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CARDIORESPIRATORY EFFECTS OF INCREASED AIRWAY PRESSURE DURING CONTROLLED AND SPONTANEOUS BREATHING AFTER CARDIAC SURGERY

H. ASKITOPOULOU, M. K. SYKES AND C. YOUNG

SUMMARY

The cardiorespiratory effects of intermittent positive pressure ventilation with zero end-expiratory pressure (IPPV), continuous positive airway pressure breathing (CPAP) and spontaneous breathing (SR) were studied in 11 patients 3–20 h after open-heart surgery. The transition from IPPV to CPAP resulted in a significant reduction in tidal volume and significant increases in respiratory frequency, Pa_{CO_2}, oxygen transport and mean arterial pressure, but there were no significant changes in cardiac output or Pa_{O_2}. There were no significant differences in any of the measurements between CPAP and SR.

The technique of continuous positive airway pressure breathing (CPAP) was introduced originally by Poulton (Poulton, 1936; Barach, Martin and Eckman, 1938; Barach, Bickerman and Petty, 1975). However, the method did not gain widespread acceptance and was replaced eventually by the use of intermittent positive pressure ventilation. Renewed interest in the use of CPAP was aroused by Gregory and colleagues (1971), who reported a significant reduction in mortality when CPAP was used to treat the idiopathic respiratory distress syndrome of the neonate. Since then, CPAP has been used for the treatment of patients with arterial hypoxaemia secondary to cardiopulmonary disease. Thus, the technique has been advocated for the management of infants following cardiovascular surgery (Hatch et al., 1973; Stewart et al., 1973; Crew et al., 1974), especially for those with pulmonary vascular engorgement, low ventilation/perfusion ratios and a low functional residual capacity with airway closure during tidal breathing (Gregory et al., 1975). CPAP has been used also in the treatment of adults. It has been advocated in the treatment of patients with acute respiratory failure refractory to conventional therapy with IPPV, PEEP and high inspired oxygen concentrations (Civetta, Brons and Gabel, 1972; Pontoppidan, Geffin and Lowenstein, 1972; Garg and Hill, 1975; Glasser, Civetta and Flor, 1975) and also for the process of weaning such patients from a ventilator (Feeley and Hedley-White, 1975; Feeley et al., 1975). However, although these and other studies have detailed the effects of CPAP on intrapulmonary shunt and lung volumes (Craig and McCarthy, 1972; Lemelin et al., 1972; Abboud et al., 1975) there have been few reports of its effects on haemodynamic performance. Accordingly we have studied the cardiorespiratory effects of CPAP in a group of patients who were being weaned from a mechanical ventilator after open-heart surgery.

PATIENTS AND METHODS

Eleven adult patients (mean age 46 yr) who had undergone elective open-heart surgery for valve replacement or coronary artery bypass graft were studied in the supine semi-recumbent position. The patients had been anaesthetized with thiopentone, nitrous oxide and oxygen, supplemented by analgesics and neuromuscular blocking drugs; i.v. papaveretum 2.5–5 mg had been used for sedation and analgesia after operation. All the patients had received routine postoperative care with meticulous fluid and blood volume replacement. However, three patients were receiving an i.v. infusion of salbutamol and the fourth was receiving isoprenaline. In all the patients the lungs had been ventilated mechanically with zero end-expiratory pressure from the end of the operation to the time of the study, the duration of ventilation varying from 3 to 20 (mean 15) h.

The patients received an inspired oxygen concentration of 39–40% from an oxygen–air mixer during the three phases of the study. At least 30 min

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before commencing the measurements the tidal volume was adjusted to produce Pa_{CO_2} 4.5–5.3 kPa at a frequency of 17–18 b.p.m. A set of cardiorespiratory measurements was made (1) during controlled ventilation with zero end-expiratory pressure, (2) 30–50 min after the resumption of spontaneous breathing with 0.98 kPa (10 cm H_2O) end-expiratory pressure (CPAP), (3) 30–50 min after the initiation of ambient pressure spontaneous breathing (SR).

