BASIC INTRODUCTION TO ASBESTOS CLAIMS

A. INTRODUCTION

The HSE state that asbestos can be found in any building built before the year 2000 (houses, factories, offices, schools, hospitals etc) and causes around 5,000 deaths every year. This is more than the number of people killed on the road.

When materials that contain asbestos are disturbed and damaged, fibres are released into the air. When these fibres are inhaled they can cause serious diseases. The diseases will not affect you immediately, they often take many years to develop but once diagnosed it can be too late to do anything about it.

B. WHAT IS ASBESTOS?

Asbestos is a naturally occurring fibrous mineral which comes in several forms – all of which are hazardous to health.

It is used worldwide and mined in many countries such as Canada, Russia, South Africa, India and parts of South America.

Asbestos used to be thought of as being a wonderful material. Asbestos fibres are:

- Resistant to heat and are fire proof
- Resistant to many chemicals
- Strong: stronger than steel
- Light weight
- A good insulator against heat, frost and noise.

Asbestos has widely been used as a cheap filler for building materials, brake linings, clutches, gaskets and seals.

There are different types of asbestos. It is a generic term used to describe 6 different varieties of fibrous mineral silicate which can be divided into two groups:-

(1) Amphibole Asbestos group: including Crocidolite (blue), Amosite (brown), Tremolite, Actinolite and Anthrophylite; and

(2) Serpentine Asbestos. The only type of Serpentine Asbestos is Chrysotile (white) but this accounts for 95% of the world's asbestos production. Serpentine asbestos is softer and more flexible than amphibole asbestos.

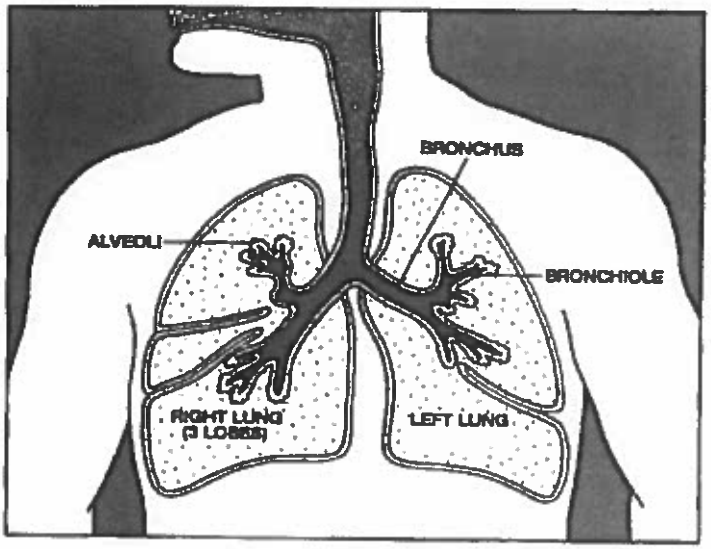
The Amphibole group accounts for most of the adverse consequences.

C. WHY IS ASBESTOS DANGEROUS?

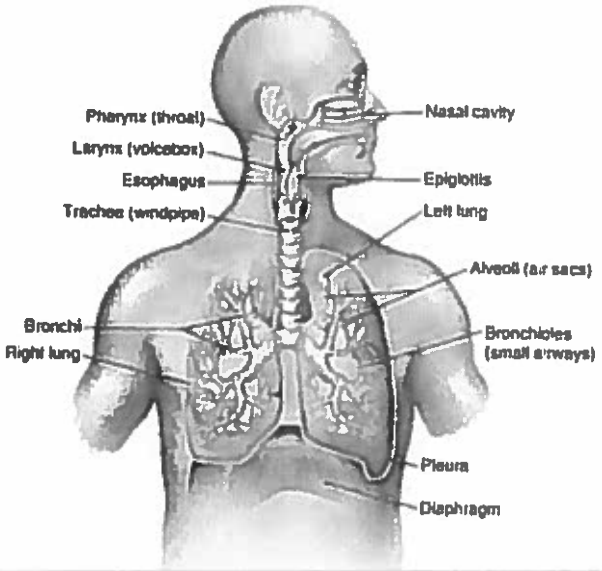
Asbestos is made up of thin fibres which can break down into small and thin fibres. The smallest fibres cannot be seen by the naked eye but can be breathed in. The fibres that are breathed in can get stuck in the lungs and damage them. A brief inhalation of asbestos may be filtered out in mucous but any particles that are not filtered out may reach the lungs (in particular, any particles reaching the alveoli are deposited at the alveolar duct bifurcations).

