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REPORT ON THE CLINICAL SYMPTOMATOLOGY AND PATHOLOGY OF ASBESTOSIS

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ASBESTOS ... ASSOCIATION
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I. ASBESTOS**

The word "asbesta" means noncombustible. Even prior to Herodot's time, mineral asbestos was already being woven into cloth (ref. 25) as the latter, around 450 B.C., described a cremation garment apparently made from it (ref. 22).

According to Strabo and Plutarch, the wicks of the virgins' lamps were made of "asbesta". We also get similar information from Plinius and Pausanius (ref. 22).

Marco Polo also observed that the Tartars used a certain kind of cloth which resisted fire. The latter was apparently made from a certain earthy mineral (ref. 22).

The most important characteristics of asbestos are as follows:

-- excellent resistance to high temperatures;

-- spinability of the soft fibers;

-- capacity of bending into desired shapes;

-- resistance of certain types to acids;

-- minimal electricity and heat transfer capacity.

It is evident that the ancients were at least partly aware of these properties which led to its increased use in the industry as of the beginning of the 20th century.

**Literature, see 31, 44, 45.

A. Chemical and Physical Characteristics of Asbestos

As we know it today, the term asbestos (in French: amiante, pierre a coton; in Italian: amianto; in German also bergflachs, etc.) is actually a technical collective term used to refer to various basic fibrous silicates of magnesium.

From a mineral point of view, these can be broken down into 2 main groups.

I. Amphibolic asbestos (= chemically and mineralogically different fibrous minerals obtained from monoclinic and rhombic amphibole). For instance, tremolite, corcidolite (= blue asbestos) and amosite are all members of the amphibolic asbestos group.

II. Chrysotile asbestos (= serpentine asbestos or olivine asbestos, a fibrous variety obtained from the rhombic serpentine group).

Each of these 2 main groups contains a number of varieties which differ from each other chemically and physically and can thus be technically analyzed more or less accurately. The following table contains the individual physical and chemical characteristics which apply.

TABLE I. KEY: a - amphibolic asbestos; b - chrysotile asbestos; c - formula; d - hardness; e - specific density; f - refraction; g - double refraction; h - melting point*; i - resistance to acids; j - thermal conductivity; k - elasticity. 1 - minimal; 2 - very high; 3 - low; 4 - little elasticity; 5 - brittle; 6 - around 1550°C; 7 - low; 8 - good when separated into fibers; otherwise low; 9 - high elasticity.

	a) Hornblendeasbest	b) Chrysotilasbest
c Formel	$Mg_3CaSi_4O_{12}$	$H_4Mg_3Si_2O_9$ oder $3MgO \cdot 2SiO_2 \cdot 2H_2O$
d Härte	5-6	3-4
e Spezifisches Gewicht	2,5-3,4	2,3-2,5
f Lichtbrechung	1,66-1,60	1,55-1,53
g Doppelbrechung	1) sehr klein - 0,03	0,01-0,02
h Schmelzpunkt*	1150-1530° C	6) um 1550° C
i Widerstandsfähigkeit gegen Säuren	2) sehr stark	7) schwach
j Wärmeleitfähigkeit	3) schlecht	8) aufgefasert gut, sonst schlecht
K Elastizität	4) wenig elastisch, 5) spröde	9) sehr elastisch

*according to Niggli: tables for general and specialized mineralogy

Amphilbolic asbestos is mostly grayish-green and yellowish-brown in color, brilliant like glass and silk, transparent, brittle or elastic.

The color of chrysotile asbestos is generally similar to that of amphilbolic asbestos though somewhat darker; its fibers are usually fine but shorter and translucent.

There can be considerable differences in the chemical make-up of the individual types. Table II (quoted according to Sundius and Bygden (ref. 127)) gives a short review of this.

