THORACIC DEFORMITY AND VISCERAL DISPLACEMENTS 
WITHOUT PLEURAL ADHESIONS.

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In the unilateral fibroid tuberculosis a displacement of the trachea, heart or all the mediastinal contents toward the diseased side frequently takes place. Elevation of the diaphragm may also be present. A chest deformity due to the sinking in of the ribs on the affected side is not infrequently found.

These thoracic changes are brought about by the loss of lung volume due to an extensive fibroid process within that organ; by the absorption of alveolar air (atelectasis); or by a combination of both these processes. Moreover, a widespread adhesive pleurisy is usually associated with pulmonary fibrosis. The tug of pleural adhesions upon the surrounding structures has been considered a cause of thoracic deformity and visceral displacement.

The classical post-mortem finding shows a contracted, indurated and usually pigmented lung containing one or more dense walled cavities, dilated bronchi, and cheesy encapsulated nodules, surrounded by so dense and adherent a pleura that the lung is removed from the chest cavity only with the greatest difficulty. However, in spite of gross pathological changes within the lung, and abnormal positions of other thoracic structures, the pleura at times may be entirely free.

In pulmonary tuberculosis, a flattening and limited motion of one side of the chest, extensive dullness, bronchial breathing above and feeble breath sounds below; or the X-ray findings of a dense shadow more or less throughout the affected side, obliterated costophrenic angle, high or peaked diaphragm, or displaced trachea or heart, do not singly or collectively constitute a sure means of diagnosing an adherent pleura. Only by the success, or failure, in introducing air into the pleural cavity can the absence, or presence, of adhesions be definitely determined.
Case I, Figs. 1, 2, 3, 4.

Mrs. C. A. Age 32, married. The first symptoms of her disease began with a cold in the spring of 1923. From an X-ray of the chest made at that time, pulmonary tuberculosis was diagnosed. In June, 1923, the sputum was found positive for tubercle bacilli. In Novem-

Case I, Fig. I.

X-ray taken July, 1925, showing extensive tuberculosis throughout right lung. Heart slightly displaced to right.
Ca se I, Fig. II.

X-ray taken July, 1927. Note deviation of trachea and further displacement of heart to right.
CASE I, FIG. III.
X-ray taken Nov., 1928. Complete dextrocardia; marked deviation of trachea; large pulmonary cavity.
CASE I, FIG. IV.
After induced pneumothorax. Note small size of collapsed lung. No pleural adhesions except at extreme apex. Heart and trachea in normal position.
ber of the same year she had an eight ounce hemoptysis. The patient rested at home and had short periods of sanatorium care. The disease slowly progressed. She had cough, expectoration, dyspnoea, slight fever, an occasional small hemoptysis.

In February, 1927, the chest examination showed:

Rt: Dullness front and back. Bronchial breathing and widespread pectoriloquy over the upper half. Medium coarse and coarse râles over the front and back.

Lt: Few medium coarse râles in the upper third. The normal cardiac dullness was replaced by resonance extending under the sternum.

Artificial pneumothorax on the right side was instituted in October, 1928. On entrance, the intrapleural pressures were—8—20 cm. of water. An almost complete collapse of the lung was obtained. The heart has shifted back to its normal position.

Comment: The facts presented illustrate a case of pulmonary tuberculosis of seven years' duration in which the right lung became so small in size that to compensate for the loss of lung volume a complete dextro-cardiac resulted. The history, physical examination and X-ray evidence would indicate an adherent pleura, and yet, except at the apex, it was entirely free of adhesions as revealed by the pneumothorax. The extremely small size of the collapsed lung would indicate that before the pneumothorax it was not a firm, dense, fibroid structure, for if it had been how could it be reduced to such dimensions? Possibly a fair proportion of the pulmonary tissue was atelectatic, as such tissue can be further compressed by outside pressure.

Case II, Figs. 1, 2, 3, 4.

Mrs. F. F. Age 27, married. First taken sick in 1923 with cough, expectoration, fever, dyspnoea. Sputum positive for tubercle bacilli. In 1927 a phrenic nerve operation on the left side was performed. The clinical results were good.

In November, 1929, the chest examination showed:

Rt: Impaired resonance to the second rib. Broncho-vesicular breathing, and increased vocal resonance in the same area. Occasional fine râle to the second rib.

Lt: Limited motion. Apex beat in the anterior axillary line. Dullness front and back. Bronchial breathing, cavernous in quality,
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and P.T.S. to the seventh v.s. and whispered pectoriloquy to the fourth rib and seventh v.s. Feeble breathing at the base front and back. A few coarse râles scattered over the front and back.

