Occupational disease and work related disease claims present practitioners with new and different challenges to those arising from an accident or other single event.

Over the past decade disease claims have generated a significant amount of case law. New disease and work related conditions have been the subject of claims.

Practitioners need a means of capturing and retaining the wealth of disease related data which crosses their desk almost on a daily basis. Kennedys’ Occupational Disease Unit has therefore produced a pocket-sized manual or reference guide which provides information on a number of occupationally induced conditions and which will hopefully be a useful aide memoire of the relevant case law.

I sincerely hope that the manual will prove to be a very useful and portable tool.

Kennedys offer a national occupational disease service from the offices in Birmingham, Chelmsford, London, Manchester, Taunton and Sheffield.

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Asbestos Related Illness

Asbestos is a naturally occurring fibrous silicate mineral with well known fire resistant properties. Its fibrous structure allows it be spun and woven into cloth. Besides its fire resistance, asbestos is an efficient heat and sound insulator.

When mixed with cement and water, “asbestos cement” can be moulded to make pipes, boards and tiles. It can be applied by hand to lag heating pipes and boilers. Certain types of asbestos fibre can even be sprayed. Sprayed asbestos insulation known as “Limpet” spray was used liberally inside ships and power stations.

TYPES OF ASBESTOS FIBRE

The main types of asbestos fibres are:

**Crocidolite** – blue asbestos

**Amosite** – brown asbestos

**Chrysotile** – white asbestos

Crocidolite and Amosite belonged to a category of asbestos fibres known as **amphiboles**. Such fibres are short and rigid and once ingested, are difficult for the lungs to expel.

Chrysotile (white asbestos) has longer but soft, curly fibres known as “serpentine” fibres. These fibres are more easily cleared from the lungs by the act of coughing.

Because they are more difficult for the body to expel, amphibole fibres have a greater propensity to cause asbestos induced illnesses, particularly asbestos induced cancers.

Crocidolite is 500 times more toxic in the causation of mesothelioma than is chrysotile. Amosite is 100 times more toxic than chrysotile in causing mesothelioma. Fortunately, 95% of all of the asbestos which was imported into the UK comprised chrysotile.

In 1930, the **Merewether and Price** report highlighted the incidence of asbestosis inside asbestos manufacturing workshops. Subsequently, their recommendations were given statutory force by the introduction of the **Asbestos Industry Regulations 1931**.

Dr Richard Doll made the first association between asbestos exposure and the incidence of lung cancer in 1955. He noted a significant increase in bronchial tumours amongst asbestotics. Then in June 1960 a paper was published by an epidemiologist, **Dr Christopher Wagner et al** identifying a new form of pleural cancer amongst persons who were living close to a crocidolite mine in the North West Cape. This paper was the first paper to be written about mesothelioma.
and its publication proved to be quite controversial amongst epidemiologists and oncologists.

However in 1965 Newhouse & Thompson produced their seminal paper in which they published their findings relative to persons living in close proximity to an asbestos factory in the East End of London. Their study established that relatively low-level exposure to crocidolite could cause mesothelioma in persons who have had no occupational exposure to asbestos. This lead to the introduction of the Asbestos Regulations 1969. These Regulations not only superseded the 1931 Regulations but applied across the board. They were not restricted to the asbestos manufacturing industry. Now, anyone using asbestos in any process was required to adhere to stringent rules of industrial hygiene.

NON MALIGNANT CONDITIONS

1. Diffuse interstitial pulmonary fibrosis a.k.a. asbestosis
This condition is caused by very significant, usually prolonged exposure to asbestos dust and fibres. The lung tissue becomes clogged with asbestos fibres causing the tissue to fibrose (become scarred). The scarring restricts the lungs’ ability to expand and so respiratory capacity is reduced. Asbestosis is dose related and progressive. The condition is incurable and sometimes fatal. An asbestotic is at an increased risk of contracting lung cancer.

2. Diffuse pleural thickening
This condition is also caused by significant exposure to asbestos. The pleura or lining of the lung thickens, thus restricting respiratory function. Pleural thickening is also dose related and progressive.

3. Circumscribed pleural thickening a.k.a. pleural plaques.
This is a benign, asymptomatic condition, which is only detectable radiologically. The House of Lords ruled in the case of Rothwell and Others v Chemical and Insulating Company Limited that asymptomatic pleural plaques do not constitute an actionable injury. This area remains a hotbed for political debate. On 8 January 2010 the Court of Session in Scotland rejected the application made by a number of major insurance companies for judicial review of the Damages (Asbestos-related Conditions) (Scotland) Act 2009, which effectively overturns Rothwell as far as claims in Scotland are concerned and the insurers’ further appeal has since also been rejected [24.04.11]. On 25 February 2010, the MoJ confirmed that the decision in Rothwell is not to be overturned and pleural plaques will remain non-compensatable (until such time as further medical evidence/research becomes available). However, the Government accepted that individuals who had made an understandable claim for pleural plaques prior to the House of Lords ruling in October 2007
and whose claims were effectively on hold, would be eligible for a newly created fixed payment scheme of £5,000. A series of further announcements were made and can be read in greater detail via the MoJ website http://www.justice.gov.uk/news/newsrelease250210a.htm

**MALIGNANT CONDITIONS**

1. **Lung cancer**
   Whilst it is clear that many asbestotics go on to contract lung cancer, the most informed medical opinion now seems to be that where there has been significant exposure to asbestos, then lung cancer should be considered to be asbestos attributable. In a case where there is asbestosis and the victim is or has been a heavy smoker, the combined effects of asbestos exposure and smoking significantly increase the risk of lung cancer.

2. **Mesothelioma**
   This is a rare cancer that is almost always associated with previous asbestos exposure. This cancer usually occurs in the pleura (i.e. the lining of the lung) but can also occur in the peritoneum (the lining of the abdominal cavity) and more rarely in other parts of the body.

   Mesothelioma is a latent condition and rarely occurs less than 20 years after first exposure. The mean latency period of a mesothelioma is between 30 and 40 years after first exposure. The condition is incurable and life expectancy is usually between 6 months and 2 years from the manifestation of first physical symptoms.

   First symptoms usually consist of chest or shoulder pain accompanied by breathlessness because the pleural cavity has accumulated a significant quantity of a straw coloured fluid. This accumulation of pleural fluid known as an effusion can be aspirated or drained resulting in a short-term improvement.

   Theoretically, chrysotile cannot cause mesothelioma. However, much of the chrysotile used commercially over the years has been contaminated by tremolite, another type of amphibole fibre. Whilst mesothelioma is dose related, it can be caused by relatively low levels of exposure.

**QUANTUM**

The ninth edition of the Judicial Studies Board (JSB) Guidelines provides tabular information as to the appropriate bracket these claims fall into. In more recent years we have seen an increase in the higher levels of compensation paid out towards the upper end of the bracket. One such example is *Mayfield v Adelaide Engineering* [2009] in which the 68 year old Claimant was
awarded £80,000 having undergone surgery and chemotherapy and who was expected to suffer worsening pain leading up to his death.

<table>
<thead>
<tr>
<th>(C) Asbestos-related Disease</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Mesothelioma</td>
<td>£35,000 to £83,750</td>
</tr>
<tr>
<td>(b) Lung cancer</td>
<td>£51,500 to £66,000</td>
</tr>
<tr>
<td>(c) Asbestosis</td>
<td>£31,500 to £69,500</td>
</tr>
<tr>
<td>(d) Pleural thickening</td>
<td>£25,250 to £51,500</td>
</tr>
<tr>
<td>(e) Provisional awards for the least serious cases within (d) where the provisional award excludes any risk of the development of mesothelioma, lung or other cancer or asbestosis.</td>
<td>£4,350 to £7,250</td>
</tr>
</tbody>
</table>

USEFUL CASE LAW

In the following list of cases you will note we have referred to cases which deal with the issue of causation, apportionment of damages and knowledge.

