Analog Simulation of Pulmonary Mechanics*

PHILIP D. MANFREDI, M.D.**
Boston, Massachusetts

INTRODUCTION

DURING THE PAST FEW YEARS, THE application of electronic analog computers to the study of biologic phenomena has gained widespread acceptance. In this paper, an analog computer was programmed to simulate the mechanics of respiration.

In order to use such a computer to simulate a given system, the mathematical equations describing that system must be available. In the case of pulmonary function, a number of theoretical equations describing various aspects of the mechanics of respiration have been derived. With the development of better techniques for measuring intra-pleural pressure, air flow, etc., the various theoretical equations have been tested and revised until they now appear to describe adequately the dynamics of the pulmonary system. Mead† has prepared an excellent summary of the current concepts concerning the mathematical analysis of the respiratory system.

Other investigators have constructed various mechanical models to simulate pulmonary function. Such models are indeed useful, but are in general difficult to construct and operate. They also lack the precision and flexibility of an electronic model. Today's analog computers are very simple to program. Once the model has been set up, changing parameters is simply a matter of turning a knob. All variables are present as voltages and can be displayed and recorded very accurately. The system is extremely flexible.

It must be understood, however, that any model, whether mechanical or electronic, is never a perfect representation of a system, but rather constitutes an abstraction of it. All the characteristics of the natural phenomena are not represented in the model either because the investigator is ignorant of them or because he knowingly disregards certain factors for the sake of expediency. However, if the limitations of such a model are realized, one can derive from it a great deal of knowledge and understanding of the system under study.

THEORY

Mechanics of Breathing. The forces that drive the respiratory system (lung plus thorax) are produced by the contraction of the diaphragm and the accessory respiratory muscles. In considering a model of the lung alone, the driving force of the system is the transpulmonary pressure, that is, the pressure difference between the mouth and the intrapleural space.

During the act of breathing, the transpulmonary pressure, $P_t$, must overcome: 1) the elastic forces of the lungs, 2) the frictional resistance of the moving tissues, 3) the resistance to air flow and 4) the inertial forces of the gas and tissues. The inertial elements are extremely small during ordinary breathing, and may be neglected. Expressed in equation form:

$$ P_t = P_{l(e)} + P_{l(res)} + P_{g(res)} $$

Equation (1) states that any moment during the respiratory cycle, the transpulmonary pressure is equal to the sum of the opposing pressures produced by the elasticity of the lung tissue, $P_{l(e)}$, the resistance to movement of the lung tissue, $P_{l(res)}$, and the resistance to gas flow, $P_{g(res)}$. It is necessary to define mathematically the three terms on the right side of equation (1).

The lung is elastic in nature and must be stretched during the process of inspiration. Under conditions of no flow (static), the transpulmonary pressure is exactly bal-
chanced by the force of retraction of the lung. In the normal tidal range, the static pressure varies nearly linearly with volume and the relationship can be expressed by equation (2).

\[ P = \frac{1}{C} V \]

where \( P \) is the applied pressure, \( V \) is the lung volume and \( C \) is the compliance of the lung. The term \( \frac{1}{C} V \), represents the pressure produced by a lung with compliance, \( C \), when expanded to a volume, \( V \), and is equal to \( P_1 \) of equation (1).

Rohrer derived theoretically the relationship between pressure and air flow (V) when flow is both laminar and turbulent:

\[ P = K_1 V + K_2 V^2 \]

The linear term is due to laminar flow and \( K_1 \) is a constant which includes the viscosity of the gas. The quadratic term represents turbulent flow and \( K_2 \) is a constant that includes the density of the gas. Later investigators experimentally confirmed equation (3) over the tidal volume range.

Tissue resistance is a result of displacement of the tissue during inspiration and expiration. Pulmonary tissue resistance is normally about 20 per cent of the total pulmonary resistance, the remainder being airway resistance. This element of resistance is included in the linear term of equation (3), so that the right hand side of equation (3) includes the opposing pressures produced by both the resistance to gas flow and the resistance of the lung tissue.

