An Electrocardiographic Analysis of Sixty-Five Cases of Mitral Regurgitation

By L. G. Bentivoglio, M.D., J. F. Uricchio, M.D., A. Waldow, M.D., W. Likoff, M.D., and H. Goldberg, M.D.

The detailed electrocardiographic analysis of a large series of patients with proved, dynamic mitral regurgitation is lacking in the medical literature. The present report intends to fill the gap and to point out and discuss some discrepancies observed between the current concepts and the results of the study.

Electrocardiography has been long an important tool in cardiac diagnosis and reports of abnormal patterns in patients with rheumatic mitral regurgitation have already appeared in the world literature. The series are, however, either too small or lack sufficient surgical or pathologic confirmation of major leak of the mitral valve.

The object of the present report is to present a detailed analysis of the electrocardiographic findings in 65 patients with rheumatic mitral regurgitation of dynamic significance.

Material and Methods

Sixty-five patients with proved dynamic mitral regurgitation constitute the object of the present study. The diagnosis was entertained clinically on the basis of fatigue and dyspnea, a grade III or more blowing systolic murmur at the apex, radiating to the axilla, and a normal to absent mitral first sound. A mid-late rumble at the apex was frequently present. The majority of the patients had also a huge left atrium with a wide sweep of the esophagus on the roentgenograms.

The clinical impression was fortified by the heart catheterization findings in 40 patients. Of these, 23 had right heart catheterization, 11 had left heart catheterization, and 6 had combined heart catheterization. Criteria for suggestion of mitral insufficiency at heart catheterization were the presence of a prominent "e-v" wave either in the pulmonary venous capillary pulse or left atrium, the "wash-out" of the catheter from the left ventricle during systole of this chamber, and a small or absent left atrial-left ventricular filling pressure gradient. When present, the gradient was larger in early diastole and smaller or absent at the end of the ventricular filling period, as previously observed in patients with mitral regurgitation.

Surgery for correction of mitral regurgitation was performed in 64 patients, 2 of whom came later to autopsy. In 1 case postmortem confirmation alone was obtained. In all the operated cases the amount of mitral regurgitation was estimated by the surgeon to be greater than 2 plus on the basis of 1 to 4 plus. The mitral valve area, estimated at the time of surgery or autopsy, was at least 1 1/2 fingers (2.5 cm.²) in size but less than 2 fingers (4.0 cm.²) in 17 patients, and 2 fingers or more with a maximum of 3 1/2 fingers in all the rest. An area of 2.5 cm.² is well beyond the critical size for dynamic mitral stenosis.

Of the total 65 patients, 22 were male and 43 were female, a ratio of 1 to 2. The ages ranged between 13 and 54 years, with an average of 33 years. The average age for males was 35 years, (22 to 54); for females it was 32, (13 to 51). The distribution of patients according to sex and age groups is reported in table 1.

All the tracings of the present study were recorded on Sanborn electrocardiographs during the period of hospitalization immediately preceding surgery or death (1 case died before surgery) with the patients in the basal state, breathing quietly in the recumbent position.

A 12-lead electrocardiogram was obtained in all cases. The electric axis of the P, QRS, and T waves was measured according to the method of Carter and co-workers. The value of the electric axis of the T wave was approximated to the nearest multiple or submultiple of 15°. The normal axis for the P wave was considered to lie between +45° and +69° of the Carter quadrant; for the QRS interval between +30° and +90°. The electric position was obtained by the criteria of Wilson and co-workers. The duration of waves and intervals is reported in seconds. The magnitude of

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Circulation, Volume XVIII, October 1958
Table 1.—Relative Incidence of Normal Sinus Rhythm and Atrial Fibrillation

<table>
<thead>
<tr>
<th>Ventricular patterns</th>
<th>NVC</th>
<th>LVH</th>
<th>RVH</th>
<th>L&amp;RVH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>9</td>
<td>8</td>
<td>9</td>
<td>2</td>
<td>28</td>
</tr>
<tr>
<td>NSR</td>
<td>8</td>
<td>1</td>
<td>5</td>
<td>1</td>
<td>15</td>
</tr>
<tr>
<td>AF</td>
<td>7</td>
<td>18</td>
<td>8</td>
<td>2</td>
<td>35</td>
</tr>
<tr>
<td>Total no. of pts.</td>
<td>7</td>
<td>26</td>
<td>9</td>
<td>2</td>
<td>46</td>
</tr>
<tr>
<td>Percentage of total</td>
<td>51%</td>
<td>31%</td>
<td>15%</td>
<td>3%</td>
<td>100%</td>
</tr>
</tbody>
</table>

