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THE PRESSURE–FLOW RELATIONSHIPS OF THE INTRATHORACIC AIRWAY IN MAN *

By ROBERT E. HYATT † AND ROGER E. WILCOX

(*From the Cardiopulmonary Laboratory, Department of Medicine and Department of Surgery, Beckley Memorial Hospital [Miners Memorial Hospital Association], Beckley, W. Va.*)

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Measurement of the pressure-flow relationships of the human respiratory tract is basic to the study of ventilatory mechanics. The relationship of respiratory air flow to the total pressure drop along the respiratory tree (alveolus to mouth) has been extensively studied and been shown to depend on lung inflation (1–3) and the type and degree of airway disease (4). Recently the total pressure drop has been partitioned into that occurring along the lower or intrathoracic airways (alveolus to trachea) and that along the upper or extrathoracic airways (trachea to mouth) (5, 6). The resistance of the upper airway with mouth breathing was found *1*) to comprise a significant portion of the total airway resistance in normal subjects, *2*) to vary from subject to subject as well as in a given subject, *3*) to depend on the type of breathing maneuver employed, and *4*) to vary inversely with lung inflation to a slight degree (6).

Only limited consideration has been given to the pressure-flow behavior of the lower airways (7). Yet in the evaluation of normal and abnormal ventilatory mechanics one is primarily interested in the behavior of the intrathoracic airways. It is the purpose of this study to analyze the pressure-flow characteristics of the lower airway in normal and emphysematous subjects and to relate the results to the usual methods of evaluating airway resistance in man.

THEORY

If one neglects the effect of gravity on the gas, the pressures along a stream of gas flowing through a nonuniform conduit system, such as the bronchial tree, may be analyzed by considering two properties of the flow, its frictional properties and its inertial properties. At any moment there are two superimposed pressure drops along the system related to these properties. A frictional pressure drop will exist related to the physical properties of the gas and to the flow geometry of the airways in a rather complicated manner. An inertial pressure drop will also exist related to the density of the gas and to the acceleration of the flow. The acceleration of the flow is of two varieties, local and convective. Local acceleration is the rate of change of velocity of the gas particles that occurs with time. Convective acceleration is the rate of change of velocity of the particles that occurs with the distance traveled by the gas particles, such as would occur with converging or diverging flow boundaries. In both cases the rate of change of velocity must be accompanied by a force or pressure gradient according to Newton's Second Law. The transient pressure drop associated with local acceleration in the airway has been shown to be negligible (8) and will be omitted from this analysis. The pressure drop associated with convective acceleration has not been thoroughly studied and may not be negligible.

Therefore, in analyzing the pressure difference between two points in a stream, as between alveolus and trachea in this study, one may consider this difference to consist of two pressure drops, one representing the energy dissipated between the points due to frictional losses in the stream and one representing the energy to accelerate or decelerate the gas particles between the two points associated with the converging or diverging flow boundaries (Bernoulli effect). The flow leaving the alveoli is essentially equal to that in the trachea. Moreover, the gas particles leaving the alveoli have extremely low velocities.

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† Present address: Section of Physiology, Mayo Clinic, Rochester, Minn.

As the particles travel toward the trachea, their velocities increase with the diminishing cross-sectional area of the conduit. Even if the frictional losses were negligible, a pressure difference would exist between alveolus and trachea to produce this velocity difference (convective acceleration). Similarly, during inspiration a pressure drop must exist to decelerate the tracheal gas particles to alveolar velocities. In either case the tracheal pressure associated with the convective acceleration of the gas will be negative with respect to the alveolus and will favor airway narrowing.

It follows that in interpreting the lower airway presssure-flow behavior it is necessary to estimate the convective and frictional pressure drops separately since each has independent significance. The convective pressure drop represents that energy stored in the flow as kinetic energy. The frictional pressure drop represents that energy lost in the flow as heat. These drops may be estimated by simultaneously measuring lateral tracheal air pressure, alveolar pressure, and total respiratory flow. A curve relating flow to the total pressure drop from alveolus to trachea can then be constructed. If in turn the