The iron content of asbestos fibres is thought to be an important factor. Chrysotile has no iron content and is therefore less of a stimulus than other Amphiboles.

D. DIAGRAMS OF THE LUNGS & THE RESPIRATORY SYSTEM



Respiratory System



E. ASBESTOS RELATED DISEASES

1. Pleural plaques
2. Diffuse pleural thickening
3. Asbestosis
4. Asbestos related lung cancer
5. Mesothelioma

1. Pleural plaques

Pleural plaques are raised small irregular patches of thickening or scarring found on the lining of the chest wall or on the diaphragm. They may or may not show on x-ray. In most cases they can be detected by CT Scan. Sometimes the plaques are "calcified" and this may be referred to as "calcified pleural plaques".

Usually symptoms will not be experienced. Pleural plaques is seen as the least serious asbestos disease and are in themselves not dangerous. They are, however, an indication that there has been exposure to asbestos and there is a chance that because of that exposure the Claimant may develop other types of asbestos disease in the future.

Ridiculously, in England and Wales damages are not awarded for pleural plaques.

2. Diffuse pleural thickening

Asbestos fibres may attack and thicken the pleura surrounding one or both lungs. When the thickening is extensive the lungs are restricted making breathing difficult. Inflammation causes severe pain – similar to pleurisy.

Early pleural thickening can be detected by CT Scan but the radiation dose is 20 times that for a chest radiograph so the Industrial Injuries Advisory Council recommend that diagnosis should be by chest radiogram. Normally thickening increases slowly.

Diffuse pleural thickening is similar in appearance to pleural plaques but the thickening is more widespread and may involve both layers of the pleura.

3. Asbestosis

Asbestosis is the fibrosis of scarring of the lung tissue caused by asbestos fibres inhaled many years earlier. It is also sometimes called "interstitial pulmonary fibrosis". This means that the lung itself is damaged by the body's inflammatory reaction to asbestos fibres. Inflammation results in scar tissue, or fibrosis, building up in the interstitium (tissue between the air sacs). The build up of the fibrosis reduces the elasticity of the lungs and they become stiff making it less easy for the lungs to inflate on breathing.

Symptoms include shortness of breath, coughing, tightness of the chest and tiredness. Breathing is more difficult partly as the lungs shrink and become progressively tougher so that less oxygen is absorbed. This can be detected by a lung function test before the fibrosis shows on an x-ray or scan.

4. Asbestos Related Lung Cancer

Workers exposed to asbestos have an increased risk of developing lung cancer.

Typical symptoms include coughing, shortness of breath and chest pain. Coughing up blood or weight loss are very suggestive. Some patients may have symptoms related to secondary cancers in their bone or liver such as back pain or jaundice.

Treatment usually involves a combination of surgery, radiotherapy and chemotherapy. It is very difficult to distinguish lung cancer caused by asbestos and that caused by smoking or other environmental pollutants.

As a result of these diagnostic difficulties the Department for Works and Pensions does not accept a diagnosis of asbestos related lung cancer unless the Claimant also has a diagnosis of asbestosis or has worked in 4 specified manufacturing industries for a specified period of time *(the manufacture of asbestos textiles; spraying asbestos; asbestos insulation work; applying or removing materials containing asbestos in the course of shipbuilding – where the exposure occurred before 01/01/75 for a period which amounts to an aggregate of 5 years or more or where the exposure occurs from 01/01/75 where the period was for 10 years or more).*

The risk of developing lung cancer has greatly increased for anyone who has inhaled asbestos fibres and also has smoked cigarettes. Once smoking stops the risk becomes less each year.

Mortality rates are summarised by Dr. Ward, DSS 1981, are as follows:-

Non-asbestos exposed, non-smoking controls	1.00
Non-asbestos exposed, smokers	10.85
Asbestos exposed non-smokers	5.17
Asbestos exposed smokers	53.24

The Relationship Between Asbestos Exposure and Lung Cancer

Allen Wilson, a lecturer for MBL, has reported:-

Most of the studies that have identified high risks of lung cancer from asbestos exposure are carried out among workers whose exposure was mainly between 1940 and 1970. Subsequent studies have generally not found clear evidence of excessive risks of lung cancer from asbestos exposure, unless the dose has been sufficiently high.

