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OCCUPATIONAL CANCER IN ASBESTOS WORKERS*

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*Dedicated to the publisher of this Journal, my admired teacher, Professor Albert Dietrich, on the occasion of his 65th birthday.

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Asbestosis, the occupational disease of the asbestos workers, obviously occurs in countries and cities where asbestos plants were established. Somewhat more and earlier knowledge on this specific disease became available in England and in America than in our country. Nevertheless, Marchand demonstrated an asbestos lung as early as 1906 to the German Pathology Association, admittedly without diagnosing the condition. Eight years later, Fahr showed a second case, the main characteristics of which were by then correctly defined.

In the course of the last ten years, the disease was thoroughly studied, principally in England, America, and Germany. We are acquainted with more extensive serial examinations performed here and in foreign countries in asbestos workers. A case jointly observed by Beger and Stroebe at Hannover in Germany resulted in significant progress, since Beger studied asbestosis thoroughly from the viewpoint of mineralogy. This prompted corresponding studies elsewhere as well (Koppenhofer, Sundius).

Many years of exposure to asbestos dust results clinically in asthmatic complaints and a painful death follows, caused by dextral heart paralysis. In a considerable number of cases, as in silicosis, the pathologic condition occurs jointly with lung tuberculosis (Egbert, Wood), and cases are generally reported in which asbestosis is combined with a lung carcinoma (Wood, Gloyne, 1933).

The overall number of post-mortems is low; however, their number increased quite rapidly during recent years. To the best of my knowledge, nine cases were subjected to autopsy and were published in Germany so far

(Marchand, Fahr, Loeschke, Stroebe-Seger, di Biasi, Wedler, Bohme, and two of my own)(*).

Carcinoma cases are occasionally mentioned in literature; however, merely three publications from American and England deal more thoroughly with the cancer problem in the asbestosis lung. Some of these reports should be regarded with the caution always necessary at the start, when a causative correlation between two diseases is at issue.

The far-reaching significance of the question for our insurance system requires a brief discussion of these studies, before describing two cases which we observed. The compilation of the six cases and of other incidentally mentioned cancers in asbestosis cases is intended to confirm the contention that occupational cancer in asbestos workers does exist.

Case 1. Lynch and Smith [Amer. J. Of Cancer, 24, 56 (1935)]. Lynch refers to a worker, 57 years old, who was employed for 22 years as a weaver in a cotton spinning factory; this was followed by 21 years in an asbestos plant. The X-ray taken shortly before his death coincides exactly with Hornig's representation of our Case 5, with the difference that the deep tumor shade is located in the right lung. The post-mortem revealed lung fibrosis with numerous asbestos fibers and asbestos corpuscles, as well as a cornified malignant squamous epithelioma in the right anterior lobe.

The author classifies the tumor as a Schneeberg lung cancer type or as a type of cancer found in coal dust lungs and other pneumoconiosis cases; he finally compares the same with cancers in workers exposed to

(*) According to Prof. Saupe's report, 3 deaths, u.b. (specifically) without carcinoma, occurred at Dresden within the area to be surveyed.

minerals containing arsenic, in which cases a causative correlation between the occupational disease and the cancer can hardly be questioned. It is certainly true that the fibrosis developed long before the start of carcinogenesis in the lungs; available information on cancer confirms that the cancer is attributable to chronic irritant effects, in this case to bronchial irritation.

Case 2. Gloyne (Tubercle 1935, 5) reports on a woman, 35 years old, who started to work with asbestos 17 years before her death; the occupation lasted for eight years. Accordingly, she had not been exposed to asbestos for nine years prior to her death. The lung asbestosis was of moderate extent. The right superior lobe showed a small, walnut-sized node, microscopically diagnosed as a malignant squamous epithelioma; its ramifications extended to the lung apex and pleura. The neoplasm obviously followed the bronchial ramifications. Giant cells were determinable at the border of the neoplasm.

Case 3. Gloyne (Tubercle 1935,5). The second case described in the same publication deals with a woman, 71 years old, with moderately severe asbestosis, ascites and left vein thrombosis. The left inferior lung lobe showed a neoplasm with numerous necroses and caverns, allegedly similar to those demonstrated in Lynch's case (Case 1). A malignant squamous epithelioma was microscopically verified in this case as well.

In his summary, Gloyne does not seem to be quite certain concerning the primary starting point, such as the bronchus, for example, despite the fact that the cancer proliferated along the bronchial ramifications; simultaneous carcinogenesis could have taken place at numerous sites.

In another case (Case 3), the tumor had obviously developed at the site where the asbestosis was in the most advanced stage. In both cases, the author was under the impression that the tumor was not large enough to cause death; neither did he feel that the patients asbestosis was severe enough to be acceptable as the only cause of death. He believes instead that the two pathologic conditions had a joint lethal effect.

