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# Significance of Primary T Wave Aberrations in the Electrocardiogram of Asymptomatic Young Men

## *Part I. Electrocardiographic data*

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ABSTRACT

The electrocardiogram (ECG) at rest and during orthostasis and exercise in 51 healthy men 18-19 years of age without history or symptoms of heart disease, but with T wave aberrations in the ECG (group T) were compared to the normal ECGs of 112 controls of the same age. These aberrations (which litterature suggests to be due to organic heart disease) consisted of either a notch in the T wave, especially in the midprecordial leads, that sometimes became inverted, or a low T wave without concomitant ST depression. The T wave aberrations at rest in group T were similar to what 25 % of the controls evidenced during orthostasis (group B). Both group T and group B had signs of increased sympathetic tone at rest with a higher heart rate and systolic blood pressure than did the subjects with normal ECG both at rest and during orthostasis. These T wave aberrations disappeared for the majority during exercise. Both group T and group B had prolonged QTc intervals. Group T had increased R wave amplitudes which did not correlate to the severity of the T wave aberration or to systolic blood pressure. Our opinion is that primary T wave aberrations in the majority of these young men were because of increased sympathetic tone.

## INTRODUCTION

A rather high frequency (0.5 - 11.9 %) of T wave aberrations in electrocardiograms (ECGs) that are "primary", i.e. not secondary to an abnormal depolarisation has been found in young men (5, 16, 19, 26, 31). The majority (Atterhög, J-H & Malmberg, P, unpublished) of these aberrations are generally low and flat but may become inverted, frequently in association with tachycardia and ST depression, and are seen (for example) in association with anxiety (24, 30) and fear (25) and may thus be explained by increased sympathetic tone. Other types of T wave aberrations which are not associated with ST depression can be either a notch in the T wave most often in the precordial

leads (which can make a more or less complete part of the T wave negative) or a flat T wave that makes a break in the trend of the T wave amplitude through the precordial leads. These T wave changes, which have been found to occur in 4.3 % of young men (Atterhög, J-H & Malmberg, P, unpublished) have sometimes been considered to be because of organic disorders of the heart (8, 23, 38). This type of T wave change has occasionally led to disablement of young persons without other signs or history of heart disease because of the difficulty in interpreting the findings (15, 27, 34, 43). Results from an earlier study (Atterhög, J-H & Malmberg, P, unpublished), however, also suggest that these types of T wave aberrations may originate from an increased sympathetic tone.

The aim of the present study was to further elucidate the significance of such primary T wave aberrations by comparing them with normal ECG reaction when sympathetic tone is increased by standing and to follow the changes in such T wave aberrations during and after dynamic exercise.

## MATERIAL

Ninety-eight percent of all Swedish men are subjected to an extensive medical examination when they are 18 - 19 years of age in connection with their military induction. A 12-lead ECG at rest is included. During a period of 10 months a sample of 51 conscripts having either a low T wave without ST depression, a more or less pronounced notch (defined below) in the T wave or a break in the trend of the T wave amplitude through CR4R to CR7 in their ECGs, were selected at an indiction center for further examination in the hospital the day after their induction. Subjects with juvenile T wave aberrations (T wave negativity in leads CR4R, CR1 and CR2) were not included. None had a history of heart disease and there were no abnormal findings in the physical examination, excluding their blood pressure recordings. The subjects were unaware of the ECG change and had never had any symptoms of discomfort from the chest region as far as they could remember.

During the same period a random sample of 112 conscripts were taken from those with normal ECGs and no history of heart disease to constitute a control group. The subjects in the two groups came from both rural and urban parts of the eastern central Sweden. The two groups were treated in the same manner and the subjects were not informed that the further examination was not a part of the routine. Disclosure of the T wave findings was not given until afterwards.

The subjects were divided into the following groups:

Group T: subjects with T wave aberrations at rest and during orthostasis, n = 51.

The normal ECG group (A+B), n = 112 which was subdivided into:

Group A: subjects with normal ECGs in the supine position and during orthostasis, n = 84.

Group B: subjects with normal ECGs in the supine position but T wave aberrations during orthostasis, n = 28.

## METHODS

The examination started one and a half to three hours after breakfast with a 12-lead ECG recorded in the supine position after 10 minutes of rest. An orthostatic test was performed including heart rate (HR), blood pressure (BP) and ECG after standardized standing with their heels and back of the head against a wall for 8 minutes. The exercise test was performed in the sitting position on an electrodynamically braked bicycle ergometer (EM 370-1, Siemens-Elema). The work load was increased by 50 W increments at six minute intervals until a HR of about 170 beats/min was reached (39, 46). The ECGs were recorded with an ink-jet electrocardiograph (Mingograph 81, Siemens-Elema) and included limb leads and CR leads at rest (CR4R, CR1,2,4,5,7) and the corresponding chest-head leads during exercise (18), The PR and QT intervals were measured in lead II and QT was corrected (QTc) for differences in HR pursuant to Bazett (6).

