Right heart pressures in bronchial asthma

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Right heart pressures, electrocardiograms, blood gases, and peak expiratory flow rates were measured in nine patients admitted to hospital with severe bronchial asthma. Low or normal right heart pressures were found despite electrocardiographic changes in five patients consisting of right atrial P waves, abnormal right axis deviation, and in one patient T-wave changes in pre-cordial leads. These electrocardiographic changes reverted towards normal on recovery of the patient from the asthmatic attack.

Electrocardiographic changes suggestive of right heart embarrassment have been noted in acute bronchial asthma, particularly right atrial P waves (P pulmonale) and abnormal right axis deviation (Harkavy and Romanoff, 1942; Miyamoto, Bastaroli, and Hoffman, 1961; Ambavagar, Jones and Roberts, 1967). These observations raise the possibility that death in bronchial asthma may be due to acute cor pulmonale although necropsy evidence is against this suggestion (Earle, 1953; Houston, de Navasquez, and Trounce, 1953). Cardiac catheter studies in asthmatics have with few exceptions been made during remission of symptoms.

In the present study a miniature cardiac catheterization technique (Bradley, 1964) has been used to measure right heart pressures during acute attacks of asthma in an attempt to determine the significance of the electrocardiographic changes. The results have been assessed in relation to the blood gases and peak expiratory flow rates. All the patients had asthma (as defined by the Ciba Guest Symposium, 1959), some had additional chronic bronchitis (as defined by the Medical Research Council, 1965), and all had been admitted to hospital because of the severity of dyspnoea.

METHODS

An attempt was made by clinical examination, chest radiographs, and electrocardiograms to exclude cardiopulmonary disease other than bronchial asthma (with or without bronchitis). Patients received conventional treatment and the investigation was performed as soon as possible after admission and after a full explanation and request for co-operation. No patient under 21 years of age or with important psychogenic factors was asked to participate.

The procedure was carried out in the general ward with the patient in the sitting position supported at 60 to 90 degrees to the horizontal because orthopnoea was always present. Immediately after catheterization the peak expiratory flow rate was measured with a Wright peak flow meter (Wright and McKerrrow, 1959) and blood (capillary or arterial) was taken for measurement of pH, PCO_2 , and standard bicarbonate by the Astrup method (Astrup, Jørgensen, Andersen, and Engel, 1960). The peak flow rate and electrocardiogram were repeated after recovery. One patient (No. 5) was re-catheterized when she had recovered from her asthma.

Cardiac catheterization was carried out with a miniature disposable cardiac catheter (Portland Plastics Ltd.) inserted through a disposable Guest cannula (Capon Heaton Ltd.). The cannula was introduced percutaneously into a median antecubital vein under local anaesthesia. Right ventricular and/or pulmonary artery pressures were measured with an electromanometer and recorded together with a continuous electrocardiogram on a direct-writing Elema Schonander Minograf 34 Recorder (Siriex Ltd.). The sternomanubrial junction was used as the zero reference point.

RESULTS

Low or normal right heart pressures were recorded in all patients. The pressures fell even further on inspiration by as much as 20 mmHg in the acute phase. These observations are illustrated in Figures 1B, 2B, and 3. Figure 3 also shows the prolonged expiration typical of bronchial asthma. Detailed results are given in the Table which emphasizes the reversible nature of the airways obstruction and describes the electrocardiograms. Electrocardiographic abnormalities were present in five of the nine patients but were not associated with higher right heart pressures than in the other four. Right atrial P waves and right

FIG. 1. Right ventricular pressures: A, Effect of posture (Donald et al. 1953) on (a) two normal subjects; (b) two patients with pulmonary hypertension. B, In four seated patients with acute bronchial asthma and one (5a) on recovery.

FIG. 2. Pulmonary artery pressure: A, Effect of posture on (a) two normal subjects (Donald et al. 1953); (b) one normal subject (Lagerlöf et al. 1951); (c) two patients with pulmonary hypertension (Donald et al. 1953). B, In seven seated patients with acute bronchial asthma and one (5a) on recovery.

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FIG. 3. (a) *Right ventricular pressure tracing and electrocardiogram—patient 3.* (b) *Pulmonary artery pressure tracing and electrocardiogram—patient 2.* (c) *Pulmonary artery pressure tracing and electrocardiogram—patient 9.* Note the inspiratory fall of pressure lasting a much shorter time than the expiratory rise.

TABLE

No.	Patient		PEFR l/min	ECG 1		ECG 2		pH	Blood		PR RR (per min)	BP	RV P pressure		PA Pressure		Notes on ECG
	Age	Sex		P	QRS	P	QRS		PCO ₂	HCO ₃			Exp.	Insp.	Exp.	Insp.	
1	67	F	80	1	+ 49	1	—	7.45	40	27	95 30	160/100	—	—	5/0	2/-4	—
2	33	M	80	1	+ 50	1	—	7.38	46	25	120 22	110/90	—	—	22/3	4/-18	—
3	27	F	40	3	+ 45	1	+ 45	7.39	35	22	120 28	110/70	8/1	4/-2	—	—	Persistent clockwise rotation transient T inversion VI-6
4	61	F	0	2.5	+ 78	2	+ 63	7.31	47	21	105 20	120/80	13/3	5/-4	—	—	—
5	37	F	40	370	1	+ 77	1	7.43	33	23	105 30	130/80	19/6	9/1	12/6	5/1	—
6	63	M	85	280	1	+ 135	0.5 + 120	7.37	32	21	75 34	170/110	11/-3	8/-4	11/3	9/1	Persistent RBBB
7	23	F	80	280	2	+ 117	0.5 + 63	7.33	40	20	160 28	120/60	—	—	19/7	1/-12	—
8	21	F	80	420	3	+ 112	—	7.40	35	23	130 20	140/80	27/4	8/2	17/10	5/-4	Clockwise rotation
9	63	F	50	160	3	+ 67	1.5 + 65	7.28	41	19	130 30	130/95	—	—	18/8	6/-2	—
								7.41	40	25	130 24				12/0	-4/-12	Persistent clockwise rotation

