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Electrocardiographic findings in mitral stenosis. Hemodynamic correlation.

The electrocardiographic changes seen in 51 patients with pure mitral stenosis (MS) were compared with hemodynamic data obtained from right and left cardiac catheterization in order to understand its genesis and verify the possibility of non-invasive assessment of the severity of mitral valve obstruction.

Three-fourths of the patients were in either functional class III or IV and none in class I. Left atrial hypertrophy was seen in 75% of patients in sinus rhythm and the majority of them had pulmonary capillary wedge pressure (PCWP) greater than 20 mmHg. Right ventricular hypertrophy (RVH) was the second most common ECG abnormality (41%) and correlated well with increased pulmonary arterial systolic pressure (PASP) (sensitivity = 86%, specificity = 73%). Those patients who had RVH associated with right axis deviation had even higher PASP. Atrial fibrillation (AF) was seen in 28% of patients and its presence may indicate more severe MS as assessed by the diastolic mitral valve gradient, PCWP and PAPS when compared to patients in sinus rhythm. However, it probably results from the combination of duration of rheumatic disease, atrial myocardial fibrosis and the size of the mitral valve orifice. Normal ECG was an infrequent finding (16%) and always implied low PCWP and PASP. It is concluded that the ECG most frequently reflects the hemodynamic derangements of patients with MS and it can accurately identify those who may have pulmonary hypertension.

For several decades, before the advent of cardiac catheterization, the electrocardiographic changes seem in patients with mitral stenosis were purely descriptive[1]. The initial works of Wood [2,3] ,Semler[4] and Imperial[5], attempted to correlate these findings with hemodynamic data. With the introduction of cardiac catheterization and echocardiography, the electrocardiogram was the considered a secondary diagnostic tool in the evaluation of mitral stenosis.

Hence, very few studies were performed to correlate the electrocardiographic findings with hemodynamic evaluation in obstructive mitral valve disease This prompted us to produce a retrospective study so as to obtain a more comprehensive understanding of the ECG in this pathology and a non-invasive evaluation of its severity.

Material and methods

Twelve-lead electrocardiograms of 51 patients with mitral stenosis were reviewed retrospectively for analysis of the P wave, QRS complex and the cardiac rhythm These patients belong to a population of 94 cases of mitral stenosis, who had had cardiac catheterization from 1972 to 1979 at the Hospital Geral de Bonsucesso. Thirty-eight of the 94 patients were excluded because of another associated significant valvular lesion, while five patients were excluded because their ECG's were not available.

Cardiac catheterization was performed in those whose hemodynamic derangements caused by the mitral valve obstruction, placed thin functional class II, III and IV of the NYHA. Seventeen patients (33%) were in class II, 31, (59%) in class III and 4 (8%) in class. Thirty three patients (65%) gave a history of rheumatic fever.

The following criteria were utilized to assess the electrocardiographic abnormalities of the P wave, QRS complex and cardiac rhythm: 1 - left atrial hypertrophy (at least two of the following criteria): a) P Wave equal to or greater than 0,12 sec in lead II; b) notched P wave in lead II; c) negative deflexion greater than 0,03 mm-sec in V_1 (Morris index) [6]; 2 -

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right atrial hypertrophy; P wave greater than 0,25 mV in lead II; 3 - right ventricular hypertrophy (at least two of the following criteria): a) QRS axix equal to or greater than +95°, in the frontal plane; b) R > S in lead V_1; c) rs or qs in lead V_6; d) RS or rS in lead V_1 (the right ventricular hypertrophy was classified according to the combination of the ECG abnormalities: type A =a + b + d, type B = b + d, type C = a + c +d); 4 - right bundle branch block: a) Rsr' in lead V_1; b) Rs in lead V_6 (with slurred s); c) QRS duration greater than 0,10 sec; 5 - Atrial fibrillation- a) Absence of P wave; b) fibrillation of the baseline; c) irregular ventricular rhythm.

The patients underwent cardiac catheterization following a six hour fast. All drugs had been discontinued for at least six hours prior to the catheterization. Pressures were measured, using transducers at the level of the mid auxiliary lime with the patient in horizontal decubitus. The mean pulmonary capillary wedge pressure was obtained by electric damping of the phasis pressure curve. The transmitral mean diastolic pressure gradient was obtained by simultaneous equisensitive pressure recording from the left ventricle and pulmonary capillary wedge position.

