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LUNG CANCER AND OCCUPATION

Professor Dr. Koelsch, Ministerial Counselor

(Industrial Physician of the State of Bavaria, Munich)

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International studies have shown rather consistently that cancer of the respiratory tract has increased considerably everywhere in recent years. Attempts to explain this unwelcome phenomenon draw from various factors: on the one hand, the improved diagnostic capability brought about by roentgenography and the increased attention to the occurrence of cancer, and the more frequent autopsies, which gives rise to better statistical coverage as well; on the other hand, the increased average age of the European population, which allows more individuals to reach the cancer-favoring age. Nevertheless, these formal explanations are not entirely satisfactory, especially since the involvement of the total population in the lung cancer mortality rate is not uniform. Persons who live in large cities are affected more than those who live in small cities, and men are more often affected than women. Still other causes must therefore be drawn on: influenza with subsequent tissue metaplasia of the respiratory tract, dust in the air as a result of increasing street traffic, sooting of the atmosphere by flue gases, air pollutants from the exhaust of motor vehicles and from the emanations from the tarred streets, increased use of tobacco (cigarettes!) etc. So far, none of these attempted explanations has been found fully satisfactory.

Naturally, certain dangerous factors related to occupation must also be referred to, such as constant irritation by dust or certain chemical irritations to which large numbers of workers are exposed. But all of these attempts to explain the increased incidence of lung cancer have, with very few exceptions, been completely unsatisfactory and, so far, have been inadequate.

In the following report we wish to deal only with the problem of the more limited connections between lung cancer and occupation. Practical experience would seem to indicate that an apparently large number of harmful factors related

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to occupation promote the development of cancer and that in many occupational groups lung cancer has an unusually high incidence. However, if we wish to gain more insight into these causal relationships, we must single out individual industrial groups or groups exposed to the same risks and consider them separately. We must gather case by case and relate them causally with the specific occupational hazards.

1. WORK WITH EXPOSURE TO DUST

A number of authors, such as Askanazy, Schmorl, Fritz, and Sweany, et al., call attention to the fact that the continuous action of dust leads to irritation of the mucosa of the respiratory tract, which in turn leads to epithelial proliferation and metaplasia, and thus to precancerous changes. Changes of this sort have doubtless been observed over and over again; they too can lead to atypical tissue proliferation and thus to a neoplasm.

Junghans wrote: As far as the etiology is concerned, it is conspicuous that among those persons affected with cancer, there are often some to be found who had an occupation that involved heavy exposure to dust. Pigment penetration and anthracochalicosis are found here in large numbers of cases.

Buschbeck took the trouble to investigate more than 3000 cases of lung cancer for their possible cause of development. He pointed out that almost half of the men had been involved in occupations in which they were exposed to the action of dust (or smoke). However, he was unable to give a satisfactory explanation. I would like in this connection to refer to the observations of Sekoulitsch, who, on the basis of his observations in 2000 cases of disease in 377 woodworkers (cabinetmakers, carpenters, wheelwrights, etc.), discovered, in addition to chronic mucous membrane irritation, an unusually high incidence of malignant tumors of the upper respiratory tract, which he attributed to the inhalation of sawdust.

For all that, they have still been observed only in isolation relative to the huge number of workers constantly exposed to dust and to the huge number of dust injuries to the lungs. On the contrary, clinical and statistical studies have shown that there are no regular relationships between pneumoconiosis or silicosis and carcinoma. The most extensive observations of this type come from the classic country of silicosis, South Africa, at the mine in Witwatersrand. We read in the report of the Miners-Phthi is Medical Bureau (1932) that in 3215 autopsies within 8 and 11 years, only 16 cases of bronchial or lung cancer were found and that the miners with silicosis (1083) accounted for 4 of these cases (0.37%), while those without siiicosis (1109) accounted for 6 of the cases (0.54%) and nonmining personnel (1023) accounted for 6 cases (0.58%). Similarly negative results were obtained in studies by Boehme, Husten, Di Biasi, Schulten, Reichmann, and Kollmeier in German coal mining, by Sladden in England and by Kennaway in America. Husten found no cases of lung carcinoma in 205 cases of pneumoconiosis and no cases of pneumoconiosis in 5 cases of carcinoma of the lung. Di Biasi also found no carcinoma in 177 cases of pneumoconiosis.