* Key to abbreviations: NVC, normal ventricular complex; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy; L&RVH, left and right ventricular hypertrophy; NSR, normal sinus rhythm; AF, atrial fibrillation.

deflections is given in tenths of millivolts with the instrument adjusted to the usual sensitivity of 1 cm./mV. Durations, magnitude of deflections, and ventricular activation times were calculated according to the recommendations of the committee on electrocardiography of the American Heart Association. Normal standards for the electrocardiograms were considered those given by Kossman. Upper limits of normal for the duration of the Q-T interval in males and females were considered those given by Ashman and Hull. Criteria for left atrial enlargement were considered (1) mean manifest electric axis of atrial activation (AP) of less than +45°, (2) duration of the P wave in the peripheral leads of 0.12 second or more, (3) notching of the P wave in the peripheral bipolar leads, and (4) diphasic P wave in which the initial deflection is upward in lead V₁. The criteria of Sokow and Lyon were used for the diagnosis of left and right ventricular hypertrophy. An S wave in lead V₁ equaling or exceeding 24 mm. was also considered among the criteria for the diagnosis of left ventricular hypertrophy. The criteria of Wilson and co-workers, summarized by Lipman and Massie, were used for the diagnosis of complete left bundle-branch block. The criteria of Barker and Valenca were used for the diagnosis of incomplete right bundle-branch block and incomplete right bundle-branch block associated with right ventricular hypertrophy. The pattern of incomplete left bundle-branch block has not been considered because of the well-known difficulty of differentiating it from left ventricular hypertrophy. No other electrocardiographic patterns were encountered in our series. All but 4 patients (M.H., E.H., E.R., M.W.) were receiving digitalis at the time the electrocardiograms were recorded. The electrocardiograms were analyzed in a tabular form on master sheets and all durations and deflections were carefully measured with the help of calipers and lenses, when necessary. The analysis of the duration and size of the deflections was limited to those leads that are considered representative for the diagnosis of the various electrocardiographic syndromes mentioned above.

Table 2.—Characteristics of the P Wave and P-R Interval

<table>
<thead>
<tr>
<th>Name</th>
<th>Axis (deg.)</th>
<th>Duration (sec.)</th>
<th>Voltage Lead II</th>
<th>Shape</th>
<th>P-R interval (sec.)</th>
<th>Ven-tricular complex</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.A.</td>
<td></td>
<td>0.12</td>
<td>1.0</td>
<td>Nt.</td>
<td>D</td>
<td>0.21</td>
</tr>
<tr>
<td>K.B.</td>
<td></td>
<td>0.14</td>
<td>0.7</td>
<td>Nt.</td>
<td>I</td>
<td>0.18</td>
</tr>
<tr>
<td>M.H.</td>
<td></td>
<td>0.14</td>
<td>1.2</td>
<td>R</td>
<td>D</td>
<td>0.20</td>
</tr>
<tr>
<td>E.H.</td>
<td></td>
<td>0.12</td>
<td>1.0</td>
<td>Nt.</td>
<td>Nt.</td>
<td>0.18</td>
</tr>
<tr>
<td>B.M.</td>
<td></td>
<td>0.10</td>
<td>1.7</td>
<td>R</td>
<td>D</td>
<td>0.17</td>
</tr>
<tr>
<td>E.R.</td>
<td></td>
<td>0.11</td>
<td>1.1</td>
<td>R</td>
<td>D</td>
<td>0.19</td>
</tr>
<tr>
<td>M.S.</td>
<td></td>
<td>0.12</td>
<td>1.5</td>
<td>R</td>
<td>D</td>
<td>0.18</td>
</tr>
<tr>
<td>M.W.</td>
<td></td>
<td>0.11</td>
<td>2.6</td>
<td>P</td>
<td>D</td>
<td>0.18</td>
</tr>
<tr>
<td>M.B.</td>
<td></td>
<td>0.13</td>
<td>1.0</td>
<td>R</td>
<td>D</td>
<td>0.21</td>
</tr>
<tr>
<td>E.D.</td>
<td></td>
<td>0.16</td>
<td>1.0</td>
<td>R</td>
<td>D</td>
<td>0.21</td>
</tr>
<tr>
<td>J.D.</td>
<td></td>
<td>0.13</td>
<td>0.4</td>
<td>Nt.</td>
<td>D</td>
<td>0.22</td>
</tr>
<tr>
<td>H.M.</td>
<td></td>
<td>0.13</td>
<td>0.3</td>
<td>Nt.</td>
<td>D</td>
<td>0.16</td>
</tr>
<tr>
<td>L.S.</td>
<td></td>
<td>0.13</td>
<td>1.0</td>
<td>Nt.</td>
<td>D</td>
<td>0.14</td>
</tr>
<tr>
<td>M.J.W.</td>
<td></td>
<td>0.12</td>
<td>1.0</td>
<td>Nt.</td>
<td>I</td>
<td>0.24</td>
</tr>
<tr>
<td>T.T.</td>
<td></td>
<td>0.14</td>
<td>1.5</td>
<td>Nt.</td>
<td>D</td>
<td>0.23</td>
</tr>
<tr>
<td>M.P.</td>
<td></td>
<td>0.15</td>
<td>2.5</td>
<td>Nt.</td>
<td>R</td>
<td>0.18</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td>0.128</td>
<td>1.15</td>
<td>—</td>
<td>—</td>
<td>0.191</td>
</tr>
</tbody>
</table>

