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FOURTEENTH ANNUAL MEETING — AMERICAN COLLEGE OF CHEST PHYSICIANS
Congress Hotel, Chicago, Illinois — June 17-20, 1948

Asbestosis:
VI. Analysis of Forty Necropsied Cases*

KENNETH M. LYNCH, M.D. and W. M. CANNON, M.D.**
Charleston, South Carolina

During the past eighteen years the necropsy studies at the Medical College of South Carolina have included forty cases in which asbestosis of some degree has been encountered. In this period several reports[1-5] have been published.

Since various previous writings give extensive presentations of the industrial relations, clinical manifestations, roentgenological diagnosis and pathology of the disease, the scope of this paper will be limited to such additional information as may be gained in relation particularly to uncleared questions.

A division of these forty cases into three groups based upon the estimated grade of pulmonary fibrosis (Figs. 1, 2 and 3) is given in Charts I, II and III. So far as possible the duration of exposure has been ascertained but that important information is missing in many of the histories. Where possible it was supplied by the employer. All traced exposure was in an asbestos factory where prior to the time of recognition of asbestosis as an industrial hazard the working conditions were undoubtedly very dusty, but where dust control has been progressively improved in the last twenty years or so and where conditions for work are recently as good no doubt as technical circumstances will allow. In Chart I, covering cases of minor asbestosis, there is one conspicuous case (49037) of failure of history of exposure to fit the disease state. There is nothing to indicate that this man actually worked ten years in asbestos dust, and the history of ten years exposure must be doubted. In case 70964 a nodular fibrosis of the lungs was also present, in reality excluding the case from comparison. Case 15652 is included in the discussion of tuberculosis in the series. Otherwise this group showed only incidentally the finding of asbestosis. Even the employment which caused it escaped the history of the case.

In Chart II, listing cases of well developed but not advanced asbestosis, is encountered a conscious relation of the employment exposure in connection with the case, although really in most of

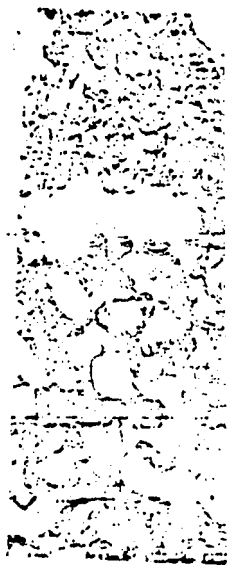
*Presented at the Thirteenth Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 8, 1947.

**From the Department of Pathology, Medical College of the State of South Carolina.

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these cases the history of the employment was traced out after the necropsy diagnosis. The shortest known exposure to cause this grade of lung fibrosis was twenty-eight months during an elapsed three years. The fibrosis was naturally of recent formation. In general the advance of the disease and the age of the lung fibrosis parallels the duration of exposure and the length of time since

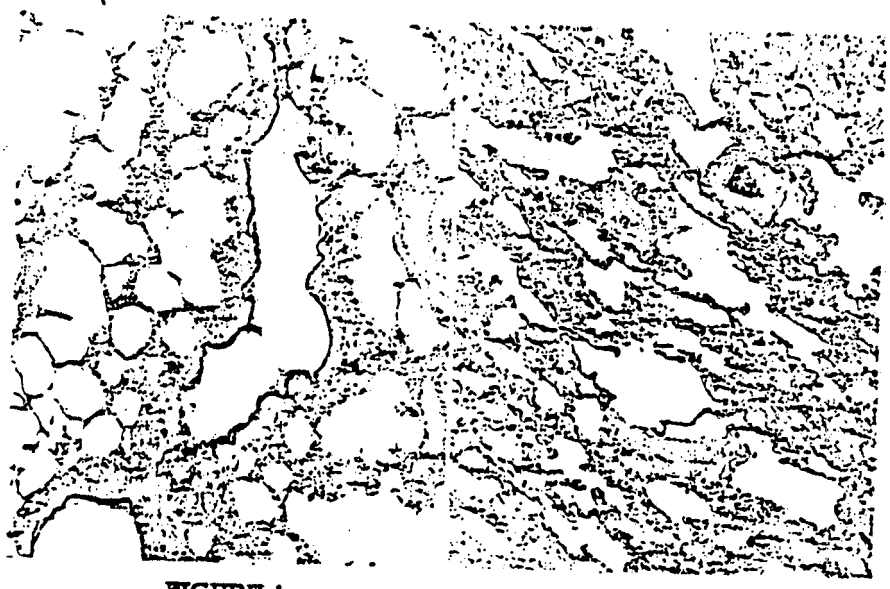


FIGURE 1

FIGURE 2

Figure 1: Grade 1 asbestosis. Human lung. X 75.
Figure 2: *Grade* 2 asbestosis. Human lung. X 75.

