of a better medical outcome often arises in cases of delayed, missed or wrong diagnosis leading to a deterioration in the patient's condition. What must be determined is whether the damage is a result of the doctor's fault or the normal outcome of the pre-existing condition – see *Laferrière v Lawson* (1991) 78 DLR (4th) 609 at 657(h) – 658(a) (SC). The answer, on traditional causation principles, will depend on the patient's prognosis at the time the correct diagnosis should have been made. If the patient was at that time more likely than not to recover if properly diagnosed and treated, then the faulty diagnosis will be treated as causative. Otherwise it will not be. If it is treated as causative then full damages are recovered and there is no discounting for the chance (which could be up to 49 per cent) that the negligence did not cause the deterioration. If it is not found to be causative, then the plaintiff fails to recover at all.

[39] Thus in *Wilsher* (see at [19] above), the Court, applying traditional causation principles, did not compensate the plaintiff for the chance that the negligence, as against the other possible causes, caused his blindness. Another example where a plaintiff failed to recover damages for loss of chance is *Hotson v East Berkshire Area Health Authority* [1987] 1 AC 750 (CA and HL). In that case there were two possible causes of injury. The plaintiff had fallen and injured his hip. There was a negligent delay in treatment and he ultimately developed avascular necrosis of the hip joint. The medical evidence was that there was a 75 per cent likelihood that this would have resulted from the fall alone. The House of Lords held that the plaintiff had failed to prove causation, reversing the lower court's award of compensation for the lost 25 per cent chance. If it was more likely than not that he would not have developed the condition he would have been entitled to full compensation. If causation was not proved the plaintiff could recover nothing.

[40] In *Hotson*, Lord MacKay of Clashfern at 786 distinguished *McGhee* on the basis that, in *McGhee*, the precise aetiology (causes) of the disease could not be evaluated, whereas in *Hotson* it could and went against the plaintiff. It may seem harsh that uncertainty can work for a plaintiff whereas precision works against him or her. Although the actual plaintiff in *Hotson* would have been covered under our accident compensation scheme whether the injury resulted from the fall or the medical error (as both events attract accident compensation cover), another claimant may not receive cover in New Zealand due to the *Hotson* principle if the injury had two possible causes and it was more likely that the injury was caused by an event which does not attract cover as opposed to the other possible causal event which does attract cover.

[41] After *Fairchild*, the question arose in England as to whether the principle in that case applies to all cases of medical negligence. This question came before the House of Lords in *Gregg v Scott*. In that case there was a negligent delay in the diagnosis of Mr Gregg's cancer and its consequent treatment. This reduced his chance of survival from 42 per cent to 15 per cent. By a majority of three to two, the House of Lords rejected the claim. Lord Hoffmann (at [90]) and Baroness Hale of Richmond (at [225]) did so on policy grounds, including the major consequences for the health system of allowing compensation for risk. Lord Phillips of Worth Matravers (at [191]) rejected the argument largely on the facts. He was not necessarily totally averse to a loss of chance argument but only in cases where the outcome is known (and in this case the patient was still alive at the time of the hearing).

[42] Of the two Law Lords who would have allowed the claim, Lord Nicholls did so on policy grounds. A doctor's duty is to maximise the patient's recovery prospects whatever the patient's prognosis. In his view, it follows that the law should fashion a meaningful remedy for the loss of even a poor prospect of recovery - see his judgment at [42] - [46]. Lord Hope of Craighead distinguished Mr Gregg's situation from that of the plaintiff in *Hotson*. In the latter case, the fate of the plaintiff depended on the state of facts existing when he was admitted to hospital whereas Mr Gregg's chance of survival with proper treatment involved hypothetical speculation on an event that still lay in the future. He saw a loss of

chance analysis as permissible in such a case. Lord Hope saw the claim as relating to the loss and damage caused by the enlargement of the tumour and the diminished chance of survival due to the delay.

[43] The House of Lords had to reconcile *Fairchild* and *Gregg v Scott* in the case of *Barker v Corus*. That case was, like *Fairchild*, a case of mesothelioma. The plaintiff had had multiple employers but there was also a period of self-employment. The question was whether liability and the resulting damages should be apportioned between the claimant and the various employers. The majority of the House of Lords held that the basis of liability in such cases is the wrongful creation of a risk or chance of causing the disease and damages should be apportioned accordingly. Lord Hoffmann, at [36], recognised that this was essentially applying loss of chance principles, but said, at [39], that these were applicable only in the type of cases covered by the *Fairchild* exception. Lord Hoffmann noted that the rejection of the loss of chance analysis in *Gregg v Scott* was not an aversion to the concept generally but a concern that the loss of chance analysis would extend the *Fairchild* analysis to all cases of medical negligence, if not beyond. This would be inconsistent with *Wilsher*. Lord Scott made similar comments at [57], as did Lord Walker at [114]. Lord Rodger, however, considered that the majority were not so much reinterpreting as rewriting *Fairchild* in applying loss of chance principles (see at [71]).

[44] Loss of chance analysis has had a mixed reception in other common law jurisdictions. It has been rejected by the majority of the Supreme Court in Canada – see Linden and Feldthusen *Canadian Tort Law* (8ed 2006) at 116. In *Laferrière v Lawson*, the defendant physician removed a lump from a patient's breast but failed to inform her it was cancerous. The removal of the lump without any further follow up was an available method of treatment. The Supreme Court rejected the claim for damages for the loss of a chance to seek further treatment. Gonthier J, for the majority, undertook an extensive analysis of the French and Belgian cases utilising loss of chance principles. In his view, it would only be in exceptional cases that a court was unable to decide, based on the facts and statistics relating to the particular case, whether a chance had been realised and thus courts had no need to resort to loss of chance principles – see at 654.

