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Title page

Title

Distinctive ECG Patterns in Healthy Black Adults

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Abstract

Six ECG patterns are found more frequently in healthy black adults than in whites. These patterns are presumably benign, but also may resemble those of malignant disease. 1) Healthy black adults show higher QRS voltage, and more often meet ECG criteria for left ventricular hypertrophy (LVH). Associated repolarization abnormalities can produce ST segment elevation (STE) that resemble ST elevation MI (STEMI). 2) The pattern of benign anterior STE, seen often in males, is more common in black subjects. Similar to LVH, this pattern may falsely suggest STEMI. 3) Both early repolarization (ER) and benign inferolateral STE are more common in black patients. Although they may convey a higher risk of fatal arrhythmias or cardiac death in white populations, it does not appear that black subjects with these patterns show a similar risk. 4) The persistent juvenile T wave inversion pattern shows asymmetric T wave inversion (TWI) in V1-V4, without ST segment deviations. It is most common in black females, and is considered benign. However, this pattern can also resemble the anterior TWI of arrhythmogenic right ventricular cardiomyopathy (ARVC). 5) A pattern of anterior TWI with associated J point elevation is a common finding in the black population, especially athletes. It could suggest hypertrophic cardiomyopathy, but can be presumed to be a benign finding in black athletes, when TWI is limited to V1-V4 and preceded by J point elevation. 6) TWI in the lateral precordial leads, usually associated with end-QRS slurring or notches is seen much more often in apparently healthy black subjects than white subjects. Unlike the anterior TWI pattern, however, it cannot be presumed benign.

In conclusion, awareness of these ECG patterns may help to avoid unnecessary diagnostic or therapeutic interventions, but also encourage appropriate investigations.

Introduction

We aim to review those ECG patterns found more often in healthy black adults of African ancestry than in those of other backgrounds (e.g. white European ancestry). Our review highlights that a robust association of "black race" with certain ECG patterns can be found across a wide variety of black populations, from African American military veterans to Bantu villagers. We focus on adults who are not highly-trained athletes.

The black population is understudied in medicine, and this is no less true in electrocardiography. The ECG patterns we review are presumed to be benign, but they also may resemble those of rare but malignant disease. Accordingly, the clinician may be led to initiate extensive medical investigation, producing unneeded anxiety and costs. On the other hand, early ECG evidence of a malignant condition may be discounted as a nonspecific or benign finding in black patients.

A note on our terminology: We use the term "black" to refer to people with African ancestry and dark skin. Of course, this belies the considerable heterogeneity within the "black population," and ignores how race was assigned in each study (e.g. self-assigned versus designated by the researchers).[2] More specifically, we describe subjects who reside in the United States as "African American," despite the heterogenous character of this population.[3] Similarly, African subjects are referred to by their nationality; e.g. "black South Africans," as African sub-populations show remarkable diversity in terms of geography, culture, language.[1] Different ECG patterns may be represented unequally across black African and American sub-populations.[4],[5],[6] Also, we have tried to avoid the term "variant" to describe the ECG patterns described in this review. Just as the term "non-white" should be avoided, as it implies that "white" is the norm,[2] the use of the term

"variant" emphasizes the "non-black" ECG patterns as normative. We acknowledge the arguments against this terminology as well and expect that future readers may find our language outdated. We trust those readers will understand our intent.

Lastly, we must note that the ECG cannot be used to predict or confirm a patient's racial background.

QRS magnitude and criteria for left ventricular hypertrophy

Higher QRS voltage and greater rates of electrocardiographic LVH are more common in Blacks

Higher QRS voltage and greater rates of electrocardiographic LVH (most markedly with the Sokolow-Lyon criteria), are more common in black subjects compared with white subjects (Figure 1).

