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Contribution of Airway Resistance to Airway Pressure during Mechanical Ventilation: an Experimental Study

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Although airway resistance (R) is an important parameter of the pulmonary condition, its determination during mechanical ventilation is not easy. Most physicians estimate R from peak airway pressure during mechanical ventilation. We assessed the relationship between R and peak airway pressure by a computer simulation. The time course of airway pressure (Ptr) during mechanical ventilation was calculated from the airway pressure at end-inspiration and respiratory flow, allowing for the buffering effect of the dead space. The parameters for computer simulation were obtained from 5 paralyzed and mechanically ventilated dogs. The predicted Ptr curve was a function of airway resistance. Since R was not directly determined by the animal experiments, we determined R by using the Ptr curve most closely approximating the original Ptr curve as the true R. The R-peak tracheal pressure relationship predicted by computer simulation showed that the peak airway pressure increased almost linearly with increases in R. However, in computer simulation, when R was increased 10-fold from the value at airway relaxation, the peak airway pressure increased only 6-fold from the corresponding value. We conclude that peak airway pressure is a relatively insensitive parameter for the estimation of airway constriction during mechanical ventilation.

Key words: computer simulation, pulmonary mechanics, animal study

INTRODUCTION

The peak airway pressure is an important parameter for determining mechanical ventilation in patients with bronchial asthma (3). If the peak airway pressure increases during mechanical ventilation in asthmatic patients, the physician presumes that airway resistance has been increased by the asthma probably denotes an impending barotrauma (3). On the other hand, an increase in peak airway pressure indicates a decrease in pulmonary compliance in patients with interstitial lung disease. Pulmonary resistance and compliance can be estimated by measuring pleural pressure using an esophageal balloon, airflow, and airway pressure (5). Among the three parameters, airway pressure and airflow can be measured by non-invasive measures but pleural pressure can not because a long-term indwelling of an esophageal balloon is impractical in the mechanically ventilated patient. The purpose of this study was to develop a method to calculate airway resistance from airflow and airway pressure during mechanical ventilation, and to analyze the relationship between airway resistance (R) and peak airway pressure.

MATERIALS AND METHODS

Five beagle dogs (5-10 kg) were deeply anesthetized with a short-acting barbiturate (Thiamylal) and then decerebrated by transection of the mid-brain at the rostral margin of the superior colliculi (6). The dogs were paralyzed with intravenous pancronium bromide (1.0 mg/kg, every hour), and then tracheostomized. Volume-cycled mechanical ventilation (Harvard 613) was performed with a tidal volume of 15 ml/kg. Arterial blood was frequently sampled from an indwelling catheter in the femoral artery and analyzed for arterial blood gas. The respiratory frequency of

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mechanical ventilation was adjusted to keep $PaCO_2$ within physiological limits. As shown in Fig. 1, ventilatory flow (F) was measured by a hot-wire flow meter (Minato, RF-2) placed between the mechanical ventilator and tracheal tube. A catheter (ID 2mm) was connected to a side hole made in the tracheal tube. Airway pressure was continuously measured from this catheter as tracheal pressure (Ptr), using a pressure transducer (Nihon Kohden, DX360). The venturi effect of this side hole system on Ptr can be neglected because the respiratory flow of mechanical ventilation was not high. The eighth through fifteenth tracheal cartilagenous rings were surgically removed and a pair of four silk strings were tied on each side of the membraneous portion of the trachea. The strings in the left side of the trachea were connected to a rod and those in the right side were connected to an isometric force transducer (Nihon Kohden TB 611T), for recording tension of the tracheal smooth muscle (Ttr). The bilateral vagus nerves were transected in the neck and the distal end of the nerves were mounted on bipolar stimulating electrodes. The signals of F and Ptr were recorded on magnetic tape. The signals were then played back and A/D converted with a sampling frequency of 25 Hz for computer analysis. The digitized data were processed by a personal computer (NEC, PC-9821 Ct 16, 166 MHz). The experimental conditions were during a steady state of mechanical ventilation and during maximal stimulation (7) of efferent fibers of the bilateral vagus nerves with a pulse train (frequency 30Hz, intensity 40V, pulse duration 1.0ms, simulation interval 20s).

RESULTS

Figure 2 are recordings made before and during electrical stimulation of the efferent pathway of the vagus nerves. During stimulation, tracheal tone and airway pressure increased and expiratory flow decreased. This suggests an intimate relationship between airway constriction, airway pressure, and expiratory airflow during mechanical ventilation. The maximum change in airway pressure always developed earlier than that of tracheal constriction. As we have speculated in a previous paper (6), this difference may arise from differences in neural control and mechanical features of the two parts of airway smooth muscle. Therefore, we compared the maximum responses of Ttr and Ptr.

