Peculiarities of the African’s Electrocardiogram and the Changes Observed in Serial Studies

By H. Grusin, M.B., B.Ch., M.R.C.P. (Lond.)

Among African medical patients with and without heart disease about 63 per cent are found to have electrocardiograms which are abnormal by accepted standards. Twenty-two per cent of a group of healthy African nurses show the same peculiarities. Over a period of months or years the electrocardiograms show spontaneous changes. Clinical and postmortem evidence indicates that the electrocardiographic changes are not related to organic heart disease. It is suggested that these electrocardiograms may be of a juvenile type or alternatively that they may be attributable to some obscure metabolic factor of nutritional origin.

The Electrocardiogram of occasional healthy American Negroes is known to show inversion of T waves in precordial leads,1 and similar findings have been reported in the South African Bantu (African).2 In this study evidence is presented that the electrocardiogram of the African differs in two main respects from that of a white man. First, patients without heart disease show peculiarities not only of the T wave but also of the S-T segment and P wave, and second, a series of striking changes takes place in the electrocardiogram for which no reason is apparent.

Material and Methods

The patients on whom these studies were made were urbanised Africans accustomed to living under squalid conditions and subsisting on a badly balanced diet. Fifty healthy young female African nurses who were adequately housed and fed served as a control group. Electrocardiograms were recorded from the nurses and from patients with and without heart disease. A random sample of subjects showing these peculiarities were selected from each group for further study. Tracings were recorded daily for the first two weeks and thereafter at weekly intervals for 1 to 12 months, and the effects of drugs and certain maneuvers on the electrocardiogram were also studied.

Results

At least 63 per cent of 159 consecutive medical patients admitted to the wards and 22 per cent of healthy nurses showed electrocardiograms which deviated significantly from the accepted normal (table 1). For purposes of description these have been classified into three patterns on the basis of S-T deviation in precordial leads.

Pattern 1. S-T Depression and Inverted T Waves in Precordial Leads (fig. 1A)

Incidence. These electrocardiographic features were found in 33.9 per cent of 159 medical patients, in 34.1 per cent of patients with heart disease and in 14 per cent of healthy nurses (table 1). They occurred in adults of all ages and both sexes. Twenty one patients showing this pattern were selected for follow-up studies and of these only seven had heart disease.

Precordial Leads (fig. 1A). In a typical right ventricular surface lead (V2) the end deflection consisted of a deeply inverted T wave with depression of J and an S-T segment bowed upwards to form a wide shoulder; the whole deflection was asymmetrical with a longer distal limb which ended in a rounded hump before settling on the isoelectric line.

In 17 patients these features were most marked in right ventricular surface leads, in three they were seen in all precordial leads and in one they were confined to left ventricular surface leads (fig. 2A, C, D).

Standard and Augmented Unipolar Extremity Leads (fig. 2A, C, D). When all precordial leads or only left ventricular surface
leads showed S-T depression and T wave inversion, unipolar extremity leads and standard leads reflected these features. In leads facing the cavity or the back of the heart the T wave was always upright and the S-T segment either isopotential or elevated (4 of 21 cases). When only right ventricular surface leads were affected unipolar extremity leads and standard leads were hardly abnormal (17 of 21 cases).

Serial Changes (figs. 1A to E, 3A to F). Within one to three weeks the end deflection began to change. The inversion became shallower and the S-T segment, taking off higher, was now boldly curved above the isoelectric line giving the T wave a cove-plane appearance. Days or weeks later more and more of the S-T segment appeared above the isoelectric line and the inversion became still shallower. From this stage the evolution of the T wave followed one of two courses. In 17 patients it never became upright during the period of observation but remained inverted and continued to change in shape, being deeper, shallower or almost upright on different occasions (fig. 3); in one patient it was still showing changes a year after his admission to hospital (fig. 2A, B). In four patients the T wave became upright in periods ranging from 14 to 49 days; when right ventricular surface leads were involved the inversion disappeared from V3 first and V1 last, and when both ventricular surface leads were affected the inversions disappeared from both at the same time or independently. In two of the four patients in whom the T wave had become upright it was found to be inverted again within a month, and similar cycles of T-wave inversion and recovery were found in the past records of patients for periods as long as three years.