The average time between the electrocardiogram and cardiac catheterization was 20.0 ± 29.4 days.

The time interval between the onset of acute rheumatic fever (when referred) and the date of the electrocardiogram was called: duration of rheumatic disease. The time interval between the recognition of heart disease and the date of the electrocardiogram was called: duration of heart disease. These were sought for each patient through a review of in-patient and out-patient records and available in the majority of them The patient's age ranged from 10 to 61 years with a mean of 37.5 ± 10,7 years. Thirteen were mages and 38 females No patients had any other form of heart disease, chronic obstructive pulmonary disease and none had left bundle branch block or hemiblocks in the electrocardiogram.

Date are expressed as the mean standard deviation. Analysis of differences among groups was made by Student's t test. Regression equation was obtained by standard formulas. Probability less than 5% ($p < 0,05$) was considered significant.

Results

The pulmonary capillary wedge pressure, transmitral mean diastolic gradient and pulmonary artery systolic pressure values of our patient population were 24.6 ± 8.5, 18.6 ± 8.3, and 56.5 ± 23.5 mmHg, respectively.

The electrocardiographic changes seem in the 51 patients with mitral stenosis are depicted in table I.

Cardiac rhythm: Thirty-seven patients (72%) were in sinus rhythm and 14 (28%) were in atrial fibrillation. The relationship between cardiac rhythm, pulmonary wedge pressure (PCWP), transmitral diastolic gradient (G) and pulmonary artery systolic pressure (PAPs) are shown in table II.

Table I - Electrocardiographic findings in 51 patients with mitral stenosis.

Normal ECG		9	(18%)
Atrial fibrillation		14	(28%)
Sinus rhythm		37	(72%)
Left atrial hypertrophy		28	(76%)
Right atrial hypertrophy		12	(32%)*
Right ventricular hypertrophy		21	(41%)*
	Type A	6	(29%)
	Type B	11	(52%)
	Type C	4	(19%)
Right QRS axis deviation		12	(23%)
Right bundle branch block		5	(10%)

* Percentage remated to 37 patients in sinus rhythm.

Table II - Relation between pulmonary capillary wedge pressure (PCWP), transmitral diastolic mean gradient (G), pulmonary artery systolic pressure (PAPs) (in mmHg) and cardiac rhythm in patients with mitral stenosis.

	Sinus Rhythm (n = 37)	Atrial Fibrillation (n = 14)	P≤ 0.05
PGWP	23.4 ± 7.9	27.7 ± 9.4	N.S.
G	17.5 ± 8.3	21.6 ± 7.8	N.S.
PAPs	53.8 ± 22.8	64.0 ± 24.7	N.S.

The differences were small and were not statistically significant. However, it may be seen that all three observed pressure parameters, had a greater mean value in the patients with atrial fibrillation.

A history of prior rheumatic disease was found in 33 of the 51 patients (65%). The duration of heart disease was determined in 49 cases (96%). The relation between cardiac rhythm and duration of rheumatic disease (DHD) is depicted in table III.

Table III - Relation between the duration of rheumatic disease (DRD), duration of heart disease (DHD) (in years) and cardiac rhythm In patients with mitral stenosis.

	Sinus Rhythm.	Atrial FIbrilation	P≤0 05
DRD (n = 33)	21.1 ± 10.4 (n = 24)	27.9 ± 8.4 (n =9)	N.S.
DHD (m = 49)	7.4 ± 8.1 (n = 35)	9.1 ± 6.7 (n = 14)	N.S.

Although the differences were not statistically significant, again the duration of disease appeared to be longer in patients with atrial fibrillation.

Left atrial hypertrophy (LAH): Of the 37 patients with sinus rhythm, 28 (76%) presented electrocardiographic criteria of left atrial hypertrophy. The pulmonary capillary wedge pressure was 25.7 ± 6.7 mmHg in patients with left atrial hypertrophy and 16.4 ± 7.6 mmHg in patients without it ($p < 0\,01$). In 89% (25/28) of patients with left atrial hypertrophy, the pulmonary capillary wedge pressure was equal to or greater than 20 mmHg, whereas in 77% (7/9) of patients without hypertrophy was less than this value (fig.1).

