Negative-Pressure Ventilation Improves Cardiac Output After Right Heart Surgery

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Background A low cardiac output state can complicate the postoperative course of patients undergoing Fontan-type operations and tetralogy of Fallot repair.

Methods and Results We investigated the effect of negative-pressure ventilation on cardiac output in 11 children in the early postoperative period after right heart surgery. All patients were initially ventilated with volume-cycled intermittent positive-pressure ventilation, and negative-pressure ventilation was delivered with the Hayek external high-frequency oscillator. Cardiac output was calculated by the direct Fick method, oxygen consumption being measured by respiratory mass spectrometry. Cardiac output was measured during intermittent positive-pressure ventilation and after 15 minutes of negative-pressure ventilation. Negative-pressure ventilation improved the cardiac output by a mean of 46% ($P=.005$). Heart rate did not change, and stroke volume increased by a mean of 48.5% ($P=.005$). Mixed venous saturation increased by 4.6% ($P<.02$), and consequently arteriovenous oxygen content difference fell significantly ($P=.01$). The systemic and pulmonary vascular resistances were reduced significantly during negative-pressure ventilation ($P<.05$ and $P<.03$, respectively).

Conclusions Negative-pressure ventilation improves cardiac output in children after total cavopulmonary connection and tetralogy of Fallot repair and may prove to be an important therapeutic option in children with the low cardiac output state. (*Circulation*. 1996;94[suppl II]:II-49-II-55.)

Key Words • cardiac output • ventilation • tetralogy of Fallot • Fontan procedure

The right ventricle frequently has abnormal anatomy and physiology in children with congenital heart disease, and abnormalities of its function are widely reported.[1] The normal cardiopulmonary interactions (the relationship between the action of breathing and phasic changes in right heart hemodynamics) have similarly been reported,[2,3] but there are relatively few data concerning this interaction in congenital heart disease, particularly in the immediate postoperative period.

IPPV has well-established beneficial effects on gas exchange,[4] but its negative influences on cardiac output may be important,[5] particularly after right heart operations such as Fontan-type procedures[6,7] and TOF repair. We have previously shown that pulmonary blood flow and hence cardiac output increase during spontaneous inspiration in patients after Fontan-type operations[8,9] and that in patients with restrictive physiology after TOF repair, IPPV reduces antegrade diastolic flow into the pulmonary artery and increases pulmonary incompetence, potentially reducing cardiac output.[10] In these patients, routine postoperative management should include IPPV with a low peak inspiratory pressure and without PEEP. Although we aim to extubate within 24 hours of the procedure, this may not be possible if the postoperative period is complicated by the low cardiac output state. In this situation, the use of alternative ventilation techniques may play a key role in improving cardiac output and preventing a rapid spiral of clinical decline.[11,12]

Negative-pressure oscillation with a cuirass ventilates the chest around a mean negative pressure and may have hemodynamic benefits over IPPV. In this study, we examined the potential use of the Hayek oscillator in improving the early postoperative hemodynamics after TCPC[13,14] or repair of TOF.

Methods

We performed a prospective study comparing cardiac output using IPPV and NPV in 11 children (6 boys; median age, 5.3 years) undergoing cardiac surgery between January and April 1995. All patients undergoing repair of TOF or Fontan-type operations were recruited into the study; 5 children had repair of TOF and 6 had fenestrated TCPC[15] for complex cyanotic congenital heart disease. Table 1 shows anthropometric data for each patient, with details of previous surgical procedures.

Intermittent Positive-Pressure Ventilation

On return from the operating theater, all children were ventilated with pressure-limited volume-cycled IPPV with the Siemens Servo ventilator 900C. Ventilatory parameters were set by the attending intensive care physician, with no child receiving PEEP.

Negative-Pressure Ventilation

NPV was delivered with the Hayek external high-frequency oscillator (Medicom Ltd). This consists of a power unit and a flexible cuirass, and in this study, a pediatric (for children weighing <20 kg) or an adult oscillator was used as appropriate. The cuirass size was chosen according to the size of the chest, and ventilatory parameters (rate and inspiratory and expiratory pressures) were adjusted to allow good chest excursion, similar minute ventilation, and end-tidal carbon dioxide measurements to IPPV. The mean chamber pressure was always negative.

