Electrocardiographic Findings in Cardiac Amyloidosis

By Albert J. Josselson, M.D., and Raymond D. Pruitt, M.D.

The electrocardiographic findings were analyzed in 15 patients found at the time of necropsy to have deposits of amyloid in the heart. Although 12 of the 15 tracings were regarded as abnormal, changes which could be held to be specific to cardiac amyloidosis have not been defined. The most common alterations encountered were auricular fibrillation, QRS complexes and T waves of low amplitude in the standard limb leads, and impaired auriculoventricular conduction.

Deposits of amyloid may be found in the hearts of individuals afflicted with primary systemic amyloidosis, with amyloidosis complicating multiple myeloma and with secondary amyloidosis. An entity exists, also, in which the heart alone is infiltrated by amyloid. The descriptive phrase, "amyloid localized to the heart," has been applied to this condition. The amount of amyloid deposited in the myocardium under this latter circumstance may be as extensive as in cases of primary systemic amyloidosis or the quantities may be functionally insignificant.

In another study, detailed consideration has been given to the clinical and pathologic findings in a series of 29 cases in which amyloid deposits were found in the heart at necropsy. The object of this presentation is to record the electrocardiographic findings as they were available in this series of cases.

Fifteen sets of electrocardiograms have been studied. Ten were obtained from the series of 29 cases just mentioned in which deposits of amyloid were localized to the heart. Four were obtained from cases in which primary systemic amyloidosis was present and one was taken from a case in which amyloid disease complicated multiple myeloma.

The degree of cardiac amyloidosis in these 15 cases is summarized in table 1. Vessels was a commonly associated finding in these 15 cases, in none was it of such extent as to be regarded as clinically significant. Congestive cardiac failure was the primary or contributory cause of death in 7 of the 15 cases.

The electrocardiograms in these 15 cases are reproduced in figures 1 to 4. An attempt has been made to assemble in each figure a group of tracings that have one or more features in common.

Figure 1 includes three tracings. In one of these cases (A-5), the disturbance may be placed in the category of right bundle branch block. In lead V1, the form of the primary ventricular deflection is of a qR type rather than of the RSR' configuration encountered more commonly in right bundle branch block. The other two electrocardiograms in this figure are perhaps the most unique in the entire series. In both, a Qr type of complex is present in lead aVR, as commonly occurs in right bundle branch block, but in neither does the form of the primary ventricular deflection in lead V1 support the presence of such a defect in conduction.

The electrocardiograms from six patients are reproduced in figure 2. Auricular fibrillation was present in each instance. The QRS complexes in the standard limb leads share the quality of low amplitude. The primary ventricular deflection in precordial lead V1 is of an rs or Rs type.

* The case numbers used herein correspond with those appearing in a study pertaining to the pathologic findings in cases of amyloid localized to the heart.
In figure 3, another instance (case 5) of auricular fibrillation is included. Only standard limb leads were recorded and from these limited tracings the presence of a left bundle branch block may be suspected but not established. The other two cases are characterized by the presence of complete auriculo-ventricular dissociation. In case A-3, the focus in which ventricular excitation arose apparently was in the left ventricle, while in case 18 that focus apparently was in the right ventricle.

In figure 4, three electrocardiograms are reproduced that have as a common feature the quality of being essentially normal.

**Comment**

Certain facts that appear pertinent to proper evaluation of these electrocardiograms are included in the legends for these figures. Figure 1 is the only illustration in which all tracings are derived from patients who had severe degrees of cardiac amyloidosis. In case A-4 (fig. 4), the heart was heavily infiltrated by amyloid, yet the electrocardiogram was essentially normal. It is impossible, therefore, to propose any exact correlation between the degree of cardiac involvement by amyloid and the changes encountered in the electrocardiogram. In the absence of such a correlation derived from quantitative considerations, the failure to find a consistently recurring electrocardiographic pattern among all cases

**Table 1.—Degree of Cardiac Amyloidosis in 15 Cases**

<table>
<thead>
<tr>
<th>Type of amyloidosis</th>
<th>Degree of involvement of heart by amyloid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild</td>
</tr>
<tr>
<td>Primary systemic</td>
<td>1</td>
</tr>
<tr>
<td>Complicating multiple myeloma</td>
<td>0</td>
</tr>
<tr>
<td>Localized in the heart</td>
<td>3</td>
</tr>
</tbody>
</table>

**Fig. 1.** Primary systemic amyloidosis. Case A-1: A 38 year old man whose blood pressure was 122/96. Cardiac amyloidosis was severe. The heart weighed 662 Gm. Case A-2: A 54 year old man whose blood pressure was 102/60. He had taken 0.1 mg. of digitoxin daily. Cardiac amyloidosis was severe. The heart weighed 460 Gm. (calculated normal weight = 245 Gm.). Case A-3: An 82 year old man whose blood pressure was 150/100. Cardiac amyloidosis was severe. The heart weighed 535 Gm. (calculated normal weight = 333 Gm.).
in the series, irrespective of the degree of cardiac amyloidosis, is not surprising. Certainly the incidence is unusually great of tracings in which the amplitude of the QRS and T deflections in the standard limb leads was low. Auricular fibrillation, present in 7 of the 15 cases, was unusually common, but it

must be remembered that in this series only those cases were included in which electrocardiograms had been recorded, and auricular fibrillation is a generally accepted indication for obtaining an electrocardiogram. Prolongation of the P-R interval, a finding in certain cases of cardiac amyloidosis reported in other series,\textsuperscript{1-3} was not encountered in our series, but complete auriculoventricular dissociation was present in two instances.