[45] The High Court of Australia has not had a pure loss of chance case in the medical negligence area. In *Naxakis v West General Hospital* (1999) 197 CLR 269 the plaintiff indicated that such an argument may be run on any retrial but the appeal was decided on other grounds. Only two of the Judges made any comment on the possible application of loss of chance principles. Gaudron J was against the notion of discounted damages for loss of chance (at 281) and Callinan J was in favour of it (at 312 – 313). The New South Wales Court of Appeal has, however, accepted a loss of chance analysis in *Rufo v Hosking* (2004) 61 NSWLR 678. Other States have also approved the loss of chance doctrine – see *Brown v Willington* [2001] ACTSC 100, *Quantock v Australian Capital Territory Health and Community Care Service* [2003] ACTSC 98, *Gavalas v Singh* (2001) 3 VR 404 (CA), and *Lemm v Daniels* [2001] QDC 231. The loss of chance doctrine has also been accepted in the United States in the medical negligence sphere – see Coltoff, Kennel and Pellegrino *Corpus Juris Secundum: Physicians and Surgeons* Vol 70 at § 92.

[46] Whatever the future developments in loss of chance in other jurisdictions, the loss of chance analysis seems to us to be incompatible with the accident compensation regime. Under a no fault regime, either there is cover or there is not. There is no ability to discount compensation and in a no fault regime no conceptual need to do so. Where the wrongdoer is not himself or herself financially liable for the injury, there is no need to discount the level of compensation to reflect the fact that other factors unrelated to the wrongdoer's conduct played a part in the injury. The focus is on "real compensation" – see *The Woodhouse Report* at [59]. Further, as indicated above in the discussion on *McGhee*, at [32], to the extent that risk of injury is considered a sufficient basis of liability (even in the limited circumstances of the *Fairchild* exception), this would be inconsistent with *Atkinson*. In terms of the causation principles set out in *Atkinson*, any risk must be realised in the occurrence of a personal injury and the personal injury must be proved to have been caused by the risk factor involved. In keeping with this principle, if the omission to treat causes an identifiable added injury, cover would be available for that injury.

[47] The next area where the traditional causation test has been challenged is where informed consent to treatment has not been obtained. In such cases, the causation question must depend on what the particular claimant would have done had the risks of the medical procedure been properly explained. Clearly, if the claimant would have continued with the procedure even if the risks had been explained, then there is no causation. If the claimant would have refused medical treatment then the non-disclosure has caused the claimant's damage.

[48] The third (and more difficult) category of case is where an explanation of risk would, on the balance of probabilities, have caused the claimant to delay treatment but treatment would nonetheless have been likely to have been undertaken with a similar risk of injury. This was the situation in *Chappel v Hart* (1998) 195 CLR 232 and *Chester v Afshar* [2005] 1 AC 134 (HL). In both cases the surgeon, who had failed to warn of the risks, was held liable. The decision in both cases was primarily based on a policy choice to impose liability in order to ensure that the duty to obtain informed consent is respected by medical practitioners - see for example *Chester v Afshar* at [16] – [18] and [25] per Lord Steyn, at [85] – [87] per Lord Hope, at [92] and [101] per Lord Walker and Gaudron J at 238, Kirby J at 272 and Gummow J at 258 in *Chappel v Hart*.

[49] Mr Corkill submitted that the High Court in this case, while recognising that the loss of chance cases were not applicable, nevertheless relied heavily on the reasoning of Lord Hope in *Chester v Afshar*. He submitted that *Chappel v Hart* and *Chester v Afshar* are not applicable in New Zealand but, even if they are, they should be limited to cases of informed consent. We accept this submission.

[50] To the extent that the informed consent cases are based on the policy imperative of upholding patient rights to be informed by imposing liability to pay damages, then they have little relevance to a no fault accident compensation regime. *Clerk & Lindsell on Torts*, however, questions (at [2 – 15]) whether the decision in *Chester v Afshar* had to be based on policy factors. The authors opine that a patient's claim in respect of non-disclosure of risk is for the physical damage

attributable to the materialisation of the risk and not to the exposure to the risk. If the risk does not materialise and no physical damage ensues, then there is no claim. If looked at in this way, then the right to be informed cases can be seen as being in accordance with the *Atkinson* test as being concerned with outcomes and not risk. Indeed, where treatment would have been delayed, the "but for" test could be seen as having been met if the chances of the risk materialising on the later occasion are slight. But for the failure to inform, the procedure would not have been undergone that day and the injury would in all likelihood not have been suffered when the procedure was rescheduled.

[51] If the *Clerk & Lindsell* approach is correct, it would, as Mr Corkill submitted, apply only to informed consent cases and not more widely. We are not, however, to be taken as endorsing that approach even in an informed consent context. In our view, it would mean that causation in fact ("but for") always equates to causation in law in that context. We would need to consider whether that was appropriate. In that regard we would need to consider Lord Hoffmann's view that to hold that there has been causation in such circumstances is similar to saying that a win at the casino was caused by going there on Tuesday because the chances were slight that there would have been a win had the punter gone to the casino on Wednesday – see Hoffmann "Causation" (2005) 121 LQR 592 at 602. See also the comments of McHugh J in dissent in *Chappel v Hart* at 246 – 247 where he said that, if the act or omission of the doctor had done no more than expose the plaintiff to a class of risk to which he or she would have been exposed irrespective of the doctor's act or omission, the law of tort should not require the doctor to pay damages. McHugh J's concern, however, may apply to a much lesser extent in a no fault compensation regime.

[52] Any further consideration of the applicability (or otherwise) of the informed consent cases under the accident compensation regime must wait until a case arises in the New Zealand context.

How have the courts otherwise dealt with the difficulty of proof and uncertainty?