Fibre Burden In Lung Tissue Found in Asbestos Related Conditions

Condition	Mesne Asbestos (Bodies/Gramme)
Asbestosis and Lung Cancer	28,600
Asbestosis	17,100
Pleural Fibrosis	3,000
Malignant Mesothelioma with Pleural Plaques	900
Pleural Plaques	300

The degree of asbestos exposure necessary to cause lung cancer is substantially higher than the dose necessary to cause asbestosis. In other words, it is relatively easy for a person to develop pleural plaques as a result of exposure to asbestos but it is difficult to get lung cancer.

What does this mean in terms of causation? Asbestosis is therefore an essential pre-condition for the development of asbestos related lung cancer.

Animal studies, epidemiological evidence and post-mortem studies all support this. The latest review in 1998 confirmed that pulmonary fibrosis is the pre-cursor of carcinoma. The upshot is that on the balance of probabilities any cancer claim without fibrosis is likely to fail. The huge amount of material confirming the link between smoking and lung cancer do not help, although the risk is highly age correlated. Adenocarcinoma accounts for about 30% of all smoking related cancers. The conclusion is that the overwhelming consensus of scientific evidence does not support an association between lung cancer and asbestos exposure in the absence of asbestosis.

The conclusion is that the overwhelming consensus of scientific evidence does not support an association between lung cancer and asbestos exposure in the absence of asbestosis. Certainly, any such claims are likely to be robustly defended.

Allen Wilson suggested that if there is a high asbestos exposure, no asbestosis and lung cancer at the moment most experts would gloss over the fact that there is no asbestosis present.

If there is a history of smoking most doctors would say that the smoking is the primary cause of the lung cancer and not just a contributory factor.

5. **Mesothelioma**

This is also referred to "diffuse" or "malignant" mesothelioma. It can affect the pleura (the lining of the lungs), the peritoneum (the lining of the abdomen) and the pericardium (the area around the heart). It is a very aggressive cancer for which there is no known cure. It can take 60 years to develop and is usually associated with exposure to asbestos – which can be exposure of a short duration.

The latency period may be as short as 10 years or longer than 60 years. There have been cases where family members of workers who had asbestos on clothing when they arrived home have developed mesothelioma.

Unlike other asbestos diseases, mesothelioma is not dose related.

An early sign of mesothelioma is a build up of fluid in the pleural space around the lung or the peritoneal space in the abdomen. This can cause pain and breathlessness. It can be very difficult to make a diagnosis of mesothelioma and more than one biopsy (very small sample tissue) is sometimes needed.

As the cancer progresses the symptoms worsen and some patients can develop difficulty swallowing or congested blood vessels as the cancer invades local structures. Treatment usually focuses on relieving the patient's symptoms. Radiotherapy can help some

symptoms. In some patients this cancer can cause fluid on the lung and draining the fluid may help.

The mesothelioma spreads slowly to cover and compress the lung or abdominal wall and organs. It can spread to other parts of the body but is less likely than other cancers to do so.

Macmillan nurses are specialists in handling this illness and have produced booklets giving advice about it.

F. THRESHOLD EXPOSURE FOR LUNG CANCER & MESOTHELIOMA

There is an exposure level below which there is no increased risk of either lung cancer or mesothelioma. Any increased risk for lung cancer is restricted primarily due to smoking.

The cumulative lifetime asbestos exposure used to be 25 fibres/ml-years (1 fibre/ml per year x 25 years). This is now believed to be too high. A threshold of 20 fibres/ml-year (or 0.8 fibres/ml x 25 years) is now considered to provide a reasonable safety margin even for asbestosis.

G. LUNG FIBRE ANALYSIS

If a Coroners Pathologist uses only optical microscope they may not be able to see and identify asbestos fibres. The Coroner may then ask the Environmental Lung Diseases Research Group at Llandough Hospital to look for asbestos using electron microscope analysis of lung tissue.

Problems that can affect fibre counts:-

1. Results are not reproducible. They can vary greatly even when the same laboratory investigates adjacent cubic centimetres of a lung using a standard technique.
2. Not more than 2 grams of lung tissue can be processed at a time and it takes 2 days to prepare grids from that tissue for examination. Fibres are not evenly distributed throughout the lung and even if 0.5 grams of tissue are taken from 4 different areas of the lung and pulled to give 2 grams it is not possible to ensure that the 2 grams of tissue examined are representative of the entire lung.

