A NEW RESPIRATOR

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The poliomyelitis epidemics in Minnesota during 1952 and in western Canada during 1953 afforded an opportunity to study numerous patients confined to tank-type respirators. The high mortality and morbidity encountered in these two groups were related in part to the encumbered position of the patients. Further, it was frequently observed that patients who were deteriorating in the tank respirator actually improved when they were removed from the tank and were ventilated by intermittent positive pressure provided by manual compression of the rebreathing bag of an anesthesia machine. In 1953 Lassen of Denmark (1) reported the maintenance of numerous apneic poliomyelitis patients, incorporating the use of manual intermittent positive pressure breathing.

The apparent advantages offered by a "pressure breathing" respirator were numerous. Increased accessibility to the patient would obviate diagnostic, therapeutic, and nursing procedures. The frequent wide range positional changes, so essential in the care of chronically apneic patients, could be accomplished with ease. The incidence of such complications as hypostatic pneumonia, nephrolithiasis, thrombophlebitis, and decubitus ulcers would be curtailed.

Developmental Requirements

Before undertaking the development of a respirator capable of optimal ventilation of apneic patients without producing undesirable physiologic side effects, a comprehension of the normal mechanics of pulmonary ventilation was essential. Upon this background one could outline the basic requirements of a mechanical ventilator, and the most physiologic methods of achieving these requirements. Where normal physiology of necessity was altered, methods capable of minimizing or counteracting these alterations could be developed.

J. H. Matthews, M.D., S. J. Cedarleaf, M.D., and C. W. Field, M.D., also, are co-authors of this paper. Their names were omitted above because of limitation of space.

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First Requirement. To provide normal alveolar ventilation.— Physiologic alveolar ventilation maintains continuously normal tensions of arterial oxygen and carbon dioxide. Since it has been demonstrated that in the normal lung the concentration of oxygen in arterial blood is adequate when breathing air or gas mixtures with oxygen tensions of 100 mm. of Hg or greater, ventilation should be regulated according to the amount of carbon dioxide to be removed (2).

The effect of abnormal concentrations of carbon dioxide on acid-base and electrolyte balance with accompanying alterations in both the cardiovascular and central nervous systems dictates the accurate regulation of the tension of this gas (3—8). To effect transfer of carbon dioxide from blood to ambient air the lung volume must be varied with rhythmic precision. Expressed in practical and measurable terms, *the optimal clearance of carbon dioxide is dependent on the minute volume of alveolar ventilation.*

The required minute ventilatory volume may be calculated for individuals on the basis of their metabolic demands. To this extent Radford (9) has constructed a most useful nomogram for the selection of both tidal volume and rate. Since alveolar ventilation regulates the P_ACO_2, the adequacy of such ventilation may be determined by analysis of total arterial blood concentration of carbon dioxide, and *p*H. Based on these determinations the tension of carbon dioxide may be calculated. Indirect monitoring of the end alveolar carbon dioxide tension is also possible (10, 11). The alveolar values closely parallel those of the blood (10), provided equal aeration of all segments of the lung and normal pulmonary blood flow exists (12).

In man normal ventilation occurs by an active inspiratory phase and a passive expiratory phase. Respiratory excursions starting at the resting expiratory level and proceeding into the *inspiratory capacity* compartment offer the following advantages over those which proceed into the *expiratory reserve volume compartment* (13): (1) the lungs and thorax enlarge in all directions resulting in a greater mean lung volume over the respiratory cycle favoring a more equal distribution of alveolar ventilation, (2) the greater mean lung volumes produced by positive pressure are accompanied by increases in the mean caliber of the conducting airway, (3) an increased mean caliber of the airway results in a decreased resistance to gas flow, especially significant when high flow rates are utilized, and (4) with a higher mean caliber of the conducting airways the critical closing pressures of the bronchioles are avoided. These factors favor the supposition that pulmonary ventilation is most efficient when the inspiratory phase is active and its excursion takes place within the *inspiratory capacity* compartment of the lungs.

Second Requirement. To achieve a precision minute volume.— Since the efficiency of artificial ventilation depends to the greatest extent on the volume of respiratory gas moved with each respiratory

excursion, it is essential that each tidal volume enter the lungs in its entirety, regardless of opposing forces.

