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Breathing as exercise:

The cardiovascular response to weaning from mechanical ventilation

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It is axiomatic that exercise increases oxygen (O_2) demand if within the bounds of cardiovascular reserve, results in a proportional increase in whole body oxygen consumption (VO_2). Exercise physiologists have long known that it does not really matter what form of exercise is performed, VO_2 will increase in response to all forms of exercise, although the degree to which the increased VO_2 demand is met by increasing cardiac output and O_2 extraction varies with conditioning and underlying disease [1]. Interestingly, the type of exercise also influences the efficiency of the performance, defined as the ratio of external work performed to VO_2 [2]. The reasons for this discrepancy probably reflect motor efficiency and vascular conditioning as much as cardiac reserve. Thus, some subjects with prior training in a specific event will increase their cardiac outputs more during that stress than less conditioned subjects. These other less conditioned subjects may not increase cardiac output as much and will support the increased O_2 demand by increasing O_2 extraction across the capillary beds.

Using this paradigm, DeBacker and colleagues demonstrated that all ventilator-dependent patients following cardiovascular surgery who were challenged by weaning to spontaneous ventilation and in whom weaning was successful demonstrated markedly different car-

diac output and O_2 extraction responses, depending on their underlying pathology [3]. Those subjects with primarily peripheral vascular disease (vascular surgery patients) responded to this challenge with an increase in cardiac output, whereas those subjects with progressively lesser levels of cardiac responsiveness increased their O_2 extract more. Importantly, it did not matter why cardiac output responsiveness was limited, the overall response was predictable. Thus, subjects with normal autonomic tone and cardiovascular responsiveness (status – post coronary artery surgery) increased their cardiac output more than subjects with denervated hearts (cardiac transplant recipients) whose chronotropic response is usually blunted. These data are interesting to the practicing clinician for several reasons.

First, they underscore the obvious fact that spontaneous breathing is exercise. This statement alone needs to be heard. Numerous studies before this present one have documented that the transition from mechanical ventilation to spontaneous ventilation induces increased cardiac stress that can induce myocardial ischemia in subjects with coronary artery disease [4, 5, 6, 7]. Similarly, the act of spontaneous inspiratory efforts can precipitate left ventricular (LV) dilation [8] and cardiogenic shock [9] in subjects without coronary artery disease. Importantly, clinically relevant cardiovascular improvements during weaning often occur in these patients only if inotropic agents are given [10] or aggressive diuresis reverses cor pulmonale [9]. Thus, clinicians need to appreciate that spontaneous ventilation can place an excessive cardiovascular burden on the heart, induce myocardial ischemia and promote cardiogenic shock. Simple clinical observation and a review of the large clinical literature on this subject adequately document this point.

Second, how cardiovascular stress is manifest in a given patient during weaning is as much a function of their cardiovascular reserves and conditioning as it is the level of metabolic load imposed by the weaning tri-

al. Jabran et al. [11] demonstrated recently that although all patients with chronic obstructive lung disease increased their cardiac outputs during weaning trials, those who failed to wean also increased their arteriovenous O₂ saturation difference, demonstrating that patients who fail to wean do so as part of cardiovascular decompensation. Thus, occult cardiovascular insufficiency occurs during weaning failure in critically ill patients and may be the primary cause of weaning failures in many patients.

Given that these statements are true and relevant to clinical practice, where should we go from here? For starters, it is incumbent on the bedside clinician to be able to identify the etiology of cardiovascular failure. Dynamic hyperinflation-induced cor pulmonale or tamponade can cause weaning failure [12]. Acute LV failure associated with LV enlargement and pulmonary edema also occurs during weaning trials [13]. Though both these scenarios will present with an increasing arteriovenous O₂ gradient, the management of each is quite different. Hyperinflation is usually treated with maneuvers tending to prolong expiratory time and match extrinsic positive end-expiratory pressure (PEEP) to the dynamic hyperinflation-induced intrinsic PEEP [14]. Whereas, acute LV failure is best treated with inotropic agents and therapies aimed at minimizing the negative swings in intrathoracic pressure during spontaneous inspiration [15]. Thus, accurate diagnosis and management of cardiovascular insufficiency during weaning trials requires careful attention to both the underlying pulmonary dysfunction and the overall cardiovascular response, not just the change in VO₂.

Furthermore, since cardiovascular insufficiency is commonplace in subjects who fail to wean from mechanical ventilation, reliable bedside monitoring tools and tests need to be developed to aid the clinician in titrating cardiovascular therapies during weaning trials. One recent technique described by Mechard et al. [16] uses arterial pulse pressure variation during positive-pressure breath as a cardiovascular forcing function to identify preload-responsive states. Since arterial pulse pressure variation approximates LV stroke volume vari-

ation from beat to beat, these authors reasoned that increasing arterial pulse pressure variation would reflect positive-pressure ventilation-induced phasic decreases in venous return. Thus, the greater the pulse pressure variation, the more preload-dependent would be the subject. They demonstrated that arterial pulse pressure variation predicted the subsequent decrease in cardiac output as PEEP was increased by 10 cmH₂O. Similarly, the decreases in pulmonary artery diastolic pressure, and presumably arterial diastolic pressure, can be used to monitor the spontaneous ventilation-induced decreases in intrathoracic pressure [17]. This measure would allow the bedside clinician to estimate the work-cost of breathing, an important determinant of the increased VO₂ during weaning.

