

consideration of coronary angiography and bypass surgery.

What does all this mean for the general practitioner? From the national figures a group practice with 6000 patients would expect to admit to hospital about 12 patients a year with a myocardial infarction. Three would die there, so 9 would leave hospital. Only one would die in the next year. Over a period of 11 years the practice might, at best, have saved the lives of 3 patients if all the survivors were treated with beta-blockers for the year following hospital discharge.

It is difficult to gauge the current attitudes of general practitioners, though some doubts have been expressed<sup>9</sup>. Amongst British consultant cardiologists there does seem to have been a positive response: a random survey of 100 consultant cardiologists, of whom 83 answered questionnaires, indicated that 60 (72%) used beta-blockers prophylactically. However, half of these only gave them to patients they felt were at 'high risk of death or reinfarction', whilst the remainder prescribed them to all suitable survivors<sup>10</sup>.

In contrast, cessation of smoking following myocardial infarction reduced the prevalence of angina at one year from 32.2% to 19.5% in males under the age of 60 years. Unfortunately after six years this benefit was lost<sup>11</sup>. The effects on prognosis have been more impressive. Over a five-year follow up period, considering the smoking habits of survivors three months after infarction, there was 87% survival (13% mortality) amongst ex-smokers compared with only 72% survival (28% mortality) amongst those who had continued to smoke. For comparison with the foregoing trials of beta-blockers, at 3 years survival was 90% in ex-smokers compared with 85% in smokers<sup>12</sup>.

In conclusion, if beta-blockers were routinely used in England and Wales in the first year after myocardial infarction, 61 700 patients could be treated at a cost of about £4 350 000. About 1900 lives might be saved. This would reduce total mortality from myocardial infarction by less than 2%. For a general practitioner working in a three-partner practice this would mean treating about 33 patients and a life saved every 11 years. Persuading survivors of infarction to stop smoking might well be at least equally effective in the long term, at lower cost and with less side effects.

**R A Greenbaum**

*Cardiology Department*

*Royal Free Hospital and School of Medicine, London NW3*

#### References

- 1 Theroux P, Waters DD, Halphen C, Debaisieux JC, Mizgala HF. Prognostic value of exercise testing soon after myocardial infarction. *N Engl J Med* 1979;**301**:341-5
- 2 Davidson DM, DeBusk RF. Prognostic value of a single exercise test 3 weeks after uncomplicated myocardial infarction. *Circulation* 1980;**61**:236-42
- 3 The Norwegian Multicenter Study Group. Timolol-induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. *N Engl J Med* 1981;**304**:801-7
- 4 Beta-blocker Heart Attack Trial Research Group. A randomized trial of propranolol in patients with acute myocardial infarction. *JAMA* 1982;**247**:1707-14

- 5 Department of Health and Social Security, Office of Population Censuses and Surveys and Welsh Office. *Hospital In-patient Inquiry*. Series MB4 No. 17, 11. London: HMSO, 1981
- 6 Department of Health and Social Security, Office of Population Censuses and Surveys and Welsh Office. *Hospital In-patient Inquiry*. Series MB4 No. 17, Table 11 available on microfiche. London: HMSO, 1981
- 7 Office of Population Censuses and Surveys. *Mortality Statistics cause - England and Wales*. Series DH2 No. 8, 24. London: HMSO, 1981
- 8 *MIMS*. Cardiovascular system. January 1987:45-52
- 9 Colling A. B Blockers after myocardial infarction: have trials changed practice? *Br Med J* 1985;**290**:322
- 10 Bater NS, Julian DG, Lewis JA, Rose G. B Blockers after myocardial infarction: have trials changed practice? *Br Med J* 1984;**289**:1431-2
- 11 Daly LE, Graham IM, Hickey N, Mulcahy R. Does stopping smoking delay onset of angina after infarction? *Br Med J* 1985;**291**:935-7
- 12 Aberg A, Bergstrand R, Johansson S, *et al*. Cessation of smoking after myocardial infarction. Effects on mortality after 10 years. *Br Heart J* 1983;**49**:416-22