Volume-pressure relationships: Expansion of the lungs and chest cavity is accomplished only through the initiation of a pressure differential between the intrapulmonary compartments and the atmosphere surrounding the body. This pressure differential is necessary to overcome several resistances, namely: (a) the resistance to the flow of gas through small tubes, (b) the frictional resistance between tissue elements, and (c) the resistance due to the elasticity of the lungs and chest wall, commonly referred to as "pulmonary compliance" (14).

The major part of the force required for inspiration in normal individuals is spent overcoming the elastic resistance of the lungs and chest cage. High pulmonary compliance is normal, and relatively small pressure differentials are required for lung inflation. In some pulmonary conditions (pulmonary edema, fibrosis) compliance is lost and volume changes per unit of static pressure change are diminished. It is evident that ventilation is severely handicapped in the presence of decreased compliance and increased resistance. Despite this handicap, it remains essential that the required minute volume be delivered, regardless of the necessity for an increased pressure differential. To summarize, *minute ventilatory volumes must remain constant while pressure differentials are permitted to vary accordingly. Pressure differentials should never remain constant while minute volumes are allowed to vary.*

Third Requirement. To supply the optimum volume-pressure relationships needed for adequate ventilation without disturbing cardiovascular function.—During normal inspiration the intrapleural pressure decreases, resulting in an elevation of the pressure gradient between the intra- and extrathoracic veins. An accelerated venous return to the right heart ensues, effecting an increased stroke volume output of the heart and an elevation of the arterial blood pressure (15). During expiration the reversal of these events occurs. Thus normal ventilation is responsible for the rhythmic variations seen in stroke volume output and arterial blood pressure.

Should the negativity of the intrapleural pressure during inspiration be decreased or lost, as is the case with "pressure breathing" respirators, the aforementioned circulatory dynamics are reversed, venous flow decreasing during inspiration and increasing during expiration. For a few cardiac cycles the stroke volume output will fall; however, reflex homeostatic mechanisms, an increase in peripheral venous pressure, and a change in circulating blood volume probably account for the rapid restoration to normal cardiovascular dynamics (16). There is little evidence to support the view that grossly unfavorable changes occur in the circulatory systems of normal individuals during "pressure breathing." With the exception of the loss of intrapleural pressure during the active respiratory phase, the intra-

pulmonic-atmospheric pressure differential remains unchanged (17).

The foregoing relationships between respiration and circulation may be grossly altered by changes in the physiology of either or both. Oligemic and normovolemic shock, excessive increases in respiratory resistance, or combinations of these factors may produce a sustained depression of the circulatory function during "pressure breathing." The degree of circulatory depression under abnormal conditions will depend upon the decrease in pulmonary compliance, the degree of circulatory shock, and the mean intrapulmonic pressure (18).

Mean intrapulmonic pressure varies with the integrated pressure curve which is dependent upon: (1) peak inspiratory pressure attained, (2) duration of inspiratory pressure, (3) shape of the inspiratory pressure curve, (4) minimum expiratory pressure attained, (5) duration of the expiratory pressure, (6) shape of the expiratory pressure curve, and (7) the pressure and similar component characteristics of a negative-pressure phase.

Fourth Requirement. To provide safely the normal ventilatory volume-pressure variations.—The most common hazard of artificial ventilation is over- or under-ventilation. This does not occur in precision or "fixed" volume respirators, but is a common failing in pressure-regulated machines.

The safe application of pressure to distend the lungs and thorax is of considerable importance. The extent to which the intra-alveolar pressure is increased will depend upon the peak, shape, and duration of the inspiratory pressure curve of the respirator, as well as the resistance and compliance of the thoracic cage and lungs. Because of the resistance to high flow rates offered by the small bronchioles, the intra-alveolar pressure will be lower than the tracheal pressure (13), provided inspiration is not excessively slow or static. A semirigid chest wall also tends to protect against alveolar rupture on a lung subjected to high intrapulmonic pressures. There is little evidence in the recent literature to suggest changing the figure of 30 mm. of Hg as the effective pressure differential required to rupture alveolar membranes (13).

