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PEEP and CPAP Following Open-heart Surgery in Infants and Children

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The cardiorespiratory effects of 5 cm H_2O end-expiratory pressure were studied in 22 infants and children an hour after open-heart surgery during mechanical ventilation with positive end-expiratory pressure (PEEP) and prior to endotracheal extubation approximately 15 hours later during spontaneous breathing (CPAP). Thermodilution cardiac output determinations and respiratory airflow, volume and pressure recordings were made to assess the effects of airway pressure changes on the respiratory waveform and oxygen delivery.

Neither PEEP nor CPAP had a significant effect on cardiac output, intrapulmonary shunting, oxygen consumption, or oxygen utilization. Patients who had had pulmonary hypertension preoperatively did not behave differently from those without pulmonary hypertension when removed from ventilatory support. Expiratory airflow was significantly prolonged when positive end-expiratory pressure existed during both controlled and spontaneous respiration. During CPAP, this "expiratory braking" was associated with an increase in tidal volume and decreases in respiratory rate and minute volume. Because of the lack of improvement in cardiopulmonary function in this group of patients, and the possibility of untoward effects from sustained end-expiratory pressure, PEEP and CPAP might properly be reserved as temporary supportive techniques should respiratory function be compromised. (Key words: Heart: cardiac output. Oxygen: consumption. Lung: function; shunting; vascular resistance. Ventilation: positive end-expiratory pressure; continuous positive airway pressure; mechanical.)

THE MAINTENANCE of positive end-expiratory pressure (PEEP) during controlled or spontaneous respiration increases functional residual capacity, may decrease the alveolar arterial oxygen tension difference and, in the presence of severe respiratory distress, may permit the inspired oxygen concentration ($F_{I_{O_2}}$) to be decreased to safer levels.[1] Both PEEP and continuous positive airway pressure (CPAP) have been used during postoperative ventilation and during weaning from respiratory support to produce these salutary effects.[2–6] Little information is available, however, regarding the effects of these techniques on cardiac output, oxygen consumption and the breathing pattern in infants and children.[7–8]

The purpose of this study was to obtain this information and to determine whether the cardiorespiratory effects of 5 cm H_2O PEEP or CPAP in infants and children following open-heart surgery provide any benefit that might justify their routine use.

Methods

Twenty-two patients, 15 months to 8 years in age, were selected for study. The subjects ranged in weight from 7 to 22 kg (mean 14 kg). Nine patients underwent repair of an atrial septal defect; six, repair of a ventricular septal defect; five, complete repair of a tetralogy of Fallot; one each, repair of a total anomalous venous return and correction of an endocardial cushion defect. Five of 15 patients for whom preoperative catheterization data were available had mean pulmonary arterial pressures greater than 25 torr (table 1).

All patients received controlled mechanical ventilation after operation until the following morning, when fully reacted, hemodynamically stable and ready

ABBREVIATIONS

BP	= blood pressure
CI	= cardiac index
HR	= heart rate
$Cao_2 - C\bar{v}_{O_2}$	= arterial less mixed venous O_2 content
$\dot{V}_{O_2}$	= whole-body O_2 consumption
Uc_{O_2}	= coefficient of O_2 utilization
$\bar{P}_{PA}$	= pulmonary arterial mean pressure
$\bar{P}_{RA}$	= right atrial mean pressure
$\bar{P}_{LA}$	= left atrial mean pressure
P_{PAD}	= pulmonary arterial diastolic pressure
PVR	= pulmonary vascular resistance
$\dot{Q}_s/\dot{Q}_t$	= ratio of shunted to total blood flow
$\dot{Q}_p$	= pulmonary blood flow
f	= respiratory rate
V_t	= tidal volume
$\dot{V}$	= minute volume
$\overline{AP}$	= mean airway pressure
AP_p	= peak airway pressure
$\bar{V}_i$	= mean inspiratory flow rate
$\dot{V}_{ip}$	= peak inspiratory flow rate
$\bar{V}_e$	= mean expiratory flow rate
$\dot{V}_{ep}$	= peak expiratory flow rate
T_i	= duration of inspiration
T_e	= duration of expiration
T_{tot}	= duration of respiratory cycle
T_i/T_e	= ratio of inspiratory to expiratory time
T_i/T_{tot}	= ratio of inspiratory time to total time
V_t/T_i	= ratio of tidal volume to duration of inspiration (same as $\bar{V}_i$)