Right ventricular hypertrophy (RVH) and right atrial hypertrophy (RAH): Twenty one (41%) of the 51 patients studies had electrocardiographic criteria of right ventricular hyper trophy, type B being the commonest form. The pulmonary arterial systolic pressure was 75.1 ± 23.6 mmHg in patients with right ventricular hypertrophy and 43.7 ±12.0 mmHg in patients without it ($p < 0.01$). Eighty-six percent (18/ 21) of patients with RVH had pulmonary artery systolic pressure greater than 50 mmHg, whereas 73% (22/30) of patients without RVH had pressure less than this value (fig.2). The pulmonary arterial systolic pressure levels for the three types of right ventricular hypertrophy were slightly but not significantly different: 82.0 ±: 25.5 mmHg for type A, 74.7 ± 24.5 mmHg for type B and 65.7 ± 20.5 mmHg for type C. The relationship between right ventricular hypertrophy in the electrocardiogram and pulmonary capillary wedge pressure and mitral diastolic gradient is shown in table IV.

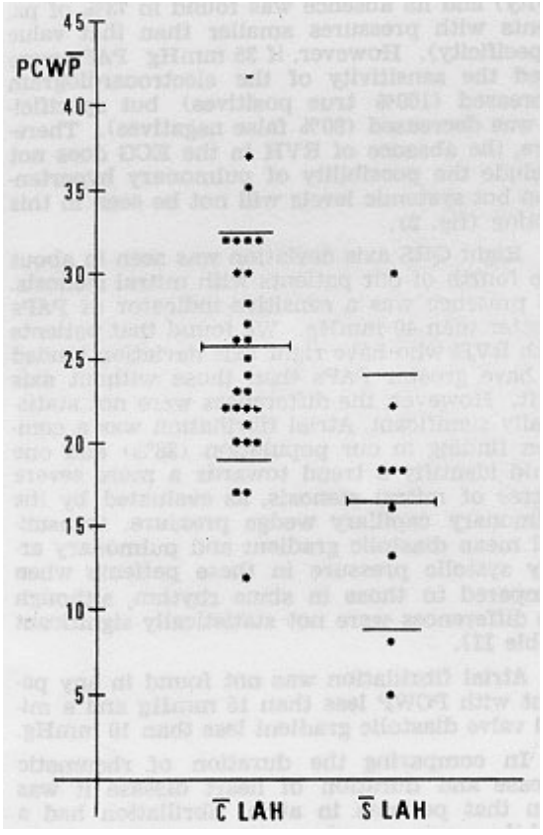

Fig. 1 - Distribution of 37 patients with mitral stenosis in sinus rhythm according to the presence or absence of left atrial hypertrophy (c LAH and s LAH, respectively) in relation to the mean pulmonary capillary wedge pressure (PCWP) (in mmHg). Mean values and standard deviation are shown by long and short horizontal lines, respectively.

Patients with RVH had significantly greater levels of these pressure data than those without it. Right atrial hypertrophy was found in twelve of 37 patients (32%) in sinus rhythm. Six had associated right ventricular hypertrophy.

Right QRS axis deviation (RAD): QRS axis equal to or greater than + 95°, in the frontal plane was considered right axis deviation and was found in twelve patients (23%).

