Cardiopulmonary interactions in healthy children and children after simple cardiac surgery: the effects of positive and negative pressure ventilation

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Abstract

***Objective*—To investigate the effects of cuirass negative pressure ventilation on the cardiac output of a group of anaesthetised children after occlusion of an asymptomatic persistent arterial duct, and a group of paediatric patients in the early postoperative period following cardiopulmonary bypass.**

***Design*—Prospective study.**

***Setting*—The paediatric intensive care unit and catheter laboratory of a tertiary care centre.**

***Patients*—16 mechanically ventilated children were studied: seven had undergone surgery for congenital heart disease, and nine cardiac catheterisation for transcatheter occlusion of an isolated asymptomatic persistent arterial duct.**

***Interventions*—Cardiac output was measured using the direct Fick method during intermittent positive pressure ventilation and again after a short period of negative pressure ventilation. In five of the postoperative patients a third measurement was made following reinstitution of positive pressure ventilation.**

***Results*—Negative pressure ventilation was delivered without complication, with no significant change in systemic arterial oxygen and carbon dioxide tension. The mixed venous saturation increased from 74% to 75.8% in the healthy children, and from 58.9% to 62.3% in the postoperative group. Negative pressure ventilation increased the cardiac index from 4.0 to 4.5 l/min/m^2 in the healthy children, and from 2.8 to 3.5 l/min/m^2 in the surgical group. The increase was significantly higher in the postoperative patients (28.1%) than the healthy children (10.8%).**

***Conclusions*—While offering similar ventilatory efficiency to positive pressure ventilation, cuirass negative pressure ventilation led to a modest improvement in the cardiac output of healthy children, and to a greater increase in postoperative patients. There are important cardiopulmonary interactions in normal children and in children after cardiopulmonary bypass, and by having beneficial effects on these interactions, negative pressure ventilation has haemodynamic advantages over conventional positive pressure ventilation.**

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The introduction of positive pressure ventilation has made a dramatic impact on the outcome of intensive care in both adults and children. Although historically predated by negative pressure ventilation, its obvious practical advantages and undisputed beneficial effects on gas exchange allowed it quickly to become the method of choice in most patients requiring mechanical respiratory support.

The detrimental effects of positive pressure ventilation on the cardiovascular system are well understood, and an appreciation of these problems is essential for optimal intensive care management.[1–3] The haemodynamic effects of different negative pressure ventilation devices have been compared with those of positive pressure ventilation and reported in many animal models,[4–8] and in some paediatric patient groups.[9] Although there is little doubt that negative pressure ventilation, while providing similar ventilatory efficiency, does not cause any more haemodynamic compromise than positive pressure ventilation,[4] the question of whether it has any potential *advantages* depends on several factors related to the device used and to the characteristics of the patient. Whole body negative pressure ventilators have a rather different effect on the systemic venous return than "cuirass" devices which enclose only the chest and upper abdomen. Similarly, it is not surprising that the cardiovascular effects of ventilation using negative inspiratory pressures may differ from those of continuous negative extrathoracic pressure with the patient receiving intermittent positive pressure breaths.

Dynamic and structural properties of the heart and lungs and their close anatomical and functional relation play an important role in determining the haemodynamic influences of negative pressure ventilation. Indeed, when considering the haemodynamic effects of any mode of ventilation it is essential to consider the heart and lungs as an integral cardiopulmonary unit rather than as two separate systems.

There are clinically obvious interactions between the heart and the lungs in selected postoperative patients with congenital heart disease; indeed we have previously reported a marked haemodynamic improvement when cuirass negative pressure ventilation replaced intermittent positive pressure ventilation in the

Table 1 Patient details, showing haemodynamics before occlusion of patent arterial duct for group 1 patients, and surgical details for group 2 patients