The inverted T waves were not always as gross as those illustrated in the pictures. The first electrocardiogram recorded from these 21 patients revealed a deeply inverted T wave in only nine and a "cove-plane" or shallow inverted T in the remaining 12. Although the serial changes described above apply to both types, there was one obvious difference in be-

<table>
<thead>
<tr>
<th>Table 1.—Comparative Incidence of Patterns in Three Groups of Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Random group of patients; consecutive medical admissions</td>
</tr>
<tr>
<td>Patients with heart disease; consecutive admissions; undigitalised</td>
</tr>
<tr>
<td>Healthy nurses</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>159</td>
</tr>
<tr>
<td>41</td>
</tr>
<tr>
<td>50</td>
</tr>
</tbody>
</table>

Fig. 1. Male, aged 30, with lobar pneumonia. No heart disease. A to E made at 10-day intervals. Pattern 1 more or less confined to right ventricular surface leads.
Fig. 2. (A) Male aged 14, with petit mal. No heart disease. (B) One year later. The electrocardiogram had never been normal during this time. Pattern 1 confined to right ventricular surface leads; standard and limb leads normal. (C) Male, aged 24, with typhoid fever. No heart disease. Both ventricular surface leads show inverted T waves; standard and limb leads abnormal. The S-T segment is elevated in aV₁. (D) Male, aged 30, with pleural effusion. No heart disease. Pattern 1 confined to left ventricular surface leads; standard leads and limb leads abnormal. (E) Two weeks later, behavior between deeply inverted T waves and shallow ones. The deeply inverted T wave changed gradually and reached the cove-plane stage in one to three weeks. From this point it changed more rapidly and, once most of the S-T segment lay above the isoelectric line, the deflection became labile and capricious; nervousness on the part of the patient, a sudden noise or the sight of an hypodermic syringe might make it deeper or shallower. In continuous tracings it showed mild changes from hour to hour and in daily records it might appear deeper on one day, shallower or almost upright on the next.

Other Studies. Exercise, deep inspiration or expiration, change in the patients' posture, the inhalation of amyl nitrite and intravenous injection of atropine 0.9 mg. had little or no effect on deeply inverted T waves. When the initial inversion of the T wave was shallow,
male, aged 27, with pellagra. No heart disease. A to F made over a period of 3½ months. P waves are bifid in leads III, aVf, V4-R.

However, the same procedures, and particularly intravenous atropine, changed the form of T which became less inverted or completely upright. Neither deep nor shallow inverted T waves were affected by carotid sinus pressure.

**Pattern 2. S-T Elevation and Tall T Waves in Precordial Leads (fig. 4)**

**Incidence.** These electrocardiographic features were found in 25.2 per cent of 159 medical patients, in 29.3 per cent of patients with heart disease and in two nurses (4 per cent) (table 1). Eighteen patients showing this pattern were selected for follow up and of these only two had heart disease.

**Precordial Leads.** In a typical left ventricular surface lead (V4) the terminal deflection showed elevation of the S-T segment and a tall bold T wave with a slowly rising ascending limb and a sharply falling distal limb end-

**Fig. 3.** Male, aged 27, with pellagra. No heart disease. A to F made over a period of 3½ months. P waves are bifid in leads III, aVf, V4-R.

**Fig. 4.** Male, aged 30, with lobar pneumonia. No heart disease. A to C obtained at intervals of 2 weeks. S-T elevation in left ventricular surface leads, in aVf and standard leads II and III; lead aVr shows S-T depression and deep inversion of T.
back of the heart showed striking S-T depression and deeply inverted T waves.

The QRS Complex. Some subjects showing this pattern had unusually tall R waves in left ventricular surface leads (fig. 5). Excluding patients with heart disease, the tallest R wave measured 35 to 47 mm. in three patients, 25 to 30 mm. in five patients, and ranged between 15 and 25 mm. in the rest. The height of the R waves was not related to the degree of S-T elevation nor to the height of the T wave and the QRS complexes were usually less than 0.05 second in width.

Serial Changes. The end deflection changed in every case, but sometimes so slowly that a difference was only appreciable after weeks or months. In five patients the electrocardiogram came to resemble the accepted normal in 12 to 60 days, but in the remaining 13 patients the S-T and T deviations were still present 6 to 12 months later. During this period the electrocardiogram showed many changes. The degree of S-T elevation was always varying and the shape of the T wave was always changing, at times being rounded and at others reverting to its bold, peaked form; in three patients it became flat or mildly inverted while the S-T segment was still elevated. Factors tending to raise the heart rate, such as anxiety on the part of the patient, usually produced a slight drop in the level of the S-T elevation and in the height of the T wave.