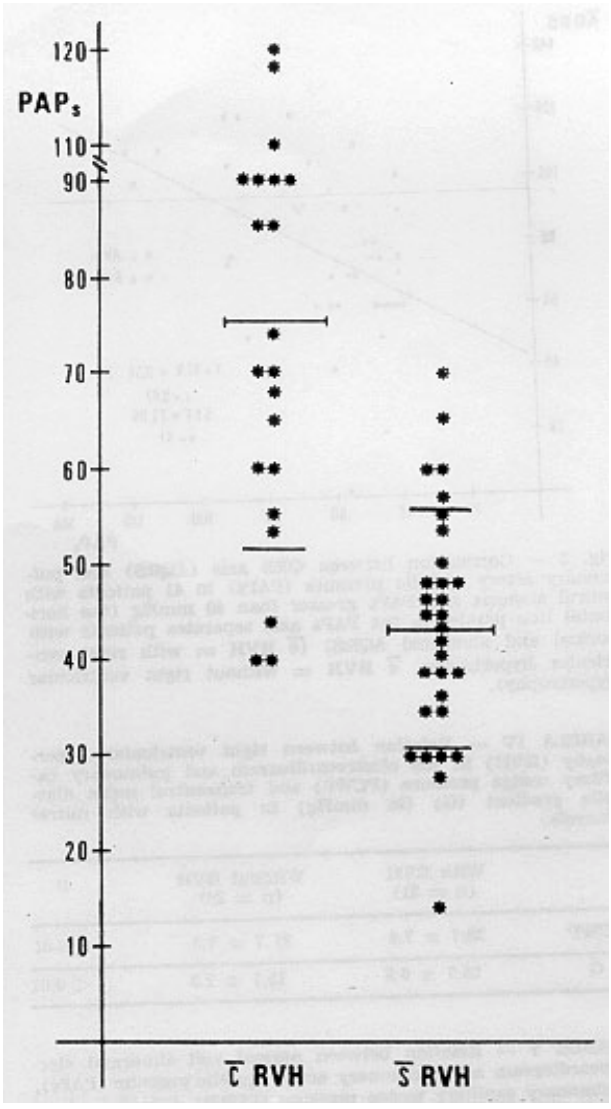

Fig. 2 - Distribution of 51 patients with mitral stenosis according to the presence or absence of hight ventricular hypertrophy (c RVH and s RVH, respectively) in relation to the pulmonary artery systolic pressure (PAPs) (in mmHg). Mean values and standard desviation are shown by long and short horizontal lines, respectively.

All had right ventricular hypertrophy in the electrocardiogram and a pulmonary arterial systolic pressure greater than 40 mm Hg. The pulmonary arterial systolic pressure in patients with RVH and right QRS axis deviation was 81.2 ± 25.7 mm Hg whereas in patients with RVH without, right QRS axis deviation, it was 67.0 ± 18.7 (N S). The ratio between QRS axis and pulmonary arterial systolic pressure in 41 patients with PAPs equal to or greater than 40 mm Hg is depicted in figure 3 and one can seen the poor relationship between these two parameters.

All patients with right QRS axis deviation had a pulmonary capillary wedge pressure equal to or greater than 20 mmHg.

Right bundle branch block: Right bundle branch block was seem in six patients (12%) three of whom had right ventricular hypertrophy in the electrocardiogram. The pulmonary

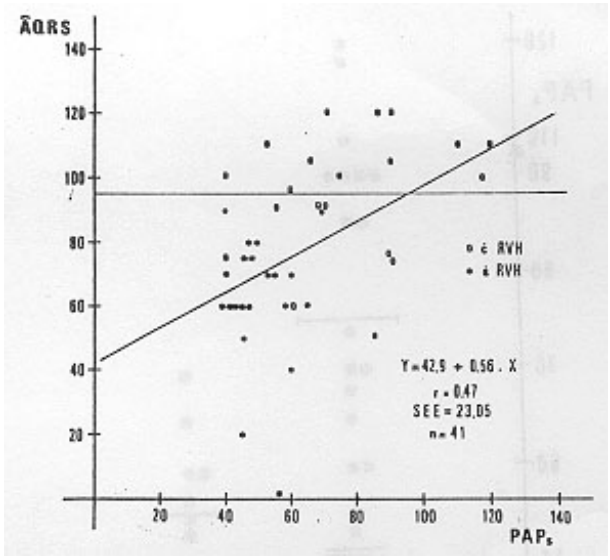

Fig 3 - Correlation between QRS axis (AQRS) and pulmonary artery systolic pressure (PAPs) in 41 patients with mitral stenosis and PAPs greater than 40 mmHg (the horizontal line parallel to the PAPs axis separates patients with normal and abnormal AQRS) (c RVH = with right ventricular hypertrophy, s RVH = without right ventricular hypertrophy).

Tabela IV - Relation between right ventricular hypertrophy (RVH) in the electrocardiogram and pulmonary capillary wedge pressure (PCWP) and transmitral mean diastolic gradient (G) (in mmHg) in patients with mitral stenosis.