* Key to abbreviations: NVC, normal ventricular complex; LVH, left ventricular hypertrophy; CLBBB, complete left bundle-branch block; L&RVH, left and right ventricular hypertrophy; Nt., notched; R, rounded; D, diphasic; I, inverted; P, peaked.

Results

Rate and Rhythm. The ventricular rate varied between 52 and 135 beats per minute, with an average of 80. There was no significant difference in the heart rate between patients with normal sinus rhythm and those with atrial fibrillation.
Sixteen patients (25 per cent) showed normal sinus rhythm. Two of these (12 per cent) were males and 14 (88 per cent) females. The incidence of normal sinus rhythm was 9 per cent among males and 32 per cent among females (table 1).

**Atrial and Ventricular Complexes.** The data concerning the P waves, P-R intervals, and QRS complexes are presented in tables 2-9. Thirty-three patients showed a normal ventricular complex, 19 showed a pattern of left ventricular hypertrophy, 6 showed right ven-
tricular hypertrophy, 3 showed incomplete right bundle-branch block, and 1 each showed left bundle-branch block, combined right and left ventricular hypertrophy, right ventricular hypertrophy and incomplete right bundle-branch block, right and left ventricular hypertrophy and incomplete right bundle-branch block (tables 2-9).

**DISCUSSION**

The sixty-five cases reported represent patients with proved, functionally pure, and advanced mitral regurgitation. The results of the present analysis are in partial disagreement with previous reports and current teaching. Preceding series admitted cases diagnosed only on clinical grounds. The difficulty in distinguishing major mitral regurgitation from combined mitral stenosis and regurgitation by clinical methods alone is generally appreciated. Tricuspid regurgitation can also masquerade as mitral regurgitation in patients with pure mitral stenosis. It is for these reasons that special diagnostic studies including cardiac ventriculography and left heart catheterization often must be employed to establish more exactly the amount of obstruction and leak. Electrocardiographic reports on patients with rheumatic mitral valve disease in which these tests are not performed, or in which direct palpation of the valve at surgery is not carried out, can justifiably be viewed with skepticism. Another factor to be considered is the generally advanced stage of the disease in our group as judged by the higher incidence of atrial fibrillation. These considerations supply possible explanations for some discrepancies encountered between this and previous reports.