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TABLE 1. Preoperative Catheterization Data for 15 Patients, Five with Increased $\bar{P}_{PA}$ (Group A) and Ten with Normal $\bar{P}_{PA}$ (Group B) (Means ± SEM)

	$\bar{P}_{PA}$ (torr)	P_{PAD} (torr)	$\bar{P}_{LA}$ (torr)	$\dot{Q}_p$ (l/min/m²)	PVR (u/m²)
Group A ($\bar{P}_{PA} > 25$ torr)	46 ± 10	28 ± 10	9 ± 1	11 ± 3	4 ± 5
Group B ($\bar{P}_{PA} < 25$ torr)	16 ± 1	11 ± 2	7 ± 2	16 ± 5	1 ± 0.3

for endotracheal extubation. The BABYbird® respirator was selected for smaller patients and either the Monahagn 220® or Bennett MA-1® controlled-volume ventilator was used for larger children. Tidal volume was adjusted to produce a minute ventilation sufficient to induce mild respiratory alkalosis ($Pa_{CO_2} \simeq 33$ torr). Half the patients were initially ventilated for a period of approximately one hour, studied, then restudied for a 30-min period of ventilation with 5 cm H_2O PEEP. Alternate patients were initially ventilated with 5 cm H_2O PEEP, followed by a 30-min period of ventilation without PEEP.

Respiratory gas flow was monitored at the endotracheal tube with an in-line heated Fleisch #0 pneumotachograph. Tidal volume was obtained by electrical integration of the flow signal and was found to be approximately 15 ml/kg. Any leak between endotracheal tube and trachea was readily detectable by baseline drift in the tidal volume trace. Airway pressure was measured with a gas pressure transducer through an 18-gauge needle inserted into the endotracheal tube at lip level. Simultaneous and continuous recordings were made of airflow, tidal volume and airway pressure, from which the following values were determined: tidal volume, respiratory rate, minute volume, peak inspiratory and expiratory flow rates, and mean inspiratory and expiratory flow rates. Mean and peak flow rates were determined by a method demonstrated by Nunn and detailed elsewhere.[9,10] In brief, mean airflow was calculated from a one-second change in volume determined with the slope of a line drawn on the tidal volume tracing from the beginning to end of either inspiration or expiration. Peak flow rates were calculated from a one-second change in volume determined from the slope of a line drawn through the steepest portion of the inspiratory or expiratory trace. The time of the total respiratory cycle, inspiratory flow, and expiratory flow were also determined from the tidal volume trace. Calibration of flow and volume was accomplished at the end of each recording period with precision-bore flow tubes and a 1-liter calibrated syringe. The peak airway pressure was obtained from the airway pressure tracing, and mean airway pressure represents the time-averaged mean of pressures encountered above and below ambient pressure throughout the respiratory cycle.

Each patient had an indwelling radial-artery catheter, and right and left atrial catheters for pressure monitoring. The right atrial catheter was placed in the mid-atrium to minimize overrepresentation of superior or inferior vena caval or coronary-sinus blood in samples drawn for intrapulmonary shunt determinations. A 4-French thermodilution catheter was inserted into the pulmonary artery via the right atrium and ventricle at the time of operation for measuring cardiac output.[11] Triplicate output determinations were made using 2-ml volumes of iced dextrose, 5 per cent, in water. Since results of thermodilution measurements are affected by residual intracardiac shunting, postoperative dye-dilution curves obtained from all patients, with and without mechanical support of ventilation, were analyzed to rule out the presence of intracardiac shunting.

Respiratory measurements, cardiac output determinations, and samples of arterial and right atrial blood were withdrawn for blood gas and hemoglobin determinations at the end of the appropriate period of ventilation with or without PEEP. Calculations were then made of intrapulmonary shunting, oxygen consumption using the Fick equation, and the coefficient of oxygen utilization (Uc_{O_2}).[12] The latter was determined from the formula:

$$Uc_{O_2} = Ca_{O_2} - C\bar{v}_{O_2} \div Ca_{O_2}.$$

The following morning, approximately 15 hours later, all patients were allowed to breathe spontaneously through a T-piece assembly with zero end-expiratory pressure (ZEEP) for approximately an hour and all measurements were repeated. Five centimeters H_2O CPAP were then added, and 30 min later measurements were again made. Gas flow rate through the T-piece/CPAP system was at least two and a half to three times the minute volume. Alternate patients had the sequence reversed. Mean values with standard errors are reported for all data. Paired comparisons of data obtained from each patient during IPPB and PEEP and during T-piece breathing and CPAP were made, and significant differences in mean values determined by the Student t test for paired data.

