The Rationale of Therapeutic Pneumoperitoneum
Physiological and Mechanical Considerations*

NORMAN L. ANDERSON, M.D., F.C.C.P.
Asheville, North Carolina

A priori reasoning has in the past convinced many chest physicians that the therapeutic approach to pulmonary tuberculosis by the abdominal route with artificial pneumoperitoneum is unscientific, unsound physiologically, and radical. Indeed, to treat thoracic disease by the injection of air into the peritoneal space seemed at the onset so formidable and ridiculous that some physicians have been reluctant to abandon established methods of collapse therapy. Other physicians unfortunately have attempted to use this form of therapy in a few unselected, far-advanced cases, neither mastering the technique nor really understanding the rationale behind its use. Their results quite naturally, have been disappointing.

Other groups of courageous phthisiotherapists have succeeded in demonstrating the favorable therapeutic influence of pneumoperitoneum by extensive and prolonged experience with this technically simple and relatively safe procedure. Their results have been, for the most part, remarkable, indicating that artificial pneumoperitoneum is perhaps as effective a collapse measure as artificial pneumothorax and at the same time, apparently much safer. The advantages of this form of therapy over artificial pneumothorax have been discussed in a previous paper.1

The discrepancy between the dangerous anti-pneumoperitoneum reasoning mentioned above and the surprisingly favorable results may be clarified by examining carefully the reasons for the effectiveness of artificial pneumoperitoneum. It is the purpose of this paper to attempt to explain just why artificial pneumoperitoneum benefits pulmonary tuberculosis.

The Peritoneal Space

The peritoneal space is, ordinarily speaking, a closed one: that is, the flexible and cohesive nature of the abdominal wall, of the viscera, and of the moist serous surfaces of the peritoneum and omentum results, for the most part, in an absence of dead space. This absence of dead space not only is further effected by the tonus of the abdominal muscles, but also by the slightly sub-

atmospheric pressure present in the upper abdominal cavity resulting presumably from the negative pressure existing in the adjacent intrapleural spaces. If these concepts of intraabdominal mechanics can be visualized, then the rationale of artificial pneumoperitoneum becomes much clearer.

That there is a negative or subatmospheric pressure present in the upper abdominal cavity has been indicated by Keppich, Melchior, Krause, and Overholt, and thoroughly discussed by Banyai, who has pioneered and championed artificial pneumoperitoneum therapy. The subject becomes more complicated when one considers the variations of this subatmospheric pressure under the influences of respiration, disease, adhesions, muscular activity, and of the ever-changing mobility of the abdominal viscera.

Nevertheless, for the purposes of this discussion, we shall postulate that the rigid compartment of the thorax transmits to some degree the subatmospheric pressure of the intrapleural space subdiaphragmatically into the non-rigid structures of the abdomen. Conversely, a change in the existing intraperitoneal pressure is directly transmitted to the lower pressures of the intrapleural space and consequently to the lungs. This is apparently what takes place when, for example, 500 cc. of air is introduced intraperitoneally. The increased pressure is immediately transmitted partially to the thorax; and the lungs, being readily collapsible, are reduced correspondingly in volume. Because of the existing negative intrapleural pressure and the collapsibility of the lungs, the majority of displacement takes place in the direction of the lungs rather than toward the rigid pelvis or toward the less-rigid, but muscular abdominal wall. This occurs with the patient recumbent or standing since the points of least resistance remain the same.

Thus it is seen that, after one injection of intraperitoneal air, the diaphragm will rise nearly two centimeters at the expense of lung volume. This reduction of lung volume often results in an immediate therapeutic benefit after a single injection of air. This may be partially explained by the resulting relaxation of the lung, improved bronchial drainage from diseased areas, and by reduction of toxemias following closure of lymphatic vessels communicating with diseased areas.

**EXPERIMENTAL**

**The Pleural Space**

After a intraperitoneal injection of air what happens to the intrapleural pressure? It is increased! This has been shown to be invariable in six cases by simultaneous measurement of intra-
pleural pressure with a pneumothorax needle in the intrapleural space during the administration of artificial pneumoperitoneum with a separate apparatus.

This intrapleural pressure does not remain increased. By the time another intraperitoneal injection of air becomes due, the intrapleural space pressure has decreased practically to its normal state despite the fact that the lung volume remains reduced. The mechanism of this compensatory reaction is obscure. The fact that this pressure does return to a nearly normal subatmospheric state indicates that the thorax is prepared for an identical response to later intraperitoneal injections of air. Consequently each successive dose of pneumoperitoneum is simply another step in progressive and continued relaxation of the lungs and reduction of lung volume. Thus, in long-standing pneumoperitoneum, the intrapleural space, instead of building up a high pressure as a result of continued pressure from below, actually remains in an almost normally functioning physiological state.