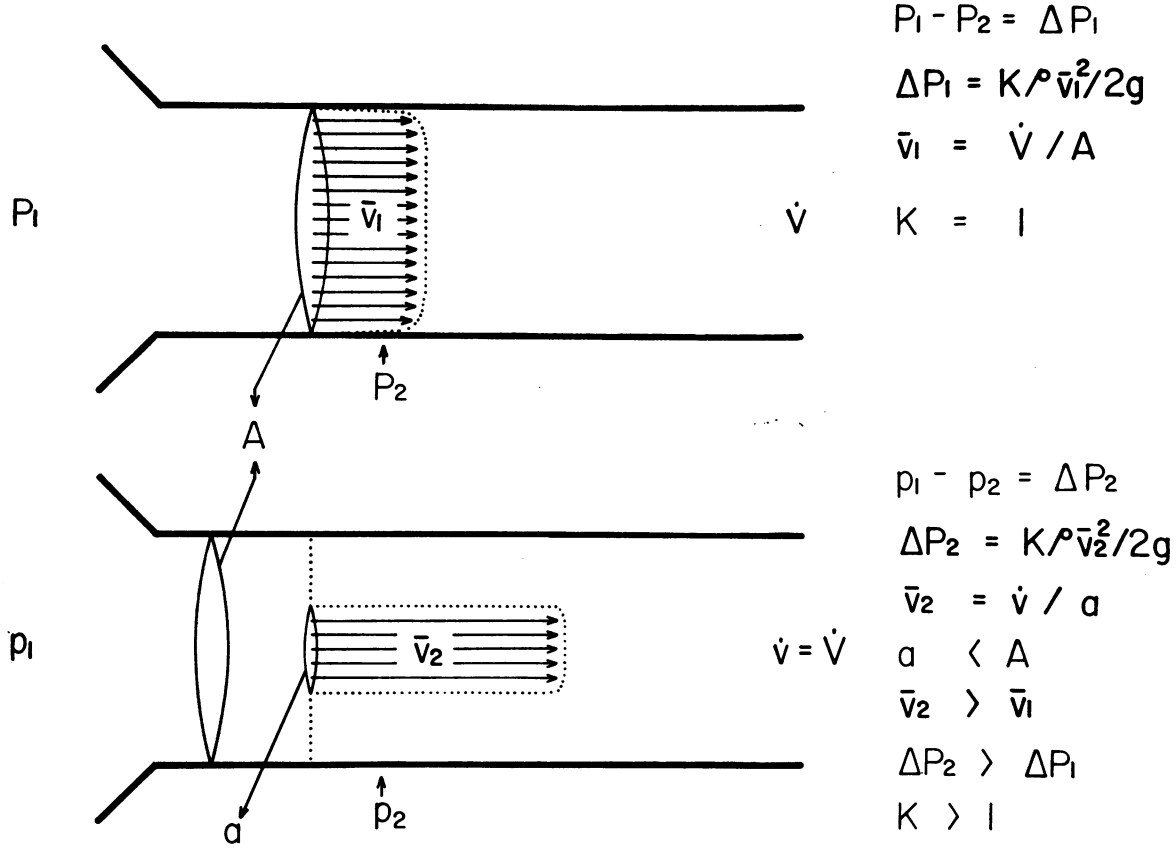

FIG. 1. EFFECT OF DIFFERENT VELOCITY PROFILES ON CONVECTIVE ACCELERATION PRESSURE DROP. At top, a uniform or blunt velocity profile (dotted line) is depicted. The pressure drop from alveolus (P_1) to trachea (P_2) due to convective acceleration is $P_1 - P_2$ or ΔP_1. A is the anatomic cross-sectional area of the trachea and $\dot{V}$ the flow. Mean sectional velocity, $\bar{v}_1$, equals $\dot{V}/A$. K equals 1 in this case. Below is shown a hypothetical velocity profile (dotted line) in which the velocity is shown distributed entirely in the center of the stream. Flow in this case, v, equals the flow, $\dot{V}$, in the above example. Anatomic area, A, is also the same. The velocity profile, however, as depicted by the velocity vectors, is quite different with high velocities in the center of the stream through the area, a, and zero velocity everywhere else. The effective cross-sectional area, a, for this case is smaller than the anatomic area, A, which was the effective area in the above case. The resultant mean sectional velocity, $\bar{v}_2$, is greater than that in the uniform case, $\bar{v}_2 > \bar{v}_1$. It follows that the pressure drop due to convective acceleration is also greater, $\Delta P_2 > \Delta P_1$. Therefore, K must be greater than 1 in this nonuniform case. Had the velocity profile in this last example been assumed blunt, both the velocity and the pressure drop due to convective acceleration would have been underestimated. The fitting constant, K, corrects for such variations in velocity profile.

convective pressure drop can be calculated for each flow, it is possible to derive a flow versus frictional pressure drop curve by subtracting the convective term from the measured total pressure drop. Such a plot would be of value, since in the clinical evaluation of airway disease one is primarily interested in the frictional losses of the flow.

The convective pressure drop may be calculated if *1*) alveolar gas velocity is assumed to be zero, *2*) the tracheal cross-sectional area is known, *3*) the gas velocity distribution across the trachea is defined, and *4*) total respiratory flow is known. The convective acceleration pressure drop, $\Delta P_{C.A.}$ in cm H_2O, may then be computed from the formula:

$$\Delta P_{C.A.} = \frac{K\rho\bar{v}^2}{2g}, \qquad [1]$$

where ρ is the gas density, g the acceleration of gravity, $\bar{v}$ the average gas velocity across the trachea, and K a constant related to a spatial integration of gas velocity over the cross-section of the trachea. The value of K is equal to unity for a perfectly blunt velocity profile and equal to 2 for a parabolic profile (9). In the general case K will vary somewhere between these values. The theory evaluating K is somewhat involved (9) and fortunately, in anticipation of the results to be presented, need not be explored here. We may compute $\bar{v}$ from the formula:

$$\bar{v} = \dot{V}/A^1, \qquad [2]$$

where $\dot{V}$ is the flow at the mouth in centimeters^3 per second and A is the tracheal cross-sectional area in centimeters^2.

To summarize, the instantaneous pressure drop from alveolus to trachea, ΔP_{A-T}, is the sum of two pressure drops:

$$\Delta P_{A-T} = \Delta P_{(friction)} + \Delta P_{C.A.} \qquad [3]$$

$$= \Delta P_{(friction)} + \frac{K\rho\bar{v}^2}{2g}. \qquad [4]$$

A knowledge of K would permit computation of the convective term and, hence, calculation of $\Delta P_{(friction)}$. The importance of assessing the tracheal velocity profile is illustrated in Figure 1.

