Racial and Sexual Differences in the Standard Electrocardiogram of Black vs White Adolescents*

Mark A. Reiley, B.A.; Jih-Jui Su, M.S.; and Barbara Guller, M.D., F.C.C.P.

Standard 12-lead electrocardiograms were recorded in 114 healthy adolescents to substantiate possible influences of race and sex on the “juvenile pattern” (increased precordial voltages of QRS complex, precordial T wave inversions, and ST-segment elevations considered pathologic in adults) in this age group. Black male subjects had the highest precordial QRS amplitudes and the highest incidence of biphasic or negative precordial T waves and ST-segment deviations. In white male subjects, these findings were less pronounced but were more evident than in black or white female subjects. Results indicate the following: (1) race-specific and sex-specific normal electrocardiographic standards should be developed in adolescents; (2) criteria for left ventricular hypertrophy are race-specific and sex-specific and should be tested against independent anatomic or physiologic information in adolescents with left ventricular overload; and (3) the “juvenile pattern” may be viewed as a predictable continuum of age-related changes starting in childhood and progressing through adolescence on to later life.

With the emergence of the use of chest leads, it became apparent that the precordial T waves of children are often opposite in direction to those obtained from similar positions in adults. These inverted T waves became known as the “juvenile pattern.” Since that time, other electrocardiographic findings that are considered pathologic in adults but occur normally in healthy children have been observed and should be included under the term, “juvenile pattern.” These findings are not unusual elevations of the ST-segment in leads V1 to V6 and increased precordial QRS voltages that may mimic left ventricular hypertrophy. Strong et al have reported normal data for the aforementioned three variations of the “juvenile pattern” in male and female adolescents. These investigators showed that this pattern is significantly more pronounced in the adolescent than in the adult.

Previously published normal adolescent electrocardiographic data do not take racial differences into account. This study substantiates the influence of race on the normal adolescent ECG, since race is one of the major constitutional variables affecting QRS voltages and components of the ST-segment and T wave. Electrocardiographic data are given for black and white adolescents of either sex. The findings are compared with those of younger and older normal populations, in order to determine whether the “juvenile pattern” of the adolescent is related to the preadolescent and adult ECGs.

Materials and Methods

Standard 12-lead ECGs were recorded in 114 adolescents aged 11 to 17 years. According to Strong et al, the normal ECG is only slightly dependent on age within this period. The adolescents were divided into the following four groups: (1) 27 black male subjects; (2) 27 white male subjects; (3) 34 black female subjects; and (4) 26 white female subjects. None had evidence of heart disease by physical examination or history. Individuals who participated regularly in competitive sports and could be labelled as athletes were excluded from the study because athletes may have increased QRS voltages and changes in the T vector.

The recorder was a three-channel direct writer (Marquette Electronics series 3100 automatic electrocardiograph). This instrument fulfills the 1975 requirements of the American Heart Association for the frequency response of electrocardiographs, which should be between 0.05 and 100 cps. Direct writers used previously to report normal electrocardiographic data in children have an upper cutoff frequency at 50 cps. If 50 rather than 100 cps is the upper limit on frequency, differences in measurements of the peak amplitude and width of the QRS complex may occur in children. Such differences do not significantly affect the diagnostic interpretation of ECGs based on manual measurements of the R and S waves. Our data can therefore be compared with those of previous authors.

The limb and precordial leads were attached according to the recommendations for placement of leads in children suggested by the 1976 Bethesda Conference on Optimal Electrocardiography. The paper speed was 25 mm/sec. The leads on the extremities were calibrated at 10 mm/mV and the precordial leads at 10 mm/mV or at 5 mm/mV. The following manual measurements were taken: frontal-plane P axis; P-R interval; P-wave amplitude in lead 2; duration of QRS complex; frontal-plane QRS axis; R-wave amplitude in