A rheumatic etiology was established in our cases, based on positive historical data, biopsy of atrial appendages, and autopsy findings.
### Table 6.—Duration of the Ventricular Activation Time

<table>
<thead>
<tr>
<th>Lead V1-V5</th>
<th>NVC</th>
<th>LVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>CLBBB</th>
<th>LVH</th>
<th>RVH-LVH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. pts.</td>
<td>28</td>
<td>19</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Range</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.04</td>
<td>0.05</td>
<td>0.02</td>
<td>0.08</td>
<td>0.09</td>
</tr>
<tr>
<td>Mean</td>
<td>0.02</td>
<td>0.02</td>
<td>0.01</td>
<td>0.06</td>
<td>0.08</td>
<td>0.02</td>
<td>0.08</td>
<td>0.09</td>
</tr>
</tbody>
</table>

* Key to abbreviations: NVC, normal ventricular complex; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy; IRBBB, incomplete right bundle-branch block; CLBBB, complete left bundle-branch block; L&RVH, left and right ventricular hypertrophy; N, normal; PRO, prolonged.

### Table 7.—Size of the S Wave in the Representative Precordial Leads

<table>
<thead>
<tr>
<th>S Wave Lead V1</th>
<th>NVC</th>
<th>LVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>CLBBB</th>
<th>LVH</th>
<th>RVH-LVH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. pts.</td>
<td>22</td>
<td>17</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>43</td>
</tr>
<tr>
<td>Range</td>
<td>3.0</td>
<td>3.0</td>
<td>2.0</td>
<td>5.0</td>
<td>34.0</td>
<td>6.0</td>
<td>2.0</td>
<td>34.0</td>
</tr>
<tr>
<td>Mean</td>
<td>7.5</td>
<td>9.9</td>
<td>2.0</td>
<td>6.3</td>
<td>34.0</td>
<td>6.0</td>
<td>2.0</td>
<td>34.0</td>
</tr>
</tbody>
</table>

* Key to abbreviations: NVC, normal ventricular complex; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy; IRBBB, incomplete right bundle-branch block; CLBBB, complete left bundle-branch block; L&RVH, left and right ventricular hypertrophy; N, normal; INC, increased.
Females were encountered twice as commonly as males. The mean age for the 2 sexes was approximately the same and fell within the first half of the fourth decade of life.

In contrast with previous reports, atrial fibrillation was encountered in 3 out of 4 patients. This is a higher incidence than the 1:2 ratio reported for patients with proved mitral stenosis and it is probably related to the marked dilatation of the left atrium usually present in mitral regurgitation. The frequency of atrial fibrillation was directly proportional in a linear fashion to the age of the patients and, presumably, to the duration of the disease. It occurred in 90 per cent of the males and 66 per cent of the females.

Normal sinus rhythm was found exclusively and in equal proportion among patients with a normal ventricular complex or left ventricular hypertrophy (and in 1 patient with combined ventricular hypertrophy). The atrial complex of all the patients fulfilled at least one of the criteria for the diagnosis of left atrial enlargement. This is in contrast with the normal P waves reported in previous studies.

For a consideration of the ventricular complex the patients were classified into 4 groups: normal ventricular complex, left ventricular hypertrophy, combined ventricular hypertrophy, and right ventricular hypertrophy. This grouping was made not only for greater simplicity but also with the realization that some electrocardiographic patterns, although morphologically different, merely represent progressive stages of an identical process. It has been repeatedly emphasized, for instance, that incomplete or complete bundle-branch block, obtained in a ventricle performing an increased amount of work, may be considered as the expression of ventricular hypertrophy. Rheumatic mitral regurgitation, in its typical form, initially increases the work of the left ventricle. Subsequently, with the production of vascular changes in the pulmonary circuit, the right ventricle is subjected to an increased load. It seems quite logical, therefore, to group left ventricular hypertrophy and left bundle-branch block together and to do the same for the right ventricular

### Table 8.—R/S Ratios in Preordial Leads

hypertrophy and incomplete right bundle-branch block.

In disagreement with the current concepts and teachings, which stress the frequent occurrence and diagnostic value of left axis deviation in mitral insufficiency,1, 20 the present study revealed the mean manifest electric axis of ventricular activation (AQQRS) to be either normal or deviated to the right and the electric position to be intermediate to vertical in the great majority of cases. Left axis deviation and semi-horizontal or horizontal position were found almost exclusively among patients with left ventricular hypertrophy and in a few cases with normal ventricular complex. No patients with left ventricular hypertrophy showed right axis deviation and conversely no patients with right ventricular hypertrophy showed left axis deviation.

