

## THE PNEUMONOCOONIOSES IN SOUTH WALES

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(With Plates II–XI)

In an article on Pneumoconiosis published in 1935 I attempted to simplify the problem of lung diseases caused by exposure to dust in industry by means of a classification which might allow of the placing of individual cases into appropriate categories. The importance of some such classification has been emphasized during the past year or so by the gradual realization of the fact that, especially in the South Wales coalfield, cases of more or less disabling lung conditions are frequently met with which cannot conscientiously be fitted into the category of "silicosis", as defined at the International Conference at Johannesburg in 1930. These cases cannot be "certified" for compensation under existing Statutes, and yet present a picture of respiratory distress and dyspnoea on exertion which marks them out as quite unfit for work.

Cases of this type call for serious consideration, and it is obvious that, in the near future, close study will have to be directed to the exact abnormalities underlying their disability, and this not only from the clinical and pathological, but also from the physiological aspects. In this paper an attempt is made to indicate, if not to explain, some of the pathological and histological features of lung specimens examined by me at Cardiff.

A glance at recent medical literature bearing on dust diseases in South Wales will suffice to show that workers in the mining industry in this area are liable to be affected, in varying degree, by the inhalation of the mineral dusts to which they are exposed. In certain small departments of the industry, in those concerned to an especial degree with drilling in hard rock, such as "hard-heading" workers, borers, and sinkers, the dust hazard is comparable in every respect to that of gold miners on the Rand, tin miners in Cornwall, or workers in any of the "refractories" industries.

Tattersall (1926) was one of the first to call attention to the occurrence of silicosis among hard-ground workers in the South Wales coal mines. At the other end of the scale, and in connexion with workers exposed to pure coal dust only, without any significant admixture with stone dust, Collis & Gilchrist (1928) described a form of pneumoconiosis in coal trimmers employed in the Cardiff Docks, and stated that "clinical observation by X-ray examination discloses that, after years of work, the lungs of coal trimmers are not normal, and exhibit signs similar to those widely regarded as characteristic of silicotic fibrosis".

Bathgate, in a recent thesis, as yet unpublished, has analysed his X-ray findings in forty-four coal trimmers examined by him in the course of panel practice, or seen through the courtesy of Dr J. C. Gilchrist, and arrives at conclusions corresponding to those of Collis & Gilchrist. It is of interest to note that eleven of these persons were found by him to be disabled and were regarded as unfit for work owing to their pulmonary condition.

Harper (1935), referring to his conclusions based on X-ray examinations, calls attention to the occurrence of somewhat similar appearances in "screeners", men who are employed above ground in screening the coal as it leaves the pits and who, therefore, like the trimmers, encounter almost pure coal dust and only a minimal amount of stone dust.

We have, therefore, evidence of two sharply contrasted types of pneumoconiosis in South Wales; the pure silicosis of hard-heading and other stone workers in the collieries and the pure anthracosis of coal trimmers at the docks and coal screeners on the surface at the coal mines.

But the average colliery worker belongs to neither of these simple categories. His life is spent in work at the coal face or in the "roads". He is exposed to mixed dusts from the coal he works in, from the rock in which the coal seams occur, and from the stone dust used, except in the anthracite collieries, as a precaution against explosions. Such a worker, though not exposed to the intense stone dust clouds encountered by those working as borers or sinkers or in hard-headings, may still occasionally meet considerable concentration of silicious dust while engaged in "ripping" the roof, or repairing the roads. He may never experience such intensive coal dust clouds as must be breathed in by the trimmer in the holds of ships; such clouds would be fraught with danger of explosion underground and are prevented, in the mines, by the elaborate methods of ventilation in constant use; but still he is exposed to some coal dust all day and every day during his working life.

And, while the collier may, now and then, inhale a certain amount of free silica, he is constantly exposed to combined silica in the form of sericite, to which W. R. Jones (1933) has called such grave attention as a possible factor in the production of silicosis, and as silicates in the shale dust added to the mine air to prevent explosions; while he is also exposed to the gases and fumes liberated in shot firing, which have been proved, in their animal experiments, by Irwin *et al.* (1934) to be capable of aggravating the effects of active dusts simultaneously inhaled.

The pathological effects of these mixed dusts, when breathed into the lungs, have been described by Cummins (1927), Cummins & Sladden (1930), Cooke (1932) and Sladden (1933) and a convenient name for the condition, suggested independently by Cummins and by Cooke, is silico-anthracosis.

From these preliminary remarks, it will be evident that the problem of pneumoconiosis in South Wales is not a simple one and that there is every excuse for the confusion undoubtedly existing in the minds of medical men practising in the valleys or called upon, in the course of their professional work,

to adjudicate upon radiological or post-mortem findings on colliers in their district.

The provisional classification published by me in 1935, and already referred to, is here reproduced as a basis for the further pathological and histological consideration which follows:

*Type 1.* Dust retention and accumulation determined by the operation of a chemically active dust inhaled into the lungs; resulting in nodular fibrosis and, in X-ray films, bilateral symmetrical mottling.

*Examples:* silicosis; asbestosis.

*Type 2.* Retention and accumulation of an inert dust, determined by lymphatic blockage through previous or simultaneous inhalation of a chemically active dust; resulting in nodular and diffuse fibrosis, dust accumulations, and, in X-ray films, bilateral symmetrical mottling and irregular diffuse shadows.

*Examples:* silico-anthracosis; silico-siderosis.

*Type 3.* Accumulation of an inert dust through excess of entry over elimination; resulting in black anthracotic but usually well-aerated lungs without much fibrosis and, in X-ray films, *nil* or partial small mottling.

*Examples:* the anthracosis of coal trimmers and screeners.

*Type 4.* Localized dust retention in areas of obstructed lymph flow due to old tuberculous foci or other lesions; resulting in extensive, sharply localized accumulations of dust in fibrotic "indiarubber-like" areas of non-symmetrical distribution, and, in X-ray films, "cricket ball" or "pseudo-neoplasm-like" shadows.

*Examples:* the fibrotic aggregations of inert dust occasionally present in individual cases in all types of pneumoconiosis, but especially frequent in silico-anthracotic persons, in coal trimmers, and in screeners.

*Type 1.* It may strike clinicians as unwarrantable to include asbestosis as well as silicosis as exemplifying Type 1, since the type of fibrosis in the former is not usually regarded as "nodular". Nodules, however, do occur though they are much rarer and much less definite than in silicosis, and they have been figured and described by Simson (1928) and by Gloyne (1933). There are, indeed, curious and interesting differences between the clinical and pathological states in asbestosis and silicosis, to which further reference will be made in this paper, but, from the point of view of classification, both conditions obviously fall into Type 1. They have a further important character in common, that both are associated with a definitely increased liability to tuberculosis, a character not so evident in the other types of pneumoconiosis.

