

Continuous Positive Airway Pressure and Pulmonary and Circulatory Function after Cardiac Surgery in Infants Less Than Three Months of Age

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Continuous positive airway pressure (CPAP) was used to support the ventilation of infants less than 3 months of age who had undergone thoracotomy for cardiovascular surgery. The functional residual capacity, which was approximately 30 per cent of predicted at zero CPAP, increased 35 per cent in cyanotic and 33 per cent in acyanotic infants with the application of 5 mm Hg pressure. Increasing airway pressure from zero to 5 mm Hg increased $P_{a_{O_2}}$ 4 per cent in cyanotic and 13 per cent in acyanotic infants. There was no change in heart rate, respiratory rate, mean arterial pressure, pH or $P_{a_{CO_2}}$ under similar circumstances, but central venous pressure increased 1.5 mm Hg in cyanotic and 0.8 mm Hg in acyanotic infants. (Key words: Anesthesia, pediatric; Surgery, pediatric; Ventilation, continuous positive-pressure; Surgery, cardiovascular.)

CONTINUOUS positive airway pressure (CPAP)** is a new method of respiratory

assistance for infants,¹ although previously the technique was applied in management of adult patients and subsequently abandoned.² In the recent past CPAP has proven beneficial in treatment of infants with idiopathic respiratory distress syndrome and following cardiac surgery.^{3,4} Ashbaugh and associates used positive end-expiratory pressure (PEEP) during mechanical ventilation of patients who had adult respiratory distress syndrome.⁵ Although CPAP is now widely used in pediatric patients, there are few data on the effects of CPAP on respiratory and circulatory function in infants less than 3 months of age who have had corrective or palliative cardiac surgery. This report summarizes our experience with 19 such infants.

Methods

From January 1970 to December 1972, 126 infants 3 months of age or less had cardiovascular operations at the University of California. All but three received CPAP postoperatively. We studied some effects of CPAP on pulmonary and circulatory function in 19 randomly selected infants. Eleven infants had cyanotic and eight, acyanotic congenital heart disease. The average age at operation was 33 ± 23 days. Two of the 19 infants died before discharge from the hospital.

POSTOPERATIVE RESPIRATORY PROTOCOL

After operation all infants were transferred to the Intensive Care Nursery with an orotracheal tube in place. Temperature, respiratory rate, heart rate, and intravascular systemic arterial blood pressure were recorded at 15-minute intervals in all 19 patients.

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** CPAP is spontaneous breathing against a positive end-expiratory pressure.

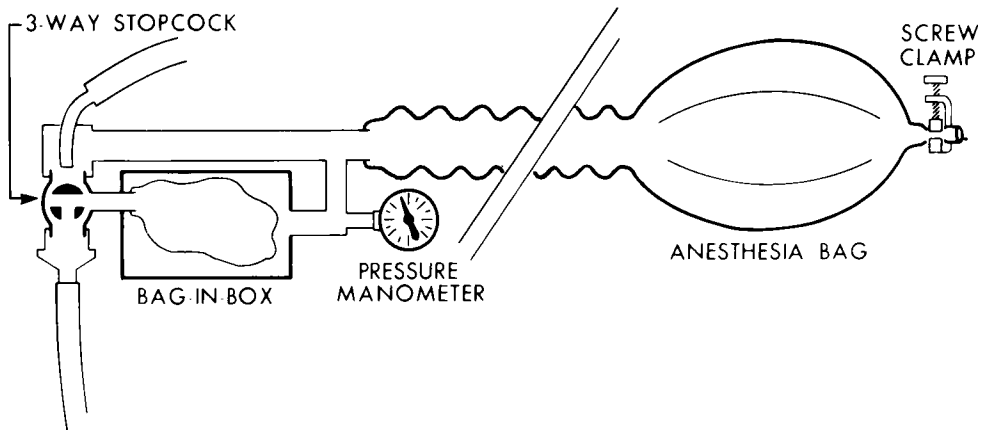


FIG. 1. System for measuring FRC. Note that the pressure around the bag-in-box is the same as that applied against the patient's airway for therapy. The specially constructed stopcock has 0.3-ml deadspace.

