The Challenge of Medical Uncertainty: Factual Causation in Anglo-Australian Toxic Tort Litigation

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1. INTRODUCTION: CONCEPTS OF CAUSATION

A waterside worker develops mesothelioma. It is caused, beyond any doubt, solely by asbestos dust to which he has been exposed while loading and unloading ships. His employers over the years, a series of stevedoring companies, admit negligently exposing him to the dust. But nobody knows the casual mechanism by which asbestos dust causes mesothelioma. Is it triggered by a single unit of exposure, a single fibre perhaps, or have all the fibres of asbestos combined to cause the disease? Can the party which caused the disease be identified?

A woman develops leukaemia. Twenty years previously there had been a leak, negligently caused, at a nuclear reactor one hundred kilometres from her home. Leukaemia has never been an uncommon disease in the community but in the last few years there has been observed a considerable increase in the incidence of the disease. Can the woman identify the negligent authority as the source of the disease?

These two examples illustrate two simple types of toxic tort.[1] In the first, which is a type common in the workplace, the substance causing the disease is known and the negligence of the defendants is established. The problem is that medical science knows no more than that the onset of the disease is dose-related, in other words that increased exposure means increased risk. But unless it is agreed how a substance causes a disease, it is difficult to prove whether it did so in a particular case or whether a certain party's negligence can be held responsible. Medical uncertainty poses problems for plaintiff and court.

In the second example the problem is both that the disease-causing mechanism is likely to be unknown and that the offending substance itself, though suspected, cannot be identified with certainty. It is known that the cancer in

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[1] In this article the term *toxic tort* will be used to denote a tort involving harm caused by exposure to pathogenic substances. The word *substance* is interpreted broadly and includes emanations from toxic substances, for instance radiation. The words *toxic* or *pathogenic* denote *disease-causing* under the circumstances obtaining at the time. Thus oxygen can be a toxic substance under certain circumstances (*Wilsher* v *Essex Area Health Authority* [1988] 2 WLR 557). The term *toxic tort* is used in a broader sense than it generally is in US writings whence it has been borrowed. Thus the injury may or may not be traumatic; exposure may or may not be chronic and repeated; and injury may or may not manifest itself after a latency period. Although a distinction may be made between *toxic* and *carcinogenic* this distinction will not be made here.

question occurs in unexposed populations. It is also known that the incidence of the cancer increases in exposed communities. How can it be established that a *particular* cancer was caused by the defendants? This is a problem typically faced both in environmental pollution and product liability cases. Again, medicine does not have the answers.

While toxic torts are simply unintentional torts to the person, their significance is that they push at the frontiers of causation doctrine, challenging settled assumptions in this area of the law by bringing into focus states of affairs which used to be regarded as exceptional. The law is able to respond in a variety of ways which will be discussed in the course of this article. Most of these have been developed in response to more conventional situations but have found new application in the toxic tort area. Before dealing with judicial techniques in confronting these causation problems, however, it is worth looking at the concept of causation itself.

What is cause?

On one level *cause* is merely a word or symbol. It is a neutral shell for meaning. The meaning which fills it is conceptual and therefore has an intrinsic volatility. Furthermore, the word symbolises not one concept but a spectrum of concepts so that any two uses of it may carry entirely different meanings. As Morris said in 1939: 'The word *cause* is almost its own antonym'.[2]

At one end of the spectrum it is the word given to the infinitely complex web of circumstances which issue in any particular event. To assert cause in this context is to assert little more than that a lineal sequence of events is not random. At the other end of the spectrum it is the single most easily apprehensible reason why a certain event occurred. It is the reason which it suits the speaker to name at the time of speaking. Thus the word has elements both of objectivity and subjectivity. Philosophers tend to align the word with its most objective meaning. Science narrows it in the search for predictable outcomes and aligns it with the mechanism which will always produce a certain outcome under given circumstances. This predictive function is of limited interest to lawyers whose wish is to trace origins within reasonable bounds and to ignore the literally infinite fanning out of causes which the rigour of the philosopher reveals. They will use it both to serve and to reinforce a community concept of justice.

Causation and proof

If one is to say that causation exists in a particular situation one must, since the meaning of causation is elusive, articulate the tests used in coming to the decision. But this articulation is itself not illuminating in a legal context unless one also states what it will take to pass the test. Evidence rather than argument is the crucial factor in establishing factual causation in legal cases

[2] C Morris, 'On the Teaching of Legal Cause' (1939) 39 *Columb L Rev* 1087, 1094.

and their consistent focus is on what type of evidence will lead the tribunal to a point where the standard of proof is satisfied and an inference of causation can be drawn. Causation, like many other concepts, has no legal existence until it has been inferred to exist, until a court has been satisfied of its existence on the balance of probabilities. Though a situation may fall far short of satisfying a scientist that causation is present it may nevertheless satisfy a judge. From that moment on, causation *will* exist for legal purposes.

Factual causation and proximate cause

It is traditional to differentiate between factual causation and proximate cause.

Factual causation is in essence positive and attributive. Its modus operandi is more philosophical than political, more objective than subjective. It sorts out the relevant from the irrelevant, using more or less objective criteria. Working within the framework of an allegation of, for instance, negligence, it determines whether the defendant's act or omission in any sense produced the harm alleged. It does not determine whether he should be punished. Without a finding of factual causation any consideration of proximity is otiose.

Tests of proximity are equally essential but temporally secondary. They are exclusionary and political and subject to accepted ideas of justice. They negate absolute liability and classify the importance of the defendant's act. Unless the defendant's act was a sufficiently direct cause of the harm, he will not be deemed to have caused or contributed to it.

Together these elements add up to the legal concept of cause, a highly idiosyncratic and pragmatic one. Concepts of factual causation show a borrowing from philosophy and science; concepts of proximity show the law's determination to make the word *causation* mean whatever the law deems it appropriate that it should mean within the bounds of its 'factual' definition.

Why does causation matter?

Causation is an essential requirement for the establishment of liability in many areas of law. Our concepts of natural justice demand this. In a legal system which compensates the plaintiff at the expense of a defendant it is considered only fair to ensure that the defendant was the originator of the harm in an acceptably direct sense. This attributive element functions as a guard against liability based on nothing more than a 'general moral deficiency' — the breach of the duty of care alone.[3] That we should take responsibility for the consequences of our acts is a well-established principle; that we should pay for acts not our own is an evil to be avoided as far as possible and not, on ordinary concepts, to be enshrined in a legal system.

[3] See E J Weinrib, 'A Step Forward in Factual Causation' (1975) 38 MLR 518.

Tests of factual causation

In the face of medical uncertainty the adequacy of accepted legal mechanisms and definitions, including the causation mechanism, is thrown into doubt. Courts are faced with plaintiffs who have been damaged in situations often clearly involving the negligence of others, but because of medical/scientific uncertainties, are unable positively to attribute the damage to the defendant. The courts are generally sympathetic but can only use the tools which they have, occasionally in remodelled forms.

In testing for causation the tool that is most ready to hand is the but-for test. Would a certain event have occurred but for a particular act or omission of the defendant or but for his negligence? The test has been taken so much for granted that it has been identified with factual causation. This identification is, however, problematic.

If we are to say that whatever emerges from the application of the test is by definition the cause, and that nothing else can be the cause, we are left with results that militate against traditional notions of justice. We have a disjunction between the word (bearing the meaning invested in it by the test) and the function it is intended to perform. Thus, in cases of overdetermined causation, where a factor other than the specified act would have been sufficient to cause the outcome without the specified act playing any role at all, the result of the test's application is that nobody can be liable. These cases might involve pre-emptive causation (where the effects of the specified event pre-empt an identical outcome) or duplicative causation (where injury is produced jointly by two or more acts sufficient in themselves to produce the harm but either combining with each other or duplicating each other's effects). Thus, if A shoots B just before B was about to be run over by a train, or if C and D light two separate fires, each sufficient to destroy E's home, and in fact they combine to destroy it, neither A nor C nor D is liable because the outcome (death, a burnt-down house) would have occurred in the absence of their acts. Their acts were not necessary to produce the harm.

Such problems are acute in toxic tort cases where instances of duplicative causation in particular are frequent.

The unease of writers with this state of affairs has found expression in attempts to modify, supplement, redefine or replace the but-for test, or to sidestep it in some other way.

Managing the but-for test

Some writers modify the test by applying it to a detailed description of the injury or the manner of its occurrence, maintaining that the injury would not have occurred at the time or in the manner or with the severity that it did but for the negligent act or omission of the defendant.[4]

[4] See R Perkins, *Criminal Law* (2nd ed, 1969) 689 (mentioned by R W Wright in 'Causation in Tort Law' (1985) 73 *Cal L Rev* 1735, 1778) and A Becht and F Miller, *The Test of Factual Causation in Negligence and Strict Liability Cases* (St Louis, Missouri, Washington University Studies, 1961) 15–17.

Others apply the test to multiple potential causes in the aggregate:

> When the conduct of two or more actors is so related to an event that their combined conduct, viewed as a whole, is a but-for cause of the event, and application of the but-for rule to them individually would absolve all of them, the conduct of each is a cause in fact of the event.[5]

Akin to the above is the addition of *material contribution* to the definition of the causal requirement. Thus the plaintiff has to prove on the balance of probabilities that the defendant's negligence caused *or materially contributed* to the injury suffered. Just what *material contribution* might mean is discussed in section 2 of this article.

Some commentators either refine or sidestep the but-for test by recourse to common sense:

> The common law tradition is that what was the cause of a particular occurrence is a question of fact which "must be determined by applying common sense to the facts of each particular case".[6]

Thus, where the but-for test reveals an infinite number of conditions without the existence of which an event would not have occurred, common sense is used to seek out its dominant or effective cause. In effect the test is one of proximate cause rather than of strictly factual causation.

Alternatively, in circumstances where the but-for test is inappropriate, common sense can fill the breach by identifying a cause according to commonsense principles. The problem is that common sense is essentially a subjective quantity, offering no fixed criteria for a finding of causation, and while its flexibility may be superficially attractive, it is too reliant on the expertise of the tribunal to be of use in medical cases where the tribunal has little or no expertise.

The main contender to replace the but-for test is the NESS test (though Australian courts tend to conflate it with the but-for test).[7] The letters denote Necessary Element of a Sufficient Set. This idea derives from the writings of J S Mill and Hart and Honoré[8] and is now propounded particularly by Richard Wright in the US.[9] It has recently been adopted by Street[10] and by Fleming.[11]

Like the but-for test, this test of factual causation is based on a concept of necessity. But the necessity is at one further remove from the injury. Instead of asking whether an act or omission was necessary for the occurrence of an

[5] W Keeton, D Dobbs, R Keeton and D Owen, *Prosser and Keeton on Torts* (5th ed, St Paul, Minnesota, West Publishing Co, 1984) 268, para 41.

[6] This judicial formulation of the position was put foward by Mason CJ in *March* v *E and MH Stramare Pty Ltd* (1991) 65 ALJR 334, 338 quoting Lord Reid in *Stapley* v *Gypsum Mines Ltd* [1953] AC 663, 681.

[7] See, for example, the judgment of Mahoney JA in *Barnes & Ors* v *Hay* (1988) 12 NSWLR 337, 354.

[8] J S Mill, *A System of Logic Ratiocinative and Inductive* (8th ed, London, Longman, 1970) and H L A Hart and T Honoré, *Causation in the Law* (2nd ed, Oxford, Clarendon Press, 1985).

[9] R W Wright, 'Causation in Tort Law' (1985) 73 *Cal L Rev* 1735, 1788.

[10] M Brazier, *Street on Torts* (8th ed, London, Butterworths, 1988) 223.

[11] J G Fleming, *The Law of Torts* (7th ed, Sydney, The Law Book Co Ltd, 1987) 173.

event, one asks whether it was necessary for the creation of a set of conditions which was sufficient on its own to produce the injury and was actually operating on the plaintiff. Thus where there are multiple sufficient causes, there are multiple sufficient sets of conditions, and any condition without which a particular set could not have been operative will be deemed a cause of the event. The test also covers multiple insufficient causes:

> In the pollution cases, the NESS test confirms that each defendant's pollution contributed to the injury, even though it was neither necessary nor independently sufficient for the injury. For example, assume that five units of pollution were necessary and sufficient for the injury and that each of seven defendants discharged one unit of pollution. Each defendant can truthfully say that its one unit was neither necessary nor independently sufficient for the injury. But each defendant's one unit was necessary for the sufficiency of *a* set of actual antecedent conditions that included only four of the other units, and the sufficiency of this particular set of actual antecedent conditions was not affected by the existence of two additional duplicative units.[12]

The NESS test is an ingenious answer to the shortcomings of the but-for test. It is also pragmatic since it broadens the area that the causation net covers without stating formally the principles on which it is operating. It is suggested that these principles are in fact principles of contribution, which will be discussed below.

These, then, are the major positions, some more highly formalized than others. In the courts one sees a favouring of the more pragmatic, less formalized positions. Anglo-Australian courts, for instance, have made heavy use of the but-for test tempered by 'common sense' or modified according to the circumstances. They have also been prepared to use the concept of the contributory cause.[13]

Finally, it should always be borne in mind that the whole legal system is coloured by the overriding standard of proof. Theoretical writers assume an absolute quality of causation. The legal system, however, knows nothing of such absolutes and concerns itself, in the civil context, only with probable causation. In effect it defines *causation* as *probable causation.*

Structure of this article

This article will consider four areas in which courts have responded to situations of medical uncertainty or where they might do so.