Substituting equations (2) and (3) into equation (1), one derives the total expression relating transpulmonary pressure to the opposing pressures produced by the lung:

\[ P_1 = \frac{1}{C} V + K_1 V + K_2 V^2 \]

Equation (4) is the simplest equation that adequately describes the dynamics of ventilation. It is simple because the factors of compliance and resistance are assumed to be constant throughout the respiratory cycle. This relationship is not valid over the entire vital capacity range, particularly in diseased lungs. The compliance only approaches linearity over a small range of lung volume. Likewise, airway resistance has been shown to be inversely related to lung volume and is also affected by intrapleural pressure.

It is obvious, then, that equation (4) is extremely simplified. A more general equation would be:

\[ P_1 = F_1(V) + F_2(V) + F_3(V^2) \]

where \( F_1 \), \( F_2 \) and \( F_3 \) are not constants but are function of lung volume, intrapleural pressure and perhaps other parameters.

However, for the evaluation of pulmonary function in most clinical situations, equation (4) is more than adequate, and it is this equation that was used to program the analog computer.

Work of Breathing. In a pressure-volume system, the work of breathing is expressed as the integral of the pressure with respect to volume:

\[ W = \int P dV \]

Otis et al. and Fenn calculated theoretically the work of breathing by assuming that flow rate during inspiration follows a sine curve. By further mathematical manipulation they were able to derive an expression relating the frequency of minimal work to alveolar ventilation. This frequency of minimal work is a function of compliance, resistance and the alveolar ventilation-to-dead-space ratio. There is also a frequency, usually a different one, which is least costly in terms of the average force exerted by the respiratory muscles. There is still some disagreement as to whether man breathes naturally at the frequency of minimal work or the frequency of minimal average pressure.

Electric Analog. In a pressure-volume system, the variables are volume, flow and pressure. In an electrical system, the analogous variables are charge, current and voltage. The electrical analog of a container with compliance, \( C \), is a capacitor with capacitance, \( C \). The electrical analog of laminar gas flow resistance is the resist-
ance to current flow, \( R \). There is no electrical analog to turbulent flow or its resistance.

The lung is composed of thousands of branching airways and small containers. The corresponding electrical circuit would be one of many resistive and capacitive elements arranged in parallel fashion, and the equation describing such a system would be extremely complicated. However, there is a theorem in electrical engineering which states that the behavior at a given frequency of a circuit composed of resistors and capacitors in any arrangement can be duplicated by a circuit consisting of a single resistor and capacitor arranged in series. The simplified circuit is called the equivalent circuit and the values of the resistor and capacitor are called, respectively, the effective resistance and the effective capacitance. The value of these two elements is a function of the frequency of the driving voltage.

So that a system as complicated as the lungs can be represented by a simple electrical model composed of a resistor and capacitor in series. The element of turbulent flow is, however, not represented.

If the driving pressure of the model is sinusoidal, the phase angle (the angle between the driving pressure and the resultant flow) is given by:

\[
(7) \tan \theta = \frac{1}{2} \pi f R C
\]

where \( f \) is the frequency of the applied pressure, \( R \) is the flow resistance and \( C \) the capacitance.

**Materials and Methods**

The computer used was an Electronic Associates, Inc. electronic analog computer, model TR-10. The data was plotted by means of a Houston Instrument Corp. Model HR-96 X-Y recorder.

The computer was wired to solve the differential equation (4). In addition, the computer was wired to generate a sinusoidal driving pressure, and a circuit was included to compute the work of breathing. The entire program, in the form of a block diagram, is show in Fig. 1.
There are nine potentiometers in the program that must be set before the problem is run. Table 1 indicates what parameter each pot. controls.

As the computer solves the equation, any of the variables present as voltages (P, V, W) can be plotted on the variplotter. Variables can be plotted against each other or against time. Before each problem is run, the variplotter is standardized in terms of centimeters of deflection per unit volt. The curves are plotted on standard graph paper.

### RESULTS

Once the model has been constructed, the number of experiments it can be programmed to perform are numerous. Some representative results are shown here.