The T waves in leads II, CR4 and CR7 at rest and during orthostasis were coded regarding maximum positive amplitude (codes Z, A, B) and notches (codes O, K, L, M, N, P). Z denotes a T wave amplitude more than 10 per cent of the R wave amplitude and A - B denotes a decreasing level of T amplitude without ST depression (ST level -0.05 mV). O denotes a normal T wave form and the letters K - P denote an increasing amount of a T wave notch without ST depression. The code system will been described in detail later (Atterhög, J-H & Malmberg, P, unpublished). A notch was defined as a negatively directed aberration of a positive or isolectric T wave not commencing at the beginning of the wave. The findings were coded with combinations of an amplitude and a notch letter as seen in Figs 1-4. R denotes an ST depression of 0.05 mV or more in addition to a negative - positive type of T aberration. The coded findings have been ranked in order of severity so that the occurrence of a notch is regarded as being more severe than a flat T wave without a change in form. That makes ZK worse than AO but less than BO, and ZL worse than BO, and so on. HR was calculated from a 30 seconds' ECG recording.

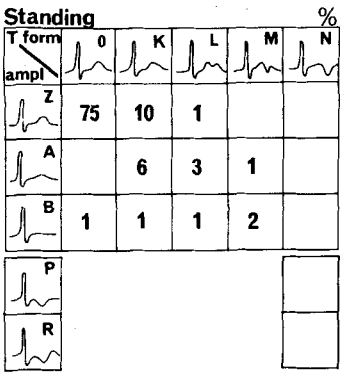


Fig. 1. The percentage freuqency of the most severe T wave aberration for each subject in either of leads II, CR4 or CR7 in the control group (n=112) during orthostasis.

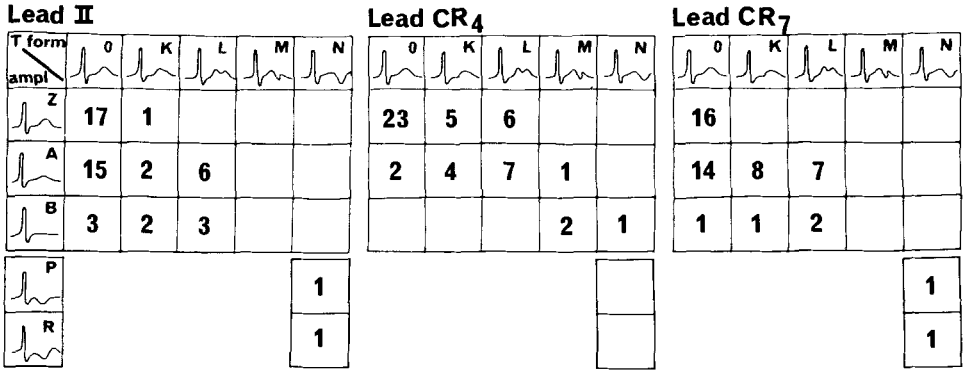


Fig. 2. Number of T wave aberrations in group T in leads II, CR4 and CR7 at rest.

"Early repolarization" syndrome (ERS) (34) was defined as an upwards concave RS-T segment elevation of 0.05 mV or more from the iso-electric baseline (i.e. the straight line between the ends of the PR intervals) in 2 leads or more, in connection with either a "slurred" downstroke of the R wave directly turning to the elevated ST segment or an "overshooting" of the upstroke of the S wave giving a small r' immediately preceding the elevated ST segment. BP was measured by ausculation with a calibrated cuff on the right upper arm by the same person throughout the whole study. The diastolic blood pressure (DBP) was read at phase 5 (disappearance of the sounds) and approximated to the nearest level of 5 mm Hg. In the case where this phase was not distinct, the DBP was read corresponding to phase 4 (muffling of the sounds).

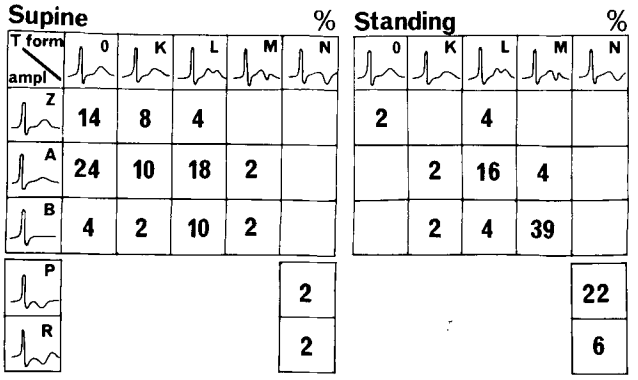


Fig. 3. The percentage frequency of the most severe T wave aberration for each subject in either of leads II, CR4 or CR7 in group T (n=51) at rest and during orthostasis.