Figure 3 shows an example of airway resistance (R) calculated by the most simplest method, i.e., Ptr divided by F, during one inspiratory phase. In the figure, F demonstrates a negative deflection, similar to that in Fig. 2.

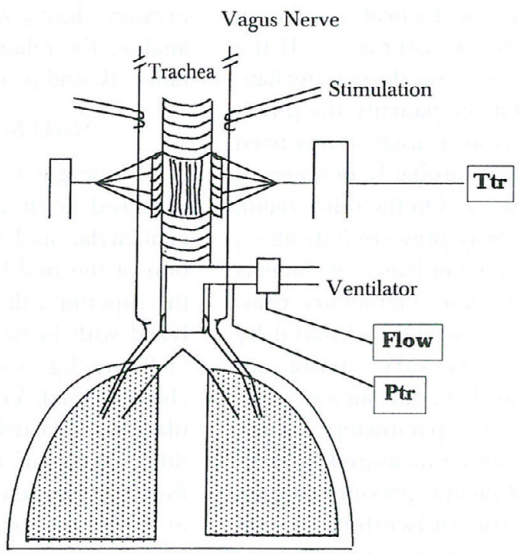

Fig. 1 The experimental setup. Ttr: tension of membraneous portion of the cervical trachea, Flow: respiratory flow, Ptr: airway pressure measured as tracheal pressure. See text for detailed explanations.

The calculated Ptr/F was smallest at the onset of inspiration and largest at the end of inspiration. Since R should be nearly constant during the respiratory cycle of quiet breathing (5), figure 3 suggests that Ptr/F does not represent true R.

In respiratory physiology, mechanical changes in respiration are expressed by Ptr, lung volume, ventilatory flow, and inertia of the lung-thoracic system (5). In our system of mechanical ventilation, the contribution of inertia to respiratory mechanics is very small and can be neglected. Thus, the following equation is valid:

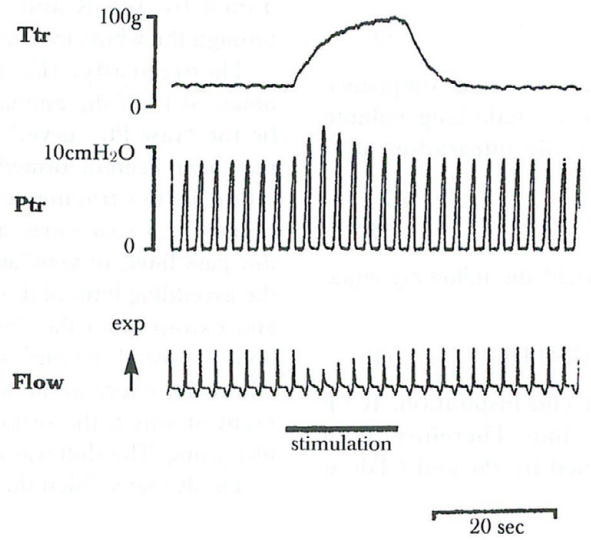

Fig. 2 Recordings before and during electrical stimulation of the efferent pathway of the bilateral vagus nerves. Abbreviations are the same as in Fig. 1.

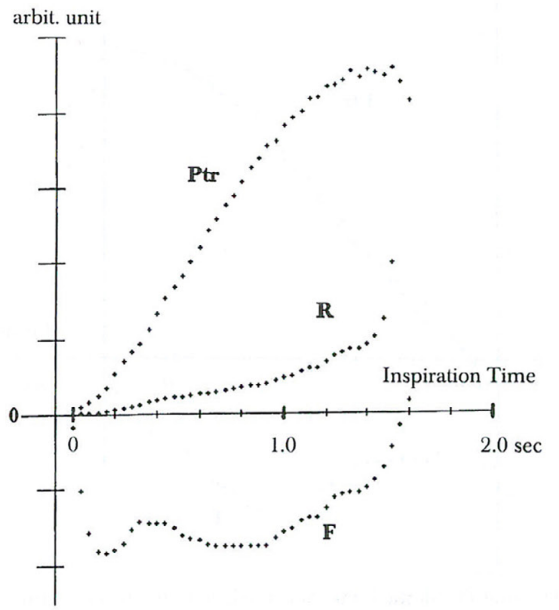

Fig. 3 Airway resistance (R) estimated by airway pressure (Ptr) divided by ventilatory flow (F) during a single inspiratory phase of mechanical ventilation. In the figure, F was demonstrated as similar to that in Fig. 2.

$$Ptr - Palv = R \times F \quad (1)$$

where Palv represents alveolar pressure. Equation 1 means that airflow is primarily determined by airway resistance and the difference between tracheal pressure and alveolar pressure.

