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Right ventricular performance in patients with acute respiratory failure

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Abstract. To examine the right ventricular response to acute respiratory failure, serial studies of biventricular performance were analysed in 34 such patients, specifically detailing the role of associated underlying disease. During the initial study, the 34 patients with acute respiratory failure had a higher right ventricular end-diastolic volume than the control group (+21%), associated with a decrease in right ventricular ejection fraction, abnormalities which tended to return to normal values in the 15 survivors. In the 9 patients who died of refractory hypoxemia with severe pulmonary hypertension, the right ventricular dilation allowed to maintain stroke volume. In contrast, in 8 patients who died of septic shock, biventricular function was progressively altered (right and left ventricular ejection fraction = −37% and −35%). In 4 patients who died of cardiogenic shock (viral myocarditis), the cardiac function was the lowest (right and left ventricular ejection fraction = −59% and −60%). Only patients with acute respiratory failure associated with septic shock or viral myocarditis are unable to maintain their stroke volume.

Key words: RV performance – Acute respiratory failure

The importance of altered hemodynamic function in acute hypoxemic respiratory failure (ARF) has been well recognized [1, 2]. Zapol and Snider [2] reported pulmonary artery hypertension and elevated pulmonary vascular resistance in ARF, even after correction of systemic hypoxemia. Although neither elevated pulmonary vascular resistance nor cardiac output reliably predicted death, survivors had progressive decreases in pulmonary vascular resistance over time, whereas nonsurvivors tended to maintain or increase their pulmonary vascular resistance [2]. The work load imposed upon the right ventricle by elevation of pulmonary vascular resistance may be a factor limiting survival in severe ARF.

Sibbald et al. [3], as well as our group [4], reported that pulmonary artery hypertension in patients with ARF was associated with an increased right ventricular (RV) end-diastolic volume and a decreased ejection fraction. The stroke volume is preserved by the Frank Starling mechanism as preload increases. In addition, Sibbald et al. [3] compared RV function in two groups of acutely ill patients with ARF with and without traumatic contusion of the RV free wall due to blunt chest injury. Characterization of the two groups of patients over a range of pulmonary artery pressures was consistent with a depression of RV performance in the patients with a contused right ventricle. Thus, it appears that the contractile state of the right ventricle can significantly influence the clinical course of superimposed ARF.

In the present study, we examined the RV response to ARF by analyzing initial and serial studies of biventricular performance in such patients, specifically detailing the role of associated underlying disease in the observed right ventricular dysfunction.

Methods

Patients

Thirty six patients with ARF were studied in the Medical Intensive Care Unit of Cochin Port-Royal University Hospital. With consideration of study the ARF was characterized by bilateral homogenous pulmonary opacification on chest roentgenographic examination and by marked arterial hypoxemia, with a calculated venous admixture higher than 25% at an inspired oxygen concentration (FiO_2) of 0.50, when

the mean pulmonary wedge pressure was lower than 10 mmHg. All patients with a previous history of cardiac or lung disease were excluded from the study.

Protocol

The initial study was always performed as near to the onset of ARF diagnosis as was technically and medically feasible, always in the first 24 h, just at the beginning of PEEP ventilation. Volume controlled ventilation (Servo Ventilator 900 C, Siemens) was used, delivering a tidal volume of 10 to 14 ml/kg of body weight; $PaCO_2$ and pH were maintained about normal values. Follow-up studies were performed at 5 and 10 days after the acute phase of the illness. Informed consent was obtained from the next of kin and the protocol was approved by the Committee on Ethics of our Institution.

Hemodynamic and thermodilution measurements

A quadruple-lumen, pulmonary artery catheter (Model 93A-431H-7.5F, American Edwards Laboratories, Santa Ana, CA) equipped with a fast-response (95 ms) thermistor was inserted. Cardiac output and RV ejection fraction were measured by a previously described thermodilution technique [5–7] (REF1 prototype, Edwards Laboratories). Systemic arterial pressure, right atrial pressure, pulmonary artery pressure, and pulmonary capillary wedge pressure were zero-referenced to the midchest and measured at end-expiration.

