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Negative pressure ventilation as haemodynamic rescue following surgery for congenital heart disease

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Abstract A low cardiac output state is an important cause of morbidity and mortality following repair of tetralogy of Fallot (ToF). This is often refractory to conventional measures. The cardiac output of these patients is highly dependent on diastolic pulmonary arterial flow which is enhanced during spontaneous respiration, but much reduced by intermittent positive pressure ventilation (IPPV).
We report the successful use of negative pressure ventilation (NPV) as haemodynamic therapy in three children with a low output secondary to restrictive right ventricular (RV) physiology following ToF repair. NPV produced a significant haemodynamic improvement, with increases in cardiac output of greater than 100 % in two of the children. By augmenting pulmonary blood flow, and hence cardiac output, NPV has a role as adjunctive haemodynamic therapy in patients with a low output secondary to diastolic RV dysfunction, in whom early extubation is not possible.

Key words Children · Cardiac output · Ventilation · Diastole

Introduction

A low cardiac output state can complicate the post-operative course of children undergoing surgery for congenital heart disease. Tetralogy of Fallot (ToF) accounts for around 5 % of all congenital heart defects, and although complete surgical correction of ToF is now associated with a low operative mortality, a minority of patients develop a severe low-output state which, in the extreme, can necessitate the use of extra-corporeal life support [1].

Over one-third of patients undergoing ToF repair have diastolic right ventricular (RV) dysfunction secondary to restrictive physiology in the early post-operative period, and a small but significant sub-group of these patients can develop a severe low-output state while on the intensive care unit (ICU) [2]. In these patients, where ventricular systolic function is usually well-preserved, the low-output state is typically refractory to conventional haemodynamic measures such as colloid therapy and inotropic support. Restrictive RV physiology is characterised by diastolic antegrade pulmonary arterial flow, which makes a significant contribution to the total cardiac output [2]. This important diastolic flow is enhanced during spontaneous inspiration but reduced, or even lost, during positive pressure inspiration [2]. Thus, patients with a low-output state secondary to restrictive RV physiology would ideally benefit from early extubation in order to establish spontaneous respiration but, paradoxically, are more likely to need ongoing ventilatory support in the presence of borderline haemodynamics.

We previously reported a significant improvement in cardiac output during a brief period of cuirass negative pressure ventilation (NPV) using the Hayek oscillator in children on the ICU in the early post-operative peri-

Table 1 Patient details and haemodynamic parameters before starting negative pressure ventilation, after 4 hours' therapy, and at the end of the treatment period

Pt (age yrs)	BP (S/D/M)		pH/BE		CVP		urine (ml/kg/hr)		CI (l/min/m^2)				duration NPV (hrs)
	pre	4 hrs	pre	4 hrs	pre	4 hrs	pre	4 hrs	pre	4 hrs	pre-d/c	1 hr off	
1 (1.5)	58/35/42	71/41/53	7.30/–6	7.41/–4	13	10	0.8	5	2.2	4.8	4.9	4.8	7
2 (0.5)	60/42/49	75/56/63	7.25/–8	7.37/–4	16	12	1.0	3	1.3	2.3	3.0	3.1	12
3[a] (0.8)	48/36/41	68/49/56	7.34/–6	7.50/0	16	14	0.8	7					78

BP (S/D/M) – blood pressure (systolic, diastolic, mean); BE – base excess on arterial blood gas; CVP – central venous (right atrial) pressure; CI – cardiac index; pre-d/c – before discontinuing NPV
[a] cardiac output not measured in this patient

od following ToF repair [3, 4]. These studies clearly suggested that NPV might have a potential role in the haemodynamic management of patients with a low output who were unlikely to be ready for early extubation. Furthermore, studies of adult medical [5] and cardiac surgical [6] intensive care patients have shown a haemodynamic improvement during a trial of cuirass NPV. We now report the first long-term application of NPV as adjunctive haemodynamic therapy in three children with a severe low-output state in the early post-operative period following ToF repair.

Patients and methods

Three children underwent complete surgical correction of ToF without intra-operative complications, and were transferred post-operatively to the ICU where echocardiography demonstrated restrictive RV physiology, with the characteristic diastolic pulmonary arterial flow coincident with atrial systole, in all of them. Conventional cardiorespiratory support was initially continued, with modest inotropic therapy (4–8 μg/kg per min dopamine) and conservative levels of intermittent positive pressure ventilation (IPPV).