Kikuth also noted that he was unable to establish any relation between pneumoconiosis and carcinoma of the lung in cases investigated in Hamburg. In relation to these extensive negative observations, which stretch over a period of years, the few positive findings play the role of isolated cases or coincidences.

For example, Pankoast and Pendergrass reported on 6 cases of lung cancer combined with silicosis; Dible discussed three such cases; Allen two cases; Fine and Jaso, Sladden, and Maxwell one case each. The case discussed by Cramer involved pneumoconiosis caused by iron, quartz and coal dust; the case reported on by Rodenacker involved anthracosis; and the two cases reported on by Dreyfuss concerned a brother and sister who had worked in iron oxide dust. I myself know

of a case (unpublished) of a sandblaster who had a combination of silicotuberculosis and lung cancer. Of course, we also find opposing interpretations. Schmorl points out that especially in the sandstone industry and in coal mining, chalicosis and anthracosis of the lung and of the bronchial lymph nodes are quite common, which results in damage to the lung, especially of the larger and intermediate bronchi.

Harris reported on cancer of the larynx (not lung cancer) in 6 coal miners who also had pneumoconiosis (in 117 cases of laryngeal cancer) between 39 and 59 years old; the length of employment was about 15 years. He also reported on 2 cases of bronchogenic carcinoma in combination with pneumoconiosis in 2 miners. Harris noted in this connection that nothing was known about the ultimate cause of the carcinoma development (dust, chemical actinic effects).

Seyfahrt reported that among 18 persons admitted to the Leipzig Hospital for bronchial carcinoma within one year (1926), there were four cigar-makers and that prior to that, 7 cigar-makers with bronchial carcinoma had been examined there. However, in my opinion, he was quite wide of the mark when he then characterized this affection as a typical occupational disease of the cigar-maker, caused by the constant irritation of the respiratory tract by dust. For in other places nothing is known of any susceptibility of this sort in cigar-makers.

Constant inhalation of dust was assumed to be the significant cause.

Observations of lung cancer in combination with asbestosis seems to me to hold some interest. Several authors have recently reported on this. I cite papers by Lynch (1 case), Egbert and Geiger (1 case), Gloyne (2 cases), Domenici (2 cases, oral report), and Baader (oral report). A total of 12 cases are known to date. Further experience is needed to show whether these represent isolated cases or specific diseases in combination with damage caused by asbestos. I consider it quite possible that there is specific causation in view of the type of action of the asbestos needles on the tissue.

Since it cannot be doubted that carcinomas can develop in other organs under the influence of chronic irritation, in theory it could be assumed that cancerous deterioration can also occur in the lungs and bronchi on the basis of the tissue changes produced by dust irritation. Schmorl noted the following in this connection: pneumoconiosis produces bronchitis deformans, whereby the bronchial wall becomes flat-knobby, and occasionally the cylindrical epithelium is converted to squamous epithelium; between the rings of cartilage there are small diverticula, deformations of the cartilage plates; in addition, there is connective tissue new growth and shrinkage of connective tissue with distortion and narrowing of the bronchi, peripheral bronchiectasis etc. Changes such as these occur especially in the upper and middle zones of the lungs, especially on the right, seldom before the 55th year, except in workers exposed to dust, in whom they can appear sooner. Adhesion then develops between the bronchial walls and the anthracochalicotic glands, whose pigment migrates toward the bronchus; finally, the softened contents of the gland break through into the bronchus (pigment rupture). Scars develop as a result of this which must be considered precancerous changes. Occasionally, chronic infectious processes seem to interact with the dust. Although there is much to be said for this explanation of Schmorl's, it is nevertheless my belief that in general the inhalation of dust cannot in itself be accepted as a practically important cause of carcinoma of the lung.

