

D. De Backer  
P. El Haddad  
J.-C. Preiser  
J.-L. Vincent

## Hemodynamic responses to successful weaning from mechanical ventilation after cardiovascular surgery

Received: 14 June 1999  
Final revision received: 18 April 2000  
Accepted: 26 April 2000

D. De Backer · P. El Haddad · J.-C. Preiser ·  
J.-L. Vincent (✉)  
Department of Intensive Care,  
Free University of Brussels,  
Erasme Hospital, Route de Lennik 808,  
1070 Brussels, Belgium  
e-mail: jlvincen@ulb.ac.be  
Tel.: + 32-2-555 33 80  
Fax: + 32-2-555 45 55

**Abstract** Weaning from mechanical ventilation is usually associated with an increase in oxygen consumption ( $\text{VO}_2$ ), which may stress the cardiovascular system. We studied relative changes in the cardiac index and oxygen extraction ratio ( $\text{EO}_2$ ) during successful weaning in patients after cardiac surgery ( $n = 52$ ), cardiac transplantation ( $n = 17$ ), or abdominal aortic surgery ( $n = 11$ ). Cardiac index was determined by the thermodilution technique and arterial and mixed venous blood gases were obtained before and 30 min after the start of weaning through a T-piece. The cardiovascular changes were evaluated in 42 patients in whom  $\text{VO}_2$  (calculated by Fick's equation) increased by more than 10%. Cardiac index increased more after abdominal

aortic surgery (from  $3.27 \pm 0.77$  to  $4.44 \pm 0.58 \text{ l min}^{-1} \text{ m}^{-2}$ ,  $p < 0.01$ ) than after cardiac surgery (from  $2.53 \pm 0.59$  to  $2.87 \pm 0.46 \text{ l min}^{-1} \text{ m}^{-2}$ ,  $p < 0.01$ ) or cardiac transplantation (from  $2.99 \pm 0.64$  to  $3.33 \pm 0.74 \text{ l min}^{-1} \text{ m}^{-2}$ ,  $p < 0.05$ ).  $\text{EO}_2$  remained stable in patients after aortic surgery (from  $25.9 \pm 7.1$  to  $25.2 \pm 5.6\%$ , NS) but increased slightly after cardiac surgery (from  $33.3 \pm 6.1$  to  $37.3 \pm 6.4\%$ , NS) and significantly after cardiac transplantation (from  $25.8 \pm 4.1$  to  $28.2 \pm 4.0\%$ ,  $p < 0.05$ ). Hence the cardiovascular response to weaning from mechanical ventilation may vary according to the type of surgery.

**Key words** Cardiac surgery · Aortic surgery · Cardiac output · Oxygen consumption · Oxygen extraction

### Introduction

Weaning from mechanical ventilation is generally associated with an increase in oxygen requirements related to the increased work of breathing [1, 2] and also sometimes to a rise in catecholamine levels [3, 4, 5]. Jubran et al. [6] recently reported in a mixed group of critically ill patients that failure in weaning from mechanical ventilation was associated with an impaired cardiac response. This was reflected by increased oxygen extraction in these patients while oxygen extraction decreased and cardiac index increased in the patients successfully weaned from mechanical ventilation. However, even in patients in whom weaning was successful, the increased oxygen consumption ( $\text{VO}_2$ ) may represent a stress to

the heart, which must increase its output. The increase in loading conditions may further jeopardize the myocardial oxygen balance, exacerbate myocardial ischemia [7, 8, 9], and lead to anaerobic myocardial metabolism [8]. Weaning can result in left [10, 11] and right [12, 13] ventricular dysfunction.

Therefore weaning from mechanical ventilation is a particularly delicate period for patients with compromised cardiac function. However, the overall hemodynamic response to weaning from mechanical ventilation after surgical procedures has not been adequately studied. Since the hemodynamic response may also differ according to the type of surgery, we compared the cardiovascular response to the increase in  $\text{VO}_2$  induced by weaning from mechanical ventilation in patients after

two types of cardiac surgery (cardiac surgery and cardiac transplantation) and after subdiaphragmatic aortic surgery. We hypothesized that the increase in cardiac index would be lower after cardiac surgery than after subdiaphragmatic aortic surgery.