	With RVH (n = 21)	Without RVH (n = 30)	p
PCWP	28.7 ± 7.4	21.7 ± 7.9	< 0.01
G	23.7 ± 6.9	15.1 ± 7.3	< 0,01

Table V - Relation between normal usd abnormal electrocardiogram and pulmonary artery sytolic pressure (PAPs), pulmonary capillary wedge pressure (PCWP) and transmitral mean diatolic gradient (G) (in mmHg) in patients with mitral stenosis.

	Normal ECO (n = 8)	Abnormal ECGS (n = 43)	p
PAPs	33.0 ± 9.8	61.0 ± 20.5	< 0.01
PCWP	14.7 ± 57	2.3 ± 7.5	< 0.01
G	9.1 ± 3.8	20.4 ± 7.5	< 0.01

artery systolic pressure was 59.3 ± 18.5 mmHg, being greater than 40 mmHg in all cases.

Normal Electrocardiogram: normal ECGs were found in eight patients (16%). The relation between PCWP, G and PAPs in patients with normal and abnormal ECGs is shown in table V. Patients with abnormal ECGs had significantly higher values of these parameters when compared to patients with normal electrocardiogram. No patient with a normal ECG had PCWP, G and PAPs values greater than 22, 14 and 46 mm Hg, respectively. Half of the patients with normal electrocardiogram were in functional class II and the other half in class III.

Discussion

The present study in 51 patients with mitral stenosis demonstrates that correlations between electrocardiographic changes and hemodynamics can be established. Left atrial hypertrophy was the most common ECG finding being present in 55% of all cases and 76% of patients in sinus rhythm. The P mitral correlates with a left atrial pressure greater than 20 mmHg in the majority of cases. However, its absence does not necessarily mean less severe mitral valve obstruction. Several patients with a normal P wave had elevated pulmonary capillary wedge pressures (fig. 1).

Right ventricular hypertrophy was the second most common ECG abnormality being present in 41% of the patients and correlated well with the level of pulmonary artery systolic pressure. RVH in the electrocardiogram was seem in 86% of patients with pulmonary artery systolic pressure greater than 50 mmHg (sensitivity) and its absence was found in 73% of patients with pressures smaller than that value (specificity). However if 35 mmHg PAPs were used the sensitivity of the electrocardiogram increased (100% true positives) but specificity was decreased (80 % false negatives). Therefore, the absence of RVH in the ECG does not exclude the possibility of pulmonary hypertension but systemic levels will not be seen in this setting (fig. 2) .

Right QRS axis deviation was seen in about one fourth of our patients with mitral stenosis. Its presence was a sensitive indicator of PAPs greater than 40 mmHg. We found that patients with RVH who have right axis deviation tended to have greater PAPs than those without axis shift. However, the differences were not statistically significant. Atrial fibrillation was a common finding in our population (28%) and one could identify a trend towards a more severe degree of mitral stenosis, as evaluated by the pulmonary capillary wedge pressure transmitral mean diastolic gradient and pulmonary artery systolic pressure in these patients when compared to those in sinus rhythm, although the differences were not statistically significant (table II).

Atrial fibrillation was not found in any patient with PCWP less than 15 mmHg and a mitral valve diastolic gradient less than 10 mmHg.

In comparing the duration of rheumatic disease and duration of heart disease it was seen that patients in atrial fibrillation had a slightly greater number of years than those in sinus rhythm, although the differences were not statistically significant. A normal electrocardiogram was an infrequent finding (16%) and always implied a law pulmonary capillary wedge and pulmonary artery pressures, and a small transmitral diastolic gradient. Correlating the electrocardiographic and pressure data, we were able to classify our mitral stenosis population in three subgroups:

1. Hypertensive mitral stenosis: Thirty four patients had transmitral diastolic gradient greater than 15 mmHg and PCWP greater than 20 mmHg. All had PAPs greater than 40 mmHg (80% had it greater than 50 mmHg). Eight pa-

tients were in NYHA functional class II, 22 in class III and four in class IV.

2. Discrete mitral stenosis: eleven patients with transmitral diastolic gradient between 10 and 15 mmHg and PCWP between 15 and 20 mmHg. Nome had PAPs greater than 50 mmHg. Five patients were in NYHA functional class II and six in class III.

