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CARDIOPULMONARY EFFECTS OF PRESSURE BREATHING DURING HYPOTHERMIA

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ABSTRACT

Continuous pressure breathing was studied in hypothermic anesthetized dogs. Alveolar ventilation decreased during continuous positive pressure breathing (CPPB) and increased during continuous negative pressure breathing (CNPB). The changes in alveolar ventilation were due to changes in respiratory rate as well as in respiratory dead space. Cardiac output fell significantly during CPPB due to a reduction in heart rate and stroke volume. During CNPB cardiac output was only slightly greater than during control as a result of a fall in heart rate and an increase in stroke volume. Oxygen consumption was reduced to 60% of control during CPPB of 16 cm H_2O, but was 25% greater than control during CNPB. Qualitatively, CO_2 production changed as did O_2 consumption, but was different quantitatively during CNPB, indicating hyperventilation due to increased respiratory rate. Mean pulmonary artery pressures and pulmonary resistance varied directly with the applied intratracheal pressure. The results indicate that the hypothermic animal can tolerate an imposed stress such as CPPB and can increase its O_2 consumption during CNPB as does the normothermic animal.

FOREWORD

The work reported herein was conducted by Duke University Medical Center, Durham, North Carolina 27702, under contract no. AF 33(657)8854 with the Aerospace Medical Research Laboratories. The work was performed in support of project 7163, Physiology Research, task 716302, Vital Organ Functions in Mammals. Mr. Donald A. Rosenbaum, Altitude Protection Branch, Life Support Division of the Biomedical Laboratory, was the technical contract monitor.

This technical report has been reviewed and is approved.

WAYNE H. McCANDLESS
Technical Director
Biomedical Laboratory
Aerospace Medical Research Laboratories

SECTION I

INTRODUCTION

Although the oxygen requirement of a hypothermic animal is less than that during normothermia it has been shown that an acidosis develops during and following periods of hypothermia. Ballinger et al. (ref 1) showed that excess lactate produced by hypoxic metabolism increased in anesthetized dogs during cooling to approximately 28 C, indicating an oxygen debt, hypoxic metabolism and a concomitant metabolic acidosis. Since Lenfant and Howell (ref 6) reported that oxygen consumption and cardiac output increase during continuous negative pressure breathing in normothermic anesthetized dogs the investigation herein reported was undertaken to determine if the hypothermic animal also responds in the same manner. The problem is of interest physiologically since little is known concerning the ability of a hypothermic animal to respond to an imposed metabolic stress. Continuous positive pressure breathing was also imposed in order to make a more complete comparison of the effects of continuous pressure breathing in normothermic and hypothermic animals.

SECTION II

METHODS

Eleven mongrel dogs weighing between 14 and 20 kg, mean wgt. of 17.2 kg, were used. Dial in Urethane, 50 mg/kg, was injected intraperitoneally and was not supplemented during the experiments. A tracheal cannula was inserted and catheters were placed under fluoroscopic guidance into the pulmonary artery and left atrium. Retrograde approach was used for the insertion of the catheter into the left atrium. A catheter was also inserted into the abdominal aorta. All catheters were hydraulically coupled with Statham pressure transducers, the outputs of which were recorded simultaneously on a Miller oscillograph.

End-tidal CO_2 was measured by an Beckman LBI CO_2 analyzer from a catheter attached to a sidearm of the tracheal cannula and to the microcatheter sampling cell of the instrument. Pulmonary ventilation was measured by collection of expired gas in plastic Douglas bags during a known time interval. Respiratory rate was recorded on an Esterline Angus recorder by using the intratracheal pressure fluctuations to activate a Statham Transducer. Tidal volume was calculated from pulmonary ventilation and respiratory rate. Oxygen consumption and CO_2 production were calcualted from the concentration of these cases in the expired gas and the pulmonary ventilation volume. Oxygen concentration was measured with a Beckman E-1 oxygen analyzer and CO_2 on

the Beckman LBI CO_2 analyzer. Anatomic dead space was calculated from the expired and alveolar CO_2 pressures and pulmonary ventilation using the Bohr formulation. Physiologic dead space was calculated from the Bohr equation using expired and arterial CO_2 pressures.

Blood samples were withdrawn anaerobically from the pulmonary artery and abdominal aorta. The arteriovenous oxygen difference was measured spectrophotometrically on hemolyzed blood samples (ref 4). Cardiac output was calculated from the oxygen consumption and arteriovenous oxygen differences using the direct Fick principle.

