

Hear disease is common both in the population at large but also in the population of working age. It is estimated that heart disease, including stroke and high blood pressure, is responsible for more costs than any other disease or injury. The cost in occupational terms of cardiovascular disease (CVD) is, however, harder to quantify but is likely to be similarly high. Heart disease can claim the ultimate cost as the most common cause of death.

CVD is the main cause of death in the UK, accounting for over 245 000 deaths per year. The main forms of CVD are coronary heart disease (CHD) and stroke. Death before retirement age is commonly attributed to CVD, accounting for 36% of premature deaths in men and 27% of premature deaths in women. CHD is responsible for 120 000 deaths a year in the UK and is responsible for 23% of deaths before the age of 65 years in men and 13% in women. Significant advances in acute medical care have, however, brought reductions in mortality from CHD over the last 10 years or more. Morbidity rates have not seen that same fall; in fact, rates have risen, especially in older individuals over the last 20 years. Two million people in the UK suffer from angina, 680 000 people have heart failure, and 270 000 will suffer a heart attack each year—highlighting the significance of CVD as a cause of morbidity.

A recent study of self reported work related illness in 2001–2¹ shows that record numbers of workers feel that they have an illness that was caused by or made worse by their work, equating to 2.3 million people and 33 million working days lost. These figures show a prevalence estimate for CVD caused or made worse by work of 80 000 during the study year, with each person reporting work related illness taking an average of 23 days off through sickness in the year. This equates to 1.84 million days lost to work related CVD, with associated costs to industry of approximately £120 million. In essence, the issue of heart disease and work is a very significant one in terms of individuals affected, industry, health service resource, and national resource.

EFFECT OF WORK ON HEALTH: AN OCCUPATIONAL HISTORY

Many doctors maintain a time honoured Hippocratic approach to methodical history taking when involved in a consultation with a patient. This should be extended to include questions about occupation as first proposed by Ramazzini almost 300 years ago (fig 1). Not only should a physician ask “what is your occupation” but also gain full explanation of what the job entails. Modern job titles often provide little or no clues as to what an individual does every day in the course of his work. When assessing the possible health risks associated with a given job it is useful to think in terms of physical, chemical, biological, ergonomic, and psychosocial hazards.

It is important not only to consider the presence of a hazard in the workplace such as noise exposure (which has been associated with raised blood pressure) or carbon monoxide (which is associated with increased rates of coronary artery disease), but to then quantify any attendant *risks* that may be linked to that hazard—is there exposure to the hazard in such a way that the disease may be linked causally to the exposure?

Useful approaches in taking an occupational history include:

- ▶ what do you do at work?
- ▶ do you use any equipment/machinery regularly?
- ▶ do you come into contact with chemicals/gases/fumes/dusts?
- ▶ what is the work environment like? including any personal protective equipment you may need to wear—for example, gloves, masks, breathing apparatus, etc (figs 2 and 3)
- ▶ what hours do you work? are they regular hours? do you work shifts?
- ▶ do you enjoy your work? if not, why not?
- ▶ are there any aspects of your work that you think are impacting on your health? what are they?
- ▶ do you have regular health checks in relation to your work—for example, COSHH (controlled substances hazardous to health)/lead medicals, etc.

Equipped with this occupational component to the clinical history it is then much easier to methodically consider the impact of work on health and health on work—the basis of occupational health practice. It has long been accepted for certain clinical specialties to consider

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Figure 1 Bernardino Ramazzini (1633–1714).

the workplace when assessing a patient. This is true of chest medicine and also dermatology. Cardiologists have not considered developing the discipline of occupational cardiology as yet but this may be something that needs consideration for the future.

SYMPTOMS/PROGNOSTIC IMPLICATIONS FOR AN EMPLOYED PATIENT

There will be differences in both complaints by the patient about his symptoms and also the significance of those symptoms according to whether a patient works or not and also according to the job that he does. Under and over reporting of symptoms in CVD is common. Over reporting may occur where an individual feels unsafe at work, but more commonly a worker will under report for fear of losing his job. Denial has been reported in professional drivers. Equally, workers with symptoms but who are denying them often move from a “high risk” job to a “lower risk” job. This can be a clue to subclinical disease as demonstrated by the high cardiac risk seen among workers who have recently moved from shift to day work; this represents an individual’s attempt to help himself move away from the cardioxious environment—the so called “healthy worker effect”.