3. Normotensive mitral stenosis: Six patients with transmitral diastolic gradient smaller than 10 mmHg and PCWP smaller than 15 mmHg. Nome had PAPs greater than 50 mmHg. Four were in NYHA functional class II and two in class III.

The mean pulmonary artery systolic pressure for patients with hypertensive, discrete and normotensive mitral stenosis according to the above classification were 66.6 ± 19.0, 41.4 ± 6.0 and 28.0 ± 7.4 mmHg, respectively, and the differences were statistically significant ($p < 0.05$) . Comparing these subgroups (table VI) it was seen that no patient with normal electrocardiogram had hypertensive mitral stenosis. Atrial fibrillation was seen in the hypertensive and discrete subgroups but not in the normotensive one. Sinus rhythm was prevalent in all subgroups but left atrial hypertensive was quite rare in the normotensive form. Right ventricular hypertrophy was almost always indicative of hypertensive subgroup.

Table VI - Electror ardiograPhic findings in 51 patients with mitral stenosis (MS) when classified accordingly to the pulmonary capillary wedge pressure and transmitral mean diastolic gradient (see text)

	Hypertensive MS (n = 34)	Discrete MS (n = 11)	Normotensive MS (n = 6)
Normal	0	3 (27%)	5 (83%)
Sinus rhythm	23 (68%)	8 (73%)	6 (100%)
Atrial fibrilmation	11 (32%)	3 (72%)	0
Left atrial hypertrophy	22 (95%)*	5 (62%)*	1 (17%)*
Right atrial. hypertrophy	10 (430/6) *	2 (25%)*	0
Right ventricular hypertrophy	20 (59%)	1 (9%)	0
Right QRS axis deviation	12 (35%)	0	0
Right bundme branch block	6 (18%)	0	0

* Percentage related to the patients in sinus rhythm.

For 30 years physicians have been looking at the electrocardiogram for signs that would allow them to imply intracardiac pressures and consequently to assess the hemodynamic status of patients with mitral stenosis. Wood [2,3] in his extensive and classic review of this pathology indicated a direct relationship between pulmonary arterial pressure and right ventricular hypertrophy in the electrocardiogram. Lewis et al.[7] had earlier found the same results. Other authors were unable to find similar results in relation to right ventricular hypertrophy [1,4] thus creating some controversy. There seems to be agreement when correlating right QRS axis shift with pulmonary artery pressure indicating that this is probably the best isolated index of elevated pulmonary vascular resistance in patients with mitral stenosis[4,8].

Our data demonstrates that electrocardiogram can predict if there is significant pulmonary artery hypertension, and that the sensitivity is greater than the specificity (86% vs 73%). Type A right ventricular hypertrophy seems to indicate greater pulmonary artery pressure level than types B and C, corroborating the findings of Mershon et al[9].

The association of right QRS axis deviation and electrocardiographic right ventricular hypertrophy correlated with elevated pulmonary artery systolic pressure but the degree of right axis deviation did not correlate with the degree of pulmonary hypertension (fig. 3). Left atrial overload is diagnosed in the electrocardiogram by changes in duration and morphology of the P wave. It is known that pressure and/or volume overload of the left atrium may cause increased duration and the notched shape of the P wave primarily in lead II [10,11], but its electrogenesis is still uncertain. Some authors feel that these changes are due solely to increase in left atrial volume and/or mass [11,13], while others refer to a delay in intra-atrial conduction time [14,l5].

Increased posterior forces of atrial depolarization leads to a "plus minus" P wave in lead V_1. When the terminal negative deflexion is equal to or greater than 0,04 mm-sec, one has a positive Morris index [6].

The sensitivity and specificity of the eletrocardiogram in detecting left atrial overload through changes in the P wave have been good specially when a true increase in the chamber volume exists but false positives and false negatives have been seen frequently[11,14]. Our data shows that three fourths of patients with mitral stenosis have electrocardiographic pattern of left atrial hypertrophy and these patients have greater hemodynamic disturbances as assessed by the pulmonary capillary wedge pressure, than those with normal P wave.

