Gradual Changes of ECG Waveform During and After Exercise in Normal Subjects

By M. L. Simoons, M.D., and P. G. Hugenholtz, M.D.

SUMMARY
The directions and magnitudes of time-normalized P, QRS, and ST vectors, and other ECG parameters were analyzed during and after multistage exercise in 56 ostensibly healthy men aged 23 to 62. By selective averaging with a digital computer system a single representative beat was obtained from each stage. Measurements were taken from this beat. During exercise, the interval between the spatial maximum of the P wave and the onset of the QRS complex decreased while the magnitude of the P wave increased. The direction of the P vectors did not change. This pattern corresponds to the electrocardiographic manifestations of predominant right atrial overload. No significant changes in the QRS duration were observed. Also the magnitude and spatial orientation of the maximum QRS vectors remained constant. The interval between the QRS onset and the maximum spatial magnitude of the T wave shortened. The terminal QRS vectors and the ST vectors gradually shifted toward the right, and superiorly. The T magnitude lessened during exercise. In the first minute of the recovery period the P and T magnitudes markedly increased. Afterward all measurements gradually returned to the resting level.

Mechanisms which may explain the observed ECG changes during and after exercise are discussed, including changes in the blood conductivity and intracardiac blood volume. None did not contribute to the variance of the ECG measurements, but a significant reduction of this variance could be obtained in some ST-segment measurements by relating them to heart rate with linear regression equations (P < 0.05). Therefore it is expected that the sensitivity of the exercise ECG for detection of ischemic heart disease would be increased when heart rate dependent normal limits for ST-segment measurements are used. Different criteria should be employed for the interpretation of the ECG during and after exercise.

Changes in the Electrocardiogram (ECG) during exercise in normal subjects were described by Simson in 1953. He observed decreased R wave amplitude and right axis deviation as well as junctional depression of the ST segment and decreased T wave amplitude. Sjöstrand showed that the depression of the QRS-ST junction during exercise in normal subjects is related to heart rate. Later, Irisawa demonstrated a marked increase of the P wave amplitude during exercise. These findings have been confirmed in recent years by quantitative ECG analysis with modern computer techniques. Blomqvist and Bruce et al. demonstrated that such ECG changes occur gradually when a multistage exercise test is performed. In the recovery period, these changes reverse, although the relation between ST-segment amplitude and heart rate in the recovery period seems to follow a different pattern than during exercise.

When a corrected orthogonal lead system is employed, the changes in the magnitude and in the direction of the heart vector can be separated. Results from one such study indicate that after exercise changes only in the magnitudes of QRS and T occur, while the directions of these vectors remain constant. On the other hand, Rautaharju et al. found changes in both magnitudes and directions of Chebychev waveform vectors of the P wave and the ST-T segment during submaximal exercise.

Although changes in various ECG measurements during or after exercise are indicated by these reports, none of the authors presents a complete description of the entire ECG at all levels during a multistage exercise test and after exercise in normal subjects. In addition little information is available on the mechanisms which cause these ECG changes.

The present study has been designed to provide a quantitative description of the entire ECG from rest until maximal exercise and in the recovery period. Both ECG measurements at time-normalized intervals of the P, QRS, and ST segments and at fixed intervals after the end of the QRS complex as well as time integrals of the negative parts of the ST segments were analyzed. These results show gradual changes in the P, QRS, and T wave which can be related to heart rate. However, the relation between many ECG measurements and heart rate in the recovery period differed significantly from the same relation during exercise.

On the basis of these observations it is postulated...
that correction of ECG measurements for heart rate
determined from the same record will considerably in-
crease the diagnostic power of exercise ECGs, while
further analysis of changes in vector magnitudes may
be of use in the assessment of the hemodynamic func-
tion of the heart as a pump.

Materials and Methods

Orthogonal electrocardiograms at rest, during exercise,
and recovery were analyzed in 56 ostensibly healthy men.
Thirty-five of these were studied at the Occupational
Health Center of Rotterdam Harbour.13 The other 21 recordings
were obtained in cooperation with the KRIS.* All subjects
were selected for the present study because they had no history
of cardiovascular disease, no chest pain, blood
pressure lower than 160/95, fasting serum cholesterol below
6.7 mM/L, blood sugar below 10 mM/L one hour after in-
gestion of 75 grams glucose, and a normal ECG at rest. The
age distribution is shown in table 1.

