

Use of other physiological variables to predict pulmonary arterial pressure in patients with chronic respiratory disease

Multicentre study

REPORT PREPARED BY J. M. BISHOP AND K. W. CROSS

Departments of Medicine and Social Medicine, University of Birmingham, Edgbaston, Birmingham B15 2TH, U.K.

KEY WORDS: Pulmonary arterial pressure, pulmonary hypertension, chronic respiratory disease, pulmonary function tests, arterial blood gases.

Chronic pulmonary disease may lead to the development of pulmonary arterial hypertension with subsequent hypertrophy of the right ventricle. Pulmonary heart disease caused in this way is a major category of heart disease, but it is often unrecognized, and there is little information about its prevalence or natural history.

A collaborative group was established by the European Office of the World Health Organization to develop research into these problems. The first objective of the group is to develop and validate non-invasive methods for measuring the degree of pulmonary arterial hypertension. Secondary objectives are to apply these procedures to determine the prevalence of pulmonary hypertension in various groups of patients, to study the

natural history of pulmonary hypertension and pulmonary heart disease and finally its response to treatment.

Pulmonary arterial hypertension cannot be reliably recognized, nor its degree of severity determined by clinical methods alone. Cardiac catheterization makes this possible but invasive procedures are not suitable for studies of prevalence, and have limitations where repeated measurements are required. Various non-invasive methods for estimating pulmonary arterial pressure have been developed, but the collaborative group did not consider that any single one had been shown to be sufficiently reliable for the present purposes. Several of these non-invasive methods are therefore being studied further. The present report, which is complementary to these studies, describes a retrospective analysis to determine the extent to which it is possible to approximate pulmonary arterial mean pressure from other measured physiological variables, either singly or in combination, in several types of chronic respiratory disease.

Methods

Patients were initially categorized as suffering from obstructive, restrictive or thromboembolic disease, based upon clinical criteria, and evidence from tests of pulmonary function as follows:

(1) Patients with chronic obstructive lung disease, defined in the usual ways by clinical and physiological methods. Patients to be included would have either chronic bronchitis, characterized by cough and sputum, during at least three consecutive months for each of two consecutive years, or emphysema, characterized by marked dyspnoea

Received for publication 27 February 1981; and in revised form 24 April 1981.

Centres participating in this study were:

Birmingham: Department of Medicine, Queen Elizabeth Hospital (Professor J. M. Bishop).

Budapest: Koranyi National Institute of Tuberculosis and Pulmonology (Professor I. Hutás).

Lyons: Department of Cardiac and Pulmonary Investigation, Saint Joseph Hospital (Dr M. Tartulier).

Nancy: National Institute of Health and Medical Research (Professor P. Sadoul).

Prague: Department of Medicine II, Institute for Clinical and Experimental Medicine (Professor J. Widimsky).
Institute of Physiological Regulations, Czechoslovak Academy of Sciences (Dr V. Jezek).

Strasbourg: Pavillon Laennec, University Hospital (Dr E. Weitzenblum).

Warsaw: Department of Medicine, Institute of Tuberculosis (Dr J. Zielinski).

Convened by Dr G. Lamm, World Health Organization Regional Office for Europe, Copenhagen.

Requests for reprints to: Professor J. M. Bishop, Department of Medicine, Queen Elizabeth Hospital, Birmingham B15 2TH, U.K.

Table 1 *Number of patients by centre and diagnostic group*

Diagnostic group	Centre								Total
	1	2	3	4	5	6	7	8	
1. Obstructive	86	1	77	76	151	175	29	—	595
2. Fibrosing alveolitis	3	—	43	10	1	8	5	42	112
3. Sarcoid	—	—	5	—	—	13	1	—	19
4. Tuberculosis	—	43	76	1	—	2	—	—	122
5. Silicosis	—	—	49	6	—	1	1	—	57
6. Thromboembolism	1	—	42	7	—	1	—	—	51

with radiological evidence of emphysema and functional evidence of airways obstruction.

