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(1936)

RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

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VOLUME 27
~~June~~ June, 1936, to December, 1936

WAGONER DEP
P. 51 L. 3
To
P. 52 L. 10

OWNED AND PUBLISHED BY
THE RADIOLOGICAL SOCIETY OF NORTH AMERICA
AS ITS OFFICIAL JOURNAL

ASBESTOSIS: A ROENTGENOLOGIC REVIEW OF 71 CASES

By J. RUSH SHULL, M.D., *Charlotte, N. C.*

ASBESTOS has been used in industry for centuries. It would seem that the pathologic action of this dust in the lungs would have been recognized before 1900, when Murray, in England, performed an autopsy on such a case. This autopsy was performed on a man, the last of 11 men who had begun work together in an asbestos plant in 1890. Murray did not report his findings until 1906. Probably the first case in English medical literature to be definitely proved as asbestosis, was reported by Cooke in 1924. This patient had tuberculosis as a complication.

Little attention has been paid to this form of pneumonoconiosis in the American literature until 1928, when the "Journal of the American Medical Association" commented editorially on Cooke's report and suggested that this condition deserved more consideration than it has been given. The first case reported in America was by Mills, in 1930. Since then many individual case reports, or small groups of cases, have been brought to our attention; but no large series from which a comprehensive study can be made has appeared.

Asbestosis may be defined as a disease of the lungs caused by the inhalation of asbestos dust and fiber. It is classified as a pneumonoconiosis and is characterized roentgenologically by an early interstitial fibrosis with progression into a terminal diffuse fibrosis. This fibrosis begins primarily in the bases of the lungs, involving the peribronchial structures. The parenchyma of the lungs is comparatively uninvolved. As the disease progresses there develops a filmy, hazy appearance of the lung-fields which has been aptly described as a "ground-glass appearance." The right side of the heart is frequently enlarged in

TABLE I.—PULMONARY ASBESTOSIS

Percentage of Cases Having	16 Slightly Advanced	35 Moderately Advanced	20 Markedly Advanced
Right-sided cardiac hypertrophy	37.5	62.8	95.0
Pericardial and pleural thickening	0	28.6	45.0
High left diaphragm	12.5	48.6	80.0
Emphysematous type of chest	6.3	65.7	95.0

CHART I.—PULMONARY ASBESTOSIS: SLIGHTLY ADVANCED CASES

	Name	Age	Color	Sex	Years' Exposure	Tuberculosis Present	Cardiac Hypertrophy Right-sided	Pericardial and Pleural Thickening	High Left Diaphragm	Trachea Displaced	Emphysematous Type of Chest	Comments
1	W. I. W.	28	W	M	4	–	Slight	–	+	–	–	No. 12, R. L. D., died of pneumonia 15 months after examination. Ill only three days.
2	C. W.*	34	W	M	2½	–	–	–	–	–	–	
3	S. E. T.	25	W	M	2½	–	–	–	–	–	–	
4	J. S.*	24	W	M		+	–	–	–	–	–	
5	B. B. R.	28	W	M	4	+**	–	–	–	–	–	
6	K. T.*	26	W	M	2							
7	L. B. H.*	26	W	M	9	+	–	–	–	–	–	
8	J. H.*	24	W	M	1½	–	Moderate	–	+	–	Yes	
9	J. G.	44	W	M	10	–	Moderate	–	–	–	–	
10	J. L. E.*	49	W	M	8	–	–	–	–	–	–	
11	W. F.	38	W	F	13	–	–	–	–	–	–	
12	R. L. D.	46	W	M	12	–	Slight	–	–	–	–	
13	T. J. C.*	36	W	M	8	–	Slight	–	–	–	–	
14	L. E. B.	34	W	M	2½	–	–	–	–	–	–	
15	L. M.	23	W	F	4	–	–	–	–	–	–	
16	C. B. J.	30	W	M	5	–	Slight	–	–	–	–	

* Re-examined 15 months later.
** Healed miliary.

the moderately advanced cases, and it is a common finding in the markedly advanced cases. The fibrosis is a result of chemical irritation caused by silica in the dust and of mechanical irritation instigated by the asbestos fibers.

Another finding characteristic of this disease is the presence of peculiar golden colored, crustation-like bodies in the lungs of these patients. Stewart and Stewart and Haddow have suggested the name "asbestos bodies" for them. Gloyne described a technic whereby the central core of these bodies was found to be a minute asbestos fiber. He also pointed out that it is important to demonstrate tubercle bacilli before one can assume that tuberculosis is a complicating factor.

Clinically, the most striking symptom is undoubtedly dyspnea. This is progressive, slow, and insidious in development and is due to inelasticity of the lungs and interference with blood supply. Cough and expectoration, especially the latter, may be nearly or quite absent except during bronchitic attacks. Anorexia, cyanosis, loss of weight, and emaciation are rather late manifestations, and are usually out of proportion to the physical signs, differing thereby from tuberculosis alone.

During the latter part of 1934 it was my privilege to examine the chests of 71 of 100 workers who had been dismissed from local asbestos plants. All had undergone physical examination before they were referred for roentgenologic study and were found to be physically disabled.

