Effects of Alcohol on the Electrocardiogram

By George Sereny, M.D., F.A.C.P., C.R.C.P. (C)

SUMMARY
The electrocardiograms of 1,000 chronic alcoholic patients were examined and analyzed. Evidence is presented that excessive consumption of alcohol, in the absence of organic heart disease, may produce changes in the electrocardiogram. The predominant abnormalities were sinus tachycardia and nonspecific T-wave changes. These abnormalities were present in the majority of the patients, provided that electrocardiograms were taken while the patients were still intoxicated. It is suggested that the changes are caused by two known effects of alcohol: stimulation of catecholamine secretion from the adrenal medulla and alteration of cell membrane permeability.

Additional Indexing Words:
Adrenal medulla Alcohol Electrocardiogram Electrolyte transport
Nonspecific T-wave changes

The association between excessive alcohol consumption and heart disease is well documented, and the various electrocardiographic abnormalities encountered in alcoholic patients have been recorded. Most of the observations1–7 relate the abnormal electrocardiograms to organic myocardial disease. Isolated reports, however, suggest that electrocardiographic changes may occur in chronic alcoholics without corresponding cardiomyopathy,8 but data are lacking in regard to the specificity of the ECG changes and their prevalence in the chronic alcoholic population.

The present study was intended to answer the following questions:
1. Does prolonged and excessive alcohol consumption produce electrocardiographic changes without cardiomyopathy?
2. Are these changes specific to alcohol?
3. What is the prevalence of the ECG changes in the alcoholic population?

Materials and Methods
One thousand male alcoholics, admitted consecutively to the Medical Unit of the Addiction Research Foundation from 1967 to 1970, were included in the study. All patients had a complete medical history and physical examination on admission and filled out a specially constructed questionnaire under the guidance of two nurses who were specifically assigned to this study.

Within 72 hr an electrocardiogram and a chest X-ray were taken in most patients, and extensive laboratory investigation was carried out. All ECG’s were read by the author. The laboratory investigation included urinalysis with microscopic examination, hemoglobin, W.B.C., differential count, sedimentation rate, BUN, fasting blood sugar, serum cholesterol, SGOT, and SCPT. Seventy-five of the patients had serum sodium, potassium, and chloride determinations. Magnesium levels were not measured. A thorough drug history was taken from each patient.

When the admission electrocardiogram was abnormal, one or more repeat electrocardiograms were taken at intervals of 5–7 days. In addition to the patient group, ECG’s were taken of 137 male nonalcoholic healthy volunteers. They were admitted to the Medical Unit for a special research project. The ECG’s were part of their medical work-up.

An abnormal electrocardiogram was considered to be probably caused by excessive intake of alcohol if the following criteria were met:
1. No history of previous coronary artery disease, rheumatic heart disease, hyper- or hypothyroidism, or congenital heart disease.

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EFFECTS OF ALCOHOL ON ECG

Table 1
Electrocardiographic Diagnoses in 1,000 Chronic Alcoholic Male Patients

<table>
<thead>
<tr>
<th>Description of ECG</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>820</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>86</td>
</tr>
<tr>
<td>Nonspecific T-wave changes</td>
<td>40</td>
</tr>
<tr>
<td>Extrasystoles, occasional and frequent</td>
<td>9</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>3</td>
</tr>
<tr>
<td>First-degree heart block</td>
<td>2</td>
</tr>
<tr>
<td>Coronary artery disease*</td>
<td>24</td>
</tr>
<tr>
<td>Right bundle-branch block</td>
<td>3</td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>6</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>1000</td>
</tr>
</tbody>
</table>

*All related diagnoses, such as old myocardial infarct and coronary insufficiency, are included in this category.

2. No symptoms or signs of coronary artery disease or congestive heart failure on admission.
3. No clinical evidence of avitaminosis.
4. Return of the electrocardiogram to normal within 7–10 days after admission.

In nine sobered-up, alcoholic patients it was possible to study the direct effect of ethanol on the electrocardiogram. These patients were free of cardiac, renal, or hepatic impairment. Ethanol was given intravenously in a dose of 1 ml/kg/hr over a 2-hr period. Electrocardiograms were taken before the infusion and immediately after its termination.

Results

The electrocardiographic findings in the 1,000 patients are listed in table 1. The abnormalities probably caused by excessive alcohol consumption fall into two main groups: sinus tachycardia and nonspecific T-wave changes. Cases with extrasystoles, atrial fibrillation, and right or left bundle-branch block were too few to allow a valid conclusion about the etiology. The frequency of their presence in this group is unlikely to be different from their frequency in the general population.