(2) Patients with diffuse interstitial lung disease (pulmonary fibrosis). Diseases in this group, characterized by restrictive changes with increased elastic recoil, may either be caused by exogenous factors, or be of unknown origin. The diagnosis was made radiologically and according to the presence of functional changes, including decreased vital capacity, transfer factor for carbon monoxide and static compliance, without features of obstruction. In some patients the diagnosis was confirmed by lung biopsy or at autopsy.

(3) Patients with thromboembolic disease. This disorder is indicated by the presence of attacks of paroxysmal dyspnoea, pleurisy, haemoptysis due to pulmonary infarction, or it may present as progressive exercise dyspnoea. There should be no evidence of other parenchymal lung disease. The condition should be confirmed by lung scan and/or pulmonary angiography.

Patients were excluded if they had evidence of cardiac failure, rheumatic or ischaemic heart disease, systemic hypertension, gross thoracic deformity, bronchial asthma, recent chest infection or other serious illness.

In the subsequent analysis the patients have been classified according to whether they had chronic obstructive lung disease (Group 1) or chronic thromboembolic disease (Group 6).

Patients in the restrictive group were placed into one of four subgroups according to whether they had chronic fibrosing alveolitis (Group 2), sarcoid lung disease (Group 3), chronic pulmonary tuberculosis (Group 4) or silicosis (Group 5). Patients with tuberculosis had inactive disease and any who underwent lung resection or thoracoplasty were excluded. Data were available from a total of

956 patients who had been investigated in one of the eight participating centres, and the composition of the groups is shown in Table 1.

TECHNICAL PROCEDURES

Each patient had undergone right heart catheterization for an indication determined by the centre concerned. Catheterization was performed in the supine position, in the resting state and pulmonary arterial mean pressure was determined by planimetry or electrical integration. Cardiac output was measured by the direct Fick Method, in a steady state.

The other physiological measurements were made within four weeks of cardiac catheterization, by methods which were currently in use in the various centres. Those which have been used in the present analysis are the arterial oxygen saturation, arterial oxygen tension and arterial carbon dioxide tension, forced expiratory volume at one second, vital capacity, packed cell volume, haemoglobin concentration and cardiac output. Complete sets of data were not obtained in all patients, and the numbers are shown in the tables.

Patients were in a steady clinical state at the time of investigation and were free of acute respiratory infection, oedema or other signs of cardiac failure. The technical procedures used were not identical in all centres, but the procedures were well established in the individual centres, all of which are active research centres dealing with large numbers of patients with respiratory disease. The indications for cardiac catheterization varied between the centres and patients were consequently selected according to different criteria. This resulted in patients with a wide range of pulmonary arterial mean pressure being included, which proved advantageous for the present investigation.

Table 2. Mean values with standard deviation and number of patients in six diagnostic groups

	\overline{PA}	SaO ₂	PaO ₂	PaCO ₂	Hb	PCV	FEV _{1.0}	VC	FEV _{1.0} /FVC%	Q
1. Obstructive	24.39	89.14	63.53	44.06	14.79	45.94	1.54	3.12	45.94	3.30
	11.47	8.34	11.88	20.27	2.34	8.06	0.97	1.14	14.97	0.92
	595	580	493	571	421	469	573	567	570	557
2. Fibrosing aveolitis	26.25	90.13	69.38	37.88	14.84	45.16	1.65	2.23	73.07	3.38
	12.29	6.43	13.13	5.46	2.34	9.38	0.76	0.92	12.61	1.05
	112	112	92	98	112	81	111	115	109	95
3. Sarcoid	16.26	95.50	79.00	36.53	13.69	39.62	2.48	3.31	71.60	3.74
	6.36	2.04	11.04	3.13	1.57	4.96	1.39	1.59	14.62	0.73
	19	20	19	19	20	13	20	20	20	14
4. Tuberculosis	22.45	90.31	71.30	43.77	14.62	44.19	1.30	2.35	56.63	3.32
	9.35	5.49	9.07	6.96	1.81	5.88	0.67	0.90	16.85	0.97
	122	91	40	44	84	52	107	121	110	86
5. Silicosis	19.42	91.21	75.29	38.61	15.90	46.60	2.05	3.08	64.41	2.70
	10.90	4.79	12.58	5.40	1.88	4.58	0.93	0.94	15.10	0.49
	57	57	17	23	57	48	56	58	56	52
6. Thromboembolic	29.10	93.60	78.81	33.94	14.68	44.84	2.51	3.66	68.00	3.12
	19.84	4.78	13.74	6.29	1.71	6.15	0.91	1.12	10.33	1.01
	51	47	41	46	49	19	51	51	51	49