Stereoroentgenograms were made of each

CHART II.—PULMONARY ASBESTOSIS: MODERATELY ADVANCED CASES

	Name	Age	Color	Sex	Years' Exposure	Tuberculosis Present	Cardiac Hypertrophy Right-sided	Pericardial and Pleural Thickening	High Left Diaphragm	Trachea Displaced	Emphysematous Type of Chest	Comments
1	R. A. W.*	25	W	M	5	+	–	–	–	–	Yes	
2	L. E. W.	25	W	M	5	++	–	–	–	R	Yes	No. 2, L. E. W., died one year after examination. Tuberculosis.
3	J. C. S.	36	W	M	8	+	Slight	–	+	–	–	
4	W. L. B.	30	W	M	15	+	Moderate	–	+	–	Yes	
5	C. A. S.*	33	W	M	8	+	Slight	Yes	+	–	Yes	
6	R. H. S.	34	W	M	5	+	–	Yes	+	–	Yes	
7	T. R.	40	C	M	5	+	–	–	+	–	Yes	
8	C. D.	31	W	M	9	+	Moderate	–	+	–	Yes	
9	J. M.	39	W	M	12	+	Moderate	Yes	+	–	–	
10	L. M.	41	W	F	4	+	Moderate	–	+	–	Yes	
11	C. M.	42	W	F	11	+	–	–	+	–	Yes	
12	O. B. L.	45	W	M	7	+	Slight	Yes	+	–	Yes	
13	R. K.*	39	W	M	10	+	–	–	–	–	–	
14	E. C. L.	23	W	M	3	+	Slight	–	–	–	–	
15	S. J.	31	C	M	7	+	Moderate	–	–	–	Yes	
16	M. L.*	37	W	M	9	+	–	Yes	–	–	–	
17	R. G. J.	38	W	M	12	++	Slight	Yes	+	L	–	No. 17, R. G. J., is in terminal stages of tuberculosis. Death imminent.
18	W. H.	24	W	M	4	+	Slight	–	+	–	–	
19	H. H.	20	W	M	1½	+	Moderate	–	+	–	Yes	
20	U. L. J.	38	W	M	10	+	–	–	+	–	Yes	
21	O. G.*	39	W	M	7	+	Moderate	–	+	–	Yes	
22	R. G.	27	C	M	7½	+	–	–	–	–	Yes	
23	P. E.	38	W	F	11	+	Slight	–	–	–	–	
24	L. E.	33	W	F	12	+	Moderate	Yes	–	R	–	
25	W. W. E.	40	W	M	10	+	Slight	–	–	–	–	
26	A. W.	46	C	M	12	+	Advanced	Yes	+	R	Yes	
27	S. D.	29	C	M	8	+	–	–	+	–	Yes	
28	J. D.	30	W	M	8	+	Moderate	–	–	–	Yes	
29	S. C. C.*	30	W	M	9	+	–	Yes	+	–	–	
30	W. J. C.	42	W	M	8	+	Advanced	–	–	–	Yes	
31	V. O. B.	36	W	M	8	+	Slight	Yes	–	–	Yes	
32	A. R. B.	34	W	M	12	+	Slight	–	+	–	–	
33	G. L. B.	37	W	F	15	+	Moderate	–	+	–	Yes	
34	E. A. C.	28	W	M	9	+	–	–	+	–	Yes	
35	C. G.*	31	W	M	8	+	–	–	+	–	Yes	

* Reexamined 15 months later

ent, using a skin-target distance of 72 es. The same technic was meticu- ly observed in each case, so that films ood contrast and equal exposure could obtained for comparison. A number e also examined roentgenoscopically. Fifty-six were white males, six were white females, and nine were negro males. The average age was 34.4 years and the time of exposure varied from 16 months to 21 years.

In eight recognized cases of tuberculosis

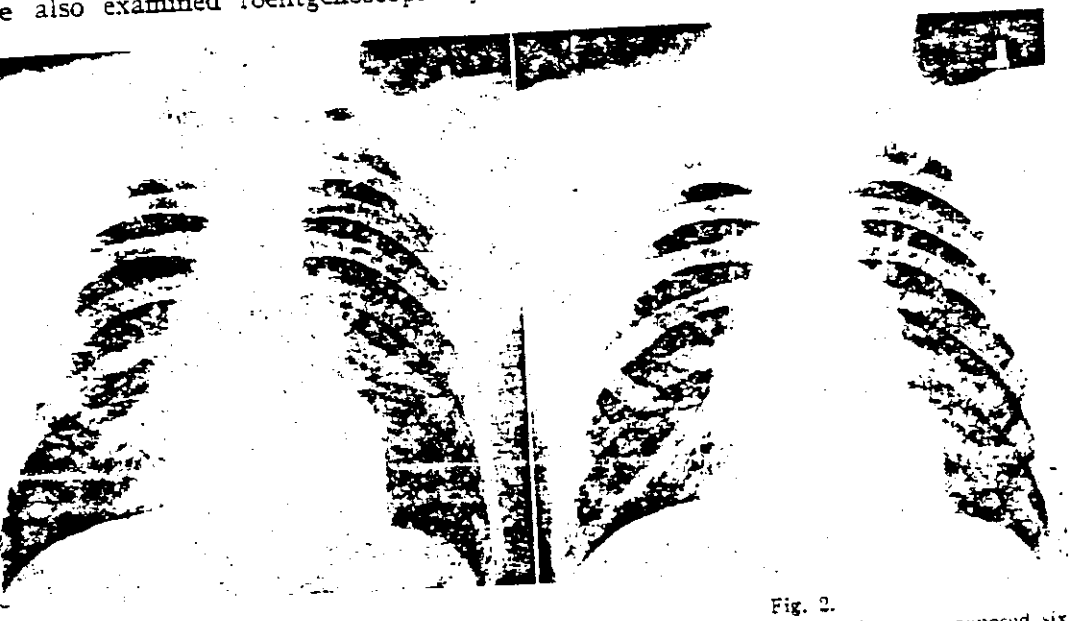

Fig. 1.

Fig. 2.

Fig. 1. Case 3756. Examined on Dec. 3, 1934; white male, aged 49 years. Patient was exposed six years: no complaints. Early type of asbestosis.
Fig. 2. Re-examination made on Feb. 15, 1936; there is slight improvement.