Arterial PCO_2 and pH were measured with Instrumentation Laboratories, Inc. electrodes and meter system at the temperature of the blood when withdrawn from the animal.

Continuous pressure breathing was accomplished by connecting the trachea in series with two 200-liter tanks. A plastic Douglas bag was inserted in the tank for collection of expired gas, the atmosphere around the bag (room air) was inspired, through appropriate valving. The tank was pressurized to + 8 and + 16 cm H_2O with a small pump and evacuated to -8 and -16 cm H_2O with the same pump. The capacity of the tank was such that the pressure fluctuations with breathing were only a few mm H_2O. Tank pressure was adjusted to compensate for changes in functional residual volume when the tank was initially connected to the trachea.

The procedure for each experiment involved observations during normothermia to indicate the general reactivity of the animal. The animal was then lowered into ice-water bath and cooled to 28 C as indicated from thermocouples in the rectum and vena cava. The temperature was maintained at ± .5 C by adjusting the bath temperature. Control observations were made followed by 10-minute exposure to each positive and negative pressure in random order. A recovery period of at least 15-minutes followed the exposure to a given pressure. Each animal was observed without pressure and to each of the four types of pressure breathing. Collection of data during pressure breathing was started when a steady state had been achieved as indicated by respiratory rate and heart rate.

The reliability of using respiratory rate and heart rate as indices of the steady state was verified by using a closed-circuit system to measure oxygen consumption during pressure breathing in a separate series of eight experiments on four dogs. A Krogh-type spirometer containing CO_2 absorber was placed within a sealable wooden box having a volume of 231 liters. By using appropriate valves and a pump the barrel-

box-spirometer system was pressurized or decompressed as desired. The large volume of the system containing the spirometer resulted in only small fluctuations in the applied pressure as a result of gas exchange between the animal and spirometer. The animals were exposed to positive and negative pressures of 8 and 16 cm H_2O in random order using 100% oxygen as the inspired gas. Duration of exposure to a given pressure varied from 6 to 10 minutes and with a 15-minute recovery period between exposures. In every experiment during continuous positive or negative pressure breathing, a steady state of oxygen consumption was reached within 30 seconds to 2 minutes (the longer times being associated with CPPB) and that there were no significant changes in respiratory rate or heart rate when oxygen consumption was in a steady state. Therefore, it is felt that in the open-circuit system use of respiratory rate and heart rate as indices of steady state during pressure breathing was valid.

SECTION III

RESULTS

The influence of continuous positive pressure breathing (CPPB) and continuous negative pressure breathing (CNPB) on pulmonary ventilation, tidal volume and respiratory rate is presented in Figure 1. Pulmonary ventilation decreased during CPPB and increased during CNPB. Tidal volume increased slightly during CPPB at 8 cm H_2O but was essentially unchanged from control at 16 cm H_2O. During CNPB tidal volume decreased 13% at -8 cm H_2O pressure and decreased 20% at -16 cm H_2O pressure. Respiratory rate decreased 30% during CPPB but was almost double the control values during negative pressure breathing. The magnitude of the changes in respiratory rate were independent of the level of tracheal pressure during negative pressure breathing.

Alveolar ventilation, figure 1, like pulmonary ventilation, decreased during positive pressure breathing and increased during negative pressure breathing. The decrease in alveolar ventilation during CPPB resulted from a combination of the fall in respiratory rate and an increase in physiologic dead space, table I. The increase in physiologic dead space during CPPB at 8 cm H_2O pressure is shown to be primarily the result of an increase in anatomic dead space when the change in tidal volume (an increase) is taken into consideration. The physiologic dead space was 44% of the tidal volume during the control period, and the anatomic dead space was 41% of the tidal volume. The distribution or alveolar dead space would then be 3% of the tidal volume. During CPPB at 8 cm H_2O pressure the physiologic dead space increased from 44% of the tidal volume during the control periods to 52% of the tidal

