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Talc Pneumoconiosis

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Since Thorel's original study in 1896,[1] when talc pneumoconiosis was first recognized as a distinct pathologic entity, numerous clinical and experimental reports have continued to appear in the literature. The bulk of these reports have related to workers in talc mills and mines and to those engaged in the rubber industry.* Three investigations of the talc industry were carried out in New York State.† Dreessen's study of 57 men exposed to tremolite revealed an incidence of fibrosis above average (67%) but of a nondisabling character. In contrast to Dreessen's [2] observations, the findings of Porro, Patton, and Hobbs,[5] based on 15 cases with 5 postmortem examinations, showed that pneumoconiosis causing disability and death does occur in talc workers and that pathological tissue changes are principally due to talc itself. In 1943, Siegal, Smith, and Greenburg [6] reported on the dust hazard in tremolite talc mining, including roentgenologic findings in talc workers, and their findings and conclusions were similar to those of the latter.

Since the roentgenograms and histories of the 32 cases reported on by Siegal and associates [6] were still available after 14 years, a follow-up study was undertaken in order to (1) ascertain the health status of this group, (2) evaluate the present operations and dust concentrations of the talc mines and mills, and (3) do a comparative study of the workers employed before and after the newer engineering techniques had been introduced (1943). The present paper deals largely with the first two objectives; the third is now in progress and will be reported at a later date.

Recorded for publication April 6, 1955.

From Division of Industrial Hygiene, New York State Department of Labor.

* References 2 and 3.

† References 4 to 6.

FINDINGS

Environmental Data.—A study of the operations and dust concentrations of the talc mills and mines at this time shows a marked improvement in the engineering techniques from that found in 1941, and this is reflected in an appreciable decrease in the dust exposure. From 1943 to 1948, a number of corrective measures were instituted to reduce the dust concentrations and included the following: (*a*) wet drilling in mines; (*b*) enclosure of elevator and chutes; (*c*) installation of automatic bagging machines; (*d*) conveyorized car loading; (*e*) discontinuance of blow rooms, and (*f*) provision of local exhaust ventilation. The dust concentrations obtained at the mills before and after improvements were instituted are summarized in Table 1.

Medical Data.—Deceased: Of the original 32 patients, 19 have since died, and the causes of death obtained from the hospital records, physicians' files, or death certificates are given in Table 2. No necropsy data are available to confirm the clinical diagnosis. The ages of the 19 that died ranged from 48 to 84 years, and in the 4 whose cause of death was believed to be due to pulmonary failure associated with talc pneumoconiosis, the ages varied between 60 and 79 years.

Living: Thirteen of the original 32 are still alive, and of these 7 have retired and 6 are gainfully employed. The ages of the retired group vary between 63 and 83 years; the ages of the actively employed, between

TALC PNEUMOCON

TABLE 1.—Compara

Mines	
Drilling	
Mucking	
General air	
Mills	
Crushing	
Screening	
Grinding mills	
Garners and separato	
Bagging	
Blow room	
Open chutes	
General air	

* Dust counts are in mill
† High count due to leak

Patient	Age, Yr.	
L. A.	Unknown	Fra
G. B.	63	Car
W. H. D.	34	(1)
		(2)
F. D.	53	(1)
		(2)
B. F.	60	(1)
		(2)
A. G.	Unknown	Unk
G. J.	68	Acu
D. K.	68	Acu
H. L.	66	Acu
F. V.	Unknown	Mes

* Causes of death were
(a) death certificates.

