THE ELECTROCARDIOGRAPHIC CHANGES IN BRONCHIAL ASTHMA AND THEIR RELATIONSHIP TO THE SEVERITY OF AIRWAYS OBSTRUCTION

By J. L. Da Costa and B. L. Chia

SYNOPSIS

The electrocardiographic findings in eighteen asthmatic patients are reported. Sinus tachycardia was invariably observed. Clockwise rotation of the transitional complex, P pulmonale and ST-T wave changes in the right ventricular leads were noted as the asthma increased in severity. Most of the changes resolved with relief of bronchospasm. The electrocardiographic changes in severe asthma were similar to those found in severe emphysema and chronic bronchitis except for the absence of right ventricular hypertrophy, low voltage changes and the lead I sign.

Prominent right atrial P waves and right axis deviation in association with bronchial asthma were noted by Kahn (1927) and subsequently confirmed by Harkavy and Romanoff (1942), Ambiavagar *et al* (1967) and Gunstone (1971). In addition, T wave alterations and QRS abnormalities were also observed by Colton and Ziskin (1937), Harkavy and Romanoff (1942), Gunstone (1971) and Rebuck and Read (1971). The reversibility of most of these changes with relief of the asthma has also been noted (Colton and Ziskin, 1937. Harkavy and Romanoff, 1942, Rebuck and Read, 1971), but their correlation with the severity of airways obstruction has been poorly documented.

This paper reports the electrocardiographic changes in 18 asthmatic patients and their relationship to the severity of airways obstruction. Furthermore, opportunity was taken to compare the electrocardiographic changes associated with airways obstruction of similar severity in asthma, emphysema and chronic bronchitis.

MATERIALS AND METHODS

Eighteen asthmatic patients (eight males and ten females) were investigated. Their mean age was 28 years (range 19 to 48 years). All had a history of recurrent reversible attacks of airways obstruction with a measurable response to isoprenaline.

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A standard 12-lead electrocardiogram, the forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) before and after bronchodilator therapy, were obtained in all patients.

The FEV_1, and FVC were measured with the patients sitting and at rest by the method described previously (Da Costa *et al*, 1971) and predicted normal values were calculated from regression equations based on normal data obtained in this laboratory (Da Costa, 1971). Electrocardiographic data in severe emphysema and chronic bronchitis was taken from a previous study done by one of the authors (Da Costa, 1973).

RESULTS

Details of the patients studied together with their electrocardiographic and spirometric data before and after bronchodilator therapy are given in Table I. The frequency of the main electrocardiographic abnormalities is shown in Table II. Most of the abnormalities resolved following adequate relief of the bronchospasm (Fig. 1). Sinus tachycardia was seen in every patient and was the earliest electrocardiographic change. It diminished steadily with relief of asthma despite concomitant adrenaline therapy. Clockwise rotation of the transitional complex tended to appear with moderate airways obstruction but was not always observed. Resolution of this change occurred in all cases after relief of airways obstruction. P pulmonale and ST segment depression and T wave inversion in leads II, III and aVf were invariably seen in severe asthma (Table III). Complete resolution could occur rapidly with relief of bronchospasm, in some patients within 2 hours.

TABLE I

SPIROMETRIC AND ELECTROCARDIOGRAPHIC DATA IN THE 18 ASTHMATIC PATIENTS

Case No.	Patient	Sex	Age (Year)	FEV_1, % of Predicted Normal		Electrocardiographic Observations*
				Pre-treatment	Post-treatment (Time)	
1	N.T.H.	M	19	20	65 (2 days)	Pp, IRBBB, CRTC, ST↓, T, ↓ CRWT except IRBBB
2	A.H.C.	F	31	20	80 (4 days)	CRTC, ST↓, T↓, CRWT
3	E.W.	M	24	28	60 (4 days)	ÂQRS—80°, IRBBB, CRTC, CRWT
4	S.H.L.	F	39	13	35 (9 hours)	Pp, CRWT
5	H.B.A.	F	26	20	35 (2 hours)	VEB, ST↓, T↓, Pp, CRTC, Latter two reversed with treatment
6	L.C.H.	M	19	20	70 (4 days)	Intermittent IRBBB, RAD, CRWT
7	L.C.K.	F	32	25	60 (2 days)	Pp, ST, ↓ T↓, CRTC, CRWT
8	K.C.E.	F	16	12	45 (2 hours)	Pp
9	L.H.T.	M	48	29	40 (5 days)	IRBBB, CRTC, CRWT
10	T.H.S.	F	20	12	25 (1 day)	ST↓, T↓, CRTC, CRWT
11	L.K.C.	F	24	17	60 (4 days)	ST↓, T↓, CRTC, CRWT
12	K.C.K.	F	19	12	45 (2 hours)	Pp, ST↓, T↓
13	G.S.H.	M	32	11	18 (2 hours)	Pp, CRTC. Latter reversed after 2 hours of treatment
14	Y.N.F.	M	16	25	33 (3 hours)	CRTC, RAD, CRWT
15	E.H.L.	M	48	17	25 (40 mins)	Pp, CRWT
16	K.U.N.	M	41	29	55 (24 hours)	VEB
17	S.B.S.	F	19	60	—	—
18	S.B.	F	36	50	70 (24 hours)	—