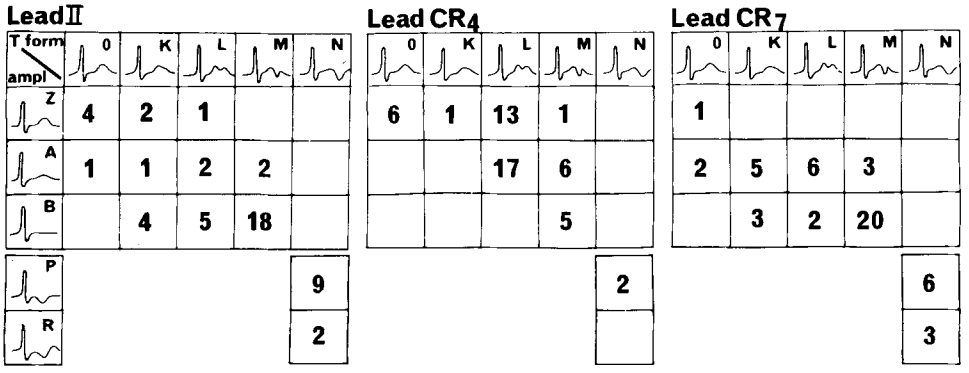


Fig. 4. Number of T wave aberrations in group T in leads II, CR4 and CR7.

The differences between group means were tested for significance using Student's t-test. The differences between frequencies were tested by Chi--square computed on 2 x 2 tables with Yate's correction. The relation between variables has been studied with linear regression analysis.

## RESULTS

Controls. ECG at rest and during orthostasis. In the control group A + B (n = 112 subjects) one subject had a pronounced respiratory sinus arrhythmia (HR variation 10 %) and one subject had a supraventricular ectopic beat on one occasion. Seven subjects had, either continuously or periodically, a

slight deviation from the normal on the mean P vector with a normal PR interval. The QRS complex was normal in all instances.

During orthostasis T wave aberrations appeared in 28 subjects (group B) i.e. 25 % of the controls (Fig 1). One subject ecidenced only a flat T wave without any form change. Eight subjects evidenced a notch that was 0.05 mV or more deep and three of them (i.e. 3 % of total control material) evidenced such a pronounced aberration as a negativity in the T wave.

Table 1. Distribution of the most severe T wave aberration in leads II, CR4 and CR7 calculated as a percentage for
a) the control group B during orthostasis (n = 28)
b) group T at rest (n = 51) and
c) group T during orthostasis (n = 51).

a) standing

| | II | CR4 | CR7 | % II CR4 CR7 |
|---|---|---|---|---|
| II | 64 | 7 | 11 | |
| CR4 | | 4 | 4 | |
| CR7 | | | 7 | |
| II, CR4, CR7 | | | | 4 |

b) supine

| | II | CR4 | CR7 | % II CR4 CR7 |
|---|---|---|---|---|
| II | 24 | | 15 | |
| CR4 | | 39 | 2 | |
| CR7 | | | 15 | |
| II, CR4, CR7 | | | | 4 |

c) standing

| | II | CR4 | CR7 | % II CR4 CR7 |
|---|---|---|---|---|
| II | 27 | | 31 | |
| CR4 | | 22 | 4 | |
| CR7 | | | 12 | |
| II, CR4, CR7 | | | | 4 |

Lead II was, in 86 % of the subjects, the place for the most severe T wave aberration, alone or in combination with other leads (Table 1a). The T wave aberration in lead CR4 was the most severe in only 12 % of the subjects .

Group B had significantly higher HR and SBP at rest than group A (Table 2). During orthostasis there was a larger increase in HR in group B than in group A whereas the differences in SBP and DBP were similar in the two groups.

ERS was found in 50 % of the subjects in group A and in 61 % in group B. This difference was not significant. Mean HR in the group A subjects with ERS was 60.5 beats/min (SD 9.8), which was significantly lower than the averaged HR of 65.3 beats/min (SD 10.0) in the subjects without ERS (p 0.05). In group B the subjects with ERS, had on the average, a 1.1 beats/min. higher HR than those without ERS. There was no significant difference between group A and group B regarding the PR intervals (Table 2).

The QT interval was shorter in group B than in group A but after correction for differences in HR the QTc interval became longer in group B.

Group T (n = 51). ECG at rest. Five subjects had normal ECGs during the examination the day after their induction. Two further subjects had a lower T wave in CR4 than in CR2 and CR5 but this aberration was not large enough to fulfill any code criteria. Twenty-nine subjects were studied as to how their T wave aberrations changed in severity between the examination at the conduction center and the present examination and the results were related to HR. The

aberrations were unchanged for 7 subjects and the HR for those decreased on the average by 3 beats/min. In 5 subjects the aberrations increased in severity with a mean HR increase of 8 beats/min. wheras the 17 subjects with a decrease in the aberration severity decreased by 9 beats/min. on the average.