## Patients and methods

The study included 80 patients (age  $62 \pm 16$  years) in whom we prospectively collected hemodynamic data during weaning from mechanical ventilation for less than 24 h after cardiac surgery ( $n = 52$ ), cardiac transplantation ( $n = 17$ ), or abdominal aortic surgery ( $n = 11$ ; Table 1). Since these maneuvers were part of our routine management, informed consent was waived by the institutional ethics committee. Only patients with an uncomplicated course in whom weaning was successful were included. All patients were routinely monitored during the surgical procedure by an arterial catheter and a pulmonary artery catheter (Swan-Ganz catheter 7 F, Baxter-Healthcare, Irvine, Calif., USA). During cardiac surgery (including heart transplantation), anesthesia consisted of high-dose fentanyl or sufentanil and midazolam; postoperative sedation was provided with a continuous midazolam infusion. During aortic surgery anesthesia consisted of variable doses of alfentanil associated with midazolam and low concentrations of inhaled isoflurane; postoperative sedation and analgesia were provided with continuous midazolam and morphine infusions associated with intermittent paracetamol injections. No patient received epidural anesthesia. In the three groups all drug infusions were stopped at least 6 h before starting the weaning procedure.

Intravenous fluids consisted of crystalloids, synthetic (hydroxyethylstarch solution; Fresenius, Germany) and natural (fresh-frozen plasma and albumin solution; Belgian Red Cross) colloids. Red blood cells were transfused to maintain a hemoglobin level above 9 g/dl. Vasoactive drugs were administered according to routine protocol: dobutamine, isoprenaline, and sodium nitroprusside are the preferred agents for inotropic, chronotropic, and vasodilating effects, respectively. After cardiac surgery 42 patients were treated with dobutamine at a mean dose of  $6 \pm 6 \mu\text{g kg}^{-1} \text{min}^{-1}$ . After cardiac transplantation 13 patients were treated with dobutamine at a mean dose of  $6 \pm 5 \mu\text{g kg}^{-1} \text{min}^{-1}$  and 15 with isoprenaline at a mean dose of  $0.05 \pm 0.03 \mu\text{g min}^{-1}$ . After vascular surgery all patients were treated with sodium nitroprusside at a mean dose of  $80 \pm 60 \mu\text{g min}^{-1}$ .

Mechanical ventilation was provided on an assist/control mode (Siemens Elema 900 C, Solna, Sweden) using tidal volumes of 8–12 ml/kg and a respiratory rate adapted to maintain arterial carbon dioxide pressure between 30 and 40 mmHg and/or a pH between 7.30 and 7.50. Positive end-expiratory pressure did not exceed 5 cmH<sub>2</sub>O. When the patient was normothermic, awake, and in a stable hemodynamic state, weaning from the ventilator was begun with a T-piece with a fractional inspiratory oxygen of 0.4 or 0.5. No change in therapy was allowed during the procedure.

Complete hemodynamic measurements, obtained less than 30 min before and 30 min after the beginning of the weaning process, included measurements of intravascular pressure at end-expiration and cardiac index determination by the thermodilution technique (COM1 or COM2, Baxter Healthcare). Cardiac index was averaged from five successive injections of cold ( $< 10^\circ\text{C}$ ) D5W during the expiratory phase. Arterial and mixed venous blood samples were simultaneously withdrawn for measurements of blood gases (ABL 500, Radiometer, Copenhagen, Denmark), oxygen saturations and hemoglobin concentration (Hemoximeter

**Table 1** Clinical data

	<i>n</i>	Age (years)
Cardiac surgery	52	$66 \pm 9$
Cardiac artery bypass graft	29	
Valve replacement	18	
Combined	5	
Cardiac transplantation	17	$47 \pm 7$
Heart	14	
Heart-lung	3	
Abdominal aortic surgery	11	$68 \pm 6$

OSM3, Radiometer). Arterial blood lactate concentration was determined by an enzymatic technique (automated analyzer Hitachi, Tokyo, Japan).

Oxygen delivery ( $\text{DO}_2$ ), oxygen consumption ( $\text{VO}_2$ ), and oxygen extraction ( $\text{EO}_2$ ) were calculated as follows:

$$\text{DO}_2 \text{ (ml min}^{-1} \text{ m}^{-2}\text{)} = 10 \times \text{CI} \times \text{CaO}_2$$

$$\text{VO}_2 \text{ (ml min}^{-1} \text{ m}^{-2}\text{)} = 10 \times \text{CI} \times (\text{CaO}_2 - \text{CvO}_2)$$

$$\text{EO}_2 \text{ (\%)} = (\text{CaO}_2 - \text{CvO}_2) / \text{CaO}_2$$

where  $\text{CaO}_2$  and  $\text{CvO}_2$  represent oxygen contents of the arterial and mixed venous blood, respectively, and CI is cardiac index.

Systemic and pulmonary vascular resistance, stroke index, and left and right ventricular stroke work indices were calculated using standard formulas.

