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The physiological basis of ST-T variations in the electrocardiogram: A review

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Summary

ST-T aberrations in the Negro involve both sexes, all age groups and all electrocardiogram (ECG) leads and are benign in the asymptomatic subject. This suggests that the variations have physiological basis and that alterations in the electrochemical processes occuring during repolarization would explain the ECG counterparts. While an isoelectric J-point suggests a temporal dissociation between phases 0 and 1 of the action potential, an elevated J-point suggests that phase 1 starts before the end of phase 0. If the current changes in phases 2 and 3 are the same, the ST-segment and T wave would form a continuum. It is suggested that in the Negro, the force producing repolarization overrides that producing slow depolarization during phases 2 and 3 and that the net result of the conflicting electrochemical processes accounts for the ST-T aberrations in the Negro race.

The physiological basis of ST-T variations in the human heart

It is well established that ventricular repolarization (VR) takes diverse forms in the adult Negro[1-2] and that these are benign in the asymptomatic subject[1,3-5]. Nevertheless, the mechanism of ST-T aberations remains unclear. Various explanations have been offered to account for ST-T variations: Littman[6] termed it the "normal or functional juvenile pattern" because of similar occurrence in normal infants and the fact that adult Negroes retain infantile bodily habitus. Wasserburger and Alt[5] called it the "Labile T wave" because latent forms not on the resting ECG can be unmasked in the emotionally labile subject. Grusin[2] attributed the ST-T variants to metabolic factors of nutritional or liver origin but were refuted by Powell[7]. Grusin also proposed a system of classification but as pointed out by Araoye[1] and Somers and Rankin[8], Grusin's classification is untenable because the three patterns he described were basically the same. Moreover, Grusin worked on a small sample of young males and his findings could not be held representative of the adult Negro population.

In a detailed study of healthy adult Nigerians, Araoye[1] proposed a system of classification based on the morphology of the J- point, ST-segment, ST-T junction ("K-junction") and the associated T wave. He also presented the age, sex and lead distribution of the variants from which a number of observations emerged that could explain the genesis of ST-T variations in the human heart:-

1. ST-T variations involved all age groups, both sexes and all ECG leads.

2. They occur with such regularity, and in such a predictable pattern as to make it possible to propose four types in the normal adult Negro. These are:-
   Type I: with isoelectric J-point and K-junction (Fig. 1);
   Type II: with ST-T fusion with or without J-point elevation (Fig. 2);
   Type III: or pseudo-infarction pattern (Fig. 3); and
   Type IV: or the non-specific ST-T changes (Fig. 4).

3. Type I and those with isoelectric J-point occur in the standard and left praecordial leads. They are commoner in the elderly than the young, and in females than males.

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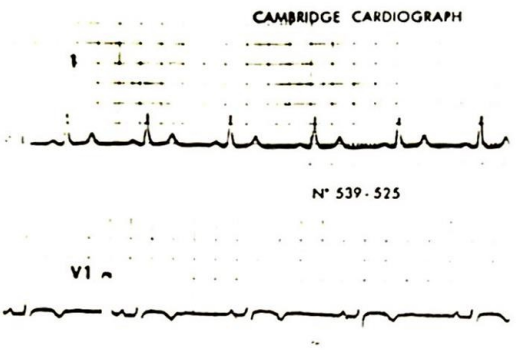