However, the three patients developed clinical features of a severe low-output state despite increasing doses of inotropes (dopamine 12–16 μg/kg per min; enoximone 10–15 μg/kg per min), with oliguria, metabolic acidosis and hypotension, in the face of relatively high central venous pressures (Table 1). At this time all patients were still fully ventilated on IPPV, with a mean airway pressure of 9–12, with a PEEP of 3–5. The FIO_2 in patients 1 and 2 was 0.5, and in patient 3 was 1.0. In view of their poor haemodynamic statuses, we did not anticipate early ventilatory weaning and so commenced cuirass NPV in the hope of improving cardiac output by preserving or augmenting diastolic pulmonary blood flow. NPV was delivered using the Hayek Oscillator (Breasy Medical, London) as previously described [3, 4, 7]. In patients 1 and 2, regular measurements of cardiac output were made using the direct Fick method (oxygen consumption being measured using respiratory mass spectrometry). Patients 1 and 2 were commenced on NPV at a relatively early stage (20 and 6 h after ICU admission, respectively), while patient 3 was started 42 h post-operatively. This child had a significantly increased alveolar-arterial gradient by the time NPV was commenced, and although the FIO_2 was reduced from 1.0 to 0.65 soon after starting NPV, inhaled nitric oxide (iNO) was added 24 h later at a maximum of 5 parts per million (ppm), with the aim of further improving arterial oxygenation.

Results

Effective ventilation was achieved without difficulty using NPV, and the application of NPV was not associated with any clinical or haemodynamic instability. All patients had marked clinical improvement in terms of blood pressure, urine output and metabolic acidosis during the treatment period (Table 1, Fig. 1). No patient required renal replacement therapy or extracorporeal life support. In patient 3 the addition of iNO allowed further reduction of the FIO_2 to 0.35. In patients 1 and 2 NPV rapidly improved the cardiac output by over 100 % (Table 1, Fig. 1). In all cases NPV was discontinued and IPPV recommenced when it was felt that haemodynamic improvement and stability had been achieved (7–78 h). Haemodynamic stability was sustained after re-institution of IPPV in all cases. All patients were subsequently extubated within 72 h, and were discharged home within 2 weeks.

Discussion

Diastolic ventricular dysfunction is becoming increasingly recognised as a cause of haemodynamic disturbance in a variety of groups of intensive care patients. On the paediatric cardiac ICU, patients with a low output secondary to diastolic RV dysfunction after ToF repair can present us with a difficult management challenge. Although the duration of the low-output state itself is typically relatively short-lived, the intensive care stay of these patients can be further prolonged [2] for other reasons: first, by the 'by-products' of our more conventional attempts at achieving adequate haemodynamics – tachycardia and vasoconstriction produced by high dose inotropes and extravascular fluid accumulation, which results from colloid administration – and, second, the complications of a long ICU stay – most commonly ventilator associated pneumonia and other infections. In these patients, by replacing IPPV with NPV, ideally early in their post-operative ICU course, this vicious cycle can be broken as illustrated in the three patients here.

