Properties of Passive Exhalations in Anesthetized Subjects

*Norman A. Bergman, M.D.**

Mechanical properties of the thorax during anesthesia were studied by analysis of passive exhalations in anesthetized, paralyzed subjects. There were several differences between properties of passive exhalations and respiratory mechanics in normal subjects and in those who had increased respiratory resistance. In normal patients the magnitudes of respiratory resistance and pressure–flow relationships of the thorax were comparable to those previously reported for conscious men. Values for thoracic compliance also approached those previously reported for conscious humans. Basic mechanical properties of the thorax in the anesthetized paralyzed patient did not appear to be remarkably different from those in comparable conscious individuals breathing spontaneously.

EXHALATION in anesthetized, paralyzed subjects is entirely independent of voluntary or involuntary muscular activity and depends solely on the mechanical properties of the lungs and chest wall. For this reason such individuals are suitable subjects for study of the basic mechanical properties of the thorax. Interpretation of studies of respiratory mechanics in conscious subjects frequently is complicated by inability to determine the manner in which factors such as muscular activity influence observed results. This study describes some properties of passive exhalations in anesthetized paralyzed subjects which form a basis for some conclusions concerning mechanical characteristics of the respiratory system. In addition, deviations of observed properties of passive exhalations from theoretically-predicted ideal conditions are evaluated.

* Associate Professor of Anesthesiology.

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Methods

Subjects of the study were patients scheduled for elective surgery under general anesthesia. They were premedicated with pentobarbital, 100–200 mg, and atropine, 0.6 mg. Anesthesia was induced with thiopental sodium. *D*-tubocurarine, 0.4 mg per pound of body weight, was then administered; when the subject was sufficiently relaxed a No. 36 cuffed rubber endotracheal tube (length 28 cm) was passed and the cuff inflated to provide an airtight seal. This dose of *d*-tubocurarine was selected because it provided profound relaxation with complete absence of spontaneous respiratory activity for the duration of the study. Following endotracheal intubation, anesthesia was maintained with nitrous oxide and either halothane or methoxyflurane. All measurements were made after the start of surgery.

The experimental apparatus consisted of two separate circuits: a ventilator circuit for mechanical ventilation between measurements and a syringe–spirometer circuit for actual performance of the measurements (fig. 1). The four-way wide-bore (I.D. 1 inch) respiratory valve ("B") permitted rapid alternation between circuits. With valve "B" in line with the ventilator circuit, nitrous oxide and either halothane or methoxyflurane were supplied to the subject by the Manley Ventilator[1] and the subject exhaled to atmosphere through the expiratory valve of the ventilator. End-expiratory carbon dioxide tension was monitored with a Beckman LB-1 infra-red analyzer.

To record passive exhalations, the three-way valves "C" to the CO_2 analyzer and "A" to the spirometer were closed. At the end of an exhalation, valve "B" was rotated to select the syringe–spirometer circuit and the subject's thorax was inflated with 500, 1,000, or 1,500 ml of the anesthetic gas mixture from the 1,500-ml syringe. Each series of inflations was preceded by a large breath. The orders

in which the different volumes were employed were varied. Thoracic inflation was maintained for approximately two seconds; then, rapid rotation of valve "A" permitted passive exhalation into the spirometer.† When exhalation was complete, rotation of valve "B" to the ventilator circuit permitted resumption of regular mechanical artificial ventilation.

Pressure in the airway was measured with a pressure transducer (Statham PM5TC), exhaled volume and flow rate during passive exhalations were measured with the volume and flow transducers of the spirometer, and all three parameters were recorded using a Minneapolis Honeywell Visicorder. The pressure transducer was calibrated with a water manometer, the volume transducer of the spirometer with the 1,500-ml syringe, and the flow transducer of the spirometer by measuring rate of change of volume during filling of the spirometer at constant flow.