That this temporarily increased intrapleural pressure returns to practically its normal state has also been repeatedly indicated in six patients by actual measurement of intrapleural pressures during the course of established pneumoperitoneum treatment. Herein lie two of the most important factors which explain the success of pneumoperitoneum: one, the initial rise of intrapleural pressure following intraperitoneal injection of air, and two, the rapid return to a nearly normal physiological state. These phenomena help explain why both functioning lungs may be partially and progressively collapsed over long periods of time while the healing process takes place. They also indicate that the mechanics and physiology of the intrapleural spaces are altered very little over a period of pneumoperitoneum therapy. Following cessation of refills, the intrapleural spaces are left essentially unaltered. This of course is not the case in long-standing artificial pneumothorax where the pleurae are invariably thickened, thus resulting in impairment of respiratory function.

The same reasoning explains partially why cases with adherent pleurae respond less satisfactorily to artificial pneumoperitoneum. In these cases lung volumes reduce with more difficulty, and higher intraperitoneal pressures must be attained before comparable rises of the diaphragm are achieved. Therefore, one of the indications for perfect suitability of a case for pneumoperitoneum therapy is the integrity of the intrapleural space. Unfortunately many cases come to this therapy when pneumothorax has failed or cannot be given because of adherent pleurae. Even so, many of them are benefited by the pneumoperitoneum.

It must not be forgotten that it may in some cases be necessary to
give maximum injections of air in order to overcome the resilience of the muscular diaphragm, thus achieving a maximum therapeutic response which is apparently related significantly to reduction of lung volume.

The Lungs

It has been shown that apical disease responds as favorably to artificial pneumoperitoneum as disease elsewhere in the lung. When one considers that pressure changes in uncomplicated cases should be transmitted equally to all parts of the closed space in the thorax, it is readily understood why apical cavities close. Another factor is that the lung is cone-shaped and it is quite possible that pressure changes from below may be augmented as they are transmitted to the smaller apex. Actually, this may be a prominent factor in the healing of apical tuberculosis following strict bed rest taken flat without a pillow, with the foot of the bed elevated, or with the patient wearing an abdominal binder. The healing of hilar tuberculous infiltrations and cavities is usually a more difficult achievement for any form of collapse therapy. This may be partly a result of the increased rigidity of the larger bronchi and blood vessels in that area, which thus prevents easy collapse. The author feels that another important factor is the proximity to the hilar area of the constantly pulsating great vessels and heart, a situation which precludes any chance of constant immobilization. These rhythmic pulsations of the heart and larger vessels in the hilar area exert a constant massaging action on the closely surrounding lung tissues which, if tuberculous, have diminished chances for healing.

The Diaphragm

The addition to pneumoperitoneum therapy of phrenic nerve crushes unquestionably results in further elevation of the diaphragm and increased collapse of the lung on the relaxed side. Furthermore, following phrenic nerve crushes, patients often seem to tolerate refills of pneumoperitoneum more comfortably. Nevertheless, phrenic nerve crushes result in altered respiratory function; and occasionally paralyzed diaphragms fail to regain normal contractility following cessation of pneumoperitoneum therapy. Phrenic nerve crushes, although often required, are therefore not necessarily considered to be a favorable adjunct to pneumoperitoneum therapy.

Abdominal Binders

The effect of abdominal binders or corsets used during the treatment of pulmonary tuberculosis with artificial pneumoperitoneum seems to be manifold. In addition to the reduction of
cosmetic inelegance resulting from a protuberant abdomen, there seems to be a soothing effect on the abdominal musculature which bears much of the burden of increased abdominal pressure. Furthermore, several days following refills of air, the diaphragmatic elevation may be maintained by tightening the binder as the air is absorbed. This results not only in a continuous elevation of the diaphragm but also in an increase in the time interval between refills. That pressures will rise and further elevation of the diaphragm can be effected by the use of abdominal binders is readily proved by comparing intraperitoneal pressures with and without a binder and comparing by fluoroscopy the elevation of the diaphragm. Nevertheless, abdominal binders are unnecessary in most patients.