It is of interest that the tracings were abnormal in 12 of 15 cases of cardiac amyloidosis in which electrocardiograms were recorded, yet in no instance did this abnormality present

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Case 23: An 88 year old woman who died in a state of congestive cardiac failure and whose blood pressure was 100/50. She received digitoxin during the terminal phase of her illness but subsequent to the recording of these electrocardiograms. Cardiac amyloidosis was severe. The heart weighed 456 Gm. (calculated normal weight = 315 Gm.). Case 24: A 68 year old man whose death was attributed to bronchopneumonia and whose blood pressure was 135/75. Cardiac amyloidosis was moderate. The heart weighed 440 Gm. (calculated normal weight = 313 Gm.). Case 22: A 90 year old man whose death was ascribed to bronchopneumonia and congestive cardiac failure and whose blood pressure was 100/80. He had been receiving digitalis. Cardiac amyloidosis was moderate. Mitral stenosis of mild degree was present. The heart weighed 440 Gm. (calculated normal weight = 370 Gm.). Case 18: An 80 year old man who died after a pulmonary embolism and whose blood pressure was 140/90. Cardiac amyloidosis was severe. The heart weighed 450 Gm. (calculated normal weight = 350 Gm.). Case 26: A 75 year old man who died in a state of congestive cardiac failure and whose blood pressure was 150/85. He had received digitalis. Cardiac amyloidosis was severe. The heart weighed 875 Gm. (calculated normal weight = 343 Gm.). Case 9: A 63 year old woman who died in a state of hepato renal failure and whose blood pressure was 170/80. Cardiac amyloidosis was mild. The heart weighed 385 Gm. (calculated normal weight = 215 Gm.).}
\end{figure}

features typical of or justifiably confused with the findings of acute or old myocardial infarction. In one instance (fig. 2, case 26), the complexes recorded in lead V\textsubscript{5} were consistent with left ventricular hypertrophy, but in the remaining 11 abnormal electrocardiograms, the peculiarities were not those characteristic of
either right or left ventricular hypertrophy. By this devious course, the limited conclusion may be derived that in cardiac amyloidosis the electrocardiogram commonly is abnormal in a manner that fails to identify it with one of the specific patterns of abnormality frequently encountered in patients of advanced age.

Fig. 3. Case A-3: An 81 year old woman on whom a final diagnosis was made of primary systemic amyloidosis with congestive cardiac failure. The blood pressure was 220/80. Treatment with digitalis had been instituted 11 days before the electrocardiograms were recorded. Cardiac amyloidosis was mild. The heart weighed 585 Gm. (calculated normal weight = 215 Gm.). Case 13: An 80 year old man on whom a final diagnosis of congestive cardiac failure was made and whose blood pressure was 160/50. Cardiac amyloidosis was severe. The heart weighed 511 Gm. (calculated normal weight = 294 Gm.). Case 5: A 78 year old man on whom a final diagnosis was made of carcinoma of the pancreas and whose blood pressure was 130/50. Cardiac amyloidosis was severe. The heart weighed 570 Gm. (calculated normal weight = 343 Gm.).

SUMMARY AND CONCLUSIONS

A study has been made of the electrocardiograms obtained from 15 patients who had cardiac amyloidosis of various types. If the clinician is to recognize the existence of cardiac amyloidosis, he is most likely to arrive at the diagnosis by defining the presence of amyloid elsewhere in the body, as in primary systemic amyloidosis, or by identifying a disease, namely, multiple myeloma, that predisposes to amyloidosis.

If the deposits of amyloid are limited to the heart, then a method for positive diagnosis by clinical means is not evident. Suspicion that such a process exists may be entertained when congestive cardiac failure is encountered in an elderly individual unafflicted with the usual causes of such failure.

Fig. 4. Case 12: An 84 year old woman on whom final diagnosis included perforated peptic ulcer, peritonitis and congestive cardiac failure. The blood pressure was 158/60. Cardiac amyloidosis was mild. The heart weighed 250 Gm. (calculated normal weight = 215 Gm.). Case A-1; A 65 year old woman on whom a final diagnosis was made of amyloidosis complicating multiple myeloma and whose blood pressure was 146/92. Cardiac amyloidosis was severe. The heart weighed 440 Gm. (calculated normal weight = 244 Gm.). Case 11: A 75 year old man on whom a final diagnosis was made of carcinoma of the bladder and whose blood pressure was 160/100. Cardiac amyloidosis was moderate. The heart weighed 405 Gm. (calculated normal weight = 333 Gm.).

Definitive evidence cannot be had from the electrocardiogram, but suspicion may be aroused by a tracing in which the standard limb leads disclose QRS complexes and T waves of low amplitude, by a tracing affording evidence that auriculoventricular conduction is impaired or blocked or by a tracing disclosing auricular fibrillation. Finally, these
tracings usually possess peculiarities that conform to none of the specific patterns of abnormality commonly encountered in patients of advanced age.

**SUMARIO ESPAÑOL**

Los hallazgos electrocardiográficos fueron analizados en 15 pacientes que durante la autopsia se le encontraron depósitos de amiloide en el corazón. Aunque 12 de los 15 trazados se consideraron anormales, cambios que se pudieran considerar específicos de amiloidosis cardíaca no se han podido definir. Las alteraciones más comunes encontradas fueron, fibrilación auricular, complejos QRS y ondas T de poca amplitud en las derivaciones regulares de las extremidades y deterioro en la conducción auriculoventricular.

**REFERENCES**

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