I am sure that there is no need at this point to call special attention to certain differential diagnostic difficulties which make it difficult in many cases to differentiate between tumor forms of silicosis and true carcinomatous lung tumors. Harries (see above) (referred to as Harris before[Tr.]), reported on three cases of pneumoconiosis which simulated mediastinal tumors. Gutz also wrote on the simulation of a lung tumor by severe anthracosis. I myself have recently seen a case of severe tumorous silicosis in which a diagnosis of carcinoma

of the lung had been made at first. Naturally, it is also necessary to rule out lung metastases in cases of carcinomas in other organs.

2. THE QUESTION OF CHEMICAL IRRITATION

In industrial medicine, it often happens that an opinion must be given about whether a single case of inhalation (accident) or a constant action (occupational disease) of irritant chemicals, for example, chlorine, sulfur dioxide, acid vapors, caustic lime, chromates, can produce or trigger the development of a cancer of the upper or deeper respiratory tract. One would think that there must be a large number of observed cases in this area, since, of course, the manufacture and use of these chemicals is extremely widespread throughout the whole world and since, in addition, the extensive use of war gas materials in the World War provide a large number of cases that could be observed.

Contrary to this expectation, however, the reports on this subject in the world literature are extremely sparse. As far as single (acute) actions of such irritants are concerned, I can say that in my own investigations in the German acid industry, in which 1000 workers are employed, and in the acid-processing industry, I was unable to discover any such case.

In the following case published by Jacobs a lung carcinoma appeared four years after acute intoxication with illuminating gas; in the four-year interim the patient had had continuous chest pains. While Kikuth, Kraus and other authors believe there are possible causal connections in this case, other authors reject this notion in this case.

I could not find anything about carcinoma of the upper and lower respiratory tract due to war gas damage in the German and American war medical literature. In the remainder of the literature available to me, I found only the following case.

Spamer reported on a case of this type: It involved a 30-year-old soldier who died 33 days after inhalation of French war gas. On his epiglottis was found a marginal ulcer and an area of thickening of the mucous membrane the size of a lentil which microscopic examination proved to be a cornified squamous cell carcinoma.

Huhnermann also published a case: It involved a 37-year-old soldier who had inhaled mustard gas (dichlorodiethyl sulfide) in October, 1918. Subsequent to this, he suffered from dyspnea and coughing, but at first there were no findings. In the spring of 1919, his symptoms included difficult breathing, loss of voice, paralysis of the vocal cords and chronic catarrh; an infiltrating carcinoma of the larynx was discovered in the fall of 1920.

In addition, Kahler mentioned his observation of the development of a papilloma of the soft palate after war gas corrosion.

The case described by Tilley may also be mentioned here; it involved an intratracheal tumor following the action of war gas at the origin of the right bronchus (papillary granuloma) based on ulcerations of the mucous membrane.

Derischanoff, in his paper on lung cancer, mentions the possibility of carcinoma being caused by irritant gases such as phosgene and chlorine; in this connection, he writes, "It is certain that considerable morphological changes occur in the epithelium of the bronchial mucosa under the influence of gassing. We find changes in epithelial differentiation, epithelial proliferation and a marked inflammatory process (Krinitzky). All of these are predisposing factors in the development of lung cancer. Nevertheless, it seems to me that no such great role can be ascribed to gassing in the development of lung cancer since in the numerous references which I have studied, I have encountered not a single case which could be attributed to gas poisoning according to the case history."

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Among his 898 cases of primary lung cancer, Brockbank found four cases in which the patient had been gassed with poisonous gases during the war.

Several of these cases, for example, the case reported on by Spaner and the case discussed by Huhnermann, are strongly contested or not accepted; it is correctly said in this connection that general observation of the thousands of soldiers who were gassed shows no such causal relationship.

Another case of carcinoma of the upper respiratory tract following chemical injury was reported by Birkholz; however, the circumstances of this case were somewhat different. This case involved a 43-year-old chauffeur who got some gasoline in his throat in April, 1916 and immediately suffered a fit of coughing and hoarse voice. The laryngeal complaints in the form of productive chronic inflammation lasted for 3-1/2 years; in 1919 a cancerous tumor was discovered in the larynx; the patient died in 1921 in cancerous cachexia.

