THE SIGNIFICANCE OF CHANGES IN THE LUNG VOLUME AND ITS SUBDIVISIONS DURING AND AFTER ABDOMINAL OPERATIONS *

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THE effect of abdominal operations upon the lungs in man has been studied for many years and much pertinent data have been accumulated. Earlier workers noted decreased vital capacity following abdominal operations (1, 2, 3, 4, 5, 6) and pointed out that upper abdominal operations caused a more marked decrease than lower abdominal operations (1, 2, 3, 6). Beecher (7, 8), in a more complete study, found in addition increased respiratory rate, decreased tidal air and decreased residual, functional residual, reserve, complemental and total volumes. He (7, 8) stressed the importance of these findings in relation to the occurrence of atelectasis, but did not study the specific factors responsible for the changes observed. It is evident that changes in pulmonary volume found after operation are the result of the action of a variety of factors. In the present discussion the effects of several factors will be analyzed, that is, position, binders, pain, distention and intravenously administered fluids, and these findings will be correlated to the occurrence of pulmonary and circulatory complications after operation.

The terms to be used in this discussion and their definitions are as follows:

Tidal Air.—The volume breathed with each respiration.

Residual Air.—The air remaining in the lungs after maximal forced expiration.

Functional Residual (Subtidal) Air.—The air remaining in the lungs after normal expiration; it is the sum of the residual and reserve airs. This is the volume of air which must be washed out by the tidal air during respiration. It is decreased in volume in atelectasis and when the diaphragm is elevated, and is increased in pulmonary congestion and, more strikingly, in emphysema.

Reserve (Supplemental) Air.—The air which, after normal expiration, is expelled by maximal forced expiration. It is a measure of the elasticity of the lungs and is small when the former is decreased. It

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also varies with the intrapleural negative pressure, decreasing as the pressure approaches atmospheric level.

Complemental Air.—The air which, after normal expiration, is taken in by maximal forced inspiration. It is a measure of the expansibility of the lungs and of the thoracic cage. It is diminished by certain intrapulmonary states such as fibrosis and passive congestion, by extrapulmonary factors such as pleuritic or upper abdominal pain, and by mechanical interference with inspiration.

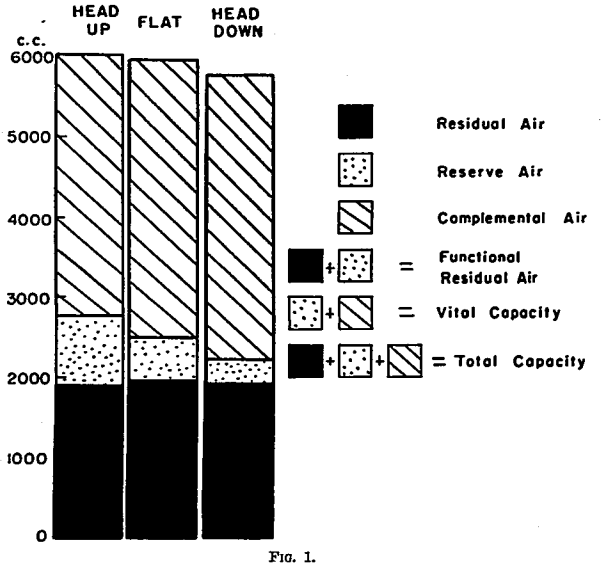

FIG. 1.

Vital Capacity.—The sum of complemental and reserve air.

Total Capacity.—The sum of residual, reserve and complemental air.

The Effect of Position.—Comparisons have been made of the volumes of various portions of the total pulmonary capacity with the patient in the sitting and lying positions; these data have been summarized by McMichael and McGibbon (9). In a more recent study, the effects of positions commonly used during and after operations were investigated (10). It was found that the residual air was unchanged and the vital and total capacities varied only slightly in the flat, Trendelenburg and head-up positions (fig. 1). However, striking changes

in functional residual, reserve and complemental air occurred (fig. 1). The volume of functional residual air was lowest in the Trendelenburg position, intermediate in the flat, and greatest in the head-up position (fig. 1). The average difference between the flat and the head-down position at an angle of 22.5 degrees with the horizontal was 17 per cent, that is, the lungs were collapsed to that extent. The volume of reserve air was greatest in the head-up, intermediate in the flat, and lowest in the head-down positions. The volume of complemental air varied inversely with the reserve air. These changes were considered to be due to a cephalad shift of the diaphragm when the patient

