Letters to the Editor

On Dying and Dying Well

From Dr Richard Lamerton
Medical Director of the Home Care Service,
St Joseph's Hospice, Hackney, London E8 4SA

Dear Sir, After the rough ride the Press gave the Archbishop of Canterbury, following his Edwin Stevens Lecture (February *Proceedings*, pp 75–81), one does not wish to criticize him further for suggesting that elderly and dying patients were too expensive to look after. From the context of his excellent speech, it is evident that His Grace was really referring to deeply unconscious patients, of any age, maintained on artificial life-support systems.

What one must take issue with, however, is his suggestion that a doctor is responsible to the DHSS; that when faced with a patient he should regard the State as preeminent. Once again the idea of 'Weltschutz' raises its ugly head.

As a citizen, I am responsible to the State; but as a doctor, only to my patient. In an election I may vote for a government which decides to build office blocks for DHAs, AHAs and RHAs instead of hospitals, but in the surgery I may not say 'I won't treat you because the money could be better spent elsewhere.'. I would remind His Grace of the words of a former Archbishop of Canterbury, William Temple:

> 'If each man and woman is a child of God, whom God loves and for whom Christ died, then there is in each a worth absolutely independent of all usefulness to society. The person is primary, not the society; the State exists for the citizen, not the citizen for the State.'

Yours sincerely
RICHARD LAMERTON
2[illegible] February 1977

Asbestos Content of Dust Encountered in Brake Maintenance and Repair

From Dr W J Smither
Chairman, Research Committee of
Asbestosis Research Council

Dear Sir, I am disturbed by the suggestion of Rohl *et al.* in their article 'Asbestos Content of Dust Encountered in Brake Maintenance and Repair' (January *Proceedings*, p 32) that submicroscopic fibres can produce disease. Such short fibres can be engulfed by a single alveolar macrophage, of typical diameter 13 μm, and therefore have a finite chance of clearance via the airways.

A large number of papers have been published on the importance of fibre length on the biological effects of asbestos, and the vast majority suggest that long fibres are much more dangerous than short ones. However, it has not been possible to produce really clearcut results owing to the difficulty of preparing dust samples of different fibre lengths. Almost all publications refer to short fibre samples that have been prepared by prolonged grinding and it has been suggested that this process has so affected the structure of the dust that the biological results obtained are not representative of what really occurs when short fibres are inhaled in a factory. This doubt will remain until some accurately graded samples are available in different lengths, prepared by a process that it is agreed does not change the crystal structure. With such materials one carefully conducted series of experiments would settle the matter.

Detailed studies on the mechanisms by which any asbestos dust is able to stimulate the production of fibrous tissue have involved large numbers of animal experiments. A number of early studies suggested that the most important factor in fibrogenesis was the silica content of asbestos which stimulated collagen production by chemical action (Beger 1934, Kuhn 1941). As early as 1946, however, King *et al.* administered chrysotile fibres, cut on a special microtome at lengths of 15 μm and 2.5 μm, to rabbits by intratracheal injection. They reported a greater tissue reaction from those animals that had received the long fibre sample. Vorwald *et al.* (1951) reported that animals which had inhaled chrysotile fibres in the 20 to 50 μm range had more lung fibrosis than those breathing only dust fibres below 3 μm in length. Szmczykiewicz & Wiecek (1960) obtained similar results when they administered 'fibrous' and 'amorphous' asbestos dust to guinea pigs by intratracheal injection. They did not however give details of the asbestos type employed. In 1968 Klosterkotter extended these studies using both the intratracheal and intraperitoneal injection of chrysotile and crocidolite, ground to an average fibre length of less than 5 μm. They found that these samples produced little or no fibrosis in either site. In contrast, longer fibres of the same asbestos type,

resulted in considerable fibrosis in both regions. Almost identical results were obtained by Hilscher *et al.* (1970) using similar techniques.

The importance of long fibres in the production of fibrosis following inhalation was further emphasized by Timbrell & Skidmore (1968) who exposed rats and guinea pigs to long and short fibres of amosite and obtained a much greater reaction with the long fibre sample. Webster (1970) treated monkeys with a finely ground crocidolite dust, with a fibre length below 5 μm, and obtained only a macrophage reaction in the lungs. Davis (1970, 1972) conducted a series of experiments using the intrapleural injection of a number of different mineral samples including long and short fibre chrysotile. The short fibres samples were either synthetic chrysotile with a maximum crystal length of 1 μm or chrysotile fragmented by ultrasonic treatment until all fibres were below 1 μm in length. While the long fibre samples produced massive fibrosis, the short fibre specimens produced almost no tissue reaction. Similar results were obtained with other mineral fibres indicating that the physical shape of many mineral particles appears more important than their chemistry in determining their pathological potentialities. From these studies it would appear that the length of the asbestos fibres is the main factor in determining fibrosis. Whether the surface chemistry of asbestos and other mineral fibres can exert an additional fibrogenic effect has not yet been determined.

