

# UCLA

## UCLA Previously Published Works

### Title

Comparison of noninvasive with hemodynamic data in patients with pulmonary hypertension due to chronic obstructive pulmonary disease

### Permalink

<https://escholarship.org/uc/item/3f5490f0>

### Journal

The American Journal of the Medical Sciences, 278(1)

### ISSN

0002-9629

### Authors

TOBIS, JONATHAN M  
DANAHY, DANIEL T  
ARONOW, WILBERT S  
[et al.](#)

### Publication Date

1979-07-01

### DOI

10.1097/00000441-197907000-00003

Peer reviewed

# Comparison of noninvasive with hemodynamic data in patients with pulmonary hypertension due to chronic obstructive pulmonary disease

---

JONATHAN M. TOBIS  
DANIEL T. DANAHY  
WILBERT S. ARONOW  
KOTA CHETTY  
FREDERICK GLAUSER

**Abstract:** Twenty-one patients with chronic obstructive pulmonary disease were studied with right heart catheterization. The mean pulmonary artery pressure ( $\overline{PAP}$ ) was compared with several noninvasive tests. The pulmonary lobar diameter/thoracic ratio correlated with the  $\overline{PAP}$ ,  $r = 0.677$ ,  $P < 0.01$ . The oxygen saturation and pH were used to derive a calculated value for  $\overline{PAP}$ . The calculated  $\overline{PAP}$  correlated with  $\overline{PAP}$  values measured below 40 mm Hg,  $r = 0.787$ ,  $P < 0.01$  but correlated very poorly with  $\overline{PAP}$  values measured above 40 mm Hg. The electrocardiogram revealed at least one criterion for right ventricular abnormality in all 18 patients with pulmonary hypertension, but was too nonspecific to correlate with  $\overline{PAP}$ . The hemoglobin concentration did not correlate with  $\overline{PAP}$ . There was such wide variation between the measured  $\overline{PAP}$  and the various noninvasive tests that these noninvasive tests could not be used to accurately predict the  $\overline{PAP}$  in an individual patient.

#### KEY INDEXING TERMS

*Chronic obstructive pulmonary disease*  
*Pulmonary artery pressure*  
*Electrocardiogram*  
*Hemoglobin*

*Pulmonary hypertension*  
*Chest roentgenogram*  
*Arterial blood gases*  
*Right heart catheterization*

Several attempts have been made to correlate clinical and hemodynamic data in patients with chronic obstructive pulmonary disease (COPD). In 1964, Enson and associates developed a formula for predicting the

mean pulmonary artery pressure ( $\overline{PAP}$ ) determined by arterial oxygen saturation and pH.<sup>1</sup> Kilcoyne and co-workers later used this equation to correlate electrocardiographic findings in 200 patients with COPD with

---

*From the Cardiology and Chest Sections and Medical Service, Long Beach Veterans Administration Hospital, and the University of California, Irvine, and the Chest Division, Medical College of Virginia, Richmond.*

*Requests for reprints should be addressed to Wilbert S. Aronow, M.D., Veterans Administration Hospital, Long Beach, California 90822.*

predicted  $\overline{\text{PAP}}$  values, without obtaining right heart catheterization.<sup>2</sup>

Radiographic techniques have been used to evaluate cardiac size<sup>3</sup> and to distinguish between right and left heart failure in patients with COPD complicated by acute respiratory failure.<sup>4</sup> Lupi and associates described another radiographic technique, the pulmonary artery lobar diameter/thoracic ratio, for predicting the presence of pulmonary artery hypertension.<sup>5</sup> Burrows and co-workers studied 50 patients with COPD and distinguished the clinical and hemodynamic findings of two groups: those with emphysematous versus those with bronchitic types of lung disease.<sup>6</sup> Rizzato and associates used a multifactorial analysis to compare measured  $\overline{\text{PAP}}$  with body surface area, age, arterial  $\text{PO}_2$ , arterial pH, arterial  $\text{PCO}_2$ , hematocrit, and pulmonary function tests (vital capacity, forced expiratory volume in one second/vital capacity, residual volume/total lung capacity) in 70 patients with COPD.<sup>7</sup> Of the nine variables they studied, only two (arterial  $\text{PO}_2$  and body surface area) correlated significantly with  $\overline{\text{PAP}}$ .