A normal ventricular complex was present in 50 per cent of the total series. The female to male ratio was 4:1 in this group. Soulié and co-workers6 found at right heart catheterization increased pulmonary resistance in a few cases with normal ventricular complexes and suggested that they may represent patterns of balanced left and right ventricular hypertrophy. A normal ventricular complex could therefore be the resultant of well-balanced potentials in hypertrophy of both ventricles. These considerations probably apply also to the present series and some of the normal tracings may belong in the group of combined left and right ventricular hypertrophy, which is unexpectedly small. Theoretically one would expect a higher incidence of combined rather than right ventricular hypertrophy in mitral regurgitation. From a quantitative point of view the electrocardiogram of mitral regurgitation may be represented by a pyramid where the base is made up by the normal ventricular complex and the apex by the right ventricular hypertrophy pattern with left and combined ventricular hypertrophy in between.

Left ventricular hypertrophy was present in approximately 30 per cent of our series. This lower incidence in comparison to previous reports6, 7 is explicable probably by our close adherence to the rigid criteria of Sokolow and Lyon15, 16 and Wilson and associates,11 which have been shown to be highly accurate in a recent anatomic-electrocardiographic correlative study.24 In some instances6 the sole evidence of left ventricular hypertrophy consists of RS-T and T changes.

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**Table 9.—Sums of R and S Waves in Precordial Leads**

<table>
<thead>
<tr>
<th>R Wave in V1+VL Wave in V6-V6</th>
<th>NYC</th>
<th>LVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>CLBBB</th>
<th>L&amp;RVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>IRBBB</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. pts.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>1.0</td>
<td>1.5</td>
<td>0.9</td>
<td>14.5</td>
<td>9.0</td>
<td>17.5</td>
<td>16.0</td>
<td>12.0</td>
<td>9.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Mean</td>
<td>3.5</td>
<td>2.8</td>
<td>5.9</td>
<td>14.5</td>
<td>9.4</td>
<td>17.5</td>
<td>16.0</td>
<td>12.0</td>
<td>4.7</td>
<td>15.0</td>
</tr>
</tbody>
</table>

**Table 9.—Sums of R and S Waves in Precordial Leads**

<table>
<thead>
<tr>
<th>R Wave in V1+VL Wave in V6-V6</th>
<th>NYC</th>
<th>LVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>CLBBB</th>
<th>L&amp;RVH</th>
<th>RVH</th>
<th>IRBBB</th>
<th>IRBBB</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. pts.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>33</td>
<td>11</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>10.0</td>
<td>18.0</td>
<td>35.5</td>
<td>3.0</td>
<td></td>
<td>14.3</td>
<td>52.0</td>
<td>61.5</td>
<td></td>
<td>40.0</td>
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<tr>
<td>Mean</td>
<td>31.5</td>
<td>33.0</td>
<td>56.0</td>
<td>19.5</td>
<td></td>
<td>33.0</td>
<td>52.0</td>
<td>61.5</td>
<td></td>
<td>33.0</td>
</tr>
</tbody>
</table>

| Group                        |     |     |     |       |       |       |     |       |       |       |
|------------------------------|     |     |     |       |       |       |     |       |       |       |
| Range                        | 10.0 | 18.0 | 3.0  | 16.9  |       | 52.0  | 61.5|       |       | 40.0  |
| Mean                         | 20.7 | 36.0 | 3.0  | 16.9  |       | 52.0  | 61.5|       |       | 26.8  |

*Key to abbreviations: NYC, normal ventricular complex; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy; IRBBB, incomplete right bundle-branch block; CLBBB, complete left bundle-branch block; L&RVH, left and right ventricular hypertrophy; N, normal; INC, increased.*
These changes, however, could be ascribable to digitalis effect rather than ventricular hypertrophy. In none of our cases was the diagnosis of left ventricular hypertrophy based on these changes alone. In this way false positives are eliminated, although a few patients with varying degrees of left ventricular enlargement may be excluded from this group.

Combined left and right ventricular hypertrophy was encountered in only 2 patients, or 3 per cent of our total group, against 37 per cent in the series by Soulié. Here again Soulié reported as typical of combined ventricular hypertrophy a tracing that seems to indicate only right ventricular hypertrophy.