The common symptoms for heart disease are the same for the worker as they are for the non-worker, but for the worker they can have both public and personal impact if the individual is in a safety critical job.

These symptoms are:

- ▶ chest pain, angina, or acute myocardial infarction
- ▶ breathlessness and fatigue from heart failure
- ▶ loss of consciousness or temporary aberration caused by arrhythmias.

Most jobs associated with safety critical issues are those where the worker operates vehicles—for example, cars, buses, planes, trains, ships, etc. In addition, operating cranes or handling dangerous industrial processes can also be classed as safety critical. Risk assessment for these activities is complex. For car driving, figures show that less than 0.1% of all accidents can be attributed to a health problem. Of those, 10–25% are said to be related to a cardiac event.

In a study undertaken over 20 years looking at the London Public Transport Service,² only six accidents were found to have been caused by a driver having a heart attack. A total of

6.8 million miles were covered corresponding to 334 000 driver hours. It therefore seems reasonable to assume that asymptomatic drivers who fulfil the Driver and Vehicle Licensing Agency (DVLA) criteria can safely be allowed to return to work without harm to others or themselves.

Loss of consciousness poses an equally complex problem. The decision as to whether an individual with a cardiac condition where there is a risk of loss of consciousness can return to a safety critical job depends on the exact nature of the cardiac condition and the likelihood that it will induce a serious arrhythmia, the probability that even with treatment the arrhythmia may recur, and the likelihood that the arrhythmia will impact on the individual losing control of the vehicle/process/machinery and that that loss of control will endanger other individuals or cause significant loss of another kind. This is the type of question that may be asked of a cardiologist when assessing the risk associated with returning to work into a safety critical role.

IMPACT OF WORK ON CARDIOVASCULAR DISEASE

It has long been acknowledged that certain occupational exposures will exacerbate or even cause cardiovascular disease. Some of these exposures are very common in the workplace, others are now quite rare.

Physical hazards

Temperature extremes

Extremes of heat or cold temperatures in the workplace have both been linked to an increased risk of acute cardiovascular events, usually where pre-existing CVD is present. Heat exposures occur in a variety of workplaces such as foundries, mining, etc. Where this heat exposure leads to heat stroke and heat exhaustion, the risk of cardiac ischaemia in an individual with CVD appears to be related to the magnitude of heat stress.^{3 4}

Exposure to extremes of cold occurs in cold stores/freezers or outdoor workers and in relation to certain sports—for example, ski instructors. Studies have found significant increases in the rate of silent ST depression in women workers working in ambient temperatures lower than 20°C. It is possible that sudden exposure to cold could induce coronary artery spasm as in the cold pressor test, which has been shown to provoke changes in myocardial perfusion not only in patients with CVD but also normal subjects.^{5 6}

Noise

There is considerable, if at times conflicting, evidence that prolonged exposure to noise (exceeding 80 Db) can cause significant elevation of blood pressure. Other factors relating

Cardiovascular disease and its impact on work: key points

- ▶ Cardiovascular disease in the population at large but also in the population at work is common
- ▶ In a study of self reported illness, 2–3 million employees in the UK report an illness caused by or made worse by work leading to 3.3 million working days lost; 80 000 are said to be due to CVD
- ▶ The impact of work on health and health on work is the basis of occupational medicine
- ▶ Cardiologists need to consider the impact of work on their patient’s case management, treatment, and prevention strategies

to noise are lack of predictability, lack of meaningfulness, that it is intermittent in nature, and of a disharmonious nature.⁷ Proponents of a causal relation between noise and hypertension suggest that this rise in blood pressure could result in modest increases in the relative risk for CVD.⁸

A few studies looked at the impact of noise exposure on other cardiac risk factors—for example, cholesterol values. Results are not definitive. A simple study found a borderline significant relation in men between industrial noise exposure and ST depression in ambulatory ECG monitoring.⁹ It is important to remember that noise exposure in the workplace remains common and further research in this area is important.

Vibration

Vibration can be segmental (affecting part of the body—for example, when using vibrating hand held tools) or whole body (for example, driving or fork lift truck operation). Evidence is strong that vibration of either type has acute effects on the arterial intima which may cause impact on the cardiovascular system.¹⁰

Work involving electromagnetic fields and radiofrequency radiation has had postulated links to cardiovascular disease but no definite link has been demonstrated so far.⁷ Further research continues.