In the presence of pulmonary disease high resistance and low compliance impede gas flow. Under these conditions a greater inspiratory pressure is needed to deliver an equivalent volume. When the pulmonary involvement is uniform, the higher pressure is unlikely to produce alveolar rupture. However, if pulmonary disease is nonuniform, unequal pressure distribution occurs; some alveoli will be subjected to pressures in excess of others, which may lead to alveolar rupture.

Subatmospheric intra-alveolar pressures also may be hazardous In addition to producing hysteresis which results in an unequal distribution of ventilation, resistance to flow is increased. Loss of com-

pliance occurs as expiration proceeds into the *expiratory reserve volume* compartment and diminishes ventilatory efficiency (13).

The effect of prolonged and severe negative intrapulmonic pressures upon circulatory dynamics has been outlined by Brecher (15). Extrathoracic veins pass rapidly from the "stage of depletion" to the "stage of collapse." During the "stage of collapse" venous return ceases and stroke volume output drops precipitously.

Developmental Solutions

Based upon the preceding physiologic principles of ventilation the solutions to the four basic requirements for a "pressure breathing" respirator were sought.

First Solution. Normal alveolar ventilation may be attained only through accurate independent control of tidal volume and rate.—To

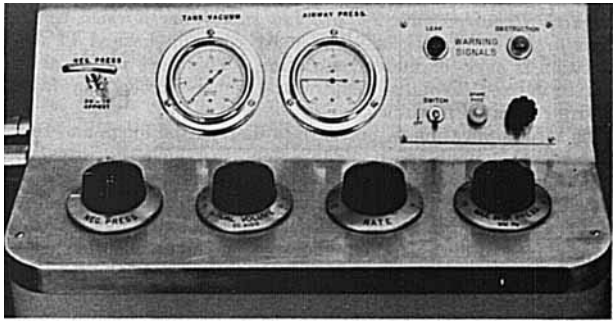

Fig. 1. Control panel of respirator depicting individual selector knobs, pressure gages and warning signals.

this end a delivery reservoir with a capacity which could be regulated with accuracy (plus or minus 15 cc.) to produce any desired tidal volume within reason (150–1,400 cc.) appeared ideal. The rate of respiration also had to be controllable and independent of volume-pressure relationships (fig. 1).

The above criteria were met by adoption of a motor driven, gas tight piston and cylinder unit (fig. 2). Tidal volume control was achieved by adjustment of the cylinder capacity through regulation of the piston stroke. Rate control was effected by interposing a variable speed transmission between the motor and the piston drive unit. To achieve the most efficient alveolar ventilation, the excursion of the piston was designed to move air into the *inspiratory capacity* compartment starting at the end expiratory level of respiration.

Second Solution. Precision volume control may be achieved by use of a rigid non-distensible delivery system.—Since volume-resistance relationships not only differ among individuals, but also within the same individual, it was essential that additional uncontrolled variables were not introduced by the respirator. As already stated, this was accomplished in part by employment of a rigid piston and cylinder unit. Further, the entire conduit system had to be of a non-distensible nature. This assured that a portion of the calibrated tidal volume would not be utilized in expanding materials of varying elastic properties, which tend to increase the "dead space" by inconstant and unknown amounts.

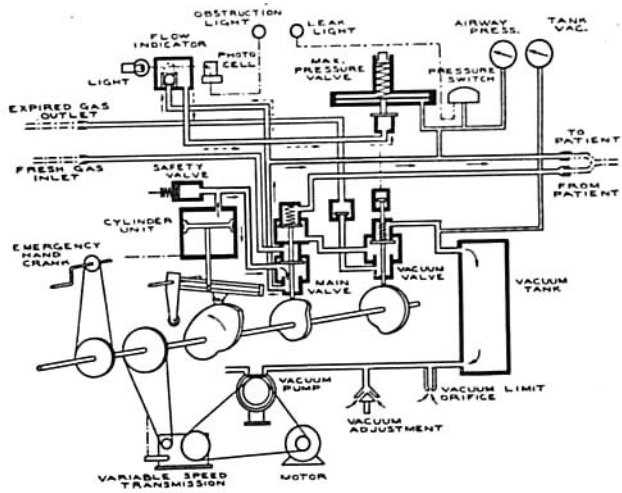

FIG. 2. Schematic diagram of respirator—inspiratory phase.