Breathing systems

Both Cape and Engström (ER312) ventilators, fitted with a pressure-operated collect valve at the patient Y-piece (Sykes, 1969), were used for measurements during controlled ventilation.

The circuit used for CPAP breathing is shown in figure 1. The pressurized gas passed into a weighted reservoir bellows and thence through a non-rebreathing valve* to the patient. The expired gas passed to a large-bore underwater blow-off valve adjusted to provide an end-expiratory pressure of 0.98 kPa (10 cm H_2O).

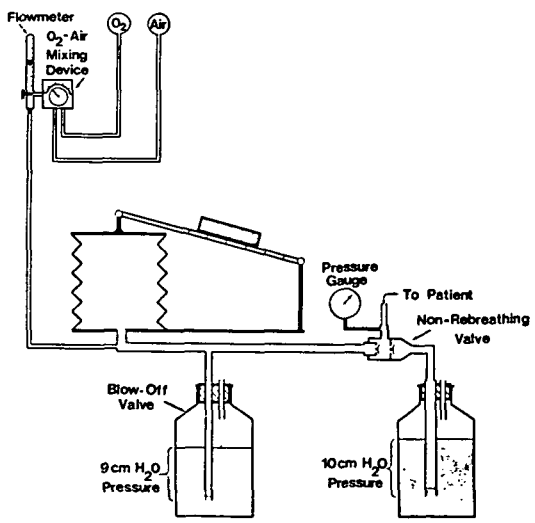

FIG. 1. CPAP circuit using weighted bellows.

The fresh gas flow was delivered at a rate which was sufficient to maintain an adequate volume in the reservoir bellows, but if these became overdistended the excess gas could escape through the second underwater blow-off valve on the inspiratory side. By keeping the blow-off pressure on the inspiratory side slightly less than that on the expiratory side, dilution of expired gas with fresh gas was avoided. The airway pressure was monitored throughout the study.

The conventional CPAP breathing system using a T-piece with a 5-litre reservoir bag in the inspiratory line requires a flow of 25–30 litre min^{-1} to minimize rebreathing in the adult patient, but even at this flow rate there is often a marked reduction in airway pressure during inspiration. The modified system utilizing the weighted bellows and non-rebreathing valve minimized the difference in pressure between inspiration and expiration and permitted fresh gas flow rates equal to the minute volume (fig. 2).

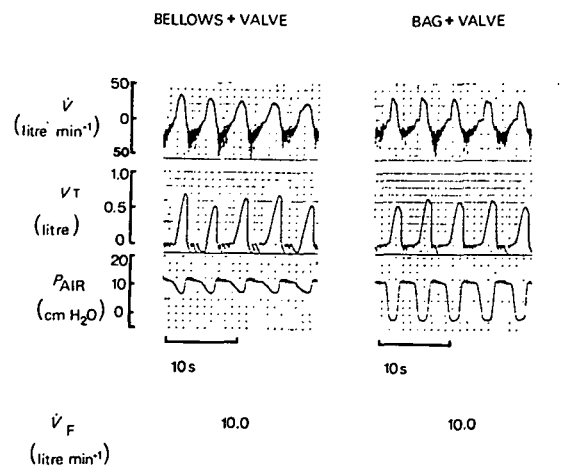

FIG. 2. Comparison of flow ($\dot{V}$), tidal volume (V_T) and airway pressure (P_{AIR}) recorded at the mouth at a fresh flow rate ($\dot{V}_F$) of 10 litre min^{-1} using the bellows and the bag CPAP systems.

During breathing at ambient pressure (fig. 3) the expiratory blow-off valve was removed, the weighted bellows were replaced by a reservoir bag and a short length of Paul's drainage tube was inserted into the circuit in place of the inspiratory underwater blow-off valve so that the pressure in the reservoir bag was always held at less than the opening pressure of the non-rebreathing valve. This ensured that the expired gas collected in the Douglas bag was not contaminated by inspired gas.