Statistical analysis was performed using one-way analysis of variance for repeated measurements with post hoc Newman-Keuls adjustment. A *p* value less than 0.05 was considered statistically significant. All values are reported as mean  $\pm$  SD.

## Results

Weaning and extubation were uneventful in all patients. No patient had any signs of myocardial ischemia. Since the  $\text{VO}_2$  response to weaning was quite variable, patients were separated into three groups according to the changes in  $\text{VO}_2$ : increased by at least 10% ( $n = 42$ ); changed by less than 10% ( $n = 34$ ); or decreased by at least 10% ( $n = 4$ ). There was no significant difference at baseline between the three groups except that baseline  $\text{VO}_2$  was significantly lower in patients whose  $\text{VO}_2$  increased than in other patients (Table 2). There was also a significant inverse correlation between the changes in  $\text{VO}_2$  ( $\Delta\text{VO}_2$ ) and basal  $\text{VO}_2$  (Fig. 1;  $\Delta\text{VO}_2 = 71.4 - 0.47 \text{VO}_2$ ,  $r = 0.47$ ,  $p < 0.0001$ ).  $\text{VO}_2$  increased by at least 10% in 24 of 52 patients after cardiac surgery, in 8 of 17 after cardiac transplantation, and in 10 of 11 after vascular surgery. In patients whose  $\text{VO}_2$  did not increase ( $n = 38$ ) there were no significant changes in cardiac index or  $\text{DO}_2$  (data not shown).

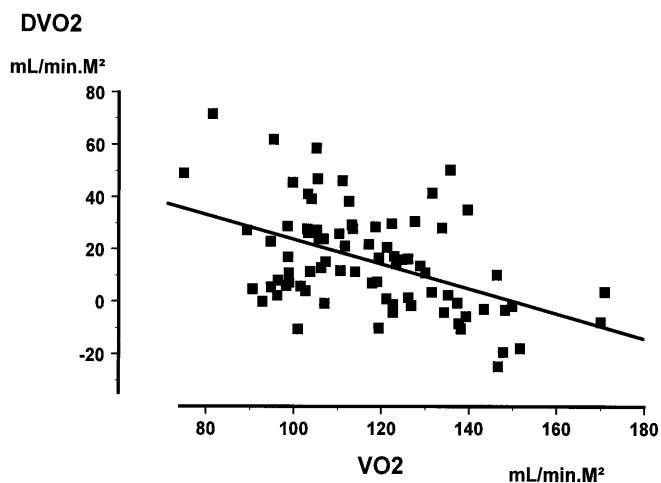
We further studied the 42 patients in whom  $\text{VO}_2$  increased. Heart rate increased significantly in aortic surgery patients but remained stable in the other patients (Table 3). Mean arterial pressure, pulmonary artery

**Table 2** Baseline characteristics in oxygen consumption ( $VO_2$ ) responses after cardiac surgery (*MAP* mean arterial pressure, *MPAP* mean pulmonary artery pressure, *PAOP* pulmonary artery occluded pressure, *RAP* right atrial pressure)

	$VO_2$ increased > 10% ( <i>n</i> = 25)	$VO_2$ unchanged ( <i>n</i> = 24)	$VO_2$ decreased > 10% ( <i>n</i> = 3)
Heart rate (bpm)	92 ± 15	89 ± 15	101 ± 14
MAP (mmHg)	78 ± 11	79 ± 12	80 ± 10
MPAP (mmHg)	17 ± 5	19 ± 6	24 ± 2
PAOP (mmHg)	9 ± 4	10 ± 5	13 ± 6
RAP (mmHg)	8 ± 3	9 ± 4	10 ± 3
Cardiac index ( $l \text{ min}^{-1} \text{ m}^{-2}$ )	2.54 ± 0.59	2.75 ± 0.63	2.61 ± 0.78
$VO_2$ ( $ml \text{ min}^{-1} \text{ m}^{-2}$ )	109 ± 13	125 ± 25*	133 ± 28**
$\Delta VO_2$ ( $ml \text{ min}^{-1} \text{ m}^{-2}$ )	27 ± 16	1 ± 5	-12 ± 1
Lactate (mEq/l)	2.9 ± 1.6	2.9 ± 1.3	3.1 ± 2.1
Cardiopulmonary bypass time (min)	118 ± 24	117 ± 21	134 ± 33
Dobutamine dose ( $\mu g \text{ kg}^{-1} \text{ min}^{-1}$ )	7 ± 6	6 ± 5	5 ± 5

