Nonperforative Pneumothorax with Negative Pressure: Traction Pneumothorax

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In the initial stage of spontaneous pneumothorax, (henceforth pn.th.) a condition which is usually caused by the rupture of an emphysematous bleb, there is an intrapleural pressure which is about equal to the atmospheric pressure. Sometimes a valve mechanism gives rise to positive pressure; such a condition is referred to as valvular or tension pn.th.

During the process of recovery, the pressure will gradually return to negative as the ruptured area heals and air is absorbed, and the lung will re-expand. Recurrences of pn.th. are frequently seen and are due to reappearance of the rupture. The negative pressure will then be reduced again.

The onset of the present case was insidious; dyspnea developed gradually and progressively, and was found to be due to bilateral pn.th.

Intrapleural pressure was abnormally low. The condition probably arose from excessive traction by retractile lung tissue, which brought about a gradual, and probably nonperforative, separation of the visceral pleura from the parietal pleura. Thus bilateral negative pressure pn.th. developed, in which highly rarified air must have been present. As might have been expected with this mechanism of pn.th. formation, spontaneous expansion of the lung was insufficient, and was hardly affected by aspiration of air from the pleural cavity. Consequently, retraction of the lungs recurred several times, with intrapleural pressure falling to subnormal.

Case Report

J. B., a 46-year-old man, was admitted on April 15, 1949 with the following history: approximately one year before the time of admis-

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Figure 1
diaphragm and excursions were limited on the right. The hilii of the lungs were of normal size. Bronchovascular markings on both sides were somewhat too dense, but difficult to evaluate because of the partial collapse of the lungs. There were fine patchy densities in the right lower chest.

Laboratory findings: erythrocyte sedimentation rate 24 mm./hr., hemoglobin 14.4 mg./100 ml., hematocrit 44 per cent, leukocytes 7,800, differential count normal. Chloride in plasma 100 mEq. per liter, CO₂ combining power 28.6 mEq./liter, urea 30 mg./100 ml.

Electrocardiogram: normal, no sign of right hypertrophy. Spirography: tidal air 300 ml., vital capacity 540 ml. Pressure as measured by pneumothorax apparatus: right —20 to —12, left —12 to —6 cm. of water (Fig. 2). An attempt to aspirate air from the left pleural cavity resulted in an increased negative pressure of —26 to —12 after 300 ml. had been removed.

The past history did not make it clear how long this bilateral pn.th. had been in existence; there had been no acute onset such as is often seen in spontaneous pn.th. Pressure was remarkably low on both sides, which made it impossible for the lung to expand after further removal of air.

Course of the disease: during the first month, several attempts were made to promote expansion of the lungs by aspiration of air from the pleural cavity. The results were unsatisfactory, as will be seen from the diagram (Fig. 2) made after a film taken on May 4 (Fig. 1), three weeks after admission.

A series of x-ray films during the observation period (April 14 through October 6, 1949) clearly indicate the recurrent character of this bilateral pn.th. Bronchoscopy was performed by the late Dr. Hildernisse on May 6. No pathologic change was found in the respiratory passages.

At this stage, we seriously considered whether any improvement might be effected by surgical treatment. Surgery might be attempted if a ruptured emphysematous bleb had produced a minute rent which refused to heal, thus preventing the pn.th. from disappearing spontaneously. In that case it might be expected that negative pressure would diminish or disappear with each increase of the pn.th. Neither happened.

On the contrary, as the pn.th. increased, negative pressure augmented. We were forced to assume that excessive retracility prevented normal expansion. Surgery would be of no avail against this. It might be possible, however, to produce adhesion of the pleurae by means of a sealing method. For this purpose we chose 40 per cent glucose, of which 5 ml. at a time was injected into the pleural space, after as much air as possible had been removed. This treatment was started when, after expansion in June, the right lung collapsed again early in July, and pronounced negative intrapleural pressure developed. By the end of July, this therapy seemed to be effective, although it proved impossible to eliminate pn.th. of the apex altogether. Towards the end of September, the degree of collapse even increased bilaterally without demonstrable cause.

On October 5, 1949, an attempt was made to promote the formation of pleural adhesions on the left side by injecting 4 ml. 1 per cent silver nitrate, after removing as much air as possible.

Perhaps as a reaction to the procedure, excessive dyspnea developed suddenly on October 6, and an x-ray film once again showed massive pn.th. on the right side, which was now nearly complete, and pleural effusion on the same side. Our preparations for another attempt at aspiration were cut short by his sudden death.

The clinical diagnosis was: traction pneumothorax caused by excessive retracility of the lungs, probably associated with interstitial fi-
brosis. Sudden death may have been due to complicating pulmonary embolism or to asphyxia.

** Necropsy findings:** We were permitted to remove only a portion of the left lung which was sent to the Pathologic Institute of Leiden University for examination. This portion of the lung was studied exhaustively by Dr. J. F. Ph. Heds.

**Gross findings:** Consistency after fixation: firm and elastic. Most of the pleura was smooth and glistening, with local membranous fibrous adhesions. A small portion of the upper lobe showed fibrous adhesions to the lower lobe. There were slight pigmented dust markings, otherwise it was pale. The sectioned surface of the lung was also firm and elastic and contained little air. The lung neither rose to the surface of the water nor sank in it, but remained floating free. It was grayish to brownish. Little blood drained from the sectioned surface. The bronchi were open and contained little or no secretion. The mucosa was pale. There were fine reticular markings of the connective tissue. The interlobular septa were too sharply marked. There were no focal lesions.

