THE RESPIRATORY EFFECTS OF RESISTANCE TO BREATHING IN ANESTHETIZED MAN

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IN conscious man, moderate resistance to breathing usually results in a decrease of respiratory minute volume.[1-4] This is largely due to a reduction in respiratory frequency which outweighs a small increase in tidal volume. It is to be expected that the response to resistance would be modified by general anesthesia. This paper is an attempt to quantify the interference with respiration resulting from known resistances to breathing during surgical anesthesia.

METHODS

Procedure. The studies were carried out during routine surgical procedures under general anesthesia with spontaneous respiration. A continuous spirometric record of ventilation was made while various resistances to respiration were interposed in the gas circuit for periods of 2–7 minutes. These periods were separated by several minutes of unobstructed respiration which served as controls. It was not practicable to study the effect of longer periods of resistance since it would be difficult to interpret changes against the long term fluctuations in minute volume which are commonly seen during anesthesia with spontaneous respiration. Short term fluctuations were avoided so far as possible by the use of deep anesthesia and by confining the observations to periods of minimal surgical stimulus.

Patients and Anesthesia. The patients were unselected and included both the elderly and those with moderately severe respiratory disease (table 1). All were given premedication; the drugs given were those of choice of the various anesthetists responsible for the patients' care.

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In every case anesthesia was induced with thiopental (100–500 mg.). The agents used for the maintenance of anesthesia are shown in table 1, and the patients have been considered as being in three groups. The first received nitrous oxide, oxygen and halothane, the second nitrous oxide, oxygen and a relaxant, while the third received only nitrous oxide and oxygen. Intermittent doses of meperidine were given to certain patients (table 1). In all instances, a cuffed tube (33–40 F) was inserted into the trachea by the oral route and usually during relaxation procured with succinylcholine (50–75 mg.).

The Gas Circuit. A circle system was used with CO_2 absorption. A high fresh gas flow rate was used (7 l./minute) to ensure that the inspired gas approximated in composition to the fresh gas supply. This necessitated a leak through a relief valve (fig. 1). The circuit incorporated a continuous flow spirometer which was, in effect, a box-bag system with continuous replenishment of the gas in the bag and aspiration at a corresponding rate from the box. The aspiration flow rate was under manual control and was adjusted to keep the kymograph trace level. Under these conditions the mean of inspiratory and expiratory tidal volume was indicated with an accuracy of better than 2 per cent, below respiratory frequencies of 35/minute and up to fresh gas flow rates of 10 l./minute.[5] Changes in the level of the trace could be due, not only to changes in the end-expiratory lung volume but also to changes in the respiratory exchange ratio, the net anesthetic gas exchange, the fresh gas flow rate and even the voltage motivating the aspiration pump. It was therefore generally impossible to interpret gradual changes in the level of the trace. However, an abrupt change in level was usually indicative of a change in the end-expiratory lung volume.

The Resistors. Three forms of resistance to breathing were studied. The first was ob-

TABLE 1

DETAILS OF PATIENTS STUDIED

Serial Number	Sex	Age, Years	Medical Condition	Operation	Position on Table	Premedication (mg.)		Anesthetic Agents Used for Maintenance
1	F	28	normal	vaginal repair	lithotomy	papaveretium hyoscine	20 0.4	nitrous oxide halothane meperidine
2	M	64	normal	cystoscopy	supine	promethazine meperidine atropine	50 100 0.6	nitrous oxide halothane
3	F	79	normal	mastectomy	supine	promethazine meperidine	37.5 75	nitrous oxide meperidine
4	F	73	normal	urethrotomy	lithotomy	meperidine	100	nitrous oxide meperidine
5	M	54	normal	herniorrhaphy	supine	papaveretium hyoscine	20 0.4	nitrous oxide meperidine gallamine
6	M	57	emphysema	herniorrhaphy	supine	meperidine atropine promethazine	100 0.6 50	nitrous oxide halothane
7	M	33	normal	herniorrhaphy	supine	meperidine atropine	100 0.6	nitrous oxide meperidine *d*-tubocurarine
8	F	51	normal	mastectomy	supine	meperidine atropine	100 0.6	nitrous oxide halothane
9	F	72	normal	mastectomy	supine	meperidine atropine	100 0.6	nitrous oxide halothane
10	M	48	normal	herniorrhaphy	supine	meperidine promethazine atropine	100 50 0.6	nitrous oxide halothane
11	M	36	normal	herniorrhaphy	supine	meperidine promethazine	100 50	nitrous oxide halothane
12	M	51	normal	herniorrhaphy	supine	meperidine promethazine	100 50	nitrous oxide halothane
13	M	63	normal	herniorrhaphy	supine	atropine promethazine	0.6 50	nitrous oxide halothane
14	F	45	normal	insertion of radium	lithotomy	meperidine atropine	100 0.4	nitrous oxide halothane
15	M	70	atheroma	amputation	supine	meperidine atropine	100 0.6	nitrous oxide halothane
16	M	67	emphysema	herniorrhaphy and orchidectomy	supine	atropine	0.6	nitrous oxide meperidine *d*-tubocurarine

