Talc Pneumoconiosis

Significance of Sublight Microscopic Mineral Particles

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Described herein is a patient with talc pneumoconiosis whose symptoms began fifteen years after heavy exposure and who progressed to massive fibrosis and death seven years later from cardiopulmonary insufficiency. Studies of pulmonary function showed restriction of lung volumes, loss of pulmonary compliance and increased venous admixture with moderate hypoxemia. Cardiac catheterization revealed pulmonary hypertension. Lung tissue was analyzed for mineral content by x-ray diffraction and electron microscopy, which established the presence of talc in the absence of histologic demonstration of mineral particles by conventional light microscopy. The significance of submicroscopic talc particles in the production of disease is demonstrated and related to similar findings in asbestosis.

The lung disease which was first related in 1934 [1] to the inhalation of talc has been the subject of many clinical and environmental investigations [2–5]. Studies of lung function generally have been concerned with the value of screening tests, such as vital capacity and carbon monoxide diffusing capacity, in large numbers of exposed workers [6–8]. Our purpose is to present more complete physiologic findings, including ventilation-perfusion relationships and data on hemodynamics and lung mechanics, in a patient with severe respiratory impairment due to talc pneumoconiosis. Lung tissue from this patient was obtained at open lung biopsy and at necropsy. Talc could not be found on histologic examination by light microscopy, but was demonstrated in lung tissue by various mineralogic technics, including x-ray diffraction and electron micrography. Particle size was shown to be below the limit of resolution of light microscopy, illustrating the significance of submicroscopic particles in the production of pneumoconiosis due to talc. The importance of such submicroscopic fibers in the pathogenesis of pneumoconiosis due to asbestos has recently been demonstrated [9,10].

MATERIALS AND METHODS

The diagnosis of talc pneumoconiosis was established by (1) history of prolonged occupational exposure to dust shown to be predominantly true talc by x-ray diffraction; (2) roentgenographic appearance of extensive pulmonary infiltrations; (3) histologic findings of granuloma formation and fibrosis; and (4) mineralogic and electron microscopic demonstration of talc particles in lung tissue.

Measurements of lung volumes, maximum voluntary ventilation,

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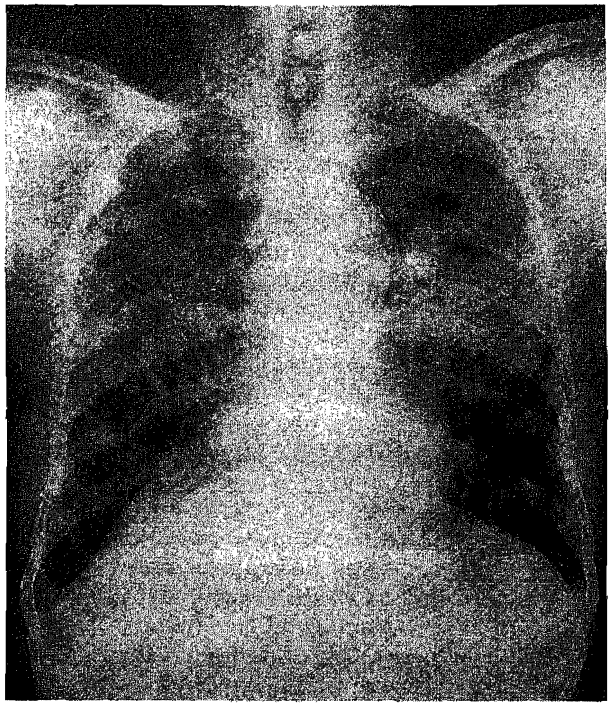

Figure 1. Chest roentgenogram demonstrating extensive fibrosis, with confluence in the mid-lung fields.

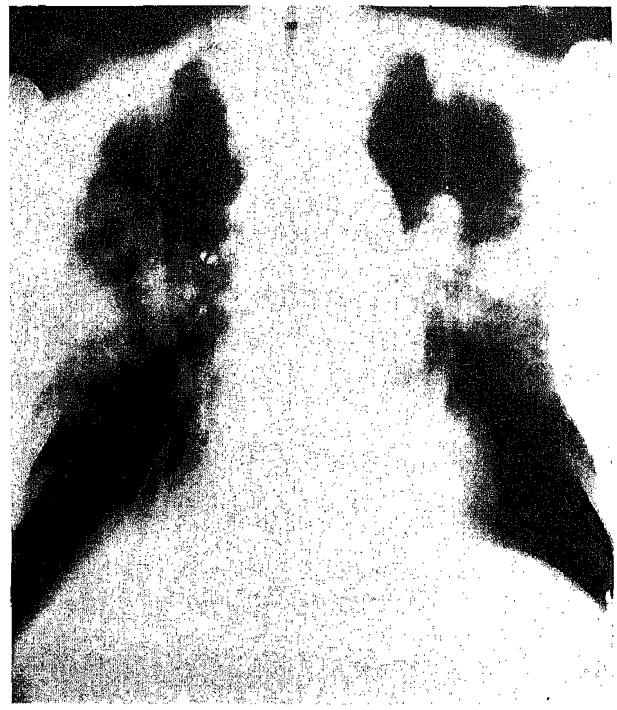

Figure 2. Tomogram of the chest showing hilar calcification and confluent fibrotic masses.