Exercise was performed on a calibrated bicycle ergometer.
The workload was increased in a stepwise manner
with 10 W/min (KRIS) or 30 W/3 min, starting at 15 W
(Harbour). The subjects were encouraged to exercise to their
maximum. The tests at the Harbour Occupational Health
Service were terminated in 20 subjects when the exercise
stage of 165 W was completed. The ECG was recorded on
analog tape.† A modified Frank lead system was used. Chest
electrodes were placed at the level of the 5th intercostal
space in the upright position. The H electrode was placed in
the neck, the F electrode at the sacrum. The ECGs were
processed off-line with a PDP-8E1 computer system. Twenty
second periods were sampled at a rate of 500/sec at rest in
a sitting position, every second or third minute during exercise,
and the first, third, sixth, and ninth minute of the
recovery period.

A total of 642 ECGs was processed. A single representat-
tive beat with a low noise level was obtained from each record
by averaging of all detected beats, except those with an
aberrant waveform. The onset and end of the different
waveforms were identified by a program developed especially
for waveform analysis in serial ECGs.† The waveform analysis
was checked visually in all ECGs. In 69 records (11%) P
wave detection proved to be incorrect. On the other hand,
QRS errors were not found. The maximum of the T wave
was detected erroneously in only one record. The incorrect
records were not used for further analysis. The mean
amplitude 20 to 10 msec before the onset of the QRS com-
xplex was used as a zero reference level for all measurements.
The following types of measurements were taken: (table 2):

1) Intervals from the maximum spatial magnitude of the
P wave until the onset of the QRS complex (PPK-Q); from
the onset to the end of the QRS complex; and from
the onset of the QRS complex to the maximum spatial
magnitude of the T wave (Q-TPK). The conven-
tionally measured P-Q and Q-T intervals were not
chosen because, in most records, at heart rates over
120 beats per minute, the P wave is superimposed on
the preceding T wave. In those records the true onset

*Kaunas Rotterdam Intervention Study; a project of the World
Health Organization.
†Totenite Inc., USA.
†Digital Equipment Corporation, USA.

Table 1

<table>
<thead>
<tr>
<th>Age Distribution of Study Population</th>
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<tbody>
<tr>
<td>N</td>
</tr>
<tr>
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<tr>
<td>2</td>
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</tbody>
</table>

of the P wave and end of the T wave could not be
detected.

2) Amplitudes at eight time normalized intervals of the
PPK-Q segment, the QRS complex, and the S-TPK

3) Amplitudes at fixed intervals after the end of the QRS
complex.5,6

4) The area of the negative part of the ST segment in
orthogonal leads X and Y.9

These measurements were plotted against heart rate, and
linear regression analysis was performed for rest and exercise
records, and for the recovery records. The relative influences
of heart rate and age were analyzed with linear multiple
regression analysis. Paired and unpaired Student's t-tests
were used to compare the differences of various parameters
at selected heart rate intervals.

Results

In figure 1 the PPK-Q, QRS, and Q-TPK intervals
have been plotted against heart rate. The means of the
time-normalized ECGs at rest and during exercise at a
heart rate of 160-180 beats per minute are presented in
figure 2.

P Wave Changes

The PPK-Q interval shortened from 108 ± 16 msec
(mean and standard deviation) at a heart rate of 70
beats/min at rest to 72 ± 10 msec during exercise at a
heart rate of 170 beats per minute (fig. 1). In the recovery
period the PPK-Q interval lengthened again,
with a similar linear relation toward heart rate. The
spatial orientation of the P vectors was not altered
significantly (fig. 2). The spatial magnitude of the P
wave increased from 0.122 ± 0.035 mV at a heart rate
of 70 to 0.221 ± 0.046 mV at 170 beats per minute. In
figure 3 the maximum P vectors have been plotted
against heart rate. These plots show that the amplitude
changes occurred gradually when the heart rate
increased. In the first minute of the recovery
period a further augmentation of the P wave was
observed in all three leads although the heart rate
decreased. Therefore the spatial magnitude of the P
wave in the recovery period was higher than during exercise at corresponding heart rates.