TABLE 1 (*Continued*)

Serial Number	Sex	Age, Years	Medical Condition	Operation	Position on Table	Premedication (mg.)		Anesthetic Agents Used for Maintenance
17	M	33	normal	herniorrhaphy	supine	meperidine promethazine atropine	100 50 0.6	nitrous oxide *d*-tubocurarine meperidine
18	F	78	normal	biopsy of gland in neck	supine	meperidine promethazine atropine	50 25 0.6	nitrous oxide gallamine
19	M	63	normal	herniorrhaphy	supine	papaveretium hyoscine	20 0.4	nitrous oxide meperidine gallamine
20	F	65	normal	cystoscopy and insertion of radium	lithotomy	meperidine atropine	100 0.6	nitrous oxide gallamine meperidine
21	F	27	normal	appendicectomy	supine	meperidine atropine	100 0.6	nitrous oxide halothane
22	M	32	normal		supine	meperidine atropine	100 0.6	nitrous oxide halothane
23	M	50	normal	cystoscopy	supine	meperidine atropine	100 0.6	nitrous oxide halothane meperidine
24	F	60	secondary carcinoma of chest	radium to cervix	supine	meperidine atropine	100 0.6	nitrous oxide halothane
25	F	52	diabetic	radium to cervix	supine	papaveretium atropine	20 0.6	nitrous oxide halothane
26	F	50	hypertension	radium to cervix	supine	papaveretium atropine	20 0.6	nitrous oxide halothane
27	F	79	diabetic	radium to cervix	supine	meperidine atropine	100 0.6	nitrous oxide halothane
28	F	14	normal	appendicectomy	supine	meperidine promethazine atropine	50 50 0.6	nitrous oxide halothane

tained by passing the respired gas through an adjustable depth of water (fig. 1). Gas only flowed when the pressure difference equaled or was in excess of the depth of water; such a device may be termed a threshold resistor. This resistor caused a reduction in pressure which was, to a first approximation, independent of gas flow rate and equal to the depth of water. Threshold resistors were incorporated on both inspiratory and expiratory sides of the circuit and each could be separately bypassed by quick action wide bore taps. The maximum reduction in pressure obtainable was 17 cm. water in each threshold resistor.

The second type of resistor consisted of a short length of narrow bore tubing placed between the patient and the unidirectional valve box, so that it should be operative during both inspiration and expiration (fig. 1). As it was intended to simulate an obstructed airway, it was designed for turbulent

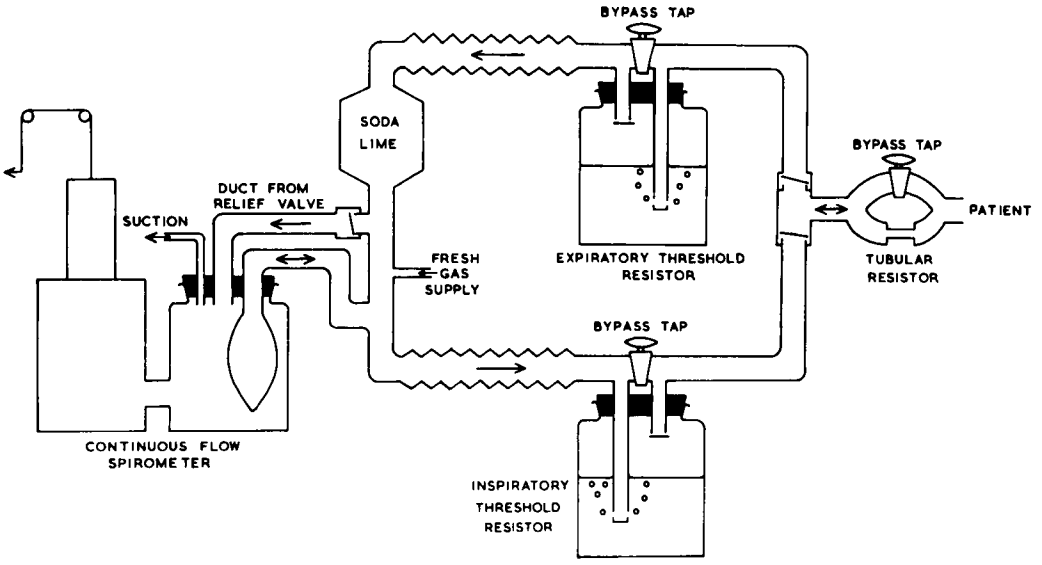

FIG. 1. The gas circuit, consisting of a circle system with leak, incorporating a continuous flow spirometer for recording the ventilation. The three resistors were each provided with a bypass of negligible resistance.

gas flow. Two tubular resistors with the following dimensions were made:

Tubular resistor *A:*

diameter, 4.5 mm., length, 25 mm., equivalent resistance −24 F endotracheal tube.