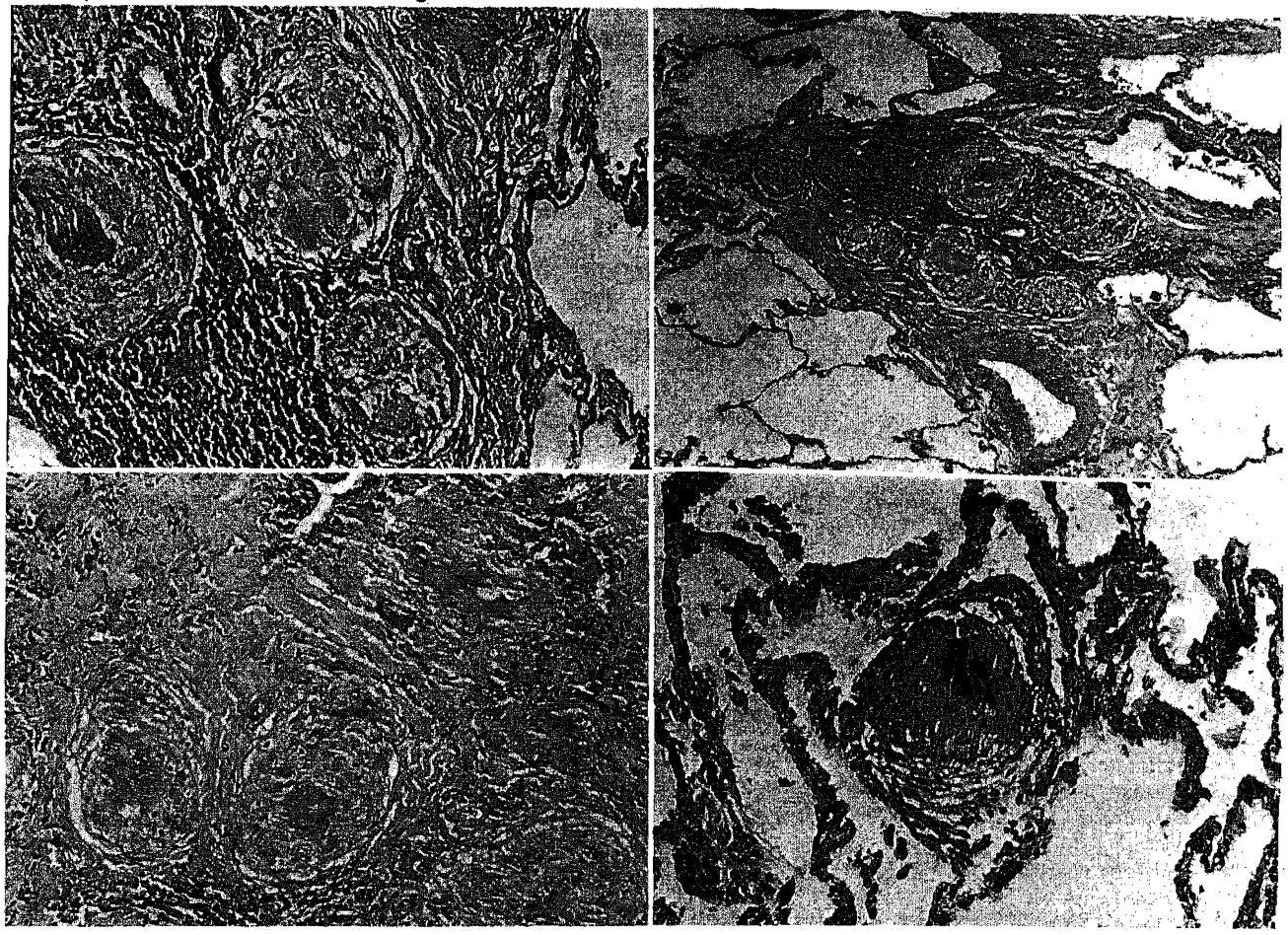

Figure 3. Lung tissue obtained by open lingular biopsy, under light microscopy, illustrating the granulomas, multinucleated giant cells, lymphocytic infiltration, fibrosis and changes in the alveolar septums.