Many jobs have become more sedentary—this in itself is associated with increased risk of CVD, with an increased relative risk of inactive compared to active work of 2.0.⁷ However, certain types of physical activity may be detrimental to CVD. Irregular heavy physical exertion (such as 6 or more metabolic equivalents) is associated with increased risk of acute myocardial infarction in the first hour after exertion. A significant increase in standardised mortality ratio for myocardial infarction related to heavy lifting has been noted when combined with “hectic” work.¹¹

Chemical hazards

Several specific occupational chemical exposures have been linked to specific cardiovascular conditions—for example, carbon disulfide, methylene chloride, and nitrate esters. Evidence is strongest where exposure levels are high.

Carbon disulfide (CS₂)

CS₂ is used as a solvent when manufacturing viscose rayon, but it is also used in the manufacture of other chemicals and as a solvent directly. CS₂ is absorbed via inhalation and also via the skin. Exposure levels associated with CVD is reported at 20–60 parts per million (ppm). Current permitted occupational exposure is now much lower than this at 4 ppm (for an eight hour shift) and a short term exposure limit—that is, a sudden exposure of 12 ppm. Epidemiological studies of viscose rayon workers showed a two- to fivefold excess in mortality from CVD in workers exposed to CS₂ in 1968. Reduction in exposure to CS₂ reduced the risk of CVD to those of control groups—that is, the effect on the cardiovascular system was reversible.⁷

Toxicity effects are caused by CS₂ reacting with amines and amino acids to produce dithiocarbamates which complex with trace metals such as copper and zinc and react with coenzymes such as pyridoxine. The effect of these reactions is an inhibition of enzyme systems. Direct and indirect mechanisms then act to produce increased CVD risk—for example, microaneurysms, increased low density lipoprotein (LDL), decreased fibrinolysis, hypertension, a negative

inotropic effect, and direct ECG changes. As exposure to CS₂ has been reduced in the workplace, the incidence of CVD in this industry has reduced.

Nitrate esters

Nitroglycerine (glyceryl trinitrate, GTN) and ethylene glycol dinitrate exposures occur in the munitions and explosives industry and among construction workers handling dynamite. Exposure levels are high during explosives mixing and cartridge filling operations. Nitrate esters are very volatile and readily absorbed through the skin and via inhalation. Modern work practices such as automation dramatically limit exposure, but it is important to remember that occupational exposures produce much higher plasma concentrations of GTN than occurs with therapeutic doses—for example, 98.1 nmol/l (median) in workers involved in the production of gun powder compared to 5.7 nmol/l after taking 1.0 mg of GTN sublingually.

In the 1950s, epidemics of chest pain and sudden death in munitions workers was noted. The deaths typically occurred 36–72 hours after withdrawal from exposure to the nitrate esters and so were thought to be caused by nitrate withdrawal syndrome. The mechanism of action of nitrate withdrawal syndrome is thought to be due to adaptation of the body's physiological response to the vasodilator effects of nitrates—that is, flushing, headaches, and palpitations.⁷ When exposure is prolonged, compensatory vasoconstriction occurs with activation of the renin–angiotensin system. If exposure to nitrates stops—for example, over a weekend or during a holiday away from work—vasoconstriction is unopposed leading to coronary spasm, angina, myocardial infarction, or sudden death. This has been termed “Monday morning angina”.

Carbon monoxide (CO)

Significant exposure to CO is more common than exposure to CS₂ or nitrate esters. Carbon monoxide binds aggressively to haemoglobin resulting in reduced oxygen delivery to tissues, including the myocardium. High levels of CO exposure (> 25% carboxyhaemoglobin) occur relatively rarely in occupational settings. Examples include fire fighting and use of diesel engines in confined spaces. Such levels of CO exposure can lead to myocardial ischaemia/infarction, arrhythmias, or even sudden death.¹² At lower levels of CO exposure, the acute cardiac effects depend on how efficiently the individual's coronary arteries respond to the hypoxic stress so as to increase coronary artery blood flow.

Exposure comes from combustion of organic materials which includes a complex mixture of gases and particulates such as CO, polycyclic aromatic hydrocarbons, nitrosamines, hydrogen cyanide, oxides of nitrogen, etc. The route of exposure is via inhalation. Typical workplace exposures result in carboxyhaemoglobin levels of 2–8% whereas cigarette smokers may have levels of 5–15%. Where smokers have CO occupational exposure, levels increase. A carboxyhaemoglobin of 5% is tolerated by healthy individuals but not by those with cardiovascular or chronic lung disease.

