Serial Electrocardiograms in Hypertensive Cardiovascular Disease

Rosalie A. Dunn, Ph.D., Robert J. Zenner, M.D., and Hubert V. Pipberger, M.D.

SUMMARY A graphic method for depicting serial changes in electrocardiograms is described and demonstrated for patients being treated in an antihypertension clinic. For these patients, the diagnostic categories, normal, left ventricular hypertrophy, and biventricular hypertrophy, are of primary interest. For each electrocardiogram, 14 measurements are used to compute posterior probabilities for each of the three categories. A triangular grid is used to plot each set of probabilities for an electrocardiogram as one point, which by its position in the triangle can be related to the three diagnostic statements are reported together with electrocardiographic measurement changes.

In certain types of heart disease, particularly those associated with ventricular hypertrophies, it would be desirable and helpful if serial changes in the electrocardiogram could be used as an indicator of the course of the disease where the patient’s latest record is viewed in relation to previous ones. Based on observations by Poblete and coworkers, this should be feasible in cases with hypertensive cardiovascular disease (HCVD). When comparing records from patients on antihypertensive therapy with those in a placebo group, they found that the electrocardiographic changes in the treated patients showed a tendency toward normalization which was seen rarely in records of untreated patients who had either increased or unchanged signs of left ventricular hypertrophy (LVH). Results of this study and many other observations on dynamic changes of LVH signs in the electrocardiogram re-emphasize the fact that the electrocardiographic diagnosis of LVH is not an all-or-none statement. Most observers have felt that a relationship between the degree of LVH and its electrocardiographic characteristics must exist. However, as reviewed by Holt et al.,

12 quantitative correlations have met with only moderate success. Of the many factors which may limit quantitative correlations, two appear most obvious. In patients with persisting left ventricular overload, hypertrophy develops, not

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only in the left but also in the right ventricle, particularly after episodes of congestive heart failure (CHF). In this situation, increase in electromotive forces of the right ventricle tends to counterbalance and cancel left ventricular forces. This is usually accompanied by diminution of high QRS voltage in leftward and posterior direction which represents the mainstay of the electrocardiographic diagnosis of LVH.18

Another factor which can lead to a decrease of QRS voltages is cardiac enlargement.19 It has been postulated that the increased intracavitary blood mass has a short-circuiting effect on cardiac potentials. In a clinical situation, biventricular hypertrophy (BVH), cardiac enlargement, and possibly other factors may influence the electrocardiogram simultaneously to a variable degree which may explain the rather modest success with quantitative correlations between anatomic indicators of LVH and the electrocardiogram.

In the electrocardiographic computer program developed in the Veterans Administration, probabilities are being computed for all diagnostic entities under consideration.14, 18 Classification of ventricular hypertrophy cases was found most efficient. Compared to conventional electrocardiographic analysis, more than twice as many cases with HCVD could be diagnosed as having LVH.14 In hypertensives, only three diagnostic categories are under primary consideration; namely, normal (N), LVH, and BVH. The question arose, therefore, whether probabilities for the presence of these three conditions could not be used in a practical and efficient display of longitudinal changes which would allow immediate evaluation of the patient's status in terms of increase or decrease of ventricular hypertrophy. A display system based on triangular coordinates was tested for its usefulness in following serial changes in hypertensive patients. Since the number of patients who are being treated for this disease is increasing at a rapid rate, an efficient display of these changes would be a welcome aid in evaluating the course of the disease and the efficacy of therapeutic measures.

Materials and Methods

Basic Elements

The basic data needed for the analysis of serial changes are provided routinely by the Veterans Administration computer program for the automated analysis of orthogonal electrocardiograms (Frank leads), which has been described14 and documented with respect to clinical usage and diagnostic performance.14 This program for analysis of single resting electrocardiograms is in daily, routine operation at this hospital as well as in several others in this country and abroad. An information-retrieval system for comparison of serial electrocardiograms has also been reported4 and has been operating in the Veterans Administration Hospital in test mode.

Information Retrieval

The storage files of previously analyzed electrocardiograms are automatically maintained by the computer and can be queried through a two-way communications terminal by specifying a combination of patient's name and social security number. The system normally handles a maximum of three records per patient, unless specific programming instructions are given to accommodate a larger number. This is done, for instance, for all records from the intensive care unit and for tracings earmarked for special studies, such as the present one.