Sinus Tachycardia

Sinus tachycardia was diagnosed on the basis of a sinus rhythm together with a heart rate of 100 beats/min or more. Eighty-six patients fell into this group, including eight who also had T-wave changes.

Nonspecific T-Wave Changes

Forty patients had abnormal but nonspecific T-wave changes, including flattened, isoelectric, diphasic, or inverted forms. The degree of T-wave inversion also varied. There seems to be no definite pattern to the frequency of their appearance in one lead or another, and the difference between the T-wave changes of the individual patients was quantitative rather than qualitative. (fig. 1). Eight patients had a combination of sinus tachycardia and T-wave changes.

In addition, 10% of the patients showed tall “peaked” T waves identical with those described by Evans1, 2 and by Levine et al.9 in known or suspected alcoholics. However, 40 of the electrocardiograms recorded from 137 young healthy volunteers showed tall peaked T waves indistinguishable from those found in the chronic alcoholic population. None of the 137 showed T-wave changes of the other types mentioned.

Prevalence

Sinus tachycardia was observed in 8.6% and T-wave changes in 4% of the total patient population. The age distribution of patients with nonspecific T-wave changes (exclusive of the “peaked” T waves) is almost identical with the age distribution of the whole patient population (table 2). Thus, age does not appear to influence the frequency of occurrence of these changes. However, the percentage of abnormal electrocardiograms differs greatly according to the time elapsed between the consumption of the last drink and the taking of the ECG (table 3). Eighty-five patients had electrocardiograms taken on the same day as the last drink. Forty-two of these patients (49%) had sinus tachycardia and 18 (21%) showed T-wave changes. When tracings were taken 24, 48, and 72 hr after the last drink, the prevalence of abnormal records fell to 6.8%, 5%, and 5.9%, respectively (table 3). This would suggest that alcohol per se will induce electrocardiographic changes and that once all the alcohol is metabolized these changes subside in most cases.
Serum Potassium

A total of 75 patients who were intoxicated on admission had serum potassium determinations on admission. The results were classified as normal (4.5-5.5 mEq/liter) in 57 cases, low (3.4-3.9 mEq/liter) in 15, and very low (3.4-3.9 mEq/liter) in 15.

Figure 1

(A) Examples of the different T-wave changes from the admission electrocardiograms of three chronic alcoholic patients. All three patients were intoxicated on admission. (B) Five days later. All the T waves returned to normal.
(3.3 mEq/liter) in three. In the low and very low potassium groups 10 patients had normal electrocardiograms; three patients had sinus tachycardia, and three had T-wave changes. All three patients with T-wave changes fell into the low group, i.e., their potassium levels were between 3.4 and 3.9 mEq/liter. Two patients in the low potassium group had ECG diagnoses unrelated to the alcohol consumption. There were no abnormal T waves in the group with a potassium level below 3.3 mEq/liter (table 4).

**Chest X-ray**

In all, 883 patients had routine chest X-rays taken within 72 hr of their admission. Generalized cardiac enlargement was reported in five and left ventricular enlargement in two patients. Four of the five patients in the former group had normal electrocardiograms. The fifth patient had sinus tachycardia.

**Discussion**

Sinus tachycardia, atrial fibrillation, conduction defects, and T-wave changes have been described in beriberi heart disease and in alcoholic cardiomyopathy.1-7 Frederiksen8 reported that out of a group of 121 young male alcoholics 36 had sinus tachycardia and four had isoelectric or diphasic T waves. Evans3 found T-wave changes in 17 of 20 alcoholic patients. He described these abnormal T waves as dimpled (a shallow inversion of the T waves), cloven, blunted, and spinnous (narrow-based, peaked, tall T waves).

However, the T-wave changes shown by Evans1,2 as specific signs of alcoholic cardiomyopathy appear similar to those seen in sleep, fever, hypothyroidism, nutritional deficiencies, electrolyte disturbances, shock, and severe infections. Brigden and Robinson3 also found T-wave changes in a group of alcoholic patients, and felt that these were nonspecific. As noted in "Results," tall "peaked" T waves were found in a fairly high proportion of healthy nonalcoholic young subjects. These T waves therefore appear to be a normal variant.