\overline{PA} = pulmonary arterial mean pressure (mm Hg); PaO₂ = arterial oxygen tension (mm Hg); Hb = haemoglobin concentration (g/100 ml); FEV_{1.0} = forced expiratory volume in 1 s (litres); SaO₂ = arterial oxygen saturation (%); PaCO₂ = arterial carbon dioxide tension (mm Hg); PCV = packed cell volume (%); VC = vital capacity (litres); FEV_{1.0}/FVC% = ratio (%); Q = cardiac output (l/min/m²).

Table 3 Pulmonary arterial pressure by age group and by diagnostic group

Sex and age group	Obstructive disease		Fibrosing alveolitis		Pulmonary tuberculosis	
	No. of patients	Mean \pm s.d.	No. of patients	Mean \pm s.d.	No. of patients	Mean \pm s.d.
<i>Males</i>						
<40	17	25.14 \pm 15.70	79	27.05 \pm 13.19	16	19.69 \pm 8.57
40-44	45	23.11 \pm 15.54		3		22.50 \pm 10.52
45-49	57	23.88 \pm 10.03	8		26.37 \pm 9.80	26
50-54	78	24.37 \pm 9.96		6	28.33 \pm 16.70	
55-59	130	24.04 \pm 10.37	6			12
60-64	111	25.22 \pm 11.51				
65-69	63	22.29 \pm 9.22				
70+	40	24.17 \pm 10.59				
<i>Females</i>						
<30	5	30.40 \pm 14.01	22	21.36 \pm 5.45	15	24.47 \pm 13.97
30-39			17	27.18 \pm 16.36		
40-49	11	30.00 \pm 14.24	14	28.71 \pm 9.80	11	25.54 \pm 12.63
50-59	18	19.94 \pm 7.89	13	32.32 \pm 16.02	13	26.08 \pm 11.03
60+	12	31.25 \pm 18.37	6	21.50 \pm 10.21		

Results

Mean values for 10 physiological variables in the six diagnostic groups are given in Table 2. Initial analysis showed that there were minor trends in the effect of age on pulmonary arterial mean pressure (Table 3) but none of these was significant and this variable has not been taken into account in subsequent analysis.

Mean pressures in females slightly exceeded those in males for each diagnostic group and each age group. The differences were small and seem likely to have resulted from the way in which patients were selected for study by different centres. In subsequent analysis the results have therefore been pooled.

CORRELATION COEFFICIENTS

The extent to which pulmonary arterial pressure was associated with each of the other nine variables was examined by calculating Spearman correlation coefficients; these coefficients were used because the variables were not normally distributed. A single regression analysis of pulmonary arterial pressure on each of the variables was followed by a stepwise multiple regression analysis. In addition to the regression equations provided by these conventional analyses the proportions of the variances in the dependent variable (pulmonary arterial pressure) accounted for by the regression equations were also obtained although the significance of each

regression coefficient could not be tested because of the non-normality of the data.

Table 4 shows correlation coefficients for the PA mean pressure against each of the nine variables, for each of the six diagnostic groups. The significant correlations for Group 1 are more important since they are based upon larger numbers of patients.

In Group 1 (obstructive disease) highly significant correlations were found with all variables except cardiac output. These variables may be grouped into indices of respiratory gas exchange (PaO_2 , SaO_2 , PaCO_2), variables indirectly related to respiratory gas exchange (Hb and PCV) and indices of lung volume (FEV_{10} , VC and FEV/VC %). PA pressure correlated most highly with indices of respiratory gas exchange and somewhat less highly with the indices of lung volume.