CHART III.—PULMONARY ASBESTOSIS: MARKEDLY ADVANCED CASES

	Name	Age	Color	Sex	Years' Exposure	Tuberculosis Present	Cardiac Hypertrophy Right-sided	Pericardial and Pleural Thickening	High Left Diaphragm	Trachea Displaced	Emphysematous Type of Chest	Comments
1	P. S.	36	W	M	10	—	Moderate	—	+	—	Yes	No. 3, S. G., died three months after examination, of an acute fulminating tuberculosis. Autopsy reported in text.
2	C. H. S.*	33	W	M	1½	—	Slight	+	—	—	Yes	
3	S. G.	30	C	M	8	—	Moderate	+	+	—	Yes	
4	R. W.	40	W	M	8	—	Slight	+	+	—	Yes	
5	P. J. V.	55	W	M	20	—	Advanced	+	+	R	Yes	No. 5, P. J. V., died one month after examination. Autopsy reported.
6	G. W. C.	38	W	M	13	—	Slight	+	+	—	Yes	
7	G. H.*	36	W	M	9	—	Moderate	+	+	—	Yes	
8	S. P.	36	W	M	6	—	Moderate	+	+	—	Yes	
9	E. A.	26	C	M	8	—	Moderate	—	+	—	Yes	
10	C. G.*	34	C	M	9	—	Moderate	—	+	—	Yes	No. 6, G. W. C., is in sanatorium with tuberculosis.
11	F. B.	31	W	M	6	—	Moderate	—	+	—	Yes	
12	L. B. J.*	35	W	M	8	—	Slight	+	+	R	Yes	
13	C. Mc. N.	47	W	M	10	+	Moderate	—	+	—	Yes	
14	T. F. E.	50	W	M	6	—	Moderate	—	+	—	Yes	
15	A. J. H.*	37	W	M	10	—	Slight	+	+	—	Yes	No. 16, W. E. G., had pneumonia four months after examination. Recovery.
16	W. E. G.*	38	W	M	8	—	Advanced	+	+	—	Yes	
17	R. H.*	41	C	M	12	—	—	—	+	—	Yes	
18	G. M. C.	32	W	M	6	—	Slight	+	—	—	Yes	
19	F. D. B.	31	W	M	7	—	Moderate	—	—	—	—	
20	G. C.	36	W	M	9	—						

* Re-examined 15 months later.

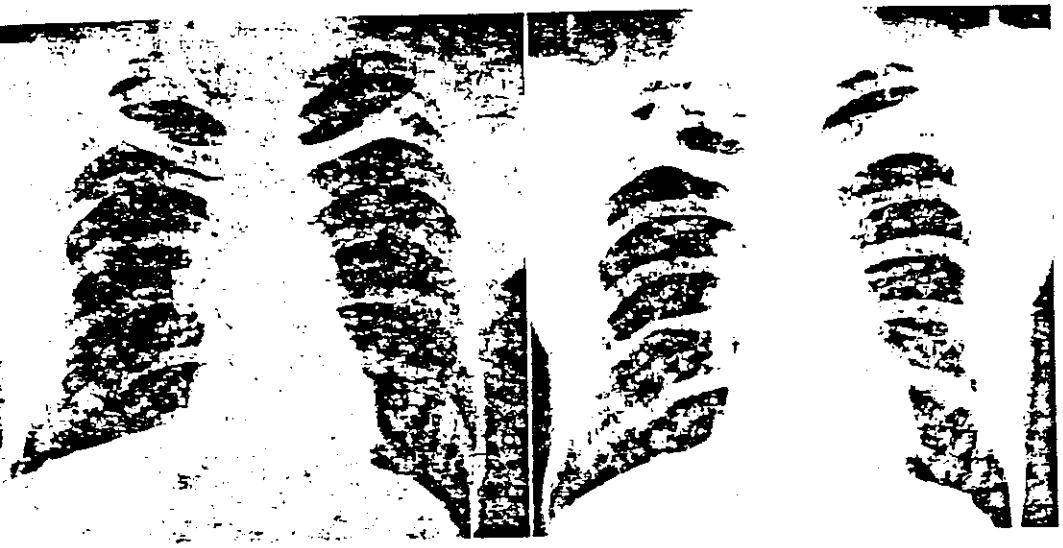

Fig. 7. Fig. 8.

Fig. 7. Case 3839. Examined on Jan. 7, 1935; white male, aged 37 years. Patient was exposed nine years. Slightly advanced type of asbestosis. Slight cough and some dyspnea, but no time loss from work. Note that in addition to the findings of asbestosis there is an active tuberculous lesion in the upper right lobe.
Fig. 8. Re-examination made on Feb. 15, 1936. Note improvement in lung-fields. No complaints. Decided gain in weight. Has worked steadily in a textile plant since discharge from asbestos mill in November, 1934.

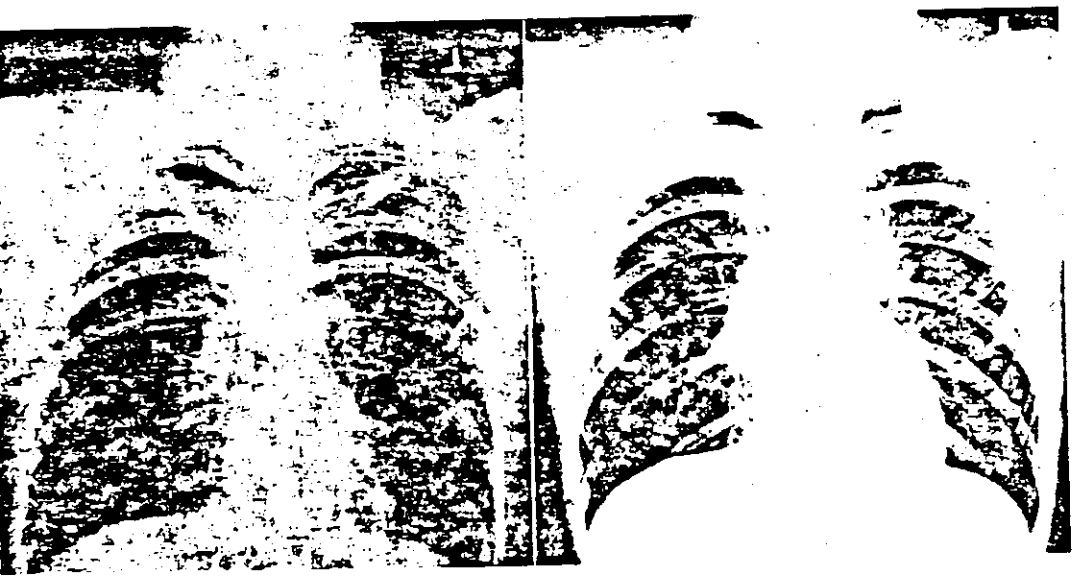

Fig. 9. Fig. 10.

