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Right ventricular function in COPD patients during weaning from mechanical ventilation

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Abstract. We studied the right ventricular function during a successful weaning period in 7 COPD patients without LV disease who had been mechanically ventilated for several days after an acute exacerbation of their disease. A Swan-Ganz ejection fraction thermodilution catheter performed measurements of right ventricular ejection fraction (RVEF) and right ventricular end-diastolic volume index (RVEDVI) before and fifteen minutes after disconnection from the ventilator at the maintenance FiO_2. Although pulmonary artery pressure (PAP) rose from 25±4 to 28.5 ± 4.5 mmHg after disconnection from the ventilator, RVEF (0.36±0.56 to 0.35±0.12) and RVEDVI (117±51 to 126±52 ml/m^2) remained similar in both conditions. We concluded that right ventricular systolic function assessed with modified pulmonary artery catheter was maintained during the weaning phase in such weanable patients. This method could easily detect any fall of RVEF or diastolic RV enlargement able to impair the weaning in some patients.

Key words: Right ventricular ejection fraction – COPD – Weaning

Weaning patients with chronic obstructive pulmonary disease (COPD) who have been mechanically ventilated (MV) for an acute exacerbation may be difficult. Recently, we showed that the rapid weaning from MV induced profound modifications in intrathoracic pressure, increasing the work of breathing, releasing catecholamines and dramatically changing biventricular loading conditions and myocardial oxygen demand-delivery balance [1]. We measured a very large increase of pulmonary artery occlusion pressure (PAOP) and pulmonary artery pressure (PAP), which led to right ventricular (RV) afterload. Consequently, an augmentation of the right ventricular end-diastolic volume index (RVEDVI) was noticed on the simultaneously performed cardiac angioscintigraphy. The gated radionuclide ^{99m}Tc right ventricular ejection fraction (RVEF) was however unchanged in the majority of our 15 patients. However, in some of these patients radionuclide RVEF dramatically fell with an important dilation of RVEDVI. Accordingly, we studied the right ventricular function during a successful weaning period in 7 COPD patients without LV disease who had been mechanically ventilated for several days (7±3) after an acute exacerbation of their disease.

Materials and methods

Seven patients were selected for this study. They were mechanically ventilated for an acute exacerbation of a previous COPD which was diagnosed on the basis of clinical history, blood gases, X-ray finding and positive pulmonary function test. Normal left heart function was assumed on the basis of history, clinical examination, ECG and echocardiography. At the time of this study, patients were still on controlled ventilation, with a stable cardiopulmonary status, and were in sinus rythm. They met the usual criteria for predicting successful weaning: maximum negative inspiratory pressure lower than −30 cm H_2O; a VT larger than 5 ml/kg; and a minute ventilation lower or equal to 10 l/min. Accordingly, all these patients weaned successfully 48 hours after the study.

A Swan-Ganz ejection fraction thermodilution catheter (model 93A 431H–, 7.5 F, Edwards Laboratories) was placed via an internal jugular vein to measure the pulmonary artery pressure (PAP) and the pulmonary artery occluded pressure (PAOP) at end-expiration. Cardiac output and RVEF were determined with a REF-1 computer (Edwards Laboratories). We averaged eight successive measurements obtained by injection of 10 ml of a 0 °C dextrose solution, ran-

domly spread in the respiratory cycle, to minimize the influence of respiratory cyclic variations. Nevertheless, in no patients were large differences observed between successive values of RVEF. RVEDVI was derived from CO, SV and RVEF. No significant tricuspid insufficiency was detected by a cardiac echo-Doppler study. Blood pressure (BP) was continuously monitored using a radial artery catheter.

Hemodynamic measurements were performed before and fifteen minutes after disconnection from the ventilator, at the maintenance FiO_2.

Statistics

Multiple paired t-tests were used, to compare data (PAP, PAOP, cardiac index (CI), heart rate (HR), RVEF, RVEDVI and BP) during mechanical ventilation (MV) and spontaneous ventilation (SV). Significance was judged according to Bonferroni's inequality, using a simultaneous significance level of 0.05, i.e. 0.05 was divided by the number of comparisons, in order to obtain the significance level that an individual p value must satisfy to achieve significance.

Results

During weaning mean BP (BP mean) rose from 99±30 mmHg to 106±31 mmHg, cardiac index from 3.5±1.1 $l \cdot min^{-1} \cdot m^{-2}$ to 4.3±1.0 $l \cdot min^{-1} \cdot m^{-2}$ (Table 1). The two latter augmentations did not reach statistical significance level. Increase of cardiac index was essentially due to a signifcant increase of HR from 89±15 to 98±17 beats/min. Mean PAP significantly rose from 25±4 to 28.5±4.5 mmHg after disconnection from the ventilator. RVEF (0.36±0.08 to 0.39 ± 0.12) and RVEDVI (117±51 to 126±52 ml/m^2) were similar in both conditions (Table 1). All patients were successfully weaned from their ventilator at the end of the study.

