The Topographic Distribution of Mineral Dusts in Some Pneumoconiotic Lungs*

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All theories that have served to explain the formation of fibrous tissue in silicosis relate the development of the collagen fibers, directly or by inference, to the site of the silica particles. Recent studies\(^1, 2\) have established that portions of some silicotic nodules may contain no demonstrable mineral particles and that dust particles formerly believed to be more or less permanently imprisoned at the site of their deposition within the lung tissue not only may be shifted from one region to another within the lung but may also be expectorated. Thus the occurrence of silicotic fibrous tissue without the demonstrable associated causative mineral particles should be considered no more anomalous than the more familiar finding of manifestly silicotic lungs with a relatively low silica content, that is, a silica content no higher than that of some non-silicotic lungs.

Because the topographic study of the distribution of mineral dust in pneumoconiotic lung tissue gives promise of providing new information regarding the pathogenesis of pulmonary dust diseases such studies have been performed on routine cases that have come to the attention of the Industrial Hygiene Foundation. It is the purpose of this paper to summarize the results of some of these studies.

**Method**

Formalin-fixed lung tissue was embedded in paraffin, sectioned and stained in a routine manner. In addition replicate sections were also stained by a combination of the Van Gieson stain for collagen and the postassium ferrocyanate method for iron. The locations of selected fields in the stained sections were noted with the aid of a "field finder."\(^\uparrow\) Following photography of these fields, the sections were incinerated at 550\(^\circ\) for one hour, and after cooling, the acid-soluble portion of the ash was removed by placing the slides in concentrated hydrochloric acid for 30 minutes. They were then washed with running tap water, followed by rinsing in distilled water and air drying. The same fields were then rephotographed but under dark field conditions. Composites were made by superimposing the 2½" x 2½" dark-field negative upon the bright-field negative or the color transparency of the same field and after careful matching, fixing them in their relative position by means of transparent tape.

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1. **Silicosis with slight asbestosis**

Case O. S. G., age 55, had 31 years exposure to fine silica sand (consistency of flour) and to a minor degree also to cement and asbestos dust. Patient had worked steadily until 17 months prior to death. X-ray at the onset of illness showed a spontaneous pneumothorax and generalized pulmonary nodulations (Figure 1). He continued to work, but intermittently, until two months prior to death. The main pulmonary findings at autopsy included severe silicosis with minimal asbestosis, emphysema, and sclerosis of pulmonary vessels.

Analysis of the dried lung indicated a total silica content of 1.77 per cent (chemical determination) and a quartz content of 1.0 per cent (x-ray diffraction).

Most of the lung sections show relatively little air-containing tissue. Much of the tissue is represented by silicotic nodules and masses which, although composed of acellular hyaline, show no lamination. Many of the nodules, particularly the smaller ones, have a stellate or irregular periphery (Figure 2). The nodules contain a surprisingly small amount of mineral dust much of which is concentrated on the periphery (Figure 4). The larger nodules and masses generally contain less dust than the smaller ones. There are regions (Figure 6) such as where air spaces are all but surrounded by coalescent nodules in which the walls of air spaces are diffusely infiltrated by dust. Asbestos bodies are seen in this region associated with foci giving a positive reaction for iron.

2. **Pure Silicosis**

Case J. C., age 72, had been a monument worker for 27 years to the age of 47. For the next ten years he was a door-to-door salesman and the following ten years were spent as a supervisor of several small apartment buildings. At the time of his death, in addition to silicosis there was a renal carcinoma with small pulmonary metastases and a penetrating duodenal ulcer with subdiaphragmatic abscesses.

Sections of lung contain sharply delimited, laminated and well collagenized nodules which vary in size from microscopic to 4 mm. in diameter and larger (Figure 7). The intervening lung tissue is emphysematous and the pleura is thickened. The nodules are composed of acellular, concentrically arranged hyaline collagen which is surrounded by a cellular "reactive" zone of varying width (Figure 8). Although the

![Figure 1: Case O. S. G. Advanced pneumoconiosis due to mixed dusts: quartz silica, cement, and asbestos.](image-url)
collagenous portions of most nodules are smooth and rounded, and even the peripheral reactive zone frequently also presents a smooth and rounded circumference, there are regions which resemble a sunburst (Figure 10). This appearance is produced by a fibrous thickening of the bases of adjoining septa that extend radially. The collagenous centers of the nodules contain irregularly scattered aggregations of mineral dust with considerable portions of the collagen free of demonstrable dust. In contrast, much of the mineral dust is concentrated in the peripheral reactive zone, extending also into adjoining septa (Figures 9 and 11).