METHODS

Isovolume pressure-flow curves. Respiratory gas flow was measured with a concentric cylinder flowmeter (10). Respiratory volumes were recorded by a Krogh spiro-

TABLE I

Clinical data on subjects

Subject	Age	Height	Vital capacity	Max. mid-expir. flow*	Total lung cap.	RV/TLC†	Flow resistance‡
	yrs	*cm*	*L*	*L/sec*	*L*	%	*cm H_2O/L/sec*
Normal							
1	30	168	4.2	2.8	6.3	33	2.0
2	35	178	4.5	3.5	5.7	21	1.2
3	36	174	5.5	4.5	7.7	28	0.6
4	39	169	4.1	4.5	6.4	39	2.2
5	50	173	3.5	2.9	6.9	50	2.2
6	53	179	5.4	2.9	7.1	25	1.0
Emphysematous							
1	31	175	2.7	0.3	7.0	63	3.9
2	34	168	5.0	0.9			3.3
3	41	166	4.1	1.7			2.0
4	42	170	4.0	1.3	6.7	38	3.7
5	44	165	3.0	0.8			12.9
6	60	178	3.0	0.6	7.0	68	6.6
7	60	170	4.3	2.1			3.5
8	60	163	3.8	1.3	7.2	55	3.6
9	61	168	3.6	1.1			4.1
10	63	178	3.6	2.2	7.8	53	2.5
11	63	173	4.1	0.9			3.7

* Reference 20.
† RV = residual volume; TLC = total lung capacity.
‡ Measured at 1 L per second during inspiration in tidal volume range. Relates total pressure drop (alveolus to mouth) to flow.

meter, the displacement of which was measured by an angular differential transformer.

After skin anesthesia, a blunt 19-gauge needle with the tapered end cut off at right angles was introduced into the trachea 2 to 3 cm below the cricoid cartilage and placed under fluoroscopic vision. Lateral tracheal gas pressure was recorded from this needle.

Intrathoracic pressure was estimated from an air-filled balloon (11) placed in the lower esophagus. The pressure drop from balloon to blunt tracheal needle was plotted against corresponding respiratory flow on an oscilloscope. In a given run only pressure (P) and flow ($\dot{V}$) points at a specific lung volume were plotted. The difference between balloon and lateral tracheal pressure can be used to represent the pressure drop from alveolus to trachea if one neglects tissue friction[2] and also corrects for the pressure of lung elastic recoil. An isovolume pressure-flow curve automatically corrects for the elastic recoil since its zero flow intercept is the static retractive force of the lung at that volume. These isovolume pressure-flow ($P\dot{V}$) curves were recorded in 6 normal and 11 emphysematous subjects. Table I lists pertinent clinical data for the subjects. Thoracic gas volumes were measured by a volume displacement body plethysmograph (12). All volumes are expressed at ambient temperature and pressure saturated with water vapor ($ATPS$). The details of obtaining isovolume $P\dot{V}$ curves are described elsewhere (6, 13).

Tracheal velocity. Measurement of tracheal velocity was based on the Pitot principle which states that the velocity at a point in a stream may be estimated by measuring the difference between the lateral presssure and the impact or stagnation pressure of the stream. This is expressed in the relationship:

$$v = \sqrt{2g\Delta P/\rho}, \qquad [5]$$

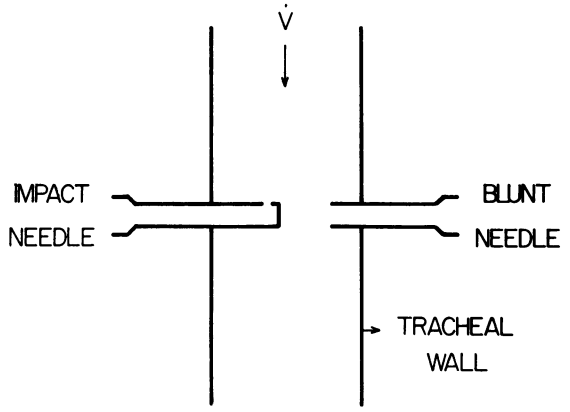

FIG. 2. ARRANGEMENT OF NEEDLES FOR ESTIMATING TRACHEAL VELOCITY. The blunt needle samples lateral tracheal gas pressure. The impact needle with a side opening is directed into the flow and samples the impact or stagnation pressure of the stream.

[1] This equation assumes that the pressure drop from trachea to mouth remains small compared to ambient pressure.

[2] Variously called the tissue component of total pulmonary flow resistance or tissue viscous resistance.

TABLE II

Expiratory velocity (cm per second) at flow of 2 L per second *

Subject	Centerline velocity	Velocity 1 mm from tracheal wall
A	2,580	2,370
B	1,500	1,740
C	1,150	1,440
D	880	640
E	1,350	1,400

* These velocities have not been corrected for the 30 per cent overestimation by Pitot tube.

where v is velocity in centimeters per second and ΔP the difference between lateral and impact pressures. In the five cases in which tracheal velocity was measured, a second needle with a side opening was introduced at the same tracheal level as the blunt needle. Simultaneous lateral and impact pressures were recorded from the blunt and side-opening needles on separate matched pressure transducers. The experimental arrangement is shown schematically in Figure 2. The difference between these pressures was determined electrically and used to compute velocity from the above expression. The dynamic response of the system (needles, connecting tubing, and gauges) was flat to ±5 per cent through 10 cycles per second.

