raphy, the transduodenal approach to the bile duct with sphincteroplasty is the obvious method of choice when dense adhesions surround the previous operative site. Peroperative intravenous aprotinin (Trasylol) minimises the development of clinical pancreatitis, although postoperatively the serum amylase rises. Endoscopic papillotomy may also be tried in specialist centres. With severe obstructive jaundice the site of obstruction should be shown by either retrograde cholangiopancreatography or percutaneous transhepatic cholangiography just before laparotomy. The transduodenal approach is ideal for stone impacted in the lower end of the common bile duct, but in high-risk cases with severe jaundice and septicaemia simple T tube drainage of the bile duct is indicated with forced diuresis and appropriate antibiotic cover. Elective surgery may be undertaken later after control of infection and T tube cholangiography.

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For Debate

Deaths from coronary artery disease and coalworkers' pneumoconiosis

DEWI DAVIES

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Summary

When pneumoconiosis makes a material contribution to a miner's death benefit is paid to his widow. Doctors from the Department of Health and Social Security have argued that extensive pneumoconiosis not only does not increase the risks of death if coronary thrombosis develops but also that it may exert a beneficial effect. This view has been put to the National Insurance Commissioners on at least three occasions recently. There are, however, several reasons for coming to the opposite conclusion. There is no evidence that progressive massive fibrosis reduces mortality or the development of atheroma, and until such evidence is produced it is unwise to believe that the presence of any severe disease improves anyone's chances of surviving coronary thrombosis. Rather, it should be recognised that any disease which produces a high mortality is inevitably associated with a lower mortality from other conditions. As doctors from the Department will continue to argue that severe pneumoconiosis does not increase the risk of death from coronary thrombosis informed readers are asked to adjudicate between the conflicting views.

Introduction

When a man with pneumoconiosis dies his widow is entitled to death benefit if the disease made a material contribution to his death. The decision is made by an insurance officer who relies on the opinion of a pneumoconiosis medical panel which has

examined the heart and lungs. Appeals from such a decision may go to a local tribunal and from there to a National Insurance commissioner. Cochrane¹ in a statistical study of advanced pneumoconiosis concluded that the regulations were being interpreted generously, but disquiet has been expressed about individual cases.² In my view there is now cause for great dissatisfaction about decisions made when men with advanced pneumoconiosis die from coronary artery disease.

Case report

A coalminer was found to have category 2 simple pneumoconiosis in 1963 at the age of 63. Disability was assessed at 10%. Shortness of breath increased steadily, and when he was last examined by the panel in the summer of 1973 he had to stop after walking 20 yards, his weight had dropped by 14 kg, and the pneumoconiosis had progressed to category B—the highest-but-one category of progressive massive fibrosis (PMF). There was also extensive pleural thickening over both lungs. Ventilatory tests were not done because he was considered to be too frail for the effort, but between 1963 and 1971 his vital capacity and forced expiratory volume in one second had fallen by 25%. His deterioration could not be attributed to anything other than fairly rapidly progressive pneumoconiosis. Disability in 1973 was assessed at 50% (40% for pneumoconiosis and 10% for emphysema).

A year later he developed a myocardial infarct. He was managed at home by his family doctor and died suddenly 20 hours after the onset. At necropsy the pathologist found considerable coronary atheroma with a recent thrombus and a posterior myocardial infarct. There was doubtful right ventricular hypertrophy. Extensive pneumoconiosis was present, and the pathologist concluded that this had materially reduced the chances of survival.

The panel broadly agreed with the findings but claimed that there was evidence of previous infarcts. When further evidence against this was later presented the point was dropped. The PMF was estimated to total 237 ml. In the right lung the masses had liquefied and had been partly coughed up. Subsequent shrinkage had not been taken into account in estimating the amount of PMF before cavitation occurred, and in my view PMF amounted to about 500 ml. Only 3% of certified patients have disease of this extent at necropsy; if the panel's estimate is taken, the proportion is about 10%.³ The pleural thickening, attributable to the underlying pneumoconiosis, could be blamed for

City Hospital, Nottingham NG5 1PB

DEWI DAVIES, MD, FRCP, consultant chest physician

further respiratory impairment. The panel concluded that death would have occurred when it did irrespective of the presence of pneumoconiosis, and death benefit was not paid.

At the local tribunal the widow's case was supported by a report that claimed that death was not inevitable from myocardial infarction alone and that pneumoconiosis increased the risk on three counts. Firstly, the miner was so disabled by pneumoconiosis that death could have occurred at any time from the lung disease alone or from a superimposed trivial accident or illness. Secondly, the lung disease was so severe that hypoxaemia was undoubtedly present. This would have two adverse effects: collateral blood entering the area supplied by the blocked coronary artery would give less protection and allow more extensive infarction; and partly damaged muscle, perfused by hypoxic blood, would be more likely to produce the ventricular fibrillation that apparently killed him. Thirdly, some degree of right ventricular strain, caused by the lung disease, reduced his chance of survival.