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TABLE 1.—*Comparative Dust Count* Data at Several New York State Talc Mines and Mills*

	Original Study (1941)				Follow-up Study (1954)			
	Low	Medium	High	Average	Low	Medium	High	Average
Mines								
Drilling	83	413	2,800	818	0	2	5	3
Mucking	2	36	475	120	3	4	5	4
General air	4	18	96	19	..	..	..	..
Mills								
Crushing	22	60	680	180	3	18	360	63 †
Screening	43	61	186	69	8	35	68	37
Grinding mills	32	75	271	92	5	15	40	20
Garners and separators	58	70	728	278	3	9	21	11
Bagging	26	129	520	151	5	16	90	28
Blow room	115	1,190	2,480	1,227		Discontinued		
Open chutes	21	88	440	125		Discontinued		
General air	65	70	85	69	5	18	40	19

* Dust counts are in million particles per cubic foot.
† High count due to leakage from conveyor enclosure.

TABLE 2.—*Causes of Death in Talc Pneumoconiosis*

Patient	Age, Yr.	Cause of Death *	Patient	Age, Yr.	Cause of Death *
L. A.	Unknown	Fractured skull	F. L.	62	Acute myocardial infarction
G. B.	63	Carcinoma of G.I. tract	C. L.	57	Carcinoma of stomach
W. H. D.	84	(1) Hypertensive cardiovascular disease (2) Cerebral hemorrhage	J. M.	65	Carcinoma of pancreas
F. D.	53	(1) Cerebral hemorrhage (2) General arteriosclerosis	W. M.	68	Pulmonary failure associated with pneumoconiosis
B. F.	60	(1) Pulmonary insufficiency—pneumoconiosis (2) Congestive heart failure	W. O.	48	Duodenal ulcer
A. G.	Unknown	Unknown	L. S.	72	Pulmonary failure due to pneumoconiosis
G. J.	68	Acute myocardial infarction	J. R. S.	79	(1) Pneumoconiosis (2) Myocarditis
D. K.	68	Acute myocardial infarction	C. T.	Unknown	Unknown
H. L.	58	Acute myocardial infarction	C. B.	58	Carcinoma of G.I. tract
F. V.	Unknown	Mesothelioma of pleura—pneumoconiosis (bilateral)			

* Causes of death were obtained from the following sources: (1) private physicians; (2) hospital records, and (3) death certificates.

45 and 64 years. A summary and analysis of the clinical and laboratory findings are seen in Table 3.

COMMENT

In view of the comparatively small number comprising this investigation, no significance can be attached to age incidence of the deceased and living groups. It is noteworthy, however, that the age range in the retired workers was between 63 and 83 years and that in the four deceased, whose cause of death was believed to be due to pulmonary insufficiency associated with talcosis, the ages varied between 60 and 79 years. Although no statistically significant correlative data can be formulated between the duration of tremolite talc exposure and longevity, it is of interest that an above average life span may occur, following prolonged exposure to talc dust and associated with pulmonary dysfunction (Tables 2 and 3).

Analysis of the medical data of the living shows many similarities to those reported in the literature.‡ Clinically, the outstanding features are dyspnea, productive cough, abnormal chest findings (diminished breath sounds, basal rales, limited chest expansion), clubbing, and a roentgenographic picture descriptive of pneumoconiosis. Clinically, the course is relatively slow but progressive, and, similar to silicosis, the clinical picture does not retrogress with marked improvement of the industrial environment or upon retirement. Dyspnea is present in 100% of the group and is sufficiently pronounced to limit

‡ References 5 to 7.