An additional source of CO is via methylene chloride which is a solvent used in degreasing or as a paint or varnish remover. Methylene chloride is metabolised by the liver in part to CO. Exposure to methylene chloride can result in high carboxyhaemoglobin levels. A carboxymeter gives an easy mechanism for workplace monitoring. Results of various studies of the impact of CO exposure and CHD are

conflicting. However, epidemiological studies suggest that high level CO exposure may cause moderate increases in CVD, whereas low levels of CO exposure in individuals with pre-existing CHD could increase the risk of cardiac ischaemia.

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Solvents and arrhythmias

A small number of chemicals have been associated with atrial or ventricular arrhythmias. Such agents are bromofluorocarbons, methyl chloroform, methylene chloride, and trichloroethylene. Glue sniffing, where very high intentional exposures occur, give the strongest support to this hypothesis. In the workplace exposure to these solvents occur in dry cleaning, degreasing, painting, and chemical manufacture. Numerous cases of sudden death in relation to high level solvent exposure either occupationally or through solvent abuse have been reported in the literature.⁷ Low level exposure to solvents has not shown increased risk of CHD except for CS₂ as discussed above.

The halogenated hydrocarbons exhibit complex effects on the heart. Low level exposures make the heart more sensitive to the effects of catecholamines so that a lower dose of adrenaline is required to produce ventricular tachycardia or fibrillation once solvent exposure has occurred. Solvents may also induce bradyarrhythmias and reduce AV nodal conduction leading to AV block.

Lead

Occupational lead exposure is still common in some industries such as construction, lead smelting, and the manufacture of lead batteries, and unusual jobs such as in the production of leaded windows or repair of old pottery. Exposure to lead is said to cause rises in blood pressure levels. Environmental exposure to lead occurs via inhalation of exhaust fumes from leaded petrol and drinking water carried in lead pipes. The concentration of lead in the blood is easily measured. Regulation of workers in the lead industry ensures, via control measures, that blood lead concentrations are being reduced as far as is reasonably practicable via reduced exposure to lead. One recent review of annual epidemiological, occupational, and population studies demonstrated that even at low concentrations, lead was



Figure 2 Testing safety systems in the chemical industry.

contributing to increases in blood pressure levels.¹³ A twofold increase in blood lead concentration was associated with a 1 mm increase in systolic pressure and a 0.6 mm increase in diastolic pressure.

The mechanisms by which lead exerts this effect is postulated as including interference with calcium metabolism, potentiation of sympathetic stimulation, and a direct effect on vascular smooth muscle.

Cobalt

Cobalt is used in the production of metal alloys for drills and bits. Cobalt was first associated with cardiotoxicity in the 1960s in beer drinkers where cobalt had been used to stabilise the foam. Mortality rates in this group were high but similar responses to occupational cobalt exposure have not been demonstrated. Removal of cobalt from the beer halted the epidemics. It is postulated that the cobalt along with excessive alcohol consumption and/or malnutrition acted together to bring about the cardiomyopathy seen in this very specific group.

Arsenic/arsine

Occupational exposure has occurred in smelters where ore containing arsenic is processed or in relation to arsenical insecticides which have been used in vineyards. Acute arsenic poisoning can cause ECG abnormalities and recurrent ventricular fibrillation has been described. Arsine gas causes red blood cell haemolysis and can cause cardiac failure. Massive haemolysis causes hypercalcaemia and the attendant cardiac changes associated with this.

Subacute arsenic poisoning caused by drinking arsenic contaminated beer has been associated with cardiomyopathy and cardiac failure. An epidemic in Manchester affected 6000 people leading to 70 deaths. The mechanism of action is unclear.¹⁴

Biological hazards

Many occupations are associated with biological hazards, especially where there is contact with animals or humans or their byproducts. It is difficult to postulate an association between an occupational biological hazard and the onset of CVD. However, individuals with coexisting CVD may be at greater risk of such infective agents because of their CVD.

Many more employees travel abroad to developing countries than ever before. They are not always made fully aware of the very unusual and specific risks of such travel abroad. Again, infective agents encountered on these periods of travel may impact on cardiovascular health and the possibility of business travel should be considered when assessing patients.