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Selected Abbreviations and Acronyms

IPPV = intermittent positive-pressure ventilation
NPV = negative-pressure ventilation
PEEP = positive end-expiratory pressure
TCPC = total cavopulmonary connection
TOF = tetralogy of Fallot

Cardiac Output Measurements

Pulmonary blood flow was calculated by the direct Fick method (see "Appendix"). This requires measurement of oxygen consumption and arterial and mixed venous oxygen content (taking into account dissolved plasma oxygen for the latter two calculations). Oxygen consumption was measured by bedside respiratory mass spectrometry. Mixed venous oxygen content was calculated from pulmonary arterial blood samples. Arterial oxygen content was calculated from peripheral arterial or left atrial samples after TOF repair and from pulmonary venous samples after TCPC. In the cases of TOF, in which no child had evidence of intracardiac shunting, pulmonary blood flow was equal to cardiac output. In the children with fenestrated TCPC, in which an obligatory right-to-left shunt exists effectively at the atrial level, we measured pulmonary blood flow and calculated the shunt fraction using systemic arterial blood samples.

Respiratory Mass Spectrometry

Respiratory mass spectrometry is a highly sensitive and accurate noninvasive method of continuous gas analysis that allows simultaneous measurement of constituent fractions of a gas mixture on the basis of their mass-to-charge ratio alone.

An Amis 2000 quadripolar mass spectrometer (Innovision A/S) was modified on our intensive care unit for use in ventilated patients. This gives on-line readings of oxygen consumption, carbon dioxide excretion, respiratory exchange ratio, end-tidal carbon dioxide, tidal volume, and expiratory minute ventilation.

Oxygen consumption ($\dot{V}o_2$) was measured by the mixed expirate inert gas dilution method of Davies and Denison.[16] This technique requires collection of expired gas and analysis of inspired and expired gas. Inspired gas was sampled by an inlet at the endotracheal tube. All expired gas was collected from the expiratory limb of the patient's breathing circuit and directed into the proximal port of a mixing box with a 4.5-L capacity. A known flow of inert indicator gas (argon) was added to this, and the resultant gas mixture was sampled from an inlet at the distal port of the mixing box. Inspired and expired gas mixtures were sampled at a rate of ≈10 mL/min down narrow-bore Teflon tubing (0.3-mm ID). Each constituent inspired gas (argon, oxygen, nitrogen, and carbon dioxide) was analyzed at least six times each second, and $\dot{V}o_2$ was calculated (see "Appendix") and recorded on-line every 30 seconds.

Before starting each study, we performed a two-point calibration exposing the distal inlet both to a four-gas calibration mixture (nitrogen, oxygen, carbon dioxide, and argon) and to zero gas (closed inlet). This calibration was repeated at 30-minute intervals throughout the study period. The tracer gas flow (pure argon) was calibrated manually.

Protocol

This study was approved by our local Ethics Committee, and written informed consent was obtained from the parents of each child. All children were nasally intubated with cuffed endotracheal tubes (Mallinckrodt Medical) after induction of anesthesia, and on return from the operating theater, they were ventilated with pressure-limited volume-cycled IPPV with a PEEP of zero.

Expired gas was collected from the expiratory limb of the patient's breathing circuit and directed into the proximal port of the airtight mixing box, whose outlet was connected to the expiratory port of the ventilator. All children received continuous intravenous infusions of morphine (40 $\mu g \cdot kg^{-1} \cdot h^{-1}$), midazolam (0.1 to 0.3 $mg \cdot kg^{-1} \cdot h^{-1}$), and vecuronium (50 to 80 $\mu g \cdot kg^{-1} \cdot h^{-1}$) and had invasive intravascular monitoring of systemic blood pressure, pulmonary arterial pressure, and central venous pressure. Heart rate, peripheral oxygen saturation, core and peripheral temperatures, end-tidal carbon dioxide, and oxygen consumption were measured noninvasively.

A blood sample was sent for hemoglobin estimation before the study was begun. Mixed venous oxygen content was calculated from pulmonary arterial blood samples, and arterial oxygen content from peripheral arterial, left atrial, or pulmonary venous samples, as appropriate.

Pulmonary blood flow measurements were made between 2 and 15 hours after surgery. A measurement, a record of drug doses, and simultaneous hemodynamic parameters were taken for each patient in a cardiorespiratory steady state during IPPV with the cuff of the endotracheal tube inflated to prevent leakage of expired gas. A cardiorespiratory steady state was defined as a 15-minute period with <5% fluctuation in mean arterial pressure, oxygen consumption, and end-tidal carbon dioxide. After the cuirass was placed over the chest and upper abdomen and secured to eliminate significant leaks, NPV was started and IPPV stopped. The rate and inspiratory and expiratory pressures during NPV were adjusted to give a similar end-tidal carbon dioxide. The inspired oxygen fraction (delivered by the Servo ventilator 900C)

