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Ventricular Function and Pulmonary Hypertension in Acute Respiratory Failure

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วรรณ สมบูรณ์วิบูลย์. การศึกษาหน้าที่ของหัวใจและความดันของปอดใน
ผู้ป่วยหายใจล้มเหลว. จุฬาลงกรณ์เวชสาร 2525 มีนาคม ; 26 (2) : 151-156

การเปลี่ยนแปลงในผู้ป่วย *acute respiratory failure* ที่พบเสมอก็คือการ
เกิด *pulmonary hypertension* และการเพิ่มของ *pulmonary vascular resistance*
ซึ่งจะมีผลทำให้เกิด *right ventricle strain* และ *dilatation* เป็นการลด *right*
ventricular ejection fraction ด้วย และจะมีผลกระทบกระเทือนต่อ *left ventri-*
cular function ได้ จึงได้ทำการศึกษาผู้ป่วย *acute respiratory failure* จาก
สาเหตุต่างๆ 26 ราย โดยการใช้ *pulmonary artery catheter* (*Swan Ganz*
catheter) เพื่อดูการเปลี่ยนแปลงของ *hemodynamics* และศึกษา *ventricular*
functions ด้วยวิธี *radionuclide angiocardiography* โดยใช้ *technetium*
99 และวิธีการที่เรียกว่า *Gated first pass cardiac blood pool*

ผลจากการศึกษานี้พบว่าผู้ป่วยทุกรายมีการเพิ่มของ *pulmonary pressure*
และ *pulmonary vascular resistance* ส่วน *ventricular function* มีทั้งปกติ
และผิดปกติ ซึ่งผู้ป่วยที่มีโรคปอดมาก่อนที่จะเกิด *respiratory failure* จะมีความ
ผิดปกติของ *right ventricular function* ทุกราย

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Introduction

It has been suggested that elevation of mean pulmonary artery pressure and pulmonary vascular resistance are uniformly found in patients with severe acute respiratory failure. (1)

In this study we attempted to answer two questions. First, did pulmonary hypertension occur in our patients with acute respiratory failure, and second, was ventricular dysfunction present? (2)

We used the Swan Ganz catheter to determine the hemodynamic changes, namely pulmonary artery pressure and pulmonary vascular resistance, studied the ventricular functions by the use of radionuclide angiocardigraphy with technetium 99 (4) in 26 patients with acute respiratory failure from various causes.

Material and method

1. Subjects : 26 patients age range from 17 to 84 years (mean of 57 years) 19 were males and 7 were females, who were diagnosed as having acute respiratory failure by clinical manifestations of dyspnea, tachypnea, cyanosis and tachycardia and with the blood gases of PaO_2 less than 50 mmHg with or without hypercapnea. These patients were admitted to the Respiratory Intensive Care Unit Mayo Clinic from November 1979 to March 1980.

2. Method

Swan Ganz catheter was passed through the central line to measure the hemodynamic changes, ventricular functions were studied by radionuclide angio-

cardigraphy with technetium 99 and gated first pass cardiac blood pool was used in every patients.

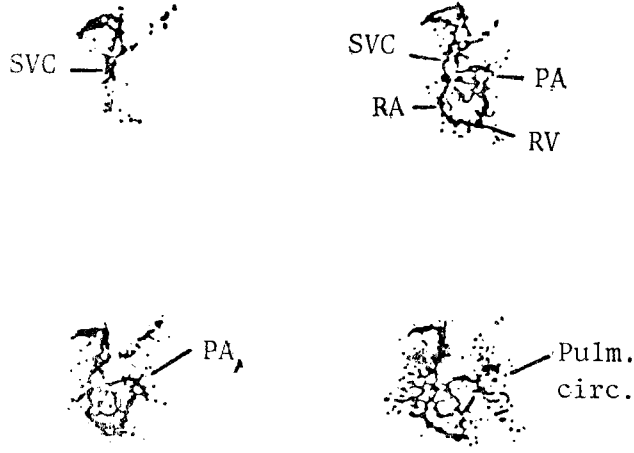
The following data were recorded. Ratio of arterial oxygen tension to fractional inspired oxygen tension ($\text{P}_a\text{O}_2/\text{F}_i\text{O}_2$)

Pulmonary artery pressure (PAP)	mmHg
Pulmonary arteriolar resistance (R_{pa})	u/m^2
Heart rate (HR)	beat/min
Stroke volume index (SVI)	ml/m^2
Right atrial pressure (RAP)	mmHg
Right ventricular stroke work index (RVSWI)	u/m^2

To further evaluate ventricular function, radionuclide angiography was performed with a portable 37 photomultiplier tube Anger camera. Red blood cells were labelled in vivo using 25 to 30 mc of technetium 99 M pertechnetate. To compensate for the count rate limitation of the Anger camera, gating of the first pass right heart studies according to the technique of McKusick and others was utilized. (4)

By monitoring the progress of the radio pharmaceutical on a cathode ray tube, data acquisition immediately following the injection of technetium was limited to the time between the arrival of activity in the right atrium and its appearance in the pulmonary bed as shown on figure 1.