B. Appearance, Production, Treatment and Use

Asbestos fibers are found mostly accumulated in basic eruptive rocks in the form of veins only a few centimeters thick; quartz should therefore not be expected to be found in asbestos products. The first rock consists mostly of serpentine, but amphibole also comes in the form of country rock. The asbestos veins can contain traces of magnetite, chromite and serpentine.

TABLE II. KEY: a - serpentine asbestos; b - amosite asbestos; c - blue asbestos. 1 - silicic acid; 2 - aluminum oxide; 3 - iron oxide; 4 - ferrosoferric oxide; 5 - lime; 6 - magnesia; 7 - water; 8 - alkalis; 9 - titanic acid; 10 - nickel; 11 - manganis oxide. d - traces.

	Serpentinasbest a %	*Amositasbest* b %	*Blauasbest* c %
1. Kieselsäure SiO_2 . .	39-42,5	49-53	50,5-51,5
2. Tonerde Al_2O_3 . . .	0,3-3,7	0,13-2,4	—
3. Eisenoxyd Fe_2O_3 . .	0,7-4,4	34-44	35,5-36
4. Eisenoxydul FeO . .	0,42	37,34	—
5. Kalk CaO	0-0,35	0-2,5	—
6. Magnesia MgO . . .	39-43	0,7-6,3	0-3
7. Wasser H_2O	13,3-16,5	2,3-3	3,5-4
8. Alkalien Na_2O, K_2O .	—	—	7,5-9
9. Titansäure TiO_2 . .	—	0,09%	—
10. Nickel Ni	d Spuren	—	—
11. Manganoxydul . . .	—	d Spuren	—

The ores are extracted primarily through surface mining.

The largest asbestos fields are located in Canada (Thetford) and these make up approximately 3/4 of worldwide production (consisting primarily of chrysotile asbestos). Other important beds are found in South Africa (chrysotile asbestos, crocidolite, amosite), in the United States (Arizona: chrysotile asbestos), in Russia (Ural, Siberia: primarily serpentine asbestos) and in Finland (amphibolic asbestos). Until the end of the 19th century, the Italian amphibolic asbestos also played a major role.

Asbestos can also be found in various regions of Switzerland, including the Wallis and the cantons of Uri, Tessin and Graubunden. However, only the chrysotile asbestos beds of Puschlav (Quadrada Alps) exploited during the two world wars gained major importance.

Worldwide production has increased from approximately 500 t (1880) to more than 430,000 t (1929) (ref. 93) and continues to increase.

Using various methods, the raw mineral is decomposed on the mining site (milling, sorting [humid or dry] by means of a revolving screen into asbestos types of various fiber lengths). Once more or less freed of any impurities, the raw asbestos is finally sent to the plant where chrysotile and amphibolic asbestos are mixed in accordance with future requirements.

Asbestos types with long fibers are spun and woven into cloth. This requires final crushing in edge mills and the carding machine as it is called then turns the individual fibers into a preliminary yarn to be used by special spinning machines which will treat it and convert it into a finished asbestos yarn through twining and winding. In certain cases, cotton or fine metallic thread is wound together with the asbestos.

Short-fiber asbestos is usually mixed with fillers and rolled into plates or rods or converted into tiles, bricks, asbestos cement, etc.

Asbestos is used in many different applications, but we shall only mention the most important ones here: fireproof clothing, fireproof packing material, seals and insulating products; filters for use in sugar factories, cider and wine factories; linings for acid-resistant containers, laboratory objects in the chemical industry; insulators and diaphragms in the electrical industry; brake and clutch linings for automobiles; asbestos cement tiles and plates (eternit), also as foundation for colors, etc.

C. Asbestos as an Industrial Dust

The treatment of asbestos is generally accompanied by a high development of dust. From an industrial hygiene point of view, Aurebault (ref. 3) was the first in 1906 to arouse interest in this matter (as mentioned in references 93, 138), as later done by Oliver, Cooke and Simson (1925-1931) (refs. 22, 102, 115).

