

The Cardiorespiratory Effects of Constant and Intermittent Positive-pressure Breathing

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Twelve patients in need of controlled mechanical ventilation alternately received IPPB and constant positive-pressure breathing with 5 cm H₂O end-expired pressure (CPPB-5). Intrapulmonary shunting, cardiac output, and alveolar and arterial oxygen washin curves were determined during both types of ventilation, and washin curves were compared with those of six patients breathing spontaneously. Intrapulmonary shunting was significantly less when patients received CPPB-5; those with shunts greater than 30 per cent had the greatest reductions in shunt. Cardiac output was slightly, but not significantly, reduced. Alveolar oxygen washin curves were unchanged with positive-pressure breathing. Arterial oxygen washin was prolonged in patients receiving either IPPB or CPPB-5, indicating new areas of over-perfusion relative to ventilation. No pneumothorax or other adverse effect was found with CPPB-5. (Key words: Constant positive-pressure breathing; Intermittent positive-pressure breathing; Cardiac output; Intrapulmonary shunting.)

RENEWED INTEREST in constant positive-pressure breathing (CPPB) as an alternative to IPPB has grown from favorable reports of its use in patients with severe respiratory problems.¹⁻⁴ There have been reports of improvement in arterial oxygenation with sustained pressures of 1-2 cm H₂O to 13 cm H₂O. The likelihood of pneumothorax and cardiovascular complications increases as mean airway pressure increases, however, and any potential value of CPPB may be negated by the complications it induces.

Recent laboratory evidence indicated that CPPB without expiratory resistance but with 5 cm H₂O end-expiratory pressure minimized

increases in airway pressure, yet was effective in reducing intrapulmonary shunting.⁵ The present study was designed to evaluate and compare the efficacy of IPPB and CPPB-5 in 12 patients with respiratory insufficiency and to determine the safety of CPPB-5 as a routine method of mechanical ventilation.

Methods

Patients selected for study were receiving controlled respiration in the surgical intensive-care ward. Seven patients had undergone cardiac surgery, four were multiple-injury patients with crushed chests, and one patient had had replacement of a dissecting abdominal aneurysm. All patients had radial artery and central venous catheters in place. Patients were ventilated with IPPB and with CPPB-5 for periods of two hours, using the Bennett MA-1 respirator. The order was reversed in alternate patients. Respiratory rate, tidal volume, and inspiratory flow rate were initially adjusted to meet each patient's requirements and were not subsequently changed during the period of study. Tidal volumes were 10-12 ml/kg and mean inspiratory rate was 17/min. This ventilation produced mean PaCO₂ values of 31 ± 8 torr during IPPB and 33 ± 8 torr during CPPB-5. Inspired O₂ concentration was maintained at 40 per cent. Two-milligram doses of morphine sulfate were occasionally given intravenously to control restlessness. No other medication was given during the period of study.

The following studies were done at the end of the initial period of ventilation. Cardiac output was determined by the dye-dilution technique using a Beckman Cardiodensitometer. Intrapulmonary shunting during breathing of 100 per cent O₂ was determined from analysis of arterial and mixed venous blood, using standard formulae.⁶ Oxygen content of blood was determined from the hemoglobin concentration and HbO₂, utilizing the IL Model 182 CO-Oximeter.

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Since CPPB-5 increases functional residual capacity, which may affect \dot{V}/\dot{Q} , alveolar and arterial oxygen tensions were monitored during a switch from 40 to 100 per cent oxygen inhalation during both IPPB and CPPB-5. The time taken to reach a new alveolar or arterial oxygen tension level, while independent of the magnitude of the change in inspired oxygen tension, reflects abnormalities in ventilation and perfusion.⁷⁻¹² Changes in arterial oxygen tension were recorded continuously. Blood was drawn from the radial artery at a constant rate of 10 ml/min using a Holter roller-head pump. The blood was constantly heparinized using a Braun infusor to a concentration of 4 units/ml. The heparinized blood was then pumped through a heated Beckman flow-through chamber which incorporates an oxygen electrode. The electrode signal was amplified with a Beckman Model 160 Physiological Gas Analyzer and recorded with a Heath potentiometric recorder. Analyzed blood was then pushed into a vented Abbott Hemoseal, filtered, and returned via drip into the venous circulation. The 90 per cent response time of the system and recorder to a "square-wave" increase in oxygen tension was 48 seconds, and the maximum rate of increase was 17 torr/sec. The 90 per cent response time to a "square-wave" decrease in oxygen tension was 28 seconds and the maximum rate of decrease was 29 torr/sec. The discrepancy is assumed to result from dampening of responses during increases in oxygen tension and augmentation of responses during decreases caused by the high oxygen consumption of the Beckman electrode #670552. The total response time of the system, however, was at least three times as fast as that of arterial blood during washin.