In the patients with fibrosing alveolitis PA pressure correlated with SaO_2 and PaO_2 but not PaCO_2 . There was no correlation with indices of lung volume. By contrast in the small group of patients with sarcoidosis there were correlations with indices of lung volume and not with those indicating respiratory gas exchange.

In the groups of patients with tuberculosis and silicosis correlations were found with SaO_2 and with restriction of lung volume. In both conditions correlations with lung volume somewhat exceeded those for SaO_2 .

In the case of patients with thromboembolic disease PA pressure correlated with SaO_2 and PaO_2 .

Table 4 Spearman's correlation coefficient for pulmonary arterial mean pressure against other physiological variables in six diagnostic groups

	SaO ₂	PaO ₂	PaCO ₂	Hb	PCV	FEV _{1,0}	VC	FEV/VC%	Q
1. Obstructive	-0.514***	-0.526***	0.569***	0.111**	0.228***	-0.386***	-0.380***	-0.271***	0.057
2. Fibrosing alveolitis	-0.594***	-0.444***	-0.037	0.404***	0.375***	0.082	0.033	0.089	0.001
3. Sarcoidosis	-0.212	-0.342	-0.212	-0.199	-0.109	-0.879***	-0.825***	-0.678***	-0.205
4. Tuberculosis	-0.338***	-0.248	0.316*	-0.189*	-0.228*	-0.542***	-0.379***	-0.118	-0.120
5. Silicosis	-0.535***	-0.285	0.401	-0.016	-0.250	-0.684***	-0.626***	-0.433***	-0.268*
6. Thromboembolic	-0.467***	-0.560***	0.215	0.129	0.327*	-0.260	-0.189	-0.345*	-0.223

*P < 0.05, **P < 0.01, ***P < 0.001.

Table 5 Regression equations for arterial oxygen saturation, and forced expiratory volume on pulmonary arterial mean pressure in six diagnostic groups

	SaO ₂	FEV _{1.0}
1. Obstructive	PAP = 100.74 - 0.859 SaO ₂	PAP = 28.91 - 3.208 FEV
2. Fibrosing alveolitis	PAP = 113.09 - 0.962 SaO ₂	PAP = 24.24 - 1.186 FEV
3. Sarcoid	PAP = 165.04 - 1.557 SaO ₂	PAP = 25.33 - 3.680 FEV
4. Tuberculosis	PAP = 97.64 - 0.847 SaO ₂	PAP = 31.08 - 6.207 FEV
5. Silicosis	PAP = 114.51 - 1.042 SaO ₂	PAP = 31.75 - 5.870 FEV
6. Thromboembolic	PAP = 163.81 - 1.439 SaO ₂	PAP = 44.80 - 6.250 FEV

but not with PaCO₂ and there was no significant correlation with indices of lung volume, apart from a weak correlation with FEV/VC%.

REGRESSION ANALYSIS

Regression analysis was undertaken for each of seven of the variables against pulmonary arterial mean pressure for each diagnostic group. FEV/VC % and cardiac output were omitted from this analysis in view of the earlier findings. Table 5 gives the regression equations of pulmonary arterial mean pressure on arterial oxygen saturation and forced expiratory volume.

Comparison of the three groups containing the largest number of patients showed that the slopes of the regression lines for PAP against SaO₂ were similar but that for fibrosing alveolitis was a little steeper than that for tuberculosis which was in turn somewhat steeper than that for patients with obstructive disease.

MULTIPLE REGRESSION ANALYSIS

This was undertaken for patients with obstructive disease (Group 1), fibrosing alveolitis (Group 2) and tuberculosis (Group 4), as the other groups were considered too small for this analysis.