A member of the panel gave verbal evidence and the chairman's extensive notes report him as saying that these views were not supported by medical facts and that it had "been shown in several medical articles that blood of poorer quality is an advantage to people suffering from lung disease and the supply of poorer quality blood to the heart gives these patients a greater chance of recovery from heart attack." This opinion was repeated later in his evidence. The widow's appeal was rejected.

Before the commissioner a senior medical officer of the Department of Health and Social Security said that pneumoconiosis had no influence on survival from myocardial infarction unless it was severe enough to produce a bedfast state or congestive cardiac failure. He quoted the papers on which the Department's views were based³⁻⁵ (see below). After his interpretation of these had been extensively criticised, he adhered to his view and continued to support the statement made by his colleague at the local tribunal.

In view of the conflict of opinion the commissioner sent the papers to an independent physician. This doctor considered that the heart disease "was sufficient to account for his death without any lung disease." The commissioner then decided that death was not materially accelerated by pneumoconiosis.⁶

Such arguments had also been used successfully in two similar cases before the Chief Commissioner in 1974,^{7,8} and his decisions were introduced as evidence at this hearing. It is therefore desirable to consider whether these persuasive arguments, which will be evidently used again, are logical or not.

Reports

Nonkin *et al*⁴ in a study of 2853 necropsies at a veteran's hospital found that no patients with obstructive emphysema died from myocardial infarction, whereas 10 or 11 deaths would have been expected. Necropsy studies by Sanders⁵ and Lindars *et al*³ showed fewer deaths from coronary thrombosis in those with PMF than in those with simple pneumoconiosis. These reports have led the Department to conclude that severe pneumoconiosis protects people from dying from coronary thrombosis and to the theory that hypoxaemia produced by pneumoconiosis stimulates the development of protective intercoronary anastomoses.⁸

The patients in Nonkin's⁴ survey died in hospital. Only 4% were classified on clinical grounds as having definite obstructive emphysema, an obvious underestimate for such a prevalent condition. The classification indicated that the emphysema was severe, so not surprisingly these patients died from this and not from any other condition. The remaining 96% had a wide range of disorders and many undoubtedly had emphysema as well. There is nothing unexpected in finding that 10% of them died from coronary thrombosis.

Sanders⁵ studied the type of heart disease found at death in men with coalworkers' pneumoconiosis. Of those with simple pneumoconiosis, 27 died from myocardial infarction and four died from cor pulmonale. In contrast, five of the men with PMF died with myocardial infarction and 28 died from cor pulmonale. The size of the groups was not given. Sanders concluded that "patients with PMF... are protected from death from coronary artery disease and are more likely to die from congestive cardiac failure than are patients with simple pneumoconiosis." His second deduction is correct but the first,

on the evidence made available, is absurd. It is accepted that PMF produces pulmonary hypertension and will cause many to die from cor pulmonale; they cannot also die from coronary thrombosis. On the other hand, simple pneumoconiosis does not produce much pulmonary hypertension, and those who die from heart disease are most likely to die from coronary thrombosis.

Lindars *et al*³ found that fewer patients with extensive PMF died from coronary thrombosis. They also found that coronary thrombosis occurred more often in those with little shortness of breath and less often when it was severe. They too failed to see what needs to be enunciated as a principle: that any disease which produces a high mortality is inevitably associated with a lower mortality from other and independent conditions. Taken to its ultimate conclusion, getting run over by a bus protects one from dying from a whole host of other nasty conditions. Failure to appreciate this has also led to a claim that chronic bronchitis protects men from developing lung cancer.⁹

An objection put forward to this reasoning is that many patients with PMF are older at death than those with simple pneumoconiosis.³ PMF, however, is a sequel of simple pneumoconiosis and most men in the Midlands have to live to a considerable age to develop it. They have to be relatively free of such conditions as chronic bronchitis and coronary atheroma or they will die before they acquire it. The patients with simple pneumoconiosis, however, will include men with diseases that tend to kill at a younger age. (The same mechanism helps to explain why cardinals of the Roman church are old when they die.) It is reasonable to believe that most people with PMF would almost certainly live even longer but for their lung disease. Getting run over by a bus at the age of 70 still shortens life.

In contrast to these wrongly interpreted findings,³⁻⁵ prospective surveys have shown that the existence of impaired lung function at entry increases the risk of developing myocardial infarction later.^{10,11} The association is not considered to be due to excessive smoking.

