10.2.1 Occupational and environmental health 1638

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10.2 Occupational health CONTENTS 10.2.1 Occupational and environmental health 1638 Raymond Agius and Debasish Sen 10.2.2 Occupational safety 1652 Lawrence Waterman 10.2.3 Aviation medicine 1656 Michael Bagshaw 10.2.4 Diving medicine 1664 David M. Denison and Mark A. Glover 10.2.5 Noise 1671 David Koh and Tar-Ching Aw 10.2.6 Vibration 1673 Tar-Ching Aw 10.2.1 Occupational and environmental health Raymond Agius and Debasish Sen ESSENTIALS Occupational diseases are those for which work or, specific- ally, exposures in the workplace are necessary causes. The most prevalent occupational diseases in developed countries today are musculoskeletal and psychological disorders (usually stress- related conditions), but generally occupationally related malig- nancies (e.g. mesothelioma related to asbestos exposure) have the most serious outcomes. The proportion of all cancers attrib- utable to occupational exposures is about 4%, with occupation- ally related cancers almost exclusively concentrated in manual workers aged 20 or over in sectors such as mining, agriculture, and industry. When presented with a patient whose illness might possibly have been caused or aggravated by work or by other environmental fac- tors, the physician can usefully adopt an approach similar to that used for determining causation in epidemiological studies, with key issues being the temporality, reversibility, exposure-response, strength of association, and specificity of the illness with exposure to the factor in question, also biological plausibility, consistency with other reports, and evidence of similar exposures producing similar illness. The prevention of occupational disease depends upon recogni- tion of the condition as occupational, assessment of ‘exposure’ and hence determination of risk, education of stakeholders, control of the problem at source, audit of the risk management procedures, and perhaps health surveillance of those exposed using suitable techniques for the early detection of disease and a check on the ef- fectiveness of the control measures. Extant health and safety legis- lation is driven mainly by risk assessment, and those who generate the risks are responsible for undertaking an assessment, the detail of which must be commensurate with the complexity of the situation and the ultimate risk. Worker compensation, or the financial recompense for harm done to an individual by work or workplace, can be for an injury or a disease and might be difficult to secure even in the 21st century. It is important that clinicians are aware of statutory compensation schemes, and patients should be advised to claim for compensation if their

disease and work exposure seem related. If the disease and/or work exposure are not scheduled, there may still be a case worthy of pursuit under common law. Around the world, most people in work do not have access to an occupational health service or an occupational physician, and this is despite the International Labour Organization's recommenda- tions in 1985 for its members 'to develop progressively occupa- tional health services for all workers' and that 'The provision made should be adequate and appropriate to the specific needs of the undertaking'. Introduction 'Disease' results from a combination of genetic, behavioural, and 'environmental' factors, generally cumulating with the passage of time. However, proportionately, genetic factors explain very little of the burden of ill health. Therefore, it behoves us as physicians to consider, as a first alternative or as a 'default', environmental fac- tors, ranging from the ambient air we breathe to our occupation, but including diet, micro-organisms, and other exogenous factors as the prime determinants of disease.

10.2.1 Occupational and environmental health 1639 As physicians we should, first and foremost, aim to protect indi- viduals and society from such ill health through preventive meas- ures. We need to be aware of the sources and nature of the physical, ergonomic, chemical, microbial, and psychological hazards, how people are exposed to them and the risk or likelihood of this hap- pening, how they bring about adverse effects, and what structural, organizational, or behavioural interventions we should advocate to protect health and prevent ill health. When presented with a patient whose illness might possibly have been caused or aggravated by work or by other environ- mental factors, the physician might need to broaden their usual approach to history taking. Taking short cuts here can result in missing the diagnosis altogether and in losing a vital opportunity of making the patient better and of improving the fate of other coworkers. This additional information might be critical in at- tributing the illness to work. Bradford Hill postulated guidelines for determining causation in epidemiological studies, which lend themselves to be adapted for clinical purposes in an analogous manner (see Table 10.2.1.1). Moreover, when taking an occupa- tional history, it is also important to determine exposure, and a job title. For example, 'engineer', is not enough; a description of what the job entails and what agents or energies are involved are a bare minimum. The full job description might need to go back several years—pleural mesothelioma, for example, can occur half a cen- tury after the first exposure to asbestos. The workplace is where many people spend a significant propor- tion of their lives. Work is important for self-esteem and well-being (physical and psychological) as well as for the economic well-being of the individual and society. Work is good for us as long as it is good work and, therefore, does not impose unacceptable risks to our health. Thus, in dealing with the individual, physicians, whatever their specialty or interest, need to explore occupational and other envir- onmental causes of disease, to protect and better manage the pa- tient concerned, and indirectly protect others from the same fate. Furthermore, the physician has a responsibility as part of managing the patient's health to do their best to help the patient achieve and maintain gainful and fulfilling employment, whether this in- volves rehabilitation back to work, resolving presenteeism, or even addressing health-related issues standing in the way of achieving a job in the first place. Definitions and scope: Occupational disease What then is an occupational disease? Put simply, it is a disease for which work or, specifically, exposures in the workplace are neces- sary causes. The answer often has far-reaching consequences, both in terms of suitability of employment, compensation costs, the in- clusion of controls against exposure, as well as legal or policy consid- erations. For silicosis (caused by airborne quartz), or mesothelioma (caused by asbestos), the disease is almost always caused by work and the key aetiological agent, silica, found in the workplace. But what about bronchial

cancer in a joiner exposed to asbestos? Is it an occupational disease even though it is impossible to assess the precise contribution of occupational versus nonoccupational factors (such as tobacco smoking) in any one individual? Epidemiologic research on the excess of illness that is attributable to work can be used to directly represent the impact of work on health at a popu- lation level, as well as occupational exacerbations of symptoms and disability in pre-existing conditions, such as asthma brought on by irritants in the workplace. The most prevalent occupational diseases in developed coun- tries today are musculoskeletal and psychological disorders (usually stress-related conditions) but generally occupationally related ma- lignancies have the most serious outcomes. However, when it comes to understanding the mechanism by which occupational diseases occur, two basic concepts immediately arise: the fact that in the case of two workers doing an identical job one will get the disease but the other may not; and the fact that with some workplace exposures, for example asbestos, the latency of the disease, in this case meso- thelioma, may mean many years before the disease appears. These factors can make the diagnosis of occupational diseases difficult. On the other hand, failures of safety aspects of work are usually very obvious, especially if they lead to a workplace accident resulting in an injury or even death. Fig. 10.2.1.1 guides the clinician to an outline of the hazards to health arising from the workplace or other environments. Table 10.2.1.1 Analogies between determining causality in an epidemiologic context (the Bradford Hill criteria) in population studies, and when dealing with an individual patient in a clinical context Epidemiologic criteria for causality Analogous clinical questions Temporality When in relation to exposure do/did the symptoms start? Reversibility Do the symptoms improve when no longer exposed, e.g. after a weekend off, or by the end of a holiday? Exposure-response Are the symptoms especially worse when undertaking tasks or in areas with high exposures? Strength of association Do other workers/patients suffer from similar symptoms associated with the same exposures? Specificity What other exposures/causal factors could be responsible for the same symptoms? (Smoking perhaps?) Other data, or information processing: Consistency Are there other reports of the same symptoms associated with or caused by the same exposure? Analogy Even if there is no evidence to hand of identical exposures or circumstances resulting in the same symptoms, have similar agents/ chemicals of similar structure been implicated in the same symptoms of, for example ... dermatitis, ... or asthma. Biological plausibility Do the symptoms ‘add up’ in terms of what is known about the mechanisms of disease?

