test was totally specific. Sensitivity ranged from 64% for teichoic acid antibody to 72% for antibody assays to both peptidoglycan and alpha-toxin. Specificity was 75% for antibodies to both peptidoglycan and teichoic acid and only 50% for alpha-toxin. The data indicate that patients who remain seronegative for all three antigens can be considered free of complicated bacteraemia.

How should these tests be used clinically? The occasional cross-reactions and background antibody activity make their reliability questionable for diagnosis of culture-negative infections. Similarly, the delayed antibody response means that they are scarcely likely to be used in routine diagnosis of *Staph aureus* infections. They appear to be helpful in separating uncomplicated infection (suitable for treatment with short-course antibiotic therapy) from infection causing metastatic sepsis before it becomes clinically apparent and therefore demands more protracted therapy. High titres and positivity in more than one test constitute a pointer to endocarditis and have been shown to guide diagnosis and response.[10] Whether such tests will remain in the province of the reference or research laboratory or become more widely used is uncertain. The necessity to perform duplicate or even triplicate assays raises doubts about the practicability of such investigations. Despite these reservations a commercial assay of teichoic acid antibody activity has recently been marketed in the United States,[20] but again the results demand critical interpretation.[21] It is not unreasonable to anticipate that these arguments may be rendered obsolete by development of staphylococcal antigen detection tests.

PREDICTING THE RISKS FROM ASBESTOS

WHETHER we like it or not, asbestos is with us for the foreseeable future. In Britain, importation of crocidolite (blue) and amosite (brown) asbestos, and of manufactured goods containing these substances, is now prohibited. Nevertheless, the legacy of earlier days remains, and many buildings and industrial plants still contain asbestos in the form of thermal and acoustic insulation. Moreover, chrysotile (white) asbestos continues to find important use in the production of asbestos cement and friction materials such as brake and clutch linings. Our aim must be to minimise the hazards from exposure to asbestos or at least to restrict them to acceptable levels.

Rational decisions in the control of asbestos require an understanding of the relation between patterns of exposure and the risk of disease that ensues—information which comes largely from epidemiological studies. The United Kingdom has stricter regulations than many other Western countries, and there is good reason to believe that the control limits now adopted by the Health and Safety Executive (0·2 fibres/ml for crocidolite and amosite, and 0·5 fibres/ml for chrysotile) will effectively eliminate mortality and serious morbidity from asbestosis.[1] The main worry, therefore, is the risk of cancer, in particular bronchial carcinoma and mesothelioma.

Whilst asbestos has been subject to closer epidemiological scrutiny than any other industrial carcinogen except perhaps ionising radiation, substantial uncertainties remain. The difficulty arises from the many variables which can influence the outcome of exposure—eg, the risk of cancer is likely to vary with age at and time since first exposure, as well as with duration and intensity of exposure. Moreover, there is good evidence that different types of fibre carry different levels of risk, and that disease incidence further depends on the circumstances of exposure. For a given fibre count, risks seem to be higher in the manufacture of asbestos textiles than in asbestos mines or the production of friction materials, possibly because different sizes of fibre are encountered in these situations. Other uncertainties arise because, of necessity, many studies have used only crude estimates of exposure, and where hygiene data have been collected in the past they often do not relate simply to the measurements which are made today with more refined techniques. For bronchial carcinoma, there is the added difficulty of allowing for the possible confounding effects of smoking at different levels of exposure. No direct information is available on the effects of very low intensities of exposure such as are found in buildings with asbestos components, and any conclusions about such exposure can only be drawn by extrapolation.

Knowledge may be limited but decisions have to be made. What should be the control limit for chrysotile in the workplace? Is it safer to remove asbestos from a school building, with a possible hazard to the demolition contractor, or to leave it in place as a source of low-level atmospheric pollution over many years? To help answer these and similar questions, attempts have been made to model the risks from asbestos exposure mathematically. The usual approach is to examine the risks of bronchial carcinoma and mesothelioma separately. The relative risk of bronchial carcinoma is considered as a linear function of cumulative asbestos exposure, independent of age and smoking. Such a model entails several fundamental assumptions, but this type of relation fits adequately with data from follow-up studies of asbestos workers. From results of such investigations it is possible to estimate the gradient of the dose/response relation for different types of asbestos encountered in different situations. The incidence of mesothelioma is usually taken to rise in proportion to a power (between 3 and 4) of the time since first exposure to asbestos. Again, the constant of proportionality can be estimated from the findings of follow-up studies.

Using a model of this type, Hughes and Weill in New Orleans have now calculated that the risk of children exposed for six years to 0·001 mixed fibres/ml in asbestos-containing school buildings is in the order of 5 premature deaths per million,[2] a figure which corresponds to an average annual rate of approximately 0·1 deaths per million exposed. They compare this with estimated annual death rates per million in the United States of 1200 for long-term smoking, 15 for bicycling, and 15 for the inhalation or ingestion of foreign bodies. Three other groups who have explored the hazard of asbestos pollution from buildings have derived similarly low risk estimates.[1,3,4] The accuracy of such predictions has yet to be established, but even if they are out by an order of magnitude, they still help to place things in perspective. On present evidence, there is no reason to believe that asbestos in buildings constitutes a major threat to the public health.

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