showed very dense subserous fibrosis and lymphocytic infiltration, with ill-defined tubular inclusions lined by polygonal cells that showed no cytological evidence of malignancy (fig. 7). The patient was seen once more in outpatients two months later, when the abdomen was said to be solid with growth. She died at home shortly afterwards and necropsy was not performed.

Case 9.—A 35-year-old woman was admitted to a gynæcological ward with a three-month history of gradual enlargement of the abdomen. No history of exposure to asbestos was recorded in the notes, but her sister was noted to have died from lung cancer secondary to asbestosis. There was dullness to percussion at both lung bases, with diminished breath sounds and coarse crepitations. The abdomen was greatly distended with fluid. Chest X-rays showed loss of translucency over both sides of the diaphragm, and the sputum contained asbestos bodies. At laparotomy, hard nodular swellings were found along the mesenteric border of the small intestine and studded over the omentum. The ovaries, liver, kidneys, appendix, and uterus were normal. The patient died seven weeks after operation.

Necropsy.—A layer of soft white growth covered the visceral peritoneum over the intestines, stomach, and pelvic viscera. There was similar growth over the under surface of the left lobe

Fig. 6—Case 9. Mainly solid large polygonal-cell neoplasm with some papillary and some small-cell areas with cleft formation. A few areas show a transitional pattern. (×110.)

of the diaphragm and over the posterior adbominal wall. The ovaries were normal, but their surfaces were covered with growth. In the chest, there were tough fibrous adhesions between the right dome of the diaphragm and the lung, and two plaques of hyaline fibrosis in the pleura over the left dome of the diaphragm. No growth was found in the hilar lymph-nodes. There was diffuse fibrosis throughout the lower lobes of both lungs, with areas of confluent slaty-black anthracosis. No primary growth was found anywhere. Histological examination showed a neoplasm composed of solid, large polygonal cells with some papillary-cell and small-cell areas, associated with cleft formation. Occasional areas showed a transitional-cell pattern (fig. 8).

Case 24.—Admitted at the age of 50. Three years earlier, a diagnosis of asbestosis had been made. One year before admission, he attended outpatients with an eight-month history of epigastric discomfort, unrelated to meals but worse at night. There were no abnormal signs and a barium-meal examination showed no abnormality. He had some relief from alkalis; but vague abdominal discomfort persisted, and for two months before admission there had been loss of appetite with increasing girth. He was ill and wasted. The percussion note was impaired at both lung bases and there were bilateral basal crepitations. The abdomen was tense with fluid, but, after paracentesis, a large epigastric mass was felt. Chest X-rays showed diffuse reticulation, maximal in the lower zones. The sputum contained asbestos bodies. He died after three months in hospital.

Necropsy.—Firm fibrous thickening of the parietal pleura was present over the posterior chest wall, particularly on the right. There was slight diffuse fibrosis of the visceral pleura throughout, with underlying subacute flecking. The lungs showed slight diffuse induration, with scattered areas of anthracosis. No macroscopic carcinoma was found in the bronchial or hilar lymph-nodes. In the mesentery of the ileum, a nodule of firm fibrous mass was found which separated easily from the posterior abdominal wall. There were numerous nodules of growth in the lesser omentum, and around the pylorus of an otherwise normal stomach. Further nodules and plaques of growth were found throughout the peritoneum, in many places indenting the mucosa of the large and small bowel, but not elsewhere infiltrating the mucosa. On histological examination the tumour shown was composed of small cuboidal cells with papillary, cleft-like, and tubular formation. There were a few areas of larger, less well-differentiated cells, and also some areas of myxomatous degeneration. This neoplasm was present in the serosa of ileum, spleen, and the peritoneal surface of the diaphragm, with one solid trabecula of cells extending through the diaphragm into the hyaline, fibrotic pleural surface. There were small clumps of cells in a cæliac lymph-node, which also contained asbestos bodies. In the lungs, there was moderate peribronchiolar and pleural fibrosis with numerous asbestos bodies. A little bronchiolar epithelial hyperplasia was seen in the right lower lobe.

