GETTING TO GRIPS WITH ASBESTOS

The widespread use of asbestos has magnified its occupational and environmental hazard to health. The risk, formerly confined to workers engaged in its manufacture, now extends to those involved in its many industrial applications and to a lesser extent to the public at large (*Cited in T.C.T.* 1967, 5, 409). Asbestosis, the lung fibrosis caused by inhalation of asbestos particles, has been recognized for 40 yr. A more sinister result of asbestos exposure, which has come to light in the last few years, is the development of mesothelial tumours of the pleura and peritoneum. This sudden discovery called for an immediate assessment of the magnitude of the hazard and although several questions still remain unanswered, measurable progress has been achieved.

Source of carcinogenic hazard

Asbestos is commercially available in various forms, namely chrysotile (white asbestos), crocidolite (blue asbestos), amosite, anthophyllite, tremolite and actinolite. Chrysotile accounts for 82% of the UK market, amosite 13% and crocidolite 5%. According to a UK Advisory Panel (*Problems Arising from the Use of Asbestos: Memorandum of the Senior Medical Inspector's Advisory Panel*, HM Factory Inspectorate, HMSO, London, 1967), epidemiological evidence points to crocidolite as the form mainly responsible for the carcinogenic hazard. However, animal experiments indicate that other forms of asbestos are carcinogenic. Amosite and crocidolite but not chrysotile were found to produce cancer in rats when injected subcutaneously (Roe *et al. Rep. Br. Emp. Cancer Campn* 1966, no. 44, Part II, p. 7), but a positive result with inhaled chrysotile was achieved in rats by Gross *et al.* (*Archs envir. Hlth* 1967, 15, 343). These three forms and anthophyllite have each produced experimental asbestosis in guinea-pigs (Holt *et al. J. Path. Bact.* 1966, 92, 185), but since the relevance of asbestosis in the aetiology of lung tumours is still obscure, this experiment throws no light on the carcinogenic potential of the four forms. As discussed by Hourihane & McCaughey (*Post-grad. med. J.* 1966, 42, 613), mechanical as well as chemical factors contribute to the production of fibrosis and may be involved in the carcinogenic process, and the possible participation of contaminants, e.g. oils, iron, nickel and chromium, cannot be excluded.

Control of use of asbestos

Various authors have emphasized the gravity of the asbestos hazard and the need for stringent controls (Gould, *New Scient.* 1967, 33, 453; Cooper, *Archs envir. Hlth* 1967, 15, 285; *Lancet* 1967, i, 1311; *British Medical Journal* 1967, 3, 62). Gould (*loc. cit.*) puts the problem in perspective by pointing out that a total of only 500 cases of mesothelioma has so far been reported throughout the world compared with 25,000 cases of lung cancer diagnosed each year in the UK alone. The *Lancet* (*loc. cit.*) welcomes the 1967 draft UK regulations, which will eventually supersede those of 1931, but hopes that certain deficiencies will be overcome before the draft becomes law. For example, to exclude completely the emission of asbestos dust from the workshop is not a practical proposition and instead a maximum allowable concentration should be imposed. Unless legislation is enforceable it tends to be neglected, argues the *Lancet* (*loc. cit.*). Meanwhile, the UK Advisory Panel (*loc. cit.*) recommends the substitution of crocidolite by other forms of asbestos wherever possible.

Occupational and environmental exposure

It has now become established that asbestos is an important atmospheric contaminant. More curious is the fact that asbestos finds its way into beer and other drinks as a result of