**Figure 2**


**Figure 3**

*(Neg. 3303) Case O. S. G. Two coalescent fibrous nodules, the lower one possessing a "reactive" peripheral zone. Van Gieson-iron stain.*

**Figure 4**

*(Neg. 3303) Case O. S. G. Composite of the above showing relatively little mineral dust in center of nodules, less in the larger one than in the smaller. Concentration of mineral aggregates in the peripheral "reactive" zone.*

**Figure 5**

*(Neg. 3304) Case O. S. G. A cluster of coalescent nodules surrounding a small island of atelectatic alveoli. The darkly staining spots are largely siderophages and asbestos bodies situated in the atelectatic tissue. The nodules are largely hyaline but show a peripheral, cellular, "reactive" zone. Van Gieson-iron stain.*
3. **Silicotuberculosis with slight asbestosis**

Case S. G., died at age 48, nearly 11 years after his last exposure mainly to silica (as diatomaceous earth) and to a lesser degree to asbestos. The total duration of dust exposure was nine and one-half years. He was forced to quit his job and to be institutionalized for six years because of tuberculosis. Following his discharge he performed light work for almost three years but was forced to seek readmission because of serious illness with positive sputum. The patient died 21 months after his last admission.

The results of a post-mortem examination indicated that death was due to an extensive tuberculous pneumonia which arose from a cavitating fibro-caseous tuberculosis of the lungs. There was a severe pneumoconiosis in which silica (as cristobalite) played a major role and asbestos, a relatively minor role. Right ventricular hypertrophy and dilatation (cor pulmonale) were present.

The silicotic nodules here are non-laminated and, as in the preceding cases, contain a moderate amount of mineral dust which tends to be concentrated at the periphery. The smaller nodules are stellate because of broad fibrous thickening of the bases of radiating alveolar septa (Figure 15). The mineral dust tends to extend beyond the nodules into the adjoining, widened alveolar septa (Figure 14). The larger nodules and masses, on the other hand, contain much less mineral dust. The outlines here are not stellate because adjoining septa are not thickened and no mineral dust is demonstrable beyond the confines of the nodules or masses.

Circumscribed areas of caseous necrosis are singularly devoid of mineral dust (Figure 16). There is likewise a striking paucity of mineral dust in areas of other tuberculous inflammation except in a region of tuberculous pneumonia where mineral dust is found in a few of the thickened alveolar septa which are still viable, and in the adjoining caseous alveolar content.

Some regions rich in asbestos bodies also contain many coarse and fine asbestos fibers of varying lengths, but no relationship can be demonstrated between the asbestos bodies and the asbestos fibers.

4. **Diatomite Pneumoconiosis**

Case E. S., died at age 71. Beginning with age 42 and continuing for 12 years to age 54 there was exposure to natural, calcined, and fluxcalcined diatomite powders in varying concentrations. Following recovery from an acute attack of coronary heart disease at age 54 he was transferred to office work with no more occupational exposure. A second attack three years later forced his retirement at age 57. For the next 14 years until his death the patient led a sedentary life, doing no work. During this last period there was a gradual increase in respiratory impairment. He died of cardiac and respiratory failure following an attack of bronchopneumonia.

