Pulmonary and Circulatory Function of the
Reexpanded Pneumothorax Lung*

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Introduction. The current trend away from pneumothorax is in part based on evidence indicating marked diminution in pulmonary function of the reexpanded lung after conclusion of treatment. However, this evidence is not extensive. The function of the reexpanded pneumothorax lung has been the subject of two spirometric and three bronchospirometric investigations.

Cournand and Richards first discussed the effects of various types of collapse-therapy upon pulmonary function in 1941. They demonstrated impaired function in five cases of fibrothorax and five cases of empyema complicating pneumothorax. In a group of 11 uncomplicated reexpanded pneumothorax lungs they found diminished pulmonary function. There remained 66.5 per cent of predicted spirometric vital capacity (VC) and 53 per cent of predicted maximum breathing capacity (MBC). Other series of cases were too small to be representative. Bucher’s and Gloor’s series in which bronchospirometry was performed indicated much less reduction of pulmonary function than Cournand and Richards found. Thus the average spirometric VC in a series of 11 cases of uncomplicated pneumothorax lungs after reexpansion was 97 per cent and the MBC was 82 per cent of the predicted value. In 10 cases complicated by long standing effusion or empyema the VC was 70 per cent and the MBC 60 per cent of predicted value. The bronchospirometric VC of the reexpanded pneumothorax lung in the first group was 78 per cent and in the second 47 per cent of the predicted VC.

Because of the conflicting results of the few published studies on the function of the reexpanded pneumothorax lung, the following study was undertaken. It is based on determination of the pulmonary function of 47 suitable patients and of circulatory function in six of them.

Methods

MBC was determined using a 150 liter Tissot spirometer with 2.5 cm. tubing. The patients were tested in the standing position and were instructed to breathe through a Bennett mask as rapidly and as deeply as possible during a period of 30 seconds. The predicted normal MBC was calculated on the basis of age, sex and body surface. Spirometric studies were carried out with the patient in the sitting position in the non basal condition using a Benedict-Roth water spirometer. From the record of quiet respiration during a three minute period the resting minute...
<table>
<thead>
<tr>
<th>GROUP 1</th>
<th>RPL with normal contralateral lung</th>
<th>8  43  7  1  22  45  66.5  50  83  .....  .....</th>
<th>72</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUP 2</td>
<td>RPL with involvement of the contralateral lung</td>
<td>11  34  5  6  29  52  55  68  42  34.5  2.98</td>
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<tr>
<td>GROUP 3</td>
<td>Bilateral RPL</td>
<td>11  35  3  8  R: 33  R: 54  49  R  54  45  41.5  2.98</td>
<td>69.5</td>
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<tr>
<td>GROUP 4</td>
<td>Fibrothorax</td>
<td>8  35  4  4  57  72  57  35  82  49  3.5</td>
<td>71</td>
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<tr>
<td>GROUP 5</td>
<td>Empyema</td>
<td>9  33  4  5  24  36  49.6  33.5  66  37  2.57</td>
<td>57</td>
</tr>
</tbody>
</table>

* * * In these groups, four, six and two patients were examined respectively.
### Pulmonary and Circulatory Function of Reexpanded Pneumothorax Lung (RPL)

<table>
<thead>
<tr>
<th>GROUP 1</th>
<th>RPL with normal contralateral lung</th>
<th>4200</th>
<th>5490</th>
<th>176</th>
<th>196</th>
<th>2.8</th>
<th>2.8</th>
<th>90.7</th>
<th>88</th>
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<tbody>
<tr>
<td>GROUP 2</td>
<td>RPL with involvement of the contralateral lung</td>
<td>5360</td>
<td>4386</td>
<td>175</td>
<td>157</td>
<td>3</td>
<td>2.8</td>
<td>90.7</td>
<td>88</td>
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<tr>
<td>GROUP 3</td>
<td>Bilateral RPL</td>
<td>4893</td>
<td>3653</td>
<td>212</td>
<td>150</td>
<td>2.3</td>
<td>2.9</td>
<td>91</td>
<td>89</td>
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<tr>
<td>GROUP 4</td>
<td>Fibrothorax</td>
<td>2308</td>
<td>4034</td>
<td>72</td>
<td>297</td>
<td>3.3</td>
<td>3.7</td>
<td>88</td>
<td>86</td>
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<tr>
<td>GROUP 5</td>
<td>Empyema</td>
<td>3440</td>
<td>5140</td>
<td>98</td>
<td>272</td>
<td>3.5</td>
<td>1.9</td>
<td>91</td>
<td>89</td>
</tr>
</tbody>
</table>

