The Unexpandable Lung*

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Artificial pneumothorax is generally regarded as a reversible procedure, and usually it is. Comparatively little attention has been directed however, to the fact that when it is stopped, very seldom does the lung fully re-expand. Pinner, Leiner and Zavod remark that the term "re-expansion" is a misnomer. Often the affected portion of the lung remains permanently collapsed, and the portion of the hemithorax thus vacated is filled variously, with fluid, by compensatory emphysema of the same or of the opposite lung, by elevation of the diaphragm, mediastinal shift or retraction of the chest wall. In all of these instances, the pleural space is obliterated, or else there is left a "dead space" between the two layers of the pleura, and the lung is said to be unexpandable.

This complication is recognized now more frequently than before, but we still do not know how often it occurs. Farber, on the basis of an admittedly limited series, estimates that about 5 per cent of lungs successfully collapsed by pneumothorax cannot be made to re-expand. This paper does not represent a statistical study; it is a report of a few cases to emphasize the importance of the clinical problem and to point to the fact that artificial pneumothorax is not always a "temporary" treatment.

When a lung fails to re-expand, generally it is because of any one or any combination of three factors: bronchial obstruction, massive pulmonary fibrosis and thickened visceral pleura.

Bronchial obstruction may be caused by external pressure but usually it means stenosis secondary to tuberculous endobronchitis. Distal to the bronchostenosis, whatever the cause, stasis and accumulation of septic products and absorption of the entrapped air produce progressive atelectasis of the affected portion of the lung, and it is incapable of re-expanding. Only too often empyema occurs as a complication of bronchial stenosis.

Massive pulmonary fibrosis may arise independently of bronchial obstruction by healing of large tuberculous areas. This

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combination of infection plus disease can prevent re-flation of the lung. As Lindbloom pointed out, fibrosis affects the interlobular septa, gradually leading to organization of most or all of the involved lung. This process of carnification cannot be reversed; consequently, even after all the air in the pneumothorax space has been exhausted, the lung will not re-expand.

The most common hindrance to re-expansion is the presence of a universally thickened visceral pleura with fibrous septa leading into the interior in all directions. Even in the absence of bronchial obstruction, such a lung cannot re-expand because the pleura, which has been enormously thickened by prolonged empyema (or occasionally hemothorax or hydrothorax), is too tough. Generally some degree of bronchial stenosis is associated with this, but even without it, air entering the bronchi under atmospheric pressure cannot distend a lung bound together by the tough fibers.

CASE I

L. C., a 23 year old white man, was first seen April 6, 1941, two days after a serious hemoptysis. Artificial pneumothorax was induced on the left side May 10, 1941. The lung was almost completely collapsed by November 11, 1941. Fluid was first noted 7 months after pneumothorax had been started. On two occasions 500 cc. were aspirated. He was last given a refill on December 10, 1943. Because of contralateral disease, an attempt was made to re-expand the left lung, but there is no obvious change in the roentgenological appearance after 10 months. His vital capacity is 1150 cc.

CASE II

E. G., a 36 year old white man was first seen January 16, 1941. Pneumothorax was started on the left side February 5, 1941, and almost complete collapse was noted on April 2, 1941. Fluid was first detected on June 14, 1941 and was removed repeatedly in 1943.

All refills of air were abandoned but no re-expansion of the lung is evident. Because the pleural space could not otherwise be obliterated, left phrenic crushing was performed on May 19, 1944, but the patient is still left with a "pleural dead space."

CASE III

E. H., a 19 year old white man was first seen February 15, 1939 for secondary syphilis for which he accepted only a few injections of an arsenical. He reported on April 25, 1940 because of hemoptysis; pneumothorax was started on the left side on May 11, 1940. Fluid was aspirated several times during 1941. Because of spread to the contralateral side, pneumothorax was started on the right side while attempts were made to re-expand the left lung in 1942. It was necessary to aspirate fluid repeatedly from the right side in 1943 and 1944. Attempt to re-expand both lungs have been unsuccessful, a pleural "dead space" existing bilaterally. His vital capacity is 1250 cc. (38 per cent of the computed normal).
CASE IV

L. W., a 27 year old white woman was first seen October 3, 1939 when she was 2 months pregnant. Because of a rapidly progressive lesion of the right lung, artificial pneumothorax was induced on December 5, 1939. Fluid was first noted on February 24, 1940. Repeated aspirations were necessary, complete collapse being evident November 8, 1940. By January 1942, there was complete re-expansions of the lower two-thirds and partial re-expansion of the upper third of the lung but a small amount of fluid persisted despite frequent attempts to remove it. The interval between treatments was extended to 10 weeks but at the end of this time, the patient complained of intense dyspnea and thoracic pain relieved only by a "refill". The lung has apparently not re-expanded at all in the past 30 months.

CASE V

N. L., a 43 year old white female, was first seen March 15, 1937 because of disease localized to the left upper lobe. After 6 months of conservative therapy, there was no change in the size of the cavity, and acid-fast bacilli were still present in the sputum; so artificial pneumothorax was induced. Adhesions preventing adequate collapse were severed 4 months later, and complete collapse of the entire lung ensued. Pure tuberculous empyema followed and persisted with febrile symptoms about 8 months, after which time, the patient became ambulatory. A small amount of fluid re-appeared from time to time, but no tubercle bacilli were isolated from it. Subsequently, 50 to 75 cc. of air administered every 15 to 20 days caused a marked rise in intrapleural pressure with moderate dyspnea. In 1940 attempts were made to re-expand the lung by prolonging the inter-treatment intervals to 12 weeks, but no re-expansion was evident roentgenologically. In 1941 treatments were suspended for a period of 7 months. At the end of that time, alarming symptoms of paroxysmal dyspnea and respiratory embarrassment began. The intrapleural pressure became exceedingly negative; it was necessary to administer oxygen by mask. Treatments were resumed and were given daily at first and subsequently at gradually lengthening intervals after which the respiratory embarrassment disappeared. In 1943 the suspicion of broncho-pleural fistula was entertained but was not sustained by gas analyses. The pleural space was eventually obliterated by a complete thoracoplasty.