Volume-resistance relationships within the patient introduced the first variable, pressure. Since it was essential that the volume remain constant, the inspiratory pressure had to be allowed to vary according to the changes encountered in respiratory resistance. There was no easy way of automatically varying the pressure. The simplest means of circumventing this obstacle was to incorporate a "signal" which would be activated by volume spillage from the "inspiratory line" (fig. 2). This signal would indicate that respiratory resistance had increased, and that measures should be taken to either re-establish the original pulmonary resistance, or if this were impossible, to increase the delivery pressure to a point where the spillage would cease. By

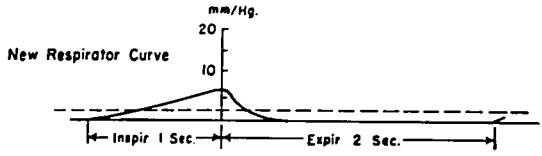

FIG. 3. Time ratio of inspiration to expiration.

this method, minute ventilatory volume could be kept constant while the delivery pressure was varied according to changes in pulmonary resistance.

Third Solution. The production of optimum volume-pressure relationships which would not disturb cardiovascular function was accomplished through the adoption of methods to reduce the mean airway pressure.—Proper phasing of the respiratory cycle: Evaluation of various existing volume-pressure curves and their influence on the mean tracheal pressure led to the design of a more optimal curve than that of Cournand's "Type III" (19). The period of inspiration was decreased and expiration prolonged, achieving an inspiratory-expiratory ratio of one to two (fig. 3).

Configuration of the volume-pressure time curve: The inspiratory segment of this curve was designed to produce a steep acceleration of the rate of pressure increase to a peak, with rapid return to atmospheric pressure. Thus less desirable degrees of pressure would exist for only a minimal time. Rapid return of the peak inspiratory pressure to that of the atmosphere was accomplished by using large

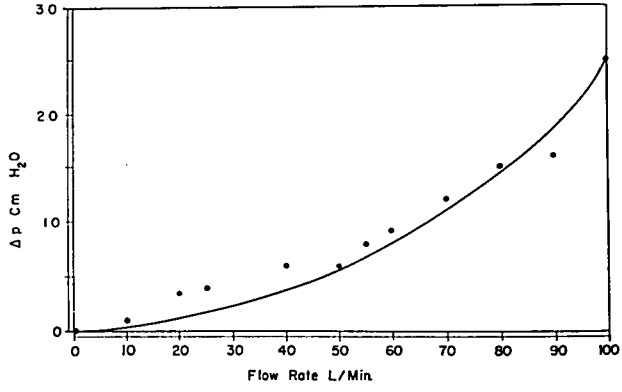

FIG. 4. Expiratory resistance within respirator.

¾-inch diameter conduits and valve orifices in the expiratory system. Thus the respirator would offer minimal resistance to flow during passive expiration (fig. 4). Utilizing this type of pressure curve, the required tidal volume was introduced at a minimal mean tracheal pressure.

To attain this type of respiratory curve, a revolving cam wheel was designed which would produce an accelerating stroke of the piston for one-third of its revolution, and then rotate free of the piston rod for the remaining two-thirds of the cycle.

Utilization of the negative pressure phase: As previously mentioned, the negative-pressure phase frequently is incorporated in impairment of circulatory dynamics. Utilization of a negative-pressure phase was capable of reducing mean-mask pressure to subatmospheric levels. Since the negative-pressure phase was intended to offset the decrease in venous return resulting from a positive intrapulmonic pressure, it appeared most optimal to place this component immediately preceding the positive pressure surge. The resultant effect therefore should be an increase in venous return, producing slight "over-filling" of the heart just prior to the obstructive effect of positive pressure. With the exception of rare instances where pulmonary hysteresis exists following inspiration, the negative phase has little ventilatory value and therefore should be of short duration (one-sixth of the entire cycle) and low amplitude (0 to −10 mm. Hg). To this end, a tank was incorporated which could be evacuated by means of a motor driven vacuum pump. The vacuum within the tank was regulated by an adjustable valve (fig. 1). The negative phase was introduced into the expiratory line at the appropriate time by means of a cam-activated valve.