*In these groups, four and two patients were examined respectively.*
ventilation and oxygen consumption were determined and the VC was measured. The predicted normal VC was calculated on the basis of age, sex and height. Residual volume was determined by the open circuit method of Darling, Cournand and Richards. Using this procedure the efficiency of alveolar mixing was determined from the percentage of alveolar nitrogen in the alveolar air after breathing of pure oxygen for seven minutes. Arterial oxygen content was measured at rest and immediately after moderate exercise by the Scholander method. Residual volume, intra-alveolar mixing index and arterial oxygen content were measured only in cases with suspicion of respiratory insufficiency.

Bronchospirometric studies were performed with a Sanborn twin spirometer and a soft rubber Zavod catheter of suitable size. The catheter was introduced after topical cocaine anesthetization, and its tip inserted into the left main bronchus. Pure oxygen was introduced and the VC, oxygen consumption and minute ventilation were measured.

Pulmonary arterial pressure (PAP) was measured by intravenous catheterization in six patients at rest and after standard exercise.

Results

The first group consisted of eight patients (Table I) with reexpanded unilateral pneumothorax in whom the contralateral lung was either normal or showed minimal disease. Four had a history of moderate or marked effusion; nevertheless, all but one had roentgenological evidence of pleural thickening. The MBC of this group was 72 per cent of the predicted value, higher than the 53 per cent found by Cournand and Richards. However, the reexpanded lung in this group lost an average of 50 per cent of its VC, and the contralateral lung 17 per cent. This later decrease of VC may be the result of distension emphysema, although this decrease may be within the range of normal variation. Leiner in his bronchospirometric studies before and after the induction of pneumothorax also found a slight decrease of VC in the contralateral side. The PAP was measured in two cases and normal values were found.

Marked restrictive insufficiency of the reexpanded lung characterized this group. The lung was fixed to the thoracic wall by mild or moderate pleural fibrosis which, however, did not invade or damage the lung parenchyma.

The second group consisted of 11 patients with reexpanded pneumothorax on one side and far advanced tuberculosis or collapse therapy on the contralateral side. In five there was moderate or marked effusion during the pneumothorax treatment. On the contralateral side, three had intrapleural pneumothorax (non-reexpanded), one had extrapleural lucite ball plombage, and one had paraffin plombage. In two phrenic crush had been performed, but at the time of bronchospirometry the paralyzing effect was over.

The average VC of the reexpanded lung in this group was 18 per cent higher than that of the preceding group. This increased value of VC
of the reexpanded pneumothorax lung when the contralateral side is severely damaged may be interpreted at the first glance as a compensatory phenomenon. However, this difference is probably not significant. There is no compensation by the contralateral lung for the loss of VC; the compensatory hyperfunction is achieved by increasing oxygen consumption. In four cases with one lung under collapse therapy and history of effusion on the reexpanded pneumothorax side there was evidence of arterial oxygen unsaturation at rest, more marked after exercise.