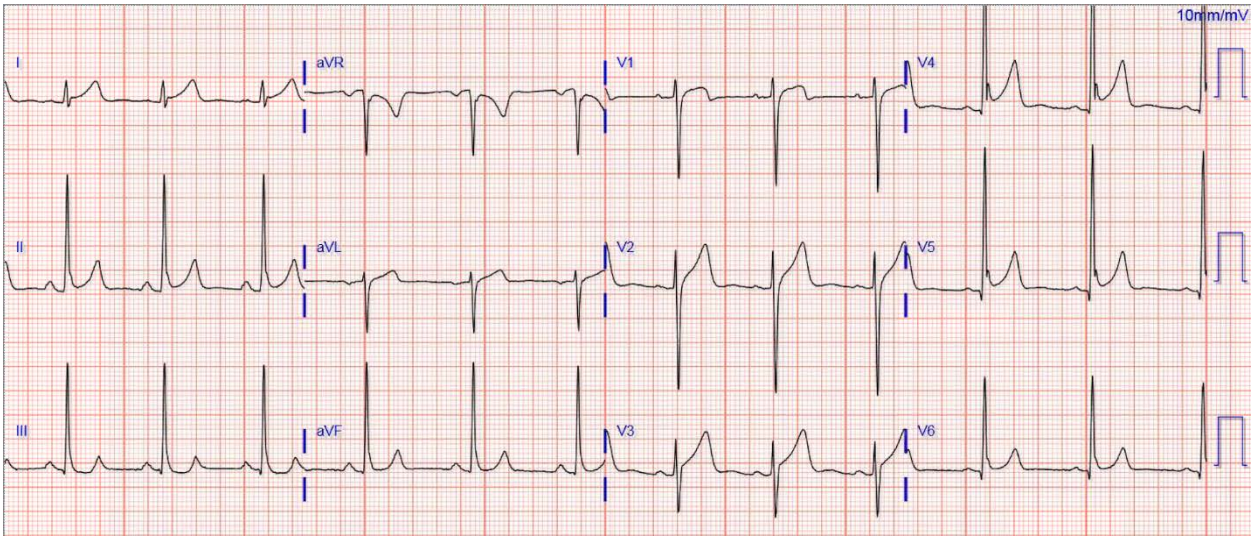

Figure 1

This racial difference is found whether the subjects are free of heart disease, have hypertension, or have overt hypertrophic cardiomyopathy. Up to 24%-30% of healthy young black adults can

meet Sokolow-Lyon criteria for LVH.[7],[8],[9] Even with adjustment for age and sex, black subjects show significantly higher QRS voltage than white subjects (e.g. 23% of black men versus 6% of white men[10]), and fulfill ECG criteria for LVH more often than white subjects.[11],[12] Similar results are seen in patients with hypertension,[13],[14] or overt hypertrophic cardiomyopathy.[15]

Of course, differences in QRS voltage, by themselves, are unlikely to be clinically relevant. However, a prominent pattern of LVH could complicate assessment of acute cardiac ischemia.

Clinical relevance: Risk of over-diagnosis of STEMI

The LVH pattern itself is not distinctive in individual black subjects versus whites. However, the higher prevalence of the ECG pattern of LVH may lead a disproportionately higher risk of overdiagnosis of STEMI in black patients.

An LVH pattern can confound accurate identification of a STEMI.[16] When LVH generates a deep S wave in V1, there may be associated ST segment elevation in V1 and V2. This secondary repolarization phenomenon, commonly described as a "strain" pattern, may mimic an anterior wall STEMI.[17] No validated criteria accurately diagnose STEMI in the setting of LVH.[16] While Armstrong et al. suggested that a ratio of ST elevation to QRS magnitude would be useful,[18] others have noted that such criteria would likely be highly insensitive.[19]

While the strain pattern can occur in a patient of any race, black patients show this pattern more often than white patients; among patients with hypertension, 28% of blacks show a strain pattern versus only 10% of whites.[20],[21] This may explain why ECG features of LVH increase the risk of

an inappropriate cardiac catheterization laboratory activation for STEMI,[22],[23] and why black patients may be at higher risk for this than white patients (Figure 2).[22,23]