Table 2. HR, SBP and DBP at rest and during orthostasis and QT interval before and after correction for HR (QTc) and PR interval for the control group without (A) and with (B) T wave aberrations during orthostasis, for the total control group (A+B) and for the group with T wave aberrations at rest and during orthostasis (group T).

| | Group A n=84 | | Group B n=28 | | Group A+B n=112 | | Group T n=51 | |
|---|---|---|---|---|---|---|---|---|
| | mean | SD | mean | SD | mean | SD | mean | SD |
| HR, supine, beats/min | 63.0 | 10.4 | 73.4 | 11.1 | 65.6 | 11.5 | 75.2 | 15.2 |
| sign. of diff. | | $p<0.001$ | | | | $p<0.001$ | | |
| SBP, supine mm Hg | 124.3 | 11.8 | 130.0 | 9.9 | 125.7 | 11.6 | 136.6 | 13.5 |
| sign. of diff. | | $p<0.05$ | | | | $p<0.001$ | | |
| DBP. supine mm Hg | 73.2 | 7.7 | 76.4 | 8.4 | 74.0 | 8.0 | 78.3 | 6.9 |
| sign. of diff. | | n.s. | | | | n.s. | | |
| HR diff. erect-supine beats/min | 17.7 | 7.7 | 23.2 | 11.1 | 19.1 | 8.9 | 18.3 | 11.6 n=50 |
| sign. of diff. | | $p<0.01$ | | | | n.s. | | |
| SBP diff. erect-supine mm Hg | 0.0 | 6.7 | -1.6 | 6.7 | -0.4 | 6.7 | -2.9 | 7.3 n=50 |
| sign. of diff. | | n.s. | | | | $p<0.05$ | | |
| DBP diff. erect-supine mm Hg | 7.4 | 7.7 | 6.4 | 6.5 | 7.2 | 7.4 | 4.1 | 5.7 n=50 |
| sign. of diff. | | n.s. | | | | $p<0.01$ | | |
| QT, s | 0.37 | 0.02 | 0.36 | 0.02 | 0.37 | 0.03 | 0.36 | 0.03 |
| sign. of diff. | | $p<0.05$ | | | | $p<0.001$ | | |
| QTc, s | 0.38 | 0.02 | 0.40 | 0.03 | 0.38 | 0.02 | 0.39 | 0.03 |
| sign. of diff. | | $p<0.05$ | | | | $p<0.05$ | | |
| PR, cs | 15.5 | 1.9 | 15.0 | 1.8 | 15.4 | 1.9 | 14.4 | 1.7 |
| sign. of diff. | | | | | | $p<0.01$ | | |

One subject had frequent supraventricular ectopic beats and sometimes 2 in series. Two subjects had periods of supraventricular ectopic rhythm and one had a wandering atrial pacemaker focus. Two subjects had occasional ventricular ectopic beats. The QRS was evaluated normal for all subjects.

The number and types of T wave aberrations in leads II, CR4 and CR7 are described in Fig. 2. A flat T wave without a notch was the most common finding in leads II and CR7 while a suggested or definite notch with even a negative

segment constituted the majority of T wave changes in CR4. The frequency of the most severe T wave aberration in either of leads II, CR4 and CR7 for each subject is shown in Fig. 3. Fourteen per cent of group T had either normal ECGs or non-codable T wave aberrations at rest during the hospital examination while 28 % of the subjects had a flat T wave without any change in the form as their most severe T wave aberration.

Forty per cent of the subjects had a notch in their T waves of more than 0.05 mV, and for 8 % this caused a negativity, partly or totally, of the T wave. In only one subject (i.e. 2 % of the material) the T wave aberration was combined with a ST depression of 0.05 mV or more.

In contrast to the T wave aberrations that occurred during orthostasis in group B the most severe T wave finding in group T occurred most often in the precordial leads (76 %), especially in CR4 (45 %, Table 1b).

Early repolarisation syndrome. An elevation of the ST segment was found to a high extent in group T (72 %) but the criteria for ERS were fulfilled only by 25 % of the subjects compared with 53 % in group A + B. This difference was significant ($p < 0.001$). The lower frequency in group T depended on a generally very minor elevation of the ST segment. Thus, the frequency of subjects with changes in the final part of the QRS wave associated with ERS (see methods) were similar in group T and group A + B (75 and 62 % respectively). Mean HR in group T subjects with ERS was 4.8 beats/min. lower than in subjects without ERS but this difference was not significant.

PR and QT intervals (Table 2). The PR interval was on the average shorter in group T than in group A + B. The QT interval was also shorter in group T, but after correction for the differences in HR the QTc became more prolonged than for group A + B. There were no differences in QT or QTc intervals in group T compared to group B alone.