Thus, many claims have been made in the literature concerning the reliability of noninvasive methods to predict  $\overline{\text{PAP}}$ .<sup>9-13</sup> We studied a series of 21 patients with COPD and correlated the hemodynamic data obtained with arterial blood gases, the chest roentgenogram, electrocardiogram, and hemoglobin concentration. Our findings are reported here.

#### Materials and Methods

Twenty-one patients with severe COPD were evaluated as part of a study of the hemodynamic effects of isosorbide dinitrate on pulmonary hypertension. Patients were chosen for the study if they had clinical evidence of COPD with severe airway obstruction by pulmonary function tests and chronic hypoxemia and hypercapnea ( $\text{PO}_2 < 65$  torr, and  $\text{PCO}_2 > 45$  torr). Informed consent was obtained from each subject. The study was approved by our institutional committee on human research.

Each patient was in a stable condition at the time of study. A right heart catheterization was performed via percutaneous approach with a flow directed balloon-tipped catheter. The catheter was placed in the main pulmonary artery with the proximal opening in the right atrium. A long-dwell 20-gauge plastic catheter was placed percutaneously in the brachial or radial artery. Pressures were recorded through a Statham P23 ID transducer on an Electronics for Medicine VR-6 recorder. The following baseline measurements were obtained on room air: arterial and mixed venous blood gases, arterial pressure, pulmonary artery pressure,  $\overline{\text{PAP}}$ , mean right atrial pressure, pulmonary artery capillary wedge pressure, and cardiac output by thermodilution. Every patient had a 12 lead electrocardiogram (ECG) and complete blood count taken at the time of study.

The ECG was evaluated for evidence of right ventricular abnormality by the following criteria: (1) right axis deviation  $> 90^\circ$ ; (2)  $R > S$  in  $V_1$ ; (3)  $R > S$  in aVR; (4)  $R < S$  in  $V_5$ ; (5) P wave voltage  $> 2.5$  mm in a standard lead; (6) T wave inversion in  $V_{1-3}$ ; and (7) ST depression in inferior leads 2, 3, aVF.

Nineteen patients had a six-foot, postero-anterior chest roentgenogram taken. These roentgenograms were examined independently by three observers without knowledge of the patient's identity. The following measurements were determined: widest cardiac diameter; widest thoracic diameter; cardiothoracic ratio; the pulmonary lobar diameter (PLD) as defined by Lupi and associates to be the width between the borders of the upper lobar artery and the pars interlobaris on the left to the outside border of the anterior trunk on the right<sup>5</sup>; the PLD/thoracic (PLD/T) ratio; and the width of the right descending pulmonary artery (RD-PA) at the level of the eighth to ninth rib interspace.

Using the formula derived by Enson and associates, a correlation was performed between the arterial blood gas values and the  $\overline{\text{PAP}}$ . This formula utilizes the arterial oxyhemoglobin desaturation percentage and

TABLE I

MEAN VALUES OF PULMONARY FUNCTION TESTS, ARTERIAL BLOOD GASES, AND CHEST ROENTGENOGRAPHIC MEASUREMENTS IN 21 COPD PATIENTS

Parameter	Mean Value	±	Standard Deviation
Vital capacity	2.16	±	0.62 liters
% of predicted vital capacity	50.3	±	13.8 %
Forced expiratory volume 1 sec	0.70	±	0.25 liters
% of predicted forced expiratory volume 1 sec	20.0	±	6.3 %
Maximum mid-expiratory flow rate	0.35	±	0.13 liters/sec
Arterial PO <sub>2</sub>	47.6	±	5.9 torr
Arterial PCO <sub>2</sub>	58.4	±	7.9 torr
Arterial pH	7.37	±	0.03
Cardiothoracic ratio	0.48	±	0.05
PLD/T ratio	0.39	±	0.03
RD - PA width	21.2	±	2.9 mm