![Figure 6](image6.png)  ![Figure 7](image7.png)

**Figure 6**: (Neg. 3304) Case O. S. G. Composite of the above showing areas within the hyaline collagen which contain no demonstrable mineral. Plentiful mineral dust is present in the peripheral "reactive" zone. There is also infiltration of the atelectatic alveoli by mineral aggregates.—**Figure 7**: (Neg. 3422) Case J. C. Single and coalescent silicicmum ligies, some of which are laminated. Although the haline reforms have a smooth, rounded outline, they are surrounded by a cellular "reactive" zone of variable width which gives the nodules a ragged, ill-defined outline. Van Gieson-iron stain.
The earliest x-rays taken at age 50, after eight years of dust exposure, showed definite accentuation of linear marking with a fine granular infiltration in all lung fields. A small band-like density first appeared over the right apical region four years later. No significant changes were noted in films taken at the time of retirement three years later, but films taken the year of his death showed far advanced diatomite pneumoconiosis with extensive bilateral coalescent lesions in both apices and bleb formation at the bases (Figure 17).

There was a severe induration of the right upper lobe which was continuous with a similar induration extending from the hilar region along the major bronchi and vessels. A section from a region of coalescent lesions in the right upper lobe exhibits large and smaller irregular, interconnected areas of dense collagen which enclose or

FIGURE 8

Figure 8: (Neg. 3429) Case J. C. Coalescent, smooth-contoured, laminated silicotic nodules surrounded by an irregular, cellular “reactive” zone. Partial atelectasis of adjoining alveoli. Van Gieson-iron stain.—Figure 9: (Neg. 3429) Case J. C. Composite of the above showing very heavy concentrations of mineral aggregates in “reactive” zone and extending into adjoining alveolar septa (lower right corner). There is relatively less mineral in the centers of the nodules.

FIGURE 10

Figure 10: (Neg. 3428) Case J. C. Portion of periphery of a silicotic nodule which has sunburst appearance due to great broadening of the bases of alveolar septa. The wide dark bands cutting across the acellular collagen are folds in the section. Van Gieson-iron stain.—Figure 11: (Neg. 3428) Case J. C. Composite of the above showing infiltration of the radially extended thickened septa by mineral aggregates. No mineral is demonstrable in the regions of the folds, presumably because poor contact with the glass does not allow the mineral particles to be embedded in the soft surface of the glass during the incineration.
surround large vessels and bronchi as well as islands of atelectatic or emphysematous parenchyma (Figure 18). A considerable amount of iron is present. Most of this iron is not associated with silica and is situated largely at the periphery of the masses although a small amount is also found within the collagen masses and in some of the atelectatic air spaces.

The fibrous masses are largely acellular and partially hyalinized. A reactive peripheral zone of lymphocytes, monocytes and dust-laden macrophages is frequently present. The fibrous masses are associated with peri-focal atelectasis. The atelectatic alveolar walls tend, in most instances to be arranged parallel to the periphery of the nodule. Such alveolar walls are generally not thickened. But there are also club-shaped, thickened alveolar septa which tend to extend radially from a collagenous mass and thereby give it a stellate or sunburst appearance (Figure 19). Alveolar walls generally are infiltrated by lymphocytes and monocytes.

Much of the collagen masses is free of mineral dust but foci of dense mineral deposits are found irregularly scattered within the masses. In general, the disposition of the mineral dust is unpredictable although it is generally abundant in the reactive cellular zone on the periphery of the collagen masses. Adjacent alveolar walls, whether thickened or not, commonly are heavily infiltrated by mineral dust (Figures 20 and 22).

There is pronounced vascular sclerosis and perivascular fibrosis with narrowing of vascular lumens (Figure 21). The profuse infiltration of the perivascular tissues and, indeed, of the vessel walls themselves with mineral dust is of great interest (Figures 20 and 22).

Analysis of the dried lung tissue for mineral constituents disclosed that the ash content was 6.98 per cent; silica, 1.62 per cent; quartz, 0.09 per cent; and crystobalite, 0.52 per cent.

5. Asbestosis

Case E. N., age 60, had worked for 29 years in an asbestos mill as bagger, oiler, sweper, grader, and tester. During this time he received significant exposure to asbestos dust. The first chest x-ray film taken after 22 years of exposure indicated an advanced asbestosis. Because he was asymptomatic he remained at work and continued in his occupation without a day of sick leave for seven years longer. Shortly thereafter he rapidly became dyspneic and cyanotic. An electrocardiogram at this time was indicative of cor pulmonale. From then until his death two years later he was

FIGURE 12: Case S. G. Advanced pneumoconiosis from inhalation of mixed dust consisting of diatomaceous earth and asbestos, complicated by tuberculosis.
totally disabled and he died with severe cardiac decompensation and generalized anasarca.