In paralyzed animals the following equation is also valid:

$$Palv = V / C \quad (2)$$

where C is a constant representing compliance of the lung. V represents static lung volume and it is calculated by the integration of F. Therefore,

$$V = \int Fdt \quad (3)$$

Equations 1, 2 and 3 yield the following equation:

$$Ptr = R \times F + 1/C \times \int Fdt \quad (4)$$

Since airflow stops at end-inspiration, $R \times F$ becomes zero at this time. Therefore, C in equation 4 is determined by Ptr and $\int Fdt$ at end-inspiration. The $\int Fdt$ at end-inspiration can be calculated from the F curve and theoretically $\int Fdt$ is the tidal volume of mechanical ventilation. Although equation 4 gives a Ptr curve as a function of time, to determine a single Ptr v.s. time curve one R value should be used. To find the most suitable R, we squared the difference between the Ptr curve determined by any R and the "raw Ptr curve" through the whole trajectory of inspiration.

Theoretically, the Ptr v.s. time curve obtained from the animal experiments should be the "raw Ptr curve." However, this curve may have been deformed by dead space ventilation in the trachea and ventilator circuit because this space may act as a reservoir (i.e., low-pass filter) of ventilated flow. We assumed the ascending limb of the Ptr curve as linear (1) and extrapolated the linear part of the curve until it crossed the time axis (1). We then shifted this Ptr curve to the left until it reached the point at which the original Ptr curve started increasing. The shift was usually 0.04s.

The R that yielded the smallest square value

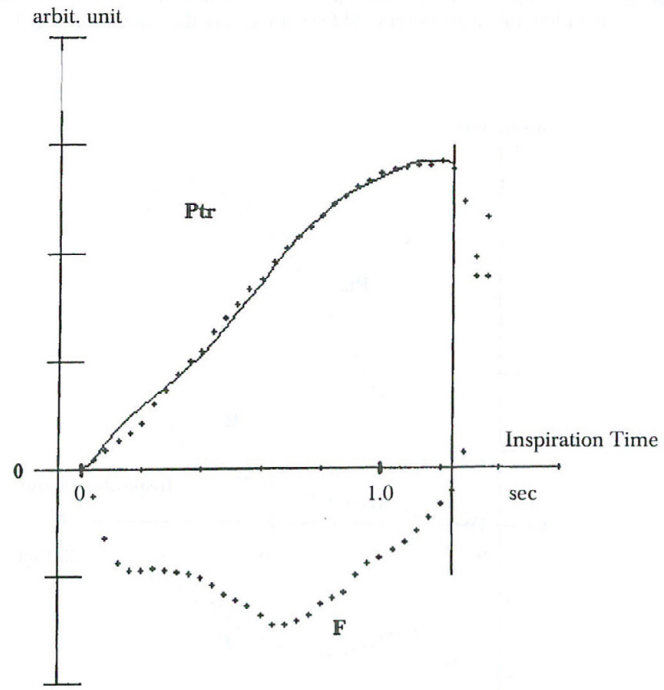

Fig. 4 The true Ptr (dashed line) and predicted Ptr calculated from the airway pressure at end-inspiration and ventilated flow. R for the predicted Ptr was 6.84 cmH2O/l/s.

of [equation 4- based Ptr minus raw Ptr] was regarded as the most suitable Ptr. In the following paragraphs we call this the "predicted Ptr."

In Fig. 4, an example of the predicted Ptr curve is superimposed on the raw Ptr curve. The predicted Ptr fits well with the raw Ptr. In the demonstrated case, the R of 6.84 $cmH_2O/l/s$ gave the best fitting Ptr curve.

The mean tracheal tension, before and during stimulation of the bilateral vagus nerves in 5 dogs, were 17.9±10.0 (mean±SD) and 90.9±43.5g. The Rs measured by this method were 10.3±3.2 and 28.7±6.5 $cmH_2O/l/s$ (Fig. 5). The differences were statistically significant (paired t test, $P<0.05$). The increases in tracheal tension also had a significant relationship with those in R. The mean C was 0.012±0.002 l/cmH_2O.

Figure 6 shows the relation between R and the peak of the predicted Ptr calculated by our simulation equation. In the simulation, C was regarded as 0.012 l/cmH_2O (the average of 5 dogs) and the flow curve was obtained by operating the ventilator without a load. The computer simulation indicated that peak airway pressure increased almost linearly with increases in R. However, when R showed a 10-fold increase (i.e., from 10.3 to 103.0 $cmH_2O/l/s$), the peak airway pressure increased only 6 fold (i.e., from 14 to 81 cmH_2O).