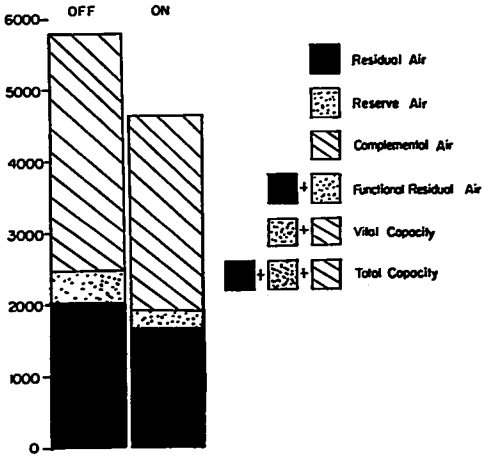

FIG. 2. Abdominal binders.

was in the Trendelenburg position. The decreases in volume of functional residual and reserve air in the Trendelenburg position are significant in two respects: (a) they indicate that this position favors atelectasis by collapsing about 20 per cent of the lung and (b) they indicate that the intrapleural pressure is less negative than normal in the head-down position. The latter factor influences the efficiency of respiration unfavorably and impairs the return of venous blood to the heart. It is apparent, therefore, that the prolonged use of the Trendelenburg position is unwise, especially in patients in whom respiration and circulation are already depressed. This includes patients in shock.

The Effect of Binders.—McMichael and McGibbon (9) showed that abdominal binders cause a cephalad shift in the position of the diaphragm and earlier workers recorded decreases in vital capacity after

the application of such binders (1, 6). A more recent study (11) has demonstrated that tight abdominal binders decrease the functional residual air by 15 or 20 per cent, the reserve air by 30 to 50 per cent, the complemental air by 7 to 17 per cent, the vital capacity by approximately 20 per cent, and the total capacity by 12 to 20 per cent (fig. 2). Lesser degrees of change occurred when binders were applied loosely. The most significant figures are those relating to the functional residual and reserve air; they indicate that binders, like the Trendelenburg position, favor atelectasis and impede respiration and the return of blood

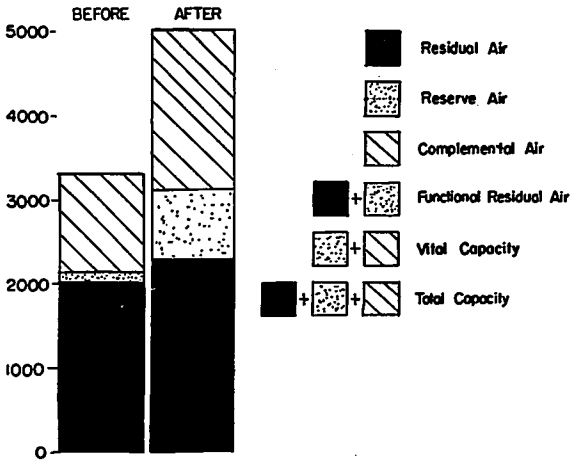

FIG. 3. Distention.

to the heart by raising the intrapleural pressure. Abdominal binders also interfere with respiration mechanically, causing increased rate and decreased tidal volume, thereby rendering the gaseous exchange in the lungs less effective. Haldane, Meakins and Priestley (12) showed that prolonged restriction of respiratory movement by tight abdominal binders may cause fatigue of the respiratory center, thereby giving rise to anoxia and respiratory arrhythmias, including periodic breathing. That abdominal binders impede the flow of blood from the legs by compressing the great veins of the abdomen also has been stated (13); the possible relation of this fact to the occurrence of phlebitis after operation has been noted (13).