For Group 1 the most highly correlated variable was SaO₂. When this variable was used in a stepwise multiple regression analysis the regression equation was:

$$\text{PAP} = 98.59 - 0.841 \text{ SaO}_2$$

Prediction of PAP by this equation explained 42.4% of the variance in PAP values. When the variables packed cell volume and FEV₁ were successively incorporated into the multiple regression a further 1.2% and 1.1% of the variance respectively could be accounted for. The final regression equation was then

$$\text{PAP} = 83.19 - 0.736 \text{ SaO}_2 + 0.173 \text{ PCV} - 1.208 \text{ FEV}_1$$

Table 6 Percentage of patients with obstructive disease with values for arterial oxygen saturation at specified levels who had pulmonary arterial mean pressure within particular range

Arterial oxygen saturation (%)	Pulmonary arterial mean pressure (mm Hg)		
	<20	20-29	>29
<85	7.0	30.4	61.6
85-89.9	27.0	49.5	23.5
90-93.9	46.7	38.0	15.3
>94	61.3	33.8	4.8

For the group with fibrosing alveolitis the most highly correlated variable was again SaO₂. The regression equation was

$$\text{PAP} = 113.33 - 0.964 \text{ SaO}_2$$

This equation accounted for 25.2% of the variance in mean PA pressure. When the variable haemoglobin was incorporated into the equation a further 1.7% of the variance could be accounted for. Addition of further variables did not improve the precision of the prediction. The final regression equation was:

$$\text{PAP} = 89.792 - 0.830 \text{ SaO}_2 + 0.771 \text{ Hb}$$

In the group with tuberculosis the variable correlating most highly was again SaO₂ and the regression equation was:

$$\text{PAP} = 104.67 - 0.916 \text{ SaO}_2$$

This equation accounted for 19.0% of the variance in PAP values. The incorporation of FEV₁ into the multiple regression accounted for a further 10.8% of the variance, but inclusion of other variables did not improve the prediction further. The final regression was:

$$\text{PAP} = 97.484 - 0.768 \text{ SaO}_2 - 4.451 \text{ FEV}_1$$

Table 7 Percentage of patients in four diagnostic groups with arterial oxygen saturation at specified levels who had pulmonary arterial mean pressure in each of three ranges

Arterial oxygen Saturation (%)	Pulmonary arterial mean pressure (mm Hg)											
	<20				20-29				>29			
	Group: 1	2	4	6	1	2	4	6	1	2	4	6
<85	7.0	(0)	(12.5)	(0)	30.4	(27.8)	(25.0)	(0)	61.6	(72.2)	(62.5)	(100)
85-89.9	27.0	4.5	28.6	(37.5)	49.5	54.5	71.4	(12.5)	23.5	40.9	0	(50.0)
90-93.9	46.7	46.4	54.1	(50.0)	38.0	25.0	43.2	(12.5)	15.3	28.6	2.7	(37.5)
>94	61.3	52.4	69.6	56.7	33.8	40.5	21.7	23.3	4.8	7.1	8.7	20.0

Figures in parentheses are based upon less than 20 patients at the specified level of arterial oxygen saturation. Group 1 = obstructive; Group 2 = fibrosing alveolitis; Group 4 = tuberculosis; Group 6 = thromboembolic.

Table 8 Percentage of patients with obstructive disease with values for FEV₁₋₀ at specified levels who had pulmonary arterial mean pressure within particular range

FEV ₁₋₀ (litres)	Pulmonary arterial mean pressure (mm Hg)		
	<20	20-29	>29
<0.7	10.4	34.9	54.7
0.7-0.99	25.6	38.8	35.7
1.0-1.29	41.8	40.5	17.7
>1.3	57.6	35.1	8.3

THRESHOLD ANALYSIS

The purpose of this analysis was firstly to determine the proportion of patients with values of arterial oxygen saturation at a certain threshold level who had either a normal pulmonary artery pressure or a specified degree of pulmonary hypertension. There were sufficient patients to allow the analysis in the patients with obstructive disease (Group 1) and those with fibrosing alveolitis (Group 2), tuberculosis (Group 4) and thromboembolic disease (Group 6). An example of the analysis is shown in Table 6 for patients with obstructive lung disease. Of the patients in whom arterial oxygen saturation was less than 85%, 61% had a mean PA pressure exceeding 29 mm Hg and only 7% had a PA pressure which was less than 20 mm Hg. At the other end of the scale in those patients in whom arterial oxygen saturation exceeded 94%, only 5% had a mean PA pressure greater than 30 mm Hg and 61% had a pressure less than 20 mm Hg. Results expressed in the same form for four groups of patients are compared in Table 7. In patients with mild to moderate

degrees of hypoxaemia pulmonary hypertension was more frequent in fibrosing alveolitis than in the other groups, and normal pulmonary arterial pressure was less frequent.