The author raises the question concerning a causative correlation, but refuses to make any attempt in his publication to answer the same.

Case 4. Egbert and Geiger [Amer. Rev. Tbc. 34, 143 (1936)]. A Hungarian male, 41 years old, worked uninterruptedly for 18 years as a weaver in an asbestos factory. A pneumonia which persisted for eight days was followed by coughing and shortness of breath. The sanitary conditions in the factory where he worked were improved to some extent only two years prior to his death. He died within eight months with the symptoms of left superior lobe cancer of the lung. In this case, bronchial carcinoma metastases were found in various organs, in addition to asbestosis; adenocarninoma was microscopically diagnosed. The authors believe that asbestos possibly caused the lung carcinomas.

Within brief intervals, two cases were under observation in our Institute; both patients were workers with asbestosis and lung carcinomas.

Case 5. Our own findings. A woman, 35 years old, worked for a total period of exactly seven years, from her 17th to her 26th year of life, in an asbestos factory, with an interruption lasting for 1-1/2 years. This included two years in the asbestos cardroom; the rest of the time was spent in the spinning and weaving workshops. After the

above period, she never returned to the factory; her domicle was at a considerable distance from the plant.

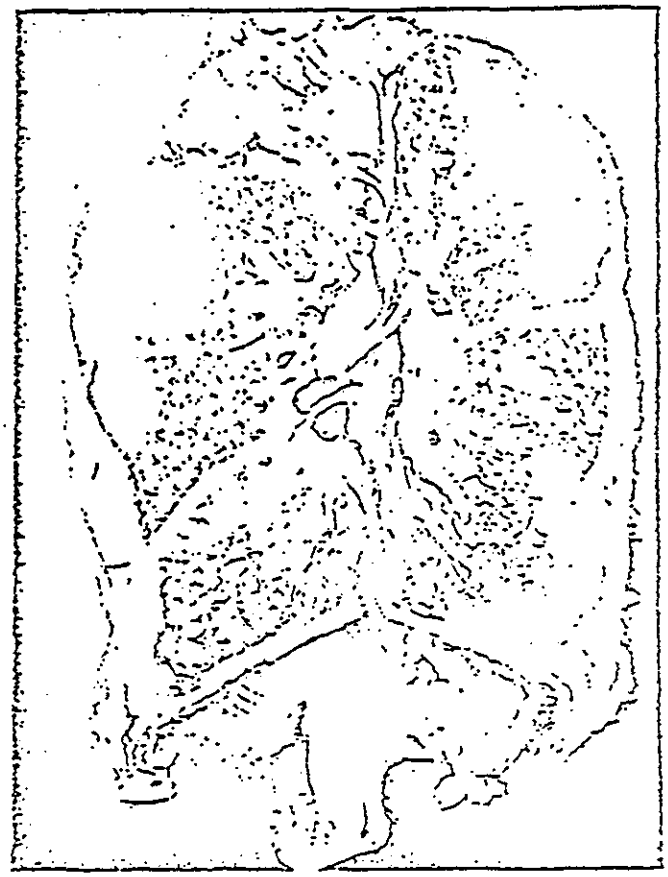
Her father's death was caused by cancer of the esophagus; her mother died after a miscarriage. Her brother and sisters were in good health, with one exception: a sister was affected by tuberculosis. Five brothers died in the war; two died in infancy. The patient was affected by childhood diseases only. After three miscarriages, her marriage remained without issue. The Wassermann blood test was negative. While employed, the patient had a bronchial catarrh, but never asked for sick leave. Four to five years before her death, she started to cough and shortness of breath developed later; this was especially severe in the spring and the fall. Three quarters of a year prior to her death, she received medical attention; subfebrile temperatures, pleuritic grating (sounds) and sluggishness of the sinistral respiration was found. The X-ray taken at that time revealed a curious shadow in the left inferior lung region, extending in the shape of a wedge from the hilus to the back. While hospitalized (see Honig's report on this case), the previous diagnosis of tuberculosis was corrected and asbestosis was diagnosed instead. The large, by then extremely dark shadow in the region of the left inferior lobe led to the assumption that a tumor, originating from the asbestosis, had developed.

The cadaver was pale and showed poor nutritional condition. The lungs as well as the superior abdominal organs showed coalescent surfaces. The lungs were inflated; the section surfaces were reddish-brown, smooth and filled with a foaming fluid containing large quantities of asbestos corpuscles. The lung section had a net-like pattern, with

brick-red, rough webbing. The left inferior lobe was very hard, brick-red or white, a walnut-sized cavern with jagged border extended from the principal bronchus and was obviously connected with the latter. Skeletized vessels and bronchi infiltrated the cavern, recalling the pattern of a buttress system.