Fourth Solution. Normal ventilatory volume-pressure excursions may be provided with safety by incorporating pressure-limiting valves and warning devices.—Gross underventilation may be detected through the use of the volume-pressure relationship. This was accomplished by employing a pressure tap from the inspiratory line to a pressure switch (fig. 2). When pressures less than 7 mm. of Hg occur, a red light is activated (fig. 1). This signal indicates a loss in the volume-pressure relation, and therefore warns the operator of the need for checking the adequacy of tidal volume delivery. This system would be activated, for example, if the patient became disconnected from the respirator or a leak developed in the delivery conduits.

The same "take-off" from the inspiratory line also leads to the maximum pressure regulator valve which is sensitive to pressure changes of 0.5 cm. of H_2O (fig. 2). Should an increase in resistance to inspiration develop, thereby causing the intrapulmonic pressure to be increased above the previously set limit, the maximum pressure valve opens, allowing gas to escape through the flow indicator system. This system consists of a cup which is elevated by the escaping gas stream,

thereby interrupting a photoelectric beam and activating an amber light. As previously explained, this is indicative of an increase in the volume-resistance relationship, resulting in a diminution of the tidal volume delivered, which dictates readjustment of the pressure regulating mechanisms or re-establishment of the previous dynamics of gas delivery.

In addition to the above, the following safety features were incorporated. The pressure regulator valve limits the maximum inspira-

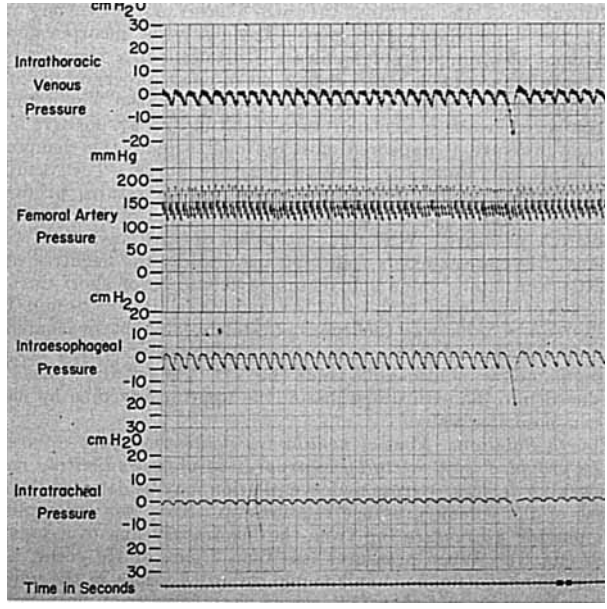

FIG. 5. Hemodynamics—spontaneous respiration (cardiac output 5.64 L/min.).

tory line pressure to 35 mm. of Hg. Negative line pressures in excess of — 12 mm. of Hg cannot be achieved because of a vacuum limiting orifice which was incorporated in the vacuum tank.

In the event of an electric power failure, an emergency hand crank was provided for manual operation.

PRELIMINARY TESTING

Methods.—Ten mongrel dogs were anesthetized with 2.5 per cent Surital® sodium and a cuffed tube was inserted into the trachea. Tidal

volumes were determined by means of a Bennett ventilation meter. Flow rate was calculated by interposing a low resistance impedance orifice in the airway and recording the pressure drop across the orifice with a differential strain gauge. In this technique the square root of this pressure differential was calculated and recorded electronically, and from this data the rate of flow was continuously calculable. The range of accuracy of the flow rates remained linear in a range of 8 to 60 liters per minute. Esophageal pressures were measured with a