Tubular resistor *B:*

diameter, 3.0 mm., length, 25 mm., equivalent resistance −16 F endotracheal tube.

When in excess of 2.5 l./minute, gas flow through these resistors was largely turbulent

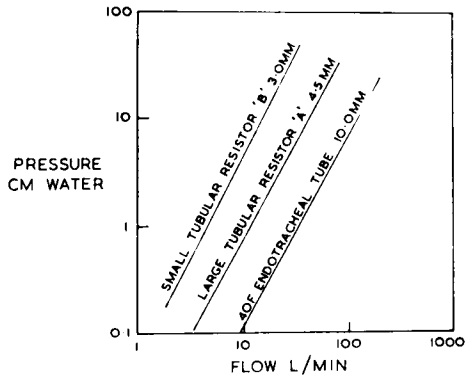

FIG. 2. Pressure/flow rate characteristics of the two tubular resistors displayed on logarithmic coordinates and compared with a 40 F endotracheal tube. The larger tubular resistor *A* corresponded to a 24 F and the smaller resistor *B* to a 16 F endotracheal tube.

(fig. 2)—the value of n being 1.8 for *A* and 1.9 for *B*. ($n = \log \triangle P/\log \triangle$ flow: $n = 1$ for wholly laminar flow: $n = 2$ for wholly turbulent flow.)

The third form of resistance consisted of external interference with respiratory movements. Weighed sandbags (1–20 kg.) were placed upon the sternum or the epigastrium of the patient who was supine or in the lithotomy position. This was intended to simulate the surgical assistant leaning upon the patient or instruments being piled upon the thoracic cage. This part of the study was confined to patients 21–28 all of whom were anesthetized with halothane.

RESULTS

Respiratory Frequency. With all types of resistance studied, the respiratory frequency was not significantly changed provided that the resistance was not sufficient to abolish respiration completely. Out of a total of 106 applications of resistance to gas flow, on 99 occasions, the frequency changed by less than ±10 per cent, on 4 occasions by −11 to −20 per cent and on 3 occasions by +11 to +20 per cent. The scatter appeared random and of the same order as the normal background

TABLE 2

FAILURE OF INSPIRATION OR EXPIRATION FOR THREE OR MORE BREATHS AFTER INTRODUCTION OF RESISTANCE TO BREATHING

Expiratory resistance (threshold), cm. HOH	no failure	failure
+2- +6	15	0
+7- +9	9	3
+10–+16	1	13

Inspiratory resistance (threshold), cm. HOH	no failure	failure
−2- −6	10	0
−7- −9	2	2
−10–−17	1	12

Expiratory plus inspiratory resistance (threshold), cm. HOH	no failure	failure
(+2-2)–(+4-4)	5	1
(+6-6)–(+13-13)	0	17

fluctuations in respiratory frequency during anesthesia.

Since there were no significant changes in respiratory frequency, the changes in ventilation are reported as changes in tidal volume. Changes in minute volume are thus approximately in proportion to changes in tidal volume.

Expiratory Threshold Resistance. Failure of expiration (expiratory apnoea), lasting for three or more breaths, occurred with the higher, but not with the lower, threshold resistors (table 2). All but four patients resumed expiration within 40 seconds.

The most striking finding was an immediate increase in the *inspiratory* tension developed (fig. 3). This occurred in the breath following the application of the expiratory resistance. It resulted in an increase in the end-expiratory lung volume until Δ volume/Δ pressure lay within the range 25–70 ml./cm. water (fig. 4).

In the face of resistance, the tidal volume was initially greatly diminished but improved within the next 90 seconds. When there was initial expiratory apnoea, compensation reached its maximum 90 seconds after the resumption of expiration (fig. 5). When the resistance was bypassed, there was considerable initial hyperventilation, which usually subsided within 90 seconds.

Since the compensatory processes appeared to be complete within 90 seconds, the mean tidal volume was measured between 2 and 3 minutes after the resumption of respiration. The resultant reduction in ventilation was then expressed as a percentage of the mean of the control tidal volume before and after the period of resistance (fig. 6). The control measurements were made immediately before the application of the resistance and two minutes after it had been removed. When expiration was not resumed within 45 seconds of imposing the resistance, it was assumed that apnoea was permanent and the bypass was opened. However, it is probable that in many instances respiration would have returned had sufficient time been allowed.

The results (fig. 6) showed considerable variation, not only between different patients, but also in the same patient at different stages of the anesthesia. However, under stable conditions, it was possible to demonstrate a linear relationship between respiratory depression and the expiratory threshold pressure. Observations on patients 5 and 8 are shown in figure 6. It was surprising to find that some patients could achieve satisfactory compensation against pressures as high as 12 cm. of water. However, of greater significance was the maximum possible reduction of ventilation and this is indicated in figure 6 by the broken line.

