INDUSTRIAL HYGIENE FOUNDATION
OF AMERICA, Inc.

Medical Series, Bulletin No. 12

THE PNEUMOCONIOSES

4400 Fifth Avenue
Pittsburgh, Pennsylvania 15213
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FOREWORD

The Foundation's first medical series bulletin entitled, "Silicosis and Allied Disorders" was prepared under the supervision of Dr. A. J. Lanza. Long out of print, this publication was considered by many to be the best available and most useful statement of what was known and not known about silicosis in 1936. Much of its contents are still valid and occasional requests for it are still received.

Dr. Paul Gross, Director of IHF Research Laboratory, and I were recently asked to assist the American College of Chest Physicians by preparing a section on The Pneumoconioses for consideration for possible use in a reference text, "Cardiac and Pulmonary Physiology," sponsored by the College.

We have elected to prepare this bulletin as a first step towards such a section, feeling that critical review of this material in its present form will be received from within and without the Foundation's membership and that meanwhile, its contents may provide useful guidance to physicians responsible for evaluating, controlling and preventing chronic lung disease in employed populations exposed to dusty environments.

We recognize that there is need for much more (and more precise) knowledge of the manner in which human lungs respond to injury. The Foundation's Laboratory will continue its efforts to explicate the pathogenesis of the pneumoconioses, of emphysema and of lung cancer caused by environmental factors. Meanwhile, we will appreciate receiving data on which to base corrections and other improvements of this text prior to submitting it for publication in the open literature.

Robert T. P. deTreville, M.D.
Managing Director, IHF

INTRODUCTION

Purpose:

The purpose of this communication is to provide systematized factual information on the pneumoconioses not readily available elsewhere. What the pneumoconioses are, how they differ from other diseases, their diagnosis, prognostication, and preventive medical practices are expected to involve all medical practitioners to an increasing degree as a result of increasing public awareness of and interest in control of the environment. It would, of course, be impossible to include here comprehensive coverage of all of these areas of importance; however, carefully selected references are cited by means of which the interested reader may further develop proper orientation and familiarity in any of the areas discussed, such as methods and interpretations of pulmonary function testing, or techniques of environmental survey and analysis, for example. Chief attention is directed to anatomical and pathological considerations, as these, rather than the clinical and physiological aspects of the pneumoconioses have been most important diagnostically and from a forensic standpoint.

Chronic obstructive bronchopulmonary disease, a symptomatic condition commonly found in the general population, often occurs in coal miners and in other workers; it may thus "complicate" a simple or complex dust reaction. This association should by no means be interpreted to mean that chronic bronchitis or symptomatic emphysema (or both) are a result of the pneumoconiotic process. Such data as exist and are presented here suggest that such a relationship has not been established, although further data are needed to further document this position and these are being gathered.

Definition:

It is an understatement to say that there is no unanimity as to what constitutes pneumoconiosis. At an international conference[2] of experts in 1950, pneumoconiosis was defined as ". . . a diagnosable disease of the lungs produced by the inhalation of dust. . . ." As a result of the acceptance of this definition, it could happen that a coal miner may be demonstrated to possess black lungs at autopsy, whereas he was not considered to have had pneumoconiosis (even in retrospect) a few hours previously during life—because it was not diagnosable.*

As described later under Coal Workers Pneumoconiosis, a few years after the above definition of pneumoconiosis was adopted, an attempt was made to broaden the term pneumoconiosis to include symptomatic emphysema of the coal workers, and this concept has recently been introduced into law in the State of Pennsylvania.** There are no good data to substantiate such an etiologic association, and in our opinion, socio-economic rather than scientific consideration appears to have been foremost in this legislation.

General Considerations:

Environmental—As described in detail elsewhere,[3] only a fraction of the dust suspended in air is respirable. Generally, particles larger than 10μ in diameter are filtered out by the nasal passages. Particles down to 3μ in diameter are largely trapped on the tracheo-bronchial mucosa by their momentum, as a result of abrupt changes in direction of the dividing tubules of the bronchial

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* As used in this Bulletin, unless otherwise stated, the term "pneumoconiosis" refers to the deposition of dust in the lungs and the pulmonary reaction to its presence.

** 77 Purdon's Statutes Section 1208 (K). (An Amendment to Section 108 (K) to the Pennsylvania Occupational Disease Act, dated Nov. 10, 1965, effective Dec. 1, 1965.)

air-conducting system and by gravity as well. Also, as the tubular airways subdivide, there is a progressive decrease in the airflow rate until, in the alveoli, it approaches zero. Larger and heavier particles thus settle out before reaching the alveoli. Particles visible with the optical microscope and less than 3μ in diameter reach and may be deposited upon the alveolar surfaces; however, they do not necessarily remain in the lung.

Alveolar Clearance—The lung possesses a very efficient self-cleaning mechanism which is estimated to be 98 to 99 per cent efficient—even when somewhat overtaxed. Dust that becomes stored in the lung is, accordingly, the result of the one to two per cent inefficiency. From the point of view of prevention, increased attention should, therefore, be directed to the effectiveness of the individual worker's clearance mechanism inasmuch as a reduction in the efficiency of dust clearance of only one per cent from 99 to 98 per cent may thereby double the amount of dust stored in the lung! It is generally stated that if 100 men are exposed to an atmosphere with known serious silica hazard for the same length of time, only 25 will develop pneumoconiosis (see page 20). What is responsible for the greater susceptibility in the 25 per cent affected is not known, but reduced effectiveness of the pulmonary clearance mechanism is undoubtedly an important factor.

There are two theories concerning the mechanism of alveolar clearance: one that has been taught for about a century is based on the vitalistic concept that dust-filled alveolar macrophages migrate by directed ameboid motion (tropism responsible for the direction is unknown!) to the terminal bronchiole there to join the proximally moving mucous blanket. The other

hypothesis is only 15 years old. According to the latter, a film of fluid (described by the Canadian anatomist, Macklin, to be about 0.2μ thick) covers the alveolar surface and moves from the periphery to the terminal bronchiole. Inhaled dust particles, coming to rest upon the alveolar surface either as naked particles or within macrophages, are transported to the muco-ciliary escalator passively by the moving film.[4] The area drained by the surface film is estimated to be 500 times greater than that of the respiratory bronchiole, which may be graphically represented as the narrow end of a large funnel. In our opinion, this concept best explains the observed tendency for particulates (which are cleared rapidly from alveoli within a few days after experimental dust exposure) to accumulate and stagnate in the respiratory bronchiole itself, and, more especially, in its alveoli. Delicate tissues in this location may thus be exposed to relatively higher concentrations of noxious materials for much longer periods of time than elsewhere in the lung. Consequently, tissue reactions to inhaled noxious materials would localize in the region of the respiratory bronchiole. The fact that such localization is easily demonstrated experimentally would appear to validate such a hypothesis, at least in the several animal species which have been used.

