# STUDIES OF BREATHING IN ANESTHESIA

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IN recent years, much interest has been aroused in the study of the nature of pulmonary mechanics in normal subjects and in individuals with various pulmonary disorders. As yet, little is known of the changes of the mechanical properties of the respiratory apparatus under anesthesia. Recent studies suggest that added resistance from anesthesia equipment may affect the ventilation of patients under general anesthesia (1, 2, 3). It is also conceivable that alterations of pulmonary mechanics may occur incident to anesthetic procedures, and thus would affect the work of breathing.

The mechanical work involved in ventilating the lungs is determined from the forces required to overcome the elasticity of the lungs and the airway and tissue viscous resistance. For practical purposes, the elastic properties of the respiratory apparatus are best identified as compliance (4), an expression of volume change per unit pressure change (normally 120–220 cc./cm., in the supine position [5]), while resistance is expressed as pressure change per unit air flow (normally about 3.0 cm. water/l./sec.). The elastic properties of the lungs (compliance) can be measured only when tracheal flow is zero, at which time the pressure difference between the intrapleural space and the mouth represents only the force required to maintain expansion of the lungs. When the lung volume is changing, additional forces must be available to overcome frictional resistance due to movement of the tissues in the thorax and movement of gases along the tracheobronchial tree.

The purposes of this investigation were: (1) to measure the magnitude of the resistance in various anesthesia apparatus at normal levels of ventilation and (2) to evaluate the nature and extent of alterations in the mechanics of breathing in patients under general anesthesia.

Anesthesia apparatus were tested in two ways. The first method involved the use of a potentiometric recording respirometer (fig. 1) for recording volume change, simulating tidal exchange of a human subject. In this system, flow was measured with a pneumotachograph, and pressure was measured with a Statham P23B low pressure transducer. These were recorded simultaneously on a Sanborn multichannel recorder.

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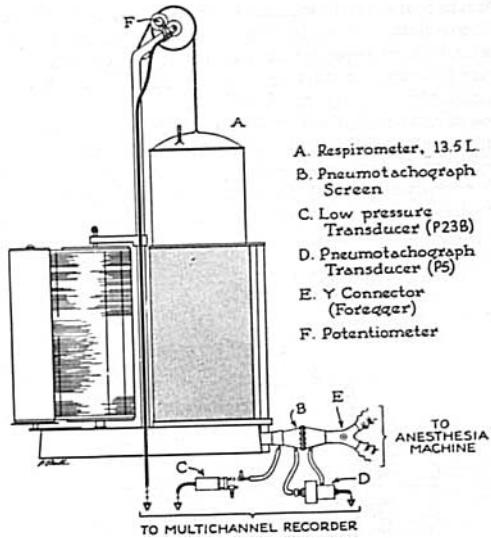


FIG. 1. Apparatus to measure resistance to air flow through anesthesia apparatus.

TABLE 1

SUMMARY OF CERTAIN PRELIMINARY DATA CONCERNING THE PATIENTS STUDIED

| Name | Age, Years | Weight, Pounds | 0.5 Sec. EC,* cc. | VC,* cc. | 0.5 Sec. EC,† / VC, Per cent | VC / PVC‡ Per cent | Operation | Comments |
|---|---|---|---|---|---|---|---|---|
| D. B. | 27 | 122 | 1,940 | 2,500 | 78 | 83 | Vaginal hysterectomy | |
| W. D. | 38 | 95 | 2,100 | 3,200 | 66 | 98 | Vaginal hysterectomy | |
| O. D. | 55 | 152 | 1,800 | 2,300 | 79 | 81 | Abdominal hysterectomy | Frequent "colds" with cough |
| L. F. | 49 | 155 | 2,200 | 3,300 | 67 | 80 | Subtotal Gastrectomy | "Asthma" in Childhood |
| F. A. | 42 | 145 | 1,500 | 2,600 | 58 | 82 | Ovarian cystectomy | "Cigarette cough" |
| N. T. | 40 | 175 | 1,600 | 2,600 | 62 | 80 | Pan hysterectomy | "Cigarette cough" |
| L. M. | 52 | 185 | 2,700 | 3,500 | 77 | 114 | Vaginal hysterectomy | |
| R. M. | 23 | 205 | 2,800 | 3,300 | 85 | 95 | Vaginal hysterectomy | |
| T. B. | 32 | 98 | 1,300 | 1,830 | 71 | 60 | Cholecystectomy | Chronic pneumonitis |
| D. M. | 44 | 183 | 2,150 | 2,800 | 73 | 80 | Pan hysterectomy | Just recovered from a "cold" |

* 0.5 Sec. EC is the expiratory capacity in the first 0.5 second of expiration during the performance of the vital capacity (VC) (27).