TABLE 3.—*Symptomatology of Talc Pneumoconiosis*

Patient	Age, Yr.	Status *	Environmental Exposure, Yr.	Clinical Picture: Symptoms † Cough	Symptoms † Dyspnea	Signs † Lungs	Signs † Heart	Signs † Clubbing	Roentgenologic Findings: Progression †	Roentgenologic Findings: Complications	Roentgenologic Findings: Plaques †	EKG	Peripheral Blood: Hgb.	Peripheral Blood: RBC	Peripheral Blood: WBC	Peripheral Blood: Diff.
F. E.	62	R	Talc mill—32 ‡	Mi P	S	Br. sounds distant Rales (basal) Emphysematous	Enl.	S	S	Bullae Cor pulmonale	Pr.	Rt.V.H. pattern	15.5	5.15	9.1	N
M. G.	64	W	Talc mill—25 Aluminum—21	None	Mi	Br. sounds distant Emphysematous	Not enl.	None	Mi	Hilar nodes enl.	Pr.	Normal	15	5.16	11.1	N
W. H.	49	W	Talc mill—18 Stool mill 8	Mo P	S	Clear to palpation and auscultation	Not enl.	Mi	Mo	Hilar nodes enl.	Pr.	.....	16.5	5.28	8.6	N
J. J.	49	W	Talc mill 30 ‡	S P	S	Br. sounds distant Rales (throughout) Emphysematous	Enl.	S	S	Cor pulmonale	Pr.	Rt.V.H. pattern	16.5	5.21	13.8	N
R. J.	59	W	Talc mill—25 ‡	Mi P	Mi	Rales and wheezes (throughout)	Not enl.	Mo	Mi	None	Not pr.	.....	15	5.01	11.8	N
C. K.	88	R	Talc mill—40 ‡	S P	S	Br. sounds distant Rales (throughout) Emphysematous	Not enl.	Mi	Mo	None	Pr.	Abnormal rhythm	15.5	5.09	9.8	N
V. L.	45	W	Talc mine 26 ‡	Mo P	S	Br. sounds distant Rales (basal) Emphysematous	Not enl.	None	S	Bullae Hilar nodes enl.	Pr.	Normal	16	5.12	7.1	N
S. P.	63	R	Talc mine—5 Talc mill—28 Zinc mine—2	S P	Mo	Br. sounds distant Rales (basal) Emphysematous	Not enl.	Mi	Mo	None	Pr.	Lt.V.H. pattern	16.5	5.2	7.8	N
C. S.	73	R	Talc mill—32 Other quarries —17	Mi P	Mo	Br. sounds distant Rales (basal) Emphysematous	Not enl.	None	Mo	None	Pr.	Normal	16	5.18	9.1	N
W. W.	69	R	Talc and zinc mines—51	Mi P	S	Br. sounds distant Rales (throughout) Emphysematous	Enl.	Mo	S	Hilar nodes enl.	Pr.	Lt.V.H. pattern	14.2	4.61	8.8	N
S. W.	63	W	Talc mill 30 ‡	Mo P	S	Br. sounds distant Rales (throughout) Emphysematous	Not enl.	Mi	Mo	Bullae	Pr.	Nonspecific T-wave changes	14.4	4.71	13.1	N
R. M.	79	R	Talc mill 24 ‡	Mo P	Mo	Emphysematous Rales (basal)	Not enl.	Mi	Mo	Hilar nodes enl.	Pr.	.....	....	....	....	..
R. R.	73	R	Talc mill—25 ‡	None	Mi	Rales (basal)	Not enl.	Mi	Mi	Hilar nodes enl.	Pr.	.....	12.2	3.5	10.2	Basophiles 6%

* Status; R—Retired; W Working.
† Mi—Minimal; Mo Moderate; S—Severe; P.—Productive; Pr.—Present.
‡ Exposure to talc only.