Group 1						*Group 2*				
Patient	*Weight (kg)*	*Age (years)*	*Diagnosis*	*Qp:Qs (pre-occl)*	*PA pressure (pre) S/D/M*	*Patient*	*Weight (kg)*	*Age (years)*	*Diagnosis*	*Operation*
1	12.6	3.0	PDA	2.6:1	23//11/18	10	7.0	0.8	TGA	Mustard
2	12.5	2.5	PDA	–	25/11/17	11	49.0	13.4	Constrictive pericarditis	Pericardectomy, TV repair
3	55.5	10.8	PDA	2.5:1	24/10/17	12	5.8	0.6	Partial AVSD, cleft MV	AVSD closure + MV repair
4	9.8	1.4	PDA	2:1	25/7/22	13	7.0	1.3	VSD	Closure VSD
5	10.1	0.5	PDA	1.6:1	25/8/17	14	12.6	4.7	Congenital MV disease	MV repair
6	53.2	14.0	PDA	–	15/4/8	15	14.1	3.7	VSD, mild PS	Closure VSD, pulmonary valvotomy
7	12.2	1.7	PDA	2:1	30/12/18	16	8.2	1.3	VSD	Closure VSD
8	31.6	5.6	PDA	2.1:1	14/5/9					
9	23.8	9.0	PDA	1.5:1	24/8/15					

–, data not available; AVSD, atrioventricular septal defect; MV, mitral valve; PA, pulmonary artery; PS, pulmonary stenosis; Qp:Qs, pulmonary to systemic blood flow ratio; TGA, transposition of the great arteries; TV, tricuspid valve; VSD, ventricular septal defect.

early postoperative period following tetralogy of Fallot repair or total cavopulmonary connection.[10] In this study we have therefore compared and contrasted the effects of cuirass negative pressure ventilation with conventional intermittent positive pressure ventilation on the cardiac output of a different group of children in the early postoperative period following cardiac surgery, and a group of anaesthetised children with structurally normal hearts.

Methods

PATIENTS

The effects of negative pressure ventilation on the cardiac output were studied on 16 mechanically ventilated children, of whom nine had a structurally normal heart (group 1), and seven had undergone surgery for congenital heart disease (group 2). At the time of study, no child had any evidence of an intracardiac shunt, and all children had radiologically clear lung fields on a routine chest *x* ray.

Group 1

Nine children (four boys, five girls, median age three years) were studied following successful transcatheter occlusion of an asymptomatic isolated persistent arterial duct under general anaesthesia. Patient details are given in table 1. No patient in this group had ever been oxygen dependent, had previously undergone heart or lung surgery, or was receiving any cardiac drugs or bronchodilator treatment at the time of study. Children were excluded from the study if a routine postocclusion angiogram showed evidence of residual ductal flow.

It is the policy of our unit to perform cardiac catheterisation under general anaesthesia. All children were orally premedicated with 1.5 mg/kg trimeprazine, 30 μg/kg atropine, and 0.5 mg/kg morphine (Triatromorph, Royal Brompton Hospital, London, UK) one to two hours before the procedure. Anaesthesia was induced with an inhalational agent (1–2% isoflurane in 100% oxygen; two patients) or with an intravenous bolus of thiopentone (Intraval Sodium, Rhône-Poulenc Rorer, Eastbourne, UK; 4–7 mg/kg; seven patients). All patients then received an intravenous bolus of atracurium (400–650 μg/kg; Tracrium, Wellcome, Manchester, UK). Anaesthesia was subsequently maintained with an intravenous infusion of midazolam (100–150 μg/kg/h; Hypnovel, Roche, Hertfordshire, UK), and regular intravenous boluses of fentanyl (2–3 μg/kg; Sublimaze, Janssen, High Wycombe, Buckinghamshire, UK), and atracurium (150–250 μg/kg). Nitrous oxide was not used for maintenance of anaesthesia as its molecular mass (44) would have rendered it indistinguishable from carbon dioxide for the purpose of respiratory mass spectrometry (see below).

The pulmonary to systemic flow ratio was calculated in seven of the nine patients before duct occlusion (table 1). All children had an aortogram before the study, and patent arterial ducts were occluded using a 12 mm umbrella device (Bard, Galway, Ireland; patient 3), or one or more 5 mm detachable coils (William Cook Europe, Denmark). Following duct occlusion, a 4 F pigtail catheter (Cordis Europa, The Netherlands) was positioned in the descending aorta for arterial blood sampling, and a 6 F Berman angiographic catheter (Arrow International, Reading, Pennsylvania, USA) in the right atrium or right ventricle for mixed venous blood samples.