The findings are confirmed by other authors[6 11,12], but not by the elegant study of Josephson et al.[16] that demonstrated the absence of correlation between the electrocardiography pattern of left atrial hypertrophy and pulmonary capillary wedge pressure in 21 patients with different forms of heart disease. They concluded that, in patients with rheumatic mitral valve disease, the electrocardiographic pattern of left atrial hypertrophy represents a lengthening of interatrial conduction time which is caused by the presence of rheumatic fibrosis and the increase in left atrial volume.

The discrepancy between Josephson's results[16] and ours, concerning the pulmonary wedge pressure vs. electrocardiographic left atrial hypertrophy problem, seems to results from facts that may introduce bias in his results, namely the small number of patients with rheumatic mitral valve disease in his study, a population with less hemodynamic disorders and the fact that his criteria for inclusion in the study was previous existence of P mitrale in the electrocardiogram and not the fact that the patients had mitral valve disease.

Atrial fibrillation is another finding that has raised some controversy regarding its genesis. Several possibilities as rheumatic fibrosis, left

atrial dilatation and hypertrophy, rheumatic and/or duration of heart disease, either isolated or associated, have been incriminated. Singer et al.[17] used biopsied atrial samples of patients with chronic atrial fibrillation for electrophysiologic studies and noted nom-uniformity of membrane potential, conduction velocity and excitability. Bailey[18] in a different study concluded that fibrosis is the pathologic substrate for this electrophysiologic non-homogeneity in patients with rheumatic heart disease, and that the perpetuation of atrial fibrillation is related to scarcity or absence of myocardial cells in the atrial. On the other hand, Lee [19] observed no difference in patients with mitral stenosis who were in sinus rhythm or atrial fibrillation as far as hemodynamic data, is concerned, which was confirmed by our present study.

Our data showed a trend towards longer duration of disease in patients with atrial fibrillation as compared to those in sinus rhythm, although the differences were not statistically significant. This information leads us to conclude that in patients with rheumatic mitral stenosis the appearance of atrial fibrillation will depend on the presence of myocardial fibrosis, serious ness of mitral valve obstruction and duration of rheumatic disease altogether and not to any of these parameters alone.

Normal electrocardiograms have been frequently found in patients with significant mitral stenosis. Semler et al[4] saw am incidence of 16% of normal ECG in patients found to have important mitral valve obstruction at surgery. Our study shows a similar experience but no patient had pulmonary artery systolic and capillary wedge pressures greater than 46 and 22 mmHg, respectively.

Nome of the patients were in NYHA functional class IV, as half were in class II and half in class III.

In conclusion, the present study indicates that a careful assessment of the electrocardiogram can provide important information that may lead one to assume the hemodynamic status of a patient with mitral stenosis, specially the existence of pulmonary hypertension which can be detected in most patients.

Resumo

As alterações eletrocardiográficas observadas em 51 pacientes com estenose mitral (EM) pura foram comparadas com as alterações hemodinâmicas obtidas por cateterismo cardíaco com o objetivo de esclarecer suas causas e verificar a possibilidade de avaliação não invasiva da gravidade da obstrução valvar.

Três quartos dos pacientes eram das classes funcionais III ou IV e nenhum da classe I (NYHA). Hipertrofia do átrio esquerdo estava presente em 75% dos pacientes em ritmo sinusal e a maioria deles tinha pressão média de capilar pulmonar (PCP) maior que 20 mm Hg. Hipertrofia ventricular direita, (HVD) foi a segunda alteração eletrocardiográfica mais comum (41%) evidenciando relação com a elevação da pressão sistólica de artéria pulmonar (PSAP) (sensibilidade = 86%, especificidade = 73%). Aqueles pacientes que tinham HVD associada a desvio do AQRS para a direita, tinham PASP ainda mais elevada. Fibrilação atrial (FA) foi encontrada em 28% dos casos e sua presença pode indicar EM mais severa, em termos de gradiente ao nível da mitral, PCP e PSAP, do que nos pacientes em ritmo sinusal. Entretanto a FA, provavelmente resulta de uma tríade: duração da doença reumática, fibrose miocárdia atrial e área da valva mitral. Eletrocardiograma normal foi um achado pouco freqüente (16%) e sempre significou baixa PCP e PSAP. Conclui-se que o ECG reflete, ma maioria das vezes o estado hemodinâmico dos pacientes com EM e pode identificar acuradamente os portadores de hipertensão pulmonar.

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