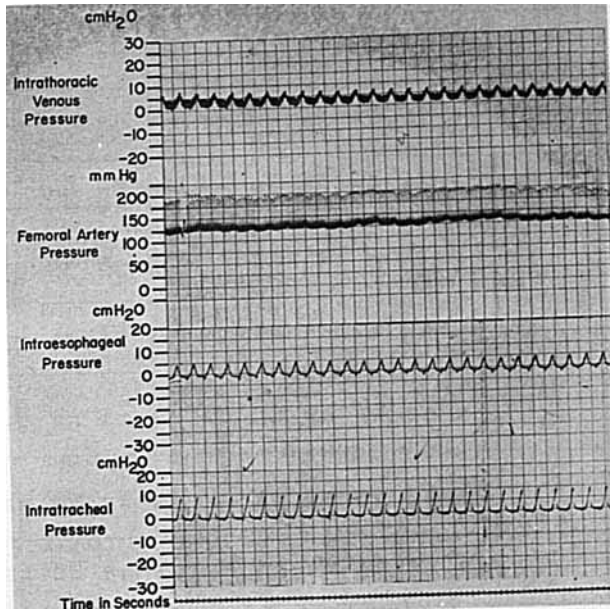

FIG. 6. Hemodynamics—positive-atmospheric respiration (cardiac output 5.98 l./min.).

Statham strain gauge (Model P 23B) connected by a long polyethylene tubing to a partially inflated balloon lying in the thoracic esophagus (20). Tracheal pressures were measured by a similar strain gauge connected to a 15 gauge needle inserted into the lumen of the endotracheal tube. The mean tracheal pressure was calculated later by planimetry of the tracheal pressure recordings.

The P_ACO_2 was recorded continuously by the gas mass spectrometer (10).

Cardiac catheters were threaded into the thoracic aorta and the

TABLE 1

VENTILATORY DATA

Dog	Breathing	Tidal Volume (cc.)	R. R.*	P_ACO_2 (mm. of Hg.)	Peak Flow Rate l/Min.	Duration		I:E Ratio	Pressures (cm. of H_2O)				
						Insp. (sec.)	Exp. (sec.)		Esophageal		Tracheal		Mean Tracheal
									Insp.	Exp.	Insp.	Exp.	
1	Ambient	300	16	36.4	18	1.5	2.5	1:1.65	−4	3	−1	2	0
	Respirator	300	16	43.6	21	1.3	2.6	1:2	4	−0.5	10	0	2.06
2	Ambient	375	15	29	19.5	.6	3.4	1:5.6	—	—	−2.86	0.75	0
	Respirator	375	16	32	14	1.2	2.4	1:2	—	—	6.5	0	1.57
3	Ambient	350	16	43	20	.6	2.8	1.1:4.6	−7	−1	−3	1.5	0
	Respirator	375	20	43	20	1.	2.	1:2	6	0	10.	0	1.25
4	Ambient	350	28	25	19	.8	1.2	1:1.5	−5	2	−1	.75	0
	Respirator	375	20	40	30	1.	2.	1:2	4	−1	8.4	0	1.51
5	Ambient	272	22	36	17.5	.6	2.	1:3.3	−7.5	1	−2	3	0
	Respirator	330	24	36	31.5	.84	1.78	1:2	3	0	9.5	0	1.00
6	Ambient	200	16	32	12	1.2	2.4	1:2	−4	0	−1	0.5	0
	Respirator	200	20	36	15	1.	2.	1:2	3	−1	8	0	2.76
7	Ambient	225	15	33	10	1.2	2.98	1:24	−4.2	0.2	−1	0.75	0
	Respirator	250	16	51	16	1.2	2.40	1:2	2.5	0	9	0	1.2
8	Ambient	210	27	55	21.5	.6	1.5	1:25	−7	2.4	−3.6	1.2	0
	Respirator	325	26	51	41	.64	1.4	1:2	4	0	10.5	0	2.53
9	Ambient	400	11	58	31	1.6	5.2	1:3.2	—	6	−1.5	1.65	0
	Respirator	500	15	58	37	1.5	3.2	1:2	—	−0.25	13.7	0	2.94
10	Ambient	220	24	51	14.5	1.1	1.5	1:1.36	−3.5	2	−2	3.5	0
	Respirator	450	16	51	31	1.3	2.6	1:2	3.5	0	16	0	3.46

* Respiratory rate.

femoral artery and connected to Statham strain gauges (Model P 23A). All strain gauges were connected to a Sanborn polyviso recorder and synchronous continuous recordings of all the above data were obtained.