Fig. 9. Case 3738. Examined on Dec. 1, 1934; white male, aged 28 years. Patient was exposed ten years. Slight cough for one year. Early type of asbestosis. Note co-existing tuberculosis.
Fig. 10. Re-examination made on Feb. 10, 1936. This patient has gained eight pounds in weight and has worked as a radio technician since his discharge from the asbestos mill in November, 1934. Note fibrosing of tuberculous lesion and increased aeration of lung-fields.

g them with a number of plates of other types of pneumonoconiosis, it has been possible to make certain observations which seem to be peculiar to this group of asbestosis cases as a whole. These are tabulated under Table I.

Furthermore, it was obvious that a classification could be effected. Since the

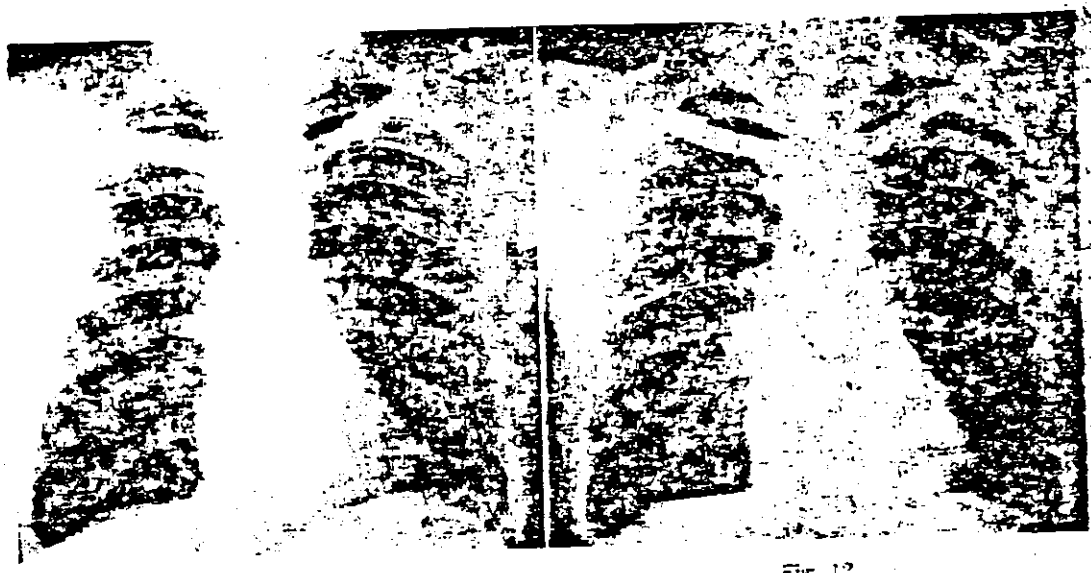

Fig. 11.

Fig. 12.

Fig. 12.—Examined in December, 1934. White male, aged 37 years. Patient was exposed eight years. Moderately advanced type of asbestosis. Note pericardial and pleural adhesions.

Fig. 11.—Preliminary examination made on February 25, 1930. No change in the appearance of the lung-fields.

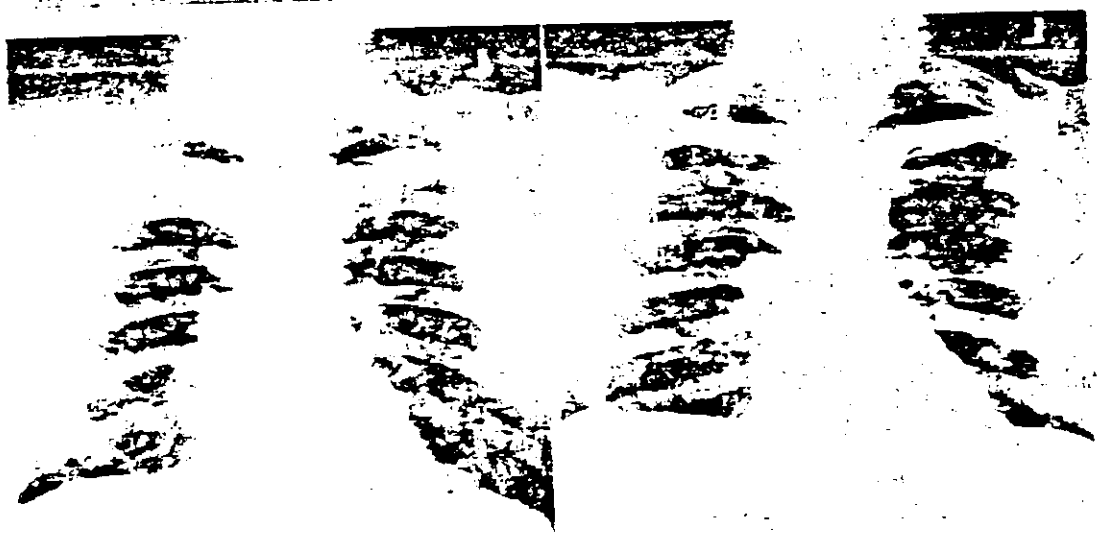

Fig. 14.

Fig. 14.—Examined in December, 1934. White male, aged 26 years. Patient was exposed two years. He had previously been on a farm. Past medical history was entirely negative. Moderately advanced type of asbestosis. Pericardial adhesions. Note miliary-like deposits suggestive of a healed tuberculosis.

Fig. 13.—Preliminary examination made in February, 1930. Patient now regularly employed in a textile plant. No change in the appearance of the lung-fields to suggest active tuberculosis.