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leads 1, aVR, aVL, aVF, V1, V2, and V6; S-wave amplitude in leads 1, 3, V1, V2, and V6; Q-wave amplitude in leads 3, aVF, V2, and V6; direction and magnitude of ST-segment deviations in leads aVF, V1, V2, and V6 to V6; frontal-plane T axis; T1 and T2 amplitudes and directions in leads 3, aVL, V1, V2, and V4 to V6; and heart rate. Measurements of voltage were made from the center of the line to the center of the line. The line of reference for the ST-segment was the preceding P-R segment. The mean electrical axis of the P wave, QRS complex, and T wave in the frontal plane was estimated utilizing the hexaxial reference frame. The axis for these waves was positioned within ±15° of the lead projection with the largest positive or negative deflection of a wave. The following criteria for left ventricular hypertrophy were computed: the sum of the R wave in lead V5 plus the S wave in lead V6; the sum of the R wave in lead V4 plus the S wave in lead V5; and the sum of the R wave in lead 1 plus the S wave in lead 3.

To evaluate the influence of sex and race on these data, sex-related differences between the means of electrocardiographic variables were tested in adolescents of the same race, while racial differences were determined in the two adolescent groups of the same sex. Statistical calculations included means, standard deviations, and a t-test to evaluate the significance of differences between means at various levels of probability. A χ² test was used to test differences in the incidence of various patterns of repolarization. For samples sized less than 50 subjects, the range given is the maximal and minimal values. For samples exceeding 50 subjects, the 96th percentile range was computed. This range is not meaningful for smaller samples, although it is desirable to define limits for data with a non-Gaussian distribution, such as for electrocardiographic data with percentile limits, rather than by the extreme values. For angular measurements (including the QRS, P, and T axis), determination of the arithmetic mean does not indicate the degree of clustering, while the precision method used by Strong et al. for the statistical treatment of angles is more appropriate and allows for comparison of means. Since much overlap in our raw angular data was evident between the four groups, we have not computed the degree of clustering, as proposed by Strong et al., and thus have not evaluated the statistical significance of differences between angular measurements.

RESULTS

Tables 1 and 2 and Figures 1 through 3 describe electrocardiographic data for normal black and white adolescents of either sex. Items that are tabulated, illustrated, or described in the section on "Results" are of clinical significance for differentiating among the four groups and include QRS amplitudes and T-wave size and polarity, as well as ST-segment deviations. (Tables that contained data of lesser clinical significance were omitted from the text but are available from us on request. These tables list the means, standard deviations, ranges, and significance of differences between means for all electrocardiographic data listed in the section on "Materials and Methods" for the four groups of adolescents, which were not mixed racially and sexually [white male, black male, white female, and black female subjects] and for the combined groups [all white, all black, all male, and all female subjects]).

<p>| Table 1—Upper limits (Maximal Values) of R-Wave, Q-Wave, S-Wave Amplitudes in Healthy Adolescents |
|--------------------------------------------------|--------|--------|--------|--------|</p>
<table>
<thead>
<tr>
<th><strong>No. of subjects</strong></th>
<th>Black Male</th>
<th>White Male</th>
<th>Black Female</th>
<th>White Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limit of R-wave amplitude, mm</td>
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<td></td>
<td></td>
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<tr>
<td>Frontal leads</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Lead 1</td>
<td>15*</td>
<td>10</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>Lead aVR</td>
<td>5</td>
<td>4**</td>
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<td>2</td>
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<tr>
<td>Lead aVL</td>
<td>11</td>
<td>11</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Lead aVF</td>
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<td>Precordial leads</td>
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</tr>
<tr>
<td>Lead V1</td>
<td>11</td>
<td>8</td>
<td>10</td>
<td>8</td>
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<tr>
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<tr>
<td>Lead V4</td>
<td>30*</td>
<td>36</td>
<td>47**</td>
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</tr>
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<td>Lead V5</td>
<td>44*</td>
<td>39**</td>
<td>25**</td>
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<td>Lead V6</td>
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<td>Upper limit of Q-wave amplitude, mm</td>
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<td>Lead V4</td>
<td>3*</td>
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<td>Upper limit of S-wave amplitude, mm</td>
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<tr>
<td>Lead V1</td>
<td>34</td>
<td>32</td>
<td>26*</td>
<td>16</td>
</tr>
<tr>
<td>Lead V2</td>
<td>34</td>
<td>36**</td>
<td>26**</td>
<td>36</td>
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<td>Lead V4</td>
<td>4</td>
<td>5</td>
<td>2**</td>
<td>2</td>
</tr>
</tbody>
</table>

*Statistically significant racial difference (P<0.05) within same sex. **Statistically significant sexual difference (P<0.05) within same race.