Discussion

Asbestosis

The first recorded case of pulmonary asbestosis was that of a patient who died in Charing Cross Hospital, London, in 1900. Details were given to the Departmental Committee on Compensation for Industrial Diseases (1907), by Dr. Montague Murray. It was not until 1930, when a further inquiry was held (Merewether 1930a and b; Merewether and Price 1930), that asbestosis was recognised as an industrial disease in this country and measures were taken to prevent it. By the nature of the work involved, these measures were unlikely to be wholly successful, and it is of interest that of the 42 cases in this series, ten (8 men and 2 women) were first exposed after 1930.

Asbestosis and Lung Cancer

Lynch and Smith (1935) reported the first case of lung cancer associated with asbestosis. In 1952, Hueper (1955) was able to collect 61 published examples. Of 99 details by Hueper (1955), 82 originated in England. The first statistical review was that of Merewether (1947), who reported the finding of lung cancer in 13·2% of 23 necropsies on patients with asbestosis. This was compared with an incidence of 1·32% among 6884 cases of silicosis. Wyers (1950) reported an incidence of 14·6% among 11 necropsies, and Gloyne (1951) found lung cancer in 14·1% of deaths associated with asbestosis. Doll (1955) provided further confirmation of a causal relationship with his investigation of necropsy reports on 105 persons who had been employed at one asbestos works. Lung cancer was found in 15 of 75 cases with asbestosis, and in 3 of the 30 cases without asbestosis. He concluded that the risk of lung cancer among men employed for twenty years or more was ten times that of the general population.

Such a direct association is not universally accepted. Jacob and Bohlig (1955), in Dresden, studied 343 cases of asbestosis and found only 4 cases of lung cancer, but 15 with associated active tuberculosis. They state that, during the past twenty years, the average number of asbestos workers throughout Germany has varied between 3000 and 40 but that only 17 cases of lung cancer have been reported among them. Braun and Trian (1956) criticise statistics based on necropsy findings without reference to the pop

risk. In an epidemiological study of over 6000 workers in the asbestos mines of Quebec, they found only 3 proved lung cancers—an incidence of 25·3 per 100,000 of the population studied. Comparing this incidence with that of 22·5 per 100,000 for the rest of the province of Quebec, they conclude that the absestos workers do not show a significantly higher death-rate from lung cancer than do comparable sections of the general population.

The present series is too small for statistical analysis, but it will be seen that the incidence of lung cancer at necropsy is very much higher than any previously reported—particularly among the men, where it is 67%. This is largely explained by selection, since 6 of the 10 men were referred directly to the medical and surgical thoracic units from other hospitals with the diagnosis, either provisional or established, of lung cancer. The same explanation does not hold for the females, who all attended at general medical outpatients, thus giving a lung cancer incidence of 26% of all deaths associated with asbestosis. It will be seen from table III that the average duration of exposure

TABLE III—PERIOD SINCE FIRST EXPOSURE AND DURATION OF EXPOSURE OF 35 CASES OF ASBESTOSIS, ASBESTOSIS WITH LUNG CANCER, AND ASBESTOSIS WITH PERITONEAL CANCER

—	Number of cases		Years since first exposure		Duration of exposure (years)	
	M	F	M	F	M	F
Asbestosis and peritoneal cancer ..	1	8	26	30·7* 21–33†	23	5·8* 1–21†
Asbestosis and lung cancer	10	4	24·6* 5–45†	25* 15–39†	14·3* 2–45†	9·7* 1/12–20†
Asbestosis alone (living)	4	9	30·0* 18–42†	23·5* 18–33†	19·1* 1/12–42†	7·2* 3–17†

* Average. † Range.

of the 4 women with lung cancer was only about nine and three-quarters years whereas the average period from first exposure to death was twenty-five years, suggesting that time of exposure is not the most important factor in the development of lung cancer. Merewether (1947) also noted that the average age of death with asbestosis and carcinoma of lung was about fifty-two years against about fifty-four years for those with asbestosis alone, though there was little difference in the duration of exposure. Hueper (1951) draws the conclusion that many of these patients die from asbestosis before their lung cancers have time to develop.

