The Electrocardiogram of the South African Bantu

By J. D. Woods, M.B., and W. Laurie, M.D.

Considerable attention has been directed to the "suggested" low incidence of arteriosclerosis in the Bantu and its relation to diet and blood lipids. Electrocardiographic abnormalities found in the Bantu have been regarded as unreliable evidence either of the presence or the nature of heart disease in these subjects and hence not inconsistent with the alleged freedom of heart disease in these people. In the present study the authors challenge the validity of such an interpretation of electrocardiographic changes.

The electrocardiogram has become so well established in diagnosis that no heart examination can be considered complete without its use.¹ The only doubt as to the reliability of this method of diagnosis in the Bantu races has been cast by Grusin,² who described tracings, abnormal by accepted standards, in large numbers of sick and healthy individuals of this race. He considered that these changes mimicked those found in myocardial infarction, acute cor pulmonale, acute pericarditis, and left ventricular hypertrophy; these changes were found in 22 per cent of healthy nurses, in 63 per cent of consecutive noncardiac medical admissions, and in 63 per cent of consecutive cardiac admissions. He considered these changes to be functional in origin and suggested that they were due to a persistence of the juvenile pattern or to some effect of malnutrition. Whatever the cause of these abnormalities, their occurrence on such a large scale, if correct, must lead to distrust of the electrocardiogram as a diagnostic tool in this race and, in view of the current interest in the supposed freedom of the Bantu from coronary disease, would confuse the position even more.

We are unable to accept Grusin's findings and are satisfied that the electrocardiogram is a valuable diagnostic aid in Bantu heart disease. In support of this statement we produce in this paper evidence of its reliability, but before describing our results we wish to comment on Grusin's findings, which he classified into the 3 following patterns.

Pattern 1. Deeply inverted T waves, usually, but not always, confined to the right precordial leads with depression of J and an upwardly bowed S-T segment, the T wave ending in a hump before settling to the isoelectric line. This pattern occurred in 14 per cent of healthy nurses, 34.1 per cent of cardiac cases, and 53.9 per cent of noncardiac medical admissions.

Pattern 2. Elevation of the S-T segment and a tall bold T wave with a slowly rising ascending limb and a sharply falling distal limb ending in a U wave. These features were almost invariably confined to the left precordial leads. This pattern occurred in 4 per cent of healthy nurses, 29.3 per cent of cardiac patients, and 25.2 per cent of noncardiac patients.

Pattern 3. This covered the wide range from the loss of the normal concavity of the S-T segment with rounding of the peak of the T wave to complete flattening of this wave. No details were given as to the leads in which these changes were found. This pattern occurred in 4 per cent of healthy nurses, none of cardiac cases, and 4 per cent of noncardiac patients.

These patterns thus embrace most changes in the precordial end deflection, ranging from tall, upright T waves with elevated S-T segments, through flattening of the T wave, to inverted T waves with depressed S-T segments, or raised S-T segments. This last observation was not commented upon by Grusin but is shown in his illustrations.

Of the 250 subjects studied by Grusin, only 50 were in good health; the remainder con-
sisted of 159 consecutive noncardiac medical admissions and 41 consecutive cardiac admissions. Abnormal electrocardiograms were almost 3 times more commonly seen in both groups of patients than in the healthy controls. It seems unwise to assume that these changes in the T wave and S-T segments can be ascribed to functional causes when so many other important factors may have been present in the patients. It would not appear unreasonable that a considerable proportion of consecutive medical admissions would be complicated by factors such as electrolyte imbalance, infections, or metabolic disorders, all of which may cause alterations in the ST-T complex.\(^4\) This would indeed appear to be the case as, of the 11 examples illustrated, no fewer than 9 patients were suffering from infections that included typhoid fever, pneumonia, acute bronchitis, and pleural effusion. In some of these, myocarditis would seem difficult to exclude.

In the group of 41 cardiac patients, it appears impossible to accept that electrocardiographic patterns resembling those found in heart disease can be ascribed to functional rather than organic causes in the absence of autopsy proof or details of the nature of the cardiac lesions known to be present.

It would seem that only in the control group could these changes properly be ascribed to functional causes, but here lack of full information on the extent of the T-wave inversions leaves doubt as to the significance of these changes. There is clearly a great difference in importance between T-wave inversion in the right precordial leads, and inversion of the T in the left precordial leads. In the case of the former, inversion of the T as far across as V4 may be normal when accompanied by an rS, or Rs, pattern.\(^4\) Such a persistence of the juvenile pattern is found in other races, such as the American Negro,\(^5\) and in the Puerto Ricans,\(^6\) and is accepted not to be organic.

Pattern 2 is admittedly similar to that found in the early stages of acute pericarditis, but in the control group serial studies did not show the subsequent changes found in this disease, and differentiation could thus have been made without great difficulty.

Pattern 3 covers such a wide range of T-wave changes that the finding of only 2 instances of this variegated pattern in the control group appears scarcely to justify its inclusion as a special pattern.