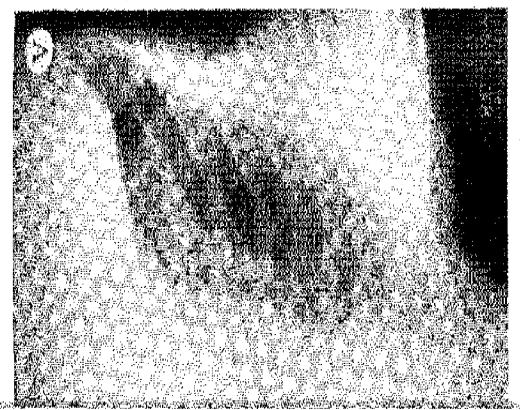

Fig. 2—A, Patient B. R. M., 1954, pericardial ar

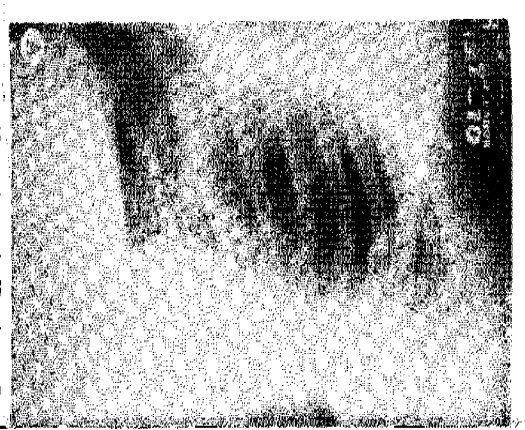

Fig. 1—A, Patient S. 1954, note giant bulla in R

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Of extreme interest are
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			mines—31	P		Rales (throughout) Emphysematous				enl.		pattern				
S. W.	63	W	Talc mill—30 ‡	Mo P	S	Br. sounds distant Rales (throughout) Emphysematous	Not enl.	Mi	Mo	Bullae	Pr.	Nonspecific T-wave changes	14.4	4.71	13.1	N
B. M.	79	R	Talc mill 24 ‡	Mo P	Mo	Emphysematous Rales (basal)	Not enl.	Mi	Mo	Hilar nodes enl.	Pr.	....	...	...	...	..
R. R.	79	R	Talc mill—35 ‡	None	Mi	Rales (basal)	Not enl.	Mi	Mi	Hilar nodes enl.	Pr.	....	12.2	3.6	10.2	Baso-philes 8%

* Status: R—Retired; W—Working.
† Mi—Minimal; Mo—Moderate; S—Severe; P—Productive; Pr.—Present.
‡ Exposure to talc only.

