THE MORPHOLOGY OF BAUXITE-FUME PNEUMOCONIOSIS *

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A lung disease associated with the manufacture of alumina abrasives was uncovered recently through an industrial survey by Shaver and Riddell.[1,2] The manufacture of these abrasive powders, the principal ingredient being Arkansas bauxite, was greatly increased in the later years of World War II, and resulted in a new and lethal pulmonary hazard with distinct clinical, radiologic, and morphologic findings. The salient features of the morbid anatomy in 6 fatal cases are brought forward in this contribution. The industrial exposure, symptomatology, case histories, radiologic findings, and chemical assay are given briefly to complete the background of this new entity.

In the industrial process involved, an abrasive, made up of an aluminum oxide known as "corundum," is being produced. The processing is carried out in electric furnaces with the mix consisting of finely ground bauxite, iron, and coke. Carbon electrodes are lowered to the surface of the mix and fusion occurs at a temperature of 2000°C. Dense white fumes are evolved during this process, leading to contamination of the furnace rooms. All fatal cases have occurred in furnace feeders or crane operators.

The clinical syndrome common to all is shortness of breath, cyanosis, substernal discomfort, and recurrent episodes of spontaneous pneumothorax from rupture of emphysematous bullae. Percussion and auscultation of the chest reveal a variety of signs dependent upon the underlying parenchymal changes and the presence or absence of air in the pleural cavities. The radiologic evidence is so startling and different from that in other pneumoconiotic films that Shaver[1] characterized this dust disease as individual and distinct from the more common silicosis. Diffuse, irregular, lace-like, and granular shadows, greatly increased width of mediastinum, and collapse of the lung are the cardinal features of the roentgenogram.

The parenchymal fibrosis of the lungs is unlike anything previously encountered (Figs. 1 and 2).

* Received for publication, May 11, 1948.
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Report of Cases

CASE 1

H. B., a Norwegian, had spent the last 11 years of his life in the abrasive-manufacturing furnace rooms. The terminal 5 years of employment were as a crane operator above the charged steel pots. Past history, with reference to the lungs, revealed an attack of influenzal pneumonia in 1918, influenza in 1926, work in underground mines for 11 years, and intermittent rock drilling in a canal area for 12 years, but neither of these occupations entailed exposure to dusts high in silica.

Gross Examination of the Lungs

The lungs were heavy, indurated, puckered, and gray-black. The scar tissue in the interlobular septa gave a granular, pebbled appearance to the lung surface covered by thickened pleura. Pigmentation was heaviest in the regions of localized subpleural scars which extended downward into the underlying parenchyma. No pleural nodules were discernible. On section, the pulmonary tissue was pigmented. Wide regions of parenchyma had been replaced by irregular, sweeping strands and masses of dense, gray-black fibrous tissue. These masses of homogeneous scar were generally located midway between the hilum and the periphery of the lung and consisted of branching bands 2 to 4 cm. in width, with finer prolongations extending peripherally through the interlobular septa to the pleura and mesially along the walls of bronchi and blood vessels to the hilum. No discrete nodules were discovered. Between the larger areas of conglomerate fibrosis and the pleura were irregular zones of coarse emphysema. In a few areas, particularly toward the hilum, the parenchyma was gray-yellow, suggestive of pneumonic consolidation. The bronchi were of normal caliber and contained a grayish coagulum of mucoid material. The vessels showed varying degrees of perivascular scar thickening. All were patent. The tracheobronchial lymph nodes were slightly enlarged, firm, black, and without white, whorled nodules.

Microscopic Description

Microscopically, the fibrosis was diffuse, obliterated the normal architecture (Fig. 3), and was hyaline, with nothing to suggest its origin. Black pigment, obviously exogenous, was distributed in masses of charged phagocytes trapped between the heavy, swollen, collagen bands. No nodule formation as in silicosis was discovered, although the hyalinization of collagen simulated that condition to a certain extent. At the margins of larger masses the fibrous tissue extended into the septa between alveoli, greatly thickening their walls and encroaching upon the air spaces. The lung parenchyma showed marked septal swelling due

to intracellular edema and numerous young fibroblasts. There was irregular focal deposition of eosinophilic collagen. In some regions the acellular fibrotic tissue contained trapped lymphoid knots and dilated capillaries. The dense abutments of fibrous tissue were covered with stretched septal cells. Dilated emphysematous sacs were caught between the dusky hyaline strands of fibrous tissue. Pyknotic alveolar cells were trapped in the bundles of dense scar tissue. The alveolar spaces contained macrophages filled with black particles, numerous giant cells surrounding empty crystal clefts, and considerable cellular débris. Small isolated groups of alveoli revealed evidence of an acute pneumonia, but this feature was never pronounced. Dust cells were present in the foci of pneumonitis. The lung parenchyma in other regions was not remarkable except for alveolar capillary congestion.

The bronchi, surrounded by dense fibrous tissue, were filled with mucus and possessed a thickened submucosa, heavily infiltrated with lymphoid cells. The smaller bronchi caught in areas of fibrosis were distorted and filled with inspissated basophilic mucus (Fig. 4).

Arteries frequently revealed endarteritis obliterans, which involved chiefly the larger branches. Medial proliferation and hyalinization were the predominant vascular changes.

The tracheobronchial lymph nodes were remarkable chiefly for large accumulations of dust-filled phagocytes within the follicular structures. Scattered throughout the lymph nodes were minute patches of diffuse hyaline fibrosis, and a few of the lymph channels were lined with thin hyaline membranes. No silicotic nodules with redundant fibrosis were encountered.

