

A study of pulmonary artery pressure, electrocardiography, and mechanocardiography in thoracic scoliosis

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Shneerson, J. M., Venco, A., and Prime, F. J. (1977). Thorax, 32, 700–705. A study of pulmonary artery pressure, electrocardiography, and mechanocardiography in thoracic scoliosis. Cardiac catheterisation was carried out in 40 patients with thoracic scoliosis in order to measure the pulmonary artery pressure. Statistical correlations were calculated between these results and the electrocardiographic and mechanocardiographic findings determined on a separate occasion. The pulmonary artery pressure was normal in 72% of subjects. It was inversely correlated with arterial oxygen tension but not with the aetiology, severity, or age at onset of the scoliosis. The accuracy of electrocardiography and mechanocardiography in predicting the pulmonary artery pressure was assessed. The closest correlates were found to be a tall P wave in lead II or III and a prolonged interval between pulmonary valve closure and tricuspid valve opening.

Clinicians and morbid anatomists in the early 19th century realised that enlargement of the heart was often associated with spinal deformity (Corvisart, 1806; Harrison, 1820). This impression was confirmed by several series of necropsies during the last century. Bachmann (1899) found right ventricular hypertrophy in 82% of 197 kyphoscoliotics. Barié (1904) was the first to attribute this hypertrophy to a raised pulmonary artery pressure, but the latter could not be measured until the advent of cardiac catheterisation for clinical use.

Bloomfield *et al.* (1946) were the first to use this technique in scoliotics. They, and several later groups (eg, Schaub *et al.*, 1954; Bergofsky *et al.*, 1959), found that pulmonary hypertension did commonly occur.

Bergofsky *et al.* (1959), by demonstrating that the wedge pressure and cardiac output were normal, showed that the pulmonary hypertension was due primarily to an increase in the pulmonary vascular resistance. They thought that this was the result of compression and kinking of small vessels in the lungs. However, Schaub *et al.* (1954) emphasised the importance of hypoxic vasoconstriction, and Reid (1965) demonstrated that the anatomical dimensions of the pulmonary vascular bed were much reduced. However, insufficient physiological data are available to differentiate between these possibilities.

Electrocardiography was first attempted systematically in scoliotics by Adorno and White (1945). They found right axis deviation in 17% of young asymptomatic scoliotics. Towers and Zorab (1969), in a study of 168 young subjects, found that both right and left axis deviation were more common than normal and that high-voltage QRS complexes were common in V_2 – V_4 .

However, in neither of these reports were the findings compared with either the pulmonary artery pressure measured *in vivo* or right ventricular hypertrophy observed *post mortem*. The present paper reports data relating the pulmonary artery pressure to electrocardiographic and mechanocardiographic findings in order to assess their sensitivity and reliability as predictors of pulmonary artery pressure. In view of the abnormal shape of the thorax, which might introduce artefacts into the results, the observed pulmonary artery pressures have also been related to various details of the scoliosis and to the vital capacity of the subject.

Subjects

Forty patients with thoracic scoliosis and unexplained dyspnoea or suspected pulmonary hypertension were studied. All gave informed consent to the investigations. The personal details of the

subjects are shown in Table 1. Twenty-five were female and 15 male. Twenty-four had previously undergone spinal fusion. The aetiology of the scoliosis was congenital (6), idiopathic (14), post-polio myelitis (8), associated with Marfan's syndrome (3), neurofibromatosis (3), fragilitas ossium (1), spinal muscular atrophy (1), arthrogryposis (1), Friedreich's ataxia (1), peroneal muscular atrophy (1), and polymyositis (1). There was no evidence of cardiac or respiratory disorders except for an ejection systolic murmur in three subjects (MH, MB, SPe) and moderately severe chronic obstructive bronchitis in one (SBa).