Sabin v BRB [2010] – whether the deceased had asbestosis or usual interstitial fibrosis (UIP) unrelated to asbestos exposure found in favour of the claimant

Fleet v Fleet [2009] EWHC – measure of damages; mesothelioma; death from mesothelioma

Revenue and Customs v Silcock [2009] EWHC – mesothelioma; show cause; allegations of negligent exposure to asbestos at work; burden of proof at show cause hearings

Horsley v Cascade Insulation Services Ltd [2009] EWHC – asbestosis; contributory negligence; discounts; future loss; general damages; measure of damages; smoking

Sienkiewicz v Greif (UK) Ltd; Willmore v Knowsley Metropolitan Borough Council [2011] SC – duty of care, mesothelioma, proof, correct test for determining causation; occupational and environmental exposure

Abraham v G Ireson & Son (Properties) Ltd [2009] EWHC – extent of asbestos exposure; causation; foreseeability; knowledge

Durham v BAI (Run Off) Ltd [2010] CA – EL Policy Trigger Litigation: causation; employers’ liability insurance; interpreting determination of event triggering insurer’s liability
Brett v Reading University [2007] CA – breach of duty of care; evidence required of breach of duty; employers’ liability

Barker v Corus (UK) Ltd [2006] HL – causation; several liability; apportionment of liability according to share of responsibility for creating material risk of mesothelioma

Bolton MBC v Municipal Mutual Insurance Company Ltd and Another [2006] CA – policy wording, local authorities; accidental bodily injury or illness occurring during currency of policy

Bone v Ministry of Defence [2006] – quantum; award to widow for death of husband from lung cancer following exposure to asbestos dust at work (1956-1979)

Pinder v Cape Plc [2006] EWHC – duty of care to public to dispose of hazardous waste appropriately; foreseeability of harm

Badger v Ministry of Defence [2005] EWHC – contributory negligence; lung cancer caused by smoking and exposure to asbestos; reduction in damages attributable to smoking

Maguire v Harland & Wolff Plc and Another [2005] CA – foreseeability; wife exposed to asbestos from husband’s clothing; liability for mesothelioma caused by exposure

Rothwell v Chemical and Insulating Limited [2005] HL (formerly known as Grieves and Others v F T Everard and Sons and Others [2006]) – causes of action; asbestos; pleural plaques; risk of future disease and consequent anxiety; actionable damage

Fairchild v Glenhaven Funeral Services and Others [2002] HL – mesothelioma; employers’ liability; exposure to asbestos while working for more than one employer; causation

Ballantine v Newalls Insulation Co Limited [2000] CA – measure of damages; industrial diseases; benefits to be deducted in full from personal injury damages award

Holtby v Brigham and Cowan (Hull) Ltd [2000] CA – joint tortfeasors; asbestosis; exposure to asbestos by more than one employer; claimant to prove causation; apportionment of responsibility on time exposure basis

Margereson and Hancock v J W Roberts Limited [1996] CA – liability of factory owner; reasonable foreseeability of risk of harm established from state of contemporary knowledge
HAVS is the collective name for injuries to blood vessels, nerves and muscles caused by hand transmitted vibration. The typical symptoms are tingling, numbness, cold induced whiteness of the fingers and weakness of grip. HAVS can be defined as a disease with three separate components:

1. Circulatory disturbances also sometimes referred to as a vasospasm, finger blanching ("white finger") or vascular injury;
2. Sensory and motor disturbances also sometimes referred to as neurological injuries; and
3. Muscular skeletal symptoms (although these are sometimes strain related rather than vibration induced).

The diagnosis of HAVS is based on:

a) The history of the symptoms provided by the patient,

b) The history of relevant and significant vibration exposure, and

c) No evidence of an underlying cause to account for the symptoms.

Note that Raynaud's Phenomenon (which is a descriptive term for cold induced episodic whiteness affecting the fingers which is the principal vascular symptom of HAVS) may be caused by Raynaud's disease that is a constitutional condition. It can also be caused by connective tissue disease, arterial disease, toxins/drugs as well as having traumatic causes that include but are not limited to hand transmitted vibration.

The level of symptomology is classified according to the Stockholm Workshop Scale that assesses both neurological and vascular symptoms.
Table 1 – The Stockholm Workshop Scale for the classification of Cold-Induced Raynaud’s Phenomena in the Hand/Arm Vibration Syndrome

<table>
<thead>
<tr>
<th>STAGE</th>
<th>GRADE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td></td>
<td>No attacks.</td>
</tr>
<tr>
<td>1</td>
<td>Mild</td>
<td>Occasional attacks affecting only the tips of one or more fingers.</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
<td>Occasional attacks affecting distal and middle (rarely also proximal) phalanges of one or more fingers.</td>
</tr>
<tr>
<td>3</td>
<td>Severe</td>
<td>Frequent attacks affecting all phalanges of most fingers.</td>
</tr>
<tr>
<td>4</td>
<td>Very Severe</td>
<td>As in Stage 3, with trophic skin changes in the fingertips.</td>
</tr>
</tbody>
</table>

Note that the staging is made separately for each hand. In the evaluation of the subject the grade of the disorder is indicated by the stages of both hands and the number of affected fingers on each hand e.g. “2L(2)/1R(1).”

Table 2 – Sensorineural Stages of the Hand/Arm Vibration Syndrome

<table>
<thead>
<tr>
<th>STAGE</th>
<th>SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 SN</td>
<td>Exposed to vibration but not symptoms.</td>
</tr>
<tr>
<td>1 SN</td>
<td>Intermittent numbness with or without tingling.</td>
</tr>
<tr>
<td>2 SN</td>
<td>Intermittent or persistent numbness, reduced sensory perception.</td>
</tr>
<tr>
<td>3 SN</td>
<td>Intermittent or persistent numbness, reduced tactile discrimination and/manipulative dexterity.</td>
</tr>
</tbody>
</table>

FORESEEABILITY/DATE OF KNOWLEDGE

This is the date when any individual employer ought first to have recognised that work with vibratory equipment gave rise to a foreseeable risk of injury.

1975 is often referred to as being the date of knowledge however it is important to consider whether this is in fact appropriate for each individual case since the date of knowledge in HAVS cases varies between industries and many cases draw a distinction between the date when the employer should have been aware of the risk and the date by when it should have instituted precautions to reduce or eliminate that risk.
Examples of various dates of knowledge are:

**Rail Industry** – knowledge of risk ought to have arisen by 1973 and by the end of 1973 a system of issuing warnings to work as ought to have been in place and by the end of the following year periodical medical surveillance ought to have been introduced to all employers exposed to the use of hand held vibratory equipment [Allen v British Rail Engineering Ltd [1998].

**Coal Industry** – date of knowledge 1973 with an obligation to take precautionary steps by issuing warnings and undertaking medical surveillance by 1975, a duty to undertake job rotation by 1976 and a duty to adapt tools to minimise vibration at some unspecified point thereafter [Armstrong v British Coal Corporation 31 July [1998].

**Woodworking Industry** – date of knowledge 1991-1992 by which point employers should have been monitoring their employee’s health with respect to vibration injuries (Doherty v Rugby Joinery [2004] EWCA Civ147).

**BREACH OF DUTY**

**Vibration Levels**
For a claimant to establish breach of duty he must first show that the defendant exposed him to “excessive vibration” (i.e. vibration which exposed him to a foreseeable risk of injury) and secondly that he failed to take appropriate and necessary precautionary/preventative measures to reduce the risk of injury to an appropriate level.