\*  $p < 0.05$  and \*\* $p < 0.01$  vs. patients who increased  $VO_2$  by more than 10%

pressure, and cardiac filling pressure increased (Table 3). Cardiac index, stroke index, left and right ventricular stroke work indices increased significantly in the three groups, but especially after aortic surgery (Table 3). In the three groups arterial pH and arterial oxygen pressure decreased and arterial carbon dioxide pressure increased.  $DO_2$  increased in all groups, but especially after aortic surgery (Table 3). Mixed venous oxygen saturation remained stable after aortic surgery but decreased significantly after cardiac surgery, including cardiac transplantation.  $EO_2$  increased after cardiac surgery and cardiac transplantation but remained stable after aortic surgery. The relationship between the cardiac index and  $EO_2$  illustrated a different pattern; both increased after cardiac surgery, including cardiac transplantation, but only the cardiac index increased after aortic surgery (Fig. 2).



**Fig. 1** Relationship between changes in  $VO_2$  ( $\Delta VO_2$ ) and  $VO_2$  at baseline ( $VO_2$ ) for the entire study population.  $\Delta VO_2 = 71.4 - 0.47 VO_2$ ,  $r = 0.47$ ,  $p < 0.0001$

## Discussion

The present study emphasizes the variability in response to weaning from mechanical ventilation after cardiac and vascular surgery. The weaning procedure in each patient was standardized, consisting in a shift from assist/control volume ventilation to spontaneous breathing through a T-piece, the measurements taken at identical time intervals in each patient, without any other intervention or change in drug therapy. Patients with respiratory complications or unsuccessful weaning episodes were not included, as this was not the aim of our study, and has already been described by Jubran et al. [6]. In this study  $VO_2$  was determined using Fick's equation rather than by indirect calorimetry since changes in ventilation alter  $CO_2$  stores and prevent the maintenance of a steady state. Since the errors in calculating  $VO_2$  from Fick's equation are between 5% and 10% [14, 15], we considered that changes in  $VO_2$  greater than 10% would represent true changes in oxygen demand. Using this cutoff limit,  $VO_2$  increased in one-half of the total patients, but in almost all patients after aortic surgery.

Previous studies have reported a variable course of  $VO_2$  after cardiac surgery [16, 20]. Wolff et al. [18] and Vuori and Klossner [17] observed no significant change in  $VO_2$  but did not report individual data. Räsänen et al. [19] observed various responses during weaning in children after cardiac surgery. Similarly, Beach et al. [20] observed an increase in  $VO_2$  in 8 but a decrease in 15 adult patients after cardiac surgery. Several factors may influence the  $VO_2$  response during weaning, including the type of ventilatory support before or even during weaning (e.g., intermittent mandatory ventilation or pressure support) [21] and the underlying respiratory function. Unfortunately, we did not determine the work of breathing in our study.

Interestingly,  $VO_2$  increased in the patients with the lowest  $VO_2$  at baseline. There was also an inverse rela-

**Table 3** Course of selected hemodynamic and blood gas parameters during weaning from mechanical ventilation in patients in whom  $\text{VO}_2$  increased (MAP mean arterial pressure, MPAP mean pulmonary artery pressure, PAOP pulmonary artery occluded pressure, RAP right atrial pressure, SVR systemic vascular resistance, PVR pulmonary vascular resistance, LVSWI left ventricular

stroke work index, RVSWI right ventricular stroke work index,  $\text{PaCO}_2$  arterial carbon dioxide pressure,  $\text{PaO}_2$  arterial oxygen pressure,  $\text{SaO}_2$  arterial oxygen saturation,  $\text{PvO}_2$  venous oxygen pressure,  $\text{SvO}_2$  mixed venous oxygen saturation,  $\text{DO}_2$  oxygen delivery,  $\text{VO}_2$  oxygen consumption,  $\text{EO}_2$  oxygen extraction ratio)