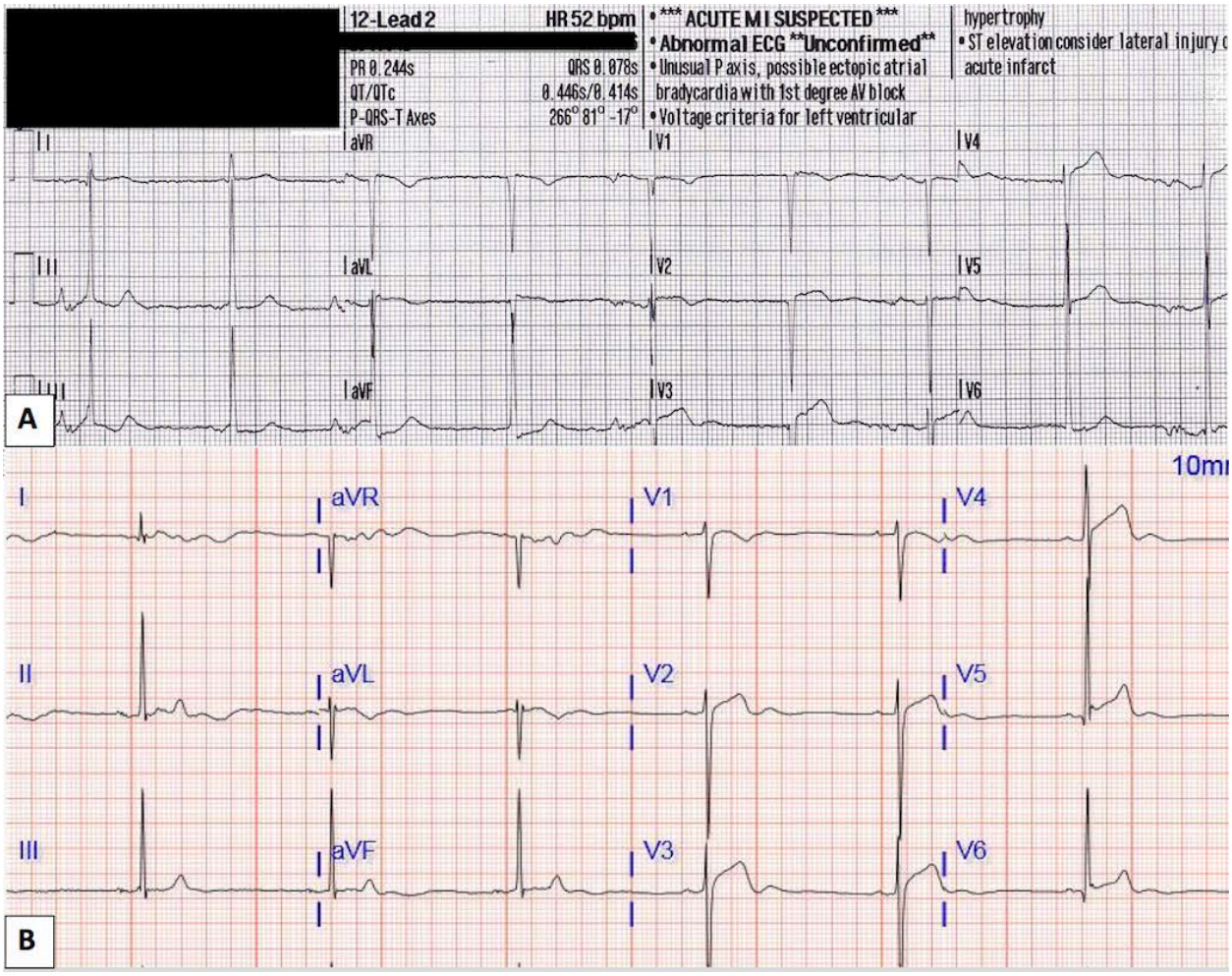

Figure 2

However, it is not clear that black race is an independent risk factor for a false-positive activation,[18] and more work is needed in this area. Additionally, healthy black patients often manifest more anterior ST segment elevation than white patients (this topic is addressed more fully in the following section),[11] and it is unclear if this baseline ST elevation is additive with the ST elevation seen with LVH "strain," or if it is a distinct phenomenon.

Benign Anterior ST Segment Elevation

A modest degree of ST segment elevation (STE) in V1-V4 has long been considered a benign finding (Figure 3).[24] This STE is more prominent in young males,[24] and had previously been termed the "male pattern."[25,26]

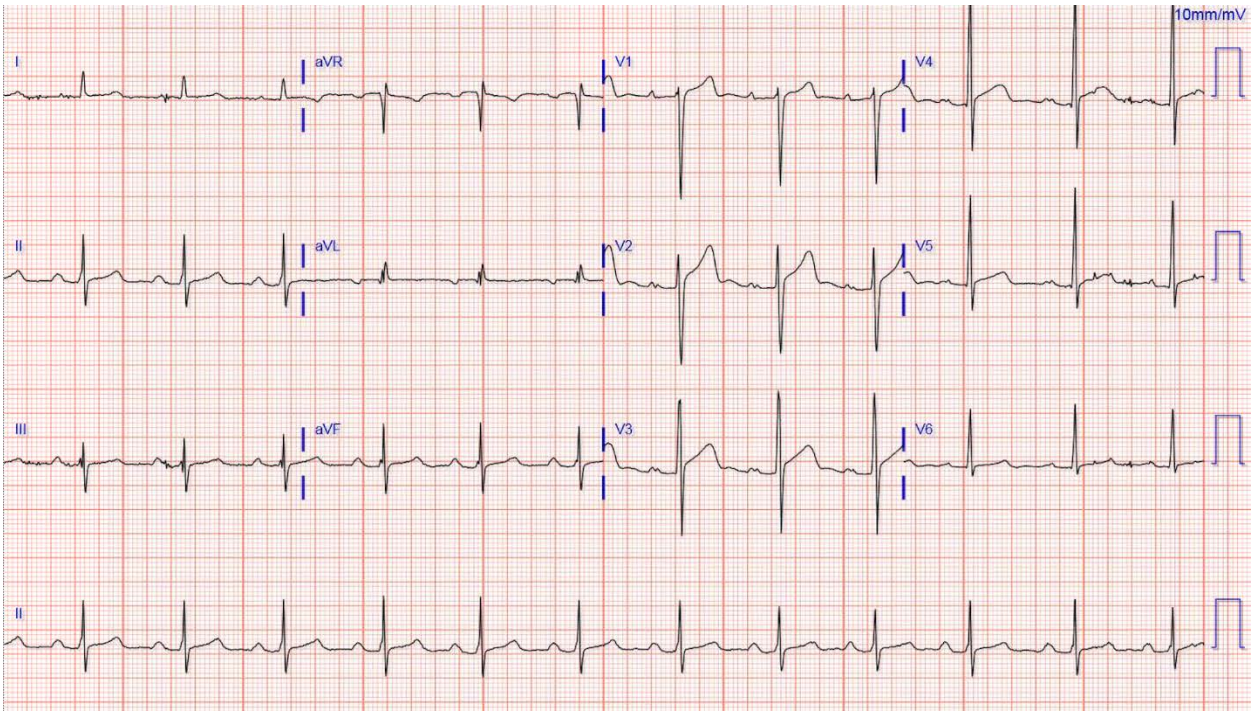

Figure 3

The STE is not seen in the inferior and lateral leads, unlike the "classic" pattern of early repolarization (ER) (now simply called benign inferolateral STE).[27] Nonetheless, this pattern of normal anterior STE does not always show the end-QRS features of notching or J point slurring that define ER. Benign anterior STE may coexist with, or be conflated with, a true ER pattern,[28] although STE in V1-V3 has usually been excluded from studies of ER.[29] The normal anterior STE pattern is also distinct from anterior T wave inversion with associated STE, e.g. the "normal variant" pattern described by Wang.[25] (We discuss that pattern in a subsequent section.)