Artificial pneumothorax was started November 18, 1929. The needle was inserted at the angle of the left scapula directly over marked dullness and loud cavernous breathing. The intrapleural pressure was too greatly negative to register on the manometer, but after 225 c.c.'s of air had been given, the pressures read —18 —16 cm, of water. Except for two or three band-like adhesions, a satisfactory collapse was obtained.

Comment: This is another case of long standing pulmonary tuberculosis, in which an adherent pleura was judged surely to be present. The pneumothorax showed otherwise. In 1929 the X-ray showed an increased density in the lower half of the left lung, and a further displacement of the heart as compared with the previous X-ray (1928). This very marked change may have been caused by an atelectasis involving the lower lobe.

CASE III, FIGS. 1, 2.

Mrs. E. Married, age 20. Her first symptoms of pulmonary disease began in June, 1924, with an haemoptysis, followed by cough, expectoration, and high fever. The sputum was positive for tubercle bacilli in 1925. The patient was an invalid for the following years until 1930, when artificial pneumothorax was begun. At this time the chest examination showed:

Rt: Impaired resonance to the second rib and third v.s. A few râles in the same area.

Lt: Dullness front and back. Bronchial breathing and whispered pectoriloquy in the upper half. Coarse râles over the front and back. At the initial insufflation in March, 1930, the pressures were too negative to register on the ordinary manometer. Treatments were continued until an excellent collapse was obtained.

Comment: Another case of pulmonary tuberculosis of six or seven years' duration showing marked displacement of the thoracic viscera to the affected side, and yet the pleura practically free of adhesions.
Case IV, Figs. 1, 2, 3.

Mr. S. Single, age 22. This young man had been sick for about two years. The sputum was positive for tubercle bacilli. He had cough, expectoration, fever.

The examination of the chest showed the right lung free of adventitious sounds, but the left showed the apex beat displaced to the left; limitation of motion, marked dullness front and back, cavernous breathing over almost the entire hemithorax, numerous coarse râles over the front and back.

On entrance with the pneumothorax needle, the intrapleural pressures were too greatly negative to measure on the pneumothorax apparatus manometer. A good collapse resulted from continued insufflations.

Comment: Of particular interest in this case is the entire freedom of pleuropericardial, or mediastinal adhesions, as shown by the presence of air between the lung and these other structures. The trachea is still displaced, but no adhesions tug on it. Obviously, the decreased negative pressure due to the shrinkage in lung volume played the chief rôle in causing the displacement of the trachea and heart.

That these visceral displacements are not caused by the tug of pleural adhesions is shown in some cases of prolonged pneumothorax treatment when the pleural space is allowed to become obliterated. The long compressed lung re-expands very little, but it and the mediastinal structures move en masse toward the chest wall, pulled over by the very negative intrapleural pressures.

Case V, Figs. 1, 2, 3, 4.

Mr. H. Age 27. First symptoms of pulmonary tuberculosis in 1921. Bilateral distribution. Artificial pneumothorax started on the right side in Oct. 1923. In 1924 a purulent effusion developed. Later, a gradual loss of space occurred, with retraction of the lung and mediastinum into the affected side.

Comment: At one time there was a dextro-cardia in the presence of a free pleural space, Case V, Fig. 4. The evident thickening of the parietal pleura as a result of the purulent effusion is probably also present as a thickened membrane encasing the lung and preventing
any expansion of that organ. The high, negative, intrapleural pressure is the chief cause of the displacement, and may also be a factor in causing the retraction of the ribs on that side, although the deformity may be due to the downward pull of the contracting parietal pleura upon the ribs, substantiating Delpech's explanation of chest deformities caused by empyema. In Case VI, Fig. 1, we have a case of pulmonary tuberculosis involving chiefly the right lung. There is a cavity present and the trachea and heart are drawn to the right. The diaphragm is high and peaked. Fig. 2 shows the character of the collapse. There are four or five large adhesions which prevent the lung from being further compressed. If we had known, before pneumothorax, where the spots of pleural symphysis were, and could have separated the pleura, without changing the intrathoracic pressure, are we to assume that the displaced trachea, heart and diaphragm would have moved back to their normal position? Not at all. Everything would have remained as formerly. The pleural adhesion played no active rôle in causing the visceral displacements.

The series here reported show, in a spectacular manner, the negligible effect of adhesions upon the visceral displacements.

The following case would indicate that high negative intrapleural pressure may alone cause a chest deformity.

Case VII, Figs. 1, 2.

Miss M. Age 31. Single. First became ill in March, 1928, with digestive disturbances, vomiting, fatigue, loss of weight. In the summer of 1929 she began to cough, expectorate, run fever, and have pains in the left chest. She was also short of breath. The sputum was positive for tubercle bacilli. In January, 1930, the chest examination showed: Rt: Dullness to the second rib and third v.s. A few fine râles in the same area. Lt: Marked flattening and limitation of motion. Apex beat displaced to the left. Dullness over the front and back. Bronchial breathing and whispered pectoriloquy to the fourth rib and fifth v.s. Below this the breath sounds were feeble. Scattered medium and coarse râles front and back.