In the following cases, chronic effects of irritant gases are blamed for the development of the tumor. Kikuth describes the following case: a worker was exposed for one year to dichloroethylene vapors in a chemical plant. Difficulty in swallowing developed, and six months later a carcinoma was found in the right upper lobe. Death occurred 14 months after the patient had begun to work in the chemical factory.

Nikoloff reported on a case of bronchogenic carcinoma in a chauffeur who worked in that capacity for 20 years and who naturally inhaled gasoline vapors and exhaust gases during this period. Nikoloff believed it was possible to assume chronic irritation by gasoline vapors and other gases.

I myself judged the following case: An industrial chemist who worked for 30 years in a hydrochloric acid factory became affected at the age of 63 with a pedunculated squamous cell carcinoma of a vocal cord. In accordance with German insurance legislation, the case had to be formally rejected as an occupational

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disease; however, there are also medical considerations that oppose a causal relationship. Besides, in my extensive studies in the acids industry, in chemical plants of all types etc., I have never been able to observe any connections between the chronic action of irritant substances and cancer of the respiratory tract.

We can also include the observations of Saupe, who examined the workers of a large metalworks in which ores with a high content of arsenic are processed. He found mild cases of dust irritation and signs of chronic arsenic poisoning, but not a single case of lung cancer.

Kotzing mentioned a case of bronchial cancer in a worker at a rubber goods factory in which the chemicals that were used there had been blamed as the cause (vulcanization accelerators, sulfur chloride, benzene, gasoline). However, a causal connection had to be rejected.

Naturally, repeated efforts have been made to deal with these problems in animal experiments. I would like to cite the experiments of Ricker and Adelheim, who observed epithelial metaplasia following inhalation of phosgene and chlorine. Miller found basal cell proliferation in animals after inhalation of diphosgene. Of course, it seems questionable whether forced experiments such as these may be used for purposes of evaluation in insurance medicine. The animal experiments performed by Koontz seem more convincing to me. Koontz performed inhalation experiments over a period of years on hundreds of dogs, whose lungs are much more sensitive than human lungs. He used phosgene, chlorine, chloropicrin, mustard gas, lewisite and methyldichlorarsin in concentrations that approach lethal concentrations. After the symptoms of irritation had run their course, permanent functional damage of the respiratory organs was not observed; in rare cases, pathoanatomical examination revealed slight fibrous changes in the bronchioles and in the lung tissue which outlasted the symptoms of irritation.

Finally, we should also mention two series of experiments with chemical substances. Garschin and Schabad injected 10% formalin solution directly into the lungs of rabbits and in this way produced atypical epithelial proliferation, which presumably was caused by the inflammatory process. Nicole allowed rabbits to inhale beta-naphthalenamine vapors and then injected them with embryonal lung tissue. In one of these animals he found a tumorously altered pulmonary lobe.

Novel observations were made in England some time ago; I had not been aware of publications on such observations prior to this. These observations involve 14 cases of lung cancer (and 16 cases of cancer of the paranasal sinuses) which appeared after 10-12 years of activity in the processing of nickel ores. The cause is still unexplained; radioactivity is out of the question here. Similar observations in other countries have not yet been published.

A great many references have recently been made of the effects of inhaled tar vapors. I myself had to give an opinion on such a case a short while ago. A 48-year-old mechanic who had worked for 24 years in a factory that produced tar and who had repeatedly suffered for a long time with chronic bronchitis (tuberculosis!), finally became affected with sarcoma. The treating physician assumed occupationally-caused bronchitis due to irritation to be the basis of the sarcomatous changes. On the basis of German insurance legislation, I had to formally reject the case as a compensable occupational disease.

In a similar case of fatal bronchial cancer which supposedly had been caused by tar vapors (Neumann), it was also necessary to reject this causal connection.

Kuroda and Kawahata recently reported on lung cancer in producer gas workers. They found 12 cases (between 38 and 45 years old) with an average number of years in this work of 15. The observed duration of the disease was about seven months.

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The authors conjectured that tarry gases might be the essential causal agency, supported, to be sure, by other irritant gases and dust. Another five cases (in which exposure to coal gases was also involved) were found by these authors in the associated smelting works. Kennaway also believed that it was possible to establish a higher susceptibility in the workers in his studies who work with coal gas and tar.