A representative record of pressure, volume and flow during thoracic inflation and subsequent passive exhalation is illustrated in figure 2. Thoracic static compliance was calculated by dividing volume exhaled (corrected to BTPS) by transthoracic pressure during the brief period of sustained thoracic inflation. Total respiratory resistances at flows of 0.5, 1.0 and 1.5 l/sec were calculated by measuring thoracic volume at instants that these flows occurred. Assuming that at any instant transthoracic pressure is proportional to thoracic volume, dividing these selected volumes by compliance gives the transthoracic pressure required to produce these expiratory flows which, by definition, is total respiratory resistance. Apparatus resistance (including endotracheal tube) was determined in an identical manner, using a 20-l carboy as a resistance-free patient analog. Values for apparatus resistance were subtracted from those for total resistance to obtain patient respiratory resistance in each subject. The time constant of exhalation was calculated as the time required to exhale down to 0.368 (1/e) of the total exhaled volume, and a second value for respiratory resistance was calculated by dividing the time constant of exhalation by thoracic compliance (appendix).[2] A semilogarithmic plot of thoracic volume against time was made for a 1,500-ml exhalation of each subject, inspected visually for correspondence to an exponential function, and a second value for time constant calculated from the slope of this graph. From simultaneous measurement of transthoracic pressure (P) and flow ($\dot{V}$) at 0.1-second intervals during passive exhalation of 1,500 ml, an attempt was made to fit the observed values to an equation of the general form:

$$P = K_1\dot{V} + K_2\dot{V}^2$$

† Wedge Spirometer, Medical Science Electronics, St. Louis, Mo.

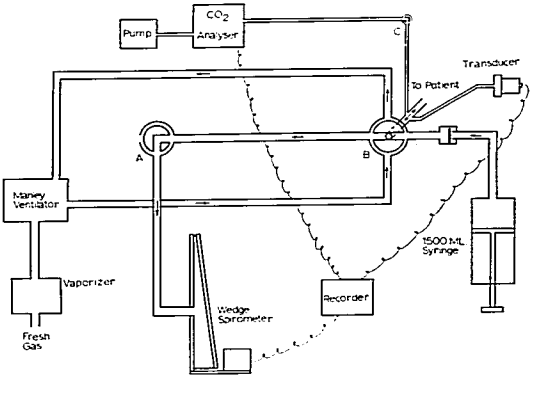

FIG. 1. The experimental apparatus. (See text.)

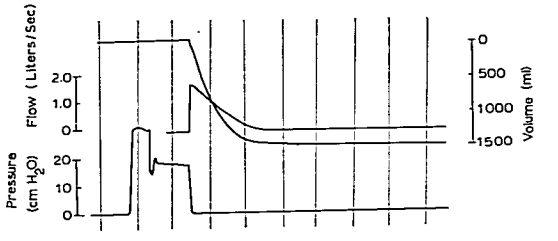

FIG. 2. Pressure, exhaled volume and expiratory flow rate during thoracic inflation and passive exhalation in a normal subject. Vertical time lines denote 1-sec intervals.

Results

Experimental results for thoracic inflations of 1,500, 1,000 and 500 ml are tabulated in tables 1, 2 and 3, and summarized in table 4. Exhaled volumes demonstrate the 7 to 9 per cent increase in volume which occurrs when dry gas at room temperature is warmed and humidified in the lung.

Subjects were classified as "normal" or "abnormal" with respect to manner of exhalation on the basis of expiratory flow patterns. In normal subjects, expiratory flow decreased at a fairly uniform rate throughout

