ANALOG COMPUTER SIMULATION OF
MAXIMUM EXPIRATORY FLOW LIMITATION

by

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ABSTRACT

This paper extends previous modeling work in our laboratory on the simulation of the panting maneuver executed in a body plethysmograph [Golden, et al. (1973)] to the simulation of a wider range of pulmonary function tests and, in particular, the maximum expiratory flow-volume (MEFV) curve. The simulation is implemented on an analog computer for ease of parameter manipulation; the effects of changes in several model parameters (such as large and small airway resistance, and airway and lung compliance) on the MEFV curve are discussed. The model structure presented here is much simpler than the ventilatory system models of Fry (1968) and Pardaens, et al. (1972) and yet yields the same information in a diagnostic sense.

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1. INTRODUCTION

A lumped parameter approach to modeling pulmonary airway dynamics is advantageous in that it reduces the complexity of analysis and frequently allows the interpretation of clinical data that are generally obtained via measurements on the system as a whole (i.e., the measurement is in itself a summation of responses from an inherently distributed system and only certain measurements are feasible from a clinical standpoint).

Numerous models of airway dynamics have appeared in the literature, many differing considerably in their ability to describe the mechanics of this system. One would expect that for a well-formulated model, both model complexity and dimensionality would increase with the amount of anatomical and functional detail incorporated into the model. There are two general approaches to this modeling problem: first, a "fundamental" approach wherein the investigator attempts to describe lung-airway mechanics in considerable detail from a fluid mechanical standpoint, and second, a "functional" approach wherein the more general aspects of the lung-airway behavior are considered. Typical of models in this latter category is the model of Rohrer (1915) who first described the relationship between alveolar pressure (P_{ALV}) and airflow at the mouth

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($\dot{V}_{A0}$) (see equation (1)). While this model adequately describes inspiratory flows of less than 5 liters/sec, it fails to predict expiratory flow limitation due to airway compression [Bouhuys and Jonson (1967)]. More sophisticated models have since been developed that include lumped upstream and downstream airway resistances as well as peripheral airway compliance.

Thus, "fundamental" models as described above usually have an entirely different set of modeling objectives than the more clinically oriented goals of "functional" models. Given the same general clinical objective, for example simulating (a) pulmonary function data obtained from the execution of a panting maneuver in a body-plethysmograph or (b) patient data obtained from a forced-expiratory maneuver, the approaches taken would differ considerably:

(1) The "fundamental" model would most probably take into account the morphometry of the bronchial tree. Weibel (1963) has developed a symmetrical model that assumes a regular dichotomous branching and this study has been utilized in other modeling studies of lung-airway dynamics [e.g., Fry (1968) and Pardaens, et al. (1972)].

(2) The "functional" model would contain rather gross lumpings of pulmonary resistance and compliance subject to the constraints that the parameters have a physical basis and the fit to observed subject data is adequate. The overriding concern here is the ability to specify a model structure that makes sense physically, and yet with only a relatively small number of parameters is able to adequately mimic patient data [e.g., Golden, et al. (1973), Feinberg, et al. (1970), Pride, et al. (1967), and Yamabayashi, et al. (1970)].

Here the "fundamental" model would be of considerable use in studying

(for a given set of parameters) the basic fluid mechanical aspects of the air-flow in the bronchial tree. It would have utility as a teaching aid and a research tool since parameter manipulation could be made at any level in the tree and effects on respiratory variables observed. The rather large number of parameters involved, however, preclude the use of parameter identification techniques [e.g., Graupe (1972)] for determination of model parameters for a given patient for diagnostic purposes. These latter objectives can possibly be achieved using an adequately specified "functional" model containing many fewer model parameters. Viewed in this pragmatic context, additional model complexity is warranted only if it lends additional diagnostic insight into lung-airway behavior.