Psychosocial hazards

It was as early as 1958 that evidence began to emerge that exposure to “occupational stress and strain” was much higher in young male coronary patients than in equivalent healthy controls.¹⁵ At the same time, Friedman and colleagues published their findings showing a significant relation between serum cholesterol concentrations and blood clotting times and a cyclical variation of worker related stress in a group of accountants.¹⁶ Many more published works have continued to demonstrate a relationship between work related stress and the development of CVD. A major contributor in this field is Karasek who introduced the concept of the “job strain model”. Karasek postulated

that strain occurs where there is excessive psychological workplace demands coupled with low job decision latitude. Social isolation has been added to this combination more recently as an additional deleterious factor.

Conversely, Karasek postulated that work, where high demands were coupled with high decision latitude, led to increased learning with the likelihood of improved coping mechanisms and improved health outcome measures—that is, high demands were not deleterious of themselves, it was the coupling with lack of control. A detailed analysis of studies of job strain and ischaemic heart disease in men¹³ showed that when taken together, the studies demonstrate a significant positive relationship between exposure to low control and/or job strain and the subsequent development of CVD. Cohort studies also demonstrate the temporal nature of the association.

The impact of job strain on blood pressure levels has also been studied. The results show a correlation between job strain and ambulatory systolic blood pressure. Where casual blood pressure readings are used, this correlation is very limited. For ambulatory blood pressure measurements, the associated rise with job strain exposure extends beyond work periods into leisure time and evidence exists that reduction in the levels of “exposure”—that is, job strain—reduces the likelihood of morbidity in this group, said by Hemborg to be “the most conclusive evidence of causality”.¹⁷ Obesity has been shown in some studies to be significantly associated with job strain. A study of US chemical operators in 1990⁹ showed that even after controlling for sociodemographic factors, those workers exposed to higher levels of job strain smoked more than comparable workers in lower job strain roles. There is, therefore, some evidence building that job strain impacts on the likelihood of development of CVD via changes in blood pressure and other cardiac risk factors. Definitive conclusions cannot be drawn yet and much more research will be needed.

Another measure of the psychological impact of work is the “effort–reward model” which looks at effort/reward imbalance in the workplace. The Whitehall study¹⁸ showed that exposure to high effort and low reward at work was associated with double the risk of newly reported CHD over 5.3 years. Work that requires employees to maintain a high level of vigilance to prevent major incidents/accidents presents a greater psychological burden than many jobs. Such work includes professional drivers, air traffic controllers, sea pilots, etc. Epidemiological studies have shown some association between this type of work and CVD outcomes.¹³

Shift work is recognised as an occupational risk factor for CVD. There is, unfortunately, no agreed definition of shift work but it usually applies to fixed work at night, roster work, and specific shift patterns—of which there are many. The number of people working shifts appears to be increasing. In Europe about 18% of the workforce works at night 25% of the time and even more work outside normal hours.¹⁹ This means that shift work is one of the most common work environment risk factors for CVD. However, shift work is unlikely to be eradicated since society requires that some work outside of normal hours continues; therefore, attempts to modify the impact of shift work on cardiovascular health will need to be by manipulating and reducing the links that shift work has with the development of CVD.

It has been postulated that the link between shift work and CVD is via three pathways—mismatch of circadian rhythms,



Figure 3 Vessel work requires a high level of personal protective equipment.

social disruption, and behavioural changes. The mismatched circadian rhythm relates in part to eating patterns—eating more calories at night than during the day has been associated with higher cholesterol values.²⁰ Myocardial infarction rates and angina are higher in the early morning. It has therefore been suggested that mismatch of oxygen supply to cardiac muscle may precipitate this increased rate of angina. Workers requiring extra cardiac effort at this time may be at greater risk. Ventricular extrasystoles are more common in night workers which may have a role to play, and the persistent lack of sleep that shift workers suffer is also postulated to be an issue, but there is no research to support this theory as yet. Shift work impacts on the availability of social support and lack of social support is well recognised as a risk factor for CVD. Finally, shift work often leads to behavioural changes which are risk factors for CVD. These include higher smoking rates, altered eating habits—snacks or missed meals—and one study showed that shift workers, though not significantly heavier than non-shift workers, had more centrally deposited adipose tissue—another risk factor for CVD.²¹ Exercise levels and alcohol consumption were no worse in shift workers than non-shift workers.

Many questions remain unanswered about the effect of shift work on CVD. Better organisation of shift work may be protective, and more research on what constitutes a “healthier shift pattern” is urgently required.