Group 2

This group consisted of seven children (four boys, three girls, median age 1.3 years) who were ventilated on the paediatric intensive care unit in the early postoperative period following open heart surgery. Cardiac output measurements were made between three and six hours after discontinuing cardiopulmonary bypass. Anthropometric data and details of cardiac surgery are given in table 1. None of these patients had previously undergone cardiac catheterisation, and so preoperative shunts—where present—had not been measured.

Children were selected for study on the basis of various criteria. Only patients who were considered haemodynamically stable at the intended time of study were included. Studies were carried out only on patients in whom rapid ventilatory weaning was not intended, and continued sedation would therefore have been part of the standard early postoperative management. All patients were receiving intravenous infusions of dopamine (3–8 μg/kg/min) at the time of study and no child was receiving more than one inotropic agent. All children were receiving an intravenous infusion of vecuronium (Norcuron, Organon-Teknika, Cambridge, UK) at a dose of 40–80 μg/kg/h, and were fully sedated with intravenous morphine (20–40 μg/kg/h) and midazolam (100–300 μg/kg/h). No changes were made to pharmacotherapy in terms of inotropic support

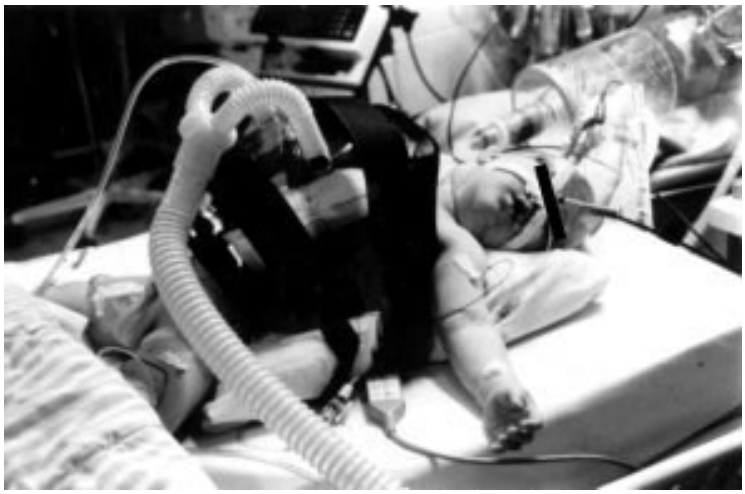

Figure 1 Cuirass negative pressure ventilation in a postoperative patient who was studied four hours after closure of a ventricular septal defect.

or colloid administration during the study period.

Patients in group 2 had indwelling peripheral arterial (five patients) or left atrial catheters (two patients), and pulmonary arterial catheters for arterial and mixed venous blood sampling, respectively.

PROCEDURES

Ventilation

All patients were intubated with cuffed endotracheal tubes (Mallinckrodt Medical, Northampton, UK), and were initially ventilated using volume cycled intermittent positive pressure ventilation (Servo ventilator 900C, Siemens Medical Systems, Solna, Sweden).

Negative pressure ventilation was delivered using the Hayek external high frequency oscillator (Medicom, Hendon, Middlesex, UK). This consists of a flexible cuirass which is attached to a fully programmable bedside power unit. The cuirass was placed over the patient's chest and upper abdomen, and sealed easily using Velcro straps placed around a bean filled pillow on which the patient was lying (fig 1).

The ventilatory settings during intermittent positive pressure ventilation and negative pressure ventilation are given in table 2; and during negative pressure ventilation these were adjusted to give similar end tidal carbon dioxide levels and minute volumes to those used during positive pressure ventilation. The inspired oxygen fraction was not altered when the mode of ventilation was changed. Patients remained intubated throughout the study period, and during negative pressure ventilation, the positive pressure ventilator was used to provide a constant flow of inspired oxygen, with a trigger sensitivity of -4 cm H_2O to deliver a brief period of pressure support of 3 to 5 cm H_2O. This was necessary to overcome airway resistance from the endotracheal tube.