This paper extends the simple lumped parameter "functional" model of Golden, Clark, and Stevens (1973) [which simulates the special case of the panting maneuver executed in a body plethysmograph] to the simulation of a wider range of pulmonary function tests that include the production of maximum expiratory flow-volume (MEFV) and isovolume pressure-flow (IVPF) curves. For an MEFV maneuver, the subject expires with maximum effort from total lung capacity to residual volume. A plot of flow at the mouth vs lung volume during this maneuver gives the MEFV curve. For an IVPF maneuver, the subject expires from total lung capacity with an effort that is sequentially increased until maximum effort is achieved. A plot of flow at the mouth vs alveolar pressure at a specific lung volume with a variable expiratory effort gives the IVPF curve. The model is programmed on an analog computer which allows fast realization and convenient parameter manipulation. The results indicate the effi-

cacy of using a lumped-parameter, four resistance model to mimic the full range of dynamic pulmonary function tests. In addition to the four resistance parameters, the airway and lung compliance curves must also be specified. Thus, the simulation indicates the influence of a number of airway lung parameters on the nature of expiratory flow limitation.

II. MODELING ASPECTS

Figure 1 shows the lumped parameter model utilized for the study of respiratory airway dynamics. Briefly, the lumped alveolar space (with volume V_L) is connected to the mouth by a single airway of variable resistive nature. The alveolar space and most of the airway are surrounded by the pleural cavity which is assumed to be at a pressure (P_{PL}) relative to atmospheric pressure. The airway is divided into three segments; the large, rigid part of the airway is modeled by a nonlinear "Rohrer resistor" characterized by the following pressure-flow relation:

$$P = K_1 \dot{V}_{A0} + K_2 \dot{V}_{A0}^2 \quad (1)$$

Here, $K_1 \dot{V}_{A0}$ is the laminar flow term containing the viscosity of the gas and the tubular geometry, while the second term, $K_2 \dot{V}_{A0}^2$, is an additional resistance term which becomes significant at higher flow rates where turbulent flow is likely to occur. The mid-section of the airway is assumed to be normally distended by the positive pressure gradient across the wall of the airway. However, when this transmural pressure passes from positive to negative (which can occur during expiration), the airway tends to collapse. So the mid-airways of the ventilatory system are modeled by a pressure-dependent resistance (R_C) characterized as a cylinder of constant length whose radius, and hence volume

(V_C) , varies with transmural pressure (P_{TM}). Poiseuille's Law is then used in the development of the following expression for the resistance of the collapsible segment:

$$R_C = f(V_C) = K_3(K_4/V_C)^2 \quad (2)$$

Here K_3 and K_4 are weighting constants; K_3 determines the overall magnitude of R_C and K_4 determines the relative volume of the collapsible segment at which R_C will be the dominant component of overall airway resistance. Finally, the small part of the airway represents the smaller peripheral airways located within the lung tissue. These airways are held distended by the overlying lung tissue at high lung volumes, but tend to collapse with the lung tissue at low lung volumes. Thus the small part of the airway is modeled by a nonlinear resistance whose magnitude is a function of lung volume (Figure 3):

$$R_S = f_1(V_L) \quad (3)$$

The electrical analog of this model is shown in Figure 2. Using Kirchoff's voltage and current laws, the following equations are obtained:

$$P_{ALV} = P_{EL} + P_{PL} \quad (4)$$

$$0 = P_{EL} - P_{TM} + R_S \dot{V}_L \quad (5)$$

$$0 = P_{TM} + P_{PL} + (R_C + K_1 + K_2 |\dot{V}_{A0}|) \dot{V}_{A0} \quad (6)$$

$$V_C(t) = V_C(0) + \int_0^t (\dot{V}_{A0} - \dot{V}_L) dt \quad (7)$$

$$V_L(t) = V_L(0) + \int_0^t \dot{V}_L dt \quad (8)$$

Here, P_{TM} is a nonlinear function of V_C (Figure 4):

$$P_{TM} = f_2(V_C) \quad (9)$$

and lung elastic recoil (P_{EL}) is a nonlinear function of V_L and P_{PL} , as determined by maneuver effort (see Figure 5):