The pattern of right ventricular hypertrophy comprised 15 per cent of the present series. The pathophysiologic and clinical features of some of these patients have been analyzed in a previous report. The finding of right ventricular hypertrophy in mitral insufficiency in a relatively high number of cases is certainly unusual and in sharp contrast with the present teaching. It is important to emphasize that right ventricular hypertrophy is not an unusual feature of rheumatic mitral regurgitation. Its presence, therefore, in patients with mitral valve disease is of less value than other clinical features in the differentiation between mitral stenosis and regurgitation.

**Summary**

The electrocardiographic data of 65 proved cases of dynamically significant mitral regurgitation are presented. In the present series rheumatic fever was the common etiologic factor. Females were encountered twice as often as males. Atrial fibrillation was observed in 3 out of 4 patients and evidence of left atrial enlargement was constant when sinus rhythm was present. The axis of the QRS complex was normal or deviated to the right in most instances. The ventricular complex was normal in 50 per cent of the cases while left ventricular hypertrophy occurred in 30 per cent, right ventricular hypertrophy in 15 per cent, and combined ventricular hypertrophy in 5 per cent of the cases.

Discrepancies between the present and previous series are discussed and possible explanations for them and for the lower than expected incidence of combined ventricular hypertrophy are proposed.

**Acknowledgment**

The authors wish to express their appreciation to Miss Evelyn deSimone, Miss Judith A. Creed, and Mrs. Lois E. Pugh for their technical assistance.

**Summario in Interlingua**

Es presentate le datos electrocardiographic ab 65 provate casos de regurgitacion mitral de grados dynamicamente significative. In iste serie, febre rheumatic esesva le factor etiologic commun. Femninhas esseva duo vices plus frequente que mascullos. Fibrillacion atrial esesva observate in 3 ex omne 4 pati-entes, e signos de allargamento sinistro-atrial esesva presente in omne casos con ritmo sinusual. In le majoritate del casos, le axe del complexo QRS esseva normal o deviava in direction dextrorse. Le complexo ventricular esesva normal in 50 pro cento del casos. Hypertophia sinistro-ventricular occurreva in 30 pro cento, hypertophia dextero-ventricular in 15 pro cento, e hypertophia ventricular combine in 5 pro cento del casos.

Discrepantias inter le presente e previe series es discutite. Es proponite explicationes possibile pro illos e etiam pro le inexpectate-mente basse incidentia de hypertophia ven- tricular combine.

**References**


An Electrocardiographic Analysis of Sixty-Five Cases of Mitral Regurgitation
L. G. BENTIVOGLIO, J. F. URICCHIO, A. WALDOW, W. LIKOFF and H. GOLDBERG

Circulation. 1958;18:572-580
doi: 10.1161/01.CIR.18.4.572

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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/content/19/2/313.2.full.pdf
insufficiency, for instance, in parents and children, in siblings, and in twins. In such observations the author sees further evidence that an inherited predisposition must be present for the acquisition of extraneous cardiovascular diseases.

**REVIEWs IN CARDIOVASCULAR DISEASE**


**Wesson, L. G., Jr.: Glomerular and Tubular Factors in the Renal Excretion of Sodium Chloride.** Medicine 36: 281 (Sept.), 1957.


**Errata**

*Various authors have requested that the following changes be made in their published papers:*


On page 348, paragraph 3 under Method, should read "If Dp is the blood pressure (systolic or diastolic) in the standing posture during drug action . . ."


On page 572, table 5, Lead V6, Group Range, 56.0 under LVII should be 36.0; table 5, Lead V1, No. pts., — (INC) under RVH & IRBB should be 1.

On page 576, table 6, Lead V5-V6, No. pts., 48 (N) and 17 (PRO) under Total should be 49 (N) and 16 (PRO).

On page 577, table 8, R/S Ratio in Lead V1, No. pts., 37 (X) under Total should be 27.

On page 578, table 9, R Wave in V1 + S Wave in V5-V6, No. pts., 18 (N) under Total should be 19.


On page 831, line 13, should read "... workers may expend energy at a higher rate off the job than on it."