Pathological Classification of Dusts - Fibrogenic versus Inert—The alveolar membrane's response to the presence of inhaled insoluble particles is a demonstrable proliferation of cohesive cells upon the alveolar surface. Simultaneously, an elaboration of argyrophilic fibers may be seen; first manifested by increased arborescence of immature (argyrophilic) fibers and subsequently, by a network of such elaborated fibers that support the pro-

liferated cells. Where exposure is to dust alone, the subjacent capillary does not participate in the reaction. The proliferated tissue therefore is, and remains, avascular. This alveolar membrane reaction effectively sequesters inhaled particles within the proliferated cell mass. The sequestration of inhaled dust is not necessarily permanent. As long as the inflammatory stroma remains argyrophilic and does not convert to collagen, it may resolve and disappear and thereby release the imprisoned cells and dust. This undoubtedly explains the continued expectoration of black sputum by coal miners many years after leaving the dusty environment.

The avascular reactive argyrophilic stroma of the alveolar membrane is of entodermal, rather than mesodermal origin. Characteristically, it tends to mature into adult collagen either late or, often, not at all during the lifetime of the individual. Some dusts, notably quartz, evoke an alveolar membrane reaction with abundant argyrophilic stroma that matures after a short interval of weeks to months into adult collagen. In contrast, the reactive tissue produced in the lung by coal dust or kaolin is characterized by relatively scanty, argyrophilic stroma that matures very little or not at all. It is on this basis that dusts have been classified as either fibrogenic or nonfibrogenic. The latter term is also called benign or "biologically inert," "inert" here being used in a relative sense. It is important to note that not all dusts can be classified as being "fibrogenic" or "inert." Many of the "in between" dusts are silicates, feldspar being a good example; and such dusts produce more abundant argyrophilic fiber formation, with limited conversion of the fibers to collagen.

Mixtures of Dusts—Practically speaking, human exposures differ from those in the experimental laboratory in an important respect: in most dusty jobs, a worker is exposed not to one dust, but to mixtures of dusts, the fibrogenic potential of which vary from low to high. Thus, in industry it is common to find workers who have been exposed to mixtures of coal and quartz dusts, the former possessing low fibrogenic potential. Among other dust mixtures to which workers may be exposed are: iron oxide plus quartz, asbestos plus cement, kaolin plus quartz, asbestos plus quartz, and limestone plus quartz, to mention only a few of the more common mixtures which have been prevalent in the past. While dust control measures have greatly reduced levels of dustiness to which workers were exposed in many industries prior to 1940, a number of potentially hazardous conditions still exist in some operations where such controls have not been maintained or, in some cases, have never been installed.

Clinical—As indicated in "Definitions," ultimately, presence or absence of pneumoconiosis is based on anatomic or pathologic grounds and, therefore, during life it may be impossible to make an accurate diagnosis. History of prolonged exposure to dusts capable of causing pneumoconiosis is often not accompanied by any evidence of disease by physical examination, and such changes as are present in radiological or physiological findings may not be pneumoconiotic in origin (see page 8). The outstanding exception consists of an important disease complication—progressive massive fibrosis (PMF) (see pages 9 and 23). The chief value to be derived from quantitation of measurable defects by pulmonary function tests and X-rays may be in locating individuals with the highest risk of developing PMF. We need to learn more of the natural history of these individuals in the absence of exposure to dust and also to avoid

any possible acceleration of their further deterioration which might result from exposure to dusty environments.

Regarding job placement, the late Dr. A. J. Lanza had said in 1937 that an individual with known tuberculosis should not be exposed to an environment suspected to have an associated silicosis hazard.[5] This admonition holds today, but we now suspect that the tubercle bacillus is only one of a number of organisms capable of producing PMF in coal miners.[6] In addition to massive respiratory exposure to coal dust, some infectious process or other appears to be essential to the production of PMF, and genetotrophic and sociological factors are also undoubtedly contributory. Regarding the question of destructive lung disease (symptomatic emphysema) and dust exposure, the chief goals of retrospective and prospective environmental health research studies are: (1) to identify among apparently healthy individuals those with high risk of developing pulmonary emphysema, (2) to provide them sheltered work situations, applying practical health conservation procedures, and (3) to measure the effectiveness of such preventive measures to the best possible degree. In contrast to acute bronchopulmonary conditions which may signal their presence clinically with alarming accompanying signs and symptoms, chronic bronchopulmonary disease is usually not diagnosable until extensive because of an effective physiological "safety factor," the reserve capacity of the lungs. Fifty per cent or more of the lung's normal function may be lost without subjective recognition of malfunction in persons performing the usual work required by modern living; a similar degree of loss may be necessary before unquivocal evidence of emphysema may be detected by clinical examination, in the absence of measure-

ment of gaseous diffusion across the alveolar membrane. Even where evaluation of lung function in a well-equipped pulmonary physiology laboratory is performed, interpretation of results (concerning functional loss and prognosis) is made difficult when there are no baseline measurements on the individual being studied.[7]

Lung function tests are not specific in regard to etiology, in general, and this appears especially true in the pneumoconioses.[6] The ranges of normal variation in pulmonary functions are great, and unless there are existing values against which to compare findings in individuals, there is no way of being sure that conclusions concerning functional loss at the time of testing are actually valid. The decrement produced by age must be considered in such an evaluation, as it may be very significant. Other factors to be evaluated are cigarette smoking and recurrent acute pulmonary infections, both of which are common findings in histories obtained from individuals with chronic obstructive bronchopulmonary disease.