Table 1 Personal details of subjects

	n	Range	Mean	SD
Age (years)	40	13- 67	30.0	13.1
Age at onset of deformity (years)	40	0- 16	7.0	5.1
Angle of scoliosis (degrees)	40	10-140	75.3	29.7
Vital capacity (litres)	40	0.55-3.75	1.52	0.74

Methods

The angle of scoliosis was determined by the method of Cobb (1948). The vital capacity was measured with a dry spirometer (Vitalograph). A standard 12-lead electrocardiogram was recorded at 25 mm/s. Phonocardiograms were obtained with a piezo-electric crystal microphone and the sounds filtered to a mid- and high-frequency spectrum. A carotid pulse tracing was made simultaneously. Where technically possible, right apex cardiograms (ACG) and jugular venous pulses (JVP) were recorded with a Cambridge Scientific Instruments pulse transducer with a time constant of 4 s and a lower frequency limit of 0.04 Hz. Echocardiograms were recorded with a Smith-Kline 20 ultrasonoscope using a 2.25 MHz 1.25 cm probe. All the outputs were displayed on a Cambridge multi-channel photographic recorder at a paper speed of 100 mm/s, with a simultaneous ECG trace.

On the next morning, at least two hours after a light breakfast, a saline-filled Bradley (1964) catheter was floated into the pulmonary artery and connected to a Consolidated Electrodynamics strain gauge manometer (4-326-L212). The output was amplified by a Devices DC2 amplifier. The response of the system to applied pressures was linear and was flat to 12 Hz. Mean pressures were obtained by electrical damping. The pressures were recorded with the patient supine, and the zero reference level was taken as 5 cm behind the sternal angle. A sample of arterial blood was withdrawn from a radial artery for gas analysis on an

IL 213 blood-gas analyser while the arterial pressures were recorded. Sixteen subjects then inspired 100% O₂ for at least five minutes and the change in mean pulmonary artery pressure was also recorded.

A systolic pressure of 30 mmHg or more and a mean pressure of 20 mmHg or more were regarded as abnormal. Electrocardiographic criteria of right heart involvement were as follows:

- (1) P wave 2.5 mm or taller in leads II or III.
- (2) Electrical axis of +91° or more.
- (3) R/S ratio >1 in leads V₁ or V₂.
- (4) T-Wave inversion in leads V₂ or V₃.

The relative intensity of A₂ and P₂ in the pulmonary area was observed phonocardiographically. The interval from pulmonary valve closure to tricuspid valve opening (P₂-T₀) was calculated from the phonocardiogram and the O point of the ACG, the U point of the JVP, or the start of valve opening on the echocardiogram.

Results

CATHETERISATION

The pulmonary artery pressure and blood gas results are shown in Table 2. Twenty-nine of the 40 patients had a mean pulmonary artery pressure less than 20 mmHg and 31 a systolic pressure less than 30 mmHg. An RV-PA gradient was present in MH (10 mmHg), MB (12 mmHg), and SPe (11 mmHg).

There was no correlation between the mean pulmonary artery pressure and the aetiology, severity (Fig. 1), or age at onset of the deformity. It was also unrelated to the vital capacity (Fig. 2). However, the mean pulmonary artery pressure rose as the Pao₂ fell, especially when this was below 70 mmHg (Fig. 3).

Sixteen subjects inhaled pure oxygen. The change in pulmonary artery pressure ranged from +1 to -16 mmHg (mean -3.0; SD 4.1). The magnitude of the pressure change was not related to either the Pao₂ or the mean pulmonary artery pressure immediately preceding oxygen administration.

Table 2 Pulmonary artery pressures and arterial gas analyses

		n	Range	Mean	SD
PA pressure (mmHg)	Systolic	40	14- 58	25.3	9.4
	Mean	40	10- 43	17.2	6.9
	Diastolic	40	6- 32	12.4	5.5
Pao ₂ (mmHg)		36	42.7-101.4	79.7	14.6
Paco ₂ (mmHg)		36	33.0-89.6	43.9	10.7

1 mmHg=0.133 kPa.