TABLE 1. Experimental Results for Thoracic Inflations of 1,500 ml*

Subject†	V_E (ml BTPS)	C_T (ml/cm H_2O	Respiratory Resistance (cm H_2O/l/sec)			Observed TC (sec)	Calculated TC (sec)	Res_{TC}	$Res_{1.0U}$	K_1	K_2
			0.5 l/sec	1.0 l/sec	1.5 l/sec						
1	1,495	67.9	6.8	8.4	7.9	0.76	0.64	11.2	11.3	3.3	3.5
2	1,587	113.4	3.0	4.2	5.9	0.82	0.80	7.2	7.1	1.2	2.5
3	1,696	66.5	5.0	4.3	5.6	0.64	0.63	9.6	7.2	3.8	0.6
4	1,643	99.6	2.8	4.0	5.1	0.74	0.70	7.7	6.9	2.9	1.4
5	1,670	68.2	3.0	3.9	5.2	0.64	0.58	9.4	6.8	1.9	1.9
6	1,609	76.6	3.4	4.1	5.3	0.68	0.69	8.9	7.0	3.8	0.9
7	1,576	55.3	5.2	7.4	7.4	0.66	0.58	11.9	10.3	4.5	3.5
8	1,585	102.2	3.0	4.2	5.1	0.76	0.70	7.4	7.1	1.8	1.7
9	1,650	86.9	1.8	2.3	3.3	0.64	0.59	7.4	5.2	1.1	1.3
10	1,594	70.8	5.0	5.8	6.6	0.66	0.68	9.3	8.7	4.1	1.1
Mean	1,611	80.4	3.9	4.9	5.7	0.70	0.65	9.0	7.8	2.84	1.84
SD	96.4	18.6	1.52	1.81	1.33	0.065	0.073	1.63	1.81	1.25	1.03
11	1,341	58.5	13.4	8.1	7.2	0.70	—	12.0	11.0		
12	1,598	71.0	15.0	9.7	7.4	0.92	0.78	13.0	12.6		
13	1,596	99.7	9.4	7.3	6.0	1.22	1.09	12.2	10.2		
14	1,600	88.9	9.2	6.5	5.7	1.06	1.08	11.9	10.1		
Mean	1,535	79.5	11.8	7.9	6.6	0.98	0.98	12.3	10.8		
SD	126	18.4	2.91	1.36	0.85	0.22	0.18	0.52	1.36		

* V_E = exhaled tidal volume; C_T = total thoracic compliance; observed TC = time required to exhale down to 0.36S of total exhaled volume; calculated TC = time constant of exhalation calculated from slope of semilogarithmic volume *vs.* time plot; Res_{TC} = total respiratory resistance calculated using thoracic compliance and observed time constant of exhalation; $Res_{1.0U}$ = respiratory resistance calculated at a flow of 1.0 l/sec uncorrected for apparatus resistance; K_1 and K_2 = coefficients for the equation $P = K_1\dot{V} + K_2\dot{V}^2$.

† Subjects 1–10 were classified as normal and subjects 11–14 as abnormal on the basis of expiratory flow patterns.

TABLE 2. Experimental Results for Thoracic Inflations of 1,000 ml*

Subject†	V_E (ml BTPS)	C_T (ml/cm H_2O)	Respiratory Resistance (cm H_2O/l/sec)			Observed TC (sec)	Res_{TC}	$Res_{1.0V}$
			0.5 l/sec	1.0 l/sec	1.5 l/sec			
1	1,033	66.6	4.8	5.5	5.6	0.60	9.0	8.4
2	1,044	104.4	3.0	4.0	—	0.67	6.4	6.9
3	1,087	57.2	5.4	5.1	6.1	0.52	9.1	8.0
4	1,110	85.4	2.4	4.0	—	0.60	7.0	6.9
5	1,088	65 9	2.4	3.6	4.5	0.52	7.9	6.5
6	1,098	64.6	4.6	4.2	4.9	0.56	8.7	7.1
7	1,057	47.1	4.2	6.8	—	0.49	8.8	9.7
8	1,082	90.2	2.2	3.5	—	0.60	6.7	6.4
9	1,093	78.1	1.8	2.1	3.4	0.53	6.8	5.0
10	1,081	70.9	3.4	4.8	5.3	0.56	8.0	7.7
Mean	1,078	73.0	3.4	4.4	5.0	0.57	7.9	7.3
S.D.	24.5	16.8	1.26	1.28	0.95	0.053	1.18	1.28
11	1,088	49.5	8.6	8.6	6.4	0.56	11.3	11.5
12	1,083	63.7	13.8	8.8	—	0.88	13.8	12.6
13	1,104	92.0	8.8	7.1	—	0.92	10.0	10.0
14	1,085	80.4	7.0	6.2	—	0.84	10.4	9.1
Mean	1,090	71.4	9.6	7.7	(6.4)	0.80	11.4	10.8
S.D.	9.5	18.7	2.94	1.25	—	0.16	1.73	1.25

