Coalworkers' pneumoconiosis in Britain today and tomorrow

Since 1965 the number of new certifications of coalworkers' pneumoconiosis (defined radiologically as category 2 or more) has declined from over 1000 to just under 500, and the proportion of working miners among those certified has been halved. The prevalence of all categories of the disease in working miners aged 55 to 59 fell from 22% in 1965 to 13% in 1979.¹ The corresponding reduction in the 35-44-year age group was from 6% to 1.3%, showing that pneumoconiosis is increasingly a disease of older men.

Several reasons account for these changes in the prevalence and age distribution of pneumoconiosis in working miners. Firstly, contraction of the underground workforce and voluntary early retirement for men with long service have reduced the numbers with pneumoconiosis. The coal industry has also been strikingly successful in reducing dust levels underground, guided by research which has described the relation between exposure to respirable dust and the risk of developing pneumoconiosis.^{2,3} Thus the industry is in the unique position of being able to base action to prevent disease on a detailed assessment of the potential health benefits and the financial and social costs of such action.

At present the emphasis in prevention of pneumoconiosis is on reduction in levels of respirable dust underground. This approach is based on the coal industry's research on ventilation and dust control. The less dust a man is exposed to, the smaller is his risk of developing simple pneumoconiosis and hence of progressing to the disabling massive fibrosis. These measures are supplemented by the industry's periodic x-ray scheme, by which all men are offered chest radiography every four years. Any man found to have early radiographic signs of simple pneumoconiosis is told of this and offered appropriate medical advice. Men with category 2 simple pneumoconiosis are advised to apply to the Department of Health's pneumoconiosis medical boards for assessment. If the disease is confirmed the coal industry is legally required to offer the man work in conditions where the dust levels are known to be low and to keep him under regular medical supervision.

On the whole these measures seem to be working well, and the risk of disablement by pneumoconiosis is appreciably less than it was a decade ago. Indeed, in some parts of the coalfield, pneumoconiosis is now very rare. Some problems remain, however. The main difficulty is in understanding the causes of progressive massive fibrosis. Recent work (so far published only in abstract) has shown that this potentially disabling condition may occur long after a man has stopped being exposed to dust,⁴ and the length of time dust resides in the lung seems to be a crucial factor. We also know that progressive massive fibrosis is especially likely to occur in men whose simple pneumoconiosis progresses unusually rapidly⁵ and that such men have often been exposed to relatively high proportions of quartz in the dust they have breathed.^{6,7} The suggestion that quartz may be responsible for some cases of progressive massive fibrosis has been supported by recent pathological studies.⁸ Quartz is not the cause of all progressive massive fibrosis, however; indeed, most cases still occur in South Wales, where in general exposures to quartz underground are very low. Speculation on the causes of non-quartz-induced progressive massive fibrosis would be premature, but one known factor is the total dose of respirable dust to which the man has been exposed. In the past

these doses have probably been higher in South Wales than elsewhere. Further investigation is needed of the peculiar characteristics of the dust and of men's exposure to it that make this part of Britain the area of highest prevalence of pneumoconiosis.

With continued control of mixed respirable dust underground coalworkers' pneumoconiosis should become increasingly uncommon in Britain. As this happens, a higher proportion of the cases that do occur are likely to be due to exposure to mixtures of dust containing relatively high proportions of quartz, since the dose of quartz required to cause disease is much smaller than that of coal. Quartz occurs in small amounts in all mixed coal-mine dust; the proportion rises to relatively high levels when cutting coal requires taking intrusions of roof or floor as well. The industry is now considering action to reduce these risks. Since nationalisation, the British coal industry has played a unique part in preventive medicine by sponsoring research to define its medical problems and by taking action based on the results of the research. This has allowed the formulation and introduction of sensible regulations for the control of dust. The falling prevalence of pneumoconiosis and continued control of dust levels despite increasing productivity stand witness to the merits of such a policy.

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Carotid body tumours

Carotid body tumours have been the subject of surgical interest for the past 100 years, but they still present three main clinical problems.

The first concerns their diagnosis. Many reported series of patients treated surgically record that the definitive operation was done after a previous diagnostic exploration or biopsy in as many as one-third of cases, indicating that at the first operation the diagnosis was unknown or incorrect. Carotid body tumours are usually hard or firm, but they may be soft and compressible. They do not usually pulsate or cause a bruit. They are frequently misdiagnosed as an enlarged cervical lymph node and discovered by a junior surgeon attempting a biopsy, sometimes under local anaesthesia. The only way for surgeons to make a correct diagnosis is to remember their existence.