If one attempts to re-expand a lung by discontinuing "refills" and finds no evidence of resumption of the former volume, generally one of two things happens. Either the pleural space is obliterated by one of the factors previously mentioned (usually formation of fluid or deviation of the mediastinal structures) or alarming symptoms ensue after a variable period of time. The intrapleural pressure becomes increasingly negative leading to respiratory and circulatory embarrassment. The patient becomes intensely dyspneic, there is a marked tachycardia, and circulatory collapse rapidly develops. That these symptoms are due to absorption of gas from the pleural space and to traction on the mediastinal vessels is indicated by the fact that they rapidly abate as soon as the intrapleural pressures have been brought back almost

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to atmospheric by instillation of air. In a sense, these symptoms are only exaggerated counterparts of those reported by many patients when the interval between refills is unduly extended. Any attempt at vigorous re-expansion of these unexpandable lungs is followed by dangerous consequences.

RECOGNITION OF THE UNEXPANDABLE LUNG

One may be warned of the imminence of the unexpandable lung by certain signs during the course of pneumothorax therapy. Small amounts of air given at a refill soon cause the post-treatment pressures to become positive; the patient needs less and less air at succeeding refills, and even with smaller amounts of air, it is possible to extend the time interval between treatments. It can also be noted at fluoroscopic examinations (and verified on roentgenograms) that there is comparatively little change in lung volume on maximal inspiration and forced expiration. After each refill, the patient complains of dyspnea.

Under these circumstances, one should at once make efforts to re-expand the lung before the irreversible phase is reached. All possible fluid should be aspirated. Bronchoscopy should be employed to determine if there is any degree of bronchostenosis which can be overcome at least long enough to permit some re-expansion of the lung.

What shall we do when a lung becomes unexpandable? The easiest way to manage this problem is simply to continue pneumothorax for the life-time of the individual. That this may not be the safest course to pursue is obvious. Pneumothorax is not efficient as a means of permanent collapse; there are always the dangers of rupture of the lung and recurrent empyema. Until a large series of patients receiving pneumothorax for a long period of time (10 to 15 years for example) has been watched to learn the incidence of these complications, we do not really know the extent of this danger. In all probability if the "dead space" is small, thoracoplasty is preferable; if the "dead space" appears too large to be collapsed easily by thoracoplasty, pneumothorax offers fewer hazards. It must be admitted that at the present time we have very little recorded experience to guide us.

PREVENTION OF THE UNEXPANDABLE LUNG

Possibly one can prevent this complication by better selection of cases and by more careful attention to details of the management of the pneumothorax patient. Inasmuch as bronchostenosis is one of the most common antecedents of the unexpandable lung, one should hesitate to perform artificial pneumothorax where bronchostenosis is impending. This means that it should not be
performed if there is strong suspicion of tuberculous endobronchitis. Unilateral wheezing, otherwise unexplained dyspnea, large amounts of sputum with marked daily variations in volume or x-ray evidence of concentration of disease about the root of the lung are symptoms, any of which should cause one to be extremely reluctant to induce pneumothorax. Unfortunately one cannot usually detect lesser degrees of bronchostenosis without bronchoscopy. It has been suggested that bronchoscopy should always be performed before artificial pneumothorax is induced. This may be an ideal prerequisite, but it is not as yet feasible in most places.

To minimize thickening of the visceral pleura, attempts should be made to reduce the effect of pleural effusions. This, of course, implies frequent aspirations. Selective collapse, where attainable through the application of collapsing (relaxing) rather than compressing pressures, may conserve the function of uninvolved lung and tend to promote eventual re-expansion.

SUMMARY AND CONCLUSION

1. There are times when a lung fails to re-expand after being collapsed by artificial pneumothorax.
2. This unexpandable state may be due to bronchial obstruction, massive fibrosis of the lung, protracted empyema or to any combination of these.
3. Early detection of this complication is possible by frequent fluoroscopic examinations and manometric observations.
4. Perhaps this complication would be encountered less frequently if all pleural effusions of any magnitude were removed by frequent aspirations, if a collapsing (relaxing) compressive type of pneumothorax were maintained, and if artificial pneumothorax were not used where bronchial stenosis exists.
5. Treatment of the unexpandable lung calls for obliteration of the pleural space by thoracoplasty or by oleothorax. If neither one is feasible, refills must be indefinitely continued.

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RESUMEN Y CONCLUSION

1. A veces no se puede obtener la reexpansión del pulmón después de haber sido colapsado por el neumotórax artificial.
2. Esta falta de expansión puede ser causada por obstrucción bronquial, fibrosis pulmonar masiva, empiema prolongado o cualquiera combinación de estos estados.
3. Mediante frecuentes exámenes radioscópicos y observaciones
manométricas es posible el descubrimiento temprano de esta complicación.

4. Tal vez sobrevendría esta complicación con menos frecuencia si se practicara la aspiración frecuente de todos los derrames pleurales de cierta magnitud, si se aplicara un neumotórax de tipo colapsante (dilatador) y no compresivo, y si no se empleara el neumotórax artificial en casos en los que existe estenosis bronquial.

5. El tratamiento del pulmón faltó de expansión exige la obliteración del espacio pleural mediante la toracoplastia o el oleotórax. Si ni el uno ni el otro son factibles deben continuarse las insuflaciones indefinidamente.

REFERENCES