CASE 2

J. F., a white male, 42 years old, was a furnace feeder through the years 1940 to 1944. Antecedent history was of no significance. This man's death was hastened by an acute respiratory illness, 1 year after removal from his industrial occupation.

Gross Examination of the Lungs

The surfaces of the lungs were covered by thickened pleura with remnants of adhesions. The parenchyma generally was replaced by heavily pigmented, diffuse, gray-black scar tissue without nodulation. Thick-walled bronchi and blood vessels traversed this diffuse scar tissue. In most places the fibrosis was so generalized that no crepitant lung remained. Near the periphery there were groups of dilated air sacs. Beneath the pleura in a few spots the tissue was soft, compressed, ragged, and markedly distorted, but the general appearance was that of collapsed bullous emphysema. At the bases of the lungs there was little parenchy-

mal distortion. There were numerous lobules of apparently acute pneumonia, red to yellow, in the basal gutters. The bronchial nodes were moderately enlarged, heavily pigmented, but not excessively hard. No trace of nodular fibrosis was detected.

Microscopic Description

There were numerous large areas of conglomerate fibrosis seen on microscopic examination. These masses of dense scarring were strung out in a coarse network. Heavy strands of hyaline fibrous tissue radiated throughout the parenchyma. At their margins, where the architecture of the lung was still recognizable, alveolar walls were extremely thick and composed of hyaline tissue. Deposits of black dust were heaviest in the large masses of fibrotic material. A foreign body giant cell reaction surrounded lozenge-shaped, empty clefts which were principally intra-alveolar in distribution and associated with the polynuclear pneumonic reaction. Giant and foam cells were not found within the septa.

Widespread septal hyalinization was noteworthy. This intrinsic septal change compressed, distorted, and obliterated the capillary pathways and in some regions these lesions were grouped together to form cores of ramifying, dense fibrous tissue. Large, subpleural, emphysematous areas with delimiting borders of hyaline septal tissue were frequent.

CASE 3

P. H., a white male, 33 years of age, had been a furnace feeder for 3 years. Past history was irrelevant. In the last year of employment there was a progressive increase of dyspnea, cyanosis, and sporadic incapacitating episodes of spontaneous pneumothorax. His condition rapidly deteriorated and death ensued in November, 1943.

Gross Examination of the Lungs

The lungs were similar in outline, weight, and tissue texture to those described in cases 1 and 2. They weighed 450 gm. each, and were dark with diffuse, rubbery texture. Pleural thickening was marked, with adhesions in the interlobar fissures. Innumerable emphysematous blebs were noted over the upper portions. The lungs cut with increased resistance. Dense bands of scar tissue were found, particularly in the upper portions, fanning out to the periphery. The bronchial tree and blood vessels were negative. The hilar lymph nodes were negative.

Microscopic Description

Microscopically, an important feature of the parenchyma was the distortion of the alveolar walls with an intense mononuclear and lymphocytic infiltrate along with swollen alveolar lining cells (Fig. 5). This

inflammatory septal lesion frequently was found between dense bands of old fibrous tissue which formed suspensory strands. The increased collagen was pericapillary in many of the affected septa. In some areas the collagen formed an eosinophilic cast of the septa; in other regions the septa were broad with a loose reticular and lymphoid stroma covered by swollen cuboidal alveolar cells. The lung parenchyma was distorted by giant emphysematous pockets arranged either in a fragmented cystic manner or as a latticework. These foci were lined with a dense fibrotic layer. Vessels, bronchi, and lymph nodes were negative.

CASE 4

R. L., a white male, 61 years old, had been a foreman in the alumina abrasive manufacturing plant for 15 years. For the last 4 years this man had worked as a furnace feeder. Previous to World War II, radiologic examination of his chest had revealed increased bronchovascular markings and some fine streaks of shadowing, but no specific diagnosis was made. This worker was symptom-free prior to 1941. His condition deteriorated progressively due to increased respiratory difficulty. He was totally incapacitated for the last 2 years of life and died in 1945.

Gross Examination of the Lungs

The previously fixed lung specimens were received in two portions, both resembling irregular blocks of black India rubber. The consistency was firm throughout. In the peripheral part of one region there were many dilated, ramifying bronchi measuring up to 0.6 cm. in diameter. A few subpleural, caseous, white foci were noted up to 0.1 cm. in diameter. Projecting from the medial and basal portions of the lungs were a number of emphysematous bullae measuring up to 2.0 cm. in diameter. The pleural surface was opaque, granular, and in some places had tough, grayish white and stringy tag ends of adhesions. The granularity was particularly marked over the vertebral borders. The opacity and stringy adhesions were enhanced over the anterior borders. The bronchi and blood vessels were incarcerated in this noncrepitant, gray-black, resilient tissue.