The more remote area surrounding the cavern was rough; the pleura above the same showed nodular thickening to 10 mm; the section surface was dry, partitioned into fields and white (see Figure 1).

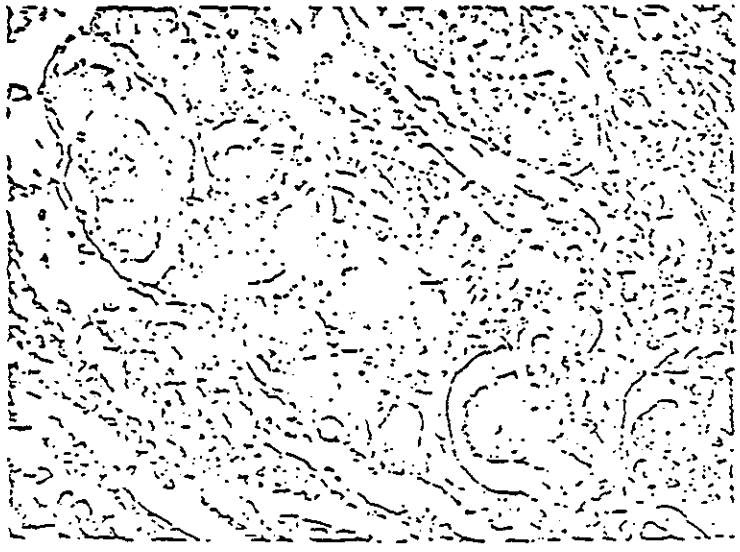
Figure 1. Left lung, in Case 5 (my post-morten report, page 1247/37). Malignant squamous epithelioma of the left inferior lobe and pleura, with central decomposition cavern. Lung asbestosis.



Metastases were detected in the kidneys, partially in the shape of full nodes, partially in the shape of a rough, white network.

The cancer-free sections of the lung showed the known asbestosis pattern under the microscope. It should be pointed out in this context that the cornifications caused by asbestosis were partially quite significant, much in excess of the condition defined as diffuse lung fibrosis, as known in cases of lung congestion. The large number of giant cells surrounding the asbestos corpuscles was especially noteworthy, also as compared with the available data on cases reported by Fahr, di Biasi, Loeschke and Stroebe-Beger. The tumor itself was a cornified, malignant squamous epithelioma; its epithelial cones and stroma contained entire bunches of asbestos fibers and asbestos corpuscles (Figure 2). The metastases showed the same structure, but without asbestos.

Figure 2. Left lung in Case 5 (my post-mortem report, page 1247/37). Malignant squamous epithelioma with asbestos corpuscles.



Case 6. Male, 55 years old. He was employed from his 36th to his 43rd year of life in the workshop of an asbestos factory, where the substance was prepared for spinning. He worked there for seven years; eight months of unemployment followed, during which he was ill with pneumonia. He was then active in a wool laundry up to four months before he died. The patient was enlisted for military duty and served during the three war years only. Previous diseases: diphtheria, scarlet fever and malaria. His weight never exceeded 65 kg. He complained about dryness in the throat, which had persisted since approximately 1919. He was later treated for a bronchial catarrh and acidosis. Considerable weight loss occurred three months before death; and his sputum was bloody. Tuberculosis was suspected. The patient discontinued his work four months before he died. Shortness of breath and coughing persisted. The respiration showed sinistral sluggishness. Breathing was subdued and weaker in that region. The sputum contained masses of asbestos corpuscles.

Figure 3.

X-ray, Case 6 (2 months before death). Deep shading of the left inferior region.

The X-ray showed a deep shade in the left inferior lobe region (Figure 3). The attending physicians, Drs. Schulte and Reichelt (Hannover) diagnosed--on the basis of these symptoms--lung asbestosis and carcinoma in the left inferior lobe, with metastasis into the diaphragm, resulting from the radiologically determinable narrowing of the esophagus. The post-mortem revealed significant adhesions in the lung, with expanded alveoli and a finely articulated, steel-gray, rough pattern. The color of the section surface from the rest of the area was reddish-brown. A dotted black and white tumor was detected in the region of the inferior lobe; the tumor had infiltrated nearby organs such as: the pericardium, the left chamber of the heart, the diaphragm, the peritoneum in the left superior abdominal area, the retroperitoneal tissues in the rear of the spleen, the lymph node group at that location, the inferior thoracic and superior lumbar sectors of the spinal column (Figure 4). A walnut-sized decomposition cavern with inflamed borders was found in the center of the tumor, forming an extension of the primary bronchus.