TABLE 1. Anthropometric Details of the Patients Entering the Study

Patient	Age, y	Weight, kg	Diagnosis	Previous Surgery	Operation
1	5.3	15.5	DILV, VA disc, sub-PS	None	Fenestrated TCPC
2	6.4	16.8	PA IVS	L BT shunt, bidir Glenn	Fenestrated TCPC
3	5.3	12.9	TOF	None	Repair TOF
4	0.9	8.4	TOF	None	Repair TOF
5	3.8	12.4	DILV, VA disc, CoA, R-sided rudimentary RV, bilateral SVC	CoA repair, PA band, enlargement VSD	Fenestrated TCPC
6	6.8	15.4	Complete AVSD, DORV, L-sided aorta, PS	None	Fenestrated TCPC
7	13	43	TOF, bilateral SVC	None	Repair TOF
8	6	21	TOF	None	Repair TOF
9	1.3	9.4	Down's, TOF, AVSD	None	Repair TOF
10	6	15.6	DILV, straddling LAV valve	PA band	Fenestrated TCPC
11*	3.8	12.4	DILV, VA conc, bilateral SVC	CoA repair, PA band	Fenestrated TCPC

DILV indicates double-inlet left ventricle; VA disc, ventriculoarterial discordance; PS, pulmonary stenosis; PA IVS, pulmonary atresia with intact ventricular septum; CoA, coarctation of the aorta; RV, right ventricle; SVC, superior vena cava; AVSD, atrioventricular septal defect; LAV, left atrioventricular valve; VA conc, ventriculoarterial concordance; L BT shunt, left modified Blalock-Taussig shunt; VSD, ventricular septal defect; and PA band, pulmonary artery band.

*Patient excluded from analysis (see "Methods").

TABLE 2. Hemodynamic Parameters of Individual Patients During IPPV and NPV

	Pulmonary Blood Flow Index, L·min⁻¹·m⁻²		Heart Rate, bpm		Stroke Volume, mL·beat⁻¹·m⁻²		Oxygen Consumption, mL·min⁻¹·m⁻²		PA Saturation, %		AV Oxygen Content Difference, mL/100 mL		Mean ABP, mm Hg		SVRI, U/m²		PVRI, U/m²	
Patient	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV	PPV	NPV
1	0.56	0.92	119	111	4.71	8.3	68	105	22	25	12.2	11.7	64	59	...	...	1.79	1.09
2	1.07	1.23	130	140	8.2	8.8	79	96	50.7	50.3	7.4	7.7	64	71	...	...	2.8	2.44
3	2.71	3.75	136*	136*	19.9	27.6	167	217	70	70	6.2	5.8	65	62	18.3	13.3	...	...
4	1.46	2.66	161	160	9.1	16.6	146	200	42	57.2	10	7.5	61	58	30.3	16.2	...	
5	4.65	6.78	144	135	32.3	50.2	203	227	71.8	78.7	4.4	3.3	51	50	...	...	0.65	0.44
6	2.87	4.76	144	150	19.9	31.7	174	210	68.9	77.4	6.1	4.4	67	67	...	...	0.5	0.33
7	2.52	3.58	100	100	25.2	35.8	121	129	70.2	77.2	4.8	3.6	69	71	22.6	15.9	1.1	0.56
8	5.86	8.42	133	120	44.1	70.2	238	238	77	80.3	4.1	3.0	59	60	7.9	5.5	...	...
9	1.10	1.49	147	145	7.5	10.3	101	126	53.1	55.5	8.7	8.4	43	50	28.5	24.8	...	...
10	2.62	3.33	114	113	23.3	38.6	146	176	76.9	77.3	5.5	5.3	54	60	...	...	0.75	0.72

PA indicates pulmonary arterial; AV, arteriovenous; ABP, arterial blood pressure; SVRI, systemic vascular resistance index; and PVRI, pulmonary vascular resistance index. ... indicates calculation not possible due to insufficient hemodynamic data.
*Patient atrially paced.

was not altered. A pulmonary blood flow measurement and a full record of ventilatory and hemodynamic parameters were made after 15 minutes of NPV. IPPV was then reinstituted, and the cuirass was removed. Exclusion criteria for the study were excessive losses from pleural or mediastinal drains and radiological evidence of lung collapse, consolidation, or significant effusions, none of which occurred in this group of children. No child had a splinted chest. Intravenous colloids were not infused, and adjustments to pharmacological management were not made during the study period. One patient (patient 11) desaturated after NPV was started, and despite good chest excursion and adequate inspiratory pressures, we were unable to maintain a satisfactory end-tidal carbon dioxide. This complication was unexplained, but the study could not be completed in this patient.

In five patients, simultaneous transesophageal echocardiography with a biplane pediatric probe (Hewlett-Packard Instruments) was performed to assess the pattern of pulmonary blood flow during IPPV and NPV.