TABLE 3. Experimental Results for Thoracic Inflations of 500 ml*

Subject†	V_E (ml BTPS)	C_T (ml/cm H_2O)	Respiratory Resistance (cm H_2O/l/sec)		Observed TC (sec)	Res_{TC}	$Res_{1.0V}$
			0.5 l/sec	1.0 l/sec			
1	505	59.5	3.8	4.5	0.42	7.1	7.4
2	533	98.6	3.0	—	0.52	5.4	—
3	522	45.4	7.4	6.0	0.46	10.1	8.9
4	566	80.1	3.0	—	0.44	5.5	—
5	528	58.7	2.6	3.8	0.38	6.5	6.7
6	526	61.9	2.8	3.8	0.39	6.3	6.7
7	511	40.9	5.2	7.7	0.36	8.8	10.6
8	536	76.5	2.0	3.4	0.44	5.8	6.3
9	514	64.2	2.0	1.9	0.35	5.5	4.8
10	560	70.1	4.4	4.3	0.45	6.4	7.2
Mean	530	65.5	3.6	4.4	0.42	6.7	7.3
S.D.	19.9	16.9	1.67	1.75	0.052	1.56	1.75
11	579	48.3	13.2	7.1	0.80	16.6	10.0
12	550	55.0	13.0	—	0.86	15.6	—
13	547	78.1	7.0	—	0.64	8.2	—
14	541	77.2	5.0	—	0.64	8.3	—
Mean	554	64.7	9.6	(7.1)	0.73	12.2	(10.0)
S.D.	16.9	15.3	4.18	—	0.112	4.55	—

TABLE 4. Summary of Experimental Results for Thoracic Inflations of 500, 1,000 and 1,500 ml

	V_E (ml BTPS)	C_T (ml/cm H_2O)	Respiratory Resistance (cm H_2O/l/sec)			Observed TC (sec)	Res_{TC}	$Res_{1.0}$
			0.5 l/sec	1.0 l/sec	1.5 l/sec			
Normal subjects	530	65.6	3.6	4.4	—	0.42	6.7	7.3
	1,078	73.0	3.4	4.4	5.0	0.57	7.9	7.3
	1,611	80.4	3.9	4.9	5.7	0.70	9.0	7.8
Abnormal subjects	554	64.7	9.6	7.1	—	0.73	12.2	10.0
	1,090	71.4	9.6	7.7	6.4	0.80	11.4	10.8
	1,535	79.5	11.8	7.9	6.6	0.98	12.3	10.8

exhalation (fig. 2), while in those classified as abnormal an abrupt change in rate of fall of expiratory flow occurred at some point during exhalation (fig. 3). For normal subjects the semilogarithmic graph of lung volume as a function of time could reasonably be approximated by a straight line during the greater part of exhalation, but the actual configuration of the graph demonstrated progressive increase in flow rate relative to lung volume, becoming more prominent toward the end of exhalation (fig. 4). In abnormal subjects, although the initial portion of exhalation could be approximated by a straight line on the semilogarithmic graph, two lines were required for adequate approximation of exhalation (fig. 5). A slight increase in flow rate relative to lung volume occurred at the end of exhalation.

Mean thoracic compliance values were 65.2, 72.6 and 80.2 ml/cm H_2O for inflations of 500, 1,000 and 1,500 ml, respectively, and there was no difference in compliances of normal and abnormal subjects.

The time constants of exhalation were 0.42, 0.57, and 0.70 seconds for inflations of 500, 1,000, and 1,500 ml in normal subjects. In abnormal subjects the time constants of exhalation were significantly greater; corresponding values were 0.73, 0.80, and 0.98 sec. The time constant of exhalation calculated from the slope of the line which best approximated the points of the semilogarithmic volume-*vs.*-time graph obtained from a 1,500-ml exhalation was 0.65 sec for normal subjects; 0.98 sec for abnormal subjects.