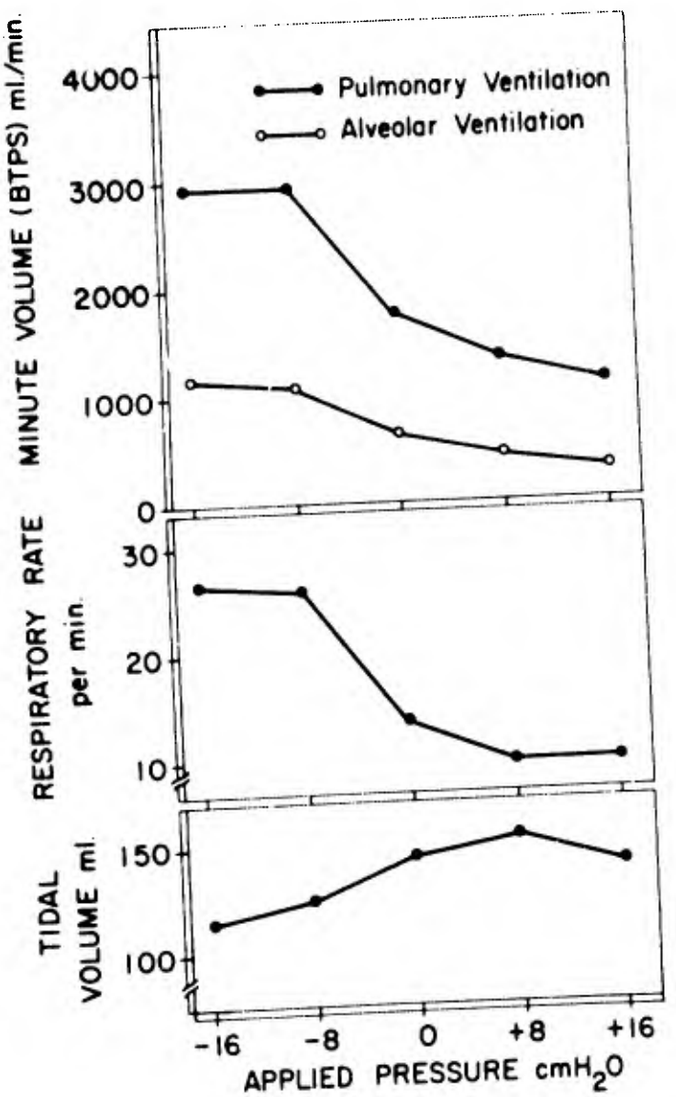

Figure 1. The effects of continuous positive and continuous negative pressure breathing on respiratory functions in hypothermic anesthetized dogs. The magnitude of the applied pressure is indicated on the abscissa, O cm H_2O applied pressure represents control.

Table 1.

Respiratory dead space as influenced by continuous pressure breathing in hypothermic anesthetized dogs.

	Control	Pressure, cm H_2O			
		+8	+16	-8	-16
Anatomic Dead Space ml*	60	75	80	46	37
Physiologic Dead Space ml*	63	80	85	52	48
$\frac{\text{Anatomic Dead Space}}{\text{Tidal Volume}}$	.41	.49	.57	.37	.32
$\frac{\text{Physiologic Dead Space}}{\text{Tidal Volume}}$	.44	.52	.60	.42	.42

* corrected for instrument dead space

volume, the anatomic dead space was 49% of the tidal volume and hence the alveolar dead space remained the same as during the control, i.e., 3% of the tidal volume, table I. Physiologic dead space during CPPB at 16 cm H_2O was 85 ml or 60% of the tidal volume, table I. Again the increase in physiologic dead space was the result of an increase in anatomic dead space, since the alveolar dead space as a percentage of the tidal volume remained equal to that recorded in the control condition. Alveolar ventilation during CPPB at 16 cm H_2O was less than half that during the control. This decrease is the result of a 30% reduction in respiratory rate and a 27% increase in physiologic dead space.

Alveolar ventilation during CNPB at 8 cm H_2O was 70% greater than during the control, figure 1. Respiratory rate was almost twice that during the control, whereas tidal volume was slightly less than control, figure 1. Physiologic dead space decreased both in absolute volume and as a percentage of the tidal volume. Physiologic dead space during CNPB at 8 cm H_2O, table I, was 42% of the tidal volume and anatomic dead space was 37%. Alveolar dead space was therefore 5% of the tidal volume, an increase over that of the control period. This would indicate that at 8 cm H_2O CNPB an increase in alveolar dead space is beginning to develop. CNPB at 16 cm H_2O resulted in qualitatively the same changes from control as those seem at 8 cm H_2O. The alveolar dead space, however, at 16 cm H_2O CNPB was 10% of the tidal volume.