Most authors agree that the different processes used in the asbestos textile industry, i.e. carding, weaving, mixing and spinning do create large quantities of dust (refs. 22, 52, 74, 114). Other occupations in which the workers are subject to dust hazards include brake lining grinding work (ref. 16), and the manufacturing of asbestos mats. Asbestos mining and crushing in open mills do not seem to constitute any source of danger (ref. 102, 114).

Various researchers such as Cooke (ref. 25), Gloyne (ref. 54), Luton and Champeix (ref. 81) performed microscopic studies of asbestos dust. These

found fibers up to 150 microns long; Koppenhoefer found certain fibers which were as short as 2-3 microns (ref. 73); similar values were also found by Kuhn during an electron microscopic study (ref. 75).

Measurement of the dust content in various plants were performed by Dreessen et al. (ref. 37), Fleischer et al. (ref. 46) and Luton and Champeix (ref. 81). For the asbestos textile industry, Dreessen considers a concentration of 5 million dust particles per cubic foot as the safety limit. Luton and Champeix determined a dust density of 4-74 mg/m^3 air.

Many authors have recommended protective measures against dust; for example (see refs. 16, 22, 52, 74, 93).

II. ASBESTOSIS

Year-long inhalation of asbestos dust can lead to the development of diffuse lung fibrosis (pulmonary asbestosis) and finally and certainly to slow death.

A. Case No. 1

In 1906, Dr. M. Murray (ref. 96) reported on a 34-year-old patient who had worked in an asbestos plant for 14 years and was now dying of a chronic lung disease. The autopsy revealed a diffuse lung fibrosis without evidence of tuberculosis. Murray expressed his opinion that asbestos dust inhalation could be at the origin of this fibrosis since he had microscopically observed asbestos fragments in the lungs of the patient.

In 1914, Fahr described a case of "chronic enduring pleural pneumonia" in a 35-year-old female asbestos worker. Nodules in the lungs which macroscopically appeared as dust induration were microscopically found to be accumulations of "crystal, drumstick or nail-shaped formations"

(asbestos particles). Fahr found an original correlation between these "crystal formations" and "pleuropneumonia" with the inhaled asbestos dust (ref. 42, 43).

One additional early report was published by W. E. Cooke in 1924 (ref. 23) and 1927 (ref. 22). A 33-year-old female had been working in an asbestos plant for 18 years. Over the past 5 years, she had been complaining of increasing cough and dyspnea and, 2 years prior to her death, cavernous lung tuberculosis was added to her symptomatology.

The primary findings during the autopsy consisted of diffuse lung fibrosis. In 1927, McDonald published a detailed histological description of Cooke's case (ref. 92).

A summary published by Egbert (1935) lists 28 publications of individual asbestosis cases (ref. 38). However, most of the chemically and pathologically important points were already mentioned in the first works published by Murray, Fahr and Cooke.

A series of works on diseases and cases of death among asbestos workers were published between the years 1906 and 1918 (Scarpa quoted according to Loriga in ref. 138; Collis quoted in refs. 77, 93, 138; Whiteclock quoted in 93, 138; Beattie quoted in 93; Brit. Fact. Dept. quoted in ref. 93; Canadian Dept. of Labor quoted in ref. 74, 138; Hoffmann quoted in ref. 77, 114, 138; Pancoast and Pendergrass quoted in ref. 103). However, most of these are of limited significance.

B. Clinical Symptomatology

The clinical symptomatology of lung asbestosis was studied during several serial examinations and individual observations (refs. 1, 16-19,

37-39, 52, 68, 74, 81-84, 93, 101-105, 112, 113, 115, 117, 118, 124, 125, 144-147, 149.