The key standards that had been published addressing vibration levels are:

- **Draft Development Standard** published by the British Standards Institution in 1975 “DD43”. This stated vibration magnitudes of below 1.0 ms$^2$ were acceptable vibration magnitudes and magnitudes above 10ms$^2$ were unacceptable vibration magnitudes. This upper limit apply to exposure durations of 2½ hours and less per day. The lower boundary of 1 ms$^2$ applied to exposure durations of 400 ms$^2$ per day roughly up to 2½ hours it should be 150 minutes.

- **Health and Safety Executive Guidance Document HS(G)88.** This guidance was published in 1994 but is based upon the British Standard BS 6842 first published in 1987.

- **HS(G)88** prescribes an “action level” of 2.8 ms$^2$ A(8). The A(8) is a reference to an assumed or normal hours working day of 8 hours. Therefore 2.8 ms$^2$ A(8) is a measure of the average vibration level over the course of the 8-hour working day.
• The action level of $2.8 \text{ ms}^2 \text{ A}(8)$ is not a “safe” level of vibration which if it is complied with it is likely to avoid all injury. The action level is based upon statistical data demonstrating that a vibration dose equivalent to the action of $2.8 \text{ ms}^2 \text{ A}(8)$ would result in 10% of the exposed population developing symptoms of HAVS within 8 years.

• The Control of Vibration at Work Regulations 2005.
  http://www.opsi.gov.uk/si/si2005/20051093.htm

These came into force on 6 July 2005 and define exposure limits as $5 \text{ ms}^2 \text{ A}(8)$ and daily exposure actions value as $2.5 \text{ ms}^2 \text{ A}(8)$. Note that these measurements differ from those set out in the earlier Standard and Guidance due to the way in which the vibration level is now measured. As such the levels are not directly comparable.

The employer is required not to expose employees or others who may be affected by his work to vibration levels above the exposure limit. Where it is not reasonably practicable to eliminate the risk from exposure to vibration at source and the exposure action limit is likely to be reached or exceed the employer is required to reduce exposure levels to the lowest level reasonably practicable.

**Liability at Common Law**

The employers’ common law duty of care is not an absolute one but rather one of reasonable care in all the circumstances including any known susceptibility or vulnerability of the employee.

Not every exposure to vibration will suffice potentially to found liability. The exposure will need to be significant and such as to give rise to a foreseeable risk of injury. Tortious exposure will almost always be in excess of $A_8$ of 1.0 ms.

An employer will need to show that he has taken reasonable steps to control exposure to vibration if he is to avoid liability at common law. These include:

• Pre-employment screening for HAVS symptoms;

• Providing training and information to employees on the nature of the risk from vibration and what signs or symptoms they should look out for and report;

• Providing routine health surveillance;

• Controlling vibration levels by, where possible, engineering out processes which expose the employee to vibration and where not using tools designed for low vibration and properly maintaining these;
• Controlling how the employees work so as to limit exposure times (e.g. by way of breaks and job rotation); and

• Ensuring tools are handled in such a way so as to limit vibration being transmitted into the hand e.g. by training employees not to grasp tools tightly.

**Liability under Statute**
The Control of Vibration at Work Regulations 2005 set out obligations to:

• Elimination or control of exposure to vibration at the workplace;

• Assess of the risk to health created by vibration at the workplace;

• Provide health surveillance where there is a risk to health from vibration or the exposure action limit is likely to be reached or exceeded; and

• Provide information, instruction and training where there is a risk to health from vibration or the exposure action limit is likely to be reached or exceeded.

<table>
<thead>
<tr>
<th>Quantum</th>
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<tbody>
<tr>
<td>(i) Most Serious</td>
</tr>
<tr>
<td>(ii) Serious</td>
</tr>
<tr>
<td>(iii) Moderate</td>
</tr>
<tr>
<td>(iv) Minor</td>
</tr>
</tbody>
</table>

**Apportionment**
HAVS is a divisible, dose related condition the causation of which is capable of apportionment between those who have exposed the claimant to vibration in line with the principles of *Holtby v Brigham & Cowan (Hull) Ltd* [2000] i.e. the defendant should only be liable for those symptoms which his breach of duty has caused.

However the position is often complicated since, unlike the linear development of NIHL, there will usually be a latent period where exposure may have been negligent but there are no symptoms. Claimants are often described as having a “vibration reservoir” which is gradually “filled” by exposure to vibration but where they do not develop symptoms. When that “reservoir” is full the claimant goes on to develop symptoms.
There are often multiple employers and therefore defendants in these cases. Often the claimant has not developed symptoms until a later period of employment. This could give rise to the argument that in the absence of any injury the claimant has no right of action against a defendant who has exposed the claimant to vibration but where there is no temporal connection with the development of symptoms. There is no definitive case law on this issue yet.

In practise, defendants and insurers often take a pragmatic approach by apportioning quantum on a time exposed basis in the absence of specific evidence on levels of exposure during the exposure period even where symptoms may not have developed until a later period of employment.

However in the case of *Brookes v South Yorkshire Passenger Transport Executive* (1) and *Mainline Group Ltd* (2) [2005] the Court of Appeal addressed a facet of this issue when looking at whether an employee’s damages should be discounted for pre-date of knowledge (and therefore innocent) exposure. The claimant in that case had not developed symptoms until some years after the negligent exposure of one of the defendants had begun. The Court ruled that there should be no discount, as without the negligent exposure the claimant would have never developed symptoms.

Some defendants and insurers are seeking to rely upon this case to support the proposition that all of the liability for damages should fall to the defendant who was the claimant’s employer when symptoms arose. There is some force in this but there is no direct case law on the point.
Industrial Asthma

This is a disease process caused by the narrowing or inflammation of the lung passageways making breathing difficult. Symptoms include wheezing, tightness in the chest and difficulty exhaling air. Most people with asthma have sudden attacks or periods of more severe symptoms interspersed with periods of mild or no symptoms at all. When a person with asthma is exposed to one of the triggers personal to that individual, the airways begin to swell and narrow, making it difficult to exhale. Asthma cannot be cured but can be controlled by medication. The earlier the diagnosis, the better the prospects for greater control and avoidance of permanent lung inflammation and airway hypersensitivity.

WHAT IS OCCUPATIONAL ASTHMA?

A respiratory disease caused by exposure to a trigger in the workplace, usually inhaled.

Potential triggers are wide ranging and can be found in almost any workplace, including offices, factories, shops and hospitals. The list of industries and jobs where there may be exposure to triggers include plastics, rubber, chemicals, textiles, electronics, baking, food processing, cleaning, spray painting, soldering and welding. It must be remembered that constitutional asthma is very commonly triggered by exposure to a wide range of common irritants such as pollen, perfume, animal fur, house dust mites and tobacco smoke. Occupational asthma can thus occur as:

- an aggravation of a pre-existing condition caused by regular exposure to a new trigger causing hypersensitivity to the trigger and resultant attacks, or
- an irritant asthma caused by exposure to certain substances or conditions in the workplace irritating the airways with immediate symptoms.

Risk factors for occupational asthma include:

- Frequent exposure to the triggers;
- Allergies;
- Family history; and
- Smoking.
Occupational asthma requires a history of exposure to a sensitising agent. The latency period depends on the substance but can range from a matter of weeks to years. Medical causation is particularly difficult because of the prevalence of the condition to the general public. Issues to be considered include:

- Temporal relationship between exposure to a workplace trigger and the onset of symptoms;
- Variability of airflow obstruction measured by peak expiratory flow rates and spirometry;
- Patch testing as evidence of the sensitivity to the alleged trigger; and
- Personal/family history.

Data released by the HSE indicates that the incidence has fallen as a result of better control but occupational asthma remains a major concern.