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1640 When faced with a case of ill health, to exclude work or the envir- onment, which might include the patient’s workplace, in the differen- tial diagnosis, it is important to determine, through the occupational history, the nature, duration, and intensity of any likely exposure, as illustrated in Fig. 10.2.1.1. The following are useful guides:

1. How was the task done? For example, was the adhesive applied with a brush while the patient leant over it?
2. Was the airborne concentration of a dust or fume such that it could be seen (although harmful particles are usually invisible to the naked eye), or the noise so loud that normal conversation was difficult?
3. Is local exhaust ventilation/extraction provided for the machine or area where work is taking place?
4. Are the risks to health connected with work such that the em- ployer has (or should have) supplied workers with certain items of personal protective equipment such as gloves or a

mask? Different categories of harmful agents require different and spe- cific forms of personal protection (e.g. rubber-based gloves are not appropriate when handling solvents). The relationship between hazard, risk, and the eventual possible onset of disease is a very important one, since it goes beyond ‘clinching a diagnosis’ and extends to the crucial aspects of preven- tion of ill health, as illustrated in Fig. 10.2.1.2. The size of the problem The International Labour Organization in 2013 estimated 2.34 million deaths each year from work-related accidents and diseases, the vast majority (2.02 million) dying from a range of work-related diseases. This equates to 5500 deaths each and every day caused by various types of work-related diseases. On top of this, the International Labour Organization (ILO) also estimates 160 million cases of nonfatal work-related diseases occurring annually worldwide. However, these figures might well be serious underestimates. In the United Kingdom, estimates for 2016/2017 indicate around 13 000 deaths each year are thought to be linked to past exposures at work; primarily to chemicals or dust. (Asbestos-related deaths, often with historic exposure, make up the greatest number dying from work-related cancers). The types and trends of reported, nonfatal diseases worldwide vary widely although some common features do exist: occupational lung diseases from exposure to workplace dusts, gases, vapours, and fumes; musculoskeletal disorders particularly low back pain; psy- chological disorders. In 2016/2017 an estimated 1.3 million people in the United Kingdom were suffering from an illness (longstanding as well as new) they believed was caused or made worse by their current work; 516 000 were new cases among those working in the previous 12 months, accounting for 25.7 million working days lost due to work-related ill health in 2016/2017. Around 79% of new work-related conditions were either musculoskeletal disorders or stress, depression, or anxiety. Fig. 10.2.1.3 shows how the incident cases distribute by major category from a general practice perspec- tive. Most of the incident cases are musculoskeletal, but mental ill health accounts for most of the sickness absence burden. The ILO estimates that, worldwide, work-related accidents and diseases result in an annual 4% loss in gross domestic product, or about US$2.8 trillion in direct and indirect costs. The cost of work- related diseases in the EU has been estimated to be at least €145 bil- lion per year, excluding compensation costs (for asbestos-related ERGONOMIC Heavy lifting, handling of loads Abnormal postures PSYCHOLOGICAL Work overload or underload High demand—poor control Other stressors BIOLOGICAL Work with infected patients or laboratory work: blood–borne viruses, tuberculosis, leptospirosis CHEMICAL Breathing in and/or handling chemicals or other hazardous substances; ingesting chemicals: mineral dusts, 'heavy metals', organic solvents, monomers, hardeners, etc. PHYSICAL High noise exposure, or vibration Hot work Radiation (ionizing and nonionizing) Fig. 10.2.1.1 Types of hazards, which may lead to occupational disease or other environment-related ill health.

 diseases, for example, of around €1.5 billion per year) and per- sonal litigation/compensation costs. In Great Britain, costs of new cases of workplace illness are estimated at £8.6 billion (2012/13 data), shared between cost to the individual worker (pain, grief, and suffering), the employer (lost productivity), and government (healthcare). History of occupational disease Some industries, such as mining, have always been considered haz- ardous. The ancient Egyptians recognized this by restricting such work to slaves and criminals. Hippocrates emphasized the rela- tionship between environment (air and water) and health, but the HAZARDS Potential to cause harm May arise from many sources PRIMARY

PREVENTION Reduction through intervention SECONDARY PREVENTION Reduction in risk to others by intervention TERTIARY PREVENTION Achieving improvement in capacity through occupational rehabilitation PRECLINICAL/BIOLOGICAL HEALTH EFFECTS CLINICAL ILLNESS Intensity of exposure leading to dose RISK or likelihood of effect/disease USUAL MEDICAL INTERVENTION PLUS REHABILITATION Fig. 10.2.1.2 From hazard, to risk, to disease, and the principles of prevention. Musculoskeletal Respiratory Hearing loss Mental ill-health Other Skin 1% 5% 4% 5% <1% 2% 3% <1% 2% 3% 55% 2% 9% 32% 51% % days sickness absence certified 48% 43% 35% % of cases reported % of cases sickness absence certified N = 6492 cases Fig. 10.2.1.3 Distribution of incident cases of work-related ill health reported by general practitioners to the THOR-GP surveillance scheme (University of Manchester) and associated certified sickness absence, 2006–2015, by diagnostic criteria. The THOR-GP Surveillance Scheme, COEH, The University of Manchester, Manchester.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1642 earliest occupational physicians served military forces, Galen; for example, being a physician to Roman gladiators. By the Middle Ages, the plight of the free miner had been recognized by Georgius Agricola (1494–1555) and Paracelsus (1493–1541). Agricola not only described the ‘galloping consumption’ of Carpathian silver miners but also proposed ways of reducing the dust in mines by im- proved ventilation. The first authoritative treatise on occupational disease was written by Ramazzini (1633–1764). His book De Morbis Artificium describes many occupational diseases ranging from mercurialism in mirror workers to repetitive strain injury in clerical workers. The Industrial Revolution in the United Kingdom brought occupational diseases to the attention of Parliament, largely through the work of phil- anthropists like Robert Owen, Robert Peel, and Lord Shaftesbury. Early legislation to control the worst vicissitudes of factory labour was emasculated by Parliament but the process had begun. The First Act of 1802 (which introduced the concept of limiting the hours of work and providing rest breaks in the working day) was followed by others leading to the 1833 Act which saw the start of His/Her Majesty’s Factory Inspectorate—the first enforcing, regulatory au- thority in this field anywhere in the world. By the early 20th century, the toxic effects of arsenic, mercury, phosphorus, and lead were common and understood in the West. Notification of these diseases became a requirement under health and safety law, and compensation for ill health was also granted. But the world of work has changed since then, particularly in the developed world. Here we now have a move away from manufacturing industries (heavy engineering, coal mining, and so on) to the provision of services, retail, and leisure. Consequently, the heavy, often dirty, industries such as mining and shipbuilding are, in some countries, like the United Kingdom, few and far between. With the growth in the service sector, whole new ways of working have also been developed (flexible working, short-term and ‘zero hours’ contracts), and with this we now have a new set of occupational dis- eases (more biopsychosocial problems). While the working condi- tions in the developed world have improved steadily since the early 20th century, working conditions for many in developing countries remain hazardous, demonstrating an important tenet of occupa- tional health practice: that is, while occupational disease can be preventable, the continued—often necessary—use of hazardous ma- terials and processes means that many such diseases are not elimin- ated, but need to be controlled. Prevention of occupational disease and other work-related ill health The prevention of occupational disease depends upon recognition of the condition as occupational, assessment of ‘exposure’ and hence de- termination of risk, education of stakeholders, control of the problem at source, audit of the risk management procedures, and perhaps health surveillance of those