1. Exposure time

The time elapsed between the beginning of employment in an asbestos dust-filled area and the appearance of symptoms indicating lung fibrosis varies. The development of pneumoconiosis first depends on the dust concentration and the exposure time, i.e. from the c/t product. However, many partly individual factors also play a role; nowadays these are somewhat difficult to determine (filtering effects of the nose and upper respiratory tract, etc.--see Ehrhard, ref. 40).

Most authors (refs. 1, 16, 18, 52, 64, 74, 81, 93, 144, 149) quote an average exposure time of 3-5 years. Of course special conditions around the individual working site must also be taken into consideration. Under particularly unfavorable conditions, some cases of asbestosis can be detected even before the aforementioned 3-year delay (refs. 18, 93, 125, 144, 149). In certain serial studies, none of the lungs were radiologically free of changes after a 10-year exposure. On the other hand, Merewether (cited in ref. 103) found only approximately 1/3 of the cases suffering from lung fibrosis after 10-14 years of employment.

The importance of the exposure time for the development of asbestosis is undisputed. Just as important, however, is an exact determination of the dust concentration (see Table III quoted according to Dreessen et al. ref. 37).

TABLE III. (quoted according to Dreessen)

KEY: a - dust concentrations in millions of particles per cubic foot; b - exposure time in no. of years; c - 10 and over; d - ill; e - exposed; f - percent; g - Emp.

a Staubkonzentration, Millionen Partikel pro Kubikfuß		b Expositionszeit in Jahren 1–4,9	5–9,9	c über 10
0–4,9	d krank	2 Arb. g	1 Arb.e	0 Arb.e
	e exponiert	81 Arb. g	19 Arb.e	5 Arb.e
	f in Prozent	2	5	0
5–9,9	d krank	0 Arb.e	6 Arb.e	13 Arb.e
	e exponiert	70 Arb.e	37 Arb.e	19 Arb.e
	f in Prozent	0	16	68
c über 10	d krank	8 Arb.e	22 Arb.e	21 Arb.e
	e exponiert	13? Arb.e	43 Arb.e	36 Arb.e
	f in Prozent	6	51	58

2. Symptomatology and diagnosis

In the first stages there is a "initial catarrh" in the area of the upper respiratory tract due to mechanical irritation, as described in (ref. 16, 81, 110). After a few months, there is an asymptomatic habituation stage which can last for several years.

The subjective symptoms appear very slowly and for a long time remain disproportionate as compared to the clinical and radiological signs. The leading symptom is an uncontrollable progressive dyspnea followed by a persistent cough and occasional mucous discharge. The patients often complain of piercing, burning and deep thoracic pain. The disease is followed by a general worsening of thepatient's health and the latter finally dies in a state of extreme cachexia.

Objective symptoms: oscultation and percussion are negative in the first stages and minimal in the later stages. There is a definite similarity to other types of pneumoconiosis.

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During a microscopic study of the sputum of both healthy and sick workers, asbestos particles are almost always detected (refs. 17, 82, 83, 86, 115, 123, 124). As a rule, a few months of exposure to the dust is required (minimum 3 weeks, ref. 81) before it begins to appear in the sputum; however, it can still be detected in the sputum several years after the end of exposure to asbestos. The presence of asbestos particles in the sputum simply indicate a previous exposure to asbestos dust and should not be considered as an indication of asbestosis (for example, see ref. 50).

However, the presence of accumulations of 20-30 radially arranged asbestos particles in the sputum have a totally different significance (ref. 124). These only appear in the case of advanced asbestos and, according to Stewart, indicate a destruction of the lung tissue due to an inflammatory process. With practically total numbness of the lungs in the most severe cases of asbestosis, there is sometimes no expectoration of asbestos particles and their detection can only be made through lung puncture (ref. 64, 122) or in the feces (ref. 57).

Severe dyspnea can be substantiated through spirometric examinations. The results of such functional tests are generally conclusive (ref. 104 and 16, 81, 106, 117, 125, 144).

In addition to the occupational history and clinical symptomatology, the radiological findings play a significant role in determining the diagnosis. Like silicosis, asbestosis goes through different radiological stages.