Cardiac output was estimated by the Hamilton dye-dilution method (21). The circulation time was calculated from the first appearance of dye in the collecting tubes to the first sign of recirculation of the dye.

The above measurements were recorded in the anesthetized dog

TABLE 2

CIRCULATORY DATA

Dog	Breathing	Pulse Rate (beats/min.)	Caval Pressure (cm. of H_2O)		Aortic Pressure (mm. of Hg.)	Cardiac Output (L./min.)	Circulation Time (sec.)
			Insp.	Exp.			
1	Ambient	132	−6	2.5	215/155	2.07	13
	Respirator	126	7	2	200/150	1.53	13
2	Ambient	138	−5	0	165/140	1.58	13
	Respirator	150	2.5	−2.5	165/140	1.34	15
3	Ambient	102	−10	−1.5	185/125	2.28	11
	Respirator	96	−3	−7	180/130	1.69	14
4	Ambient	108	−10	−5	170/120	5.6	9
	Respirator	210	2	−4	165/140	5.98	9
5	Ambient	138	4	10	175/140	1.64	13
	Respirator	170	14	10	185/165	1.68	17
6	Ambient	168	−1	5	185/145	2.56	11
	Respirator	153	2	−1	180/150	1.72	15
7	Ambient	150	1	18	195/155	1.33	13
	Respirator	132	9	8.5	190/145	1.5	15
8	Ambient	186	−1	1	175/135	—	—
	Respirator	174	1	−1	175/135	—	—
9	Ambient	156	−2	1	145/120	—	—
	Respirator	168	2	−1	150/125	—	—
10	Ambient	172	−4	6	180/105	3.26	9
	Respirator	168	8	−2	210/125	2.91	12

with ambient breathing. The animal was then paralyzed with intravenous Flaxedil® in dosages sufficient to produce apnea, whereupon ventilation was provided with the intermittent positive pressure respirator. Respiratory paralysis and intermittent positive pressure breathing were continued for one hour, during which time continuous recordings were made of air flow rate, P_ACO_2, tracheal, esophageal, caval, aortic, and femoral artery pressures. Tidal volume was adjusted to maintain the P_ACO_2 at the partial pressure which existed in

the ambient breathing state. Intermittent doses of intravenous Flaxedil were given to maintain muscular flaccidity. After one hour, a final cardiac output estimation was made and the experiment was terminated. Figures 5 and 6 depict the pertinent hemodynamic alterations occurring when a dog is shifted from ambient to respirator breathing.

Findings.—Table 1 represents a compilation of respiratory data measured. Ventilation, as determined by the P_ACO_2, remained essentially unaltered in 8 of the 10 dogs, as they were transferred from ambi-

TABLE 3

EFFECT OF INCREASES IN MEAN TRACHEAL PRESSURE UPON CARDIAC OUTPUT

Dog	Breathing	P_ACO_2 (mm. of Hg.)	Mean Tracheal Pressure (cm. of H_2O)	Cardiac Output (l./min.)
1	Ambient	36.4	—	2.07
	Respirator	43.6	2.06	1.53
2	Ambient	29	—	1.58
	Respirator	32	1.57	1.34
3	Ambient	43	—	2.28
	Respirator	43	1.25	1.69
4	Ambient	25	—	5.6
	Respirator	40	1.51	5.98
5	Ambient	36	—	1.64
	Respirator	36	1.00	1.68
6	Ambient	32	—	2.56
	Respirator	36	2.76	1.72
7	Ambient	33	—	1.33
	Respirator	51	1.2	1.5
8	Ambient	55	—	—
	Respirator	57	2.53	—
9	Ambient	58	—	—
	Respirator	58	2.94	—
10	Ambient	51	—	3.26
	Respirator	51	3.46	2.91

ent to positive pressure breathing. In these 8 dogs the average peak tracheal pressure attained was 10.5 cm. of H_2O, resulting in an average mean tracheal pressure of 2.2 cm. of H_2O.