As reported previously,2 the program for serial electrocardiograms compares diagnostic statements together with electrocardiographic measurement changes which are most pertinent for the diagnoses under consideration. The display of changes in serial tracings of patients with HCVD, which is described in the present report, is generated upon special request.

Triangular Grid Display

To follow change in those patients with HCVD, an additional analysis is available whereby probabilities are computed for three diagnostic categories, N, LVH, and BVH. They are of primary concern because: 1) N is considered the baseline state; 2) due to increase in left ventricular work in patients with hypertension, LVH is usually the first electrocardiographic abnormality to be observed; and 3) if the blood pressure remains at a high level, BVH develops, particularly after episodes of CHF. The actual measurements used in the computation, consisting of 11 QRS and 3 ST-T amplitudes, are listed in table 1, together with the mean values for each of the three categories and the pooled standard deviations. The electrocardiographic variables were obtained from a data base consisting of 510 normals,17 939 patients with HCVD,14 and a group of 200 patients with documented BVH.13 The method to compute posterior probabilities is given in the Appendix. The model used for these computations assumes that each electrocardiogram fits into one of three alternative categories, N, LVH, and BVH. The numerical value or probability level associated with a particular category indicates how typical the electrocardiographic pattern is for that category. Usually, the decision process specifies that the category with the highest probability level is "the answer." Thus, an electrocardiogram with probability 0.65 for N, 0.20 for LVH, and 0.05 for BVH would be classified as normal, and the probability level would indicate it is 3 ½ times as likely to be normal as it is to be the nearest alternative, LVH.

After being computed, the three probabilities representing one electrocardiogram are graphed as one point in a triangular grid, a homogeneous coordinate system as

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Normal</th>
<th>LVH</th>
<th>BVH</th>
<th>Pooled standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Rx</td>
<td>1.116</td>
<td>1.460</td>
<td>1.432</td>
<td>.48</td>
</tr>
<tr>
<td>2. Ry</td>
<td>0.912</td>
<td>0.930</td>
<td>1.339</td>
<td>.45</td>
</tr>
<tr>
<td>3. Q</td>
<td>-0.372</td>
<td>-0.291</td>
<td>-0.224</td>
<td>.21</td>
</tr>
<tr>
<td>4. 2/8 QRS</td>
<td>-0.254</td>
<td>-0.164</td>
<td>-0.016</td>
<td>.24</td>
</tr>
<tr>
<td>5. 3/8 QRS</td>
<td>0.862</td>
<td>1.042</td>
<td>0.971</td>
<td>.40</td>
</tr>
<tr>
<td>6. Max QRS</td>
<td>1.311</td>
<td>1.196</td>
<td>1.458</td>
<td>.51</td>
</tr>
<tr>
<td>7. Max QRS</td>
<td>1.336</td>
<td>1.629</td>
<td>1.823</td>
<td>.51</td>
</tr>
<tr>
<td>8. 6/8 QRS</td>
<td>0.409</td>
<td>0.517</td>
<td>0.649</td>
<td>.28</td>
</tr>
<tr>
<td>9. 5/8 QRS</td>
<td>0.922</td>
<td>1.088</td>
<td>1.328</td>
<td>.50</td>
</tr>
<tr>
<td>10. 5/8 QRS</td>
<td>0.808</td>
<td>0.984</td>
<td>1.262</td>
<td>.46</td>
</tr>
<tr>
<td>11. Jmax</td>
<td>0.222</td>
<td>0.301</td>
<td>0.359</td>
<td>.19</td>
</tr>
<tr>
<td>12. Jmax</td>
<td>0.079</td>
<td>0.110</td>
<td>0.154</td>
<td>.06</td>
</tr>
<tr>
<td>13. 3/8 ST-T</td>
<td>0.064</td>
<td>-0.017</td>
<td>-0.091</td>
<td>.07</td>
</tr>
<tr>
<td>14. Ts</td>
<td>0.256</td>
<td>0.053</td>
<td>-0.134</td>
<td>.18</td>
</tr>
</tbody>
</table>

*All measurements represent amplitudes, expressed in millivolts.
blood pressure was poorly controlled and repeatedly reached 250/150 mm Hg. He has been on diuretics, Reserpine, and Aldomet but did not follow the prescribed regime regularly until 1971 when his blood pressure came down to 165/108 mm Hg. An electrocardiogram taken at this time (#1 in fig. 2) corresponds to a probability for LVH of 79%. To improve blood pressure control, the patient was readmitted in 1972 when the readings averaged 160/109 mm Hg with a probability for LVH of 72% (#2 of fig. 2). Since that time, the patient has been on 50 mg Hydrodiuril and 0.25 mg Reserpine daily with controls at regular intervals. In 1973 his blood pressure came down to an average of 140/100 mm Hg which was reflected in the electrocardiogram by a decrease in the probability for LVH to 40% (#3 in fig. 2).