The third group consisted of 11 patients with bilateral reexpanded pneumothorax. There were six effusions on the right and four effusions on the left side. In six instances of measured residual volume five showed evidence of mild and one of severe emphysema. There was also evidence of arterial hypoxia in four. In these cases the PAP was also measured. Patient No. 62 had normal PAP in spite of the fact that she had bilateral pneumothorax of six years standing. Patient No. 63 with a history of bilateral pneumothorax of three years standing, right effusion and fibrosis of the right upper lobe had elevation of systolic PAP rising from 22.5 mm. Hg. at rest to 33 mm. after exercise. Patients No. 60 and 66 with history of bilateral effusion showed fairly conclusive evidence of hypertension of the lesser circulation at rest which was more marked during exercise: the systolic PAP rising from 27 and 30 at rest to 56 and 59 respectively.

The fourth group consisted of eight patients with unilateral fibrothorax. Four had effusion during collapse therapy, in another four the pleural peel was formed during reexpansion. The contralateral side was overdistended because of mediastinal shift to the fibrothorax side. In five there was active cavernous disease and in two the contralateral side had been previously treated with collapse therapy (one thoracoplasty, one phrenic crush). In four with involvement of the contralateral side there was moderate or advanced degree of emphysema and arterial oxygen unsaturation.

Bronchospirometric investigations showed that the ventilatory and respiratory functions of the fibrothorax lung were severely damaged in all cases, but that there was great inequality in the impairment of these two functions. In four the fibrothorax lung participated on the average in 38 per cent of the total ventilatory function, while its average oxygen consumption was only 13 per cent. On the other hand, in four other cases the respiratory function was better preserved than its ventilatory function, the fibrothorax lung showed greater oxygen consumption (32 per cent of total) than ventilation (21 per cent).

The fifth group consisted of nine cases of empyema as a complication of artificial pneumothorax. There were two of uncontrolled empyema and seven brought under control by repeated aspirations, oleothorax or open drainage. In four both the ventilatory and respiratory functions were poor. In five other cases there was only moderate pleural thickening after control of empyema and not only was the pulmonary function relatively preserved but the lung showed respiratory hyperfunction.
Discussion

Forlanini was of the opinion that after reexpansion the pneumothorax lung would regain its normal function. Studies of the collapsed pneumothorax lung particularly emphasized the differences between an uncomplicated and a complicated pneumothorax. An uncomplicated long-standing collapse may cause a slight fibrosis of the interalveolar septa, cuboidal metaplasia of the alveolar cells and slight pleural fibrosis possibly due to the irritant action of the air. The vessels of precapillary size in collapsed but otherwise normal parenchyma showed hyaline thickening of the wall. In pneumothorax complicated by effusion severe interstitial pulmonary fibrosis is sometimes found.

Some claim, however, that there is never an uncomplicated pneumothorax, and the expression “reexpanded lung” is a misnomer. The air in the pneumothorax space is reabsorbed but the lung does not reexpand to the pre-treatment volume. A pleural dead space results which later is filled by organized fibrinous peel. Shifting and fixation of the mediastinum, elevation and fixation of the diaphragm, and retraction of the chest wall also compensate for the shrinkage of the so-called reexpanded lung. However, Mitchell found that in 312 patients after abandonment of pneumothorax there was no thickening of the pleura in 20 per cent, slight thickening in 57 per cent, and moderate to marked in 23 per cent.

Considering the fact that pneumothorax is generally instituted in limited lesions, the fibrosis of the original lesions and accompanying distension emphysema cannot contribute much to the decrease of pulmonary function.

Pulmonary pleurogenic fibrosis is a much more important factor in impairment of pulmonary function of the reexpanded pneumothorax lung. It may present itself as a rigid frame of white scar tissue of cartilaginous consistency or as a filmy sheet covering the visceral surfaces. This “fibrosis of Brouardel” invades the lung like the teeth of a comb through the interstitial spaces and obliterates bronchi and vessels. Bronchograms show a picture of “broken twigs” or “autumnal trees” without foliage. Angiograms appear to indicate arterial occlusion.