The lungs appeared enlarged and showed a greatly coarsened texture with numerous thick bands of scar tissue occurring in an increasingly prominent manner toward the lower portion of the upper lobes and still more outspokenly toward the bases. There were subpleural emphysematous spaces 2 to 4 mm. in diameter. Subpleural fibrous plaques 5 to 6 mm. in diameter were also present. The pleura was greatly thickened. Although the bronchi appeared normal, the blood vessels were appreciably thickened.

Microscopically there is irregular fibrous thickening of the parenchyma with severe distortion of the architecture (Figure 23). Few recognizable alveoli are found. Instead, there are very large, irregular, distended spaces; most of them bounded by thick fibrous septa. Larger areas of fibrosis are found in the regions of vessels and

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**FIGURE 13**  
*Figure 13:* (Neg. 3309) Case S. G. A stellate silicotic nodule with a broad cellular reactive zone about a central acellular, hyaline focus. Van Gieson-iron stain.

**FIGURE 14**  
*Figure 14:* (Neg. 3309) Case S. G. Composite of the above showing heavy concentration of mineral dust in broad reactive zone with infiltration of thickened radial septa by dust aggregates.

**FIGURE 15**  
*Figure 15:* (Neg. 3314) Case S. G. Large, well delimited area of caseous necrosis stretching across upper portion of field and a smaller one in lower right corner. There is a silicotic nodule between these two areas of caseous necrosis. Another silicotic nodule occupies the lower left corner. Van Gieson-iron stain.

**FIGURE 16**  
*Figure 16:* (Neg. 3314) Case S. G. Composite of the above showing complete demineralization of the areas of caseous necrosis.
bronchi, generally with considerable vascular sclerosis. Relatively little mineral dust is present in the altered parenchyma (Figures 24 and 26) and much of the scarred tissue contains no demonstrable dust. The greater part of the mineral dust appears to be non-fibrous. At high magnifications, however, scattered asbestos fibers up to 50 to 75 microns in length are seen among the crystalline plates and refractile granules.

Because no definitive information was available regarding the stability of chrysotile asbestos fibers under our conditions of incineration and acid-treatment, such data were obtained by two methods. In the first method slightly over 5 g. of finely divided chrysotile asbestos was placed into a Gooch crucible and after weighing (following a sojourn in a drying oven), incinerated at 550° C. for one hour. The crucible with its content of asbestos was then placed in a beaker containing concentrated hydrochloric acid for 30 minutes. The crucible rested on glass beads to allow for some circulation of the acid. After removal from the acid the contents of the crucible was washed with running distilled water until the wash water was chloride free (negative test with AgNO₃). Following drying overnight in an oven the crucible and content were weighed again. The total weight loss from this procedure was found to be only 6.3 per cent.

The second method consisted of preparing a thin smear of ultrafine asbestos dust upon a glass slide and photographing a selected field under dark field conditions before and after incineration with its subsequent acid treatment and wash. Comparison of the two pictures shows that the finest particles visible in the print before incineration are also visible in the pictures taken after the incineration and acid treatment.

Thus, we are reasonably certain that the paucity of mineral dust in our asbestotic tissue is real and not artefactual.

**Discussion**

Simpson and Strachan differentiated between an arrested silicotic nodule and one in which the inflammation, and therefore further growth, was still progressing. The criterion for the progressiveness of a silicotic nodule, according to these writers, is the presence of a “reactive zone” on the periphery of a nodule. As seen in the present case material the reactive zone consists of lymphocytes, monocytes, fibroblasts, and dust-laden macrophages. One of the more significant findings in this study is the frequent concentration of mineral dust in the reactive zone on the periphery of a

![Figure 17: Case E. S. Far advanced diatomite pneumoconiosis with coalescent lesions and numerous bullae.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21320/ on 04/29/2017)
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silicotic nodule or mass and the frequent permeation of the mineral particles into the adjoining less involved or non-indurated tissues. It would appear that the presence of the high mineral dust concentration in the peripheral portion of the silicotic nodule offers a logical explanation for the existence of the reactive zone in that region and also throws light on the mechanics of the growth of the nodules.