...important feature of all these cases is unquestionably the diffuse fibrosis; this sign is taken as the "yard stick" in compiling our classification. After the groupings are arranged, the other roentgenologic findings are tabulated under each group.

The following seems to be a satisfactory and working classification:

(1) Slightly advanced cases.
(2) Moderately advanced cases.
(3) Markedly advanced cases.

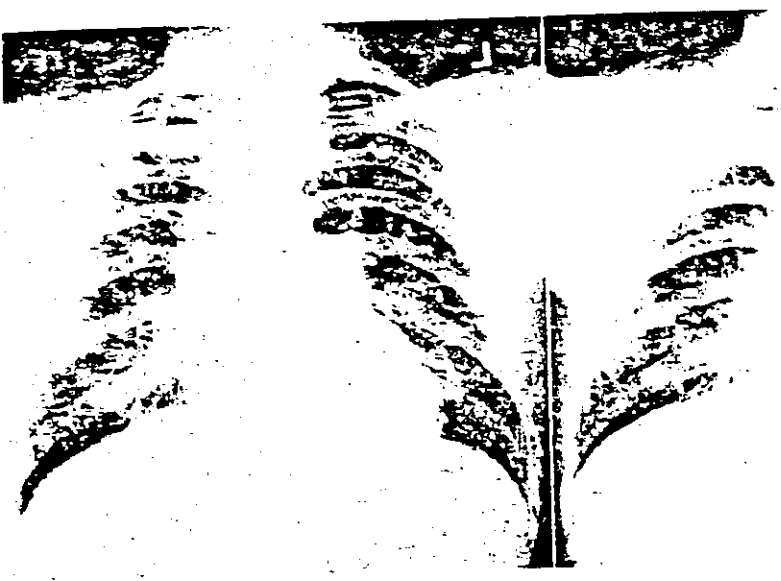

Fig. 15.

Fig. 16.

Fig. 15. Case 3708. Examined on Nov. 24, 1934; white male, aged 35 years. Patient was exposed seven years. Chief complaint was dyspnea. Moderately advanced type of asbestosis.
Fig. 16. Re-examination made on Feb. 17, 1936. There is a slight increase in aeration of the lung-fields.

Fig. 17.

Fig. 18.

Fig. 17. Case 3743. Examined on Dec. 1, 1934; white male, aged 38 years. Patient was exposed eight years. Cough and dyspnea were the chief complaints. Markedly advanced type of asbestosis.
Fig. 18. Re-examination made on March 15, 1935. Patient had an attack of lobar pneumonia in January, 1935. Note changes in lung-fields and in cardiac outline.

1. *Slightly Advanced Cases* (Chart 1).—In this group are 16 cases. In all, there is a beginning interstitial fibrosis in both lung bases producing a filmy, hazy appearance. The roentgen film cannot be considered diagnostic; a history of exposure is necessary to aid in arriving at a diagnosis. There are no cases of pericardial and pleural thickening, in none is the trachea displaced, rarely is there an emphysematous type of chest, and less than half showed right-sided cardial hypertrophy. In this group there

Fig. 19.

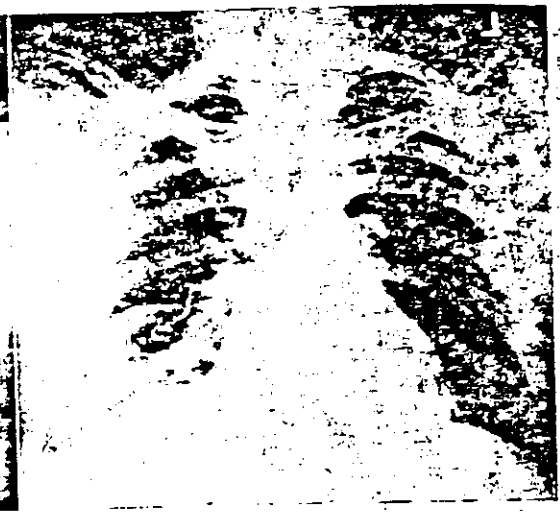

Fig. 20.

Fig. 19. Case 3751. Examined on Dec. 3, 1934; white male, aged 33 years. Patient was exposed one and one-half years. He had cough for two months with moderate dyspnea. Markedly advanced type of pulmonary asbestosis.

Fig. 20. Re-examination made on Feb. 10, 1936; no improvement. Note slight increase in cardiac base. (This patient's father had worked in the same plant for five years prior to dismissal in September, 1934. His lungs showed only an early or slightly advanced type.)

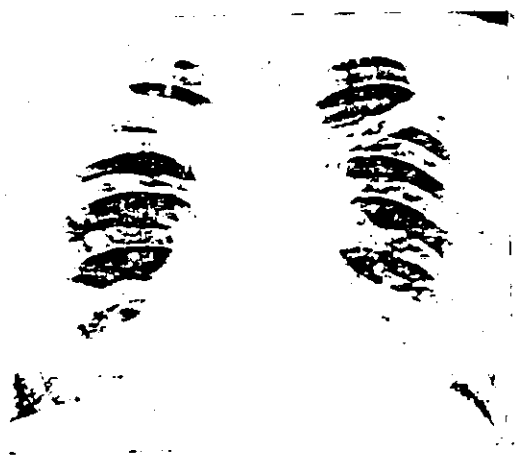

Fig. 21. Case 3775. Examined on Nov. 11, 1934; white male, aged 50 years. Patient was exposed seven years. Markedly advanced type of asbestosis. Dyspnea was marked. Note left diaphragm and characteristic basal involvement. This film is typical of markedly advanced asbestosis as evidenced by basal involvement, fuzzy cardiac outline, high left diaphragm, and the characteristic ground-glass appearance of the lung-fields.

are two females and 14 males, all white. The average age is 32.2 years and the average time of exposure is 5.4 years.