The velocity distribution across the trachea was determined by sampling impact pressures along the tracheal radius at varying flows. The position of the impact needle was adjusted under fluoroscopic vision. The blunt needle was kept stationary. The velocity profile of the trachea was evaluated for inspiratory and expiratory flows ranging from 0.5 to 4.5 L per second.

The accuracy with which this modified Pitot tube estimated velocity was tested in a plastic tube of known radius. A subject breathed through the tube, generating various flows. Velocity was determined at carefully measured points along the tube radius by using the same needles employed in the human studies. The total flow based on these velocity determinations was calculated by arithmetic integration and compared to the corresponding flow recorded by the respiratory flowmeter. Over a wide range of flows the Pitot tube used in this study was found to overestimate velocity by approximately 30 per cent. This measurement error affected the magnitude of velocity noted but not the pattern of the velocity distribution across the trachea.

The dimensions of the trachea were obtained from anterior-posterior and lateral radiographs. The trachea cross-section most nearly approached an ellipse. Major and minor radii corrected for X-ray beam divergence were determined for the site of measurement and used to calculate tracheal cross-sectional area.

RESULTS

Tracheal velocity profile. Table II records expiratory velocities in the five subjects at two sites in the trachea, centerline and a point ap-

proximately 1 mm from the tracheal wall. These measurements were made at a flow of 2 L per second. In each case the velocity near the wall is approximately equal to that at the center. In three subjects wall velocity slightly exceeds centerline velocity, presumably representing random measurement error. However, the mean values of expiratory velocity for the entire group at the centerline and wall are quite similar, 1,492 and 1,518 cm per second, respectively. Velocities at intermediate sites were similarly close to centerline velocity. The values during inspiration showed the same pattern. These data indicate that under the conditions of this study and at the flows studied, the tracheal velocity profile during inspiration and expiration is essentially blunt. It seems reasonable to assume that K is equal to 1. Thus, average gas velocity across the trachea, $\bar{v}$, may be calculated by dividing flow at the mouth, $\dot{V}$, by tracheal cross-sectional area, A. Then,

$$\Delta P_{C.A.} = \frac{\rho \dot{V}^2}{2gA^2}. \qquad [6]$$

Table III estimates the error resulting from the assumption of a blunt profile. The measured expiratory centerline velocity was first corrected for the 30 per cent overestimation by the Pitot tube determined from the model study. This corrected velocity was then multiplied by the tracheal area derived from radiographs to give calculated flow. All measurements were made at 2 L per second. The assumption of a blunt profile overestimates flow in four of the five subjects and leads to an average overestimation of about 10 per cent. These estimates assume the tracheal cross-sectional area measurements to be accurate and the pressure drop from trachea to flowmeter small (cf. footnote 1).

Isovolume P V̇ curves. Figure 3 presents isovolume $P\dot{V}$ curves of the lower airway in a normal subject. The solid lines define the relationship of flow to the total pressure drop from esophageal balloon to lateral tracheal pressure tap. The dashed curves in the figure indicate the effect of subtracting the convective acceleration term, $\Delta P_{C.A.}$, and represent the frictional pressure drop versus flow behavior of the lower airway. The tendency for frictional flow resistance to decrease with increasing lung inflation is seen. Figure 4 from an emphysematous subject shows the decrease in maximal expiratory flow and increase in expiratory and inspiratory resistance of the lower airways characteristic of this disease. At these low flows the convective pressure drop is small compared to the large frictional drop and is not shown.

Table IV presents pertinent data on 33 isovolume $P\dot{V}$ curves obtained in this study. Included is the total alveolus to trachea pressure drop at maximum expiratory flow. In no case was maximum expiratory flow associated with a pressure in excess of 18.3 cm H_2O. The resistance values in this table are for the lower airway.

DISCUSSION

Theoretical estimates based on the critical Reynolds number indicate that turbulence probably occurs in the trachea at flows slightly less than 0.5 L per second (14). Recent studies of bronchial casts by Dekker (15) and West and Hugh-Jones (16) support these estimates. Flows below 0.5 L per second were not carefully evaluated in the present study. Moreover, the present data do not identify the type of flow producing the nearly blunt profile found. It could result from either turbulent or laminar flow.

TABLE III

Estimate of error in assuming blunt profile

Subject	Expiratory centerline velocity		Tracheal area	Calculated flow	Measured flow
	Measured	Corrected *			
	cm/sec		*cm²*	*L/sec*	*L/sec*
A	2,580	1,806	1.22	2.20	2.0
B	1,500	1,050	2.89	3.03	2.0
C	1,150	805	2.65	2.13	2.0
D	880	616	2.61	1.60	2.0
E	1,350	945	2.22	2.08	2.0

* Corrected for 30 per cent overestimation by Pitot tube used.