$$P_{EL} = f_3(V_L, P_{PL}) \quad (10)$$

Thus, equations (1) through (10) comprise the mathematical model for the ventilatory system. These equations represent an extension of the model of Golden, et al. (1973) to allow for the simulation of vital capacity maneuvers and presentation of model data in the form of MEFV and IVPF curves. As previously mentioned, the model of Golden, Clark, and Stevens is capable of simulating quasi-static respiratory maneuvers such as the panting maneuver executed in a body plethysmograph for the measurement of total airway resistance (R_{AW}). The model structure presented in Fig. 1 is essentially identical to that utilized in previous studies [Golden, et al. (1973)]. However, the nature of some of the resistive and compliant model parameters were changed to accomplish the modeling objectives of this study. Specifically, in equation (2), the values of the weighting coefficients (K_3, K_4) were altered so as to produce a collapsible segment resistance [$R_C(V_C)$] that is more consistent with published results in the literature.* Small airway resistance R_S (equation (3)) was changed from a constant value (for an approximately constant lung volume) to a nonlinear function of lung volume (Fig. 3). The collapsible segment compliance curve (i.e., the relationship between transmural pressure P_{TM} and collapsible segment volume V_C) was altered from its highly stylized form in the previous model to the form shown in Fig. 4 which is in closer agreement with published data.† Similar changes were also made for the lung compliance curve

* Discussed in the following section.

† Ibid.

curve (i.e., the relationship existing between lung elastic recoil P_{EL} and lung volume V_L) as can be seen in Fig. 5 and equation 10. Also, pleural pressure P_{PL} is not constant as in the study of Golden, et al. (1973) but varies with time during the expiratory maneuver (Fig. 7).

III. COMPUTATIONAL ASPECTS

The mathematical model of pulmonary airway dynamics was programmed on an Electronics Associates, Inc. MiniAC analog computer system and was run at ten times real time to allow for the slow response time of the plotter. A block diagram of the computer program is given in Figure 6.

Typical resistance and compliance values for a normal subject were taken from the literature. Specifically, values for large airway resistance (K_1 and K_2) were adapted from Bouhuys and Jonson (1967) and Mead (1961). The values of K_1 and K_2 were taken to be $K_1 = 0.5 \text{ cm H}_2\text{O}/(\ell/\text{sec})$ and $K_2 = 0.2 \text{ cm H}_2\text{O}/(\ell/\text{sec})^2$. Values for collapsible airway resistance (K_3 and K_4) were also adapted from the literature. Mead, et al. (1967) found that increased large airway resistance had no discernible influence on maximal flow at volumes below 50% vital capacity (VC). Furthermore, Bouhuys (1974) states that the MEFV curve represents flows that correspond to plateaus on IVPF curves at lung volumes below about 80% VC. These results were approximated when values $K_3 = 0.6 \text{ cm H}_2\text{O}/(1/\ell \cdot \text{sec})$ and $K_4 = 0.125$ were utilized. The non-linear function for small airway resistance was adapted from data reported by Macklem and Mead (1967) and Bouhuys (1974) and is shown in Figure 3. R_S is small and approximately constant (equal to $0.3 \text{ cm H}_2\text{O}/(\ell/\text{sec})$) above 40% VC, but increases rapidly below 40% VC.

Physiological data published by Murtaugh, et al. (1971) and Hyatt and Flath (1966) was used in the formulation of the compliance curve for the collapsible segment (Figure 4). Likewise, pressure-volume data published by Colebatch, et al. (1973) was used in the formulation of the static lung compliance (P_{EL} static) curve (Figure 5). The dynamic lung compliance (P_{EL} dynamic) curve (Figure 5) was formulated using the static lung compliance curve and data published by Glaister, et al. (1973) and Clements, et al. (1961). The choice of lung elastic recoil was found dependent on respiratory maneuver.