PEFR 1 = peak expiratory flow rate initially (l./min). PEFR 2 = peak expiratory flow rate on recovery (l./min). ECG 1 and ECG 2 = electrocardiographic data initially and on recovery; P = height of the P wave in leads II or III (whichever was the greater) in millimetres; QRS = the mean QRS axis in degrees. PCO₂ = carbon dioxide tension (mmHg). HCO₃ = standard bicarbonate (mEq/l.). PR and RR = pulse and respiratory rates/min. BP = systemic arterial pressure (mmHg). RV = right ventricle; PA = pulmonary artery; Exp. = expiration; Insp. = inspiration. RBBB = right bundle-branch block.

FIG. 4. Legend on page 44.

FIG. 4. Selected electrocardiograms of asthmatic patients. ECG 1, during acute asthma; ECG 2, on recovery.

axis deviation were the commonest changes observed and each of these might be present without the other (Fig. 4). Other features noted were clockwise rotation, right bundle-branch block, and T-wave inversion. As indicated in the Table, most of these changes were transient.

Right heart pressures were uninfluenced by the small degrees of respiratory acidosis (patient 4) or the metabolic acidosis (patients 6 and 8).

DISCUSSION

The pressures recorded were surprisingly low in view of the electrocardiographic changes and do not agree with the findings of other workers, as

discussed later. Pressure measurements made from the sternomanubrial junction as the zero reference point are about 4 mmHg lower than those using the mid right atrium. However, even if this figure is added, the highest pressure recorded was only 31/8 mmHg.

Most cardiac catheterizations are carried out on supine subjects, whereas in this study the asthmatics could not lie flat. The effect of posture on right heart pressures has been studied by Donald, Bishop, Cumming, and Wade (1953) and by Lagerlöf, Eliasch, Werkö, and Berglund (1951) (Figs 1a and 2a): on sitting up from the supine position the pulse pressure fell in all patients studied, but in patients with pulmonary hypertension the systolic and diastolic pressures both fell. It is an open question, therefore, whether the pressures in the present series would be higher had the patients been flat, as they were in the studies now to be discussed.

Williams and Zohman (1960) and Irnell (1964) noted moderate pulmonary hypertension on exercise in some but not all of the asthmatic patients they studied. Zimmerman (1951) and Helander, Lindell, Söderholm, and Westling (1962) noted a rise in pulmonary artery pressure in half of their patients in whom asthmatic attacks had been artificially induced. These patients were studied in the supine position; patients in whom bronchoconstriction is artificially induced may be distressed by the need to remain flat and this could contribute to the rise in pulmonary artery pressure. Harris and Heath (1962a) state that the pulmonary circulation, like the systemic circulation, is influenced by emotional factors.

All the 14 patients studied by Helander *et al* showed systemic arterial desaturation, only two having an oxygen saturation of more than 92% during the induced attack. Ambiaavagar *et al* (1967) report a patient in status asthmaticus and respiratory failure (Pco_2 75 mmHg and Po_2 94 mmHg while breathing oxygen) whose right ventricular pressure (measured with the patient reclining at 45 degrees) was 50/10 on expiration and 20/20 on inspiration. This patient was far more ill than any in the present series and subsequently required artificial ventilation.

The pulmonary hypertension noted by other observers in bronchial asthma could be due to hypoxaemia, which is commoner in bronchial asthma than was once thought (Palmer and Diamant, 1967; Tai and Read, 1967; Waddell, Emerson, and Gunstone, 1967). In this present study arterial oxygen was not measured but an attempt was made to eliminate hypoxaemia. Harris and

Heath (1962b) reviewed the literature concerning the effect of oxygen on the pulmonary circulation and stated that, as well as oxygen, isoprenaline and aminophylline can lower the pulmonary artery pressure. All the patients in the present series were treated with one or more of these three agents, so in the completely untreated patient pulmonary artery pressures might indeed have been higher. Yet despite treatment severe bronchial obstruction persisted throughout the catheterization as did the electrocardiographic changes.

The absence of pulmonary hypertension leaves undetermined the cause of these changes. The over-distension of the lungs and descent of the diaphragm might account for the axis changes but the right atrial P waves and other changes are less easily explained. Relevant to this discussion is the observation by Mayerson and Davis (1942) that in normal subjects passive tilting to the vertical position caused the following electrocardiographic changes—an increase in the amplitude of the P waves, a decrease in the amplitude of the T waves, and a shift of the QRS axis to the right and of the T-wave axis to the left. They quote observations suggesting that sympathetic activity may be partly responsible for these changes. That the position of the heart changes considerably in bronchial asthma is well demonstrated in the chest radiographs reproduced by Riding and Ambivagar (1967). Furthermore, an erect posture is usually assumed by patients with severe bronchospasm in whom there is much sympathetic (or sympathomimetic) activity.

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