Table 2 Ventilatory variables during intermittent positive pressure ventilation (IPPV) and negative pressure ventilation (NPV)

Variable	*IPPV*	*NPV*
Minute volume (ml/kg)	150 to 220	140 to 220
Frequency (breaths/min)	15 to 22	30 to 50
Tidal volume (ml/kg)	10 to 15	5 to 7
P_{insp} (cm H_2O)		
Group 1	+16 to +20	−18 to −24
Group 2	+18 to +23	
P_{exp} (cm H_2O)	0 to 2	+1 to + 4
P_{aw} (cm H_2O)	+ 5 to + 9	− 6 to −9
Pressure support (cm H_2O)		+ 3 to + 5
Fio_2 Group 1	0.25 to 0.35	as IPPV
Group 2	0.3 to 0.6	as IPPV

P_{insp}, inspiratory pressure; P_{exp}, expiratory pressure; P_{aw}, mean airway pressure; Fio_2, inspired oxygen fraction.

Patient monitoring

All patients had surface monitoring of heart rate and peripheral oxygen saturation, non-invasive monitoring of end tidal carbon dioxide, and invasive monitoring of systemic blood pressure. Patients in group 2 had additional monitoring of pulmonary arterial pressure.

Cardiac output measurement

Pulmonary blood flow was calculated using the direct Fick method. This requires steady state measurement of oxygen consumption and of arterial and mixed venous oxygen content. All children in group 1 had angiographic confirmation of complete arterial duct occlusion, and no patient in either group had echocardiographic evidence of any additional intracardiac shunt at the time of study. Pulmonary blood flow was therefore assumed to be equal to systemic, and hence cardiac, output.

Oxygen consumption was measured using on line respiratory mass spectrometry. The Amis 2000 mass spectrometer (Innovision A/S, Odense, Denmark) was adapted for use in ventilated patients, and the mixed expirate method was used for on line measurement of oxygen consumption.[11 12] We have previously described in detail our method of measurement of oxygen consumption, together with the modifications required for gas collection within the ventilator circuit.[10]

Protocol

The protocol for this study was approved by the ethics committee at the Royal Brompton Hospital, and informed consent was obtained from parents.

Before starting the cardiac output study, blood was sent for haemoglobin estimation. The cuff of the endotracheal tube was inflated (leakage around the tube was excluded by sampling for carbon dioxide with a probe connected to a mass spectrometer inlet) to ensure collection of all expired gas. Oxygen consumption monitoring was started, and a cardiorespiratory steady state was established with a stable systemic blood pressure and heart rate. At the end of a 15 minute period of steady state recording during intermittent positive pressure ventilation, a cardiac output measurement was made. A cuirass of the appropriate size was then fitted over the patient's chest, and negative pressure ventilation was begun, using the same inspired oxygen fraction. Rate and pressures during negative pressure ventilation were adjusted to maintain a similar minute volume and end tidal carbon dioxide to intermittent positive pressure ventilation, and at the end of

Table 3 Haemodynamic variables for individual patients during initial period of positive pressure ventilation (IPPV), negative pressure ventilation (NPV), and in selected patients following reinstitution of positive pressure ventilation ($IPPV_2$)