R amplitudes. The R amplitudes in leads I, II, aVL, aVF, CR5 and CR7 in the ECG at rest were significantly higher in group T than in the control group (Table 3).

Table 3. Mean R and S amplitudes and SD in some ECG leads in groups A+B and T and significance of differences between group means.

| | | R amplitude, mV | | | | | | | S ampl. mV CR1 | $S_{CR1}+R_{CR5}$ |
|---|---|---|---|---|---|---|---|---|---|---|
| | | I | II | III | aVL | aVF | CR5 | CR7 | | |
| Group A+B | mean | 0.64 | 1.50 | 1.05 | 0.24 | 1.27 | 2.74 | 1.97 | 1.03 | 3.78 |
| | SD | 0.26 | 0.41 | 0.55 | 0.20 | 0.49 | 0.74 | 0.37 | 0.52 | 0.88 |
| Group T | mean | 0.83 | 1.78 | 1.18 | 0.28 | 1.48 | 3.38 | 2.36 | 1.05 | 4.43 |
| | SD | 0.29 | 0.44 | 0.54 | 0.19 | 0.47 | 0.79 | 0.48 | 0.46 | 0.84 |
| Sign. of diff. | | p< 0.001 | 0.001 | n.s. | n.s. | 0.05 | 0.001 | 0.001 | n.s. | 0.001 |

The mean R amplitude in CR5, and the mean sum of R in lead CR5 and S in CR1, was higher in group T than the criteria for high amplitude R waves coded 3.1 in the Minnesota code modified for CR leads (56). There were significantly more subjects in group T that fulfilled these amplitude criteria in leads II, CR5 and CR1 + CR5 than there were in group A + B (Table 4). The total percentage of subjects with codes of high R wave amplitudes in the control group was 40.7 % and in group T was 72.6 % ($p < 0.001$). The R waves in group B were somewhat higher than in group A but these differences were not significant in any leads.

Table 4. Percentage of the groups A+B and T that fullfilled the amplitude criteria in code 3.1 ("High amplitude R waves") in a "Minnesota code" modified for CR leads (56) and significance of differences between the groups.

| | I | II | III | aVL | aVF | CR5 | CR7 | $S_{CR1}$+ | Any ampl |
|---|---|---|---|---|---|---|---|---|---|
| | R | R | R | R | R | R | R | $R_{CR5}$ | item |
| | 2.0 mV | 2.0 mV | 2.0 mV | 1.2 mV | 2.0 mV | 3.3 mV | 3.3 mV | 4.0 mV | |
| Group A+B, % | 0 | 9.7 | 6.2 | 0 | 6.2 | 16.8 | 0 | 32.7 | 40.7 |
| Group T, % | 0 | 31.4 | 7.7 | 0 | 17.6 | 54.9 | 3.9 | 64.7 | 72.6 |
| Sign. of diff. p< | | 0.01 | n.s. | | 0.10 | 0.001 | n.s. | 0.001 | 0.001 |

ECG during orthostasis. All the T wave changes became more pronounced during orthostasis (Fig. 3). There was no subject without a T wave aberration. The one subject coded ZO had a lower T in CR4 than in the neighboring leads. Excluding him, all the subjects evidenced a notch as a part of the most severe T wave aberration which for 92 % of the subjects were deeper than 0.05 mV. For 71 % a T wave negativity occurred that was total in 22 % and concomitant to a ST depression of 0.05 mV or more in 6 %. The number and type of these T wave aberration in leads II, CR4 and CR7 are shown in Fig. 4. The most severe T wave aberrations which appeared during orthostasis were to a greater extent located in lead II than they were when the subject was in the supine position (Table 1c). Sixty-two per cent of the subjects had their dominant T wave aberration in lead II while 30 % had their most severe T wave aberration in CR4.

Exercise. All the subjects with ectopic beats or supraventricular rhythm at rest in control group (A + B) and group T had a steady sinus rhythm during exercise. One subject in group T evidenced occasional supraventricular beats while another evidenced ventricular ectopic beats. All subjects in group A + B had normal ECG reactions during and after exercise. The T wave changes in group B during orthostasis disappeared and did not return at rest in recumbent position after exercise.

For the majority of subjects in group T the T wave aberrations were accentuated during exercise on low loads but decreased on the highest load (mean HR 169.2, SD 7.2) (Table 5). In a recumbent position immediately after exercise the ECG was normal in 67 % of group T and in only 8 % the T was their wave change more pronounced after exercise than before.