Benign anterior STE is more common in the black population

Multiple studies support that healthy black men manifest more STE in the anterior leads compared with white patients.[30],[31],[32],[33],[34],[11] Furthermore, despite the old description of a "male" pattern, this racial difference is seen in females as well, with higher STE in V2 and V3 seen in African American females compared with white females.[34] Indeed, black females may manifest STE in V2 or V3 equal to,[31] or higher,[33] than that in white males.

Clinical relevance: Benign anterior STE may mimic STEMI

Dramatic (but benign) anterior STE may falsely suggest an anterior STEMI. While Wang suggested that benign anterior STE typically shows a concave ST segment,[25] this is not a reliable finding.[35] A better tool is the subtle STEMI score, first derived as a 3-factor calculation (QTC, the height of the R wave in V4,and the height of the ST segment in V3 at 60 ms following the J point).[36] (Available on-line at https://www.mdcalc.com/subtle-anterior-stemi-calculator) A 4-factor calculation, using the total QRS amplitude in V2, has also been derived.[37] and validated.[38]

Additionally, patients with marked benign anterior STE should be identified by the clinician, and encouraged to carry with them a paper or electronic copy of their baseline ECG.

The Early Repolarization Pattern

We restrict the term early repolarization (ER) to describe an ECG pattern characterized by end-QRS slurring and/or notching in the inferior and/or lateral leads (Figure 4).[39] However, some experts allow benign inferolateral ST elevation, with or without end-QRS notches or slurs, to be

described as “ER pattern with ST-segment elevation.”[27] We avoid this term, but will discuss studies that consider benign infero-lateral STE without end-QRS phenomena. We are careful to distinguish those from studies that examined true ER.

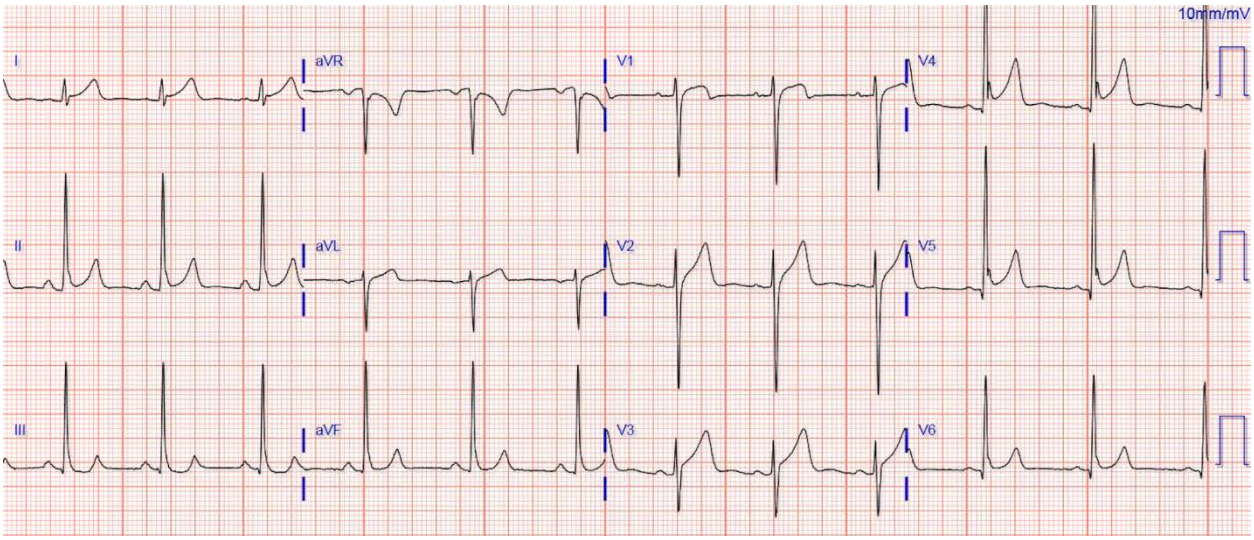

Figure 4

ER is more common in black populations

Both ER and the pattern of benign inferolateral STE seem to be more common in black patients compared to white patients. Early literature suggested that ER is common in black populations,[40],[41],[42],[4],[43],[44],[45],[46] Many of these studies were limited by unclear definitions or examples. Nonetheless, both the studies that consider benign STE (with or without end-QRS notches or slurs), [47],[48],[49],[50] as well as studies that consider true ER,[47],[51],[52] almost all show higher prevalence in black patients.[47],[48],[51],[53],[54]