Saupe's classification takes all the likely possibilities best into consideration (ref. 74, 110). Additional radiological classifications were

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also suggested by Elman (ref. 17), Luton, Shull (cited in reference 139) and Stone (ref. 125).

Saupe differentiates between 4 different stages based on the transitional pictures:

Asbestosis 0-I: (preliminary stage). Somewhat uncharacteristic thickening of the lung image, particularly in the center and lower fields. Fine striped shadows and very light spotting in the complementary areas, particularly where the heart meets the diaphragm.

Asbestosis I: An extremely fine network of lung signs in the middle and lower fields, already covered with small light spots.

Asbestosis II: Marked lung signs, numerous minute spotted shadows, first darkening of the middle and lower fields, particularly around the heart. Broad and dense highlights, generally without calcium deposits.

Asbestosis III: Definite shadowing in the middle and lower fields, increased radiation in the upper fields, cardiac periphery difficult to differentiate, broad moderately dense highlights.

The shadows observed in asbestosis patients are soft and faint and differ considerably from those of silicosis which are very dense and calcareous and fairly sharp (snow-like, single-shot lungs, pseudo tumor-type balls). This makes up the most important radiological difference between silicosis and asbestosis. Somewhat less definite changes therefore do not necessarily indicate a less serious problem (ref. 118).

There are few reports available on the behavior of the circulatory system. The statement "no evidence was found which would relate heart impairment to asbestos dust exposure" (ref. 37) opposes the findings of heart enlargement (right-handed hypertrophy) in some of the cases (ref. 125, 139).

With the exception of a possible acceleration of the sedimentation reaction (ref. 90, 137), the blood analyses showed nothing specific.

With regards to the theory whereby asbestos decomposes in the organism releasing silicic acid which could be responsible for the fibrosis, studies of the SiO_2 elimination in asbestosis patients were somewhat significant (ref. 37). However, these did not produce any higher values than those obtained with reference persons.

Gloyne and Dewirtz (refs. 34, 54) were the first to arouse our attention with regards to the so-called asbestos wartswhich might result from a foreign body reaction with hyperkeratosis and acanthosis of the epiderm and development of giant foreign body cells. The small papules contain asbestos fibers but no asbestos particles.

III. PROGRESS, THERAPY AND PROGNOSIS

The progress of the disease is chronic and can last for several years.

The therapy is purely symptomatic and, in addition to protection against the dust, the only effective measure consists of regular prophylactic radiological examinations at 1-2 year intervals (ref. 17, 18, 139).

Once the disease reaches a moderately severe stage, it continues to progress (even without further exposure to the dust) over periods of time which vary with the individual (ref. 93, 95, 149). The prognosis must therefore be made seriously (see Bridge quoted in 17, 37, 143).

Like silicosis, asbestosis then begins to show the usual complications (including emphysema, bronchopneumonia and right cardiac insufficiency). Bronchial carcinoma and lung tuberculosis deserves special mention in relation to asbestosis.

IV. ASBESTOSIS AND CARCINOMA

Contrary to silicosis (ref. 108), asbestosis is often accompanied by lung carcinoma. This combination was first suggested by Gloyne and Wood (ref. 60, 62, 149).

In 1938, Nordmann even suggested that bronchial cancer among asbestosis patients be recognized as an occupational disease (ref. 97). In 1941, he attempted to confirm his thesis through animal experiments (ref. 98). Almost half of 100 mice exposed to asbestos dust for 7-13 weeks, 68-80 hours, showed "abnormal epithelial growth symptoms" in the bronchial mucus membrane, partly with pavement epithelial metaplasia considered by Nordmann as a precancerous change. After 240 days, 10 animals were still alive, including 2 with a cornified carcinoma of the pavement epithelium. His calculations indicated a 20% incidence of carcinoma!