Although we were not primarily concerned with circulatory changes, it was found that the blood pressure (systolic) was never reduced by more than 10 mm. Hg. during the periods of expiratory resistance. Furthermore, there was no obvious engorgement of the peripheral veins and, even in response to direct questioning, the surgeons would admit no change in the calibre of the axillary of other exposed veins. Capillary oozing from cut surfaces was apparently uninfluenced.

Inspiratory Threshold Resistance. Transient inspiratory apnoea was caused by a level of inspiratory pressure similar to the expiratory pressure required to produce transient expiratory apnoea (table 2). However, inspiratory apnoea was invariably accompanied by expiratory apnoea and there was thus no marked

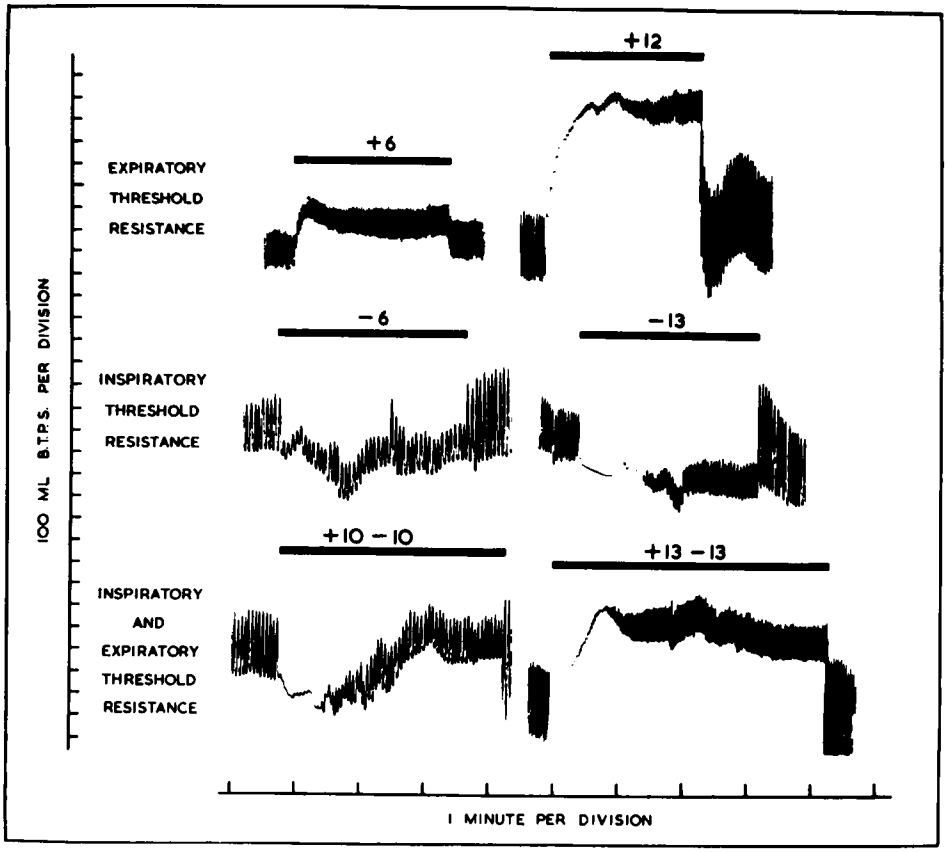

FIG. 3. Typical spirograms of patients exposed to various forms of threshold resistance to breathing. The black bars indicate the periods during which resistance was present and the figures show the threshold pressure in cm. water.

change in end-expiratory lung volume as with expiratory resistance (fig. 3). There was actually a small decrease in lung volume (mean 50 ml.) which was independent of the inspiratory threshold pressure (fig. 4). Two patients changed their lung volume in an atypical manner. Figure 4 shows one whose lung volume decreased 200 ml. while a second (not shown) reduced his lung volume by 400 ml.—both in the face of an inspiratory threshold pressure of 4 cm. of water.

Inspiratory resistance caused an immediate increase in the developed inspiratory tension—in this respect being similar to expiratory resistance. The reduction in ventilation was always less than would be accounted for by

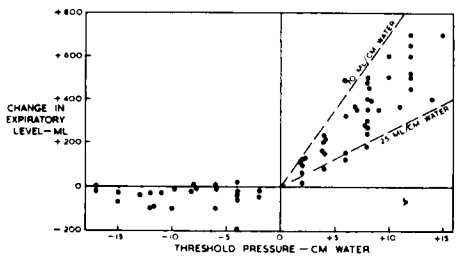

FIG. 4. Changes in the expiratory level (end-expiratory lung volume) plotted against threshold pressures of inspiration (—) and expiration (+).