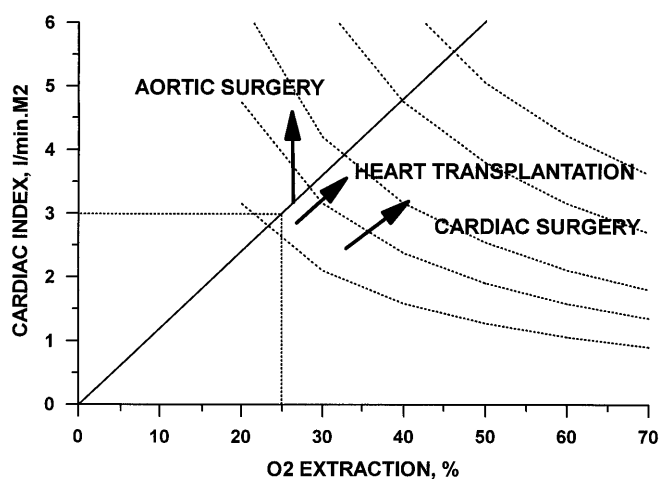
	Cardiac surgery		Cardiac transplantation		Aortic surgery	
	Mechanical ventilation	Spontaneous ventilation	Mechanical ventilation	Spontaneous ventilation	Mechanical ventilation	Spontaneous ventilation
Temperature ( $^{\circ}\text{C}$ )	36.8 $\pm$ 0.6	36.9 $\pm$ 0.5	36.8 $\pm$ 0.7	36.7 $\pm$ 0.7	37.0 $\pm$ 0.5	37.2 $\pm$ 0.6
Heart rate (bpm)	92 $\pm$ 15	92 $\pm$ 16	110 $\pm$ 8***	110 $\pm$ 5***	89 $\pm$ 26	99 $\pm$ 19*
MAP (mmHg)	78 $\pm$ 11	82 $\pm$ 9*	78 $\pm$ 15	89 $\pm$ 14	91 $\pm$ 16 <sup>5*</sup>	105 $\pm$ 9 <sup>5*</sup>
MPAP (mmHg)	17 $\pm$ 5	22 $\pm$ 6**	17 $\pm$ 4	20 $\pm$ 6	20 $\pm$ 6	24 $\pm$ 5
PAOP (mmHg)	9 $\pm$ 4	13 $\pm$ 6**	9 $\pm$ 2	10 $\pm$ 5	7 $\pm$ 2	11 $\pm$ 5*
RAP (mmHg)	8 $\pm$ 3	10 $\pm$ 5**	7 $\pm$ 2	9 $\pm$ 5	7 $\pm$ 2	9 $\pm$ 3*
Cardiac index ( $\text{l min}^{-1} \text{m}^{-2}$ )	2.53 $\pm$ 0.59	2.87 $\pm$ 0.46**	2.99 $\pm$ 0.64***	3.33 $\pm$ 0.74 <sup>4*</sup>	3.27 $\pm$ 0.77 <sup>4*</sup>	4.44 $\pm$ 0.58 <sup>4*</sup>
Stroke index ( $\text{ml/m}^{-2}$ )	28 $\pm$ 7	32 $\pm$ 6**	28 $\pm$ 7	30 $\pm$ 8*	38 $\pm$ 8 <sup>4*,5*</sup>	46 $\pm$ 7 <sup>4*,5*</sup>
SVR ( $\text{dyne s}^{-1} \text{cm}^{-5}$ )	1312 $\pm$ 137	1158 $\pm$ 273**	1061 $\pm$ 230	1156 $\pm$ 498	1205 $\pm$ 228	1002 $\pm$ 185*
PVR ( $\text{dyne s}^{-1} \text{cm}^{-5}$ )	156 $\pm$ 64	139 $\pm$ 50	140 $\pm$ 102	145 $\pm$ 84	183 $\pm$ 106	134 $\pm$ 42
LVSWI ( $\text{g m}^{-1} \text{m}^{-2}$ )	26 $\pm$ 7	30 $\pm$ 7**	27 $\pm$ 11	32 $\pm$ 6*	43 $\pm$ 12 <sup>4*,5*</sup>	59 $\pm$ 14 <sup>4*,5*</sup>
RVSWI ( $\text{g m}^{-1} \text{m}^{-2}$ )	3 $\pm$ 1	5 $\pm$ 2**	3 $\pm$ 2	4 $\pm$ 2*	6 $\pm$ 3 <sup>4*,5*</sup>	10 $\pm$ 3 <sup>4*,5*</sup>
Arterial pH	7.43 $\pm$ 0.05	7.40 $\pm$ 0.05**	7.42 $\pm$ 0.09	7.37 $\pm$ 0.09*	7.41 $\pm$ 0.04	7.36 $\pm$ 0.06**
$\text{PaCO}_2$ (mmHg)	34 $\pm$ 5	37 $\pm$ 4**	34 $\pm$ 5	41 $\pm$ 7*	34 $\pm$ 3	38 $\pm$ 5**
$\text{PaO}_2$ (mmHg)	98 $\pm$ 27	86 $\pm$ 23*	122 $\pm$ 31	107 $\pm$ 31	111 $\pm$ 32	91 $\pm$ 24
$\text{SaO}_2$ (%)	96.8 $\pm$ 1.6	95.3 $\pm$ 2.0**	96.9 $\pm$ 2.4	96.4 $\pm$ 2.6	97.4 $\pm$ 2.2	95.4 $\pm$ 2.3*
$\text{PvO}_2$ (mmHg)	47 $\pm$ 6	33 $\pm$ 4**	41 $\pm$ 3	40 $\pm$ 4	39 $\pm$ 5	40 $\pm$ 5
$\text{SvO}_2$ (%)	65.0 $\pm$ 5.4	60.2 $\pm$ 6.2**	73.0 $\pm$ 5.0 <sup>4*</sup>	70 $\pm$ 3.9 <sup>4*</sup>	73.0 $\pm$ 6.8 <sup>4*</sup>	72.1 $\pm$ 5.7 <sup>4*</sup>
$\text{DO}_2$ ( $\text{ml min}^{-1} \text{m}^{-2}$ )	335 $\pm$ 63	374 $\pm$ 54**	457 $\pm$ 78 <sup>4*</sup>	503 $\pm$ 80 <sup>4*</sup>	462 $\pm$ 132 <sup>4*</sup>	612 $\pm$ 142 <sup>4*</sup>
$\text{VO}_2$ ( $\text{ml min}^{-1} \text{m}^{-2}$ )	109 $\pm$ 5.8	137.3 $\pm$ 6.4**	116 $\pm$ 10	139 $\pm$ 9**	113 $\pm$ 19	150 $\pm$ 21**
$\text{EO}_2$ (%)	33.3 $\pm$ 6.1	37.3 $\pm$ 6.4	25.8 $\pm$ 4.1 <sup>4*</sup>	28.2 $\pm$ 4.0 <sup>4*</sup>	25.9 $\pm$ 7.1 <sup>4*</sup>	25.2 $\pm$ 5.6 <sup>4*</sup>