In this connection, I should also mention the experimental studies of Campbell and Argyll, who allowed their experimental animals to inhale street dust with 2% tar or tar dust with carbon monoxide gas for approximately one year. After about six months they observed malignant skin warts and secondary lung metastases, but no primary lung cancer, in mice (but not in guinea pigs or rabbits). I will also mention the experiments of Kimura and of Schabad, who produced atypical epithelial proliferation by intrabronchial insufflation of tar. Carschin and Pigalew injected gas tar directly into the lungs and in this way produced atypical epithelial proliferation which, however, did not become malignant. The authors consider these tumors not to be the result of the specific action of the tar, but rather of the chronic inflammatory irritation. Murphy and Sturm proceeded differently; by brushing tar on the skin of young mice, they obtained typical epithelial new growths in the lungs which were similar to tumors that occasionally appear spontaneously in older animals. However, they did not assume a specific effect of the tar.

The question of the effect of tobacco smoke, which once again is receiving stronger emphasis, is very closely related to the question of "tar damage". Earlier, I pointed out the view of Seyfarth that "bronchial carcinoma is a typical occupational disease of cigar workers" (dust). However, smoking, especially smoking in which the smoke is inhaled, is addressed even more as a cause of lung

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cancer. In 1933, Ferrari referred to the tar content of tobacco smoke as a cancerogenic substance and pointed out the correlations between steadily increasing consumption of tobacco and the increase in lung carcinoma. Lickint wrote in 1929, and again in 1935, on bronchial cancer in smokers; he too assumed a specific action of the tarry substance contained in tobacco smoke. On the other hand, and in contrast to Merten, Campbell was unable to produce new growths of the respiratory tract by tobacco inhalation in animal experiments. Like tar, paraffin also has a recognized cancerogenic effect. In conclusion, therefore, I would like to report an observation made by Gartner which has no significance as far as occupational pathology is concerned, but which nevertheless allows us to draw certain conclusions. This case involves paraffin granulomas of the lung in a 64-year-old man who for years had used the paraffin-containing agents (sprays)Mistol and Rhinolaryngin because of chronic pharyngitis.

The production of the world literature on lung cancer and chemical irritants is thus quite inadequate; moreover, several of the cases cited are challenged by criticism. Let us first consider the anatomical conditions. It is known from experience that a single inhalation of an irritant gas for a single hour causes only slight catarrhal inflammation of the mucosa of the respiratory tract accompanied by the formation of a mucous discharge. This inflammation subsides after a short period of time. As far as chronic irritant effects are concerned, repeated inhalation or inhalation over a period of several years of an irritant gas of suitable concentration can sometimes lead to the development of severe inflammation of the mucous membranes with concomitant discharge of a more or less highly purulent exudate and to the development of ulceration. Six to eight weeks are generally required for the healing of gas ulcers of this type. That continuous

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chronic inflammatory processes of various types with repeated regeneration processes and tissue metaplasia ("erosion of the epithelium") and especially ulceration and cicatrization can possibly cause the development of mucosal cancer after a long latent period is a view that is repeatedly advocated and for whose correctness a large number of arguments can be cited.

In general, however, it can be said that the international observations leave a great deal of doubt in our minds about whether there are closer causal connections between the inhalation of irritant gases and bronchial or lung carcinoma. In view of the huge numbers of gas poisonings in the war, however, these few observations do not indicate very close causal connections, especially in view of the now rather long intervals of time.

In the above statements we have cited many examples of cancer of the lung and bronchi in which acute or chronic effects of irritant chemicals may perhaps have played a causal role. In my opinion, however, these are still only isolated cases or mere "possibilities". However, international mass observations in industry and in gas warfare has taught us that from the standpoint of insurance medicine a causal connection between acute or chronic inhalation of irritant gases and cancer of the upper or lower respiratory tract can be accepted only in rare instances and only when special conditions are satisfied (young age of the affected individual, continuous medical observation, demonstrated ulceration or cicatrization, infiltrating tumor and the like). Observations on the effect of tar vapors are more serious but do not yet provide clear proof.