Two patients with normal blood pressures and hearts of normal size showed R waves measuring 45 mm. in V₆ on numerous occasions during a period of a year. At other times the R waves appeared to have lost 5 to 10 mm. in height (fig. 5), but over such a long period of time it was impossible to be sure that this reduction in height was not due to some factor causing a change in the position of the heart relative to the electrode.

The pattern did not always show features as marked as the examples illustrated. All gradations were seen to graphs which would have been accepted as normal if previous or subsequent records had not been available. The elevation of J in left ventricular surface leads ranged from 1 to 4 mm. and the height of the T wave from 7 to 17 mm.; lesser degrees of

---

*Fig. 5. Male, aged 23, with acute bronchitis. No heart disease. A obtained on admission and B six weeks later. S-T elevation and tall T waves in both right and left ventricular surface leads; the R wave in V₆ has apparently become shorter.
S-T elevation were difficult to appreciate in right ventricular surface leads and could only be diagnosed in retrospect as the electrocardiogram changed.

Other Studies (fig. 6). The inhalation of amyl nitrite usually reduced the degree of S-T elevation and the height of the T wave which sometimes became inverted; within two or three minutes, as the heart rate slowed, the end deflection usually reverted to its original form. When amyl nitrite caused an appreciable drop in the height of T, the R wave also became shorter.

Similar effects on the end deflection and the R wave were observed after exercise (running) and after a deep inspiration but not with postural changes or carotid sinus pressure.

Pattern 3. Rounded or “Flat” T Waves in Precordial Leads (fig. 7)

The two patterns already described were striking and easily detected but in some patients without heart disease the electrocardiogram showed more subtle changes. In these the precordial T wave though upright had lost its contours, the concavity of the upstroke was ironed out and the apex rounded; all gradations were seen from such rounded T waves to completely “flat” ones. The tracings showed changes over the weeks; in some, the T wave gained height and recovered its lines and in others the pattern of T inversion or S-T elevation supervened. Pattern 3 was recognizable in 4.4 per cent of the random group of medical patients but the incidence of lesser degrees of change could not be accurately assessed.

Combined Patterns

In a few patients patterns 1 and 2 were seen together in the same electrocardiograms, leads V₁ to V₄ showing S-T depression and in-
verted T waves and leads V5 and V6 S-T elevation and tall T waves. In the majority of patients with pattern 1, however, S-T elevation, if present at all, was only found in leads facing the cavity or the back of the heart and not at “free” ventricular surfaces anteriorly, laterally or posteriorly.

**Other Data**

The three patterns were seen at rapid and slow heart rates. Sinus rhythm was the rule except in four patients who showed nodal rhythm. The P-R interval lay within normal limits. The electrical position of the heart was horizontal in 15 patients, vertical in 23 and “indeterminate” in one. The corrected Q-T interval (Q-Tc)3 in 10 patients ranged between 0.34 and 0.43 second but the length of the interval did not vary significantly with changes in the end deflection. The QRS complex showed no abnormality in patterns 1 and 3.

The P wave showed changes. In 10 patients it lost its sharp outline and became rounded, indistinct or split (fig. 3). In some patients these changes were seen in all leads but in others they were confined to standard or precordial leads.

**Relation between the Electrocardiogram and the Clinical State**

All the nurses and the majority of patients showing these patterns had no evidence of heart disease by clinical or radiologic standards nor were the patterns encountered more frequently in cardiac patients than in the random group of medical admissions. (See table 1.) Usually there was no association between the nature and severity of the patients’ medical illness and his electrocardiographic changes. Amongst the nurses the patterns were found less frequently and the deviations were not as gross as those seen amongst the medical patients.

**Postmortem and Other Evidence**

Postmortem evidence was obtained from two patients who had shown one of the two main patterns in their electrocardiogram during life. One had died of pellagra and the other of a spinal tumor. Detailed macroscopic and microscopic section revealed no abnormality of the heart, coronary vessels or lungs. In several other patients showing one or other pattern in their electrocardiogram during life, routine postmortem examination failed to reveal a cardiac lesion.