Patient total respiratory resistances were 3.9, 4.9, and 5.7 cm H_2O/l/sec, calculated at flow rates of 0.5, 1.0, and 1.5 l/sec following 1,500-ml inflations in normal patients. Corresponding values in abnormal patients were 11.8, 7.9, and 6.6 cm H_2O/l/sec. Thus, in normal subjects transthoracic pressure required to produce a unit flow increased as flow rate increased, and the pressure–flow relationship in these subjects was best described by the equation:

$$P = 2.84\dot{V} + 1.84\dot{V}^2$$

Since in abnormal patients during passive

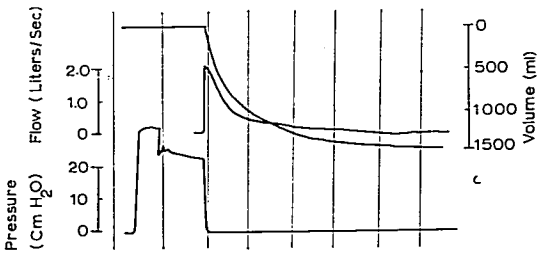

FIG. 3. Pressure, exhaled volume and expiratory flow rate during thoracic inflation and passive exhalation in an abnormal subject. Rate of decrease in expiratory flow changes abruptly in contrast to the fairly regular fall in expiratory flow rate throughout exhalation seen in normal patients (fig. 2).

exhalation transthoracic pressure required to produce a unit flow actually decreased as flow rate increased, an equation of the general form $P = K_1\dot{V} + K_2\dot{V}^2$ could not be fitted to data from these subjects.

Total respiratory resistances calculated from the time constants of exhalation were 6.7, 7.9, and 9.0 cm H_2O/l/sec for inflations of 500, 1,000, and 1,500 ml in normal subjects. Since these values include apparatus resistance, they should be compared with resistance calculated from simultaneous volume and flow measurements uncorrected for apparatus resistance and at a flow of 1.0 l/sec. These values were 7.3, 7.3, and 7.8 cm H_2O/l/sec for the three inflation volumes. In abnormal subjects respiratory resistances calculated from time constants of exhalation were 12.2, 11.4, and 12.3 cm H_2O/l/sec, and corresponding values calculated from simultaneous volume and flow measurements uncorrected for apparatus resistances were 10.0, 10.6, and 10.8 cm H_2O/l/sec for inflations of 500, 1,000, and 1,500 ml.

End-expiratory carbon dioxide tensions of all subjects were in the range of 30–38 mm Hg; they did not vary appreciably during the study in any subject.

Discussion

The subjects were men 34 to 76 years old. Of the 11 subjects classified as "normal" on the basis of expiratory flow patterns, one subject (no. 2) had a history of asthma and chronic cough and an abnormal chest radiograph. The other ten were clinically free of pulmonary disease. All four subjects who had "abnormal" expiratory flow patterns also had physical and radiographic evidence of pulmonary disease. It is known that resistance in the respiratory system is nonlinear, and the equation $P = K_1\dot{V} + K_2\dot{V}^2$ has been proposed to describe pressure–flow relationships for the respiratory system.[3] The equation describing pressure–flow relationships in normal anesthetized subjects in the present study, $P = 2.84\ \dot{V} + 1.84\ \dot{V}^2$ is in very good agreement with equations obtained by previous workers from data in relaxed, passively-ventilated conscious volunteers. Mead proposed the equation $P = 2.5\ \dot{V} + 3.3\ \dot{V}^2$,[4] while Otis *et al.* derived the equation $P = 3.5\ \dot{V} + 1.5\ \dot{V}^2$.[5] Thus,

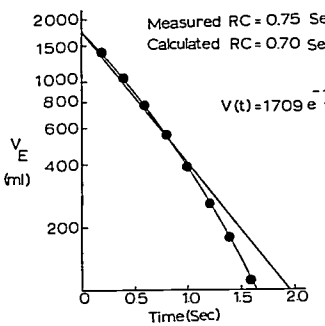

FIG. 4. Thoracic volume as a function of time during a passive exhalation in a normal subject. The greater portion of exhalation can reasonably be approximated by a straight line, but observed values indicate progressive increase in flow relative to lung volume throughout exhalation.

anesthesia with muscular paralysis does not appear to alter pressure–flow relationships of the respiratory system from those during the conscious state.