Vorwald and Karr were unable in thousands of experimental animals to induce lung tumors through exposure to silicates.

Incidence of Lung Carcinoma among Asbestosis Patients:

According to Wedler (ref. 135, 137), Koelsch (ref.72) and Welz (ref. 140), the 34 cases of asbestosis autopsied in Germany as of now included 10 cases with lung carcinoma or pleural endothelioma. The annual report of factories (England quoted in ref. 2) provides a summary of the years 1924-1946 with 235 deaths due to asbestosis, including 31 with carcinoma (bronchial carcinoma or pleural endothelioma). In the United States, Lynch and Cannon described over 40 cases of asbestosis autopsied, including 1 case of lung carcinoma (ref. 88).

The following table provides a comparison between the frequency of lung cancer in asbestosis and that in silicosis.

TABLE IV.

Silicosis autopsies obtained from literature (ref. 108)	2204, including 32 cases of carcinoma = 1.4%
Our own	271, including 3 cases of carcinoma = 1.1%
Asbestosis autopsies* (refs. 2, 72,88,135,137,140)	309, including 44 cases of carcinoma = 14.2%

The following table compiled by Homburger and supplemented by additional literature references provides a general view of the individual observations of carcinoma among patients suffering from asbestosis published prior to the year 1948.

The average exposure time is 16.5 years (minimum 19 months, maximum 42 years).

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*Compared to incidence of bronchial carcinoma in relation to autopsies performed in the Pathological Institute of the Zurich University (Prof. H. von Meyenburg) quoted according to Walther: Between the years 1936 and 1945, the latter varied between 0.94 and 2.78%.

There is an average latency period of 20 and 1/4 years between the beginning of exposure to the dust and the time of death (minimum 6 years, maximum 42-1/2 years).

In addition to this, conclusions cannot be drawn as yet as to whether the primary tumor actually develops in the area of the most severe asbestos fibrosis (Nordmann et al.) and whether the histological development is actually a carcinoma of the pavement cells in view of the small number of well-treated cases.

It is difficult to find an explanation of the relatively high incidence of bronchial carcinoma among asbestosis patients. Based on the known composition of asbestos, there can hardly be any possibility of a chemically active carcinogenic factor being involved. The possibility of a mechanical genesis of the tumor is discussed by Linzbach, Wedler (ref. 79), Nordmann (ref. 97), Welz (ref. 140), etc. The constant mechanical irritation caused by the fine sharp asbestos needles would eventually injure the cells and lead to an increased proliferation of the respiratory ciliated epithelium. According to the aforementioned authors, this persistent regenerating process could become malignant; however, this question has not as yet been clarified.

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TABLE V.

	Author	Year	Sex, age	Occupation in the asbestos industry	Exposure time	Time elapsed between beg. of exp. and time of death	Type of carcinoma	Seat of primary tumor	Metastases	Comments
1.	Lynch & Smith (85)	1935	M 57	weaver	21 yrs	4 mos	pavement cell	R LL	several nodules, R LL	originating carcinoma with bronchial pavement epithelial metaplasia
2.	Cloyne (61)	1935	F 35	spinner	8 yrs	9 yrs	cornified pavement cell	R UL	pleura	
3.	Cloyne (61)	1935	F 71	mattress mfg., opener	19 mos	15 yrs	cornified pavement cell	R LL	none	
4.	Egbert & Geiger (39)	1936	M 41	weaver	18 yrs	2mos	adeno	L LL	general	
5.	Cloyne (63)	1937	M 59	packer	21 yrs	? mos	oat-cell	L LL	L UL, pleura	
6.	Nordmann (97)	1938	F 35	spinner, incl. 2 yrs carding dept	7 yrs	9 yrs	cornified pavement cell	L LL	liver, kidneys	father died of carcinoma of esophagus
7.	Nordmann (97)	1938	M 55	production of prel. yarn	7 yrs	12 yrs	cornified pavement cell	L LL	general; pericardium, ventricle wall, diaphragm, peritoneum, retroperitoneum, WS	bronchial epithelial growth on right +++
8.	Lynch & Smith (87)	1939	M 50	weaver	13 yrs	3 yrs	pavement cell, partly adeno	R LL	pleura, mediastinal lymph nodes	another case of death by asbestosis shows severe metaplasia of pavement epithelium in the bronchial epithelium, also observed among certain asbestosis patients