Radionuclide measurements

Multigated blood-pool imaging was then performed using a previously described technique [8].

The *left* ventricular ejection fraction (EF) was then calculated by the formula EF = EDC − ESC/EDC, where EDC is end-diastolic counts and ESC is end-systolic counts. Laboratory normal value for left ventricular ejection fraction is 65% ± 6%.

Calculations

Using the pulmonary artery catheter to measure cardiac output (CO), we calculated both cardiac index (CI [l/min/m^2]) and stroke volume index (SV [ml/m^2]). Using thermodilution curve computerization, RV end-diastolic volume (RVEDV) was calculated by the formula: RVEDV (ml/m^2) = SV/RVEF, and right ventricular end-systolic volume (RVESV: ml/m^2) by subtracting SV from RVEDV. At the same time as radionuclide angiographic examination, left ventricular end-diastolic volume (LVEDV) was calculated by the formula: LVEDV (ml/m^2) = SV/LVEF, where LVEF is left ventricular ejection fraction and left ventricular end-systolic volume (LVESV: ml/m^2) by subtracting LVEDV from SV.

Control group

These hemodynamic studies were also done in 10 control subjects, average age 45 ± 7 years, who were undergoing during routine cardiac catheterization for aortic valvular dysfunction without alteration of ventricular function.

Statistical analysis

The frequency of underlying diseases and of fatalities were compared by means of the chi-square test with Yates correction. The paired t-test corrected for the use of small numbers was used to compare group mean values. The Bonferroni method was used to decrease the chance of mistakenly declaring significance due to multiple comparisons. The method of least squares was used for the calculation of linear regression. All values are expressed as the mean ± SD.

Results

The clinical characteristics of the 36 patients. The mean age was 49 years. All had an underlying disease known to be associated with acute respiratory failure: gram negative sepsis in 13; bacterial pneumonia in 8; viral pneumonia in 6; fat emboli in 3; pancreatitis in 3; and inhaled smoke, massive blood transfusion and pneumocystis carinii pneumonia in one case respectively. All patients required mechanical ventilation with positive end-expiratory pressure at an average level of 10 cm H_2O (7 to 16).

Fifteen out of the 36 patients survived their acute illness. The other 21 patients (58%) died of refractory hypoxemia with severe pulmonary artery hypertension ($n = 9$), septic shock ($n = 8$), or cardiogenic shock ($n = 4$) due to viral myocarditis. The survivors and nonsurvivors did not differ significantly with respect to age (mean 46 vs 52 yrs), level of PEEP (mean 9 vs 12 cm H_2O), hemodynamic and respiratory monitored variables.

Hemodynamic data [Table 1]

During the initial study, the 36 patients with ARF had a higher RV end-diastolic volume than the control group (+21%, $p < 0.05$) associated with a lower right ventricular ejection fraction (−16%), but this difference lacks statistical significance. A significant negative correlation between the mean pulmonary artery pressure and the RV ejection fraction was found ($y = -0.98x + 65.7$, $r = 0.67$, $p < 0.001$). Left ventric-

Table 1. Behavior of biventricular performance in the 36 patients with ARF vs control group

	RVEDV	RVEF	LVEDV	LVEF	SV
Do ($n = 36$)	110 ± 33^a	0.41 ± 0.06	74 ± 24	0.61 ± 0.06	45 ± 10
PAH ($n = 9$)	145 ± 27^b	0.29 ± 0.04^a	66 ± 18	0.64 ± 0.05	42 ± 8
SS ($n = 8$)	115 ± 21^a	0.31 ± 0.04^a	79 ± 20	0.43 ± 0.04^a	35 ± 7^a
CC ($n = 4$)	120 ± 25^a	0.20 ± 0.02^b	96 ± 15^a	0.25 ± 0.03^b	24 ± 6^b
Recov. ($n = 15$)	96 ± 23^c	0.46 ± 0.05^c	72 ± 21	0.61 ± 0.05	44 ± 8
Control ($n = 10$)	91 ± 18	0.49 ± 0.05	71 ± 14	0.63 ± 0.04	45 ± 7