Oxygen consumption rate and CO_2 production rate are shown in Figure 2. Oxygen consumption decreased during CPPB and at 16 cm H_2O was 40% less than during control. Oxygen consumption during CNPB was 25% greater than during the control period. The decrease in oxygen consumption during CPPB was dependent on the level of pressure applied, decreasing as the pressure increased. During CNPB the increase in oxygen consumption rate was independent of the level of pressure applied. CO_2 production rate, figure 2, decreased during CPPB and similar to O_2 consumption was dependent upon the level of pressure. Imposition of CNPB resulted in a 60% increase in CO_2 production, but like O_2 consumption, CO_2 was independent of the applied pressure. The respiratory exchange ratio was 0.70 with not applied pressure, 0.65 at 8 cm H_2O CPPB and 0.61 at cm H_2O CPPB. The application of CNPB resulted in a relatively greater increase in CO_2 production than in O_2 consumption, such that the R.Q. was increased from the control level of 0.70 to 0.93 with 16 cm H_2O CNPB with a concomitant reduction of alveolar CO_2 values as shown in Figure 3.

The alveolar and arterial CO_2 pressures and pH as influenced by pressure breathing are presented in figure 3. Alveolar and arterial CO_2 pressures increased during

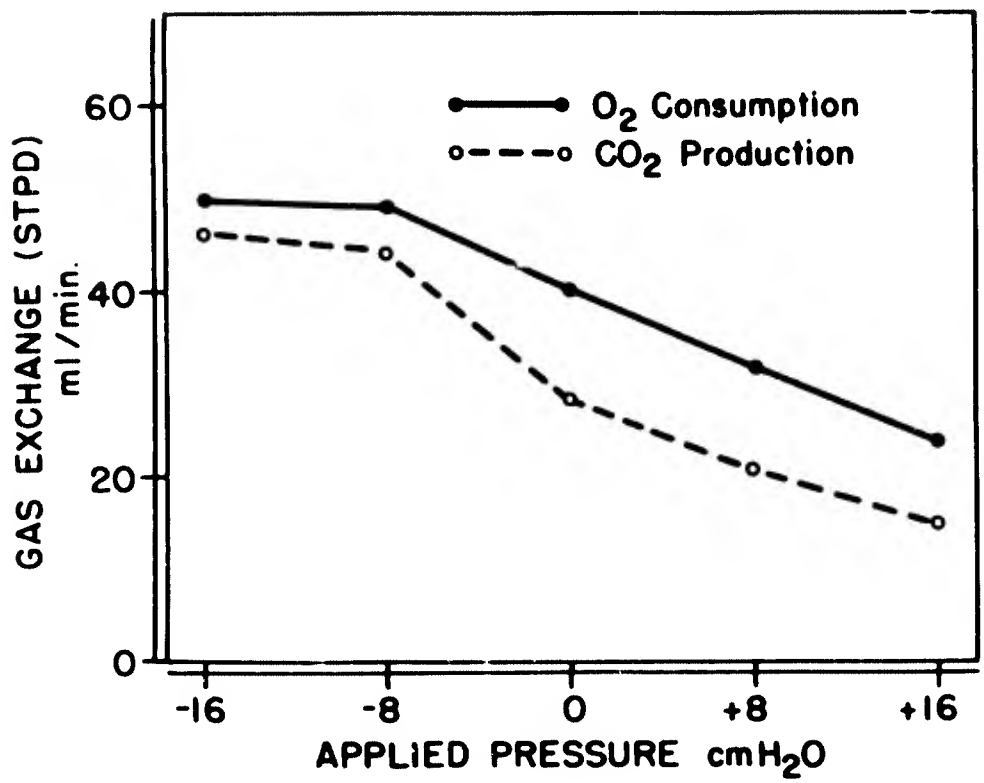

Figure 2. Metabolic gas exchange during continuous positive and continuous negative pressure breathing in hypothermic dogs. The magnitude of the applied pressure is indicated on the abscissa, O cm H_2O applied pressure represents control.