The shape of the silicotic nodule, whether smooth or stellate, has been used by diBiasi\textsuperscript{4} as a criterion to differentiate between nodules of pure

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Figure18}
\caption{Figure 18: (Neg. 3423) Coalescent nodules and plaques of non-laminated collagen associated with many vessels of varying size most of which are sclerotic. Atelectasis and focal emphysema of nearby parenchyma. Van Gieson-iron stain.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Figure19}
\caption{Figure 19: (Neg. 3457) Case E. S. A collagenized nodule adjacent to a large vein. The reactive zone is not sharply defined. Adjoining septa are clubbed and radially arranged so as to give sunburst appearance. Van Gieson and iron stain.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Figure20}
\caption{Figure 20: (Neg. 3457) Case E. S. Composite of the above showing mineral infiltration into the reactive zone, into the radiating septa, and into a portion of the vein wall.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Figure21}
\caption{Figure 21: (Neg. 3450) Case E. S. Extensive, irregular fibrosis which includes the pleura. Adjacent surviving parenchyma is atelectatic. There are a number of sclerotic arteries with narrowed lumen.}
\end{figure}
silicosis (smooth-contoured) and those of mixed silicosis. A mixture of silica with other dusts has been presumed to result in stellate nodules. Although the present study offers no data in refutation of diBiasi’s contention, nevertheless the photomicrographs suggest a purely mechanical explanation for the genesis of either a smooth-contoured or a stellate nodule.

If alveolar walls at the periphery of a nodule become infiltrated with irritating mineral particles, interstitial edema followed by cellular infiltration and finally fibrosis of the structures result. Whereas initially it is turgescence which prevents collapse of the alveolar walls, later the rigidity becomes permanent by collagenization of the septa. Because the mineral infiltration is heaviest at the bases of the rigidly extended alveolar septa, the fibrosis also is greatest in this region. Thus the nodule receives a stellate or sunburst periphery.

In contrast, if the mineral particles do not infiltrate into the adjoining alveolar walls, the latter remain soft and pliable and tend to be closely applied circumferentially against the nodule by pressure of the surrounding structures. Thus the nodule retains a smooth contour.

It is highly probable that laminations in a silicotic nodule represent lines of growth and arrest. Contrariwise, the absence of laminations in a nodule suggest that its growth has been a steady one, without growth arrest. The extent to which circumferentially applied atelectatic alveolar walls also contribute to the laminated appearance of silicotic nodules must await future study. All observations to date point to a centrifugal dissemination of mineral dust in a growing silicotic nodule but it is not known at present the extent, if any, to which new silica particles from the surrounding lung tissue may contribute to the peripheral growth of a nodule.

That silicosis is a progressive disease is generally recognized. The observations recorded in the present study anent the peripheral localization

**FIGURE 22**

*Figure 22:* (Neg. 3458) Case E. S. Composite of the above. Much mineral dust diffusely infiltrates practically all tissues including those not appreciably involved by fibrosis. The thickened arteries also show mineral infiltrates in their walls. This is a picture of fulminating dissemination of mineral dust 17 years after the last dust exposure.—*Figure 23:* (Neg. 3365) Case E. N. Irregular band-like and septal fibrosis associated with severe emphysema, typical of asbestosis. Van Gieson-iron stain.
of mineral particles in the reactive zone of silicotic nodules and the tendency of these particles to infiltrate adjacent less involved or near-normal structures offer a logical explanation for this progressiveness.

It is of interest to point out that in the case of J. C. who had his last exposure to silica at age 47 and who died 25 years later at age 72 there are laminated pulmonary silicotic nodules with a reactive cellular peripheral zone (indicative of relatively recent reactivation) and dissemination of the mineral dust into adjoining tissues. Similarly, in the case of E. S. who had his last exposure to diatomite dust at age 54 and who died 17

Figure 24: (Neg. 3439) Case E. N. Large, irregular scar enclosing numerous sclerotic vessels and involving adjoining alveolar septa irregularly. Van Gieson-iron stain.—

Figure 25: (Neg. 3439) Case E. N. Composite of the above showing very little mineral in the scar tissue.