	Cardiac index (l/min/m²)			*$\dot{V}O_2$ (ml/min/m²)*			*a-v Difference (ml/dl)*			*MV saturation (%)*		
Patient	*IPPV*	*NPV*	*$IPPV_2$*	*IPPV*	*NPV*	*$IPPV_2$*	*IPPV*	*NPV*	*$IPPV_2$*	*IPPV*	*NPV*	*$IPPV_2$*
1	2.70	2.82		119	116		4.39	4.11		73.8	75.5	
2	5.41	6.10		179	190		3.31	3.12		74.0	75.8	
3	4.10	4.57		164	163		4.00	3.50		77.3	78.9	
4	4.36	4.91		218	223		5.00	4.54		71.1	74.2	
5	4.29	4.18		146	141		3.40	3.36		78.3	77.7	
6	3.78	4.10		197	219		5.20	5.24		71.6	72.0	
7	3.00	3.35		134	132		4.50	4.00		68.7	71.9	
8	3.08	3.77		135	156		4.39	4.14		75.4	77.2	
9	5.23	6.09		205	198		3.92	3.25		77.0	78.4	
10	2.40	3.20		155	181		6.46	5.69		58.4	63.3	
11	1.50	1.83	1.59	88	108	108	5.98	5.90	6.79	55.2	55.1	50.4
12	1.10	1.38	1.24	103	125	131	9.36	9.05	10.6	46.0	48.8	41.0
13	2.54	2.77	2.70	213	209	216	8.37	7.54	7.86	54.0	58.1	57.2
14	6.82	8.00	8.30	290	310	306	4.25	3.84	3.70	71.0	74.2	75.6
15	2.37	4.08	2.71	120	143	138	5.06	3.50	5.10	67.1	71.9	63.2
16	2.77	3.26		173	187		6.25	5.74		61.7	64.7	

$\dot{V}O_2$, oxygen consumption; a–v difference, arteriovenous oxygen difference; MV saturation, mixed venous oxygen saturation.

a 15 minute steady state period of negative pressure ventilation a second cardiac output measurement was made. The cuirass was then removed and positive pressure ventilation was reinstituted. A routine aortogram was performed in group 1 patients to confirm successful arterial duct closure. In five patients in group 2 patients a third cardiac output measurement was made after a further 15 minute period of intermittent positive pressure ventilation. No other therapeutic interventions were made during the study period.

STATISTICAL ANALYSIS

Data are expressed as mean (SD). Results within the two groups were analysed using an analysis of variance, with Bonferroni's correction for multiple comparisons. Between group data (group 1 *v* group 2) were compared using the Mann-Whitney U test. In all cases, the null hypothesis was rejected for p values of greater than 0.05.

Results

The study proceeded without complication in all patients, and efficient ventilation was achieved easily during negative pressure ventilation. All children in group 1 had a satisfactory result from arterial duct occlusion, with no residual leak detectable on angiography following the procedure.

Data for individual patients are given in table 3, and group data are shown in table 4. Similar gas exchange was achieved during both modes of ventilation, as shown by the systemic arterial carbon dioxide tension and oxygen saturation, both of which were unchanged during negative pressure ventilation. Mean arterial blood pressure and heart rate within each group were also unchanged.

GROUP 1 (AFTER DUCT OCCLUSION)

The mean oxygen consumption was unchanged during negative pressure ventilation, but the mixed venous saturation and oxygen content increased significantly ($p = 0.02$), and so the arteriovenous oxygen content difference fell from 4.23 (0.7) to 3.9 (1.2) ml/dl ($p < 0.01$). The cardiac index fell slightly during negative pressure ventilation in patient 5 (by 2.3%), but increased in the remainder. The mean increase in cardiac index during negative pressure ventilation was 10.8(7.1)% ($p < 0.01$; fig 2).

GROUP 2 (POSTOPERATIVE PATIENTS)

The oxygen consumption in the postoperative patients increased by 10.6% during negative pressure ventilation ($p < 0.01$). The mixed venous oxygen saturation and content increased during negative pressure ventilation, from 58.9(8.0)% to 62.3(9.1)% ($p = 0.03$). The arteriovenous oxygen difference thus fell

Table 4 Haemodynamic data for group 1 and group 2 during intermittent positive pressure ventilation (IPPV, A_1 and A_2), negative pressure ventilation (NPV, B_1 and B_2), and following reinstitution of IPPV (C_2).