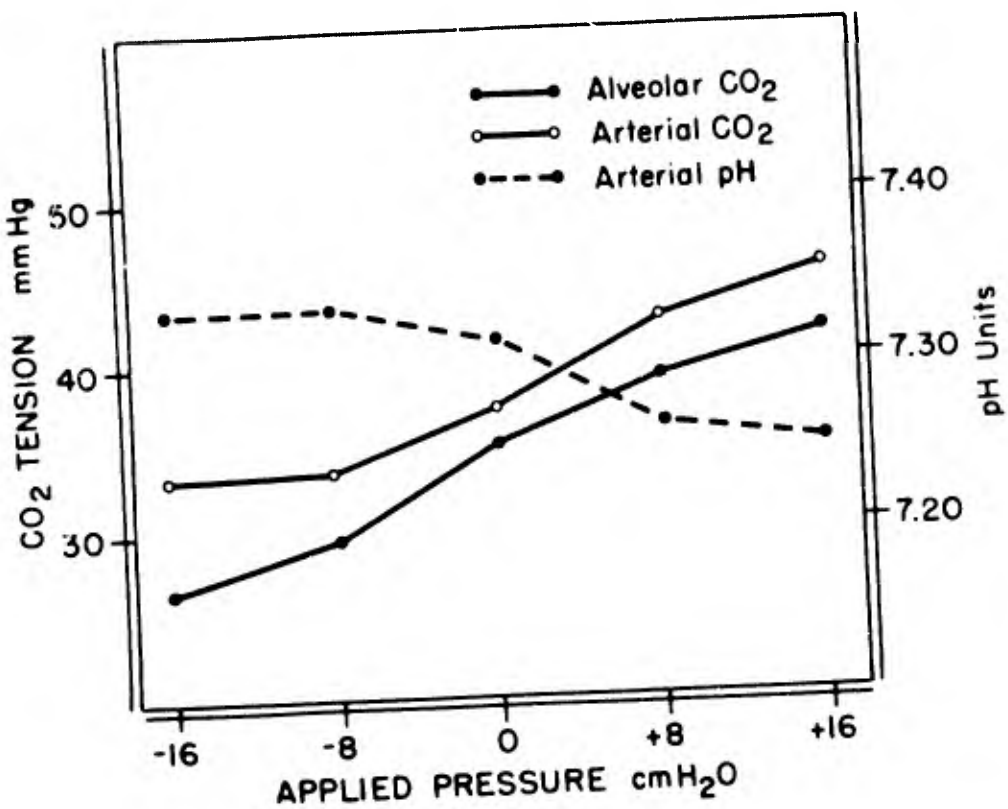

Figure 3. The effects of continuous positive and continuous negative pressure breathing on blood gases and pH in hypothermic dogs. The magnitude of the applied pressure is indicated on the abscissa, O cm H_2O applied pressure represents control.

CPPB and decreased with CNPB. The increases in CO_2 pressures parallel the reduction of alveolar ventilation during CPPB, figure 1, and the decreased CO_2 production rate, figure 2. As a result of an increase in arterial PCO_2, there was a reduction in pH during CPPB. The alveolar and arterial CO_2 values during CNPB were less than during control. Again, these changes reflect the increase in alveolar ventilation and the increase in the respiratory exchange ratio. As a result of the decrease in arterial PCO_2 the pH during CNPB was greater than control. The arterial-alveolar CO_2 pressure gradient was greatest during 16 cm H_2O CNPB at which time the alveolar dead space-tidal volume ratio was also the greatest.

Cardiac output decreased significantly with positive pressure breathing and increased slightly with negative pressure breathing, figure 4. The fall in cardiac output with CPPB was correlated with the imposed pressure such at 8 cm H_2O cardiac output was reduced by 33% and at 16 cm H_2O it was less than half that during the control. During negative pressure breathing cardiac output was increased approximately 10% over control levels and was relatively independent of the level of pressure.

Heart rate, figure 4, decreased from control during CPPB but was not different from control during CNPB. As occurred with cardiac output, the magnitude of the changes in heart rate during CPPB was correlated with the level of CPPB. Systemic pressures were not significantly influenced by either CPPB or CNPB.

Stroke volume, calculated from the cardiac output divided by heart rate, decreased 24% during CPPB of 8 cm H_2O and 45% during CPPB at 16 cm H_2O pressure. The decreases in stroke volume were of greater magnitude than the decrease in heart rate which would indicate that the fall in cardiac output during CPPB is the result primarily of a decrease in stroke volume and secondarily attenuated by the fall in heart rate.