Total respiratory resistance as measured in the present study includes airway resistance, lung tissue resistance, and resistance of the chest wall. The magnitude of total respiratory resistance of normal subjects in the present study was 3.9 cm H_2O/l/sec, calculated at a flow of 0.5 l/sec. This may be compared with the value of 4.6 cm H_2O/l/sec obtained by DuBois and associates using a method of forced oscillation of the lung in conscious subjects[6] and with the value 5.0 cm H_2O/l/sec calculated by Otis and associates from data obtained during passive ventilation of conscious subjects in a tank respirator.[5] An appreciable fraction of total respiratory resistance is known to reside at the glottis and in the upper air passages,[7] and in the present study these structures were bypassed with an endotracheal tube whose resistance was included in that of the apparatus. It is concluded, therefore, that total respiratory resistance in anesthetized, paralyzed subjects is not notably different from that in comparable conscious individuals breathing spontaneously.

Differences in expiratory flow patterns during exhalation which formed the basis

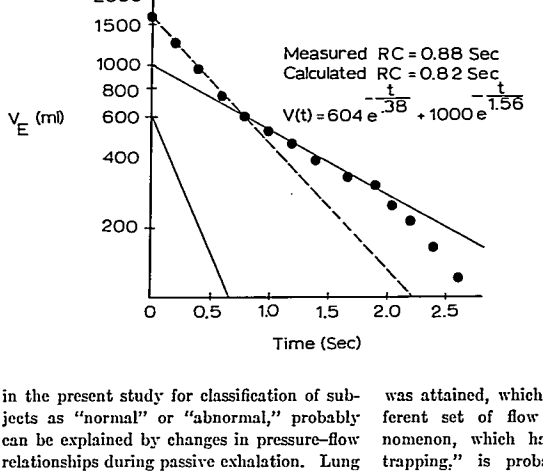

FIG. 5. Thoracic volume as a function of time during a passive exhalation in an abnormal subject. The initial and greater portion of exhalation can be described by a straight line (dotted line) but at least two lines (solid lines) are required for a reasonable approximation of the exhalation. Increase in flow relative to lung volume occurs toward the end of exhalation.

in the present study for classification of subjects as "normal" or "abnormal," probably can be explained by changes in pressure–flow relationships during passive exhalation. Lung volumes and expiratory flow rates continuously decrease during exhalation, and since diameter of conducting air passages is determined in part by lung volume and transpulmonary pressure, bronchial caliber continuously decreases during exhalation also.[8] The plot of lung volume as a function of time during passive exhalation in normal subjects (fig. 4) indicated that, in these individuals, rate of lung emptying relative to lung volume increased during exhalation. A likely explanation for this observation is that during exhalation flow resistance decreased more rapidly than transthoracic pressure, so that given increments of transthoracic pressure were capable of producing more effective lung emptying as exhalation proceeded. In abnormal subjects, initial expiratory flow was usually rapid, but at some point during exhalation an abrupt decrease in rate of change of flow occurred (fig. 5) so that subsequent decrease in expiratory flow rate was markedly slower and exhalation was prolonged. Under these circumstances it is postulated that during exhalation flow resistance decreased at a slower rate than transthoracic pressure, and that at some point in exhalation a critical level of bronchial caliber was attained, which abruptly imposed a different set of flow conditions. This phenomenon, which has been designated "air trapping," is probably due to structural changes in the walls of the conducting air passages, and is frequently demonstrable in spirograms obtained from conscious individuals with bronchopulmonary disease.[9] Flow in the respiratory system should become increasingly turbulent as flow rate increases, and it would be predicted that magnitude of respiratory resistance should be greatest at high flow rates and should diminish as flow rates decrease. This prediction was confirmed in normal subjects in the present study, since respiratory resistance was greatest when measured at 1.5 l/sec, less at 1.0 l/sec, and least at 0.5 l/sec. The reverse situation was encountered in the abnormal subjects; resistance was least when measured at high flow rates and increased as flow rate decreased. This is not a contradiction to known physical laws describing flow of fluids through tubes, however. If resistance were measured in these abnormal subjects using different flow rates at a fixed thoracic volume with constant dimensions of the conducting airways, then the expected relationship between resistance and flow would have been observed.[10] The apparent paradox occurred because under conditions of this study the highest flow rates always occurred at the beginning of a passive exhalation when

bronchial dimensions were greatest, and because of the probable greatly-disproportionate reduction of bronchial caliber relative to flow rate during exhalation in abnormal subjects. Patterns of exhalation observed in this study represent mechanical behavior of the thorax uninfluenced by muscular activity.