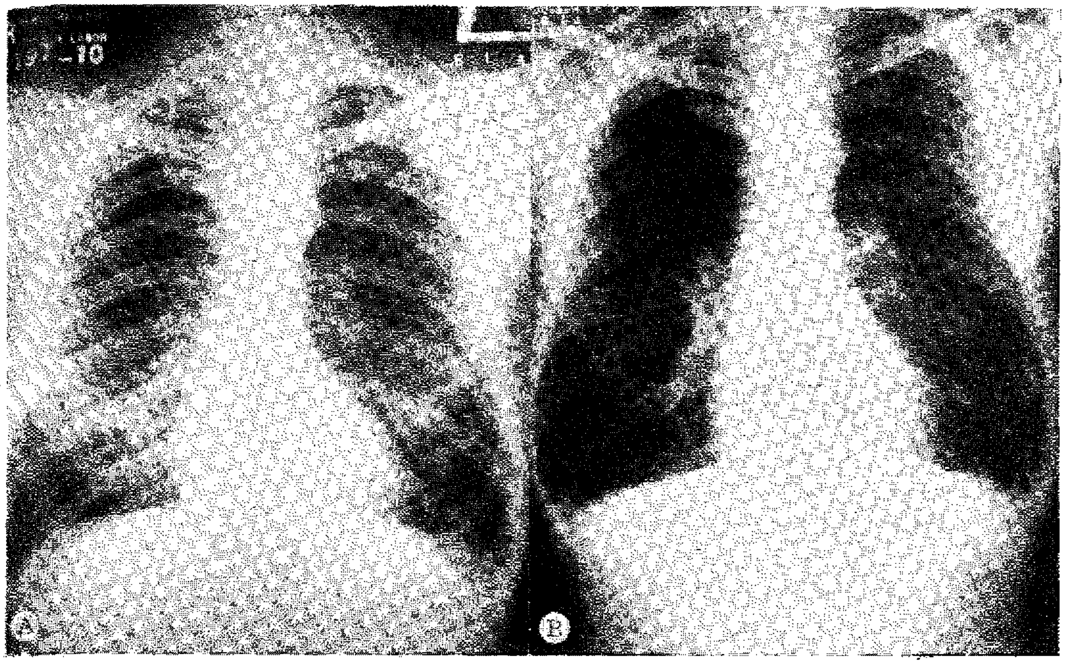
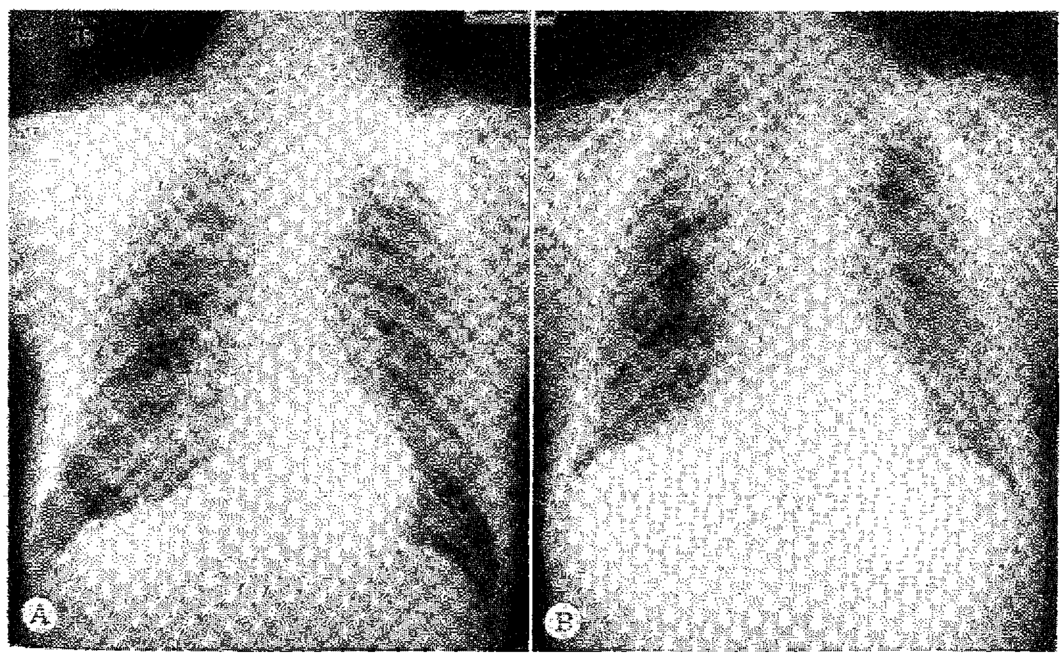


Fig. 1.—*A*, Patient S. W., 1941, characteristic basal infiltrations are seen. *B*, Patient S. W., 1954, note giant bulla in R. U. L. and prominent hilar markings.

the ordinary routine physical activity of the worker to a moderate degree.

Of extreme interest are the findings in the chest x-rays. The basic lesion is an interstitial infiltration of variable intensity, located basally and in the midlung fields and of bilateral distribution, with the apices relatively spared. In several, the infiltration is nodular in character. Emphysematous bullae of varying sizes are observed in four (Fig.

Fig. 2.—*A*, Patient B. M., 1941, pericardial and diaphragmatic plaques are seen. *B*, Patient B. M., 1954, pericardial and diaphragmatic plaques are more pronounced.