So far as South Wales coal miners are concerned, it is curious that the pure silicosis now known to occur so frequently and so severely in borers, sinkers and others working in stone, passed so long unnoticed. Its presence in the South Wales coalfield is now established and the cases tend to be, on the whole, more severe than the present-day cases on the Rand, and only to be compared with those formerly met with at Johannesburg, in the old days before the Silicosis Bureau existed, as described and figured by Watt *et al.* (1916).

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It is unnecessary to describe the classical lesions in silicosis as this has been done so well already, and especially by Strachan & Simson (1930) in their contribution to the International Silicosis Conference; but a glance at Plates II and III, illustrating the extent of the fibrosis and accompanying lung deterioration in a South Wales hard-ground worker will suffice to remind us that it is not only in South Africa that such cases occur.

*Type 2.* Of greater practical importance in South Wales is Type 2 where the chemically inert coal dust is retained and accumulated to an inordinate extent through previous or simultaneous inhalation of a chemically active dust, the latter leading to lymphatic obstruction so that the inert dust cannot escape from the lung. These silico-anthracotic persons are very numerous throughout the coalfield and exhibit the whole gamut from working health to severe dyspnoea and marked disability. Plates IV and V illustrate the macroscopic and microscopic appearances seen in cases of this type.

Sladden (1933), in his extremely valuable paper on the silica content of lungs, suggests that "in the 'ordinary' coal miner exposed to minimal doses of silica year by year, the silica accumulates only slowly, a substantial part being in earlier years passed on from the lungs. A prolonged or more intensive 'bombardment' of the lungs causes progressive damage to the lymphatics, and favours an accelerating rate of accumulation of dust, silicious or other, so that after age 50 the damage to the lungs from the silica fraction may have assumed serious proportions".

While it is doubtless true that the silica fraction initiates most of the damage, it seems to me that the accumulated coal dust itself ultimately amounts to a foreign deposit of sufficient extent to act like other foreign bodies in exciting a certain amount of tissue reaction; slight fibrotic proliferation and other alterations of a sort calculated to add to the pathogenic effect.

The tendency of these silico-anthracotic cases to develop tuberculosis, though masked by the detoxicating effect of the coal dust, is nevertheless quite definite. Dr Enid Williams found six out of one hundred retired colliers in good health but for dyspnoea, to be sputum positive, and Dr P. K. Sen, in an unpublished thesis, reports 12 per cent positive sputa in 100 cases of pneumoconiosis in South Wales.

*Type 3.* Type 3 of my classification comprises those inert dust accumulations which are so frequently found in the lungs of coal trimmers, screeners and other workers exposed to heavy concentrations of coal dust. These dust collections represent the excess of dust-entry over dust-disposal in and from the lungs. The dust, being chemically inert except for the small proportion of silica native to it (Sladden quotes Redmayne as placing this at 0.6 per cent), excites little or no nodular fibrosis, but there may be considerable diffuse fibrosis around the large coal-dust accumulations where these occur. It is to cases of this kind that the term Anthracosis should be confined. It is common to hear the word used for the small localized carbon deposits in the lungs of city dwellers, but the latter condition is quite unimportant.



To what extent the intense anthracosis of trimmers and screeners may be clinically important is still unsettled and the evidence is conflicting. The Industrial Pulmonary Disease Committee of the Medical Research Council (1934) in its *Report* on coal trimmers exposed to anthracite dust at Swansea, states that "the Committee was unable to find, in this series of cases, any evidence that the inhalation of anthracite or other coal dust had caused fibrosis of the lungs". The X-ray films, which I saw as a member of the Committee, fully justified this claim so far as such a claim is allowable on radiological and clinical examination alone. The findings of Collis & Gilchrist, of Harper, and of Bathgate, already referred to, likewise based on radiological data, tell a different story; perhaps because they deal with groups of individuals less critically selected. Five coal trimmers included in Sladden's tables all showed but a low silica content on analysis, and he concludes "that coal dust alone, apart from silica, cannot be regarded as a cause of serious fibrosis of the lungs, and such minimal fibrosis as does occur in this class of worker can be explained as due to the action of traces of silicious dust present even in high-grade 'pure' coal". With this view I am in general accord, but it is still possible that, apart from fibrosis, coal dust itself may cause lung injury. The as yet unpublished findings of Bathgate, which he has kindly allowed me to refer to, suggest that this is so; and it is just possible that a study of the coal trimmers in Cardiff Docks might bring to light differences from the findings of the Medical Research Council at Swansea.

In regard to tuberculosis liability, Type 3 seems to be exonerated. In seventy-six coal-trimmer patients, Bathgate found no instance of a positive sputum, and my own studies of the lungs of some half-dozen coal trimmers dying of respiratory disease have been entirely negative for tuberculosis.

*Type 4.* This type is characterized by localized dust retentions in areas of obstructed lymph flow in the lungs, resulting from old stabilized tuberculous or other lesions. Such dust deposits may complicate the picture in any of the first three types of pneumoconiosis, destroying the symmetrical X-ray appearances so characteristic of dust inhalation effects and bringing about vast irregularly placed shadows suggestive of acute tuberculous infiltration or advanced malignant disease. It is not sufficiently realized to what extent these old chronic scars or dry cavities interfere with local lung drainage. Such an accumulation of coal dust is well seen in Plate VI, the photograph of a lung from a silico-anthracotic coal miner. Some observers appear to be prepared to certify "silicosis" on X-ray appearances depending on these accumulations. While, however, such shadows do appear in true silicotics now and then, depending on old lesions irregularly placed in the lungs and tending to become the centres of dust accumulation, they are equally common and quite as large and dense in coal trimmers and screeners. It is, therefore, quite wrong to accept them as evidence of silicosis.

In the above account of the classified types of pneumoconiosis, much stress has been laid upon fibrosis, whether nodular or diffuse. Fibrosis, how-

ever, though the direct outcome of the reaction of the tissues to active dusts, is not, in itself, the only abnormality in pneumoconiotic lungs, nor does the amount of fibrosis necessarily correspond with the degree of dyspnoea noted.

The object of this paper is to call attention to certain less frequently described alterations which seem to play an important role in cases of pneumoconiosis. The fibrotic changes have frequently been described and need not be discussed here, but consideration will be given, instead, to emphysema, bronchitis and the changes in the lymphatic glands.