In seven patients central venous pressure was recorded from a catheter in the right atrium or thoracic vena cava.

The infants breathed 100 per cent oxygen at a CPAP of 5 mm Hg through the CPAP system.¹ Inspired oxygen concentrations were reduced progressively to maintain arterial oxygen tensions between 50 and 75 mm Hg in acyanotic and above 25 mm Hg in cyanotic infants. Morphine sulfate (0.1 mg/kg) was given intravenously for analgesia and sedation as needed. Hourly, chest physiotherapy was performed, isotonic saline solution (0.5 ml) was injected into the endotracheal tube, the infants' lungs hyperinflated and the airways suctioned.⁶ Chest-tube drainage losses were replaced with whole blood, plasma, or packed cells according to the hematocrit. Roentgenograms of the chests were obtained to rule out pulmonary air leaks and/or improper endotracheal tube placement. When the inspired oxygen concentration had been reduced to 40 per cent, the end-expiratory pressure was reduced 1 mm Hg every two hours so long as arterial oxygen tensions remained within the desired ranges. If ventilation and circulation were stable, the endotracheal tube was removed approximately 4 hours after airway pressure had been reduced to zero.

STUDIES

Within 6 hours after operation we measured the following variables at both zero and 5

mm Hg CPAP: arterial P_{O_2} , P_{CO_2} , and pH , arterial pressure, pulse rate, central venous pressure (seven patients) and functional residual capacity (FRC) (12 patients). The CPAP was varied in random sequence and held stable for 10 minutes before measurements were made. These studies were repeated 24 hours later in four patients, and 48 and 72 hours later in two patients. We used the system diagrammed in figure 1 to measure FRC by helium dilution. The pressure inside the bag was equal to that against which the infant was breathing (0 or 5 mm Hg). The stopcock and bag-in-box were flushed twice with 5 ml of 10 per cent helium in oxygen, following which the bag was filled with 60 ml of the same gas mixture. At the end of expiration the stopcock was turned and the infant allowed to rebreathe the helium- O_2 mixture for 30 seconds. The concentration of helium present in the bag at the end of 30 seconds was measured with a helium catharometer (Med Science) and compared with the original concentration of helium in the oxygen mixture. FRC was corrected for CO_2 , water vapor, and body temperature. Duplicate determinations of FRC that differed by more than 5 per cent were discarded. The presence of a loss of helium around the uncuffed endotracheal tube and/or nonequilibrium of the bag helium with FRC were checked during each study. The possible leak of gas from the lung

was checked by continuously monitoring CO₂ concentration in the posterior pharynx with a Beckman infrared analyzer. If the CO₂ concentration of the posterior pharynx exceeded 0.5 per cent, a concentration that would account for a maximal leak of 1 ml of gas from the system in 30 seconds, the studies were discarded. Equilibration of the oxygen-helium mixture with FRC was checked by comparing the expired helium concentrations after 15 and 30 seconds of rebreathing. If the difference between the two concentrations was less than 5 per cent, equilibration was assumed. FRC was calculated using the formula

$$\text{FRC} = \frac{V_i \times \text{He}_i}{\text{He}_f} - v_f$$

where *i* stands for initial, *f* for final, *V* for volume of gas in the bag and *He* for helium concentration. Significances of changes in these variables with application of CPAP were evaluated by the *t* test for paired data.

Results

None of the 19 infants received mechanical ventilation. Their endotracheal tubes were removed 13 to 168 hours after operation, mean 46 hours. The inspired oxygen concentration was greater than 60 per cent for

0.5 to 20 hours (mean 6.1 hours) and the oxygen concentration above atmospheric for 1 to 177 days (mean 10) postoperatively. There was no laryngeal complication. Two patients died 5 to 9 weeks postoperatively of their cardiac lesions. The remaining infants were discharged from the hospital alive.