At the initial insufflation in March, 1930, the intrapleural pressures were —8 —16 cm. of water.

Comment: The physical examination showed a marked flattening of the left chest and limited motion; the X-ray film revealed a
CASE VII, Fig. I.
Advanced pulmonary tuberculosis with marked deformity of left chest.
CASE VII, FIG. II.
After five injections of air. Note normal contour of left chest.
small left apex, with a sloping inward of the ribs and narrowing of the interspaces. After the fifth refill, when the pressures had been brought to $-2 - 0$, the X-ray film showed a complete disappearance of the thoracic deformity, the ribs having returned to their normal position, although several adhesions are present in the upper third of the thorax. At the same time inspection of the chest showed the left side was equal in contour, and its motion but slightly restricted.

DISCUSSION.

PLEURAL ADHESIONS.

That displacements of the thoracic organs may take place without a symphysis between the pleural membranes is not a new observation. Since the use of the Roentgen Ray, and artificial pneumothorax, the partial or complete freedom of the pleura has been clearly shown. In 1913 Bertier reported cases in which artificial pneumothorax was instituted for the treatment of the dextrocardia without pleural adhesions. Sargent and Stoichitza found cases with deformities of the thorax without pleural adhesions, although in a series of seventeen cases with deviation of the trachea, twelve had a total symphysis of the pleura, two a partial, while in the remaining three pneumothorax was not attempted. They believe, however, that the sclerosed lung is the active agent in producing the thoracic changes, whereas the pleura, if adherent, plays a passive rôle. Bard, Ameuville and Rist believe that the pleura does not play a prominent part in chest deformities. Guinard and Hinault found in sixty-nine cases of successful pneumothorax that twenty had mediastinal displacements before the collapse was attempted. Hedblom has discussed chest deformities, and reports a case of dextrocardia in association with an extensive pulmonary tuberculosis and a large right tuberculous empyema.

The series of cases reported has illustrated very graphically the fact that great displacement of the trachea, mediastinum, heart or diaphragm, and even chest deformity may take place without pleural adhesions, and that when adhesions are present they evidently play no part in producing the visceral displacement.

FIBROSIS OR ATELECTASIS.

There is a very close resemblance between the physical and X-ray findings in the first four cases of this series, and those cases of
massive atelectasis (collapse) brought about by an occlusion of a large bronchus by fibrous changes or by tumor, a number of which I have already reported.7 The great displacement of the heart and trachea, the limited motion, flattening of the affected side of the chest, the marked dullness, extensive bronchial breathing, and the dense, homogeneous X-ray shadow throughout the side, make one speculate whether, in the presence of a free pleural space, we are dealing with a lung completely fibrous, or whether a large part of the morbid changes may be due to atelectasis.

Favoring an atelectasis in these cases, the following facts may be cited:

1. At post-mortem, the fibroid lung is practically always surrounded by a densely adherent pleura, whereas in pure atelectasis the pleura is rarely involved.

2. The great negative intrapleural pressures found in this series are similar to the pressures found in cases of massive atelectasis as reported by Habliston.8

3. In the cases here reported, a combination of fibrosis and atelectasis should be considered, because in the upper parts of the lung, where the tuberculous cavities were present, band-like adhesions were found after pneumothorax, whereas the lower portions of these lungs were free from pleural symphysis, although the physical signs and X-ray film would indicate otherwise.

4. Pure atelectatic lung tissue is rather soft and can be further compressed by outward pressure, but a dense indurated lung remains firm and incompressible. Hence the completeness of the collapse would argue against the lung being fibrous throughout its entire structure.

The pathologist speaks of collapse induration, referring to those areas around a focus of disease where a small bronchus has become occluded, and the alveoli supplied by it atelectatic. If there are many tuberculous lesions distributed through the lung, there may be many and widespread areas of collapse induration. The stenosis of many bronchi in a lung is illustrated by the following case. A patient with tuberculosis exhibited all the signs and X-ray evidence of massive atelectasis of the left lung. Believing that the main stem bronchus was occluded, a lipiodol injection was made to see if such was the case. But the main bronchus was found patent and led into four large
dilated bronchi, while beyond this zone no lipiodol entered, indicating, I believe, an occlusion of all the bronchi in that immediate neighborhood. Whether the areas beyond the structures were still atelectatic, or had become transformed into fibrous tissues, I do not know.

Large bronchi may become occluded by fibrous changes within and around their walls, causing even a whole lobe to become atelectatic. The following cases illustrate this point.