[53] There will often be some difficulty in establishing a causal link between medical error and injury because of scientific uncertainty over the causal

mechanism – see Khoury at 48 - 50. Uncertainty can arise because of the objective limitations of scientific medical knowledge about a particular biological process (particularly where there may be multiple possible causes) or from the difficulty of providing a scientific explanation for the sequence in a particular case. While a causal relationship may exist between a possible cause in a certain percentage of cases in the population as a whole, it may be impossible to say whether it causes the condition in the case at hand. There may also be difficulties proving causation where the outcome may be dependent on hypothetical human actions, either of the claimant or a third party.

[54] We propose to deal in this section with the ways in which courts have traditionally dealt with uncertainty under the following headings: evidential onus, inferences, statistics and proximity.

EVIDENTIAL ONUS

[55] As pointed out by the Court in *Ithaca (Custodians) Limited v Perry Corporation* [2004] 1 NZLR 731 at [44] – [47] (CA), the term burden of proof has been used in two quite distinct senses – see Williams "Burdens and Standards in Civil Litigation" (2003) 25 Syd LR 165. The first is a reference to the legal burden. The legal burden is what must ultimately be proven by a person in order to win the case. Equally, it can refer to the evidential burden. The term evidential burden is, in turn, used to refer to two quite distinct notions. In the first sense, it means the burden of adducing evidence on an issue on pain of having the trial Judge determine the issue in favour of the opponent. The second sense in which the phrase is used refers to the burden resting upon a party who appears to be at risk of losing on a given issue at a particular point in a trial. This merely involves a tactical evaluation of who is winning at a particular point which can shift depending upon the trial dynamics. This is often referred to as the tactical burden.

[56] As we have noted above at [14], the switching of the legal burden to the defendant was rejected in *Atkinson* and has not found favour with the House of Lords – see at [19] above. A reverse onus was also rejected in Canada, apart possibly from in exceptional cases such as occurred in *Cook v Lewis* [1951] SCR 830 where the

plaintiff had been shot and all three defendants had negligently discharged their rifles in his direction - see *Snell v Farrell* (1990) 72 DLR (4th) 289 at 301 (SC). The High Court of Australia has not favoured a reversal of the legal burden either – see *Chappel v Hart* at 273 per Kirby J and at 247 per McHugh J.

[57] The shifting of the evidential onus has met with a greater acceptance, however. In *Chappel v Hart*, Kirby J, in the majority, stated at 273:

> Nevertheless, the realistic appreciation of the imprecision and uncertainty of causation in many cases – including those involving alleged medical negligence – has driven courts in this country, as in England, to accept that the evidentiary onus may shift during the hearing. Once a plaintiff demonstrates that a breach of duty has occurred which is closely followed by damage, a prima facie causal connection will have been established. It is then for the defendant to show, by evidence and argument, that the patient should *not* recover damages. (Emphasis in original).

[58] Gummow J, also in the majority, held that the evidential burden shifted to the defendant once the plaintiff had established a prima facie case – see at 257. The shifting of the evidential onus was even favoured by McHugh J in dissent – see at 247 - 248. He said that, once the plaintiff proves that the defendant breached a duty to warn of the risk and that risk eventuated and caused harm to the plaintiff, the plaintiff has made out a prima facie case of causal connection. An evidentiary onus then rests on the defendant to point to other evidence suggesting that no causal connection exists.

[59] In Canada in *Snell v Farrell*, Sopinka J, for the Court, pointed out that in many malpractice cases the facts lie particularly within the knowledge of the defendant. In these circumstances very little affirmative evidence on the part of the plaintiff will justify the drawing of an inference of causation in the absence of evidence to the contrary and even though positive or scientific proof of causation has not been adduced - see at 300. Even if some evidence to the contrary is adduced by the defendant, the trial judge is entitled to take account of Lord Mansfield's famous precept in *Blatch v Archer* (1774) 1 COWP 63 at 65; 98 ER 969 at 970:

> It is certainly a maxim that all evidence is to be weighed according to the proof which it was in the power of one side to have produced, and in the power of the other to have contradicted.

[60] Sopinka J said that this is what he believed Lord Bridge had in mind in *Wilsher* when he referred (at 1090) to a "robust and pragmatic approach to the …facts". Sopinka J also referred to the cases of *Diamond v British Columbia Thoroughbred Breeders' Society* (1965) 52 DLR (2d) 146 at 158 (BCSC), *Pleet v Canadian Northern Quebec R Co* (1921) 64 DLR 316 at 319 - 320 (Ont SC), *Guaranty Trust Co of Canada v Mall Medical Group* [1969] SCR 541 at 545 and to Buckley LJ's affirmation of the principles in *Dunlop Holdings Limited's Application* [1979] RPC 523 at 544 (CA), which in turn referred to Stephens *A Digest of the Law of Evidence* (12ed 1946) art 104 cited in Cross *Cross on Evidence* (4ed 1974) at 86, which states that, in considering the amount of evidence necessary to shift the burden of proof, the court has regard to the opportunities of knowledge with respect to the facts to be proved which may be possessed by the parties respectively. However, the peculiar means of knowledge of one of the parties does not relieve the other of the burden of adducing some evidence with regard to the facts in question, although very slight evidence will often suffice.

[61] Sopinka J said, however, that these references speak of the shifting of the secondary or evidential burden of proof or the burden of adducing evidence. He said that he finds it preferable to explain the process without using the term secondary or evidential burden. This is because it is not strictly accurate to speak of the burden shifting to a defendant when what is meant is that evidence adduced by the plaintiff may result in an inference being drawn adverse to the defendant. Whether an inference is or is not drawn is a matter of weighing evidence. The defendant runs the risk of an adverse inference in the absence of evidence to the contrary. He noted that this is sometimes referred to as imposing on the defendant a provisional or tactical burden. As noted above at [55], the term tactical burden has been employed by this Court.

