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ASBESTOSIS AND LUNG CARCINOMA

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All pathological and anatomical studies performed have shown that asbestosis is often accompanied by lung cancer. The following table contains a few such statistics.

TABLE.

author	year	country	autop-sies pert.	lung carcin-oma	%
Nordmann	1938	Germany	12	2	17
Wedler	1943	Germany	29	6	20
Cartier	1955	Canadian asbestos mines	40	6	15
Doll	1955	England	75	15	20
Hueper	1955	worldwide literature	738	114	15.5

A few authors observed considerably higher figures when performing their own autopsies: according to Hueper, O'Donnel observed ten cases of lung carcinoma in Pennsylvania during 21 autopsies (=48%); in 1951, Konig found five cases of lung carcinoma during 16 autopsies (=31%).

The figures are somewhat smaller if we do not limit our statistics to autopsy cases but rather to the medical diagnosis given at the time of death. For instance, the official report from England mentions 31 cases of lung carcinoma out of 235 persons who died of asbestosis (equals 13%) between the years 1927 and 1936, as quoted by Behrens.

In addition to these statistics, the correlation between asbestosis and lung carcinoma is also confirmed by anatomical findings: in asbestosis the carcinoma is most often localized in a lower lobe whereas lung carcinoma generally develops in an upper lobe. Cancer due to asbestos often

takes the form of squamous cell carcinoma, and not adenocarcinoma and non-differentiated small-cell tumors, as is generally the case. As observed by Gloyne, Nordmann, Boemke, Guttner et al., the development of asbestos cancer is often multicentric in the asbestosis tissue and rarely originates from a circumscribed area. The desquamation and epithelial hyperplasia as well as accumulations of giant foreign body cells which often accompany asbestosis are considered as precancerous changes.

The evaluation of animal experimentation performed on mice (Nordmann and Sorge, Gardner, Lynch, McIver and Cain) is made difficult by the high incidence of spontaneous tumors and all researchers found a certain occurrence of lung carcinoma in mice following the administration of asbestos dust, often leading to precancerous changes.

The latency period between the beginning of the exposure to asbestos dust and the time of death due to lung cancer was usually quite long; Nordmann calculated that the average latency period was 18 years, Wedler between 12 and 42.

Although in spite of many differences all individual pathological and anatomical studies do tend to indicate a high incidence of lung carcinoma among persons suffering from asbestosis, the picture obtained from mass studies performed on living humans is much less clear. Out of 333 persons suffering from asbestosis in all stages, including the very early stages, among workers of the Dresden asbestos industry, Jacob and Bohlig found only four cases of lung cancer prior to the year 1955. For the year 1957, they found five cases of lung carcinoma

confirmed by the autopsy out of 517 cases of asbestosis, i.e. approximately one percent of all asbestosis cases. We get similar figures from North America. Braun and Truan observed 5000 employees of the Canadian asbestos industry over a six-year period. During that time, nine died of lung carcinoma, including three where cancer was suspected. Based on this figure, lung cancer among asbestos workers is hardly higher than for the general Canadium population. However, these studies only provide an idea of the incidence of lung carcinoma among the employees of the Canadian asbestos mines and mills where the incidence of asbestosis is fairly low as determined by Cartier. Out of 4000 employees of the Canadian asbestos mines and mills, Cartier found only 128 cases of asbestosis. The figures quoted by Braun and Truan therefore do not allow us to draw conclusions as to the incidence of lung carcinoma among persons suffering from asbestosis.

The considerable difference between the results obtained by pathological anatomists and those of mass studies are often attributed to the fact that the autopsies are often only performed in cases of special interest so that there is a certain imbalance in cases observed. I feel that this is due more to the fact that death of asbestosis patients due to lung cancer usually occurs quite late, in the later stages of asbestosis. Since the number of living asbestosis patients includes several young men, it would seem that the number of cancer cases observed during mass examinations of living humans is considerably lower than the autopsy results. However, given the very large difference between the results of these two

types of statistics, we wonder this is sufficient to explain the situation.