Pp = P pulmonale
IRBBB = Incomplete right bundle branch block
CRTC = Clockwise rotation of transitional complex
ST = Depressed ST segment in leads II, III and aVf
T = Inverted T wave in leads II, III and aVf
CRWT = Complete reversal with treatment
* = All cases showed pretreatment sinus tachycardia
VEB = Ventricular ectopic beats
RAD = Right axis deviation.
ÂQRS = Mean frontal plane QRS axis

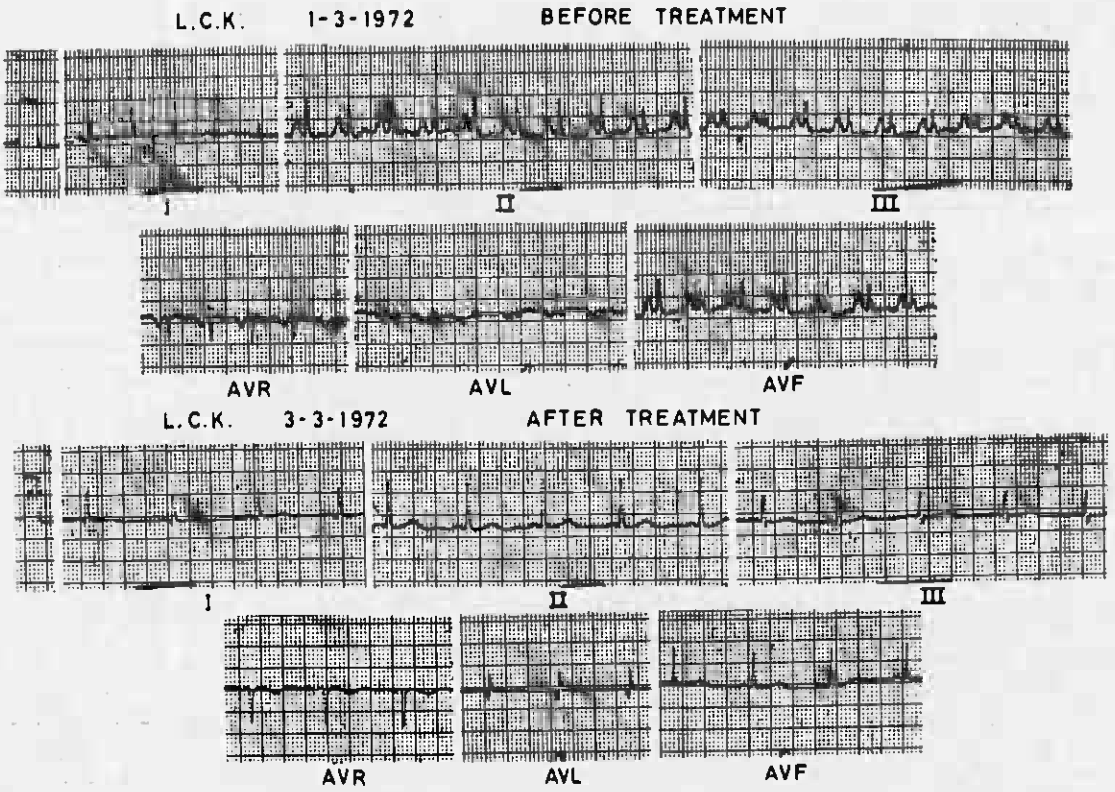

Fig. 1.