Microscopic Description

Microscopically, there were four salient features in this lung: (1) Of cardinal importance was the fine, diffuse, septal fibrosis and marked hyalinization. This septal change probably represented the earliest lesion, and the agmination of these infiltrated hyalinized septa led to massive conglomerate fibrosis (Fig. 6). Extracellular carbon drifts and birefringent particles accompanied the massive scar tissue (Fig. 7). Cubical alveolar lining cells were a feature of the secondary parenchymal changes. This reaction was most intense in association with the dense, conglom-

erate fibrosis. Occasional giant cells arose from these cubical cells. (2) There was massive fibrosis as in the other cases. This diffuse, radiating fibrosis was old, in some places hyalinized. No amyloid was encountered. (3) A banal inflammatory infiltration of the alveolar septa by mononuclear and plasma cells was noted. In many regions this reaction was within the dense, broad bands of scar tissue. In many regions the septa showed stuffed, swollen, club-shaped outlines filled with a mixed chronic inflammatory infiltrate. (4) Evidence for tuberculosis was given by clear-cut histopathologic criteria, consisting of caseous foci, with surrounding giant cell systems. In these regions there was a definite attempt at a peripheral defense fibrosis with drifts of black pigment incorporated. The morphologic features of tuberculosis were dominant and virgin in these areas and certainly not modified as in the koniophthisis of Belt.[3] The shadows of scarred pulmonary parenchyma were still present despite caseation, which indicated tuberculosis to be a late sequel. Endobronchial tuberculosis was found also. Extreme emphysema was noted in some regions and endarteritis obliterans was noted in many of the vessels. Lymphoid follicles, free of reaction, were scattered throughout the lung. The bronchial lymph nodes showed reactive hyperplasia, minimal hyaline fibrosis in the littoral spaces, and freedom from nodule formation.

CASE 5

N. C., a white male, 41 years old, had been a furnace feeder for 6 years and a crane operator in the furnace room for 4 years. Past history was irrelevant. For a period of 1½ years after December, 1945, there was progressive increase in respiratory symptoms and signs accompanied by lace-like shadows in the radiologic chest plate. This workman died 2 years after removal from industrial exposure.

Gross Examination of the Lungs

The right lung weighed 675 gm., with the pleura adherent over the upper portion. The remaining pleural surface was opaque. Along the lateral border there were a number of blebs measuring up to 1.5 cm. in diameter. The entire lung was of homogeneous rubbery consistency and cut sections revealed a diffuse, indurated, gray to rusty brown color. Neither nodules nor any gross evidence of tuberculosis could be found. The bronchi and vessels were negative. The left lung weighed 620 gm. and was similar in all respects to the right lung. The lymph nodes in the hilar regions were soft, gray-black, and somewhat enlarged.

Microscopic Description

On microscopic examination, the entire lung parenchyma was stiffened by disseminated, variegated, septal scarring. Many of the alveolar walls

contained young fibroblasts and capillary buds, or showed spotty hyalinization. In other regions fragmented, eosinophilic, collagenous material filled the alveolar walls. Overlying septal cells were cubical in areas and some alveolar lining cells were charged with coal-black refractile particles. In a few areas piling up of septal cells was noted; frequently buds were formed, filling the distorted alveolar spaces. The cuboidal septal cells often rested upon dusky fibrovascular buds. Retained secretion was frequently surrounded by cuboidal cells and occasionally by foreign body giant cells.

Reticulum, demonstrated by Laidlaw's method, was pericapillary in distribution in the least thickened of the alveolar septa. In regions of marked collagenous sclerosis there was still persistence of the alveolar pattern.

Thickening of the bronchial submucosa and a lymphocytic-eosinophilic inflammatory infiltrate were frequent findings. The vascular channels showed thickening of the outer media in the larger branches; the smaller showed endarteritis. The lymph nodes showed a preserved architecture, and no indications of silicosis were found.

CASE 6

R. M., a white male, 51 years old, had worked on the hot-change process for 4 years and then as a laborer in a winery for 1 year. Past history was otherwise noncontributory. Radiologic examination prior to his work as a laborer showed diffuse pulmonary shadows and bullous emphysema. One year later, a sudden pneumothorax developed with acute dyspnea and cyanosis, terminating in death.

Gross Examination of the Lungs

Microscopically, the left lung weighed 325 gm. and was greatly shrunken and compressed by a rough pleura, 0.4 cm. thick, which was plastered over the lower portions of the gray-black, indurated parenchyma. The cardinal feature was the presence of giant bullae around the periphery of the lung and emphysema in the upper portions. Some of these bullae reached a diameter of 4.0 to 5.0 cm. The lower part of one lung was firm and rubbery. The cut section revealed a uniform collapse of the parenchyma and a fish-net appearance in some regions. A few of the bronchi in the lower lobe were dilated.

The right lung weighed 375 gm. and was compressed throughout. It showed similar pronounced bullous formation. Cross section throughout the parenchyma revealed gray-black scarring with a rough, granular texture. The interlobar fissures were obliterated and replaced by a thickened scarred septum. Rough, gray-black, granular atrophy of the lung parenchyma was particularly prominent in the upper portions. Bronchi, vessels, and lymph nodes were negative.

Microscopic Description

Microscopically, the lung architecture in many regions was completely altered by diffuse interstitial fibrosis of a relatively mature type. The normal delicate filigree pattern was erased and the septa were thickened variably with fibroblasts and dense collagen. The septal fibrosis merged into irregular bands of old vascularized sclerotic tissue along the larger vessels and bronchi. This distribution was well demonstrated by the use of van Gieson's and Mallory's connective tissue stains. In many regions the interstitial fibrosis was sharply delineated and assumed many bizarre configurations like Chinese lettering. Smaller vascular channels showed thickening of their walls by collagenized tissue. Septal walls were broadened, stiffened, blunt-ended, and frequently were covered with hyperplastic cells. The septal lesions showed pronounced fibroblastic proliferation mingled with mononuclear cell infiltrate, and the broad, irregular bands of scar tissue were considered to be mats of such septal fibrotic lesions (Fig. 8). In the intertwined, spreading bands of old scar tissue and in the alveoli there were irregular deposits of black dust. Lymphoid follicles in the regions of interstitial fibrosis were intact. Reticulum, revealed by silver impregnation, lacked regular density and was arranged in a haphazard manner. In regions where septal fibrosis was not profound, alternating collapse and emphysematous pockets were found. Marginal emphysema was well advanced. The bronchioles within the scar reaction were dilated. A few showed squamous metaplastic changes and their lumina contained excessive secretion. Vascular channels showed thickening of their walls, narrowing of their lumina, and collagenous sclerosis outside the media of the vessels. The lymph nodes showed hyperplasia of the sinus reticulum and minimal amounts of fibrotic scarring of lymph sinusoids.