Fig. 1: Type I ST-T morphology

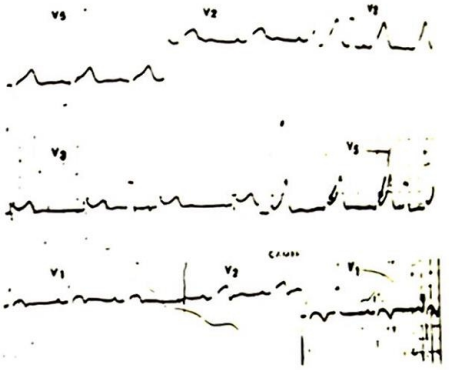

Fig. 3: Type III ST-T morphology

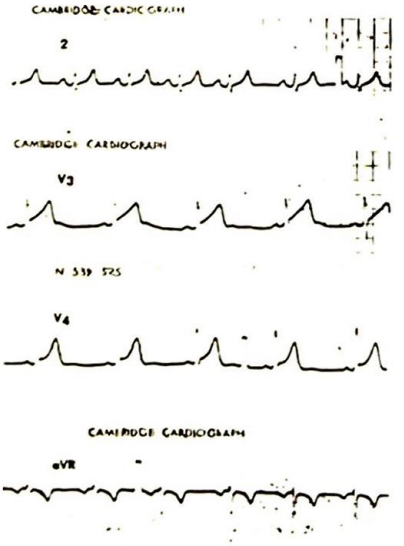

Fig. 2: Type II ST-T morphology

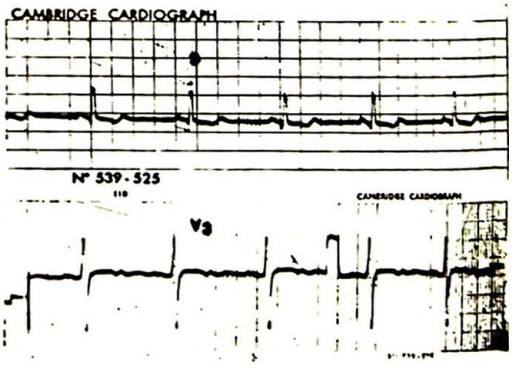

Fig. 4: Type IV ST-T morphology

3. Type I and those with isoelectric J-point occur in the standard and left praecordial leads. They are commoner in the elderly than the young, and in females than males.
4. The variants with J-point elevation involve the praecordial leads but are rare in the limb leads of males and rarer still in those of females.
5. The subtypes reflecting T wave inversion are distributed predominantly in aVR and V1.
6. The indeterminate (non-specific) ST-T variants involve commonly leads aVL, III and V1 but are rare in other leads.

These observations suggest that the phenomenon of ST-T variations is physiological and could be explained in terms of vector distribution during the course of ventricular repolarization (VR).

The work of van Dam and Durrer[9], Noble[10], Trautwein[11] and Katz[12] on cardiac action potential (CAP) made it possible to relate cellular physiology with the surface ECG counterpart. The CAP phase 0 corresponds to the QRS complex; Phase 1 corresponds to the J-point; phase 2 to the ST-segment and phase 3 to the T wave. Accordingly, variations in the magnitude, direction and temporal dispersion of the vectors of phases 1, 2 and 3 would reflect in the J-point, ST-segment and T wave respectively. An isoelectric J-point suggests that phase 0 ends before phase 1 begins. An elevated J-point, otherwise termed "early repolarization" or "accelerated repolarization" implies that phase 1 starts before the end of phase 0. An isolectric tracing suggests that the net rate of current propagation is steady. Accordingly, if the current flow in phase 2 is steady and that of phase 3 is rapid — crescendo and decrescendo, the surface ECG would show an isoelectric ST-segment, a distinct K-junction and T wave — a picture compatible with Type I ST-T morphology (Fig. 5).

However, if the rate of VR in phases 2 and 3 are the same or uniform, the ST-segment and T wave would fuse without leaving a K-junction as is found in Type II ST-T morphology. (Fig. 6). If phase 0 is prolonged, or trails into phase 1, or the latter starts before the end of phase 0, and accelerates into phases 2 and 3 the surface ECG would show an indistinct J-point and K- junction — features of Type III ST-T morphology. (Fig. 7).

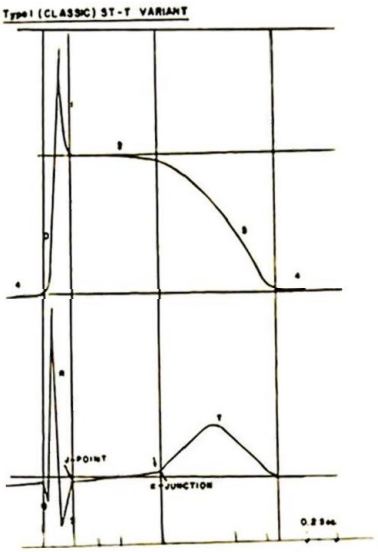

Fig. 5: Type I ST-T variant
Hypothetical Cardiac Action Potential (Top panel) with well defined phases 1, 2 and 3. The corresponding surface ECG (Bottom panel) shows definite J-point, ST-segment, K-junction and T wave.

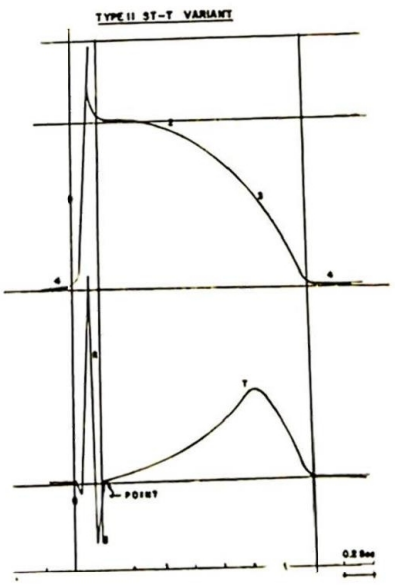

Fig. 6: Type II ST-T variant
Hypothetical Cardiac Action Potential (Top panel) in which phases 2 and 3 form a continuum. The corresponding surface ECG (Bottom panel) shows a definite J-point, but absent "K- junction".