\*  $p < 0.05$ , \*\* $p < 0.01$  vs. mechanical ventilation; \*\*\* $p < 0.05$ , <sup>4\*</sup> $p < 0.01$  vs. cardiac surgery; <sup>5\*</sup> $p < 0.01$  aortic surgery vs. heart transplantation

tionship between the change in  $\text{VO}_2$ , and the  $\text{VO}_2$  during mechanical ventilation. All other parameters, including arterial blood gases, were similar at baseline. Similar observations have been reported by Beach et al. [20], but the number of patients in their study was too limited to achieve statistical significance. In the children studied by Räsänen et al. [19] during weaning from mechanical ventilation after cardiac surgery, the only significant differences between subgroups of children whose  $\text{VO}_2$  increased were baseline  $\text{VO}_2$ , body temperature, and spontaneous respiratory rate. The increase in  $\text{VO}_2$  during weaning from mechanical ventilation is associated with a stress response that can be reflected by the increase in catecholamine levels [3, 4, 5]. The higher  $\text{VO}_2$  at baseline in patients whose  $\text{VO}_2$  did not increase during weaning was probably related to a greater stress prior to weaning.

Although the increase in  $\text{VO}_2$  was similar after cardiac surgery, cardiac transplantation, and subdiaphragmatic aortic surgery, the cardiovascular response differed: the increase in  $\text{VO}_2$  was met by combined increases in cardiac index and oxygen extraction after cardiac surgery and transplantation but by an increase only in cardiac index after abdominal aortic surgery. This

means that either cardiac function was impaired in patients after cardiac surgery and transplantation, or that oxygen extraction was altered in patients after aortic surgery. Several factors could account for myocardial depression. First, patients after cardiac surgery may be more prone to develop ischemic complications as weaning may precipitate myocardial ischemia [7, 8, 9] or left ventricular failure [10, 11] and right ventricular dysfunction [12, 13]. However, patients undergoing abdominal aortic surgery often also have a compromised cardiovascular function. Nevertheless, none of the patients showed evidence of myocardial ischemia. Second, cardiopulmonary bypass with cardioplegia could result in myocardial stunning, accounting for a lower cardiac index after cardiac surgery and cardiac transplantation than after abdominal aortic surgery. Third, the anesthetic techniques differed. Larger doses of opiate analgesics were used after cardiac surgery and cardiac transplantation, but these drugs do not have significant myocardial depressant effects [22]. The different intravenous anesthetic agents administered during cardiac surgery and transplantation (fentanyl and sufentanil) and during abdominal aortic surgery (alfentanil) may have influenced the cardiovascular response. Weissman and colleagues



**Fig. 2** Relationship between cardiac index and oxygen extraction demonstrating that in aortic surgery the increase in  $\text{VO}_2$  was met only by an increase in cardiac index while for cardiac surgery and cardiac transplantation it was due to combined increases in cardiac index and oxygen extraction. *Dotted lines* Lines of identical  $\text{VO}_2$  for any given hemoglobin level; *oblique line* proportional increases in cardiac index and oxygen extraction

[23, 24] studied the effects of these agents on cardiovascular response after an increase in  $\text{VO}_2$  induced by chest physical therapy in postoperative patients. They reported that fentanyl administration does not affect the increase in  $\text{VO}_2$  but does limit the increase in cardiac index [23], whereas alfentanil does not limit the increase in cardiac index despite similar effects on blood pressure [24]. Nevertheless, these differences were probably minimized in our study, as the patients were fully awake at the time of weaning.