H. KEY EVENTS TIMELINE

Year	Description
1901	The Factory Workshop Act 1901. Section 79 of the 1901 Act was headed "Dangerous and Unhealthy Industries". It provided "Any manufacturer, machinery, process or description of manual labour, used in factories or workshops that is dangerous or injurious to health". Subsequently, the Asbestos Industry Regulations 1931 were made under this Section of the Act. The Act was amended and changed in various forms until the introduction of the Health and Safety at Work Act 1974.
1930	The Merewether and Price Report 1930. The UK Government commissioned Merewether and Price to investigate and report on the state of health of individuals working with asbestos. Their report was published in 1930. It showed that chronic exposure to high concentrations of asbestos could lead to asbestosis. Subsequently the 1931 Asbestos Industry Regulations were published and came into force on 01/03/1932. The general thrust of the 1931 Regulations was directed towards the asbestos manufacturing industries. However, Merewether and Price did recognise that other users of asbestos products, such as laggers and shipbuilders, were heavily exposed to asbestos dust and could be at risk.
1931	The Asbestos Industry Regulations 1931 SI number 1140 (in force 01/03/1932). The Regulations applied to the asbestos manufacturing industry and were intended to prevent the release of asbestos dust into the workplace.
1961	The Factories Act 1961. The main flow of the 1961 Regulations was that they were industry specific and didn't apply outside the asbestos industry. This prolonged the period during which insulaters, plumbers, boiler makers, shipyard workers and others were exposed to asbestos dust.
1967	Writ issued in the first successful personal injury claim for a victim of negligent exposure to asbestos dust. The case went to the Court of Appeal in 1971. The case paved the way to subsequent asbestos claims and claims for pleural plaques compensation in the UK. The issuing of the Writ led to publicity, one of the effects of which was the issuing of the Asbestos Regulations 1969.
1969	The Asbestos Regulations 1969 SI number 690 (in force 14/05/1970). These Regulations applied to building sites and factories where asbestos was used, not to the asbestos manufacturing industry alone. They imposed "control limits" for each type of asbestos.
1970	Voluntary Import Ban on the import of raw blue asbestos.
1974	The Health and Safety at Work Act 1974 requires employers to conduct their work in such a way that their employees will not be exposed to health and safety risks and to provide information to people about their workplace that might affect their health and safety.
1980	The Voluntary Asbestos Import Ban. In 1980 the asbestos industry agreed to a similar ban to that of 1970 this time in relation to brown asbestos.

1983	1983 The Asbestos Licensing Regulations 1983 meant that Contractors working with asbestos insulation or coating were required to get a license from the Health and Safety Executive.
1985	Asbestos (Prohibitions) Regulations 1985 SI number 910 (in force 01/01/1986). The importation of Crocidolite (blue) and Amosite (brown) asbestos banned. The supply of blue/brown asbestos or any product containing them for use at work banned. Spraying or the installation of insulation containing any type of asbestos banned.
1985	The Asbestos Products Safety Regulations 1985 (subsequently amended in 1987). These Regulations prohibited the supply and use of blue and brown asbestos containing products and require that any products containing other asbestos types were labelled in accordance with the Schedule to the Regulations. The label was to give a warning that asbestos was present and to give details about precautions to be taken when handling the products.
1987	The Asbestos Products Safety (Amended) Regulations 1987. These amended Regulations implemented EC Directives on the marketing and use of asbestos.
1987	The Control of Asbestos at Work Regulations 1987. These Regulations introduced statutory control procedures to prevent workers from exposure to asbestos at work. They required employers to prevent exposure of employees to asbestos or reduce that exposure to the lowest reasonably practicable level.
1988	The Asbestos Prohibition (Amended) Regulations 1988 in addition to the prohibition of import, supply and use of blue and brown asbestos introduced a prohibition on the spray application of asbestos paint and other compositions containing any asbestos type.
1990	The Control of Asbestos in the Air Regulations 1990 imposed an emission limit of 0.1mg/M3 for asbestos emissions to the air by industrial installations utilising asbestos processes. The Regulations also included further general provisions to prevent significant environmental pollution from activities involving the working of products containing asbestos.
1992	Asbestos (Prohibitions) Regulations 1992 SI number 3067 (in force 01/01/1993). Importation into the UK and any new use of all forms of Amphiboles banned. Also, certain uses of Chrysotile banned.
1992	Control of Asbestos at Work (Amendment) Regulations 1992 SI number 3068 (in force 01/01/1993). Control limits for all forms of asbestos lowered. Regulation 5(1)(a) – where practicable substitute a less hazardous substance for asbestos (including Chrysotile).
1999	Asbestos (Prohibitions) (Amendment) Regulations 1999 SI number 2373 (in force 24/11/1999). The importation, supply and use of Chrysotile and products containing Chrysotile banned. A very few specialised uses are exempt for a limited period. The sale of second hand asbestos cement sheets and boards covered with paint or plaster containing Chrysotile banned. Supply included sale, lease, hire, loan, give or exchange. Products in use which contain any form of asbestos which remain in good condition may continue in use until the end of their service life.
1999	The Asbestos Prohibition (Amended) (Number 2) Regulations 1999 extended the prohibition of the import, supply and use of all vehicle