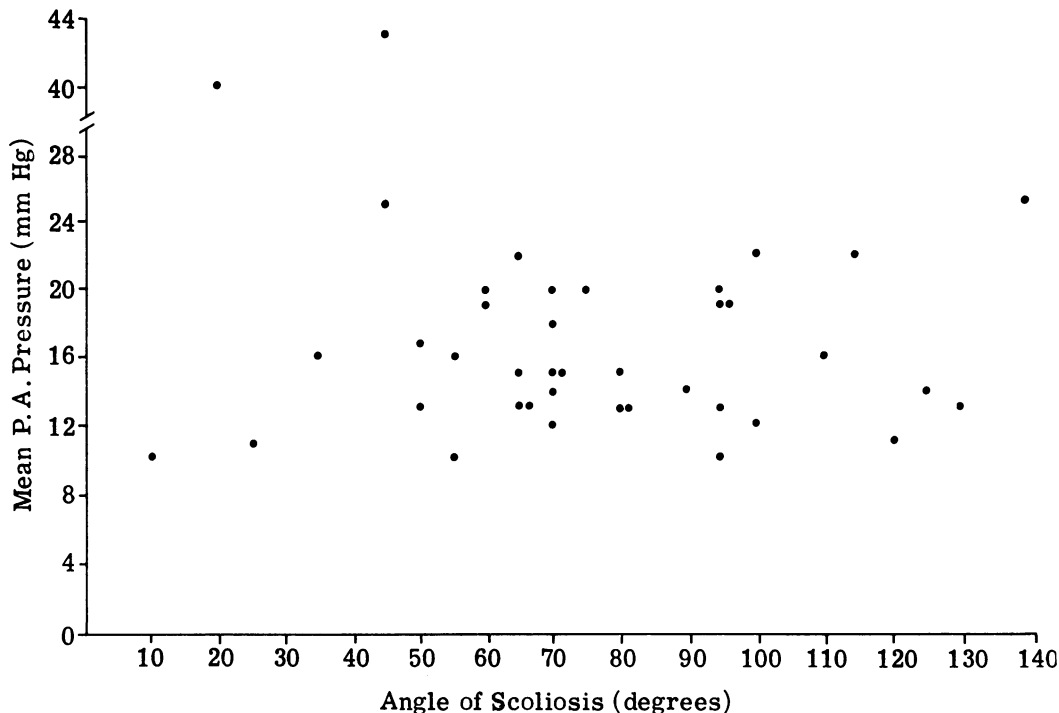


Fig. 1 Relationship between mean pulmonary artery pressure and angle of scoliosis.