2. *Moderately Advanced Cases* (Chart 2).—There are 35 patients in this group. There is definite interstitial fibrosis radiating to the periphery and producing a ground-glass appearance to the lung-fields. Bronchiovascular markings are increased and pericardial and pleural thickening are noted. Right-sided cardiac enlargement is more frequent and emphysematous types of chests are common. The series contains five white females and 30 males, five of whom are colored. The average age is 37.1 years and the average time of exposure is 8 years.

3. *Markedly Advanced Cases* (Chart 3).—There are 20 patients in this group. In only one is there no roentgenologic evidence of right-sided cardiac hypertrophy and in only one other is evidence of an emphysematous type of chest lacking. Nearly half have pericardial and pleural thickening and the majority show a high left diaphragm (Table I). The average age in this group is 37.1 years and the average time of exposure 8.7 years. All are males, 16 white and four colored.

In the entire series only six showed evidence of slight displacement of the trachea,

Fig. 22.

Fig. 23.

ig. 22. Case 3700. Examined on Nov. 22, 1934; white male, aged 36 years. Patient was exposed ten rs. He had an unproductive cough with a negative sputum. Note the shaggy heart. Markedly ad-ced type of pulmonary asbestosis.
ig. 23. Re-examination made on Feb. 18, 1936. Chief complaint was dyspnea. Note spread of fibrosis.

of these three had x-ray evidence of rculosis. As would be expected, the ee of cardiac hypertrophy is largely de-ent on the length of exposure. Other ngs do not seem to depend on this fac-o the same extent. Just what influence degree of exposure, *i.e.*, the type and unt of dust inhaled, exerts on the roent-logic picture is difficult to say, as it not possible to determine this factor. would expect more serious and exten-pathologic changes in cases in which e has been a greater degree of exposure. loubtedly there are other factors which r. an important rôle in the amount of nage done to lung tissue: the patient's stitutional make-up, his intelligence, vious disease of the lungs, and other ses may exert an influence in the rapid-with which the disease progresses. I do believe tuberculosis plays a significant t in the development or prognosis of as-tosis. Roentgenologic evidence tends to hold this statement. Two of the pa-nts with definite evidence of tuberculosis wed unquestionable improvement on examination 15 months subsequently. th are employed in other industries.

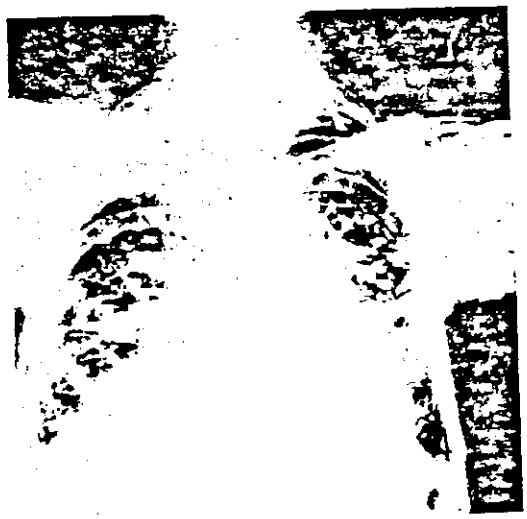

Fig. 24. See caption under Figures 25 and 26.

The series of re-examined patients is too small, and perhaps insufficient time has elapsed, to draw definite conclusions on the progress of the disease from a roentgenologic standpoint. When more patients have been re-examined over a longer period of time, such a report will appear. However, the observations made on the 21 re-examined patients are worthy of note.

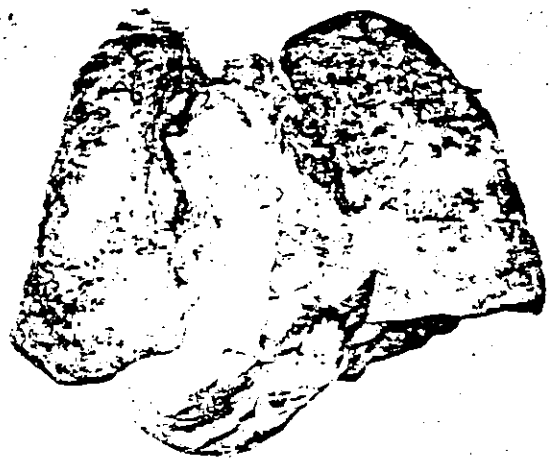

Fig. 25.

Fig. 26.

Figs. 24, 25, and 26. Case 3792. Examined Dec. 12, 1934; white male, aged 55 years. Patient was exposed 25 years. He had been totally disabled for the past two years. Marked dyspnea, troublesome cough, emaciation, and peculiar palor of the skin. Markedly advanced type of asbestosis. Note the shaggy heart and very little air space remaining. This patient died one month after this examination. Photographs of lungs and summary of pathologic findings after autopsy are found in the text.

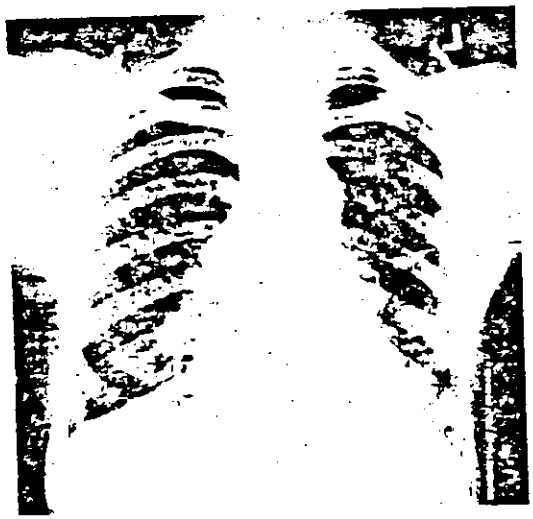

Fig. 27. Case 3746. Examined on Dec. 3, 1934; negro male, aged 30 years. Patient was exposed seven years. Chief complaint was cough of five years' duration and recent continued hemoptysis. There is some cardiac enlargement but no roentgenologic evidence of tuberculosis. He died three months later and at autopsy tuberculosis was found. (See report.)