Alveolar oxygen was monitored by continuously withdrawing gas from a Rahn end-tidal sampler at a flow of 200 ml/min using a Beckman Microcatheter sample pump. The gas was drawn through a Beckman C-2 Pauling paramagnetic oxygen analyzer and recordings were made at 30-second intervals. Response time of the C-2 oxygen analyzer to a "square-wave" change in oxygen tension was 4 seconds. Following respiratory and blood-gas measurements, ventilation was changed to either IPPB or CPPB-5 for 1-2 hours. All measurements were then repeated.

TABLE 1. Effects of IPPB and CPPB-5 on Cardiac Output and Shunting (Mean Values \pm SE)

	Number of Patients	IPPB	CPPB-5	P
Cardiac output (l/min)	12	5.5 \pm 0.8	4.6 \pm 0.6	N.S.
$\dot{Q}_v/\dot{Q}_a \times 100$	12	25 \pm 3.6	20 \pm 2.7	<0.02
$\dot{Q}_v/\dot{Q}_a > 30$ per cent	6	37 \pm 1.2	28 \pm 2.4	<0.005
$\dot{Q}_v/\dot{Q}_a < 30$ per cent	6	13 \pm 1.4	12 \pm 1.4	N.S.

Each patient served as his own control, receiving both IPPB and CPPB-5, in an attempt to control intragroup variability. The initial method of ventilation was alternated with the other in an attempt to control sequential changes which may occur with progressive therapy. Statistical analysis was by Student's t test for paired data. The effects of spontaneous respiration on alveolar and arterial oxygen washin times were determined by studying six additional postoperative patients recently weaned from respirators, breathing spontaneously, but prior to nasotracheal extubation.

Results

The effects of IPPB and CPPB-5 on cardiac output and shunting are listed in table 1. Intrapulmonary shunting was 20 per cent less in the 12 patients treated with CPPB-5, compared with IPPB alone. CPPB-5 had little effect on shunting in patients with shunts of less than 30 per cent. Six patients with shunts of more than 30 per cent, however, experienced a 24 per cent reduction in shunting with CPPB-5, which was highly significant.

Reduction in shunting was associated with a slight but not significant reduction in cardiac output with CPPB-5. A change to IPPB or CPPB-5 did not affect the cardiac outputs of four patients with outputs below 3 l/min. Mean P_{aO_2} values were 215 \pm 46 torr with IPPB and 241 \pm 43 torr with CPPB-5. Because of the slightly lower cardiac output (CO) associated with CPPB-5, however, the total oxygen delivery ($CO \times CaO_2$) was somewhat less (802 ml/min) with CPPB-5 than with IPPB (946 ml/min).

Figure 1 shows the mean alveolar and arterial oxygen washin curves of seven patients following changes from 40 to 100 per cent

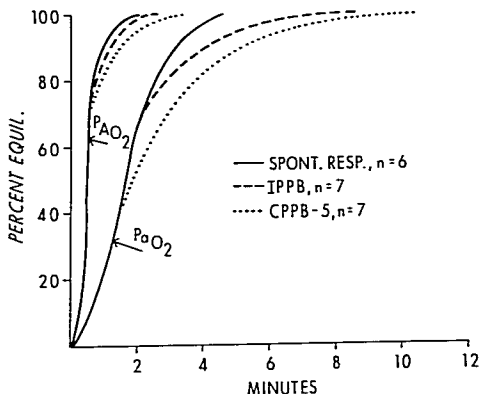


FIG. 1. Alveolar and arterial oxygen washin curves plotted as percentages of full equilibration during IPPB, CPPB-5, and spontaneous respiration (mean values).