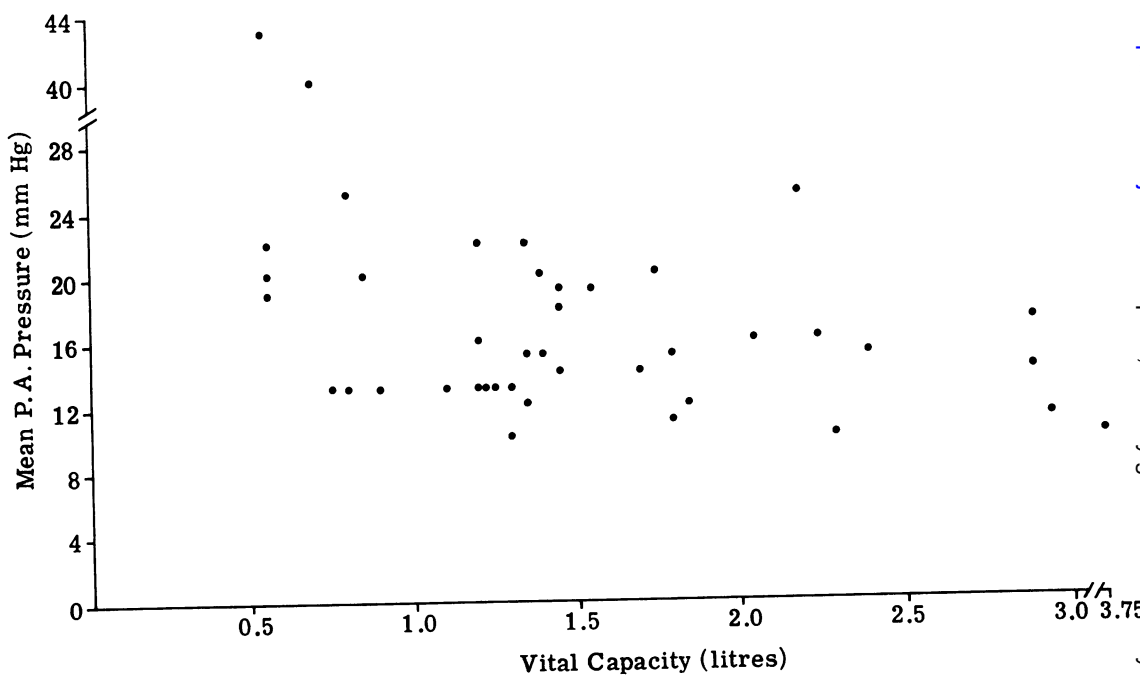


Fig. 2 Relationship between mean pulmonary artery pressure and vital capacity.

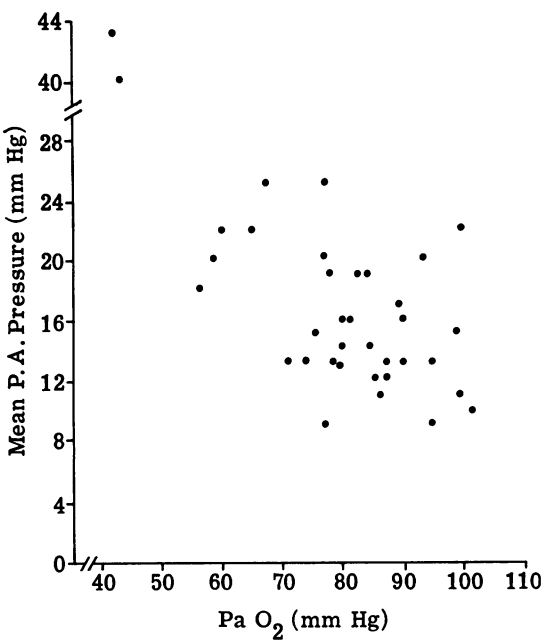


Fig. 3 Relationship between mean pulmonary artery pressure and PaO_2 .

ELECTROCARDIOGRAPHY

Twenty-eight of the 40 patients had none of the criteria of right heart involvement. The details of the other 12 subjects are shown in Table 3. All five of those with tall P waves had pulmonary hypertension. Of the seven with right axis deviation, four had normal pressures. No patient had an R/S ratio greater than 1 in V_1 or V_2 . T-wave inversion in V_{2-3} was seen in one patient with pulmonary hypertension (MF), in two with normal pressures (MM, SR), and in one who had an RV-PA gradient of 12 mmHg and a mean pulmonary artery pressure of 16 mmHg (MB).

Table 3 ECG abnormalities

Subject	$r \geq 2.5$ mm	Electrical axis $\geq 91^\circ$	$R > S_{V_{1-3}}$	T-wave inversion V_{1-3}
MH	3.5	—	—	—
IP	2.5	—	—	—
PG	—	+120°	—	—
JF	—	+125°	—	—
MB	—	—	—	Yes
DW	—	+110°	—	—
LP	2.5	—	—	—
MM	—	+110°	—	Yes
JC	—	+120°	—	—
MF	6	+110°	—	Yes
RH	3	+110°	—	—
SR	—	—	—	Yes

The two patients with the severest pulmonary hypertension had abnormal ECGs, but in five of the nine with mildly raised mean pressures at rest the ECG was normal. Two of the three patients with an RV-PA gradient had abnormal ECGs, but so did five subjects with no gradient and normal pressures.

PHONOCARDIOGRAPHY

The relative intensity of A_2/P_2 in the pulmonary area was $>2:1$ in 16 subjects, between $2:1$ and $1:1$ in four, $1:1$ in three, and $<1:1$ in one. The relationship of the ratio to the pulmonary artery systolic pressure is shown in Figure 4. All the patients in whom P_2 was as loud or louder than A_2 had pulmonary hypertension except MB, who had a pulmonary artery systolic pressure of 26 mmHg and an RV-PA gradient of 12 mmHg.