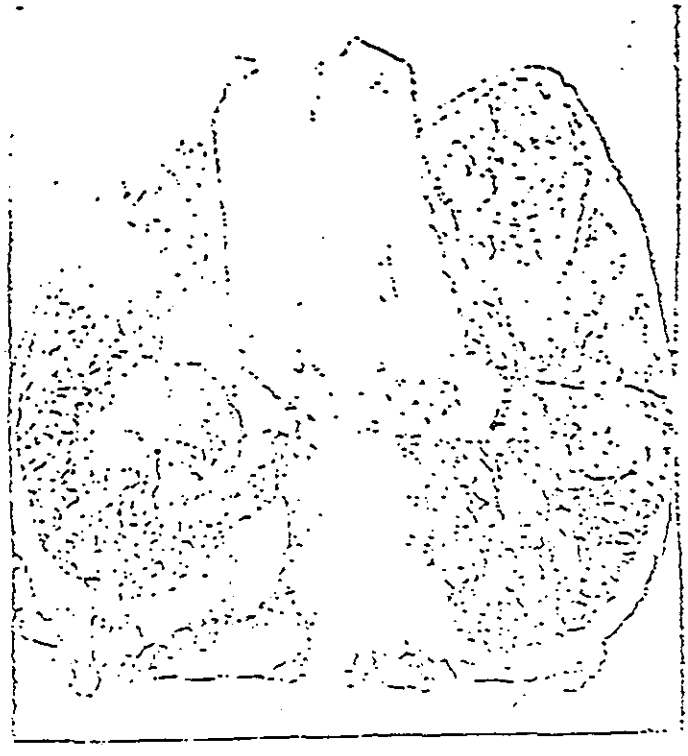
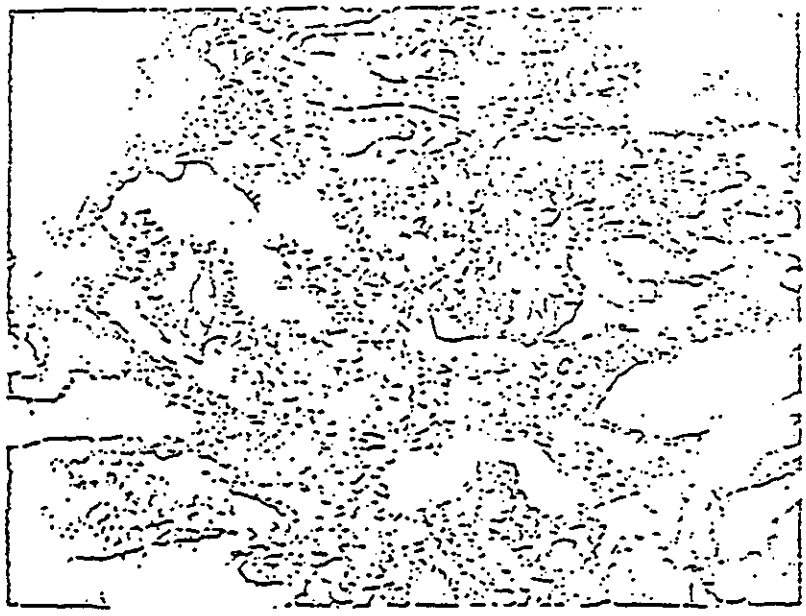


Figure 4.

Left lung, Case 6 (Post-mortem report, page 1335/37). Malignant squamous epithelioma of the left inferior lobe and of the pleura, with central decomposition cavern. Lung asbestosis.

Figure 5. Atypical bronchial epithelium growth from the right anterior lobe in Case 6. (Post-mortem report, page 1335/37).



The microscopic examination of cancer-free sections showed asbestosis; its severity and extent was comparable to findings in cases when the lethal outcome was not a sequela of complication. The left inferior lobe contained a significantly decomposed, cornified, malignant squamous epithelioma, in which asbestos fibers and asbestos corpuscles were determinable. The right anterior lobe showed an area with considerable hardening (Figure 5). The microscopic examination revealed significant bronchial epithelium growth, which infiltrated delimited areas in the form of a true carcinoma. The marcrophage and giant cell count in the lungs, within the area of the cornifications and in the alveolar spaces, was as high as in Case 5.

We compiled the cited cases as well as those observed by us, diagnosed as asbestosis and carcinoma cases, on the following table.

Table 1. The occupational diseases of asbestos workers.