Changes in the QRS Complex

No significant change was observed during or after
exercise in the QRS duration. However, the duration
of the S wave in leads X and Y increased from a mean
of 14 (range 0-52), and 18 msec (0-50) respectively at
a heart rate of 70 to 21 (10-52) and 24 (0-52) msec at a
The magnitude of the T wave decreased gradually from 516 ± 97 µV at rest to 415 ± 176 µV at maximum exercise (P < 0.01). Its spatial orientation did not change. In the first minute of the recovery period the T wave spatial magnitude increased markedly to 629 ± 154 µV (P < 0.01). The differences between the time normalized vectors at rest and during exercise have been plotted in figure 5. This figure shows that the direction of the changes was independent of heart rate. The magnitude of the changes gradually increased during exercise. The changes of the ST vectors were similar in direction to the changes of the terminal QRS vectors. The whole ST segment as well as the T wave in leads X and Y was shifted downward during exercise.

Regression Analysis

The results of the linear regression analysis of 32 parameters at rest and during exercise with heart rate as the independent variable are summarized in table 3. A significant (P ≤ 0.05) reduction of the standard errors of the amplitudes ST-1, ST-2, ST-3, ST-30 and the standard errors of the negative ST integrals in X and Y were obtained when adjustment for heart rate was made. Predictions which adjusted for both heart rate and age by linear multiple regression analysis did not yield better results than prediction from heart rate alone. Regression analysis with heart rate expressed as a fraction of the maximal heart rate, or with workload as the independent variable, did not reduce the standard errors.

Discussion

Comparison with Other Studies

The results of the current study confirm and extend earlier observations of changes of various ECG measurements during and after exercise in normal individuals. However, these studies1-12 each describe a small number of ECG measurements. A systematic analysis of the entire ECG waveform was performed in the present investigation.

The spatial magnitude of the P wave increased gradually during exercise, while the PPK-Q interval shortened. This is in agreement with earlier findings during6, 4, 12 and after11 exercise. However, Irisawa et al.5 report immediate reduction of the P amplitude in the standard leads after mild exercise, while a further augmentation of the P magnitude was observed in the first minutes of the recovery period in the present study (fig. 3).

In an earlier study from this laboratory,14 a small, but statistically significant shortening of the QRS complex of 2.7 ± 2.0 msec was found in young individuals, using the same waveform analysis algorithms. In the present study the QRS duration did not change, a finding also described by Rautaharju et al.16

heart rate of 170 beats per minute (fig. 2). The R wave duration shortened by the same amount. No significant changes were observed in the magnitudes of the time-normalized QRS vectors during exercise. Also the spatial orientation of the maximum QRS vectors did not change (fig. 2). However, the initial and terminal parts of the QRS complex shifted rightward and superiorly. This shift caused a heart rate dependent right axis deviation in the ECG.

ST-T Segment Changes

The Q-TPK interval shortened during exercise and increased again in the recovery period. The ST segment shifted toward the right, superior, and posterior. These changes were also linearly dependent on heart rate (fig. 4).
There is no apparent explanation for this discrepancy, other than the differences in the populations studied.

The observed shift of the QRS and ST-T vectors toward the right and superiorly during exercise is in agreement with data published by Blomqvist and by Rautaharju et al. This shift results in a depression of the QRS-ST junction followed by an ascending ST segment. This junctional depression has often been reported in different leads and is generally considered as a normal response to exercise. The junctional depression proved to be linearly related to heart rate. It should be noted that, while time-normalized amplitudes in leads X and Y decreased during exercise, the amplitudes at ST-70 and ST-90 increased (table 3). Also the variances of these measurements increased at higher heart rates. This was caused by the gradual shortening of the Q-T interval. At a heart rate of 60, ST-90 corresponds approximately to ST-4/8, while it corresponds to ST-6/8 at a heart rate of 180 beats/min. Also the slopes measured at fixed intervals after the QRS complex became steeper during exercise. On the other hand the slopes of the time-normalized ST segment hardly changed during exercise (fig. 2), a finding which is in agreement with the results obtained with Chebychev waveform vectors for the description of the ST-segment changes during moderate exercise.