As Watt *et al.* (1916) say, "the cardinal symptom" of silicosis "is shortness of breath on exertion". The same authors call attention to the rigidity of the chest in such cases and draw an interesting comparison between emphysema and silicosis as follows: "In emphysema, the chest is distended and barrel shaped, in fibrosis of the lungs it is typically contracted." This observation, made by Watt & Irvine in 1912, is worth noting and is borne out by the recent work of Hurtado and his colleagues (1933) who compared the radiological chest volume in various respiratory disabilities. They state that "in undoubted cases of pulmonary emphysema there is a conspicuous decrease in the ability to expand the chest" . . . "Reduction of chest expansion is *not* as characteristic of pneumoconiosis and pulmonary fibrosis as it is of pulmonary emphysema." This inability to expand in emphysema depends, of course, upon the fact that the chest is already nearly completely expanded. Meakins & Christie (1934) may be quoted in this connexion. They write: "Whatever the etiological factor, the cause of the impairment of function in emphysema evidently lies in a loss of pulmonary elasticity. As this loss of elasticity progresses, the lungs can no longer resist the traction of the chest wall and they distend until a position approaching full inspiration is reached. The lung can now no longer deflate during expiration by the normal process of passive elastic recoil but has to be actively compressed by the extrinsic muscles of expiration."

*Emphysema in pneumoconiosis.* In South Wales cases of all three types, emphysema appears to play an important part in causing the marked dyspnoea noted as an invariable feature whether of pure silicosis, silico-anthraco-sis, or advanced anthracosis. Clinically, the importance of this emphysema has been stressed by P. K. Sen as the essential factor in coal-miner's dyspnoea and he establishes the fact that the dyspnoea is often out of all proportion to the extent of lung fibrosis as assessed by X-ray findings. Pathologically, I can state positively that, in the great majority of pneumoconiotic lungs examined by me at Cardiff, marked emphysema has been a conspicuous feature. This state of emphysema is clearly visible on cutting into suitably fixed lung specimens. It varies from fine alveolar distension to honeycomb pitting all over the surface of those portions of the lungs not solidified by dust accumulation and fibrosis, as may be seen in Plates II and III. In many cases these honeycomb pittings co-exist with subpleural blebs or more frequently with air-distended bullae, such as have been described and figured by William Snow Miller in emphysema (1926). My studies of these honeycomb distensions and bullae



lead me to the opinion that the former result from a giving way of the sacculi alveolares, while the bullae appear to be formed by a fusion and distension into one cavity of a respiratory bronchiole with its whole atrial and saccular apparatus included. This concept is supported by the fact that the walls of the bullae usually exhibit partial septa arranged like irregular shelves in their concavities. Microscopically, I have often found them to be lined at some point, usually close to the orifice, by low cubical epithelium; but the walls are often quite smooth as a result of distension. These bullae sometimes project as hernial distensions of the visceral pleura yet, in most cases, are fixed and immovable. It is much rarer to find movable blebs under the pleura; but these latter, as pointed out by Miller, can be shifted about by digital pressure and are due to rupture of alveoli and escape of air into the tissues.

*Elastic tissue in pneumoconiotic lungs.* All these considerations have led me to examine the arrangement of elastic tissue in these pneumoconiotic specimens and to compare them with normal lung tissue. For this purpose, Verhoeff's stain and Murray's modification of Weigert's method were employed.

Text-book descriptions of the arrangement of elastic tissue in the lung are not very enlightening, and I have been driven to seek out some sort of concept for myself. Maximow & Bloom (1931) state that "the interalveolar septa carry a fair number of elastic fibres. These are continuations of the elastic membranes of the bronchioles, are decidedly fewer in number than the reticular fibres and are morphologically different from them in that they are usually straight and thin with only occasional branches." This is doubtless correct, and I think I have succeeded in tracing these elastic strands from the bronchioles into neighbouring alveolar walls. But there is also direct connexion between the elastic tissue in the walls of small blood vessels and the alveolar septa, as is shown in Plate VII, fig. A from a normal (child's) lung. I have also been able to confirm the beautiful observations of William Snow Miller on the elastic layers in and under the visceral pleura, and the intimate association of the latter with the underlying alveoli. I am led to think that the whole areolar structure of the lung and its alveoli is rendered extensible by elastic fibres from the walls of the bronchioles and the blood vessels, which form a mutually interdependent recoil system throughout the lobules. Any local destruction of bronchioles or blood vessels lets down this network and brings into play unequal stresses and tensions. The effect of loss of elasticity in any extensive area of lung must tend, it would seem, to the distension, rather than the collapse, of terminal bronchioles and alveolar ducts. This, perhaps, may underlie the honeycomb distensions and bullae already referred to. In the pneumoconiotic lung, stained for elastic tissue, the first thing that strikes the eye is an apparent increase in elastic tissue as compared to the normal. This has been noted by Gloyne (1933) also, in connexion with asbestosis. A closer examination shows, however, that the elastic tissue is only more visible because it is no longer an effective part of an evenly distributed recoil mechanism. It is found lying in detached strands or skeins which are obviously the residue

of destroyed or collapsed bronchioles or arterioles. This is illustrated in Plate VII, fig. B, and Plate VIII.

These isolated strands and skeins have lost their *points d'appui* and are no longer effective elements in lung elasticity owing to the destruction of key points in their mesh.

The histological appearances suggesting injury to the elastic tissue seem to fall into line with the marked dyspnoea and emphysema noted in the cases and specimens examined.

Do these findings suffice to indicate some essential difference between the silicotics of the Rand and the silicotics and silico-anthracotics of South Wales? To me, they appear to amplify rather than to conflict with the observations of Watt and Irvine. There is an element in pneumoconiosis which is absent in classical pulmonary emphysema. That element is lung fibrosis and pleuritis. It is to the fibrosis and pleuritis that the shrinkage is due. This fibrosis also prevents the relaxation of the emphysematous pneumoconiotic chest to the typical "inspiratory" position characteristic of ordinary emphysema. In pneumoconiosis, the dyspnoeic state is the resultant of consolidation and fibrotic shrinkage on the one hand and emphysematous distension on the other.

*Bronchitis.* Dr J. S. Haldane drew a sharp distinction between "the undoubted cases of silicosis among coal miners who . . . have worked in highly siliceous rock and . . . the large number of other cases . . . among men who have not worked in such rock". Of the latter he said: "I think these cases are primarily cases of either bronchitis or ordinary phthisis, the collection of dust with such extra fibrosis as is found on post-mortem or X-ray examination being due to paralysis by bronchitis of the normal process of dust elimination." The respect and affection of all who knew the late Dr Haldane claims for his views on this subject, ably traversed by Prof. Kettle at the same meeting, a close examination from the pathological point of view. It is exceedingly difficult to be sure of the real cause of absence of ciliated epithelium in the main bronchi of lungs removed at autopsies in the Welsh valleys and sent to the laboratory in preservative solutions. Under such circumstances, the delicate epithelial lining often perishes in transit. Where the lungs are removed by a trained pathologist and at once squeezed and then re-distended by means of a 10 per cent solution of formalin in saline, it is usual, even in markedly silicotic lungs, to find the ciliated epithelium well preserved. Even in specimens posted from the valleys, it is often possible to find well-preserved ciliated epithelium in the more remote subdivisions of the bronchi, though it may be absent from the larger tubes.