There was no change in heart rate, arterial pressure (systolic, diastolic, or mean), central venous pressure, or respiratory rate when the airway pressure was varied from 0 to +5 mm Hg in either cyanotic or acyanotic infants (table 1).

Table 2 shows the changes in blood gases and *pH* that occurred with changing airway pressure. Arterial oxygen tension (PaO₂) increased 10 to 32 per cent (mean +13 per cent) at the higher airway pressure in acyanotic infants and changed from -9 to +25 per cent (mean +4 per cent) in cyanotic infants (fig. 2). In contrast, arterial PaCO₂ and *pH* did not change consistently in either cyanotic or acyanotic infants with application of 5 mm Hg airway pressure. After operation FRC was only 33 per cent of predicted^{7,8} when the airway pressure was zero. Both cyanotic and acyanotic infants showed a 34 per cent increase in FRC when airway pressure was increased to 5 mm Hg (*P* < 0.01) (fig. 3). Figure 4 shows the FRC's of four patients measured at 0 and 5 mm Hg during the first six hours postopera-

TABLE 1. Changes in Vital Signs with Application of CPAP (Mean ± 1 SD)

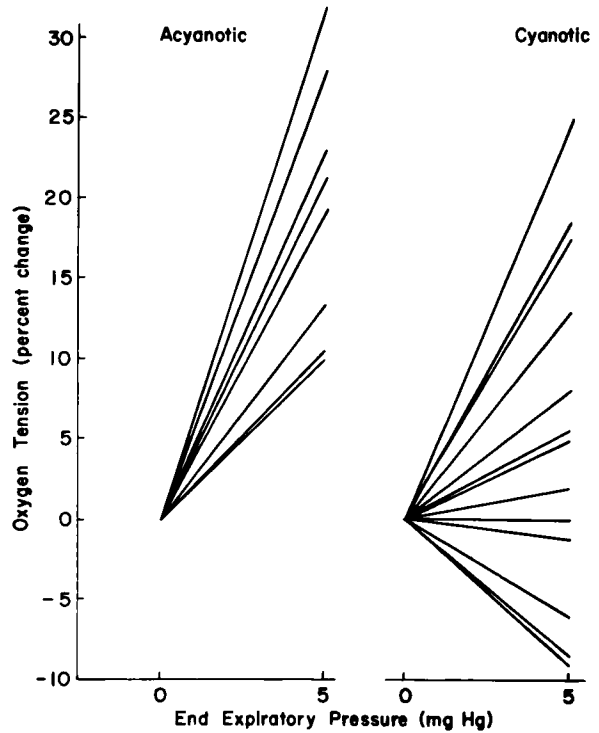
Airway pressure (mm Hg)	Heart Rate (Beats/Min)		Respiratory Rate (Breaths/Min)		Mean Arterial Pressure (mm Hg)		Central Venous Pressure (mm Hg)	
	0	5	0	5	0	5	0	5
Cyanotic	151 ± 18	149 ± 21	63 ± 18	60 ± 15	54 ± 19	57 ± 25	5 ± 1	6.5 ± 1.2
Acyanotic	151 ± 15	142 ± 8	52 ± 14	49 ± 12	68 ± 15	67 ± 18	6 ± 1	6.8 ± 1.1

TABLE 2. Changes in Blood Gases and *pH* with Application of CPAP (Mean ± 1 SD)

Airway pressure (mm Hg)	Acyanotic		Cyanotic	
	0	5	0	5
PaO ₂ (mm Hg)	72 ± 20	84 ± 23*	35.2 ± 2	36.8 ± 0.8
PaCO ₂ (mm Hg)	46 ± 7.7	42 ± 6.8	43.9 ± 11.2	43.6 ± 10.6
<i>pH</i>	7.36 ± 0.05	7.36 ± 0.05	7.35 ± 0.05	7.37 ± 0.05

* Significant at 0.05 level.