Since the year 1936, I have had an opportunity to periodically examine 250 employees of an asbestos plant at intervals of one to two years (less frequently during wartime) and found 92 cases of asbestosis of which, according to the most recent findings, 32 were at the 0-I stage, 26 were at the I stage, 14 were at the I-II stages or II, 20 were at the II-III stages or III. Of these 92 cases of asbestosis ranging between the early and advanced stages, I only knew of one case of lung carcinoma autopsied by Professor di Biasi of Bochum in 1957. Professor Boemke of Dortmund and Professor di Biasi later reported two additional cases of lung cancer among workers of my observation series autopsied in 1951. During autopsies performed in 1958, Professor Boemke found three additional cases previously examined by me and where asbestosis was accompanied by lung carcinoma. The number of lung cancer cases detected in asbestosis patients of my observation groups between the years 1936 and 1958 thus increased to six. I continued to perform these regular mass examinations until the year 1956, but the total number of cancer cases observed in 1958 was more conclusive. A few additional cases of asbestosis were observed during these two years. I know of this mostly due to my own observations, as well as reports from the National Work Physician, Dr. Buckup, as well as surveys performed in the textile and clothing industries; these are already included in the grand total of 92 asbestosis cases. Of course we can assume that a few early or very mild cases of asbestosis are not included in that figure.

If we assume based on earlier observations that around three to four new, early or very mild cases of asbestosis go undetected each year, the total number of asbestosis cases for the period of 1936 to the end of 1958 would be approximately 100. This would include six cases of lung carcinoma (equals 6%), which is considerably higher than the figure quoted by Bohlig and Jacob.

Thanks to the assistance of Professors di Biasi and Boemke, I was able to report on these cases based on the autopsy findings. The first five cases have not as yet been published in detail.

Case 1. E.V., born 1896. 4-17-36: Employed for period of 15 years as master weaver in an asbestos plant. For two years has been complaining of coughing, expectoration, and shortness of breath. Mild asbestosis roentgenologically detected. Numerous asbestos fibers in expectoration as well as some asbestos particles. Complaints increased in 1940, including crepitation. Asbestosis II. 7-31-41: Increased crepitation. Vital capacity 2000 cm^3. Asbestosis continues to progress and compact shadow areas hardly differentiated from the heart shadows appear on the left which merge above with the enlarged hilus shadows. Asbestos II-III. According to the report of the family physician, increasing stomach discomfort around the beginning of September 1941. 10-8-41: Hospitalization. Loss of weight of 25 lbs., shortness of breath, mild icterus, large smooth tumor on the upper abdomen protruding through the right diaphragm. Bronchitis, cachexia. Roentgenological diagnosis: asbestosis, epigastric carcinoma (pyloris, liver). Patient died on 11-7-41 at the age of 45. The autopsy (Professor di Biasi) was first performed on 4-15-42 at which

time the organs had already begun to putrefy. The lungs and particularly the lower lobes hardened on the whole and covered with grayish-white strands. Pleural growth bilaterally. Grayish-red almond-sized cluster in the center of the left lower lobe around the bronchus. Whitish circumscribed thickening of the wall in the bronchus itself. Lung tissue generally distended. Bronchus lymph nodes enlarged, tough and gray in color. Liver covered with numerous tumor nodes sometimes as large as a plum. No cancerous stomach changes. Minimal enlargement of left cardiac ventricle, greatly enlarged on the right. Blood congestion of spleen and kidneys. Moderate arteriosclerosis, numerous gall stones. Microscopically; small and medium sized connective tissue clusters in the lungs, partly peribronchial and partly arterial, some without connection to vessels and bronchia. Connective tissue thickening of lobular sheath and sometimes of alveolar walls. These changes are moderate in the upper parts of the lungs and more advanced in the lower parts. Numerous asbestos needles and asbestos particles in the connective tissue. Moderate small-cell stromal cancer around the left lower lobe. Small isolated metastases in other parts of the lungs. Pneumonia in both lower lobes. Bifurcating glands extensively affected by cancer. Hepatic nodes found as metastases of bronchial cancer. Presence of severe asbestosis with lung cancer recognized and death considered to be a consequence of the occupational disease.

A cancer disease had therefore been detected here prior to V.'s death, but not the presence of lung cancer. The lung cancer in the asbestosis tissue was so minimal that it could not be detected by the X-rays.