Comparison of angiopneumographic and bronchspirometric studies showed that in the lung with diminished oxygen consumption there is always comparable reduction of the circulation. Vascular fibrosis causes an increase in the vascular resistance of the corresponding area of the lung and shunts the blood to a greater or lesser extent away from affected zone towards unaffected areas. According to some authors the fact that the pulmonary arteries are not filled during angiography does not indicate that they are poorly perfused with blood, since at operation they bleed profusely. However, the blood they transmit is oxygenated blood coming through anastomoses with the bronchial arteries (left-right shunt). In angiography the contrast medium does not enter the pulmonary artery to any large extent because it meets a counter current from the bronchial arteries.
The evolution of pulmonary pleurogenic fibrosis depends on the character of the effusion (allergic, infectious or suppurative) and its duration, but in general it is unpredictable. The evidence that there are relatively well ventilated lungs with poor circulation and lungs with good circulation and poor ventilation, as was shown in the study of our fibrothorax group, indicates that both the degree and distribution of this fibrosis varies considerably. The prevalence of one of the two processes: compression of the fibrous frame of pachypleuritis or vascular fibrosis was a deciding factor in this ventilo-respiratory dissociation. Bruce in his study of fibrothorax lung found that oxygen consumption of the "reexpanded" fibrothorax lung was more reduced than ventilation and the elimination of carbon dioxide. In one case oxygen consumption was completely abolished and the lung functioned only in the elimination of carbon dioxide.

With increasing restriction in the indications for pneumothorax and improvement of methods of its management there are fewer complications and subsequent pleurogenic fibrosis is now much rarer than it was 15 years ago. In Courmand and Richards' study in 1941 the decrease of pulmonary function of the reexpanded pneumothorax lung was less than in other types of collapse therapy. It is therefore surprising that their investigation became the basis of the subsequent anti-pneumothorax trend. In our material pulmonary function values of the reexpanded pneumothorax lung were in general higher than those of Courmand and Richards, but lower than those of Bucher and Gloor.

SUMMARY

The reexpanded lung after pneumothorax was found to lose an average of 50 per cent of its vital capacity. If the collapse was not complicated by fibrothorax or empyema, it generally preserved its respiratory function. The loss of vital capacity was not compensated by the contralateral side as was oxygen uptake. The reexpanded lung is capable of considerable respiratory hyperfunction in case of disease or collapse in the contralateral side.

In the reexpanded bilateral pneumothorax the loss of total vital capacity was about 50 per cent. In some cases increase of residual volume and hypoxemia due to distension emphysema was found.

The main factor in the impairment of pulmonary function of the reexpanded pneumothorax lung is pulmonary pleurogenic fibrosis. Fibrothorax and empyema severely damage the pulmonary function, although in some cases respiratory function is partly preserved if the pleural fibrosis does not invade the interstitial spaces. The higher ventilatory equivalent in these group is evidence that oxygen consumption is damaged more than ventilation. The reduction of the respiratory function of the reexpanded pneumothorax lung is usually compensated for by increase in oxygen uptake of the contralateral lung, i.e., the compensation is achieved through the pulmonary artery system. Nevertheless pulmonary hypertension was found only in a few cases of long-standing bilateral collapse with effusion.
El pulmón reexpandido después de neumotórax, se encontró que pierde un término medio de 50 por ciento de su capacidad, vital. Si el colapso no se complicó con fibrotórax o empiema, su función respiratoria generalmente se conserva. La pérdida de capacidad vital no fue compensada por el lado contrario como lo es la captación de oxígeno. El pulmón reexpandido es capaz de considerable hiperfunción en caso de enfermedad o colapso del contralateral.

En el neumotórax bilateral reexpandido la pérdida de capacidad vital fue alrededor de 50 por ciento. En algunos casos se encontró aumento del volumen residual e hipoxemia debida a distensión por enfisema.

El principal factor del daño a la función pulmonar en el neumotórax reexpandido, es la fibrosis pleurógena.

El fibrotórax y el empiema dañan severamente la función pulmonar aunque en algunos casos la función pulmonar es conservada en parte si la fibrosis no invade los espacios intersticiales. El más alto equivalente ventilatorio en estos grupos, evidencia que el consumo de oxígeno es más perjudicado que la ventilación.