CENTRAL BLOOD POOL
RADIONUCLIDE ANGIOGRAM
Stannous pyrophosphate-25 mCi Technetium 99



On these images one can see first the right atrium, then the tricuspid valve plane, then the right ventricle and finally the pulmonary outflow tract. On the end diastolic frame a manually delineated region of interest over the right ventricle yielded the total end diastolic counts. A computer was used to determine the counts in this region of interest for each of 14 frames. The frame with the lowest count in the region of interest is the end systolic frame.

On the end systolic frame a second region of interest was outlined by light pen and total end systolic counts obtained. The background is negligible and can be ignored. Thus, right ventricular ejection fraction was calculated according to the formula : ejection fraction is equal to total end diastolic counts minus total end

systolic counts divided by the end diastolic counts. Multiple gated equilibrium images were then acquired to permit calculation of left ventricular ejection fraction and for the assessment of regional wall motion abnormalities.

The ventricular functions were determined by

1. radionuclide angiography for the ejection fraction of the ventricles
2. correlation of ventricular filling pressure and stroke work index

Results

1. pulmonary artery pressure and pulmonary arteriolar resistance are significantly elevated in patients with acute respiratory failure as compared to normal subjects.

HEMODYNAMIC STUDIES

26 PATIENTS WITH ACUTE RESPIRATORY FAILURE

		NORMAL* (N = 21)	ARF (N = 26)
		MEAN ± S.E	
P_aO_2/F_iO_2	300		174 ± 15
PAP MM Hg	17 ± 1		29 ± 2 ⁺
RPAI U/M ²	1.5 ± 0.1		4.1 ± 0.4 ⁺
H R BEAT/MIN	79 ± 2		109 ± 5 ⁺
SVI ML/M ²	49 ± 2		32 ± 2 ⁺
RAP MM Hg	6 ± 1		11 ± 1 ⁺
RVSWI U/M ²	5.4 ± 0.4		6.0 ± 0.6

* DATA FROM BARRATT-BOYES ET AL.

+ SIGNIFICANTLY DIFFERENT FROM NORMAL (P < 0.05)

2. Ventricular functions by radio-nuclide angiography. Patients were considered to have abnormal ejection fractions when their left ventricular ejection fraction was less than 55 % or right ventricular ejection fraction less than 50 %, values which were 2 standard deviations below the mean of normal from the data of Walton and others (5) Using these values we were able to divide our patients into 4 groups according to the ejection fractions