	asbestos containing brake linings including those for motorcycles.
2002	The Control of Asbestos at Work Regulations 2002 updated and extended many of the earlier Regulations. The main change was to introduce an explicit duty to manage asbestos in all non-domestic properties. So great was this change that a 2 year lead-in period was granted. A duty to manage came in force in May 2004.
2003	The Asbestos Prohibition (Amended) Regulations 2003 changed definitions which were included within the 1999 Amendments. The definitions were designed to make definitions less ambiguous and to reduce legal argument.
2003	The Asbestos Licensing (Amended) Regulations 2003. These Regulations were implemented to reduce the mandatory notification period of 28 days to 14 days. Method statements to be submitted to the HSE for approval at least 14 days before the commencement of asbestos removal/remediation work.
2006	The Control of Asbestos Regulations 2006 combine the Control of Asbestos at Work Regulations 2002, the Asbestos (Licensing) Regulations 1983 and the Asbestos (Prohibitions) Regulations 1992 and all of their amendments into one set of Regulations. These regulations aimed to minimise the use and disturbance of asbestos containing materials within workplaces. Essentially this legislation bans the import and use of most asbestos products and sets out guidelines on how best to use those currently in situ. Also action levels were removed and were replaced with a new single control limit of 0.1f/cm3 and a short term exposure limit (stel-acop standard, not in the Regulations) of 0.6f/cm3 measured over 10 minutes. A new World Health Organisation asbestos fibre counting method was introduced to replace the European Reference Method (ERM).
2012	The Control of Asbestos Regulations 2012 came into force 06/04/12 to take account of the European Commission's view that the UK had not fully implemented the EU Directive on exposure to asbestos (Directive 2009/148/EC). The changes were relatively minor and included an additional requirement for non-licensed asbestos work. The changes mean that some non-licensed asbestos work now requires notification and has additional requirements for managing this work, e.g. record keeping and health surveillance.
2013	HSE published revised guidance on managing and controlling asbestos

I. Task

Facts:

Mrs F's husband developed mesothelioma as a result of asbestos poisoning. He worked for 2 consecutive employers where he was exposed to asbestos in his work. Both employers breached their duty of care for him by exposing him to asbestos, but it couldn't be determined which breach actually led to the poisoning, or if they both did.

In other words:

1. C was employed by two different companies (A & B) at different times;
2. Both A & B owed a duty of care to C;
3. Both A & B breached their duty to C when he worked for them;
4. C suffers from an injury directly related to the breach of duty;
5. Any other cause of injury can be effectively ruled out; and
6. C cannot prove when the injury developed or who was responsible.

Topic for discussion:

If the "but for" test is applied what would be the outcome?

What should the Court have decided in relation to liability?

J. FAIRCHILD v GLENHAVEN FUNERAL SERVICES LTD & OTHERS [2002] UKHL 22 (20 June 2002)

Lower Courts

The Claimant suffered from mesothelioma (a condition which isn't dose related). The lower Courts took into account that the malignant transformation of a cell into a tumour is thought to have been initiated by a single asbestos fibre and as there was more than 1 exposer from whom the fibre could have come, the Claimant couldn't prove from where the fibre had come. The Judges applied the "but for" test and determined that neither party can be found liable because it cannot be proven that the outcome would have occurred without either of their actions.

House of Lords

In the House of Lords, Lord Bingham used the principle in McGhee v National Coal Board to formulate his own specific formula for determining liability in cases like this. He breaks the facts into 6 specific steps that must be present for his decision to apply, and states that when they are present the Claimant is entitled to recover against both Defendants. He emphasizes that this only applies when all 6 steps are present.