exposed using suitable techniques for the early detection of disease and a check on the effectiveness of the con- trol measures. These procedures are outlined next. The legal duty on those who generate occupational and environmental health risks to manage them will vary from country to country. Occupational and environmental hazardous agents likely to harm human health might attack the body in various ways: through inhalation; ingestion; absorption through the skin; or a direct effect on organs of sense, for example, the eyes and ears. Occupational health risk management comprises recognition, evaluation, control, monitoring, and review. Fig. 10.2.1.4 illustrates the overall manage- ment of occupational health risks, and although a physician in a spe- cialty other than occupational medicine will contend directly with only a small part of this algorithm, awareness of it is important for the better management of the index patient and for the prevention of ill health in others. Extant health and safety legislation is driven mainly by risk as- sessment, and those who generate the risks are responsible for undertaking an assessment, the detail of which must be commen- surate with the complexity of the situation and the ultimate risk. Before embarking on any evaluation, especially where an occupa- tional cause is suspected, care must be exercised to determine what the known toxicological/health effects are, previous evidence from similar circumstances and/or environments, and any epidemio- logical evidence if available, as well as what the legislation requires. This is the ‘recognition’ aspect, and highlights the necessity not only to be well informed but also competent to interpret the available in- formation and act appropriately Table 10.2.1.2 shows how, after recognizing the possibility of a hazard being present in the workplace, evaluating the likelihood of harm arising, through the assessment of risk(s), is undertaken in a systematic manner. Similar approaches can be used in protecting the community at large such as in establishing air quality standards. Clinically, the issue of ‘what’ to assess might be self-evident; for example, blood lead in a case of suspected lead poisoning. However, from the standpoint of preventive risk assessment, a systematic approach must be pursued to determine the specific purpose and value of the monitoring. For example, is biological monitoring or biological-effect monitoring required? The former is the detection of a chemical or its metabolite in a biological sample as a measure of ex- posure (measurement of lead bound to red blood cells is a good ex- ample). The latter is measurement of a change in some biochemical or physiological variable to indicate the effect of the contaminant on the body (such as measurement of red cell or serum cholinesterase activity in relation to organophosphorus pesticides). However, even these measurements do not necessarily indicate harm but require an understanding of toxicology and epidemiology, as well as the pres- ence of relevant clinical findings. Often it is not possible to measure uptake or a biological effect short of ‘harm’, and one can only detect early manifestations of ill health, such as asthmatic symptoms from exposure to colophony fume in soldering. ‘Gold standard’ tests are rare in occupational health practice. Choice of an appropriate technique for making these measurements should be based, where possible, on indices of sensitivity and speci- ficity, as well as on the economics and acceptability of the technique. Remedial action: If the clinician suspects that work might be having an adverse impact or poses a significant risk to a patient’s health then they should advise the patient, and, with consent, the general practitioner and even the employer accordingly. If the clin- ician does not feel confident enough to do this, then advice can be sought from occupational physician colleagues in the relevant in- dustry, or in the health service, or from regulatory bodies, which also have a professional advisory role (such as the Health and Safety Executive in the United Kingdom). Advice given to the patient should include information on precautions that might be needed

10.2.1 Occupational and environmental health 1643 on returning to work, and whether any further investigations are required. Information on occupational rehabilitation and fitness to work is available from other specialists and agencies including voluntary/charitable bodies (such as the Royal National Institute of Blind People—RNIB, in the United Kingdom). European health and safety (H&S) legislation is clear: the em- ployer must adapt the workplace to be a safe environment for the employees. The employees should not have to adapt themselves to the stresses and strains of their work. Antidiscriminatory legis- lation, such as the Equality Act 2010 in the United Kingdom, now requires preplacement medical assessment of workers and not pre-employment 'medicals', only after a job offer is made. Fitness to work within existing H&S legislation remains vitally important to fit the job to the individual. Occupational cancer Occupational cancer is the leading cause of work-related deaths worldwide. Very often, it is difficult to establish the association be- tween occupation and cancer due to the long latency and multifac- torial causation of the disease. However, some of the associations Q1. WHAT HAZARDS DO WE HAVE? Q2. HOW DO WE ASSESS THE RISKS? Q3. HOW CAN WE MANAGE THE RISKS? Q4. HOW CAN WE CHECK TO SEE IF OUR INTERVENTIONS HAVE WORKED? Occupational health referrals Prior knowledge of Hazards Systematic surveys OCC hygiene measurements Employee/Union concerns Hierarchy of CONTROLS Risk-based HEALTH surveillance etc TRAINING & retraining on OH risks MONITOR all in Q1. (Active and Passive) AUDIT against standards & Occupational Health Plan Measurements/ Biomonitoring results Fig. 10.2.1.4 The overall strategy for managing Occupational Health Risks. Table 10.2.1.2 Specific considerations in assessing health risks Assessing the risks Where exposure can occur? The whole workplace? Where specific hazards exist? Who is likely to be affected? Those working full or longer shifts? Those working at increased metabolic/breathing rate? Those undertaking unusual/unscheduled tasks, e.g. maintenance work? Those already unwell? Are environmental measurements needed? . . . 'background' and/or 'personal'? How can the hazard enter the body? How likely is the exposure to cause a specific harmful
effect, and how severe? Route of entry and hence likely damage? Epidemiology and clinical toxicology, especially exposure-response relationships When and what to measure/ evaluate? Before, during, after exposure? Pre-, postshift; end of working week? (Cumulative effect; different metabolic
half-lives of hazards?)