1*A* and *B*). A most striking finding is the presence of opaque densities, varying from single linear strands in the region of the diaphragm to massive deposits, bizarre in shape, extending over a large part of the lung field and occasionally on the pericardium (Fig. 2*A* and *B*). These deposits, referred to as talc plaques by Siegal and associates,[4] are observed in all but one of the cases studied. Cardiac enlargement is present in four persons, in two of whom the cardiac configuration is suggestive of cor pulmonale. The pulmonary arteries and branches are prominent in a number of the chest x-rays. When comparisons are made with the roentgenograms taken in 1941, it is of interest to note that, although variable progression occurred in the majority of the patients (Fig. 3*A* and *B*), the most outstanding revelation is that what in retrospect had appeared to be severe pulmonary involvement in the original series cannot be so considered in the light of a 14-year interval. Furthermore, the hilar adenopathy which is seen in six of the present series in retrospect was also present in three of the x-rays in 1941. As mentioned previously, two of the present chest x-rays showed cardiac configurations suggestive of cor pulmonale, but of even added significance, a markedly decreased cardiac silhouette is noted in one patient (G. S.) and is strongly suggestive in another. The cause for this is not clear. It is possible that pleural pericardial adhesions, associated with the plaque formation, and subsequent retraction and limitation of the pericardium may possibly account for this decreased cardiac silhouette.

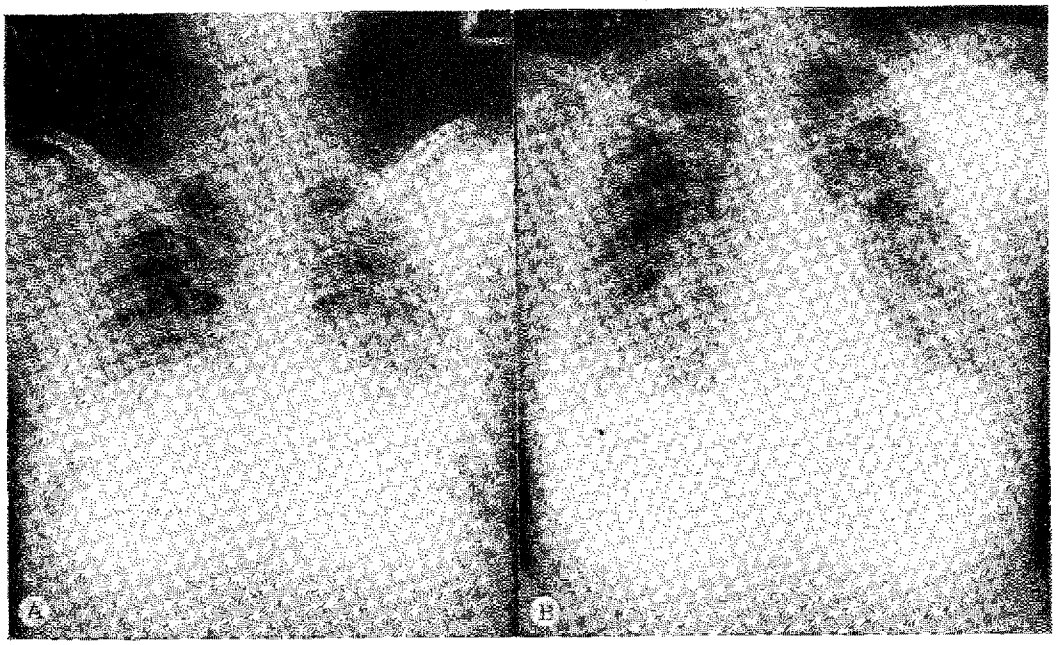


Fig. 3.—*A*, Patient J. J., 1941, marked basal infiltrations are noted. *B*, Patient J. J., 1954, progressive infiltration extending to upper lobes is seen; there is a dilated heart; clinical picture is one of cor pulmonale.

No correlation between symptomatology and x-ray findings is noted, and this has been observed by others.§ Although the series is small, there appears, nevertheless, a trend between the degree of clubbing and chest x-ray involvement; those with severer clubbing show more extensive pulmonary involvement.

Six out of 11 show abnormal electrocardiographic configurations, such as (*a*) pattern of right ventricular hypertrophy (2); (*b*) pattern of left ventricular hypertrophy (2);

§ References 6 and 8.