TABLE V (cont'd)

9.	Wedler (Bohne) (137)	1943	M 58	master	10 yrs	3 mos	cancerous-type tissue	R ML	subcutaneous, axillary above 9th rib	
10.	Linzbach Wedler (79)	1941	M 60	preparation dept.(3 yrs)	18 yrs (incl. 3 yrs +++)	1 yr	cornified pavement cell	R LL	none	multiple metaplasia of the pavement epithelium
11.	Holleb & Angrist (67)	1942	M 52	pipe insulation works	25 yrs	9 wks	noncornified pavement cell	R UL	mediastinal lymph nodes, kidneys, suprarenal glands	pavement cell metaplasia in right main bronchus
12.	Holleb & Angrist (67)	1942	M 58	pipe insulation works	25 yrs	10 yrs	oat-cell	R LL	general: mediastinal peripancreatic lymph nodes, liver, kidneys, cerebral cortex, spinal cord	
13.	Desmeules et al. (33)	1947	M 57	machine convertor	25 yrs	10 yrs	alveolar cell pleuropulmonary epithelioma of alveolar origin	L L (pleura)	pleura, pericardium	
14.	Desmeules et al. (33)	1947	M 50	emptying asbestos sacks	22 yrs	1 mo	pavement cell, partly cylindro-cellular	R L	R L, pleura mediastinum	chronic cough for past 15 yrs
15.	Homburger (69)	1943	M 45	?	5 yrs	1 yr	pavement cell	R L	R L, pleura, diaphragm, pericardium, thoracic wall	bronchiectasis
16.	Homburger (69)	1943	M 43	?	20 yrs	17 mos	anaplastic	L LL	pleura	

TABLE V (cont'd)

17.	Homburger (69)	1943	F 49	no known contact with asbestos; diagnosis of asbestosis was a histological one			pavement cell	R L	liver, suprarenal glands, stomach, hilary lymph nodes	19 mos prior to death there was dyspnea and coughing; clinical diagnosis was bronchial carcinoma on right with liver metastases
18.	Welz (140)	1942	M 43	preparation of asbestos (4 yrs.), cabinet making, packer	11 yrs (incl. 4 yrs +++)	11 yrs	pavement cell	L LL	pleura, ribs, axillary lymph nodes	metaplasia of pavement epithelium in the noncarcinomatous bronchial sections
19.	Welz (140)	1942	M 56	mixer, carding	21 yrs	8 wks	skirrhus (?)	R LL	hilary and portal lymph nodes, liver, vertebrae, diaphragm	up to 1 yr prior to death, no subjective complaints, "not even coughing"
20.	Wedler (Teutschlander) (137)	1943	F 40	with long interruption, total	10 yrs	?	pleural blastoma, pseudo-alveolar mesothelioma	R	peritoneum	
21.	Wedler (Alwens) (137)	1943	M 60	asbestos rubber dept.	42 yrs	6 mos	primary mucus formation in the gland cell carcinoma of the pleura, largely adenomatous	L	peritonium chest musculature	
22.	Cureton (ref. 30)	1948	F 37	pipe insulation plant	7 yrs	15 yrs	pavement cell	L LL	pleura, myocardium diaphragm, liver, ovary, mediastinal lymph nodes, kidneys	

NOTE: One additional case of carcinoma in a lung asbestosis patient was published by R. Stoll et al. while the present article was under press (AMA Arch. Int. Med. 88, 831:1951).