Clinical relevance: “Malignant” ER

Although ER is, in general, a benign finding, some studies have demonstrated subtypes of ER that predicted a higher rate of ventricular fibrillation[29] or a higher rate of cardiac death.[55] It does

not appear that black patients with ER have a similar risk of these outcomes, and indeed they may have a lower risk compared with whites. Stavrakis et al. found that ER was associated with increased all-cause mortality only in "non-African American" subjects (versus the combined group of Caucasian and "other" subjects),[56] while Muramoto et al. did not find increased mortality in either African Americans or "non-African Americans".[51], [53]

Similarly, while benign inferolateral STE is more common in African American than non-African American patients, increased mortality may only be seen in the non-African American subjects,[48] or be unrelated to race.[50] Studies that evaluate J point elevation (JPE) as an ER "surrogate" suggest that only white patients with JPE had higher rates of sudden cardiac death.[49],[57] but these studies lacked details on JPE definition, and are hard to compare directly to the rest of the ER literature.[58]

The Persistent Juvenile T Wave Pattern

The persistent juvenile T wave pattern consists of asymmetric T wave inversion seen in V1 through to V3 or V4. These inversions show a slow descent, with a relatively brisk upstroke, and there is no associated ST segment deviation (Figure 5).[59],[60],[61] It is named because it resembles the normal child's ECG,[62],[63] As a child ages towards adolescence, the T waves gradually "flip" upright, starting in the left-most leads first. When this otherwise "juvenile" pattern is seen in an adult, it has been termed the "persistent juvenile T wave pattern."[64]

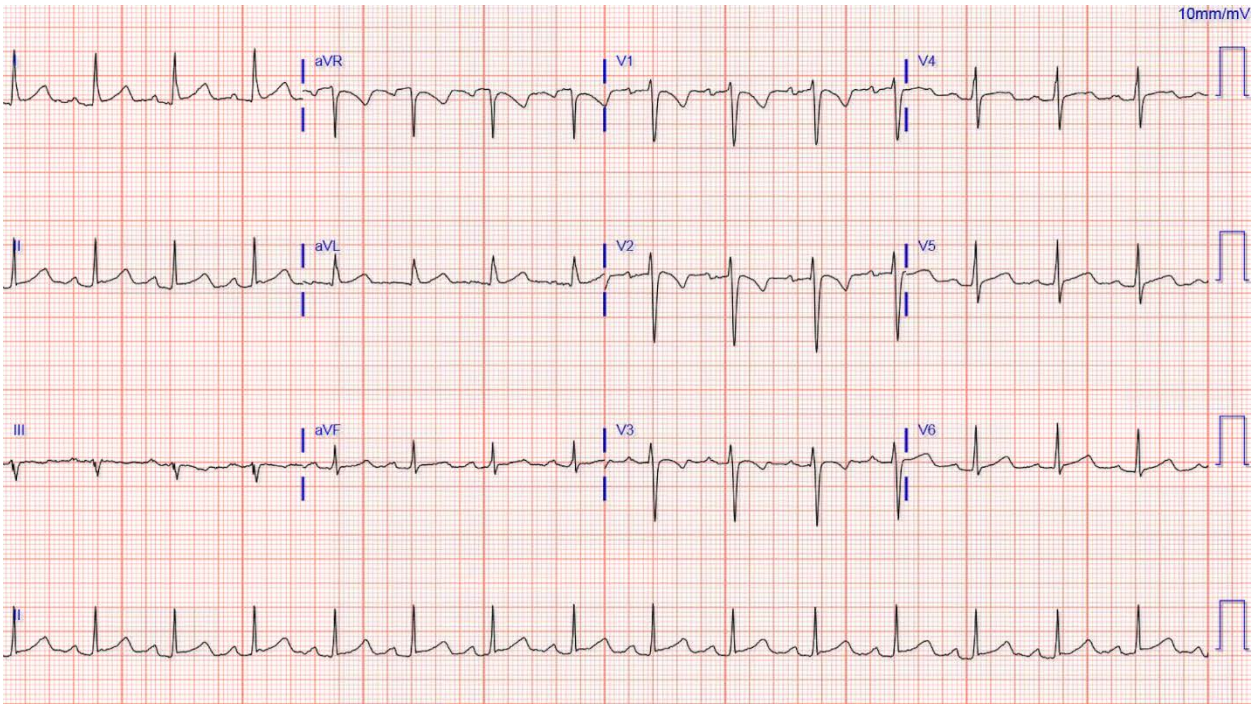

Figure 5

Juvenile T wave pattern is more common in black patients

This pattern was recognized as early as 1946, found predominantly in black subjects, and especially women, with prevalence of 2.1% to 4.6%.[65],[30],[44] [66] The older literature can often be difficult to interpret, as many studies term a variety of patterns (i.e. overt ST deviation in addition to T-wave inversion) as "juvenile pattern".[40],[67],[68] Contemporary literature supports the view that the juvenile pattern is more common in blacks than whites,[69],[70],[71],[9] and more often in females than males,[72] and is especially common in black females.[59],[69] Lastly, this pattern is not clearly related to strenuous athletic training,[73],[74] although the evidence is mixed.[70]

Clinical relevance: The juvenile pattern resembles arrhythmogenic right ventricular cardiomyopathy.