The first area is the concept of causation itself and the increasingly often used related concept of material contribution. The latter term is ill-defined and this article will attempt some clarification of the senses in which it has been used and assess the consequences of its application.

The second area is that of evidence. The role which expert evidence should play in decision-making has been much discussed in case law, the main issues being whether and when it should give way to a commonsense interpretation

[12] Wright, op cit 1792–3.
[13] See section 2 of this article.

of circumstantial evidence and under what circumstances an expert assessment of *possible* causal linkage can give rise to a judicial finding of *probable* causal linkage. In cases where medical certainty is not possible these are crucial issues. Furthermore, it is proposed to discuss the willingness of courts to draw inferences and form presumptions concerning disease-causing mechanisms.

The third area concerns the civil standard of proof — the balance of probabilities. In many toxic tort cases the only evidence which can be offered is probabilistic; the plaintiffs can establish no more than that the defendant exposed them to an increased risk or probability of contracting a disease. This situation obtains in the case of our leukaemia victim. What must be asked is whether evidence expressed as probability can satisfy the requirement of evidence establishing a casual link on the balance of probabilities or whether a more direct attribution of damage to the defendant must be proved.

Finally it will be suggested that problems of medical indeterminacy might be resolved through a redefinition of the damage which the plaintiff is required to prove.

2. TESTING FOR CAUSATION

THE CONCEPT OF MATERIAL CONTRIBUTION: CUMULATIVE EFFECTS AND CONCURRENT CAUSATION

The waterside worker with mesothelioma was negligently exposed to asbestos dust by a series of employers. One may argue that if there is no evidence to establish that one of those employers was the predominant cause of the injury or that injury would not have been sustained but for the actions of that employer, then liability cannot be established. A number of recent cases have been decided on these grounds.[14] Increasingly, however, courts have evinced dissatisfaction with this type of reasoning and its rigid adherence to the but-for test, and have embraced a concept of concurrent causation. In doing so they have shown regard for scientific reality where substances, particles of substances, or periods of exposure to them are constantly acting in concert with each other or duplicating or exacerbating each other's effects. The but-for test in its classical form is peculiarly unsuitable for such situations due to its high level of specificity. In calling for the identification of precise causative factors or links with a particular defendant and insisting that recovery cannot take place unless they are proved to be essential to the onset of a condition, it requires detailed information concerning the aetiology of the disease. This information is often not available.

In a British line of cases starting in the middle of this century the House of Lords showed itself acutely aware of the problems faced by plaintiffs in work-induced toxic tort cases. Its response was multi-faceted, one aspect of it being

[14] See, for example, *Chance* v *Alcoa*, unreported decision, District Court, Western Australia, 22 June 1989 and *Wintle* v *Conaust (Vic) Pty Ltd & Ors* [1989] VR 951.

to despecify that which had to be proved. This was done through the assertion that the plaintiff had to prove, on the balance of probabilities, not that the defendant *caused* the injury but that the defendant *materially contributed to* it. It is arguable that to establish material contribution is not as onerous a task as to establish but-for causation, but this will depend on the definition given to *material contribution.*

Difficulties with the term 'material contribution'

The concept of material contribution is ill-defined. The word *material* is defined in a way which begs for further definition and the word *contribution* is interpreted in two fundamentally different ways. Case law shows that each of these interpretations is subject to further categorization.

The first, more radical and more generalized, meaning of the word *contribution* looks beyond causation of the disease itself to causation of certain of the background conditions which caused the disease. A type of indirect causation is at issue here and there is little evidence of the but-for test being used to establish it.

This meaning of the word embraces such concepts as contribution to the aggregate of a disease-producing substance, to the total exposure to that substance or to other background conditions which promote the disease. There is no need for the contribution to play an individually decisive role in the genesis of the disease.

The second, more conventional, interpretation of the word has it describing partial causation of the disease itself or causation of its timing or severity. The but-for test is used to establish this type of causation, though it may be used in a modified form. This meaning embraces the concepts of precipitating contribution, accelerating contribution and aggravating contribution.

A precipitating contribution is some exposure to a harm-causing substance in excess of previous or concurrent exposure. While it is itself insufficient to cause the disease, it has the effect of *tipping the balance*, ie combining with the insufficient previous exposure to precipitate the onset of the disease. It is truly a but-for cause of the disease even though the precipitating exposure may be very small.

An accelerating contribution is some exposure which hastens the onset of the disease. As it is a necessary condition of the timing of the disease, it satisfies a modified form of the but-for test.

An aggravating contribution is a condition which is necessary to the manifestation of the disease at a particular level of severity. Thus it also satisfies a modified form of the but-for test.

The case law which has thrown up these competing interpretations has not discussed their relative merits nor apparently recognised the fundamental differences between them. As it is, they exist silently side by side in the same cases. It is to be hoped that they will receive more judicial discussion in the future.

The following is a discussion of the major British and Australian cases, indicating the development of, and support for, the various notions of

material contribution. The concept came to the fore in the UK in the decision of the House of Lords in *Bonnington Castings Ltd* v *Wardlaw.*[15]

The rise of the contribution principle in Britain

Bonnington was the case of a steel dresser who had contracted pneumoconiosis as a result of his employment at the defendants' foundry. The condition was attributable to dust from two sources: a pneumatic hammer and swing grinders. The worker took action against the employers for breach of statutory duty and common law negligence in exposing him to dust from the swing grinders. While most of the worker's exposure had come from the hammer it was not established that any negligence on the employers' part caused this exposure. In the case of the swing grinders, however, negligence was established. The issue then arose of whether the employers' breach caused the disease.

Viscount Simonds, in commenting on the burden and standard of proof in such cases, held that the 'ordinary standard of proof in civil actions'[16] was that the plaintiff must prove on the balance of probabilities that the breach of duty caused or materially contributed to the injury.[17] Since the House made no distinction in this regard between statutory and common law breaches, its comments can be taken to apply to causation in all types of civil cases where there are no statutory indications to the contrary.

The defendants argued in *Bonnington* that to establish causation the plaintiff would have to show that more dust came from the swing grinders than from the pneumatic hammer, ie to prove on the balance of probabilities that it was more likely than not that dust from the grinders caused the condition. They were taking a traditional position which precluded the notion of concurrent causes.

The court, however, endorsed the plaintiff's argument that a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years was the cause of the disease. It was enough if the defendants could be shown negligently to have contributed a part of the dust as long as that part was not negligible. They would thus materially have contributed to the development of the disease.

Definition of 'material'

In this case Lord Reid gave the word *material* a definition which has been used repeatedly in later cases:

> The real question is whether the dust from the swing grinders materially contributed to the disease. What is a material contribution must be a question of degree. A contribution which comes within the exception

[15] [1956] AC 613.
[16] Id 620.
[17] In assenting to this the Court overruled the carlier authority of *Vyner* v *Waldenberg Brothers Ltd* where it had been held that in cases where breach of statutory duty had occurred 'the onus of proof shifts on to the [defendant] to show that the breach was not the cause [of the injury]' [1946] KB 50, 55.

> de minimis non curat lex is not material, but I think that any contribution which does not fall within that exception must be material.[18]

This definition was reiterated by the House in *Nicholson* v *Atlas Steel Foundry & Engineering Co Ltd*,[19] a case involving almost identical facts to those of *Bonnington*.

Lord Reid further admitted that probably '*much the greater proportion* of the noxious dust'[20] came from the hammers, but that the 'quota' which came from the swing grinders was enough to be deemed a material contribution, thereby indicating that relatively small quantities might satisfy the *de minimis* principle. The concept cannot, however, be said to have been satisfactorily defined.

Definition of 'contribution'

Lord Tucker posited most clearly the more radical interpretation of the word *contribution*:

> The inference to be drawn from these facts is that the silica dust discharged from the swing grinders *contributed to the harmful condition of the atmosphere*.[21]

There is no suggestion that a but-for test is being used. Lord Reid, in emphasising the 'quota' of silica dust which was contributed to the 'general atmosphere' and hence found its way into the plaintiff's lungs, would appear to have supported Lord Tucker's interpretation.

Lord Keith of Avonholm, however, appeared to opt for the more conventional interpretation and its associated use of the but-for test. He stated:

> I think the natural inference is that had it not been for the cumulative effect [of the 'guilty' particles added to the 'innocent' particles] the pursuer would not have developed pneumoconiosis *when he did* and *might not have developed it at all*.[22]

Thus we already see three interpretations being posited — contribution as an addition to a harm-causing aggregate (per Lords Tucker and Reid), contribution as acceleration, and contribution as precipitating cause (per Lord Keith). The first would appear to be dominant.

In *Nicholson* the House of Lords endorsed the *Bonnington* material contribution principle, presumably the broader and dominant interpretation of the term. Viscount Simonds, however, apparently embracing the view that the but-for test does underlie the material contribution idea, implied that material contribution could be linked with aggravation of a condition.[23]

[18] *Bonnington* 621.
[19] [1957] 1 WLR 613.
[20] *Bonnington* [1956] AC 613, 622 (emphasis added).
[21] Id 623 (emphasis added).
[22] Id 626 (emphasis added).
[23] *Nicholson* [1957] 1 WLR 613, 617.

McGhee v *National Coal Board*

In this case[24] the House of Lords both filled out the reasoning in *Bonnington* and *Nicholson* and extended it. Here again the judges took different views of contribution.

This was a case concerning dermatitis contracted by a workman engaged in emptying brick kilns. It was admitted that the dermatitis was attributable to his work in the kilns. The plaintiff alleged that the employers, in failing to provide adequate washing facilities, thus forcing him to cycle home covered in dust, had breached their duty of care. The employers admitted the breach of duty but contended that it was not proved that it had caused the onset of the disease.

Medical witnesses agreed that the dermatitis was caused by repeated minute abrasion of the outer horny layer of the skin followed by some injury to or change in the underlying cells, the precise nature of which had not yet been discovered by medical science. Sweat and exertion during a period when the skin remained unwashed would exacerbate the problem. The effect of abrasion of the skin was cumulative, in the sense that the longer a subject is exposed to injury, the greater the chance of his developing dermatitis.

The two views of contribution were represented by the judgments of Lords Wilberforce and Simon.

Lord Wilberforce, in adopting the judgment of Lord Keith of Avonholm in *Bonnington* (who held that the natural inference was 'that had it not been for the cumulative effect the pursuer would not have developed pneumoconiosis when he did and might not have developed it at all'),[25] appeared to endorse the use of a but-for test. Though he noted that there was no proof that but for the addition of the 'guilty' dust the disease would not have been contracted, he remarked that this problem was overcome by inference.[26]

On the other hand, Lord Simon of Glaisdale indicated that he did not see the notion of necessity in the *Bonnington* test. To his mind the case established

> that where an injury is caused by two (or more) factors operating cumulatively, one (or more) of which factors is a breach of duty and one (or more) is not so, in such a way that it is *impossible to ascertain* the proportion in which the factors were effective in producing the injury or *which factor was decisive*, the law does not require the . . . plaintiff to prove the impossible, but holds that he is entitled to damages for the injury if he proves on a balance of probabilities that the breach or breaches of duty contributed substantially to causing the injury.[27]

This passage states clearly the more radical interpretation of *contribution*

[24] [1973] 1 WLR 1.
[25] *Bonnington* [1956] AC 613, 626.
[26] *McGhee* [1973] 1 WLR 1, 7. In fact the only express inference that was made by Lord Keith, on the balance of probabilities, was that the 'guilty' dust had materially contributed to the disease and/or that it had accelerated the onset of the disease. The words 'might not have developed it at all' do *not* indicate any satisfaction of the civil standard of proof.
[27] Id 8 (emphasis added).

and was quoted with approval in the 1988 House of Lords decision in *Wilsher* v *Essex Area Health Authority*.[28] Previously, however, in the Court of Appeal decision in the same case, Mustill J, in referring to *Bonnington*, apparently considered that the notion of necessity had been present: 'The uncertainty was whether the fault had *tipped the scale*'.[29] And in *Kay* v *Ayrshire and Arran Health Board*, Lord Griffiths of the House of Lords included the idea of aggravation in that of causation, thus indicating that it might be part of the definition of material contribution.[30]

On balance, though *Bonnington* seems to stand for the broader, more radical view of contribution, there is still support for more restricted readings.

Australian cases

In Australia *Bonnington* has been taken, in the toxic tort cases, to stand for the wider interpretation of *contribution* (though the narrower view has been posited in other types of cases).[31]

In *Power* v *Snowy Mountains Hydro Electric Authority*,[32] decided the year after *Bonnington*, the New South Wales Supreme Court in banco decided in favour of the plaintiff explicitly on *Bonnington* principles.

The plaintiff was already suffering from silicosis. Due to the negligence of his defendant employers he was exposed to more additional silica dust than he should have been, aggravating his condition. Evidence showed that contact with silica dust is more serious for a person who has silicosis than for one who has not. It is unclear from the report whether the plaintiff was seeking compensation for his present condition of silicosis or merely for the aggravation of the disease. If the former were the case, then the plaintiff would have been seeking full recovery on proof of a contribution to a contributing cause.