The assumption that prolonged excessive alcohol intake can affect the electrocardiogram in the absence of cardiomyopathy has received scant consideration in the medical literature. Priest et al.8 reported T-wave changes in 20 of 37 chronic alcoholic

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**Table 2**

<table>
<thead>
<tr>
<th>Age group</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of admitted patients</td>
<td>40</td>
<td>190</td>
<td>330</td>
<td>320</td>
<td>120</td>
</tr>
<tr>
<td>No. of patients with T-wave changes</td>
<td>2</td>
<td>12</td>
<td>12</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Incidence of T-wave changes as % of each age group</td>
<td>5</td>
<td>6.3</td>
<td>3.6</td>
<td>3.5</td>
<td>3.3</td>
</tr>
</tbody>
</table>

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**Table 3**

<table>
<thead>
<tr>
<th>Time between last drink and ECG (days)</th>
<th>Total tested</th>
<th>No. of patients</th>
<th>% of patients with abnormal ECG’s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sinus tachycardia</td>
<td>T-wave changes</td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>85</td>
<td>42</td>
<td>18</td>
</tr>
<tr>
<td>1</td>
<td>488</td>
<td>25</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>141</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>135</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>849</td>
<td>75</td>
<td>33</td>
</tr>
</tbody>
</table>

*In 85% of the patients the electrocardiograms were taken within 4 days of their last drink.

**Table 4**

<table>
<thead>
<tr>
<th>ECG diagnosis</th>
<th>Potassium levels (mEq/liter)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥3.3</td>
</tr>
<tr>
<td>Normal</td>
<td>2</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>1</td>
</tr>
<tr>
<td>Nonspecific T-wave changes</td>
<td>—</td>
</tr>
<tr>
<td>Other</td>
<td>—</td>
</tr>
</tbody>
</table>
patients none of whom showed serious cardiac symptoms or evidence of heart failure. They suggested that ECG abnormalities may precede the development of myocardial impairment. In the present work, none of the patients with abnormal electrocardiograms had a history of organic heart disease, or symptoms or signs of congestive heart failure. Chest X-rays showed that all but one patient had a normal heart size.

Furthermore there is a clear difference in the frequency of abnormalities in electrocardiograms taken while the patients were still intoxicated and those taken after return to sobriety (table 3). This strongly suggests that these electrocardiographic abnormalities are to a significant extent connected to blood alcohol levels and not to myocardial damage.

The final proof that alcohol indeed does produce changes on the electrocardiogram can be seen in the electrocardiograms of three of nine patients who received alcohol infusions. Electrocardiograms of two of these patients are presented in figures 2 and 3. Both sinus tachycardia and T-wave alterations similar to those seen in the patient group of alcoholics with abnormal electrocardiograms were reproduced by alcohol infusion.

The mechanism by which alcohol per se produces electrocardiographic alterations can be only speculation. Although the data on serum potassium levels are not sufficient to

Figure 2

Chronic alcoholic male patient, age 37, without symptoms or signs of organic heart disease. (A) Diphase T waves in V5 and flat T waves in V6 24 hr after the last drink was consumed. (B) Three days later. All the T waves were normal. (C) Immediately after an alcohol infusion. T waves became inverted in V5 and V6. (D) Four days after the infusion. The T waves are returning toward normal.
permit a definite conclusion, it would appear that serum potassium levels do not play a significant role in the alteration of T waves by alcohol. However, it is known that alcohol interferes with the active transport of sodium and potassium across cell membranes.\textsuperscript{10, 11} The depolarization and repolarization process, recorded on the electrocardiograms by the QRS and the T waves, is dependent upon the movements of Na and K ions across the cell wall.\textsuperscript{12, 13} It is quite conceivable that alcohol, through its effect on electrolyte transport, could produce T-wave alterations.

The presence of sinus tachycardia in a large number of patients might be explained by the effect of alcohol on the adrenal medulla. Alcohol is reported to produce increased liberation of epinephrine.\textsuperscript{14, 15, 16} This effect depends on the route of administration and on the rate at which the blood alcohol concentration rises;\textsuperscript{17} this may explain why sinus tachycardia is not found in all intoxicated patients.

The present results indicate that the excessive consumption of alcohol, in the absence of underlying organic heart disease, may produce electrocardiographic abnormalities. These may at times imitate the changes produced by coronary artery disease, but the prognostic significance of the abnormal electrocardiogram would be quite different in the two conditions.

Acknowledgments

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