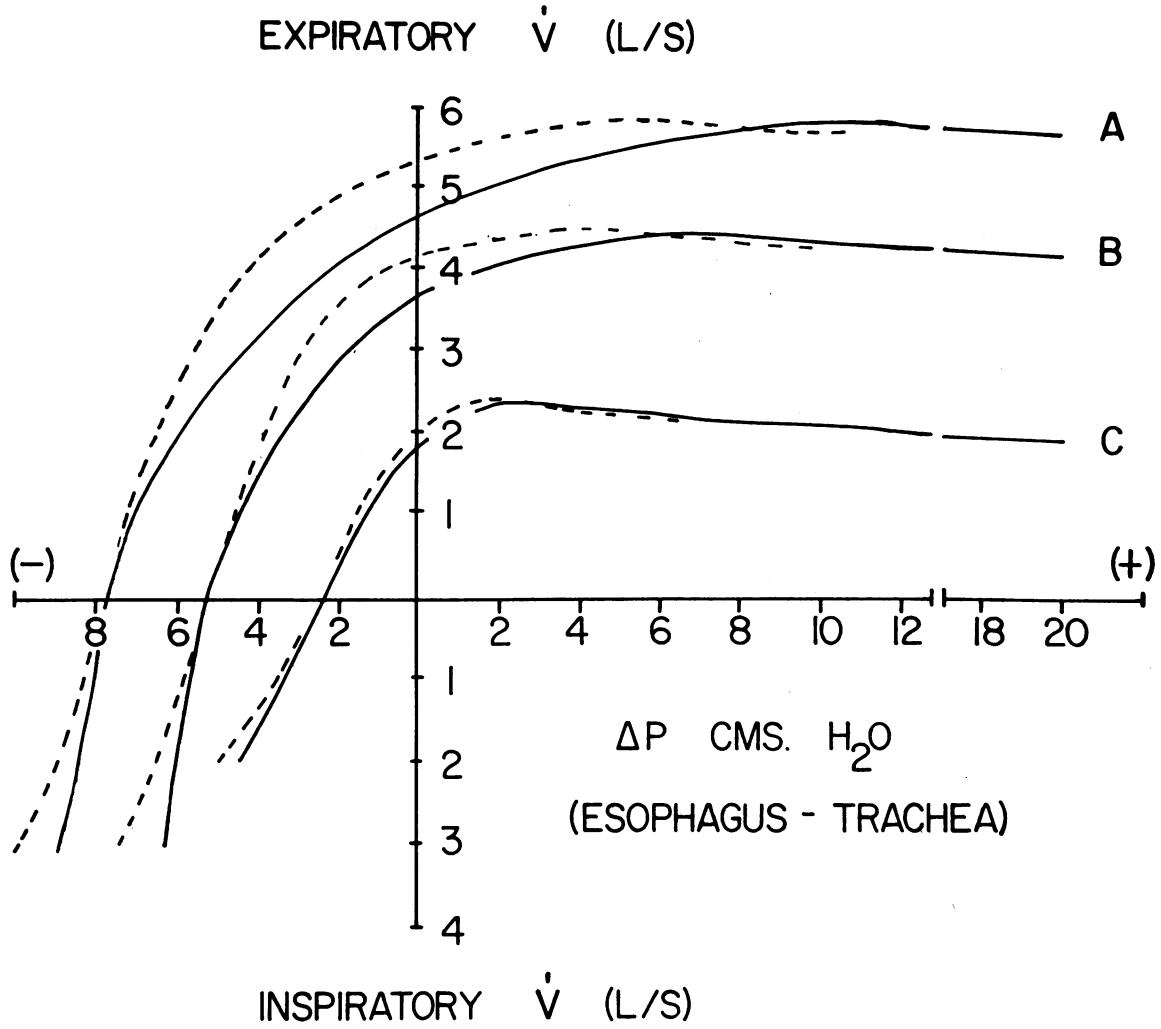

FIG. 3. LOWER AIRWAY ISOVOLUME PV̇ CURVES FOR NORMAL SUBJECT 2. Curves A, B, and C measured at 49, 44, and 28 per cent of vital capacity, respectively. Solid curves relate total pressure drop to flow and dashed curves relate frictional pressure drop to flow. Flow given in L per second (L/S).

The important observation is that a nearly blunt profile appears to obtain in the trachea in man. This greatly simplifies correction of the lower airway $P\dot{V}$ relationships for the convective acceleration pressure drop. It does not necessarily follow, however, that the velocity profile is blunt in the smaller airways.

The general contours of the total pressure versus flow curves of the lower airway are similar to those previously published for the entire airway (2, 13). The effect of correcting the lower airway $P\dot{V}$ curves for the convective term is seen in Figure 3. The contours of the curves relating frictional pressure drop to flow (dashed lines) are quite similar to those relating total pressure drop to flow (solid lines) until flows of about 2 L per second are reached. Since airway resistance is defined as the ratio of airway pressure drop to flow, it is apparent that failure to correct for the convective acceleration pressure drop leads to an overestimation of expiratory and an underestimation of inspiratory frictional resistance. Mead (7) has questioned whether this factor may explain in part the reported differences in resistance between the respiratory phases. The present data in normal subjects corrected for convective acceleration showed lower airway inspiratory frictional resistance to be approximately 85 per cent of expiratory resistance at 1 L per second. Owing to the marked alinearity of the expiratory

limbs of the isovolume $P\dot{V}$ curves, this difference becomes even greater at higher flows. In the emphysematous subjects, the inspiratory frictional resistance of the lower airway averaged 67 per cent of the expiratory resistance.