Clearly, anterior T wave inversion can be caused by a number of cardiopulmonary conditions. However, some of these conditions may present with suggestive symptoms, and/or can be excluded using laboratory or imaging studies

Arrhythmogenic right ventricular cardiomyopathy (ARVC) – at least in the early stages – may show certain ECG signs in the absence of marked symptoms. The differential diagnosis of the juvenile T wave pattern in a patient with mild or absent symptoms should include ARVC. Anterior T wave inversion is now considered one of the major criteria for diagnosis of ARVC,[75] and is far more sensitive than the finding of an epsilon wave. ARVC should be considered when T wave inversion extends throughout V1-V3.[60],[70],[76] Unfortunately, there is scant literature that suggests the prevalence of ARVC in Black populations. A US-based registry of ARVC cases has enrolled very few black patients;[77] only one patient was included in the most recent study (versus 432 white patients).[78] Accordingly, the prevalence of ARVC in black non-athletes with an ECG pattern that otherwise suggests persistent juvenile T wave inversion is unknown, and the clinician risks both over- and under-testing.

Precordial T wave Inversion with J point Elevation

Inverted or biphasic T waves, in combination with J point elevation, may be seen in the precordial leads of healthy black subjects more often than in white subjects. This was suggested in studies starting in the mid-20th century that examined both African and African American cohorts. The prevalence of precordial T wave inversions in these studies ranged from 10% of hospitalized African Americans[67] to 25%- 34% in African populations.[40],[42] Other studies found that precordial T wave inversion were markedly more common in black populations than in white populations,[65],[4],[30]

Inconsistent definitions, examples, and terms, have made it difficult to analyze this older literature. For example, these precordial inversions received a variety of names; "juvenile pattern,[67]" "Pattern I,[40]" "the other variant,[79]" the "normal ST-T wave variant,[24],[80]" and as "benign T wave inversion.[81]" We will focus on the contemporary literature.

The T wave inversions may be seen predominantly in the anterior leads (V1-V3) or in the lateral leads (V4-V6). [30,40,65,67],[24],[80] There are different implications for T wave inversions in these two regions, so we discuss them separately. We first discuss the anterior pattern in this section, and the lateral pattern in the next.

Anterior T wave inversion

The TWI is usually biphasic, with terminal TWI. J point elevation is seen in those same leads, often with convex-upwards STE (figure 6). This J point (± STE) distinguishes this pattern from the anterior TWI seen in the persistent juvenile T wave pattern.