At any rate, a new trial was ordered on the basis that the negligence of the defendants had contributed to the pathogenic state of the atmosphere and hence to the plaintiff's condition.

In *Thompson* v *Johnson and Johnson Pty Ltd & Anor*, a case of toxic shock, Vincent J of the Supreme Court of Victoria stated:

> The evidence does not suggest that tampons of themselves are responsible for [the development of toxic shock] but that they contribute to the estab-

[28] [1988] 2 WLR 557, 568 per Lord Bridge of Harwich. It should be noted, however, that *Wilsher*, while endorsing a broad reading of *McGhee* in respect of contribution principles, has been instrumental in establishing other principles in *McGhee* as being narrower than has on occasion been believed. See the discussion in section 3D of this article of the 'harm-within-the-risk' concept.

[29] *Wilsher* v *Essex Area Health Authority* [1987] QB 730, 752.

[30] [1987] 2 All ER 417, 422.

[31] See, for example, the judgment of McHugh JA in *Alexander & Ors* v *Cambridge Credit Corporation Ltd (in receivership) & Anor* (1987) 9 NSWLR 310, 352 where he states that 'there is no conflict between the "material contribution" requirement and the application of the "but for" test in a practical, commonsense manner as the exclusive test of causation'.

[32] (1957) 57 SR (NSW) 9.

lishment of an environment which is conducive to the production of the toxin.[33]

This was enough to establish a casual link in this case[34] and is similar to the reasoning in *Bonnington*. The concept of *tipping the balance* is not stated to be significant here.

The situation was similar in *Chance* v *Alcoa* (Supreme Court of Western Australia),[35] a case of dry eye which might either have been caused by caustic soda burns or might have been idiopathic or both. Here it was medical evidence which stated that the burns could have made a contribution to the onset of the condition but no view was stated as to whether the burns were a precipitating cause. The medical evidence was accepted expressly on *McGhee* (and hence *Bonnington*)-type reasoning.

Contribution: a separate path of development

Independent of the *Bonnington* line of cases but covering similar ground and again supporting a broader reading of *contribution* are the Australian case of *Barker* v *Permanent Seamless Floors Pty Ltd*, a 1983 decision of the Full Court of the Queensland Supreme Court,[36] and its British predecessors.

In *Barker* the respondent became aware in 1980 that he was suffering from toxic hepatic necrosis and that this condition was consistent with exposure to five toxic chemicals, to all of which he had been exposed since 1976. The appellant was Permanent Seamless Floors, his employer. Ciba Geigy, which had supplied one of the chemicals, was also an appellant. Connolly J stated:

> The medical evidence is not conclusive but it suggests at least the probability that the disease is the result of the cumulative effect of constant exposure to the chemicals in question.[37]

Ciba Geigy had claimed that as it had supplied only one of the five toxic chemicals to which the respondent was exposed, and as any one of the other four might have caused or contributed to the damage, there was no evidence to establish a cause of action against Ciba Geigy. The supplier was effectively relying on the but-for test and on the evidential impossibility of proving conclusively that *it*, the supplier, caused the damage.

In dismissing this reasoning Connolly J turned to a line of nuisance cases stretching into last century. In *Blair and Sumner* v *Deakin*, a pollution case before the Chancery Division, Kay J considered that the law would be most unjust if it stood for the proposition that none of the contributors to a nuisance could be sued if it were found than none of them singly poured 'into this stream foul matter enough by itself to create a nuisance, but . . . what they all pour in together does create a nuisance'.[38]

[33] [1989] Aust Torts Reports 80–278, 68 959.
[34] Though the plaintiff ultimately failed on other grounds.
[35] [1990] Aust Torts Reports 81–017.
[36] [1983] 2 Qd R 561.
[37] Id 562.
[38] (1887) 57 LT 522, 525.

A more illuminating case still is *Thorpe* v *Brumfitt*, a dispute over a right of way. Here James LJ of the Chancery Court observed:

> Then it was said that the Plaintiff alleges an obstruction caused by several persons acting independently of each other, and does not shew what share each had in causing it. It is probably impossible for a person in the Plaintiff's position to shew this.
>
> Nor do I think it necessary that he should shew it. The amount of obstruction caused by any one of them might not, if it stood alone, be sufficient to give any ground of complaint, though the amount caused by them all may be a serious injury. Suppose one person leaves a wheelbarrow standing on a way, that may cause no appreciable inconvenience, but if a hundred do so, that may cause a serious inconvenience, which a person entitled to the use of the way has a right to prevent; and it is no defence to any one person among the hundred to say that what he does causes of itself no damage to the complainant.[39]

Of importance is the observation that it may be impossible to show the exact contribution made by a single party and that it should not be necessary to show it.

Even more suggestive is the example of the wheelbarrows. Each wheelbarrow is a particle, separate from the other wheelbarrows. The nuisance is the mass of wheelbarrows but not *all* of them would have caused any particular nuisance at all. Ten or twenty would be enough to block a road. The others are just part of the mass and none of them could have been said to *tip the balance.* Yet the reasoning of James LJ would not allow a wheelbarrow owner to claim that his wheelbarrow was on the periphery and therefore did not cause any harm. As long as the barrow belongs to the nuisance-causing mass its owner will be liable.

In *Pride of Derby and Derbyshire Angling Association* v *British Celanese Ltd*,[40] it was further observed by Harman J in the Chancery Division that even if one defendant had done enough to be sued alone, this would not absolve other contributors. This and the above reasoning was affirmed by the Court of Appeal.

On the basis of these judgments Connolly J rejected Ciba Geigy's argument, holding that there was evidence in proof of the fact that Ciba Geigy's product at least contributed to the toxic hepatic necrosis in being part of the chemical cocktail which produced the damage.[41]

This line of cases, then, shows an abandonment of the but-for test in instances either of multiple insufficient causes or even where one contribution is in itself sufficient to produce damage but others are present.

It shows great potential in toxic tort situations where the occurrence of a disease is dose-related but where it is unknown which particular but uniden-

[39] (1873) LR 8 Ch App 650, 656–7.

[40] [1952] 1 TLR 1013, 1023–4.

[41] It is significant here that his Honour saw the harm as being caused by elements in a single causal mass rather than by one unidentifiable cause among a group of discrete possible causes, each of which tended to increase the risk of injury. This distinguishes the case from *Wilsher* v *Essex Area Health Authority* [1988] 2 WLR 557 discussed in section 3D of this article.

tifiable unit in a mass of identical or similar units might be the actual causative factor. This complexity distinguishes the situation from that in *Bonnington* where it was the *accumulation* itself of contributing factors which was inferred to be causal, again without the causal mechanism needing to be identified.

It remains to be seen whether this reasoning, based on this line of cases, will be endorsed by the High Court. Until it is, the decision will only be of persuasive authority outside Queensland. It does, however, carry the authority of having been quoted by Fleming as defining causation under the NESS test.[42]

Conclusions

The concept of material contribution is thus coming into use in situations where previously a choice was made between two or more concurrent 'causes'. In doing so it is introducing a little conceptual flexibility into situations where either-or decision-making could do little justice to complex scientific facts. Exactly what the concepts of *material* and *contribution* entail, however, is still uncertain. The best view seems to be that the words, like *causation* itself, cover a spectrum of meanings. This enhances their usefulness while making it uncertain when and how they will be applied.

It should be noted that whichever of the two views of contribution outlined above is used, a legal problem arises. In the seminal British cases full recovery was allowed upon proof of contribution in spite of the fact that on any reading, the damage caused was essentially *partial.*

This is pointed out by Stapleton who defines the 'traditional requirement' as being that 'the "damage" which forms the gist of the action is the damage to which a causal connection has to be proved'.[43] She rightly argues that *Bonnington* and *McGhee* are radical in that the damage is not the whole of the illness but what she calls a portion of it. The plaintiff recovers for the full extent of the illness even though substantial damage probably would have occurred without the contribution.

Judicial discomfort with the dicta in *Bonnington* and *McGhee* has also been evident. The view that material contribution can lead to full recovery is at odds with other decisions[44] which indicate that where there are, for instance, successive employers, or successive periods of employment, the first causing damage but entailing no liability because there is no breach of duty, and the second adding to the damage through breach of duty, the tortfeasor should not have to pay for damage which he did not cause. In other words, though his contribution was to the aggregate of the damage, this aggregate should be broken up when it comes to calculating damages.

The problem was discussed by Mustill J of the Queen's Bench in *Thompson* v *Smiths Shiprepairers Ltd,*[45] a case of industrial deafness. His Honour

[42] Fleming, op cit 176.
[43] J Stapleton, 'The Gist of Negligence' Pt 2 (1988) 104 LQR 389, 407.
[44] For example, *Baker* v *Willoughby* [1969] 3 All ER 1528.
[45] [1984] 1 All ER 881.

narrowed the applicability of *Bonnington* and *McGhee* by reference to their facts. He held that there should be recovery in proportion to the contribution made by the tortious acts to the overall injury in cases where it was *known* that part of the injury was *not* caused by the tortious acts. This principle seems to be correct on grounds of logic and justice, though it reduces the vigour of the material contribution concept.

3. EVIDENCE AND INFERENCE

A. BALANCING CIRCUMSTANTIAL AND SCIENTIFIC EVIDENCE: THE ROLE OF COMMON SENSE

Medical uncertainty is likely to come before the court as expert opinion evidence. The role of this type of evidence has received sustained attention in the Australian courts since the seminal case of *Adelaide Stevedoring* v *Forst*[46] in 1940, and in particular the dissenting judgment of Dixon J (as he then was). In that case the debate concerned the weight to be given to commonsense conclusions based on circumstantial evidence as against the testimony of experts. The judgments of Rich ACJ and Dixon J represented the two poles of the argument.

Adelaide Stevedoring v *Forst*

The case was brought under the *Workmen's Compensation Act* 1932–1935 (SA), and went to the High Court. In concerned the death from heart attack of a 62 year-old waterside worker on the job. The issue was whether his work had contributed to his death. A three-to-one majority held that it was reasonable to find that this was so. Dixon J dissented, but his views have nevertheless been influential in later decisions.

The circumstantial evidence in this case was the sequence of events leading up to the injury. Rich ACJ maintained that it was proper for the investigation to begin here:

> I do not see why a court should not begin its investigation, i.e., before hearing any medical testimony, from the standpoint of the presumptive inference which this sequence of events would naturally inspire in the mind of any common-sense person uninstructed in pathology.[47]

Medical evidence would then have the role of rebutting the presumptive inference if it were able to do so. The tests of this ability would be stringent:

> [If] physiological and pathological opinion shows no more than [that] the current medical views find insufficient reason for connecting coronary thrombosis with effort. . . . that . . . is not enough to overturn or rebut the presumption which flows from the observed sequence of events.[48]

[46] (1940) 64 CLR 538.
[47] Id 563.
[48] Id 564.

Rich ACJ distinguished this situation from that where medicine offered 'strong positive reasons' to rebut the lay presumption, but held that merely 'a blank negation' would not override our 'intuitive inferences'.[49]

In typical cases of medical uncertainty — the workman with mesothelioma, the woman with leukaemia — the whole point is that pathological opinion is neither able to make a positive connection with a particular potential source of the disease nor offer any 'strong positive reasons' to rebut a presumption of causation. Given a court prepared to make benign 'intuitive inferences' (and this would apply particularly where a jury was the finder of fact), this type of reasoning would lead to a situation extremely favourable to the injured party.

In contrast with Rich ACJ, Dixon J put primary weight on the medical evidence. He resisted the view of the Supreme Court of South Australia that inferences which medical experts were unwilling to draw could be drawn from the circumstantial evidence. Furthermore, to Dixon J, the pathological question was not only crucial; it had to be asked first:

> It is impossible to treat the question raised as anything but an unmixed question of fact, medical and scientific in character, *and therefore to be decided upon expert testimony*. . . . The problems are medical, and the fact that in the present state of medical knowledge and opinion a uniform and decisive answer cannot be given on each occasion . . . is anything but a ground for the courts of law attempting to supply by legal reasoning a solution to what is entirely a question of fact.[50]

Dixon J allowed common sense based on a sequence of external events a role only when 'positive knowledge or common experience supplies some adequate ground for believing that the events are naturally associated'.[51]

What we see in the judgment of Dixon J is an apparent willingness to open the law up to the sophisticated and objective modes of information-gathering and reasoning represented by medical science. In doing so, however, he was also making it and the plaintiff subject to the inconclusiveness of science.

Development after *Adelaide Stevedoring*

Despite the strong words of Dixon J in *Adelaide Stevedoring*, so-called commonsense and circumstantial evidence have been permitted to maintain an influential position in legal reasoning.

In *Nicolia* v *Commissioner for Railways (NSW)*,[52] for instance, it was held by the High Court that a cause of death, ie substantially a medical question, could be judicially determined without recourse to any accepted medical evidence. 'The common knowledge and experience of mankind'[53] was sufficient to support the determination. It should be emphasized, however, that this was

[49] Ibid.
[50] Id 568 (emphasis added).
[51] Id 570.
[52] (1971) 45 ALJR 465.
[53] Id 466 per Barwick CJ.

in the total absence of any acceptable scientific evidence. The courts are obviously unwilling to be paralysed by a complete evidential lacuna.