**APPLICABLE LAW**

The most significant legislation is the *Control of Substances Hazardous to Health Regulations [1998] [COSHH]* as amended in 2002 and 2004 and specific guidance notes released by the HSE. COSHH requires an employer to control substances that are hazardous to health. The standard is a very high one. The aim of the regulatory regime is to provide a uniform code of the control of any substance that may be harmful to health.

The HSE also publish a list of “Occupational Exposure Limits” [OEL] for over 700 substances including short term [15 minutes] and long term [eight hours] exposure limits.

The definition of a substance hazardous to health is extremely wide. It includes substances with specified occupational exposure limits, biological agents, infectious bacteria and viruses, dust of any kind in substantial concentrations in air or any other substance which because of its chemical or toxicological properties creates a risk of injury to health.

Date of knowledge under common law is unlikely to provide grounds for defence in occupational asthma cases. In *Dugmore v Swansea NHS Trust* [2002] the Court of Appeal held that a date of knowledge defence was not relevant when applying Regulation 7 of COSSH. The case concerned the risk of wearing latex gloves causing sensitisation. The Court of Appeal held that the question of knowledge of the risk was irrelevant as:

*“Every employer shall ensure that exposure of his employees to substances hazardous to health is either prevented or where this is not reasonably practicable, be adequately controlled”.*
The *Dugmore* decision means that foreseeability is not an issue to be considered under COSHH.

“Regulation 7, sub-section 1 uses the language of strict liability and providing that an employer shall ensure that exposure is either prevented or adequately controlled. The primary duty is to prevent exposure altogether unless this is not reasonably practicable... nowhere is there any reference to the reasonable foreseeability of risk. Nor is the duty dependent upon what a risk assessment would have revealed”.

In these circumstances, where a causative connection is established between the substance and the asthmatic condition, it will be virtually impossible for any employer to escape liability.

**QUANTUM**

<table>
<thead>
<tr>
<th>(D) Asthma</th>
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<tbody>
<tr>
<td>(a) Severe and permanent disabling asthma</td>
<td>£28,250 to £43,000</td>
</tr>
<tr>
<td>(b) Chronic asthma</td>
<td>£17,250 to £28,250</td>
</tr>
<tr>
<td>(c) Bronchitis and wheezing</td>
<td>£12,600 to £17,250</td>
</tr>
<tr>
<td>(d) Relatively mild asthma-like symptoms</td>
<td>£7,000 to £12,600</td>
</tr>
<tr>
<td>(e) Mild asthma, bronchitis, colds and chest problems</td>
<td>Up to £3,400</td>
</tr>
</tbody>
</table>
Industrial Deafness

Traditionally those at particular risk of hearing damage were those in heavy industry such as shipbuilding, metal work, drilling and quarrying or where the use of noisy machinery is utilised such as engineering, woodworking, textiles and printing. Many heavy industries have disappeared or been scaled down and new plant and methods of working introduced with consequent reduction in noise levels. Nevertheless in spite of this and the increased awareness of the risk and controls over noise levels, noise induced deafness still occurs during the course of employment and insurers continue to receive a steady stream of deafness claims. The main difference from the thousands of claims of the seventies and eighties is that present day claims concern, in the greater part, lower levels of hearing loss.

HOW IS NOISE DEAFNESS CAUSED?

The cochlea, a spiral cavity in the inner ear, is filled with fluid and lined by cells with very fine hairs. For reasons that are not entirely clear, some of these cells are more sensitive than others to unwanted sound or noise and are damaged as a result of exposure. Over time hearing acuity can be compromised by exposure to such noise.

The first symptom of noise induced hearing loss is usually difficulty hearing a conversation against a noisy background such as in a busy restaurant. Sometimes this hearing loss is accompanied with intermittent high-pitched ringing in the ears called tinnitus. Typically, by the time the symptoms have become sufficient to prompt medical consultation and to be measured by audiometry, the damage will be severe and with every further session of noise exposure progressive.

Sound can measured scientifically in terms of intensity but also be related to particular frequency bands. Pitch or frequency is measured in hertz [Hz]. The higher the pitch will sound, the higher the frequency of it. Sound intensity is measured in decibels [dBA].

Noise induced hearing loss will begin to affect the hearing of certain frequencies. This can be noted with a dip on an audiogram usually at the 4 – 5 KHz’s range. With continued exposure to noise, the loss of sound perception progresses both in severity and into lower frequency ranges as well.

NIHL CLAIMS

During the 1970s and early 1980s thousands of deafness claims were litigated many on behalf of shipyard workers who had been exposed to decades of noise. In the lead case of Thompson v Smiths ShipRepairers [1984] it was decided that from 1963 employers exposing employees to
levels of noise in excess of 90 dB[A] were in breach of duty until such time as adequate ear protection was provided. Claimants were thus only able to recover for damage suffered between those dates. The many who had been exposed to noise prior to 1963 were thus only compensated for the proportion of their hearing loss attributable to the negligent exposure post 1963.

The decision was based on a detailed review of the medical and other information available to an employer about the need to protect employees against noise. On the key question of date of knowledge it was decided that until the early sixties, the need to protect employees was not generally known and so negligence was not established in respect of prior employment.

As a result of the Thompson decision as well as various agreements reached between Insurers and certain Unions for payment of scaled compensation, there was comparatively little industrial deafness litigation until the late nineties when attention was directed to the potential for damage at levels of noise less than the 90 dB[A] threshold.

In Baker v Quantum Clothing, Meridian Limited and Pretty Polly Limited [2011] the Supreme Court considered the alleged hearing loss suffered by textile workers, prior to the introduction of the Noise at Work Regulations 1989 on 1 January 1990, where the noise exposure had been between levels of 85 to 90dB[A].

At first instance it was held that at common law, the acceptable standard for the average employer to adhere to was 90dB[A], as per the 1972 Code of Practice published by the Department of Employment. The second European draft Directive, which ultimately became the Noise at Work Regulations 1989, was published in 1987. From that date, average employers should have been aware as to the risks of noise exposure between 85 and 90dB[A]. A period of two years was allowed for employers to implement protective measures, meaning that the average employer had no common law liability before 1 January 1990 (although this precise date was not made clear, but has since been clarified by the Supreme Court). A distinction was made for employers with greater than average knowledge who were held to have a date of knowledge from 1983. The same two year implementation period was applied and they were therefore in breach from 1985. Section 29(1) of the Factories Act 1961, which stated that “every place at which any person has at any time to work ... shall, so far as is reasonably practicable, be made and kept safe for any person working there”, did not add materially to the common law duty.

However, on appeal to the Court of Appeal, whilst the common law position was agreed, the implementation period for protective measures was restricted to six to nine months and, as such, liability arose from January 1988 and late 1983 respectively. The fundamental difference in the Court of Appeal judgment related to Lady Justice Smith’s interpretation of Section 29. It was held

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that the definition of “safe” was objective, unchanging and independent of any foresight of injury. The only qualification was for an employer to show that it was not reasonably practicable to reduce or avoid the noise exposure. As a consequence, by late 1976 an average sized employer in the knitting industry could and should have been able to make an informed assessment of the risk arising from exposure below 90dB[A].

The Supreme Court restored the first instance decision by a majority of 3:2. The Supreme Court held that common law employers with average knowledge were potentially liable from 1 January 1990 and those with greater than average knowledge from 1 January 1985.

Section 29 had not previously been considered at the highest level and it was held as follows:

- Whether a workplace was safe involved a judgment which was objectively assessed but by reference to the knowledge and standard of the time.
- Section 29 could apply to the activity and was not restricted just to the physical condition of the premises.
- There was no such thing as an unchanging concept of safety.
- The fact that a single person has suffered injury due to some feature of the workplace was not, on its own, proof that the workplace was unsafe. The successor legislation to the Factories Act, the Health and Safety at Work Act 1974, did not aim to create an environment that was entirely risk free and therefore it would be strange if Section 29 had a more stringent effect.
- The purpose of the Factories Act was to reinforce the common law obligation of an employer to take care of the safety of its workers. The Court of Appeal had found that statute must differ from the common law, otherwise it would be otiose. However, the Supreme Court held that there was no principle of law that a statutory duty cannot be interpreted as being co-terminous with a common law duty.