TABLE 3. Hemodynamic and Ventilatory Parameters During IPPV and NPV

Parameter	IPPV	NPV	P
End-tidal CO_2, %	4.53	4.61	.29
Pulmonary blood flow index, L·min⁻¹·m⁻²	2.54	3.68	.005*
Heart rate, bpm	131.4	132.8	.33
Stroke volume index, mL·beat⁻¹·m⁻²	19.4	28.8	.005*
Oxygen consumption, mL·min⁻¹·m⁻²	144	172	.008*
Arteriovenous oxygen content difference, mL oxygen/100 mL blood	6.10	6.93	.014*
Mixed venous saturation, %	60.3	64.9	.017*
Mixed venous oxygen content, mL oxygen/100 mL blood	10.92	11.69	.014*
Mean arterial blood pressure, mm Hg	59.8	60.9	.51
Right atrial pressure, mm Hg	13.8	13.4	.58
Systemic vascular resistance index, U/m²	21.52	15.15	.043*
Mean pulmonary arterial pressure, mm Hg	10.5	10.8	.29
Left atrial pressure, mm Hg	8.3	8.6	.47
Pulmonary vascular resistance index, U/m²	1.28	0.93	.028*
Peak inspiratory pressure, cm H_2O	+18	−19	
Mean airway pressure during IPPV, cm H_2O†	+7		
Mean chamber pressure during NPV, cm H_2O†		−7	

Mean values of group data are shown.
*Statistically significant change.
†Peak inspiratory pressure during IPPV ranged from +16 to +22 cm H_2O, and during NPV, from −18 to −24 cm H_2O.

Statistical Analysis

Grouped data are expressed as mean±SD. The Wilcoxon matched-pairs signed-rank test was used for statistical analysis. The null hypothesis was rejected for values of $P<.05$.

Results

There were no adverse hemodynamic consequences from use of the oscillator, and chest drain losses were not increased during its use. In one patient (see "Methods"), adequate ventilation could not be maintained, and the data are excluded from further analysis.

Results for individual patients are shown in Table 2 and for grouped data in Table 3. There was no significant change in end-tidal carbon dioxide during NPV ($P=.29$). The mean pulmonary blood flow during IPPV was 2.54±1.67 L·min⁻¹·m⁻², and the mean during NPV was 3.68±2.43 L·min⁻¹·m⁻² (see Fig 1). Thus, NPV increased pulmonary blood flow by 46.1±20.5% ($P=.005$). Patient 3 was atrially paced. In the remainder, the heart rate did not change significantly ($P=.33$), and the increase in pulmonary blood flow was achieved by an increase in stroke volume from 19.4±12.5 to 28.8±19.8 mL, with a mean increase of 49.4±24.2% ($P=.005$). The V_{O_2}

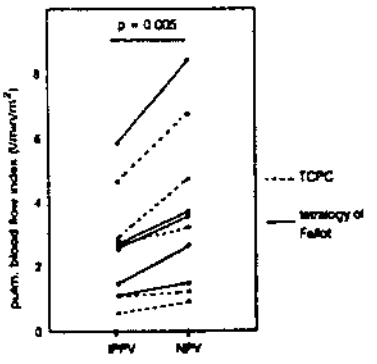

FIG 1. Pulmonary (pulm.) blood flow index during IPPV and after 15 minutes of NPV. Values for each patient are shown. Mean increase in pulmonary blood flow was 46%. There was no significant difference between the increase in pulmonary blood flow in patients after TOF repair and TCPC.

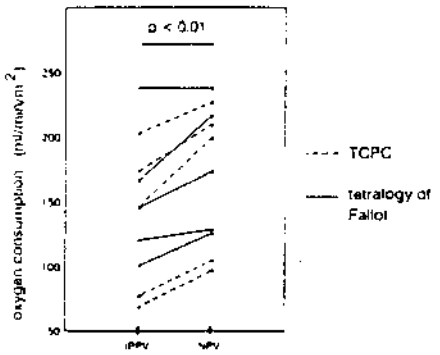

FIG 2. Oxygen consumption during IPPV and after 15 minutes of NPV. Values for each patient are shown.

increased slightly, from 142±54.7 to 171±56.1 mL·min⁻¹·m⁻² during NPV ($P=.02$, see Fig 2). The arterial oxygen content was unchanged during NPV, but the mixed venous oxygen content increased by a mean of 0.77±0.83 mL oxygen/100 mL blood ($P=.01$), which was reflected by an increase in mixed venous saturation of 4.6±4.6%. The arteriovenous oxygen content difference therefore fell significantly ($P=.01$, see Fig 3). There was no significant change in mean systemic arterial ($P=.51$) or right atrial pressures ($P=.58$) or in mean pulmonary arterial ($P=.29$) or left atrial pressures ($P=.47$). The systemic vascular resistance in the TOF group thus fell significantly ($P<.05$), as did the pulmonary vascular resistance, calculated in the patients with indwelling left atrial catheters (5 TCPC+1 TOF, $P<.03$, see Fig 4).