FIG. 2. Effects of end-expiratory pressure on percentage changes in P_{aO_2} in cyanotic and acyanotic infants.



tively and 24 hours later. Figure 5 shows the changes in FRC on four successive days in one patient who had cyanotic heart disease.

Discussion

Following cardiac surgery infants frequently require ventilatory support to maintain adequate gas exchange.^{3,9,10} Fifty-three per cent of patients less than 6 months of age reported by Downes *et al.* required ventilatory support postoperatively; 57 per cent of these were less than 4.6 months of age. Forty-nine per cent of those with postoperative respiratory failure died. Therefore, we considered that all our infants less than 3 months of age who had undergone cardiovascular surgery were at risk of respiratory failure.

Our data show that FRC is low immediately after operation and increases over the next several days. The low FRC is almost certainly due to atelectasis. Several structural differences between the lung and chest region of the newborn and that of the older child or adult should make the former more prone to develop atelectasis following an insult to

the chest such as thoracotomy. The terminal air units are of small diameter in the newborn and in small infants.¹¹ Therefore, a greater transpulmonary pressure is necessary to pre-

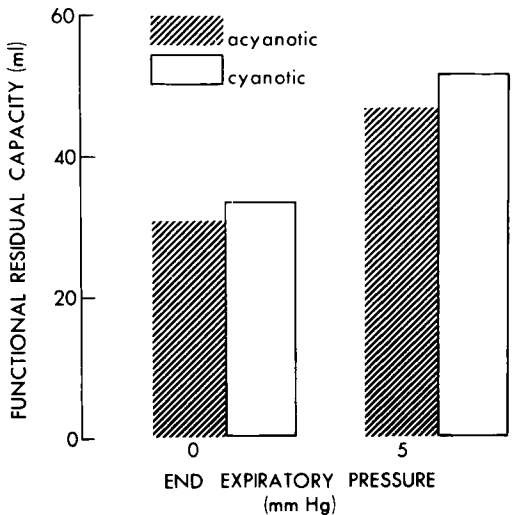


FIG. 3. Effects of CPPB on functional residual capacity. Mean values for acyanotic and cyanotic infants at zero and +5 mm Hg end-tidal pressure.

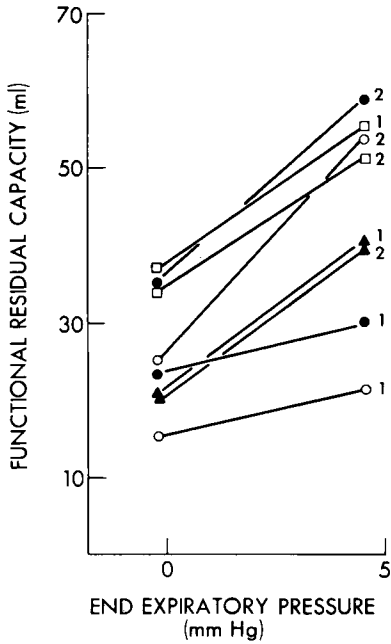


FIG. 4. Effects of time on functional residual capacity. Values for four patients immediately after¹ and 24 hours after operation.²

vent alveolar collapse with expiration. The chest wall, which contributes to¹¹ transpulmonary pressure, is more compliant, and pressure rises nearly to zero at end-expiration in infants, as opposed to -6 cm H_2O in adults.⁸ Furthermore, infants probably have airway closure within the tidal volume range.¹² Once atelectasis does develop it is more likely to persist due to inward collapse of the soft chest wall as the diaphragm descends. All of these factors are present, or are potentially present, in infants less than 3 months of age.