Within his Judgment he states:

2. The essential question underlying the appeals may be accurately expressed in this way. If

> (1) C was employed at different times and for differing periods by both A and B, and
> (2) A and B were both subject to a duty to take reasonable care or to take all practicable measures to prevent C inhaling asbestos dust because of the known risk that asbestos dust (if inhaled) might cause a mesothelioma, and
> (3) both A and B were in breach of that duty in relation to C during the periods of C's employment by each of them with the result that during both periods C inhaled excessive quantities of asbestos dust, and
> (4) C is found to be suffering from a mesothelioma, and
> (5) any cause of C's mesothelioma other than the inhalation of asbestos dust at work can be effectively discounted, but
> (6) C cannot (because of the current limits of human science) prove, on the balance of probabilities, that his mesothelioma was the result of his inhaling asbestos dust during his employment by A or during his employment by B or during his employment by A and B taken together,

is C entitled to recover damages against either A or B or against both A and B?

To this question (not formulated in these terms) the Court of Appeal (Brooke, Latham and Kay LJJ), in a reserved judgment of the Court reported at [2002] 1 WLR 1052, gave a negative answer. It did so because, applying the conventional "but for" test of tortious liability, it could not be held that C had proved against A that his mesothelioma would probably not have occurred but for the breach of duty by A, nor against B that his mesothelioma would probably not have occurred but for the breach of duty by B, nor against A and B that his mesothelioma would probably not

have occurred but for the breach of duty by both A and B together. So C failed against both A and B.

The crucial issue on appeal is whether, in the special circumstances of such a case, principle, authority or policy requires or justifies a modified approach to proof of causation.

Principle

8. In a personal injury action based on negligence or breach of statutory duty the Claimant seeks to establish a breach by the Defendant of a duty owed to the claimant, which has caused him damage. For the purposes of analysis, and for the purpose of pleading, proving and resolving the claim, lawyers find it convenient to break the claim into its constituent elements: the duty, the breach, the damage and the causal connection between the breach and the damage. In the generality of personal injury actions, it is of course true that the Claimant is required to discharge the burden of showing that the breach of which he complains caused the damage for which he claims and to do so by showing that but for the breach he would not have suffered the damage.

9. The issue in these appeals does not concern the general validity and applicability of that requirement, which is not in question, but is whether in special circumstances such as those in these cases there should be any variation or relaxation of it. The overall object of tort law is to define cases in which the law may justly hold one party liable to compensate another. Are these such cases? A and B owed C a duty to protect C against a risk of a particular and very serious kind. They failed to perform that duty. As a result the risk eventuated and C suffered the very harm against which it was the duty of A and B to protect him. Had there been only one tortfeasor, C would have been entitled to recover, but because the duty owed to him was broken by two tortfeasors and not only one, he is held to be entitled to recover against neither, because of his inability to prove what is scientifically unprovable. If the mechanical application of generally accepted rules leads to such a result, there must be room to question the appropriateness of such an approach in such a case.

Conclusion

34. To the question posed in paragraph 2 of this opinion I would answer that where conditions (1)-(6) are satisfied C is entitled to recover against both A and B. That conclusion is in my opinion consistent with principle, and also with authority (properly understood). Where those conditions are satisfied, it seems to me just and in accordance with common sense to treat the conduct of A and B in exposing C to a risk to which he should not have been exposed as making a material contribution to the contracting by C of a condition against which it was the duty of A and B to protect him. I consider that this conclusion is fortified by the wider jurisprudence reviewed above. Policy considerations weigh in favour of such a conclusion. It is a conclusion which follows even if either A or B is not before the court. It was not suggested in argument that C's entitlement against either A or B should be for any sum less than the full compensation to which C is entitled, although A and B could of course seek contribution against each other or any other employer liable in respect of the same damage in the ordinary way. No argument on apportionment was addressed to the House. I would in conclusion emphasise that

my opinion is directed to cases in which each of the conditions specified in (1) - (6) of paragraph 2 above is satisfied and to no other case. It would be unrealistic to suppose that the principle here affirmed will not over time be the subject of incremental and analogical development. Cases seeking to develop the principle must be decided when and as they arise. For the present, I think it unwise to decide more than is necessary to resolve these three appeals which, for all the foregoing reasons, I concluded should be allowed.