

ing is massive, and it has the added advantage that clotting agents may be injected.⁶ If bleeding continues skilled judgment is needed, and joint consultation is essential; a balance has to be struck between early (and perhaps unnecessary) surgical intervention and prolonged and repeated resuscitation and transfusion, which may well weaken the patient's resistance.

Since most patients do not require intensive treatment they may be nursed perfectly satisfactorily in an ordinary ward. In spite of the pioneer work of Avery Jones,⁷ who showed that early feeding reduced mortality, the dangerous myth persists that patients who bleed from the gut should be given "nil by mouth." There is also widespread faith in alkalis, despite the clinical observation that in a patient who has bled previous dyspeptic pain (and therefore presumably acid) disappears. The immediate and long-term effects of bleeding on gastric acidity bear re-examination. A similar uncritical attitude has grown up to the use of cimetidine; several trials⁸ have shown that it is valueless in the treatment, as opposed to the prevention, of bleeding, and its "routine" use intravenously should be condemned on the grounds of both illogicality and expense. For most patients admission to hospital should mean a few days of observation rather than treatment, and if bleeding does not recur they can be safely discharged home.

¹ Cotton PB, Rosenberg MT, Waldram RPL, Axon ATR. Early endoscopy of oesophagus, stomach, and duodenal bulb in patients with haematemesis and melaena. *Br Med J* 1973;ii:505-9.

² Schiller KFR, Truelove SC, Williams DG. Haematemesis and melaena, with special reference to factors influencing the outcome. *Br Med J* 1970;ii:7-14.

³ Conn HO. To scope or not to scope. *N Engl J Med* 1981;304:967-9.

⁴ Peterson WL, Barnett CC, Smith HJ, Allen MH, Corbett DB. Routine early endoscopy in upper-gastrointestinal-tract bleeding: a randomised, controlled trial. *N Engl J Med* 1981;304:925-9.

⁵ Hunt PS, Hansky J, Korman MG. Mortality in patients with haematemesis and melaena: a prospective study. *Br Med J* 1979;ii:1238-40.

⁶ Ring EJ, Oleaga JA, Baum S. Interventional radiology in gastro-intestinal haemorrhage. In: Berk JE, ed. *Developments in digestive disease*. Philadelphia: Lea and Febiger, 1977:59-72.

⁷ Jones FA. Haematemesis and melaena with special reference to bleeding peptic ulcer. *Br Med J* 1947;ii:441-6, 477-82.

⁸ La Brooy SJ, Misiewicz JJ, Edwards J, et al. Controlled trial of cimetidine in upper gastrointestinal haemorrhage. *Gut* 1979;20:892-5.

Smoking, coal, asbestos, and the lungs

Over the past 20 years the number of British coalminers with pneumoconiosis has fallen substantially. In part the fall is due to the fact that fewer men work in the pits, but it is also a response to effective measures taken to reduce dust levels underground—on the evidence of research showing the association between exposure to respirable dust and the risks of developing pneumoconiosis.¹ Some 500 men a year are still diagnosed as having the disease by the pneumoconiosis medical boards, though the average ages at which men show signs of the different stages of the disease have been increasing steadily, largely reflecting the higher dust levels of earlier years.² Asbestosis, the other important pneumoconiosis in Britain, continues to be diagnosed by the pneumoconiosis boards in about 200 people a year; no decline in incidence has yet been seen, but current dust-control policies in the industry are expected to produce such an effect in the near future.

In theory occupational diseases are wholly preventable, but in practice so long as society requires an industry's products some men will fall ill and even die as a result. A cost in terms of

illnesses and accidents is exacted for the benefits provided by most productive industry. Society and its elected representatives need to be informed of the risks in order that work can be made as safe as possible, consistent with the need to continue or increase production³; but the ultimate responsibility in a democratic society for occupational morbidity and mortality rests with every individual.

Two problems that exemplify the complexity of these issues are bronchitis in the coal industry and lung cancer in the asbestos industry. Chronic bronchitis is one of the most common disabling diseases in Britain and, though mortality from it has been falling, it still ranks high as a cause of death. Its relation to cigarette smoking is well known,⁴ but it is also related strikingly to the Registrar General's social groupings (which are based on occupation), to dust exposure in industry, and to area of residence,⁵ implying some influence of general atmospheric pollution (at least in the past), overcrowding, and other social factors. Cigarette smoking, with its effects on both smokers and those who surround them, is also strongly related to social class.⁶ In clinical practice patients disabled by chronic bronchitis who have never been smokers are extremely rare. Talk of occupational bronchitis, as though occupation were the sole cause of the potentially disabling or fatal disease in an individual, is misleading. Claims that exposure to dust does not contribute to the disease are, however, equally false, since there is good evidence in the coal industry of relations between measured dust exposure and symptoms, impairment of lung function, and mortality from chronic bronchitis.^{7 8} To disentangle the relative effects of dust and cigarettes in causing the disease is difficult epidemiologically⁹ and impossible in the individual exposed to both; nevertheless, both have played their part—at least in the past. Whether current levels of dust exposure in the coal industry will be sufficient to cause important clinical effects on the airways remains to be seen, but the evidence suggests that this is unlikely.