† Normally, the ratio $\frac{0.5 \text{ Sec. EC}}{\text{VC}} \times 100$ should be more than 60 per cent.

‡ The VC should normally exceed 85 per cent of predicted. PVC equals predicted vital capacity.

In order to test certain apparatus at various rates of flow, including higher rates of flow than could be conveniently obtained by the previous method, a rotameter type flowmeter (Fisher and Porter) was used to measure air flow from a tank of compressed air. In this system, flow was read directly from the flowmeter.

Four patients who had no evidence of pulmonary disease by history or clinical examination and six patients with a history suggestive of mild respiratory disturbances, but with negative physical findings, were selected for study. Patients' clinical data and routine vital capacity studies are listed in table 1 as a means of defining the patient material

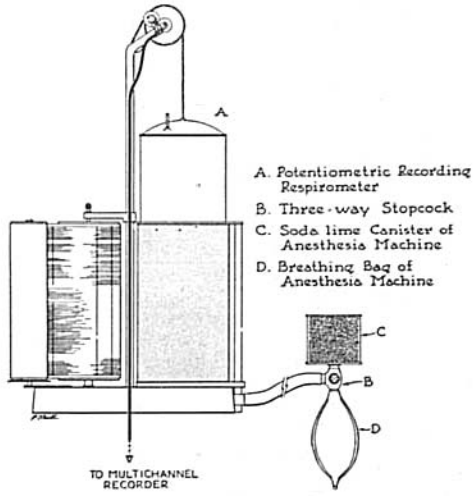


FIG. 2. Apparatus to measure respiratory volume changes during anesthesia.

of this study. Following routine hypodermic preanesthetic medications, in most instances consisting of 1/6 gr. morphine, 1/150 gr. atropine, the patients were taken to the operating room. A Cournand needle was placed in the radial or brachial artery. An intraesophageal pressure recording balloon on a polyethylene tube (6) was passed to the lower third of the esophagus and the tube was connected to the positive side of a Statham P–6 low pressure differential transducer. The negative side of the transducer was attached to an 18 gauge needle inserted into the airway at the mouth. Thus, pressure recorded in this manner represents essentially the pressure difference across the lungs. Simultaneous respiratory volume changes were recorded from a potentiometric recording respirometer and flow was

measured with a pneumotachograph placed between the mouthpiece and the respirometer. The valves and soda lime canister of the respirometer were removed to diminish resistance. Control measurements were made with the patients lying supine thirty minutes to one hour after the preanesthetic medications were given. Cyclopropane was used for induction and intubation in all cases. Magill no. 9 cuffed endotracheal tubes (inside diameter 8.5 mm., length 21.4 cm.) with curved catheter connections (inside diameter 8.5 mm.) were used. Induction was followed by ether-oxygen anesthesia, using a closed circle absorption system. During anesthesia, the respirometer with a pneumotachograph at the outlet was connected between the anesthesia

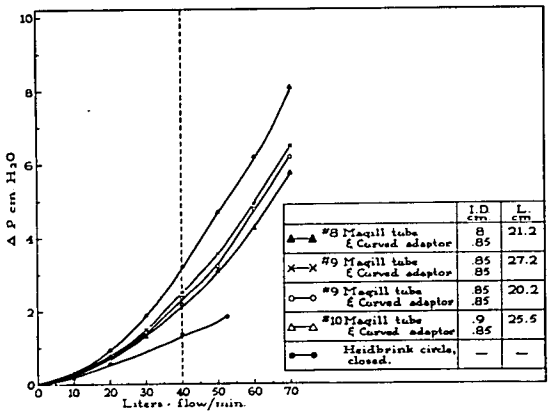


FIG. 3. Resistance to air flow through three adult sized endotracheal tubes with adapters and three commonly used anesthesia machines (represented here by Heidbrink machine.

machine and the breathing bag with a 3-way stopcock (fig. 2). Volume measurements were made by intermittently switching the patient to the oxygen filled respirometer. Arterial blood samples were drawn for blood gas and *p*H determinations. Blood carbon dioxide and oxygen contents were measured by the method of Van Slyke and Neill (7). *p*H was measured at 37.5 C., with a Cambridge electron ray meter. Carbon dioxide tension was calculated from *p*H and whole blood carbon dioxide content using the nomogram of Singer and Hastings (8).