Case 2. O.D., born 2-15-05. 1939: Employed 15 years in the asbestos industry including one and one-half years in the carding plant and later as a foreman. Mild asbestosis without symptoms of insufficiency. In 1940, moist pleural inflammation. 1954, moderate asbestosis, right cardiac overload, bundle branch block on right. 1230 mm sedimentation. Bronchitis and mild insufficiency. 28a recognized with 40% reduction of capacity. 5-7-56: Bronchitis, shortness of breath after stress. EKG unchanged. Increase of asbestosis as shown by X-rays. Diaphragm badly defined bilaterally and hardly mobile. Increased striations and minimal fine-core spotting of upper fields. Middle and particularly upper fields covered with fine spots with tendency towards confluence on both sides of the heart. Hilus shadows expanded bilaterally. Right hilus shadows and horizontal interlobar space extending downward. Emphysema of upper fields. 40% reduction of capacity due to asbestosis. No additional information available after that. D. died on 2-23-58 of "asbestosis with Pleuritis exsudativa." Autopsy performed by Professor Boemke: 4 liters amber-yellow fluid in the thoracic cavity. Right lung extending far towards the left. Left pleura freely mobile, soft and smooth; right pleura firm and grayish-white in color thickened by several small nodes. Cardiac ventricle highly distended and hypertrophic, particularly around the pulmonary section. Mild pulmonary sclerosis. Intermediate lobular spaces grown together. Lungs compactly elastic, surface of cut filled with fields partly covered with a gray reticulum. In the upper part of the lower lobe, marked grayish-white spots disappearing in the adjacent lung tissue. Advanced liver congestion with central lobular

necrosis. Remaining inner organs filled with congested blood. Broad connective tissue strands and reticular areas microscopically detected in both lungs with numerous asbestos particles, partly isolated and partly in the form of dense groups, accumulated between them and sometimes in the bronchia as well. Lung tissue with broad alveoli whose septa are thin and continuous or torn between the connective tissue areas. The larger bronchia show highly distorted openings with connective tissue splitting of their walls by round cell accumulations. Bronchial epithelium desquamated. In right lower lobe, diffusely arranged growth and epithelial cells arranged in compact groups and covering the lung tissue in an infiltrating fashion. Mitoses present in larger epithelial cell accumulations. Such accumulations can also be found in and around the pleura and, to a lesser degree, in the right upper lobe. Diagnosis: asbestosis of both lungs with marked fibrosis. Solid carcinomatous tumors in the right lung. No circumscribed point of origin. Emphysema bilaterally. Chronic bronchitis. Microscopically irregularly arranged carcinomatous formations in right lower lobe and right side of the lungs and pleura. Occasional connection with fibrotic lung sections. In right upper lobe, occasional cancer nests detected microscopically. Circumscribed tuberculosis of a hilar lymph node.

The carcinoma which was more or less limited to severe asbestosis changes of the right upper lobe and the right pleura went undetected while the patient was alive. Perhaps an analysis of the pleural effusion and the expectoration towards the end of the patient's life would have allowed the carcinoma to be detected.

Case 3. J. Sch., born 1-15-06. He was employed since 1924 in the asbestos plant as bricklayer; after 1929 he worked in the plate factory and later as a foreman. In 1936 showed early symptoms of asbestosis, in 1948 mild asbestosis. 4-17-56: lung signs increased bilaterally in the form of a reticulum. Minimal fine-core spotting of middle and upper fields. Hazy area on the left with a few bean-sized spots. Hilus shadows enlarged. Low area of emphysema on both sides. Vital capacity 2200 cm^3. Insufficiency after stress. Medically reported as occupational disease. 10-20-56: examination by work physician (Dr. Buckup). Definite mild asbestosis, 30% capacity. Two general views put at my disposal by Dr. Buckup and taken 8-23-56 and 8-26-57 are basically similar to that taken 4-17-56 (Fig. 1). On an enlargement of the views we can see a definite fine spotting and streaking over all parts of the lungs. There is no circumscribed area of compaction.

At the beginning of March 1958, the patient complained of back pain with radiation in the abdomen and the testicles. There was a sudden loss of weight. 3-15-58: hospitalization in the Dortmund Medical Clinic (Professor Wenderoth). X-rays showed asbestosis of the lungs and areas of density about the size of a small apple (carcinoma) in the middle and lower parts of the lungs on the left. Bone degenerative process in the 11th dorsal vertebra and the second lumbar vertebra as well as in the second rib on the left. Sedimentation reaction 105/135 mm. Subfebrile temperature with asbestos particles in the expectoration (Professor Boemke). Symptoms of congestion around one leg. Sch. died

on 5-21-58. The autopsy was performed by Professor Boemke: severe asbestosis of both lungs with fibrosis and stiffness of lung tissue as well as contraction of the left lung. Adenocarcinomatous growth in both lungs in the form of gland tube-like growths. Lymphangeosis carcinomatosa of both lungs, more marked on the left, and of both the visceral pleura and the pleura bilaterally. 1800 cm^3 hemorrhaging effusion in the left pleural space. Chronic prurulent bronchitis, prurulent bronchiolitis. Pulmonary arteriosclerosis. Contracted left lung highly compact. Whitish strand-like areas on the cut surface and increasingly white clusters towards the hilus. Occasional air-filled areas in the parenchyma. Similar changes on the right though less developed.