There is still another type of lung cancer of chemical causation that must be discussed. It must be considered a true (specific) occupational cancer. These are cases of lung cancer in chromate workers of which I first became aware in 1911.

Thirty-eight cases are now recorded in Germany. I cite the publications of Alwens, Jonas, Pfeil, and Teleky. Diseases of this type have not been reported on in foreign countries as yet. I am also aware of one such case in Switzerland, which I learned of by private communication. So far, the affected individuals have been workers who were exposed to the vapors of chromate solutions or to dust containing chromate. Chrome iron ore itself does not appear to have a disease-producing effect since it is practically insoluble in the body. The period of development is 20-40 years, the course is insidious and can extend over several years. Since only a small portion of the personnel becomes affected with the disease, it seems likely that personal disposition is involved. More advanced age seems to favor development. The first disease symptoms are insignificant for a long time: bronchitis; bronchopneumonic states follow this; occasionally pleuritis is found. The radiographic findings are decisive, of course; in the first stages, there is enlargement and solidification of the hilar glands (often only unilaterally) later there is localized solidification in the lung tissue, which then no longer differs in any respect from bronchial cancer or lung cancer of other origin. There is certainly no need at this point to deal further with the difficulties involved in differential diagnosis.

Continuous x-ray examination of the endangered groups of workers is basic to prophylaxis. Enlargement of the hilar glands (especially unilaterally) is suspicious and is cause for immediate removal from the chromate plant. Naturally, constant hygienic improvements in the factories must go hand in hand with this, especially the extensive elimination of vapors and dust at their sites of development. In accordance with German social legislation (Third Occupational Disease Law of December 16, 1936), lung cancer in chromate workers is a notifiable and compensable occupational disease.

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3. EFFECTS OF RADIATION

For many years we have known of the lung cancer in miners at Schneeberg (Saxony) and Joachimsthal (Czechoslovakia). These two places are 26 km apart and lie on the same ore veins. It has been known for probably 500 years that these miners mostly die of severe lung diseases (called "miners' sickness" or "miners' lung sickness").

More exact studies were conducted in Schneeberg in 1878-1879 by Hesse and Harting; at that time, about 28-32 men among 600-700 members of the crew died each year, and 75% of these deaths were due to lung cancer. In the period between 1875 and 1912, 276 miners died of lung cancer, 10 of tuberculosis and 183 of other lung diseases. In the last (official) study by Thiele, Rostocki, Saupe, Schmorl and others in 1922-1925, 154 miners were given clinical and radiological examinations; 17 cases of tumor shadows in combination with pneumoconiosis were found. Lung cancer was found in 13 autopsies that were performed in this period, and in eight other cases of death there were symptoms of lung cancer. The affected miners ranged in age from 37 to 69 years (mean age: 55 years); the time that had been spent in mining was about 20 years or more. The clinical symptoms and the differential diagnosis do not need to be discussed here. As far as the pathologic anatomy was concerned, most of these cases involved a carcinoma or sarcoma arising from the hilus or from a bronchus, and in most cases this was combined with a more or less well-developed case of pneumoconiosis.

The first studies in Joachimsthal were published in 1929 by Loewy, although it had been known for a very long time that many miners there died prematurely of severe lung disease. The autopsy results were similar to those obtained in Schneeberg; in the year 1929-1930, 19 miners died there; among 13 patients on whom autopsies were performed, there were 9 with lung cancer; another six workers

also died of severe lung diseases. In the year 1931-1932 there were 12 deaths, including 9 from lung cancer. At the same time, there were often pneumoconiotic changes. According to data supplied by Ziel, such changes were found in about 60% of the workers.