In the Markedly Advanced Group there were five re-examinations: three gained some weight, one lost, and the other remained the same. One showed no change in the roentgen picture and in three there was an increase in the amount of fibrosis. There was no improvement in the heart shadow in two, and in two it had enlarged. Clinically four showed no improvement. The fifth (See Figs. 29 and 30) showed definite improvement.

In the Moderately Advanced Group, seven were re-examined. Three had gained a substantial amount of weight, two remained the same, and two had lost. Three showed improvement in the lung picture and two a definite spread of the disease. One showed an increase in the size of the heart and another showed definite improvement.

In the Slightly Advanced Group, nine were re-examined. Three had gained considerable weight and only one had lost. Five showed improvement in the lung picture and only one any increased fibrosis. In none was there any alteration in the size of the heart. Three of these cases had roentgen evidence of tuberculosis. Clinically these patients were much improved.

Generally speaking, it would seem that improvement can be expected in the early cases when they are removed from asbestos

plants. As the disease progresses, improvement is less likely, and when the condition becomes markedly advanced the patient usually becomes a permanent invalid and the prognosis must be considered entirely unfavorable. Lanza states: "It is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor does infection seem to be as closely and intimately associated with asbestosis as with silicosis."

AUTOPSY FINDINGS IN ASBESTOSIS

Autopsy was performed on two patients in the Markedly Advanced Group. One (S. G.) died of an acute fulminating tuberculosis process, with continued hemaphysis, three months after examination. There was no roentgenologic evidence of this condition. The other (P. J. V.) died one month after examination, of asbestosis. Pathologic examinations of the lungs were made by Dr. J. B. Bullitt, Professor of Pathology in the School of Medicine at the University of North Carolina.

Fig. 28. Case 3746. Roentgenogram of lungs following autopsy. Lung mapping was attempted but was unsatisfactory. Synopsis of autopsy findings reported in text.

Case 1 (S. G.). "*Gross:* The lungs are somewhat distorted by being molded in the container. The conditions in the two lungs are essentially the same. The pleura over practically the whole surface is rough and apparently had been adherent to the patietes; in some areas it is as much as two millimeters thick. The interlobular divisions are obliterated by adhesions except as below described. The cut surfaces show nearly uniform character from base to apex, though the degree of damage is greater in the central and base portions. Narrow grayish lines block the tissue into irregular small areas. A few small gray spots, resembling tubercles, are scattered here and there from base to apex. Near the central portion of each lung is an irregular shaped area of solidification, about two by three centimeters, which is apparently caseous pneumonia. Similar areas of smaller size are found in the apical and in the basal portions. In this area are small cavities, about three or four millimeters in diameter. Except in these areas, the tissue has an elastic feel, similar to but somewhat less than that of normal lung. I find nothing to justify the massive hemorrhage that he is said to have had shortly before death. The heart showed nothing of import. The hilal lymph nodes are enlarged and mottled with caseous areas.

"*Microscopical:* In all parts of both lungs there is considerable fibrosis. In great part, this consists of small irregular shaped nodules with lobular distribution, but also there is much fine fibrosis thickening the walls of alveoli that are still functional. Most of this fibrosis looks like old healed scars, but much of it shows some infiltration with mononuclear cells, suggestive of a slowly progressive process. In practically all these scars there is some pigment deposit—in some places a considerable amount, and also there are numbers of fine asbestos shreds. I am confident that much of this fibrosis is due to the asbestos, though part of it might be healed tuberculosis. In addition to this asbestos, the mi-

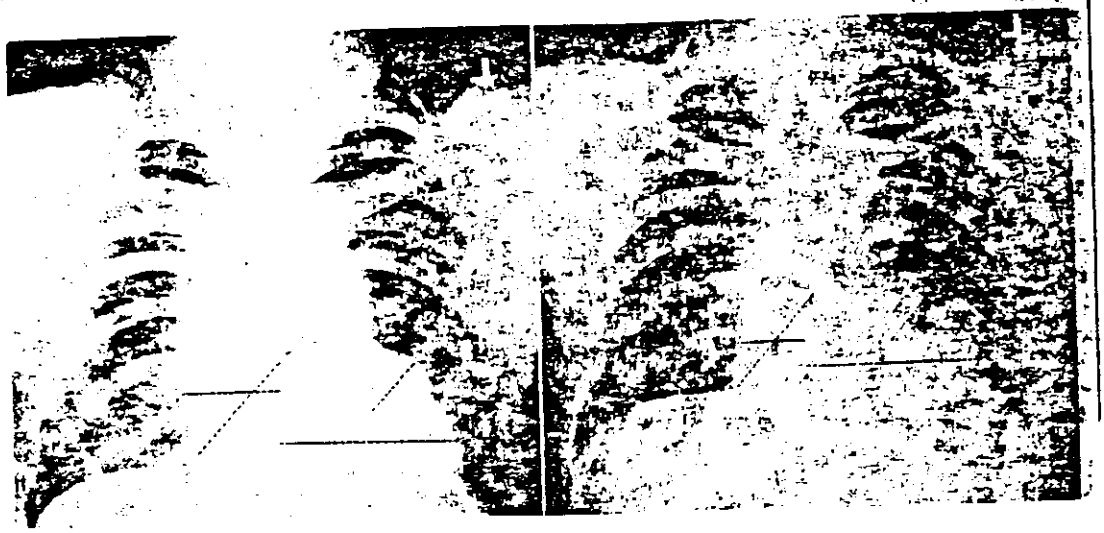

Fig. 29. Fig. 30.

Fig. 29. Case 3778. Examined on Nov. 4, 1934; negro male, aged 40 years. Patient was exposed 12 years. He had had cough and dyspnea for two years. Markedly advanced type of asbestosis with typical cardiac enlargement. Measurements: MR 5.5, ML 10.2, and Base 12.0.