(*c*) abnormal rhythm
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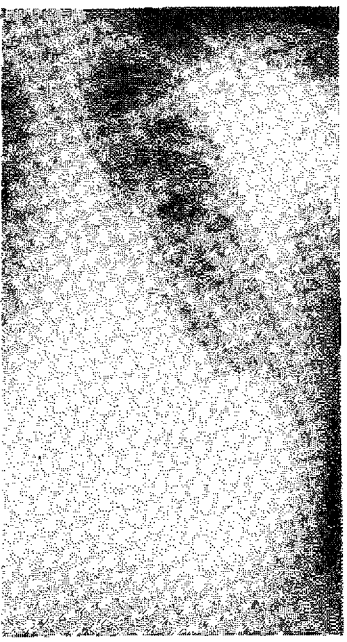

noted. *B*, Patient J. J., 1954, is a dilated heart; clinical

ion between symptomatology dings is noted, and this has l by others.§ Although the , there appears, nevertheless, a the degree of clubbing and volvement; those with severer more extensive pulmonary in-

l show abnormal electrocardio- urations, such as (*a*) pattern icular hypertrophy (2); (*b*) ventricular hypertrophy (2);

6 and 8.

(*c*) abnormal rhythm (1), and (*d*) nonspecific T-wave alterations (1). There is a suggestive trend between clubbing and abnormal electrocardiographic findings. In five, whose electrocardiograms are normal, only one shows clubbing, and this to a minimal degree; in the six with abnormal electrocardiograms, clubbing varies from a moderate to a marked degree, being severest in the two with the right ventricular hypertrophy pattern. It is noteworthy that in the latter two, the clinical picture is characteristic of cor pulmonale. The incidence of the latter in tremolite pneumoconiosis is unknown; however, histologic evidence reported by several investigators [5] confirms an etiological relationship. Jaques and Benirschke [9] have reported not only generalized thickening of all the pulmonary arteries, with cardiac enlargement associated with progressive pulmonary talcosis, but also a granulomatous involvement of myocardium per se. In view of the paucity of the available pathological data on human pulmonary talcosis, the mechanism underlying the development of cor pulmonale is not clearly defined.

The peripheral blood findings are not remarkable and indicate that the peripheral blood does not reflect the physiopathologic changes associated with talc pneumoconiosis.

The workers presently observed are well developed and well nourished, in contrast to the observations made by Siegal and associates,[6] and no reason can be offered to explain this apparent difference.

The results of the present study confirm the findings of other investigators that talc per se does produce a distinct fibrosing pneumoconiosis, and it has been suggested that this slowly developing fibrogenic disorder may be allied to asbestosis.[10] According to McLaughlin, the evidence favors that talc pneumoconiosis is caused only by the fibrous varieties of talc. The asbestine variety of the talc found in our study may therefore explain the close resemblance of the x-ray picture to that of asbestosis. It is significant that asbestos bodies, first emphasized in talcosis by Gardner, are almost invariably present in cases of talcosis in which talc fibers are of an appreciable length.[10] This has been observed histologically by several investigators [5] and by ourselves.‖ In essence, this pneumoconiosis consists of a dense fibrosis which is particularly prevalent peribronchially and perivascularly and occurs in sheets, occasionally assuming a nodular configuration; the latter finding has been suggested as representing a reaction to the quartz content of the talc. The hilar lymph nodes are generally described as showing no enlargement or fibrosis, and on microscopic examination they are reported to contain talc-laden macrophages and the talc content to be of considerable magnitude.

It is appreciated that some of the workers may have been exposed to a relatively high concentration of quartz at some phase of their operation, but it appears highly unlikely that this was uniformly present for all the workers, particularly in view of the consistent finding of low free silica content in both the mills and the mines. Furthermore, evidence has been presented by recent investigators ¶ that moderately advanced pneumoconiosis is caused by talc in the absence of any silica in the talc mixture or in the atmosphere. It is not unlikely that some of the commercial preparations of talc which have different mineralogic and chemical characteristics, as pointed out by Hogue and Mallette,[11] may require a long exposure before chest x-ray evidence or symptoms appear. From observations of Siegal and associates,[6] 10 years are required between exposure and onset of symptoms.