oxygen inhalation during both IPPB and CPPB-5, and those of six other patients breathing spontaneously at a mean rate of 24 respirations/min. Arterial and alveolar washin curves are plotted beginning with the first observed change in tension to correct for circulation and system lag times. During spontaneous respiration, alveolar washin was complete in 2 minutes; arterial washin generally paralleled alveolar washin, and was complete in 4.6 minutes. During IPPB and CPPB-5, at a rate of 17 respirations/min, alveolar washin was complete in 2.8 and 3.4 minutes, respectively. Arterial washin was considerably delayed during IPPB, however, reaching final equilibrium in 8.6 minutes. Arterial washin with CPPB-5 was even slower, requiring 10.2 minutes for final equilibration.

Although a loose correlation ($r = 0.39$) between arterial washin time and cardiac output was evident, no relationship between arterial washin time and the size of the intrapulmonary shunt could be demonstrated.

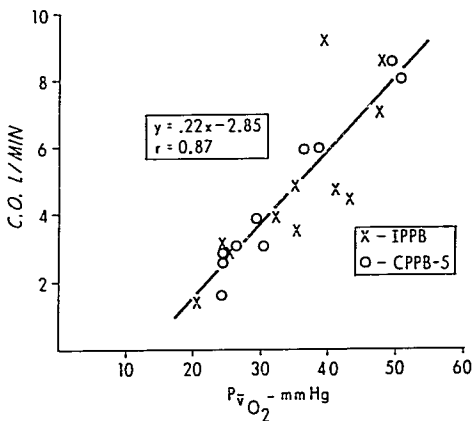
Discussion

Since intrapulmonary shunting was less during CPPB-5 than during IPPB, more alveoli must have been better ventilated and perfused. The increases in mean and end-expiratory pressures associated with CPPB-5 result in increased FRC. Hypoinflated alveoli tend to expand, and normally inflated alveoli will distend when subjected to increased pressure.

Were perfusion of the lung to remain unchanged, the \dot{V}/\dot{Q} ratio would increase and reduce that portion of the total shunt caused by a ventilation-perfusion inequality. It might be inferred that weaning patients from high inspired oxygen tensions to room air while they are on CPPB-5 should be accomplished with less of an increase in total shunt than weaning during spontaneous respiration. There is supporting laboratory evidence that \dot{V}/\dot{Q} abnormalities do contribute less to the total physiologic shunt during IPPB and CPPB-5 with air inhalation than during spontaneous breathing of air.⁸

Delays in arterial oxygen washin during IPPB and CPPB-5, however, suggest that changes in perfusion do occur. While our patients' arterial washin parallels alveolar washin during spontaneous respiration, IPPB and CPPB-5 produced progressive delays in arterial oxygen washin (fig. 1). The exponential behavior of the terminal portions of the curves was characteristic of that seen with chronic obstructive pulmonary disease associated with increases in FRC.^{9, 11, 12} In normal subjects, Lenfant and Okubo, studying alveolar and arterial washin curves, found that during spontaneous respiration, PaO_2 rose extremely fast to a plateau, with the peak distribution of pulmonary blood flow occurring in less than 0.4 minutes and completed in 4 minutes, and arterial washin closely followed the distribution functions of lung volume and total ventila-

FIG. 2. Simultaneous cardiac output and $P\bar{v}O_2$ determinations in patients ventilated with 40 per cent oxygen.