Chemical Investigation

The chemical approach to bauxite-fume pneumoconiosis offers a means of seeking the specific dust responsible for this unique pulmonary fibrosis. Investigation by chemical analysis is continuing under Dr. C. M. Jephcott, of the Division of Industrial Health, Ontario Department of Health. A few facts culled from the chemical analyses are presented in Tables I and II to offer a linkage with the described pathologic features.

Spectrographic determinations revealed that the majority of the fume particles are not greater than 0.5 μ in diameter. Analysis by roentgen diffraction shows that the fumes consist mainly of amorphous material.

From Tables I and II it is evident that amorphous silica and alumina represent the two prominent constituents. Further work has shown that

spectrographic analyses of the ashed lung residue from the fatal cases give results similar to those of the furnace fumes, thus offering a close chemical correlation between cause and effect.

TABLE I
Analysis of Furnace Fumes

Chemical	Range
Silica	29.0%—44.0%
Alumina	41.0%—62.0%

TABLE II
Chemical Analysis of Lungs

Case	Silica in ash	Alumina in ash
1	30.5%	30.4%
2	24.8%	40.5%
3	25.3%	Not examined
4	21.2%	28.9%
5	28.6%	40.2%
6	31.0%	25.7%

COMMENT

It is important to recapitulate the lung changes encountered in this entity. The specific pathologic findings were confined to the lungs. The outstanding features of the lungs from the gross examination were the relatively normal size and the gun-metal color. On palpation, the diffuse, widespread induration was of note. A fine fish-net pattern was present in some regions; elsewhere, radiating bands or masses of rubbery black tissue were more evident. Of further importance was the absence of shotty or confluent nodulation. Only in case 4 were caseous foci of active tuberculosis encountered and these had not been altered by the diffuse scarring. The diffuse fibrosis in these lungs was probably responsible for the spreading out of the anthracotic pigment and accounts for their gray-blue color. The frequency and size of emphysematous vesicles were noteworthy. The bullae were not only found incarcerated within the fibrosed parenchyma but were especially prominent in the subpleural regions and frequently reached giant size. Tags of adhesions and pleural thickening were common, particularly over the upper lobes, but pleural thickening was frequently encountered wherever the lungs had been collapsed by repeated spontaneous pneumothorax.

The hilar and tracheobronchial glands were not enlarged or hard, bore the usual amount of anthracotic pigmentation, and were completely free of nodulation.

Histopathologic examination showed a constant pattern of diffuse fibrosis. It is our belief that the initial lesion was intracellular septal edema with early fibroblastic proliferation. The next salient feature was infiltration of inflammatory cells, principally lymphocytes and mononuclear cells within the thickened alveolar walls. The fibroblastic proliferation was succeeded by collagen deposition. The irregular "starched" trabeculae made up of hyalinized alveolar walls were seen to best advantage in the material from case 5. At this stage of the morphogenetic

process, fibrosing septal walls might remain distinct, giving the stiffened reticular pattern, or these alveolar walls might be matted together and produce wide bands of scar. The fibrous scar was eventually hyalinized, either focally or diffusely. Doubly refractile, particulate matter was frequently demonstrated by polaroid examination. Distribution of these particles was irregular and haphazard in these lungs. The sites of their deposition and the particle size and shape were not of a decisive nature.

Anthracotic pigment was present in varying amounts and in a variable pattern. Its usual distribution was often disturbed and in many regions the carbon pigment was incarcerated in the dense bands of scar tissue. Within the diffuse scar tissue, bronchiolectasia and dilated alveoli filled with basophilic, trapped mucus frequently were noted. Septal cells in these sites were cuboidal, this transformation being particularly frequent where the alveoli were incarcerated in bands of collagen. Squamous metaplasia at the bronchiolar level was uncommon and not present to the same degree as the occurrence of cuboidal cells. These cellular changes are not specific, being frequently noted in diverse chronic pulmonary conditions such as those resulting from bronchial obstruction, or from cadmium smoke poisoning.

Obliterative endarteritis was frequent and reached the greatest intensity in the regions of diffuse fibrosis. Collagenous sclerosis was particularly marked in the outer portions of the vessels. There was no invasion of the walls by pigment-laden macrophages.

Confluent bronchopneumonia, tuberculosis, and lipidosis were nonspecific features found in individual cases. Acute infections are frequent but play no part in the progression of this disease. Foreign-body giant cells surrounding lozenge-shaped clefts were prominent in case 2 in the regions of pneumonic reaction. Tuberculosis in case 4 presented no special features. This pneumoconiotic background may offer a nidus for tubercle bacilli but the resulting reaction is not modified as in some other pneumoconioses, for example, siderosilicosis or the coal miner's lung. Alveolar bleb formation and emphysematous vesicles were prominent. This feature is apparently encountered relatively early in this disorder, as revealed by case 6. In the walls of some of the emphysematous blebs dense hyalinization was present. The fibrosis was pre-eminently in an interalveolar and interlobular pattern. Abundant septal scar tissue and lack of nodules are the characteristic parenchymal features in "alumina dust" fibrosis of the lung.