TABLE II

THE MAIN ELECTROCARDIOGRAPHIC ABNORMALITIES IN THE 18 PATIENTS WITH ASTHMA

Electrocardiograph Abnormalities	No. of patients with resolution of electrocardiographic Abnormality	Total No. of Patients
Sinus tachycardia	14	18
Clockwise rotation of transitional complex	10	10
ST—T wave changes	6	7
P pulmonale	6	7
Incomplete right bundle branch block	1	4
Right axis deviation	1	2
Ventricular ectopic beats	—	2
Left axis deviation	—	1

The main electrocardiographic findings in severe emphysema, chronic bronchitis (Da Costa, 1973) and asthma with airways obstruction of similar severity are compared in Table IV. It was of interest to note that right ventricular hypertrophy (Goodwin and Abdin, Grades 2—4) and the lead I sign were not seen in any asthmatic patient. On the other hand the frequency of P pulmonale, ST-T wave changes and rotation of the transitional complex beyond V_4 was the same in all three conditions.

DISCUSSION

The findings in this study again emphasize the frequency of electrocardiographic abnormalities in asthma. ST-T wave changes were associated with severe airways obstruction. These abnormalities usually disappeared rapidly with subsidence of the acute asthmatic attack and are not simply a manifestation of hypoxemia or hypercapnia as they are often present in the absence of hypercapnia or hypoxemia (Rebuck and Read, 1971). It has been postulated that other disturbances secondary to asthma may account for the changes, for example, acute reversible pulmonary hypertension, limitation of cardiac output or cardiac compression and poor electrical conductivity with 'verticalization' of the electrical field in the hyper-inflated lung

TABLE III

THE RELATIONSHIP BETWEEN THE PRESENCE OF ST-T WAVE CHANGES AND P PULMONALE IN THE ELECTROCARDIOGRAM AND THE FEV_1 (% PREDICTED NORMAL) BEFORE AND AFTER TREATMENT IN THE ASTHMATIC PATIENTS

Case No.	Electrocardiographic Abnormality			
	ST-T Wave Changes		P Pulmonale	
	Pre-Treatment*	Post-Treatment*	Pre-Treatment*	Post-Treatment*
1	± (20)	— (65)	+ (20)	— (65)
2	+ (20)	— (80)	— (20)	— (80)
4	— (13)	— (35)	± (13)	— (35)
5	± (20)	+ (35)	— (20)	— (35)
7	± (25)	— (60)	± (25)	— (60)
8	— (12)	— (45)	+ (12)	+ (45)
10	+ (12)	— (25)	— (12)	— (25)
11	+ (17)	— (60)	— (17)	— (60)
12	+ (12)	+ (45)	+ (12)	— (45)
13	— (11)	— (18)	+ (11)	± (18)
15	— (17)	— (25)	+ (17)	+ (25)

*FEV_1, % predicted normal, in parenthesis. + = present — = absent.

TABLE IV

COMPARISON OF THE MAIN ELECTROCARDIOGRAPHIC FEATURES IN SEVERE AIRWAYS OBSTRUCTION (FEV_1 LESS THAN 30% PREDICTED NORMAL) ASSOCIATED WITH ASTHMA, EMPHYSEMA AND CHRONIC BRONCHITIS

	No. of Cases	FEV_1, % Pred. Normal	Right Ventricular Hypertrophy Grades**		P Pulmonale	Lead I Sign±	ST-T Wave Changes	Transitional Complex Beyond V_4	Arrhythmias
			1	2-4					
Severe Emphysema	15	<30	9*	3	6	10	5	13	4
Chronic Bronchitis	7	<30	2	2	3	2	5	5	2
Asthma	16	<30	4*	—	7	—	7	10	2

*Excludes one case with right bundle branch block. **Grading of Goodwin and Abdin (1959).
±Iso-electric P wave, QRS less than 1·5 mm. and T wave less than 0·5 mm.

(Spodick 1959). Gunstone (1971) however, recently reported similar electrocardiographic changes in 5 severely asthmatic patients who had normal right heart pressures.

It was noteworthy that except for the lack of the lead I sign of Fowler (1965) the electrocardiographic changes in severe asthma resembled those found in emphysema and chronic bronchitis with a similar degree of severe airways obstruction (Table IV). Furthermore, low voltage was not noted in any of the asthmatic patients as has been recorded previously in emphysema (Selvester and Rubin, 1965). Thus it seems likely that the lead I sign and low voltage in the electrocardiogram are not specifically indicative of the anatomic influence of hyperinflated lungs and the increased antero-posterior diameter of the chest wall as has been suggested recently by Shmock *et al* (1971). Other unknown factors in emphysema must be involved in the production of these characteristic changes.

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