No.	Year	Author	Age (years) Sex	Start of asbestos work	Duration of work with asbestos	Pathologic-anatomic diagnosis
1	1935	Lynch and Smith, America	57 Male	21 years ago	21 years	Malignant squamous epithelioma, right anterior lung lobe
2	1935	Gloyne, England	35 Female	17 years ago	8 years	Malignant squamous epithelioma, right superior lung lobe
3	1935	Gloyne, England	71 Female	15 years ago	1 year 7 months	Malignant squamous epithelioma, right anterior lung lobe
4	1936	Egbert, and Geiger, America	41 Male	18 years ago	18 years	Adenocarcinoma, bronchus, left inferior lung lobe, with numerous metastases
5	1938	Nordmann (Hornig)	35 Female	18 years ago	7 years	Malignant squamous epithelioma, left inferior lung lobe (as in Case 1) with metastases in the liver and kidneys
6	1938	Nordmann	55 Male	18 years ago	7 years	Malignant squamous epithelioma, left inferior lung lobe (as in Case 1) with infiltration into the cardiac muscle.

Germany: 12 sections, including 2 carcinomas . . . . . approx. 17%

England (Wood): 12 cases of death, including 2 (3?) with carcinomas . . . . . approx. 20%

As the data show, lung cancer occurred in numerous cases jointly with asbestosis. This could be accidental (Gloyne, Wood). However, the six cases subjected to intensive observation show several identical characteristics. Accordingly, a cause-effect correlation is presumably present between the asbestosis lung and the occupational disease in question.

The small number of cases is explained as follows: the number of workers exposed to the risk in asbestos plants is significantly lower than the overall number of persons active in such plants. For example: the estimated number of persons exposed to the asbestos risk in Germany merely amounts to a few thousand. Moreover, the work force includes numerous women who, jointly with some men, are frequently employed by the plants for a brief period only. According to British, American, and German authors, noticeable symptoms or objective signs of dust inhalation become evident after several years of exposure. Therefore, no large number of case histories related to our problem was available.

Also, attention focused on the occupational hazard of asbestosis only a short while ago. The bulk of scientific studies completed in England originates from the years 1927/29 (see Literature). Apart from the earlier work of Marchand (1906) and Fahr (1914), lung asbestosis was more thoroughly discussed in Germany starting from 1931/32 only; the same applies to America. Our Table definitely shows that the publications dealing with our problem in particular originate from the last three years. As a matter of course, the decisive evaluation of a new pathologic pattern is undertaken when the basic characteristics of the disease at issue are already known.

Since the available statistical material is limited, the question whether the cancer of asbestos workers is an occupational disease can be settled merely on the basis of cases with lethal outcome. In Germany, 12 post-mortems, including two cases of cancer, were performed, i.e., 17% of the cases showed cancer. In three cases, the post-mortem took place at Hannover; two showed cancer. The prognosis of another patient (the fourth case) is unfavorable. The latter is treated by occupational disease specialists; it is believed that no carcinoma is present. Accordingly, carcinomas were found in 50% of the cases. The number of cases subjected to post-mortems in Germany is probably much higher; many of them were not reported. No survey of the 100 cases observed by Wood in England is available. In 30% of the latter asbestosis combined with tuberculosis, was diagnosed; the diagnosis of 17 cases has not yet been confirmed; in the remaining 53 cases, lung asbestosis was definitely diagnosed. Twelve patients died, two of them had lung cancer and one had "a few deposits of growth in the pleura." Disregarding the last-mentioned, questionable case, two out of twelve asbestosis patients, i.e., 17%, had cancer. The small number of cancer cases should not be used as an argument against a causative correlation; it corresponds to the present status of the problem's scientific study. The percentages mentioned by me, calculated according to a small number of cases, do not claim to represent the actual percentage; they are, nevertheless, definitely useful for the further study of the problem at issue.

We trust that efforts made by factories and occupational disease specialists to improve sanitary conditions in plants will prevent any rapid increase of cases under observation by eliminating the risk demonstrated by us here.

The next objection raised against the cause-effect correlation is the generally large number of lung carcinoma cases, and, in particular, its incidence in those of the 50 to 70 age groups. The Table indicates in this context that one-half of the cancer patients were young; two were 35 years old and one man was 41 years old. The fact that a regular period of 15 to 21 years passed in all cancer cases from the start of asbestos work until death occurred is even more significant than the high incidence of cancer in young patients. The average duration of the period is 18 years—a fact which is correct in half of the cases. The oldest patient in the series survived for the briefest period, i.e., for 15 years. However, older workers became exposed to asbestos much later in life. Therefore, the age when an old patient dies does not indicate a specific age-related susceptibility to cancer; instead, it seems to be a function of carcinogenesis in asbestosis cases. Four out of six cancer patients were exposed to asbestos dust during part of said period and only in its initial stage. They had inhaled asbestos into their lungs; carcinogenesis was not inhibited by the fact that these persons later avoided exposure to dust inhalation. The periods of work lasted for at least seven to eight years; a woman, 71 years old, worked for 1-1/2 years only. She, nevertheless, developed severe asbestosis. The investigation of the sputum while the patient was alive as well as the anatomic examination of the lung definitely proved that leaving the plant does not mean that asbestos is eliminated from the lung; nor does it prevent the development of lung fibrosis and cardiac muscle hyperplasia with corresponding, even if possibly tolerable, symptoms. The

above pathologic pattern strikingly recalls the development of experimental tar cancer: as known, the period following the discontinuation of cutaneous tar application can no longer prevent the development of the carcinoma.