Mean pulmonary artery pressure, figure 5, was increased by CPPB and decreased by CNPB. Pulmonary resistance, expressed as P.R.U. was approximately doubled during CPPB at 8 cm H_2O and was three times greater than control during CPPB at 16 cm H_2O.

Ventilation perfusion ratio, calculated from the ratio of alveolar ventilation to cardiac output, during control was 0.58. CPPB had no significant effect on this ratio. Therefore, the reductions in alveolar ventilation and cardiac output, during CPPB are of the same order of magnitude. However, during CNPB the ventilation-blood flow ratios were significantly greater than that of the control, indicating that during CNPB alveolar ventilation increased to a greater extent than did cardiac output.

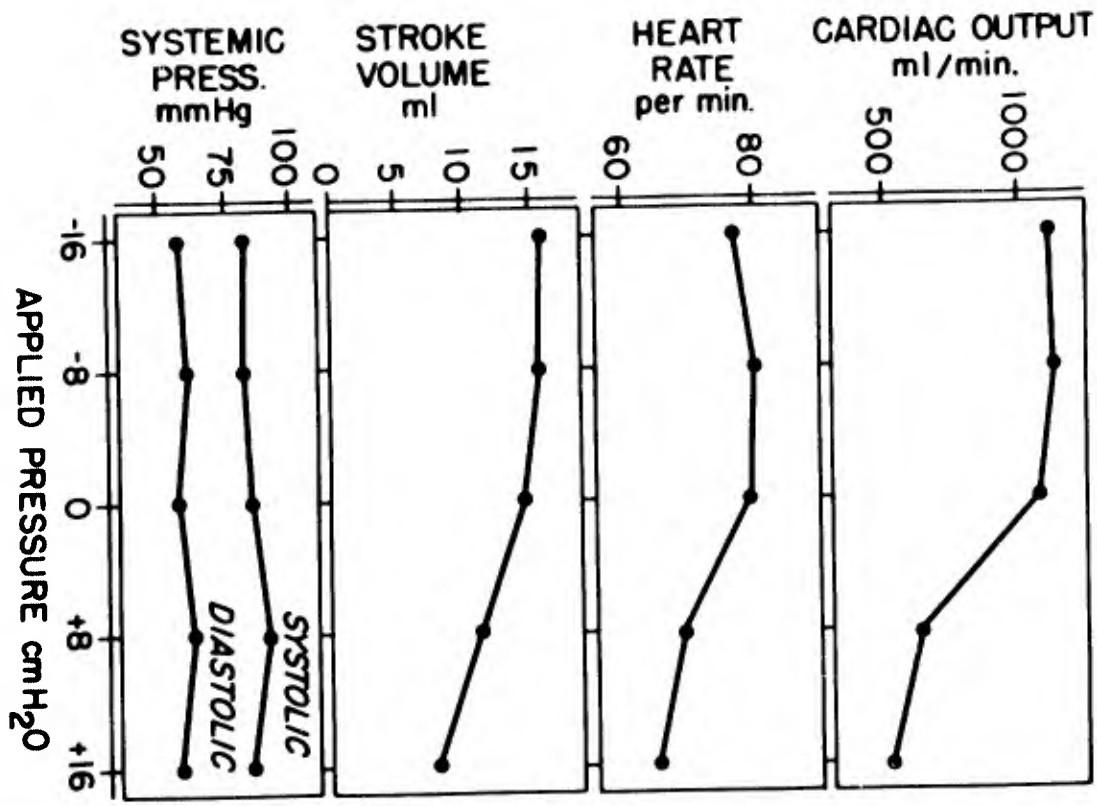

Figure 4. Cardiovascular responses to continuous positive and continuous negative pressure breathing of hypothermic dogs. The magnitude of the applied pressure is indicated on the abscissa, O cm H_2O applied pressure represents control.