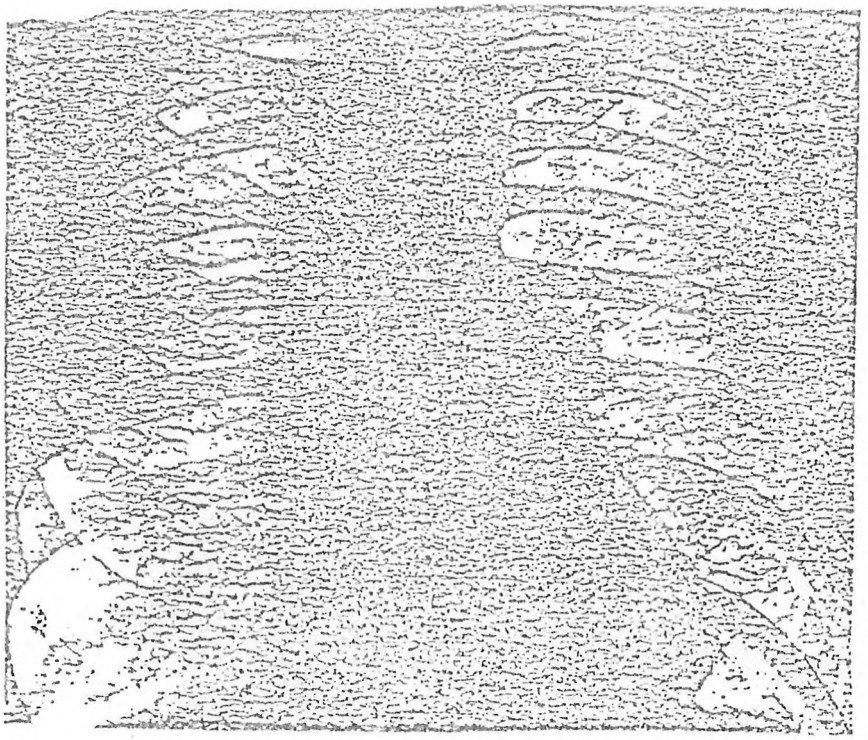

FIGURE 1. [illegible].Sch. 8-20-57 (X ray taken by Dr. Buckup) Clinicall confirmed mild asbestosis. Lung carcinoma not yet to be detected.

Fibrotic areas in both sides of the lung root lymph nodes. Carcinomatous metastases in intrathoracic and intra-abdominal lymph nodes, in the liver, the right kidney, the wall of the urinary bladder, in all vertebrae and left rib with fracture of the latter. Arteriosclerosis, sclerosis of the coronary artery and distension of all cardiac cavities. hydropericardium, thrombosis of the left iliac, femoralis and lower cava. Microscopic fibrous enlargement of the alveolar septa with contraction of the openings. Abundant asbestos particles, sometimes densely grouped, in the septa and the alveoli. Alveolar surfaces partly desquamated. Polynuclear giant cells often present in addition to asbestos particles. Extensive multicentric epithelial cell growth forming irregular gland tube-like accumulations with a polymorphous core appearance and numerous atypical mitoses. Broad connective tissue processes and asbestos particles in the lung root lymph nodes and regular epithelial cell growth. Occupational disease 28b was considered to be the cause of death.

During an examination performed shortly prior to the patient's death in the hospital, a lung tumor about the size of a small apple was roentgenologically detected in the form of a dense cluster, whereas the X-ray taken nine months prior to this had shown no density in that area. The tumor therefore must have developed very rapidly. Numerous metastases also indicated the presence of a malignant tumor.

Case 4. W. L., born 1-31-00. Employed in the asbestos industry since 1927, the first 11 years in the carding department and later as master spinner.