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1644 are clear; for example, asbestos exposure and mesothelioma. Other associations are much more complex and less easily proven, such as the relationship between shift work and breast cancer. There is a considerable body of evidence on what causes oc- cupational cancer. The International Agency for Research on Cancer (IARC) based in Lyon, France, is part of the World Health Organization and has an established programme for the systematic evaluation of scientific evidence on the carcinogenicity of specific hazards and exposures. This it publishes in its extensive monograph series as an authoritative source of information on human carcino- gens. To date, IARC has classified nearly two hundred hazards as being established or probably carcinogenic to humans. Epidemiological evidence has been used to determine estimates of the proportion of all cancers attributable to occupational expos- ures. In round numbers the proportion is to the order of 4%, with a range of 2–8% for a developed country like the United States or the United Kingdom. Occupationally related cancers are almost ex- clusively concentrated in 20% or so of the population, comprising manual workers aged 20 or over, in sectors such as mining, agricul- ture, and industry. In this group, perhaps as many as one case of lung or bladder cancer in every five

might be attributable to workplace exposure. A wide range of industrial processes have been causally associ- ated with human cancer. Attribution of carcinogenesis by industrial process is often the first evidential step, and many legally recognized causes for compensation (‘prescribed’ industrial diseases) still rely on a definition by process. However, this approach has the short- coming of not necessarily being specific as to what chemical ex- posure needs to be controlled. Moreover, with the passage of time, once changes are implemented the risk associated with the original process could, and hopefully should, subside. The physician seeking to determine and thence prevent new circumstances with a risk of causing cancer must be aware that the causal agents, or analogues of them, might be responsible for causing cancer in other processes which are new and had not been recognized as risky. Therefore, in Table 10.2.1.3, the focus is on the chemical or physical hazard with processes listed by way of illustration while bearing in mind that these or similar hazardous agents might nowadays be manifest else- where. The agents named are not necessarily word for word as in the IARC documentation or as in legal schedules, partly for concise- ness and partly to consider more recent epidemiologic evidence. The IARC evaluation of carcinogenicity in humans sometimes applies to the group of chemicals as a whole, and not necessarily all individual chemicals within the group. Musculoskeletal disorders Statistical data from the European Union, including the United Kingdom, as well as the United States, indicate that musculoskeletal disorders have the highest reported incidence (Fig. 10.2.1.3) out of the major categories of occupational or work-related ill health—in the United Kingdom there were an estimated 507 000 workers suf- fering from work-related musculoskeletal disorders (new or long-standing) in 2016/2017. However, musculoskeletal symptoms are very common in the general population, a large proportion of which are in employment so making a work connection is often difficult to confirm. In some situations, a combination of occupational, psychological, personal, social, and home factors can be involved. This makes the diagnosis of the more common disorders associated Table 10.2.1.3 Some chemical and physical hazards causally associated with human cancer for which exposure has been mostly occupational. Examples of the index processes are in square parentheses while suspected target organs are in round parentheses Hazard Human target organ Arsenic and arsenic compounds Skin, lung (liver, haematopoietic system, gastrointestinal tract, kidney) Asbestos (most commonly in demolition nowadays) Lung, pleura, peritoneum, gastrointestinal tract, larynx Benzene (manufacture, petroleum refining) Haemopoietic (lymphatic) system Beryllium Lung Chromium VI (i.e. hexavalent compounds) Lung (gastrointestinal tract) Coal tars/pitches (coke production and coal gasification) Skin, lung (bladder, gastrointestinal tract, haemopoietic/lymphatic system) Diesel exhaust particulates or other particles containing polycyclic aromatic hydrocarbons (vehicle exhaust, some foundry processes) Lung, bladder Formaldehyde (disinfection, preservation, manufacture of resins and binders) Nasopharynx Inorganic acid mists (strong) containing sulphuric acid (isopropyl alcohol manufacture) Larynx Ionizing radiation (radiography, nuclear, and other industries) Haemopoietic/lymphatic system, bone, skin, and other organs depending on exposure and type of radiation Mineral oils, untreated or mildly treated Skin (respiratory tract, bladder, gastrointestinal tract) 2-Naphthylamine, and analogous aromatic amines (dye manufacture, rubber industry) Bladder (liver) Certain nickel compounds (nickel refining) Nasal sinuses, lung (larynx) Radon and its decay products (uranium mining, but also domestic/residential) Lung Silica (crystalline, e.g. quartz) Lung Vinyl chloride Liver, lung, brain, lymphatic and haematopoietic systems (gastrointestinal tract) Wood dusts, especially hardwoods (furniture manufacturing) Nasal sinuses

10.2.1 Occupational and environmental health 1645 with work, such as low back pain, nonspecific upper limb pain, and work-related stress, particularly difficult because of the reliance on the subjective reporting of symptoms and the evidence of both cul- tural and psychosocial influences, many of which are unrelated to work, on such symptoms being reported. The epidemic of so-called ‘repetitive strain injury’ in Australia during the 1980s highlighted the complex interaction of illness beliefs and behaviour, as well as employment in determining the workers’ symptomatic complaints. Musculoskeletal disorders can occur in all types of industry with high rates in post and courier services, agriculture, specialized con- struction work, and aspects of healthcare. The occupational risk factors implicated include: awkward posture; manual handling of loads; keyboard and other repetitive work; and psychosocial factors. Musculoskeletal disorders cases are very variable in their clinical pro- gress, though generally more predictable in this regard than mental disorders. Thus, some affected workers might be unable to return to work for a time, primarily because of pain, and/or need adjustments to their work. However, in general, cases are not very serious and treatment might be limited to pain management and adjustments in the workplace to reduce the risk of recurrence. People tend to recover and return to work, although conditions are often episodic. In the United Kingdom and elsewhere in the developed world, low back pain is possibly the most common musculoskeletal condi- tion experienced by people at work, making up 38% of musculoskel- etal disorders by affected area in the UK in 2016/2017. Poor lifting and manual-handling techniques and sitting for prolonged periods in the course of work activities (e.g. professional drivers) are con- tributory factors. Nurses, porters, and bricklayers are groups with a high prevalence of low back pain. The total cost of sickness absence, early retirement, and treatment for low back pain in many coun- tries is considerable. Historically this was exacerbated by the belief by patients and their medical attendants that rest was needed for recovery. However, there is now very good evidence that, with the exception of serious spinal diseases and nerve root problems, special investigations are unnecessary, and rapid mobilization and return to work should be advocated. Moreover, the rising incidence of back pain reported to be work related, over a period of decades, cannot be adequately explained by physical conditions at work or by work practices. Cultural and psychological factors probably have to be in- voked satisfactorily to account for the high frequency of back pain. In the upper limb, other musculoskeletal disorders such as carpal tunnel syndrome and De Quervain’s tenosynovitis might be related to work, as well as to recreational activities, in tasks which involve strenuous gripping. Work can aggravate as well as cause musculoskeletal disorders and the physician has a responsibility to rehabilitate people with musculoskeletal disorders back to work. Fortunately, the advent of new ‘biologics’ has made rehabilitation easier for patients with arth- ritic disease, such as rheumatoid arthritis. They need to be assessed for the effect that the condition might have on the performance of their work duties. The fluctuating nature of most forms of chronic arthritis makes precise predictions difficult. Physical disability can improve despite persistence of the disease. This is due to the bene- ficial effects of treatment, to the patient’s adaptation to the conse- quences of the disease, and to successful rehabilitation at work. Mental or psychological disorders related to work The reported incidence of work-related stress and mental disorders in the United Kingdom, the wider EU, and also the United States over recent years has increased dramatically (both in absolute rates as well as in comparison with other work-related ill health), and in certain industry sectors such as health and social care, education, and finance, it is now the single biggest cause of sickness absence from work. Several factors in the working environment have been Sources of stress Intrinsic to job (DEMAND, CONTROL) Org structure/climate (CHANGE management) Role in org (CONTROL) Personal PERSONALITY PSYCHOLOGICAL HEALTH PSYCHOLOGICAL Physical & Mental ill health