IMPACT OF HEART DISEASE ON WORK

A diagnosis of heart disease can have an impact on work capacity/capability in many ways. It is important that, where possible, this impact is evidence based rather than based on preconceived ideas, assumptions, and practice that is in effect discriminatory.

PRE-EMPLOYMENT OR CAREER ADVICE

Individuals with congenital heart disease and also young people with acquired heart disease may ask for specific career advice. Generally, this is likely to relate to careers that have very specific medical standard requirements at pre-employment, such as the armed forces and other safety critical roles. Early discussions can help to develop

Hazards in the workplace: key points

- ▶ Certain hazards at work have long been recognised as exacerbating or even causing cardiovascular disease
- ▶ Hazards in the workplace are usefully considered in terms of:
 - physical
 - chemical
 - biological
 - ergonomic and psychosocial exposures
- ▶ Although physical and chemical hazards are better controlled, work conditions for many employees remain potentially harmful to health
- ▶ Shift work and changing workforce demographics—for example, an aging workforce—will need to be considered in relation to cardiovascular risk

appropriate expectations and informed career choices for these individuals.

RETURN TO WORK AFTER THE DEVELOPMENT OF HEART DISEASE

Studies on the success of returning to work after the onset of heart disease have shown that several different issues are involved.

The nature of the original cardiovascular event that led to the individual stopping work

The most common condition is CHD, which can range from acute myocardial infarction to angina treated with coronary artery bypass, angioplasty, or drugs alone. With CHD, whichever form it takes, the important issues for return to work are the persistence of chest pain during exercise, the risk of arrhythmia, and the level of left ventricular function, especially if this may impact on exercise capacity. In addition the possibility of silent ischaemia needs consideration for high risk individuals. It is interesting that although angioplasty ensures a more speedy return to work than coronary artery bypass surgery (CABG), long term employment prospects are the same in both treatment groups.²²

For uncontrolled hypertension, symptoms such as headache, dizziness, or general malaise may make return to work difficult. Physical and psychological impacts of work may induce worsening hypertension and compound the problem. Where possible, treatment regimens should be effective and stabilised before return to work.

Symptoms of general malaise and cardiac syncope are strong indicators of relapse. These symptoms therefore require careful evaluation. Whereas cardiac failure once meant that a return to work was unlikely, improvements in treatment regimens now mean that more people with heart failure can return to work. Whatever condition gave rise to the cardiac failure needs treatment and the more effective this treatment is, the more likely is the individual to return to work.

The residual loss of function following the cardiac event

Functional capacity of the individual should be assessed before he or she returns to work. For cardiac disease an exercise stress test will give the information required for individuals with coronary artery disease and hypertension. Assessment of individuals with cardiac failure may need additional investigation.

The prognosis of the causative CVD

Prognostic indicators are well documented for most cardiovascular problems. Where the prognosis is poor and risk of recurrence high, return to work may be inappropriate and create unrealistic expectations for the patient and his family.

The nature of the individual

Psychosocial factors may play a much bigger role in whether an individual returns to work than medical/clinical factors. Research has shown some useful pointers regarding the likelihood of an individual returning to work after a cardiac event.

- ▶ Whatever the prognosis of the CVD, the longer an individual is off sick, the harder it is to return them to work
- ▶ If the cardiac event happened at work, return to work can be more difficult
- ▶ Age—the older the patient, especially if a pension is available, the more unlikely it is he will return to work
- ▶ Type of job—where the job is seen as unrewarding/unfulfilling, a patient is less motivated to return to work. In addition if the job is seen as dangerous or damaging to health, return will be more difficult
- ▶ Suitability for redeployment/retraining—where redeployment is difficult to achieve because of certain individual factors such as education, adaptability or even personality, the likelihood of returning that individual to work is reduced
- ▶ Attitude of employer
 - fear of further illness at work and subsequent litigation can act as a barrier to return to work; many employers take a defensive stance in these situations
 - failure to consider rehabilitation/redeployment will make return to work more difficult
 - the existence of a “cardionoxious” work environment in the organisation which can be physical or related to the culture of the organisation will hinder a return to work
- ▶ Attitude of the individual
 - fear of future illness
 - lack of motivation/understanding of the illness
 - benefits of the “sick role”
- ▶ Attitude of the state
 - sickness benefit may be generous and with low paid jobs better than being at work
 - safety related medical standards and acceptability of “risk” are dictated by the state to a large extent and, if overcautious, can prevent some people returning to work.