We, therefore, believe that the small number of findings is amply compensated by the regularity of the phenomena revealed by them. Independently from the patient's age, cancer occurs in young persons with lung asbestosis after approximately 18 years, whenever the patient in question worked in asbestos dust for several years.

A comparison of the cases reveals additional coincidences. The cancer localized in the lung is definitely a so-called primary cancer. Moreover, a malignant squamous epithelioma was found in 5/6 of the cases, and in 5/6 of the cases said carcinoma was localized in the inferior lobe. As known, lung cancer usually develops in the bronchial mucous membranes as adenocarcinoma or a small-cell, rather indifferent tumor.

From the viewpoint of incidence, the malignant squamous epithelioma shows the third highest incidence.

In most cases, the real causes of lung carcinomas are unknown. However, scars and bronchiectasis neoplasms could be precursors of lung carcinomas. In such cases, a slow restructuring of the lung tissues is believed to precede carcinogenesis, since numerous cancers develop at the sites where prolonged tissue regeneration took place. Gloyne defines the cancers diagnosed by him as small; he contends that vital tissues were never excessively involved. He is, nevertheless, unable to exclude the possibility that lung tumors could be of multiple origin. A confirmed

primary adenocarcinoma was present only in the case reported by Egbert and Geiger. As the figures show, it is mainly localized in the bronchus, in the shape of a round node. On the other hand, Lynch and Smith diagnosed the tumor detected by them at first as a case of tuberculosis. We believe to have detected the smallest tumor in the right anterior lobe of our Case 2. The cancerous bronchial epithelium growth in the non-cornified layers of the epithelial cells (Figure 5) showed a regular development pattern within a larger area, and was connected with bronchial epithelium ramifications, also known to occur in cases of lung necrosis. We, therefore, believe that the multiple, metaplastic squamous epithelioma of the bronchial system is characteristic for asbestosis; another known form, the adenocarcinoma, occurs in exceptional cases only and, therefore, confirms the rule. Findings and diagnosis are similar in case of cancer originating from cirrhosis of the liver, a phenomenon which is no longer doubted by anyone.

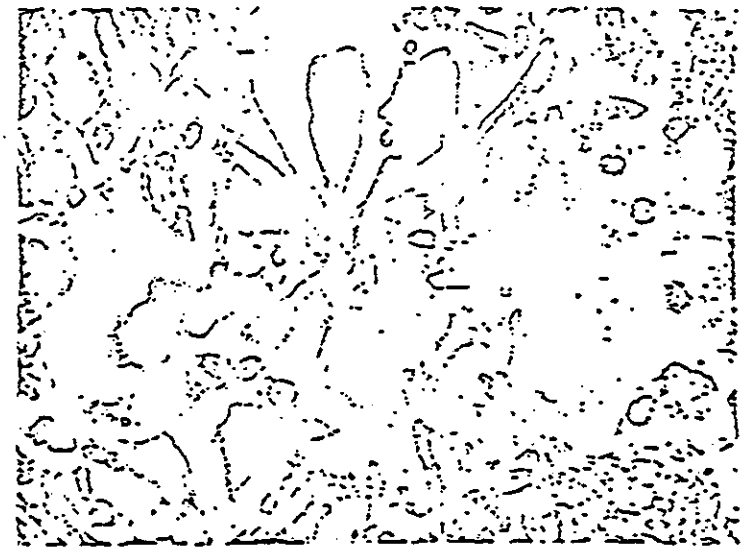
The objection could be voiced in this context that tissue restructuring in the lung is by no means sufficient for cancer etiology, since it would presumably result in a much higher lung cancer incidence, for example: in cases of silicosis or tuberculosis. It seems, therefore, that lung asbestosis has specific characteristics. In our opinion, such characteristics are, in fact, present.

Tuberculosis and silicosis show a certain similarity, due to the restructuring of the lung which occurs in these diseases. Finally, the lung tissues are substituted by a cell-rich and later connective tissue-rich scar in both diseases. These modifications involve massive accumulations of tubercle bacilli or mineral dust particles at certain locations.