As part of our larger study of heart disease in the Bantu it was necessary to clarify the value of the electrocardiogram in these people, and an investigation was carried out of the tracings in health and heart disease.

### METHODS AND MATERIAL

**Control Group.** Fifty healthy female nurses between 21 and 38 years of age, and 50 healthy male nurses and orderlies, of ages between 19 and 36, were used as controls. In addition to physical, radiologic, and electrocardiographic examinations various blood investigations were carried out. These included estimation of the serum electrolytes, liver function tests, complete blood counts, and the Wassermann reaction.

**Cardiac Group.** The electrocardiograms of 100 cardiac patients were studied. No cases were included in which the clinical diagnosis appeared to be doubtful and in 18 instances postmortem or operative proof was obtained. Cases with the possibility of more than 1 etiologic factor were excluded, e.g., hypertension complicating pulmonary heart disease, and in view of the possibility of complicating ischemia from involvement of the coronary arteries or their ostia by atherosclerosis or syphilis, no case giving a history of exertional precordial pain was included. With 4 exceptions added to bring the numbers up to 100 cases, all
ELECTROCARDIOGRAM OF THE SOUTH AFRICAN BANTU

Table 2.—Diseases Causing Hypertrophy of One or More Chambers of the Heart

<table>
<thead>
<tr>
<th>Electrocardiographic changes</th>
<th>Rheumatic</th>
<th>Syphilitic</th>
<th>Pulmonary</th>
<th>Hypertensive</th>
<th>Congenital</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.S.</td>
<td>M.I.</td>
<td>A.I. or M.I.</td>
<td>A.I.</td>
<td>A.I.</td>
<td>M.S.</td>
</tr>
<tr>
<td>Normal</td>
<td>4</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>p &gt; 0.11 sec.</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p &gt; 2.5 mm. tall</td>
<td>3</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right ventricular hypertrophy</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Delayed right ventricular conduction</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right bundle-branch block</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>4</td>
<td>3</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incomplete left ventricular hypertrophy</td>
<td>1</td>
<td>1</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flat TS in left precordial leads</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High voltage</td>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diminishing R across chest</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grusin’s pattern 2</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Totals</td>
<td>16</td>
<td>1</td>
<td>12</td>
<td>2</td>
<td>9</td>
</tr>
</tbody>
</table>

M.S. = Mitral stenosis. M.I. = Mitral incompetence. A.I. = Aortic incompetence.

Results

Control Group

On the whole, the tracings were normal by accepted standards; the 5 per cent showing deviations are discussed below. Excepting sinus arrhythmia no abnormalities of rhythm were found. The duration of the QRS tended to be short, ranging from 0.04 to 0.08 second with an average in both sexes of 0.06 second. The Q-T interval averaged in males 39.7 seconds and in females 38.8 seconds, figures not unlike those obtained in other races.

Abnormalities in Control Group. Partial heart block was seen in 1 male, the P-R interval being 0.28 second. It seems probable that some undiagnosed pathology was present, but he left our service before further investigations could be carried out.

The precordial T wave was inverted as far across as V3 in 1 female. In an additional female and in 1 male, the T wave was inverted in leads V1 and V2. An rS or RS pattern accompanied all these T-wave inversions. The electrocardiograms were repeated at a later date and leads were taken in addition to the V leads. It was found that all the leads showed upright T waves with the exception of 1 in the female who showed inversion of the T wave as far across as V3.

Grusin’s pattern 2 was seen in 2 males in leads V4 and V5 only; the pattern persisted unchanged for more than 6 months.

Cardiac Group

The causes of the heart diseases under study have been given in table 1. Table 2 shows the electrocardiographic changes found in diseases where the lesions caused hypertrophy of 1 or more chambers of the heart, while table 3 gives the findings in cases where the heart muscle was damaged either by ischemia or by infarction.

Changes due to Hypertrophy. With the few exceptions mentioned below, the findings...
in table 2 resemble closely those that would be expected in similar circumstances in any other race.

1. Right ventricular hypertrophy associated with aortic incompetence. This may have been due to failure to detect an additional mitral stenosis, as this tracing was obtained from a female aged 36 years in whom a rheumatic etiology was suspected.

2. Diminishing R waves across the chest. This occurred in the following: (a) A man aged 26 who suffered repeated attacks of congestive failure following mitral valvotomy. Embolism of a coronary artery seemed the likely cause (fig. 1). (b) A woman aged 27 who had sustained a syphilitic cerebral thrombosis, and later developed aortic incompetence. (c) A woman aged 20 with a huge heart caused by mitral incompetence. Microscopy showed only hypertrophy of the muscle fibers. While the r wave failed to increase, no Q wave was present.

3. Grusin’s pattern 2 occurred in 1 male suffering from mitral stenosis. Serial changes revealed no change suggestive of pericarditis.

Changes due to Myocardial Infarct. Pericarditis. Pericardial effusion, thought to be tuberculous, was found in 7 cases. Occlusive pericarditis, proved to be tuberculous, was present in the other 3 cases.

Myocardial Infarction. The youngest patient was aged 22 years. He had an anterior infarct near the interventricular septum, and the electrocardiogram showed left bundle-branch block without an abnormal Q wave.