The Supreme Court’s decision is reassuring to both textile industry employers and insurers and provides welcome clarity to the basis on which deafness claims should be reserved. It has been confirmed that the average employer will have no common law liability prior to 1 January 1990 for noise levels below 90dB[A]. Consequently the decision prevents a floodgate of potential claims.
### QUANTUM

The following table is based upon the JSB Guidelines:

<table>
<thead>
<tr>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Total Deafness and Loss of Speech</td>
<td>£72,000 to £92,000</td>
</tr>
<tr>
<td>(b) Total Deafness</td>
<td>£59,500 to £72,000</td>
</tr>
<tr>
<td>(c) Total Loss of Hearing in One Ear</td>
<td>£20,500 to £30,000</td>
</tr>
<tr>
<td>(d) Partial Hearing Loss/Tinnitus</td>
<td></td>
</tr>
<tr>
<td>(i) Severe tinnitus/hearing loss</td>
<td>£19,500 to £30,000</td>
</tr>
<tr>
<td>(ii) Moderate tinnitus/hearing loss</td>
<td>£9,750 to £19,500</td>
</tr>
<tr>
<td>(iii) Mild tinnitus with some hearing loss</td>
<td>£8,250 to £9,570</td>
</tr>
<tr>
<td>(iv) Slight or occasional tinnitus with slight hearing loss</td>
<td>£4,850 to £8,250</td>
</tr>
</tbody>
</table>
Carbon Monoxide (CO) Poisoning

CO poisoning arises as a result of incomplete combustion of gas. Commonly claims arise against local authorities as the landlord of premises, which contain defective boilers and gas fires. Occupational claims may also arise against bus or haulage companies as a result of carbon monoxide fumes from faulty exhaust systems penetrating the driver’s cab of buses or lorries.

LIABILITY

Local authorities are subject to statutory regulations to provide gas services; to maintain all gas installations and to ensure the safety of the gas supply/installation. Local authorities are subject to onerous statutory duties to ensure that gas supplies and installations in their housing stock are safely installed and maintained. In reality, local authorities subcontract the provision of gas supplies and installations but they are unable to delegate their statutory duties to their sub-contractors. They remain liable to their tenants but invariably have rights of indemnity as against their contractors in respect of any liability they may incur by reason of the negligent acts and omissions of those contractors. Consequently, it is important to consider the specific contractual terms between the landlord and his sub contractor.

LIMITATION

The claimant’s time for bringing a claim begins to run from the date he/she first knew or ought reasonably to have ascertained that his/her symptoms were caused by the exposure to carbon monoxide fumes from the defective appliance.

Disclosure of the claimant’s medical records will be required before determining the claimant’s date of knowledge.

LEVELS AND SYMPTOMS OF CO POISONING

Levels of this gas are measured in parts per million ("ppm"). CO levels become toxic when levels greater than 50 ppm are reached provided the exposure is continuous over an eight-hour period. Higher than 50 ppm and a person is likely to suffer from symptoms of exposure.

Mild exposure over a few hours with a CO level between 70 ppm and 100 ppm will include flu-like symptoms such as headaches, sore eyes and a runny nose.

Medium exposure with a CO level between 150 ppm to 300 ppm will produce dizziness, drowsiness and vomiting.

Extreme exposure with a CO level of 400 ppm and higher will result in unconsciousness, brain damage and death.
INVESTIGATING CO POISONING CLAIMS

When investigating these types of claims consideration should be given to:

1. Documentation (such as tenancy agreements). These will confirm who owns or has responsibility for the property and the defective appliance. The gas service agreement will determine the contractual obligations between the local authority and any sub-contractor. The sub-contractor should be asked to produce evidence of all inspections; services repair records and so forth in order to establish any negligence on their part.

2. Expert engineering evidence will be required to determine the level and duration of CO exposure.

3. It is important to analyse what symptoms are alleged and whether there is corroborative evidence of the same within the claimant’s medical records. Analysis of the symptoms alleged should be cross-referenced with the levels of CO exposure to see if the alleged symptoms are consistent with the degree of known exposure. A report from a medical toxicologist should be sought and read in conjunction with the engineering evidence. It is important to retain a medical toxicologist rather than a toxicologist because you will wish to have the claimant medically examined and his or her medical records analysed.

4. The extent of the exposure. If the exposure is extreme and cognitive deficit is claimed, seek evidence relating to the claimant’s pre-morbid intellectual ability such as school/college/employment records. Neuropsychological testing will enable the defendant to establish whether the exposure has resulted in a reduction to the claimant’s pre-morbid cognitive ability and how this will impact upon the claimant’s future earnings capacity.

5. The claimant’s health prior to the alleged exposure including whether the claimant smoked or suffered with any respiratory problems such as asthma, chronic airways disease etc.

QUANTUM

The extent of injury that arises out of CO poisoning is so wide ranging that the damages awarded can be nominal to high value figures where exposure has resulted in brain injury and consequential psychiatric problems. The claims of (1)B(2)E(3)F(4)G(5)H(6)(7)K v Centre Islands Holiday Limited [2009] recently settled with damages ranging from £1,500 to £6,750 for differing levels of exposure. Whereas in the case of Peter George Etheridge (By His Litigation Friend The Official Solicitor) V Millar & Anor [2000] an out of court settlement was reached at £585,000.
Dermatitis is irritation of the skin or derma, which causes the skin to become inflamed. Dermatitis can occur either constitutional (endogenous dermatitis) or due to exposure to an external substance (exogenous dermatitis).

There are two main types of contact dermatitis:

1. **Irritant Contact Dermatitis**
   Irritants cause approximately 80% of cases of contact dermatitis and irritant reaction is caused due to the penetration of the epidermis by an external substance, which either irritates or sensitises the living cells below. The extent of cell damage depends on the strength of the substance involved and the period of which the skin is exposed.

   Chronic irritant contact dermatitis is caused by multiple exposures, often due to several irritants at low levels over time. This can take many months, or even years to appear. The usual course of events is that each exposure adds to the gradual disruption of the outer layer of skin, and each time inflammatory mediators are released. The epidermis gradually thickens and the fat layer in the skin is gradually damaged. The affected skin looses its ability to function as a barrier, and consequently further exposure to an irritant produces further damage, with the final result being dryness, scaling and thickening of the skin.

   Acute irritant contact dermatitis is usually caused by a single exposure to a substance which causes irritation within either minutes or hours of exposure. The usual course of events is that the irritant substance penetrates the skin damaging the membranes of the skin cells. The cell damage prompts the release of chemicals that trigger the immune system into action resulting in the appearance on a mild reaction consisting of transient redness, or any on a more severe reaction to painful burns with blistering.

   Each individual has their own susceptibility level. Both sexes are equally susceptible to irritant contact dermatitis.

2. **Allergic Contact Dermatitis**
   Allergic contact dermatitis accounts for the remaining 20% of contact dermatitis cases. Allergic contact dermatitis is often difficult to tell from irritant contact dermatitis, but its aetiology is different.

   The cause of allergic contact dermatitis is an immune reaction known as “delayed hypersensitivity” reaction. The characteristic feature of the immune reaction is a delay between the first exposure to an allergen and the subsequent reaction. Consequently allergic contact dermatitis occurs in two stages, firstly there is the “sensitisation”. This is where the substance
penetrates the skin before binding to a skin immune cell which then travels to the lymph nodes. The allergen is “shown” to another type of immune cell which proliferates and causes “memory” cells that remember the particular allergen.