In a series of twenty-two cases, which included all three types of pneumoconiosis, I found intact ciliated epithelium in the main bronchi in five and in the bronchioles in ten more. The failure to find it in the others was probably due to deterioration in transit or to the sections not including suitable bronchiolar tissue. It would appear certain, therefore, that the essential machinery

for dust elimination, the ciliated epithelium, is often retained to the end, even in advanced cases of silicosis and anthracosis. Many specimens show, however, clear evidence of a terminal septic bronchitis, albeit with ciliated epithelium still in position in spite of the purulent contents of the tubes. Such a case is illustrated in Plate IX. This patient had retained his ciliated epithelium through many years of dust exposure, and had attained to advanced pneumoconiosis although the dust-eliminating machinery was still present. In this and other cases the bronchitis seems to be the result, rather than the cause, of pneumoconiosis, though there may well occur direct injury to terminal bronchioles, with resultant localized collapse, in cases of exposure to very heavy dust clouds, as in the case of coal trimmers in the docks.

*The tracheo-bronchial lymphatic glands.* In these glands it is usual to find clear evidence as to whether a given case is truly silicotic or merely anthracotic. Silico-anthracotic cases occupy an intermediate position and may be difficult to classify from an examination of the glands alone.

In silicotic cases the lymphoid tissue of the gland soon disappears in those areas where dust has entered, whorled nodules gradually taking its place. In cases of pure anthracosis, the lymphoid tissue is preserved unchanged in spite of the proximity of the dust, so that the lymph spaces become beautifully blazed by arborescent collections of dust and dust cells lying in the midst of the normal glandular structure. These appearances are contrasted in Plates X and XI.

It is worth noting that, in asbestosis, all are agreed that it is very rare for the tracheo-bronchial glands to be affected. The long asbestos fibres may, indeed, be phagocyted but, owing to their size and shape, cannot be carried far along the lymphatic channels. It may be suggested, perhaps, that herein lies an explanation of the clinical and pathological differences between asbestosis and silicosis. Asbestos particles stay where they are first deposited in the lungs and there set up their effects. Silica particles are carried to the lymph glands and to the pleura before the lung drainage has become too hampered to allow of this diffusion of dust-carrying phagocytes. It is rather later that the silica becomes "held up" in the primary lobules and sets up its characteristic effects around the alveolar ducts and respiratory bronchioles.

#### DISCUSSION

In order to enable the reader to compare the findings in a series of twenty-two specimens, representing all three main types of pneumoconiosis, I have tabulated my records according to the appearances referred to above, adding, when the information was available, the results of chemical analyses of the lungs for total silica in the dried lung and in the ash.

Table I will serve to emphasize the contrast, in regard to nodular fibrosis and silica content, between the classical silicosis of hard-ground workers (Type 1) and the anthracosis of coal trimmers (Type 3); and it will be seen that

Table I. Analysis of 22 cases of pneumoconiosis according to classified types

	Nodular fibrosis	Diffuse fibrosis	Preservation of bronchial ciliated epithelium	Fragmentation of elastic tissue	Presence of tubercle bacilli or lesions	Crossed prisms bright particles	Accumulation of coal dust	Silica % in dried lung	Silica % of ash
<b>Type 1 cases: Silicosis</b>									
No. 1	++	++	-	++	-	+++	++		
No. 4	++	++	-	++	-	+++	++		
No. 7	++	++	++	++	-	+++	++		
No. 12	++	+++	++	±	++	+++	+		
No. 13	++	+++	++	++	-	+++	+		
No. 96	+++	++	-	+++	-	+++	++	3.79	42.0
No. 159	+++	++	±	+++	±	+++	+	2.45	30.6
<b>Type 2 cases: Silico-anthracosis</b>									
No. 2	+	++	-	++	-	+++	++		
No. 3	±	+	++	?	±	+++	++		
No. 5	++	+	-	++	±	+++	++		
No. 6	±	++	-	+++	-	+++	++		
No. 8	+	+++	-	+++	-	+++	+		
No. 9	±	++	++	+	-	+++	+		
No. 11	±	++	++	++	+	+++	+		
No. 45	±	+++	+++	+	-	+++	+++	1.38	13.68
No. 47	++	+++	+++	++	-	+++	+++	1.18	20.2
No. 89	+	+++	+++	++	-	+++	+++	1.27	12.2
<b>Type 3 cases: Anthracosis in coal trimmers</b>									
No. 138	±	++	++	++	-	?	++	0.438	3.8
No. 139	-	±	++	++	-	-	++	0.05	0.09
No. 120	-	±	++	++	-	-	++	0.43	5.0
No. 142	±	++	++	++	-	-	++	0.52	9.8
No. 168	-	+	+++	±	-	-	++	0.18	2.7

the silico-anthraccotic cases fall into an intermediate position in regard to these criteria. It is interesting to note, however, that, in respect to fragmentation of elastic tissue associated, perhaps, with pulmonary emphysema, all three types are equally affected. It would appear that the pure anthracotics come off better than either of the other types in regard to retention of the ciliated bronchial epithelium, which rather suggests that the presence of stone dust may be harmful to the bronchial tubes; but it should be added that the lungs of Type 3 cases came, for the most part, from the Cardiff Royal Infirmary and were not distorted by package for the post, while several were distended with 10 per cent formalin in saline. It is interesting, too, to find that tuberculosis was, in this small series, more frequent in the silico-anthraccotics than in the pure silicotics.

#### CONCLUSIONS

1. An attempt has been made to classify the pneumoconioses met with in South Wales.
2. The amount of fibrosis and lung damage in the pneumoconioses of this coalfield varies directly with the intensity and duration of exposure to silicious dust as expressed in the silica content on analysis of dried lung, and the number of "bright particles" seen with crossed prisms.
3. All three main types of pneumoconiosis show some degree of lung injury attributable to dust accumulation, though the amount of fibrosis is minimal in pure anthracosis.
4. Pneumoconiotic lungs of all types show appearances suggesting damage to the elastic structure of the lung, a finding which may, perhaps, have a bearing on the emphysema and dyspnoea met with. The observations here recorded appear to justify the conclusion that the emphysema associated with pneumoconiosis in South Wales is not necessarily only a compensatory effect of lung fibrosis, since it may be well marked in persons suffering from pure anthracosis with only minimal fibrosis. The precise etiology of this type of emphysema invites further investigation.
5. Bronchitis is common in silicotic and silico-anthraccotic cases, but seems to be the result rather than the cause of the pneumoconiotic state which renders the lungs specially liable to catarrhal infections. Ciliated epithelium is usually retained to the end, even in the worst cases of pneumoconiosis.