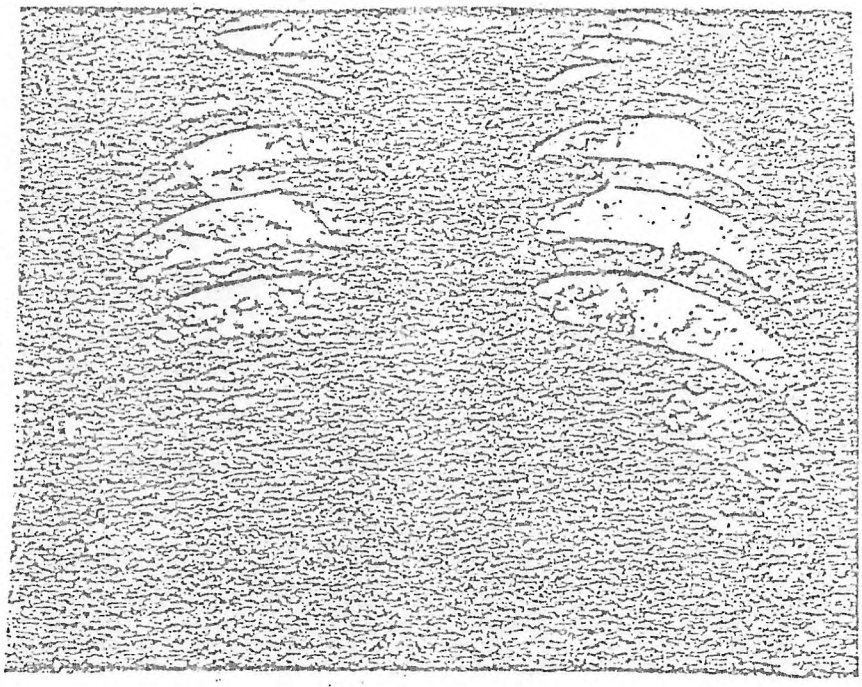

FIGURE 2. W. L. 6-30-58 (X-ray taken by Dr Zernecke) Clinically severe asbestosis. Cancer-like atelectasis in right lower field.

Early stages of asbestosis in 1936, moderate asbestosis in 1940, severe asbestosis in 1950 with related shadows around the heart in the lower field sections. Qualified for compensation with 50% E.M., 1951 60%. 1954, weakness, emaciation, extreme shortness of breath, thoracic stiffness, severe bronchitis. Vital capacity 1050 cm^3. 2-12-55: sedimentation 42/69 mm. The treating physician assumed that there was lung carcinoma in addition to asbestosis. 6-30-58: X-ray taken in the Hutten hospital of Dortmund. Dr. Zernecke was kind enough to provide us with the X-ray pictures taken. The lower field is almost entirely covered with coarse confluent shadow masses which cannot be differentiated on both sides from the diaphragm shadow and on the right from the heart

shadow and disappears upward in the lung root shadow. Atelectasis in right lower field. In the middle and upper fields, upward-decreasing spots and streaks. Emphysema in upper field. Long calcarious processes in both lung fields. Heart appears expanded towards the right (Fig. 2). Cyanosis, shortness of breath at rest, bronchitis. Sedimentation 50/72 mm. EKG: injury to the right side with early stages of intraventricular irritation. P. pulmonale. Patient died on 8-16-58 at the age of 58. Autopsy performed by Professor Boemke: extensive asbestos fibrosis in both lungs increasing downward with partly solid and partly adenomatous carcinomatous growths in both lower lobes. Abundant asbestos particles in both lungs, partly in groups and partly surrounded by giant cells. Chronic substantial emphysema. Numerous bronchiectases, particularly in the lower lobes. Severe chronic prurulent bronchitis. Pulmonary arteriosclerosis. Old superficial growth of pleura together with thoracic wall and diaphragm bilaterally with induration and obliteration. Extensive superficial calcification of pleural cortex. Fibrotic induration of lung root lymph nodes with partly decomposed asbestos particles. Moderate sclerosis of the aorta and its branchings. Sclerosis of the coronary artery, severe hypertrophy of the right cardiac ventricle. Dilatation of the heart. High blood congestion of inner organs. Death due to cardiac arrest promoted by severe asbestosis and lung carcinoma. Microscopic study: diffuse transformation of connective tissue in the lower parts of the lower lobes and the subpleural parts of the upper lobes. Remaining parts of the lungs covered with narrow streaky spots and reticular core-filled connective tissue fiber processes. Alveolar and intralobar

septa as well as part of the perivascular and peribronchial tissue enlarged by the connective tissue. Areas of atelectasis and collapse induration between the connective tissue fiber processes. Emphysema. In the indurated asbestosic parts of the lower lobes, alveols with definite tendency towards proliferation of the epithelium in terms of thickening and cushion or beat-like expansion up to massive filling. Next to this, small isolated accumulations and short solid strands consisting of small round to polygonal epithelial cells with cores of various hyperchromatic shapes as well as atypical adenomatous formations. The glandular tube-like formations covered with atypical cylinder cells.