The course of this patient, which was quite erratic during the early part of his disease, has shown consistent decreases in blood pressure since 1971 and a corresponding decrease in degree of LVH as shown by the triangular electrocardiographic display.

**Patient No. 0420**

Sequential electrocardiograms shown in figure 3 were obtained from a 53-year-old black male who was found to be hypertensive for the first time in 1945. Antihypertensive therapy was started on a regular basis only in 1965. In 1967 he was admitted because of chest pain, but myocardial infarct was ruled out. By history, the patient had repeated episodes of CHF. His blood pressure in the hospital averaged 180/110 mm Hg. An electrocardiogram taken at this time showed a probability for BVH of 99.5% (#1 of fig. 3).

Since 1972 his blood pressure has been well controlled, rarely exceeding 150/100 mm Hg. An electrocardiogram taken in 1973 (#2 of fig. 3) showed marked improvement with a shift from BVH toward LVH with a probability for the latter of only 65%. The blood pressure at this time was

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**Results**

First, some examples of the display method will be shown which provide an illustration of the changes which may be observed in individual hypertensive patients over a period of time. These examples will be followed by grouped data from record samples of normal subjects and patients with HCVD.

**Case Reports**

**Patient No. 0068**

The records displayed in figure 2 were obtained from a 57-year-old black male who was found to be hypertensive for the first time in 1969. He had suffered a cerebrovascular accident leading to right hemiparesis. In subsequent years, the
157/93 mm Hg. When a third electrocardiogram was recorded in 1974, blood pressure was 141/92 mm Hg (#3 of fig. 3).

In summary, this is a patient with a history of HCVD of approximately 30-years' duration whose blood pressure has been satisfactorily controlled only for the last four years. It is noteworthy that he had several episodes of CHF which led, almost as a rule, to BVH. As a consequence of better blood pressure control in more recent years, electrocardiographic signs of BVH subsided almost completely.

**Patient No. 0098**

This patient is a 65-year-old black male who was found to be hypertensive in 1966. His blood pressure averaged at that time 200/110 mm Hg. He was treated with Reserpine and Hydrodiuril, and blood pressure decreased to 171/105 mm Hg later in the year when the first electrocardiogram (#1 of fig. 4) was taken in our laboratory. The probability for LVH was 70% at this time. Blood pressure continued to decrease and was 138/84 mm Hg at the time of the next electrocardiographic recording (#2 in fig. 4). Probability for LVH had decreased to 46% with a concomitant rise in the probability for being normal to 52%. In 1973, blood pressure rose again, and the average reading for three subsequent clinic visits was 153/95 mm Hg. This was reflected in his electrocardiogram with a probability for LVH of 68% (#3 of fig. 4) which required an increase in antihypertensive medication.

**Patient No. 0147**

This patient is a 59-year-old black male who was found to be hypertensive in 1951. His status remained unchanged until 1957 when he underwent a bilateral lumbar sympathectomy. The blood pressure remained high in spite of vigorous and varied regimens. The diastolic pressure was rarely below 110 mm Hg. There were no signs or symptoms of CHF, and the heart size was consistently reported normal. The first electrocardiogram taken at our laboratory in 1971 (#1 of fig. 5) showed a probability for LVH of 41%. Blood pressure at that time was 180/104 mm Hg. The second electrocardiogram taken eight months later showed a slight increase in the probability for LVH to 57% with an increase in blood pressure to 188/117 mm Hg (#2 in fig. 5). The third electrocardiogram recorded in 1974 (#3 in fig. 5) showed a further increase in LVH with a probability of 67%. Blood pressure at that time had risen to 205/130 mm Hg.