TABLE I Lung Volumes and Mechanics

Data	Predicted Values	May 1960	May 1961	June 1961
Lung volumes				
Inspiratory capacity (cc)	...	1,075	1,145	1,050
Expiratory reserve volume (cc)	...	470	590	470
Vital capacity (cc)	3,800	1,460 (39)	1,510 (40)	1,475 (39)
Total lung capacity (cc)	5,020	...	2,355 (47)	...
Residual volume (cc)	1,220	...	620 (51)	...
Residual volume: total lung capacity × 100 (%)	<35	...	26	...
Maximum voluntary ventilation (L/min)	86	52 (61)	58 (68)	57 (67)
Dynamic compliance (L/cm H_2O)	0.15–0.20	0.027	0.028	0.023
Pulmonary resistance (cm H_2O/L/sec)	1.0–3.0	2.2	1.0	1.0

NOTE: Figures in parentheses represent percentage of predicted value.

index of intrapulmonary mixing, ventilation and arterial oxygen saturation and carbon dioxide content were performed according to the methods of Baldwin, Cournand and Richards [11]. The pH was measured with a Cambridge glass electrode, carbon dioxide tension (pCO_2) was obtained nomographically. Oxygen tension in the arterial blood was measured by Riley's direct technic [12]. Using two levels of inspired oxygen concentration, the ventilation-perfusion relationships and diffusing capacity of the lung for oxygen were calculated [13,14]. Lung compliance and pulmonary resistance were measured according to the method of Mead and Whittenberger [15]. Right heart catheterization was performed in the usual manner. Mineralogic methods and electron microscopy are discussed under "Mineral Analysis."

CASE REPORT

The patient, a white man, was born in 1899. From 1941 to 1950 he cleaned the factory ventilators which collected excess talc dust used in moulding and packaging rubber condoms. He used no mask and left this work because of rubber dermatitis.

Beginning in 1965, he noted increasing dyspnea. By 1961 he could not walk up one flight of stairs. He had occasional cough productive of scant white sputum and in the winter, wheezing and increased cough and expectoration. He was treated for tuberculosis in a sanitorium for two years because of his roentgenologic findings, but tubercle bacilli were never demonstrated and his illness progressed. The patient was then seen by one of us, who elicited the history of talc exposure. A provisional diagnosis of talcosis was made, and the patient was admitted to The Mount Sinai Hospital in New York in May 1961.

He was found to be underweight and markedly tachypneic. Diminished respiratory excursion and wheezing were noted, without clubbing or cyanosis. Routine hematologic and chemical studies of the blood were normal. Roentgenograms and tomograms of the chest showed calcified mediastinal nodes with symmetrical primarily mid-lung linear and confluent densities (Figures 1 and 2).

Open lung biopsy (lingula) was performed on May 18, 1961. The pleural cavity was free except for apical adhesions. Tiny nodules were palpable in a pink lingula. Culture of lung tissue for tubercle bacilli was negative.

Histologic examination (Figure 3) showed fibrosis of lung parenchyma with large granulomas characterized by aggregates of epithelioid and multinucleated giant cells, surrounded by a thin rim of lymphocytes. Between these foci, the lung was emphysematous, with thin alveolar septums. The pleura was thickened and fibrotic, and contained numerous granulomas similar to those in the pulmonary parenchyma. Talc bodies or crystals could not be demonstrated by light microscopy.

Following this hospitalization, progressive cor pulmonale developed and the patient died elsewhere one year later of cardiorespiratory insufficiency. Postmortem examination was performed, and the lungs were preserved in a frozen state for mineralogic analysis at a later date.

RESULTS

Pulmonary Function Studies. Pulmonary function tests were performed on several occasions over a fourteen month period. Results are shown in Tables I to III. Vital capacity was consistently reduced to approximately 40 per cent of normal. Residual vol-

TABLE II Ventilation-Perfusion Relationships

Measurement	Normal	Observed
Ventilation (L/min/M^2)	3.3	6.6
Index of intrapulmonary mixing (% N_2)	<2.5	<2.5
Oxygen consumption (cc/min/M^2)	130	174
Physiologic dead space (PDS/TV × 100) (%)	<30	38
Alveolar-arterial pO_2 gradient, room air (mm Hg)	<15	23
D_LO_2 (cc/min/mm Hg)	>15	17
Venous admixture (% cardiac output)	<6	18

TABLE III Arterial Blood Gas Studies (1961)

Data	Room Air (Normal)	Room Air		14% Oxygen		25% Oxygen	100% Oxygen	Exercise	
		May	June	May	June	June	May	May	June
Oxygen content (vol %)	19	19.1	18.1	16.4	15.1	18.8	22.2	19.1	17.0
Oxygen capacity (vol %)	20	21.5	20.5	21.6	19.8	20.5	21.6	21.6	20.2
Oxygen saturation (%)	95–97	90	90	78	78	93	100+	90	81
pO_2 (mm Hg)	85–100	68	...	46	...	...	...	...	...
Serum CO_2 content (vol %)	55–60	53	52	50	52	54	...	...	...
pCO_2 (mm Hg)	37–41	37	35	34	36	36	...	...	...
pH	7.37–7.41	7.41	7.42	7.43	7.42	7.42	...	...	...