In two nurses and 10 patients the blood potassium and calcium levels and the carbon dioxide combining power were estimated and repeated at intervals during the evolution of the electrocardiographic changes. The results were always within normal limits.

**Discussion**

There is a marked resemblance between the end deflections described here and those seen in various types of organic heart disease. The appearances of the S-T segment and T wave mimic those of myocardial infarction, left ventricular hypertrophy, acute cor pulmonale and acute pericarditis, yet in these African subjects no organic damage has been found in the heart at postmortem examination to account for these features. It must be assumed, therefore, that they are of a functional nature similar to those which have been produced experimentally by physical and chemical agents.4

Electrophysical considerations aside, the nature of the lesion remains to be explained. Littmann,4 who described similar precordial T-wave inversions in healthy American Negroes, attributed them to the infantile bodily habitus of his subjects. Brink2 found inversion of precordial T waves from V1 to V5 in 5 per cent of healthy Africans and considered that they were due to vagotonia.

In this study no correlation is found between the electrocardiographic patterns and the electrical position of the heart or the bodily configuration of the patient. When the T wave is at a labile stage the changes which occur spontaneously and those which follow immediately on an injection are probably due to nervous factors. On the other hand there are several features of these patterns which cannot be explained on a neurogenic basis, namely, that the deviations are gross and may
remain unchanged for weeks, that they occur at rapid and slow heart rates and are unaffected by carotid sinus pressure.

The S-T segment and T-wave changes of these patterns though they sometimes disappear often linger in the electrocardiogram for months or years. With what other known electrocardiographic patterns can they be compared? Those changes caused by drugs or associated with disordered metabolic states are transient and disappear when the operative factor is removed or corrected. Possibly the closest parallel to these patterns is to be found in the precordial T-wave inversions of children which exist for years and are found to have disappeared in adult life. Various explanations have been advanced to account for these inverted T waves. On the one hand they have been ascribed to physical factors such as the electrical position of the heart in childhood and on the other to chemical differences between the two ventricles. It is tempting to speculate that the electrocardiogram of the African may be of a juvenile type in the developmental sense and it would be interesting to know whether the T waves of children at the time when they become upright show the same lability and responses as do those described here.

Finally in assessing any departure from the accepted normal in Africans the problem of undernutrition is an ever present bogey. Even when apparently healthy, many of them suffer from occult liver disease, and it is possible that some metabolic factor produces these electrocardiographic changes by acting locally on the heart or by causing a change in the patient’s internal environment.

**Summary**

1. At least 63 per cent of 150 African patients admitted to medical wards and 22 per cent of apparently healthy African nurses showed electrocardiograms which deviated from the accepted normal.

2. Spontaneous fluctuations occurred in these electrocardiograms over a period of 1 to 12 months.

3. The changes observed have been arbitrarily classified into three patterns.

4. The electrocardiographic peculiarities were apparently not due to organic heart disease.

5. The etiology of these changes is unknown and theories as to their causation are discussed.

**Acknowledgments**

I wish to express my thanks to Drs. B. van Lingen, W. H. Craib and J. C. Gilroy for their advice in the preparation of the manuscript, to Dr. P. S. Kincaid-Smith for mounting the electrocardiograms and for much help in other ways, to Dr. J. Haggison for examining the sections of the hearts, and to Protea Distributors Ltd., Johannesburg, for the loan of a Cambridge Cardiette.

**Sumario Español**

1. Por lo menos 63 por ciento de 150 pacientes africanos admitidos a los pabellones médicos y 22 por ciento de enfermeras aparentemente saludables mostraron electrocardiogramas con desviación del normal aceptado.

2. Fluctuaciones espontáneas ocurrieron en estos electrocardiogramas sobre un periodo de 1 a 12 meses.

3. Los cambios observados han sido arbitrariamente clasificados en 3 patrones.

4. Las peculiaridades electrocardiográficas no fueron aparentemente debidas a cambios orgánicos en el corazón.

5. La etiología de estos cambios se desconoce y las teorías sobre la causa se discuten.

**REFERENCES**


Peculiarities of the African's Electrocardiogram and the Changes Observed in Serial Studies

H. GRUSIN

Circulation. 1954;9:860-867
doi: 10.1161/01.CIR.9.6.860

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1954 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/9/6/860