An alternative explanation is that a defect in extraction capabilities could occur in patients after aortic surgery. During exercise healthy volunteers increase  $\text{VO}_2$  by combined increases in cardiac index and oxygen extraction [25, 26] whereas patients with overt heart failure increase  $\text{VO}_2$  by predominant increases in oxygen extraction [27]. The patients whom we studied after cardiac surgery and transplantation had a similar cardio-

vascular response as healthy volunteers, suggesting that although myocardial function could have been depressed, overall cardiac function was sufficiently preserved. Patients studied after aortic surgery also demonstrated an increase in cardiac index but not in oxygen extraction, perhaps reflecting a defect in peripheral extraction capabilities. Such a pattern is typically observed in inflammatory states, including sepsis. Abdominal aortic surgery also elicits a major inflammatory response [28, 29] which can be associated with a decreased afterload, facilitating the increase in cardiac output but simultaneously reducing oxygen extraction capabilities.

It is also possible that incision pain, by limiting abdominal or thoracic movement, induced different changes in abdominal and thoracic pressures between those patients undergoing cardiac surgery and those with subdiaphragmatic aortic surgery. This in turn may have led to altered blood flow distribution and hence oxygen extraction capabilities between the groups.

Finally, differences in the vasoactive drug regimen could have influenced the cardiovascular response [30]. Almost all patients after cardiac surgery and cardiac transplantation were treated with inotropic support with dobutamine or isoprenaline, while patients after abdominal aortic surgery were all treated with sodium nitroprusside without  $\beta$ -adrenergic agents. We felt it unethical to alter this vasoactive regimen to evaluate its influence on the hemodynamic response to weaning.

In conclusion,  $\text{VO}_2$  increased significantly during successful weaning from mechanical ventilation in one-half of the patients whom we studied after cardiac surgery. The increase in  $\text{VO}_2$  was met by combined increases in cardiac index and oxygen extraction after cardiac surgery and cardiac transplantation, and by an increase only in cardiac index in patients after abdominal aortic surgery. No significant differences were observed in the cardiovascular response between cardiac transplantation and other forms of cardiac surgery. The present study shows that the hemodynamic response to weaning from mechanical ventilation can differ according to the type of surgery, and helps in interpreting cardiac index and mixed venous oxygen saturation during this period.

## References

1. Kemper M, Weissman C, Askanasi J, Hyman AL, Kinney JM (1987) Metabolic and respiratory changes during weaning from mechanical ventilation. *Chest* 92: 979-983
2. Pinsky MR (1994) Cardiovascular effects of ventilatory support and withdrawal. *Anesth Analg* 79: 567-576
3. Skillmann JJ, Malhotra IV, Palotta JA, Bushnell LS (1971) Determinants of weaning from controlled ventilation. *Surg Forum* 22: 198-200
4. Kennedy SK, Weintraub RM, Skillmann JJ (1977) Cardiorespiratory and sympathoadrenal responses during weaning from controlled ventilation. *Surgery* 82: 233-240
5. Oh TE, Bhatt S, Lin ES, Hutchinson RC, Low JM (1991) Plasma catecholamines and oxygen consumption during weaning from mechanical ventilation. *Intensive Care Med* 17: 199-203