TABLE IV Hemodynamic Studies

	Pulmonary Artery Pressure (mm Hg)			Right Ventricular Pressure (mm Hg)		Cardiac Output (L/min)	Cardiac Index (L/min/M²)
	Systolic	Diastolic	Mean	Systolic	Diastolic		
21% oxygen	34	17	25	35	4	5.13	3.22
14% oxygen	47	26	30	...	...	5.40	3.40
25% oxygen	39	23	28	...	...	4.91	3.09
Exercise (3 min)	...	...	30	...	...	...	...
Exercise (3 min)	...	...	41	...	...	...	...

TABLE V Mineralogic Analysis

Fraction	Tissue Weight (gm)		
	Thawed Wet	Dried, 56°C	Ashed 52 Hours
1	24.182	6.963	1.883
2	22.264	6.272	1.356
3	22.093	6.774	1.982
Total	68.539	20.009 (29.2%)*	5.221 (7.6%)

Residue	Powder (−300 Mesh)	Granules
Weight (gm)	1.132	4.089
% of ashed residue	21.7	78.3
% of wet weight	1.6	6.0

* Per cent of original weight.

ume (RV) and total lung capacity (TLC) were proportionately reduced, giving a normal RV:TLC ratio. Maximum voluntary ventilation was consistently reduced to two-thirds of normal. Air trapping was not seen on the spirogram. Dynamic pulmonary compliance was markedly diminished, and pulmonary resistance was normal.

The patient hyperventilated at rest, and during and after standard exercise. Resting hyperventilation was unaffected by increasing the oxygen concentration of the inspired air. The breathing reserve was diminished during and on recovery from standard exercise. Resting oxygen consumption was increased.

Oxygen saturation was moderately reduced at rest (90 per cent). After exercise, it remained unchanged on one occasion and was further reduced on another. Serum carbon dioxide content and pCO_2 were reduced, and pH was slightly alkalotic, indicating a partially compensated respiratory alkalosis.

Distribution of inspired air, as measured by the index of intrapulmonary mixing, was normal. The alveolar-arterial oxygen tension gradient was increased with the patient breathing 21 per cent oxygen and normal when breathing 14 per cent oxygen. Venous admixture was increased to 18 per cent of total cardiac output. The diffusing capacity of the lung for oxygen at rest was normal. Physiologic dead space was increased.

Hemodynamic data are shown in Table IV. Pulmonary artery pressure was moderately elevated while breathing room air at rest (systolic 34, diastolic 17, mean 25 mm Hg). It rose further on breathing 14 per cent oxygen and did not fall on breathing 25 per cent oxygen. Pulmonary artery pressure was measured twice during exercise. On each attempt, exercise had to be discontinued after three minutes because the patient was too dyspneic to continue. Mean pulmonary artery pressure reached 30 and 41 mm Hg, respectively.

Right ventricular end-diastolic pressure and cardiac index at rest were normal.

Mineralogic Analysis. Preparation: The lungs were frozen at necropsy. At a later date, three samples were taken from the lower lobe of the left lung for mineral analysis. After thawing, wet weight was determined. The specimens were dried at 56°C to constant weight (dry weight), and then ashed to constant weight in an electronic ashing device (Table V).

Gross appearance: The ashed residue was made up of two distinct types of material (Figure 4): light gray hard granules, 0.5 mm to 1 cm in diameter and 78 per cent by weight, and a brown-gray fine powder, —300 mesh in diameter (53 μ in the ASTM sieve series), 22 per cent by weight.

Optical examination: Under the polarizing microscope the fine powder was seen to consist of round and irregular particles, 3 μ average size, with rare fibers. However, most particles were of a size range below the limit of resolution of the light-optical system (<0.5 micron), indicated by scattering of light. These size characteristics explain the absence of talc on routine histologic examination.

The granular material consisted of large, irregular refringent particles. This morphology and the nature of extinction character between crossed Nicol prisms suggest that these granules derived from fibrotic and calcified tissue.

X-ray diffraction: Using a Norelco x-ray diffractometer, patterns obtained from the residue powder were compared with standard mineral types. Comparison with a talc standard at 9.4 Å (Figure 5) indicated that the mineral talc comprised 20 to 50 per cent of the residue powder. Up to 0.80 per cent of the wet weight of lung was talc. Some anthophyllite was present as well.

Electron microscopy: Electron microscopic examination of the extracted residue powder was made with an RCA EMU 3G electron microscope. Dust was dispersed in distilled water and pipetted onto formvar-coated 200 mesh copper grids. Examination of the air-dried grids was made at accelerating voltages of 100 kv and transmission photographs obtained.