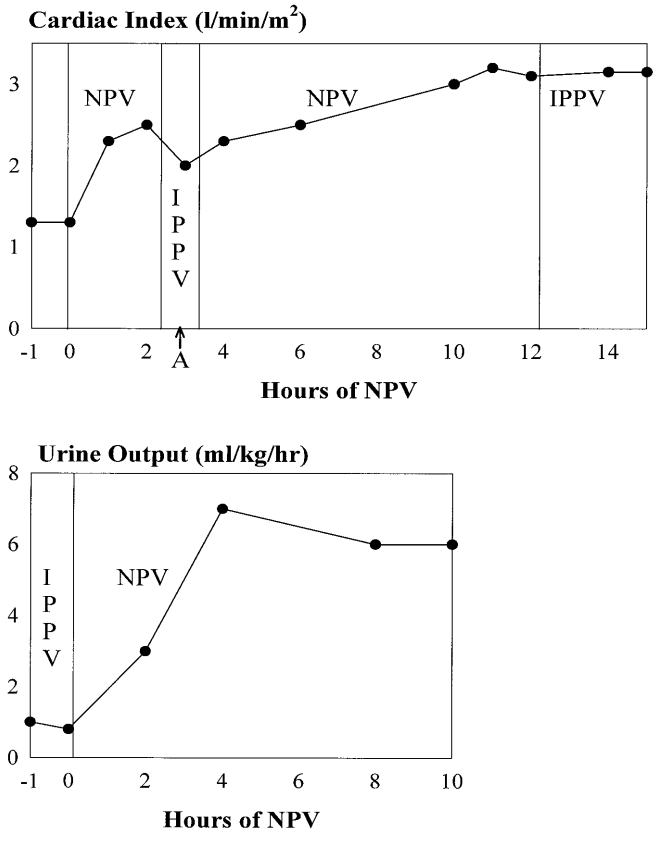

Fig. 1 Clinical data from two patients receiving therapeutic NPV. In patient 2 (*above*) the cardiac index is shown prior to starting NPV, and at intervals during treatment. There was a dramatic improvement which was attenuated when NPV was briefly stopped at 3 h (A) for wound inspection and endotracheal suction. When a period of stability had been achieved, NPV was discontinued and IPPV re-started, and the haemodynamic improvement was maintained. In patient 3 (*below*), the urine output is shown prior to, and during, the first 10 h of therapy with NPV. Before commencing NPV, the patient had been receiving an intravenous infusion of frusemide for several hours. This was discontinued after 4 h of NPV, but the marked diuresis which accompanied the onset of NPV continued

There may be a few concerns regarding the 'safety' (or appropriateness) of cuirass NPV in patients following open heart surgery. The children in this group had median sternotomies and had two or more chest drains present at the time of therapy, and would all have had oedematous, and therefore potentially friable, chest wall tissue. In addition, an increased bleeding tendency is an inevitable consequence of the abnormal platelet function and increased fibrinolysis following heparinisation and cardiopulmonary bypass [8]. The children presented here did not experience any increase in chest drain losses or any localised bleeding at points of contact between the cuirass and the chest wall during the period of NPV. The cuirass was briefly removed (and IPPV re-commenced) at 2–4 hourly intervals for skin care and for routine chest physiotherapy with endotracheal suction. This manoeuvre also periodically relieved the skin from contact with the cuirass. Although other investigators have raised concerns that prolonged use of cuirass NPV could potentially cause skin defects which may limit the duration of therapy [5], we did not encounter any such problems.

Negative pressure ventilation was successfully used as an adjunctive haemodynamic tool in three children with low-output states following ToF repair. By overcoming the adverse effects of IPPV on pulmonary blood flow, which can be so detrimental in the presence of diastolic RV dysfunction, the application of NPV resulted in marked clinical improvement in all cases.

References

1. Meliones JN, Custer JR, Snedecor S, Moler FW, O'Rourke P, Delius R (1991) Extracorporeal life support for cardiac assist in pediatric patients. Review of ELSO registry data. Circulation 84 (suppl III): III-168–III-172
2. Cullen S, Shore D, Redington A (1995) Characterization of right ventricular diastolic performance after complete repair of tetralogy of Fallot: restrictive physiology predicts slow postoperative recovery. Circulation 91: 1782–1789
3. Shekerdemian LS, Bush A, Shore DF, Lincoln C, Redington AN (1999) Cardiorespiratory responses to negative pressure ventilation after tetralogy of Fallot repair: a hemodynamic tool for patients with a low-output state. J Am Coll Cardiol 33 (2): 549–555
4. Shekerdemian LS, Shore DF, Lincoln C, Bush A, Redington AN (1996) Negative-pressure ventilation improves cardiac output after right heart surgery. Circulation 94 (suppl II): II49-II55
5. Scholz SE, Knothe C, Thiel A, Hempelmann G (1997) Improved oxygen delivery by positive pressure ventilation with continuous negative external chest pressure. Lancet 349: 1295–1296
6. Sideno B, Vaage J (1997) Ventilation by external high-frequency oscillations improves cardiac function after coronary artery bypass grafting. Eur J Cardiothorac Surg 11 (2): 248–257

7. Shekerdemian LS, Bush A, Lincoln C, Shore DF, Petros AJ, Redington AN (1997) Cardiopulmonary interactions in healthy children and children after simple cardiac surgery: the effects of positive and negative pressure ventilation. Heart 78 (6): 587–593

8. Khuri SF, Valeri CR, Loscalzo J, Weinstein MJ, Birjiniuk V, Healey NA, MacGregor H, Doursounian M, Zolkewitz MA (1995) Heparin causes platelet dysfunction and induces fibrinolysis before cardiopulmonary bypass. Ann Thorac Surg 60 (4): 1008–1014