What the specific factors are in the causation of talc pneumoconiosis are not known. There is need for more epidemiologic studies where environmental conditions are well controlled and the life course of the worker is closely followed. This would permit a better statistical analysis of data than is now possible. There is also a need for further experimental and biochemical research in this field, and it is more than likely that the utilization of all three approaches will lead to the estab-

‖ Kleinfeld, M.: Unpublished data from personal observation.

¶ References 9 and 10.

lishment of better defined criteria and a clearer understanding of the mechanism and pathogenesis of talc pneumoconiosis.

SUMMARY AND CONCLUSIONS

The health status of a group of 32 talc workers, whose roentgenograms and histories were available after 14 years, were restudied, and the following data were obtained.

1. Nineteen of this group had died; their ages at death varied between 48 and 84 years. In four, whose death was believed due to pulmonary failure associated with talc pneumoconiosis, the ages varied between 60 and 79 years. Of the living, six are gainfully employed.

2. The outstanding clinical features involve the pulmonary and cardiovascular systems. Dyspnea and cough are frequent. Emphysema, basal rales, diminished chest expansion, and clubbing are the striking physical findings.

3. The characteristic chest x-ray is that of a diffuse infiltration of the basal and midlung fields, with occasional nodularities seen. Emphysematous bullae, hilar adenopathy, and cor pulmonale are not infrequent. Talc plaques, located in the region of the diaphragm, pericardium, and extending over a large part of the lung field, are consistently observed but do not reflect the degree of pulmonary involvement. Likewise, no correlation between symptomatology and x-ray findings is observed.

4. Abnormal electrocardiograms are seen in six and include patterns of right ventricular hypertrophy (two) and left ventricular hypertrophy (two); abnormal rhythm (one), and nonspecific T-wave alterations (one). The two persons demonstrating a right ventricular hypertrophy pattern had other features suggestive of cor pulmonale. There is a suggestive trend between degree of clubbing and abnormal electrocardiographic findings.

5. The peripheral blood findings are not remarkable and do not reflect the changes in the pathophysiology of this disorder.

6. In general, the workers appear well developed and well nourished in contrast to observations reported in 1941.

Although progression was observed in all, the clinical course is relatively slow, and in spite of the presence of moderate infiltration and pulmonary dysfunction, six of the workers are still actively employed.

The marked improvement in engineering methods is reflected in an appreciable decrease in the dust exposure. The pathological picture, however, did not retrogress with improvement of the industrial environment or upon retirement.

Assistance was rendered in the present study by Dr. Jesse R. Patton, of Ogdensburg, N. Y., by Mr. John Kean, Superintendent, and Miss J. Bishop, of the X-Ray Department of Edward Noble Hospital, Gouverneur, N. Y., and by our Industrial Hygiene Engineering Unit.

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Pneumoconios South India

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INTRODUCT

Pneumoconiosis has b condition in the Kolar G 1940 and continues to be only because up to now, a be considerable dust ret lungs of underground wor of 10 to 15 years, little countered. It does not itself as a clinical disease of the word but rather as dition. The potential dange with tuberculosis, which later.

The type of mining c Kolar Gold Field require working of quartz. The reefs, varying from a few width, which dip at a sha considerable length of tim depths, down to 9900 ft. and are being worked. A dry- and wet-bulb tempera the use of water in dust cluded.† The quartz reef ing widths of lode matte

Recorded for publication

Formerly Senior Assist Kolar Gold Field.

* The Kolar Gold Field is border of Mysore State, South 57′ N. and 78° 18′ E., at a he sea level.