These photographs (Figure 6) clearly show the presence of talc plates (arrows). Also visible are some fibrous particles with the morphologic characteristics of the asbestos type minerals, anthophyllite or tremolite. These minerals are almost universally associated with talc. The juxtaposition of fibers and plates is probably an artifact produced during separation. The morphology of the talc plates is identical with that of talc standards [16,17]. The larger talc particles were observed to be about 1 to 2 μ in greatest dimension; talc particles less than 1 μ in size occurred frequently.

COMMENTS

True talc is a specific compound, hydrous magnesium silicate, $Mg_6(Si_8O_{20})(OH)_4$, which forms crystal plates. As commercially available, depending on where it was mined, "talc" powder also contains varying amounts of asbestos minerals, which form chains or fibers and resist heat and chemical agents. These include anthophyllite (hydrous magnesium silicate in another form, $Mg_7(Si_8O_{22})(OH)_2$) and tremolite (calcium magnesium silicate, $Ca_2Mg_5(Si_8O_{22})(OH)_2$). Commerical talc may therefore have an

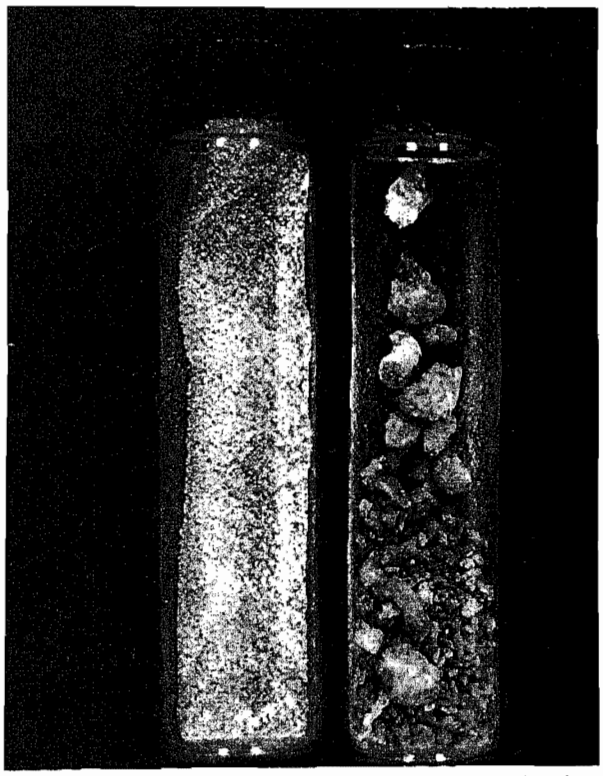

Figure 4. Residues after ashing of lung. On the left is a fine gray powder, —300 mesh in size, which is the mineral residue; 20 to 50 per cent of this is true talc. On the right are granules which are the debris of fibrotic and calcified tissue.

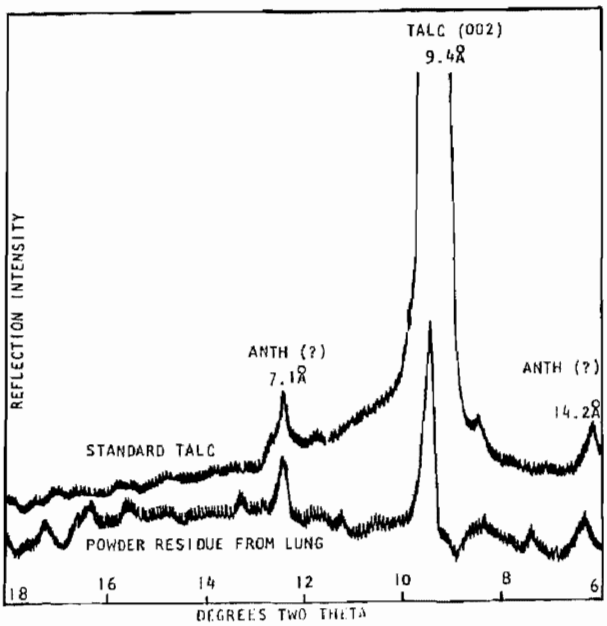

Figure 5. X-ray diffraction tracing of mineral dust residue compared with talc standard, showing a predominance of true talc (9.4 Å) with some anthophyllite (7.1 and 14.2 Å) which is also present in the standard. Abscissa indicates angle at which maximum x-ray reflection occurs; ordinate shows the relative intensity of x-ray reflection.