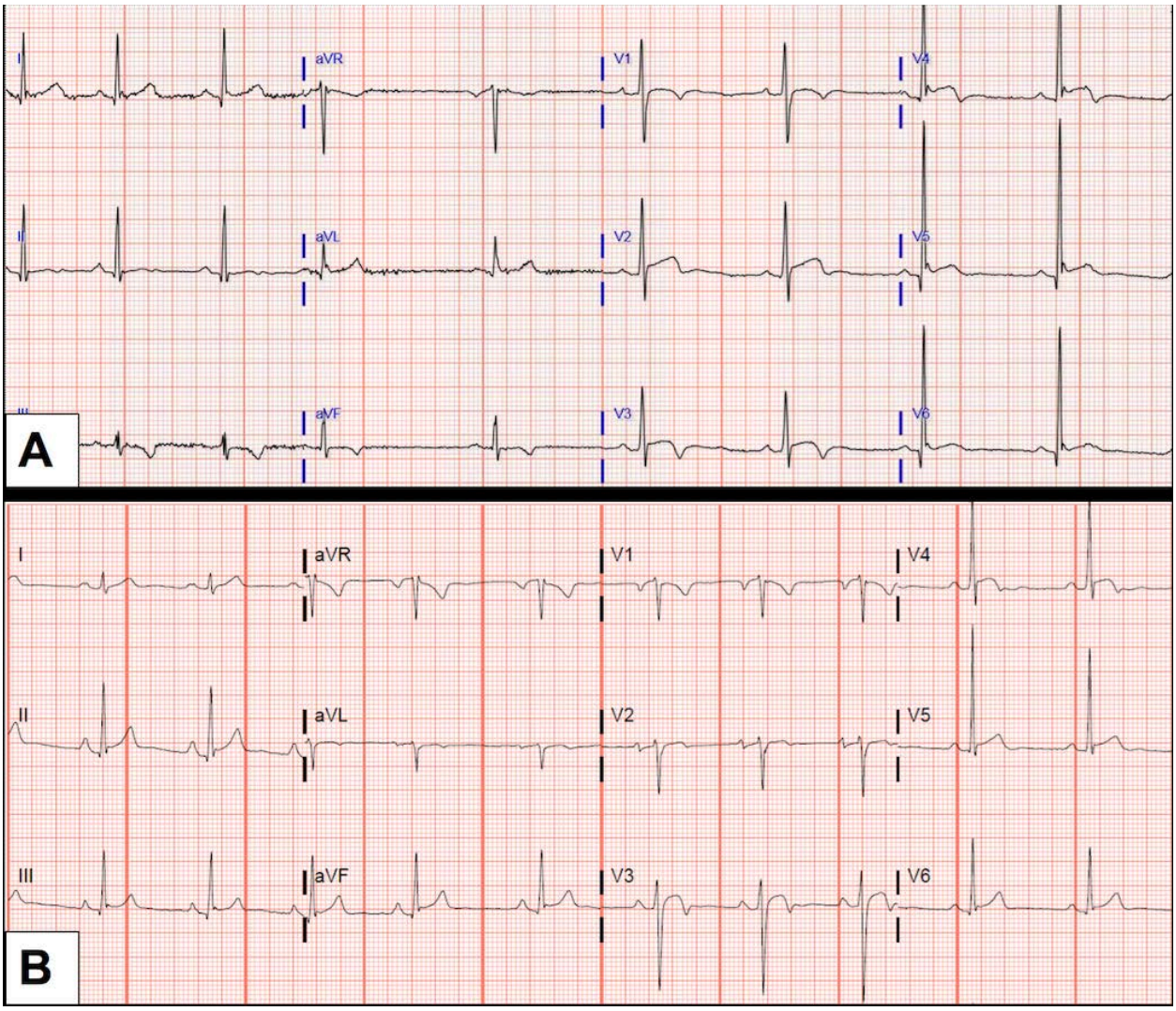

Figure 6

Anterior T wave inversion is more common in the black population

Older studies found that anterior TWI was found in 3%-10% of black Africans or African Americans,[30,40,65,67] but not in white subjects.[65] More contemporary literature finds that anterior TWI can be found in 4.2% of healthy, non-athletic black males.[82] By contrast, anterior TWI is seen in only 1.9% of white male athletes,[82] and rarely extends past V2.[83] The prevalence in non-

athletic middle aged white men is even lower (0.5%).[72] Similarly, this pattern is found in 14% of black European female athletes, but only 2% of white female athletes.[71]

Clinical relevance: Anterior TWI could suggest HCM or ARVC.

This pattern was found in over 12% of black European male athletes,[82] and is recognized as a benign feature in the black athlete.[84] This may be true in athletes of any race: one study has demonstrated that the combination of J point elevation >0.1mV and TWI limited to V1-4 ruled out arrhythmogenic right ventricular cardiomyopathy and hypertrophic cardiomyopathy, regardless of ethnicity.[85] In the absence of J point elevation, HCM or ARVC must still be considered. The literature hasn't specifically addressed whether this pattern of anterior TWI with J point elevation can be presumed benign in non-athletes, black or white (figure 6b).

Lateral T wave Inversion

TWI in the lateral leads is associated with myriad conditions.[86] Many etiologies would be suggested by a patient's symptoms (e.g. stress cardiomyopathy, acute ischemia, pulmonary embolus), while others may be relatively asymptomatic, but would be suggested by their medical history (e.g. chronic cardiomyopathy, medications). In particular, left ventricular hypertrophy caused by chronic hypertension may produce a "strain" pattern, with characteristic asymmetric lateral TWI, usually with an associated downsloping ST segment.[87]

However, lateral TWI may also occasionally be found in apparently healthy (and specifically non-hypertensive) patients. (figure 7) The ECGs of these subjects will usually show the end-QRS notching and/or slurs of the ER pattern, often with associated J-point elevation.[45] TWI may also

be concurrently seen in the inferior leads.[24,81] Similar to the ER pattern, the lateral TWI pattern can be labile over time.[24]

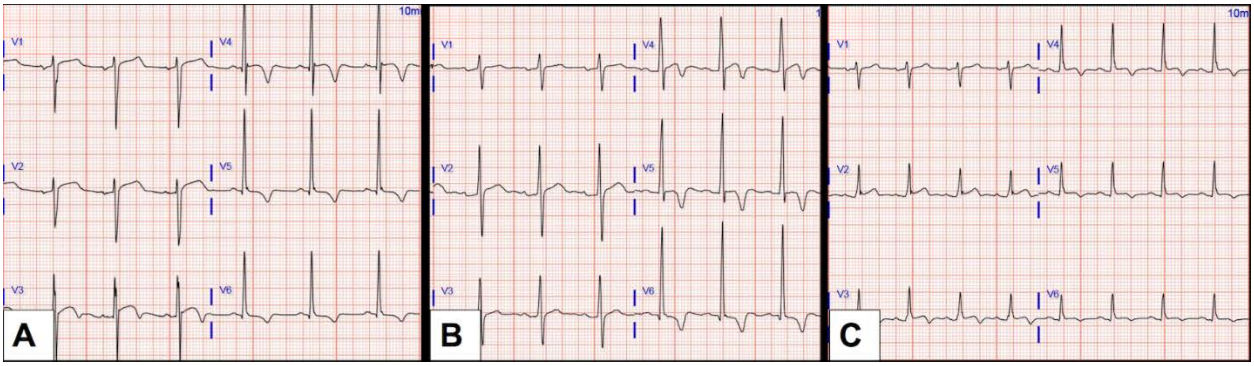

Figure 7

Lateral T wave inversion is more common in the black population

Older literature suggested that apparently healthy black men occasionally had TWI in the lateral precordial leads.[45] Contemporary literature has found this pattern in 3.3% - 4.2% of male black non-athletes.[80],[82],[8] It is much less frequently seen in black females, and is rare in white men or women.[80]