This patient's blood pressure was extremely difficult to
control in spite of vigorous treatment. Signs of LVH increased together with rising blood pressure levels. The persistent left ventricular overload seemed to be relatively well tolerated without indications of CHF, increase in heart size, or evidence of BVH.

Patient No. 1002

This patient is a 57-year-old white male who was found to be hypertensive in 1953. He was one of the first patients in the U.S. who was treated successfully with Hexamethonium. In spite of initial difficulties in reducing and stabilizing his blood pressure, his status has been stable since 1962. This was accompanied by gradual decrease in heart size which was enlarged when first seen. The first electrocardiogram taken in our laboratory in 1964 showed a probability for LVH of 67% (#1 in fig. 6). Blood pressure at that time was 133/102 mm Hg. The following records were taken in 1971, 1973, and 1974 (#2, #3, and #4, respectively, in fig. 6). Probabilities for LVH varied between 66 and 72%. Blood pressure readings ranged from 138/98 to 120/83 mm Hg.

This is a patient with excellent blood pressure control. The stable status is well depicted in the triangular display of LVH probabilities.

From the individual examples given above, it can be seen that the course of the disease, in rather general terms, is well depicted in the triangular displays. The first record of each case is to be considered as the point of departure; subsequent records show the rate of change and its direction which may indicate improvement or deterioration. The observations reported here represent only long-term follow-up, measured in terms of months or years. As described previously,19 acute lowering of blood pressure in hypertensive patients does not lead to significant changes in the electrocardiogram. Except for well-known variations in certain measurements,9 variability over short periods is not significant, especially in probability levels. Even observations made over longer periods of time without significant change in blood pressure or heart size do not change the plotted probabilities significantly, as seen in figure 6.

In order to provide an indication of the sensitivity and specificity of this display method, 273 records were selected at random from our electrocardiographic record file of normal subjects and patients with sustained hypertension. One hundred normals, taken from a larger series previously reported,19 had no evidence of past or present cardiovascular disease. An additional 100 records were taken from patients with sustained blood pressure elevation of 150/90 mm Hg or more without past or present symptoms of CHF. For the purpose of the study, they were considered pure LVH cases. A third group of 73 HCVD patients was assumed to be typical for BVH. All showed symptoms and signs of CHF, such as shortness of breath at rest and/or on exertion, orthopnea and/or paroxysmal nocturnal dyspnea, pedal edema and/or hepatomegaly.

The records of the three groups were classified according to the scheme shown in figure 7 where the area within the triangle was divided into three equal parts. The distribution of cases is given in table 2 and in figures 8–10. As seen in the first column of the table, 85% of the normals fell within the normal range with 13% misclassified as LVH and 2% as

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** Plot of probabilities of four electrocardiograms from patient 1002 with excellent blood pressure control. This stability agrees with the probabilities computed from electrocardiographic measurements.

![Figure 7](http://circ.ahajournals.org/)

**Figure 7.** Triaxial grid divided into three equal zones for classification. Perpendicular lines to the sides are drawn from the center of the triangle, represented by the dot (p, q, r) = (1/3, 1/3, 1/3). Thus, for an electrocardiogram to be classified normal (N), the corresponding probability (p) would necessarily be greater than 1/3, while the probabilities for left ventricular hypertrophy (LVH) (q) and for biventricular hypertrophy (BVH) (r) would each be less than 1/3.

<table>
<thead>
<tr>
<th>Cutoff classification</th>
<th>Clinical classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>LVH</td>
</tr>
<tr>
<td>Normal</td>
<td>86</td>
</tr>
<tr>
<td>LVH</td>
<td>13</td>
</tr>
<tr>
<td>BVH</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

Agreement between clinical diagnosis and ECG classification is shown in the diagonal with figures in italics.
BVH. Out of the group with pure LVH, 52% were so classified. Thirty-six percent were considered N; and 12%, BVH. The records from patients assumed to have pure BVH were classified correctly in 69% of the cases with 27% as LVH and 4% as N.

In figures 8–10, the actual distribution of the cases is shown. The majority of the normals are clustered at the apex with some spread toward LVH. There are no common characteristics in the normals misclassified as LVH, but it would be of interest to follow these patients to find out whether they develop hypertension or other cardiovascular disease.

The pure LVH cases are distributed more widely with a sizable number of cases in the corner of N. The BVH cases in figure 10 were mostly clustered in the BVH corner with a moderate spread toward LVH.