The absolute value of the convective acceleration term is not great. In a trachea of 1 cm radius, $\Delta P_{C.A.}$ equals 0.5 cm H_2O at 2 L per second and 3.2 cm H_2O at 5 L per second. However, the convective term may constitute a sizeable portion of the total lower airway pressure drop, particularly in normal subjects. For curve A, Figure 3, it amounts to 17 and 34 per cent of the total drop at expiratory flows of 1 and 3 L per second, respectively. Failure to consider the convective term could lead to errors in estimating the magnitude of small changes in frictional airway resistance induced by various experimental procedures, such as inhalation of cigarette smoke and bronchodilator drugs.

There are certain assumptions in the correction for convective acceleration in this study. First, it is assumed that tracheal cross-sectional area at the site of pressure measurement, i.e., in the extrathoracic trachea, remains constant throughout the breathing maneuvers employed. The recent work of Dekker and Ladeboer (17) supports this assumption. Utilizing roentgenographic techniques, they noted no change in the dimensions of the upper cervical trachea during

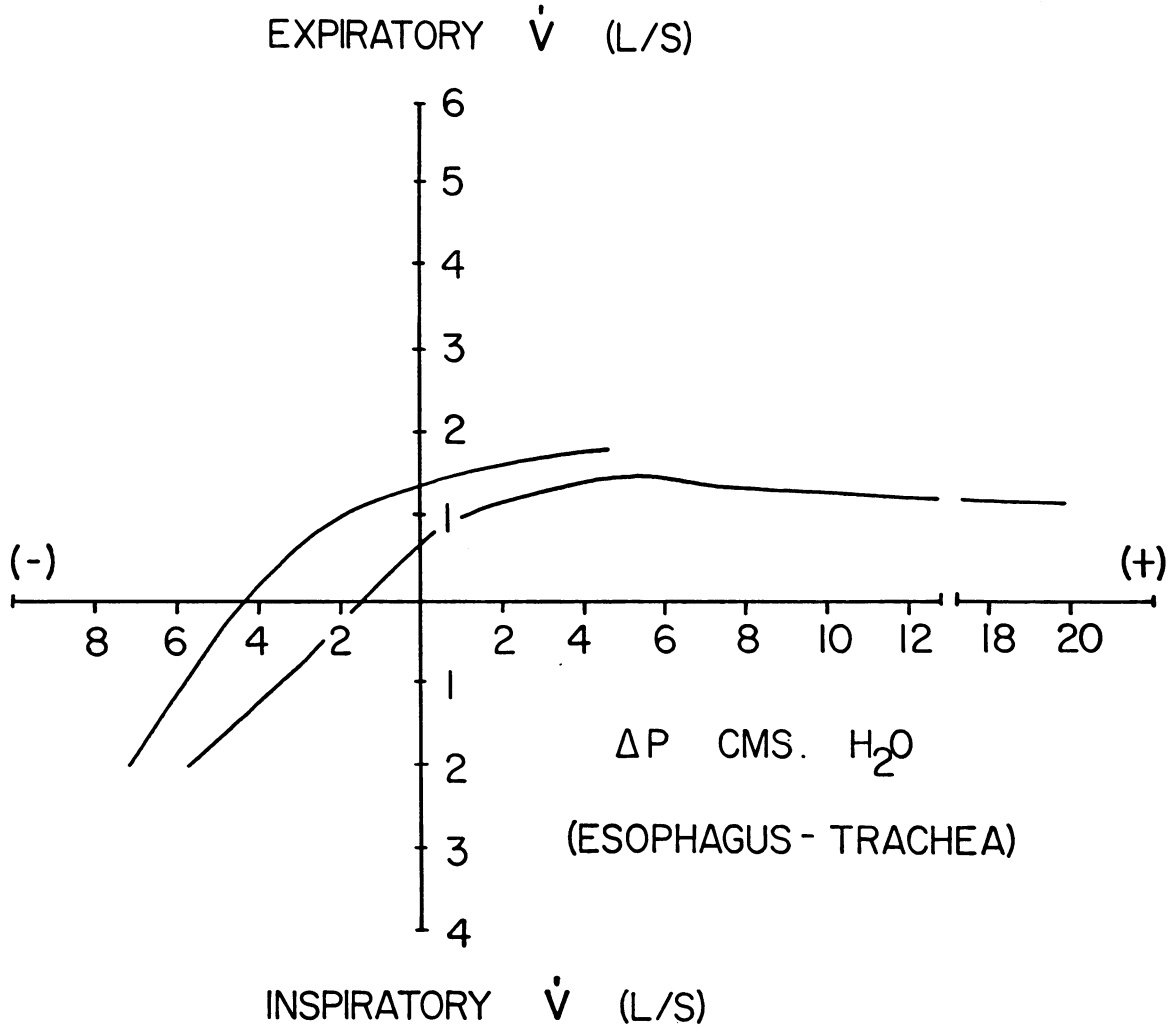

FIG. 4. LOWER AIRWAY ISOVOLUME PV̇ CURVES FOR EMPHYSEMATOUS SUBJECT 8. Top curve was measured at 80 per cent of vital capacity and has no maximum. Lower curve measured at 52 per cent of vital capacity. Curves relate total pressure drop to flow in L per second (L/S).