La reducción de la función respiratoria del pulmón reexpandido es generalmente compensada por el aumento de captación del oxígeno por el otro lado y la compensación es efectuada por el sistema de la arteria pulmonar. Sin embargo, la hipertensión pulmonar se encontró sólo en pocos casos de colapso bilateral de larga duración, con derrame.

RESUME

Le poumon reexpandu après l'abandonement du pneumothorax artificiel perd la moitié de sa capacité vitale. Si le collapse n'était pas compliqué par le fibrothorax ou l'empyème sa fonction respiratoire est preservée. La perte de la capacité vitale n'est pas compensée par le poumon contralateral comme est compensée la consommation d'oxygène. Dans le cas d'affection du poumon contralateral, le poumon reexpandu est capable d'une hyperactivité respiratoire considerable. Dans le pneumothorax bilateral la perte de la capacité vitale totale est de 50 pour cent. Dans ce groupe on a noté dans quelques cas l'augmentation du volume residual et une hypoxémie due à l'empyème de distension.

La fibrose pulmonaire pleurogène est le facteur le plus important de l'endommagement de la fonction pulmonaire, comme on le voit dans le fibrothorax et l'empyème. Cependant quelquefois la fonction respiratoire est sauve si la fibrose pleurale ne pénètre pas dans les espaces interstitiaux. Le haut coefficient d'utilisation d'oxygène dans ces groupes montre que la consommation d'oxygène est plus endommagée que la ventilation. L'insuffisance ventilatoire du poumon reexpandu est compensée par l'accroissement de la consommation d'oxygène, c.à.d. la compensation est faite par le système de l'artère pulmonaire. Cependant l'hypertension dans las petite circulation n'a pas été trouvée que dans quelques cas où il y'avait une histoire d'un collapse bilateral avec un exudat.
ZUSAMMENFASSUNG

Es zeigte sich, dass die nach Pneumothorax wieder entfaltete Lunge im Durchschnitt 50% ihrer Vitalkapazität verloren hatte. Der Kollaps nicht durch einen Fibrothorax oder ein Empyem kompliziert, bewahrte sie im allgemeinen ihr respiratorisches Vermögen. Der Verlust an Vitalkapazität wurde nicht durch die Gegenseite kompensiert wie bei der Sauerstoffaufnahme. Die Wiederausgedehnte Lunge ist zu beträchtlicher respiratorischer Hyperfunktion fähig in Fällen von Erkrankung oder Kollaps der Gegenseite.

Beim wieder ausgedehnten bilateralen Pneumothorax betrug der Verlust der gesamten Vitalkapazität ungefähr 50%. In einigen Fällen fand sich eine Zunahme der Residualluft und eine Hypoxämie infolge des Dehnungs-Empysems. Den Hauptfaktor bei der Beeinträchtigung der Lungenfunktion der wieder ausgedehnten Pneumothorax-Lunge stellt die pulmonale pleurogene Fibrose dar. Fibrothorax und Empyem schädigen die Lungenfunktion in erheblichem Grade, auch wenn gelegentlich die respiratorische Funktion teilweise erhalten ist, falls nämlich die pleurale Fibrose nicht in die interstitiellen Spalträume eindringt. Das höhere Atemäquivalent in dieser Gruppe ist das Zeichen dafür, dass die Sauerstoffaufnahme mehr Schaden gelitten hat als die Ventilation.

Die Verringerung der respiratorischen Funktion der wieder ausgedehnten Pneumothorax-Lunge wird für gewöhnlich ausgeglichen durch eine höhere Sauerstoffaufnahme der Lunge der Gegenseite, d.h. der Ausgleich kommt zustande durch das pulmonale Arteriensystem. Trotzdem wurde ein pulmonaler Hochdruck nur in wenigen Fällen von lang dauerndem beidseitigem Kollaps mit Erguss festgestellt.

REFERENCES