---

#### Perlite and other 'nuisance' dusts

---

It is the practice of national and international agencies concerned with the health and safety of workers to classify certain dusts as dangerous and to lay down safety limits for the levels of exposure which are permitted without respiratory protection. Crystalline quartz was one of the first materials to be recognized as dangerous and the internationally agreed limit is 0.1 mg/m<sup>3</sup> for particles less than 5 microns in diameter or 0.3 mg/m<sup>3</sup> for total dust. The limits for asbestos are of course much lower because of the risk of lung cancer and mesothelioma. Other non-fibrous mineral dusts are for the most part classified as 'nuisance' dusts, with limits of 5 mg/m<sup>3</sup> respirable and 10 mg/m<sup>3</sup> total. These limits are regarded by some people as meeting a social acceptability standard rather than a health and safety one. When such a dust contains more than 1% quartz, either because the rock from which it is derived contains quartz as one of its constituents or because the deposit is contaminated with quartz from adjacent strata, then safety standards are laid down using a formula based on the percentage of quartz in the dust.

This policy has led to the concept that nuisance dusts are completely safe unless they contain quartz or one of the fibrous minerals. The public have come to mistrust this sort of official 'line', having been assured of the efficacy of safety regulations regarding materials like asbestos only to be told a few years later that incurable diseases like mesothelioma could still occur at levels of exposure well within the

0141-0768/87/  
070403-02/\$02.00/0  
© 1987  
The Royal  
Society of  
Medicine

previous safety limits. There is, of course, no scientific basis for the concept that 'nuisance' dusts are completely safe or are only dangerous if they contain quartz.

Kaolin is a good example. Treated until now as a 'nuisance' dust, the cases of serious lung disease arising in the Cornish kaolin industry were regarded as due to quartz exposure in certain out-of-date commercial practices. Recent *in vitro* studies indicate that prolonged high-level exposure to kaolin itself could produce lung damage<sup>1</sup>, although early animal work had not produced extensive lung fibrosis<sup>2</sup>. The pathology of the disease in Cornish kaolin workers indicates that silicosis is present in some cases, but that there are also lung fibrosis and massive lesions which are not apparently due to quartz and are probably due to the kaolin (kaolinite)<sup>3</sup>. A similar situation exists in the coal mines in Britain where it is still not clear what components (in addition to quartz) are responsible for the lung damage. Recent studies on gypsum producers in Britain reveal a similar situation but with less serious disease<sup>4</sup>.

The second cause for concern relates to contamination. While the commercial products may be labelled kaolin, bentonite, mica, etc., the deposits from which they are derived contain other minerals and the separation procedures usually leave 5% or more of these in the commercial product. The toxicity tests (*in vitro* and animal studies) are usually done on specially purified material for obvious scientific reasons. Although commercial kaolins now usually contain less than 1% quartz, they may contain between 5% and 10% of other contaminants including feldspar and mica<sup>1</sup>. These are known to be relatively inert. The same cannot be said of fibrous contaminants like tremolite or antigorite. At the VIth International Symposium on Inhaled Particles held in Cambridge in 1985<sup>5</sup> there were several papers and posters on the extent of tremolite/antigorite contamination of Canadian chrysotile mines and the effect this has on the lung fibre burden in the miners, the inhabitants of the mining towns and the users of Canadian chrysotile. The tremolite/antigorite fibres are amphibole and survive indefinitely in the lung tissue whereas chrysotile disappears. The lung burden studies support the view that it is the amphibole fibres like crocidolite amosite and tremolite which are the main cause of the lung fibrosis, lung cancer and mesothelioma in people exposed to asbestos<sup>6</sup>. More worrying for the users of 'nuisance' dusts was the paper given at Cambridge<sup>7</sup> on investigations into the high risk of lung cancer and mesothelioma in workers at a vermiculite plant in Montana and the incidence of mesotheliomas in the neighbourhood. Contamination of this vermiculite deposit by tremolite/antigorite fibres of the sinister size range seems to be the main problem. These amphibole fibres are straight and thin and only a small proportion in the dangerous size range are detected by light microscopy using the methods agreed for monitoring asbestos fibre exposure.