In the field of glass fibre, Wright & Kuschner (1977) compared, by intratracheal injection, two samples. In their short sample, more than 99% of the fibres were less than 5 μm in length while their long sample contained over 80% fibres above 10 μm long. They found more fibrosis with the long fibre sample.

Following the discovery of the importance of fibre length in fibrogenesis this factor was also considered in relation to dust carcinogenicity. Stanton & Wrench (1972) found that partial pulverization of crocidolite to reduce the average fibre length did result in the reduction of carcinogenicity. A few tumours were produced by the pulverized dust samples, but it was pointed out that even after considerable pulverization some long fibres remained. In 1973 Maroudas *et al.* confirmed this association between fibre length and carcinogenicity and suggested that only fibres that were 20 μm or more in length and less than 2.5 μm in diameter were carcinogenic. They suggested that with mineral fibres carcinogenicity depends on the fibres providing anchorage for mesenchymal cells. Smith *et al.* (1972) suggested that hamsters injected intraperitoneally with chrysotile ground to a fibre length less than 1 μm did not develop mesotheliomas while those injected with a long fibre sample of the same dust did. Von Scheuer *et al.* (1973) reported experiments in which a number of mineral samples were injected into the peritoneal cavity of rats. These samples included a standard sample of UICC chrysotile A and another of the same material more finely ground. The tumour incidence produced by the two samples appeared similar but no details were given of the fibre length distribution of the finely ground sample.

Referring to the optical properties of the chrysotile residues found in brake lining dust, whilst I do not disagree with the statements made regarding the loss of birefringence, there is at least one method of dealing with this situation. All such dust, whether from brake drums or manufacturing processes, can be submitted to 3–4 hours low temperature ashing at 100–105°C using monatomic oxygen at 39.6 mHz with pressure of 0.5 mm Hg. This type of incineration removes much of the organic material absorbed into the crystal lattice of the fibre and thus restores its optical activity, at the same time removing the resin particles which may obliterate the fibres.

Yours faithfully

W J SMITHER

22 February 1977

REFERENCES

Beger P J
(1934) *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* 290, 280–353

Davis J M G
(1970 *British Journal of Experimental Pathology* 51, 617
(1972) *British Journal of Experimental Pathology* 53, 190–204

Hilscher W
(1970) *Zusammenhange zwischen Asbestose und Faserlange Naturwissenschaften* 57, 356–357

King E J, Clegg J W & Rae V M
(1946) *Thorax* 1, 188–197

Klosterkotter W
(1968) In: Biologische Wirkungen des Asbestos. Internationale Konferenz, Dresden. Deutches Zentralinstitut für Arbeitsmedizin, Berlin; pp 47–52

Kuhn J
(1941) *Archiv für Gewerbepathologie und Gewerbehygiene* 10, 473–485

Maroudas N G, O'Neill C H & Stanton M F
(1973) *Lancet* i, 807–809

Sczymczykiewicz K & Wiecek E
(1960) In: Proceedings of the 13th International Conference on Occupational Hygeine. New York; pp 801–805

Stanton M F & Wrench C
(1972) *Journal of the National Cancer Institute* 48, 797–822

Timbrell V & Skidmore J W
(1968) In: Biologische Wirkungen des Asbestos. Internationale Konferenz, Dresden. Deutches Zentralinstitut für Arbeitsmedizin, Berlin; pp 52–56

Von Scheuer E, Huth F & Pott F
(1973) *Archiv für Geschwulstforschung* 41, 120–136

Vorwald A J, Durkan T M & Pratt P C
(1951) *Archives of Industrial Hygiene and Occupational Medicine* 3, 1–43

Webster I
(1970) In: Pneumoconiosis: Proceedings of the International Conference, Johannesburg 1969. Ed. H Shapiro. Oxford University Press, London; pp 117–119

Wright G W & Kuschner M
(1977) In: Inhaled Particles Vol 4. Ed. W H Walton. Pergamon Press, Oxford; pp 455–474