[62] We also note that Lord Mansfield's maxim has been applied in New Zealand in a number of different contexts – see *Tindall v Far North District Council* HC AK CIV 2003-488-000135 20 October 2006 at [138], *Ghuman v Registrar of Auckland District Court* [2004] NZAR 440 at [50] (HC), *Todd Petroleum Mining Co Ltd v Shell (Petroleum Mining) Co Ltd* CA155/05 23 September 2005 at [119] and *Police v Chappell* [1974] 1 NZLR 225 at 227 (SC). Where it is applied, the legal or

ultimate burden remains with the plaintiff, but, in the absence of evidence to the contrary adduced by the defendant, an inference of causation may be drawn.

[63] While *Atkinson* is clear that in New Zealand the legal burden to prove causation in accident compensation cases remains with the claimant and does not shift to the Corporation, the case does not rule out a shift of the evidential burden, in either of the senses set out above at [55], passing to the Corporation. The Canadian Supreme Court approach is for a tactical burden to pass to the defendant when some evidence of causation has been adduced by the plaintiff. It is unclear whether the High Court of Australia was speaking of a true evidential burden or of a tactical burden passing. We favour the view that it is a tactical burden that passes as the Supreme Court of Canada held. Indeed, we endorse that Court's approach, including the reliance on Lord Mansfield's precept, as being applicable in the accident compensation context.

[64] An important factor that favours the Supreme Court of Canada's approach applying in that context is the essentially inquisitorial role of the Corporation, both when an initial claim is made and in the review function – see *Medical Law in New Zealand* at [24.12]. The inquisitorial approach should generally mean that, to the extent this is practical, all aspects of the claim (including causation) have been investigated by the Corporation before matters reach the courts. If that occurs, the situation in *Cochrane v Accident Compensation Corporation* [2005] NZAR 193 (HC) would be avoided. In that case, the medical evidence at the review stages had not been directed to the legal test of causation. As a consequence, a rehearing was ordered in the District Court. That may unfortunately also be the result in this case – see at [113] below. In our view, it is in keeping with the non-adversarial nature of the claim and review process that the Corporation should investigate all possible aspects of a claim, at least in a rudimentary fashion and as far as practicable. It would thus be in a position, once the matter comes before a court, to lead evidence on all points that were investigated, whether strictly obliged to or not.

[65] The requirement for a plaintiff to prove causation on the balance of probabilities means that the plaintiff must show that the probability of causation is higher than 50 per cent. However, courts do not usually undertake accurate probabilistic calculations when evaluating whether causation has been proved. They proceed on their general impression of the sufficiency of the lay and scientific evidence to meet the required standard of proof - see Khoury at 35. The legal method looks to the presumptive inference which a sequence of events inspires in a person with common sense – see Greenberg "The Cause of Disease and Illness: Medical Views and Uncertainties" in Freckelton and Mendelson 38 at 52 and *March v E & M H Stramare Pty Ltd* (1991) 171 CLR 506 at 509 per Mason CJ which was approved in *Sew Hoy & Sons Ltd (In Receivership and in Liquidation) v Coopers & Lybrand* [1996] 1 NZLR 392 at 407 (CA) per Thomas J, *Smith v State Insurance* [1993] DCR 947 at 958 – 959, and *Ross v Accident Compensation Corporation* DC WN AI573/00 23 August 2001.

[66] The legal approach to causation is different from the medical or scientific approach. In *March v Stramare,* Mason CJ at 509 in the High Court explained that the scientific concept of causation has been developed in the context of explaining phenomena by reference to the relationship between conditions and occurrences whereas in law problems of causation arise in the context of ascertaining or apportioning legal responsibility for a given occurrence. At law the cause is not the sum of the conditions which are jointly sufficient to produce the occurrence. See also the discussion of Spigelman CJ at 286 and Stein JA at 294 in *Seltsam,* the comments of Gonthier J in *Laferrière v Lawson* at 656 – 657, those of Sopinka J in *Snell v Farrell* at 300, *Chappel v Hart* at 238 per Gaudron J and at 255 per Gummow J, *Naxakis v West General Hospital* per Gaudron J, *EMI (Australia) Ltd v BES* [1970] 2 NSWR 238 at 242 (CA), Stapelton "Scientific and Legal Approaches to Causation" in Freckelton and Mendelson at 14 and *Personal Injury in New Zealand* at [IP30.04] and [IP155.07].

[67] The different methodology used under the legal method means that a court's assessment of causation can differ from the expert opinion and courts can infer

causation in circumstances where the experts cannot. This has allowed the court to draw robust inferences of causation in some cases of uncertainty – see at [32] above. However, a court may only draw a valid inference based on facts supported by the evidence and not on the basis of supposition or conjecture – see, for example, *Jones v Great Western Railway Co* (1930) 47 TLR 39 at 45 and *Smith v Auckland Hospital Board* [1965] NZLR 191 at 214 (CA) per McGregor J and at 220 per Gresson J. Judges should ground their assessment of causation on their view of what constitutes the normal course of events, which should be based on the whole of the lay, medical, and statistical evidence, and not be limited to expert witness evidence – see Khoury at 203. For a more detailed discussion on inferences, see Khoury at 39 – 43 and 143 – 228.

[68] Spigelman CJ in *Seltsam* said that the only time that a Judge is not able to draw a robust inference of causation are cases where medical science says that there is no possible connection between the events and the injury or death – see at 275. 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. However, if medical science is prepared to say that there is a possible connection, a Judge may, after examining all the evidence, decide that causation is probable. He referred in this regard to the comments of Herron CJ in *EMI (Australia) Limited v BES* at 242. In the case at hand Spigelman J, reversing the trial judge's findings, did not consider the evidence sufficient to infer causation. He was joined in that view by Davies A-JA. Stein JA dissented.