If passive exhalations conformed to the ideal exponential equation (appendix), it could be predicted that the time required for a passive exhalation would be independent of the exhaled volume. The present study again showed deviations from ideal conditions because, as volume exhaled increased, the time constant of exhalation also became larger. Increase in the time constant, however, was only a fraction of increase in volume exhaled. In normal subjects, doubling the exhaled volume from 500 to 1,000 ml prolonged the time required to exhale only 1.4 times, while exhalation of 1,500 ml required only 1.7 times as long as exhalation of 500 ml. In abnormal subjects, increase in the time constant with increasing volume was less, but the same general relationship occurred. Again, this phenomenon can be attributed to the presence of nonlinear resistances in the respiratory system. Normally, about 4.5 time constants would be required for exhalation of 99 per cent of the tidal volume if a passive exhalation were exponential throughout its course. Increasing the rate of lung emptying relative to lung volume during a passive exhalation would seem to be an important physiologic mechanism to facilitate completion of exhalation and thus avoid air trapping and hyperinflation of the lung at rapid respiratory rates. It is apparent that the best way to avoid air trapping in artificially-hyperventilated subjects is to achieve hyperventilation primarily by increases in tidal volume rather than by increases in respiratory rate.

One of the objectives of the present study was to determine how nonlinearity of resistance in the respiratory system affected the ideal exponential characteristics of a passive exhalation. Although significant deviations from ideal conditions were demonstrated, it is concluded that as a first approximation the time course of a passive exhalation could adequately be described by an exponential equation in many situations. This simplification would be of value in such applications as design of electrical lung analogs and analysis of the function of ventilators and other items of equipment.

In a previous publication we proposed a method for estimating respiratory resistance in apneic subjects, based on the exponential characteristics of passive exhalations (appendix).[2] The present study confirms that this is a simple, objective method for estimation of respiratory resistance which does not require measurement of gas flow rates and therefore can be performed with relatively simple equipment in anesthetized subjects or in patients during prolonged artificial ventilation. Differences between measured resistances and those estimated from time constants of exhalation can again be attributed to nonlinearity of resistance in the respiratory system.

Total thoracic compliance increased with increasing tidal volume in the present study, and at the largest inflation volume its value approached those which have been reported for healthy conscious subjects.[11] Inspection of the pressure–volume diagram of the thorax suggests that incremental volume inflation of the thorax should require progressively increasing pressure steps once the linear portion of the curve has been traversed.[12] Increase of total thoracic compliance with tidal volume in the present study could be explained by factors such as recruitment of additional respiratory units or displacement of progressively greater quantities of blood from the thorax, and illustrates that the value for compliance depends on the manner in which it is measured. Previously reported values for thoracic compliance in anesthetized subjects have almost always been lower than those in comparable conscious subjects. It has been suggested that values obtained in anesthetized subjects represent the true pressure–volume characteristics of the thorax, while those in conscious subjects are falsely high because of persistence of involuntary muscular activity.[13] As stated previously, values for respiratory resistance in anesthetized subjects in the present study were similar to those previously reported for conscious subjects. It is concluded, therefore, that basic mechanical properties of the thorax in anesthetized, paralyzed subjects are not remarkably different from