Once sensitisation has occurred subsequent exposure to the allergen causes the immune cell to recognise the allergen, thus activating the immune cells causing them to multiple and the ongoing immune reaction results in an eczema-like inflammation of the skin at the site of contact. This phase usually occurs within 48 to 72 hours after exposure.

Once the allergy has established itself, even minor future exposures to the allergen can set it off. However avoidance of the allergen will result in resolution of the rash.

The communist allergens are nickel, fragrances, rubber/latex, some types of plant, formaldehyde, skin medications (including topical corticosteroids), some make-up and hairdressing chemicals.

As with irritant contact dermatitis, allergic contact dermatitis is more common in atopic individuals.

Approximately 75% of all contact dermatitis cases affect the hands. Contact dermatitis is more common in women, with approximately 20% of females experiencing contact dermatitis on their hands at some point during their lives. This may be because of the increased risk from cosmetics and other substances used in hairdressing, beauty products and cleaning.

**MEDICAL CAUSATION**

There are many causes of contact dermatitis involving a wide range of substances and consequently it is essential to clarify the medical causation. There are several types of eczema-like reactions that can produce a similar appearance to contact dermatitis including:

- **Atopic Eczema**;
- **Seborrhoeic Eczema**;
- **Discoid (or immular) Eczema**;
- **Pompholyx, dyshidrotic or vesicular Eczema**; and
- **Stasis or venous Eczema**.

Whilst the above are not necessarily caused by either irritant or allergic contact dermatitis, they can be exacerbated by such.
Before making any diagnosis, the medical expert involved should also consider other skin conditions such as:

- Drug related appearance, usually suggested by a history of rash that occurs after starting a new drug treatment.
- Fungal infection, this can usually be determined by taking skin scrapings and examining them under a microscope.

Patch testing can also be used to check for allergies. Suspected allergens are usually applied to the back under aluminium discs or patches which are then left in place for 48 hours, before being removed and the skin inspected.

**ALLEGED OCCUPATIONAL CONTACT DERMATITIS**

Where a person’s occupation is suspected of causing dermatitis then it is essential for the medical expert (usually a Consultant Dermatologist) to be provided with information in relation to the claimant’s full medical history and any previous allergic reaction to any substances. As it is essential for the expert to ascertain exactly what substances are used/touched during a claimant’s occupation, involving not only a description of the materials, but also the various processes involved. The expert should also establish the duration and frequency of the task carried out and ascertain what protective clothing, barrier creams or cleaning controls that are implemented.

When investigating claims generally insurers should arrange for a detailed description of the claimant’s medical and working history to be obtained, together with any changes in the claimant’s working environment, including such items as lighting, heating, ventilation, cleanliness and housing-keeping etc. The insured should also be asked to provide information in relation to the operating procedures and engineering controls and any changes which have been made to them and reasons for the same. A detailed description of the claimant’s working area together with any photographs or sketch plans should be obtained together with details of who else was using any of the materials involved. Full information in relation to the instruction and training provided to the claimant together with the type and level of supervision provided.

It is occasionally difficult to investigate contact dermatitis claims due to the passage of time involved and the potential sensitisation period if one is dealing with allergic contact dermatitis. However the investigation does need to be done methodically and carefully and it is important that no immediate conclusions should be jumped at purely on the basis of a Letter of Claim without any proper investigation and medical evidence to show causation.
RELEVANT STATUTORY REGULATIONS

The main regulations applicable to contact dermatitis are as follows:

- Control of substances hazardous to Health Regulations 2002.
- Duties in COSHH are owed by employers to their employees and others whom may be affected by the work carried out by the employer, save that Health Surveillance (Regulation 11) is only owed to employees. Under Regulation 6 there is a duty on an employer to make a suitable and sufficient risk assessment, and under Regulation 7, to ensure that the exposure of employees to substances are prevented, or where that is not reasonably practicable, adequately controlled.
- Where one might have expected the Personal Protective Equipment Regulations 1992 and the Provision and Use of Work Equipment Regulations 1998 to apply, COSHH takes precedence.

CAUSATION

The leading case in relation to causation is McGhee v National Coal Board [1973] HL. McGhee was a miner, employed on hot and dusty duties buy the Coal Board, who had failed to provide any washing facilities. As a result the claimant had to cycle home caked in sweat and grime. This increased the risk of his contracting dermatitis. The House of Lords held that simply increasing the risk by a more than negligible amount (i.e. by a material amount) due to a breach of duty was sufficient to result in legal liability.

However difficult questions regarding non-negligent exposure, constitutional vulnerability and the basis for diagnosis of the type of contact dermatitis will often arise and should be considered carefully before conceding liability.

More recently in Dugmore v Swansea NHS Trust [2002] CA – a case involving an NHS nurse who became sensitised to latex from wearing latex rubber gloves as part of her work equipment – the Court of Appeal has created an absolute duty on behalf of employers. This was a case decided under COSHH Regulation 7. The Court held there is a duty to ensure that exposure is prevented or controlled, and this was an absolute duty, not subject to the usual sanction of reasonable foreseeability. The Court held that the defence of reasonable practicability only qualified the duty of total prevention and not the duty to ensure the exposure is adequately controlled.
The 9th Edition of the JSB Guidelines for the Assessment of General Damages in Personal Injury Cases provides three separate brackets for general damages.

| (a) Dermatitis of both hands, with cracking and soreness, affecting employment and domestic capability, possibly with some psychological consequences, lasting for some years, perhaps indefinitely. | £9,000 to £12,600 |
| (b) Dermatitis of both hands, continuing for a significant period, but settling with treatment and/or use of gloves for specific tasks. | £5,700 to £7,500 |
| (c) Itching, irritation of and/or rashes on one or both hands, but resolving within a few months with treatment. | £1,125 to £2,600 |
Occupational stress is on the increase, however, unlike many other disease areas, companies and insurers have got good prospects of defending these claims, provided the proper investigations are carried out and the right medical experts instructed.

Most claims are advanced in negligence but statutory breaches are often pleaded including the Management of Health and Safety at Work Regulations 1999 (failure to carry out a risk assessment and/or a breach of the Working Time Regulations).

In relation to the latter, defendants can rely on Sayers v Cambridgeshire County Council [2006] EWHC to argue that non compliance with the working time regulations does not, in itself, establish a cause of action.

Comprehensive guidance on the law applicable to stress at work claims was provided by the Court of Appeal in the case of Hatton v Sutherland [2002] EWCA civ 76.

While aspects of this judgment have been questioned in subsequent cases, it remains a very helpful guide to the general approach courts will adopt with these types of cases.

**PROTECTION FROM HARASSMENT ACT 1997**

This act was introduced to deal with stalking, however, as a result of the House of Lords’ decision in Majrowski v Guys & St Thomas’ NHS Trust [2006] UKHL, which decided that employers could be vicariously liable for their employees harassment under the 1970 Act, these claims have increased.

To succeed in a Harassment Act claim, there must be a course of conduct. There is no requirement for a recognised psychiatric illness, and the limitation period in relation to these claims is a generous six years.

Following the House of Lords’ decision in Majrowski, there were considerable fears about the potential for the Protection from Harassment Act to give rise to a substantial increase in claims, however, this fear has lessened somewhat by the decision of the Court of Appeal in Conn v Sutherland City Council [2007] EWCA.

In this decision, the Court of Appeal held that the conduct complained of fell well below the threshold required to justify labelling the conduct as harassment for the purposes of the 1997 Act.

**RECENT RELEVANT CASE LAW**

Veakins v Kier Islington Ltd [2009] CA – proceedings for damages in which a Judge erred by focusing on whether a prosecuting authority would have pursued the allegations criminally rather than focusing on primary requirement that the conduct complained of was oppressive and unacceptable.