[69] We agree that the question of causation is one for the courts to decide and that it could in some cases be decided in favour of a plaintiff even where the medical evidence is only prepared to acknowledge a possible connection. We refer to *Dais v Accident Compensation Corporation* DC WN 178/2002 5 August 2003, *Smith v State Insurance Ltd* at 959 and *Estate of Albert Francis McQueen v Accident Compensation Corporation* DC HM 190/2005 28 June 2005.

[70] Finally on this topic, we note that the generous and unniggardly approach advocated in *Harrild v Director of Proceedings* [2003] 3 NZLR 289 at [19] (CA) per Elias CJ, at [39] per Keith J and at [130] per McGrath J was used by the High Court

in this case to modify the causation test. This, in our opinion, is not an appropriate application of the principle, given the plain words of the 1998 Act and the rejection of the increased risk test in *Atkinson*. The generous and unniggardly approach referred to in *Harrild* may, however, support the drawing of "robust" inferences in individual cases. It must, however, always be borne in mind that there must be sufficient material pointing to proof of causation on the balance of probabilities for a court to draw even a robust inference on causation. Risk of causation does not suffice.

STATISTICS

[71] It would not be unusual in medical negligence cases for a claimant to be able to point only to a statistical link between particular events and the injury. Although Lord Nicholls was in the minority in *Gregg v Scott*, his comments on the use of such statistics are, in our view, of interest. He said that, in cases of medical negligence, assessment of a patient's loss may be hampered, to greater or lesser extent, by one crucial fact being unknown and unknowable: how the particular patient would have responded to proper treatment at the right time. The patient's previous or subsequent history may assist and there may be other indications, but there will be times when statistical evidence will be the main evidential aid – see at [27] per Lord Nicholls.

[72] Lord Nicholls recognised that statistical evidence is not strictly a guide to what would have happened in a particular case. Statistics record retrospectively what happened to other patients in more or less comparable situations. They reveal trends of outcome and are general in nature. The different way other patients responded in a similar position says nothing about how the claimant would have responded. Statistics do not show whether the claimant patient would have conformed to the trend or been an exception from it. They are an imperfect means of assessing outcomes even of groups of patients undergoing treatment, let alone a means of providing an accurate assessment of the position of one individual patient – see at [28] of his judgment.

[73] Lord Nicholls said that in practice statistical evidence of a diminution in perceived prospects will, despite its imperfection, often be the nearest one can get to

evidence of diminution of actual prospects in a particular case. When there is nothing better, courts should, in his view, be able to use these figures and give them such weight as is appropriate in the circumstances. This conclusion is even more compelling when it is recalled that the reason why the actual outcome for the complainant patient if treated promptly is not known is that the defendant by his or her negligence prevented that outcome becoming known. The value of any statistics will of course depend on their quality: the methodology used in their compilation, how up to date they are, the number of patients involved in the statistics, the closeness of their position to that of the claimant, the clarity of the trend revealed by the figures and so on – see at [32].

[74] Spigelman CJ in *Seltsam* also made some useful comments on this topic, directed to the use to which epidemiological evidence can be put in assessing causation. He said that evidence of possibility, including epidemiological studies, should be regarded as circumstantial evidence which may, alone or in combination with other evidence, establish causation in specific cases – see at 274 - 278. He referred in this regard to the Federal Judicial Centre's *Reference Manual on Scientific Evidence* (1994) which in turn refers to the criteria formulated by Sir Austin Bradford Hill, then Professor Emeritus of Medical Statistics of the University of London, in his Presidential Address to the Section of Occupational Medicine, "The Environment and Disease: Association or Causation?" (1965) 58 Proc R Soc Medicine 295. These criteria are also discussed with approval in Freckelton "Epilogue: Dilemmas in Proof of Causation" in Freckelton and Mendelson 429 at 443 – 444.

[75] Professor Hill's key principles are as follows:

(a) *Statistical Association.* There must be some degree of statistical association between a cause and its effect. In general, the higher the risk estimate, the less likely the finding is a result of confounding or bias.

(b) *Dose Response Effect*. If the risk of the disease rises with increasing exposure, a causal interpretation of the association is more plausible.

Causation is more likely if greater amounts of the putative cause are associated with corresponding increases in the occurrence of the disease or harm.

(c) *Temporality*. The exposure or risk factor must precede the effect. Strength of temporality, such as when a cause immediately precedes its effect, supports an inference of causation.

(d) *Consistency*. Results from other epidemiological studies of the exposure-disease association should be similar. If similar results are found in different populations using various study designs, the plausibility of a causal interpretation is increased. An alternative explanation of bias or confounding would have to apply to each of the different studies, a highly implausible explanation.

(e) *Analogy*. Substantiation of relationships similar to the putative causal relationship increases the likelihood of causation.

(f) *Biological Coherence*. Does the exposure-disease association make biological sense given what is known of the natural history of the disease? Do animal experiments support the association? Do other types of collateral evidence support the association, such as secular trends of the exposure factor in the disease? Spigelman CJ commented in *Seltsam* that unfortunately, for many diseases, little is known about their aetiologies (exposition of the origin of any disease), so the informational background by which to judge biological coherence is often limited. Thus, failure of this broad principle does not necessarily weaken the plausibility of a causal interpretation.

(g) *Experimental Evidence*. Causation is more likely if removing the exposure in a population results in a decrease in the occurrence of disease or harm.

(h) *Specificity*. When there is but a single putative cause for the disease or harm, causation is supported.