Racial Comparisons

Black Male vs White Male Subjects. Clinically important differences in the "juvenile pattern" were

Table 2—ST-Segment and T Wave in Precordial Leads of Healthy Adolescents

<table>
<thead>
<tr>
<th><strong>No. of subjects</strong></th>
<th>Black Male</th>
<th>White Male</th>
<th>Black Female</th>
<th>White Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. with ST-segment deviation (percent)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Lead V1</td>
<td>11 (41)</td>
<td>14 (62)</td>
<td>11 (33)</td>
<td>3 (12)*</td>
</tr>
<tr>
<td>Lead V2</td>
<td>25 (93)*</td>
<td>22 (81)</td>
<td>24 (71)</td>
<td>18 (69)</td>
</tr>
<tr>
<td>Lead V4</td>
<td>25 (93)*</td>
<td>19 (70)</td>
<td>23 (67)</td>
<td>12 (46)*</td>
</tr>
<tr>
<td>Lead V5</td>
<td>21 (78)**</td>
<td>13 (48)</td>
<td>17 (50)*</td>
<td>9 (35)</td>
</tr>
<tr>
<td>Lead V6</td>
<td>15 (56)**</td>
<td>5 (19)</td>
<td>15 (44)**</td>
<td>5 (19)</td>
</tr>
</tbody>
</table>

*Statistically significant sexual difference (P<0.05) within same race. **Statistically significant racial difference (P<0.05) within same race.

†No negative or biphasic T waves in leads V1 and V6.
observed between black and white male subjects. The most significant racial differences were higher R-wave amplitudes in the middle and left precordial leads, as well as in lead 1 (Fig 1; Table 1). Black subjects had higher means and ranges for the criteria of left ventricular hypertrophy, i.e. the sum of the R wave in lead V₆ plus the S wave in lead V₁, the sum of the R wave in lead V₄ plus the S wave in lead V₂, and the sum of the R wave in lead 1 plus the S wave in lead 3 (Fig 2). Black male subjects had a slightly different QRS axis in the frontal plane (mean, 59°; range, 20° to 90°) than white male subjects (mean, 69°; range, 50° to 95°). Black male adolescents had lower means and ranges for the precordial T-wave amplitudes (Fig 3). Precordial ST-segment deviations were smaller and less frequent in white subjects (Table 2).

Black Female vs White Female Subjects. Qualitative differences similar to those listed for black and white male subjects were also observed between black and white female subjects, but the racial variations in the "juvenile pattern" were less pronounced in female than male adolescents. No important racial differences in QRS voltages existed for the two groups of female subjects (Table 1), except that black female subjects had higher means and ranges for one criterion of left ventricular hypertrophy, i.e. the sum of the R wave in lead V₆ plus the S wave in lead V₁ (Fig 2). Although white female adolescents had a lower incidence of biphasic or inverted precordial T waves (Table 2), T-wave amplitudes tended to be similar in both races (Fig 3). Black female subjects had higher precordial ST-segment elevations.

Black vs White Subjects. In general, the racial

**Figure 1.** Mean precordial R-wave amplitudes in adolescents. Four curves shown pertain to black male (BM), white male (WM), black female (BF), and white female (WF) subjects. Statistical significance of differences observed among these four groups of adolescents and upper range of R-wave amplitudes are listed in Table 1.

**Figure 2.** Criteria for left ventricular hypertrophy in adolescents, with total range (size of bars) and mean values (horizontal lines within bars) in black (B) and white (W) male (M) and female (F) subjects. Differences among four groups of adolescents were largest for sum of R wave in lead V₆ plus S wave in lead V₁ (RVₛ + SV₁) (P < 0.01), RVₛ + SV₁, sum of R wave in lead V₄ plus S wave in lead V₂; and R₁ + S₁, sum of R wave in lead 1 plus S wave in lead 3.