Similar arguments apply to the relation between exposure to asbestos, cigarettes, and lung cancer. Here the two causal factors seem to act in a multiplicative manner. Exposure to asbestos probably has a linear relation with the risk of developing lung cancer.^{10 11} This risk is very high indeed in the heavily exposed smoker, but the risks are also substantial in a heavily exposed non-smoker. Two forms of action would, therefore, reduce the number of workers at risk of lung cancer: reduction of asbestos levels in the industry and reduction of smoking. Cutting the proportion of smokers would have more effect in workers exposed to asbestos than in the general population.

Dust control has been much improved in both industries in recent years, and further improvements will be made in the near future voluntarily and in response to public pressure or legislation. As well as reducing the risks of the specific pneumoconioses in the workers, this will also reduce the risk of chronic bronchitis in miners and lung cancer in asbestos workers. Yet these latter diseases will continue to be a problem in industry as well as in the general population so long as people continue to smoke. An important reduction in their incidence will occur only when the same public and governmental pressure is exerted on smoking as on dust control. Banning tobacco advertising would be a sensible next step.

¹ Jacobsen M, Rae S, Walton WH, Rogan JM. New dust standards for British coal mines. *Nature* 1970;227:445-7.

² National Coal Board. *Medicine service annual report 1979-80*. London: National Coal Board, 1981.

³ Anonymous. The risks of assessing risks. *Br Med J* 1980;281:1374.

⁴ Royal College of Physicians. *Smoking and health now*. Tunbridge Wells: Pitman Medical, 1971.

- ⁵ Holland WW. Epidemiology of chronic bronchitis. In: Scadding JG, Cumming G, Thurlbeck WM, eds. *Scientific foundations of respiratory medicine*. London: Heinemann, 1981.
- ⁶ Capell PJ. Trends in cigarette smoking in the United Kingdom. *Health Trends* 1978;**10**:49-54.
- ⁷ Rae S, Walker DD, Attfield MD. Chronic bronchitis and dust exposure in British coalminers. In: Walton WH, ed. *Inhaled particles*. 3. Old Woking, Surrey: Unwin, 1971.
- ⁸ Rogan JM, Attfield MD, Jacobsen M, Rae S, Walker DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. *Br J Ind Med* 1973;**30**:217-26.
- ⁹ Jacobsen M. Smoking and disability in miners. *Lancet* 1980;ii:740.
- ¹⁰ Saracci R. Asbestos and lung cancer: an analysis of the epidemiological evidence on the asbestos-smoking interaction. *Int J Cancer* 1977;**20**:323-31.
- ¹¹ Liddell D. Asbestos and public health. *Thorax* 1981;**36**:241-4.

Noise at work

Enormous numbers of people are exposed to potentially damaging levels of noise at work. In Britain, in manufacturing industry alone about 600 000 work in noise levels above an average of 90 decibels*—noisy enough to make shouting necessary for talking to someone standing at arm's length. Over 2½ million more work in levels over 80 decibels.

The Health and Safety Commission has now¹ proposed legislation based on the 1972 voluntary code of practice² and the specific regulations that apply to a few industries. The central provisions are that exposure to noise must be reduced to the lowest level that is "reasonably practicable"—whether by reduction of the actual noise or, failing that, by ear protection—and that no one must be exposed to more than 90 decibels. Is this level too high?