## RESULTS

Resistance to air flow was obtained in three commonly used anesthesia machines (fig. 3). In all machines, a closed circle absorption system with the breathing bag two-thirds full (vol. 2,500 cc.) was

TABLE 2

PULMONARY COMPLIANCE, RESISTANCE, RESPIRATORY RATE, AND VENTILATION BEFORE ANESTHESIA*

| Name | Time | Pulmonary Compliance, cc./cm.$H_2O$ | Mean Resistance, cm.$H_2O$/l./sec. | Respiratory, rate/min. | Ventilation, l./min. |
|---|---|---|---|---|---|
| D. B. | 0740 | 116 | 2.68 | 20 | 7.6 |
| W. D. | 0730 | 164 | 2.32 | 18 | 6.3 |
| O. D. | 0730 | 118 | 5.56 | 13 | 6.0 |
| L. F. | 0730 | 110 | 5.68 | 18 | 7.9 |
| F. A. | 1000 | 122 | 4.72 | 15 | 8.0 |
| N. T. | 1000 | 122 | 3.49 | 20 | 9.2 |
| L. M. | 0715 | 140 | 3.65 | 17 | 8.2 |
| R. M. | 1000 | 160 | 3.22 | 20 | 8.0 |
| T. B. | 0745 | 88 | 3.21 | 22 | 5.9 |
| D. M. | 0805 | 142 | 2.76 | 20 | 8.0 |

* All patients in supine position.

tested. Under these circumstances, the resistance was 1.8 cm. water/l./sec. at flow rates of 20 liters per minute and 2.3 cm. water/l./sec. at the 60 liter-per-minute level of flow. There was no significant residual pressure at zero flow.

Three commonly used adult sized endotracheal tubes (Magill no. 8, no. 9, and no. 10) and curved catheter connections were tested. Resistance data obtained at different flow rates are summarized in figure

TABLE 3

PULMONARY COMPLIANCE, RESISTANCE, RESPIRATORY RATE, AND VENTILATION AT INDICATED TIMES AFTER CYCLOPROPANE INDUCED ETHER-OXYGEN ANESTHESIA