Diminishing R wave across the chest. This feature was found in a man aged 44 who was admitted in acute cardiac failure with no history of precordial pain. During a very stormy illness lasting 2 months electrocardiogram showed transient elevations of the S-T segment and persistent diminution of the R wave across the precordium as far as between V4 and V6, where a small localized Q wave was found. V5 showed an RS pattern, the qR pattern not starting until V6. He recovered but was readmitted 4 months later and died before a history or an electrocardiogram could be obtained. Autopsy revealed an old postero-lateral infarct affecting the epicardial two thirds of the muscle and several areas of fibrosis about the size of a matchhead in the anterior wall of the left ventricle. At no time did lead aVF show changes suggestive of infarction (fig. 2).

The other 2 infarcts occurred in men aged 31 and 44 years respectively. In one case a large infarct near the apex was associated with a left bundle-branch block; in the other a large anterior infarct of the left ventricle was associated with classical electrocardiographic changes.

DISCUSSION

From our study of the electrocardiogram of the healthy, normal Bantu it appears that, with few exceptions, the patterns do not differ to any significant extent from those in other races. We agree with Grusin that in health the patterns he described are probably due to functional causes and are of no significance.

In our admittedly small series of 100 cases of cardiac disease few differences from the disease pattern in other races were found, and these, it would seem, could be accounted for by misdiagnosis rather than by any racial trait, the error lying more likely with the observer than with the electrocardiogram.

Certain observations may be made on the low incidence of Grusin’s patterns among the group of cardiac cases. It is not uncommon to find, among the Bantu, cases of "failed hyper-

<table>
<thead>
<tr>
<th>Pericarditis</th>
<th>Myocardial infarct</th>
<th>Total abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Low voltage</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Depression of S-T segments</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Elevation of S-T segments</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Inverted precordial T waves</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Flat precordial T waves</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Biphasic precordial T waves</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal Q waves</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Diminishing R across precordium</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total cases</td>
<td>10</td>
<td>4</td>
</tr>
</tbody>
</table>
tension," i.e., cardiac patients with normal blood pressures in whom previous histories reveal that they had previously been treated for hypertensive cardiac failure. Also, in the postmortem room we commonly come upon normotensive cases of cardiac failure in whom there is evidence of left ventricular hypertrophy associated with renal changes compatible with the presence of hypertension. In the series of 31 cases of hypertensive cardiac failure presented in this paper, 4 cases, now normotensive, were found from previous records to have had hypertension in the past. In 3 of these the electrocardiogram showed left ventricular hypertrophy. This phenomenon is known to occur in other races, but frequently the diastolic pressure remains above the limits of normal. In the Bantu the fall is often greater and to well within the accepted limits of normal; in the absence of previous records diagnosis becomes difficult. It is conceivable that this factor may have influenced some of Grusin’s tracings.

**Conclusions**

It would appear that there is no reason for the assumption that the electrocardiogram of the South African Bantu differs in any important respect from that obtained in other races. It would follow that when patterns suggesting specific types of heart disease are found, they must be given the same consideration as would be given were the changes found in individuals of other races.
Summary
The electrocardiograms of 100 healthy Bantu adults of both sexes and the electrocardiograms of 100 Bantu suffering from heart disease have been studied. It is concluded that neither in health nor in disease do the electrocardiograms differ significantly from those obtained in similar circumstances from other racial groups.

Acknowledgment
We wish to thank the Director of Hospital Services for Natal, Dr. J. L. Parker, and the Superintendent of Edendale Hospital, Dr. T. M. Adams, for permission to publish this paper. We also wish to thank the Matron and Nursing Staff of this hospital for their cooperation in providing the control group of subjects.

Summario in Interlingua
Esseva studiate le electrocardiogrammas de 100 normal bantu adulte de ambe sexos e de 100 bantu con morbos cardiac. Le conclu-
sion es que le electrocardiogramma del bantu —tanto in stato de bon sanitate como etiam

sub conditiones patholie—non differe significativamente ab le electrocardiogramma de altere gruppos racial sub simile circumstantias.

REFERENCES


Two patients were observed to develop central scotomata while receiving digitalis. The first patient, a 67-year-old woman, had received 6 tablets daily of digitalis leaf for 1 week when visual deterioration to 6/60 bilaterally and some nausea were noted. Moderate improvement in vision occurred after stopping the daily maintenance dose of 1 digitalis tablet 5 months later. The second patient, a 60-year-old man, developed visual weakness to 6/9 (O.D.), 6/12 (O.S.) with fluttering colored hallucinations, vomiting, and bigeminy after taking Digitalan for 3 or 4 years in unknown quantities. Considerable improvement in these symptoms occurred on stopping the drug and administering potassium iodide plus vitamin B. The various toxic effects of digitalis therapy are described.

Rogers
The Electrocardiogram of the South African Bantu
J. D. WOODS and W. LAURIE

Circulation. 1959;19:251-256
doi: 10.1161/01.CIR.19.2.251
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/19/2/251

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/