What are the causes of this occupational cancer that is localized so peculiarly in such a well-defined mining district? In the Schneeberg mines, silver, nickel, cobalt, bismuth and arsenic-bearing ores are mined in shafts as deep as 300 meters. In recent years, pneumatic drills have been used. The average air temperature underground is between 9 and 16°C. The mines are usually very damp; various types of mold fungus can very often be observed on the wood shoring. Despite the dampness of the rock, highly splintery dust develops during the drilling of the rock and the crushing of the ore. The work must be considered heavy. In addition, the miners, who live in the more or less distant villages, have a long walk to and from work in all kinds of weather. Furthermore, very tiring trips up and down ladders to the mines are usually required. One first thinks of arsenic dust or of diethyl arsine gas, which is said to arise from the action of fungus (Penicillium and Aspergillus were found in abundance in the timbering of the mine). However, these two interpretations were not confirmed. More recent investigations leave no doubt that in this case the lung cancer is attributable to radium emanations. The mine air and mine water have a considerable radium emanation content (up to 50 Mache units per liter of air, up to 70 per liter of water). However, high radium levels were not found in the drilling dust; therefore, the effect does not result from inhaled dust, but rather from the mine water. It should be noted that the well-known radium bath at Oberschlema, whose sources contain up to 12,000 Mache units per liter, is very close to Joachimsthal.

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Pitchblende, from which radium is prepared, is mined in the Joachimsthal mines (9000 kg of ore yield 3000 kg of uranium and 1 g of radium). 1.7, 450, 1380 and 5080 Mache units per liter were found in the mine water and 0.76, 8.6, 24.6 and 212 Mache units per liter were found in the mine air. High values were also found in the ore deposits and in the area around the radium factory. Our calculations indicate that a miner inhales about 33,6000 units daily and about 10 million units per year; but these estimates are probably quite low. Moreover, radium emanation penetrates the skin. It is worth noting that there were far fewer pneumoconiotic changes in Joachimsthal than in Schneeberg, but much higher emanation values; however, cancer development begins after a relatively shorter period of work in Joachimsthal.

There is no longer any doubt that the Schneeberg and Joachimsthal lung cancer is attributable to the radioactivity of the mine water and mine air. In these cases the inhalation of dust is of secondary importance. The animal experiments undertaken by Loewy et al., which it was hoped would contribute to the explanation of the cause, failed to produce useful results.

Observations such as these have not been reported from other mining districts with similar geologic conditions. Such districts would include Cobalt City (Canada), Katanga (Central Africa), Skuterud (Norway), Dobschina (Czechoslovakia) and Allemont (France).

In this connection, we should also mention two cases of lung cancer that occurred in two persons who were involved with the preparation of radium preparations (Neitzel). In one of these cases, the lung cancer had been preceded by skin burns, and carcinoma of the finger had preceded the lung cancer of the other case.*

*Note: In the last few years there have also been reports on two cases of total fibrosis of the lung with suffocation in a chemist and in a laboratory assistant in a radium laboratory (Belt and Doenecke).

The preventive measures here are not easy. The measures of dust control and of personal cleanliness are desirable, of course, in the interest of general hygiene, but they are not of crucial importance. Studies are currently in progress on the influence of artificial ventilation (mine air conditioning). Respiratory protective devices with animal and vegetable charcoal filters are recommended; in practice, however, it is hardly possible to perform hard work all day long with a well-sealed mask on one's face. The proposal of short working times with regular changes to harmless working positions is more expedient; in addition, there should be frequent breaks and vacation as well as change of occupation at the right time. Finally, continuous medical supervision is necessary.

If we now summarize the above observations on lung cancer and occupation, we come to the following conclusions:

SUMMARY

The acute and chronic irritations of the respiratory tracts caused by the breathing of irritating dust, gases and vapors are not, in general, the causes of lung carcinoma.

Nevertheless, isolated, special cases can appear but should not be considered as a common occurreuce.

Among those groups most exposed to carcinoma of the lungs we must cite those working in asbestos dust and especially those in contact with irritating chemicals- the chrome workers.

International research is needed which should be widened to take in the presence of cancer of the intestines and of the stomach.

Tar vapor is probably also disastrous.

The question of the influence of nickel dust should be cleared up though it is of much less practical importance.

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The mine workers of Schneeberg and Joachimsthal are especially predisposed to carcinoma of the lungs. The incriminating causes are: the emanation of radium in the air and water, and perhaps also dust containing radium.

The workmen and employees of the radium factories and laboratories are exposed to the same dangers.

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