This tissue reaction bore no resemblance to any previously described process such as the acute interstitial fibrosis of Hamman and Rich.[4] The arrangement of the scar tissue is not that of carnifying pneumonitis of

infectious origin. There is no evidence of any specific infective granulomata or of sarcoid. Infarction and bronchial stenosis, either of neoplastic or inflammatory origin, could be eliminated in the consideration of these cases. In case 1, with a history of influenza, scars of an influenzal nature would not have persisted for the period of 25 years which elapsed before symptoms developed. No granulomatous reaction, such as that to beryllium, was present nor were asbestos bodies found; septal fibrosis is not encountered in these two lung disorders. None of the other dust diseases such as baritosis, bagassosis, or byssinosis need be considered because of the nature of the history.

All regional lymph nodes, both hilar and tracheobronchial, were free of specific inflammatory stigmata. There was neither diffuse nor concentric nodular fibrous tissue or hyaline material obliterating the lymph node architecture.

Due to the clear-cut industrial exposure and to the unique type of pulmonary fibrosis, it is accepted that this disorder occurring in workers in alumina abrasives manufacture is a dust-caused disease. The excessive, diffuse fibrosis throughout the lung tissue favors a "chemical dust" as the cause. The high silica content in the furnace fumes and in the lungs originally forced consideration of silica as the specific incitant, but this was abandoned for several reasons. Two principal objections are the existence of the silica in an amorphous form and the pattern of the pulmonary fibrosis. Only case 1 had a history of hard rock drilling, and there is no pathologic evidence of previous intense scarring from dust diseases. On morphologic grounds it is extremely difficult to believe that very finely divided vitreous silica or alumina could alter a pre-existent nodular fibrosis from rock-dust inhalation to a diffuse fibrosis.

The lungs in this pneumoconiosis lack the size, the nodulation, the configuration, and granite-like character of the silicotic lung. None of the indelible hallmarks of the silicotic reaction are present in any of these lungs. The lack of nodulation may be regarded as evidence against koniophages transmitting the inhaled particles of amorphous silica. The effect of this dust is primarily upon the septa, whereas in silicosis the original insult is borne by the lymphatics and lymphoid collections of the lung parenchyma. In this disease, the lymphoid collections are intact. Even in acute silicosis,[5,6] or in the burning of silica-bearing rock at Gauley Bridge,[20] the histopathologic reaction in the lung is reminiscent of the silicotic pattern.

The polymorphism of silicotic fibrosis, due to diffuse fibrotic plaques, has recently been stressed by Costero.[7] The silicotic nodule in his cases assumed a diffuse pattern produced either by an organizing pneumonitis

or transformation of atelectatic zones into fibrous tissue. In the pneumoconiosis under discussion, neither of these features is encountered and the fibrosis in these cases is an autochthonous process.

It has been shown chemically that these lungs contain large quantities of amorphous alumina and silica. It is necessary to consider the part played by these two substances in the genesis of the fibrotic process. That silica and certain silicates are capable of producing nodular pulmonary fibrosis is well known. Gardner[2] has shown that extremely finely divided silica does not cause a diffuse fibrosis in the lungs of animals and that in most cases the silica is eliminated from the tissues too promptly to produce any lasting harm. Extremely fine silica is inhaled in an amorphous vitreous form from the "dense white fumes," but no human disease has as yet been traced to such finely divided silica. If this is a form of silicosis, the problem still remains to explain why the anatomic form of the reaction is diffuse, not nodular. It may be postulated that another dust or combination of dusts can be held responsible for the causation of this disease.

The admixture of dusts, such as hematite[8] or anthracotic pigment,[3,19] may modify a frank silicotic process but never completely erases it. King[9] recently has demonstrated that an antidotal mixture of aluminum hydroxide with quartz lowered solubility but did not prevent experimental silicosis. Hence if the aluminum dust combined with the amorphous silica is responsible for the production of this tissue pattern, it is evoking a fundamental biologic reaction of a previously unheralded type.

In Germany[10-16] cases have recently appeared when there was a greatly accelerated production of explosives in which only alumina dust was utilized. Apparently a clinical, radiologic, and morphologic disease was produced, similar to bauxite-fume pneumoconiosis. The German experiences with "aluminum dusty lung" have been referred to by Perry[17] in a recent report. A personal communication to one of us (A.C.R.R.) indicates the existence of a similar condition in Sweden.[18]

It is suggested, therefore, that the mechanism is that of an amorphous dust evoking a rapid, sclerosing process within the pulmonary septa and interfering with the koniophage transmission mechanism usually responsible for dust elimination. The end-result is a diffuse interstitial fibrosis with absence of nodule formation. We favor considering the amorphous alumina dust in intense concentration as the dominant etiologic agent, but the final answer as to whether these unusual morphologic lesions owe their development to the combined effect of amorphous silica and alumina in the fumes or to the alumina dust alone will have to be obtained from experimental, chemical, and pathologic studies now in progress.

Years ago, Cummins, quoted by Belt,[3] stated that the "lung was an oc-

cupational log book—it retains a qualitative and an indelible record of the mineral particles breathed during life and after death constitutes a sort of palimpsest of the industrial history." With the emphasis on industrialization and the widespread use of diverse minerals, this statement has even greater significance. With new industries and accelerated manufacturing processes, new lung hazards will be followed by distinctive lung lesions.

