

**TITLE:** AIRWAY PRESSURE RELEASE VENTILATION: IMPORTANCE OF EXPIRATORY (RELEASE) TIME

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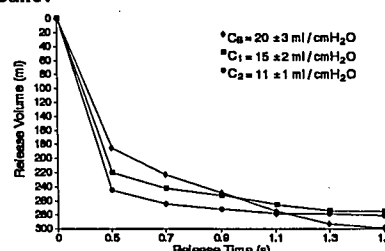
Airway pressure release ventilation (APRV) provides intermittent temporary decrease in lung volume to augment alveolar ventilation. A release time (TR) of 1.5 s has been used in previous experimental and clinical investigations. However, patients with poorly compliant lungs should require a shorter TR, without affecting tidal volume (VR). Use of minimum TR would maintain increased transpulmonary pressure and lung volume with optimum gas-exchange for a given level of ventilatory support. We wished to determine the interdependence of respiratory system compliance (CRS), TR, and gas exchange during APRV.

After institutional approval, five swine were anesthetized with  $\alpha$ -chloralose, tracheally intubated and paralyzed with pancuronium. The carotid artery was cannulated to obtain samples for blood gas analysis. Respiratory gas flow was measured with a pneumotachometer and airway pressure (PAW) with a gas-pressure transducer. The flow and PAW signals were interfaced with a computer for calculation of VR, TR, and CRS. Continuous positive airway pressure (CPAP) was adjusted to produce a VR of 12 ml/kg when released to atmospheric pressure for 1.5 s. Release rate was adjusted to produce an arterial  $PCO_2$  of 35 to 40 torr. Respiratory system compliance was

reduced with incremental banding of the chest. When CRS was reduced, CPAP level was increased to maintain constant release volume. Release rate remained unchanged throughout the study. Release time was reduced in 0.2 s decrements to 0.5 s, or until baseline  $PaCO_2$  increased by 5 torr. Data were collected at baseline and 10 min after each reduction in release time. Data are summarized as mean  $\pm$  SD and were statistically evaluated using an analysis of variance for repeated measures.

The CPAP level ranged from 13 to 30 cm  $H_2O$  and rate ranged from 10 to 18 breaths/min. Respiratory system compliance was decreased from a baseline of  $20 \pm 3$  ml/cm  $H_2O$  (C8) to  $15 \pm 2$  ml/cm  $H_2O$  (C1) and to  $11 \pm 1$  ml/cm  $H_2O$  (C2). The effect of release time on mean release volume with varied CRS is summarized in the figure. Baseline  $PaCO_2$  increased significantly as release time was reduced to 0.5 s. However, changes in  $PaCO_2$  associated with variation in CRS were not clinically significant.

Egress of air from the lungs during APRV depends on the time constant of the respiratory system ( $\tau$ ) and on the resistance of the breathing circuit. Our results show that decrease in  $\tau$  associated with reduction in CRS will permit reduction in expiratory time without compromising ventilation. A reduction in expiratory time will provide less time for airway collapse and deterioration in gas exchange.



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**TITLE:** PULMONARY CAPILLARY PRESSURE CHANGES FOLLOWING SMOKE INHALATION IN SHEEP

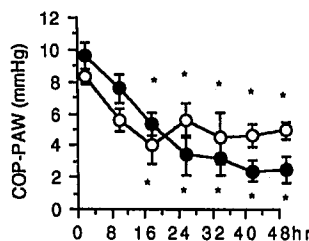
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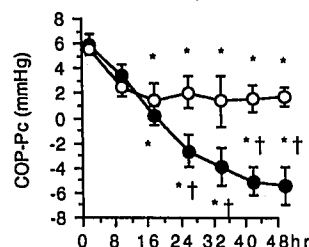
We determined pulmonary capillary pressure (Pc) and the longitudinal distribution of pulmonary vascular resistance in chronically instrumented sheep with inhalation injury by using analysis of pressure decay curves following pulmonary artery occlusion (1).

The animals were divided into two groups. Group I (N=8) sheep were insufflated with 4 x 12 breaths of cotton smoke and Group II animals (N=5; sham-control) with room air. There was an elevation of Pc from 13.6 to 19.4 mmHg and a increase in the lung lymph flow from 8.4 to 36.4 ml/h after smoke inhalation ( $P < 0.05$ ). The total pulmonary resistance (RT), pre- (Ra) and post- (Rv) capillary resistance significantly increased in Group I (RT:1.84 to 3.80, Ra:1.16 to 2.20, Rv:0.68 to 1.60 mmHg·min/l,  $P < 0.05$ ). There was alteration of the distribution of resistance between the arterial and venous beds in Group I ( $P < 0.05$ ) but no alteration of the distribution of resistance in Group II. Although lactated Ringer's solution (3 ml/kg/hr) reduced the colloid oncotic pressure in the animals of Groups I and II, lung lymph flow increased only in those exposed to smoke. The COP-PAW gradient decreased to a similar extent in both Groups (Figure 1). The COP-Pc gradient decreased to a greater extent in Group I than in Group II between 24 and 48 hours after smoke inhalation ( $P < 0.05$ )(Figure2).

The present experiments suggest that the estimates of Pc, using the inflection point of pressure tracings, can predict filtration pressure better than wedge pressures. This Pc can easily be measured in human patients when pulmonary arterial wedge catheters are in place.



**Figure 1.** Colloid oncotic pressure minus pulmonary artery wedge pressure (COP-PAW). There is no difference in COP-PAW between Group I (closed circles) and Group II (open circles).  
Values are means  $\pm$  SE.  
\*  $P < 0.05$  vs. baseline



**Figure 2.** Colloid oncotic pressure minus pulmonary capillary pressure (COP-Pc).  
Group I: closed circles  
Group II: open circles  
Values are means  $\pm$  SE.

\*  $P < 0.05$  vs. baseline  
†  $P < 0.05$  vs. control

**References:** 1. J. Appl. Physiol. 54(3): 846-851, 1983