From these data, it appears that the best classifications can be obtained for the two extreme categories, N and BVH, with a wider spread for LVH which was the most inhomogeneous group clinically. Of course, a certain percent of classification errors should be anticipated from any statistical scheme which attempts to distinguish among these three diagnostic categories.

Discussion

In following the course of patients with HCVD, it appears both desirable and useful to have a fast and readily available procedure for evaluating patients' present status in relation to the past and, more importantly, in relation to the efficacy of antihypertensive medication. In our experience with the described display method over the last two and one-half years, very useful information could be obtained by comparing numerical diagnostic probabilities for having LVH or BVH or for being normal. It appears obvious that one is practically always dealing with a certain degree of LVH or BVH in a given hypertensive patient at a given time. This degree may vary from minimal to most advanced. It is also obvious that an electrocardiographic interpretation resulting in a yes-no interpretation for the presence or absence of hypertrophy without further differentiation is only of limited clinical use. Computation of diagnostic probabilities on a numerical scale proved, in our experience, more informative and promising as a clinical tool for continuous patient follow-up with definite advantages over the rather simplistic N-LVH separation. Since rapidly increasing numbers of electrocardiograms are being analyzed by computer means, the described procedure lends itself easily to clinic or office use provided computer access is available through an appropriate terminal. The method, however, is not applicable to patients who have suffered myocardial infarction or to those with other abnormalities such as right ventricular hypertrophy or ventricular conduction defects.
As pointed out earlier, the electrocardiogram of a patient with LVH may be modified by a number of known and possibly some unknown factors. Cardiac dilatation and hypertrophy of the contralateral right ventricle are known to exert a significant influence. In fact, multiple regression using left ventricular mass (recorded from 140 patients) as the dependent variable and three electrocardiographic measurements as independent variables has yielded multiple correlation coefficients greater than 0.7. (Pipberger HV, Dunn RA, Holt JH Jr: Unpublished observations) The degree of this influence needs further investigation particularly in terms of the effect on diagnostic probabilities in the described procedure. Basically, a new and unknown record is compared with the large record samples which formed the basis for the development of the classification procedures. Although electrocardiographic information was excluded from the selection criteria of the original data base in order to avoid any bias, the clinical criteria used for case selection were rather broad as, for example, sustained hypertension as an indication of left ventricular overload and hypertrophy. Only the original BVH sample consisted exclusively of autopsy cases. Factors which may modify the electrocardiogram in a secondary fashion, such as cardiac dilatation, were not taken into account in the formation of the original data base. In order to refine the classification and display method described, collection of more records appears desirable from cases which would allow quantitative correlations between the degree of hypertrophy and the electrocardiographic parameters used for computing diagnostic probabilities for LVH and BVH. Since pure LVH is not frequently seen at autopsy, noninvasive procedures, such as echocardiograms, ventriculograms, and other radiologic studies, may lead to more quantitative estimates of the degree of hypertrophy. A study is in progress to correlate electrocardiographic changes directly with left ventricular size by using ventricular angiograms and echocardiograms.

Despite some individuals in whom the correspondence between clinical and graphic patterns was less obvious, the relationship between computed probabilities for ventricular hypertrophy on the one hand and the clinical course of the disease on the other appeared quite satisfactory for the majority of cases considered. The reason for this relatively good agreement may be due to the fact that the emphasis of the display is more on the direction of change rather than on relative position of an individual record. Since the heart represents one of the main affected end-organs in hypertensive disease, an additional measure of cardiac changes during the course of the disease may prove a helpful clinical indicator both for evaluation of the efficacy of treatment and for prognosis.

The grouped data showed a relatively wide spread of findings which should not be surprising. Some of the limitations of the method indicated above undoubtedly contribute to the overlap between groups. The pure LVH sample, for instance, represents a wide spectrum of cases ranging from mild to severe. The only distinction between the LVH and BVH groups was the absence of CHF in the former. A more detailed definition of the various record samples might improve differentiation among the groups. As noted earlier, overlap among the three diagnostic entities does not necessarily affect the utility of the display in individual cases because the direction of change is most informative in the follow-up of individual patients.