[76] We consider that statistical evidence may be of use in the assessment of causation but the limitations of such evidence must be clearly borne in mind. There is always a risk that statistical evidence gives an illusion of precision that is lacking. Statistics as to what has happened to other patients in similar situations are also not necessarily a guide to what might have happened in the case at hand - see at [72]. Such evidence can, however, as pointed out by Spigelman CJ, provide evidence of possibility which might translate into the requisite degree of probability, depending on the strength of that statistical evidence and any other relevant evidence pointing to causation. We did not hear argument on the Hill factors but it might be that they deserve further consideration to see if they might provide a possible framework for assessing the weight to be applied to certain statistical evidence in some cases.

PROXIMITY

[77] Finally in this section, we comment on the emphasis placed by the High Court on the proximity of the medical misadventure and Mrs Ambros' death. Proximity will always be a relevant factor (see at [75](c) above) but its significance in any particular case will depend on all the circumstances.

[78] In some circumstances proximity alone may be deemed sufficient to prove causation. For example, if a person suffers an allergic reaction just after being injected with penicillin (a known risk with penicillin) then, in the absence of evidence of a supervening cause, a court would almost certainly infer causation. Proximity, however, would not suffice where, for example, the reaction was not a known possible result, particularly where there were other possible causes of the reaction. We refer in this regard to the discussion in the vaccination cases of *Loveday v Renton and Wellcome Foundation Limited* [1990] 1 Med LR 117 at 184 - 5 and *Grant v Secretary of the Department of Health and Human Services* 956 F 2d 1144 at 1148 (Fed Cir 1992). By contrast, see *Gardiner v Motherwell Machinery and Scrap Co Ltd* [1961] 1 WLR 1424 at 1429 (HL) referred to in *Dais v Accident Compensation Corporation*.

Should we accept Mr Gray's suggested test?

[79] Mr Gray suggested the modified causation test, set out at [9] above, to deal with the unfairness of requiring a claimant to prove causation in cases of rare diseases where scientific uncertainty is often at its greatest. His test creates a presumption of causation able to be displaced by contrary evidence. As we understand it, Mr Gray's test represents an obligatory presumption that the courts must make, as a matter of law, when certain facts exist and in the absence of conflicting evidence. See Mackay (ed) *Halsbury's Laws of England* (4ed 2002) Vol 17(1) at [576] – [578], Casey *Garrow and Casey's Principles of the Law of Evidence* (8ed 1996) at [18.1] - [18.3], Mathieson (ed) *Cross on Evidence New Zealand Edition* (looseleaf ed) at [1.65] and [4.7] and *Re Berry (a bankrupt)* [1978] 2 NZLR 373 (CA).

[80] While, for reasons explained above (at [64], we consider that a liberal view should be taken of when a tactical burden may pass to the Corporation, we do not consider that the scheme of the legislation would allow a presumption of causation to arise in circumstances where the evidence would not (without the presumption) reach the required standard for proving causation. There is also nothing in the common law developments since *Atkinson* which would support the introduction of a presumption in cases of failures of treatment. Indeed, as has been seen at [23], and following, the approach has been to relax causation requirements rather than to create presumptions. Even the relaxation of causation requirements has been only in very limited circumstances.

[81] Limb (b) of Mr Gray's suggested test is effectively a restatement of the High Court's proximity factor. Limb (a) would presume causation just from the fact of failure of diagnosis or treatment and injury without the need for excluding inevitability of consequence or supervening cause. We agree that proof of these factors may suffice to pass a tactical burden to the Corporation. The factors chosen are similar to those suggested by McHugh J in *Chappel v Hart* – see above at [58]. However, it would be straying even further from the statutory wording and *Atkinson* than the High Court did in this case if those factors (without even the requirement to

rule out supervening cause) are, in the absence of contrary evidence, deemed sufficient to prove causation.

[82] Although, under Mr Gray's formulation, it is only an evidential rather than the legal burden that passes to the Corporation, it is uncertain how much contrary evidence is needed to displace the presumption and, in cases of true uncertainty, it is likely that it would be difficult for the Corporation to displace it. Mr Gray's test would thus pass the burden of uncertainty onto the Corporation rather than the claimant, a position incompatible with *Atkinson* where a reverse onus was rejected and where a risk that the injury was caused was deemed insufficient to prove causation.

[83] Mr Gray's suggested test would also differentiate between different types of medical error. For example, the test does not apply to erroneous treatment but only to failures of treatment. As the same level of uncertainty can apply in each case, there seems no obvious reason to differentiate between the two.

[84] For the above reasons, we do not consider that Mr Gray's suggested test is compatible with the 1998 Act.

What should happen now?

[85] The next issue is what should follow from the fact that the High Court did not apply the correct test for causation. We propose to deal with this under the following headings:

(a) The submissions of the parties.

(b) What was the evidence on causation before the High Court?

(c) What were the factual findings of the High Court?

(d) Was the evidence before the High Court sufficient to prove causation?

(e) Does this Court have jurisdiction to make findings on causation?

(f) Should the matter be referred back to the High Court or the District Court?

The submissions of the parties

[86] The Corporation submits that the factual findings of the High Court lead inexorably to a conclusion that Mr Ambros has not proved causation on the balance of probabilities. The High Court found that Mrs Ambros' death was only possibly avoidable. However, the Corporation recognises that the issue of causation has not been canvassed in any proper depth. It therefore accepts that the matter should be referred back for further evidence to be led on that subject. In the Corporation's submission, the District Court is the proper Court for a reference back as it is the specialist body with regard to ACC appeals and a reference back to that Court will preserve appeal rights.

[87] Mr Ambros submits that he has satisfied the correct test for causation. In support of that submission, he points to both "statistical" and "close proximity" evidence and the fact that there was no evidence of any supervening event. In his submission, the evidence proves that, as his wife had survived the first of her heart attacks for a reasonable period, she would have lived were it not for the negligent failures to diagnose and treat her. If we reject the submission, he submits that the matter should be referred back to the High Court as that Court would be in a position to apply the corrected standard of proof without disturbing any other elements of the case.