Results

No significant change in cardiac output, oxygen consumption, oxygen utilization, or intrapulmonary shunting was induced by adding either 5 cm H_2O

TABLE 2. Cardiorespiratory Effects of PEEP and CPAP in 22 Infants and Children Following Open-heart Surgery

	IPPB	PEEP, 5 cm H_2O	ZEEP	CPAP, 5 cm H_2O
BP (torr)	122/63 ± 6/3	121/52 ± 6/3	119/63 ± 3/3	119/65 ± 4/2
HR (/min)	133 ± 5	135 ± 6	118 ± 4	123 ± 4
CI (l/min/m²)	3.7 ± .3	3.5 ± .3	4.1 ± .2	4.2 ± .3
$Ca_{O_2} - c\bar{v}_{O_2}$ (vol per cent)	5.9 ± .4	5.8 ± .4	4.9 ± .5	5.0 ± .4
$\dot{V}_{O_2}$ (ml/min/m²)	202 ± 15	189 ± 12	184 ± 14	189 ± 13
Uc_{O_2}	.35 ± .02	.35 ± .02	.28 ± .02	.28 ± .02
$\bar{P}_{RA}$ (torr)	8.4 ± .7	9.6 ± .7*	5.5 ± .6	5.9 ± .7
$\bar{P}_{LA}$ (torr)	11.0 ± .8	11.5 ± .8	7.1 ± .9	8.3 ± .9*
$\dot{Q}_s/\dot{Q}_t$ (per cent)	8.7 ± 1.2	8.3 ± 1.2	15.5 ± 1.1	15.1 ± 1.1
Pa_{CO_2} (torr)	33 ± 2	33 ± 1	38 ± 2	40 ± 2
f (/min)	17 ± 1.0	17 ± 1.0	31 ± 2	25 ± 2*
V_t (ml)	197 ± 20	195 ± 12	134 ± 15	151 ± 17
$\dot{V}$ (l/min)	3.35 ± .30	3.32 ± .27	4.15 ± .30	3.78 ± .31*
$\overline{AP}$ (cm H_2O)	6.0 ± 1.0	9.8 ± 1.2*	0.4 ± .1	4.9 ± .3*
AP_p (cm H_2O)	24.7 ± 2.0	28.1 ± 2.4*	0.7 ± .2	6.1 ± .6*
$\bar{V}_t$ (l/min)	13.7 ± 1.5	13.7 ± 1.2	10.7 ± 1.2	12.8 ± 1.5
$\dot{V}_{tp}$ (l/min)	18.6 ± 1.9	21.7 ± 2.2	15.6 ± 1.9	16.3 ± 2.0
$\bar{V}_e$ (l/min)	10.4 ± 1.5	8.5 ± 0.8*	7.6 ± 0.8	7.4 ± 0.9*
$\dot{V}_{ep}$ (l/min)	21.1 ± 2.5	20.0 ± 2.6*	10.3 ± 1.3	11.0 ± 1.5*
T_i (sec)	.83 ± .01	.87 ± .01	.73 ± .26	.74 ± .22
T_e (sec)	1.40 ± .07	1.58 ± .09*	1.01 ± .38	1.20 ± .53*
T_{tot} (sec)	3.48 ± .18	3.58 ± .19	1.93 ± .78	2.50 ± .90*
T_i/T_e	.37 ± .06	.36 ± .02	.42 ± .07	.39 ± .02*
T_i/T_{tot}	.24	.24	.38	.30*
V_t/T_i	.23	.22	.18	.21*

* $P < .025$.

PEEP during controlled ventilation or 5 cm H_2O CPAP during spontaneous respiration with a T-piece (table 2). Five patients who had pulmonary hypertension responded no differently from ten patients without pulmonary hypertension to PEEP and CPAP (table 3).

Increases in mean and peak airway pressures induced by PEEP or CPAP were usually accompanied by small sustained increases in right and left atrial pressures. The duration of expiratory airflow (T_e) was significantly prolonged with PEEP during both controlled and spontaneous respiration. Neither the duration nor the flow rate of inspiration was affected by the addition of PEEP or CPAP. During spontaneous respiration, however, the presence of CPAP was accompanied by decreases in repiratory rate and the ratio of inspiratory duration to the total respiratory cycle (T_i/T_{tot}). Faster respiratory rates during T-piece breathing were associated with shorter durations of exhalation but with no change in the duration or flow profile of inhalation.