Perlite is a naturally occurring mineral and, like vermiculite, is heat expanded for use in insulation and lightweight plasters and also as a soil improver. Perlite consists of beads of volcanic glass. Some deposits are relatively pure while in others there is a mixture of many volcanic ash components; it can even be compacted into a granite-like rock. On heat

treatment the little beads soften and swell because of retained gas to form sponge-like particles which fragment and yield respirable dust. Although this kind of material will damage macrophages and is likely to cause acute lung damage at overwhelming levels of exposure (such as can occur with a volcanic eruption), the dust particles are unlikely to survive long enough in the lung to cause fibrosis after prolonged exposure at socially acceptable levels. They can be regarded as similar in this respect to other amorphous silicas. There is no evidence that the usual heat treatment causes the formation of cristobalite as happens when kieselgur is calcined.

A recent clinical study of perlite workers in the USA (supported by the industry) has confirmed that prolonged exposure produces little if any X-ray change or loss of lung function<sup>8</sup>. This perlite was extracted from part of the immense band of volcanic ash which lies between the mountain ranges of the western United States. Hydrothermal changes in this ash band, by dissolving the glass and re-crystallizing it with various metals, have yielded a variety of other minerals, including the most dangerous of the mineral fibres so far investigated<sup>6</sup>. This is erionite, which is the cause of so many mesotheliomas in rural areas in Cappadocia. But there is no evidence that the present perlite production involves the disturbance of fibrous silicates. The report does not deal with the mortality patterns of the workers or the people living round the workings, so that we do not know whether a situation exists similar to that which has caused such alarm in Montana. The nature of these geological processes does need to be borne in mind before other volcanic ash deposits are opened up for the extraction of perlite or other materials.

P C Elmes

Formerly Director MRC Pneumoconiosis Unit  
Llandough Hospital, Penarth

#### References

- 1 Davis R, Griffiths DM, Johnson NF, Preece AW, Livingstone DC. The cytotoxicity of kaolin towards macrophages *in vitro*. *Br J Exp Pathol* 1984;**65**: 453-66
- 2 Hale LW, Gough J, King EJ, Nagelschmidt G. Pneumoconiosis of kaolin workers. *Br J Ind Med* 1956;**13**: 251-9
- 3 Wagner JG, Pooley FD, Gibbs A, Lyons J, Sheers G, Moncrieff CB. Inhalation of china stone and china clay dust: Relationship between mineralogy of dust retained in the lungs and pathological changes. *Thorax* 1986; **41**:190-6
- 4 Oakes D, Douglas R, Knight K, Wusteman M, McDonald JC. Respiratory effects of exposure to gypsum dust. *Ann Occup Hyg* 1982;**26**:833-40
- 5 British Occupational Hygiene Society. VIth International Symposium on Inhaled Particles. Cambridge, September 1985. *Ann Occup Hyg* (in press)
- 6 Wagner JC, Pooley FD. Mineral fibres and mesothelioma. *Thorax* 1986;**41**:161-6
- 7 Amandus HE, Wheeler R, Armstrong B, McDonald AC, McDonald JC, Sebastien P. Mortality of vermiculite workers exposed to tremolite. In: *VI International Symposium on Inhaled Particles*. Cambridge: BOHS, 1985: abstract 81-2
- 8 Cooper WC, Sargent EN. Study of chest radiographs and pulmonary ventilatory function in perlite workers. *J Occup Med* 1986;**28**:199-206