**Figure 3.** Mean precordial T-wave amplitudes in adolescents. In lead V₄, racial and sexual differences between whites (W) and blacks (B) were statistically significant (P < 0.05). In leads V₂ and V₆, differences between male (M) and female (F) subjects of same race were statistically significant (P < 0.005).
differences observed between male and female subjects were also present between whites and blacks as sexually mixed groups. The "juvenile pattern" (i.e., increased precordial QRS voltages, biphasic or inverted T waves, and ST-segment elevations) were more frequent in blacks. In blacks the following 96th percentile ranges were observed for ST-segment deviations in the precordial leads: −0.5 to 1.5 mV in lead V1; 0.5 to 4.0 mV in lead V2; −0.5 to 2.0 mV in lead V4; 0 to 2.0 mV in lead V5; and 0 to 1.0 mV in lead V6. In whites, these ranges were −1.0 to 1.5 mV in lead V1, 0.5 to 2.5 mV in lead V2, 0.2 to 2.0 mV in lead V4, 0.2 to 1.0 mV in lead V5, and 0 to 0.5 mV in lead V6. The duration of the QRS complex was the same for blacks and whites (mean, 0.08 second).

**Sexual Comparisons**

**Black Male vs Black Female Subjects.** Significant differences between black male and female subjects existed for the means and upper limits of R-wave voltages in leads V4 through V6 and for S-wave voltages in leads V2 and V6 (Table 1; Fig 1). Means and upper limits for two of the criteria of left ventricular hypertrophy (sum of R wave in lead V6 plus S wave in lead V1 and sum of R wave in lead V5 plus S wave in lead V2) were also higher in male subjects (Fig 3), but differences between the sexes were not clinically important. Biphasic or negative T waves in the right and midprecordial leads (Table 2) were more frequent in male subjects, but this difference was not statistically significant. In the precordial leads, ST-segment elevations were higher in male subjects, except in lead V6.

**White Male vs White Female Subjects.** As was observed in the comparison of black male vs female subjects, white male adolescents demonstrated higher means and ranges for R-wave and S-wave voltages than white female adolescents (Table 1). Significantly higher R-wave voltages for male (vs female) subjects were recorded in leads aVR and V5. The combined voltages of the R wave in lead V5 plus the S wave in lead V2 were also higher in white male subjects (Fig 2). Left precordial T waves were lower in female subjects who also had a lower incidence of biphasic or inverted precordial T waves (Table 2). ST-segment deviations over the precordium were larger in male subjects.

**Male vs Female Subjects.** When black and white male subjects were combined and compared with black and white female subjects, precordial QRS voltages and criteria for left ventricular hypertrophy were higher in male subjects (Table 1; Fig 1 and 2). Male adolescents had higher precordial T waves (Fig 3) and larger ST-segment deviations. In male subjects the following 96th percentile ranges were observed for the ST-segment deviations in the precordial leads: −1.0 to 2.0 mm in lead V1; 0.5 to 4.0 mm in lead V2; 0.3 to 3.0 mm in lead V4; 0.2 to 2.0 mm in lead V5; and 0 to 1.0 mm in lead V6. In female subjects, these ranges were −0.5 to 1.2 mm in lead V1, 0.5 to 2.0 mm in lead V2, −0.5 to 1.5 mm in lead V4, −0.3 to 1.0 mm in lead V5, and 0 to 1.0 mm in lead V6.

**Discussion**

Race and sex substantially influence the ECG in the adult.13-15,18 This study indicates that sex and race influence the ECG in the adolescent as well and that normal electrocardiographic standards in adolescents should be sensitive to sex and race. Although the samples in this study are adequate to support these conclusions statistically, our data base of 114 adolescents is too small to serve as normal standards for this age group, and a larger base of race-specific and sex-specific data for normal ECGs should be developed in teenagers.

As shown later in this section, left ventricular hypertrophy may be falsely diagnosed from the QRS voltages of an adolescent's ECG if the criteria are not adjusted for sex and race; and misinterpretations of myocardial necrosis, pericarditis, or myocardial ischemia may be made if the normal variation of the pattern of the ST-segment and T wave with sex and race in this age group is not considered. We are proposing criteria for left ventricular hypertrophy in adolescents on the basis of our findings. These criteria will have to be tested against independent anatomic and physiologic information in adolescents with left ventricular overload.