Table 5. Reaction of the T wave aberrations during and immediately after exercise in the recumbent position in 51 subjects with T wave aberrations at rest.

| Reaction | Change of the T wave finding at a low load n | Change of the T wave x finding at highest load n | Change of the T wave finding immediately after exercise n |
|---|---|---|---|
| Impairment | 29 | 14 | 4 |
| Unchanged | 17 | 8 | 5 |
| Partly regress | 4 | 17 | 7 |
| Normalization | 1 | 12 | 35 |

The ERS which occurred at a high frequency at rest disappeared during exercise. After exercise there was a gradual return of the whole ST-T interval to the pre-exercise ECG for all the subjects but one. For the majority the ECG changes were completed within 10 minutes of rest. In three cases there was a ST-depression during and after exercise which was evaluated as being be more pronounced than normal. One of those was of a coronary insufficiency type (12) which occurred in a man with high R wave and flat T wave at rest. He had already developed an ST-T depression at low loads and the change had not returned after four minutes rest. He fulfilled the complete test, however, without symptoms, and his work capacity was normal. Blood pressure at rest was 150/90 mm Hg and HR 82 beats/min.

All subjects carried out the exercise without complaining of pain, discomfort or dyspnea. Fatigue on completion of exercise, especially in the legs, appeared frequently however. In no instance was hyperventilation noted at rest, or during or after exercise.

HR and BP and the relations between these variables and the ECG findings. HR, SBP and DBP at rest were higher in group T than in the controls (Table 2). During orthostasis the two groups T and A + B had a similar increase in HR, whereas SBP decreased more, and DBP increased less, in group T. One subject fainted in group T.

In Table 6 mean HR at rest and during orthostasis and mean SBP at rest have been calculated for the most severe T wave aberrations. HR increased with severity of amplitude and form change at rest but the differences were in no instance significant. There was no connection found between SBP and the severity of the T wave aberration. The severity of the T wave abnormality during

orthostasis seemed not to be connected with the degree of tachycardia as they all had about the same HR.

Table 6. HR at rest and during orthostasis and SBP at rest in the different T wave amplitudes (Z, A, B) and severity of notches (O, K, L, M) for the most severe T wave aberration in either of leads II, CR4 and CR7. The most severe T wave aberration for each subject in either of leads II, CR4 and CR7 were coded both regarding amplitude (Z, A, B) and severity of notches (O, K, L, M). The number of these codes (n), HR at rest and during orthostasis and SBP at rest are given for the different codes.

| | At rest | | | | | Standing | |
|---|---|---|---|---|---|---|---|
| | | HR beats/min. | | SBP mm HG | | | HR beats/min. |
| T wave code | n | mean | SD | mean | SD | n | mean |
| Z | 13 | 69 | 14 | 132 | 8 | 3 | 92 |
| A | 27 | 74 | 15 | 129 | 15 | 8 | 94 |
| B | 10 | 81 | 16 | 135 | 14 | 25 | 92 |
| O | 21 | 69 | 13 | 136 | 15 | - | |
| K | 9 | 78 | 19 | 134 | 15 | 3 | 99 |
| L | 17 | 76 | 12 | 137 | 10 | 11 | 94 |
| M | 2 | 86 | 1 | 138 | 11 | 21 | 93 |
| P | 1 | 94 | | 140 | | 10 | 96 |
| R | 1 | 110 | | 155 | | 4 | 108 |

Mean R wave amplitude in leads II and CR7 has been calculated for the different T wave aberrations in these two leads respectively and also for the most severe T wave aberration in either of leads II, CR4 and CR7 (Table 7).

There is a significant increase of R amplitude with a decrease of T wave amplitude from code Z to A if the R amplitude is measured in the same lead as the T wave aberration as well as if the $R_{II}$ and $R_{CR7}$ amplitudes are compared to the most severe T wave aberrations in either of leads II, CR4 and CR7. There was no significant correlation, however, between R-amplitude and magnitude of a T notch if either the R amplitude is measured in the same lead as the notch or is compared to the maximum T wave aberration in either of leads II, CR4 and CR7.

Linear regression analysis was performed on the R amplitude as a function of SBP or HR but no correlations were found either in group T or in the control groups and there was no increase in correlation after correction for body height. No significant correlation was found between PR interval and HR in any of the groups (r=0.10).

Table 7. Mean R amplitude in leads II and CR7 for the different T wave aberrations in leads II and CR7 respectively and mean $R_{II}$ and $R_{CR7}$ amplitude for the most severe T wave aberration in either of leads II, CR4 and CR7 are given. The T wave aberrations were coded as in Table 6.