On the other hand, where acceptable medical evidence was available, it had to be taken account of, as *EMI (Australia) Ltd* v *Bes*,[54] another workers' compensation case, shows.

Here the injured party was struck by a falling ladder at work. Twelve days later, after suffering headaches in the intervening period, taking medication for them and evincing signs of emotional disturbances, he had a car accident which killed him. It was claimed that his accident was caused, through the medium of the drugs, by his injury at work.

Here the court took very seriously the role of medical evidence. Arguments such as those of Rich ACJ in *Adelaide Stevedoring* appeared to have been put aside:

> The case is one in which the learned judge was not entitled to determine the issues on a basis of intuitive reasoning based on common experience of mankind, his Honour being bound in a case such as this to look for some assistance from the medical scientists . . .[55]

Here it was a matter of 'using the temporal experiences as a background leading up to an examination of the medical scientific evidence'.[56]

On the other hand, there was resistance to the notion of giving medical evidence an almost exclusive significance in decision-making, a notion which had been encouraged by the dicta of Dixon J in *Adelaide Stevedoring*. Herron CJ quoted the High Court in *St George Club Ltd* v *Hines* (supported by the *majority* reasoning in *Adelaide Stevedoring*):

> The determination by a court of a matter before it must depend upon a consideration of the whole of the evidence [ie not of medical evidence viewed in isolation] . . . although . . . more weight may be required to be given to one or more aspects of the evidence to the exclusion of others.[57]

This leaves the weighting to be given to the various components of the evidence largely to the discretion of the court, allowing both flexibility and an element of unpredictability. It would appear to allow testimony of medical uncertainty to be overridden by the persuasive power of circumstance.

In *Fernandez* v *Tubemakers of Australia*,[58] Glass JA confirmed the *Nicolia* principle of the inessentiality of expert evidence and its subjection to normal legal principles:

> The evidence will be sufficient if, but only if, the materials offered justify an inference of probable connection. This is the only principle of law.[59]

He reasserted the role of common sense in this type of case, thus turning some

[54] [1970] 2 NSWR 238.
[55] Id 239 per Herron CJ.
[56] Id 241.
[57] (1961) 35 ALJR 106.
[58] [1975] 2 NSWLR 190.
[59] Id 197.

way back towards the type of reasoning earlier used by Rich ACJ in *Adelaide Stevedoring*.

In the context of trauma to a worker's hand being alleged to have caused Dupuytren's contracture, Glass JA stated:

> It is true that the medical evidence fails to supply any aetiological detail which would permit the jury to be satisfied that trauma had probably set in motion a chain of anatomical and physiological events which culminated in the shortening of the tissue in the plaintiff's hand. *But I do not think that it is incumbent upon a plaintiff to demonstrate the mechanism by which a cause had probably produced an effect.* In any event, I see no reason why common sense might not instruct that changes in the subcutaneous tissue of the palm may well follow the application of violence to a hand . . .[60]

Mahoney JA supported both the judgment of Glass JA and his reasoning, stating that the question to ask was whether a 'reasonable mind', given the evidence, could conclude that the possible was the actual cause.[61]

The reasonable mind enjoys, then, an enviable latitude to exercise its common sense in all questions of causation. This was confirmed in 1991 in *March* v *E and M H Stramare Pty Ltd*,[62] a High Court decision which considered the inadequacies of the but-for test and, by a majority, held that its use was subject to overriding commonsense principles.

Of course, to enjoy a latitude is not necessarily to exercise it in favour of the plaintiff. The 1988 Queen's Bench decision of *Loveday* v *Renton*[63] in the UK, concerning damage allegedly caused by inoculation, saw the tribunal of fact, a judge in this case, resolutely refusing to give circumstantial evidence weight as against inconclusive medical evidence. A similar refusal was seen in *Wintle* v *Conaust*[64] in the Victorian Supreme Court, another non-jury decision.

It is suggested that circumstantial evidence, viewed in the light of common sense, should in fact be used sparingly in toxic tort decisions. Given that the tribunal of fact will be almost without exception scientifically ignorant, there is an acute risk of injustice to the defendant in the use of the test. The other problem is that common sense is so subjective a commodity that it can be used as an excuse for decisions that affront logic and the notion of justice.

B. LEAPING THE EVIDENTIAL GAP: THE IMPORTANCE OF INFERENCE

Perhaps the most significant method used by the courts to avoid the problem of medical uncertainty and in many cases to implement a policy of benevolence to the plaintiff is the practice of drawing inferences.

Unless the defendant admits what is to be proved, all legal decisions are ultimately matters of inference. There is always a point at which evidence stops and the gap between data and 'truth' must be jumped. The gap is

[60] Id 198 (emphasis added).
[61] Id 200.
[62] (1991) 65 ALJR 334.
[63] *The Times*, 31 March 1988, 18.
[64] [1989] BR 951.

narrowed by almost half in civil cases through the fact that proof on the balance of probabilities, ie 51%, will be deemed to be full proof. Inferences of this nature occur not only at the point of the final decision but at the point of any decision in the course of the trial.

This reliance of the system on inference gives it great flexibility for there is no fixed rule about what will found an inference. Such flexibility means that the process of inference-drawing can often be used by judges in the interests of justice as they see it. Some courts will refuse to draw inferences which other courts will readily draw.

In the toxic torts area there has been observable in the UK since the middle of this century a more overt willingness to allow policy to affect the process of inference-drawing than in Australia. And even when using UK decisions, Australian courts have tended to remain aloof from certain of the inferences which have been drawn. This is not to say that they have not drawn policy-based inferences of their own.

Inferences concerning causal mechanisms

In many toxic tort cases the first step in establishing causation is to lead evidence which will give rise to inferences concerning the medically unidentified causal mechanism.

In the case of *Bonnington Castings Ltd* v *Wardlaw*, 'the medical evidence was that pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years'.[65] This statement does not indicate a causal mechanism. Lord Reid therefore drew an inference:

> That means, *I think*, that the disease is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other.[66]

This conclusion is still vague but was usable in the context of the case. Lord Keith of Avonholm emphasized the medical uncertainty: '*Prima facie* the particles inhaled are acting cumulatively'.[67]

The decision to infer that action by an accumulation of particles meant that the whole of the material inhaled was causally relevant was crucial to the outcome of the case, based as it was on material contribution to a disease-causing whole. The same applies to *Nicholson*,[68] where the fact situation was substantially identical.

In *McGhee* v *National Coal Board*,[69] however, and in later cases, complications arose, probably due to a greater medical awareness of what the causal mechanisms might be. These sharpened the focus on the causation/contribution issue.

In *McGhee*, a case of dermatitis caused by 'innocent' and 'guilty' exposure to brick dust, Lord Reid spoke at length of known causes of dermatitis:

[65] *Bonnington Castings* v *Wardlaw* [1956] AC 613, 621.
[66] Ibid (emphasis added).
[67] Id 626 (emphasis added).
[68] *Nicholson* v *Atlas Steel Foundry and Engineering Co Ltd* [1957] 1 WLR 613.
[69] *McGhee* v *National Coal Board* [1973] 1 WLR 1.

> Dermatitis can be caused, and this dermatitis was caused, by repeated minute abrasion of the outer horny layer of the skin followed by some injury to or change in the underlying cells, the precise nature of which has not yet been discovered by medical science. . . . The evidence does not show . . . just how dermatitis of this type begins. . . . It may be that an accumulation of minor abrasions of the horny layer of the skin is a necessary precondition for the onset of the disease. Or it may be that the disease starts at one particular abrasion and then spreads, so that multiplication of abrasions merely increases the number of places where the disease can start and in that way increases the risk of its occurrence.[70]

This type of known uncertainty leaves the court in a difficult position. Should it take the *Bonnington* or *Barker* line that an effect produced by an aggregate of toxic agents raises the inference of material contribution by all the units which make up the aggregate? This would be a policy-driven inference. Should it refuse to allow recovery based on the impossibility of establishing what exactly caused the disease? Or should it use other policy-based lines of reasoning to avoid sending the plaintiff away empty-handed? In fact the court in this case used a policy-based harm-within-the-risk analysis which will be discussed below.

For the moment it is instructive to note how recent English and Australian cases have responded to the dilemma.

Recent responses to causal uncertainty

In the mesothelioma case of *Andrews* v *Lohse*,[71] decided by the Supreme Court of Queensland in 1986, there were four defendants who had employed the plaintiff in turn between 1974 and 1983. Expert evidence indicated that 'each one of the defendants gave the plaintiff sufficient exposure to asbestos to explain, in the absence of any exposure while employed with the other defendants, the onset of the plaintiff's disease'.[72] The exposure was in breach of a duty of care. This was a case of multiple sufficient causes, so if any causation was to be established the traditional but-for test had to be jettisoned, and so it was.

In was accepted by de Jersey J that 'medical knowledge in this area cannot provide categorical proof of the precise manner of the causing of such a condition',[73] thus putting the case into the same category of cases as *Bonnington* and *McGhee*. Here, however, this evidential lacuna was not perceived as causing difficulties. Although it was argued that in fact a causal link had not been proved in that the condition had not been linked to *one* particular defendant, the judge found it possible to hold that *each* of the three first defendants had materially contributed to the injury. He held that the contributions of the first and third defendants were approximately equal and that the contribution of the second defendant was greater than that of the other two.

[70] Id 3–4.
[71] [1986] Aust Torts Reports 80–043.
[72] Id 67,887.
[73] Id 67,889.

Thus, in *Andrews*, the judge ignored the requirement insisted upon by some courts that the onset of the disease must be attributable *either* to one offending incident *or* to another, and regarded the defendants as jointly liable. Damages were then apportioned according to the percentage of liability established against each defendant, ie according to which defendant caused the greatest exposure to the asbestos. This fact suggests that the quantity of dust inhaled, at least before the onset of the disease, was linked with the extent of contribution. This would indicate that, although each exposure was seen as sufficient in itself to cause the disease, an inference was drawn that the effects of exposure were cumulative, thus bringing the case within the *Bonnington* principle.

In the British case of *Bryce* v *Swan Hunter Group*[74], decided by the Court of Appeal, the problems referred to in *McGhee* loomed larger in the judge's mind. This was also a case of exposure to asbestos dust while in the employment of successive firms. The deceased had been employed from 1937 until 1975 in shipyards owned by three defendant companies. He had died of mesothelioma caused by the asbestos dust to which he had been exposed during the course of his employment. Not all exposure had been negligently caused. The deceased's widow brought an action against the employers claiming that their negligence had caused her husband's death. The defendants contended that even if they had been in breach of duties they owed as employers of the deceased, there was no evidence that those breaches had caused the disease.

It was common ground that the asbestos dust was the cause of the mesothelioma.

Phillips J, after observing that the medical evidence indicated:

1. that the risk of mesothelioma is dose-related;
2. that the present state of medical knowledge does not enable one to draw a conclusion as to precisely why it is that an increase in exposure to asbestos dust increases the risk of contracting the disease;
3. that it may be that each fibre acts separately and the more that are inhaled the more likely therefore that one will trigger the disease; and
4. that it may be that the fibres have a cumulative effect in overwhelming the body's defences so as to facilitate the malignancy,

considered that it followed that it was not possible for the plaintiff to prove on a balance of probabilities that the fibres inhaled by Mr Bryce as the result of breaches of duty by any one of the defendants were a cause of his mesothelioma.

It appears, then, that although the facts here are not dissimilar to those of *Bonnington*, the court has refused to draw the inference drawn in *Bonnington* that if a disease is dose-related, then the substance causing it can be deemed to be acting cumulatively in the sense that each unit of it is a material contributory factor in the genesis of the disease. It has refused to draw the inference even though it admits that this may well be the truth:

[74] [1988] 1 All ER 659.

> It may be . . . that, as the quantity of fibres in the lung increases, they also have a cumulative effect in damaging or overwhelming the body's defence mechanism so as to facilitate the occurrence of malignancy.[75]

Upon refusing to draw this inference the court must then try to decide which exposure was the primary cause of the disease — an impossible task.

What the court did here was to find for the plaintiff on the basis of increased risk, a principle developed in *McGhee*.[76] It may be that its hard line on the issue of factual causation was influenced by its belief that it was bound by that principle to reach a finding favourable to the plaintiff.