In the following discussion of results, race-related and sex-related differences in the ECG in adolescents will be considered separately. The statistical analysis of the data was designed to elucidate race-related differences in adolescents of the same sex and to test sex-related differences in those of the same race.

**Racial Differences**

Differences in QRS voltages are already apparent between black and white American preadolescent schoolchildren18 and in South African biracial populations of the preadolescent age.10 Adolescents of this study exhibited much larger racial deviations than those described in children.10,16 Especially in black male teenagers, R-wave and S-wave voltages were significantly higher than in their white counterparts (Table 1). The mean precordial R-wave amplitudes were increased in each lead in black
male subjects and exceeded those of white male subjects by as much as 0.85 mV. The increase in QRS voltage in blacks was directed to the left and posteriorly and therefore affected the normal values for the criteria of left ventricular hypertrophy, which were based on R-wave and S-wave amplitudes in the limb and precordial leads (Fig 2). As evident from Figure 2, the racial differences in criteria for left ventricular hypertrophy were prominent; blacks had higher limits than whites for the sum of the R wave in lead V6 plus the S wave in lead V1, the sum of the R wave in lead V2 plus the S wave in lead V5, and the sum of the R wave in lead 1 plus the S wave in lead 3. These large racial variations in QRS voltages that were found in this study suggest that the use of additional criteria for the diagnosis of left ventricular hypertrophy which are not based on amplitudes of the QRS complex would improve the diagnostic performance of the standard ECG in adolescents. In the adult, standard electrocardiographic criteria for the diagnosis of left ventricular hypertrophy by the clinician perform best if they are based on the point-score system of Romhilt and Estes.23 If their criteria are adapted to adolescents, a total of five points from the following point-score system suggests the diagnosis of left ventricular hypertrophy: (1) four points if the R-wave amplitude in lead V5 or V6 or the S-wave amplitude in lead 3, V1, or V2 exceeds age-specific and sex-specific normal limits; (2) three points if the age-specific and sex-specific pattern of the ST-segment and T wave in the left precordial leads is outside normal limits and suggests left ventricular strain; (3) three points of there is left atrial enlargement; (4) two points if there is left axis deviation of $-30^\circ$ or more; and (5) one point if the duration of the QRS complex is increased. These criteria need to be evaluated in adolescents in whom left ventricular overload is proven by independent nonelectrocardiographic information.

The etiology of racial differences in QRS voltage is unknown. Some authors attribute it to a difference in the pattern of ventricular activation between black and whites, since these investigators noted a shorter duration of the QRS complex in blacks, although QRS voltages in blacks were higher.13,14,16 These investigators speculated that the higher QRS voltages in blacks were due to activation of more myocardial fibers during a given interval of time.13 In our study the duration of the QRS complex was equivalent in blacks and whites. According to the recent report by Mazzoleni et al,24 the high QRS voltages that we observed in our blacks may reflect an increase in myocardial mass. Mazzoleni et al24 demonstrated that increases in myocardial mass that are not associated with fibrosis produce higher QRS voltages without affecting the duration of the QRS complex. In considering this hypothesis, it should also be noted that constitutional variables which were not controlled factors in our study influence the direction and amplitudes of QRS voltages; for example, body weight correlates with the position of the frontal-plane QRS axis. There were no significant differences in the direction of the frontal-plane QRS axis between our racial groups. Therefore, it is unlikely that body weight caused the racial differences in our electrocardiographic findings. Height is one of the most important factors correlating with R-wave and S-wave amplitudes of the standard ECG in the pediatric patient;25 however, the differences in body height of black and white adolescents are small26 and can hardly account for the large racial variations in QRS voltage which we observed. It is possible that the configuration of the chest contributed to the racial differences in R-wave amplitudes. The configuration of the chest is known to influence the QRS complex of the ECG in the adult.18