TABLE IV

Lower airway isovolume $P\dot{V}$ curves

Subject	Curve no.	Lung inflation*	Max. expir. flow	Pressure at max. expir. flow†	Resistance‡	
					Expiration	Inspiration
		% VC	L/sec	cm H_2O	cm H_2O/L/sec	
Normal						
1	a	47	4.0	4.0	0.9	0.7
	b	44	3.4	3.7	0.8	1.0
	c	22	1.9	3.0	1.2	0.9
2	a	49	5.9	10.5	0.8	0.8
	b	44	4.6	6.8	0.8	0.6
	c	33	3.3	2.6	0.7	0.6
	d	28	2.4	2.4	1.0	1.0
	e	20	0.9	2.1	3.0	1.7
3	a§	55	7.6	16.3	0.3	
4	a	54	5.4	8.2	0.2	0.1
5	a	43	3.7	10.0	1.5	0.9
6	a§	57	5.6	18.3	0.5	
	b§	50	4.1	7.8	1.1	
	c§	41	3.4	3.8	1.1	
Emphysematous						
1	a	61	1.1	3.0	3.3	2.2
	b	46	0.9	0.0	4.0	2.0
2	a	64	1.8	5.8	3.8	2.4
	b	54	1.6	2.0	3.0	2.0
3	a	63	3.3	6.7	1.2	1.0
4	a	38	1.8	13.5	3.6	2.9
	b	35	1.5	2.5	4.2	2.4
	c	13	0.7	3.7	5.0	4.8
5	a	66	0.9	0.8	8.6	6.0
	b	53	0.5	2.0	13.2	6.0
6	a	53	1.3	4.0	8.3	4.1
7	a	63	3.0	4.0	2.4	2.6
8	a	52	1.7	5.8	2.5	2.0
9	a	67	1.6	5.5	3.8	3.0
	b	64	1.3	2.0	5.1	2.7
10	a§	53	3.3	1.1	1.2	
	b§	33	2.0	1.4	2.3	
11	a	32	2.0	2.4	1.8	1.9
	b	22	0.9	−0.8	3.0	2.1

* Volume at which curve measured is expressed as per cent of *VC*. 100 per cent would equal the point of maximal inflation.

† Pressure refers to difference between esophageal balloon and lateral tracheal pressure.

‡ Resistance calculated from pressure drop from balloon to lateral tracheal tap and measured at 1 L per second.

§ Only expiratory limb obtained.

such violent respiratory maneuvers as cough and artificial wheezing. We have observed fluoroscopically the region of the trachea where our measurements were made during the respiratory maneuvers employed. No change in anterior-posterior diameter was detected. If tracheal area changed with increasing flows, the flow versus velocity relationships would not be linear. They were linear for the flows studied. Nor was there a consistent tendency for inspiratory velocity to differ from expiratory velocity at the same flow levels. This indicates that tracheal area was the same during both inspiration and expiration.

It is also assumed that the velocity profile remains blunt at all flows. Our data support this assmuption, but we have no data for flows under 0.5 and over 4.5 L per second.

Errors in estimating tracheal cross-sectional area could also have a significant effect on the data, since, as can be seen from Equation 6, $\Delta P_{C.A.}$ varies inversely with the fourth power of the radius. There is a tendency for the trachea to narrow in the region where our measurements were made which adds to the difficulty of obtaining exact diameter measurements. Also, as Harris has shown (18), anterior-posterior tracheal diameter is affected by the attitude of the head and somewhat by the degree of lung inflation. These variables were not rigidly con-

trolled when we obtained our tracheal radiographs. Hence, the measured tracheal areas may differ somewhat from those existing during the experimental procedures. This could bear on the accuracy of the calculations presented in Table III.

In the study of pulmonary aerodynamics one is frequently interested in assessing the pressure gradient related to the frictional losses along the intrathoracic airways. In this study the resistance of the extrathoracic airway was excluded by measuring lateral tracheal pressure. Frictional losses of the lower airway were then determined by calculating and subtracting the value of the convective term from the total lower airway pressure drop. Another approach would have been to measure the pressure difference between alveolus and impact tracheal needle, which would have yielded the frictional $P\dot{V}$ relationships directly.

In routine clinical work it is not feasible to perform tracheal punctures. Resistance is frequently calculated from the $P\dot{V}$ relationships measured by an esophageal balloon and lateral oral pressure tap. If the diameter of the tube containing the oral tap is known and the profile is assumed to be blunt, the convective term can be handled as in this study. Similarly, an impact oral tap might be employed, again yielding the frictional resistance directly. However, it must