TABLE II

CORRELATIONS BETWEEN  $\overline{\text{PAP}}$  VALUES < 60 mm Hg MEASURED AND CALCULATED ± 1 STANDARD DEVIATION

	$\overline{\text{PAP}}$ Measured	$\overline{\text{PAP}}$ Calculated	r value
Entire Group:	37.9 ± 13.4 mm Hg	34.1 ± 10.3 mm Hg	0.689*
< 40 mm Hg:	29.3 ± 8.0 mm Hg	30.4 ± 8.4 mm Hg	0.787*
40 - 60 mm Hg:	47.5 ± 8.2 mm Hg	38.0 ± 10.4 mm Hg	0.253**

\*P < 0.01.  
\*\*P not significant.

hydrogen ion concentration to calculate the  $\overline{\text{PAP}}$  as follows:  $\overline{\text{PAP}} \text{ calc.} = 40.363 - 0.713\text{H} - 3.008\text{S} + 0.1028\text{HS}$ , where  $\overline{\text{PAP}} \text{ calc.}$  = mean pulmonary artery pressure calculated, H = hydrogen ion concentration in  $\mu\text{Eq/L}$  and S = percent oxyhemoglobin desaturation. As the equation is reported to be inaccurate for  $\overline{\text{PAP}} > 60$  mm Hg, we did not include any patients with values above this level.

### Results

All 21 patients studied had obstructive lung disease as determined by clinical history and pulmonary function tests. Table I indicates the mean values ± 1 standard deviation of the pulmonary function tests, baseline arterial blood gases, and chest roentgenographic measurements in these 21 subjects.

Table II and Figure 1 show the correlation between the  $\overline{\text{PAP}}$  values measured and the

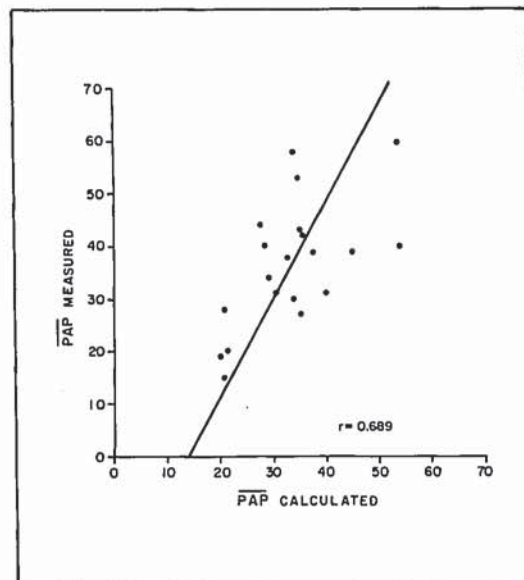


Fig. 1: Correlation of  $\overline{\text{PAP}}$  measured with  $\overline{\text{PAP}}$  calculated for each individual patient with COPD.

TABLE III  
MEAN HEMODYNAMIC VALUES IN THE COPD PATIENTS

Parameter	Value
$\overline{\text{PAP}}$	$37.9 \pm 13.4$ mm Hg
Mean pulmonary artery capillary wedge pressure	$11.7 \pm 3.2$ mm Hg
Mean right atrial pressure	$8.6 \pm 3.9$ mm Hg
Mean arterial pressure	$89.4 \pm 11.4$ mm Hg
Mean cardiac index	$3.54 \pm 0.89$ mm Hg

TABLE IV  
COMPARISON BETWEEN ECG CRITERIA FOR RIGHT VENTRICULAR ABNORMALITY AND PRESENCE OF PULMONARY HYPERTENSION IN 18 PATIENTS WITH PULMONARY HYPERTENSION DUE TO COPD

ECG Criterion	Number of Patients	% Present
Right axis deviation $> 90^\circ$	14	78
R $>$ S in $V_1$	4	22
R $>$ S in aVR	5	28
R $<$ S in $V_5$	13	72
P $>$ 2.5 mm in standard lead	6	33
T wave inversion in $V_{1-3}$	3	17
ST depression in 2, 3, aVF	4	22
Seven criteria present	0	0
Six criteria present	1	6
Five criteria present	0	0
Four criteria present	4	22
Three criteria present	6	33
Two criteria present	6	33
One criterion present	1	6

$\overline{\text{PAP}}$  values calculated by using Enson's formula.

Table III indicates the mean hemodynamic values in the 21 patients with COPD. Of the 21 patients, 18 (86%) had pulmonary hypertension as defined by  $\overline{\text{PAP}} > 20$  mm Hg.