IMPACT OF DRUGS/DEVICES ON RETURN TO WORK

The cardiologist is far better informed about current cardiovascular treatments and devices than the occupational physician. Side effects and their impact in the workplace do need to be considered rather than simply considering their effect on the individual alone. Shift work, travel to work, and business travel may all have an impact when medication is taken. Altering the time of taking tablets can significantly improve the onset of side effects or even treatment responses such that they are less intrusive in the workplace.

Pacemakers may be affected by high level magnetic fields in jobs where such fields are generated, such as in electricity generating or transmission or welding. Nuclear magnetic resonance imaging machines may also pose a problem. The

position in which a pacemaker is placed may be significant for some job types. One example is a professional driver where the pacemaker is directly below where the seat belt lies during driving.

In some individuals it will be clear that return to work will not be possible and that the person may be considered for ill health retirement. A decision about suitability for the ill health pension is very significant for the individual, the employing organisation, and the associated pension fund. Different companies have different approaches to this process. The role of the cardiologist would be as a specialist medical adviser providing information, with the informed consent of his patient, on the clinical diagnosis, likely prognosis, and treatment options.

THE FUTURE OF WORK

Current trends in working life for most people are not improving. Many workers are spending longer hours at work, often with deteriorating work conditions and work environments. A study in Europe in 1996 showed that 23% of employees worked more than 45 hours a week. Similarly, in the USA working hours have increased by 3.5 hours up to 47 hours a week from 1977 to 1996.

Changes have also occurred in relation to the nature of work in industrialised nations over the past four decades. The main changes relate to increased workload demands and increasing pace of work. Some increase in job decision latitude or control over work tasks has occurred but at an insufficient rate to compensate for the increased demands. It remains clear that men and women are working harder now than 25 years ago. New systems of work organisation have been introduced by employers to bring about increased productivity, better quality of goods/services, and of course increased profitability. These systems go under various names—lean production, total quality management, or modular manufacturing. Very often such changes are introduced without consultation with workers—80% of US workers want to have a say in decisions about their job. It may be that a weakening of unions has led to these changes.

The features of lean production such as downsizing, outsourcing, 24 hour working, increased overtime, and increased worker flexibility have all been made easier by the loss or reduction of union influence. Downsizing and excessive overtime can have negative effects on workers' health; in addition such changes often act as serious barriers in returning an individual back into work after a period of illness. These same issues need to be taken into account when assessing individuals at pre-employment to ensure work suitability; they may also have an impact on decisions relating to ill health retirement as it may be very difficult to introduce reasonable adjustments or redeploy in such organisations. It is important to remember that many of these organisational changes result in negative effects on health and indeed contribute to increased CVD risk, particularly in the lower socioeconomic groups.²³

Two other groups at work need consideration—women and the elderly worker. Women now make up a large proportion of the working population. Women tend to work in service industries—office work, sales, health care, and teaching. This type of work is often associated with a higher prevalence of job strain and with a lower level of job control than in most men's jobs.¹³ The additional impact of family responsibilities for many women adds to these stressful

exposures. Despite the increasing involvement of women in paid work, women still spend more hours in child caring and housework than men. A study in western Europe showed that women spent an average of 35 hours a week in child care and housework before 1975 and 31 hours after 1975. Men spent eight and 11 hours, respectively, during the same period.¹³ In the Framingham study,²⁴ employed women with three or more children had a higher incidence of CVD than employed women with no children or housewives with three or more children. Women who work shifts or long hours appear to have an increased rate of hospitalisation for myocardial infarction whereas for men, moderate overtime may be protective. This sex difference may be explained by the difficulties women find in combining family responsibilities with irregular or long working hours.

By 2020 the UK population will include 19 million people over the age of 60 years. This is the population bulge that followed the second world war and has major implications for the population at large. In 1975, 95% of 55–65 year old men worked; by 1999 the figure had fallen to 60%. Since then the UK government has undertaken various initiatives to get older people to continue working longer. If these initiatives are successful, individuals will continue to work beyond the retirement age of 65. Since many CVDs are more common in an aging population, it is likely that these demographic changes will have impact on all aspects of heart disease and work.

Finally, the aim of this article was to alert practising cardiologists to the important and often complex interplay between heart disease and work. It will have been successful if, in future consultations, consideration is given at every stage to the individual's work and its implication for case management, treatment, and prevention.

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