| T wave aberration code | T wave aberration in lead II | | T wave aberration in lead CR7 | | Most severe T wave aberration in either of leads II, CR4, CR7 | |
|---|---|---|---|---|---|---|
| | n | $R_{II}$ ampl, mV mean | n | $R_{CR7}$ ampl, mV mean | n | $R_{II}$ ampl, mV mean | $R_{CR7}$ ampl, mV mean |
| Z | 18 | 1.51 | 16 | 2.21 | 13 | 1.57 | 2.04 |
| A | 23 | 2.04 | 29 | 2.50 | 27 | 1.91 | 2.54 |
| B | 8 | 1.64 | 4 | 2.08 | 10 | 1.67 | 2.33 |
| sign. of diff. betw. the codes | Z-A p 0.001<br>Z-(A+B) p 0.001 | | Z-A p 0.05<br>Z-(A+B) n.s. | | Z-A p 0.01<br>Z-(A+B) p 0.05 | | Z-A p 0.001<br>Z-(A+B) p 0.01 |
| O | 35 | 1.73 | 31 | 2.30 | 21 | 1.75 | 2.35 |
| K | 5 | 1.90 | 9 | 2.52 | 9 | 1.79 | 2.31 |
| L | 9 | 1.91 | 9 | 2.46 | 17 | 1.80 | 2.44 |
| M | | | | | 2 | 1.78 | 2.30 |
| P | 1 | 1.70 | 1 | 2.30 | 1 | 1.70 | 2.30 |
| sign. of diff. betw. the codes | O-K n.s.<br>O-(K+L) n.s. | | O-K n.s.<br>O-(K+L) n.s. | | O-(K+L) n.s.<br>O-(K+L+M) n.s. | | O-(K+L) n.s.<br>O-(K+L+M) n.s. |

## DISCUSSION

An aberration of the T wave in an ECG indicates an altered repolarisation course in the heart muscle and there are a great number of cardiac and extracardiac conditions that can cause such changes (23). The subjects in group T in this study were selected, therefore, in respect to the occurrence of such T wave aberrations that are supposed to indicate organic heart disease (8, 23, 38).

It is very likely that there is more than one cause of T wave aberrations represented in this material, but since there is no reliable method to differentiate them this discussion has to constitute a common picture represented by group T.

The subjects with T wave aberrations at rest had no history of symptoms of heart disease so the ECG findings were quite accidental. Moreover, there was no increased number of concomitant ECG findings in group T that might give evidence of heart disease. Since it is known that food (37) and hyperventilation (45) may provoke T wave abnormalities, the examinations were performed more than one hour after light breakfast and it was checked that the subjects did not hyperventilate.

The T wave aberrations were found to change in severity from one day to the other and this is a common finding in young men (Atterhög, J-H & Malmberg, P, unpublished). This variability indicates an association between T wave aberrations and vegetative tone but does not necessarily imply the whole explanation

of the aberrations since organic disease may still be present nonetheless. Adrenergic stimulation can accentuate ST and T changes which are also of organic origin (18) and thereby contribute to a variability of "organic" ECG changes. An increase in sympathetic tone by orthostasis may provoke ECG changes in patients with myocarditis even if the ECG is normal at rest (9). On the other hand, the present study shows that the occurrence of what has been supposed to be "organic" T wave changes during orthostasis does not necessarily imply a heart disease. Twenty-five per cent of the controls evidenced such T wave aberrations during orthostasis and it is not very likely that so high a proportion of "normals" has had undetected heart disease. It is more likely that the T wave aberrations found in group B were related to the higher sympathetic tone which characterized these subjects as indicated by a higher HR and SBP at rest and a more accentuated increase in HR during orthostasis.

Group T showed T wave aberrations at rest which were similar to those found in group B during orthostasis. Furthermore, group T was characterized by a HR and SBP which was somewhat higher than in group B, implying a high sympathetic tone. It must, however, be emphasized that the mean differences in HR and SBP between group T and the controls were quite small, although significant. This is in accordance with earlier findings in a prevalence study on T wave aberrations (Atterhög, J-H & Malmberg, P, unpublished) and also shows that T wave aberrations of "organic" types can be explained by an increased sympathetic tone. Taggart and his coworkers have also examined subjects similar to our group T and found that T wave aberrations in at least II, III, aVF and left precordial leads may be related to increased sympathetic discharge or hypersensitivity to catecholamines, and are not necessarily connected with heart disorders (43).

Similar T wave changes as in this study have been reported in vasoregulatory asthenia (VA) (17). That disorder is regarded as being connected with disturbed sympathetic tone since training (17) and sympatholytics (2) decrease both ECG changes and symptoms in this syndrome. No subject in this study suffered from VA as this disorder, with its concomitant ECG changes, is characterised by chest symptoms like palpitations and a low physical work capacity in relation to heart volume and total haemoglobin as well as by a pronounced HR reaction in response to orthostasis. This picture was not evidenced by any subject in group T.