Group A contained 5 patients with normal biventricular ejection fractions

Group B contained 7 patients with isolated reductions in left ventricular

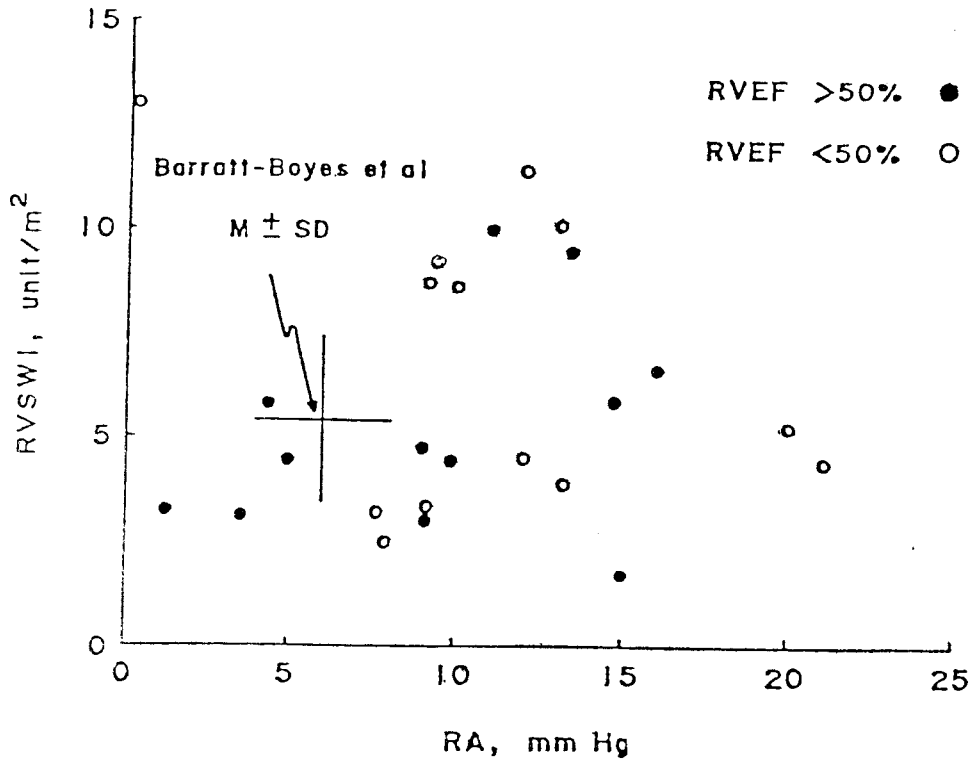
ejection fraction below 55 %

Group C consisted of 7 patients with isolated reductions in right ventricular ejection fraction below 50 %

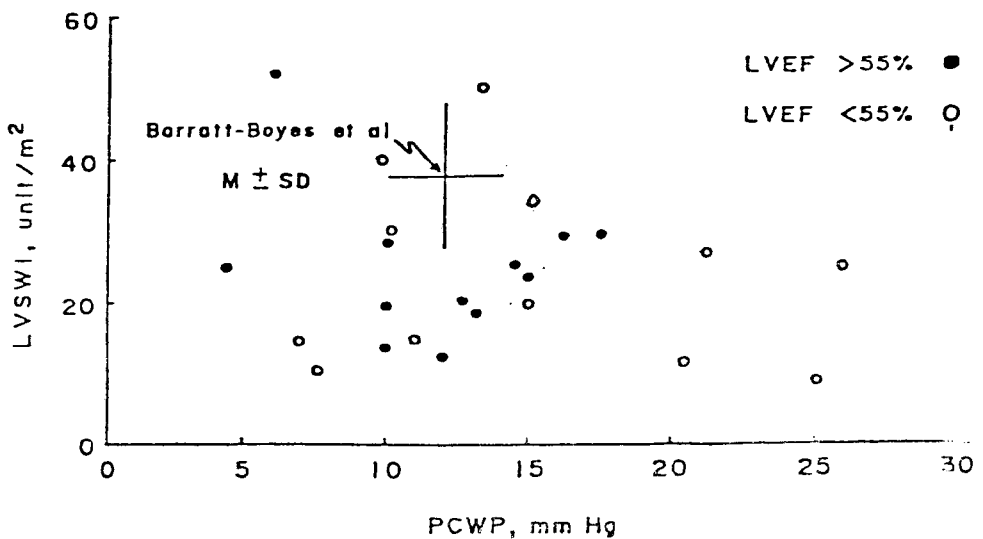
Group D consisted of 7 patients with reduced biventricular ejection fraction.

We further examined ventricular function by using the correlation between ventricular filling pressure and the stroke work index as shown here for the right ventricle. Normal values ± one standard deviation for the right atrial pressure and right ventricular stroke work index are shown also on graph 1

RV STROKE WORK INDEX AND RA PRESSURE
IN ACUTE RESPIRATORY FAILURE



LV STROKE WORK INDEX AND WEDGE PRESSURE
IN ACUTE RESPIRATORY FAILURE



No clear separation can be seen between these two groups. However, some patients with reduced right ventricular ejection fraction had an increased right atrial pressure and decreased right ventricular stroke work index, suggesting severe right ventricular dysfunction. Similar data for the left ventricle are shown on graph 2

Again, no clear separation between these two groups could be seen, but six out of 12 patients with reduced left ventricular ejection fractions clearly had severe left ventricular dysfunction.

Discussion & Summary

We have found that mean pulmonary artery pressure is increased in acute respiratory failure. This is associated with an increase in pulmonary arteriolar resistance. Heart rate and stroke volume index are significantly altered. Four patterns of ventricular function were noted. Ventricular function was normal in association with modest elevations of pulmo-

nary artery pressure in our Group A patients.

Isolated reductions in left ventricular ejection fraction were seen in patients with primary cardiac disease, particularly where there were abnormalities of regional wall motion. Isolated reduction in right ventricular ejection fraction and right ventricular dilatation was associated with primary pulmonary disease while biventricular dilatation and reductions in ejection fraction were seen in patients with a mixture of diagnosis. It should be noted that using plots of ventricular stroke work index against atrial pressure do not consistently predict the ejection fraction for the ventricle.

It is tempting to conclude that, in some patients with acute respiratory failure, pulmonary hypertension may represent an increased afterload on the right ventricle leading to dilatation and failure. Sequential studies and planned to conclusively prove this hypothesis.

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