BEHAVIOURAL PHYSICAL HEALTH PHYSICAL Relationships at work (SUPPORT) Career development Individual characteristics Symptoms Disease Fig. 10.2.1.5 From stressors to disease. After Cartwright S and Cooper CL (1997). Managing Workplace Stress. Sage Publications Inc.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1646 identified as potential psychosocial hazards. Fig. 10.2.1.5 illustrates the broad categories of stressors and the key stages in the patient's journey through symptoms of lack of well-being to psychological or physical 'disease', such as depression or coronary ischaemia. There are various models to account for the 'stress' that people perceive when they are unable to cope. Karasek's model is based on 'demand' versus 'control' wherein jobs with high demand and low control are the most stressful. In Siegrist's model, the stress pro- voking imbalance adversely affecting employee well-being is the one between 'effort' and 'reward'. Prolonged exposure to one or more of the 'stressors' categorized in Table 10.2.1.4 can result in a range of symptoms of psychological dis- tress such as feelings of anxiety, irritability, or aggressive behaviour, lack of concentration, lack of confidence, and an inability to make decisions, sleep disturbance, and fatigue. There might also be asso- ciated physical symptoms, such as frequent headaches and nausea. Occupational stress is often identified as a result of the individual's inappropriate (maladaptive) coping strategies, such as frequent short-term absences, alcohol and other substance abuse, and poor time-keeping, or by uncharacteristically poor work performance. Effective management of occupational stress usually requires an integrated approach which includes attention to both the workplace and the individual and should include intervention at the three fol- lowing levels: • Primary intervention focuses on the identification of particular sources of stress in the working environment and the institution of measures such as policies and procedures to eliminate or reduce these. These should not be viewed solely as ill health prevention, but as generally good management practices. • Secondary intervention focuses on improving the coping skills of employees, including managers, by the use of specific forms of stress management training (e.g. relaxation/mindfulness, con- flict management, assertiveness, time management) and health promotional activities. These are particularly appropriate where workplace stressors are intrinsic to the particular occupation, and therefore not removable (e.g. the potential for aggressive confron- tation with members of the public). • Tertiary intervention is concerned with rehabilitation of psycho- logically distressed individuals. When anxiety or depression is manifest, pharmacological intervention and behaviour therapy may be needed. Counselling might help, although its evidence base is weak. The source of stress can often be multifactorial, and not, therefore, solely work related, but it has an impact upon work performance and might be exacerbated by the demands of work. Infections Patients, notably those working in healthcare, are at an increased risk of occupational infections. Blood-borne infections such as hepatitis B and HIV pose practical problems for the safety of staff during contact with infected patients, and the safety of patients during contact with infected staff, especially during so-called 'exposure-prone procedures'. Numerous cases of HIV infection have occurred in healthcare workers following contact with in- fected blood or body fluids from patients. These have involved needlestick injuries, mainly from contaminated hollow-bore nee- dles, or substantial blood contamination of damaged skin. Cases of HIV transmission from healthcare workers to patients are extremely rare, but hepatitis B is far more easily transmissible, explaining the higher incidence of outbreaks from doctor to patient and vice versa. Healthcare staff should be protected from hepatitis B by vaccination, but this does not cover other hepatitis viruses. Other occupational infections affecting staff in microbiological labora- tories and other healthcare workers include tuberculosis, salmon- ellosis, brucellosis, syphilis, and malaria. Working in tropical environments

also exposes workers to the risk of tropical diseases. Occupational and environmental infections involve a range of organisms from viruses, rickettsiae, bacteria, and fungi to larger organisms such as parasites and insects. Occupations involved, together with the diseases involved, are forestry and gamekeeping (Lyme disease), sewage work (leptospirosis and some viruses), and farming (bovine tuberculosis, Q fever, and brucel- losis). Although rare in the United Kingdom, anthrax still occurs Table 10.2.1.4 Occupational factors that can act as stressors (psychosocial hazards) Work overload Quantitative: too much to do in the given time Qualitative: demands beyond the skills or organizational capacity of the worker Work underload Quantitative: not enough work to do Qualitative: monotonous, boring tasks, or below the skills of the worker Timing and control Shift work Limitations on organizing one's own work Responsibility Role ambiguity, conflict Unclear responsibility and accountability Organizational culture, and relationships Lack of communication, participation Bullying and other harassment Financial and future prospects Inadequate reward or other recognition of 'worth' Job insecurity Poor training, personal development, or other prospects for advancement Hazards and comfort Physical, chemical, and biological hazards Other environmental discomfort or concern at work

 worldwide and can be responsible for fatalities typically following exposure to infected hides or bones, or indirectly during work such as construction on contaminated soil containing spores, where tan- ning or leather factories had once existed. Occupational dermatoses Since the skin offers such a large area to physical and chemical ex- posures in the workplace, it is not surprising that skin damage is a common occupational disease. An occupational dermatosis is a pathological condition of the skin for which occupational exposure can be shown to be a major contributory factor. The two most common skin conditions caused by/made worse by work are irritant and allergic contact dermatitis (eczema), the ir- ritant form accounting for 95% of all dermatoses. In Great Britain it is estimated that there are tens of thousands of new cases of oc- cupational dermatitis seen by general practitioners (GP) each year, while some 2000 of the more serious cases are reported by derma- tologists. The diagnosis and management of skin conditions in gen- eral is covered elsewhere (Section 23) but some key facts are worth reiterating: • a very detailed chronological occupational and nonoccupational exposure history is essential; • about one-third of cases are thought to be allergic in origin; • in many cases, although it will be easy to decide whether the dermatitis (eczema) is allergic or irritant, this distinction between allergic and irritant can sometimes be difficult to make; • dermatitis that was allergic in origin may be aggravated and sus- tained by exposure to irritants at work and at home; • atopic subjects are at increased risk of irritant dermatitis. The best form of prevention of dermatoses is to eliminate the con- tact and/or control exposure at source by applying the principles described earlier. In industrial situations, the hands and forearms are most at risk. The use of proper gloves (along with gauntlets or arm bands to prevent powders entering under the cuff), coupled with a high standard of hygiene, can minimize contact, and provide adequate protection. Where there is moving machinery, wearing gloves can pose a potential danger. Also when gloves are used, they might be taken off for tasks requiring manual dexterity, with the re- sult that contaminated hands are placed back inside the gloves. Many materials have been used to manufacture gloves, including cotton, leather, nylon, glass fibre, acrylonitrile, rubber, neoprene, butyl rubber, polyurethane, PVC, PVA, and tetrafluoroethylene. These confer specific protection for defined occupational exposures but the ingredients that go into their manufacture, for example ac- celerating agents such as thiurams, might also be a cause of derma- titis through skin contact. When cleaning skin, agents should be chosen that clean ad- equately in a short period of time