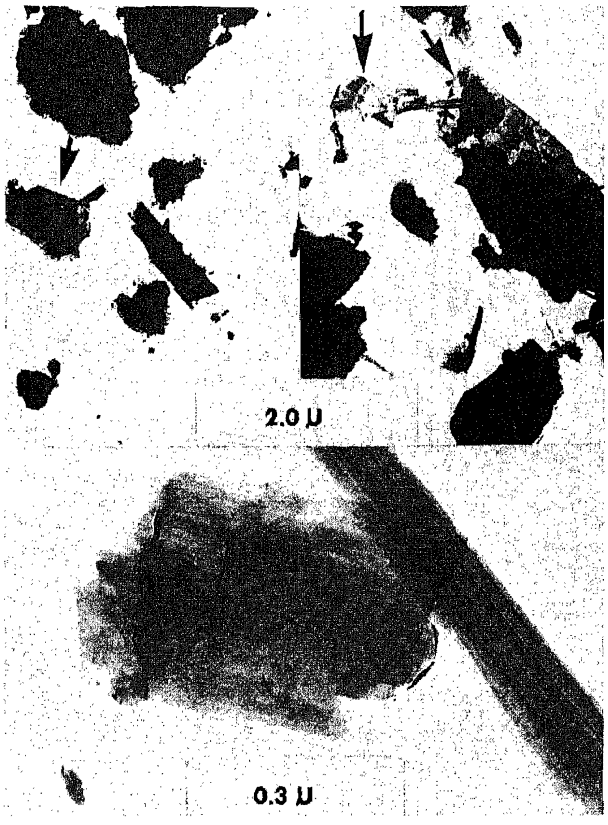

Figure 6. Transmission electron photographs of recovered talc plates and asbestos-type fibers. The upper two photographs clearly show (arrows) the presence of talc plates. The lower photograph shows a small talc particle touching an asbestos-type fiber. Scales are as marked.

asbestos-like effect on lung tissue [18,19]. The dust to which our patient was exposed was shown to be predominantly true talc, with some anthophyllite.

The degree of pulmonary fibrosis produced by the various minerals present in commercial talc depends on particle size. Schepers and Durkan [20] demonstrated this by insufflating these minerals into the tracheas of guinea pigs. Tremolite and anthophyllite, with visible fibers up to 50 μ long, produced extensive fibrosis, whereas the smaller crystals of true talc produced a primarily cellular response. All three induced perivascular, peribronchiolar and subpleural lesions. The extent to which the changes observed were due to sublight microscopic particles could not be appreciated. Both commercial talc and true talc produce pneumoconiosis, although "fibrous" commercial talc of high tremolite and anthophyllite content may be more fibrogenic [5,6,21]. Particles below 5.0 μ in diameter have been considered most harmful [22], but knowledge on this question is still inadequate. In addition to particle size, the concentration of dust obviously is important. Wet drilling has been employed to reduce the concentration of dust [21].

Talc was first implicated in lung disease in 1896 [23]. Pneumoconiosis caused by silicates, rather than by free silica, was first emphasized in 1927 [24]. In 1934 Merewether [1] found interstitial infiltrates in chest roentgenograms of rubber tire workers exposed to talc dust. Dreessen and Dalla Valle in 1935, in a survey of sixty-six workers in two Georgia talc mills and mines, found eight with disabling pneumoconiosis and fourteen with x-ray changes [2]. Many surveys of exposed workers have since appeared, the most recent being those of Kleinfeld et al. [5,7,8,25].

Talc pneumoconiosis has been described in many varied occupations, such as: (1) talc mining and milling [2,4]; (2) rubber cable and tire manufacture, especially among lay-up men who layer rubber goods with talc for storage [1]; (3) plate casting, in which the moulds are dusted with talc before pouring [26]; (4) cosmetic manufacture [27]; (5) textile manufacture [28]; (6) sailors dusting life rafts with talc [29].

Talc is widely used in other industries, as filling for paper, soaps and paints; in electric switchboards; in insulation of steam pipes; and in leather finishing.

The pathologic findings previously described in talc pneumoconiosis are those of progressive chronic inflammation with granuloma formation and fibrosis, causing destruction of large areas of lung parenchyma [19,27,30]. Pleural adhesions, bleb formation, pulmonary atheromatosis, right ventricular dilatation and hypertrophy, hilar adenopathy and marrow hyperplasia are seen. The earliest change is thickening of the alveolar walls with fibrocytic and histiocytic infiltration. Solid masses form as alveolar walls thicken and alveolar spaces disappear. Necrobiosis may occur in the large avascular fibrotic masses [27,30].

The pathologic changes are similar to those in chronic sarcoidosis. However, in talcosis doubly refractile needle-shaped talc crystals, 0.5 to 5 μ in length, may be visible on light microscopy. Particles smaller than 0.5 μ are not recognized by light microscopy and may account for significant disease, as illustrated by the case presented here. Indeed, it is unlikely that larger particles can be present without submicron particles and the pathogenetic contribution of each is not yet well understood.

