THE RATIONALE OF PNEUMOPERITONEUM
TREATMENT OF EMPHYSEMA

Artificial pneumoperitoneum as a useful measure in the management of hypertrophic emphysema has been gaining recognition during the past few years. In judiciously selected cases, the therapeutic results are highly satisfactory in the great majority of instances.

In the excellent paper by Kory and his associates published in this issue, a precise, objective, analytical study is presented on pulmonary function and circulatory dynamics resulting from pneumoperitoneum treatment of patients with emphysema. All interested in the clinical use of this measure should familiarize themselves with the basic, illuminating, pertinent information offered in their communication.

The intelligent application of pneumoperitoneum treatment requires a thorough understanding of the pathogenesis of this disease. Chronic hypertrophic emphysema is characterized by the following pathologic structural changes: (1) Destruction of the perialveolar and peribronchial elastic fibers. (2) Dilatation or rupture of the alveoli, with the consequent formation of large air spaces. (3) Destruction of some of the perialveolar capillaries. (4) Relative increase in the size of the lung. (5) Distention of the thoracic cage. (6) Abnormally low position of the diaphragm.

Functionally, one finds: (1) lessened negativity of the intrapleural pressure; (2) impaired, deranged diaphragmatic function; (3) decreased respiratory excursions of the ribs; (4) interference with normal blood circulation.

The development of hypertrophic emphysema is brought about by the excessive intrapulmonary pressure during severe coughing of long duration, by degenerative changes in the myoelastic elements of the lung and by partial bronchial occlusion due to bronchospasm or other pathologic changes. It is well to recall in this connection that when cough is strenuous, the intrapulmonary pressure may stand as high as from 80 to 200 mm. of mercury over and above atmospheric pressure. Chronic lung infections may lead to degenerative changes in the supportive and myoelastic elements of the lung either by direct toxic influences or by the associated perivascular fibrosis. In pulmonary infections, in allergy with the lung as the shock organ and pulmonary fibrosis of all sorts there is a tendency to reflex bronchospasm. The latter may result in a check-valve mechanism which permits the ingress of air into distal portions of the lung but prevents its egress. In this manner, the trapping of some of the air inhaled results in stretching and tearing of the alveoli. Similar check-valve effect may be caused by edema, granulation or fibrosis of the bronchial wall or by the accumulation of inflammatory exudate. The condition which develops in connection with the trapping of the air may well be designated as alveolar pneumatic hypertension.
Distention of the thorax as well as the enlargement of the lung in emphysema are brought about by the loss of elasticity of this organ. With the loss of the centripetal (hilusward) traction of the elastic elements, there is a decrease in or complete disappearance of the negativity of the intrapleural pressure. The inspiratory muscles, not being obliged to counterbalance the inward pull of the intrapleural pressure, are bound to distend the thoracic cage. The normal position of the diaphragm is the direct result of the traction force of the intrapleural negative pressure. In consequence of the disappearance of the upward-traction of the intrapleural pressure in emphysema, the diaphragm occupies a constant low (inspiratory) position. In this abnormally low position, the diaphragm is functionally handicapped or completely defunctionalized. The diaphragm is responsible for from 37 to 47 per cent of the ventilatory function of the lung. Pulmonary insufficiency resulting from derangement of diaphragmatic function is aggravated by the faulty distribution of the inhaled air. The latter is mostly drawn to the peripheral areas of the lung where large numbers of alveoli are destroyed, while the intact, centrally located alveoli receive none or only small amounts of the tidal air. Another factor which contributes to hemorespiratory insufficiency is the decrease in the return flow of venous blood from the periphery to the right auricle of the heart. Thus, smaller amounts of blood are available for oxygenation in the pulmonary capillaries. Also, diminished or absent distention of the lung on inspiration fails to dilate the pulmonary vascular bed so as to establish a gradient which normally facilitates the blood flow from the right ventricle to the lung.

Pulmonary insufficiency is corrected in emphysema by artificial pneumoperitoneum in the following manner:

1) Elevation of the diaphragm and reestablishment of approximately normal intrapleural negative pressure.

2) With more negative intrapleural pressure the size of the thoracic cage is reduced; the function of the respiratory muscles of the chest wall is enhanced.

3) More competent oxygen-carbon dioxide exchange results from the improved distribution of tidal air to anatomically and functionally intact, centrally located alveoli.

4) Blood supply to the lung through the pulmonary artery becomes more adequate.

5) Pneumoperitoneum is likely to result in reflex relaxation of spastic peribronchial smooth muscles.

6) Increased efficacy of the cough mechanism, with improved expectoration, rids the lower air passages of inflammatory exudate and insures better accessibility of the alveoli for the inspired air.

7) Maintenance of the elevated position of the diaphragm refunctionalizes this important respiratory muscle.

Artificial pneumoperitoneum has definite limitations in the treatment of hypertrophic emphysema. So as to avoid disappointment to the patient as
well as to the physician it is well to remember the following possible causes of failure with this method:
1) Anatomically and functionally irreversible, extensive loss of alveoli.
2) Widespread pulmonary fibrosis.
3) Sustained bronchospasm.
4) Diaphragmatic adhesions which prevent its elevation.
5) Atrophy of disuse of the diaphragm in long standing emphysema.
6) Heart failure which cannot be corrected.
7) Faulty technic.

In reference to technic, the importance of giving small amounts of air with each treatment cannot be overemphasized. It is mandatory to give less than 1,000 cc., preferably, from 500 to 600 cc., depending upon the size of the abdominal cavity and the depth of the position of the diaphragm. Intraperitoneal inflations which are too large are bound to immobilize the elevated diaphragm and thus defeat the purpose of treatment.

I advise my patients to wear a snuggly-fitting abdominal support. The latter reduces the size of the abdominal cavity and enhances the elevation of the diaphragm. It is worn night and day. With the use of abdominal supports lesser amounts of air are required for efficient pneumoperitoneum treatment.

Andrew L. Banyai.