Measurements

Before commencing each set of measurements the inspired oxygen and the end-tidal carbon dioxide concentrations were monitored continuously to ensure that conditions were steady. During this period

*Inspiratory and expiratory flow resistance 1.2 and 0.7 cm H_2O respectively at a flow of 30 litre min^{-1}. Valve deadspace = 20 ml.

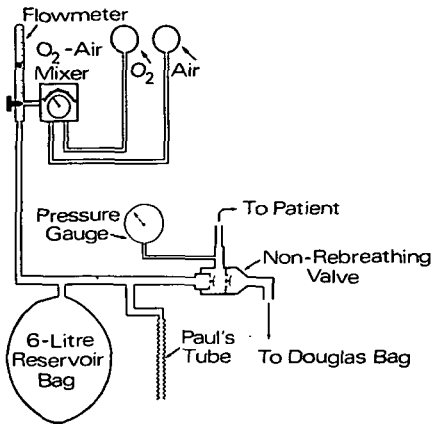

FIG. 3. The system for spontaneous breathing at ambient pressure. The Paul's drainage tube acts as a low resistance non-return valve and prevents overflow through the inspiratory and expiratory flaps of the non-rebreathing valve.

the Douglas bag was flushed out with expired gas. Arterial, pulmonary artery and left atrial pressures were recorded from the monitoring lines inserted at operation and the expired gas was collected into a Douglas bag over a 5-min period. The inspired and mixed-expired oxygen concentrations were measured using a paramagnetic oxygen analyser (Servomex OA101 MK11) and the end-tidal and mixed-expired carbon dioxide concentrations with an infra-red analyser (Hartmann–Braun URAS 4).

At the beginning of each gas collection, the cardiac output was determined in duplicate by the dye-dilution method using a Gilford system, the output being calculated from the dye curves by a computer program developed by Simons and White (1976). Five-millilitre arterial and pulmonary artery blood samples were taken slowly during the second half of each gas collection into heparinized syringes. In two patients in whom there was no indwelling pulmonary artery catheter a right atrial sample was taken. The blood samples were analysed for Po_2, Pco_2, and pH on two separate electrode systems (IL 313 and Radiometer ABL 1). The arterial and venous oxygen contents were measured directly by the Lex-O_2-Con fuel cell analyser (Selman, White and Tait, 1975) and the haemoglobin concentration was determined by the cyanmethaemoglobin method. The IL electrodes and the gas analysers were calibrated with gases which had been analysed previously on a Haldane apparatus and both sets of electrodes were checked daily with tonometered blood samples. A blood–gas factor (ranging from 1.00 to 1.03) was derived for each set of measurements and applied to Po_2 of blood. Corrections for body temperature were applied to the electrode readings (Kelman and Nunn, 1966) and gas volumes were corrected to BTPS.

Calculations using standard respiratory formulae were made on an Elliot 4100 computer using the program described by Adams (1970). The venous admixture effect was calculated from a modification of the standard shunt equation:

$$\frac{\dot{Q}_s}{\dot{Q}_t} = \frac{Cc'_{O_2} - Ca_{O_2}}{(Cc'_{O_2} - Ca_{O_2}) + (Ca_{O_2} - C\bar{v}_{O_2})}$$

where:

$Cc'_{O_2} - Ca_{O_2}$ = end-pulmonary capillary to arterial oxygen content difference calculated from alveolar Po_2 derived from the alveolar air equation and arterial Po_2; $Ca_{O_2} - C\bar{v}_{O_2}$ = arteriovenous oxygen content difference derived from the oxygen content measurements.