Figure 26: (Neg. 3434) Case E. N. Irregularly thickened walls of tremendously enlarged air spaces. The darkly staining foci are due to the presence of iron and asbestos bodies.—

Figure 27: (Neg. 3434) Case E. N. Composite of the above showing little mineral dust, much of it associated with the iron. Most of the dust is in the form of granules and plates. Scattered asbestos fibers are also present.
years later at age 71, the disease at the time of death was in a state of 
fulminant progression as may be judged from the degree of dispersion of 
the mineral dust throughout the lung tissue.

In contrast to silicosis, asbestosis is not a progressive disease. Basically 
silicosis owes its characteristic progressiveness to the fact that silica 
particles deposited in one site, may be transported by tissue fluid (generally 
as edema fluid) to uninvolved portions of the lung to initiate new lesions.\(^2\) 
Asbestos fibers, in contrast, cannot pass the intricate barrier presented by 
the lattice work of tissue fibers as readily as particles of the same diameter 
which are substantially spherical. Also, since asbestos has a very consid-
erable solubility, dissolution of the fibers offers a plausible explanation for 
the paucity of mineral found in asbestotic scar tissue. Both of these factors 
prevent the transport of significant amounts of asbestos fibers from their 
site of deposition to new locations and therefore explain the non-progres-
siveness of asbestosis.

The absence of mineral dust from areas of tuberculous inflammation, 
particularly from areas of necrosis as demonstrated in Case S. G. would 
seem to offer a facile differentiation between a silicotic and a tuberculous 
nodule. To be certain of such differentiation, however, many more cases 
of silicotuberculosis would have to be studied in order to determine the 
frequency of the exception to this finding. Furthermore, it is also well to 
consider that some silicotic nodules may be almost completely demineralized 
in the absence of an associated tuberculous inflammation.

Vascular sclerosis is seen frequently in silicosis,\(^9\) and seems to be espe-
cially pronounced in case E. S. in which cristobalite is the most important 
fibrogenic agent. The striking feature of this case is the extensive infiltr-
ation of mineral particles into the walls of the diseased vessels, a phenom-
enon not previously observed in any other pneumoconiosis. Zaidi et al\(^9\) have 
demonstrated the greater fibrogenic potential of cristobalite over quartz 
but whether or not the infiltration of vessel walls by mineral dust is a 
characteristic feature of cristobalite pneuomoconiosis can only be decided 
by further study.

**SUMMARY**

By means of a photographic method it has been possible to study the 
topographic distribution of otherwise occult mineral dust deposits in the 
lobes. The more important features of mineral dust distribution in several 
types of silicosis as well as of asbestosis are described and illustrated.

The progressive character of silicosis is explained on the basis of the 
transport of silica from the site of deposition to new locations where fresh 
inflammatory reactions are initiated. Such transport is demonstrated in 
ancient hyaline nodules which exhibit a peripheral "reactive" zone of 
younger, cellular inflammatory tissue in which mineral dust particles are 
heavily concentrated. Another striking indication of how the transport of 
mineral dust may lead to the development of new inflammatory foci is seen in 
the demonstration of heavy mineral dust infiltrations into uninvolved 
parenchyma adjacent to silica nodules.

The non-progressive character of asbestosis is explained by the pauc-
city of demonstrable mineral in asbestotic scar tissue and by the difficulty 
of mobilizing and transporting asbestos fibers through the impeding lattice 
work of stromal fibrils.
The significance of rounded versus stellate, and of laminated versus non- 
laminated silicotic nodules is discussed in the light of the present observa-
tions and demonstrations.

Areas of tuberculous inflammation, particularly of caseous necrosis, 
though occurring in the midst of silicotic tissue are shown to be free of 
demonstrable mineral dust particles.

An example of the cristobalite type of silicosis is shown to possess a 
fulminating progressiveness 17 years after the last exposure to this dust. 
An interesting side light to this disease is the demonstration of mineral 
dust infiltrating vessel walls.