Of a number of excellent references on pulmonary function evaluation, David V. Bates and Ronald V. Christie's[8] Respiratory Function in Disease, and E. A. Gaensler and G. W. Wright's[9] "Evaluation of Respiratory Impairment," which review the various means of evaluating respiratory impairment are highly recommended. Both give examples of proper use and interpretation of pulmonary function tests. Techniques and laboratory equipment differ widely, but there is general agreement concerning the ability to compare data derived from different techniques, provided that individuals performing the tests had developed their own laboratory controls and were sufficiently experienced. "Pulmonary function studies on an individual at one point in time are useful, but even more important are repeated studies in which individuals serve as their

own controls. In epidemiological studies performed in different industrial environments for research purposes, it is most important to follow cohorts, including those who leave the industry, perhaps because of some specific health problem of importance, as well as those who remain, representing those less susceptible to harm for one or another reason."[7]

Radiological—Because of the importance of the chest film to the detection of pneumoconioses, great care has gone into the preparation of an "international classification of persistent opacities in the lung fields provoked by the inhalation of mineral dusts"[10-11] shown in Table 1.*

More recently, however, the U. S. Public Health Service[11-12] has modified this classification along lines shown in Table 2. In addition, a classification to aid interpretation of X-ray films in individuals suspected of having asbestosis is being prepared[12] in support of U. S. Public Health Service asbestos chest studies currently in progress. Although not yet published, tentative guidelines dated October 1, 1966 have been circulated for comment and may be available upon request to qualified research personnel through the Department of Health, Education and Welfare, Bureau of Disease Prevention and Environmental Control, National Center for Urban and Industrial Health, Occupational Health Program, Cincinnati, Ohio 45202.

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* In agreeing to the reproduction of this Table here, Dr. Luigi Parmeggiani (Chief, Occupational Safety and Health Branch, Conditions of Work and Life Department of the International Labour Office) also offered the Foundation permission to publish illustrations based on the set of standard X-ray films which illustrate the International Classification. It would be difficult to attempt to compare X-ray films to such printed illustration, however; those wishing to obtain sets of standards should order them directly from Dr. Parmeggiani. Cost of the set of 14 standard 14" x 17" films is $25.00.

Table 1 [10]

INTERNATIONAL CLASSIFICATION OF PERSISTENT RADIOLOGICAL OPACITIES IN THE LUNG FIELDS PROVOKED BY THE INHALATION OF MINERAL DUSTS [a]

(Geneva, 1958)

	No pneumoconiosis	SUSPECT	PNEUMOCONIOSIS												
Type of opacity			Linear opacities	Small opacities									Large opacities		
Qualitative features	O	Z	L	p			m			n			A	B	C
Quantitative features				1	2	3	1	2	3	1	2	3			
Additional symbols	(co) / (cp)		(cv)	(di)			(em)			(hi)			(pi)	(px)	(tb)

[a] Including coal and carbon dusts.

DEFINITIONS AND COMMENTS

The object of the classification is to codify the radiological appearances of the pneumoconioses in a simple, easily reproducible way. It is intended to describe the radiographic appearances of the persistent opacities associated with pneumoconiosis, not to define pathological entities, nor to take into account the question of working capacity.

Where there is an appreciable difference in the appearance of the two lungs, the two appearances may be described separately, beginning with the right lung.

No pneumoconiosis	O	No radiographic evidence of pneumoconiosis.
Suspect opacities	Z	Increased lung markings.
Pneumoconiosis		
Linear opacities	L	Numerous linear or reticular opacities, the lung pattern being normal, accentuated or obscured.
Small opacities [b]		*The following types are defined according to the greatest diameter of the predominant opacities.*
	p	Punctiform opacities. Size up to 1.5 mm.
	m	Micro-nodular or miliary opacities. Greatest diameter between 1.5 and 3 mm.
	n	Nodular opacities. Size between 3 and 10 mm.
		The categorisation depends on the extent and the profusion of the opacities.
		Category 1: A small number of opacities in an area equivalent to at least two anterior rib spaces and at the most not greater than one-third of the two lung fields.
		Category 2: Opacities more numerous and diffuse than in category 1 and distributed over most of the lung fields.
		Category 3: Very numerous profuse opacities covering the whole or nearly the whole of the lung fields.
Large opacities [c]	A	An opacity having a longest diameter of between 1 and 5 cm, or several opacities each greater than 1 cm, the sum of whose longest diameters does not exceed 5 cm.
	B	One or more opacities, larger or more numerous than those in category A, whose combined area does not exceed one-third of one lung field.
	C	One or more large opacities, whose combined area exceeds one-third of one lung field.
Additional symbols		
Recommended additional symbols [d]	(co)	abnormalities of the cardiac outline. To be replaced by (cp): cor pulmonale, if this condition is strongly suspected.
	(cv)	cavity.
	(di)	significant distortion of the intra-thoracic organs.
	(em)	marked emphysema.
	(hi)	marked abnormalities of the hilar shadows.
	(pi)	significant pleural abnormalities.
	(px)	pneumothorax.
	(tb)	opacities suggestive of active tuberculosis.

[b] The choice of order of the [illegible] of the convexities of the pleurons. [c] The background of small opacities should be specified as far as possible. [d] The use of these symbols is optional.

Table 2

INTERNATIONAL RADIOLOGICAL CLASSIFICATION OF CHEST FILMS MODIFIED FOR U.S.P.H.S. CHEST STUDIES

	FILM QUALITY		NO PNEUMO-CONIOSIS	SUSPECT	PNEUMOCONIOSIS											
TYPE OF OPACITY					SMALL OPACITIES								LARGE OPACITIES*			
QUANTITATIVE FEATURES	UNSAT. FILM	POOR FILM	0	Z	1			2			3			A	B	C
QUALITATIVE FEATURES					p	q	r	p	q	r	p	q	r			
ADDITIONAL SYMBOLS	ax	ca	cn	co	cv	di	em	es	hi	pl	px	rl		tb	tba	nt	np
FILM QUALITY	UNSATISFACTORY FILM – IMPOSSIBLE TO READ POOR FILM - DETAILED CLASSIFICATION DIFFICULT															
NO PNEUMO-CONIOSIS	0 - NO RADIOGRAPHIC EVIDENCE OF PNEUMOCONIOSIS IN LUNG FIELDS.															
SUSPECT OPACITIES	Z - SHADOWS WHICH MAY BE PNEUMOCONIOTIC BUT WHICH ARE INSUFFICIENT FOR THE FILM TO BE PLACED IN ONE OF THE CATEGORIES CLASSIFYING THE SMALL OPACITIES.															