The significance of other mineral particles invisible to the light microscope, e.g., asbestos fibers, has recently been appreciated [9,10]. The presence of such particles in inspired dust would ordinarily not be recognized. Furthermore, in experimental asbestosis, even when instilled fibers were large enough to be visible by light microscopy, the majority of fibers in the alveoli were seen only by electron micrography, suggesting that the larger fibers break up into smaller fragments or fibrils. These

sublight microscopic particles are then phagocytosed, especially by alveolar macrophages, and segregated into intracytoplasmic vacuoles. There, coating with hemosiderin granules results in the typical asbestos body. "Talc bodies" have also been described in talcosis due to fibrous talc [20]. Repeated cycles of phagocytosis and release may be related to the cellular proliferation and fibrosis observed histologically [10]. In a recent review of "ferruginous" bodies, Gaensler and Addington [31] emphasized the fibrogenicity of the smallest uncoated fibers, which are invisible to light microscopy.

Clinical Features. As in our patient, dyspnea is the first and most distressing symptom; it may rarely be present with a normal chest roentgenogram [6]. Other symptoms are productive cough, hemoptysis, chest pain, wheezing and fatigue.

Physical signs are nonspecific and may include decreased thoracic and diaphragmatic excursion, decreased or harsh breath sounds, rhonchi, wheezes, prolonged expiration, rales, dullness on percussion, bronchial breathing, tachypnea, cyanosis and clubbing.

Early roentgenologic changes include reticulation, linear infiltration and/or nodules which are less dense and well defined than those in silicosis. These nodules are variable in size and may conglomerate into large masses, as in our patient (Figures 1 and 2). The apices and costophrenic angles seem least involved. Fibrosis may be extensive and bullae may be seen. Calcified pleural plaques were found in 6.3 per cent of workers in tremolite talc mines [4].

The clinical diagnosis is based on the history of exposure to talc dust and the roentgenologic findings and should be confirmed by the demonstration of talc. Although sputum and scalene node biopsy specimens occasionally show talc crystals [26,32], talc is best demonstrated by obtaining lung tissue for routine histologic, electron microscopic and mineralogic examination. As in our case, histologic appearance on light microscopy may be nonspecific and suggest sarcoidosis or idiopathic interstitial fibrosis, but a specific diagnosis can be established by electron microscopy and appropriate mineralogic procedures.

The clinical course of talc pneumoconiosis is variable. There may be only slight fibrosis after many years of exposure, and the condition may seem to stabilize when exposure ceases. Kleinfeld et al. [33] found slow progression to be the rule, despite termination of exposure. Other cases rapidly progress to massive fibrosis and cor pulmonale after relatively brief exposures [26,32]. Death is often from cardiopulmonary insufficiency, as in our patient. The incidence of malignancy of the lung and pleura is increased in talcosis, as is true in asbestosis [34].

Pulmonary Function. Alivisatos et al. [32] found arterial oxygen unsaturation at rest in three of seven patients with talc pneumoconiosis, and unsaturation after exercise in all seven. Vital capacity and maximum voluntary ventilation were reduced, resting ventilation was increased, and all affected patients were severely dyspneic after standard exercise, findings similar to those in our case. Diffusing capacity, lung mechanics and hemodynamics were not studied.

Kleinfeld et al. [6–8] reported reduced vital and total lung capacity and decreased carbon monoxide diffusing capacity in many of their exposed workers. As expected, pulmonary function tests were more deranged in those with abnormal roentgenograms, but functional impairment was found without roentgenographic abnormality [6–8] as is also the case in asbestosis [35].

In our patient, lung volumes were uniformly reduced, with no evidence of air trapping, hyperinflation or airway obstruction. The maximum voluntary ventilation was less reduced than the vital capacity. Pulmonary compliance was markedly decreased. These results are consistent with the replacement of lung tissue by the extensive fibrosis found on roentgenographic and histologic examination.

Ventilation-perfusion relationships in our patient were impaired, with increased venous admixture and physiologic dead space. Diffusing capacity for oxygen was normal at rest. This finding is at variance with the reports of Kleinfeld et al. [6–8], who used the single breath carbon monoxide technic which would not give information concerning ventilation-perfusion relationships and venous admixture.

Pulmonary hypertension was observed and persisted with inhalation of 25 per cent oxygen, suggesting some degree of fixed involvement of pulmonary vessels.

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REFERENCES

1. Merewether ERA: Annual Report, Chief Inspector of Factories and Workshops for England and Wales. London, His Majesty's Stationery Office, 1933, p 63, 1934, p 65.
2. Dreessen WC, Dalla Valle JM: Effects of exposure to dust in two Georgia talc mills and mines. Pub Health Rep 50: 131, 1935.
3. Porro FW, Patton JR, Hobbs AA: Pneumoconiosis in