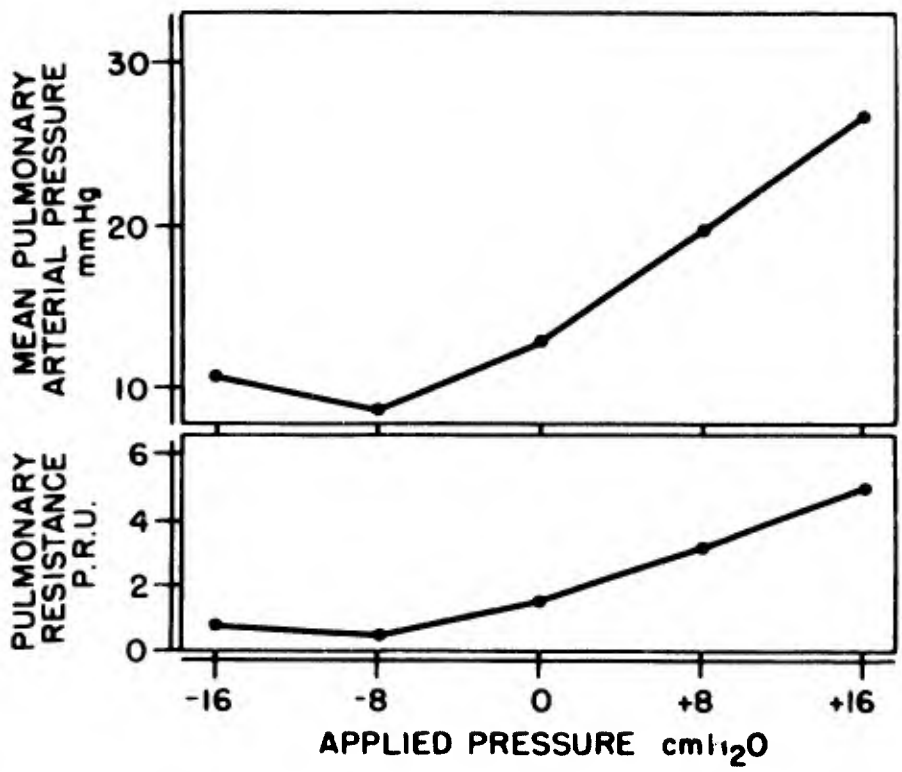

Figure 5. Pulmonary vascular changes induced by continuous positive and continuous negative pressure breathing in hypothermic dogs. The magnitude of the applied pressure isindicated or. the abscissa, O cm H_3O applied pressure represents control.

SECTION IV

DISCUSSION

The changes in respiratory dead space observed with positive pressure breathing indicate that as a result of increased intrapulmonary pressure there was a distension of the conducting airways and a concomitant increase in anatomic dead space. Shepard et al. (ref 9) have reported that as a result of positive pressure breathing in normothermic human subjects, anatomic dead space increased at transmural pressures greater than 10 cm H_2O. Kaufman (ref 5) found a 68% increase in anatomic dead space during positive pressure breathing (18.5 cm H_2O) in a group of six adult male subjects. Folkow and Pappenheimer (ref 2) showed that series dead space, that portion of respiratory dead space associated with the volume of the respiratory passages leading to the lung alveoli, increased with increasing transpulmonary pressure, reaching 150% of its normal value when the transpulmonary pressure was raised to 15-20 cm H_2O in anesthetized cats. They also found a 50% increase in series dead space in two human subjects.

Negative pressure breathing resulted in a decrease in anatomic dead space, presumably as a result of reduction in volume of the distensible bronchial tree. In normothermic supine subjects, Haab and Cimino (ref 3) found that anatomic dead space decreased during negative pressure breathing, whereas Kaufman (ref 5) reported a significant increase in anatomic dead space during negative pressure breathing. Kaufman attributed the increase in anatomic dead space during pressure breathing to reflex inhibition of constrictor tonus normally present in the respiratory air passages. The data on these hypothermic, anesthetized dogs indicate that the anatomic dead space varied directly with the transthoracic pressure.

Physiologic dead space in these hypothermic anesthetized dogs was not significantly changed during positive pressure breathing but increased significantly over control values during CNPB at 16 cm H_2O. This increase in physiologic dead space was reflected in the arterial-alveolar CO_2 gradient, which was also significantly greater at 16 cm H_2O CNPB than during control.