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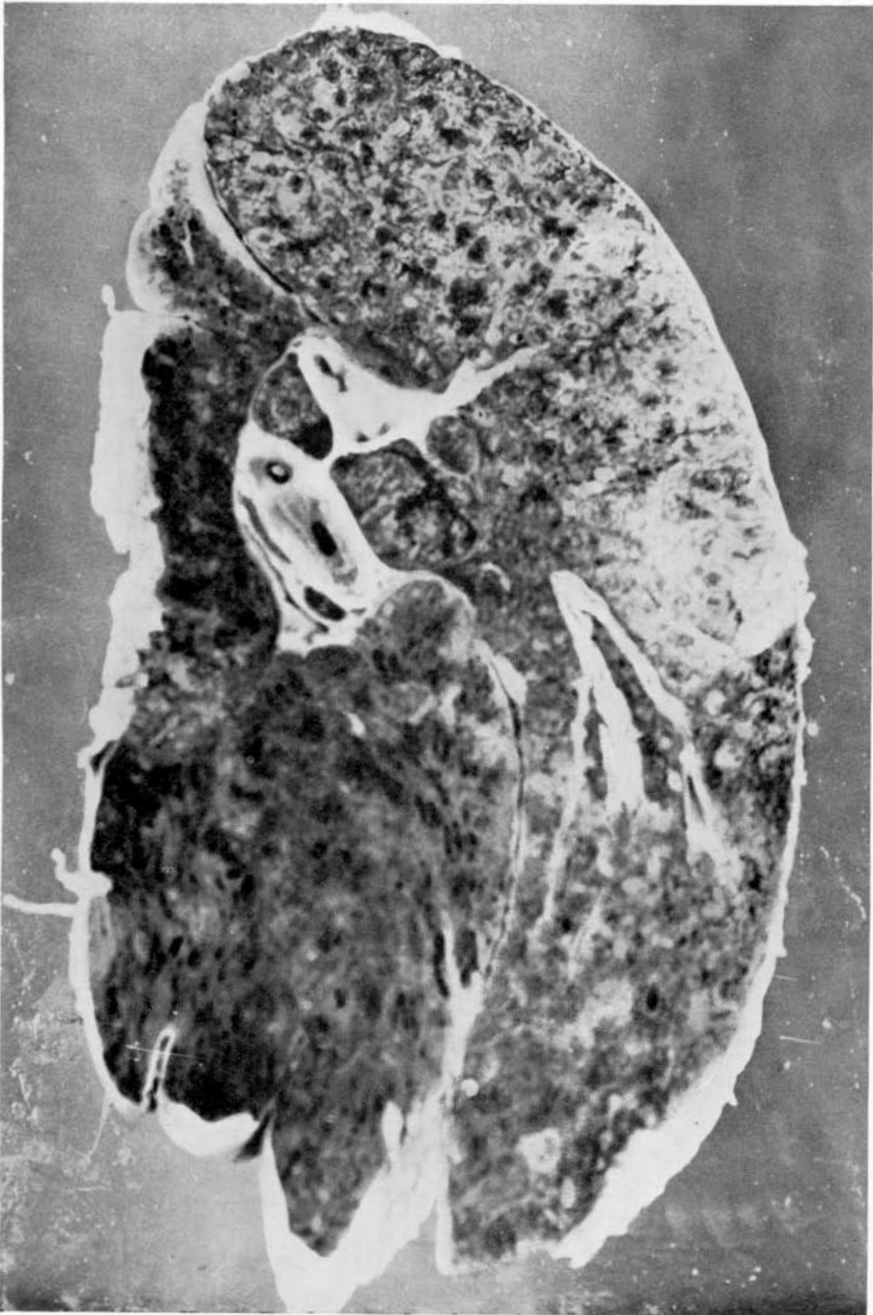
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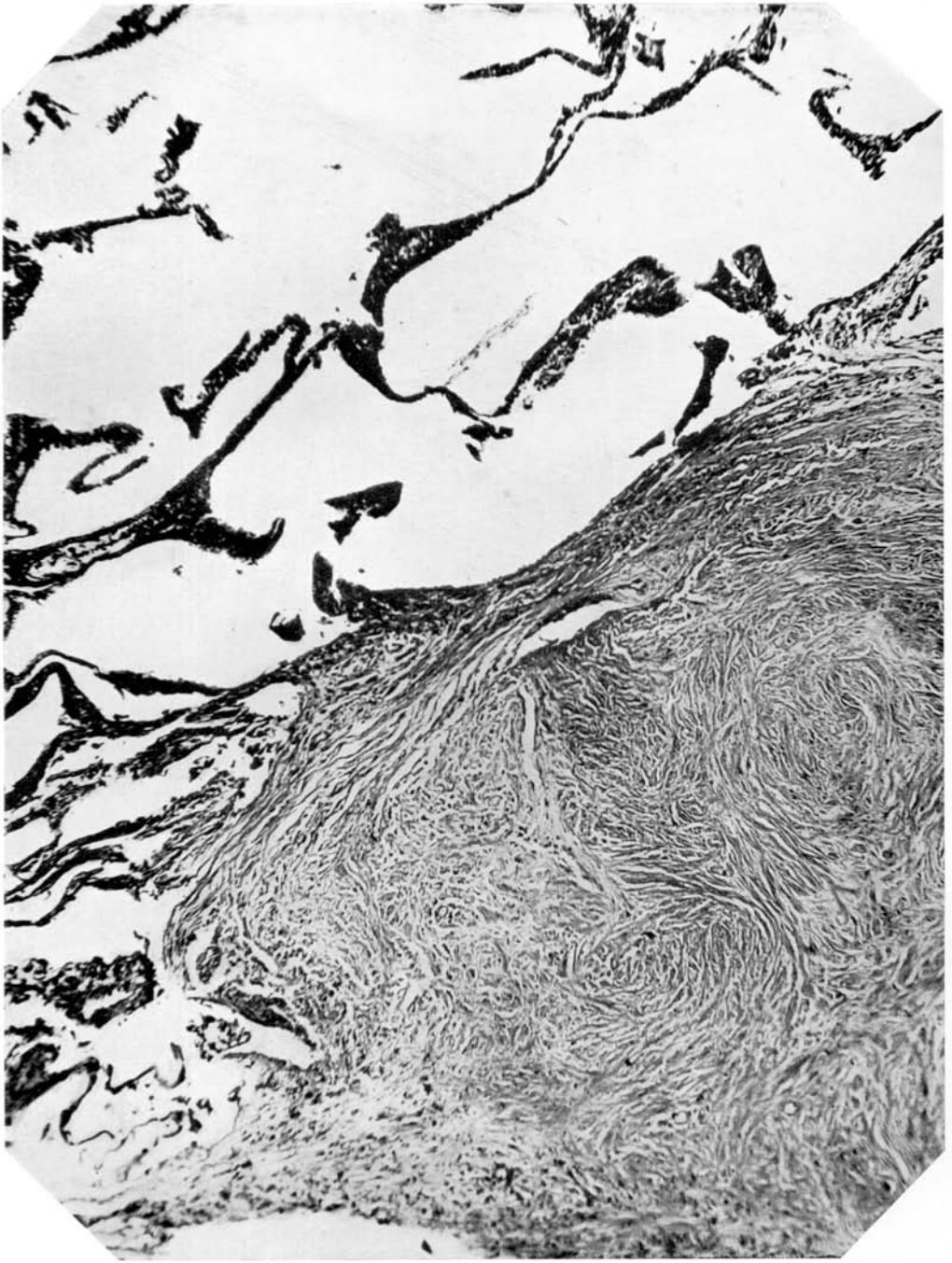
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*Example of Type I. Silicosis. Lung of case No. 159, showing extensive and confluent nodular fibrosis of upper lobe and cortex, together with "honeycomb" emphysema of lower and central parts.*