Discussion

Postoperative intrapulmonary shunting during mechanical ventilation was slight and was not affected by the addition of 5 cm H_2O PEEP. Relatively large tidal volumes sufficient to produce mild hypocarbia may have accounted for sufficient alveolar recruitment during mechanical ventilation to minimize any further recruitment with PEEP. When mechanical ventilation was stopped the following morning, and the patients were allowed to breathe spontaneously through a T-piece, intrapulmonary shunting increased from 7 to 15 per cent. Tidal volumes were smaller during spontaneous respiration an hour following withdrawal of mechanical ventilation, but the mean respiratory rate was much faster. This resulted in an overall increase in minute volume. The Pa_{CO_2} however, increased to a mean of 38 torr during T-piece breathing and to 40 torr with the addition of CPAP, and probably reflected an increase in the dead space-to-tidal volume ratio.

Because of no residual intracardiac shunting postoperatively, patients with preoperative hypertension experienced nearly a threefold decrease in pulmonary blood flow (Tables 1 and 3). Although pulmonary pressures and vascular resistance were not determined in these patients after operation, decreases in pulmonary blood flow in the presence of pulmonary vascular disease may further increase pulmonary vascular re-

sistance, presumably on the basis of a decrease in the caliber of the pulmonary arterioles when not distended by high flow, and by an overall decrease in the size of the perfused vascular bed, with increases in pulmonary vascular resistance persisting for years.[13,14] Little is known about the immediate status of the pulmonary vascular bed in relation to ventilation following correction. Increases in end-expiratory airway pressure are known preferentially to increase ventilation in dependent portions of the lung—portions that normally enjoy relative overperfusion.[15] Lungs of patients with increased pulmonary vascular resistance, however, may be characterized by comparative underperfusion of dependent portions or an overall decrease in the vascular cross-sectional area, and benefit least from increased end-expiratory pressure.[15,16] Although our patients with increased pulmonary vascular resistance had relatively larger degrees of shunting postoperatively, they did not behave differently from those without increased pulmonary vascular resistance when removed from ventilatory support (table 3).

Pulmonary stretch receptors lying within airway smooth muscle are thought to initiate vagally mediated, centrally induced changes in the duration of exhalation, respiratory rate, and depth of respiration.[17] Electromyographic studies in the dog indicate that both 4 and 8 cm H_2O CPAP will decrease electrical activity of the diaphragm, increase abdominal muscle activity, and prolong exhalation, without affecting the time of inhalation.[18] Similarly, in awake man, Josenhaus *et al.* found that increasing levels of PEEP during mechanical ventilation caused linear decreases in the effective elastance of rib cage muscles and diaphragm, while the elastance of the abdomen increased linearly with increasing levels of PEEP; tidal volume contributions attributable to abdominal musculature and diaphragm were nearly half those during spontaneous breathing.[19] Thus, increases in mean airway pressure through induced alterations in respiratory muscle tone permit a greater proportion of the tidal exchange to be affected by a more compliant cage and less by the diaphragm—abdomen component. In addition, these changes in tone will affect the ratio of inspiratory to expiratory time.[20]

In our patients, the duration of expiratory flow was significantly prolonged when PEEP was added to mechanical ventilation, and is responsible for the lower calculated mean expiratory flow rate. This prolongation of expiratory airflow or "expiratory braking" in the patient whose trachea is intubated is due in part to antagonistic contraction of inspiratory muscles

TABLE 3. Effects of PEEP, 5 cm H_2O, and CPAP, 5 cm H_2O, on $\dot{Q}_s/\dot{Q}_t$ and CI in Five Patients with (Group A) and Ten Patients without (Group B) Preoperative Pulmonary Hypertension (Means ± SEM)

	IPPB	PEEP, 5 cm H_2O	ZEEP	CPAP, 5 cm H_2O
$\dot{Q}_s/\dot{Q}_t$ (per cent)				
Group A	10 ± 3	10 ± 3	18 ± 1	16 ± 3
Group B	8 ± 1	8 ± 1	15 ± 1	15 ± 2
CI (l/min/m²)				
Group A	4.2 ± 0.7	4.1 ± 0.7	4.4 ± 0.5	4.3 ± 0.6
Group B	3.8 ± 0.3	3.6 ± 0.3	4.1 ± 0.3	4.1 ± 0.2

during exhalation, which permits, with central neuronal surveillance, an orderly dissipation of the tidal exchange.[20] Peak expiratory flow rate was not increased with PEEP as might be expected if total compliance were decreased at a higher level of tidal exchange induced by PEEP. This circumstantial evidence of "expiratory braking" occurred during controlled ventilation without the cyclic respiratory muscle activity that occurs during spontaneous respiration. If alveolar recruitment with PEEP had occurred and lung compliance increased, "expiratory braking" might be assessed as decreased and prolonged expiratory flow due to a more compliant lung. Intrapulmonary shunting, however, did not decrease significantly with PEEP and does not support the concept.