A similar analysis was performed in relation to FEV₁₋₀ for Groups 1, 2 and 4. Of patients with obstructive disease (Table 8) whose FEV₁₋₀ was less than 0.7 l, 54.7% had a PA mean pressure greater than 29 mm Hg, and 10.4% had a PA mean pressure less than 20 mm Hg. If FEV₁₋₀ was greater than 1.3 l only 8.2% had a PA mean pressure greater than 29 mm Hg and 57.6% had a value less than 20 mm Hg.

The effects of age upon this relationship in patients with obstructive disease was examined. Table 9 shows the results of analysis in three age groups as indicated, and little difference is evident between the three groups. A linear regression analysis of each of these contingency tables revealed no statistically significant trend of pulmonary arterial pressure with age for a given level of FEV₁. The greatest tendency towards such a trend occurred in FEV₁ group 0.7 to 0.99 but this

Table 9 Effect of age upon percentage of patients with obstructive disease with values for FEV_{1.0} at specified levels who had pulmonary arterial mean pressure within particular range

FEV _{1.0} (litres)		Pulmonary arterial mean pressure (mm Hg)		
		<20	20-29	>29
<0.7	a	(5.9)	(52.9)	(41.2)
	b	5.6	30.6	63.8
	c	18.2	30.3	51.5
0.7-0.99	a	(11.1)	(27.8)	(61.1)
	b	31.4	34.3	34.3
	c	26.3	43.4	30.3
1.0-1.29	a	30.0	50.0	20.0
	b	45.8	41.7	12.5
	c	45.7	34.3	20.0
>1.3	a	60.5	32.9	6.6
	b	53.3	38.5	8.2
	c	59.3	30.9	9.8

Age group: a = less than 49 years; b = 50-59; c = greater than 60.

Figures in parentheses are based upon less than 20 patients at the specified level of forced expiratory volume.

Table 10 Percentage of patients in three diagnostic groups with FEV_{1.0} at specified levels who had pulmonary arterial mean pressure in each of three ranges

FEV _{1.0} (litres)	Group:	Pulmonary arterial mean pressure (mm Hg)								
		<20			20-29			>29		
		1	2	4	1	2	4	1	2	4
<0.7		10.4	(20.0)	(0)	34.9	(40.0)	(46.7)	54.7	(40.0)	(53.3)
0.7-0.99		25.6	(27.8)	26.9	38.8	(61.1)	53.8	35.7	(11.1)	19.2
1.0-1.29		41.8	(38.9)	47.6	40.5	(27.8)	47.6	17.7	(33.3)	4.8
>1.3		57.6	34.3	53.3	35.1	34.3	40.0	8.3	31.3	6.7

Figures in parentheses are based upon less than 20 patients at the specified level of forced expiratory volume.

Group 1 = obstructive; Group 2 = fibrosing alveolitis; Group 4 = tuberculosis.

did not achieve the usual levels of statistical significance.

The findings in obstructive disease are compared with the other diagnostic groups in Table 10 in which data are presented in the same form as Table 8. Patients with fibrosing alveolitis were more likely to have pulmonary hypertension with a relatively well preserved FEV_{1.0} than patients in the other diagnostic groups.

Discussion

The purpose of this investigation was to

determine the extent to which it would be possible to predict pulmonary arterial mean pressure from those measurements of pulmonary function which are commonly made in patients with chronic respiratory disease. The study was a retrospective one and its technical and logical limitations must be recognized. Patients were recruited from eight centres in five different countries and they had been studied over a period of several years. Indications for the initial investigation varied between the centres so that the kinds of patient and the severity of their disease differed widely between centres.

The data presented represent the largest collection so far published for the purpose, and furthermore relate not only to obstructive lung disease but also to other chronic respiratory diseases as well. It is well recognized that correlations and predictions made from such data relate only to the patients included, and that predictions cannot necessarily be applied to new, prospectively studied, groups of patients. Furthermore the data for each patient relates only to one instant in time, and it is accepted that serial observations demonstrating trends with time might well provide more reliable predictions.