**COSTS**

Because of the high risks involved in pursuing Stress at Work/Harassment Act claims, claimants will look to recover 100% uplift on their costs when the work is carried out under a CFA.
Work related upper limb disorder is a generic term used to describe symptoms of pain, discomfort, weakness and swelling in the soft tissues of the fingers, hands, wrists arms and shoulders. It covers a number of well-defined clinical conditions of the upper limbs such as tenosynovitis, de quervain’s stenosing tenovaginitis, lateral epicondylitis, medial epicondylitis, carpal tunnel syndrome and trigger finger.

**ISSUES IN LITIGATION**

For a claimant to succeed in an action for damages he must prove the following:

1. Foreseeability – that his employers ought reasonably to have foreseen that there was a risk of injury for the work that he was required to perform;

2. Breach of duty – that there were steps that his employers could and should have reasonably taken which would have prevented the injury or minimised the risk of it occurring; and

3. Causation – that his employer’s breach of duty caused the injury complained of or made a material contribution to it.

**FORESEEABILITY**

The Department of Employment published the document “Beat Conditions, Tenosynovitis” in November 1972. This document was reissued by the HSE in September 1977 as guidance note MS10 which specifically drew a link between rapid repetitive twisting and gripping movements and the development of this condition.

By 1990 the HSE issued a further document entitled “Work Related Upper Limb Disorders – A Guide to Prevention” identifying factors which were solely or cumulatively responsible for upper limb disorders.

There is a significant amount of literature contained on the HSE website which would relevant to the issue of foreseeability.

http://www.hse.gov.uk/msd/uld/index.htm
BREACH OF DUTY

Statute
The following statutory duties are often encountered in WRULD claims:

The Health and Safety (Display Screen Equipment) Regulations 1992 which have great significance to computer and keyboard work.

 Regulation 3 Management of Health and Safety at Work Regulations 1999 (requiring employers to carry out a risk assessment).

 Regulation 4 of the Manual Handling Operations Regulations 1992 (requiring an employer to avoid, so far as is reasonably practicable, the need for his employees to undertake any manual handling operation which carries a risk of injury. Where this is not reasonably practicable the employer should carry out a suitable and sufficient risk assessment and reduce the risk to the lowest level reasonably practicable.

 Regulation 11 of the Workplace (Health, Safety and Welfare) Regulations 1992 requires that every workstation is arranged so that it is suitable for any person likely to work there and for any work that is likely to be done there.

Common Law
You will need to consider:

• Warnings given by the employer of the risk of injury involved in the work;
• Whether there has been a gradual introduction to repetitive work;
• Whether the job involves rotation of tasks;
• What rate of work the claimant performed and what rest breaks were allowed/taken;
• Ergonomics and posture; and
• What the employer’s response was once he became aware of the claimant’s symptoms.

CAUSATION
Care needs to be taken when considering whether an upper limb condition has been caused or exacerbated by work. It is vital that a hand surgeon diagnoses what condition the claimant is suffering from and understands the nature of the claimant’s job and the tasks that he or she is
undertaking. It is also crucial to consider whether there has been any material change to the claimant’s working practice around the time that the symptoms developed. If not then many experts will be unwilling to draw a causal link between the symptoms and the claimant’s work.

An improvement of symptoms away from work can point towards the condition having a cause at work but it is also possible that work activities may be exacerbating a constitutional condition.

### SPECIFIC CONDITIONS

**Tennis Elbow** – generally (but not always) a constitutional condition. Can be exacerbated by work.

**Golfer’s Elbow** – similar to Tennis Elbow in that it is generally a constitutional condition but one which can be caused by heavy work. Symptoms can be exacerbated by work however.

**Tenosynovitis** – commonly caused by repetitive forceful use of limbs at work or in sport.

**De Quervain’s** – generally a constitutional condition but one that can be exacerbated by work including keyboard use.

**Carpal Tunnel Syndrome** – rarely caused by work but frequently exacerbated by repetitive manual activities.

The carpal tunnel is a channel in the palm side of the wrist through which pass the tendons which enable the fingers and wrists to bend. The tunnel also contains the median nerve, which controls some of the muscles in the hand that in turn move the thumb.

Carpal tunnel syndrome is more common in women (11 per 100) than in men (3 per 100). The symptoms include numbness, tingling and burning in the hand and fingers. Occasionally, it causes pain in the forearm, which can radiate to the shoulder and even the neck. Usually, it is only the thumb, index and middle fingers that are affected. Symptoms may be mild, intermittent or continuous. In the severest and long lasting cases, the thumb muscles can waste away and the median nerve may be permanently damaged. The condition tends to develop in people over the age of 45. It is particularly prevalent in someone who is overweight and in people whose job requires them to use their hands a lot. The incidence of carpal tunnel syndrome is increased in people with thyroid problems, diabetes or who take oral contraceptives.

Where symptoms are severe, carpal tunnel release surgery may be effective. This entails severing the carpal ligament to make more space for the nerves and tendons, which occupy the carpal tunnel. Such surgery is usually ‘open’ surgery performed as a ‘day-case’ under local anaesthetic.
Surgery can also take the form of endoscopic surgery where a small incision is made in the palm side of the wrist and a machine called an endoscope is used to release the median nerve. Surgery is considered to have a 75% success rate.

**Diffuse Arm Pain** – no anatomical changes can be identified but the Courts have been prepared to find that such symptoms are work related. See cases of *Alexander & Others v Midland Bank plc* and *Gallagher v Bond Pearce*.

**QUANTUM**

<table>
<thead>
<tr>
<th>(a) Continuing bilateral disability with surgery and loss of employment.</th>
<th>£14,350 to £15,200</th>
</tr>
</thead>
<tbody>
<tr>
<td>(b) Continuing, but fluctuating and unilateral symptoms.</td>
<td>£9,750 to £10,750</td>
</tr>
<tr>
<td>(c) Symptoms resolving in the course of two years.</td>
<td>£5,700 to £6,300</td>
</tr>
<tr>
<td>(d) Complete recovery within a short period.</td>
<td>£1,450 to £2,300</td>
</tr>
</tbody>
</table>
SILICOSIS

Silicosis was once the commonest form of pneumoconiosis. The condition is caused by the inhalation of free silica. It is a latent condition generally associated with occupational exposure to silica that develops over time. Silica in crystalline form is toxic to the lining of the lungs and causes an inflammatory reaction. Over time this inflammation causes the lung tissue to become irreversibly thickened and fibrosed (scarred).

Free silica (SiO\textsubscript{2}) or crystalline silica occurs in three common forms in industry: quartz, tridymite and cristobalite. There is also a cryptocrystalline variety in which the “free silica” is bound to an amorphous silica (non-crystalline). This includes tripolite, flint and chert. Diatomite is the most common form of amorphous silica capable of producing lung disease. Some of these forms can be altered by heat to the more dangerous crystaline varieties such as tridymite and cristobalite.

Common sources of crystalline silica dust include sandstone, granite, slate, coal, pure silica and sand. People who work with these materials such as foundry workers, potters and sandblasters, are at most risk. Industrial exposure occurs in mining, quarrying, stone cutting, sand blasting, foundries and in the manufacture of glass and ceramics.

TYPES OF SILICOSIS

- **Chronic silicosis** – this is the most common form of the disease and the symptoms develop over many years (i.e. 10-20 years) from exposure to silica dust. The lung tissue becomes damaged by fibrosis and nodules of chronic inflammation and scarring provoked by the silica dust form in the lungs and chest lymph nodes.

- **Acute silicosis** – this rare condition results from short-term exposure to very large amounts of silica dust. The lungs become very inflamed and may fill with fluid, causing severe shortness of breath and low blood oxygen levels. The condition is often fatal.