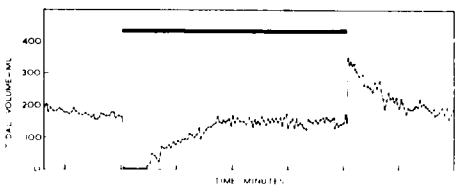

FIG. 5. The volume of individual breaths plotted against time, before during and after the application of a threshold resistance of 8 cm. water to breathing (indicated by the black bar).

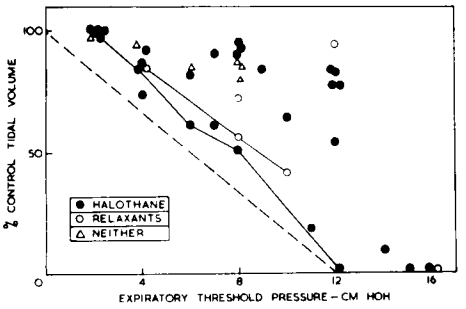

FIG. 6. Depression of ventilation caused by various levels of expiratory threshold resistance. The continuous lines indicate serial observations on two patients (5 and 8). The broken line indicates the maximum depression of respiration observed.

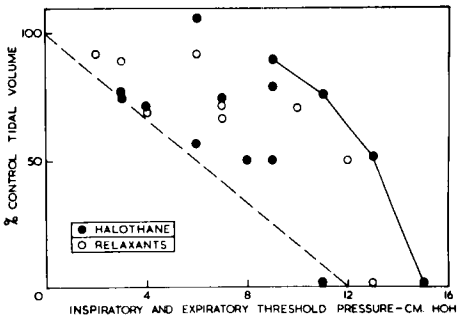

FIG. 8. Depression of ventilation caused by various levels of inspiratory and expiratory resistance. The continuous line indicates serial observations on patient number 15. The broken line indicates the maximum depression of respiration observed (as in figs. 6 and 7).

the decline in pressure across the inspiratory resistor had the end inspiratory tension been unaltered. Apart from this initial response, further improvement occurred over a period of 90 seconds as was seen with expiratory resistance. Transient hyperventilation was seen on bypassing the resistor.

Reduction in ventilation was calculated as for expiratory resistance and plotted against inspiratory threshold pressure (fig. 7). Certain patients showed an astonishing ability to compensate. Patient 15 (age 70) inspired through 11 cm. of water with 9 per cent reduction in ventilation while patient 13 (age 63) inspired through 17 cm. of water with only 14 per cent reduction. Depression was more marked with the relaxants than with halothane. In spite of individual variation, the maximum observed depression in ventilation corresponded fairly well with that found during expiratory obstruction.

No obvious circulatory changes were observed during inspiratory obstruction.

Combined Inspiratory and Expiratory Threshold Resistance. Initial apnoea (inspiratory or expiratory) was caused by a lower reduction in pressure than with the single resistances (table 2). Thereafter the patients varied in their response. In some the inspiratory resistance appeared dominant and the spirogram resembled that of a pure inspiratory resistance (fig. 3, bottom left).

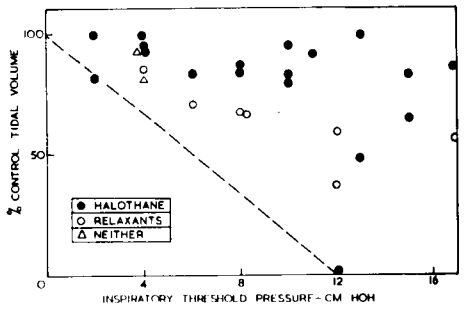

FIG. 7. Depression of ventilation caused by various levels of inspiratory threshold resistance. The broken line indicates the maximum depression of respiration observed (as in fig. 6).

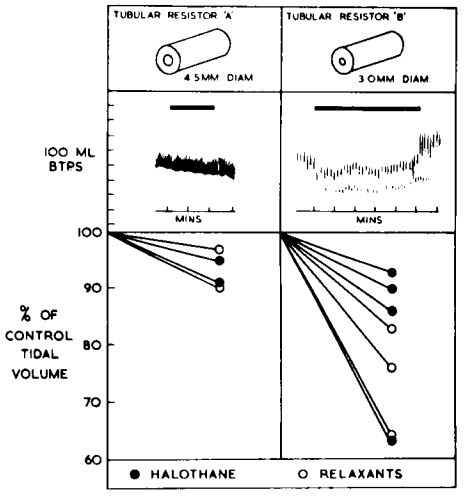

FIG. 9. Spirograms and histograms of depression of ventilation caused by tubular resistances to breathing.

TABLE 3

RESPIRATORY EFFECTS OF EXTERNAL INTERFERENCE WITH RESPIRATORY MOVEMENTS

	Change in Lung Volume, ml. (BTPS)	Change in Tidal Volume, Per Cent	Change in Respiratory Frequency, Per Cent	Change in Minute Volume Per Cent
5 Kg. on sternum (mean of 9 applications)	−70	−4.1	+2.8	−1.7
5 Kg. on epigastrium (mean of 8 applications)	−45	−19.2	+2.0	−17.8

However, in the majority the inspiratory resistance was overcome before the expiratory and consequently the lung volume increased (fig. 3, bottom right).