PNEUMOCONIOSIS

SMALL OPACITIES

THE CATEGORIZATION DEPENDS ON THE EXTENT AND PROFUSION OF THE OPACITIES:

1. A NUMBER OF OPACITIES IN AN AREA EQUIVALENT TO AT LEAST THE SECOND AND THIRD ANTERIOR RIB SPACES OF EITHER SIDE, AND AT THE MOST, NOT GREATER THAN ONE THIRD OF THE TWO LUNG FIELDS COMBINED.
2. OPACITIES MORE DIFFUSE THAN IN CATEGORY 1 WHICH MAY BE DISTRIBUTED OVER THE WHOLE OR NEARLY THE WHOLE OF THE LUNG FIELDS.
3. VERY NUMEROUS OPACITIES DISTRIBUTED OVER THE WHOLE, OR NEARLY THE WHOLE OF THE LUNG FIELDS.

THESE ARE CLASSIFIED ACCORDING TO THE GREATEST DIAMETER OF THE PREDOMINANT OPACITIES AND DENOTED BY THE FOLLOWING SYMBOLS:

p - GREATEST DIAMETER UP TO AND INCLUDING 1.5 mm.

q - GREATEST DIAMETER FROM 1.5 mm UP TO AND INCLUDING 3 mm.

r - GREATEST DIAMETER FROM 3 mm UP TO AND INCLUDING 1 cm.

LARGE OPACITIES

A - AN OPACITY HAVING A GREATEST DIAMETER EXCEEDING 1 cm AND UP TO AND INCLUDING 5 cm. OR SEVERAL OPACITIES EACH GREATER THAN 1 cm., THE SUM OF WHOSE GREATEST DIAMETERS DOES NOT EXCEED 5 cm

B - ONE OR MORE OPACITIES LARGER OR MORE NUMEROUS THAN THOSE IN CATEGORY A WHOSE COMBINED AREA DOES NOT EXCEED ONE THIRD OF THE VISIBLE RIGHT LUNG.

C - ONE OR MORE OPACITIES WHOSE COMBINED AREA EXCEEDS ONE THIRD OF THE VISIBLE RIGHT LUNG FIELD.

EGGSHELL CALCIFICATIONS (ES)

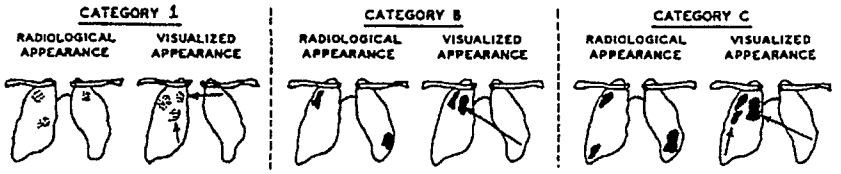
SPECIFIC CRITERIA FOR IDENTIFYING EGGSHELL CALCIFICATIONS AS EVIDENCE OF SILICOSIS:

(1) THE PRESENCE OF SHELL-LIKE CALCIFICATIONS MEASURING UP TO 2 mm. IN THICKNESS IN THE PERIPHERAL ZONE OF AT LEAST TWO LYMPH NODES.
(2) THESE CALCIFICATIONS MAY BE SOLID OR BROKEN.
(3) IN AT LEAST ONE OF THE LYMPH NODES THE RING-LIKE SHADOW MUST BE COMPLETE.
(4) THE CENTRAL PORTION OF THE LYMPH NODE MAY SHOW, IN ADDITION, SPECKLED CALCIFICATION.
(5) THE AFFECTED LYMPH NODE MUST BE AT LEAST 1 cm. IN ITS GREATEST DIAMETER.

CODE FOR ADDITIONAL SYMBOLS

- ax - SUSPECT COALESCENCE OF SMALL PNEUMOCONIOTIC OPACITIES
- ca - SUSPECT CANCER OF THE LUNG
- cn - CALCIFICATION IN SMALL OPACITIES
- co - ABNORMALITIES OF THE CARDIAC OUTLINE. TO BE REPLACED BY "cp - cor pulmonale" IF THIS CONDITION IS STRONGLY SUSPECTED
- cv - CAVITY
- di - SIGNIFICANT DISPLACEMENT OR DISTORTION OF THE INTRA-THORACIC ORGANS
- em - EMPHYSEMA
- es - EGGSHELL CALCIFICATION OF LYMPH NODES
- hi - APPRECIABLE ENLARGEMENT OF THE HILAR SHADOWS
- pl - ABNORMALITY OF THE PLEURA
- px - PNEUMOTHORAX
- rl - PNEUMOCONIOSIS MODIFIED BY THE RHEUMATOID PROCESS
- tb - OPACITIES SUGGESTIVE OF INACTIVE TUBERCULOSIS, EXCLUDING THE CALCIFIED PRIMARY COMPLEX
- tba - OPACITIES SUGGESTIVE OF ACTIVE TUBERCULOSIS
- nt - NON-TUBERCULOUS INFECTION
- np - PROBABLY NOT PNEUMOCONIOSIS

* THE BACKGROUND OF SMALL OPACITIES SHOULD BE SPECIFIED AS FAR AS POSSIBLE.



Types of Pneumoconiosis

Nonfibrogenic (also called "benign") Pneumoconioses or "Dirty Lungs":

"Dirty lungs" are caused by nonfibrogenic or "inert" dusts. This condition is not associated clinically with either symptoms or impairment of lung function. Dusts capable of producing such asymptomatic changes include coal, carbon, kaolin, cement, iron oxide, tin oxide, barium sulphate, natural (uncalcined) diatomaceous earth, and others. Although evidence is lacking, it is repeatedly stated that massive amounts of such "inert" dusts in the lungs may damage enough alveolar tissue to produce symptoms. Whether or not this is true, it has been demonstrated clinically as well as experimentally that when the amount of "inert" dust in the lung is truly massive, an infection, even though of low virulence, may result in widespread, progressive and fatal pulmonary fibrosis. Examples of such disease have been seen in coal workers (see PMF, page 21), and in workers exposed to kaolin dust, as well.

Some nonfibrogenic pneumoconioses are of considerable practical importance because chest radiographs in such cases always present an interesting diagnostic challenge. These may easily be misinterpreted by physicians unfamiliar with their appearance. The result is usually needless alarm of the patient and, at times, iatrogenic neurosis. This has occurred in cases of siderosis in which there are sharply defined miliary shadows in the chest radiograph. Differential diagnosis of "benign" pneumoconioses is discussed by Johnstone and Miller[13] who describe the necessary steps in the diagnosis of an occupational disease.