It is correct to assume that these accumulations of particles occur through the lymphatic system. Accordingly, practically intact tissues, besides decomposing sectors, are found in silicosis and tuberculosis.

Figure 6.

Giant cells surrounding asbestos corpuscles in Case 6 (Post-mortem report, page 1335/37), right lung.



At variance from the above, lung alterations in asbestosis cases were initially defined as diffuse fibrosis. Our own findings in asbestosis cases coincide with the above. We fully agree with di Baisi who states that larger scars in asbestosis are not unusual; they were present in our cases as well. The diffuse fibrosis of the lung in asbestosis is not similar to the regular fibrosis in the congested lung. The term "diffuse fibrosis" is probably acceptable because the nodules and nodes characteristic for common fibrosis are not present in asbestosis cases. These nodules have a fibrous, concentrically formed nucleus; frequently more recent granulation tissues adhere concentrically to the same. The scars in asbestosis, on the other hand, are triangular; all consist of fibers and cells.

Due to the shape and size of the asbestos fibers, accumulations of the same occur in asbestosis as well. Only minor quantities reach the

lymph nodes of the mediastinum. Our careful investigations revealed no asbestos corpuscles or fibers, except in the lymph node groups. Accordingly, the inhaled asbestos needles were distributed in the entire organism through the lymphatic system. The above becomes even more obvious when not only the asbestos corpuscles, but also the large quantities of asbestos fibers are taken into consideration.

The diffuse distribution of the asbestos fibers, therefore, presupposes a likewise diffuse, chronic irritation, which triggers the proliferation of the lung tissues. According to Beger's investigations at Hannover, a silicate is allegedly at issue here which is released when the asbestos fibers are dissolved. The morphologue di Biasi adopts the idea, at least insofar as the pointed asbestos fibers cause mechanical irritation. Morphologically, the fiber-rich connective tissue proliferates; a series of cellular elements is involved as well, such as the formation of foreign body giant cells, as well as cell proliferations directly attributable to the asbestos fibers and asbestos corpuscles. The histologic preparations in our two cases seem to indicate especially extensive proliferations of such giant cells, determinable in the entire lung. Macrophage groups and other granulations were present jointly with the giant cells in the area of the cornification. Lymphocytes and plasma cell infiltrations were likewise present.

Accordingly, asbestosis goes hand in hand with growth and restructuring phenomena, regularly distributed over the entire lung. This growth stimulus persists--as evident from the above statistical evaluations--even when asbestos dust is no longer introduced, due to a work assignment change.

Under such preconditions, a diffuse cancerous growth could be expected. In our case, in Case 1 (Lynch and Smith) and in Case 3 (Gloyne), which show similarities according to the figures and descriptions, the growth of the malignant squamous epithelioma is macroscopically much more uniform in all directions, in particular within the pleura, than the growth of a nodular bronchial carcinoma, a representative of which is listed on our Table as well. Therefore, the diffuse growth stimulus involving the entire lung in asbestosis results in a subsequent diffuse proliferation of the carcinoma. According to other opinions, the malignant squamous lung epithelioma originates directly from the alveolar epithelium. The following question will be discussed later: how far does the growth potential of the covering bronchial epithelium approximate the growth of the scirrhous organs in the alveolar epithelium during proliferation? If we adopt the aforemention line of thought, the conclusion is reached that an immediate link exists between the carcinoma and the alveolar epithelia, proliferating in the form or macrophages and giant cells. No additional conclusions can be reached on the basis of the diagnoses with modern morphologic methods in favor or a cause-effect correlation between asbestos and cancer.

No complete solution of the cancer problem can result from the first report on an occupational cancer; the report serves only as another example. We can merely stipulate that no inconsistency exists between these findings and the laws of carcinogenesis, as known today. Findings in cases of similar occupational cancers agree with our results. Malignant squamous epitheliomas are quite frequently detected in such

cancers as well. The occupational disease at issue is statistically far more important than other coniosis cases, due to the high incidence of death caused by lung cancer. This prompts us to conclude that the CHEMICAL nature of the minerals is the most significant and decisive factor in lung cancer. The effect of arsenic and radioactivity in minerals was considered earlier as a contributing factor in such cases. The possibility exists that asbestos likewise contains so-called carcinogenic substances.

We summarize below the results of our investigations, obtained on the basis of our statistical data:

In lung asbestosis, cancer develops regardless of age, i.e., in young persons as well, after approximately 18 years, affecting workers who were exposed to asbestos dust for several years. In the cases known so far, the cancer was detected in most patients (5/6 of all cases) in the inferior lung lobe; it was diagnosed as a malignant squamous epithelioma with cornification. Lungs exposed for man years to the irritant effect of asbestos fibers show the morphologic symptoms of a growth stimulus, in the form of diffuse fibrosis, with desquamative alveolar epithelium and with foreign body cells around the asbestos corpuscles. The above restructuring of the lung tissue provides a pathogenetically well-known basis for cancer. It is not known so far which properties of asbestos are responsible in final analysis for the development of the neoplasm.