Details of the right-to-left shunt fraction in patients with a fenestrated TCPC are given in Table 4. Overall, there was no significant change in the shunt fraction ($P=.5$). In three patients the shunt fraction was reduced, reflecting an absolute increase in systemic blood flow, and in two patients the shunt fraction increased slightly, indicating a rise in the pulmonary relative to systemic blood flow.

The pulmonary blood flow during IPPV and the pattern seen during NPV were markedly different in the patients with TCPC and TOF, as illustrated in Figs 5 and 6, respectively.

Discussion

The postoperative course in most patients after right heart surgery is uncomplicated. Inotrope requirements are low, and ventilation can be rapidly weaned. However, in a significant minority of patients who have undergone TOF

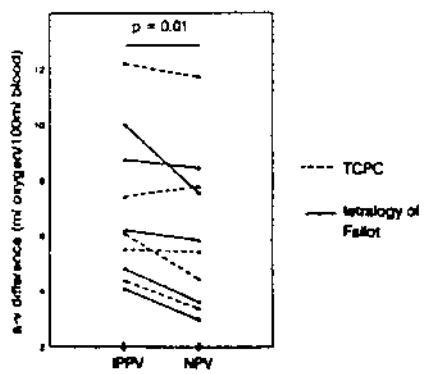

FIG 3. Arteriovenous (a-v) oxygen content difference during IPPV and after 15 minutes of NPV. Values for each patient are shown.

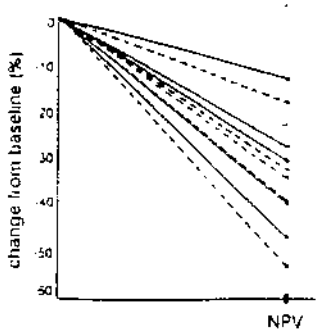

FIG 4. Percentage reduction in systemic (SVR, solid lines) and pulmonary (PVR, dashed lines) vascular resistances during NPV. The value during IPPV for each patient is taken as zero. SVR is shown for patients with TOF and PVR for patients with TCPC.

repair or Fontan-type operations, the early postoperative period is complicated by the low cardiac output state with hypotension, metabolic acidosis, and oliguria. In these patients, although it may be potentially detrimental to the hemodynamics, it is inappropriate to wean ventilation.

We have previously shown that the low cardiac output state after Fontan-type operations and repair of TOF is most often caused by inadequate pulmonary blood flow and not systemic ventricular dysfunction.[10] Furthermore, it is often refractory to conventional supportive therapy; colloid infusions elevate filling pressures, encouraging pleural and peritoneal fluid accumulation, and the administration of inotropes can lead to peripheral and myocardial ischemia, tachycardia, and unwanted increases in systemic and pulmonary vascular resistances. A more direct method of increasing pulmonary blood flow, and hence cardiac output, in these patients would have clear advantages.

We and others have previously examined in detail the influences of spontaneous and mechanical ventilation on hemodynamics after right heart surgery. Using Doppler techniques, we have previously shown that pulmonary arterial flow in spontaneously breathing convalescent patients after the Fontan operation is 64% higher during inspiratory than expiratory cardiac cycles.[7] An increase in mean airway pressure, eg, during a Valsalva maneuver, leads to a marked reduction and even retrograde flow out of the lungs in patients with the total cavopulmonary anastomosis.[8] These findings support the observation of Williams et al[7] that the level of PEEP is inversely related to cardiac output in the early postoperative period after the atriopulmonary connection. Clearly, an increased mean airway pressure is undesirable in the Fontan circulation, and an improvement in hemodynamics may therefore be possible if it could be reduced or a negative mean airway pressure could be maintained.[11,12]

TABLE 4. Right-to-Left Shunt Fraction in Five Patients With Fenestrated TCPC During IPPV and NPV

Patient	Right-to-Left Shunt Fraction	
	IPPV	NPV
1	0.03	0.04
2	0.11	0.09
5	0.33	0.30
6	0.23	0.30
10	0.17	0.06
	$P=.5$	