The carcinoma here was not macroscopically clear during the autopsy. Only the microscopic analysis allowed a definite diagnosis to be determined and showed partly solid and partly adenomatous carcinomatous growths in both lower lobes. No single point of origin of the cancerous growths could be determined and a multicentric development was assumed. No metastases had yet developed.

It was assumed that lung carcinoma was also partly responsible for the patient's death but this could not be radiologically proven. Also to be noted were the radiologically-detected calcium deposits on both sides of the pleura as often observed in asbestosis patients (Jacob and Bohlig).

Case 5. H. H., born 1911. 1928-1940 employed as carder, spinner and weaver in an asbestos plant. 1936, early signs of asbestosis. 1940, mild to moderate asbestosis. No complaints, good performance.

H. retires from the asbestos work as recommended by the physician. 1947-1955, working as a miner. 6-15-55, "moderate pneumoconiosis changes." The fact that he worked as a miner led to suspicion of silicosis. 6-21-55, feverish bronchitis. 6-29-55, severe condition requires hospitalization due to pneumonia of the right upper lobe. Death on 6-30-55.

Autopsy performed by Professor di Biasi: solid superficial pleural growth bilaterally. 200 cm^3 fibrinous-serous effusion on the right. Chronic prurulent bronchitis. Both lungs covered with numerous soft to compact grayish-black stripes and clusters, particularly solid and air-free in the lower lobes (asbestosis suspected). Grayish-white cluster about the size of a cherry in the lower peripheral part of the left upper lobe. Large number of such nodules ranging from the size of a bean to that of an almond in the left lower lobe, more marked anteriorly. Fibrinous lung inflammation of the upper part of the right upper lobe. Isolated grayish-white well defined nodes up to the size of a lentil in the right upper and lower lobes. Lung root lymph nodes soft bilaterally. Both cardiac ventricle and auricle expanded on the right; left auricle expanded to a lesser degree. Hypertrophy of right ventricle. Minimal coronary artery sclerosis. Spleen, liver and kidneys filled with congested blood. Outside the mucous membrane of the cardia in the stomach, bluish-white seemingly firm nodule about the size of a plum grown together with the liver and with a structure similar to fibrosarcoma or spindle cell sarcoma. Two chronic tumors in the pyloris.

The microscopic analysis of the lungs showed diffuse induration of the lung tissue increasing characteristically downward with extensive spreading in the lower lung parts. All parts of the connective tissue covered with asbestos needles and asbestos particles, sometimes including alveoli as well. Occasional severe catarrh-like or fibrinous prurulent pneumonia seats. Isolated hyalin-indurated silicotic nodes. Several parts covered with firm partly-tubular cancer, particularly in the anterior part of the left lower lobe with a probable multicentric origin. The lung root glands showed no silicosis nor cancer but short asbestos needles and isolated long asbestos particles.

Diagnosis: severe asbestosis with right cardiac overload and chronic bronchitis. Lung cancer of probable multicentric origin. The tumor between the stomach and the liver showed no evidence of any correlation with asbestosis. Fibrinous pneumonia. Pneumonia seat.

The cancer was limited to the asbestosis lung tissue and consisted of small clusters. It is therefore understandable that it was not detected even during the last X-ray taken prior to the patient's death. Asbestosis, however, was fully confirmed by the microscopic analysis.

We shall report only a few details concerning a case of lung carcinoma among workers of the asbestos industry previously studied by me. The autopsy results published by Professor Roemke are as follows:

Case 6. A.P., born 5-29-1895. 7-21-44: was employed for a period of 19 years in an asbestos plant, first in the carding department and later as head foreman. Coughing and shortness of breath. No bronchitic sounds. X-rays: mild asbestosis unchanged since 1936. There are no

results available on examinations performed in the later years. From mid-July on, shortness of breath, congestion of the lungs, edema of the feet, discomfort limited to the upper part of the abdomen. 8-4-51: hospitalization due to severe cardiac insufficiency with shortness of breath and edema. Patient died on 8-5-51. Autopsy: severe asbestosis of both lungs with superficial tumors in the pleura and the diaphragm, including several asbestos particles and asbestosis as well as fibrosis of the hilar lymph nodes. Extensive large-cell non-differentiated carcinomatous growth in all lung lobes, particularly in the lower lobes; no circumscribed point of origin. Metastases in both lung root lymph nodes and the suprarenal glands. Emphysema, bronchitis, bronchiolitis, hypertrophy of the right ventricle, dilatation of the heart (particularly on the right), coronary artery sclerosis.