**Case VIII, Figs. 1, 2.**

Miss R. A case of chronic pulmonary tuberculosis of many years' duration who finally died from her disease. The autopsy showed many of the bronchi of the left lung to be distorted and narrowed by fibrous changes within and without their walls. The lung was both fibrous and atelectatic, and contained cavity and dilated bronchial sacs. Of chief interest, however, was the eparterial branch from the right main bronchus. This bronchus leading to the right upper lobe was almost occluded by fibrous changes. The lobe had shrunken to extremely small size, was almost wholly atelectatic and contained a small cavity. *The pleura was not adherent.*

**Case IX, Figs. 1, 2.**

On the other hand Mr. R., who died of pulmonary tuberculosis, showed at autopsy an occluded right bronchus leading to a densely fibroid right upper lobe which had shrunken to half the size of the palm of the hand. The pleura over this lobe was very adherent to the chest wall.

These cases show that large bronchi may become occluded during the course of pulmonary tuberculosis; that as a result a whole lobe may shrink to extremely small size, and that the pleura over atelectatic tissue may be free and over fibrous tissue adherent.

I am inclined to believe, therefore, that in the type of cases that has been reported here, the freedom of the pleura, and the compressibility of the lung, plus the physical and X-ray findings, indicate that a large part of the pathological change in the structure is due to atelectasis caused by tuberculous fibrosis in and around a large bronchus.
Many cases of unilateral pulmonary tuberculosis show a flattening and limitation of motion of the diseased side. The deformity may be due to the sinking in of the interspaces, or at times to an actual falling downward of the ribs and consequent narrowing of the interspaces, as shown in Case V. In this patient, the ribs resumed their normal position after the greatly negative intrapleural pressure had been overcome by the introduction of sufficient air to raise the pressures to the normal variations, although adhesions were present. At the same time the deformity noted on inspection disappeared and the side moved equally on respiration with the opposite healthy side. Other cases might be cited to illustrate this interesting phenomenon. Evidently we are justified in assuming that much of the deformity and limited motion is due to the actual sucking in of the chest wall by the vacuum created within the chest by the marked loss of lung volume. As soon as this loss of volume, with its resulting high negative pleural pressure, is compensated by the proper amount of air introduced into the pleural cavity, the deformity is overcome and the chest assumes its normal shape and contour.

Conclusions.

1. Deformities of the thorax and visceral displacement may take place without pleural adhesions.

2. The thoracic changes are brought about by a loss of lung volume caused by fibrosis, atelectasis or both combined. An adherent pleura plays a passive rôle.

3. In the cases here reported, the evidence points towards atelectasis as playing an important part in the picture.

4. Chest deformity, flattening, and restricted motion due to pulmonary disease may be overcome in some cases by bringing high negative intrapleural pressures to normal by the introduction of air into the pleural space.

5. Even after many years of chronic tuberculosis, the pleura may be found entirely free. When therapeutic pneumothorax is indicated it should not be withheld because the physical signs indicate gross thoracic changes, or because the X-ray film shows what is usually interpreted as an adherent pleura.
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NOTE: In order to lessen the cost of production numerous illustrations have been omitted from this article.

BIBLIOGRAPHY.


DISCUSSION.

Dr. Willard B. Soper, New Haven, Conn.: There is one point that seems to be very interesting in view of the general experience of having the relatively good lung, the better lung of the two, stand up surprisingly well when the added burden is thrown upon it. The French lately have been writing, some of them, with the following conception in mind: The mediastinum is in a state of equilibrium as the result of opposed traction from the two sides. The more diseased lung has exerted the stronger pull and has drawn the mediastinum to its side. By introducing air on the more diseased side the tension there is satisfied; which allows the mediastinum to shift back, and in proportion as it shifts back it satisfies tension from the better side, thereby benefiting the latter. This mechanism very possibly explains the great improvement we see in the uncollapsed lung despite a considerable lesion in it. Just as the phrenicotomy acting from below through a shifting of the diaphragm relieves vertical tension, so does the shifting of the mediastinum relieve lateral tension.

Dr. Charles D. Parfitt, Gravenhurst, Ontario, Canada: Dr. Packard's
very interesting paper recalls to mind the first case of pneumothorax which he and I did together over seventeen years ago, in which we feared adhesions, there was such marked displacement to the left. To our surprise, we found that the fluid was aspirated quite out of the manometer tube and that there was a negative pressure in excess of 40 centimeters of water. A more recent case in which there was great displacement of the heart to the right was the one case I have referred for thoracoplasty without having attempted an initial pneumothorax. In this patient, unfortunately, the pleura was perforated at the operation and a pneumothorax occurred, which I think contributed to her death six days later. Pneumothorax should always be attempted before thoracoplasty, even when there would seem to be adhesions.