6. Jubran A, Mathru M, Dries DJ, Tobin MJ (1999) Continuous recordings of mixed venous oxygen saturation during weaning from mechanical ventilation and the ramifications thereof. *Am J Respir Crit Care Med* 158: 1763–1769
7. Abalos A, Leibowitz AB, Distefano D, Halpern N, Iberti TJ (1992) Myocardial ischemia during the weaning period. *Am J Crit Care* 3: 32–36
8. Elia S, Liu P, Hilgenberg A, Skourtis C, Lappas D (1991) Coronary haemodynamics and myocardial metabolism during weaning from mechanical ventilation in cardiac surgical patients. *Can J Anaesth* 38: 564–571
9. Chatila W, Ani S, Guaglianone D, Jacob B, Amoateng-Adjepong Y, Manthous CA (1996) Cardiac ischemia during weaning from mechanical ventilation. *Chest* 109: 1577–1583
10. Lemaire F, Teboul JL, Cinotti L (1988) Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology* 69: 171–179
11. Richard C, Teboul JL, Archambaud F, Hebert JL, Michaut P, Auzepy P (1994) Left ventricular function during weaning of patients with chronic obstructive pulmonary disease. *Intensive Care Med* 20: 181–186
12. Bastien O, Durand PG, George M, Gurbala A, Estanove S (1988) Evolution of right ventricular performance after CABG. *Intensive Care Med* 14: 499–502
13. Bizouarn P, Blanloeil Y, Billaud-Debarre C (1997) Right ventricular function during weaning from mechanical ventilation after coronary artery bypass grafting: effect of volume loading. *Intensive Care Med* 23: 1231–1236
14. Thys D (1988) Cardiac output. *Anesthesiol Clin North Am* 6: 803–824
15. De Backer D, Moraine JJ, Berré J, Kahn RJ, Vincent JL (1994) Effects of dobutamine on oxygen consumption in septic patients: Direct vs indirect determinations. *Am J Respir Crit Care Med* 150: 95–100
16. Vuori A, Jalonen J, Laaksonen V (1979) Continuous positive airway pressure during mechanical and spontaneous ventilation: effects on central haemodynamics and oxygen transport. *Acta Anaesthesiol Scand* 23: 453–461
17. Vuori A, Klossner J (1981) Central haemodynamics and oxygen transport during CPAP with and without mandatory ventilations. *Acta Anaesthesiol Scand* 25: 282–285
18. Wolff G, Grädel E (1975) Haemodynamic performance and weaning from mechanical ventilation following open-heart surgery. *Eur J Intensive Care Med* 1: 99–104
19. Räsänen J, Puhakka K, Leijala M (1992) Spontaneous breathing and total body oxygen consumption in children recovering from open-heart surgery. *Chest* 101: 662–667
20. Beach T, Millen E, Grenvik A (1973) Hemodynamic response to discontinuance of mechanical ventilation. *Crit Care Med* 1: 85–90
21. Hörmann C, Baum M, Luz G, Putensen C, Putz G (1992) Tidal volume, breathing frequency and oxygen consumption at different pressure support levels in early stage of weaning in patients with chronic obstructive pulmonary disease. *Intensive Care Med* 18: 226–230
22. Prakash O, Verdow PD, de Jong JW, et al (1980) Haemodynamic and biochemical variables after induction of anaesthesia with fentanyl and nitrous oxide in patients undergoing coronary artery by-pass. *Can Anaesth Soc J* 27: 223–229
23. Klein P, Kemper M, Weissman C, Rosenbaum SH, Askanazi J, Hyman AL (1988) Attenuation of hemodynamic response to chest physical therapy. *Chest* 93: 38–42
24. Harding J, Kemper M, Weissman C (1993) Alfentanil attenuates the cardiopulmonary response of critically ill patients to an acute increase in oxygen demand induced by chest physiotherapy. *Anesth Analg* 77: 1122–1129
25. Silance PG, Simon C, Vincent JL (1994) The relation between cardiac index and oxygen extraction in acutely ill patients. *Chest* 105: 1190–1197
26. Sutton JR, Reeves JT, Wagner PD, et al (1988) Operation Everest II: Oxygen transport during exercise at extreme simulated altitude. *J Appl Physiol* 64: 1309–1321
27. Weber KT, Kinasevitz GT, Janicki JS, Fishman AP (1982) Oxygen utilization and ventilation during exercise in patients with chronic cardiac failure. *Circulation* 65: 1213–1223
28. Roumen RM, Hendriks T, Van der Ven-Jongekrijck J, et al (1993) Cytokine patterns in patients after major vascular surgery, hemorrhagic shock, and severe blunt trauma: relation with subsequent adult respiratory distress syndrome and multiple organ failure. *Ann Surg* 218: 769–776
29. Froom AH, Greve JW, Van der Linden CJ, Buurman WA (1996) Increased concentrations of cytokines and adhesion molecules in patients after repair of abdominal aortic aneurysm. *Eur Respir J* 162: 287–296
30. Fernandez-Mondéjar E, Vasquez-Mata G, de la Higuera Torres-Puchol J, Fornieles-Pérez H, Torres-Ruiz JM (1990) Cardiac index and oxygen consumption during weaning in patients who need inotropic support. *Intensive Care Med* 16: 137–139