tion.⁹ Since alveolar washin was not significantly delayed by positive-pressure breathing in our patients, explanations other than ventilation defects are needed to account for the delay in arterial washin: 1) A reduction in cardiac output could account for some delay. 2) Positive-pressure ventilation induces a variable, and at times intermittent, flow to pulmonary capillary blood, since peak airway pressures often exceed pulmonary artery pressures. An overall increase in pulmonary capillary transit time would favor oxygen uptake in overperfused areas, would have little effect on uptake from areas of normal perfusion, but would retard overall uptake. 3) Partial redistribution of pulmonary blood flow through shunt pathways of lesser resistance during inflation could also induce a flattening of the arterial oxygen washin curve, and has been cited as a possible explanation of increased shunting in patients with intracardiac defects receiving CPPB.⁴ 4) Mixed venous blood may equilibrate only partially with alveolar gas in newly inflated alveoli that are markedly overperfused; as PA_{O_2} increases during breathing of 100 per cent oxygen, an increasing alveolar-pulmonary capillary oxygen gradient would expedite the transfer of O_2 to the blood. Because of the increased uptake of O_2 by the blood, an inordinate decrease in alveolar size during expiration will occur. In these alveoli, unless alveolar ventilation increases during inspira-

tion, PA_{N_2} will remain higher than in adequately ventilated alveoli and will retard O_2 equilibration of venous blood with inspired O_2 . If O_2 uptake by the blood increases sufficiently to exceed replacement during alveolar inspiration, alveolar collapse may occur. Briscoe has suggested that alveoli with sufficiently low \dot{V}/\dot{Q} ratios may behave like a shunt during oxygen breathing.¹⁰ The final tension of the arterial blood would be approached exponentially and achieved when a fixed rate of oxygen uptake from overperfused areas is reached. Any persisting alveolar-capillary oxygen tension gradient from these areas would affect the calculation of true or anatomic shunt.

The absence of cardiorespiratory complications with 5 cm end-expired pressure in this group of patients and the significantly lower shunts observed with CPPB-5 suggest that this method of therapy can be used safely and with benefit in critically ill patients. Oxygen delivery was more than adequate during inhalation of 100 per cent oxygen with both IPPB and CPPB-5. If arterial desaturation were present, however, as during inhalation of air or possibly with various mixtures of air and oxygen, small increases in Pa_{O_2} which CPPB-5 may effect could significantly increase oxygen delivery to the body by increasing Ca_{O_2} , assuming cardiac output was unchanged. The present study does not answer this important question.

Complications related to sustained airway pressures greater than 5 cm H₂O have been reported, however, and should forestall the routine application of CPPB, particularly for patients with small shunts, for whom CPPB has little to offer. Reported complications include tension pneumothorax, subcutaneous and mediastinal emphysema, and decreased urinary output.^{2,4} Finally, arterial saturation may be reduced still further when patients with intracardiac shunts are exposed to positive-pressure ventilation, presumably by redistribution of blood through cardiac shunts during lung inflation.⁴ Strong, Keats, and Cooley have warned of the potential hazards of excessive positive-pressure ventilation in patients with low pulmonary blood flows associated with cardiac defects.¹² They have demonstrated, however, that patients with tetralogy of Fallot can tolerate CPPB-5 without systemic hypotension and with improvement in Pa_{O₂}. The need for CPPB in patients with uncorrected cardiac defects should be carefully evaluated, however, since marked reductions in blood pressure will increase shunting.

Certain extra precautions should be taken during CPPB to forestall complications: 1) While reductions in cardiac output associated with CPPB are usually small, the likelihood of marked depression exists, particularly in the hypovolemic or marginally compensated cardiac patient. We have found that measurement of the mixed venous oxygen tension in patients ventilated with 40 per cent oxygen is a useful reflection of cardiac output (fig. 2). With due regard to the effects of temperature, pH, and 2,3-diphosphoglycerate (2,3-DPG)¹⁴ levels on the oxygen dissociation curve, mixed venous oxygen tensions below 25–30 torr dictate prompt evaluation of the patients' circulatory status and should include consideration of withdrawing CPPB. 2) Patency of chest tubes must be maintained during CPPB to prevent tension pneumothorax, particularly in patients with chest injuries. The negative intrapleural pressure induced by tubes under constant suction placed near contused or emphysematous lung, in conjunction with CPPB, may exert sufficient transpulmonary pressure to rupture the lung. Either water-seal drainage or minimal constant negative pressure in chest tubes will make this source of rupture in patients receiving CPPB less likely.

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