Mortality

These studies say nothing about case mortality from coronary thrombosis. I have been unable to find any studies that compare this in people with or without lung disease of any kind, let alone pneumoconiosis. Such a study would be valuable. Unless evidence is available it seems unwise to believe that the presence of any severe disease improves anyone's chances of surviving a coronary thrombosis. A true protective effect from PMF can be claimed only if it is shown to reduce case mortality or the development of atheroma and, in turn, the number of infarcts. There is no such evidence.

The Department's advisers hold the view that severe pneumoconiosis does not have an adverse effect unless it produces heart failure or makes a man bedfast. They go further in claiming that it may have a beneficial effect. Their explanation is that hypoxaemia secondary to lung disease improves intercoronary anastomoses.

There is evidence that anastomoses are found in most patients with uncomplicated cor pulmonale, and hypoxaemia may be the determining factor. But the greatest stimulus to the development of anastomoses is coronary atheroma.¹² There is a strong probability that most people with infarcts already have collaterals, though they prove inadequate to prevent infarction. There is no evidence that the presence of chronic lung disease would produce better anastomoses.

Hypoxaemia reduces overall myocardial function and aggravates left ventricular failure.¹³ Precapillary shunts in the lung and pulmonary hypertension from PMF also contribute to left heart stress. Destruction of the pulmonary capillary bed, even if it has been insufficient to produce obvious right ventricular hypertrophy, increases the effects of the pulmonary oedema that often complicates myocardial infarction. Hypoxaemia increases the extent of infarction in dogs,¹⁴ and in man oxygen reduces the extent of electrocardiographic abnormality

after infarction.¹⁵ Hypoxaemia may also increase the risk of severe arrhythmia. Rawles and Kenmure,¹⁶ however, failed to show benefit from administering oxygen after infarction. They excluded all patients with evidence of heart failure, lung disease, and breathlessness, so their findings can be applied only to uncomplicated cases.

Conclusion

It seems reasonable to believe that if pneumoconiosis is severe enough to produce chronic hypoxaemia, pulmonary hypertension, or a large reduction in the pulmonary capillary bed it is likely to reduce the chances of survival. In contrast, the views put forward by the Department are, in my opinion, irrational and based on entirely faulty deductions. In a field in which there is little firm evidence, it is important that what is available is interpreted sensibly. If the arguments used by the Department are faulty there is no mechanism now available to recompense the widows in the three cases cited⁴⁻⁶ without resort to High Court actions. There are probably many more such cases that did not get beyond the insurance officer or the local tribunal.

I thank Dr D C Banks for critical comments.

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Clinical Topics

Barbiturate prescribing: psychiatrists' views*

B M BARRACLOUGH

British Medical Journal, 1976, **2**, 927-928

Summary

When questioned by post most of a one-in-five sample of consultant psychiatrists replied that they did not favour the use of barbiturates as hypnotics or as sedatives.

Introduction

It is wise that doctors should monitor their use of drugs and modify their prescribing when the best interests of patients indicate that this is necessary. Like many drugs, barbiturates may be a threat to health because of side effects and abuse; the large amount of barbiturate prescribed and its association with suicide make it a drug of special concern.

When used as a hypnotic, barbiturates produce abnormal sleep rhythms that may persist for weeks after withdrawal.¹ Hangover effects the next day reduce performance.² The elderly patient may become confused.³ When used for daytime sedation rapid dependence may result, and the patient seeks to improve his mood further by increasing the dose, sometimes to a dangerous extent. In any case, the price is impaired intellectual and motor performance, sometimes to a dangerous extent. Paradoxical excitement may also occur.⁴ Barbiturate withdrawal

*Results of a survey conducted under the direction of the Research Committee of the Royal College of Psychiatrists.

MRC Clinical Psychiatry Unit, Graylingwell Hospital, Chichester, West Sussex PO19 4PQ, and Royal College of Psychiatrists, London SW1X 8PG

B M BARRACLOUGH, FRACP, MRCPsych, honorary consultant psychiatrist

may precipitate grand mal fits.⁵ Liver chemistry is affected by barbiturates, which alter the metabolism and hence the therapeutic effects of other drugs.⁶ Young people have suffered permanent damage to their bodies by injecting the contents of capsules or of ground-up tablets.

Alcohol and barbiturates are potentially fatal when taken together and the combination causes many deaths. There is a higher death rate from barbiturates than from alternatives.⁷ From 1965 to 1974 19 000 adult deaths, two-thirds of them suicides,⁸ resulted from barbiturate poisoning, about 0.3% of all deaths for this period (see figure).^{9 10}

Concern about the need to restrict the supply of barbiturates has resulted in attempts by the medical profession to limit prescribing by using educational measures. Probably these measures and the availability of alternatives account for the fact that general practitioner prescribing of barbiturates is

Barbiturate prescriptions and barbiturate deaths in England and Wales, 1965-74.