	Group 1		Group 2			
Variable	A_1 *IPPV*	B_1 *NPV*	A_2 *IPPV*	B_2 *NPV*	C_2 * *$IPPV_2$*	*p<0.05*
Haemoglobin (g/dl)	11.8 (1.2)		11.7 (1.3)			-
$PaCO_2$ (kPa)	5.1 (1.2)	5.3 (1.3)	5.2 (1.4)	5.3 (1.4)	4.9 (1.2)	-
Sao_2 (%)	97.9 (2.3)	98.1 (1.9)	98.3 (1.1)	98 (1.4)	98.1 (1.5)	-
CI (l/min/m²)	4.0 (1.0)	4.5 (1.1)	2.8 (1.8)	3.5 (2.2)	3.2 (2.9)	A_1vB_1; A_2vB_2; A_1vA_2
Increase in CI (%)		10.8 (7.1)		28.1 (21)		B_1vB_2
$\dot{V}O_2$ (ml/min/m²)	166 (35)	171 (40)	163 (71)	180 (68)	180 (68)	A_2vB_2; A_2vC_2
MV saturation (%)	74 (4.0)	75.8 (3.0)	58.9 (8)	62.3 (9.1)	57.5 (13.1)	A_1vB_1; A_1vA_2; A_2vB_2; B_1vB_2; B_2vC_2
a-v Difference (ml/dl)	4.23 (0.7)	3.9 (1.2)	6.53 (1.8)	5.88 (1.9)	6.87 (2.6)	A_1vB_1; A_1vA_2; A_2vB_2; B_1vB_2; B_2vC_2
MAP (mm Hg)	75 (11)	75 (12)	61 (7)	59 (9)	60 (12)	A_1vA_2; B_1vB_2
Heart rate (beats/min)	108 (27)	108 (31)	138 (21.5)	138 (22)	129 (20)	A_1vA_2; B_1vB_2

Values are mean (SD). Significant differences are indicated by p<0.05.
*Five patients;
$PaCO_2$, arterial carbon dioxide tension; Sao_2, systemic arterial oxygen saturation; CI, cardiac index; $\dot{V}O_2$, oxygen consumption; MV, mixed venous; a-v difference, arteriovenous oxygen content difference; MAP, mean arterial pressure.

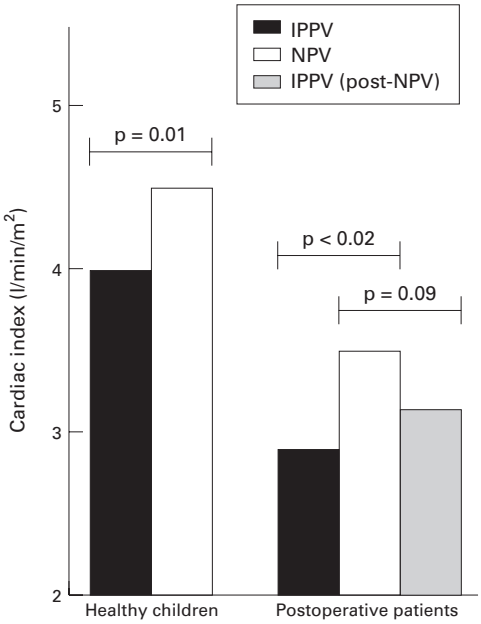

Figure 2 Cardiac index during intermittent positive pressure and negative pressure ventilation in the two patient groups.

from 6.5 (1.8) to 5.88 (1.9) ml/dl ($p = 0.03$). The cardiac index increased in all patients during negative pressure ventilation, from 2.8 (1.8) to 3.5 (2.2) l/min/m^2, and the mean increase was 28.1(21)% ($p < 0.001$; fig 2). Following reinstitution of intermittent positive pressure ventilation, the oxygen consumption was unchanged, and the mixed venous saturation and oxygen content fell in the five patients in whom a third measurement was made ($p = 0.01$). The cardiac index fell in four patients, and increased by 4% in the remaining child, but the overall change did not reach statistical significance.

GROUP 1 *V* GROUP 2

The mixed venous oxygen saturation was significantly lower for group 2 than for group 1 during both intermittent positive pressure and negative pressure ventilation (group 1 *v* group 2: $p = 0.001$ and $p < 0.01$ for positive and negative pressure ventilation, respectively). The cardiac index during intermittent positive pressure ventilation was significantly lower for group 1 than for group 2 ($p = 0.02$); but the difference during negative pressure ventilation was not significant ($p = 0.07$). This was reflected by the *increase* in cardiac index during negative pressure ventilation, which was significantly higher in the postoperative patients than in the healthy children ($p = 0.02$).