The two most important physical signs of carotid body tumours are that they occur at the carotid artery bifurcation,

level with the hyoid bone, and are closely related to the carotid artery pulsation. A single lump just in front of the anterior edge of the upper third of the sternomastoid muscle, closely related to an arterial pulse, must always be regarded with suspicion and investigated by Doppler or conventional arteriography. The arteriographic features—a vascular tumour between a widened carotid bifurcation—are diagnostic. Both sides of the neck should be investigated as carotid body tumours may be bilateral.¹⁻³ They may also be familial.^{1,4}

The second and third problems are inseparable: the pathological nature of the tumour and its treatment. Macroscopically these tumours have been divided into three varieties—those loosely attached to the vessels, those adherent to the adventitia of the carotid artery but dissectable from it, and those so intimately adherent that they are difficult to remove without making a hole in the adjacent arteries.⁴ In my experience these are technical subdivisions related more to the size of the tumour, the degree to which it surrounds the main arteries, and the difficulty of the operation than to real pathological variations. Local invasion does occur in a few cases (5%) and must be accepted as true local malignancy, but even in these cases the histological confirmation of malignant change can be difficult.^{5,6} Fewer than 5% of all tumours are eventually associated with distant metastases.³ These are only estimates of the incidence of malignant change because all the published series are small.

If left untreated carotid body tumours slowly but inexorably get larger. Eventually they will obstruct the adjacent internal carotid artery and possibly cause a stroke, but the precise chances of this catastrophe occurring have not been documented.

No surgeon should attempt to remove a carotid body tumour unless he is experienced in the surgical techniques used for carotid artery surgery for atheromatous disease. This means that any surgeon who finds such a tumour unexpectedly should stop the operation, without a biopsy, and arrange for the patient to have further investigations and re-exploration by an experienced vascular surgeon. To proceed unprepared with excision without the correct vascular instruments and surgical skill is hazardous and might cause a stroke—an unacceptable complication of what the patient had probably been told was to be a simple biopsy of a node.

In most cases the tumour can be removed without damaging the carotid artery by dissecting within or just deep to the adventitia.⁷ This dissection is always bloody but the oozing stops as soon as the tumour is free. An essential preliminary is the isolation of the vessels above and below the tumour so that they can be controlled in the event of sudden bleeding. Large tumours which completely surround the common or internal carotid artery and tumours which seem to be infiltrating the artery wall must be excised en bloc.^{8,9} If this is to be done safely the cerebral circulation must be maintained with a shunt and the excised vessel replaced with a reversed autogenous graft of saphenous vein. It is the impossibility of prediction before the operation that such a major procedure will be required that must restrict performance of the operation to vascular surgeons. The patient should be made fully aware of the risks before surgery.

Apart from the risk of stroke, which should be minimal (less than 1%), the only other risk is damage to the hypoglossal or vagus nerves resulting in an awkward tongue or a husky voice. There are no clinically apparent problems from the removal of both carotid bodies in bilateral cases, but a bilateral subintimal dissection around the carotid sinuses may leave the patient with an unstable blood pressure.

There is no evidence to indicate whether radiotherapy after incomplete removal of a locally invasive lesion delays recurrence, but radiotherapy to distant metastases, especially in bone, can reduce pain and cause regression. Patients with metastases may live for many years, and aggressive palliation of symptoms is worth while.

Most of the surgical difficulties would be averted if clinicians were to remember this tumour when examining a patient with a lump in the neck and advise surgical removal when the tumour is small.

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Vancomycin: a reappraisal

Vancomycin was introduced into clinical use in 1958 and appeared a promising antistaphylococcal agent. Two years later, however, methicillin, the first antistaphylococcal penicillin, was developed and first this and later the cephalosporins and lincomycin overshadowed vancomycin. In Britain its use declined almost to zero. In the past few years, however, vancomycin has re-emerged as a valuable antibiotic in three clinical settings: endocarditis, multiresistant staphylococcal infections, and as the first-line treatment of pseudomembranous colitis.

Vancomycin was isolated from *Streptomyces orientalis* in the mid-1950s. It is bactericidal for many Gram-positive bacteria and acts by inhibiting cell-wall synthesis but at a different site from that of the penicillins. Vancomycin may also alter the permeability of the cell membrane¹ and selectively inhibit synthesis of RNA.² Since vancomycin is not absorbed after oral ingestion and causes pain and necrosis when given intramuscularly it must be given intravenously (except for treating colitis). High and prolonged drug concentrations can be achieved after its intravenous infusion as it has a long half life (about seven hours).

Before the development of penicillinase-resistant penicillins and other potent antistaphylococcal agents vancomycin was used fairly extensively for treating severe staphylococcal infections such as septicaemia and endocarditis. After methicillin (and other beta-lactamase-stable penicillins) had been used for several years methicillin-resistant strains of *Staphylococcus aureus* appeared; and in practical terms such strains are best considered also resistant to the cephalosporins. Vancomycin