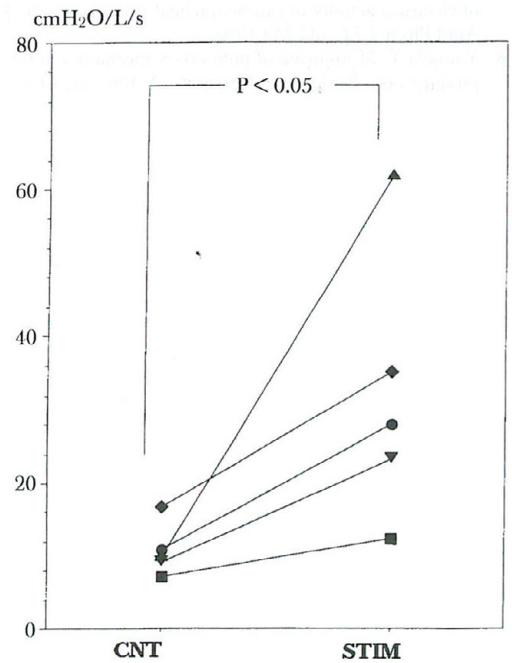

Fig. 5 Airway pressure before (CNT) and during electrical stimulation of the efferent pathway of the bilateral vagus nerves (STIM).

DISCUSSION

In this study we analyzed the relationship between airway resistance and airway pressure during mechanical ventilation, using a computer simulation. The data for computer simulation, i.e., airway pressure at end-inspiration and respiratory flow curves, were obtained from animal experiments. The predicted Ptr curve showed a close fit to the real one. This finding demonstrated that airway resistance can be obtained from airway pressure at end inspiration and respiratory flow. Our simulation equations are similar to those proposed by Bates et al. (1) as a single compartment model. We found that a similar method has been used in a recent computer-assisted respiratory monitoring system (e.g., Nihon Kohden OMR-8101) (6). Therefore, our simulation system is not quite new. However, our analysis gave important data concerning the relationship between the peak airway pressure and airway resistance during mechanical ventilation.

In clinical practice, peak airway pressure has

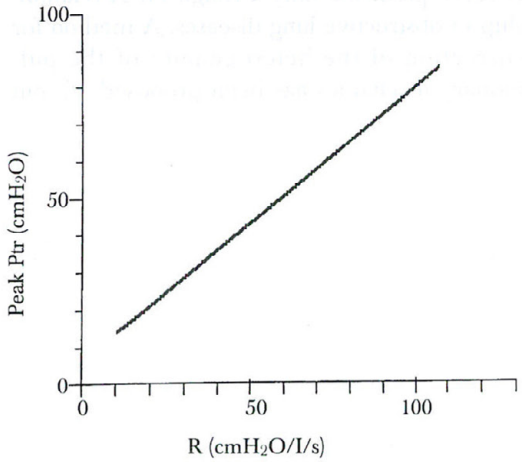

Fig. 6 Relationship between airway resistance (R) and peak airway pressure generated by a computer simulation.

been regarded as the simplest and most important parameter for estimating the pulmonary condition during mechanical ventilation(3). As seen in Fig. 3 the airway pressure reached its peak before the end of inspiration. This finding suggests that the peak airway pressure does not simply represent pulmonary compliance but it may be function of respiratory air flow. We estimated the relationship between peak airway pressure and airway resistance in Fig. 6. We found that the relationship was almost linear. However, when airway resistance was increased 10-fold, the peak airway pressure increased only 6-fold. Therefore, peak airway pressure is a relatively insensitive indicator of airway resistance monitoring. Care should be used in assessing airway obstruction from changes in peak airway pressure during mechanical ventilation.

The lung is composed of several units with different mechanical properties (1). Although in healthy subjects the lung can be assumed to act as a single compartment model, the inhomogeneity of mechanical properties can become severe in lung diseases. Furthermore, in obstructive lung diseases, auto PEEP, i.e., air trapping of ventilatory volume, may considerably alter pulmonary mechanics (4). Considering pulmonary heterogeneity, our Ptr-R curve predicted only a rough Ptr-R relationship in obstructive lung diseases. A method for correction of the heterogeneity of the pulmonary mechanics has been proposed (2), but this method is yet not satisfactory for routine use in mechanical ventilation.

In conclusion, airway resistance can be calculated from the peak airway pressure and respiratory flow. The peak airway pressure is a relatively insensitive parameter for the estimation of airway constriction.

ACKNOWLEDGMENT

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