Discussion

We have shown that negative pressure ventilation using a cuirass device improves the cardiac output of children with a healthy cardiorespiratory system and, encouragingly, further improves the cardiac output of children in the early postoperative period after cardiopulmonary bypass.

There is little doubt that in selected patients following repair of tetralogy of Fallot[13] or Fontan-like operations,[14] where pulmonary blood flow can be critically dependent on the pressure changes generated by spontaneous breathing, negative pressure ventilation may have an important role in patient management as a haemodynamic tool. Such profound physiological cardiopulmonary interactions do not exist in all patients requiring mechanical ventilation. Having previously shown a 15% to 80% improvement in cardiac output during negative pressure ventilation after right heart surgery,[10] we applied a similar protocol in a group of children undergoing other types of cardiac surgery, and in a group of healthy children with structurally normal hearts.

In 1948, Cournand *et al* showed that the cardiac output was reduced in patients receiving intermittent positive pressure ventilation by mask. This study—one of the first to investigate cardiopulmonary dynamics physiologically during mechanical ventilation—showed that the cardiac output was inversely related to right ventricular filling pressures, which were in turn directly influenced by the ventilatory settings.[15] We now know that the effects of intermittent positive pressure ventilation on the cardiovascular system are complex, multifactorial, and at least partially influenced by the cardiorespiratory status of the model being studied.

In the absence of a left to right shunt, the cardiac output is equivalent to systemic venous return, and for a given preload, right heart filling is influenced by the pressure gradient between the peripheral veins and the right atrium. By reducing this gradient, an increase in intrathoracic pressure thus lowers the cardiac output,[16] and the converse occurs with a reduction in intrathoracic pressure. There is little doubt that systemic venous return is a major determinant of cardiac output, but several studies have suggested that this may not be the only factor. Henning showed that in a healthy animal model receiving positive pressure ventilation with high levels of positive end expiratory pressure, an infusion of colloid did not restore the cardiac output despite an adequate increase in right ventricular volume, and suggested that there must be other determinants of cardiac output during mechanical ventilation.[17] If the lungs are expanded beyond the functional residual capacity, a secondary increase in pulmonary vascular resistance results in an increase in right ventricular afterload. This may independently limit right ventricular stroke volume, and hence cardiac output.[18 19]

Various studies have reported the effects of positive pressure ventilation using high levels of positive end expiratory pressure on left ventricular performance. It has been suggested that impaired diastolic left ventricular compliance as a direct mechanical consequence of lung inflation,[20] or as a result of leftward displacement of the interventricular septum during right ventricular filling,[21] could limit the cardiac output during positive pressure ventilation. In certain patient groups, therefore, we should bear in mind the possibility of depression of left ventricular function with increasing mean airway pressure.

Cardiopulmonary bypass and reperfusion result in an intense generalised inflammatory reaction. Activation of neutrophils, endothelium, platelets, complement, and the coagulation cascade leads to diffuse endothelial damage with an increase in vascular permeability.[22 23] Multiorgan dysfunction occurs (albeit to the greater extent reversible), and a certain amount of pulmonary and myocardial injury is therefore an inevitable consequence of bypass. Alveolar and interstitial oedema, which may or may not be clinically apparent, reduce dynamic lung compliance—the ratio of the tidal volume to the peak inspiratory pressure[24]—and increase ventilation/perfusion mismatch. Therefore in post-bypass patients receiving positive pressure ventilation, of whom a significant proportion already require myocardial support, a higher mean airway pressure may be necessary to provide equivalent gas exchange in the less compliant lungs, potentially further jeopardising the cardiovascular system.