The use of this formula minimized errors from shifts of the oxyhaemoglobin dissociation curve. Arterial and pulmonary capillary oxygen saturations were derived from the dissociation curve described by Severinghaus (1966). The combining factor for oxygen with haemoglobin was taken as 1.39 ml g^{-1} and the solubility factor for dissolved oxygen as 0.01 mmol kPa^{-1}. Oxygen consumption (mmol min^{-1}) and oxygen transport (mmol min^{-1}) were derived from the cardiac output and the measured values of oxygen content:

$$\text{oxygen consumption } (\dot{V}o_2) = \dot{Q} \times (Ca_{O_2} - Cv_{O_2})$$

$$\text{oxygen transport} = \dot{Q} \times Ca_{O_2}$$

The statistical evaluations were performed with a two-way analysis of variance.

RESULTS

Ventilation and oxygen transfer (table I)

There was a significant increase in Pa_{CO_2} on discontinuing IPPV. In five of the patients there was a possibility of contamination of expired gas by fresh gas and the studies were discarded. In the remaining six patients the transition from IPPV to CPAP was associated with a significant reduction in tidal volume (V_T). Respiratory frequency (f) increased so that minute volume ($\dot{V}_E$) was changed little. There were no further changes when the positive end-expiratory pressure was removed although Pa_{CO_2} remained significantly greater than during IPPV. Inspired oxygen

TABLE I. *Ventilatory changes and oxygen transfer (mean values ± SD). (For abbreviations see text)*

	$\dot{V}_E$ (litre min^{-1}) (BTPS)	V_E (ml) (BTPS)	f (b.p.m.)	Pa_{CO_2} (kPa)	$P_{A_{O_2}}$ (kPa)	Pa_{O_2} (kPa)	$(P_{A_{O_2}} - Pa_{O_2})$ (kPa)	Pv_{O_2} (kPa)	$(Ca_{O_2} - C\bar{v}_{O_2})$ (mmol litre^{-1})	$\dot{Q}_s/\dot{Q}_t$ (%)
IPPV	7.561	399	17	4.91	31.55	17.29	14.25	5.93	2.37	10.0
	±0.753	±30	±1	±0.44	±1.72	±4.22	±4.50	±0.72	±0.49	±5.5
CPAP	7.039	344	20	5.97	30.68	17.34	13.33	6.45	2.28	9.8
	±2.141	±44	±5	±0.63	±1.48	±3.27	±3.16	±0.51	±0.45	±3.1
SR	7.456	375	20	5.93	30.38	16.99	13.35	6.08	2.32	9.5
	±1.269	±77	±5	±0.51	±1.86	±3.46	±2.81	±0.52	±0.45	±3.5
n	6	6	11	11	11	11	11	11	11	11
P										
IPPV *v.* CPAP	n.s.	<0.01	n.s.	<0.001	<0.028	n.s.	n.s.	n.s.	n.s.	n.s.
CPAP *v.* SR	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
IPPV *v.* SR	n.s.	n.s.	n.s.	<0.001	<0.023	n.s.	n.s.	n.s.	n.s.	n.s.

TABLE II. *Circulatory changes (mean values ± SD). (For abbreviations see text)*

	$P_{\overline{ART}}$ (mm Hg)	$P_{\overline{PA}}$ (mm Hg)	$P_{\overline{LA}}$ (mm Hg)	Heart rate (beat min^{-1})	Cardiac index (litre min^{-1} m^{-2}	$\dot{V}_{O_2}$ (mmol min^{-1})	Oxygen transport (mmol min^{-1})	Hb (g dl^{-1})
IPPV	84.2	22.7	11.3	94	2.30	9.51	26.43	12.8
	±13.5	±6.5	±5.9	±18	±0.67	±3.20	±8.70	±1.4
CPAP	94.6	27.8	14.9	101	2.66	10.58	31.22	12.9
	±13.9	±9.2	±7.1	±16	±0.87	±3.93	±11.79	±1.4
SR	91.8	25.4	14.6	96	2.45	10.00	28.89	13.0
	±17.9	±10.1	±8.8	±18	±0.77	±3.48	±11.34	±1.3
n	11	5	3	10	11	11	11	11
P								
IPPV *v.* CPAP	<0.007	n.s.	—	n.s.	n.s	n.s.	<0.014	n.s.
CPAP *v.* SR	n.s.	n.s.	—	n.s.	n.s.	n.s.	n.s.	n.s.
IPPV *v.* SR	<0.075	n.s.	—	n.s.	n.s.	n.s.	n.s.	n.s.