The risks of damage after given exposures are known quite precisely, largely as a result of the work of Burns and Robinson.³ After a lifetime's exposure to 100 decibels, the document states, 32% of people will have a hearing threshold level of 50 decibels or more (averaged over 1, 2, and 3 kHz), and with 90 and 80 decibels the proportions will be 11% and 3%. A hearing threshold of 50 decibels is the point at which the DHSS starts to pay disability benefits; but this is a considerable level of disability. A hearing level of 30 decibels or more represents impairment of the understanding of conversation even in low background noise, and is now recognised as such by the Industrial Injuries Advisory Council.⁴ A careful look at figure 1 in the document shows that on this basis at least 40% of workers exposed to 90 decibels will have some handicap. Not all of this will be due to noise, but the steepness of the curve from 90 down to 80 decibels, at which some 26% are affected, points to the size of the problem. Moreover, some of those in noisy jobs, now more than in the past, have appreciable amounts of noise in their leisure hours. An estimated 10-12% of the large population of disco attenders have noisy jobs⁵; and, though these kinds of pursuits are unlikely to extend over a working lifetime, such combined exposure should not be forgotten.

The Health and Safety Commission says that it selected levels over 90 decibels for mandatory action "to ensure that the greater effort is directed to areas of greatest need." But this would leave unprotected the far greater proportion of workers exposed to noise of 80-90 decibels, as figure 2 in the document shows: the proportions suffering damage are smaller but the absolute numbers could be large. A further point, not dealt with by the commission, is that the damaging effects of noise are not necessarily confined to hearing. Other health effects are less certain⁶; but higher blood pressures and incidences of

*"Decibels" refers throughout to dB (A) over an eight-hour period.

hypertension, for example, have been reported in some though not all industrial studies, and recordings of industrial noise have produced increases in diastolic blood pressure and total peripheral resistance lasting longer than the noise.⁷ Work performance, accident rates, and behaviour may also be adversely affected, though confounding factors are hard to exclude in real-life studies; and Broadbent cites a study by G Jansen suggesting that steelworkers working in noise had more domestic disputes than others.⁸

Reducing noise and even providing personal protection, however, cost money—how much is not clear, but the notes in the background paper accompanying the consultative document suggest large sums.⁹ With limited resources more spent on reducing noise could mean less for other health and safety measures. The commission maintains that few if any workers will be exposed for long to exactly 90 decibels since in practice the design target will need to be two or three decibels lower than the limit. It also emphasises the general requirement to reduce noise as far as is reasonably practicable, and adds that it "will keep the question under review, and will consider whether to propose some lower value if this seems correct in the light of future developments." The present proposal, then, could indeed be seen as "a sensible first step in legislation."

Nevertheless, a general obligation to keep noise levels as low as possible and below the statutory limit would not influence the poorer or less conscientious firms as powerfully as would a lower statutory limit; and once the figure of 90 decibels is enshrined in legislation we cannot realistically expect any early change. The document speaks of the crippling costs to industries if the regulations were very stringent and the likelihood that some would have to close. The social implications of different limits, in terms of direct and indirect costs and benefits, need to be spelt out quite fully in the coming months, so that society can make an informed choice. We need too to hear more of the "strongly held and differing views" about adoption of 90 decibels as the main action level, to which the commission refers so laconically. Its final decision to propose 90 decibels must reflect Britain's present economic circumstances and a substantially lower limit may not be feasible. But would not 85 decibels, which would nearly treble the number of workers protected by legislation, be preferable? At the very least, this could be written into the regulations as a "warning level." At all events, we hope that many of those in the health professions will send their views to the Health and Safety Commission before the end of April 1982.†

¹ Health and Safety Commission. *Protection of hearing at work. Content of proposed regulations and draft approved code of practice and guidance note*. London: HMSO, 1981.

² Department of Employment. *Code of practice for reducing the exposure of employed persons to noise*. London: HMSO, 1972.

³ Burns W, Robinson D. *Hearing and noise in industry*. London: HMSO, 1970.

⁴ Industrial Injuries Advisory Council. *Occupational deafness*. London: HMSO, 1978. (Cmnd 7266.)

⁵ Bickerdike J, Gregory A. *An evaluation of hearing damage risk to attenders at discotheques*. Leeds: School of Constructional Studies, Leeds Polytechnic, 1979 (with Noise Advisory Council memorandum, June 1980).

⁶ Gloag D. *Pollution and people* [two articles on noise]. *Br Med J* 1980;**281**:1325-7 and 1404-6.

⁷ Andrén L, Hansson L, Björkman M, Jonsson A. Noise as a contributory factor in the development of elevated arterial pressure. *Acta Med Scand* 1980;**207**:493-8.

⁸ Broadbent D. Human performance in noise. In: Harris C, ed. *Handbook of noise control*. 2nd ed. New York: McGraw-Hill, 1978:ch 17,1-19.

⁹ Health and Safety Commission. *Some aspects of noise and hearing loss: notes on the problem of noise at work and report of the HSE working group on machinery noise*. London: HMSO, 1981.

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