The pleural pressure driving function was also found to vary with the particular respiratory maneuver. For the panting simulation, a high frequency (1.6 Hz), low volume (< 0.1 l) sinusoid, offset by a negative static pleural pressure was programmed to react with the static lung elastic recoil function. For the maximum expiratory maneuver, a filtered, two-break point temporal waveform (Figure 10a) and the dynamic lung elastic recoil function were implemented. This P_{PL} waveshape was adapted from patient data recorded in The Methodist Hospital Pulmonary Function Laboratory, Houston, Texas (Figure 7), as well as data reported by Milic-Emili, et al. (1964). In the MEFV maneuver, peak pleural pressure (due to expiratory muscular effort) is found to be highly variant from subject to subject due to normal physical variables (age, height, sex) and, in addition, training for the maneuver (Milic-Emili, et al. (1964)). This accounts for the significant variability in peak expiratory flow (for normal subjects) seen in the literature since flow is generally considered to be limited by peak pleural pressure in this range (Bouhuys (1974)). Figure 7 shows actual patient data for a representative untrained normal subject who

was able to generate a pleural pressure that is slightly in excess of +60 cm H_2O . Because of this high degree of variability, the choice of peak pleural pressure used in association with the driving waveform ($P_{PL}(t)$) for the maximum expiratory maneuver in the model is somewhat arbitrary. Therefore, a value of 100 cm H_2O (considered as an intermediate value) for peak pleural pressure was used in this study. To generate IVPF curves, the methods employed by Hyatt, et al. (1958) were mimicked. These involved sequentially increasing expiratory effort until maximum effort (and therefore pleural pressure) at that volume was achieved. So, in the simulation a ramp-to-plateau pleural pressure waveform (Figure 10b) was utilized. The slope of the ramp and the dynamic nature of the lung elastic recoil (with P_{EL} static and P_{EL} dynamic in Figure 5 indicating the extremes corresponding to minimum and maximum effort, respectively) were increased sequentially until maximum pleural pressure at that volume was achieved. Flow at the mouth and alveolar pressure were recorded for each case.

Table 1 shows the other values (arbitrarily chosen) that are used for the normal case.

IV. RESULTS

Figure 8 demonstrates the ability of the model to mimic the shallow panting maneuver performed in a body plethysmograph by normal and diseased patients. These results are very similar to those obtained in our previous studies which compared model and patient data (e.g., Golden, et al. (1973), Fig. 6). Each maneuver is driven by a sinusoidal pleural pressure that produces variations

of ± 5 cm H₂O in alveolar pressure. Each patient simulation is conducted at three different lung volumes (90%, 50%, and 10% vital capacity) and therefore three different values of lung elastic recoil. Since total airway resistance is reflected as the inverse of the absolute value of the slope of the $P_{ALV} - \dot{V}_{AO}$ curve (an increase in airway resistance produces a decrease in the absolute value of the slope), the curves indicate the volume actually lung recoil) dependent nature of total airway resistance due mainly to increased small airway resistance (Figure 3). Hysteresis, seen in some maneuvers, is due to parallel resistive-compliant nature of the airways. Figure 8a (the normal case) indicates the curve shape and the increase in total airway resistance with the decrease in lung volume. The small airway resistance becomes dominant at low lung volumes. Figure 8b indicates that tripled large airway resistance (approximately doubled total airway resistance) is accurately reflected at high and middle lung volumes. Again, at low lung volumes the increased small airway resistance is dominant and no discernible change is seen. Figure 8c indicates the results of the same magnitude of increased airway resistance as in Figure 8b, but in the small airways. This change was accomplished by a simple upward shift in the R_S curve (Figure 3) at all lung volumes. The simulation at 6 liters shows much less total airway resistance contribution than in Figure 8b. At 4 liters, the model also exhibits a small contribution to total airway resistance, but the combination of increased small airway resistance and decreased lung elastic recoil with decreased lung volume produces observable expiratory flow hysteresis, dynamic compression, and expiratory flow limitation. And, as in other cases, at 2 liters small airway resistance dominates all other effects.