without having too strong a degreasing effect. Detergents or solvents if used for cleaning will remove fat from the skin, thus damaging its integrity and exposing it to further insults. Frequent handwashing, so-called wet work, can have the same effect. No so-called barrier cream actually provides a barrier to pene- tration of substances into the skin. In fact, in some situations they may actually enhance penetration and, occasionally, sensitization may occur to some of the constituents of the cream. There are many ‘after-work’ creams, essentially moisturizers, which have the benefit of increasing the hydration of the skin following cleaning at the end of the day. They are of particular benefit in occupations where exces- sive drying of the skin may occur. Their use should be encouraged following ‘wet work’ and where hot air dryers are used, as these tend to dry the skin unduly (towels are preferred for hand drying). Besides occupational dermatitis, and skin cancers or other skin damage caused by actinic (UV) radiation, other dermatoses might arise from work. The more important are briefly listed in Table 10.2.1.5. Cardiovascular system Cardiovascular disease is a major cause of mortality and morbidity in industrialized countries (see Section 16). The association between personal risk factors and cardiovascular disease is well known, but now the evidence is also growing of occupational and environmental influences. There is good evidence from the classical studies of bus drivers and conductors that sedentary workers have a higher risk of is- chaemic heart disease than those who are more active. There is some evidence linking job stress and heart disease. The Whitehall II cohort studies suggest that (lack of) control over one’s job is an important factor in determining subsequent risk of myocardial
Table 10.2.1.5 Benign occupational dermatoses, other than dermatitis Urticaria Type 1 hypersensitivity reaction and may be associated with rhinitis, asthma, and most importantly anaphylaxis. Itching, dermographism Glass and ceramic fibres, fibreglass, latex Psoriasis Identical to psoriasis from other causes. Can mimic dermatitis of the hands but without vesicles At sites of injury or friction in manual workers Infections Bacterial Viral Fungal Streptococcus to anthrax Poxvirus (Orf), human papilloma virus Tinea pedis, cattle ringworm, chronic candida (in wet work) Acne Typical acne features if slightly worse with multiple comedones, for example Oil, coal tar, chlorphenols, halogenated aromatics Vitiligo Areas of depigmentation often covering large areas of skin Para substituted phenols, and hydroquinones Scleroderma Skin changes may also include lesions Vinyl chloride, trichloroethene Pigmentation Altered pigmentation of skin Mercury and silver (argyria)

 infarction. Overall, the risk is reported to be increasing by the ex- posure to various chemical substances, noise, stress, and working long hours and shift work. Exposure to chemicals in the workplace or general air pollution can contribute to cardiovascular ill health in several ways: provoking inflammation; damage to vascular endothelium; inducing dysrhyth- mias; renal effects and effects secondary to anoxia. Among physical agents, vibration is known to cause peripheral vascular disease and acute high exposure to noise is known to raise blood pressure. Workers on rotating shifts have an increased risk of ischaemic heart disease. Genitourinary system The kidneys play a crucial role in detoxification and excretion and, therefore, bear the brunt of many exposures to toxic chem- icals. Some toxic substances reach the kidney unchanged, but most are metabolized to some extent or other. Some, such as cadmium, become sequestered in the renal cortex while others, such as the carcinogenic aromatic amines or their metabolites, can be present in the bladder long enough and at a high enough concentration to induce malignant change in the transitional cell epithelium. Sudden, severe exposures to some chemicals can cause acute nephropathy. Such compounds may damage the kidney directly due to their intrinsic nephrotoxicity

or may induce secondary damage due to prerenal effects, such as the haemolysis following arsine ex- posure, or oxalate crystals in the distal nephron after ethylene glycol poisoning. The metals most commonly implicated in renal disease are so- called heavy metals, such as mercury, cadmium, and lead (see Section 21). Mercury exposure resulting in acute tubular necrosis or the nephrotic syndrome is most unusual these days at least in the Western world. Similarly, modern industrial exposures to cad- mium rarely result in the proximal or distal tubular dysfunction or renal cortical damage that was more prevalent in the past. Lead nephropathy is also a rarity nowadays, but was not uncommon in the early part of this century. Lead is capable of causing damage to all parts of the nephron. Subtle tests of renal enzymes are needed to assess the effects of 'heavy metal' exposure on the kidney. Chlorinated aliphatic solvents such as carbon tetrachloride and chloroform can cause a hepato-renal syndrome. The renal damage is largely an effect on the proximal tubular epithelium which can lead to tubular necrosis and acute oliguric renal failure. The weight of evidence from case–control studies of workers exposed to solvents suggests an excess risk of chronic proliferative glomerulonephritis. The mechanism is unclear, but the demonstration of antiglomerular basement membrane anti- body suggests possible autoimmune damage to the glomerular basement membrane. Although the prostate possesses the curious ability to concen- trate (and excrete) heavy metals, inconsistent evidence exists of oc- cupationally related prostatic disease. Cancers of the urinary tract associated with occupational exposure to aromatic amines and polycyclic aromatic hydrocarbons were described earlier. Gastrointestinal tract Though ingestion is a means of entry for occupational and envir- onmental hazardous substances, and important when skin con- tamination is transferred to food or cigarettes, there are defence mechanisms that limit the damage to the gastrointestinal tract from such pollutants, and minimize their absorption. The mucous lining of the gut and diarrhoea and vomiting form part of these defence mechanisms. Acute gastroenteritis might follow the ingestion of chemicals such as soluble salts of heavy metals. The liver is frequently at risk from occupational and environ- mental exposures, as it is the target organ for detoxification and me- tabolism of absorbed compounds. A wide variety of infectious and chemical agents cause different types of hepatocellular injury, which can eventually lead to cirrhosis and liver failure, although there has been a decline in developed countries through improved working conditions, and a shrinking manufacturing industry. Haemopoietic system Lead poisoning is one of the oldest recognized occupational diseases and is still common, especially in the developing world. Exposures to inorganic lead are widespread, ranging from nonferrous smelting to the burning off of old paint during refurbishment/ renovation work in construction. In the domestic environment a particular hazard is the ingestion, usually by children, of lead com- pounds from old (now banned) paints. Lead causes anaemia mainly by inhibiting the enzymes involved in haem synthesis and also by haemolysis. The metal binds to erythrocytes and determination of blood lead levels is used in the monitoring of lead-exposed workers. A diagnosis of lead poisoning is supported by symptoms of malaise, colic, and constipation, signs of anaemia, and peripheral motor neuropathy (rare, usually only in severe cases), and microscopic evidence in the erythrocytes of basophilic stippling (from abnormal haemoglobin), elevated blood lead, low haemoglobin, raised free erythrocyte protoporphyrin, and raised urinary δ-aminolaevulinic acid. Indications of excessive lead absorption should lead to re- moval of the affected worker from further occupational exposure, with full investigation into, and control of, the circumstances of ex- posure to lead at work. Haemolysis and subsequent anaemia can follow occupational exposure to ionizing radiation in the industrial use of radioactive sources to test the integrity of welds, in the healthcare industry, and in nuclear power stations, as well as from chemical exposures (such as to arsine). Benzene is encountered in the petroleum industry, and is

used as a starter chemical for the production of other aromatic organic compounds. Its serious effects on the haemopoietic system include aplastic anaemia, leukaemia, and probably multiple myeloma. Methaemoglobinaemia can result from exposure to occupational and environmental agents such as nitrates, and nitro and amino derivatives of aromatic compounds. Specific examples are aniline, aminobenzene, nitrobenzene, and nitrates in drinking water (from soil leachate). Babies are particularly susceptible to this. It is treated by the intravenous administration of methylene blue.