The low FRC of our patients is previously undescribed and approximates those found in infants with the idiopathic respiratory distress syndrome¹³ and in infants undergoing correction of diaphragmatic hernia or tracheoesophageal fistula (unpublished data). The application of 5 mm Hg end-expiratory pressure increased FRC towards normal in both cyanotic and acyanotic patients. However, FRC remained markedly decreased despite pressure. Figure 5 shows an increase in FRC in one patient for four days postoperatively. The progressive increase is similar to that seen in adults following abdominal surgery.¹⁴

There are two potential errors in our FRC measurements that might have led to inaccurate results: 1) A leak of helium might occur around an uncuffed endotracheal tube and give falsely large values for FRC. Since the concentration of CO_2 in the pharynx was less than 0.5 per cent, no more than 1 ml of gas or 0.1 ml of helium would have leaked out, an insignificant amount. 2) Failure of gas in the bag and lungs to reach equilibrium during the 30 seconds of rebreathing would lead to a falsely smaller value for FRC. However, our measurements of helium concentration after 15 or 30 seconds of rebreathing agreed closely; therefore, equilibration had occurred by 15 seconds.

The increase in Pa_{O_2} in acyanotic patients was almost certainly due to an improvement in ventilation-perfusion ratios, since FI_{O_2} 's were the same before and after the application of pressure (there is a remote possibility that

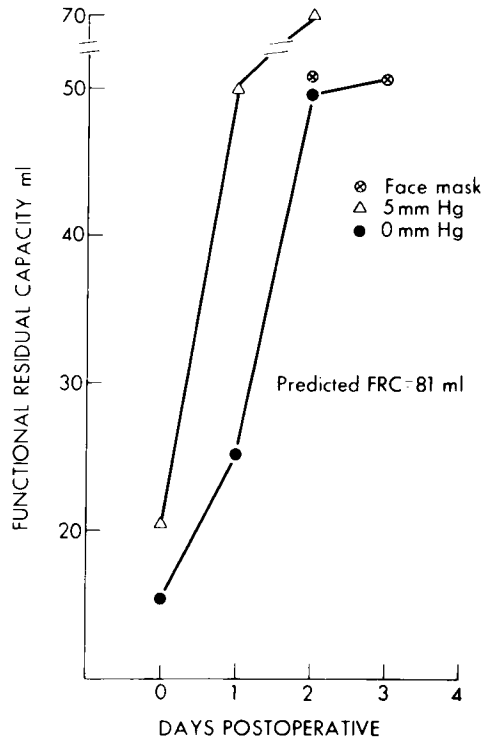


FIG. 5. Effects of time on functional residual capacity. Values for one patient on four consecutive days. Day zero is the day of operation. Note that FRC was measured with a nasal mask on the second and third postoperative days. On each of the first two postoperative days FRC was smaller at 0 than at 5 mm Hg end-expiratory pressure.

cardiac output increased or oxygen consumption decreased). It may be that the increased FRC took the closing volume out of the tidal-volume range, thus making alveoli available for gas exchange for longer periods during each respiratory cycle.¹⁴

Arterial oxygen tensions increased in eight of 13 infants with cyanotic heart disease, decreased slightly in three, and remained unchanged in two. The decreases in Pa_{O_2} in these three patients may have resulted from reduced pulmonary blood flow and/or an increase in shunt flow. The increases in Pa_{O_2} in the eight infants probably reflected improved ventilation-to-perfusion relationships.

We found no significant change in Pa_{CO_2} , pH, base excess, heart rate, respiratory rate or arterial pressures with the application of CPAP. Central venous pressure increased an average of only 1.5 mm Hg in cyanotic and 0.8 mm Hg in acyanotic infants, which suggests that the pressure applied did not interfere with venous return. A second possibility is that extrapulmonary right-to-left shunting of blood occurred. This is unlikely because Pa_{O_2} did not change. There was no progressive increase in metabolic acidemia, indicating no significant interference with delivery of oxygen to tissues.

CPAP effectively supports ventilation of infants after cardiac surgery by increasing FRC and improving ventilation-to-perfusion relationships. The level of CPAP used here does not appear to have a significantly effect venous return, cardiac output, heart rate, respiratory rate or intravascular pressures.

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