Fig. 30. Re-examination made on Feb. 14, 1936. Note diminished cardiac outline and increased aeration of the lung-fields. Dyspnea now chief complaint, but not as marked as a year ago. Measurements: MR 4.5, ML 10.0, and Base 10.5.

This is the only case in the markedly advanced group that has shown any improvement clinically or roentgenologically.

croscope shows many very small typical tubercles in various parts of the lungs; moreover, the caseous areas mentioned above are characteristically tuberculous."

Case 2 (P. J. V.). "*Gross:* Both lungs are essentially alike. The interlobar clefts are obliterated by dense adhesions. The greater part of the pleural surfaces are roughened and appear to have been adherent to the parietes, though some areas are smooth and glistening and resemble the normal. From apex to base, on the cut surfaces, the tissue is blocked off into small polygonal areas by narrow white lines. In occasional places these lines broaden to as much as one to three millimeters; also at several places, just beneath the pleura, the pulmonary tissue is solidified into about five or six millimeters in thickness and twenty to thirty millimeters in breadth. Except for the fibrosis above described, the pulmonary tissue appears essentially normal to the naked eye, but when pinched between the fingers it has a distinctly increased density, especially in the lowe lobes.

"The hilal lymph nodes are much en larged and their cut surfaces are slightly mottled with gray.

"The heart is distinctly larger than th average normal. The right ventricle is di lated, probably sufficiently to prevent per fect competence of the valves. The muscl is pale and slightly streaked. It is appar ently not quite as firm in texture as normal The aorta and the heart valves show a mod erate amount of atheroma.

"*Microscopical:* The microscope demon strates an amount of fibrosis greatly in ex cess of what the naked eye would lead on to expect. This is more marked in th lower lobes but is present in all parts c both lungs. It consists of nodules an bands of connective tissue which hav clearly resulted in the obliteration of muc pulmonary tissue. Much of it consists c old, hard scars with no present inflamma tion, but in much of it the process seem

gressive, as indicated by a low grade date (mononuclear cells). The rubbery ques on the pleural surfaces are partly de up of thickened pleura, but mostly of ual lung tissue obliterated by fibrous ue. A good deal of pigment is present most areas. In all areas there are num- us spicules of asbestos. Some of the rs in this lung might well pass for healed ercles, but I find no present tubercles, d careful search fails to demonstrate any d-fast bacilli in any area. The lymph des at the hilum show hyperplasia, some le fibrosis, a moderate amount of pig- nt, and a few spicules (very small) of estos, but no evidence of tuberculosis.

Except for moderate atheroma, the ta and heart show nothing of impor- ce under the microscope."

ASBESTOSIS AND SILICOSIS

The roentgenographic picture of silicosis es not resemble asbestosis. In the for- r, the upper third of the lungs is in- lved and the fibrosis is parenchymal and t interstitial. The lung-fields show char- teristic nodulation, and, in advanced ses, a coalescence producing dense opaque eas in contrast with the typical, hazy, ound-glass appearance of the asbestosis ng-fields. Silicosis is definitely a pro- essive disease, even when the patient has en removed from dust exposure. This is ot the case in asbestosis, certainly in the ghtly and moderately advanced groups. asbestosis, there is a definite tendency to eural and pericardial involvement which strikingly absent in silicosis.

MEDICO-LEGAL STATUS OF ASBESTOSIS IN NORTH CAROLINA

Just a word about the status of asbesto- s in North Carolina from a medico-legal andpoint. In the case of McNeely *vs.* sbestos Co., 206 N. C., page 568, the tate Supreme Court held the plaintiff ffered an "injury by accident." The ourt said: "He alleged in his complaint nd offered evidence tending to show that his injury was produced and proximately caused by negligence of the defendant in that it maintains no dusting or suction system such as is approved and in general use in other asbestos plants. Consequently his allegation and proof both established the fact that his injury was caused by the negligence of the employer and hence was not the usual incident or result of the particular employment in which the workman is engaged. That is to say, the injury was not produced by the inherent nature of the work itself and classifiable as an occupational disease, but was produced by the active negligence of the employer and his failure to exercise reasonable care." The Court held in effect that the injury itself was an accident in that it was an unlooked for and untoward event which was not expected or designed by the plaintiff, and therefore was compensable under the Compensation Act. As a consequence of this decision, the State Legislature of 1935 amended the Compensation Act to include twenty-five occupational diseases, including asbestosis.

CONCLUSIONS

1. Asbestosis is a definite disease entity.
2. Inhalation of air laden with asbestos dust and fibers produces characteristic changes in the lung.
3. The time required for the development of the disease is variable. The earliest patient in my series had worked in an asbestos mill only 16 months.
4. The disease *asbestosis* differs from the disease *silicosis*, clinically, pathologically, and roentgenologically.
5. While the roentgenogram is the most reliable diagnostic aid, the interpretation and correlation with clinical signs and symptoms is often difficult. Without the history of exposure a certain number of slightly advanced cases will not be recognized.
6. A fair percentage of the slightly advanced and the moderately advanced cases do tend to improve and the attendant disa-

bilities to become lessened when removed from asbestos dust.

7. Asbestosis does not predispose to tuberculosis.

8. From my observations asbestosis is not primarily a progressive disease.

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RADIOGRAPHIC FINDINGS IN PULMONARY ASBESTOSIS

1. Emphysematous Type of Chest.
2. Flaring of Lower Ribs.
3. Trachea not Displaced.
4. Diffuse Fine to Coarse Fibrosis Reaching the Periphery, Interstitial in Character, Differentiating it from the Roentgenologic Appearance of Silicosis.
5. Hazy Ground-glass Appearance of Lung-fields.
6. Increased Density of All Pleural Markings.
7. Shaggy Appearance of the Cardiac Outline.
8. Tendency Toward Right-sided Cardiac Enlargement.
9. Disproportionate Rise of Left Diaphragm.
10. Degree of Cardiac Involvement is not Consistent with that of Pulmonary Involvement.