When respiration was reestablished, improvement occurred during the first 90 seconds and post-obstruction hyperventilation was seen. The respiratory depression was rather greater than that seen with single resistors (fig. 8) although the line of maximum observed depression from figures 6 and 7 is not greatly exceeded. Patient 15 again displayed considerable ability to overcome resistance in spite of his age (joined points, fig. 8).

Tubular Resistance. These resistors caused no change in end-expiratory lung volume and initial apnoea was never seen (fig. 9). There was slight improvement in ventilation during the first 90 seconds of exposure but this phenomenon was not so marked as with the threshold resistors. Post-obstruction hyperventilation was seen in proportion to the degree of depression produced. This was in contrast to the removal of threshold resistors when hyperventilation occurred even if there had been no significant depression of ventilation.

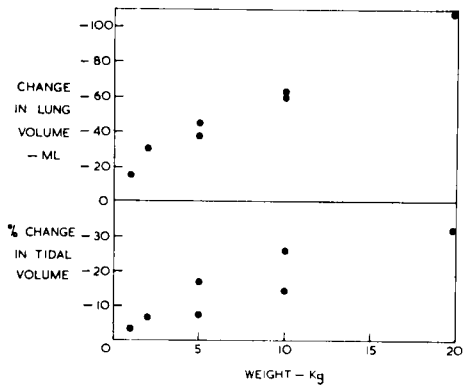

FIG. 10. Changes caused by placing weights on the epigastrium.

The mean reduction of ventilation was 7 per cent with resistor *A* and 21 percent with resistor *B*. This might be predicted from the observations made with the threshold resistors. The mean reduction in pressure across the tubular resistors could be derived from figure 2 on the assumption that the mean inspiratory or expiratory gas flow rate was of the order of three times the minute volume. For resistor *B* this would indicate a mean pressure decrease of some 10 cm. of water—which did in fact cause a comparable depression of ventilation (fig. 8).

External Interference with Respiratory Movements. The presence of a 5 kg. sandbag on the sternum caused no significant change in ventilation although the end-expiratory lung volume showed a mean reduction of 70 ml. (table 3). A single application of 10 kg. to the sternum of patient 28 caused a 6 per cent increase in ventilation. In contrast, a 5 kg. sandbag on the epigastrium caused a highly significant decrease in ventilation (table 3). Application of a series of bags to patient 28 showed that the depression of ventilation and change of lung volume were related to the weight (fig. 10). There was no evidence of improvement of ventilation when weights remained on the epigastrium, neither was there any evidence of post-obstructive hyperventilation. This was in marked contrast to the changes seen when breathing through a threshold resistor.

Discussion

Clinical. The effect of a threshold resistance on minute volume was found to be extremely variable and furthermore it did not appear possible to predict the type of patient who could most easily overcome it. However, the maximum depression encountered was sharply defined as follows: Fractional Depression of Ventilation = Threshold Pressure (cm. water)/12.

In the present study this degree of depression was narrowly exceeded only twice in 83 exposures. On the other hand, considerably less depression was common. Neverthless, when formulating specifications for gas circuits, it would be wise to use an expression indicative of the maximum depression which might result from airway resistance.

Undirectional and pressure relief valves, if well designed, have the characteristics of threshold resistors. During anesthesia with spontaneous respiration the maximum respiratory peak flow is of the order of 35 l./minute which, with a well designed valve, should not cause a pressure drop of more than 0.5 cm. water. However, some types of valves in general use cause a pressure drop of 2 cm. water at 35 l./minute [6]—sufficient to cause a 17 per cent reduction in ventilation.

The tubular resistors represented sources of turbulent flow such as kinked or blocked endotracheal tubes and those which were too small for the patient. Here, the effect was less than might be expected. Tubular resistor *A* had negligible effect upon ventilation and yet corresponded to a size of endotracheal tube (24 F) which would never be considered for use in an adult. Tubular resistor *B* corresponded to a 16 F endotracheal tube which might well be regarded as incompatible with life in an adult and yet the effect was little worse than that of many relief valves. This is in accord with observations on the degree of respiratory obstruction which may be tolerated by conscious subjects. In diphtheria, carcinoma of the larynx and sub-glottic stenosis the effective lumen of the upper respiratory tract may be reduced to that of a 24 F endotracheal tube without symptoms—*provided that the patient lies quietly in bed and remains calm.* Fear initiates a vicious circle of increased oxygen consumption and relative under-ventilation. Fortunately, during anesthesia, conditions are favorable for tolerating a narrowed airway. The oxygen consumption and minute volume are reduced and panic is impossible. Therefore peak flow rates are kept low and the effect of a flow dependent resistor is minimized. It is of interest that in 1950 Beecher [7] showed that a free lying endotracheal tube of size smaller than usual appeared satisfactory. A 32 F tube, for example, caused no rise in arterial P_{CO_2} when substituted for a larger tube.