The ability of the model to demonstrate the relationship between data presented in the form of MEFV and IVPF curves is shown in Figure 9. The IVPF curves tend to plateau at low lung volumes due to dynamic compression of the collapsible airway. At high lung volumes, the expiratory flow is limited by the pleural pressures that can be generated by contraction of the respiratory muscles. As Bouhuys (1974) indicates, points on the effort independent portion of the MEFV curve correspond to plateaus of the IVPF curves below approximately 80% vital capacity in normal subjects. Figure 9 thus demonstrates that the model is capable of providing a realistic simulation of this relationship between MEFV and IVPF curves.

Figures 10a and 10b show the temporal waveforms of selected parameters during the MEFV and IVPF maneuvers. The waveforms for pleural pressure, lung volume, and flow at the mouth are consistent with patient data (Figure 7) and data reported in the literature (Milic-Emili, et al. (1964)). The waveforms for collapsible segment volume and collapsible segment resistance are included to add insight into the dynamic nature of airway compression in the model. For the MEFV maneuver, V_C starts to decrease immediately, achieves its minimum value after the peak pleural pressure is attained, and increases slightly through the remainder of the maneuver due to the decrease in pleural pressure in the latter phases of the maximum expiratory maneuver. For the IVPF maneuver, V_C decreases throughout. R_C is inversely related to the square of V_C (as is shown in both maneuvers) and is most sensitive at low values of V_C .

The remaining figures demonstrate the results of parameter manipulation. Hyatt, et al. (1958) indicated and Mead, et al. (1967) demonstrated that if a

normal subject performed a maximum expiratory maneuver with various additional mouthpiece resistances, maximum flow is uninfluenced at lung volumes below at least 50% vital capacity. In addition, the added mouthpiece resistance increases the effective large airway resistance (R_{LAW}) and has the greatest effect at high lung volumes. This decreases peak flow and allows for the generation of a higher peak pleural pressure during maximum expiratory muscular effort (as shown by Mead, et al. (1967)). Airway compression and therefore limitation are unaffected so that the time duration of the maneuver remains approximately the same. The MEFV curves and pleural pressure driving waveforms for increased large airway resistance are shown in Figure 11. They show that with an increase in R_{LAW} and the pleural pressure waveform, an effort independent of the portion of the MEFV curve can be maintained.

Figure 12 shows the corresponding effect of an equal increase in small airway resistance (a simple upward shift in the R_S curve (Figure 3)) to approximate the results of the study by McFadden, et al. (1969). Small airway resistance can, of course, be altered while other resistance and compliance parameters are kept constant. Both total airway resistance and flow limitation are increased so that the peak pleural pressure and the time duration of the maneuver are also increased. The resulting MEFV curve demonstrates the representative effect of increased small airway resistance; peak flow is achieved early and then falls off rapidly producing a curve that is concave to the volume axis. This change in the form of the MEFV curve in simulated airway obstruction is consistent with published data (Bouhuys (1974)).

The results of changes in airway compliance are shown in Figures 13a

through 13c and agree with the results of Pardaens, et al. (1972). A change in the upper portion of the airway compliance curve appears to affect mainly the peak flow of the MEFV curve (Figure 13a), while a change in the lower portion of the airway compliance curve affects the slope of the MEFV curve in the flow limited region and can even produce gas trapping (Figure 13b). Gas trapping is the inability of the subject to expire to residual volume due to severe flow limitation. Figure 13c includes the combined effects of changes in upper and lower portions of the airway compliance curve.

Finally, Figure 14 shows the results of changes in lung compliance. The MEFV curves indicate simulations utilizing P_{EL} static, E_{EL} dynamic (normal), and P_{EL} dynamic (abnormal). Peak expiratory flow and rate of flow limitation are affected by changes in lung compliance as also reported by Pardaens, et al. (1972). Figure 14 demonstrates the relative importance associated with the use of dynamic rather than static lung compliance in the modeling of dynamic maneuvers.