| Name | Time | Position* | Lung Compliance, cc./cm. $H_2O$ | Mean Resistance, cm.$H_2O$/l./sec. | Respiratory Rate/min. | Ventilation, l./min. | Time | Position* | Lung Compliance, cc./cm. $H_2O$ | Mean Resistance, cm.$H_2O$/l./sec. | Respiratory Rate/min. | Ventilation, l./min. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| D. B. | 0940 | Lith. | 83 | 4.74 | 26 | 8.6 | 1130 | Lith. | 80 | 4.95 | 22 | 8.8 |
| W. D. | 0930 | Lith. | 111 | 3.81 | 34 | 11.2 | 1100 | Lith. | 133 | 3.22 | 27 | 12.7 |
| O. D. | 0900 | Trend. | 25 | 12.02 | 35 | 9.8 | 0945 | Trend. | 20 | 15.54 | 32 | 10.5 |
| | 1045 | Trend. | 100 | 4.47 | 36 | 13.0 | | | | | | |
| L. F. | 0900 | Sup. | Controlled respiration | | | | 1145 | Sup. | 83 | 8.88 | 18 | 6.0 |
| F. A. | 1150 | Trend. | 95 | 7.51 | 33 | 11.5 | 1230 | Trend. | 105 | 7.66 | 28 | 12.3 |
| | 1310 | Trend. | 98 | 4.32 | 27 | 14.5 | 1320 | Sup. | 112 | 5.14 | 25 | 11.2 |
| N. T. | 1400 | Sup. | 120 | 4.71 | 23 | 9.0 | 1605 | Trend. | 97 | 6.64 | 37 | 11.2 |
| | 1830 | Trend. | 87 | 8.85 | 33 | 11.0 | 1940 | Trend. | 99 | 5.45 | 38 | 13.8 |
| | 2140 | Trend. | 99 | 6.68 | 38 | 9.5 | 2230 | Sup. | 110 | 4.77 | 33 | 14.2 |
| L. M. | 0820 | Lith. | 100 | 3.89 | 20 | 6.0 | 0930 | Lith. | 101 | 3.49 | 25 | 8.6 |
| | 1043 | Lith. | 99 | 4.63 | 24 | 8.9 | 1050 | Sup. | 107 | 6.60 | 21 | 8.6 |
| R. M. | 1250 | Lith. | 94 | 3.70 | 30 | 12.0 | 1350 | Lith. | 107 | 3.05 | 34 | 12.8 |
| | 1500 | Lith. | 140 | 2.05 | 33 | 13.5 | 1600 | Sup. | 110 | 1.60 | 37 | 16.6 |
| T. B. | 0900 | Sup. | 71 | 1.50 | 42 | 11.3 | 1020 | Sup. | 60 | 1.84 | 45 | 9.5 |
| | 1045 | Sup. | 54 | 2.08 | 44 | 5.9 | 1115 | Sup. | 63 | 2.95 | 32 | 10.3 |
| D. M. | 1000 | Trend. | 45 | 5.54 | 33 | 16.5 | 1040 | Trend. | 68 | 3.69 | 31 | 14.7 |
| | 1125 | Trend. | 65 | 5.09 | 29 | 16.0 | 1230 | Trend. | 65 | 6.33 | 35 | 17.5 |
| | 1300 | Trend. | 39 | 11.68 | 29 | 15.6 | 1320 | Sup. | 50 | 13.76 | 26 | 14.8 |

* Sup.—Supine; Lith.—Lithotomy; Trend.—Trendelenburg.

3. These data are in close agreement with the findings reported by Orkin (1) and by Proctor (2).

The sum of resistances to air flow of the anesthesia machine plus endotracheal tube and connections described was less than 4.5 cm. water/l./sec. at flow rates of 20 liters per minute or less. However, resistance is noted to increase more abruptly beyond this level of air flow. The range of mean air flow of the anesthetized patients varied in this study from 11.7 to 38.1 liters per minute, and averaged 23.5 liters per minute. Therefore, the sum of added resistances, at these levels of flow, from the anesthesia equipment would vary from 3.0 to 5.5 cm. water/l./sec. and average 4.8 cm. water/l./sec.

The results of the studies obtained on patients under general anesthesia are listed in tables 2, 3 and 4. Each patient served as his

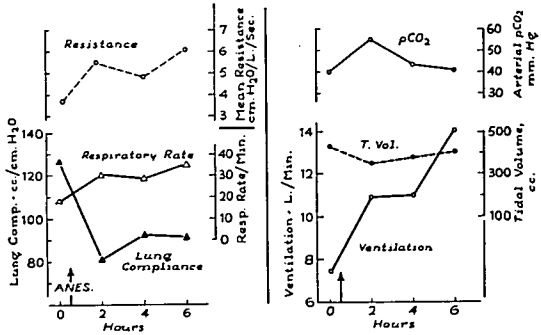


FIG. 4. Average mechanical and physiological changes of respiratory apparatus in ten patients under cyclopropane induced ether-oxygen anesthesia; arrow indicates starting of anesthesia.

own control. Figure 4 shows the average changes in the whole group. There was an overall decrease of compliance and increase in resistance during anesthesia; thus the work of breathing was increased both because of increased elastic resistance as well as increased tissue, or airway viscous resistance. The minute volume progressively increased and the respiratory rate showed a sustained rise as compared with the preanesthetic level. However, the average tidal volume decreased from a preanesthetic level of 417 cc. to 350 cc. during the early phase of anesthesia, and returned to 405 cc. during the latter part of anesthesia. The arterial $pCO_2$ was transiently elevated in most instances during the first hour of anesthesia. However, the marked average increase was a result of the high values from patients O. D. and L. F. (table 4); when these data were excluded, the average increase for the remainder of the group was not significant.

