Case No. 8M [Harold Wood Hospital 55085].—This man died at the age of 47 with 'carcinomatosis abdomini'. He had been exposed to mixed asbestos dusts for 25 years from 1929 to 1954. For the greater part of that time he was most meticulous in the wearing of a respirator in his position as foreman. For the next five years he was foreman of the gardening staff. Pulmonary asbestosis was never diagnosed although he was radiographed annually. His terminal illness began when he presented to one of us six months before death complaining of burning abdominal pain. He was admitted to hospital for investigation. At laparotomy the surgeon reported mucoid looking carcinomatous deposits in the peritoneum, liver, and abdominal organs. There were several pints of viscous free fluid. A biopsy was taken, on which Dr. H. Bennison reported mainly anaplastic growth with occasional capillary and tubular forms. A primary pancreatic growth was thought likely.

The patient died two days later. At necropsy Dr. Bennison found that the coils of the bowel were held together by numerous soft nodules of growth, which were also present on the parietal peritoneum, on the liver surface, in the abdominal nodes, and in the pancreas.

Histological examination of necropsy material confirms pulmonary asbestosis. The bowel is invaded by growth from outside. This shows the same histological structure of clefts in a solid, predominantly fibrous tumour with the capillary and alveolar pseudo-epithelial formation seen in the other cases. There are occasional birefringent particles. Although the tumour does not appear to be highly malignant, it is clearly invading the muscle of the bowel.

Case No. 9M [London Clinic 1961].—This patient died at the age of 57 with ' carcinomatosis peritonei'. He had been exposed to mixed asbestos dust for about 27 years. His exposure was minimal at any one time because his work had always been clerical, supervisory, and technical. Asbestosis was diagnosed in 1948, but he did not wish to apply for certification. The terminal illness began about six months before his final admission to the London Clinic in 1961 because of abdominal pain which radiated to the left flank.

A peritoneal biopsy (2117/61) was reported to show 'an extensive fibrous reaction amongst which there are clefts and acinus-like formations lined by cells with serosal characters'.

At necropsy Dr. F. E. Camps showed a pulmonary embolus, pneumonia, minimal pulmonary asbestosis, a hiatus hernia, and diffuse infiltration of the peritoneum by hard white tissue.

The histological appearances of the necropsy tissue are very similar to those of the first case described, case 6M (London 40336/47), showing both stroma and alveolar formations. The lungs show a moderate degree of asbestosis.

Case No. 10M [London Hospital 27245/56].—This case was fully described by Keal (1960) as his case number 24. He had been exposed in the same factory as the previous cases in this series to mixed asbestos dust for four years from 1931, and less heavily for 17 years from 1937. Pulmonary asbestosis was diagnosed on periodic examination in 1954. Histological examination of material from the London Hospital necropsy, kindly provided by Dr. J. Landells, confirms pulmonary asbestosis and bronchopneumonia. One section shows ileum entirely surrounded by tumour. This is predominantly alveolar with a mono-layer of cuboidal lining cells which are regular in size. Some typical 'adenocarcinomatous' formations are seen, and some dilated alveoli contain papillary processes. There are however some epithelial lined clefts quite unlike carcinoma in their irregular slit-like configuration.

Case No. 11M [Hammersmith Hospital 227252].—This case was fully described by Heard and Williams (1961) as their case number 6. He originally presented to one of us (W.J.S.) because he was concerned over his 'middle-aged spread'. He had had abdominal discomfort and distension for eight months. Dr. B. Heard has kindly provided histological sections from the biopsy (June 18, 1959) and necropsy (December, 1959). Histological examination of the biopsy specimen shows a nodule about 1 cm. across with fronds of very vascular tissue and some small alveolar and solid masses. One nodule contains birefringent needles 5 × 1 microns.

Discussion

In the past five and a half years we have seen 52 cases of asbestosis at necropsy. Nineteen (36%) had carcinoma of the lung, none of which was pleural in origin. Fourteen (27%) had abdominal tumours, nine (17%) of which appeared to originate in the peritoneum. There was a single case of carcinoma of the breast (2%). A possible total of 9,550 people have been occupationally exposed to asbestos at this one factory since 1913. Including two cases, the necropsies of which neither of us attended, this gives an incidence of peritoneal tumour of about 1 in 1,000 of those exposed. It should be noted, however, that as many as 46 years may pass before the onset of the syndrome, and in none of our cases was the interval less than 20 years. A number of cases may have died elsewhere since no special effort has been made to follow up all the people occupationally exposed and the eventual incidence may be higher. It seems clear that this is another clinical condition that must be recognized as part of the asbestosis syndrome, just as carcinoma of the bronchus is now recognized. Furthermore, the condition is associated with industrial exposure though there may not be enough pulmonary fibrosis to qualify for certification.

In previous series (Keal, 1960; Bonser *et al.*, 1955) of somewhat similar cases, the majority of patients were women. Indeed Keal concluded that most of his cases were ovarian carcinoma, as did the pathologists in two of the three female cases reported here, but eight of our 11 cases were men, and we have also