In the case of patients with obstructive disease our results are essentially in line with those of previous workers⁽¹⁻³⁾. Of the physiological variables studied by far the best correlation was found with arterial oxygen saturation. Using this variable alone it was possible to explain 42% of the variance in PA pressure values. This is a considerably greater proportion of the variance than was explained in the studies of Rizzato *et al.*⁽⁵⁾ but less than explicable in those of Simon *et al.*⁽⁴⁾. Addition of other variables into a multiple regression analysis added little to the certainty with which PA pressure could be predicted. Earlier workers incorporated body surface area, hydrogen ion concentration and residual volume and found that they added small amounts to the precision of the prediction. These variables were not available in our studies, but unlike previous workers we did have FEV₁₋₀. Simon *et al.*⁽⁴⁾ found that age was a significant factor but this was not so in the present investigation.

We were eventually able to explain 44.7% of the variance in PA pressure measurements by the multiple regression equation incorporating arterial oxygen saturation, packed cell volume and FEV₁₋₀. It would have been of interest to express this result in terms of the confidence limits for the predicted value for PA pressure. This would only have been possible if the PA pressure values had been normally distributed but this was manifestly not the case in our data.

Few attempts to predict PA pressure by this method in other diseases have been reported. In the case of fibrosing alveolitis arterial oxygen saturation was again the best predictor but accounted for only 25.2% of the variance with very little being added by the incorporation of other variables. In

the case of tuberculosis arterial oxygen saturation was again the best predictor but here the addition of FEV₁ brought the total proportion of the variance that was explicable to 29.8%. The latter finding is consistent with the observations of Naszlady *et al.*⁽⁶⁾.

With the reservations expressed above, it may be concluded that these regression equations, together with the statements possible from consideration of threshold values, allow predictions to be made in groups of patients. The precision of the prediction of mean PA pressure in patients with obstructive disease is quite high, with 45% of the variance being explicable. This could provide a useful means of selecting patients in whom direct measurement is likely to reveal pulmonary hypertension. On the other hand the use of the equation alone would result in too large an error to permit accurate prediction of PA pressure in an individual. We are investigating in a prospective study the possibility that addition of measurements made by other independent non-invasive techniques may allow an approach to be made to a more useful and precise prediction.

Many research workers took part in the investigations which produced the data forming the basis of this report. They are too numerous to mention individually, but we acknowledge the importance of their skilful efforts.

References

- [1] Enson Y, Giuntini C, Lewis M, Morris T, Ferrer M, Harvey R. The influence of hydrogen ion concentration and hypoxia on the pulmonary circulation. *J Clin Invest* 1964; 43: 1146-62.
- [2] Grassi V, Innocenti P, Toso M, Timio M, Muiasan G. Broncopneumopatie croniche ed ipertensione arteriosa polmonare. *Mal Tor Cardiovasc* 1969; 5: 16-26.
- [3] Lockhart A. Hemodynamique pulmonaire dans la bronchite chronique. *Bull Physiopath Resp* 1973; 9: 1069-99.
- [4] Simon H, Fink H, Fricke G, Ferlinz R, Kikis D. Einfluss der Blutgaspartialdrucke und der Lungenfunktionsparameter auf den Pulmonal-arterienmitteldruck beim obstruktiven Syndrom. *Klin Wochenschr* 1973; 51: 362-4.
- [5] Rizzato GF, Rampulla C, Mandelli V, Benza GC, Mantero O, Morpurgo M. Can pulmonary artery pressure be predicted without right heart catheterisation in chronic obstructive lung disease? *Acta Cardiol* 1975; 4: 251-65.
- [6] Naszlady A, Littauer A, Varkonyi J, Levendel L. Long-term study of pulmonary hypertension in chronic obstructive bronchopulmonary disease. *Progr Resp Res* 1975; 9: 29-36.

ould be possible
n pressure from
function which
s with chronic
a retrospective
imitations must
aited from eight
d they had been
ears. Indications
ed between the
and the severity
een centres.