TABLE 4

CHANGES IN BLOOD GASES AND TIDAL VOLUME BEFORE AND AT INDICATED TIMES AFTER CYCLOPROPANE INDUCED ETHER-OXYGEN ANESTHESIA

| Name | Before Anesthesia | | | | During Anesthesia | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Time | Tidal Volume,* cc. | Arterial $pCO_2$, mm. Hg | Arterial $O_2$ Sat., per cent† | Time | Level of Anesthesia‡ | Tidal Volume, cc. | Arterial $pCO_2$, mm. Hg | Arterial $O_2$ Sat., per cent | Time | Level of Anesthesia | Tidal Volume, cc. | Arterial $pCO_2$, mm. Hg | Arterial $O_2$ Sat., per cent |
| D. B. | 0740 | 380 | 37 | 94 | 0930 | 2 | 330 | 44 | 100 | 1130 | 1 | 400 | 37 | 100 |
| W. D. | 0730 | 350 | 40 | 95 | 0930 | 2 | 330 | 43 | 100 | 1100 | 1 | 470 | 39 | 100 |
| O. D. | 0730 | 460 | 48 | 95 | 0900 | 3 | 280 | 100 | 100 | 0945 | 2 | 330 | 86 | 90 |
| | | | | | 1045 | 1 | 360 | 49 | 100 | — | — | — | — | — |
| L. F. | 1000 | 440 | 40 | 96 | 0810 | 3 | — | 99 | 100 | 1145 | 2 | 330 | 64 | 100 |
| | | | | | 1220 | 1 | — | 48 | 100 | — | — | — | — | — |
| F. A. | 1000 | 530 | 36 | 94 | 1150 | 2 | 350 | 38 | 100 | 1230 | 1 | 440 | 36 | 100 |
| | | | | | 1310 | 1 | 530 | — | — | 1320 | 1 | 450 | 36 | 100 |
| N. T. | 1000 | 460 | 38 | 96 | 1400 | 1 | 300 | — | — | 1605 | 2 | 300 | 53 | 100 |
| | | | | | 1830 | 2 | 330 | 46 | 100 | 1910 | 1 | 360 | 44 | 100 |
| | | | | | 2140 | 2 | 250 | 48 | 100 | 2230 | 1 | 430 | 38 | 100 |
| R. M. | 1000 | 400 | 36 | 94 | 1250 | 1 | 400 | 43 | 100 | 1350 | 1 | 380 | 40 | 100 |
| | | | | | 1500 | 1 | 410 | 40 | 100 | 1600 | 1 | 450 | 34 | 100 |
| L. M. | 0715 | 480 | 36 | — | 0820 | 2 | 300 | 43 | 100 | 0930 | 1 | 340 | 36 | — |
| | | | | | 1043 | 1 | 370 | 37 | — | 1050 | 1 | 410 | 37 | — |
| T. B. | 0745 | 270 | 39 | 95 | 0900 | 2 | 270 | 47 | 100 | 1020 | 1 | 210 | 40 | 100 |
| | | | | | 1045 | 3 | 135 | — | — | 1115 | 1 | 320 | 38 | 100 |
| D. M. | 0805 | 400 | 44 | 98 | 1000 | 1 | 500 | 39 | 94 | 1040 | 1 | 470 | 37 | 100 |
| | | | | | 1125 | 1 | 550 | — | — | 1230 | 1 | 500 | — | — |
| | | | | | 1300 | 1 | 540 | 36 | 87 | 1320 | 1 | 570 | 36 | 100 |

* Tidal volumes were corrected to BTPS.
† Arterial samples were drawn while breathing room air for control study, and breathing ether-oxygen mixtures during anesthesia.
‡ Numbers indicate planes of surgical stage.

The responses to anesthesia can be divided into two types (figure 5) as shown from the data in tables 3 and 4. One type of response was characterized by a marked increase of viscous airway or tissue resistance; and a second type was characterized by a lack of significant change in pulmonary resistance. Moreover, in a few instances, the resistance was lower after anesthesia and intubation than it was during the preanesthetic control period. Pulmonary compliance, however, decreased in all cases. Both the respiratory rate and ventilation invariably increased but without direct relationship to changes either in compliance or in resistance.