Table IV indicates the prevalence of the seven ECG criteria for right ventricular abnormality and the presence of pulmonary hypertension in the 18 patients with pulmonary hypertension due to COPD. No consistent pattern was observed between the level of  $\overline{\text{PAP}}$  and the ECG criteria. Only one patient had six criteria present; his  $\overline{\text{PAP}}$  was 53 mm but was not the highest in the group. Of the three patients with normal resting  $\overline{\text{PAP}}$  values, one had P waves  $> 2.5$  mm in standard

lead 2, and another had ST depression in leads 2, 3, and aVF.

Table V and Figure 2 indicate the correlations between the roentgenographic measurements and the  $\overline{\text{PAP}}$  values. Of the three patients with a normal  $\overline{\text{PAP}}$ , two had enlarged RD-PA width and the third was 16 mm, the upper limit of normal.

The mean hemoglobin level was  $16.6 \pm 2.4$  gms percent. The correlation between the  $\overline{\text{PAP}}$  and hemoglobin level was  $r = 0.099$  (P not significant).

### Discussion

These 21 patients had severe COPD as determined by clinical history, pulmonary

TABLE V

CORRELATIONS BETWEEN ROENTGENOGRAPHIC MEASUREMENTS AND  $\overline{\text{PAP}}$

Roentgenographic Measurement	Mean Value $\pm$ 1 Standard Deviation	r Value
PLD/T ratio	0.39 $\pm$ 0.03	0.677*
Cardiothoracic ratio	0.48 $\pm$ 0.05	0.41**
RD-PA width	21.2 $\pm$ 2.9 mm	0.232**

\* $P < 0.01$ .

\*\* $P$  not significant.

function tests, chronic hypoxemia and hypercapnea by resting arterial blood gases. Each patient was studied with a right heart catheterization in preparation for an evaluation of isosorbide dinitrate in patients with pulmonary hypertension. As the course of the study progressed, we found it difficult to predict a patient's resting  $\overline{\text{PAP}}$  on the basis of noninvasive clinical information. Therefore, we approached the problem systematically by

comparing the measured  $\overline{\text{PAP}}$  versus criteria for pulmonary hypertension derived from the electrocardiogram, chest roentgenogram, hemoglobin, and arterial blood gases.

All the patients with pulmonary hypertension had at least one ECG criterion for right ventricular abnormality. However, the ECG was nonspecific and did not correlate with the degree of elevation of the  $\overline{\text{PAP}}$ . This is consistent with other studies that evaluated