*Histology of Type I. Silicosis.* Confluent nodular fibrosis from case No. 159, showing alveolar emphysema adjoining the nodules.

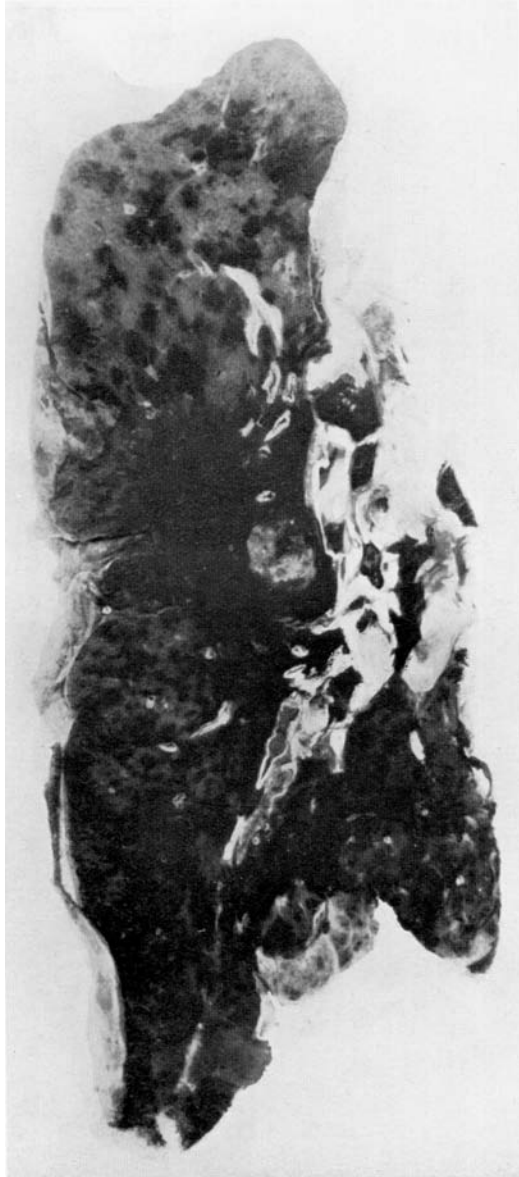


*Example of Type 2. Silico-Anthracosis.* There is excessive accumulation of coal dust with some nodular and much diffuse fibrosis. This case exhibited a terminal tuberculosis.





*Histology of Type 2. Silico-Anthracosis.* A dense accumulation of coal dust is seen around an area of perivascular fibrosis.



*Type 4. Localized dust retention.* The lung of a collier in which coal dust has accumulated around an old inactive cavity, probably tuberculous in origin.

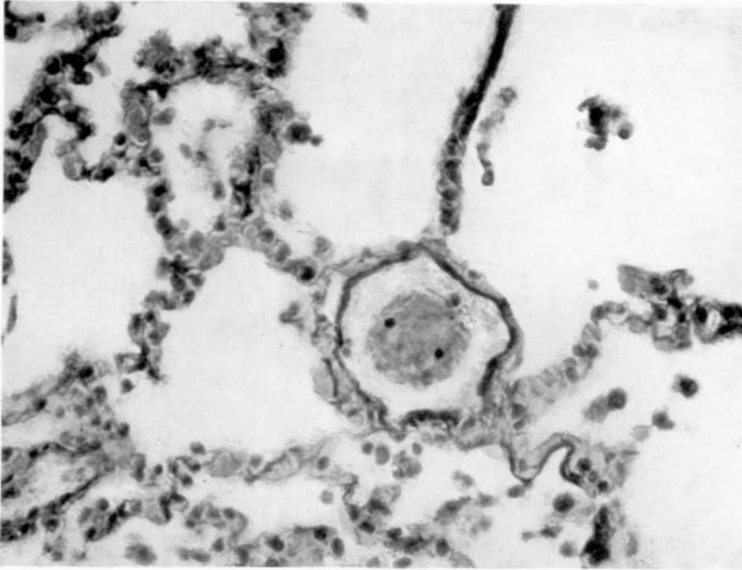


Fig. A. Illustrates the passing of elastic fibres from the wall of a small blood vessel to the alveolar septa in normal lung. Verhoeff's stain.  $\times 750$ .