During artificial ventilation, peak flow rates are much higher—often as high as 100 l./minute. Flow dependent resistors will therefore cause a large reduction in pressure although this may be of little importance.

There would appear to be no grounds for the prevalent belief that moderate pressure on the chest is harmful during anesthesia. This finding is not really surprising. Not only is respiration largely diaphragmatic during anesthesia, but the ratio $\triangle$ strain/$\triangle$ stress of a spring is not changed by a standing load provided that the elastic limit is not exceeded. On the other hand, weight on the epigastrium is clearly undersirable and this is probably true of liver retraction and some forms of insertion of abdominal packs—as during cholecystectomy. The reduction in ventilation from these sources might be considered an additional indication for artificial ventilation during upper abdominal surgery.

It is difficult to compare our results with those obtained on conscious subjects. Not only have laminar resistors been used in previous studies, but also the reaction of the conscious subject is variable. In general, depression of minute volume appears to be somewhat greater in the anesthetized than in the conscious subject.[2, 3, 4] However, this may merely reflect a greater increase in oxygen consumption during consciousness and the change in ventilation equivalent may not differ greatly in the two circumstances.

In this work no attempt has been made to assess the long term effect of resistance to breathing. It is possible that fatigue of the respiratory muscles may occur and result in a serious reduction in minute volume as it does in the conscious subject.[2] Demon-

stration of this would be difficult as it would not appear possible to distinguish the onset of fatigue from the natural long term fluctuations in ventilation which occur during anesthesia with spontaneous respiration.

Although, in this work, we were not primarily concerned with circulatory phenomena, there was a striking absence of changes which might be expected to result from resistance to breathing. It would appear inevitable that expiratory resistance would increase the mean central venous pressure but this was not detected by inspection of peripheral veins or those exposed by the surgeon. Even in response to direct questioning the surgeons would admit no change in venous engorgement or in capillary oozing from cut surfaces. The maintenance of the arterial blood pressure was less surprising since Maloney *et al.*[8] have already demonstrated the ability of a fit patient to compensate for impeded venous return.

Work of Breathing. Assuming an average minute volume of 4 l./minute during anesthesia with spontaneous respiration, it may be shown that there will be an energy expenditure of 0.0001 Cal./minute/centimeter of water pressure drop in overcoming a single threshold resistor. The normal work of breathing is of the order of 0.001 Cal./minute, which, with an efficiency of 7 per cent, requires some 4 ml. oxygen per minute. Assuming that the efficiency of the respiratory muscles is reduced to 3 per cent by the various tasks imposed upon them,[3] the resistances we have used would have caused only a slight increase in carbon dioxide output (up to 10 per cent of total) and the resultant arterial P_{CO_2} would still be largely determined by the ventilation achieved in the presence of the resistor.

Mechanisms of Compensation. It is of interest to consider the explanation of the compensations which appear to be made. On the imposition of inspiratory and expiratory threshold resistors, the tension developed by the inspiratory muscles is augmented within a single respiratory cycle. Clearly this is due to a reflex modulation of respiratory muscle tension rather than to changes in blood gas tensions. Altered intra-thoracic blood volume would not appear to play a major role, as inspiratory and expiratory resistances must have opposite effects in this respect although the overall effect on minute volume is very similar.

The augmentation of inspiratory effort with both inspiratory and expiratory resistors cannot be explained by the classical theories of reflex control of breathing. What follows constitutes a satisfactory explanation although it cannot, at the present time, be regarded as proven. It is nevertheless consistent with current theories on the action of the muscle spindles (Campbell, E. J. M., Dickinson, C. J., and Howell, J. B. L., personal communication). In support, there is evidence that conscious man is able to detect small changes in the length/tension relationship of the inspiratory muscles.[9]

It is known that the respiratory muscles, being striated, contain spindles and these receptors are supplied with both afferent and efferent (gamma) nerve fibres.[10, 11] In the presence of inspiratory resistance, the development of a given tension by the inspiratory muscles causes less than the normal degree of shortening of the spindle. Nevertheless the intrafusal fibres of the spindle (driven by gamma motor neurone discharge) contract and thereby cause increased discharge from the annulo-spiral endings. This augmented annulo-spiral activity reflexly increases the background excitation of the motor neurone pools of the inspiratory muscles. Thus, when the normal length/tension relationship of the inspiratory muscles is altered by resistance, the inspiratory effort is augmented so that, as far as possible, the tidal volume is maintained at the control value. This is closely analagous to a servo system which develops additional power to perform its allotted task.