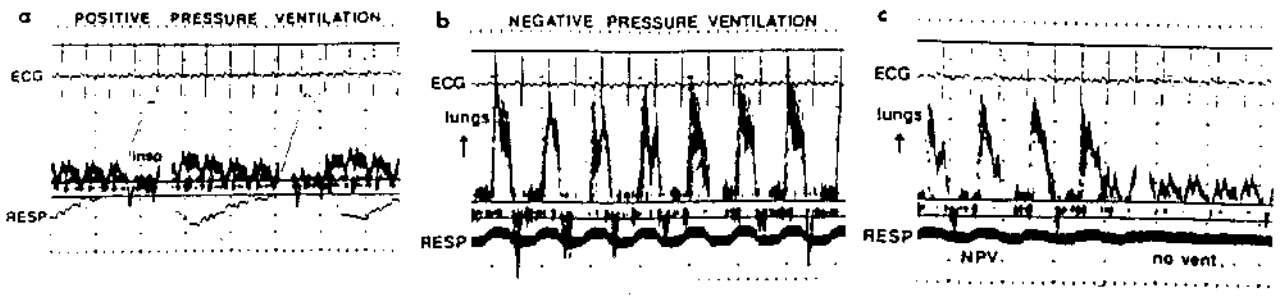

FIG 5. Changes in Doppler-derived pulmonary arterial flow in a patient after fenestrated TCPC (a) during IPPV, (b) with NPV, and (c) after removal of the cuirass. Antegrade flow is lost during the inspiratory (insp) phase of IPPV. There is a marked increase in forward flow during the inspiratory phase of NPV (shown as the upward stroke of respirometer [RESP] trace), which is immediately lost on removal of the cuirass. Vent indicates ventilation.

The reason underlying the low cardiac output state after repair of TOF is different, but it may be equally important. In our recent study of postoperative diastolic function, right ventricular restrictive diastolic physiology was present in ≈40% of patients, and its presence was associated with prolonged postoperative recovery complicated by the low cardiac output state.[16] In these patients, pulmonary arterial Doppler profiles showed characteristic antegrade late diastolic flow coincident with atrial systole, the hallmark of the poorly compliant right ventricle with restrictive diastolic physiology. Although abnormal, the a wave represents up to one third of total pulmonary arterial forward flow and reduces the time available for pulmonary regurgitation. It is, of course, generated by the atrial systolic pressure exceeding pulmonary arterial diastolic pressure, and this flow is usually abolished during the inspiratory phase of IPPV. This, coupled with our finding that the a wave increases during spontaneous inspiration in other groups of patients,[17] strongly suggests that ventilation with a negative mean airway pressure might enhance pulmonary blood flow in the presence of right ventricular restriction.

Our data support our hypothesis that cardiopulmonary interactions may be crucial to the maintenance of cardiac output after right heart surgery. Despite what we would consider "optimal" management of IPPV, with low peak inspiratory pressures and using no PEEP, we have shown a large increase in cardiac output with NPV without any of the unwanted side effects of conventional pharmacotherapy.

Some of our patients had an adequate or high cardiac output during the period of study, but a number had a critically low cardiac output and, encouragingly, the percentage increase in cardiac output was independent of baseline indexes. The patients who had undergone repair of TOF (of whom three had restrictive right ventricular physiology) and TCPC benefited equally from NPV, which stresses the important influence of ventilatory movements on pulmonary blood flow in both groups. The dramatic changes in the pattern of pulmonary blood flow during NPV are illustrated in Figs 1 and 2 in one child after TCPC and in one with restrictive physiology after repair of TOF, respectively.

Interestingly, the increase in cardiac output led to a reduction in systemic and pulmonary vascular resistances with relatively little change in heart rate and venous pressures. This suggests that these latter parameters were adaptive to reflect the cardiac output before the institution of NPV. The major adaptive response to the increased cardiac output during NPV was a fall in effective vascular resistance in the systemic and pulmonary vascular beds. In the lung, this may well be a direct hydraulic response to the reduction in mean airway pressure during NPV, but the fall in systemic vascular resistance probably reflects im-

