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TITLE: FEEDBACK CONTROL OF VENTILATION USING EXPIRED CO_2

AUTHORS: K. B. Ohlson, D. R. Westenskow, Ph.D. and W. S. Jordan, M.D.

AFFILIATION: Departments of Anesthesiology and Electrical Engineering, University of Utah College of Medicine, Salt Lake City, Utah 84132

Introduction. Using feedback control of mechanical ventilation, a patient's arterial carbon dioxide concentration ($PaCO_2$) can be held within tightly specified limits. The ventilation rate or volume can be adjusted automatically to compensate for changes in the patient's metabolism. This paper describes experience gained using feedback control of end tidal PCO_2 in animals. The controller compensated correctly for changes in CO_2 production but failed to perform correctly when the pulmonary blood flow or pulmonary dead space changed abruptly.

Methods. The feedback system uses an Intel 8085 microprocessor to control the minute ventilation of a Siemen's 900B Servo-Ventilator. End tidal PCO_2 is measured at the airway by IR absorption using a Siemen's 930 CO_2 analyzer. Measured end tidal PCO_2 is compared with the desired end tidal PCO_2 to produce an error signal. A proportional, integral, derivative controller uses the error signal to adjust the timing cycle of the servo-ventilator. The volume of ventilation is reset every 5 seconds. $\dot{V}CO_2$, end tidal PCO_2, minute volume and dead space are plotted and printed every 5 seconds.

The controller was evaluated in 6 mongrel dogs (18-20 kg). The animals were anesthetized with 25 mg/kg thiopental and their tracheas intubated with a double lumen endotracheal tube. Anesthesia was maintained with 1.5% halothane and 50% nitrous oxide in oxygen. A Swan-Ganz catheter was placed through the jugular vein to the pulmonary artery for measurement of pressure and thermal dilution cardiac output. An indwelling pH, PCO_2 sensor (Biochem International, Inc.) was placed in the left femoral artery for continuous monitoring of $PaCO_2$. Periodic blood samples were analyzed using a Radiometer ABL-1 blood gas machine.

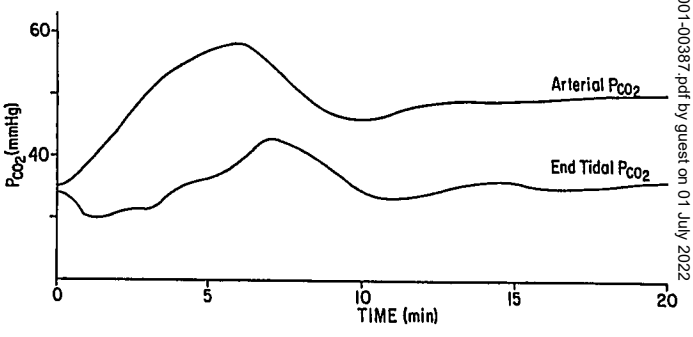
The animals were ventilated using feedback control to keep end tidal CO_2 at 5.0%. Once steady state was reached, measurements were taken of cardiac output, arterial pressure, wedge pressure, temperature, heart rate and arterial blood gases. $NaHCO_3$ was infused at the rate of .21 mEq/kg/min. After 8 minutes a complete set of measurements was taken. Ten minutes after the start of infusion, the infusion was stopped. Measurements were taken 8 minutes after the infusion stopped. The same procedure was repeated infusing .35 mEq/kg/min.

To further evaluate the controller, 3 procedures were used which disturbed the end tidal-arterial PCO_2 relationship: 1) the balloon on the Swan-Ganz catheter was inflated and wedged thus blocking blood flow through a main branch of the pulmonary artery, 2) one lumen of the endobronchial tube was occluded so only the right lung received ventilation and 3) air (1.0 ml/kg) was infused rapidly into the right atrium creating an air embolism. During each procedure continuous recording was made of $PaCO_2$, end tidal PCO_2, $\dot{V}CO_2$, minute ventilation and dead space.

Results and Discussion. During the infusion of .21 mEq/kg/min and .35 mEq/kg/min of $NaHCO_3$, the $PaCO_2$ changed by $-.06 \pm 2.3$ torr (mean $\pm$ SD) and by -2.2 ± 1.2 torr, respectively. Neither change was statistically significant. Therefore, the feedback controller held the $PaCO_2$ relatively constant despite an average increase in $\dot{V}CO_2$ from 122 to 161 ml/min and from 131 to 188 ml/min. End tidal PCO_2 appears to be an adequate feedback control parameter to compensate for changes in metabolism.

With the wedging of the pulmonary artery balloon during feedback controlled ventilation changes occurred as shown in the figure. Following pulmonary artery occlusion, the mean difference between end tidal and arterial PCO_2 increased from 1.0 to 27.6 torr. The end tidal-arterial PCO_2 differences resulting from occluding the left lung and the air embolism were 14.9 torr and 12.0 torr, respectively. These procedures demonstrate the fallacy of controlling ventilation based on end tidal measurements during changes in the distribution of pulmonary blood flow and ventilation. If the controller kept the CO_2 excretion constant during these non-metabolic changes $PaCO_2$ would remain constant.

An improved feedback controlled ventilator would use end tidal PCO_2 to control ventilation when metabolism changes and use CO_2 excretion to control ventilation when the end tidal PCO_2 changes but metabolism remains constant. Three input measurements are thus needed: end tidal PCO_2, CO_2 excretion and oxygen consumption.



Arterial PCO_2 and end tidal PCO_2 following occlusion of a main branch pulmonary artery.

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