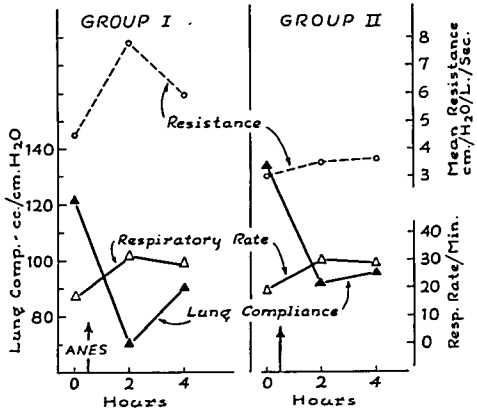


FIG. 5. Two types of responses to anesthesia; arrow indicates starting of anesthesia.

In order to evaluate the effect of changes in respiratory rate alone on pulmonary compliance and resistance in these subjects, studies were performed in three patients prior to anesthesia, in whom it was noted that increases in respiratory rate up to 50 per minute were not accompanied by changes in compliance. Other investigators (9, 10) have reported similar findings in normal subjects.

## DISCUSSION

*Resistances of Anesthesia Apparatus.*—On studying the effects of added resistance to breathing in normal conscious adults, Cain and Otis (11) found that breathing against resistance of 38.5 cm. water/l. /sec. resulted in considerable increase in the work of breathing and decrease in total ventilation which, after 12 minutes, was accompanied by a slight elevation of alveolar $pCO_2$ and a slight decrease of $pO_2$. On the other hand, at a resistance level of 14 cm. water/l./sec., there was only a slight increase in the work of breathing during quiet respiration. In this study, however, the magnitude of the resistance from the

anesthesia equipment (maximum 5.5 cm. water/l./sec. at a flow rate of 38 l./min.) was much lower than the levels studied by Cain and Otis. In addition, in view of the fact that in some cases the pulmonary resistance, after endotracheal intubation, was lower than the preanesthetic level, it becomes apparent that skillful insertion of a proper sized endotracheal tube would alter the resistance of upper airways so that the resistance from the tube should not be additive to the total resistance to breathing. Thus, only the resistance of the anesthesia machine can be considered as additive resistance. Moreover, at the levels of air flow encountered during anesthesia, this level of resistance did not exceed 2.0 cm. water/l./sec. at a maximum flow rate of 38 l./min.

*Changes in Pulmonary Viscous Resistance.*—Increase in airway resistance usually results from narrowing or partial obstruction of the tracheobronchial tree. This could be due to accumulation of secretions, reflex bronchospasm, or bronchial edema. During anesthesia, mucus production may be increased by local irritation from ether vapor and by mechanical stimulation from intubation. With narcotic depression of cough and ciliary action (12) and prolonged recumbent position, elimination of secretion is hindered. It can also be expected that in patients with bronchopulmonary disease and increased irritability of the tracheobronchial tree, hypersecretion may result from the slightest stimulation. It is interesting to note that those patients who presented a history of respiratory symptoms also showed marked increases in pulmonary resistance during anesthesia (tables 1, 2, and 3).

Posture changes with shifting of the respiratory midposition may be another cause of increase in viscous resistance. With the Trendelenburg position, elevation of the diaphragm and viscera may lower the resting midposition (13) (14). According to Butler and associates (15), increased viscous resistance in ventilation at lowered midpositions was a result of airway narrowing.

*Changes in Respiration and Ventilation.*—The increased respiratory rate observed during anesthesia in these studies could have resulted from any of a number of factors. Increase in airway resistance is known to cause increase in the rate of respiration. Patients O. D. and N. T. may be examples. There is no definite way however of excluding other causes. According to the observations of Marshall and associates (16, 17), increase in respiratory rate may represent a compensatory response to a decreased compliance since the work of breathing is less at small tidal volumes with a lung that is relatively less compliant. Among the cases studied here, changes in rate of breathing may have been related in some but not in all instances to changes in compliance. Finally, the well known reflex stimulation of the respiratory center during ether anesthesia seems to be the most appropriate explanation for the respiratory rate increase in many of the patients.

During the early phases of anesthesia, the respiratory rate increase was not accompanied by a proportional increase in ventilation. This was probably a result of depression of respiratory center by the pre-

anesthetic and anesthetic agents. Consequently, alveolar ventilation became inadequate and arterial $pCO_2$ elevated. Later, as the effect of the preanesthetic opiates subsided and the patients were maintained at lighter planes of anesthesia (table 4), minute ventilation and tidal volume increased, resulting in a greater alveolar ventilation and lowering of the $pCO_2$ to normal values.