Table 2 is a compilation of the circulatory data obtained. Of the 8 dogs with unaltered ventilation, cardiac output determinations were obtained on 6. An average fall of 17 per cent in the cardiac output resulted with the transfer from ambient to "pressure" breathing. The relationships between mean tracheal pressures and cardiac outputs are shown in table 3.

Aortic blood pressure remained essentially unchanged in these studies. No material change in pulse rate was apparent except in experiments 4 and 5. The caval and atrial pressures were inconsistent in the magnitude of change occurring during intermittent positive pressure respiration. The average circulation time increased by twenty per cent.

Discussion.—The most significant feature revealed in the above findings was the exceptionally low mean tracheal pressures required to effect the same degree of ventilation as existed during ambient breathing. The average value of 2.2 cm. of H_2O was obtained without employing the negative pressure phase of the respirator.

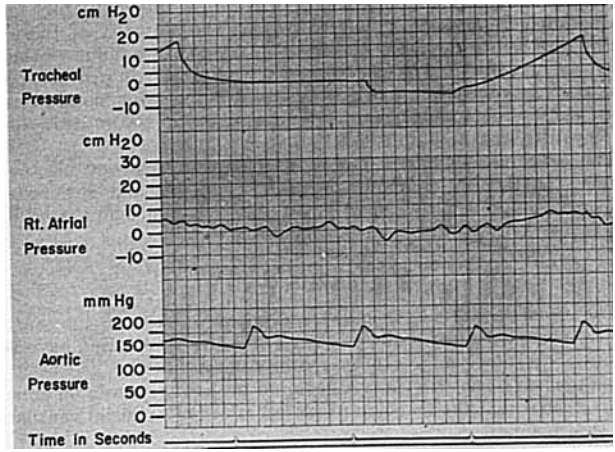

FIG. 7. Positive-negative respiration. Upper tracing depicts the application of the negative phase. In this animal the cardiac output (1.29 l./min.) has been reduced markedly by hemorrhage, yet the aortic pressure (lower tracing) is maintained.

The primary purpose of this laboratory study was to determine the level of mean tracheal pressure for normal ventilation. Certain circulatory data also were monitored. Conclusions based upon these findings are unwarranted since variables inherent in the method for producing respiratory paralysis were introduced.

The average fall of 17 per cent in cardiac output may be incident to changes in muscular tonus and the direct effects of the muscle relaxant on the cardiovascular system. Changes in the level of anesthesia also contributed an additional variable.

Studies specifically designed to reflect changes in the cardiovascular system are in progress. These consist of monitoring cardiovascular

functions under controlled conditions during positive-atmospheric and positive-negative pressure breathing. This series is too small to draw conclusions at this moment. The trends appear to agree with current concepts: introduction of the negative-pressure phase produces only a slight increase in blood pressure and cardiac output in the normal anesthetized dog, and animals in severe oligemic or normovolemic shock benefit markedly by the negative-pressure phase. Figure 7 graphically demonstrates the negative pressure phase of this respirator.

The clinical applications of this ventilator to a series of patients furnish the basis for a future report. However, the enthusiasm acclaimed by nursing, physician, and technical personnel, as well as the patients, prompts the inclusion of the following information.

To date this respirator has been employed for the ventilatory maintenance of more than 25 apneic patients. The majority of these patients were suffering from either acute systemic tetanus or bulbospinal poliomyelitis. The efficiency of the respirator in providing long term artificial ventilation was demontsrated in the seven months during which successful management of a patient with severe bulbospinal poliomyelitis was accomplished.

The continuous maintenance of normal acid-base and electrolyte balance, the facilitation of diagnostic, therapeutic and nursing care problems, and the amazing diagnostic ability of the machine itself have thus far exceeded all expectations.

Summary

The fundamental physiologic requirements for safe intermittent positive pressure and positive-negative pressure respiration have been reviewed. A new positive pressure breathing machine has been designed to meet these criteria. A group of preliminary tests on animals demonstrated that the machine produced effective artificial ventilation in a safe manner.

Acknowledgment

The individual designers, engineers, and machinists under the guidance of Mr. Tage Falk, (M.E.) share a large proportion of the responsibility and credit for the development of the respirator.

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