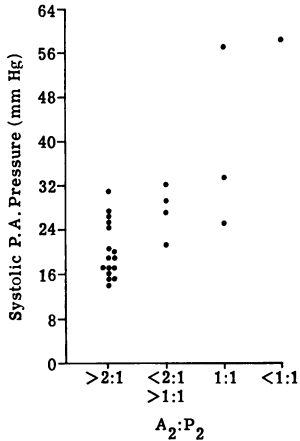


Fig. 4 Relationship of systolic pulmonary artery pressure to $A_2:P_2$.

In expiration the second sound (S_2) was single in 13 subjects, split by 0.01–0.02 s in seven, and by more than 0.02 s in four. Two of these last four had mild right ventricular outflow obstruction. There was no correlation between the width of expiratory splitting of S_2 and the pulmonary artery systolic pressure.

A mid-systolic murmur was present in the three patients with an RV-PA gradient and in two patients with mid-systolic clicks. One of these had an abnormal mitral and tricuspid valve echo.

P_2 - T_0 INTERVAL

The P_2 to T_0 interval was determined in 18 patients. In four of these, two of the methods previously described were used, and in one all three.

The pulmonary artery systolic pressures (± 5 mmHg) predicted from P_2-T_0 by the equation of Burstin (1967) were compared with the actual values. In 17 subjects there was good agreement. In only one was the estimate too high.

Discussion

Previous reports have shown pulmonary hypertension to be frequent in middle-aged scoliotics (Schaub *et al.*, 1954; Bergofsky *et al.*, 1959) but uncommon in those under 20 years (Towers and Zorab, 1969; Riseborough, 1973). The age of the patients reported here fell between these two groups, as did the occurrence of pulmonary hypertension.

The inverse relationship of the pulmonary artery pressure to arterial oxygen tension in the present study has been noted previously in emphysema (Whitaker, 1954), tuberculosis (Söderholm, 1957), and normal subjects at altitude (Peñaloza *et al.*, 1962). The fall in pressure while breathing 100% oxygen indicates that some of the hypertensive effect of hypoxia is reversible. This is probably the result of reversal of hypoxic vasoconstriction (Fishman *et al.*, 1960). However, the pulmonary artery pressure was not invariably brought down to normal levels by breathing pure oxygen. Thus it is possible that a structural change, such as muscularisation of pulmonary arterioles (Hasleton *et al.*, 1968), may also be present.

The presence of a tall P wave in leads II and III of the ECG has been shown here to be a reliable index of pulmonary hypertension. A similar conclusion was reached by Millard (1967) in chronic bronchitics and emphysematous patients. In contrast to the present study, however, he found that right axis deviation was the best criterion of right ventricular hypertrophy. This was unreliable in these scoliotics: four of the seven with electrical axes of $+91^\circ$ or more had normal pressures and no RV-PA gradient. The electrical axis in scoliotics may be influenced by the deformity, presumably by a change in the position or rotation of the heart.

Mechanocardiographic techniques proved useful in predicting pulmonary hypertension. Despite the anatomical displacement of the pulmonary arteries an $A_2 : P_2$ ratio of 1 or less was a useful indicator of pulmonary hypertension. In a study of 162 normal subjects, Harris and Sutton (1968) found by phonocardiography that P_2 was as loud or louder than A_2 in 6% irrespective of the age of the subject. That this was so in 17% of the 24 subjects reported here reflects the common occurrence of pulmonary hypertension in this group.

Burstin's (1967) prediction formula for the P_2-T_0 interval was found to predict the pulmonary artery systolic pressure accurately in 17 out of 18 cases. It is, therefore, probably the best single non-invasive measure of pulmonary hypertension in scoliosis and is unaffected by the deformity of the thorax. Serial readings may be valuable in detecting the development of pulmonary hypertension.

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