MECHANISM FOR THE IMPROVEMENT IN CARDIAC OUTPUT DURING NEGATIVE PRESSURE VENTILATION

The major mechanisms by which positive pressure ventilation can influence the cardiac output have been discussed, but how was the improvement in cardiac output achieved in our study groups during cuirass negative pressure ventilation? Lockhat *et al* showed that the cardiac output of normal dogs receiving thoracoabdominal negative pressure ventilation was significantly improved by augmentation of the systemic venous return—a phenomenon not present during whole body negative pressure ventilation or continuous positive pressure ventilation.[5] Skaburskis *et al* observed the same mechanism in dogs with non-cardiogenic pulmonary oedema receiving cuirass negative pressure ventilation.[7] It is highly unlikely that major changes in right ventricular afterload or left ventricular performance would have resulted from the relatively conservative mean airway pressure which we used during intermittent positive pressure ventilation. The Hayek oscillator is currently unique in its property of *actively* inducing the expiratory phase of ventilation. By doing so, it does not rely on the passive recoil which normally enables expiration in conventional positive and negative pressure ventilators.

We speculate that in our models of cuirass negative pressure ventilation, it is the differential effects on systemic venous return which are the most important determinants of the increases in cardiac output that were seen in both of our study groups. The increase in venous return may be further augmented in the post-bypass group by two mechanisms: first, negative pressure ventilation may improve lung compliance by ventilating around much lower mean airway pressures than intermittent positive pressure ventilation; second, by delivering active expiration, negative pressure ventilation may overcome the limitations to passive recoil which are present in the bypass injured lung.

LIMITATIONS OF THE STUDY

As far as we are aware, this is the first study that has compared and contrasted the effects of cuirass negative pressure ventilation and intermittent positive pressure ventilation on the cardiac output of healthy children and children who have undergone cardiac surgery. Cardiac output was measured using the direct Fick method, generally considered to be the most accurate method, and all our haemodynamic data were directly measured rather than derived or assumed.

For ethical reasons the protocol was specifically designed not to involve additional instrumentation or invasive monitoring, other than that routinely used in the catheter laboratory or on the paediatric intensive care unit. Thus, in the absence of pulmonary arterial and left atrial pressure monitoring, the pulmonary vascular resistance could not be calculated. It would have been informative to monitor the intrathoracic pressure when comparing two very different methods of ventilation, and particularly valuable with this protocol as it was necessary to deliver the inspiratory gas with a brief period of pressure support. This additional pressure support was necessary to overcome the flow resistance of the endotracheal tube and to allow an adequate minute volume, but may have partially obviated the beneficial effects of negative pressure ventilation.

In this study, our guidelines of intrathoracic pressure were the "mean airway pressure" during intermittent positive pressure ventilation, and "mean cuirass chamber pressure" during negative pressure ventilation. In previous studies involving invasive instrumentation of animals undergoing thoracotomy, or of adults undergoing open heart surgery, intrapericardial balloon catheters have been used to measure intrathoracic pressures.[24] Other groups have investigated the measurement of oesophageal pressure as a less invasive alternative to pericardial pressure, and have reported a reasonable correlation.[25] This may be an option for future studies in other clinical situations.

The children with a patent arterial duct had been exposed to higher than normal pulmonary blood flow before the study because of an (albeit small) arterial duct. They cannot therefore be considered truly "normal," as it is impossible to know the residual effects of the shunt on their subsequent cardiopulmonary haemodynamics following duct occlusion. Although the lack of invasive monitoring did not allow calculation of pulmonary vascular resistances, all patients had a normal preocclusion pulmonary arterial pressure, and as such they are as close to normality as is likely to be achieved in a clinical study of this kind.

CONCLUSION

We have shown that negative pressure ventilation leads to a modest but significant improvement in cardiac output when compared to conventional intermittent positive pressure ventilation in healthy children, and further improves the cardiac output following open heart surgery. While none of the patients in these study groups required augmentation of

their cardiac output, the early postoperative period following cardiopulmonary bypass for even simple defects can be a time when a small decline can precipitate significant haemodynamic instability. In this situation, a non-pharmacological means of improving the cardiac output by manipulation of important cardiopulmonary interactions may occasionally be a welcome management option. In selected cases, therefore, negative pressure ventilation using a cuirass device may be preferable to intermittent positive pressure ventilation in terms of combining similar ventilatory efficiency with additional haemodynamic protection.

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