The last thorough examination was performed in 1944, i.e. seven years prior to the patient's death. Carcinoma was first detected during the autopsy which showed that asbestosis had developed extensively during the intermediate years and led to right cardiac overload.

One further case of lung carcinoma in asbestosis reported by Professor Boemke came from the same region as the six previous cases but had not been previously examined by me since he left the asbestos plant in 1923. For that reason, his case is not being studied.

These six cases therefore confirm the known fact that lung carcinoma in asbestosis is generally localized mostly in the lower lobes with a multicentric development in the asbestosis tissue. This had already led

to metastasizing in four of the cases. In two cases the metastases were limited to the pleura and in four cases the carcinomatous tissue could only be observed in the lower lobes highly affected by asbestosis; only the microscopic analysis allowed a definite diagnosis to be reached.

Contrary to literature references, our observations included only one case of squamous cell carcinoma with a larger number of glandular or non-differentiated carcinomas.

Also worthy of note is the known frequent involvement of the pleura in the carcinomatous tumors. In two cases towards the end of the patient's life, there was extensive effusion with small nodular thickening of the pleura.

The age at the time of death ranged from 44 to 58 giving an average of 51. This is somewhat higher than that quoted by Nordmann. Our six fatal cases included none under 44 years of age, whereas half of Nordmann's cases had not reached the age of 41; in Wedler's case this was one-third. I did not observe a higher rate among the younger population as found by Nordmann and Wedler.

The latency period between the beginning of exposure and the time of death ranged between 20 and 34 years, giving an average of 29 years. This is considerably higher than that found earlier (Nordmann: 18 years). The exposure mostly took place at a time when asbestosis had not as yet been detected and proper preventive measures had not yet been developed.

Lung carcinoma often develops inside asbestosis growth and is therefore sometimes difficult to detect radiologically until it begins to grow out of the tumor as seen in Case #3. However, there was no

circumscribed shadow to be observed in that area nine months prior to this. It is therefore evident that carcinoma developed rather rapidly.

It is easy to statistically overlook the presence of a cancer disease if a long period of time elapses between the last examination and the time of death. With the exception of the roentgenological findings, blood in the expectoration, loss of weight and rapidly increasing accelerated sedimentation reaction are often the most evident clinical symptoms of lung carcinoma. Bronchoscopy, bronchography and lung puncture can also be performed if the roentgenological examination is negative but the condition of the patient often makes this impossible. Gloyne, Lynch-Smith, Wedler and Bohne, Alwens, Jacob and Bohlig reported on cases where the carcinoma which accompanied asbestosis was not detected while the patient was still alive.

The lung carcinoma cases studied here only include those with marked or (in 4 cases) severe asbestotic changes confirmed both clinically and anatomically. The seriousness of the changes, in five cases, was emphasized by an anatomically-confirmed right cardiac overload.

The small number of observations which I performed is not sufficient alone to allow a statistical evaluation to be made. The fact that three asbestosis patients died of lung carcinoma in the year 1958 shows how such secondary factors can change the rate. On the whole, however, the findings reported here prove the invalidity of some of the statistics published by Jacob and Bohlig as well as Braun and Truan and show a higher incidence of lung carcinoma in the more advanced stages of asbestosis.

They also show the considerable problems encountered in trying to obtain a complete summary of all cases of lung carcinoma detected during serial examination of asbestos workers.

Anatomical tests are a must if we are to be totally aware of lung cancer cases among our asbestosis patients. A thorough microscopic analysis can also be helpful in determining an accurate diagnosis (see case 4).

A further publication will report statistics based on a larger number of cases.

I am very grateful to Professors di Biasi and Boemke for their extensive support during this study as well as Drs. Zernecke and Buckup, and Professor Wenderoth for providing us with the necessary X-rays and medical records. I also thank the management of the asbestos plant for their great assistance in allowing me to examine their employees.

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