RVEDV = right ventricular end-diastolic volume (ml/m^2); RVEF = right ventricular ejection fraction; LVEDV = left ventricular end-diastolic volume (ml/m^2); LVEF = left ventricular ejection fraction; SV = stroke volume (ml/m^2); Do = first day of ARF; PAH = pulmonary artery hypertension; SS = septic shock; CC = cardiogenic shock; Recov. = recovery. [a] $p < 0.05$ and [b] $p < 0.01$ for comparison with control group; [c] $p < 0.05$ for comparison with PAH

ular performance and stroke volume were not significantly different in the control and ARF patients.

During the recovery phase, no significant differences in right ventricular performance were noted between the 15 survivors and control group.

In contrast, serial hemodynamic studies demonstrated a progressive increase in RV end-diastolic volume (+59% [at 10 d after the onset of ARF], $p < 0.01$ vs control group), associated with a fall in RV ejection fraction (−41%, $p < 0.01$ vs control group) in the 9 patients who died of refractory hypoxemia with severe pulmonary artery hypertension. The negative correlation between the mean pulmonary artery pressure and the RV ejection fraction described for all patients during the initial study persisted all over the study period.

In the 8 patients who died of septic shock, biventricular performance was altered: right and left ventricular ejection fractions were low (−37% and −35% [at day 5 after the onset of ARF], respectively; $p < 0.01$ vs control group), and RV end-diastolic volume increased (+26%; $p < 0.05$) while the increase in LVEDV (+11%) lacks statistical significance. In the 4 patients who died of cardiogenic shock, the dramatic fall in right and left ventricular ejection fraction (−59% and −60%, respectively; $p < 0.01$ vs control group) was associated with biventricular dilation (+32% and +35%, respectively; $p < 0.05$ vs control group). In the last two groups of patients, stroke volume was markedly lower than control group (−24%; $p < 0.05$ and −47%; $p < 0.01$, respectively), despite high levels of inotropic drugs and no correlation was found between the mean pulmonary artery pressure and the RV ejection fraction.

Discussion

ARF is associated with appropriate RV pump function, despite the observed RV dysfunction, defined as decreased RV ejection fraction and RV dilation. Only ARF patients with concomitant underlying disease as septic shock or viral myocarditis are unable to maintain their stroke volume.

Similar to the effect of an increased pressure load on the right ventricle during acute exacerbations of chronic obstructive pulmonary diseases [9], we found in ARF that the occurrence of pulmonary artery hypertension was followed by both an increase in RV end-diastolic volume and a depression of RV ejection fraction. Such changes were inversely correlated with changes in mean pulmonary artery pressure, used in clinical studies to reflect the pressure that the right ventricle must overcome during ejection [3, 8], although the right ventricular afterload is probably a more complex function [10].

The observed right ventricular overdilation without a concomitant increase in the right ventricular stroke volume, suggests, in agreement with Sibbald et al. [3], that patients sustaining a progressive increase in pulmonary artery hypertension complicating ARF were characterized by a position either on the plateau portion of a Frank-Starling relationship or on a downshifted curve, as would be seen with the concurrent emergence of right ventricular contractile depression. Despite evidence for depressed right ventricular systolic function in such patients, preload augmentation remains an ideal compensatory mechanism, as left ventricular systolic and pump function appears unaffected.

By contrast, if severe contractile depression of the right ventricle coexists, as must be the case with a septic shock or a viral myocarditis, the ability to utilize the Frank-Starling mechanism to maintain right ventricular pump function seems potentially limited, as previously demonstrated with right ventricular infarction [11] and right ventricular contusion [3].

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