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Title : VENTRICULAR FUNCTION AND PULMONARY HYPERTENSION IN ACUTE RESPIRATORY FAILURE

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Introduction. It has recently been shown that severe acute respiratory failure (ARF) is associated with elevations of mean pulmonary artery pressure (PA), pulmonary vascular resistance and right ventricular stroke work (1). These results were obtained from patients in whom normal blood gas values had been restored by oxygen therapy and mechanical ventilation. It has been repeatedly suggested that this increase in PA may lead to right ventricular strain and dilatation, with a reduction in right ventricular ejection fraction. It has also been said that dilatation of right ventricle might lead to interference with left ventricular function (2). These hypotheses have been supported by little direct evidence and were inferences drawn from atrial pressure to ventricular stroke work relationships.

Methods. To more directly examine ventricular function, 20 consecutive patients who required pulmonary artery catheterization during the course of ARF were studied. Fifteen were males and 5 females. Ages ranged from 17-84 years, mean 56 years. Multiple gated acquisition, cardiac blood pool isotopic imaging was performed using 25 mCi. of Tc-99m in vivo labeled red blood cells, and a portable 37 PM tube gamma camera with parallel hole collimation, interfaced with an on-line computer. Gated first pass right heart studies in the 30° RAO projection and gated equilibrium cardiac studies in the anterior and 45° LAO projection were performed. Right and left ventricular ejection fractions (RVEF and LVEF) were calculated. Within the span of 1 hour prior to these estimations of RVEF and LVEF, right atrial pressure (RAP), pulmonary artery pressure, pulmonary artery occlusion pressure (PAOP), and cardiac output by thermodilution were also measured as was systemic blood pressure. Arterial blood samples were drawn for estimation of arterial oxygen tension (PaO₂) at known inspired oxygen concentrations (FIO₂). Heart rate was estimated from an EKG tracing. Body surface area was determined from height and weight.

Stroke volume index (SVI) was calculated, dividing the cardiac output by body surface area x heart rate. Right and left ventricular stroke work indices (RVSWI and LVSWI) were estimated from the difference between mean arterial pressure and the mean atrial pressure corresponding to that ventricle, multiplied by SVI.

Results. The mean PaO₂/FIO₂ ratio was 189 for the 20 patients; range 81-338. ARF was associated with primary pulmonary disease in 13 of 20 patients. ARF associated with pulmonary edema and primary cardiac disease was seen in the other 7 patients. Of 13 patients with primary pulmonary disease, normal biventricular ejection fractions (Table 1) were seen in 4, isolated reductions in RVEF in 5, and biventricular reductions in ejection fraction in 4. Six of 7 patients with primary cardiac disease had isolated reduction in LVEF. Biventricular reduction in ejection fraction was seen in the other patient. Neither single values of PA, nor increase of RAP in relation

to RVSWI was consistently successful in predicting decreased RVEF. Nor were increases in PAOP, relative to LVSWI, consistently successful in predicting decreased LVEF (Table 1).

Discussion. Our results showed that our patients with primary pulmonary disease and acute respiratory failure had either normal ventricular function, isolated right ventricular dysfunction or biventricular dysfunction. By contrast, patients with acute respiratory failure associated with primary cardiac disease had isolated left ventricular dysfunction or biventricular dysfunction. Using plots of ventricular stroke work index against atrial pressure we could not consistently predict what the ejection fraction of each ventricle would be. We conclude that while some of the hypothesized events described in the introduction can be supported from our data, further sequential studies of RVEF and LVEF together with pulmonary hemodynamics in patients in acute respiratory failure are needed for final confirmation.

References.

1. Zapol WM, Snider MT: N Engl J Med 296:476-480, 1977.
2. Laver MB, Strauss HW, Pohost GM: Crit Care Med 7:509-519, 1979.
3. Barratt-Boyes BG, Wood EH: J Lab Clin Med 51:72-90, 1958.
4. Walton S, Rowlands DJ, Shields RA, Testa HJ: Intens Care Med 5:121-126, 1979.

TABLE 1
Pulmonary Hemodynamics and Ventricular Ejection Fractions: 21 Normal Subjects* and 20 Patients With ARF

Diagnosis	PA	RAP	RVSWI	PAOP	LVSWI
*Normal (N=21)	17 +1	6 +1	5.4 +0.4	12 +1	38 +2
ARF (N=20)	^a 27 +2	^a 10 +1	5.3 +0.7	13 +1	^a 24 +3
ψNormal EF (N=4)	22 +3	10 +2	4.1 +0.6	11 +3	^a 26 +3
ψLVEF<55%† (N=6)	^a 24 +3	8 +2	4.7 +2.8	12 +3	^a 23 +4
ψRVEF<50%† (N=5)	^a 28 +4	11 +3	7.1 +2.0	11 +2	27 +6
ψLVEF<55% and RVEF<50%† (N=5)	^a 34 +7	^a 12 +2	5.6 +1.8	18 +3	20 +9

*See reference 3, ψsubgroups of ARF, †see reference 4, ^adifferent from normal (reference 3) p<0.05.