V. DISCUSSION

The mathematical model presented by Golden, et al. (1973) has been extended in this paper to allow for the simulation of maximum expiratory tests as well as plethysmographic panting maneuvers. Initial conclusions having to do with the simulation of the panting maneuvers verify those results reported by Golden, et al. (1973). For example, increasing large airway resistance significantly increases total airway resistance but does not affect expiratory flow limitation; increasing small airway resistance has comparatively little effect on

total airway resistance, but significantly affects expiratory flow hysteresis and limitation; reduced lung elastic recoil (either at lower lung volumes in the normal or in disease states such as emphysema) has little effect on total airway resistance, but significantly affects expiratory flow limitation.

With regard to maximum expiratory maneuvers, the model can produce data that is in general agreement with data found in the literature. The MEFV curve represents flows that correspond to plateaus on IVPF curves at lung volumes less than approximately 80% vital capacity (Bouhuys (1974)). An increase in large airway resistance (as in Figure 11) is accompanied by corresponding increases in attainable pleural pressure during a maximum expiratory maneuver and that maximum flow is unaffected below at least 50% vital capacity (Mead, et al. (1967)). An increase in small airway resistance (Fig. 12) causes a decrease in peak flow and produces an MEFV curve that is concave to the volume axis (McFadden, et al. (1969)). Also, relatively small changes in lung and airway compliance (Figure 13) have significant effects on the shape of the resulting MEFV curves (Pardaens, et al. (1972)).

Thus, the results shown in this study indicate that our model is capable of simulating standard breathing maneuvers associated with the clinical assessment of pulmonary function. It is structurally much simpler than the models of Fry (1968) and Pardaens, et al. (1973) and yet yields the same information in a diagnostic sense. Being of simpler form it has fewer model parameters and potentially may be used in conjunction with parameter identification techniques to obtain a set of describing parameters for individual patients. This simulation can also be seen as a teaching aid to help in the understanding

of pulmonary ventilatory mechanics and is also directly extendable to a unified study of pulmonary mechanics in normal and disease states thereby providing a potentially useful tool for the interpretation of clinical pulmonary function tests.

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FIGURE CAPTIONS

- FIGURE 1 Lumped parameter model for the pulmonary ventilatory system (see text and Golden, et al. (1973)).
- FIGURE 2 Electrical equivalent model of the pulmonary ventilatory system (shown in Figure 1).
- FIGURE 3 Relationship between small airway resistance and lung volume for the normal case.
- FIGURE 4 Airway compliance for the normal case.
- FIGURE 5 Lung compliance (both static and dynamic) for the normal case.
- FIGURE 6 Block diagram for the analog computer system -- variable function generators (VFGs) include VFG₁ (transmural pressure (P_{TM}) as a function of collapsible airway volume (V_C)), VFG₂ (lung elastic recoil (P_{EL}) as a function of lung volume (V_L)), and VFG₃ (small airway resistance (R_S) as a function of lung volume (V_L)).
- FIGURE 7 Patient data for a typical normal case showing temporal waveforms (flow at the mouth ($\dot{V}_{AO}$), relative lung volume [lung volume (V_L) minus residual volume (RV)], and pleural pressure (P_{PL})) for the MEFV maneuver [recorded at The Methodist Hospital, Pulmonary Function Laboratory, Houston, Texas].

FIGURE 8 Simulated panting maneuvers at representative lung volumes (the driving pleural pressure waveform is a small amplitude sinusoid varying about a mean value).

- (a) Normal airway resistance values
- (b) Tripled large airway resistance ($K_1 = 1.5 \text{ cm H}_2\text{O}/\text{l}/\text{sec}$, $K_2 = 0.6 \text{ cm H}_2\text{O}/(\text{l}/\text{sec})^2$) representing an increase of $1.4 \text{ cm H}_2\text{O}/\text{l}/\text{sec}$ in total airway resistance at a flow of $1 \text{ l}/\text{sec}$.
- (c) Increased small airway resistance (a positive shift of $1.4 \text{ cm H}_2\text{O}/\text{l}/\text{sec}$ in the small airway resistance curve (Figure 3)) representing an increase of $1.4 \text{ cm H}_2\text{O}/\text{l}/\text{sec}$ in total airway resistance (equivalent to the increase in Figure 8(b)).