Acknowledgment

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Appendix

The calculation of posterior probabilities for orthogonal electrocardiograms was described by Cornfield et al. For a general statement of the model, let x denote a k-dimensional vector of measurements for patients; m, the number of possible disease categories; and i, a particular disease group from the m possibilities. If f(x|i) denotes the conditional probability (or density function) of x for those in disease category i, and g_m, the unconditional (prior) probability of being in disease category i, then the posterior probability of being in category i, given the measurement vector x, is Bayes' theorem

\[
P(i|x) = \frac{g_i f(x|i)}{\sum_{j=1}^{m} g_j f(x|j)}
\]

For this application, m, the number of groups, equals three: viz., normal, LVH, and BVH; k, the number of measurements, equals fourteen, listed in the first column of table 1. The density function f(x|i) has been taken as a multivariate normal with mean vector \(\mu_i\) and pooled covariance matrix \(\Sigma\). Estimates of the mean vectors for normal, LVH, and BVH are listed in columns 2, 3, and 4, respectively, of table 1. The diagonal elements of the estimated covariance matrix are listed in the final column. The prior probabilities used in this application are \(g_1 = g_2 = g_3 = 1/3\).

Thus, the computational procedure becomes simplified when it is noted that

\[
\log f(x|j) / f(x|i) = lx - [(\mu_j - \mu_i) / 2] A \Lambda \n
\]

where A is the vector of linear discriminant coefficients = \(\Sigma^{-1} (\mu_j - \mu_i)\).

References

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The Conduction System in Tricuspid Atresia With and Without Regular (d-) Transposition

SAROJA BHARATI, M.D., AND MAURICE LEV, M.D.

SUMMARY
This is a serial section examination of conduction system in six hearts with tricuspid atresia. Four had regular (d-) complete transposition and two did not have transposition. The conduction system was more or less the same in all the hearts. The atrioventricular (A-V) node was in the normal position posteriorly and was short. The A-V bundle was situated in the left ventricular aspect of the subendocardium and passed posteriorly to the ventricular septal defect. Even though this type of conduction system is abnormal in some respects, it is not the type one finds in single ventricle with small outlet chamber with regular (d-) transposition. In those hearts an anteriorly located A-V node is present. These findings further substantiate the concept that tricuspid atresia with or without transposition is not a form of single (primitive) ventricle.

WE HAVE PREVIOUSLY STUDIED THE GROSS MORPHOLOGIC CHARACTERISTICS OF TRICUSPID ATRESIA with and without regular (d-) transposition. These anomalies were not found to be forms of single (primitive) ventricle with small outlet chambers, contrary to the views of Anderson et al. The present work investigates this controversy further by comparing the conduction systems of these anomalies with those in hearts with a single ventricle.

Materials and Methods
We examined the conduction system in two cases of tricuspid atresia without transposition (cases 1, 2) and four cases of tricuspid atresia with regular (d-) transposition (cases 3-6). In cases 2-6 a block was fashioned from the sinoatrial (SA) node in a manner previously described and every tenth section was retained. In case 1 the SA node was not studied. In all cases a block was fashioned from the distal (downstream) part of the atrial septum beginning at the proximal wall of the coronary sinus, taking in the adjacent part of the right and left atrial walls and the proximal (upstream) part of the ventricular septum and the adjacent posterior wall of the left ventricle and the entire right ventricle. These blocks were fashioned and serially sectioned at various angles to the pulmonary valve anulus more or less in the Mahaim method. In case 2 the block was completely serially sectioned and all sections were retained. In case 1 every seventh, in case 3 and 6 every fifth, in case 4 and 5 every tenth section was retained. Cases 1 and 2 were alternately stained with hematoxylin-eosin and Weigert-van Gieson stains, while cases 3-6 were stained consecutively with hematoxylin-eosin, Weigert-van Gieson and Gomori trichrome stains. In this manner 670 sections were examined in case 1, 3019 in case 2, 572 in case 3, 273 in case 4, 324 in case 5 and 466 in case 6.

Findings
Case 1
Gross Diagnosis
This patient had tricuspid atresia without transposition (fig. 1). Other defects included atrial and ventricular septal defects, a small right ventricle, and hypertrophy of both atria and left ventricle. The ventricular septal defect was situated beneath the right and noncoronary cusps. It measured 0.8 cm at its greatest dimension. It was not confluent with the aorta. It entered the right ventricle beneath the arch (crista) formed by the septal and parietal bands.
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