SILICOSIS

Uncomplicated (non-infectious) silicosis is an occupational disease which has no pathognomic signs and symptoms. The diagnosis is made on the basis of the occupational history (including the number of years exposure to adequate concentrations of dust of a respirable size containing significant amounts of free quartz, i.e., levels of exposure in excess of Threshold Limits[14]) and findings on chest X-ray examination. In addition, other aspects of the clinical investigation are essential to distinguish between findings related to dust exposure and those which may be irrelevant. Skillful clinical appraisal is essential in differential diagnosis and prognostication, as many chronic diseases (e.g., miliary tuberculosis or widely disseminated metastatic carcinoma of the lungs) may on a single occasion provide X-ray densities suggestive of silicotic fibrosis.

A diagnosis of silicosis usually does not mean impairment, and ability to work (or decreased work capacity) must therefore be stated. Evaluating pulmonary impairment in the presence of chronic bronchopulmonary disease may be a difficult matter. (See General Considerations.) Drs. Gaensler and Wright[9] indicate that respiratory impairment may be categorized best in terms of a few relatively broad classes: Class O has no measurable impairment (0-10%), while Class 4, at the other extreme, includes those with obviously severe impairment who have difficulty caring for themselves and might be classified as 90-95% impaired on simple clinical observation. Between these extremes, three other groups are recognized: Class 1 with

minimal impairment from all causes in whom respiratory impairment could not be construed to contribute significantly to disability; Class 2 which has moderate impairment for heavy labor or strenuous recreational activities but which is usually significant only when accompanied by impairment of other organs; finally, Class 3 which includes severely impaired patients who manage to be gainfully employed only under special circumstances and in whom respiratory insufficiency may well be the sole reason for total disability. In addition, a suffix "X" may be added to the above classes to indicate variable impairment due to factors other than measurable physiologic disturbances (e.g., a persistent tuberculous cavity with a positive sputum).

These classes were reportedly developed at the request of the Committee on Rating of Mental and Physical Impairment of the American Medical Association, which has modified them as follows:[15]

Class 1	(0% by definition if under 20%)
Class 2	(20-30% impairment of the "whole man")
Class 3	(40-50% impairment of the "whole man")
Class 4	(60-90% impairment of the "whole man")

The AMA guide urges that the one final impairment value in an individual be expressed to the nearest 5%; however, Drs. Gaensler and Wright recommended against such close calculations that "imply a precision of measurement which, in fact, does not exist;" further, they indicate that the pulmonary reserve is so great that small degrees of impairment could not possibly reflect in terms of disability. Finally, they express doubt that the mathematical addition or

combination of multiple systems impairments by preconceived formulae is an appropriate way to express the total medical effect upon the whole man. It is important that the physician attempting to use the AMA guide recognize such shortcomings in the rating system and use his best clinical judgment, supported by appropriate pulmonary function laboratory findings.[7,8]

Physical Examination—Except in the late stages of silicosis, complicated by infection as described below, little is revealed by clinical examination of the chest. As stated above, recognition of silicosis in the individual by means of physical examination alone is very often impossible. A clear history of repeated or long-term exposure to free crystalline silica of respirable size should be sought and the chest X-ray is indispensable in arriving at a positive diagnosis in silicosis. (X-ray interpretation and classification are discussed on pages 9 and 23).

Pathological Diagnosis:

Silicosis is a fibrous, nodular disease of the lungs, caused by the presence upon the alveolar surface of crystalline silica (most often quartz). As a matter of practical experience, it is rare and becoming more so that a worker is exposed to only one dust during his lifetime. If other inhaled dusts (such as asbestos, cristobalite, or tridymite) are also fibrogenic, the net effect is summative. Otherwise, the superadded inhaled dusts probably do not contribute significantly to the uncomplicated clinical silicotic disease.

In uncomplicated silicosis the lung contains widely distributed sharply defined hard nodules 2 to 4 mm in diameter, the number and size depending upon the severity and duration of the dust exposure. These nodules may be more numerous in the upper, than in the lower lobes. Upon gross examina-

tion of the cut surface of the lung, the color of the nodules may vary from gray to black, depending upon the admixture of other dusts, particularly the carbonaceous ones. These nodules are the repository of the residual inhaled silica, sequestered within concentrically arranged dense collagenous tissue. The nodules tend to increase in size with continued exposure, the growth occurring in the so-called peripheral reactive zone in which the newly deposited silica is abundantly present. With the cessation of exposure, the peripheral reactive zone tends to become progressively more narrow and finally to disappear. The nodules are then largely acellular and the disease is in an inactive, nonprogressive state. It is important to appreciate that in uncomplicated silicosis, the lung tissue between the nodules is anatomically and functionally normal.*

Silica is readily transported from the lung tissue to lymph nodes of the hilar and paratracheal regions as well as beyond these areas. As a result, nodes so involved tend to become enlarged and stony hard, their color depending upon the presence of other dusts.

The mechanism by which the crystalline silica produces its pathologic effect is not known. Of the numerous theories which have been proposed, the one now being given most serious consideration involves surface action and immunologic mechanisms.[6]

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* Mention should be made here of so-called "acute silicosis," a rare, diffuse, inflammatory lesion of the lungs which has occurred sporadically, often in epidemics (e.g., Gaulley Bridge),[5] where workers have been overwhelmingly exposed to crystalline silica dust having a large component under 1μ in diameter. The lungs initially respond to such exposure by acute inflammatory changes, followed by a diffuse type of fibrosis not characterized by typical nodules but by confluence of micronodules, and in some cases, dense fibrosis in the upper lobes. Evidence of complicating infection may be present. Another diffuse, non-nodular form of silicosis is "Diatomite Pneumoconiosis" caused by cristobalite, tridymite and quartz which are produced by calcination of amorphous diatomaceous earth.[13]

Chronic Obstructive Bronchopulmonary Disease (Including Symptomatic Emphysema and Chronic Bronchitis):

In discussing the symptomatic emphysema and chronic bronchitis encountered in silicotic subjects, it should be stressed at the outset that we do not intend to imply either an etiologic or an aggravating relationship between the existing silicosis and these complications. There exists today in a large segment of the adult population and in housewives as well as workers in industry trades, recognizable chronic obstructive bronchopulmonary disease; and the fact that this condition is observed not infrequently in silicotics should not be surprising. Tuberculosis, which is much less prevalent in the general population than emphysema and chronic bronchitis, remains the most dreaded complication of silicosis. (It accounted for roughly 75% of the deaths of silicotics a generation ago.) The important difference in the relationship of these two diseases to silicosis is essentially this: whereas tuberculosis, in combination with silicosis is synergistic (resulting in a new and therapeutically-resistant entity, silicotuberculosis), the chronic obstructive bronchopulmonary disease encountered in silicotics is no different clinically or pathologically than that found in the general population. Although it is true that the air spaces adjoining silicotic nodules, similar to those adjacent to other types of scars, may exhibit so-called "traction," or paracicatricial emphysema, this usually has no clinical significance. There is no relationship between such anatomic findings and symptomatic disease associated with the panlobular and centrilobular forms of emphysema.[7]