tension ($P_{I_{O_2}}$) was maintained close to 37.5 kPa throughout the study. $P_{A_{O_2}}$ was significantly less during CPAP and SR than during IPPV because of the increase in Pa_{CO_2}. However, there were no significant changes in Pa_{O_2} ($P_{A_{O_2}} - Pa_{O_2}$), mixed venous oxygen tension ($P\bar{v}_{O_2}$), arterio–venous oxygen content difference ($Ca_{O_2} - C\bar{v}_{O_2}$) or the percentage shunt ($\dot{Q}_s/\dot{Q}_t$).

Circulatory changes (table II)

Mean arterial pressure ($P_{\overline{ART}}$) was significantly greater during CPAP and SR than during IPPV. Mean pulmonary artery pressure ($P_{\overline{PA}}$) and mean left atrial pressure ($P_{\overline{LA}}$) were consistently greater during CPAP and SR than during IPPV, but the differences were not significant because relatively few measurements were made. Cardiac index was slightly greater with CPAP and SR than IPPV but the differences were not significant. There was, however, a significant increase in oxygen transport on changing from IPPV to CPAP.

A seperate analysis of the five patients with mitral valve disease showed a pattern similar to the main study although $\dot{Q}_s/\dot{Q}_t$ was slightly smaller with CPAP and SR. However, none of the differences reached statistical significance.

DISCUSSION

Although the majority of patients undergoing open-heart surgery are subjected routinely to mechanical ventilation for periods up to 24 h after operation, there are no controlled studies which justify the practice. *A priori* it would seem reasonable to maintain normal blood–gas values and minimize oxygen consumption whilst acute cardiovascular changes are taking place; the presence of an endotracheal tube undoubtedly decreases the hazards of re-operation for bleeding. However, against these advantages must be set the

potential technical complications associated with mechanical ventilation and the increased monitoring work-load.

Two factors causing a deterioration in lung function after operation are reductions in tidal volume and in functional residual capacity (Hedley-Whyte et al., 1965; McClenahan, Young and Sykes, 1965; Geha, Sessler and Kirklin, 1966; Eltringham et al., 1968; Alexander et al., 1973). Although IPPV maintains normal tidal volumes, a reduction in functional residual capacity (FRC) with a possible increase in airway closure can be overcome only by the application of positive end-expiratory pressure.

It has been suggested that, in patients with normal neuromuscular function, the reduction in FRC can be counteracted best by the use of CPAP rather than by adding a positive end-expiratory pressure (PEEP) to IPPV. It has been postulated that the use of CPAP will bring the tidal breathing range to the steep part of the pressure–volume curve, so that tidal exchange can be accomplished with minimal respiratory work, whilst mean intrapleural pressure will be maintained at minimal values. Theoretically, therefore, for any given FRC cardiac output should be greater with CPAP than with IPPV and PEEP. In the present studies there was a significant increase in mean arterial pressure and in oxygen transport when IPPV was discontinued. However, there were no significant changes in cardiac output or in any of the other cardiovascular measurements, either in the group as a whole or in the five patients with mitral valve disease. Previous studies have shown that the depression of cardiac output resulting from an increase in intrapleural pressure is minimized when cardiac filling pressure is great (Sykes et al., 1970; Qvist et al., 1975). Furthermore, the transmission of airway pressure to the pleural space is reduced when lung compliance is reduced (Price et al., 1951).

Other studies have shown that there is little reduction in cardiac output when PEEP at values up to 0.98 kPa (10 cm H_2O) is applied during IPPV after open-heart surgery and that the cardiovascular effects are least in patients with a high pulmonary vascular resistance (Seed, Sykes and Finlay, 1970; Trichet et al., 1975). The present studies confirm that both IPPV and 0.98 kPa (10 cm H_2O) CPAP have remarkably little effect on cardiac output, arterio–venous oxygen differences or oxygen transport, when the filling pressure is maintained at an optimal value.