- **Accelerated silicosis** – occurs after exposure to large amounts of silica over a shorter period of time (5-10 years). Inflammation, scarring, and symptoms progress faster in accelerated silicosis than in simple silicosis.

- **Progressive massive fibrosis** – can occur in either simple or accelerated silicosis, but is more common in accelerated form. Progressive massive fibrosis is caused by severe scarring and destroys normal lung structures.
LEPTOSPIROSIS/WEIL’S DISEASE

Leptospirosis is an infectious condition, which usually causes flu like symptoms 7-14 days after contact with the source of the infection. However, in extreme cases, symptoms can occur in as little as 3 or as long as 30 days from the initial contact source.

CAUSES

Leptospirosis is a zoonotic condition, that is to say it is transmitted from animals to humans. Animals such as rats, mice, pigs, cattle, horses, dogs, sheep and bats can become infected with the bacteria leptospires and contaminate a fresh water source such as a river, lake, pond, canal, or even a drain by urinating into it. If a human comes into contact with the contaminated fresh water source, infection can pass through the eyes, mouth, nose or unhealed cuts on the skin. The infection can also pass by close contact with a sick animal (particularly rats, mice, dogs or cattle) or by contact with the blood of an infected animal.

Leptospirosis is more commonly found in rural areas. However, it can also be found in urban environments, particularly where there are rats and poor sanitation/standing water.

SYMPTOMS

Leptospirosis has two phases. The acute phase produces symptoms such as fever, severe joint pain, headache and chills. The second phase is where the leptospires bacteria infect the body’s organs such as the brain, kidneys, liver, eyes and lungs.

Fortunately, only 5-10% of Leptospirosis victims go on to develop second stage symptoms because there will be an early diagnosis and treatment. The condition is responsive to antibiotics. Early diagnosis will be accelerated by a patient telling his GP of any close contact he has had with animals or a contaminated water source.

The more serious form of Leptospirosis is known as Weil’s disease. This causes severe illness. There is usually a longer incubation period after which symptoms may start suddenly. These may consist of fever, headache, muscle aches, conjunctivitis, vomiting and diarrhoea or constipation. Weil’s disease can cause jaundice.

In the most serious cases the condition can be fatal.
WORKERS AT RISK

Persons working in an occupation where they are likely to come into contact with animal urine or blood, such as veterinarians, farm workers and slaughterhouse workers are at risk.

Canal workers, tunnelers and other workers who are likely to come into contact with a freshwater source which is likely to be contaminated by rat’s urine are also at risk.

PRECAUTIONS

Some workers who are considered to be at high risk of leptospirosis are frequently issued with cards by their employers (which they carry on them at all times) which advise that they may have come into occupational contact with contaminated water. Such cards are intended to alert a treating doctor to the nature of the worker’s employment and to assist in the early diagnosis of the disease.

INVESTIGATING A CLAIM

When investigating an occupational leptospirosis claim, consider other possible sources of the claimant’s material exposure. Is the claimant a coarse fisherman? Does he undertake recreational fresh water diving? Does he live on or walk over farm land? Does he walk his dog over farmland and/or along the banks of rivers, canals, lakes etc?

Consider what steps the employer has taken to avoid the risk of infection. Has the employer carried out risk assessments?; carried out analytical tests of still water in a tunnel etc? Has the employer provided protective equipment to minimise the risk of injury? What training has been given?

EXPERT

Employ a microbiologist or an expert in infectious diseases rather than a toxicologist. Also consider instructing an occupational hygienist.
FIBROTIC HYPERSENSITIVITY PNEUMONITIS

Fibrotic Hypersensitivity Pneumonitis (otherwise known as Extrinsic Allergic Alveolitis) is an inflammation of the alveoli (the small sacs within the lungs where gas exchange occurs between blood and air). The condition is caused by the inhalation of antigens such as dusts, moulds and avian proteins.

The inhalation of an antigen can provoke an exaggerated immune response, which may be categorised as acute, sub acute and chronic.

Acute Hypersensitivity Pneumonitis may develop within hours of a significant level of exposure to the offending antigen. Symptoms include, cold like symptoms, cough, malaise, tightening of the chest, dyspnoea and headache. Such symptoms normally resolve fairly quickly after exposure to the antigen ceases.

The symptoms of Sub acute HP are similar to those of Acute HP but the condition takes longer to develop following exposure. The symptoms are however (a) are less severe and (b) longer lasting.

Patients who have Chronic HP usually develop an insidious cough, progressive fatigue and may suffer weight loss. The symptoms of Chronic HP may be irreversible. Progressive fibrotic changes may appear radiologically in the upper lobes and there may be reduced lung volume. Some patients have hypoxemia at rest.

HP is frequently referred to by different names based on the antigen responsible for triggering the condition. These include Bird Fancier’s Lung (caused by exposure to birds and their droppings); Farmer’s Lung (rotting hay); Berylliosis (beryllium); Chemical Worker’s Lung (plastics, resin, paints etc); and Miller’s Lung (dust from grain).
Q Fever

Q Fever is a zoonosis – a bacterial infection that can cause illness in people, particularly those working with livestock. The infectious agent is the coxiella burnetti bacteria. It was first identified in abattoir workers in Brisbane, Queensland in 1937. The “Q” stands for “query” as, when it was first identified, the causative nature was unknown.

Q Fever can spread to humans from animals, most commonly from livestock such as sheep, cattle, goats and other domestic animals. The primary mode of transmission is the inhalation of windblown contaminated aerosols, particularly from infected sheep placentas on open farmland. The infectious airborne particles can travel significant distances and are highly resistant to environmental conditions, often surviving in the open air for many months. Infection can also result from the inhalation of dust, contact with unpasturised contaminated milk, meat and wool.

Although Q Fever remains relatively rare with only about seventy cases in the UK per year, it is actually the world’s most infectious disease because infection can result from the inhalation of single bacteria. As a result Q Fever has been considered by various governments as a potential biological threat.

In animals, the infection is largely asymptomatic. In humans, the incubation period is usually two to three weeks with the condition falling into two categories:

1. Acute Q Fever.
2. Chronic Q Fever (that is where the condition persists for more than six months).

In Acute Q Fever, symptoms are typically flu like with fever, myalgia and a dry cough but in some individuals, they can have mild pneumonia with symptoms of hepatitis.

In cases of Chronic Q Fever which are more likely to occur with those with a pre-existing health condition, making them more susceptible to infection, the most common symptom is endocarditis (that is, the inflammation of the inner lining of the heart), which if left untreated can result in heart failure.

Cases can occur in people who are occupationally exposed. Those occupations, which are “at risk” include farmers, shearsers, tannery workers, abattoir workers and meat packers.

If correctly diagnosed at an early stage, antibiotics can shorten the course of the symptoms. It is thought that once infected, a person develops a life long immunity against re-infection.
THE LEGAL PERSPECTIVE

Claims for Q Fever are now making their way through the Court process.

Cases include meat processing workers who had contracted the condition after inhaling infected sheep's blood after the plastic wrapping in which the meat had been transported in, had been put into a compactor. When the compactor was activated, the blood was in effect sprayed over a number of workers who subsequently developed the condition.

Other litigated cases include veterinary students on a farming course who contracted the condition whilst visiting a farm to study birthing ewes. They developed influenza type symptoms which following serologic testing was confirmed to be Q-fever.

As with many other disease cases, the normal issues relating to limitation, breach of duty and causation apply.

The assessment of damages is also contentious as there are very few reported cases, but in cases of Chronic Q fever where the claimant has permanent debilitating symptoms including fevers, chills, headaches, myalgias, malaise and continual fatigue, general damages would be up to £40,000. In such cases they are invariably also substantial claims for special damages including care, loss of services and loss of earnings.
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