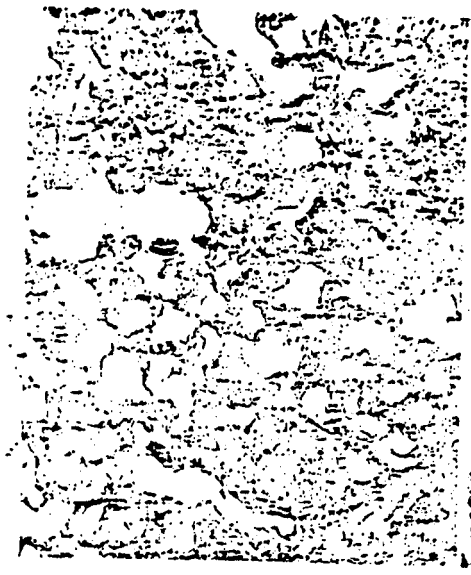


FIGURE 3

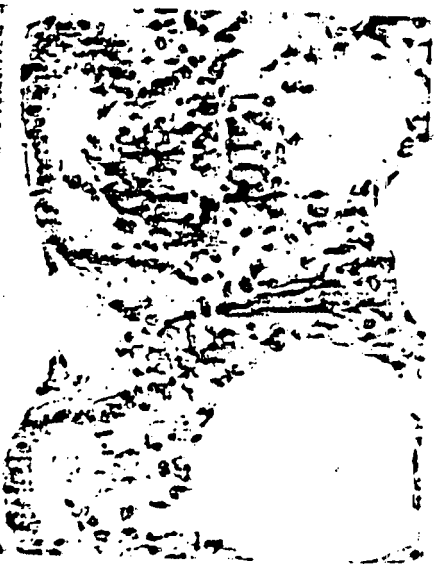


FIGURE 4

Figure 3: Grade 3 asbestosis. Human lung. X 75.—*Figure 4:* Asbestosis bodies located in terminal bronchiole and vestibular area. Human lung. X 525.

CHART I — MINOR GRADE — 12 CASES

No.	Sex & Age	Exposure	Asbestos Bodies	FIBROSIS Old	FIBROSIS Recent	FIBROSIS Nodular	Pleura	Major Disease
10672	M 32	No Data	++	.....	+	.....	++	Encephalitis, Acute
11726	M 50	No Data	+	+	+	.....	+	Aortic Insufficiency, Cor. Sclerosis
12892	F 29	No Data	++	.....	+	.....	+++	Pulmonary Tuberculosis
15652	M 32	No Data	+	+	+	.....	.....	Typhoid
20532	F 80	No Data	+	+	.....	.....	.....	Arterioscl. Gangrene of Feet
30514	M 35	No Data	++	+	.....	.....	.....	Arterioscl. Cerebral Hemorrhage
30678	F 35	No Data	+	+	.....	.....	+	Sarcoma of Uterus
36007	M 45	No Data	+	+	.....	.....	+	Drowned
48619	F 36	No Data	+	.....	+	.....	.....	Carcinoma of Groin
49037	M 28	10 yrs. ?	+	?	?	.....	.....	Endocarditis
70904	M 50	No Data	++	+	+	++	++	Arterioscl., Pneumonia
50454	M ?	No Data	+	.....	+	.....	.....	Injury