**Vital Capacity**

Further observations of physiological alterations during pneumoperitoneum therapy indicate that the vital capacity is ordinarily diminished much less than would be expected considering the reduction in lung volume. Both Banyai and Bennett recorded an average decrease of twelve per cent in vital capacity in established cases. Our experience parallels these findings although in some cases the vital capacity may actually be increased. The simplest explanation of this is that the more diseased and non-functioning lung areas are collapsed selectively, leaving healthy alveoli more space to function. There are doubtless other factors, however, which mediate toward altered respiratory function. These factors include displacement of the diaphragm and mediastinum, as well as lateral displacement of the heart which may result in reduced cardiac reserve. Electrocardiographic studies by Benatt and Berg have shown that the normal tracing develops a QTc pattern and the S-T deflection in Lead II becomes flattened. No signs of coronary thrombosis or pulmonary embolism have been noted. The pattern is apparently reversible and becomes normal again with the descent of the diaphragm. Most of the changes are secondary to an anti-clockwise rotation. These authors conclude that in general pneumoperitoneum treatment in a tuberculous patient with a normal heart does not entail any damaging cardiovascular changes.

**Weight**

The question of weight gain or loss has been a perplexing one. Many of our far-advanced cases treated with pneumoperitoneum gain as much as forty pounds during the healing process. A minority, however, in spite of marked improvement of their tuberculosis, parallel bilateral pneumothorax cases by failing to gain weight. Now why should some healing patients gain and others
lose weight during the course of identical treatment?

We have tried to show roentgenographically in six patients, by means of the barium meal, that some do not gain weight because of regurgitation of food, or from anorexia resulting from diaphragmatic herniation or because of unnatural tension and torsion of the esophagus. This has been impossible to prove since all cases examined were normal in every respect with the exception of one which exhibited mild cardiopasm.

All cases, however, both those that gained weight and those that did not, were shown by the barium meal to have visceroptosis. The stomachs for the most part were downwardly and centrally displaced into the pelvis. Since this visceroptosis was present in both types of patients, it was not thought to be primarily responsible for lack of weight gain in a minority.

A clue was uncovered by the measurement of vital capacity changes in patients before and after therapy was begun. Those cases which failed to gain weight usually belonged to the minority group which exhibited marked lowering of vital capacity. Apparently the reduction of respiratory exchange is partially responsible for impaired metabolism and nutrition.

Another attendant observation was that patients with adherent pleurae—those that required higher pressures to achieve therapeutic responses—had more difficulty in gaining weight. This could also be partially explained by reduction in vital capacity resulting from the adherent pleurae.

**SUMMARY**

1) An attempt has been made to clarify some of the physiological and mechanical changes resulting from artificial pneumoperitoneum.

2) The rationale of successful artificial pneumoperitoneum therapy is discussed.

3) Two salient factors appear to be: (1) the initial rise of intrapleural pressure resulting from intraperitoneal injection of air, and (2) the compensatory return of the intrapleural pressure to normal between refills.

4) The response of apical disease to this therapy is considered in contrast to disease elsewhere in the lungs.

5) The role of phrenic nerve crush as an adjunct to pneumoperitoneum therapy is briefly considered.

6) Abdominal binders are evaluated.

7) Vital capacity and cardiac changes during artificial pneumoperitoneum therapy are discussed.

8) Some of the reasons for weight gain and weight loss during this therapy are presented.
In many institutions for the tuberculous, artificial pneumoperitoneum has become a recognized method of collapse therapy. Contrary to considered medical judgement regarding possible harmful physiological changes resulting from its use, it has been shown that the complications and physiological alterations resulting from this therapy are by no means as serious as anticipated.

Norburn Clinic, Asheville, North Carolina.

RESUMEN

1) Se ha intentado aclarar algunas de las alteraciones fisiológicas y mecánicas que siguen al neumoperitoneo artificial.

2) Se discute la razón lógica de la neumoperitoneoterapia artificial satisfactoria.

3) Los dos factores sobresalientes parecen ser: (1) la elevación inicial de la presión intrapleural causada por la inyección intraperitoneal de aire y (2) la vuelta compensatoria de la presión intrapleural a lo normal, entre las insuflaciones.

4) Se considera la respuesta a esta terapia de las lesiones apicales, en contraste a las lesiones de otras partes de los pulmones.

5) Se considera suscitamente el papel de la trituración del frénico como adjunto a la neumoperitoneoterapia.

6) Se avalúa el empleo de fajas abdominales.

7) Se discuten las alteraciones de la capacidad vital y del corazón durante la neumoperitoneoterapia artificial.

8) Se presentan algunas de las razones que explican los aumentos y las pérdidas de peso durante esta terapia.

El neumoperitoneo artificial ha llegado a ser un método reconocido de colapso terapia en muchas instituciones para tuberculosos. Contrario a la creencia de que cambios fisiológicos perjudiciales pueden resultar con su empleo, se ha demostrado que las complicaciones y alteraciones fisiológicas que siguen a esta terapia no son ni por mucho tan graves como se había supuesto.

REFERENCES