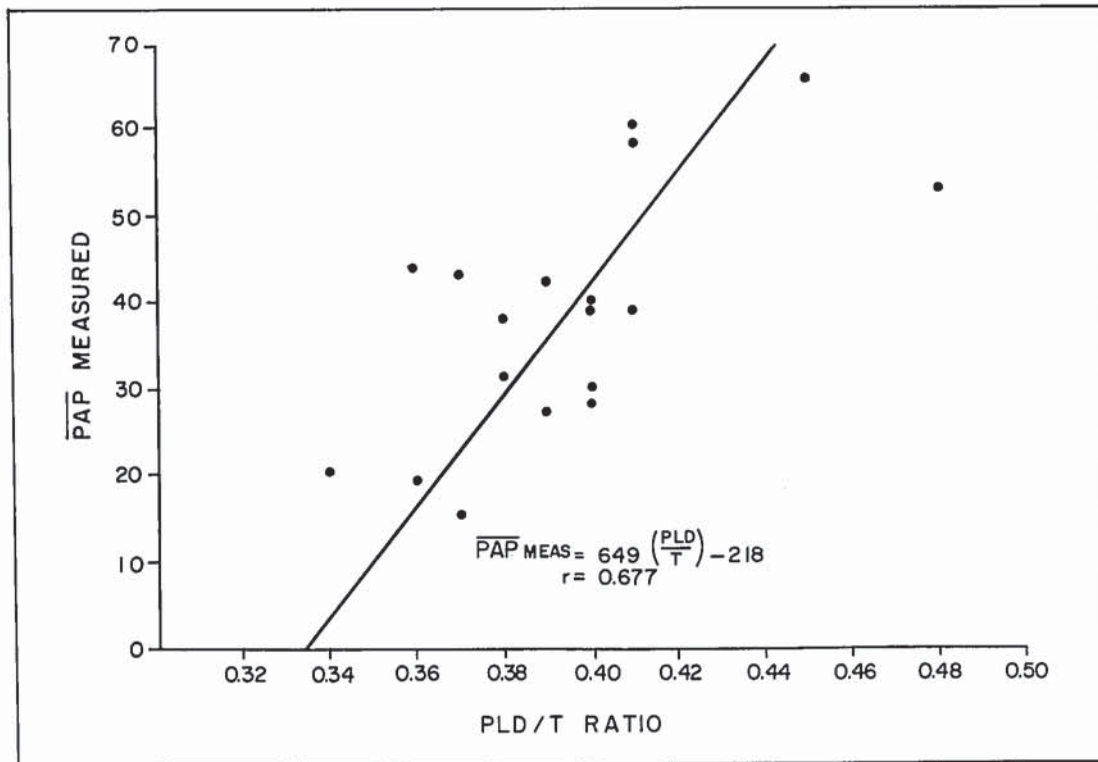


Fig. 2: Correlation of  $\overline{\text{PAP}}$  measured with pulmonary lobar diameter/thoracic (PLD/T) ratio for each individual patient with COPD.

the ECG in patients with COPD.<sup>2</sup> Right ventricular hypertrophy by ECG is a late manifestation of pulmonary hypertension but does not predict the degree of resting  $\overline{\text{PAP}}$  elevation.

Although the chest roentgenogram is qualitatively useful to demonstrate findings of pulmonary emphysema and enlarged main pulmonary vessels, it was not helpful in our study in quantitating the degree of pulmonary hypertension. The best correlation with  $\overline{\text{PAP}}$  was observed with the PLD/T ratio, which was abnormal in 12 of the 16 patients (75 percent) with pulmonary hypertension who had suitable roentgenograms. Roentgenograms in three patients were excluded from PLD/T ratio measurements because the pulmonary artery outlines near the hilar region were too indistinct to measure accurately.

Lupi and associates defined a PLD/T index > 38 percent as abnormal from a group of 150 patients with diverse cardiac disorders.<sup>5</sup> These investigators concluded that an "abnormal index suggested pulmonary arterial hypertension (PAH) but correlated poorly with the extent of hypertension." They did not study patients with COPD but they suggested that the PLD/T ratio might not be useful in COPD because the broadened lower thorax dimension could mask an enlarged pulmonary lobar diameter. Our data extend their findings and shows that the PLD/T ratio is useful in COPD to predict that pulmonary hypertension exists. However, this ratio is not sensitive enough to predict the  $\overline{\text{PAP}}$  in an individual patient. The PLD/T ratio correlated with the  $\overline{\text{PAP}}$  similarly in our patients with broadened or narrowed lower thorax dimensions.

The right descending branch of the pulmonary artery (RD-PA) was abnormally large in 19 of 20 patients, including two patients with normal  $\overline{\text{PAP}}$  values. The mean RD-PA value of 21.2 mm observed in our COPD patients is greater than the range for normal males 41 to 70 years old, which Chang reported as 10 to 16 mm, mean 14.3 mm.<sup>8</sup>

However, the size of the RD-PA in mm correlated poorly with the  $\overline{\text{PAP}}$ . We are not aware of any study which has measured RD-PA width in COPD subjects who did not have pulmonary hypertension. It is possible that the pulmonary arteries dilate in COPD before the development of pulmonary hypertension.

The level of hemoglobin varied widely among the COPD patients and did not correlate with the measured  $\overline{\text{PAP}}$ .

Kilcoyne and Ferrer claimed that "when the calculated values for  $\overline{\text{PAm}}$  were compared with the values obtained by cardiac catheterization in 20 patients, the agreement was excellent, the two determinations rarely varying by more than 5 mm."<sup>2</sup> Although there was an excellent agreement between the mean of the group of  $\overline{\text{PAP}}$  values measured and calculated in our series of COPD patients, the formula for calculated  $\overline{\text{PAP}}$  could not accurately predict the measured  $\overline{\text{PAP}}$  in individual patients. The  $\overline{\text{PAP}}$  calculated varied by 10 mm Hg from the  $\overline{\text{PAP}}$  measured in 7 of our 20 cases (35 percent) and was off by as much as 24 mm Hg. The formula was most accurate for our three patients with normal  $\overline{\text{PAP}}$  values.