Depending upon the severity of the chronic obstructive bronchopulmonary disease, there is usually an associated reduction in the total vascular bed of the lungs, resulting in cor pulmonale. This may lead to cardiac failure

and death. Although vascular sclerosis has been described in silicotic lungs, it is doubtful that silicosis without a coexisting panlobular emphysema or chronic bronchitis could give rise to pulmonary hypertension. Nevertheless, at a recent Symposium on Coalworkers' Pneumoconiosis[16] held in Morgantown, West Virginia, May 18-20, 1967, mention was made of clinical research in this area using cardiac catheterization as a means of detecting increased right heart pressure.

Tuberculosilicosis (or Silicotuberculosis):

The combination of silicosis with tuberculosis tends to produce extensive fibrous solidification of the lungs. Cavitation is, of course, also common. The recognized tendency for a superimposed tuberculous infection to cause enlargement of the shadows of the individual silicotic lesions on the chest radiograph has led experts to believe that most, if not all, confluent silicotic lesions are caused by superadded tuberculous infection. Likewise, the apparent progressiveness of some silicotic lesions after removal of the worker from further exposure to silica dust has also been attributed to a superimposed tuberculous infection.

Fibrous pleuritis, a not uncommon finding in silicotics, is caused by intercurrent infection, unrelated to the presence of silicotic nodulations.

Other Infections:

The question of whether or not silicotic lungs are more susceptible to bacterial pneumonia than are normal lungs has been investigated.[17,18] The investigating laboratories found that silicotic animals had a lower mortality rate from pneumococcic pneumonia than did normal animals. The explanation for this finding may lie in a more vigorous antibody response demonstrated in silicotic than in normal animals.

Cancer:

Probably because some lung cancers are known to have arisen in pulmonary scars, it has been suggested that there may be a greater risk for the development of cancer in silicotic lungs than in the lungs of the general population. However, epidemiologic studies to date have shown the opposite.[1, 19] Pulmonary scar cancers originate from epithelial bronchiolar or alveolar remnants imprisoned in the scars. Such remnants are not found in silicotic nodules.

COALWORKERS' PNEUMOCONIOSIS

Uncomplicated ("Simple") Coalworkers' Pneumoconiosis and Anthracosis ("Black Lung"):

Like any of the other pneumoconioses, that occurring in coalworkers is the storage of, and tissue reaction to, dust—in this case, black dust—in the lungs. In coalworkers this black dust is chiefly coal, but in city dwellers' lungs, the black pigment may be soot or fly ash. The important feature about this form of pneumoconiosis is that the tissue reaction to the dust is minimal, hence it is classified as "benign."

Uncomplicated or "simple" coalworkers' pneumoconiosis is asymptomatic, but there are three important modes by which it may become complicated: first, the concomitant inhalation of significant amounts of quartz dust with the coal dust will result in collagenous fibrosis of the dust depots; a second type of complication is the coincidental finding of chronic obstructive bronchopulmonary disease; and a third, the association of infection with massive coal dust deposition (see PMF on page 21). In a detailed review of "Coal," Braun*[1] tabulated findings in Bituminous Miners and Anthracite Miners in England (South Wales) and the United States calling attention to the importance of "rank" (Table 3).

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* Daniel C. Braun, M.D., Assistant Medical Director, U. S. Steel Corporation.

Table 3

Percentage of Workers with Nodulation or Consolidation
According to Length of Exposure

(Comparison of Findings in South Wales and USA)

Bituminous Miners

Length of Exposure	South Wales—1938 (Hart and Aslett)	USA—1941 (Flinn)
Under 10 years	0%	0%
Over 20 years	3.2%	3.2%

Anthracite Miners

Length of Exposure	South Wales—1938 (Hart and Aslett)*	USA—1935 (Sayers)
Under 10 years	3.9%	"under 2%"
Over 20 years	29.0%	23.0%

* Braun makes the following quote from Hart and Aslett's work:

> "Rank of coal is, in fact, the most important obvious factor found to be related to the varying incidence of X-ray abnormalities among colliers at these different mines. The highest incidences were found at the anthracite and the lowest at the bituminous end of the scale of rank."

The "rank" of a coal is roughly inversely proportional to the amount of volatile matter it contains. In South Wales, anthracite coals with less than 5% volatile matter have the highest rank, and bituminous coals with over 30% volatile matter have the lowest rank.

Emphysema and Chronic Bronchitis Occurring with Coalworkers' Pneumoconiosis:

Among the most common causes of breathlessness in coal miners is chronic obstructive bronchopulmonary disease. Because of the difficulty of differentiating between symptomatic emphysema and chronic bronchitis clinically and because they are commonly associated, no distinction is made between them in the British medical literature. By means of thick, macrosections of coalworkers' lungs, Dr. J Gough demonstrated that the deposition of coal dust was frequently associated with focal emphysema. At first, this focal emphysema was thought to be responsible for the breathless ness observed in some coal miners; and on the basis of this assumption, the term "coalworkers' pneumoconiosis" was suggested to cover not only the anthracosis but also symptomatic chronic bronchitis, a common complicating finding. Since, it has been found that the focal emphysema around coal dust depots is not responsible for breathlessness.[7] Vital statistics suggest that men who work in dusty jobs have higher death rates than those who do not. This applies to miners, foundry workers, furnace men and cotton workers, but Higgins[7] has pointed to the interesting fact that in each of the above cases in which the standardized mortality ratio for an occupation is "high," the wives of the men with high ratios also have high ratios, thus raising the important question: are the high ratios in the men attributable to occupational factors or are they due to social factors, associated with the occupation, that also affect their wives? There appears to be agreement that the emphysema responsible for the breathlessness of the coal miner is pathologically no different from that found in the lungs of the general population. A recent investigation resulted in the conclusion that not centrilobular, but panlobular emphysema was responsible for the symptomatology associated with this disease.[20] Thus, in

the light of present-day knowledge, it would appear that the use of the term, "coalworkers' pneumoconiosis" as a cover for a symptomatic emphysema and chronic bronchitis is an anachronism, and erroneous: it attributes the cause of the breathlessness to coal, without mentioning the probable association with other factors, such as cigarette smoking—much more important in its etiology and aggravation to all appearances at present.[7,16]