Figure 2 demonstrates the effects of changes in $K_1$ on the tidal volume and air flow. In this experiment, the variables are plotted on the vertical axis versus time on the horizontal axis. The top curve is the transpulmonary pressure, $P_1$, which was made to vary between 0 to $5\text{ cm. H}_2\text{O}$ ($1\text{ volt}=1\text{ cm. H}_2\text{O}$). The middle curve is lung volume, $V$, obtained for values of $K_1$ equal to 2, 4, 8 and 16. The bottom curve is flow rate, $V$. Compliance was arbitrarily set equal to 0.2, and $K_2$ was set equal to 2.0.

Figure 3 represents a P-V diagram obtained by plotting volume on the horizontal axis and pressure on the vertical axis. $P_1$ was again made to vary between 0 and $5\text{ cm. H}_2\text{O}$. $K_1$ and $K_2$ were set equal to 2.0. Three loops were drawn for values of compliance equal to 0.05, 0.1 and 0.2.

In Fig. 4, the computer was wired to compute the work of breathing, and this was plotted versus time. Work was computed for values of frequency equal to 8, 16, and 32.

---

**Table 1**

<table>
<thead>
<tr>
<th>Potentiometer</th>
<th>Determinant</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>initial value of $P_1$</td>
</tr>
<tr>
<td>2</td>
<td>frequency of $P_1$</td>
</tr>
<tr>
<td>3 and 4</td>
<td>Magnitude of $P_1$</td>
</tr>
<tr>
<td>5</td>
<td>coefficient of resistance, $K_2$</td>
</tr>
<tr>
<td>6</td>
<td>compliance, $C$</td>
</tr>
<tr>
<td>7</td>
<td>coefficient of resistance, $K_1$</td>
</tr>
<tr>
<td>8</td>
<td>initial value of volume, $V$</td>
</tr>
<tr>
<td>9</td>
<td>multiplies work per cycle by the frequency</td>
</tr>
</tbody>
</table>

---

![Figure 2: Plot of pressure volume and flow versus time for different values of $K_1$.](image-url)
12, 16, 20 and 24 cycles per minute. By varying tidal volume, alveolar ventilation was kept constant at 4.2 liters per minute. The values of C, K₁, and K₂ were set equal to 0.2, 2.0 and 2.0 respectively.

**DISCUSSION**

Figures 2 and 3 represent some of the results obtained in one type of experiment, the purpose of which was to observe the effects on lung volume and air flow produced by changes in C, K₁, and K₂. For different values of these constants, two types of curves were obtained. One (Fig. 2) was produced by plotting P₁, V and V versus time, and another (Fig. 3) was produced by plotting pressure versus volume. Throughout the experiment, the driving pressure was a sinusoidal pressure varying between 0 and +5 cm. H₂O.

Figure 2 demonstrates the effects on breathing produced by changes in K₁. As would be expected, an increase in K₁ results in a decrease in average flow rate. However, Fig. 2 also demonstrates that changes in K₁ affect tidal volume. An increase in K₁ causes a decrease in tidal volume both by decreasing end-inspiratory volume and by increasing end-expiratory volume. Why should changes in the flow-resistive element produce changes in the end-tidal volumes which are attained when flow is zero?

The reason is that an increase in K₁ causes a marked shift in the phase angle between pressure and flow, so that at the point of end-inspiration the driving pressure has passed its peak and is somewhat less than its maximum value. Since at this instant of no flow the pressure is decreased, the static volume maintained by this pressure is decreased. The same argument applies to the end-expiratory volume. So that it becomes clear that by producing a phase shift, changes in K₁ can affect end-tidal volumes.

Figure 2 also graphically demonstrates the changes in the phase angle produced by changes in K₁. Substituting the values of f, C, and K₁(R) used in this experiment into equation (7), the phase angles computed...
are in agreement with measurements made on Fig. 2.

Figure 3 demonstrates the changes in the P-V diagram produced by changes in lung compliance. The slope of the axis of the P-V loop is a measure of compliance, and the slopes of the three loops in Fig. 3 are in agreement with the three values of C set in the computer.