Thus, although some bedside tools are presently available to monitor cardiac vascular status, the list of such tools is short and the validity of each technique in different situations is unknown. This is an area where new investigation is necessary and where real progress can be expected, primarily because of the excellent basic physiological foundations present for their interpretation. Finally, the exact mechanisms by which LV failure can occur, as manifest by an increasing arteriovenous O₂ difference, remains to be defined. Non-invasive bedside tests that can accurately identify and differentiate different pathophysiological processes need to be developed. This is an important issue because process-specific therapies are often very effective at improving cardiovascular support, whereas non-specific therapies are often of marginal benefit and can be harmful.

At the end of the day we are left with the satisfaction of knowing that cardiovascular dysfunction may be the missing piece in predicting weaning success, and that cardiovascular monitoring can be useful in the diagnosis and management of ventilator-dependent patients, especially when weaning trials are being considered. The fact that increasing arterio-venous O₂ content monitoring identifies patients at risk to fail wean trials can aid clinicians in identifying the etiology of the cardiovascular dysfunction and, hopefully, improve outcome in this high-cost, high-mortality patient population.

References

1. Adams WC, McHenry MN, Bernauer EN (1974) Long-term physiologic adaptations to exercise with special reference to performance and cardiorespiratory function in health and disease. *Am J Cardiol* 33: 765-775
2. Chester EH, Belman MJ, Bahler RC, Baum GL, Schey G, Buch P (1977) Multidisciplinary treatment of chronic pulmonary insufficiency 3. The effect of physical training on cardiopulmonary performance in patients with chronic obstructive pulmonary disease. *Chest* 72: 695-702
3. DeBacker D, El Haddad P, Preiser J-C, Vincent J-L (2000) Haemodynamic responses to successful weaning from mechanical ventilation after cardiovascular surgery. *Intensive Care Med* (this issue)
4. Richard C, Teboul J-L, Archambaud F, Hebert J-L, Michaut P, Auzepy P (1994) Left ventricular function during weaning of patients with chronic obstructive pulmonary disease. *Intensive Care Med* 20: 181-186

5. Hurford WE, Lynch KE, Strauss HW (1991) Myocardial perfusion as assessed by Thallium-201 scintigraphy during discontinuation of mechanical ventilation in ventilated patients. *Anesthesiology* 74: 1007–1016
6. Chatila W, Ani S, Guaglianone D, Jacob B, Amoateng-Adjepong Y, Mantous CA (1995) Cardiac ischemia during weaning from MV. *Chest* 109: 1421–1422
7. Hurford WE, Favorito F (1995) Association of myocardial ischemia with failure to wean. *Crit Care Med* 23: 1475–1480
8. Buda AJ, Pinsky MR, Ingles NB, Daughters GT, Alderman EL (1979) Effect of intrathoracic pressure on left ventricular performance. *N Engl J Med* 301: 453–459
9. Lemaire F, JL Teboul, L Cinoti, G Giotto, F Abrouk, G Steg, I Macquin-Mavier, WM Zapol (1988) Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology* 69: 171–179
10. Beach T, Millen E, Grenvik (1973) Hemodynamic response to discontinuance of mechanical ventilation. *Crit Care Med* 1: 85–90
11. Jubran A, Mathru M, Dries D, Tobin MJ (1998) 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
12. Pinsky MR (1994) Though the past darkly: ventilatory management of patients with chronic obstructive pulmonary disease. *Crit Care Med* 22: 1714–1717
13. Rasanen J, Nikki P, Heikkila J (1984) Acute myocardial infarction complicated by respiratory failure. The effects of mechanical ventilation. *Chest* 85: 21–28
14. Ranieri VM, Giuliani R, Cinnella G, et al. (1993) Physiologic effects of positive end-expiratory pressure in patients with chronic obstructive lung disease during acute ventilatory failure and controlled mechanical ventilation. *Am Rev Respir Dis* 147: 5–13
15. Rasanen J, Vaisanen IT, Heikkila J, et al. (1985) Acute myocardial infarction complicated by left ventricular dysfunction and respiratory failure. The effects of continuous positive airway pressure. *Chest* 87: 158–162
16. Michard F, Chemla D, Richard C, Wysocki M, Pinsky MR, Lecarpentier Y, Teboul JL (1999) Clinical use of respiratory changes in arterial pulse pressure to monitor the hemodynamic effects of PEEP. *Am J Respir Crit Care Med* 159 (3): 935–939
17. Teboul J-L, Besbes M, Andrivet P, Axler O, Douguet D, Zelter M, Lemaire F, Brun-Bussian C (1992) A bedside index assessing the reliability of pulmonary artery occlusion pressure measurements during mechanical ventilation with positive end-expiratory pressure. *J Crit Care* 7: 22–29