In Australia, however, the courts are not bound by British precedent. In *Wintle* v *Conaust*,[77] decided by the Full Court of the Victorian Supreme Court, the applicant sought an extension of the period within which an action for damages might be brought. For twenty-six years he had been a wharf worker and had been exposed to asbestos dust while working for the five respondents, who were involved in stevedoring operations at the port during the relevant period. In 1988 he had been diagnosed as suffering malignant mesothelioma of the pleura. He had medical evidence that his mesothelioma had been caused by his exposure to asbestos between 1961 and 1970. This evidence was not impugned, but in spite of this the Court, by a majority, found that there was insufficient evidence to establish causation. On the basis that the evidence did not show the number of occasions, and the length of each of them, on which each respondent exposed the applicant to asbestos dust, the majority held that there was no 'foundation for proof of a relevant causal connection with any one particular respondent'.[78] It was working on the assumption that there was no possibility of concurrent causation and that either one defendant or another must therefore be solely liable. This was determinative. It was not enough to establish negligence of all the respondents and exposure by all of them of the applicant to the dust which remained the unquestioned cause of the disease. It was 'not enough to say that the appellant suffered asbestos exposure from no other employer and therefore, as it must have been one or more of the five named respondents, they may all be treated as vulnerable to a successful claim's being made against them'.[79]

In seeking to demonstrate the deficiency 'in relevant probative utility' of the medical evidence, the majority adopted the reasoning of Phillips J in *Bryce*. As seen above, Phillips J had relied on the uncertainty as to why the occurrence of mesothelioma was dose-related to reach the conclusion that it was impossible for the plaintiff to prove on a balance of probabilities that fibres inhaled as a result of one party's negligence were a cause of his disease.

So again we see a refusal to draw the crucial inference as to the disease's causative mechanism upon which any finding of liability must be based.

[75] Id 665.
[76] See next section.
[77] [1989] VR 951.
[78] Id 952 per Crockett and Gray JJ.
[79] Ibid.

C. 'EKING OUT THE EXPERT EVIDENCE' — HOW A POSSIBILITY CAN FOUND A FINDING OF PROBABILITY

The above cases create uncertainty as to the inferences which a court is likely to draw in circumstances where the expert evidence is neither conclusive nor expresses any probability concerning causal relationships. Some light can be cast on the theoretical background to such decisions by the Australian cases dealing with expert evidence.[80]

In these cases the courts had to determine under what circumstances a statement of possibility would be sufficient to ground an inference of probable causation; in other words, how far the evidential gap had to be narrowed before it could be leapt.

Again the seminal pronouncement is one by Dixon J in *Adelaide Stevedoring* v *Forst*:

> I think that upon a question of fact of a medical or scientific description a court can only say that the burden of proof has not been discharged where, upon the evidence, it appears that the present state of knowledge does not admit of an affirmative answer and that *competent and trustworthy expert opinion regards an affirmative answer as lacking justification, either as a probable inference or as an accepted hypothesis.*[81]

These terms are open to much interpretation. What, for instance, is 'competent and trustworthy medical opinion'? Does a 'probable inference' or an 'accepted hypothesis' fulfil the requirement of a finding which is more probable than not, or does such a test violate the civil standard of proof? Is Dixon J suggesting that inference can take over at this early point?

Assuming that inference on the part of the tribunal will bridge the gap between hypothesis and fact, the standard of proof is probably not theoretically lowered, but it seems that the plaintiff might not have to go as far as usual to get to the point where the necessary inference may be drawn. A finding can be made, ostensibly on the balance of probabilities, on evidence (of an hypothesis, for instance) which arguably establishes no probability at all.

In *EMI (Australia) Ltd* v *Bes*, Herron CJ of the Supreme Court of New South Wales, who was expressly guided by the dicta of Dixon J, looked at the desirable level of expert persuasion in the light of the other evidence in the case. His words represent a slight extension of the approach of Dixon J:

> It is not incumbent upon the applicant . . . to produce evidence from medical witnesses which proves to demonstration that the applicant's contention is correct. . . . If medical science is prepared to say that it is a possible view then . . . the judge after examining the lay evidence may decide that it is probable. . . . It may be . . . 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 . . . that no medical witness states with certainty the very issue which the judge himself has to try.[82]

[80] Referred to in section 3A.
[81] *Adelaide Stevedoring* v *Forst* (1940) 64 CLR 538, 569 (emphasis added).
[82] [1970] 2 NSWR 238, 242.

In 1975, a thorough and most useful statement of the weight to be given to expert declarations of possible causal links was given. This statement, by Reynolds JA in *Fernandez* v *Tubemakers of Australia*,[83] remains as something of a landmark:[84]

> Difficulty arises when an expert witness speaks only in terms of possibility in circumstances where it can be seen that he declines to draw the inference which the lay tribunal is invited to draw. It seems to me that the answer to the question which is posed in such cases begins with an understanding of the real content of the medical opinion relied upon. An expression of opinion that a condition could be or might be related to a suggested cause will have different meanings in different contexts. If nothing is known as to the aetiology of a condition or disease, no cause can be excluded as a matter of logic, and so it might be said that any suggested cause might have or could have caused it. In such case the assertion is not in the full sense an expression of expert opinion and has no probative force.
>
> If very little is known of the relevant aetiology, a similar expression of opinion may mean that present scientific knowledge does not exclude the possibility of a causative relationship. If much is known and the knowledge is explained and expounded to the tribunal of fact, an expression of opinion which does not pass beyond possibility may be regarded as a precise and guarded scientific statement which leaves the ultimate question or probability to the tribunal to pronounce upon, having regard to all the facts.[85]

Thus many expert statements of possibility will not provide any ground at all for inference drawing. As long, however, as enough is known about the causes of the condition in point, then a mere statement of possibility is sufficient to allow the tribunal of fact to conclude that this possibility was the operative one, assuming that the other available evidence leads to this conclusion. As Reynolds JA frankly put it: 'It remains . . . to inquire whether the expert opinion can be eked out by common knowledge so that the inference may be drawn at which the expert baulked.'[86]

This last sentence suggests the sometimes willed benignity of the decision-making in this area. Clearly the expert evidence cannot be seen as leading the tribunal even to the edge of the evidential gap. Circumstantial evidence supplies the further ground from which the inferential leap can be undertaken.

In 1940 Dixon J had not considered the need to eke out the uncertainty of scientific evidence. In failing to do so he had left the evidential gap so wide that the act of leaping it would have laid the court open to charges of legal fiction-making. Later courts have drawn back from that position and employ their ingenuity in narrowing the gap before they leap it.

[83] [1975] 2 NSWLR 190.
[84] It is cited with approval by Slattery CJ in *Karolis* v *Prince of Wales Hospital & Anor* [1986] Aust Torts Reports 80–053, 67,976–7.
[85] *Fernandez* [1975] 2 NSWLR 190, 194.
[86] Id 196.

Conclusions

In the light of these cases, and particularly the comments of Reynolds JA, it is suggested that courts have much latitude in the use they make of any well founded statement of scientific possibility. The fact that other hypotheses exist need not deter a court from allowing itself to be persuaded of the probative force of one opinion. Scientific uncertainty need not preclude a plaintiff from establishing cumulative causation, as it appears to have done in *Wintle* and *Bryce*. In the latter case the judge stated, presumably on the basis of expert evidence, that it is possible that risk rises with exposure because the more fibres that are inhaled the greater the risk that one will cause cancer, and alternatively that it is possible that the fibres have a cumulative effect (which would bring the case within the *Bonnington* principle). In this situation it does *not* then follow automatically that the plaintiff cannot prove the 'guilty' fibres to have been the cause of his condition. The court may find any additional evidence to be lacking in probative force, but the failure of the case will lie in that fact and not in the uncertainty of medical science.

D. THE SLIDE INTO FICTION

It was suggested in the last section that had the dictum of Dixon J in *Adelaide Stevedoring* v *Forst*[87] been interpreted narrowly a legal fiction would have arisen. For the courts to have been enabled to find causal linkage solely on a scientific opinion stating it to be justifiable as a probable inference or an accepted *hypothesis* would have widened the gap between adduced evidence and the court's finding of fact to a point where the finding of fact would have strained credulity.

Whether the creation of legal fictions in common law is desirable is questionable. They tend to arise where the law as it exists is seen to be out of step with conventional notions of justice. Thus, in this context, they are the creatures of policy and are interlopers in a system whose fundamental principle is that of reason or logic.

Clear examples of legal fiction-making can be seen in *McGhee* v *National Coal Board*[88] and later cases, again in the interests of sidestepping causation problems. They led to the bemused comment by Nourse LJ in *Fitzgerald* v *Lane & Anor*[89] that 'a benevolent principle smiles on . . . factual uncertainties and melts them all away'.

The harm-within-the-risk test

It was to create a solution to situations of medical indeterminacy that the House of Lords in *McGhee* developed the harm-within-the-risk test which had originated in earlier breach of statute cases.[90] At its broadest this test allowed recovery where a risk had been negligently caused by the defendant and the

[87] (1940) 64 CLR 538.
[88] [1973] 1 WLR 1.
[89] [1987] 3 WLR 249, 262.
[90] See, for example, *Lee* v *Nursery Furnishings Limited* (1945) 172 LT 285, 287.

plaintiff suffered a harm which was within the ambit of the risk. It was classified as a test of causation but is anomalous since the evidence adduced in court does not have the effect of linking the negligent behaviour with the outcome. Whereas in most situations evidence is adduced as to the truth of the fact sought to be proved and the court merely infers that the evidence establishes that truth, in this case the evidence has nothing to say about causation or even about the probability of causation. The linkage which is central to the concept of causation is never *shown* to exist on the balance of probabilities; it is *inferred* to exist on the balance of probabilities.

McGhee v *National Coal Board*

In *McGhee* Lord Reid posited what he called a 'broader view of causation'. His Lordship recounted the medical evidence based on experience:

> The fact that the man had to cycle home caked with grime and sweat added materially to the risk that this disease might develop. . . . That must be because what happens while the man remains unwashed can have a causative effect, though just how the cause operates is uncertain.[91]

He then went on to reject the distinction drawn by the Lord Ordinary in the First Division of the Court of Sessions between materially increasing the risk that the disease would occur and making a material contribution to its occurrence.

In the circumstances this rejection is appropriate. This is because the two conditions under which this test is logically unworkable were not operative in this case or in Lord Reid's reasoning.

Conditions under which the test is unusable

The first of these conditions is where the actual injury is or may be caused by a different substance from that which created the risk. When this is the case, contribution and increased risk are different concepts since injury can be caused which is within the risk but which is not contributed to by the negligence of the defendant. For instance, an employer may create a risk for his employees of getting dermatitis and an employee may in fact get dermatitis, but its cause may be the soap that he uses to wash with at home. For the test to work, description of the risk must include the causation requirement (eg dermatitis caused by brick dust). If it does so, however, the whole test becomes tautologous in that the test already expresses what it is sought to discover. These problems were recognized in the House of Lords decision in *Wilsher* v *Essex Area Health Authority*.[92]

The second of these conditions is where an attempt is made to satisfy the classical but-for test (rather than a broad view of contribution) by reference to harm within the risk. Such an attempt can only lead to the type of comment made by Lord Wilberforce (who himself appeared to want to apply the but-for

[91] *McGhee* [1973] 1 WLR 1, 4.
[92] [1988] 2 WLR 557.

test) that 'in the present case to bridge the evidential gap by inference seems to me something of a fiction.'[93] This is best illustrated by an example.

Take the dust-covered workman. Let us assume that without additional exposure caused by the firm's negligence, 30% of workers will develop dermatitis of the relevant kind. With the additional exposure 45% will develop this dermatitis. Clearly the additional exposure significantly increases the risk of getting the disease, yet the probability of the disease being caused by the additional exposure is only 15%, whereas all other operating factors are responsible for a 30% risk. Thus in any given case it is more probable than not that the dermatitis would have occurred *without* the additional exposure.

Take, on the other hand, a 20% risk of developing the disease under normal conditions which rises to a 45% risk with the additional exposure. Here the 25% additional risk means that in any given case it *will* be more likely than not that the increased exposure caused the disease.

Thus it is impossible to tell whether there is a correlation between increased risk and but-for cause of the disease without evidence of the relative probabilities. In other words the test, as a but-for causation test, is useless. It can be used only as a measure of when a judicial policy should come into force, or where 'contribution' is not interpreted as satisfying the but-for test. In no case can but-for causation be proved by satisfaction of the test without additional evidence being presented.[94]

The situation in *McGhee*

In *McGhee*, however, it was certain which substance caused the disease and Lord Reid was concerned to establish contribution to an aggregate of harm-producing exposure. Under these conditions, and assuming there was no onset of the disease before the additional exposure, it could be taken that *all* affected workers had been affected in a contributory way by any additional exposure to the dust, ie all the workers' conditions included the quota of guilty exposure.

The problem with the judicial pronouncements in *McGhee* was that they were categoric and defiant:

> But it has often been said that the legal concept of causation is not based on logic or philosophy. It is based on the practical way in which the ordinary man's mind works in the everyday affairs of life. From a broad and practical viewpoint I can see no substantial difference between saying that what the defender did materially increased the risk of injury to the pursuer and saying that what the defender did made a material contribution to his injury.[95]

[93] *McGhee* [1973] 1 WLR 1, 7.

[94] It is true that Lord Wilberforce, in the minority in this respect, also advocated the shifting of the onus of proof from the plaintiff to the defendant upon the establishment of a breach of a duty and the occurrence of harm within the relevant risk. This would not, however, solve the problem of causation. Should the defendant fail to satisfy the court of lack of causation, the court would still be *deeming* the cause-effect link to exist rather than finding it to exist on a balance of probabilities.

[95] *McGhee* [1973] 1 WLR 1, 5 per Lord Reid.