The area of the triangle formed between the axis of the P-V loop and the horizontal axis is a measure of the work done against elasticity during inspiration. As is seen in Fig. 3, this work is exactly halved by halving the compliance. It is also apparent that the work done against flow and tissue resistance, represented by the area inside the P-V loop, is also decreased by a decrease in lung compliance. This is due to the fact that for a given transpulmonary pressure a decrease in compliance produces a decrease in tidal volume. Average air flow is therefore also decreased, and the work required to produce air flow is less.

Figures 2 and 3 represent only two of the many curves that the computer can be programmed to draw. Analysis of these curves demonstrates how the computer can contribute to the basic understanding of pulmonary physiology.

Figure 4 represents another type of experiment in which the computer calculated the work of breathing. For constant values of alveolar ventilation, dead space, C, K₁, and K₂, the computer was made to calculate the work of breathing at different frequencies of respiration. The work of breathing (per minute) is plotted on the Y-axis versus time on the X-axis. In Fig. 4 for the values of lung constants given, the frequency of minimal work is seen to be around 16 cycles per minute.

Other curves (not shown) demonstrated that by increasing K₁ or K₂, the frequency of minimal work has shifted to the slower side. This is exemplified clinically by a person with bronchiolar constriction, for example, whose lung has an increased airway resistance. Such a person tends to take deep breaths at a slow frequency in order to minimize the work of breathing. And, similarly, by decreasing compliance in the model, the frequency of minimal work was seen to be shifted toward higher values.

![Figure 4: Work of breathing plotted versus time for different values of frequency.](image-url)
This situation is exemplified clinically by a person with pulmonary fibrosis who has a decreased lung compliance. Such a person takes shallow breaths at a higher frequency than normal.

The curves in Fig. 4 also contain information concerning the distribution of work. The peak of each curve represents all the work done during inspiration for an entire minute. If expiration is passive, as it is in this experiment, this represents the total work of respiration for one minute's time. The height of the plateau at which each curve levels off is a measure of the amount of work lost to flow and its plateau represents the work done against the elasticity of the lung.

This experiment demonstrates how the frequency of minimum work is affected by changes in the elastic and resistive elements of the lung. The distribution of the total work into elastic and resistive components are also illustrated. Finally, this experiment demonstrates the ease with which an electronic model can be adapted to solve various types of problems.

**CONCLUSION**

An electronic analog computer is programmed to solve a simple differential equation which describes the dynamics of pulmonary ventilation. The equation used, and therefore the model constructed, is simple in that the values of lung compliance and resistance are held constant throughout the respiratory cycle. Although the model is admittedly simple, its performance in various experimental situations is nevertheless instructive and demonstrates the values of a model of this type in the investigation of respiratory physiology.

The ease with which the model can be altered to fit various experimental conditions is evident. To vary any of the physical constants of the model, it is necessary only to adjust a potentiometer. All of the variables are readily available for display or computation purposes.

This model will act as a basis on which to construct a more complicated and accurate model of the respiratory system, so that in the future, circuits will be added which will generate lung compliance and resistance as functions of pressure, volume, frequency etc. It will also be possible to include circuits that will simulate the diffusion of gases between the alveoli and the blood, so that eventually the respiratory system as a total functioning unit may be simulated.

Once such a model has been set up, many experiments will be devised and tested in terms of their clinical value. Other applications of the model will become evident. It may be possible, for example, to apply the model to the administration of gas anesthesia.

In conclusion, it appears that the model described in this paper will form the basis on which to build a more complete and complicated model of the respiratory system. Such a model will have widespread application both in the basic understanding of pulmonary physiology and in the clinical evaluation and treatment of lung disease.

**Summary**

1) An electronic analog computer was programmed to simulate the mechanics of respiration.

2) The performance of the electronic model in various experimental situations is demonstrated.

3) Future development of the model, and its applications are discussed.

**Resumen**

1) Un computador electrónico fue programado para simular la mecánica respiratoria.

2) Se describe el funcionamiento de este modelo en situaciones diversas.

3) El desarrollo futuro de este modelo y sus aplicaciones son comentados.