Our exercise test excluded coronary insufficiency as a cause of the T wave aberrations, with the possible exception of one subject. That disorder is indeed rare in this age but does occur (20, 40). A normalisation of the T wave during exercise is an unspecific ECG reaction that may also occur after a cardiac infarction for example (3) and gives no clue regarding the genesis to the T wave aberrations. Aberrations like those presented in this study have

been shown to react in a diverse manner with some abberations increasing and others even normalizing on infusions of adrenaline (43). As a possible explanation to the diverse reaction it was suggested that the abberations followed a bimodal course of reaction on increasing sympathetic discharge (43). Normal T waves react in a bimodal way to the beta-receptor stimulating drug isopenaline with first as appearance of an aberration and then a normalozation (47). It has been supposed that rapidly reversible T wave abnormalities probably resulted from some regional deficiency of sympathetic stimulation (47). Thus a possible explanation for the abolition of T wave aberrations during exercise is that the induced increase in sympathetic discharge equalizes any asynchronous repolarisation rates in different parts of the heart and thereby normalizes the T wave.

An asynchronous repolarisation might also explain the prolonged QTc in group T. Group T had a shorter QT interval before correction for differences in HR, but QTc was prolonged when compared to the controls. This is common in various disorders with myocardial damage (22). Since group B, which like group T, also had a high HR had a prolonged QTc interval it is more likely that either the HR correction of QT according to Bazett is not applicable to this study or that the long QTc in the two groups was because of an increased sympathetic tone which under certain circumstances may induce a prolongation (22). One reason may be that sympathetic tone is nonuniformly distributed, leading to asynchronous repolarisation and T wave abnormalities. Thus, similar T wave aberrations as in this study have been elicited in dogs by selective stimulation or ablation of the sympathetic nerves to the heart and a prolongation of QTc could be observed when the left ganglion stellatum was blocked (48). These induced changes also abate on administration of isoprenaline (21). Furthermore, the T wave abnormalities and QTc prolongations found in patients with lesions in the central nervous system have been suggested to be due to altered autonomic tone (1) and the appearing T wave aberrations may be quite similar to the T wave changes in this study (28).

In addition, the occurrence of ERS has been suggested to be due to regional differences in sympathetic tone, namely an enhanced activity of the right sympathetic nerves to the heart (29). The lower frequency of ERS in group T than in the controls in association with a higher HR does not support this suggestion, however. The frequency of ERS increases with the vagal tone (36), and as the subjects with ERS in this study had a tendency to low HR the lower frequency of ERS in group T might indicate that subjects with T wave aberrations also have a lower parasympathetic tone in addition to a higher sympathetic tone. The 53 % of subjects with ERS in group A + B in this study exceeds by far the prevalence of 2 % reported earlier (32). This difference can probably be explained firstly by our use of CR leads, which give somewhat higher

amplitudes in the left precordial leads than do V leads; and secondly by the high frequency response in our registration system. Differences in criteria and materials may also have contributed.

No correlation was found in any of the groups in this study between the PR interval and HR which might have helped to explain the shorter interval in group T. Conflicting population study reports have been published on this topic with evidence both pro (11, 22) and con (4, 7, 44) relative to a correlation between PR interval and HR. The decrease in PR interval that occurs when the HR is increased by exercise is mainly related to a decline of the vagal tone (4), however, and thus a decreased parasympathetic tone might also be indicated by the shorter PR interval in group T.

Forty-seven percent of the control subjects in this study fulfilled the criteria for high R wave amplitudes (49), and this is in accordance with earlier findings that adolescents in a high frequency have R wave amplitudes accepted for left ventricular hypertrophy in adults (41). These R amplitudes were still higher in group T, and this cannot be explained by differences in age, height or weight (33) as group T was on the average shorter (which would decrease the R waves) and had the same weight and age. Since the R wave amplitude in part reflects the muscle mass of the left ventricle, the increased amplitudes in group T (especially in connection with T wave abnormalities) might indicate an incipient hypertrophy (10). This is probably not because of the higher BP in group T, as the average BP level was only moderately higher in this group than in the controls. Recent studies on spontaneously hypertensive rats, however, have shown that an increase in left ventricular weight may appear before arterial pressure has reached hypertensive levels and, among possible mechanisms, adrenergic factors have been brought up (13, 47). In young patients with borderline hypertension, asymmetric septal hypertrophy and signs of increased sympathetic drive have been found (35). Although no correlations were found either between the R amplitudes on the one hand and SBP, HR or the severity of the T wave aberrations on the other hand, nor between the severity of the T wave aberrations and SBP, it cannot be excluded that incipient heart hypertrophy may occur in group T. The significance of the high R amplitudes can only be evaluated by a prospective study.

In conclusion, it was found that asymptomatic young men with T wave aberrations in a pattern suggestive of organic heart disease (according to the litterature) show signs of increased sympathetic tone and give no evidence of organic heart disease. As this type of T wave aberration can also be found in conditions with high sympathetic discharge we find it likely that "organic" T wave aberrations in the majority of young men are a repolarisation disorder due to increased, and possibly nonuniform, sympathetic influence on the heart.

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