CHART II — MEDIUM GRADE — 14 CASES

CHART II — MEDIUM GRADE — 14 CASES

No.	Sex & Age	Exposure	Asbestos Bodies	FIBROSIS Old	FIBROSIS Recent	FIBROSIS Nodular	Pleura	Major Disease
9647	M 40	28 mos. in 3 yrs.	++		++			Gunshot
10944	F 40	No Data	+	++			+++	Pelvic Abscess
11511	F 55	No Data	++	+	++		+	Cor. Sclerosis
11613	M 62	No Data	+	++	++			Arterioscl. Cerebral Hemorrhage
11837	M 40	No Data	+++	++	++			Carcinoma of Neck
11967	F 45	No Data	++	++		+	++	Arterioscl.
17726	M 44	No Data	+++	++	?			Mesenteric Thrombosis
18062	F 65	No Data	++	++	+	+	+++	Aortic Aneurysm
20186	F 36	4 yrs. 14 yrs. before	+	++	+		+	Aortic Aneursym
22047	M 30	4 yrs. 3 yrs. before	+++	+	++		+++	Pulmonary Tuberculosis
25237	M 45	13 yrs. 3 yrs. before	++	++		+	++	Pulmonary Carcinoma
46916	M 37	No Data	++	++		+		Aneurysm
47715	M 37	10 yrs. 1 yr. before	++	++				Gunshot
66100	M 50	22 yrs. 1 yr. before	+++	++	++		+	Pulmonary Carcinoma

CHART III—

No.	Sex & Age	Exposure	Asbestos Bodies
9676	M 30	4½ yrs.	+++
10392	M ?	11½ yrs.	++
11599	F 25	No Data	+++
12192	F 30	No Data	+++
17620	M 38	No Data	+++
19094	M 45	10 yrs. 4 yrs. before	+++
20514	M 57	20 yrs. 1 yr. before 22 yrs. cottonmill	+++
22674	M 55	4 yrs. 11 yrs. before	+++
24693	M 45	No Data	+++
25145	M 35	½ yr. 10 yrs. before	+++
27030	M 55	13 yrs. 3 yrs. before	+++
83512	F 35	No Data	+
109411	M 38	3 yrs. 13 yrs. before	+
117595	F ?	? prior to 1920	+++

- ADVANCED GRADE — 14 CASES

FIBROSIS				
Old	Recent	Nodular	Pleura	Major Disease
+	+++		+	Pneumonia
+++	+	+++	+++	Pulmonary Fibrosis
+++	++		+	Pellegra
+++	+++	+	+++	Pulmonary Tuberculosis
+++				Hypertensive Disease
+++	+	+	+++	Pulmonary Fibrosis Tuberculosis
+++		++	++	Pulmonary Carcinoma Fibrosis
+	+++	+	+	Syphilitic Aortitis Pneumonia
++	+++			Glomerulonephritis, Chronic
+++			+	Hypertensive Disease
++	+++		+	Cor. Sclerosis; Lipoid Pneumonia
+++			+	Lung Abscess
	+++		++	Pyelonephritis
+++				Pulmonary Fibrosis

beginning. Also the prominence of asbestosis bodies in the lungs generally consistent with the duration of exposure. In this group appears two of the three cases of cancer of the lung which are especially presented in another place. There is no other indication in this chart that asbestosis of this degree is directly or indirectly responsible for death of the subject.

In Chart III, showing data on the fourteen advanced cases of the series, is seen the first positive evidence of the direct killing effect of the disease. At least four of the cases show a major part played by pulmonary fibrosis in the death of the individual. In the few cases where other factors responsible for the appearance of recent cellular fibrosis of the lungs could be ruled out, there is evidence to support Gardner's[6] experience with experimental asbestosis in guinea pigs that the fibrous disease does not progress indefinitely after cessation of exposure. There are five cases of known but not recent exposure, for instance cases 147595 and 25145, Chart III, showing advanced old fibrosis but none recent. There are twelve cases all told showing old but not fresh fibrosis. On the other hand, recent exposure, no matter of how long the duration, characteristically shows fresh fibrosis (see cases 9676 and 10392, Chart III).

The "Asbestosis Body"

The so-called asbestosis body has become a characteristic element in human asbestosis and is also found in the lungs of some animals which have been the subjects of experimental asbestos dust exposure[6] but not in others. It consists of a central asbestos fibre with a shiny yellow-brown coating appearing in a variety of architectural forms. Similarly coated bodies of smaller size may be found in silicosis and in miscellaneous occurance of no apparent relation to asbestos dust exposure.[4]

The location of asbestosis bodies (Fig. 4) in the terminal bronchioles and in the vestibular area of the lobule is significant in the pathogenesis of the disease. Those of smaller size appear in the peribronchial lymph nodes, where there is foreign body reaction but usually little if any fibrosis.