Feeley and colleagues (1975), in a prospective randomized trial, found that weaning patients with various types of acute respiratory failure to 0.49 kPa CPAP resulted in a significantly smaller increase in alveolar–arterial Po_2 difference than did spontaneous breathing at ambient pressure. The present studies suggest that there is little to be gained by weaning open-heart surgery patients with reasonably normal respiratory function from IPPV to CPAP in the period soon after operation. Although these conclusions may not be applicable to the open-heart surgery patient with impaired lung function, the results do confirm that both IPPV and CPAP produce minimal cardiovascular disturbances in the patient with an adequate blood volume.

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EFFETS CARDIORESPIRATOIRES DE L'AUGMENTATION DE LA PRESSION DES PASSAGES D'AIR PENDANT LA RESPIRATION CONTROLEE ET SPONTANEE APRES CHIRURGIE DU CŒUR

RESUME

Les effets cardiorespiratoires de la ventilation au moyen de respirateurs à pression positive intermittente (IPPV) avec une pression expiratoire finale zéro de la respiration sous pression positive continue des passages d'air (CPAP) et de la respiration spontanée (SR) ont été étudiés sur 11 patients entre 3 et 20 h après chirurgie à cœur ouvert. La transition de l'IPPV à la CPAP a entraîné une réduction importante du volume courant et une augmentation significative de la fréquence respiratoire, du Pa_{CO_2}, du transport d'oxygène et de la pression artérielle moyenne, mais il n'y a eu aucune variation importante dans le débit cardiaque ou le Pa_{O_2}. Il n'y a eu aucune différence significative dans les diverses measures entre CPAP et SR.

HERZ UND ATMUNG BETREFFENDE WIRKUNGEN VOM VERSTÄRKTEN DRUCK DER LUFTWEGE, WÄHREND KONTROLLIERTER UND SPONTANER ATMUNG NACH HERZCHIRURGIE

ZUSAMMENFASSUNG

Die Herz und Atmung betreffenden Wirkungen von aussetzender Überdruckbelüftung mit Null-Endausatmungsdruck (AÜDB), andauernder Luftwegüberdruckatmung (ALÜA) und spontaner Atmung (SA) wurden in 11 Patienten 3–20 Stunden nach offener Herzchirurgie untersucht. Der Übergang von AÜDB zu ALÜA verursachte ein bedeutende Verringerung des tidalen Volumens und eine bedeutende Erhöhung der Atmungsfrequenz, Pa_{CO_2}, des Sauerstofftransports und des durchschnittlichen Arteriendrucks, aber es gab keine signifikanten Veränderungen in der kardialen Leistung oder Pa_{O_2} und kam zu keinen bedeutenden Unterschieden im Resultat zwischen ALÜA und SA.

EFECTOS CARDIORESPIRATORIOS CAUSADOS POR UN AUMENTO DE PRESION EN LAS VIAS RESPIRATORIAS DURANTE RESPIRACION CONTROLADA Y ESPONTANEA DESPUES DE CIRUGIA CARDIACA

SUMARIO

Se estudiaron los efectos cardiorespiratorios de ventilación por presión positiva intermitente (IPPV) con presión cero al final de la expiración, respiración con presión continua positiva en las vías respiratoria (CPAP) y respiración espontánea (SR) en 11 pacientes a 3–20 h después de cirugía de corazón abierto. La transición de IPPV a CPAP dio como resultado una significativa reducción en el volúmen de marea y aumentos significativos en la frecuencia de la respiración, Pa_{CO_2}, transporte de oxígeno y presión arterial media, pero no se produjeron cambios significativos en el volúmen-minuto cardíaco ni Pa_{O_2}. No se produjeron diferencias significativas en ninguna de las mediciones tomadas entre CPAP y SR.