Since expiratory muscle activity is not normally used to overcome moderate inspiratory resistance, no substantial decrease in functional residual capacity is to be expected. The small decrease shown in figure 4 suggests that there is still slight inspiratory muscle tone at the normal resting end-expiratory level, (Howell, J. B. L., personal communication). Two patients in this study appeared to develop expiratory muscle activity but this cannot be regarded as typical.

The response to expiratory threshold resistance is more complicated. Exhalation against resistance causes temporary diminution of the muscle spindle discharge since the intrafusal fibres relax while the inspiratory muscles as a whole are unable to elongate. However, within 10–20 milliseconds the intrafusal fibres contract restoring the tension within the spindle. The next inspiratory effort is associated with a further increase in tension within the spindle, and the augmented annulo-spiral activity again increases the background excitation of the motor neurone pools, resulting in the development of greater tension in the inspiratory muscles. Thus, after the failure (or relative failure) of an expiration the following inspiration carries the lung volume to a higher level at which the elastic recoil is sufficient to overcome the expiratory resistance. Expiratory resistance was, in fact, shown by Campbell, Howell and Peckett[12] to be a valid method for the measurement of compliance. During the present study, the surgeons failed to detect any increase in expiratory muscle activity during the periods of expiratory obstruction. This is in accord with the conclusions of Campbell[13] who found that active expiration was not required to overcome expiratory pressures up to 10 cm. water.

The servo mechanism based on the spindles is less easy to demonstrate in the presence of other forms of resistance. Nevertheless, it is reasonable to assume that it operates since the respiratory depression found with the tubular resistors was in accord with that found with the threshold resistors. This mechanism is probably an important safeguard of the anesthetized patient who may be exposed to respiratory resistance. Since the myoneural junctions of the intrafusal fibres of the muscle spindles are affected by relaxants, it would appear that, in high dosage, these agents are particularly dangerous in the presence of resistance.

The second compensation, occurring over the first two minutes of breathing against resistance, is probably caused by elevation of the arterial P_{CO_2}, which is an inevitable consequence of hypoventilation. To a small extent the P_{CO_2} will be further elevated by the increased CO_2 production from the additional work of breathing against resistance. The post-obstruction hyperventilation is likely to be caused by the sudden withdrawal of the additional load in the presence of the augmented reticulo-spinal discharge due to the elevated P_{CO_2}. The P_{CO_2} rapidly returns to the control value and the ventilation then reverts to the level it had attained before the application of the resistance.

Summary

The respitarory effects of resistance to breathing have been studied in 28 patients anesthetized for routine surgery but permitted to breathe spontaneously. Resistances were imposed for periods of 2–7 minutes and ventilation was then compared with the control periods of unobstructed breathing.

The passage of inspired and/or expired gas through a water trap (threshold resistor) produced less than the expected reduction in ventilation since there was an immediate, presumably reflex, augmentation of the developed inspiratory tension. Thereafter there was a further increase in ventilation probably due to CO_2 retention—full compensation being achieved in 2–3 minutes. There was a wide scatter in the ability of patients to compensate. Some patients attained a minute volume close to the control value against as much as 17 cm. water, while the worst response was apnoea at 12 cm. water with proportionate reduction against lower pressures.

A second study was concerned with flow-dependent resistors—two short, narrow tubes which offered the same resistance as 24 F and 16 F endotracheal tubes respectively. The reduction in ventilation was in accord with the results obtained on threshold resistors and amounted to a mean reduction of 7 per cent with the 24 F resistor, and 21 per cent with the 16 F resistor.

Finally a study was made of the effect of external pressure on the sternum or epigastrium. Weights up to 20 kg. on the sternum reduced the functional residual capacity but had no signicant effect upon the ventilation. On the other hand 5 kg. on the epigastrium caused a mean reduction of ventilation of 18 per cent.

It would appear that reflex compensation

for resistance to breathing is well marked during anesthesia and in its absence the effects would be much more pronounced. In the present study, the ability to compensate was not found to be impaired by halothane, small doses of relaxants, old age or early emphysema.

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PREOPERATIVE EVALUATION An attempt has been made to obtain a preoperative evaluation of the chronically ill patient by the use of the tilt-table test. After initial recordings of the blood pressure, pulse and respiration in the supine position, 86 patients were tilted 60 degrees in the head-up position. Vital signs were recorded at one minute intervals for three minutes. Patients were then returned to the supine position. Pneumatic tourniquets were placed around the thighs and inflated to slightly higer than diastolic pressure. The patient was tilted for another three minutes. Twenty-three of the patients demonstrated an abnormal response to this test. Eighteen of the patients were subsequently demonstrated to have significantly reduced blood volumes. Adrenocortical hypofunction was observed in six patients. (*Stanley, T. V., and Watts, R. W.: Preoperative Evaluation of Chronically Ill Surgical Paient by Tilt Table and Adrenal Responses Correlated with Blood Volumes, Surg., Gynec. Obstet. 111: 163 (Aug.) 1960.*)