Time will not here permit full discussion of all matters of interest about these bodies. From previous publications it may be said that their formation is not an essential in the pathogenesis of experimental asbestosis in some animals although it is a characteristic microscopic feature in the naturally occurring human disease. Injury to the lung by the asbestos fibre occurs experimentally before the coating deposits in some animals[7] and without it altogether in others. It appears that the coating may be a defensive occurrence, segregating the fibers from direct tissue

contact. From study of the history of these cases it is apparent that these bodies remain deposited in the lungs more or less permanently, at least for as long as twenty-seven years (Fig. 5, case 147595, Chart III). That they undergo slow but definite change is also apparent.[1]

Findings of these bodies in the sputum is indicative of nothing more than the fact of previous inhalation of asbestos, not of the condition of the lungs. They are usually more numerous in cases of current or recent long exposure. They may be found for years after the cessation of such exposure, but in even advanced asbestosis of long duration they may not be found at all or only in sparse numbers. Since nothing valuable is to be gained from lung puncture for examination of pulmonary material for these bodies that cannot be obtained by simpler measures, there seems to be no justification for that procedure as a measure to be used in diagnosis.

Pleural Fibrosis

One of the outstanding features of disease encountered in advanced asbestosis is pleural thickening and more or less adhesive obliteration of the pleural space. Since the deposit of asbestos dust does not reach into the pleura, the reason for pleural fibrosis is not clear.

That it is not an essential of the disease state is shown by some of our cases. That it may be a secondary condition, possibly of



FIGURE 5



FIGURE 6

Figure 5: Asbestosis bodies within old fibrous and almost obliterated air spaces, from exposure 27 years previous (147,595). Human lung, X 525.—*Figure 6:* Grade 3 asbestosis (147,595). No pleural fibrosis.

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basis in intercurrent or secondary infection is indicated by other cases. In case 147595, Chart III, for instance, although there was heavy asbestos deposit and associated advanced grade of old fibrosis from exposure known to have terminated twenty-seven years before death, apparently from asbestosis itself, the pleural area was quite normal (Fig. 6). In sixteen of the forty cases there was no pleural fibrosis; even in six of the advanced cases there was none. In ten of those showing pleural fibrosis, there was some other lung disease upon which the pleural state may be blamed.

Nodular Fibrosis

In one of our previous reports[2] is cited a case (10392) of fatal fibrosis of the lungs from eleven and one-half years asbestos dust exposure. Associated with general pulmonary fibrosis in this case was the occurrence of localized hyaline nodules of scar tissue resembling silicosis. From the exhaustive history obtained in long observation of this man before he died, no exposure to silica dust was uncovered.

Later[3] we reported a case of carcinoma of the lung in advanced asbestosis which also showed silicosis type hyaline fibrous nodules within which were shown asbestosis bodies. In that report we used the term "asbestosilicosis." The subject concerned had worked in an asbestos factory for twenty-one years and previously in a cotton mill for twenty-two years.

All told, our series contains eight cases in which nodular fibrosis occurs, five in the advanced group, two in the medium group, and one among those of minor grade. Gloyne[9] also records and illustrates the occurrence of nodular fibrosis "resembling the silicotic whorl."

Although the reason for this prominent appearance of nodular fibrosis in our cases is not clear, we have previously assumed that an unknown silica dust exposure probably was involved.

However, the question of possible occurrence of nodular fibrosis in asbestosis is again raised by King, Clegg and Rae,[8] who report that nodular fibrosis of the lungs occurred from experimental intratracheal injection of rabbits with asbestos fibre of fifteen or more microns in length, while diffuse fibrosis resulted from similar injection of fibre of 2.5 microns length. This is not in accord with Gardner's[6] findings of wholly non-nodular diffuse fibrosis of the lungs in guinea pigs from inhalation of asbestos dust consisting of long fibre and practically no effect from dust of asbestos ground to eliminate long fibres. In experimental asbestosis of rabbits and guinea pigs there is the additional difference that similar, although smaller, asbestosis bodies are formed in the lungs of the latter but not in the former. King and his asso-

ci[illegible]s thought that certain black pigment associated with the [illegible]estos fibre in rabbit's lungs might represent a comparable [illegible]mation. Since there are other indications of difference in [illegible]sponse of different species to asbestos dust in Gardner's ex[illegible]eriments, this question will have to be subjected to further examination before it can be proven that nodular fibrosis of the lungs may not occur in natural asbestosis of human beings.