Such statements were unhelpful as they fuelled the belief that what the case established was that any materially increased risk or harm occurring within the risk represented a material contribution to the disease. This was patently a legal fiction.

The reining in of the test in Britain

In *Bryce* v *Swan Hunter Group plc & Ors*,[96] this inherent difficulty with the harm-within-the-risk test was emphasized by the fact that Phillips J of the Queen's Bench explicitly separated it from the question of factual causation. Given that he felt unable to draw any inferences as to the cause of the mesothelioma from the available evidence it seems absurd that, by talking about logic and law under separate headings, he felt both able and obliged to find causation on the basis that increased risk had been created.

That logic and law should not suffer this complete divorce was re-established in the House of Lords decision in *Wilsher* v *Essex Area Health Authority*,[97] a case which has done much to rein back the effects of the fiction which grew from the earlier case law.

Here the plaintiff was born prematurely and required extra oxygen. A catheter through which oxygen was administered was inserted into the wrong blood vessel, preventing blood oxygen from being correctly monitored. As a result, too much oxygen was administered during the first thirty hours of his life. After the mistake was corrected there were still occasions when the blood oxygen was considered too high. The plaintiff developed retrolental fibroplasia which resulted in blindness. One possible cause of the condition was that too much oxygen had been administered within the first thirty hours or at a later stage.

In this case Mustill LJ of the Court of Appeal, in reliance on a principle of law which he understood to have been established in *McGhee*, stated that:

> If it is an established fact that conduct of a particular kind creates a risk that injury will be caused to another or increases an existing risk that injury will ensue; and if the two parties stand in such a relationship that the one party owes a duty not to conduct himself in that way; and if the first party does conduct himself in that way; and if the other party does suffer injury of the kind to which the risk related; then the first party is taken to have caused the injury by his breach of duty, *even though the existence and extent of the contribution made by the breach cannot by ascertained.*[98]

Lord Bridge in the House of Lords disagreed, maintaining that material contribution in *McGhee* 'was a legitimate inference of fact'.[99] The inferential

[96] [1988] 1 All ER 659. For the facts of the case, see section 3B of this article.
[97] [1988] 2 WLR 557.
[98] *Wilsher* v *Essex Area Health Authority* [1987] QB 730, 771–2 (emphasis added).
[99] *Wilsher* [1988] 2 WLR 557, 569.

reasoning, according to Lord Bridge, was that where there is expert evidence that the longer brick dust remains on the body, the greater the risk of dermatitis (*although the doctors cannot identify the process of causation scientifically*), one can infer, as a matter of common sense, that the consecutive periods when brick dust remained on the body probably contributed cumulatively to the causation of dermatitis. Thus if a disease is dose-related it is reasonable to infer contribution by those tortfeasors who are responsible for the dose.[100] In the light of this reasoning the distinctions made in *Bryce*,[101] though valid, appear legally irrelevant. Furthermore, the fictional elements of the harm-within-the-risk test are put aside.

It is submitted that Lord Bridge is right that the necessity for the plaintiff to *prove causation* on the balance of probabilities was not denied by the court in *McGhee* and his Lordship certainly does not do so in this case. Such proof lies at the centre of any attempt to recover for toxic injury. However, his reference to the 'robust and pragmatic approach'[102] adopted by his fellow Law Lords in that case indicates no more than a quizzical glance at their equation of creation of increased risk with material contribution. His judgment appears to represent no denial of the harm-within-the-risk principle as long as it can reasonably be expressed as being subservient to the principle of causation.[103] This could not be done in *Wilsher*.

It had been held in the Court of Appeal that where a disease could have been caused by a number of substances and the negligence of the defendant had contributed a further one, this negligence, having undoubtedly increased the risk of injury, could be deemed to have caused or contributed to the onset of the disease.

The Vice-Chancellor in the Court of Appeal, and the House of Lords, considered that this would be an unjustified extension of the *McGhee* principle. They held that there was an additional need for convincing evidence that, on the balance of probabilities, *that* substance caused or contributed to the disease. As observed above, even if the court is applying the harm-within-the-risk test, it is crucial not to look at the question of negligence before it is established what substance(s) or incident(s) caused the injury.[104] To see causation in every case where risk was increased would be to stray too far from the

[100] This is a broad reading of *contribution* deriving from *Bonnington Castings* v *Wardlaw*.
[101] [1988] 1 All ER 659.
[102] *Wilsher* [1988] 2 WLR 557, 569.
[103] As Andrew Boon says: 'Despite the cautious interpretation of the material increase of risk adopted by the House of Lords in *Wilsher*, the principle in *McGhee* has apparently been confirmed as being of general application in issues of factual causation': 'Causation and the Increase of Risk' (1988) 51 MLR 508, 514.
[104] It may be argued that the line of reasoning pursued in *Barker* v *Permanent Seamless Floors* [1983] 2 Qd R 561 should be followed here since there too multiple substances as well as incidents were involved. That case is, however, distinguishable since there it was a matter of a *mixed* cause, each substance being found to contribute to a disease causing mass. Here, in *Wilsher*, the possible causes of the condition were discrete, different in nature and not forming any kind of identifiable aggregate.

requirement of actual causal linkage. The test could only be used where it could lead to a realistic inference of causation.

The Australian situation

In Australia, the harm-within-the-risk test has been avoided in toxic tort cases.

In *Andrews* v *SC Lohse & Co and Ors*,[105] a mesothelioma case with multiple defendants, it is significant that de Jersey J of the Queensland Supreme Court was explicit in his assertion that the evidence in the case went beyond establishing merely that asbestos exposure materially increased the risk of injury. He thus consciously avoided using the test, though he might have done so. In doing this he intimated that it was at least preferable to establish an actual causal link on the basis of evidence rather than to resort to the notion of harm-within-the-risk. The evidence he referred to was apparently undocumented expert opinion evidence.

In *Wintle* v *Conaust*,[106] decided after the House of Lords decision in *Wilsher*,[107] the court treated the harm-within-the-risk test as overruled, though it is arguable that this is not entirely so.[108] And in *Chance* v *Alcoa of Australia Ltd*,[109] although *McGhee* was referred to, the case was not discussed in terms of harm-within-the-risk at all.

Observations on alternatives to the harm-within-the-risk test

In *Wilsher* it was not open to the court to make an inference of causation. In *McGhee, Bryce* and *Wintle*, however, it is submitted that such an inference might have been drawn without recourse to the harm-within-the-risk test.

It would seem that a *Bonnington*-type test is most useful in a situation where a substance has admitted cumulative effects. Such a test does not remove the evidential gap but it has a greater logic. Assuming that the longer a worker is exposed, the more likely he is to develop a condition, one would deem every unit of exposure, both at work and after work, to be a contributing factor. Thus the defendant would be liable for any significant contribution negligently caused.

The situation is more difficult if the disease could be caused by a single particle acting independently of all other particles. In that case only the defendant who was responsible for that particle would be liable. Actual proof of causation could not possibly be established any more than it could be established that a single microbe from several carriers of a disease actually caused the disease in its new victim.

[105] [1986] Aust Torts Reports 80–043, 67,883. For the facts of the case see section 3B of this article.
[106] [1989] VR 951.
[107] [1988] 2 WLR 557.
[108] See fn 105.
[109] [1990] Aust Torts Reports 81–017.

In this case every defendant who exposed the plaintiff to a single particle would be potentially liable. Each exposure would be a sufficient cause of the disease. One would simply be confronted by multiple sufficient causes/ tortfeasors.

In this case the NESS test should be able to be used but, while the test caters for instances where the courts might wish to find causation, it does not reveal its own logical underpinning. It is suggested that the logic of finding causation in such cases resides in notions of contribution.

When it is simply unknown whether increase in risk or contribution is at issue and where the disease is quite clearly dose-related, it would seem desirable to opt provisionally for some concept of contribution. It is a sleight of hand, but is logically less offensive than to say that risk equals contribution at a point in the legal reasoning where it is quite clear that such a proposition is absurd.

In cases such as the present, the substance which causes the disease is known but it is scientifically impossible to pinpoint the causal mechanism either because the mechanism itself is unknown or, when it is known, because there is no possibility of identifying which particular chemical or biological event caused the disease. Here considerations of causation should be lifted to a higher level of generality. This happened in *Bonnington*, where it was accepted that the dust-laden atmosphere caused the disease.

Although it may be said that in that case the dust was held to have a cumulative effect in the sense that every particle contributed to the disease, this was merely an inference given the available evidence. The point is that the court described the cause in terms which embraced all the possible causative mechanisms.

It is submitted that such a move is necessary. Even when it is known that there are various mechanisms by which a substance can cause harm, as long as none of the competing explanations is capable of being proved, then either the plaintiff will have no means of recovery unless and until scientific inquiry evolves explanations of the causative process, or the law must rationalize its position. The fact that we realize that our knowledge does not extend to the most intricate mechanisms of nature should not mean that the common law, created as it was by and for human society, should suddenly find itself helpless. It must do what it can with the certainties it has. If specifics are known to be unknown, then the known generalities must be used. If we cannot specify which particle of asbestos or which microbe or which moment of exposure caused the disease, but we do know that asbestos dust or exposure to a biological agent or a certain period of exposure did so, then those who negligently contributed to or caused the relevant exposure should be liable. There is an evidential gap here. We bridge it by embracing the half-fiction that the specific can be represented by the general. This is reasonable as long as the specific remains unknowable. As soon as it is knowable then the general must be discarded. It should be noted, however, that where the substance or toxic aggregate which has caused the harm is unknown, as in *Wilsher* or *Chance*, there is no meaningful generality to look to.

4. EVIDENTIAL PROBABILITY AND THE STANDARD OF PROOF

THE CONVERGENCE OF EVIDENCE AND STANDARD

The leukaemia victim is faced with a problem which she shares with all those suffering from diseases which may have various possible aetiologies. Since a disease commonly caused by legally innocuous events is medically indistinguishable from the disease caused by a defendant's breach, how can it ever be proved that that breach caused the disease? How can the requirement that the harm be attributed to a person be fulfilled so that the aims of compensation and deterrence can be attained?

We have seen that with the right kind of expert evidence the plaintiff might have been able to claim that the additional exposure created by the defendant was a contributory cause of the disease. She might have been aided here by the willingness of some courts to draw inferences from relatively thin scientific evidence.[110] If contribution cannot be claimed, however, and if her claim to recovery is based simply on the fact that she lived in a population which received the additional exposure, the causal uncertainties thrown up by her situation will present a significant barrier to recovery.

In this case it may be that the plaintiff can look to the standard of proof itself as an aid. Assuming that the plaintiff has no personal link with the defendant's activities, that no particularistic evidence can be brought to link her disease with them, recovery might conceivably be possible on the basis of probability alone. This will be probability based on epidemiological studies; it will be *evidential* probability; it will be data showing that the background risk of any person developing the disease (ie the risk attributable to all other operative factors) was lower than the additional risk of developing it caused by the defendant's acts. It can be argued that to establish this is to establish the level of probability required for the civil standard of proof and thus to win the case. There are, however, questions begged by this statement of the law.

The first question concerns what the civil standard of proof actually is. The second concerns the admissibility of conflating evidence expressed as probability with the probability element of the standard of proof.

What is the standard of proof?

Though the civil standard of proof is generally stated to be the balance of probabilities and this is generally taken to mean 51% certainty or higher,[111] there is some authority in the case law supporting the view that the standard is

[110] The problem might also have been solved by the most liberal reading of the harm-within-the-risk test, but given the limitation put on the test by the House of Lords decision in *Wilsher* v *Essex Area Health Authority* [1988] 2 WLR 557 (ie that the causative substance must be identified on the balance of probabilities before the test can come into play) the leukaemia victim would have no chance of recovery.

[111] This is particularly the case in the United Kingdom where, for instance, in *Davies* v *Taylor* [1974] AC 207, 219 Lord Simon of Glaisdale stated: 'Beneath the legal concept of probability lies the mathematical theory of probability. Only occasionally does this break surface — apart from the concept of proof on a balance of probabilities, which can

other than this. It is proposed briefly to discuss this apparent variation and its significance in toxic tort litigation. If evidence of a statistical nature, framed in terms of probabilities, is to be used it is particularly important clearly to articulate the standard of proof which is to be met.

Variation in the standard of proof

It has long been stated that there are variations within the 'balance of probabilities' standard. In *Bater* v *Bater*, Denning LJ (as he then was) said that

> in civil cases, the case may be proved by a preponderance of probability, but there may be degrees of probability within that standard.[112]

Although these words clearly state that variation *within* the civil standard, not variation *of* the standard, is involved, Denning LJ goes on to speak of 'a higher degree of probability'. It may be, then, that he is envisaging that courts should sometimes require, say, 65% probability, and sometimes only 51% probability. Alternatively, Denning LJ may be confusing the probability component of the standard with the evidence required to reach the standard. Thus the true position would be that the weight of the evidence, rather than the standard of proof itself, is what is variable.[113] For the plaintiff, however, this would make little difference.