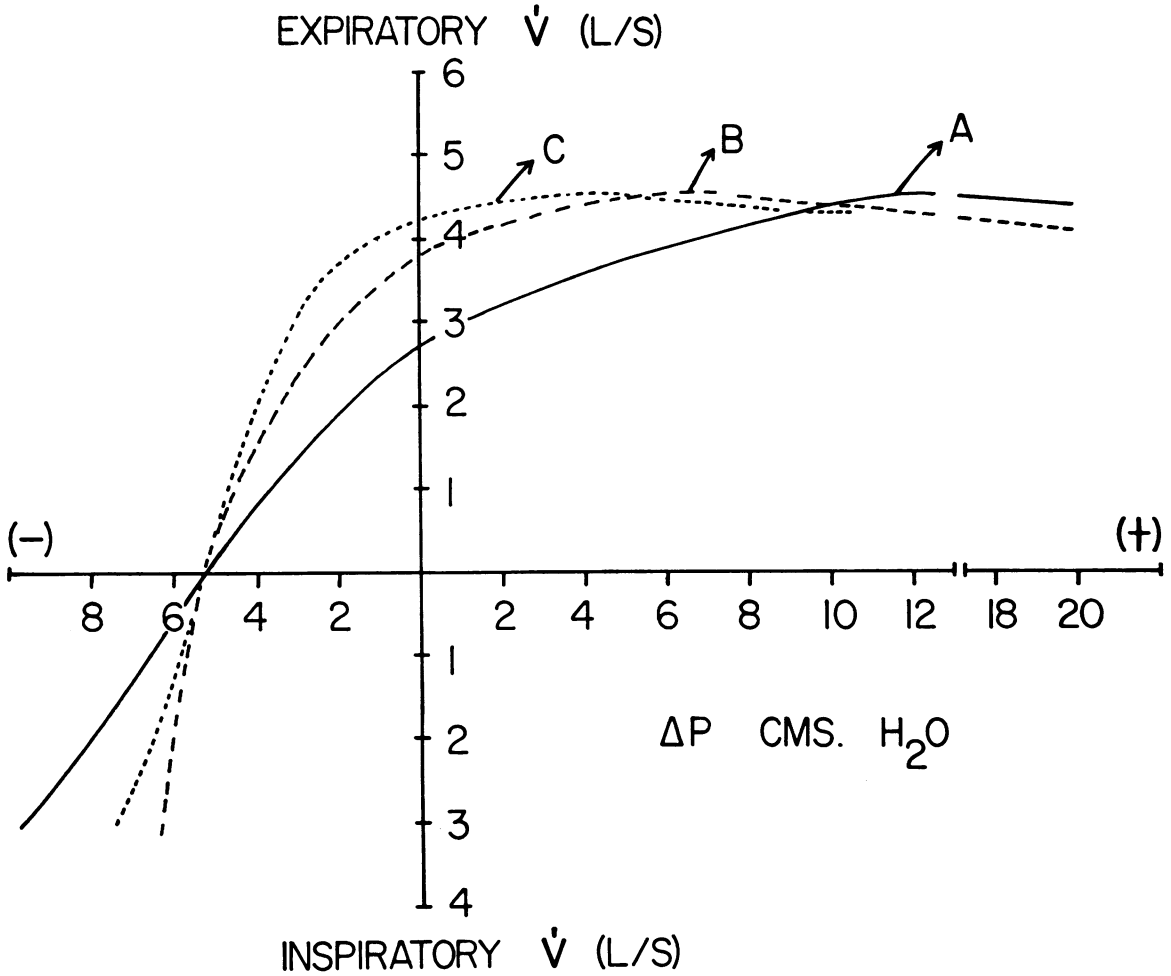

FIG. 5. MAJOR COMPONENTS OF TOTAL AIRWAY PRESSURE DROP IN NORMAL SUBJECT 2. All curves measured at 44 per cent of vital capacity. Flow is in L per second (L/S). Curve A relates flow to total pressure drop from esophageal balloon to lateral oral pressure tap. Curve B relates flow to pressure drop between esophageal balloon and lateral tracheal pressure tap. Curve C is curve B with calculated convective acceleration pressure drop subtracted.

be remembered that in this case one is also measuring the variable frictional resistance of the upper airway.

Figure 5 summarizes the major components of the total airway pressure drop when measured by an esophageal balloon and lateral oral pressure tap in a normal subject. Curve A relates flow to the pressure drop between esophageal balloon and lateral oral pressure tap. Curve B relates flow to the pressure drop from esophageal balloon to lateral tracheal pressure tap. Curve C is curve B corrected for the pressure drop due to convective acceleration and, hence, relates flow to the frictional pressure drop from esophagus to trachea. Expiratory resistance at 1 L per second for the total airway can be seen from $P\dot{V}$ curve A to be 1.6 cm H_2O per L per second. Calculation of lower airway resistance from curve B yields a value of 0.7 cm H_2O per L per second. In contrast, calculation of lower airway resistance from curve C yields a value of 0.6 cm H_2O per L per second, the true value for the frictional resistance of the intrathoracic airways. Obviously, tissue friction has been neglected in these calculations.

In a previous study (6), the frictional resistance of the upper airway was found to account for approximately 45 per cent of total airway resistance in normal and 18 per cent in emphysematous subjects. The values for total airway resistance in that study included the convective term. Correcting these data for the convective drop indicates that the frictional resistance of the upper airway actually accounts for about 49 per cent of the total frictional resistance of the airway in normal and 19 per cent in emphysematous subjects.

It should be pointed out that the body plethysmograph as conventionally used (12, 19) yields the total airway frictional resistance. If, however, one measures the pressure drop from alveolus to mouth in the body plethysmograph, the pressure drop due to convective acceleration must be taken into account.

SUMMARY

The gas velocity profile in the trachea was evaluated in five conscious subjects by simultaneous direct measurement of lateral and impact tracheal pressures. Velocity at various points along the tracheal radius was measured by a modified Pitot tube. The profile was found to be nearly blunt during both inspiration and expiration for flows ranging from 0.5 to 4.5 L per second. Isovolume pressure-flow curves of the lower airway (alveolus to trachea) were obtained in 6 normal and 11 emphysematous subjects. From a knowledge of the tracheal velocity profile it was possible to separate the lower airway pressure drop into two components, one related to the frictional losses in the gas and one to the convective acceleration of the gas (Bernoulli effect). Failure to consider the pressure drop due to convective acceleration leads to an overestimation of expiratory frictional resistance and an underestimation of inspiratory frictional resistance, particularly in normal subjects.

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