Carcinoma of the Lung in Asbestosis

In 1935, we[3] made a report of the occurrence of carcinoma of the lung in a case of asbestosis. Prior to that time there had been no particular reference to that disease in relation to dust exposure except, as there recalled, among the workers in the mines of the Erz mountains in Bohemia and Saxony. Published papers were cited laying suspicion upon radium examination and upon arsenic in the ore concerned. By 1939, when we reported a second, a total of eight cases of carcinoma of the lung in asbestosis had been recorded.

In various published papers in the meantime, additional cases have been reported and discussions made (see Gloyne,[10] Egbert and Geiger,[11] Nordman,[12] Anderson and Dibble,[13] Vorwald and Karr,[14] Klotz,[15] Halleb and Angrist[16] and Homburger[17]) some of which indicate a relationship of carcinoma of the lung to asbestosis and to silicosis, and some of which argue to the contrary.

In our necropsy service has been encountered an additional case, making a total of three in forty cases of asbestosis during a period of eighteen years. This incidence of 7.5 per cent is to be compared with a general incidence of 1 per cent of carcinoma of the lung in 2,683 necropsies in the last ten years. Of further interest in the question is the fact that each of our three cases had medium to advanced grades of asbestosis.

In addition to these necropsies we have recently had a case proven to be carcinoma of the lung by bronchoscopic biopsy in a man who had been exposed to asbestos dust irregularly for twenty years and who showed asbestosis bodies in his sputum. It may be interesting to compare this record with that of the experience of Klotz,[15] who reported four cases of carcinoma of the lung in fifty subjects of silicosis during eleven years, an incidence of 8 per cent. while in a total of 4,500 autopsies during the same period the incidence of lung carcinoma was only 1.18 per cent.

Apparently to the present there are at least sixteen necropsied cases of carcinoma of the lungs in asbestosis, which, considering the comparatively small necropsy experience in the disease, seems excessive. From reports of the natural occurrence and from the question raised by experimental evidence, it seems that continued

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Tuberculosis in Asbestosis

The question of whether asbestosis favors the development or progress of tuberculosis gains little evidence to support the affirmative here. Certain British reports indicate a high tuberculosis rate among asbestos workers and others do not. No acceptable proof of the contention in the form of properly controlled studies comparing tuberculosis in other groups of people living under a similar condition to the asbestos workers has been offered. On the other hand, Gardner reports from his experimental work that no influence upon tuberculosis occurred from asbestosis, and from surveys of asbestos workers no unusual incidence of tuberculosis has been found.

In our own series, from superficial analysis it might appear that tuberculosis had been favored by the condition. Six of the forty showed some form of active tuberculosis, an incidence of 15 per cent, while the incidence of occurrence of active tuberculosis in 2,683 necropsies during the same period was but 5 per cent.

A careful analysis of the individual cases, however, reveals that only four of the eight had active tuberculosis of the lungs. One of these (12192) had an advanced grade of pulmonary fibrosis showing the nodular localization characteristic of silicosis, which condition is well accepted as favoring the development of tuberculosis. Another (12892) had only a minimal and early grade of asbestosis. Of the other two, one (22047) exhibited a medium grade of asbestosis and one (19094) an advanced degree of old diffuse fibrosis.

SUMMARY

Forty necropsied cases of human asbestosis have been analyzed and certain findings related to uncleared questions are here presented.

In this series occurs evidence that the disease does not progress beyond a limited time after exposure to asbestos dust ceases, although the fibrosis caused persists and ages into dense scar tissue. In minor grades of the disease, there is little to indicate any recognizable influence. Characteristically some other unrelated condition caused the fatal disease. In practically all such cases the state of asbestosis was undiagnosed until disclosed at necropsy. This was likewise true in most of those of medium grade lung damage. Only in advanced form was asbestosis a conspicuous state and in only a part of those of that grade was it a major event or the sole factor in the fatal disability.

Asbestosis bodies remain deposited in the lung indefinitely, at least as long as twenty-seven years. In old deposits they show some evidence of change. Smaller forms do occur in peribronchial lymph nodes.

Pleural involvement as a part of the disease is not constant nor essential. It is indicated as of secondary occurrence.

Local fibrous nodular lesions comparable to those characteristic of silicosis occurred in such prominence as to question whether they may not also be produced by asbestos.

Carcinoma of the lung was also of such prominence as to require continued consideration as possibly inducible in a susceptible subject by severe asbestosis until disproven by further investigation.