*Changes in Pulmonary Compliance.*—A decrease in pulmonary compliance means an increase in rigidity of the lungs which may result from a variety of factors. Increased stiffness of the lungs, resulting from augmented bronchomotor tone by vagal stimulation and by broncho-constrictor drugs, has been produced in experimental animals (18, 19). There is no evidence that this factor was significantly operative in this series.

According to Nissel *et al.* (20), complete bronchial obstruction with reduction of lung volume in cats was accompanied by decreased compliance. Areas of complete segmental obstruction by secretions cannot be excluded in some of these cases. Of particular interest is the fact that patients O. D. and D. M., whose compliance showed a marked decrease during anesthesia, were found at the same time to have low arterial oxygen saturation. These findings are suggestive of the presence of intrapulmonary shunts, resulting from blood circulating through completely obstructed or nonventilated portions of the lung.

Increased respiratory rates accompanied by marked increases in pulmonary resistance results in uneven ventilation and decreased compliance. According to Mead and associates (9) and also Otis and associates (10), obstructed portions of the lung participate less in the total ventilation during high frequency breathing and, in effect, a smaller total lung volume is being ventilated.

Changes of pulmonary circulatory status may affect pulmonary compliance. Decreased compliance in patients with mitral stenosis on lying flat was said to be attributed to pulmonary congestion (17). Increased rigidity of the lungs was also noted to be associated in patients with elevated pulmonary arterial pressure and in normal subjects with increased central venous pressure following rapid infusion of albumin (5). Certain pulmonary circulatory changes may occur secondarily to anesthesia or changes in posture. Johnson has found increases of pulmonary blood volume during light ether anesthesia and during anesthesia with patients in the Trendelenburg position (21). During ether anesthesia, there were also evidences of increased pulmonary arterial pressure (21) and rises in central venous pressure (22), (23). Furthermore, obstructed breathing with an exaggeration of negative intrapleural pressure during inspiration may lead to pulmonary congestion and intra-alveolar edema. Thus, in some patients of this study, the decrease in compliance may have been a result of either pulmonary hypertension or, possibly, pulmonary congestion.

Finally, posture changes, such as the lithotomy or the Trendelenburg position during anesthesia with a resultant lowered respiratory

midposition may play a direct role in decreasing the pulmonary compliance (15). On breathing at a lowered midposition, Butler and associates suggested that the increases in elastic work were due to narrowing of some of the smaller airways which resulted in air trapping and rendered the lung less compliant.

Although compliance of the thorax was not measured in this study, the effects of anesthesia as well as posture changes on the compliance of the thorax need to be emphasized. In anesthetized and completely apneic patients, Nims, Conner, and Comroe (24) found that the compliance of the thorax was decreased as compared with the patients in the conscious and "relaxed" state. The authors suggested that the decreased compliance in apneic patients was due to abolition of normal inspiratory muscle tone, therefore measurements obtained in this manner represent true thoracic compliance. The head-down position which necessitates an increase in relaxation pressures of the thoracic apparatus at any level of lung expansion in order to displace the diaphragm and abdominal viscera also causes a measured decrease in compliance of the thorax (25, 26).

## Summary

In order to elucidate factors involved in the work of breathing during general anesthesia, studies on pressure volume changes of the lungs as well as the measurements of resistance to air flow in anesthesia apparatus were made. It was concluded: resistance produced by properly selected and employed equipment was of no physiological significance in these subjects at normal levels of air flow.

The causes of increased airway or tissue viscous resistance were various. Narrowing of airways as a result of partially obstructive secretions, bronchial edema, reflex bronchospasm, or breathing at a lower midposition in some cases are apparent. The mechanisms of increased tissue viscous resistance are not clear.

Decreases in lung compliance may have resulted from any of the following: small areas of complete bronchial obstruction by secretions; augmented respiratory frequencies in the presence of increased airway or tissue viscous resistance; pulmonary hypertension or pulmonary congestion; or changes in respiratory midposition. In any given case, the exact mechanism leading to changes in compliance or resistance may not be apparent.

The respiratory rate, which was generally increased, varied without direct relation to either changes in compliance or in resistance in most of these patients.

These studies suggest the need for further investigation into the mechanisms of altered work of breathing under general anesthesia.

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