We found a significant racial difference in the pattern of repolarization of the adolescent; over the right and midprecordial leads (V1 through V4), biphasic or inverted T waves, as well as ST-segment elevations, occurred predominantly in blacks (Table 2). Such racial differences have previously been noted in adolescents4,5,10 and also in adults.17 It has been suggested that the "juvenile ST-T wave pattern" is a normal variant up to 30 years of age in blacks, while it is abnormal in white adults.17

In view of the racial variations of the "juvenile ST-T wave pattern" in adolescents, cautious interpretation of the ECG is indicated in black adolescents who are evaluated for participation in sports, who should not be excluded from enrollment in a competitive athletic program on the basis of an apparently "abnormal" ECG. It is particularly important in black teenagers to avoid electrocardiographic misinterpretations of myocardial disease, pericarditis, or myocardial ischemia, because ST-segment elevations and inverted T waves occur not infrequently in blacks and because precordial T waves are lower in blacks than in whites, as shown in Figure 3. Aside from using race-specific and sex-specific normal data for the pattern of the S-T segment and T wave in black adolescents, there are certain electrocardiographic features that help in the differential diagnosis between a normal variant and pericarditis and between a normal variant and myocardial ischemia. In pericarditis the ST-segment deviations occur predominantly in the limb leads, and the direction of the ST-segment vector is to the left of the T vector, according to Spodick.27 In the presence of inverted T waves, normalization of
“pathologic” T-wave changes with exercise, inspiration, or administration of propranolol or other drugs may be used to exclude ischemic heart disease.

The etiology of racial differences in the “juvenile ST-T wave pattern” has not been elucidated. If blacks were simply more prone to functional T-wave changes, black adolescents should have a different response to hyperventilation than white adolescents; however, Thomsen and Wasserburger28 found that hyperventilation induces precordial T-wave inversions in white as readily as in black adolescents who have upright T waves in the ECG at rest. Athletes have changes in the T vector in the absence of heart disease,19 but both black and white athletes were excluded from our study. It is possible that extracardiac factors contribute to the racial difference in the “juvenile ST-T wave pattern.” Lepeschkin1 recently postulated that the “juvenile T-wave changes” may be ascribed to changes in electrical conductivity near the heart, while others have previously speculated that these changes are related to postural changes of the heart within the thorax.6

Sexual Differences

Our findings support the recommendations of Strong et al9 that in adolescents the use of electrocardiographic standards sensitive to sex is indicated because the presence of left ventricular hypertrophy in female subjects may not be recognized if standards for male subjects or mixed standards are used. In our study, these differences in QRS voltage were most evident between black male and female subjects, while the differences were statistically less significant between white male and female subjects. Especially in the right and midprecordial leads, R-wave voltages were much higher in black male than female subjects. Significant differences were also noted between sexes in the pattern of repolarization; male subjects of either race had a higher incidence of precordial ST-segment elevations and negative or biphasic T waves than female subjects.

“Juvenile Pattern” vs “Adult Pattern”

Recent studies assessing trends with age in the ECG in adults report a consistent pattern of changes which was traced from the third decade in life to the sixth through ninth decades.13,14,18 With increasing age the R-wave amplitude decreases at a rate of approximately 3.5 percent per decade in women and 6.5 percent in men.13,14 Concomitantly, amplitudes of the ST-segment and T wave become smaller, and there is a lower incidence of nonpathologic T-wave inversions over the precordium, a trend with age which is also evident if the normal data on the ST-segment and T wave from this study are compared with those in the adult.17,18 To assess the dynamic changes in electrical activity of the heart related to age, one may estimate the QRS and T-wave voltages in the adolescent from figures given in the adult. A reasonably close approximation of R-wave voltages for adolescents may be calculated from the aforementioned percentages pertaining to the rate of change. Using mean R-wave amplitudes reported by Simonson18 for white male subjects, one may increase values for the third decade by 6.5 percent to obtain estimates for the R-wave amplitude in the second decade. The so predicted mean R-wave amplitude in lead V₆ is 12.8 mm, while we observed 12.3 mm in our study. Such predictions pertain to T-wave voltages as well. Thus, it appears that the “juvenile pattern” or “adolescent pattern” is not separate from the normal adult pattern. The electrocardiographic variations noted in adolescents in this and other studies8,19 are part of a continuum of changes starting in late childhood and progressing on to later life.

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