Infective Pneumoconiosis (Progressive Massive Fibrosis—PMF):

A small, but significant, percentage of British coalworkers have developed what is recognized roentgenologically and pathologically as a progressive massive fibrosis of the lungs (PMF). This chronic condition has proved difficult to treat medically. It is often fatal, although its progress may be slow. In about a third of the cases of PMF, tubercle bacilli have been found in the lungs either by culture or by smear.[1] Although at one time it was thought that nearly all cases of PMF represented cases of tuberculo-anthracosis it is now believed that other infections may also play a role in conjunction with massive dust deposits.[6] For many years there has been controversy regarding the basic nature of PMF, another widely held concept having been that it was simply anthrasilicosis.[1] However, with the experimental reproduction of a model of this disease (PMF) in guinea pigs, the lungs of which had been burdened with coal-mine dust and then infected with tubercle bacilli of low virulence,[1,21] the controversy tended to subside.

Pathology:

In simple coalworkers' pneumoconiosis, there is multifocal black discoloration of the pleural, as well as of the cut surfaces of the lung. As the amount of black dust deposited in the lungs increases, the foci tend to become more

numerous, larger, and finally to fuse, thereby rendering all surfaces diffusely black. Although focal emphysema is frequently present in heavily dusted lungs, this is, by no means, a constant finding. The hilar lymph nodes are usually slightly enlarged, black and firm in consistency, the last probably resulting from some degree of silica exposure in addition to the black pigment.

With increasing amounts of crystalline silica dust inhaled along with the coal dust, foci of dust deposition tend to develop more collagenous fibrous tissue and to become harder until they resemble full-fledged silicotic nodules. These nodules differ from the smooth, round, gray nodules produced by pure silica not only in their black color, but also in their stellate outlines and generally larger size. The nodules frequently fuse to form the large stony-hard, black masses in the lungs characteristic of anthrasilicosis.

The appearance of the lungs in infective anthracosis is very similar to that of anthrasilicosis. Cavitation, when present, is usually indicative of infection. However, cavitation when found in anthracotic lungs in the absence of tuberculosis is thought to result from ischemic necrosis.[1]

Coincidental centrilobular and panlobular emphysema found in pneumoconiotic lungs of coal workers is very similar to the emphysema found in lungs free of excessive amounts of black pigment.

General Considerations:

Estimating Prognosis by X-Ray—PMF—In Coal Workers—Drs. P. D. Oldham and C. E. Rossiter[6,22] recently have confirmed the opinion of Dr. A. L. Cochrane that the size of the conglomerate lesions in PMF was of prognostic value in estimating reduced life expectancy. They find that size of PMF and residual volume correlate best with fatal outcome, while clinical evidence of abnormal physiological dynamics were of much less value in such a prediction. Radiographic findings thus continue to be of prime importance to chest physicians attending cases of PMF.

ASBESTOSIS

There are three commonly used types of asbestos.[1] In this country, about 95% of the asbestos used is chrysotile, mined in Canada. The other two types, used largely for insulation and composite materials, are amosite and crocidolite which come mostly from South Africa. Whereas in the asbestos textile industry the worker's exposure to mineral dust is limited practically to chrysotile, in other segments of the asbestos industry the dust exposures are usually mixed. Not only are the workers in this industry usually exposed to more than one kind of asbestos dust, but also to other dusts as well. It is common, for instance, to find cement and quartz dust associated in various proportions with asbestos dust. The tissue reaction to the dust may accordingly be modified depending upon the nature and amounts of the associated dusts in the lungs. The older literature contains many references to the dictum that only asbestos fibers 10μ or longer are pathogenic, that the short-fibered asbestos dust does not cause asbestosis. Experimental studies have failed to support this concept.

According to Smith,* "pulmonary fibrosis resulting from prolonged inhalation of asbestos fiber will produce a typical X-ray pattern. In early, or first-stage asbestosis, the X-ray shows a fine diffuse homogeneous infiltration throughout both lower lung fields. It should be noted that this infiltration is bilateral, that it is generalized at both bases, and that the nodular or conglomerate patterns of other pneumoconioses, such as silicosis, are not seen in asbestosis. There is a considerable amount of pleural reaction associated

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* Smith, Kenneth W., M.D., then Medical Director, Johns-Manville Corporation, presently, Medical Director, Brush Beryllium Company.

with this disease, which may account for the 'ground glass' pattern which has been used to describe the typical X-ray picture.

"...In moderately advanced, or second-stage, asbestosis, the infiltration has increased but still is confined to the lower lung fields. The 'ground glass' pattern is more apparent, and the heart borders are becoming indistinct or shaggy. There is some irregularity of the diaphragmatic outlines and beginning obliteration of both the cardiophrenic and the costophrenic angles.

"In far-advanced, or third-stage, asbestosis, the infiltration still is homogeneous and bilateral, has spread to the middle and possibly the upper portion of the lung fields, but the apices remain clear. The cardiac outline is almost completely obliterated, as are the domes of the diaphragm and the costophrenic sulci. With this picture in mind, it is advisable to reiterate the observations of many physicians, namely, that the X-ray picture should never be used to estimate the presence or the extent of impaired pulmonary function or disability. Many cases with X-ray evidence of third-stage asbestosis have been known to carry on their usual work and live fairly comfortable lives for several years. On the other hand, no case of definite disability has been seen unless there was the typical X-ray pattern of asbestosis. X-ray changes typical of asbestosis seldom are seen unless there has been a period of at least ten years of continuous exposure.

"...In some cases it has been suggested that the appearance of calcified pleural plaques on the X-ray, coupled with a potential exposure to asbestos fibre are pathognomonic of asbestosis. Reviewing a quarter century of serial X-ray films, the author noted calcified pleural plaques appeared rarely in several thousands of workers in an asbestos mine in Canada. The fibre from

this mine has been used for a great many years in numerous American plants. Cases of asbestosis have occurred in this mine and in these plants, but in only one of the plants is there any evidence of pleural plaque formation. If inhalation of the asbestos fibre alone could cause these plaques, one would expect to find plaques wherever the fibre is used. In view of the above-mentioned evidence the causal relationship of pleural plaques to other pulmonary and other cardiac conditions in addition to asbestosis is worthy of investigation.