It is our opinion, on the basis of the character of the pulmonary fibrosis and chemical analysis, that this disorder represents a distinctive pneumoconiosis.

Conclusions

Pulmonary fibrosis has been found in 6 autopsies on workers in the alumina abrasive industry. The clinical course was one of short industrial exposure and rapid development of disease. The highly characteristic lung changes are those of diffuse non-nodular interstitial fibrosis frequently accompanied by emphysematous bullae. The exact cause has not yet been uncovered, but the hypothesis is that an intense exposure to amorphous aluminum dust may play a dominant rôle in this bizarre fibrosis.

Grateful acknowledgment is made to Dr. W. L. Donohue for his help with the photomicrographs.

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DESCRIPTION OF PLATES

PLATE 63

FIG. 1. Case 6. Collapsed, shrunken lung with widespread fine scarring. Large marginal bullae are seen. There is no evidence of nodulation.

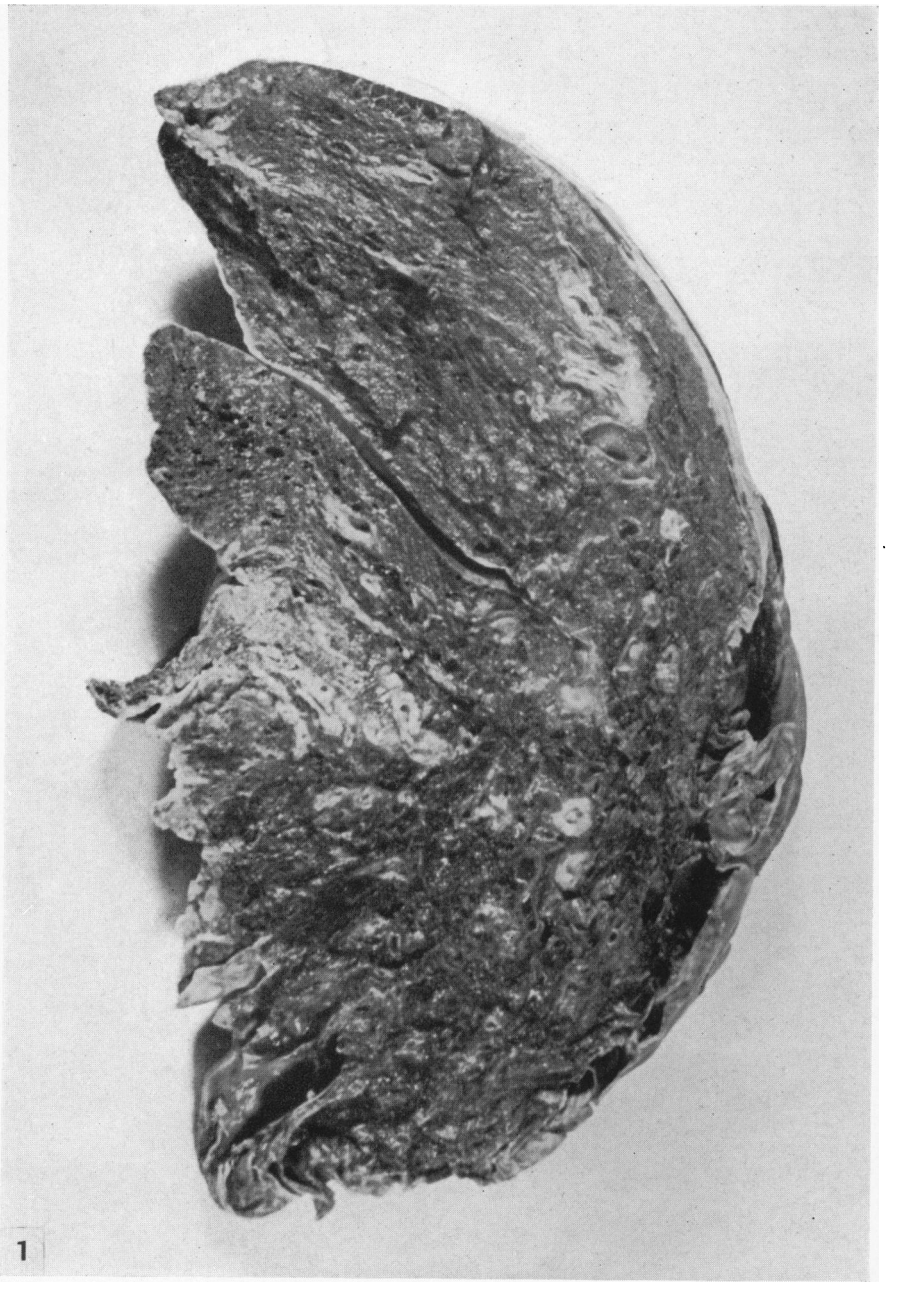

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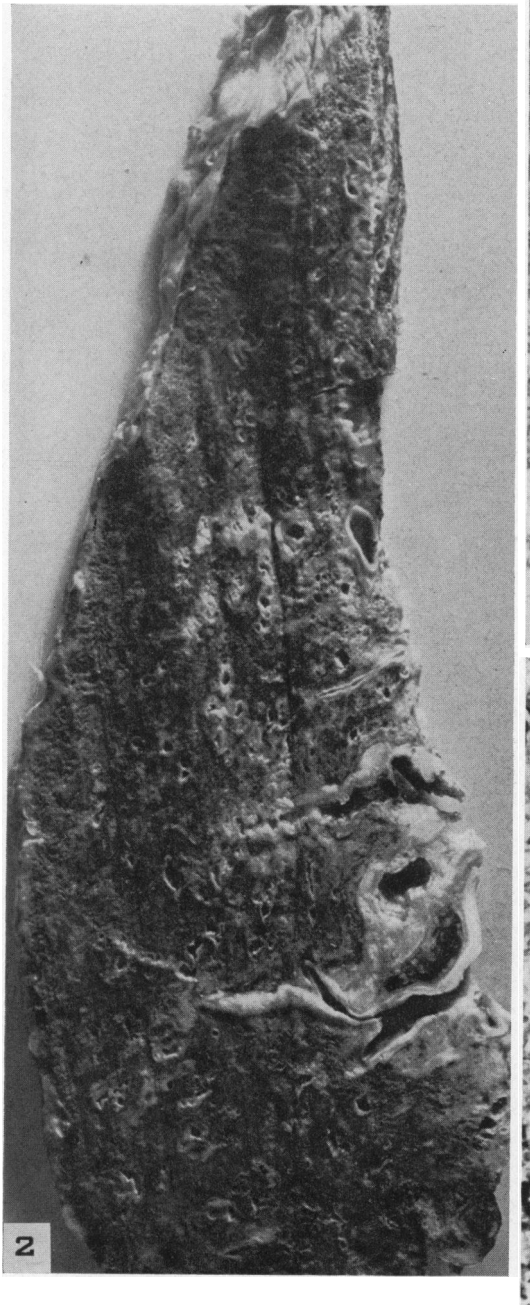
PLATE 64

FIG. 2. Lung from case 4, showing a diffuse, black, rubbery parenchyma with trapped dilated bronchial spaces. There are no nodules in the parenchyma or lymph node. × 2.

FIG. 3. Case 1. Complete obliteration of the lung pattern by scar tissue and dilated air sacs. Scar tissue is accompanied by lymphocytes. × 30.

FIG. 4. Case 1. Distorted bronchial sacs filled with trapped mucus incarcerated by non-nodular fibrous tissue. × 200.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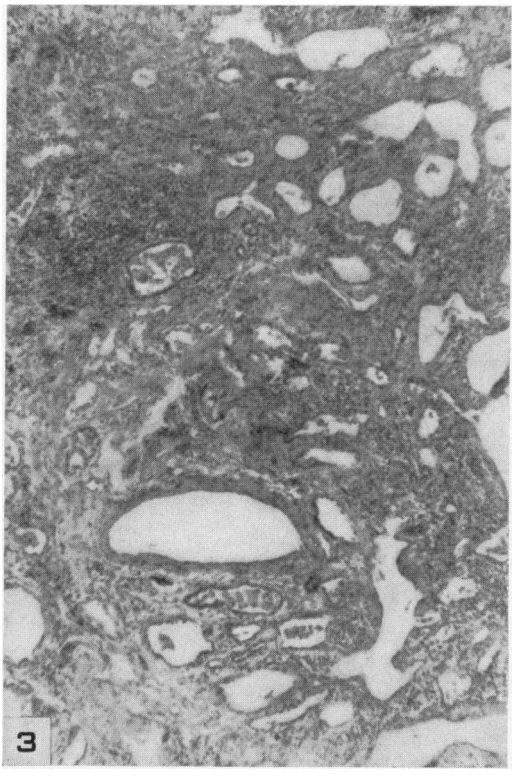
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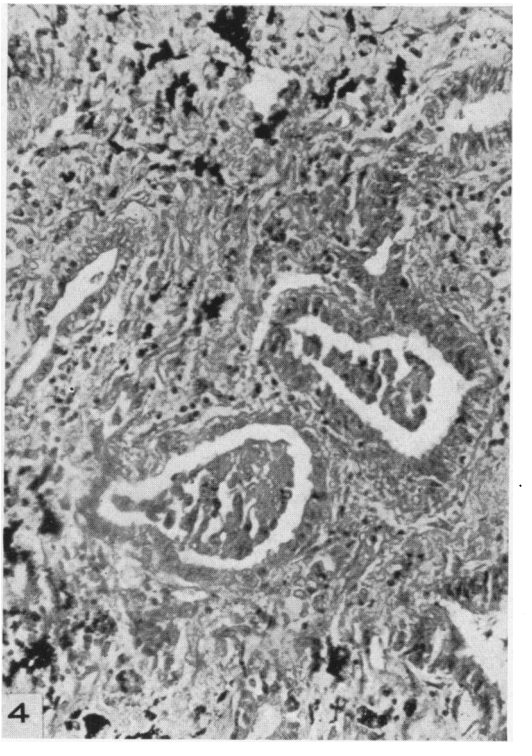