**Microscopic findings:** Fifteen specimens were taken from various sites, viz., from the pleura and from all bronchi: the lower lobe bronchus, the carina of the posterobasal, anterobasal and laterobasal branches and the posterobasal, anterobasal and laterobasal branches themselves; also from peripheral and central parts of the parenchyma of the lung, and some specimens from the remainder of the upper lobe, including what was probably part of the medial branch of the lingula. Staining methods used were: hematoxylin-eosin, mucicarmine (for mucus), Van Gieson (for connective tissue), Sudan (for fatty tissue) and Weigert's elastic tissue stain.

**Main bronchi:** Epithelium had disappeared from much of the mucosa, probably as a result of handling while the lung was being removed. Those areas which were still intact appeared to be normal, there was no metaplastic epithelium, nor were there any leukocytic elements in the epithelium. Parts of the basement membrane were somewhat thicker than usual. The tunica propria contained some disseminated, locally more or less clustered lymphocytes, as well as a few disseminated eosinophil granulocytes. The mucous glands had been normally active, their ducts were free from metaplasia. The muscularis was somewhat loose in texture, with local hyperplasia and increase of connective tissue, containing disseminated lymphocytes between the fascicles.

The less bronchi were similar in appearance, but contained more lymphocytes.

The bronchiolo contained metaplastic epithelium locally; here, too, there was evidence of lymphocyte activity, although there was no actual infiltration. The few lymphoid centers in the tunica propria showed an incipient reaction center with swollen reticulum cells. The mucous glands and mucus secretion were normal. A striking feature was the marked desquamation of alveolar epithelium, which often contained giant cells, notably in the lesser bronchi and bronchioles. Elastic tissue staining revealed a thickening of the framework of bronchi and bronchioles; Van Gieson's staining method showed a marked increase of connective tissue. Small flat alveoli in the parenchyma of the lung were mostly covered with a continuous layer of alveolar cells which had assumed the appearance of epithelium. Desquamation of alveolar cells and giant cells derived from them were often found in the alveoli. There was no fatty degeneration of alveolar cells. Van Gieson's staining clearly showed that the alveolar septa were thickened and interstitial connective tissue had increased. The elastic tissue had largely been destroyed. Elastic fibers were often loose and rolled into bundles or clusters. Slight hyperplasia of connective tissue was present in the interlobular septa and in the pleura. There was no infiltration, but there was diffuse increase of lymphocytes and some granulocytes, all of them interstitial. The vessels and capillaries were normal.

**Conclusion:** Massive atelectasis with regression of elastic tissue and marked interstitial fibrosis. Hyperplasia of alveolar epithelium with giant cells. Mild chronic bronchitis and bronchiolitis with metaplasia of the epithelium of the lesser bronchi.

No definite opinion on the case as a whole could be based on the data obtained from this portion of the lung. That would have required more material from other parts of the lungs, lymph nodes, etc. There were significant pathologic changes, viz., regression of the elastic tissue and marked interstitial fibrosis.

It is difficult to decide which was the primary lesion, either a congenital defect of the elastic tissue with secondary fibrosis, or maybe a primary chronic interstitial process of the lungs of unknown etiology (Hamman-Rich syndrome)? In the latter case, the possibility of an antecedent virus infection cannot be ruled out.

**Discussion**

It is likely that the mechanism of pneumothorax formation in this case was non-perforative. As early as 1837, Laenec suggested that under certain conditions the pleura might be capable of "sweating" gases, a process which he called "exhalation gazeuse" or "exhalation d'un fluide aériforme." It is doubtful, however, that
Laennec was referring to lungs of excessive retractility. It seems more likely that these were necropsy cases, in which no rupture could be found in the pleura—which does not prove that there had never been one.

Later workers, Proust, have rejected the theory of “exhalation gazeuse.” Only Sattler again suggested that the visceral pleura and the alveoli might allow the leakage of gas at weakened sites, even though there is no actual perforation. Traction of the lungs cannot possibly overcome the adhesive force between the visceral and parietal pleura (about five times that of atmospheric pressure) when they are impermeable and hermetically sealed together. But this changes as soon as only the smallest amount of air has penetrated into the pleural space. The chances that diffusion of gas may occur increase with the retractive force of the lungs. In this way, a partial pn.th. with pronounced negative pressure may develop so slowly that even a bilateral pn.th. might be without symptoms and remain more or less unnoticed for a long time. The patient will, of course, suffer increasingly from shortness of breath, as in our case. This genesis would account for the presence of a pronounced negative pressure in the pn.th. This seems to be the most likely explanation for the mechanism of pn.th. formation in this case. Such conditions are found only in lungs with a pronounced tendency to collapse, as seen in interstitial fibrosis (Hamman-Rich), silicosis and Boeck’s disease. However, neither in the older (compiled by Enneking’), nor in more recent literature241 similar cases of traction pn.th. have been reported.

At high altitudes, comparable conditions are encountered. As the atmospheric pressure drops, the development of pn.th. is promoted, especially the form described here of traction pn.th. This must be borne in mind when studying conditions in aviation and mountaineering.

REFERENCES

PROPHYLACTIC QUINIDINE AFTER MYOCARDIAL INFARCTION

Twenty-six patients with a recent myocardial infarct were given quinidine sulfate 0.3 gm. (5 grains) six hourly, for three weeks after admission to the hospital and 44 similar patients served as controls. Allocation of quinidine to control groups was random and the two groups were comparable on admission. Of the 26 quinidine-treated patients, seven died (26.9 per cent) and 12 of the 44 control patients (27.3 per cent). There is no evidence, therefore, from this small-scale trial that routine quinidine reduces the death rate immediately after myocardial infarction, nor is there statistical evidence that it reduces the incidence of cardiac arrhythmias.