While the lateral TWI pattern has been noted in black athletes, [5] it does not appear to be related to athletic training. It is found in black athletes and black controls at statistically similar rates (4.1 % vs 3.4%).[82]

Clinical relevance: Lateral TWI may indicate HCM.

As mentioned above, this pattern can often be seen in apparently healthy black men. However, it cannot be presumed to be benign. Lateral TWI is seen in about 70% of black patients with HCM, and so evaluation with echocardiography should be considered, and possibly cardiac magnetic

resonance imaging, depending on the clinical context (e.g. a history of unexplained syncope). It is worth emphasizing that these ECG findings can precede development of overt hypertrophy. If imaging does not demonstrate overt HCM, the patient still requires periodic reevalution to follow for an evolving HCM phenotype.

Nonetheless, lateral TWI will likely be far less specific for HCM in black subjects than in white subjects, because lateral TWI are about 10 times more common in black athletes or healthy black (negative echocardiography) controls than in white athletes.[82],[8] The high prevalence of this T wave pattern in otherwise healthy black subjects could raise a concern with over-testing for HCM in this population.

This potential for over-testing was relatively small in white athletes. It may still be tolerable when such testing is limited to elite black and white athletes in organized sports; the absolute number of cases is not likely to strain health-care resources. However, it is unclear if 3%-4% of apparently healthy black men and women could realistically be screened with echocardiography ± cardiac magnetic resonance.

The utility of lateral TWI for ECG screening of HCM relies on understanding the pre-test probability of the disease.[88] However, the prevalence of HCM in the general black population is understudied: Only 5% of patients in a U.S. registry of HCM were black.[89] While the prevalence of HCM could be lower in the black population, there is also concern that this represents under-diagnosis and consequent under-enrollment in studies.[90] Evaluation of lateral TWI in black athletes requires a thorough investigation to exclude cardiomyopathy and also avoid unnecessary

restriction from sports. However, the markedly higher rate of lateral TWI in black subjects raises the concern of generating unnecessary costs and anxiety, for athletes and non-athletes alike.[90] Further study is needed to clarify these issues.

Conclusion

There are several forms of ST and/or T-wave changes which are more prevalent in black populations. Clinical presentation will dictate further investigation of these ECG patterns, and this approach has been refined in the black athlete population. However, some experts have argued for applying sports screening guidelines to a broader non-athlete population.[91] It is unclear how the criteria developed for interpreting the ECGs of black athletes should be applied to black non-athletes, and vice versa, and there is the potential for both type I and type II errors. Widespread ECG screening in low-risk populations can potentially increase cardiac testing without affecting rates of intervention or morbidity.[92],[93] Awareness of these differences may help to avoid unnecessary diagnostic or therapeutic interventions of benign ECG patterns, but also encourage prompt appropriate investigation of suspect patterns.

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Figure Legends

Figure 1. ECG of healthy 26-year-old Black male. The precordial QRS voltage satisfies several Sokolow-Lyon criteria for left ventricular hypertrophy. Early repolarization is also seen in the inferior and lateral leads.

Figure 2. A 38-year-old Black man presented with severe acute onset chest pain. (A) EMS activated the cardiac catheterization lab based on the anterior STE, as well as the computer interpretation seen on his ECG. However, the ECG was similar to prior ECGs (B), and was attributed to LVH due to hypertension. (The apparent new Q waves in (A) are due to high placement of the precordial leads.) The patient was ultimately diagnosed with type A aortic dissection.

Figure 3. Example of right-precordial STE, "male pattern."
23-year-old black male, healthy. Note the STE in V2 and V3. There are no J waves or end-QRS slurring. (STE = ST segment elevation)

Figure 4. A 26-year-old healthy male, no history of hypertension. ER is seen as the end-QRS notching in V4 and V5, and end-QRS slurring in leads II, III, aVF, and V6. There is ST-segment elevation in anterior leads as well (male variant of STE).
ER = early repolarization STE = ST segment elevation

Figure 5. ECG from a healthy 20-year-old African American female. Note asymmetric T wave inversion in V1-V3 without ST segment elevation.

Figure 6. Patterns of anterior T wave inversion in black subjects. (a) A 28 year-old man with a normal echocardiogram, (b) a healthy non-athletic 21 year-old man

Figure 7. Examples of lateral TWI in black subjects. (a) 34-year-old man without hypertension (b) 36-year-old man without hypertension (c) 47 year-old man with controlled hypertension