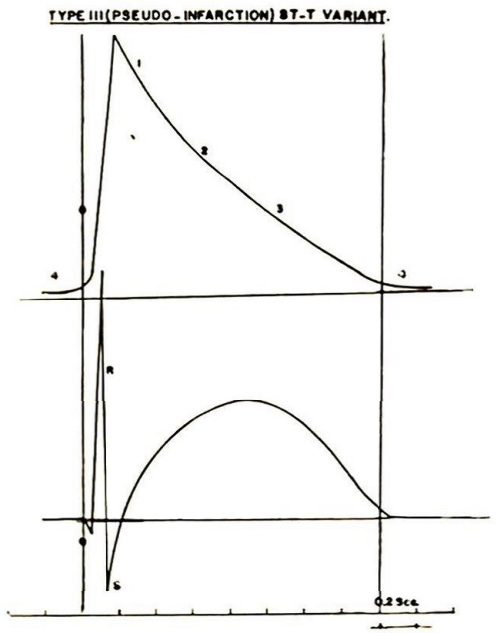

Fig. 7: Type III (Pseudo-Infarction) ST-T variant

Hypothetical Cardiac Action Potential (Top panel) in which phases 1, 2 and 3 form a continuum. In the corresponding surface ECG (Bottom panel) with ST-T coving, both the J-point and "K- junction" are absent.

Two opposing electrochemical phenomena take place during phases 2 and 3 and probably account for the magnitude of the ST-segment and T wave respectively. The first is potassium inward flow through K^+ channels thereby causing outward current (Ko) and hence VR. The second is sodium inward current (Nai) called "slow or secondary inward current" through "slow channels." The latter also permit the inflow of other cations such as Ca^{++} and Mg^{++} — a reaction that can be blocked by Ca^{++} antagonists. Thus, the force producing VR during phase 2 or 3 can be represented by Ko-Nai. As Nai tends to zero, VR becomes rapid. As Trautwein[11] pointed out, acceleration of VR in phase 3 is due to Nai inactivation while Ko is activated. It appears as though Ko overrides Nai during phase 2 in the Negro, or that Nai aborts very early during phase 2 thereby permitting early acceleration of VR. Unopposed, Ko would generate high VR voltage reflecting in J- point elevation, ST-segment elevation and tall T waves which are characteristic of Negro ECG. It is possible too that Ko acceleration and/or Nai inactivation during phases 2 and 3 account for the phathologic ST-T changes occurring in disease.

The concepts above do not contradict the reports that certain factors influence ST-T changes. VR being an electrochemical phenomenon involving the living membrane would be subject to variations in the nervous impulses, and biochemical activities abounding the myocardial tissue. Consequently, maneuvres such as anxiety, fear, and emotional reactions[13] relay their influence through the autonomic nervous system. Drugs, hormones, and electrolytes act by altering the cell environment and hence the ionic mobility across the membrane. The latter of course would have its functional integrity altered by disease conditions[14], age and sex[15]. Since the ECG is the overall manifestation of the impulse conducted from the heart to the body surface, it would be affected by physical factors such as the anatomical relations between the heart, lungs and chest wall[17], body posture[18], and the electrical conductance of the tissues.

In terms of vector orientation, the J-point vector in the sagittal and transverse planes is in the direction of leads V2 and V3 — praecordial leads with maximally elevated J-point. In a large series of 1,033 adult (Yoruba) Nigerians, Araoye[1] observed J-point elevation in 90% and 84% in V2 and V3 respectively. In the frontal plane, the J-point vector is in the direction of lead 2.

The ST-segment and T wave are generated by a progression of VR from the epicardium to the endocardium[19]. The temporal summation and spatial orientation of this potential (ST-T vector) determines the ST-T amplitude and morphology in any lead. In the adult Nigerian, the ST-T vector is away from aVR just as in other races. It is however mostly away from V1 because only 25% of the adult Nigerians of both sexes had upright TV1 (T in V1). The rest had inverted, isoelectric, notched, or biphasic TV1—so-called "persistent Juvenile pattern"[6]. However, the T vector does not appear to be oriented posteriorly and laterally as suggested by Hiss and his associates[19]. Instead, and in accordance with the principles of dynamics, the T vector is probably concurrent with the lead or leads with the tallest T wave, and perpendicular to those with the smallest or negative T wave. Of the 1,033 normal adult Nigerians studied by Araoye[1], 74% had the tallest T in the V2/V3/V4 zone. Thus, in the transverse plane both the J-point and T wave vectors are in the directions of V2 to V4. In the frontal plane

both point towards lead 2.

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