the talc industry. Amer J Roentgen 47: 507, 1942.
4. Siegal W, Smith AR, Greenburg L: The dust hazard in tremolite talc mining, including roentgenological findings in talc workers. Amer J Roentgen 49: 11, 1943.
5. Messite J, Reddin G, Kleinfeld M: Pulmonary talcosis. A clinical and environmental study. Arch Industr Health 20: 408, 1959.
6. Kleinfeld M, Messite J, Shapiro J, Kooyman O, Swencicki R: Lung function in talc workers. A comparative physiologic study of workers exposed to fibrous and granular talc dusts. Arch Environ Health 9: 559, 1964.
7. Kleinfeld M, Messite J, Shapiro J, Swencicki R, Sarfaty J: Lung function changes in talc pneumoconiosis. J Occup Med 7: 12, 1965.
8. Kleinfeld M, Messite J, Shapiro J, Swencicki, R: The effect of talc dust inhalation on lung function. Arch Environ Health 10: 431, 1965.
9. Suzuki Y, Churg J: Structure and development of the asbestos body. Amer J Path 55: 79, 1969.
10. Holt PF, Mills J, Young DK: Experimental asbestosis with four types of fibers: importance of small particles. Ann NY Acad Sci 1932: 87, 1965.
11. Baldwin E de F, Cournand A, Richards DW Jr: Pulmonary insufficiency. I. Physiological classification, clinical methods of analysis, standard values in normal subjects. Medicine (Balt) 27: 243, 1948.
12. Riley RL, Proemmel DD, Franke RE: A direct method for determination of oxygen and carbon dioxide tension in blood. J Biol Chem 161: 621, 1945.
13. Riley RL, Cournand A: Analysis of factors affecting the concentration of oxygen and carbon dioxide in the gas and blood of the lungs. I. Theory. J Appl Physiol 2: 77, 1951.
14. Riley RL, Donald KW, Cournand A: Analysis of factors affecting the concentration of oxygen and carbon dioxide in the gas and blood of the lungs. II. Methods. J Appl Physiol 2: 102, 1951.
15. Mead J, Whittenberger JL: Physical properties of human lung measured during spontaneous respiration. J Appl Physiol 5: 779, 1953.
16. Cralley LJ, Key MM, Groth DH, Lainhart WS, Ligo RM: Fibrous and mineral content of cosmetic talcum products. Amer Indust Hyg Ass J 29: 350, 1968.
17. Beutelspacher H, Van Der Morel HW: Atlas of Electron Microscopy of Clay Minerals and Their Admixtures, New York, Elsevier Pub Co, 1968, Fig 135 and 136, p 142.
18. Schulz RZ, Williams CR: Commercial talc: animal and mineralogical studies. J Industr Hyg & Toxicol 24: 75, 1942.
19. Schepers GWH, Durkan TM: The effects of inhaled talc mining dust on the human lung. Arch Industr Health 12: 182, 1955.
20. Schepers GWH, Durkan TM: Experimental study of the effects of talc dust on animal tissue. Arch Industr Health 12: 317, 1955.
21. Weiss B, Boettner EA: Commercial talc and talcosis. Arch Environ Health 14: 304, 1967.
22. Sayers RR, Jones RR: Pub Health Rep 53: 1453, 1938.
23. Thorel C: Die Specksteinlunge. Beitr Path Anat 20: 85, 1896.
24. Badham C: Notes on a fine type of pneumoconiosis produced by silicates and other minerals. Report. Director General Public Health N. S. Wales, Australia, 13: 102, 1927.
25. Kleinfeld M, Giel CP, Majeranowski JF, Messite J: Talc pneumoconiosis. A report of 6 patients with post-mortem findings. Arch Environ Health 7: 101, 1963.
26. Hunt AC: Massive pulmonary fibrosis from the inhalation of talc. Thorax 11: 287, 1956.
27. Millman N: Pneumoconiosis due to talc. Occup Med 4: 391, 1947.
28. Mann B, Deasy JB: Talc pneumoconiosis in the textile industry. Brit Med J 2: 1460, 1954.
29. Lyons HA: Talc pneumoconiosis. Milit Surgeon 113: 393, 1953.
30. Seeler AO, Gryboski JS, MacMahon HE: Talc pneumoconiosis. Arch Industr Health 19: 392, 1959.
31. Gaensler EA, Addington WW: Current concepts: asbestos or ferruginous bodies. New Eng J Med 280: 488, 1969.
32. Alivisatos GP, Pontikakis AE, Terzis B: Talcosis of unusually rapid development. Brit J Industr Med 12: 43, 1955.
33. Kleinfeld M, Messite J, Tabershaw IR: Talc pneumoconiosis. Arch Industr Health 12: 66, 1955.
34. Kleinfeld M, Messite J, Kooyman O, Zaki MH: Mortality among talc miners and millers in New York State. Arch Environ Health 14: 663, 1967.
35. Bader ME, Bader RA, Teirstein AS, Miller A, Selikoff IJ: Pulmonary function and radiographic changes in 598 workers with varying duration of exposure to asbestos. J Mount Sinai Hosp NY 37: 492, 1970.