Although tuberculosis of the lungs occurred with more frequency than in the general necropsy series, careful analysis of pertinent evidence does not add much weight to the idea of relationship. while some evidence occurred that improvement of tuberculosis may proceed in the face of advancing asbestosis in at least one case.

RESUMEN

Se han analizado cuarenta casos autopsiados de asbestosis humana y se presentan aquí algunos hallazgos relativos a ciertas cuestiones aún no aclaradas.

Ocurren pruebas en esta serie de que la enfermedad no progresa sino por un tiempo limitado después de haber cesado la exposición al polvo de asbesto, aunque la fibrosis producida persiste y se convierte en cicatrices espesas. En grados menores de la enfermedad, hay poco que indique alguna influencia reconocible. Generalmente alguna otra condición distinta causó la enfermedad fatal. En casi todos esos casos la asbestosis pasó desapercibida hasta que se descubrió en la autopsia. Fue esto cierto también en la mayor parte de los casos en los que el daño al pulmón fue de grado mediano. Solamente en la forma avanzada fue las asbestosis un estado conspícuo, y sólo en parte de los casos de ese grado fue el suceso principal o el único factor en la incapacidad fatal.

Los cuerpos de asbestosis quedan depositados indefinidamente en el pulmón, por lo menos por veinte y siete años. En depósitos viejos se notan algunos signos de alteración. Pequeñas formas si ocurren en los ganglios linfáticos peribronquiales.

La invasión de la pleura como parte de la enfermedad no es ni constante ni esencial y cuando ocurre es un hallazgo secundario.

Lesiones nodulares fibrosas locales, comparables a las que son características de la silicosis, ocurrieron con tal prominencia que despertaron sospechas de que quizás puedan ser producidas también por el asbesto.

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El carcinoma del pulmón también ocurrió con tal prominencia que no se debe abandonar la posibilidad de que sea producible en un sujeto sensible por la asbestosis grave, a menos de que sea refutado esto por investigaciones ulteriores.

Aunque la tuberculosis pulmonar ocurrió con más frecuencia que en la serie general de autopsias, el análisis cuidadoso de los datos pertinentes no apoya la idea de que exista alguna relación entre estas dos enfermedades, mientras que, por lo menos en un caso, hubo datos que indicaron que la tuberculosis puede seguir mejorando aún cuando la asbestosis continúa avanzando.

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Discussion

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Prior to 1930 there was almost no mention in American medical literature of any specific disease due to asbestos. In the British and French literature, authors recorded high frequency of chest

symptoms among asbestos workers and speculated on the relation of symptoms to the inhalation of asbestos or of silica or of iron or to other conditions associated with asbestos work. There were reports of a high tuberculosis morbidity among those exposed, and general high mortality. Some authors gave fair descriptions of the pathology or of the clinical picture of what we now call asbestosis but correlation of the pathology, roentgen appearance and the essential factor in etiology was lacking. There was consensus, however, that somehow asbestos work was harmful. The international encyclopedia on industrial diseases, entitled "Health and Occupation," published in 1930, deplores the lack of more accurate and detailed data on the asbestos hazard, and goes on to say that the increasing utilization of asbestos urgently calls for study of the condition.

In 1930, Lynch and Smith published the first necropsy protocol together with the detailed occupational history which left no doubt that the disease is a lung fibrosis and is directly due to the inhalation of the asbestos in the dust. R. S. Mills, in the same year, and later, Lanza, Gardner, McConnell, Pendergrass, Shull, Sparks, Stone and others made important contributions. In the seventeen years since that confession of confusion in the encyclopedia and Dr. Lynch's pioneer publication, American medicine has progressed with speed in the knowledge of this condition.

Today, we have had the privilege of hearing the latest report of the continuing observations by Dr. Lynch. Some problems, he tells us, need further elucidation, among them, the relation to carcinoma. Although we must keep an open mind, the evidence warrants the *tentative* conclusion that asbestos workers do have a higher incidence of lung cancer. Now that early lung cancer often is amenable to surgery, this gives added value to the periodic chest x-ray survey among these workers and calls for more meticulous film interpretation than is essential in most survey work.