[88] Mr Gray, as amicus curiae, submits that it is not appropriate in an appeal by way of case stated for this Court to make the factual findings necessary to assess causation if the High Court test is overturned. In his submission, the matter should be referred back to the High Court for reconsideration in light of the correct test. If the High Court decides further evidence is needed, then Mr Gray agrees with the Corporation that the matter should be referred back to the District Court.

What was the evidence on causation before the High Court?

[89] There were reports from four independent cardiologists in evidence, Dr David McHaffie, Dr Mark Webster, Dr Guy Armstrong and Dr Chris Nunn. These were principally directed to questions of adequacy of care but did touch on some issues relating to diagnosis and treatment.

[90] Dr McHaffie said that, if the medical staff had appreciated that Mrs Ambros did not have the "usual" coronary artery disease (the patchy degenerative condition called atheroma), they would have attached greater urgency than usual to considering angiography. If an angiogram had been carried out on 14 July and if it had demonstrated coronary dissection, it would have been possible to diagnose her condition. Dr McHaffie had earlier said that the series of serum blood tests gave strong support to a diagnosis of myocardial infarction.

[91] Dr Webster reported that SCAD is rare and that it can only be diagnosed by coronary angiography. He said that the natural history of SCAD is even less predictable than that of coronary disease. If the condition had been diagnosed, then anti-anginal medication such as beta blockers might have been administered, although there is little randomised trial evidence that they alter the natural course of the condition. The medications with evidence based efficacy (aspirin and heparin) were in his opinion used on Mrs Ambros appropriately. Dr Webster also said that resuscitation of Mrs Ambros might have been successful if her electrocardiograph (ECG) had been monitored throughout her hospital stay.

[92] Dr Armstrong said that one could speculate that Mrs Ambros' cardiac arrest would have been detected earlier and treated successfully if she had been on telemetry monitoring. Dr Nunn said that Mrs Ambros' death was not the result of treatment. Rather, her very rare condition had progressed in a sudden unpredictable way leading to her death**.**

[93] The statistical evidence referred to by Mr Ambros (see at [87] above) is contained in an article by Koller and others "Immunosuppressive Therapy for Peripartum-Type Spontaneous Coronary Artery Dissection: Case Report and

Review" (1998) 21 Clin Cardiol 40 – 46. This article was cited in the pathologist's report which was in evidence.

[94] The article describes the successful treatment of one SCAD sufferer with immunosuppressive therapy during the postpartum period. The authors had also undertaken a literature review of cases of SCAD in the peri-partum period. They found 42 cases and combined these with their own case. In the resulting sample of 43, there was a mortality rate of 48.8 per cent (meaning a survival rate of 51.2 per cent) – see Koller at 42. Of those who died, 19 died within 24 hours of symptom onset and all deaths occurred within four days – see Koller at 42.

[95] The same article also gives figures for SCAD generally. SCAD presents as sudden death in 50 per cent of cases and acute fatal myocardial infarction in an additional 18 per cent, with an overall survival rate of 33 per cent. In 65 per cent of cases, it is not diagnosed until autopsy – see Koller at 44. The survival figures for SCAD generally thus appear lower than for cases of SCAD which occur peri-partum.

[96] The article notes that treatment for SCAD is possible and that it has shown some good results. Treatment options include angioplasty or stent deployment, coronary artery bypass surgery, thrombolytic therapy and immunosuppressive therapy. However, several authors have noted difficulty in graft replacement of dissected arteries in coronary artery bypass surgery – see Koller at 45.

[97] Dr Hart, Mrs Ambros' consultant, pointed out that one of the authors of the paper is a rheumatologist and that one might wonder what a rheumatologist was doing treating such a case. He said that the rheumatologist was there to use immunotherapy to try and settle the condition "because nobody knows how to treat it".

What were the factual findings of the High Court?

[98] The High Court held that Dr Hart failed to recognise and act promptly upon a series of six serum chemistry tests from 14 – 17 July 2000. In the Court's view, there was a consistent pattern evident by 2.30 pm on 14 July pointing towards a

myocardial infarction. Dr Hart also failed to recognise and act upon the results of an ECG test undertaken on 15 July which showed further signs consistent with a myocardial infarction including ST depression and a T-wave inversion, which by then were evidence of an evolving inferior wall abnormality. Further Dr Hart failed to recognise, following her exercise ECG on 18 July, the seriousness of Mrs Ambros' condition.

[99] The High Court also held that, independently of those failures, Dr Hart's treatment of Mrs Ambros had fallen short of the required standard in three respects. He failed to transfer Mrs Ambros back to the Coronary Care Unit for specialist monitoring following her ECG test on 15 July or, at latest, on 18 July. He also failed to review or reclassify Mrs Ambros' risk profile after the ECG on 18 July or to request urgency for the proposed angiogram at Greenlane Hospital. He also failed to implement any revised management plan designed to lessen the risk of another heart attack pending the proposed angiogram which had been arranged for 20 July.

[100] In the High Court's view, had Mrs Ambros been monitored more carefully, there was a possibility that her death could have been prevented. Appropriate electronic monitoring was likely to have disclosed either a deterioration prior to the second heart attack occurring, or, at the least, the fact that the heart attack had occurred. The Court considered that the matter could not be put more highly than a possibility that death might not have occurred, given the rare disease from which Mrs Ambros was suffering.

[101] The High Court made no specific factual findings on whether an earlier angiogram should have been ordered and on whether such an angiogram would have allowed a proper diagnosis of Mrs Ambros' condition to have been made. Nor did it make any findings on whether an earlier diagnosis would have led to a different treatment regime or as to the likely effects of any such treatment.

Was the evidence before the High Court sufficient to prove causation?