The concept of persuasion

The standard of proof is most surely modified in a loose sense by the requirement of real persuasion on the part of the fact-finder, rather than a mere balance of probabilities. This concept in its modern form can be traced back in Australia to a statement of Dixon J (as he then was) in *Briginshaw* v *Briginshaw & Anor*:

> The truth is that, when the law requires the proof of any fact, the tribunal must feel an actual persuasion of its occurrence or its existence before it can be found. It cannot be found as a result of a mere mechanical comparison of probabilities independently of any belief in its reality.[114]

As a basis for this, Dixon J quoted *Wigmore* on *Evidence*:

> It [the quality of persuasion necessary] is said to be that state of mind in which there is felt to be a "preponderance of evidence" in favour of the demandant's proposition.[115]

be restated as the burden of showing odds of at least 51 to 49 that such-and-such has taken place.' The Court of Appeal in *Hotson* v *East Berkshire Health Authority & Ors* [1987] 2 WLR 287, 292 (per Sir John Donaldson MR) also stated this mathematical level of probability.

112 *Bater* v *Bater* [1951] P 35, 37.

113 This is suggested by his quotation of Best CJ (reference unspecified): 'In proportion as the crime is enormous, so ought the proof to be clear.'

114 (1938) 60 CLR 336, 361.

115 *Wigmore* on *Evidence* (2nd ed, 1923) vol v, sec 2498 (as cited in the judgment of Dixon J).

It is suggested that the two statements are in fact not altogether compatible. Actual persuasion of an occurrence or of the existence of a fact is not the same as a state of mind recognizing that there is a preponderance of evidence in its favour. The first has a pronounced subjective element, the latter only minimal subjectivity.

Dixon J went on:

> But reasonable satisfaction is not a state of mind that is attained or established independently of the nature and consequence of the fact or facts to be proved. The seriousness of an allegation made, the inherent unlikelihood of an occurrence of a given description, or the gravity of the consequences flowing from a particular finding are considerations which must affect the answer to the question . . .[116]

In short, Dixon J seems to have envisaged that *persuasion* or *reasonable satisfaction* are required in any case but that in certain cases more will be needed to establish that state of mind. On this latter point Dixon J would appear to be supported by Denning LJ in *Bater*. Both these mechanisms, speaking loosely, raise the standard of proof in that they place an additional weight of proof on the plaintiff.

Later, in *Jones* v *Dunkel & Anor*, Dixon J stated:

> 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.[117]

This conclusion was affirmed by the majority in *West* v *Government Insurance Office of New South Wales*.[118] In the recent mesothelioma case of *Wintle* v *Conaust*,[119] the majority of the Victorian Supreme Court endorsed the words of Dixon J in *Briginshaw*. It is submitted that this effective raising of the standard of proof is out of line both with other Australian authority[120] and with British authority[121] and also with the logic behind the more-probable-than-not standard. The reason for the 51% threshold is that it ensures the making of the fewest possible mistakes. If the threshold is raised the likelihood of the defendant wrongfully being found liable is decreased but the overall number of erroneous decisions is bound to increase.[122] In toxic tort cases the raised threshold makes the difficult task of adducing adequate evidence virtually impossible for the plaintiff.

Furthermore, *any* variability in the standard of proof or in the level of evidence required to reach it entrenches its essential subjectivity. Each finder

[116] *Briginshaw* (1938) 60 CLR 336, 362.
[117] (1959) 101 CLR 298, 305.
[118] (1981) 148 CLR 62, 66.
[119] [1989] VR 951, 953.
[120] See, for instance, the majority judgments in *Holloway* v *McFeeters* (1956) 94 CLR 470 and judgments of all members of the High Court in *Goodwin* v *The Nominal Defendant* (1979) 54 ALJR 84.
[121] See fn 112.
[122] See Richard Eggleston, *Evidence, Proof and Probability* (2nd ed, Weidenfeld and Nicolson, London, 1983) 42 and James Brook, 'Inevitable Errors: the Preponderance of the Evidence Standard in Civil Litigation' (1982) 18 *Tulsa LJ* 79, 86.

of fact will define it for him- or herself. This means that it could never be stated in advance what level of probability would need to be established by the plaintiff. This in turn means that epidemiological evidence expressing a statistical probability of causation, no matter how flawless, could never be of itself sufficient to win the causation issue for the plaintiff.

In the following sections on the validity of statistical evidence couched in terms of probability, it will generally be assumed that the proper standard of proof is the balance of probabilities or 51%. It is necessary, however, to bear in mind alternative views of the standard since these, and particularly the belief or persuasion requirement, underlie much of the resistance to the use of probabilistic data in the absence of particularistic data.

The use of evidence expressed as probability

The convergence of evidence and standard occurs where the evidence itself consists of a quantified probability of a particular event having caused a particular outcome. It may be that there is epidemiological evidence establishing a more than doubled risk of the plaintiff suffering a certain disorder after exposure to a certain substance. Alternatively, it may be that the circumstances of the case allow a probabilistic assessment of the cause-effect link, in other words that the available evidence makes it certain that there is an over 51% likelihood of the outcome being caused by a particular event.

A typical case of the latter type is where two cars collide, killing both drivers, and where there is no further evidence tending to establish negligence by one or the other. This situation can be argued to have occurred in *TNT Management Pty Ltd* v *Brooks*[123] (a High Court decision). The dictum of Murphy J in this case is significant in that it represents the use of *purely* probabilistic data to meet the standard of proof.

TNT Management — an Australian dictum on probability and the standard of proof

In this case the respondent's husband was killed when his semi-trailer collided with a pantechnicon driven by the appellant's employee, who was also killed. There were no eye-witnesses. The only evidence was the position of the vehicles on the road. The majority of the court drew from this the conclusion that it was reasonable for the trial judge to have found the appellant liable. Murphy J, however, based his agreement with the majority decision on grounds rejected by them. He argued probabilistically, maintaining that there were three possible explanations of the accident: either the plaintiff's negligence caused it, or the defendant's negligence caused it, or it was caused by the combined negligence of both parties. The plaintiff, who carries the burden of proof, must establish that the predominant likelihood was that the defendant's negligence caused the accident. To use language borrowed from the *Bonnington* line of cases,[124] Murphy J aggregated but-for cause with the

[123] (1979) 53 ALJR 267.
[124] See section 2 of this article.

concept of contribution. Thus, assuming that the probability of each party's negligence being the sole cause of the accident was equal, the proper course, according to him, was to add the probability of the defendant's sole responsibility to the probability of his joint responsibility (contribution). The result would then inevitably be that this aggregated causality would be greater than 51%.[125]

Such reasoning raises the objection that there is no genuine attribution of liability taking place here, that the outcome is not rooted in the circumstances of the individual case and therefore that the tribunal of fact cannot reach any requisite level of persuasion. This objection is important since it is also an objection to the use of epidemiological data to decide toxic tort cases.

The final clause of this objection was addressed by Murphy J in the case. He referred to the Dixonian view of the standard of proof expressed in *Briginshaw*[126] and considered it to be inconsistent with 'a system which applies the balance of probabilities as the standard of proof'.[127]

He also repudiated the idea of a shifting standard, higher when the consequences of liability would be more serious:

> The latter represents an abandonment of the balance of probabilities standard and its replacement by a test which differs from case to case and contains a subjective element.[128]

The dictum of Murphy J concerning the use of probabilistic evidence has not founded any decisions in Australia.[129] In the US, however, '[courts] have allowed litigants to place increasing reliance on epidemiological or statistical proof in answering cause-in-fact questions'.[130] Academic opinion which has long addressed itself to the role this evidence might play has been divided. For any victim of a disease, the incidence of which can be proved to increase by more than 100% after exposure to a certain substance, who can bring no further evidence of causation, the issue is crucial.

Objections to probabilistic evidence

One of the major concerns expressed with probabilistic evidence is that it cannot offer certainty.

In the US, Richard Wright is a major exponent of 'actual causation' in opposition of probabilistic views of causation. He distinguishes causal prediction (evidence of increased risk) from causal explanation (evidence

[125] It is questionable whether the plaintiff can reasonably assert that the area of joint responsibility is indivisible. It is arguable that, within this area, the defendant's action was causative only to the extent of his/her contribution, in this case 50% of the joint responsibility.

[126] (1938) 60 CLR 336; this view is detailed above.

[127] *TNT Management Pty Ltd* v *Brooks* (1979) 53 ALJR 267, 271.

[128] Ibid.

[129] Though it has aroused much interest in Australia and the UK. See, for instance, the writings of L Jonathan Cohen and Sir Richard Eggleston.

[130] Steve Gold, 'Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence' (1986) 96 *Yale LJ* 376, 377. A case in point is *Allen* v *United States*, 588 F Supp 247 (1984) (CD Utah).

pertaining to an individual case) and then turns his attention to 'naked statistical evidence':

> So long as tort liability continues to be based on individual responsibility, liability will be imposed on a defendant only if it is believed that the tortious aspect of his conduct actually contributed to the specified legal injury. This belief is the essence of a causal explanation, as distinguished from mere probabilistic statements of increased risk. The belief will arise in a particular case only if there is sufficient evidence that not only supports the suggested causal explanation involving the defendant's tortious conduct, but also makes it the most plausible suggested explanation. Ordinarily, only particularistic evidence fitting the relevant causal generalizations can accomplish this task.[131]

Wright, even more than Dixon J, insists on the presence of *belief*. One might almost forget that the civil rather than the criminal standard of proof is at issue. Indeed it seems as if Wright is regarding causation as an absolute philosophical notion rather than a pragmatically utilized legal notion which must, through the standard of proof, necessarily be subject to concepts of probability. If it is accepted that belief is legally unnecessary, the basis of Wright's argument disappears.

What seems particularly to worry the writers who reject the use of probabilistic evidence, however, is the disconcerting foreknowledge that if the finding of causation will inevitably be right in a certain percentage of cases, it will inevitably be wrong in a further percentage of cases. The illusion of certainty created by a proclamation of 'belief' is precluded.

Cohen, for instance, cites the case of 1000 rodeo visitors, 501 of whom have not paid for their tickets and 499 of whom have. Should the organizers of the rodeo wish to sue all 1000 visitors, they would win in each case, given that there was no further evidence of who had paid or who had not, even though it was certain that in 499 cases this decision would be wrong or that there was a 49.9% chance that in each case the decision would be wrong.[132]

Cohen (whose main objection is to commonly utilized notions of probability)[133] asserts that what is 'particularly offensive' about such reasoning is that 'it implies an official acceptance by the law that a man may legitimately lose his whole fortune in a lawsuit when there are 499 chances out of 1000 . . . that he is perfectly in the right. . . . It argues an extraordinary cynicism in the law if one claims that it officially and *de jure* recognizes so large a probability of error'.[134]

It does not appear from case law and academic writings that this perception of fundamental cynicism is widespread, though there is a growing feeling that the uncertainty component in the standard of proof should be more widely

[131] R W Wright, op cit 1826.
[132] L Jonathan Cohen, *The Probable and the Provable* (Oxford, Clarendon Press, 1977).
[133] See his discussion of 'Pascalian' versus 'Baconian' probability in *The Probable and the Provable*, ibid.
[134] L Jonathan Cohen, 'The Logic of Proof' (1980) *Crim LR* 91, 98.

recognized. This feeling finds expression in calls for recovery proportional to the likelihood of the defendant having caused the harm.[135]

Probative value of the two types of evidence — are they really different?

It is submitted that there is no substantive difference between particularistic and probabilistic evidence beyond the propensity of the latter to clarify in the mind of the fact-finder the realities of our standard of proof. Whatever state of mind is to be required of the decision-maker, it can be generated equally by both kinds of evidence. Indeed, probabilistic evidence may avoid the pitfalls which catch particularistic evidence:

> Abundant evidence from psychological research . . . suggests that in many contexts decision makers' intuitive, common-sense judgments depart markedly . . . from the actual probabilities. People use a number of simplifying operations, called "heuristics", to reduce the complexity of information which must be integrated to yield a decision. These simplifying strategies often lead to errors in judgment.[136]

As probability is ultimately what must be established in any case there is a considerable danger that mistakes will be made by decision-makers wrongly processing the particularistic data.

Furthermore, there is no doubt that case-specific information is itself fundamentally probabilistic. Saks and Kidd, quoting Tribe,[137] maintain: 'no conclusion can ever be drawn from empirical data without some step of inductive inference — even if only an inference that things are usually what they are perceived to be . . . .'[138] Questions of probability, then, lie hidden in every statement of fact, and whether the probability is quantified or not makes no difference:

> From the viewpoint of a disinterested fact finder, all information is indirect, distant, abstract, and imperfectly credible. The fact finders, in terms of their truth-seeking role, simply have a set of input information on which to base a judgment, and depending on the characteristics of the evidence and the way it is processed, that finding will have a greater or lesser probability of being correct.[139]

If a person is charged with possession of illegally imported goods and there is a choice between using perfectly reliable data that 90% of all such goods in the country are illegally imported or the testimony of a witness whom we judge to be 80% reliable that she saw the importation and delivery, we cannot say that the statistical data is more likely to lead us into error, or is essentially different from, that of the witness.[140] Nor is a judgment based on it less likely

[135] See David Rosenberg, 'The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System' (1984) 97 *Harv LR* 851.

[136] Michael J Saks and Robert F Kidd, 'Human Information Processing and Adjudication: Trial by Heuristics' (1981) 15 *Law & Soc'y Rev* 123, 127.