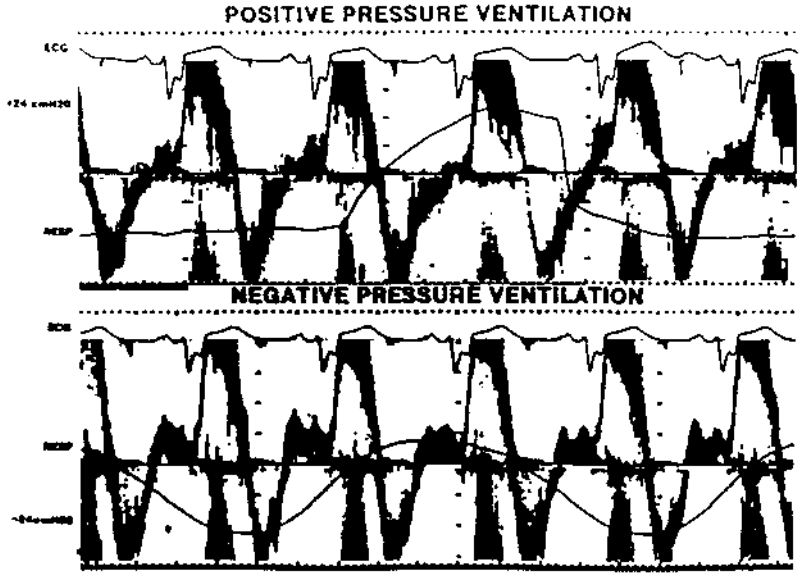

FIG 6. Increase in Doppler-derived pulmonary arterial flow during NPV in a patient with restrictive right ventricular physiology after TOF repair. Characteristic antegrade diastolic flow (the a wave), coincident with atrial systole, is seen during the expiratory phase of IPPV. This is lost during the inspiratory phase of IPPV (shown as the upward stroke on the respirometer [RESP] trace), and the pulmonary regurgitant fraction is increased. During NPV, the a wave is preserved and much increased throughout the respiratory cycle, and there is a reduction in the pulmonary regurgitant fraction.

proved flow to previously constricted vascular beds. Regional blood flow was not measured in this study, but it would be interesting to assess cerebral, renal, and splanchnic blood flow in longer-term studies.

Critique

This study does not attempt to demonstrate the long-term utility of the oscillator as either a ventilator or a hemodynamic tool. Nonetheless, the oscillator has been established as an alternative to more conventional techniques of NPV in children with neuromuscular disease and in premature infants with bronchopulmonary dysplasia. In this study, it was of paramount importance to establish a respiratory steady state, so we did not explore the specific ability of the Hayek oscillator to ventilate the lungs in the longer term in these patients.

This study group consisted of older infants and children. However, its practical design has enabled our subsequent successful use of the oscillator in younger infants and in neonates with congenital heart disease.

The reason for choosing a short period of NPV was to avoid the confounding effects of possible metabolic and cardiovascular changes that occur naturally during the first few hours after cardiopulmonary bypass. However, there is no reason why NPV should not work, and our anecdotal evidence of its longer-term use as a therapeutic tool is already very encouraging.

Methodologically, we feel that the direct Fick method is the only adequate way of measuring the cardiac output in these patients. Thermodilution is invalid in the Fontan circulation and may be inaccurate in patients who have undergone repair of TOF, in which pulmonary regurgitation is common. Nonetheless, in the fenestrated TCPC, in which we directly measured only the effective pulmonary blood flow, we also assessed the right-to-left shunt fraction. Although overall, this was unchanged during NPV, it increased in two of the five (reflecting a relative increase in shunting through the fenestration) and decreased in three. The total systemic blood flow, however, increased in all of the patients.

The changes in oxygen consumption might be superficially surprising. There were no obvious signs of waking in terms of increases in heart rate, blood pressure, or metabolic acidosis. Indeed, in seven children subsequently studied with the same protocol (but not reported here), we found no difference in serum epinephrine and norepinephrine levels during IPPV and NPV. We believe that the increase in oxygen consumption can be explained on the basis of delivery dependence, which is increasingly recognized in adults[18] and which we have reported to occur in children over a wide range of cardiac output.[19] That is, with increasing delivery of oxygen to the tissues, there is a linear response to increased oxygen consumption.

Conclusions

NPV clearly improves the cardiac output in children after repair of TOF and TCPC. We do not normally aim for a "supranormal" level of oxygen delivery in children after cardiac surgery and so would not advocate the routine use of NPV in all cases. In the child with clinical indicators of a low cardiac output, however, in whom weaning from positive-pressure ventilation is not possible, we believe that NPV has a valuable place in intensive care management.

Appendix

Hemodynamic Equations

$$\dot{V}O_2 = \dot{V}_{T_r}\{[FiO_2(1-F_MCO_2-F_{MT_r}) - F_MO_2(1-FiCO_2-F_{T_r})]/D\}$$

and

$$D = F_{MT_r}(1-FiO_2-FiCO_2) - Fi_{T_r}(1-F_MO_2-F_MCO_2)$$

where $\dot{V}_{T_r}$ is the flow of added tracer gas (argon) and F_MO_2, F_MCO_2, and F_{MT_r} are measured concentrations of oxygen, carbon dioxide, and argon, respectively, at the outlet of the mixing chamber. FiO_2, $FiCO_2$, and Fi_{T_r} are inspired concentrations of oxygen, carbon dioxide, and argon, respectively.