As in other reported series, there is no great preponderance of any one cell type. Of 15 lung cancers, 5 were squamous, 8 anaplastic, and in 2 others the type was not stated. It is worth noting that the 4 women with lung cancer were all nonsmokers.

Absestosis and Peritoneal Cancer

Leicher (1954) reported the case of a 53-year-old asbestos worker who had suffered from asbestosis complicated by pulmonary tuberculosis for several years, and also died in 1952 from tuberculous meningitis. At necropsy, the peritoneum was found to be studded with nodules of growth and there were plaques of growth over the peritoneal aspect of the diaphragm. This was thought to be a "primary cortical cell tumour of the peritoneum". No typical asbestos bodies or fibres were seen in the tissues of the tumour, but asbestos was demonstrated there by X-ray-diffraction techniques. Bonser et al. (1955) appear to have made the only previous reference to a high incidence of abdominal neoplasms associated with asbestosis. Out of their series of 72 cases, 4 (1 man and 3 women) had peritoneal cancers. Of the 3 women, 2 had been thought to have ovarian primaries (Bonser 1959).

TABLE IV—CAUSES OF DEATH OF 15 MEN AND 15 WOMEN WITH ASBESTOSIS

—	Men	Women
Ovarian or peritoneal cancer ..	1	9 (60%)
Carcinoma of lung	10 (67%)	4 (27%)
Pneumonia	2	..
Other causes	2	2

In the present series, 9 of 15 deaths in women were associated with ovarian or peritoneal cancer, an incidence of 60%. In one case (no. 5) the diagnosis was only clinical, and there was nothing in the history or examination to incriminate any particular organ as the site of the primary lesion. In the remaining 8 cases, either laparotomy or postmortem examination was performed. Only 1 (case 1) had a definite ovarian neoplasm without peritoneal involvement. In every other case there was diffuse peritoneal growth with ascites. In 4, the pelvis contained a mass of growth and the ovaries were not identified. In 2 other cases, the ovaries were said to be studded with growth but were not, apparently, the site of the primary lesion; in 1 case the ovaries were cystic but not involved by growth.

Case 24 was the only man to show a similar picture of diffuse peritoneal cancer. An intensive examination revealed no macroscopic evidence of a primary growth, but subsequent microscopy showed bronchial epithelial hyperplasia in the right lower lobe, and tumour-cells in a hilar lymph-node. In addition, there was a solid trabecula of growth extending through the diaphragm, which may have arisen on either the pleural or the peritoneal aspects. In this case, and in case 9 where plaques of growth were also found on both aspects of the diaphragm, the possibility of a missed bronchial primary is more difficult to refute.

It is possible that these cases do not represent a new pathological entity but are all cases of secondary carcinoma from bronchial primaries. The occurrence of secondary deposits from bronchial carcinoma, many months before there is clinical or radiological evidence of the primary lesion, is familiar to all. Even at necropsy, a small primary growth in the wall of a peripheral bronchus is easily missed unless specifically sought. The absence of clinical or radiological evidence of lung cancer in the 10 cases of peritoneal cancer described does not eliminate this possibility, nor will the negative record of such a primary entirely convince the sceptical. Yet the findings differ from those usually associated with carcinomatosis from lung cancer in which it is common to find deposits in liver, adrenals, and kidneys, but most unusual to see diffuse peritoneal involvement without such deposits. However, Bonser et al. (1955) and Bonser (1959) attach importance to the plaques of hyaloserositis tethering the base of the lung to the upper surface of the diaphragm in many cases of asbestosis, as in case 9 and case 24 of this series. They consider that the consequent alteration of lymphatic drainage of the region offers a means of spread to the abdomen of cancer arising in sequestrated bronchial epithelium. On the other hand, this explanation is difficult to relate to the predominance of women in the present series, since lung cancer is commoner among the men.

2 of Bonser's 3 patients with asbestosis and peritoneal cancer were thought to have primary ovarian growths. Case 1 of this series was undoubtedly ovarian, and, in 4 others, the ovaries were involved in a mass of growth filling the pelvis and may well have been the site of the primary lesion. In all cases, the histological findings have been compatible with ovarian carcinoma and this has been