During spontaneous respiration, the addition of CPAP caused respiratory rate to decrease and tidal volume to increase in our patients. During spontaneous breathing, when central and reflex control of ventilation can determine respiratory rate, CPAP had no significant effect on "inspiratory drive" as measured by the mean inspiratory flow (V_t/T_I).[8,21] It is likely that CPAP decreased the respiratory rate and the ratio of T_I/T_{tot} because of an increase in the duration of exhalation by initiating reflexes originating in the chest wall and lung.[21] Decreases in respiratory rate with CPAP have also been reported by others.[22,23]

The lack of any salutary change in cardiopulmonary function following addition of 5 cm H_2O PEEP during postoperative mechanical ventilation or 5 cm H_2O CPAP during spontaneous breathing via T-piece in this group of patients raises questions about the appropriateness of their routine postoperative use. Whether PEEP or CPAP may alter the incidence of post-extubation respiratory complications cannot be extrapolated from the data. Similarly, the data from these patients without respiratory complications should

not be used to forestall the application of PEEP or CPAP in those situations of respiratory insufficiency for which their salutary effects are well documented. Yakitis, however, reported that intraoperative use of PEEP to forestall postoperative hypoxemia usually increased Pa_{O_2}, but the beneficial effects were rapidly dissipated on withdrawal of PEEP and were of no prophylactic value in altering the usual postoperative hypoxemia.[24] While PEEP is recommended for use following cardiac operations in children by some investigators, the evidence for any advantage other than a transient increase in Pa_{O_2} is meager.[2-5] Potential adverse effects of increased airway pressure from both IPPB and PEEP are possible, particularly in the absence of pulmonary disease, but were not encountered in the present study. Increases in Pa_{O_2} may be accompanied by decreases in cardiac output with a net decrease in oxygen delivery. This is more likely to occur when pulmonary vascular resistance is low.[15,25,26] While end-expiratory pressure can impede the encroachment of excess fluid in the lung, it may mask any pulmonary manifestations of hypervolemia which may become evident when PEEP or CPAP is discontinued: a simultaneous decrease in transmural filling pressure may also occur, compromising cardiac performance.[15,25,27] A period of spontaneous ventilation without end-expiratory pressure is usually employed prior to extubation to evaluate the efficiency of the cardiorespiratory system unaltered by PEEP or CPAP. In newborn infants receiving 2 cm H_2O CPAP, however, this evaluation period of spontaneous breathing at ambient pressure may not be well tolerated, presumably because of altered upper airway resistance, and extubation of the trachea during maintenance of CPAP has been recommended.[28] Aidinis *et al.* have demonstrated that PEEP is capable of increasing cerebral blood volume and intracranial pressure by pressure transmission through the valveless jugular cerebral venous system leading to neurologic dysfunction, and is more likely to occur in the absence of pulmonary disease.[29] Periods of systemic hypotension accompanied by PEEP-enhanced decreases in cerebral perfusion pressure may precipitate further neurologic dysfunction in patients who have intracranial disease. Similarly, CPAP has been shown to increase superior sagittal sinus pressure slightly in infants with respiratory distress syndrome whose tracheas are intubated. When CPAP is administered by the rigid-hood or plastic-bag technique with which cervical constriction is possible, post-hemorrhagic hydrocephalus is very likely to occur.[30]

Thus, while PEEP or CPAP may produce a gratifying change in the Pa_{O_2} or induce alveolar recruitment, these results are not always evident, as in this group of patients. Because postoperative PEEP and CPAP may induce changes in atrial or intracranial pressure, cardiac output, or shunting, and complicate clinical assessment, they might properly be reserved as temporary supportive techniques when respiratory function is compromised. A cost/benefit assessment of any increase in mean airway pressure can then be made by clinical observation, and by evaluation of any induced changes in blood gas values and cardiac output.

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