Discussion

Maintenance of RVEF and RVEDVI, during the weaning phase, in this group of easily weanable COPD patients is the most important finding of this preliminary study. Rapid weaning from MV increased CI, HR and BP, likely due to a catecholamine discharge, as showed in previous studies [1, 2]. This catecholamine release could also explain the maintenance of the RVEF despite the increase of PAP, and thus of RV afterload. Hemodynamic performance during weaning has been investigated mainly in patients after open-heart surgery [3, 4] showing that increase in cardiac output was the best predictor of a successfull weaning. In our study, CI increased in six of seven patients, and all weaned easily. Similarly, PAOP increased significantly, but by a lesser extent than in our previous patients [1] (from 10±2 mmHg to 14 ± 3 mmHg). Therefore, RV-LV interdependence mechanism played a minor role, and the moderate increase

Table 1. Hemodynamic data before (MV) and after (SV) disconnection from the ventilator

Patients		PAP mmHg	PAOP mmHg	BP mean mmHg	HR beat/mn	CI $l \cdot m^{-1} \cdot m^{-2}$	RVEF	RVEDVI ml/m^2
1	MV	22	10	90	69	2.7	0.29	134
	SV	23	12	85	75	2.9	0.29	134
2	MV	21	11	80	80	3.5	0.37	116
	SV	26	15	86	90	5.0	0.41	137
3	MV	30	15	96	85	4.1	0.49	98
	SV	26	20	109	92	5.4	0.53	111
4	MV	27	8	65	101	3.1	0.36	86
	SV	30	15	72	116	4.8	0.56	73
5	MV	22	10	82	113	3.6	0.28	114
	SV	26	16	94	118	4.2	0.27	133
6	MV	30	10	140	82	5.5	0.31	216
	SV	32	12	145	84	4.9	0.26	214
7	MV	22	8	140	92	2.1	0.43	53
	SV	26	11	150	111	3	0.40	68
Mean ± SD	MV	25±4	10±2	99±30	89±15	3.5±1.1	0.36±0.08	117±51
	SV	28.5±4.5	14.5±3	106±31	98±17	4.3±1.0	0.39±0.12	126±52
p value		[a]	[a]	NS	[a]	NS	NS	NS

MV = Mechanical ventilation; SV = spontaneous ventilation; PAP = mean pulmonary artery pressure; PAOP = Pulmonary artery occlusion pressure; BP mean = mean blood pressure; HR = Heart rate; CI = Cardiac index; RVEF = Right ventricular ejection fraction; RVEDVI = Right ventricular end-diastolic volume index; [a] = significant at the simultaneous 0.05 level; NS = Not significant

of PAOP we measured corroborated the absence of significant impairment of LV compliance. However, increased in PAOP and mean PAP was significant, and indeed increased the RV afterload. Since RVEF did not change (form 0.36 to 0.39) and the RV end-diastolic volume index did not increase during the spontaneous breathing, contractility was probably increased. The large discharge of catecholamines we observed during the weaning phase in our previous series of patients, as already found by Kennedy et al. [2], could explain this RV contractility increase and the maintenance of RVEF. Indeed, weaning these patients is like performing a sudden exercise [5]: patients without cardiac impairment can make it, but patients with previous left heart dysfunction cannot. In addition, major intrathoracic pressure changes associated with an increase in venous return and RV afterload did not alter the RV performance in this series of patients.

After this preliminary study of patients weaning successfully, it should be interesting to assess the changes of RV function (RVEF, RVEDVI) in patients with unsuccessfull weaning. In previously published investigations, right ventricular volumes and EF have been assessed using the gated blood pool equilibrium technique. However, this technique has a number of severe limitations concerning the right ventricle which should be eliminated by using the thermal technique. A fall of RVEF and an augmentation of RVEDVI could result in an encroachment upon the LV by the dilated RV, and then in LV diastolic filling impairment. Our hypothesis is that detection of RVEF diminution could perhaps allow an easier weaning by application during this critical period of an adjusted treatment of the RV systolic function (inotropic and/or pulmonary vasodilators drugs).

In conclusion, the Swan-Ganz ejection fraction thermodilution catheter seems most useful for assessing the changes of hemodynamic parameters – especially RV's – during weaning from mechanical ventilation. In our previous series, this appeared crucial in COPD patients who failed to wean despite satisfactory ventilatory criteria. The modified pulmonary artery catheter could possibly detect in "unweanable patients", a significant fall of RVEF, attributed to a failure of the RV to counterbalance the afterload increase. It could also detect an important diastolic RV enlargement, impairing LV filling. An appropriately adapted therapy for RV systolic function might then help the heart to tolerate more effectively this critical period. This hypothesis, however, needs further investigation.

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