It would be useful to be able to accurately calculate by noninvasive methods the right-sided pressures in patients with cor pulmonale. This would facilitate acute and chronic management programs. However, despite multiple attempts, this does not appear to be feasible. The  $\overline{\text{PAP}}$  is not solely dependent on one or two variables, eg,  $\text{PO}_2$  and pH. However, at  $\overline{\text{PAP}}$  values between 20 to 40 mm Hg, there is a good correlation between these determinants. Other noninvasive tests, eg, chest roentgenogram, hemoglobin, ECG, and pulmonary function tests do not change as rapidly as intravascular pressures are liable to.

Our study corroborates the results of Rizzato and associates who measured the right-sided pressures in 70 patients with COPD.<sup>7</sup> These investigators found a poor correlation between their measured  $\overline{\text{PAP}}$  values and  $\overline{\text{PAP}}$  values calculated by Enson's equation, especially when the  $\overline{\text{PAP}}$  was above 40 mm Hg. Whereas the mean  $\overline{\text{PAP}}$  calculated

and observed are close, the individual variation is wide, thereby limiting the equation's reliability for an individual patient.

In summary, we found the chest roentgenogram, electrocardiogram, hemoglobin, and arterial blood gases consistent with a diagnosis of pulmonary hypertension secondary to COPD. However, these noninvasive techniques were not very sensitive in predicting the level of  $\overline{\text{PAP}}$ , as determined by right heart catheterization.

#### Acknowledgment

The authors thank Clifford Rousseve, Paul Troop, and Lia Padilla for their assistance.

#### References

1. Enson Y, Giuntini C, Lewis ML, et al: The influence of hydrogen ion concentration and hypoxia on the pulmonary circulation. *J Clin Invest* 43:1146-1162, 1964.
2. Kilcoyne MM, Davis AL, Ferrer MI: A dynamic electrocardiographic concept useful in the diagnosis of cor pulmonale: Result of a survey of 200 patients with chronic obstructive pulmonary disease. *Circulation* 42:903-924, 1970.
3. Murphy ML, Boger J, Adamson JS, et al: Evaluation of cardiac size in chronic bronchitis and pulmonary emphysema. *Chest* 71:712-717, 1977.
4. Bone RC, Goheen JR, Ruth WE: Radiographic, hemodynamic and clinical comparison of pulmonary venous hypertension complicating acute respiratory failure in severe chronic airway obstruction. *Chest* 71 (suppl 1-6):284-287, 1977.
5. Lupi E, Dumont C, Tejada VM, et al: A radiologic index of pulmonary arterial hypertension. *Chest* 68:28-31, 1975.
6. Burrows B, Kettel LJ, Niden AH, et al: Patterns of cardiovascular dysfunction in chronic obstructive lung disease. *N Engl J Med* 286:912-918, 1972.
7. Rizzato GF, Rampulla C, Mandelli F, et al: Can pulmonary artery pressure be predicted without right heart catheterization in chronic obstructive lung disease? *Acta Cardiol* 30:251-265, 1975.
8. Chang CH: The normal roentgenographic measurement of the right descending pulmonary artery in 1,085 cases. *Am J Roent* 87:929-935, 1962.
9. Abraham AS, Cole RB, Bishop JM: Reversal of pulmonary hypertension by prolonged oxygen administration to patients with chronic bronchitis. *Circulation Research* 23:147-157, 1968.
10. Harvey RM, Enson Y, Betti R, et al: Further observations on the effect of hydrogen ion on the pulmonary circulation. *Circulation* 35:1019-1027, 1967.
11. Ferrer MI: Cor pulmonale (pulmonary heart disease): Present-day status. *Am Heart J* 89:657-664, 1975.
12. Abraham AS, Cole RB, Green ID, et al: Factors contributing to the reversible pulmonary hypertension of patients with acute respiratory failure studied by serial observations during recovery. *Circulation Research* 24:51-60, 1969.
13. Nash ES, Briscoe WA, Cournand A: The relationship between clinical and physiological findings in chronic obstructive disease of the lungs. *Med Thorac* 22:305-327, 1965.