Folkow and Pappenheimer (ref 2) reported that in two human subjects parallel dead space (well ventilated but poorly perfused alveoli) most unexpectedly developed during positive pressure breathing. The dead space was equivalent to that expected if 10% of the ventilated alveoli were not perfused with blood. Physiologic dead space during negative pressure breathing has not been reported in normothermic anesthetized dogs. However, Lenfant and Howell (ref 6) have shown in anesthetized dogs during negative pressure breathing that the O_2 content of arterial blood was lower than control values even though the inspired O_2 tension was maintained above 300 mm Hg. They believed this observation could be explained by assuming that atelectasis developed at small lung volumes. By applying the shunt equation they calculated that at a

transthoracic pressure difference of -15 cm H_2O, 32% of the pulmonary flow perfused atelectatic alveoli. Our data on physiologic dead space in hypothermic anesthetized dogs agrees with their interpretation of the phenomenon observed in normothermic animals. The discrepancy between the result of anesthetized dogs and unanesthetized human subjects may reflect some compensatory mechanisms occurring in the unanesthetized subject that do not occur in the anesthetized dog.

The decrease in respiratory rate with positive pressure breathing is most likely the result of increased neural activity from the Hering-Breuer reflex as a result of lung distension. A decrease in respiratory rate in normothermic intact dogs during the application of 18 cm Hg CPPB has been reported by Marotta and Harner (ref 7), whereas respiratory rate was essentially unaltered by CPPB in vagotomized dogs. In 1962, we reported (ref 8) that hypothermic dogs also retain vagal afferent reflex activity. Therefore, in positive pressure breathing in hypothermic animals the decrease in rate is assumed to be the result of lung distention. The increased respiratory rate during negative pressure breathing might also be attributed to activity of the vagal afferents due to forceful deflation of the lungs.

A decrease in heart rate in normothermic dogs during positive pressure breathing has been reported by Marotta and Harner (ref 7) and by Lenfant and Howell (ref 6). Furthermore, Marotta and Harner have shown that heart rate of vagotomized dogs remained essentially unchanged throughout CPPB. Lenfant and Howell's data show essentially no change in heart rate during negative pressure breathing in normothermic dogs. The data herein reported indicate that the heart rate changes in hypothermic animals during pressure breathing are similar to those in normothermic animals.

Systemic arterial pressure increased approximately 9% during CPPB at 8 cm H_2O, but was not significantly different from control during negative pressure breathing. These results are somewhat different from those of Lenfant and Howell (ref 6) who reported a fall in arterial pressure during positive pressure breathing but no change during negative pressure breathing. Marotta and Harner (ref 7) found that femoral arterial pressure decreased significantly during CPPB. Responses of vagotomized animals paralleled those of intact animals. Changes in systemic arterial pressures in these hypothermic animals are similar to those of normothermic animals during pressure breathing.

The fall in cardiac output during CPPB and the slight increase over control values during CNPB agree with the results of Lenfant and Howell (ref 6) for normothermic dogs. The decrease in cardiac output during CPPB resulted mostly from a fall in stroke volume, which varied directly with the magnitude of applied pressure during CPPB and indicated a decrease in venous return as a result of elevated intrathoracic pressure.

The decreased O_2 consumption during positive pressure breathing paralleled the reductions in alveolar ventilation and in cardiac output. The magnitude of decrease in O_2 consumption during positive pressure breathing was dependent on the level of applied pressure and was greater than reported for normothermic dogs by Lefant and Howell (ref 6). During negative pressure breathing O_2 consumption was 25% greater than during control periods and the increase was not dependent on the level of applied pressure. This increase in O_2 consumption in response to negative pressure breathing is considerably greater than that in normothermic dogs (ref 6). The increased O_2 consumption in response to negative pressure breathing indicated that at 28 C the anesthetized dog is able to meet additional demands, in terms of O_2 usage, even though a metabolic acidosis may be present with lesser O_2 requirements. This implies that although regulation may be marginal it is still possible to supply additional energy when a stress such as negative pressure breathing is imposed.

Carbon dioxide production rate during the control period was less than O_2 consumption such that the respiratory quotient was 0.70. Carbon dioxide production decreased with positive pressure breathing and increased with negative pressure breathing as did the O_2 consumption. The changes of CO_2 production and O_2 consumption during positive pressure breathing were such that R.Q. was decreased slightly from that of the control period. However, during negative pressure breathing CO_2 evolution was relatively greater than O_2 consumption and the R.Q. increased from 0.70 during control to 0.93 during negative pressure breathing at 16 cm H_2O. The increased CO_2 production was accompanied by an increase in respiratory rate and is most probably hyperventilation initiated by deflation reflexes, since the arterial and alveolar CO_2 tensions were also less during negative pressure breathing than during control.

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13 ABSTRACT

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