$$Qp\ (\text{in L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}) = [\dot{V}O_2/(CaO_2 - CvO_2)]/BSA$$

$$CaO_2\ (\text{in mL/100 mL blood}) = [(SaO_2 \times 1.34 \times Hb)/100] - (PaO_2 \times 0.003)$$

$$CvO_2\ (\text{in mL/100 mL blood}) = [(SvO_2 \times 1.34 \times Hb)/100] + (PvO_2 \times 0.003)$$

$$SVRI\ (\text{in U/m}^2) = (MAP - RAP)/Q_s$$

$$PVRI\ (\text{in U/m}^2) = (MPAP - LAP)/Q_p$$

where Q_p and Q_s are pulmonary and systemic blood flow indexes, respectively; BSA is body surface area; Hb is hemoglobin; SVRI is systemic vascular resistance index; PVRI is pulmonary vascular resistance index; MAP is mean arterial blood pressure; MPAP is mean pulmonary arterial pressure; LAP is left atrial pressure; and RAP is right atrial pressure.

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References

1. Norgård G, Vik-Mo H. Right ventricular size and function as assessed by echocardiography and angiography in patients with different volume load. *Pediatr Cardiol.* 1995;16:209-215.
2. Riggs TW, Snider R. Respiratory influences on right and left ventricular diastolic function in normal children. *Am J Cardiol.* 1989; 63:858-861.
3. Norgård G, Vik-Mo H. Effects of respiration on right ventricular size and function: an echocardiographic study. *Pediatr Cardiol.* 1992; 13:136-140.
4. Pick MJ, Hatch DJ, Kerr AN. The effect of positive end-expiratory pressure on lung mechanics and arterial oxygenation after open heart surgery in young children. *Br J Anaesth.* 1976;48:981-987.
5. Dorinsky PM, Whitcomb ME. The effect of PEEP on cardiac output. *Chest.* 1983;84:210-216.
6. Fontan F, Baudet E. Surgical repair of tricuspid atresia. *Thorax.* 1971;26:240-248.
7. Williams DB, Kiernan PD, Metke MP, Marsh HM, Danielson GK. Hemodynamic response to positive end-expiratory pressure following right atrium-pulmonary artery bypass (Fontan procedure). *J Thorac Cardiovasc Surg.* 1984;87:856-861.
8. Penny DJ, Redington AN. Doppler echocardiographic evaluation of pulmonary blood flow after the Fontan operation: the role of the lungs. *Br Heart J.* 1991;66:372-374.
9. Redington AN, Penny D, Shinebourne EA. Pulmonary blood flow after total cavopulmonary shunt. *Br Heart J.* 1991;65:213-217.
10. Cullen S, Shore D, Redington A. Characterization of right ventricular diastolic performance after complete repair of tetralogy of Fallot:

restrictive physiology predicts slow postoperative recovery. *Circulation*. 1995;91:1782-1789.
11. Penny DJ, Hayek Z, Rawle P, Rigby ML, Redington AN. Ventilation with external high frequency oscillation around a negative baseline increases pulmonary blood flow after the Fontan operation. *Cardiol Young*. 1992;2:277-280.
12. Penny DJ, Hayek Z, Redington AN. The effects of positive and negative extrathoracic pressure ventilation on pulmonary blood flow after the total cavopulmonary shunt procedure. *Int J Cardiol*. 1991;30:128-130.
13. de Leval MR, Kilner P, Gewillig M, Bull C. Total cavopulmonary connection: a logical alternative to atriopulmonary connection for complex Fontan operations. *J Thorac Cardiovasc Surg*. 1988;96:682-695.
14. Kawashima Y, Kitamura S, Matsuda H, Shimazaki Y, Nakano S, Hirose H. Total cavopulmonary shunt operation in complex cardiac anomalies: a new operation. *J Thorac Cardiovasc Surg*. 1984; 87:74-81.
15. Bridges ND, Lock JE, Castaneda AR. Baffle fenestration with subsequent transcatheter closure: modification of the Fontan operation for patients at increased risk. *Circulation*. 1990;82:1681-1689.
16. Davies NJH, Denison DM. The measurement of metabolic gas exchange and minute volume by mass spectrometry alone. *Respir Physiol*. 1979;36:261-267.
17. Redington AN, Penny D, Rigby ML, Hayes A. Antegrade diastolic pulmonary arterial flow as a marker of right ventricular restriction after complete repair of pulmonary atresia with intact ventricular septum and critical pulmonary valve stenosis. *Cardiol Young*. 1992;2: 382-386.
18. Knox JB. Oxygen consumption-oxygen delivery dependency in adult respiratory distress syndrome. *New Horiz*. 1993;1:381-387.
19. Shekerdemian LS, Bush A, Redington AN. Delivery-dependent oxygen consumption in children after cardiac surgery. *Crit Care Med*. 1996;24(suppl 1):A153. Abstract.