10.2.1 Occupational and environmental health 1649 Reproductive system Children can be affected by parental exposure to physical and chemical hazards. More recently, there has been much concern that various organic environmental contaminants ranging from pesticides to phthalates and other plasticizers, and compounds with oestrogenic properties might cause reproductive harm to the community. In men there are indications that exposure to these ‘endocrine disrupters’ has resulted in falling sperm counts, and an increased incidence of hypospadias and testicular cancer. Risk of adverse effects on human reproduction can include re- duced fertility, spontaneous abortion, low birth weight, and child development disorders. In the United Kingdom, for example, new and expectant parents make up a significant percentage of the work- force. It is estimated that there are some 350 000 pregnant, working women in any one year in the United Kingdom. Consistent evidence supports that work exposures to some chemical, biological, and physical hazards, and certain working conditions and some occu- pations have the potential to adversely affect reproductive health. Several potential risk factors have been identified: exposure to chemicals (heavy metals, solvents, pesticides), radiation, local heat affecting sperm count and quality, having a heavy physical workload, and working irregular work schedules. Rubella can be a problem particularly for female healthcare workers and those working in microbiological laboratories, especially if adequate procedures are not in place for occupational health screening and immunization of this occupational group. Neurological disorders Damage to the central nervous system can be caused by a wide range of occupational or other environmental toxins, besides sub- stances of abuse such as ethanol and other solvents. There is very good evidence that exposure to lead (even at low level by occupa- tional standards), usually through drinking water but possibly also through inhalation, contributes to cognitive deficiency in children. Exposure to chemicals including lead, primarily encountered by workers in manufacturing, construction, and agricultural jobs, can cause transient and persistent effects on the central nervous system (Table 10.2.1.6). Transient central nervous system dysfunction is most commonly caused by exposure to volatile organic solvents, to organophosphate insecticides, or to carbon monoxide. Many low molecular weight fat-soluble organic solvents, especially if chlor- inated, are chemically very similar to the halogen-substituted an- aesthetic gases, and it is not surprising that they also have similar biological effects. In each instance, these substances, acting through different mechanisms, might cause central nervous system dys- function ranging from acute intoxication manifested by light-headedness and dizziness to loss of consciousness and even death. Persistent central nervous system sequelae can occur following one exposure episode if exposure levels are high and the duration of ex- posure is prolonged. Persistent central nervous system dysfunction, manifesting as neurobehavioural performance deficits, has been reported, par- ticularly in painters, following chronic exposure to moderate con- centrations of various agents encountered in the workplace, and occasionally in the environment. This syndrome, chronic toxic encephalopathy, consisting primarily of memory impairment, im- paired psychomotor function, and mood disorders, has been seen following chronic exposure to lead, styrene, carbon monoxide, and certain organic solvents. In

more severe cases the deficits per- sist, but do not progress, following cessation of exposure. If behavioural symptoms are present without evidence of abnormal neurobehavioural test performances (i.e. organic affective syn- drome) reversal of these manifestations usually occurs following cessation of exposure. Other rare central nervous system effects in- clude a Parkinsonian syndrome which might be a consequence of toxic exposures to manganese. Exposure to a range of toxic agents can cause damage to the per- ipheral nervous system causing either motor or sensorimotor poly- neuropathy (Table 10.2.1.7). Less commonly nowadays, exposure to lead at high levels for long periods can cause upper-extremity motor neuropathy, consisting of wrist extension weakness or wrist drop. Other specific substances (e.g. acrylamide, n-hexacarbon aliphatic solvents, and certain organophosphorus compounds) can Table 10.2.1.6 Some central nervous system health effects caused by workplace or other environmental toxins Disorders Manifestations Causal agent Acute intoxication Light-headedness Carbon monoxide Loss of consciousness Organic solvents Death (rare) Organophosphates Organic affective syndrome Fatigue Organic solvents Irritability Lead Depression Mercury Chronic toxic encephalopathy Impaired neurobehavioural function (symptoms as described) Organic solvents Lead (usually organometallic) Psychosis Marked emotional instability Carbon disulphide Manganese Mercury Toluene and related solvents Parkinsonian syndrome Tremor, rigidity, akinesia Manganese Carbon disulphide Carbon monoxide Visual disturbance Impaired acuity or peripheral field defect n-Hexane Methanol Organic mercury Colour vision loss Organic solvents Cerebellar or other damage Ataxic gait Acrylamide Organochlorine insecticides Methyl mercury Seizures Lead (usually organic) Organic mercury Organochlorine insecticides Organotin compounds

 act as axonal toxins causing a mixed sensorimotor polyneuropathy manifesting the asymmetrical, distal sensory loss. Occupational disease caused by vibration or noise Certain jobs involving the use of vibrating hand tools or pneumatic drills may be responsible for the occurrence of peripheral neuropathy, and loss of motor function if progressive, as part of ‘hand– arm vibration syndrome’ (HAVS). These disorders arise from a combination of physical trauma to the nerve itself as well as damage to blood vessels. Some workers with HAVS suffer adverse effects on hand function and are no longer fit for work. Noise-induced hearing loss is discussed in Chapter 10.3.4. Respiratory disorders The respiratory tract is, in many occupations, the most important route of exposure to hazardous substances. In many instances of oc- cupational diseases, it is the target organ. These conditions are dealt with in Section 18. Medically unexplained symptoms A growing number of occupational health complaints are charac- terized by the lack of a firm diagnosis or a clear occupational causal pathway. These include, in particular, complaints that consist of a range of nonspecific symptoms, typically headache, fatigue, nausea, depressed mood, cognitive confusion, and sometimes eye and nasal irritations. They are reported in diverse situations such as in air-conditioned offices with poor control (accounting for many cases of ‘sick building syndrome’), proximity to low-frequency electromagnetic fields, and perceived exposure to very low (often undetectable) levels of chemicals. Moreover, the prevalence of musculoskeletal complaints in many workplaces has been shown to be related to the presence of psychosocial hazards. Important elements are health beliefs and atti- tudes of the individual as well as the social and cultural environment. These influence their response to real or perceived exposure to haz- ards by determining their selection of which information to attend to and their subsequent interpretation of that information. Specific examples of syndromes with these features which occur in both an occupational and wider community setting may be ‘multiple chem- ical sensitivity’ and ‘chronic