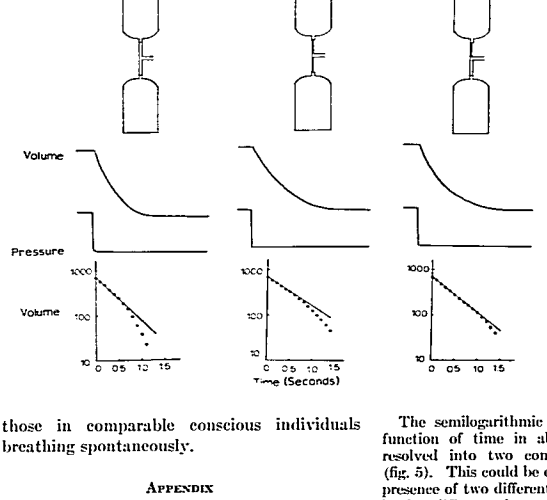

FIG. 6. Time course of passive exhalations for a pneumatic lung analog consisting of two 19.7-l. carboys exhaling through resistances made from thick-walled vacuum tubing. In *A* the resisters for each carboy have equally large internal diameters. In *B* the resisters both have small internal diameters, while in *C* one carboy exhales through a large-bore resister while the other exhales through a small-bore resister. It is not possible to deduce the presence of unequal resistances, and hence unequal time constants, for the two units exhaling in parallel in *C* from analysis of the time course of exhalation.

those in comparable conscious individuals breathing spontaneously.

APPENDIX

Exponential Characteristics of Passive Exhalations

Theoretical calculations presented in a previous publication show that, ideally, a passive exhalation could be described by the equation:

$$V(t) = V_0 e^{-t/RC} \quad (1)$$

where $V(t)$ is volume of gas in the lung at any time, t, seconds after the beginning of exhalation, V_0 is initial lung volume above resting level, R is respiratory resistance and C is respiratory compliance.[2] The semilogarithmic plot of such an equation is a straight line, and the product RC is the time constant of the expression.[14] At a time in seconds which is numerically equal to the product RC, equation (1) becomes:

$$V = V_0 e^{-1} = 0.368 V_0$$

A relatively simple method of estimating respiratory resistance in anesthetized subjects, which does not require measurement of gas flow rates, was suggested by this relationship. Respiratory compliance (C) can be determined from transthoracic pressure during a thoracic inflation and from volume subsequently exhaled. The time constant of exhalation (the product RC) can be determined from a rapid recording of the exhalation by measuring time required for lung volume to fall to 0.368 of total exhaled volume. Since both C and RC have been measured, R (respiratory resistance) can be calculated.

The semilogarithmic plot of lung volume as a function of time in abnormal subjects could be resolved into two component exponential terms (fig. 5). This could be explained by postulating the presence of two different groups of pulmonary units having different time constants and exhaling asynchronously, rather than by an abrupt change in flow conditions during exhalation. Evidence against the first alternative is suggested by properties of electrical circuits analogous to the respiratory system, where it is known that any combination of resistances and capacitances in series and/or parallel can be replaced with an equivalent circuit composed of one resistance and one capacitance. If such a circuit is charged to a predetermined level, examination of the time course of the subsequent discharge does not permit deductions as to the possible number, arrangement, or values for the passive components comprising the circuit, since the time course of discharge would be that of the simple equivalent circuit. In order to determine whether a similar principle would apply to compliance elements emptying through pneumatic resistances, a simple lung analog was employed. Two carboys which had been pressurized with a predetermined volume of air were emptied in parallel through the analog resisters made from thick-walled rubber tubing. In the first example, resistances for the two carboys had equal large internal diameters (fig. 6*A*); in the second the resistances had equal small internal diameters (fig. 6*B*); in the third one carboy emptied through a large-bore resistance while the other emptied through a small-bore resistance (fig. 6*C*). The patterns of emptying of the lung analogs were identical using each of the three resistances; only the slope of the volume *vs.* time plot varied. These observations confirm that it is not possible to deduce the distribution of time constants in pneumatic

units emptying in parallel on the basis of information obtained from a single passive exhalation. Accordingly, the shape of the volume-time plot obtained with abnormal patients in the present study must be attributed to changes in flow conditions during exhalation rather than to the presence of two groups of pulmonary units having different time constants and exhaling asynchronously. The pattern of increasing flow rate relative to lung volume toward the end of exhalation, seen in the subjects of the present study, was also observed during emptying of the lung analog (fig. 6). This pattern, therefore, is not peculiar to the respiratory system but must be a general property of compliance elements emptying through the type of nonlinear resistance found in both the respiratory system and the lung analog. Pressure-flow relationships of the analog resisters were also characterized by the equation $P = K_1\dot{V} + K_2\dot{V}^2$.

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