[137] Laurence H Tribe, 'Trial by Mathematics: Precison and Ritual in the Legal Process' (1971) 84 *Harv LR* 1329, 1330.

[138] Saks and Kidd, op cit 151.

[139] Id 152.

[140] Based on an example from Saks and Kidd, ibid.

to be right. Data is data and in this case the more probative, more diagnostic, information is the statistical data.

What is perhaps not sufficiently stressed by writers is that statistical evidence itself, like all evidence, must be subject to probabilistic analysis. It is wrong to accept a statistical statement of probability as the final statement determining whether the standard of proof has been reached, even if that statement is the only available evidence. As assessment must first be made of its probative value, and the expressed probability reduced by the amount of any uncertainty as to the statement's reliability.

None of the above allays discomfort at the statistical certainty that mistakes will be made and that many plaintiffs will either be left uncompensated or will make windfall gains at the expense of 'innocent' defendants. But this is not a problem with the evidence; it is a problem with the all-or-nothing rule.

The exclusive use of statistical evidence would, however, make at least one difference. It is probably true that to use it as determinative would tend to petrify the standard of proof. Where particularistic evidence is used and a complex process of inference-drawing takes place, there is room for unspoken, unadmitted, perhaps unperceived, reinterpretations either of the standard of proof or of the level of evidence required to satisfy it. With the removal of attention from the state of the fact-finder's mind, however, and its placement on the evidence itself, a great deal of that judicial flexibility is lost. But while it is true that the human face of justice may thereby become slightly dimmed it is illogical to exclude evidence which tends to establish precisely what is required by the burden of proof. Those unhappy with the use of probabilistic evidence should first challenge the legal requirements which invite its use.

In summary, the leukaemia victim would probably not recover in Australia or England on the basis of probabilistic epidemiological evidence alone. It would not be considered that she had established her case with sufficient particularity. In the United States her chances would be better.

It is submitted that there is no logical reason why, with sufficient well-based statistical evidence, she should not succeed. Production of statistical evidence, even in the absence of other evidence, is a valid way of 'proving causation'. And, as it is based on epidemiological data establishing increased risk, it does not require knowledge of causal mechanisms.

5. CONCLUSION

RE-EVALUATING THE DAMAGE: DISPOSING OF THE PROBLEM OF MEDICAL UNCERTAINTY

The conclusion cannot be escaped that legal antidotes to the problems of medical uncertainty are piecemeal. Concepts of causation can be broadened to include the relatively undemanding notion of contribution. Contribution itself can be broadened to include increased risk. An inference of causation may be drawn if sufficient evidence of the right type is adduced. Statistical

evidence expressing 51% or higher probability of causation *might* be permitted to elide with the standard of proof without further particularistic evidence needing to be adduced. In all these ways the problems of unspecifiable causal mechanisms can be camouflaged or avoided.

There is, however, another more radical and less piecemeal way of sidestepping the problems caused by a lack of knowledge of causal mechanisms. It has aroused considerable interest recently, particularly in the United States and Britain, and has been the subject of a much discussed UK court case — *Hotson* v *East Berkshire Health Authority*.[141] It is the notion of recovery for the loss of a chance or, expressed differently, recovery for increased risk.

The essence of this notion is that it should be possible to prove causation not of the medical condition suffered by the plaintiff but of the loss of that plaintiff's chance of a better outcome. All that would need to be established, then, would be that the defendant's breach caused the risk of an unfavourable outcome to be increased above a background level. The deceased mesothelioma victim would have lost his past chance of living a normal lifespan. The woman with leukaemia would have lost her future chance of the same. The complainant whose symptoms were aggravated would have lost his chance of less disabling manifestations of his disease. The plaintiff whose condition had been accelerated would have lost the chance of extra time without the disease. The lost chance, then, would be the damage itself, not just the means by which compensation could be calculated.[142]

The attractions of this view are many, since causation of loss of chance is a great deal easier to prove than causation of a medical condition. In *McGhee* v *National Coal Board*[143] it was effectively all that could be proved; hence the eagerness of the court to allow recovery on the basis of increased risk. In effect, many of the problems thrown up in the course of this article would be obviated.

Because the balance of probabilities standard would now apply to the proof of loss of chance it would not need to be applied to the proof of causation of the condition. Logically *any* material loss of chance,[144] once proved, would lay the ground for recovery (though in order for the recovery to be more than nominal the loss would have to be quantified). Immediately, then, the need for knowledge of precise causal mechanisms would disappear along with the

[141] *Hotson* v *Fitzgerald & Ors* [1985] 1 WLR 1036 (Queen's Bench), *Hotson* v *East Berkshire Health Authority* [1987] 2 WLR 287 (Court of Appeal) and *Hotson* v *East Berkshire Health Authority* [1987] 3 WLR 232 (House of Lords).

[142] See generally Stephen F Brennwald, 'Proving Causation in "Loss of a Chance" Cases: A Proportional Approach' (1985) 34 *Catholic UL Rev* 747, Joseph H King Jr, 'Causation, Valuation, and Chance in Personal Injury Torts Involving Preexisting Conditions and Future Consequences' (1981) 90 *Yale LJ* 1353, J Stapleton, op cit and David P T Price, 'Causation — The Lords' Lost Chance?' (1989) 38 ICLQ 735. More recent articles debating the issue are: T Hill, 'A Lost Chance for Compensation in the Tort of Negligence by the House of Lords' (1991) 54 MLR 511 and W Scott, 'Causation in Medico-Legal Practice: A Doctor's Approach to the "Lost Opportunity" Cases' (1992) 55 MLR 521.

[143] [1973] 1 WLR 1.

[144] Possibly under 51%.

sometimes questionable inference-drawing which has allowed courts to infer the existence of causal mechanisms when even expert evidence could not attest to their existence.

Secondly, the but-for test need no longer be applied to the link between the defendant's action and the condition in question. As the but-for test requires an intimate knowledge of causal mechanisms this would be of considerable benefit to the plaintiff.

Thirdly, the requirement for particularistic evidence could no longer be determinative since a calculation of loss of chance would of necessity be couched in terms of statistical probability. Statistical data, properly used, would in many cases be the best data.

Finally, the damages awarded would be proportional, not to the damage expressed as loss of chance, but rather to the full loss occasioned by an individual. No longer would full damages be awarded where there was a 49% probability that there was no causal link between the defendant's act and the medical condition or where there was no more than a contribution to a harm-causing state of affairs. The damages awarded would exactly correspond with the quantified loss of chance.

Mass and individual loss of chance

In a given community, if the risk of a particular disease occurring were increased 20% by the behaviour in question then all sufferers from the disease would be able to recover. This would be in spite of the fact that 80% of people who contracted the disease would have done so through the background risk. The damages awarded to each plaintiff would be 20% of the total estimated loss to that plaintiff occasioned by the medical condition or by the aggravation or acceleration of the condition, whichever was in question. Thus the cost which each plaintiff would recover would be modest while the overall payout to a group, each of whose members had lost a percentage chance of avoiding the outcome, would correspond precisely with the cost of the defendant's breach of duty to that group (ie it would be exactly the same as if the correct but unidentifiable 20% of the population whose disease was caused by the breach were fully compensated and the other unidentifiable 80% got nothing). The type of evidence to be adduced in cases like this would be epidemiological evidence.

In cases of alleged individual loss of chance (for instance in cases of workplace injury), expert opinion evidence would need to be adduced to indicate the level of loss faced by the plaintiff.

In certain of the cases we have looked at there is no explicit finding of increased risk, but it can probably be inferred that increased risk was assumed by the courts. In the cases of contribution to a harm-producing aggregate, for instance, the contribution at issue is not quantified but we may probably assume that in satisfying the *de minimis* rule the contribution must have had the capacity to increase the risk of the harm occurring. The problem is, however, that the contribution was not quantified because it was not quantifiable. If it is not known how much the risk was increased, how can any calculation of

damages be made? In general, courts do not seem to be unduly disturbed by such uncertainty in apportioning damages. For instance, in *Thompson & Ors* v *Smiths Shiprepairers (North Shields) Ltd*[145] and *Andrews* v *SC Lohse & Co and Ors*,[146] damages were awarded on extremely rough calculations of relative levels of contribution. This could be done here too, though it is not suggested that this situation is ideal.

The theory, of course, needs much further consideration and fine-tuning, which it is not within the scope of this article to do. Should recovery be allowable when the physical condition, risk of which has been increased, has not yet eventuated? Should *any* loss of chance be sufficient to found an action for damages? Should it be limited to, say, 25% loss of chance or more? Should one be concerned that under this system plaintiffs whose condition was fully attributable to the defendant's breach would recover only partial damages or that all those whose disease was in fact not caused by the defendant's breach would automatically recover?

Finally, how should the risk be calculated? This question brings the issue back to one of causation and to the British case of *Hotson.*

Hotson v *East Berkshire Health Authority*[147]

In this case a thirteen year-old boy was injured in a fall at school. Negligently conducted medical examinations failed to reveal a displacement of the left epiphysis. When the full extent of the damage was ascertained full recovery was impossible in spite of surgery. The boy suffered avascular necrosis, a condition leading to permanent disability. It was argued by the plaintiff that permanent damage could have been avoided if the boy had been diagnosed and operated on immediately, that the negligence of the defendant had destroyed his chance of recovery. The defendants adduced expert evidence that from the start the injury was so bad that the boy's disability was inevitable; there had been no chance of recovery to lose. The central medical issue was whether, immediately after the accident, enough blood vessels in the affected area were still operative so as to be able, given treatment, to prevent avascular necrosis from occurring or whether they had already been destroyed.

The judge at first instance made two crucial findings. One was that it was more probable than not that the accident had already destroyed the blood vessels. He estimated the probabilities at 75/25. His second finding was that the boy had therefore lost a 25% chance of recovering fully.

The Court of Appeal accepted this latter estimate of a 25% chance of recovery and considered that, as there was no reason why the categories of loss should be closed, the plaintiff had made out a case for compensation for the loss of this 25% chance caused by the defendant.

The decision was overturned by the House of Lords, which in effect held that it was not permissible for the trial judge to have used his own state of

[145] [1984] 1 All ER 881.
[146] [1986] Aust Torts Reports 80–043.
[147] See fn 142.

doubt as to the health of the relevant blood vessels as a measure of the plaintiff's chance of recovery. In other words the House did not accept that, because the judge had found a possibility of 25% that the blood vessels were initially intact, this necessarily meant that a 25% chance of recovery had been lost. As Lord Mackay of Clashfern said:

> The concluding sentence in the judge's fourth finding in fact makes it plain . . . that he took the view, weighing that testimony along with all the other matters before him, that it was more probable than not that insufficient vessels had been left intact by the fall to maintain an adequate blood supply to the epiphysis and he expressed this balance by saying that it was 75 per cent. to 25 per cent. . . . Although various statistics were given in evidence, I do not read any of them as dealing with the particular probability which the judge assessed at 75 per cent. to 25 per cent. . . . It is not, in my opinion, correct to say that on arrival at the hospital he had a 25 per cent. chance of recovery. If insufficient blood vessels were left intact by the fall he had no prospect of avoiding complete avascular necrosis whereas if sufficient blood vessels were left intact on the judge's findings no further damage to the blood supply would have resulted if he had been given immediate treatment . . .[148]

It is submitted that this reasoning is correct.[149] To say that there was a 25% probability that there was a chance is not to say that there was a 25% chance. The extent of a chance cannot be quantified by the probability of it existing.

The trial judge had established that on the balance of probabilities there had been no loss of chance of recovery and as this is precisely the question which must be answered in loss of chance cases it was logically correct that the plaintiff could not recover. Assuming that the balance of probabilities test left a residual possibility that a chance of recovery had existed, the boy had lost his opportunity to test this possibility. This is certainly a loss of chance but it is not the loss that was being tested for. It is not the loss of a 25% chance of recovery. Thus, the decision in itself was not a rejection of the loss of chance argument per se.

What needs to be done in cases of this kind, where loss of chance is alleged, is to adduce expert evidence quantifying the loss and thus identifying the damage for which compensation is sought. Causation of this damage must *then* be proved on the balance of probabilities.

The Law Lords in *Hotson* were cautious on the question of the admissibility of defining increased risk or loss of chance as damage. They did not consider it necessary to decide the issue in this case, as loss of chance had not been proved anyway. They did not, however, preclude the possibility that it might form a head of damage.[150]

Given the deep dissatisfaction expressed with conventional all-or-nothing recovery, the acceptance of the loss of chance concept in contract law[151] and the fact that acceptance of it leaves virtually undisturbed conventional

[148] *Hotson* v *East Berkshire Health Authority* [1987] 3 WLR 232, 240.
[149] Although it has been strongly criticised by, for instance, J Stapleton, op cit.
[150] See *Hotson* [1987] 3 WLR 232, 238 (per Lord Bridge) and 240 (per Lord Mackay).
[151] See *Chaplin* v *Hicks* [1911] 2 KB 786.

notions of causation, standards of proof and onus of proof, it is anticipated that it is a line of thought that may well prove fruitful in the future. And not the least of its benefits will be that it offers a means of recovery to plaintiffs in situations where medical science cannot offer an explanation of the mechanism which caused their disease.