"...There is no typical clinical picture for asbestosis. The disease is insidious in its onset and slowly progressive with continued inhalation of the fibre. There is a gradual increase in cough and expectoration, some anorexia and weight loss, then slowly increasing dyspnea. Cyanosis and clubbing of the fingers are rare findings."[1]

It is important to note here that attempts should not be made to transfer the above findings, which relate to asbestos exposure, to workers exposed to mixed dusts: admixture of other dusts, such as silica, may greatly affect the appearance of the X-ray and also the histopathological findings. For a discussion of mixed dust exposure see page 6. Guidelines for X-ray interpretation in asbestosis are being developed by the U. S. Public Health Service[12] (see page 9).

Unlike most pneumoconioses, which are multifocal in character, asbestosis involves alveolar walls diffusely. Nevertheless, in the experimental animal—and it has also been claimed for human asbestosis—the disease is initially also multifocal, each focus being centered in the proximal portion of the primary lobule, the respiratory bronchiole.

When the disease is active, as it is shortly following the deposition of the dust in the lung, the alveolar walls become thickened by cohesive proliferated

cells that are supported by argyrophilic fibers. This is the florid stage. With time, usually measured by many years, the septa lose their cellularity and the argyrophilic stroma becomes converted into adult collagen. This finding indicates the inflammation had healed. In healing, however, the septum frequently becomes avascular and stiff. The involvement from one alveolus to the next may not be uniform. Some severely thickened collagenous alveolar walls have shown an almost angioma-like plethora of capillaries on one or both surfaces of the dense and acellular collagen. This has been a not uncommon finding in asbestotic lungs after the patient has been subjected to prolonged exposures to high oxygen concentrations prior to death. The uncomplicated, healed asbestotic lungs are small, stiff lungs, presumably because of the widespread contracture of the collagen in the alveolar septa.

Complications:

One of the more common coincidental findings encountered in asbestotic lungs is chronic obstructive bronchopulmonary disease. Claims for a cause-and-effect relationship have frequently been made when emphysematous changes have been found associated with asbestosis. As with the other pneumoconioses, these claims have not been adequately supported in fact. More epidemiologic studies and data regarding the pathogenesis of emphysema are needed, however, to exclude any possible cause-and-effect or aggravating relationships.

Another complication associated with asbestotic lungs that has received much attention is a pleural fibrosis that may attain a thickness of one centimeter or more and the consistency of shoe leather. Not infrequently, the pleural inflammatory fibrous tissue becomes calcified, forming pleural plaques. As with other findings, the inevitable question has arisen whether or not the

association represents a cause-and-effect relationship. The main obstacle in the path of acceptance of such relationship is the fact that it has not been possible to show a relationship between the thickened pleura and the physical presence of the dust. If asbestos dust were responsible for the thickened fibrotic pleura, one would expect to find the specific pathogenic dust concentrated there just as one finds quartz dust concentrated within silicotic nodules. Not only is there no concentration of asbestos dust, but no dust of any kind is demonstrable in the thickened pleura with the optical microscope. An occasional asbestos body has very rarely been found in such pleural lesions. A recent extensive epidemiologic investigation of calcific pleural plaques in Finland has shown that these occur in population groups living in communities containing asbestos mines and mills with a prevalence that is not significantly higher than the prevalence of pleural plaques in stable rural communities that do not have asbestos mines or mills.[23] This observation effectively supports the opinion of Smith[1] that if asbestos exposure alone were able to produce pleural plaques, it should be found wherever asbestos is inhaled, which is not the case.

Two types of cancer have been associated with the inhalation of asbestos dust, carcinoma of the lung and mesothelioma of the pleura or peritoneum. Both kinds of malignant tumors have been produced in experimental animals by the inhalation or injection of asbestos dust. Epidemiologic studies have convincingly demonstrated a much higher prevalence of pulmonary carcinoma among British asbestos textile workers who had been exposed to asbestos dust prior to 1935 than among the general population.[24] However, Knox et al have shown more recently that in the thirty-odd years since the British asbestos textile plant concerned

instituted good housekeeping measures, the risk of developing lung cancer among its workers has not been higher than that of the general population.[25] Smith[1] in 1963 reviewed his 18 years of experience in the American asbestos industry and gave his impression that among workers exposed to chrysotile fiber, there were no more cases of pulmonary malignancy than among the general population.

The current status of bioeffects research on asbestos was the subject of meetings sponsored recently by the New York Academy of Science[25] and Industrial Hygiene Foundation.[26] Epidemiologic studies have shown a much higher prevalence of pleural and peritoneal mesotheliomas among those exposed to asbestos dust than among the unexposed general population. Although most of the former were workers exposed to mixtures of different kinds of asbestos dust, reports from South Africa indicate that non-occupational (but equally heavy) exposure to the crocidolite type of asbestos was incriminated in the production of mesotheliomatous tumors with a delay of an average of about 43 years before appearance of the tumors, in which evidence of asbestosis was absent or minimal. In other cases reported from the United States, the possibility that the workers inhaled sufficient crocidolite dust to have been a factor in the mesothelioma production could not be ruled out; further and more careful epidemiological studies are needed to determine if exposures associated with types of asbestos other than crocidolite may produce mesotheliomas in man.

Asbestos Bodies Versus Ferruginous Bodies:

The hallmark of an asbestotic pulmonary inflammation is the presence of asbestos bodies. These are golden brown symmetrical, elongated structures composed largely of ferritin, or ferritin-like iron-containing proteins, precipitated around a central transparent or translucent filament. They are often

segmented and have clubbed ends. They measure 1-3μ in diameter and 20μ or more in length. In the lungs, they are usually found free in the air spaces, often in clusters. Shapes and sizes vary greatly.

In recent years these iron-containing, or ferruginous bodies have been found at autopsy in the fluid expressed from the lungs in 30 to 48% of the general population in Capetown, South Africa; Miami, Florida; Pittsburgh, Pennsylvania and Montreal, Canada.[26, 27] The lungs in which these bodies have been found generally did not exhibit significant disease. Attempts have been made to link the finding of the so-called "asbestos" or ferruginous bodies to an assumed widespread pollution of urban atmospheres with asbestos dust on the assumption that the formation of ferruginous bodies is a specific reaction to the presence of asbestos fibers; however, recent investigations have demonstrated that filamentous dusts other than those of asbestos may also evoke the formation of ferruginous bodies. One of these filamentous dusts producing such bodies in the lungs of hamsters is aluminum silicate.[28]