**Résumé**

1) L'auteur a élaboré un appareil électronique capable de simuler les éléments mécaniques de la respiration.

2) Les réalisations de ce prototype électronique ont été présentées dans différentes conditions expérimentales.

3) L'auteur discute l'avenir de cet appareil et de ces applications.
ZUSAMMENFASSUNG
1. Ein elektronisches Analogie-Rechengerät wurde in dem Sinne programmiert, daß es im-
stande war, die Mechanismen der Atmung zu simulieren.
2. Die Leistung des elektronischen Modells in verschiedenen experimentellen Situationen wird
demonstriert.
3. Zufällige Entwicklungsergebnissen des Modells und seiner Anwendungs-Räume
werden diskutiert.

REFERENCES
1 BRISCOE, W. A. AND DUBOIS, A. B.: "Relationship between Airway Resistance, Airway
2 CHENG, T. O., GODFREY, M. P. AND SHEPARD, R. H.: "Pulmonary Resistance and State of
Inflation of Lungs in Normal Subjects and in Patients with Airway Obstruction," J. Appl.
3 COMBRO, J. H., FORSTER, R. E., DUBOIS, A. B. AND BRISCOE, W. A.: The Lung, Year Book
7 MEAD, J. AND WHITTENBERGER, J. L.: "Physical
Properties of Human Lungs Measured during
11 RAHN, H., OTIS, A. B., CHADWICK, L. E. AND
FENN, W. O.: "The Pressure-Volume Diagram
12 ROKNER, E.: "Der Stromanalogastudent in
den Mammalianen Atemwegen und der Einfluss
der Unregelmäßigen Verweigung des Bronchialsystems auf den Atmungsverlauf in

For reprints, please write Dr. Manfred, New Eng-
land Center Hospital, Boston.

RIGHT VENTRICULAR FORCES IN RIGHT VENTRICULAR HYPERTENSION

Results of the standard electrocardiogram, cube
vectorcardiogram, and the corrected vectorcardio-
gram derived by the Frank system, in 50 patients
with valvular pulmonary stenosis were compared with
hemodynamic data recorded at cardiac catheteriza-
tion. Right ventricular hypertension at rest was
found to range from 40 to 166 mm.Hg. Right ven-
tricular peak pressure, right ventricular work, and
pulmonary valve area were used as criteria for compar-
ison. Two new vectorcardiographic parameters
were derived, based on the measurement of the
maximum rightward directed spatial force.

A significant correlation between right ventricu-
lar peak pressure and the maximum rightward spa-
tial vector (r=0.87) and a sum of maximum right-
ward vectors (r=0.90) was found, using data de-
derived from the Frank system. Similar correlations
for the cube system were less significant (r=0.82
and 0.83). These results indicate the superiority of
the orthogonal corrected lead system of Frank in
these electrocardiographic-hemodynamic corre-
lations. The progressively changing configuration of spatial
forces with increasing severity of hypertension
gives further insight into the alterations in the de-
polarization process in right ventricular hypertro-
phy. These findings also prove that with new cri-
terio employing the measurement of truly spatial
magnitudes, reliable estimation of resting right ven-
tricular pressure may be obtained.

GAMBRO, R., HUGENHOLTZ, P. G. AND NADAS, A. S.: "Cor-
rected (Frank), Uncorrected (Cob), and Standard Electro-
cardiographic Lead Systems in Recording Augmented Right
Ventricular Forces in Right Ventricular Hypertension," Brit.

HEART SIZE AFTER VALVULAR REPLACEMENT

The persistence of and in some cases the increase
in cardiomegaly in these patients resulted from im-
paired left-ventricular performance. This appears to
be more common after replacement of the mitral valve with a Starr-Edwards prosthesis than after
replacement of the aortic valve. It is not known
whether this is related primarily to left-ventricular
dysfunction present preoperatively, or to deleterious
factors introduced at operation.

RASCELLI, G. C., KINCAID, O. W. AND KIRKLIN, J. W.: "Heart Size After Isolated Replacement of Mitral or Aortic