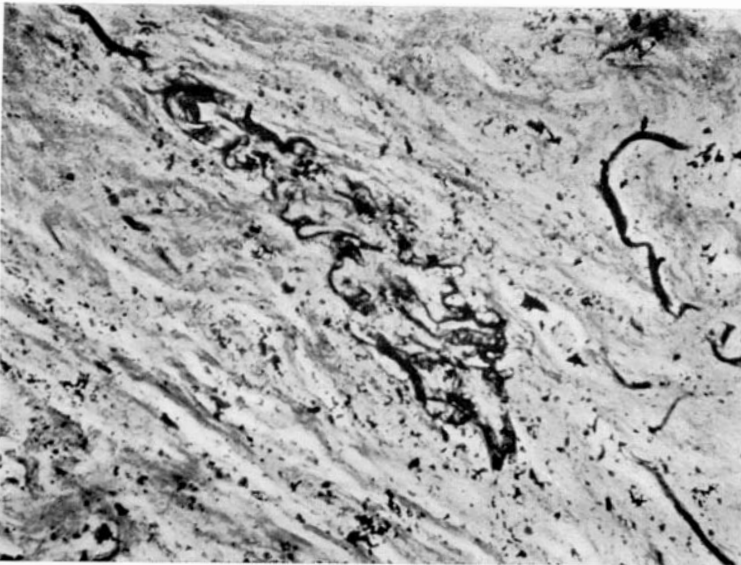
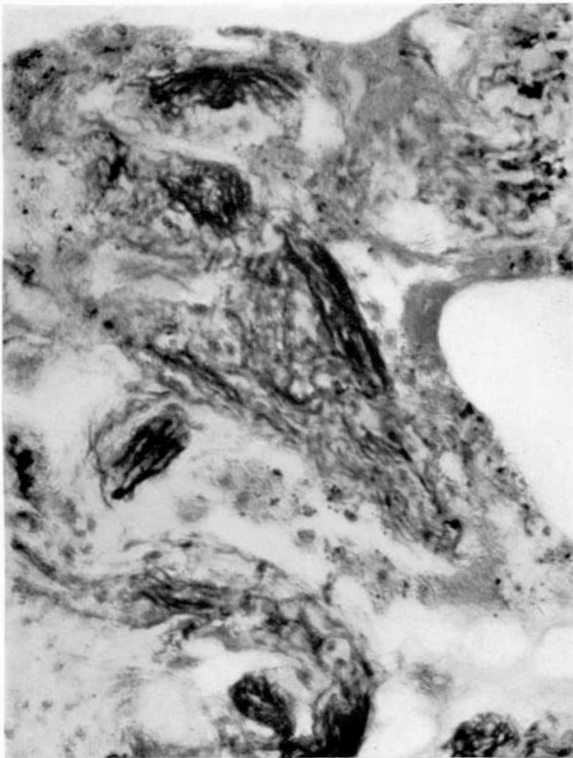
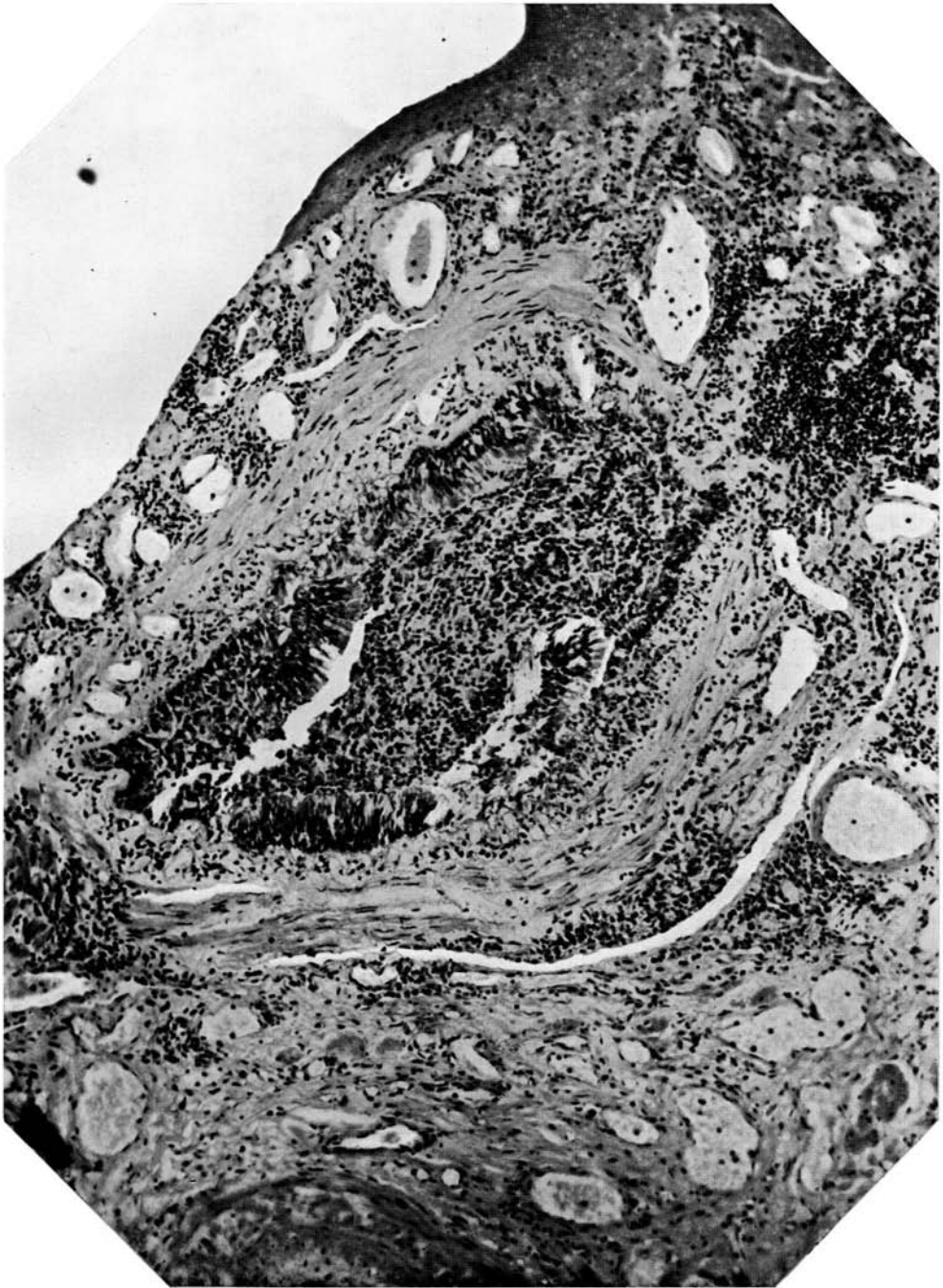


Fig. B. Skein of elastic tissue, the remains of a vessel wall, and some isolated fragments in the diffuse fibrosis of a silico-anthraxotic lung. Verhoeff's stain.  $\times 750$ .