4

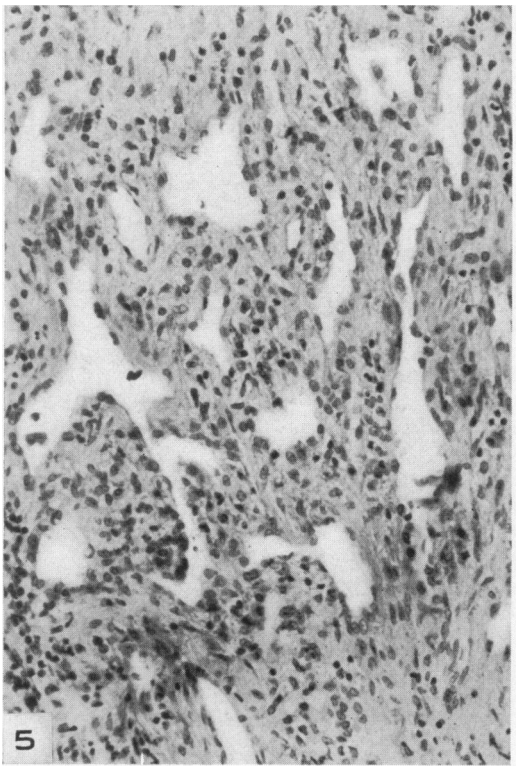
PLATE 65

FIG. 5. Case 3. Alveolar walls are swollen and thickened by mononuclear cells and fibroblastic nuclei. Obliteration of the capillary pathway may be noted. × 450.

FIG. 6. Case 6. Matting together of swollen, partially collagenized, distorted septal walls. × 450.

FIG. 7. Case 4. Irregular collagenization and hyalinization of septa. No nodular configuration is noted. × 200.

FIG. 8. Case 6. Dense bands and streaks of old scar tissue surrounding emphysematous pockets. × 120.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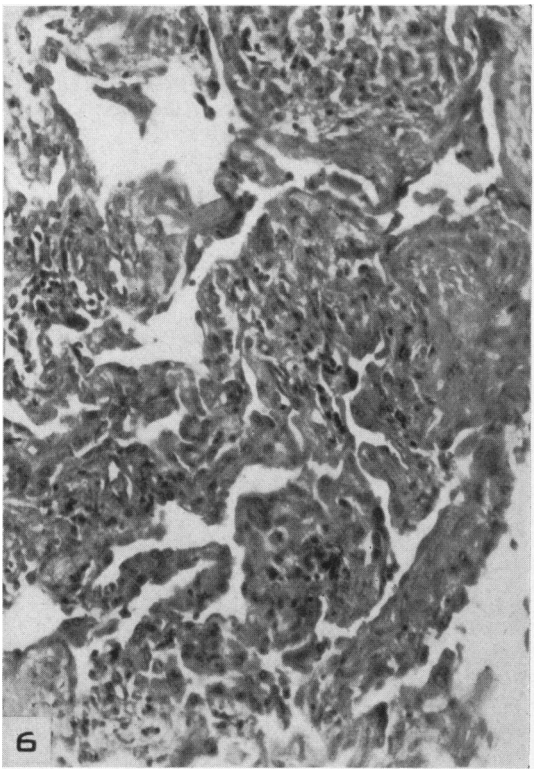
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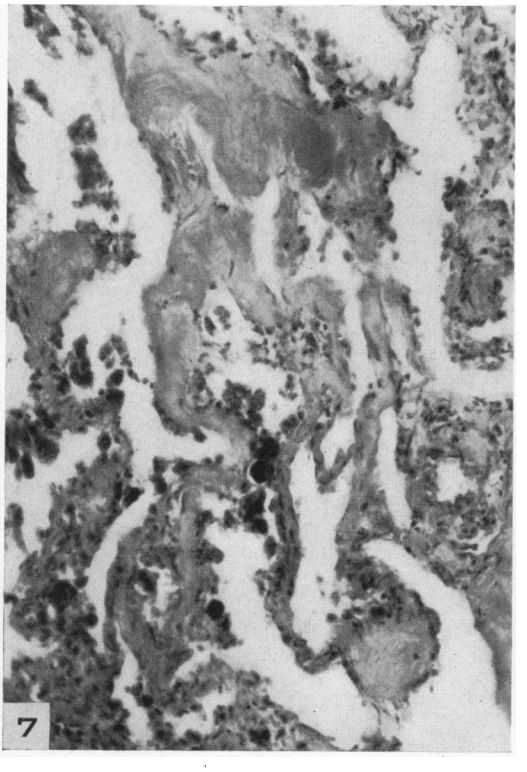
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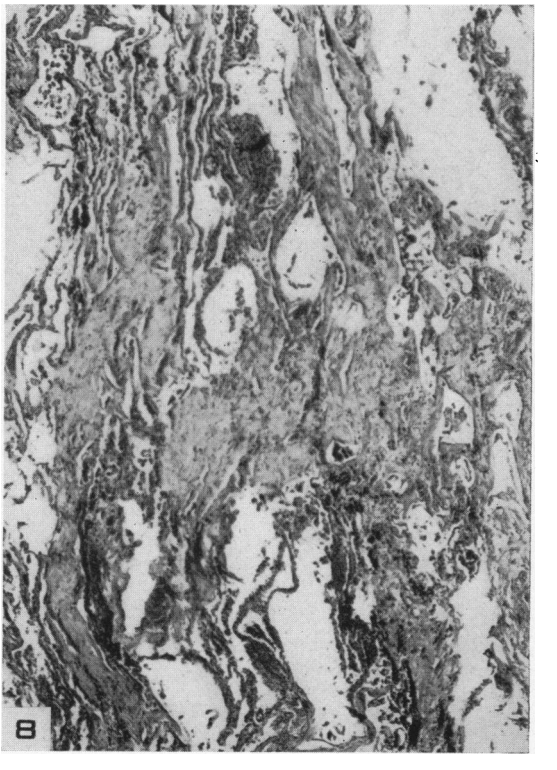
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