FIGURE 9 A simulated MEFV curve and a constructed IVPF curve for the normal case (the interrelationship of the two forms of data presentation is indicated).

FIGURE 10 Temporal waveforms (pleural pressure (P_{PL}), lung volume (V_L), flow at the mouth ($\dot{V}_{AO}$), collapsible airway volume (V_C), and collapsible airway resistance (R_C)) for model simulations for the normal case.

- (a) For the MEFV maneuver.
- (b) For the IVPF maneuver.

FIGURE 11 Simulated MEFV maneuvers for the normal case and for the case of tripled large airway resistance-associated pleural pressure waveforms are indicated in the insert.

FIGURE 12 Simulated MEFV maneuvers for the normal case and for the case of increased small airway resistance (a positive shift of $1.4 \text{ cm H}_2\text{O/l/sec}$ in the small airway resistance curve (Figure 3) to produce a change in total airway resistance equivalent to that seen in Figure 11). Associated pleural pressure waveforms are indicated in the insert.

FIGURE 13 Simulated MEFV maneuvers with altered airway compliance.

- (a) Altered upper portion of the airway compliance curve as indicated in the insert.
- (b) Altered lower portion of the airway compliance curve as indicated in the insert.
- (c) Combined changes in the upper and lower portions of the airway compliance curve as indicated in the insert (the arrow indicates the direction of increasing airway compliance).

FIGURE 14 Simulated MEFV maneuvers with altered lung compliance curves as indicated in the insert (the arrow indicates changes from normal static lung compliance to normal dynamic lung compliance to abnormal dynamic lung compliance).

TABLE 1

<u>PARAMETER OR EXPRESSION</u>	<u>SYMBOL</u>	<u>NORMAL VALUE (WHERE APPLICABLE)</u>
Lung volume	V_L	--
Total lung capacity	TLC	6.5 liters
Residual volume	RV	1.5 liters
Vital capacity	VC	5.0 liters
Functional residual capacity	FRC	2.3 liters
Pleural pressure	P_{PL}	--
Lung elastic recoil	P_{EL}	--
Alveolar pressure	P_{ALV}	--
Transmural pressure of collapsible airway	P_{TM}	--
Large airway resistance	R_{LAW}	0.7 cm H ₂ O/l/sec at a flow of 1 l/sec
Large airway Rohrer resistance components	$\begin{cases} K_1 \\ K_2 \end{cases}$	0.5 cm H ₂ O/l/sec 0.2 cm H ₂ O/(l/sec) ²
Collapsible airway volume	V_C	--
Collapsible airway volume (maximum)	$V_C \text{ MAX}$	0.1 liters
Collapsible airway resistance	R_C	--
Collapsible airway resistance components	$\begin{cases} K_3 \\ K_4 \end{cases}$	0.6 cm H ₂ O/(l/l·sec) 0.125
Small airway resistance	R_S	--
Airflow into the mouth	$\dot{V}_{AO}$	--
Airflow into the alveolar space	$\dot{V}_L$	--
Compliance of the lung	C_L	--
Compliance of the airway	C_{AW}	--
Maximum expiratory flow-volume	MEFV	--
Isovolume pressure-flow	IVPF	--
Forced expiratory volume at one second	FEV_1	--

FIGURE 1

FIGURE 2

FIGURE 3

FIGURE 4

FIGURE 5

FIGURE 6

FIGURE 7

FIGURE 8

FIGURE 9

FIGURE 10A

FIGURE . 10B

FIGURE 11

FIGURE 12

FIGURE 13A

FIGURE 13B

FIGURE 13C

FIGURE 14