fatigue syndrome’. Current approaches to effective management of these and other similar conditions favour a ‘biopsychosocial’ approach, which rejects the artificial distinction be- tween a physically and a psychologically based complaint and treats both physical and psychological symptoms simultaneously. Compensation for occupational diseases Worker compensation, or the financial recompense for harm done to an individual by work or workplace, can be for an injury or a dis- ease and might be difficult to secure even in the 21st century. In the so-called ‘first-world’ nations (i.e. the industrialized society), a worker can receive compensation prescribed by the state, or through litigation in the civil courts. By the end of the 19th century, many nations in Europe and the United States had passed workman’s compensation laws of one sort or another. Such schemes were usually restricted to specified diseases or occupations. The United Kingdom, for example, has the Industrial Injuries Benefit Scheme by which, depending on the disease in ques- tion (from the list of Prescribed Disease) and the diagnosed degree of disability, compensation is paid by the state. There are over 70 Prescribed Diseases known to be a risk to health from certain jobs, and in 2011, figures from the Department of Work and pensions, United Kingdom, confirm 5920 cases of occupational diseases as being compensated, with pneumoconiosis, diffuse mesothelioma, and osteoarthritis as the three most common diseases. The principles underlying such statutory compensation schemes are that they should be ‘no fault’, that the disease should be, with reasonable certainty, caused by work, and that the benefit claimed should offset job loss, wage-earning deficit, and disability, or pro- vide death benefit to the next of kin. Advice on proposed additions to the list of compensable diseases is made in the member states by government-appointed advisory groups. In the United Kingdom, this group is the Industrial Injuries Advisory Council, which reports to the Secretary of State for the Department of Work and Pensions. It is important that clinicians are aware of such schemes. If the disease and work exposure seem related, and are scheduled, patients should be advised to claim for compensation. If the disease and/or work exposure are not scheduled, there may still be a case worthy of pursuit under common law. Occupational health services The notion that employers should provide healthcare for workers is hardly new and the role of the physician in caring for, as well as advising on, diseases caused by work has been mentioned and goes back into the history of occupational medicine. Table 10.2.1.7 Peripheral nervous system syndromes caused by workplace or other environmental toxins Disorders Manifestation Causal agent Motor neuropathy Wrist weakness, foot drop Lead Mixed sensorimotor neuropathy Symmetrical distal sensory loss, mild motor dysfunction Acrylamide Arsenic Carbon disulphide Carbon monoxide DDT n-Hexane Methyl n-butyl ketone Mercury Organophosphorus compounds (various agents including triorthocresyl phosphate) Thallium

10.2.1 Occupational and environmental health 1651 The first recognizable occupational health service in England began in the mid-18th century when the London (Quaker) Lead Company recognized the adverse effect of lead mining on workers and provided health and welfare services in north-west England. Since then, occupational health provision has expanded along dif- ferent lines in different countries. Around the world, most people in work do not have access to an occupational health service nor an occupational physician, and is this despite the International Labour Organization’s recommenda- tions, in 1985, under ILO Convention 161, for its members ‘to de- velop progressively occupational health services for all workers’ and that ‘The provision made should be adequate and appropriate to the specific needs of the undertaking’. In the United Kingdom, approximately one in 14 consultations with a GP by those of working age are for conditions due to work or that affects ability to work. GPs and secondary care specialists in hospitals where these patients are sometimes referred therefore need to think of work as a

possible cause of ill health and ill health as a barrier to work. Both primary and secondary care physicians need to communicate freely with occupational physicians and occu- pational health services if these exist in their patients' places of work in order to keep their patients happy, healthy and in work for as long as possible or to return them to work after an illness as quickly as the medical evidence justifies. In practice, delivery of good occupational and environmental health practice is a multidisciplinary approach. It might include physicians, nurses, or hygienists who monitor and control exposure to chemical, physical, and biological agents in the workplace; tox- icologists; ergonomists; and psychologists able to assess psycho- social aspects of work. These specialists will promote occupational health, but for long-term success that is sustained, it is crucial that both managers and the workforce consider it an integral part of their working practices and philosophy. Unfortunately, occupational health services are not available universally and interpretation of the requirements varies greatly between countries and employment sectors. Initially, most services arose from a mixture of philanthropy and self-interest; the theory being that the healthy worker was likely to be more productive, a concept that holds sway today. Present-day services range from total healthcare including primary care and hospital medicine (as in some large multinationals operating in developing countries), to outsourced independent occupational health services. Services in the United States of America and much of Europe might in- clude general health promotion and education, but much inequity in healthcare exists even between enterprises within the same country. Recent increase in the provision of occupational health services has followed the enactment of effective health and safety legislation (e.g. the Health and Safety at Work etc. Act (1974) in the United Kingdom). Some countries, such as the Nordic countries, the Netherlands, and Australia, require the provision of occupational health services by law. Statutory provision of such services in the United Kingdom and the United States is limited to particular industrial sectors and specific occupational exposures, such as ionizing radiation, heavy metals, asbestos, and carcinogens. In general, primary legislation such as that produced in the United Kingdom then 'enable' a variety of government departments to create further secondary legislation in the form of 'regulations', requiring action from employers in par- ticular occupational and environmental circumstances. In the developing world occupational and environmental health often remains an almost unknown concept. National governments in the developed world have approached the question of preventing ill health at work in different ways, with the European Union's dir- ectives increasingly dominating the scene in Europe. The EU dir- ectives increasingly drive the occupational health and safety agenda in member states, requiring them to adopt these principles and then modify or create national legislation in response. The EU phil- osophy has been to encourage the delivery of occupational health to all by 'competent' persons, although the mode of delivery, even the definition of competent, has been left to member states to inter- pret. Most important, the level of service provided should be based on a thorough risk assessment of the work processes in that organ- ization and a clear and logical procedure of risk management. To deliver even such a basic service requires multidisciplinary teams including trained physicians, hygienists, and nurses. Few com- panies have such services, and many are too small even to contem- plate such provision. Future development of occupational health services might de- pend primarily on the economic climate, but also on perceptions of what constitutes occupationally mediated disease, as well as political will. Perversely, an exponential rise in legal action, insurance costs, and compensation may also play a significant part in persuading employers, and even nations, that competent occupational health services are an absolute requirement of profitable organizations. If this is to encompass small and medium-sized enterprises, provi- sion must come either from larger employers (such as the National Health Service) or from private providers. Conclusion Clinicians can make important contributions in two ways to 'occu- pational health' and hence to the health

and economic well-being of their patients and others. In the first instance they should have a high index of suspicion for occupational (and environmental) causes of disease in their patients. These can manifest to physicians in any specialty. Having reached a clinical diagnosis, especially in the case of work-related ill health, they need to liaise with an occupa- tional physician or other occupational health professional, the rele- vant enforcing authority, or the employer directly so as to remove their patient from the harmful ‘exposure’ and to control exposure to prevent similar occurrence in the patient and in other workers. Secondly, whether or not the ill health is work related, the clinician has an important role in giving advice about return to work and achieving occupational rehabilitation. However, the delivery of ‘occupational health’ and of ‘environ- mental health’ is achieved through more than just a clinical ap- proach. Occupational health is concerned with managing the health of working people. While occupational physicians deal with the ef- fects of work on health, and the influence of health on work, other professional groups, including nurses, hygienists, toxicologists, psychologists, and safety engineers, also have important roles to play in keeping people healthy and at work. In common with other prob- lems in public health, the solutions mainly lie in a population based

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