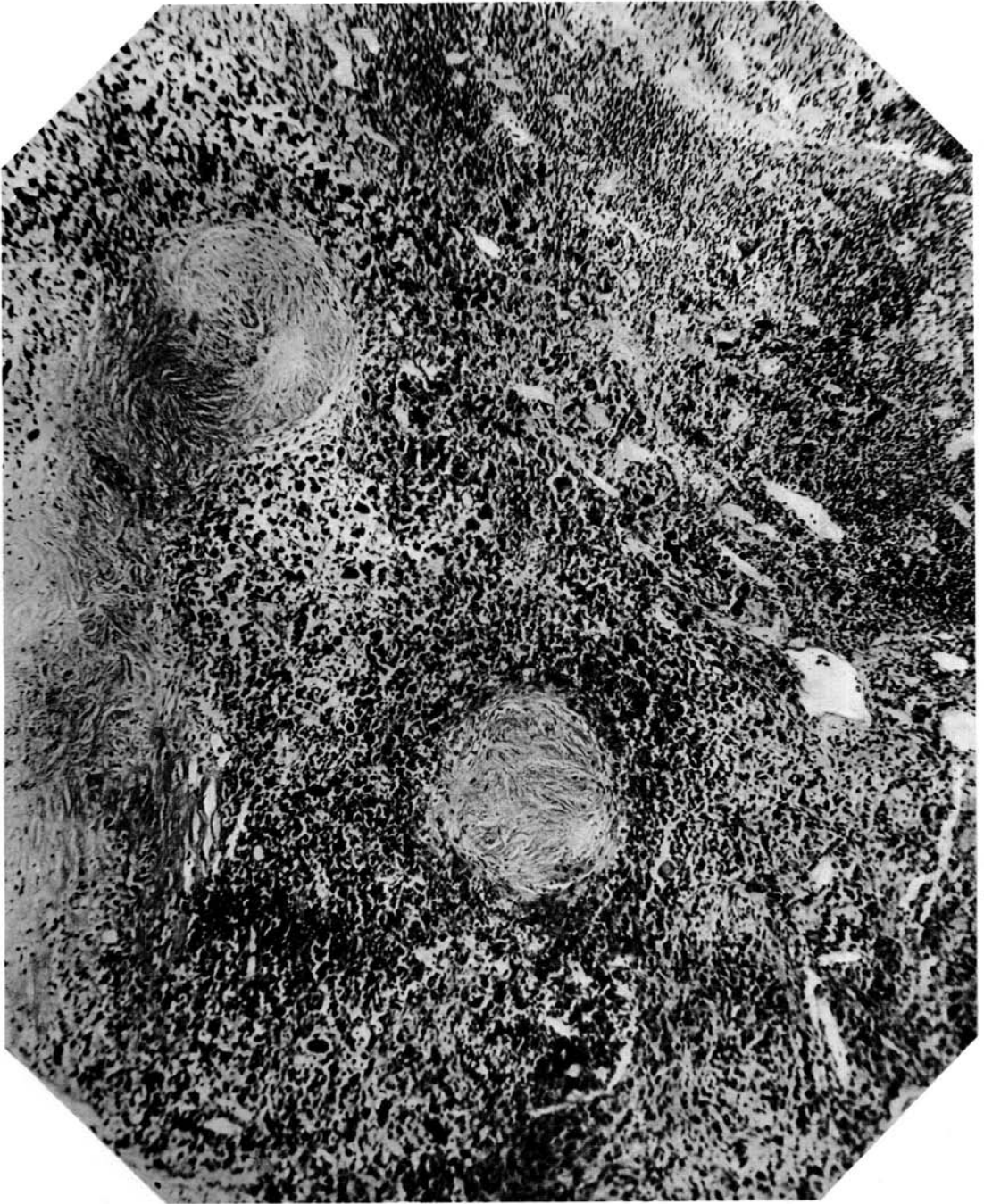


Skeins of elastic tissue in the collapsed part of a pneumoconiotic lung, apparently residual tissue from a disintegrated bronchiole. Verhoeff's stain.  $\times 750$ .



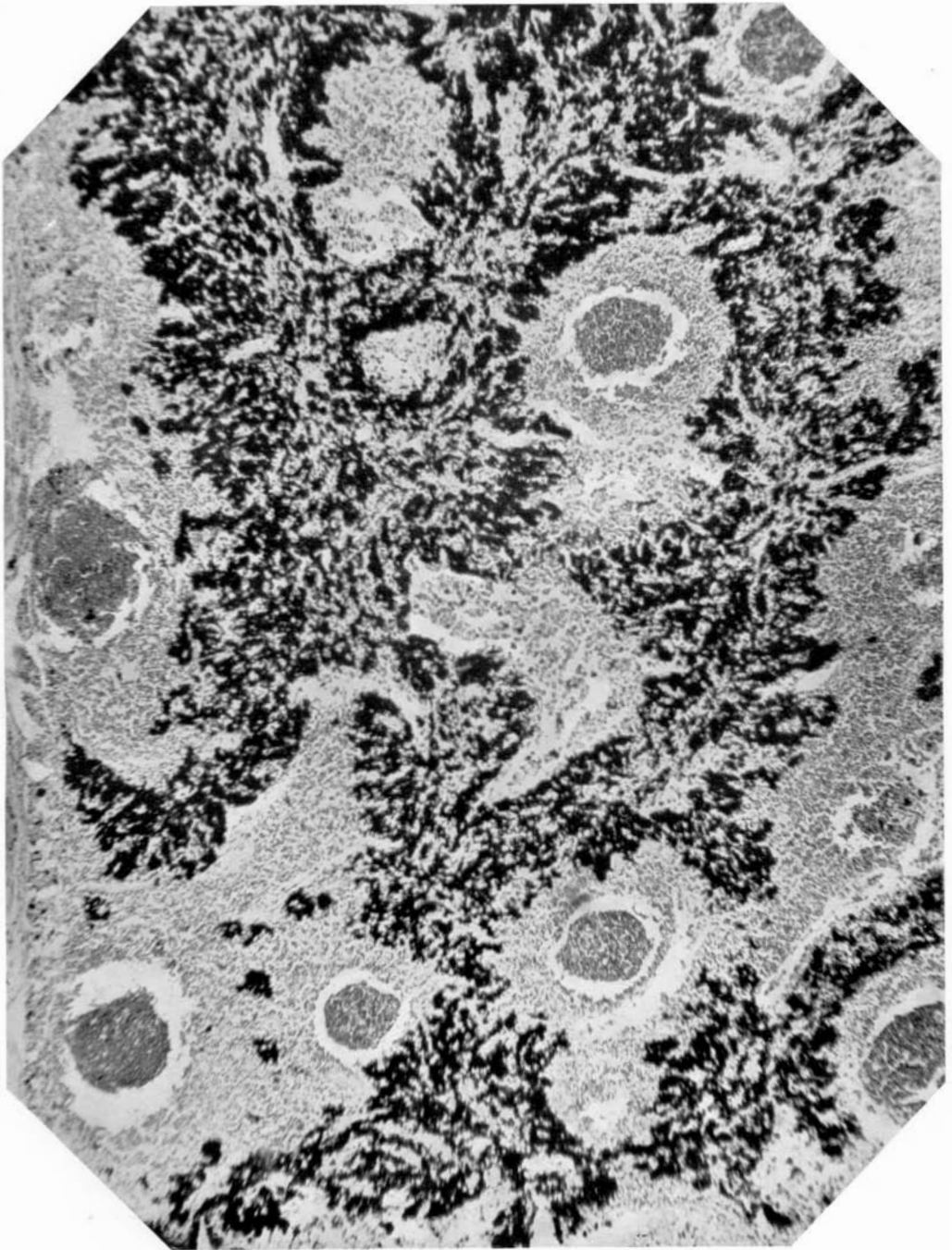
Septic bronchitis in the lung of a silico-anthracotic coal miner. The bronchiolar epithelium is well preserved but the lumen is filled with purulent material.





Tracheo-bronchial gland from a silicotic hard-ground worker. The normal glandular tissue is replaced by fibrous whorls.





Anthracotic gland from a coal trimmer. The normal glandular tissue is preserved in spite of the presence of numerous replete dust cells in the lymph spaces.