Evidence that a cancer of this type could actually exist according to facts known today is not the principal recondition for including the

cancer in question amont the occupational diseases of asbestos workers; instead, evidence is needed to show that the incidence of the cancer in its characteristic form is sufficiently high to warrant its qualification as such. I would have concluded that a cause-effect correlation must be present here, even if I had been the only observer; with reference to earlier findings, I am able to confirm that we are indeed facing an occupational cancer which affects asbestos workers.

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Anaesthetized cats were given intratracheal insufflations of fine bismuth carbonate, bismuth subnitrate and lead glass powders by means of a powder blower inserted through a bronchoscope. Fluoroscopy and roentgenograms demonstrated that in the dry state these radio-opaque minerals were not carried into the terminal air spaces but remained adherent to the walls of bronchi lined with ciliated epithelium. The process of removal could be readily followed. The column of dust on the mucosal surface moved upward with a spiral motion. Neutral bismuth carbonate was usually completely eliminated in 1 to 4 days, the acid subnitrate was excreted slightly more rapidly and the lead glass had disappeared in from 7 hrs. to 2 days. The reaction of the mineral did not affect the rate of elimination, a result at variance with those of other observers. If the particles were suspended in fluid either saline or thin syrup, they were carried into the terminal alveoli from which they were slowly excreted by the bronchial route over periods of weeks. No evidence of drainage to lymph nodes could be discovered. The authors suggest that inhalation of dust in the dry state might be less dangerous than if it were moist. The presence of sufficient amounts of water might permit the mineral particles to penetrate beyond the protective ciliary epithelium and allow gradual accumulation in the lungs.—*L. U. Gardner.*

OCCUPATIONAL CANCER IN ASBESTOS WORKERS. *M. Nordmann. Ztschr. f. Krebsforsch., vol. 47, pp. 288-302 (1938).*

The author reports on 2 cases of lung cancer of which he made the pathological-anatomical examination. He reports likewise on the clinical aspects of Hornig's case (see following abstract) and on a 55 yr. old man who had worked in asbestos from his 26th to 43rd years. Autopsy showed asbestosis and in the left upper lobe a markedly necrotic, cornified squamous celled epithelioma. The author mentions 4 additional cases of cancer with asbestosis in the literature and one must agree with him that, in view of the small number observed and the very few cases of asbestosis autopsied (12 cases in Germany), the number of cancers found is a very high one. Of these 6 cases, the site of the cancer in 5 cases was the lower lobes, in five there was a squamous-celled epithelioma. Three of the victims were less than 43 yrs. old; in all, work with asbestos had begun 15-21 yrs. before. After all, the author seems quite right in seeing a causal relation between asbestosis and lung cancer.—*L. Teleky.*

CLINICAL CONSIDERATIONS ON THE QUESTION OF INDUSTRIAL CANCER OF ASBESTOS WORKERS. *F. Hornig. Ztschr. f. Krebsforsch., vol. 47, pp. 281-287 (1938).*

A 35 year old woman who worked in an asbestos factory from 1919-1928 fell ill of asbestosis; in 1937 a sharply defined shadow was seen roentgenologically in the upper left lobe. This shadow was considered by the author to be a carcinoma and in the decision on the case, a causal relation between the presence of asbestosis and the development of carcinoma was accepted. Confirmation at autopsy of the diagnosis of lung carcinoma is presented in a paper by Nordman (preceding abstract).—*L. Teleky.*

EXPERIMENTAL INVESTIGATION OF THE SCHNEEBERG LUNG CANCER QUESTION. *H. R. Döhnert. Ztschr. f. Krebsforsch., vol. 47, pp. 309-339 (1938).*

In a very exact manner, with regard to the pertinent literature, and with careful consideration of all factors, the author reports on the tumors found in mice kept in the Schneeberg mines. In the experiment were 45 animals of whom 28 died. Of these, 26 were given careful pathological and anatomical examination; the other two were sent to another Institute for examination of their radioactivity. In 7 animals, tumors were found; in two of them, multiple tumors in various organs. Since the experimental animals came from almost tumor-free stock, the number of tumors found is a high one. In two cases there were true epithelial lung tumors, in two, ulcers of the mediastinum, in the other 3 cases there were tumor-like, more or less generalized changes of the lymphatic system. After careful consideration, the author blames the ra-