From the viewpoint of public health, of diagnosis, and of workmen's compensation adjudication, observation is needed to establish the etiologic relation or the absence of any relationship between tuberculosis and asbestosis. Many of the older clinical observations (though not all) favored the conclusion that there is a relationship similar to that between silicosis and tuberculosis. The report we have heard today does not confirm this view. One statement, or rather the implication of one statement, of Dr. Lynch must be interpreted in the light of other clinical experience. Dr. Lynch reported one case in which tuberculosis improved in the face of advancing asbestosis. However, we occasionally find tuberculosis improving in spite of the presence of silicosis and we do know that silicosis adversely affects tuberculosis. Tuberculosis sometimes

heals under the most unfavorable conditions of the infected organ. That tuberculosis may heal in the face of asbestosis, therefore, should not add any weight to *either* side in evaluating the effect of asbestosis on tuberculosis.

Dr. Lynch may not be conscious of a sermon to the clinician contained in his post-mortem reports. He said that in most of the well-developed, though not in advanced cases, the history of asbestos exposure was traced *after* necropsy diagnosis. To me, that means that the clinicians neglected the occupational histories of their patients, probably because there was another serious disease present. That the diagnosis was made post-mortem suggests there were not enough routine chest films made of these workers when they were well and certainly not sufficient chest examination when they became ill. We should take to heart this failure to recognize the occupational disease during life. Many new substances were introduced in industry in the last decade; new compounds known only to the chemist today will be used in industry tomorrow. Some of these substances may produce chest symptoms. The *clinician*, practising in the locality of the industry, and not the pathologist should be the first to clearly state that an industrial process is a health hazard. The *clinician* must be the first to know, what is equally important from a health viewpoint, that some suspected industrial process *is not* a health hazard. Let us reflect on the significance of the fact that twenty years ago hardly one of us would have suspected that the lung symptoms presented by one of our patients working on refrigerator insulation were directly due to asbestos. Perhaps today some of us are missing the etiologic factor in cases that now are under our observation because we neglect the occupational history. Dr. Lynch's studies on the pathology would be of only passing interest if he had not combined them with investigations into the occupational history in each individual case. It is the correlation which makes them a major contribution to medicine.

There are other conclusions of Dr. Lynch's applicable to problems of the clinician. Dr. Lynch finds indications that when exposure ends the disease develops but little further. The prognosis, therefore, is definite, and that must be a comfort to the victim that makes it easier to get him readjusted to a new occupation and makes Workmen's Compensation adjudication much simpler. Dr. Lynch is speaking, of course, of the fibrosis; his statement must not be applied to the secondary cardiac embarrassment once that has begun. That may progress even without exposure. If I misinterpret Dr. Lynch's conclusion I trust he will emphatically correct me. The pathologic finding that fibrosis persists and ages into scar tissue explains the outstanding physical sign of the disease,

limitation of chest expansion and also means that no recession of symptoms or signs directly related to the fibrosis is to be expected.

Another finding is that length of exposure and severity of the disease are correlated. This sounds self-evident but in silicosis we cannot make that statement. This characteristic should enable us to concentrate and strengthen asbestosis case-finding among those workers who need it most, i.e., those with long exposure, and to be alert when examining a patient who declares: "Oh no, that dust does not bother me, I'm used to it, worked there without trouble for twenty years." Furthermore, this correlation of exposure-time with severity enables a workmen's compensation board to equitably allocate liability when two or more employees with asbestos hazards are involved in the same case. Then, too, Dr. Lynch's conclusions mean that when we have negative medical findings on a patient exposed for only a few months we can reassure him with confidence on the absence of dust disease.

As if to balance these conclusions which make our jobs easier, the newer observations indicating that nodular fibrosis may occur in asbestosis makes matters more complicated. Now, knowing that nodulation occurs in asbestosis not only the pathologic but also the roentgenologic distinction from silicosis becomes more blurred. We must be more cautious or perhaps, give up trying to reason from a film or a pathologic finding as to the type of dust exposure.

This has been an informative presentation. It necessarily is an unusual privilege to be first to hear repots of pioneer investigation from men whose publications have their place in the history of diseases of the chest. I want to express my thanks to Dr. Lynch for that privilege today.

Discussion

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I wish to compliment Dr. Lynch on the contribution he has made to our knowledge of asbestosis.

Pulmonary asbestosis differs from silicosis by size, etc., of the particles inhaled and by chemical composition. Asbestosis is caused by an alkali which is magnesium silicate, along with calcium and iron. In silicosis the offending dust particle is silicon dioxide. In asbestosis the shape of the particle causes them to be arrested more often in the bronchioles and the alveoli anl they are not so