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RESUMEN

Por un procedimiento fotográfico ha sido posible estudiar la distribu-
tión topográfica de depósitos de polvos minerales que de otra manera 
permanecerían ocultos. Se ilustra la distribución característica de polvos 
minerales en los tipos diversos de silicosis y de asbestosis.

El carácter progresivo de la silicosis, se explica sobre la base del trans-
porte de sílice desde el lugar de depósito inicial hacia nuevos sitios donde 
se inician reacciones inflamatorias nuevas. Ese transporte se demuestra 
por antiguos nódulos hialinos que muestran una zona periférica “reactiva” 
de tejido más joven inflamatorio en la que se han concentrado partículas 
minerales en gran proporción.

Otra notable indicación de cómo el polvo mineral transportado puede 
conducir a la creación de nuevos focos inflamatorios se ve en la demostra-
ción de grandes infiltraciones de polvo mineral hacia tejido no comprome-
tido adyacente a los nódulos de sílice.

El carácter no progresivo de la asbestosis se explica por la escasez de 
mineral demonstrable en el tejido cicatricial de asbestosis y por la dificultad 
de movilizar y transportar las fibras de asbestos a través de las mayas 
que dificultan esto y a través de las fibrillas del estroma.

A la luz de las observaciones actuales se discute la significación de los 
nódulos redondos contra estelares y laminados versus no laminados.

Las áreas de inflamación tuberculosa, particularmente de necrosis caseo-
sa, aunque ocurren en medio de tejido silícítico se demuestra que están 
libres de polvo mineral. Un ejemplo del tipo de silicosis llamada “cri-
sto-balito” se demuestra que posee una evolutividad progresiva 17 años después 
de la última exposición al polvo de sílice. Una interesante demostración 
colateral de esta enfermedad es la presencia de polvo de mineral infiltrando 
las paredes de los vasos.

RESUME

Il été possible d'étudier par photographie la distribution topographique 
des dépôts occultes de poussière minérale dans les poumons. Les auteurs 
décrivent les aspects les plus caractéristiques et les modalités les plus im-
portantes de distribution des poussières minérales dans différents types de silicose et d’asbestose.

Le caractère progressif de la silicose s'explique par la migration de la silice de son point de dépôt vers les localisations nouvelles où paraissent des réactions inflammatoires récentes. Cette migration est démontrée dans les anciens nodules hyalins, où existe une zone périphérique “réactionnelle” de tissu inflammatoire cellulaire jeune, dans lequel les particules de poussière minérale sont fortement concentrées. Une autre démonstration frappante de la migration de la poussière minérale est offerte par le développement de nouveaux foyers inflammatoires avec infiltrations importantes de poussière minérale dans une zone du parenchyme jusqu'alors non atteinte et adjacente aux nodules de silice.

Le caractère non évolutif de l’asbestose est expliqué par le peu de minéral mis en évidence dans le tissu cicatriciel asbestosique et par la difficulté du cheminement et du transport des fibres asbestosiques à travers les lacets obstructifs des fibrilles du stroma.

Les auteurs discutent la signification des nodules silicotiques arrondis par rapport à ceux qui sont étoilés, et de ceux qui sont lamines par rapport à ceux qui ne le sont pas, en fonction, des remarques et les démonstrations qu’ils viennent de présenter.

Les zones d’inflammation tuberculeuse, particulièrement la nécrose caséeuse, bien que survenant au milieu de tissu silicotique, ne contiennent aucune particule de poussière minérale visible.

Un cas de silicose cristobalite se présente avec un potentiel évolutif foudroyant 17 ans après la dernière exposition à ces poussières. Un autre point intéressant de cette affection est l’existence de poussière minérale infiltrant les parois vasculaires.

ZUSAMMENFASSUNG


Der nicht fortschreitende Charakter der Asbestose wird erklärt durch die geringe Zahl von nachweisbarem Mineral in asbestotischem Narbengewebe und durch die Schwierigkeit, Asbestfasern zu mobilisieren und zu transportieren durch das störende Gitterwerk der Grundgewebsfibrillen.


REFERENCES