[102] In this case it is accepted that there were only two possible causes of Mrs Ambros' death. Either she died as a result of her underlying condition (SCAD)

or she died as a result of medical error by a failure to diagnose, monitor and treat her appropriately. In order to succeed, Mr Ambros is required to prove the latter on the balance of probabilities. Has he done so?

[103] While its factual findings were sufficient for the High Court to conclude that causation was proved on the basis of its test (as set out at [4] above), finding that it was possible that Mrs Ambros may have been saved does not equate to a finding that she would more likely than not be alive had it not been for the medical errors made.

[104] The issue therefore is whether Mr Ambros is right and the evidence before the High Court proved causation even in accordance with the correct test. As Mr Gray pointed out, on the basis of the statistics in the article referred to above at [93], Mrs Ambros had at least a 51 per cent chance of survival. This means that her survival was more probable than not. Indeed, Mr Gray submits that, as Mrs Ambros had survived for six days without diagnosis or treatment, her chance of survival (based on the evidence in that article where all who died did so within four days) was arguably even higher.

[105] The High Court, because of its formulation of the test of causation, made no findings based on the statistical evidence contained in the article. Both the pathologist and Dr Hart, however, appeared to accept in evidence that Mrs Ambros' case could be equated to those discussed in the article. There are limitations with the statistics. First, the sample used in the article was small but that appears to be because the condition is so rare. Indeed, Dr Hart in evidence suggested that the article covered all reported pregnancy related cases at the time. Thus the sample is not skewed even if the numbers are small. Secondly, and most importantly, however, the relationship between treatment and survival is not clear. For a start, there does not appear to be a recognised preferred treatment for the condition and, apart from the article which refers to good results in some cases from treatment, there was no evidence before the Court on the effectiveness or otherwise of any possible treatment. Further, while it is safe to assume that those whose condition presented as sudden death had not had treatment, that cannot be assumed for the others who died. Neither can it necessarily be assumed that those who survived for

more than four days did so because of any treatment administered rather than because of the natural course of the condition itself.

[106] On the other hand, the article states that 19 of the 21 women who died, did so within 24 hours of the onset of symptoms. The other two died within four days. Sudden cardiac death was the initial presentation in 12 of the cases. Therefore only nine of the women had a chance to receive treatment but there must be some doubts on how effective any treatment would have been for those who died within 24 hours. There were no special pre-disposing factors identified in the article to distinguish those who died from those who lived and there was nothing in the evidence to suggest that there were special factors relating to Mrs Ambros (for example, pre-existing illness) that would take her outside of the normal run of cases of pregnancy related SCAD. We recognise that Mrs Ambros had elevated blood pressure on her admission to hospital but there is nothing in the article that suggested that this would cause her to be at greater risk of death. Mrs Ambros too can be considered very unusual in surviving for six days after the onset of symptoms and then dying.

[107] In our view, despite the limitations of the article, a combination of the statistics in that article, the proximity between the omissions and Mrs Ambros' death, the availability of possible treatment and the increased monitoring had her condition been diagnosed properly sufficed to make the question of causation arguable. There was thus a tactical burden on the Corporation to adduce evidence on causation, even if a less liberal view is taken of when a tactical burden might pass to the defendant than McHugh J suggests in *Chappel v Hart* – see at [58] and [79] - [83]. The absence of counter evidence from the Corporation would have allowed the High Court to assume that no such counter evidence existed but this would not necessarily have led to a finding in Mr Ambros' favour. The High Court could still have found, even on the basis of drawing a robust inference, that the evidence was insufficient to prove causation to the requisite standard.

[108] Mr Ambros has submitted that this Court should make the requisite findings on causation. This raises the issue of whether we have jurisdiction to do so.

Does this Court have jurisdiction to make findings on causation?

[109] Section 166(1) of the 1998 Act provides that a party to an appeal before the High Court may, with the leave of the High Court, appeal to this Court by way of case stated for the opinion of that Court on a question of law only. Section 166(3) provides for such appeals to be dealt with in accordance with the rules of this Court, being currently the Court of Appeal (Civil) Rules 2005. Rule 47 provides that all appeals are by way of rehearing. Rule 48 gives a wide range of powers to the Court when hearing and disposing of appeals.

[110] At first blush, there is a conflict between the application of the rules and the case stated procedure. As generally understood, a right of appeal by way of case stated is not a right of appeal to this Court by way of rehearing. It is a form of consultation with the appellate court to obtain an answer on a point of law - see *Harris Simon & Co Ltd v Manchester City Council* [1975] 1 WLR 100 at 105 per Lord Widgery CJ (QB).

[111] It seems to us that, in order to resolve the possible conflict, the rules must be read in a manner that is consistent with the nature of a case stated. This is particularly the case as any appeal to this Court is confined to a question of law. This means that it would be inappropriate for us to undertake a full review of the evidence in order to make factual findings on causation. We should limit ourselves to answering the question in the case stated and referring the matter back to be resolved in accordance with our opinion.

[112] Even if we had jurisdiction to make findings on causation, however, we would not have considered it appropriate to do so in the absence of full argument and when the necessary underlying factual findings (see at [101] and [107] above) have not been made by the courts below.

Should the matter be referred back to the High Court or the District Court?

[113] Given that the case was stated by the High Court, we consider that the matter should be referred back to that Court. Further, Mr Ambros should have the

opportunity to have the High Court assess his argument that causation has already been proved to the requisite standard. If, however, the High Court decides that the parties should be allowed to adduce further evidence it seems to us, for the reasons given by the Corporation (see at [86] above), that the most suitable course would be for the High Court to refer the matter back to the District Court**.**

Result and costs

[114] The appeal is allowed. The answer to the question set out at [6] above is in the negative.

[115] The matter is referred back to the High Court to be dealt with in accordance with the principles set out in this judgment.

[116] No costs were sought by the Corporation and none are awarded.