The Effect of Abdominal Pain.—Clinical observations indicating that pain of abdominal wounds restricts respiration form the basis of many discussions of postoperative pulmonary complications; measure-

ments of pulmonary dynamics designed to evaluate this factor, however, are few. Attempts to evaluate the role of pain have been based on studies of the vital capacity before and after relief of pain caused by abdominal operations. Thus Overholt (6) and Capelle (14) reported increases of 10 to 25 per cent in vital capacity following abdominal surgical procedures. Zollinger (15) and Starr and Gilman (16) found more striking increases in vital capacity following anesthetization of nerves leading from the wound, as did Capelle (14) and Collins (17), who anesthetized the wound itself. It is clear that ab-

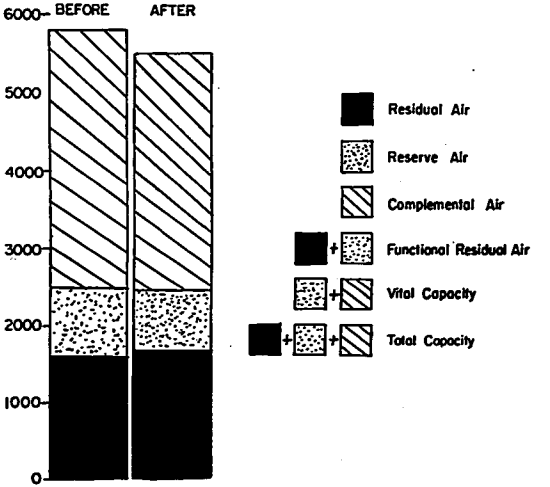

FIG. 4. Intravenous fluids 1800 cc. 185 cc. per minute.

dominal pain, by tending to decrease the depth of respiration, favors the development of atelectasis and also causes anoxia by impairing gaseous exchange. Pain also may discourage coughing and thereby lead to the accumulation of secretions. Zollinger (15), Collins (17) and Starr and Gilman (16) have suggested various types of local anesthetic mixtures for the induction of prolonged wound anesthesia.

The Effect of Distention.—Clinicians have often commented on the fact that elevation of the diaphragm due to abdominal distention interferes with respiration and favors the occurrence of postoperative pulmonary complications, but here again physiologic measurements are few. In one case studied (18), abdominal distention caused a considerable decrease in functional residual, reserve and complemental air, and

in vital and total capacity (fig. 3). Distention therefore causes atelectasis, reduced expansibility of the lungs and an increase in intrapleural pressure which impairs respiration and venous return. The return of blood to the heart is also retarded by increased intra-abdominal pressure acting directly on the inferior vena cava and its tributaries; thus the femoral venous pressure is elevated when abdominal distention occurs (19, 20). Acute distention severe enough to increase the intra-abdominal pressure to approximately 40 cm. of water may result in either cessation of respiration (19, 21) or a fall in blood pressure level due to diminished return of blood to the heart (22).

The Effect of Intravenous Infusion.—It has been stated that rapidly administered intravenous infusions or even large infusions given slowly are likely to cause pulmonary edema. It is probable, however, that this danger is exaggerated. Exclusive of patients with uremic acidosis, pulmonary edema did not occur in any patient during the last 6000 intravenous infusions of 1500 cc. or more given at this hospital (practically none was given to patients with overt congestive heart failure). Edema of the lungs occurred in two instances, two and six hours, respectively, after infusion; both were elderly hypertensive patients. Large infusions given rapidly are likely to impose considerable strain on the heart (23), but infusions of 1800 cc. given as rapidly as 185 cc. per minute in normal subjects (24) cause no significant change in pulmonary dynamics (fig. 4). Similarly in animals it is necessary to give 50 to 100 per cent of the animal's body weight in intravenous fluids to produce pulmonary edema (25, 26). However, these observations should not be regarded as vitiating the established clinical concept that patients with severe renal, pulmonary, cardiac or central nervous system disease should receive intravenous infusions slowly and in small amounts.

SUMMARY

During and after operations, various factors, that is, Trendelenburg position, distention and tight abdominal binders, elevate the diaphragm and thereby lower the functional residual air, causing atelectasis. This type of mechanically induced atelectasis cannot be cured by inhalation of carbon dioxide or by forced deep breathing. It is important since atelectasis frequently is the forerunner of postoperative pneumonia. The factors mentioned also increase intrapleural pressure, thereby impairing respiration and impeding the return of blood to the heart. Binders and distention may also impede the venous return by causing pressure on the inferior vena cava. Binders and wound pain diminish the expansibility of the lungs, inducing shallow and therefore inefficient respiration which favors anoxia, fatigue of the respiratory center and the accumulation of secretions. Intravenous infusions have no effect on the lungs of normal individuals.

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