Possible Cause of ECG Changes During Exercise

The factors which may contribute to the changes of

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**Table 2**

*Measurements from the Electrocardiograms Used in this Study*

<table>
<thead>
<tr>
<th></th>
<th>Units</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPK-Q</td>
<td>msec</td>
<td>Interval spatial maximum P wave — onset QRS complex</td>
</tr>
<tr>
<td>QRS</td>
<td>msec</td>
<td>QRS duration</td>
</tr>
<tr>
<td>Q-TPK</td>
<td>msec</td>
<td>Interval onset: QRS complex — spatial maximum T wave</td>
</tr>
<tr>
<td>P-1/8 . . . P-8/8</td>
<td>μV</td>
<td>8 time-normalized amplitudes of the P wave, with the maximal spatial magnitude of the P wave</td>
</tr>
<tr>
<td>QRS-1/8 . . . QRS-8/8</td>
<td>μV</td>
<td>8 time-normalized amplitudes of the QRS complex</td>
</tr>
<tr>
<td>ST-1/8 . . . ST-8/8</td>
<td>μV</td>
<td>8 time-normalized amplitudes of the ST-T segment, with the maximal spatial magnitude of the T wave</td>
</tr>
<tr>
<td>ST-30 . . . ST-90</td>
<td>μV</td>
<td>Amplitudes of the ST segment at 30, 50, 70, and 90 msec after the end of the QRS complex</td>
</tr>
<tr>
<td>S×A</td>
<td>mV × msec</td>
<td>Area of the negative part of the ST segment</td>
</tr>
</tbody>
</table>

*The mean amplitude 20-10 msec before the onset of the QRS complex is the baseline for all measurements.*

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**Figure 2**

Means of time-normalized instantaneous P, QRS, and ST vectors at rest and during exercise at heart rate of 180-180 beats/min. The Cartesian coordinates (X, Y, Z) have been plotted as well as the polar coordinates (M = magnitude; α = angle in the frontal plane; AP = angle toward the frontal plane). Vertical calibration of X, Y, Z, M is in mV; angles in degrees. α = 0 : x-axis; + = vectors pointing toward inferior; A: vectors pointing anterior; P: posterior.
Figure 3
Plots of X, Y, Z components, and magnitude (M) of the maximal P vectors at rest and during exercise, and the magnitude in the recovery period against heart rate. Regression equations are presented in table 3. (P = 3/8)

Figure 4
Plots of instantaneous 2/8 ST vectors during exercise against heart rate. Regression equations are presented in table 3.
**ECG CHANGES DURING & AFTER EXERCISE**

**Figure 5**

Plots of differences between rest and exercise ECGs. Means have been plotted of the records when the heart rate during exercise was 90-50 (thin line), 60-80 (dotted line), and 90-110 (heavy line) beats per minute higher than at rest. See legend figure 2. Note the gradual augmentation of the magnitude of the changes (M), while the mean directions of the changes (α, AP) are the same at different heart rates.

**Table 3**

Regression Analysis of ECG Measurements and Heart Rate During Exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>N</th>
<th>Mean</th>
<th>sd</th>
<th>A</th>
<th>B</th>
<th>r</th>
<th>see</th>
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<tr>
<td>PPK-Q</td>
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<td>92</td>
<td>19</td>
<td>133</td>
<td>-0.34</td>
<td>0.38</td>
<td>36*</td>
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<tr>
<td>QRS</td>
<td>405</td>
<td>84</td>
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<td>81</td>
<td>0.02</td>
<td>0.09</td>
<td>8</td>
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<tr>
<td>Q-TPK</td>
<td>404</td>
<td>230</td>
<td>31</td>
<td>330</td>
<td>-0.81</td>
<td>0.88</td>
<td>15**</td>
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<tr>
<td>P -3/8 X</td>
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<td>55</td>
<td>41</td>
<td>9</td>
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<td>0.29</td>
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<tr>
<td>Y</td>
<td>356</td>
<td>176</td>
<td>62</td>
<td>54</td>
<td>0.95</td>
<td>0.51</td>
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<td>356</td>
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<td>56</td>
<td>23</td>
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<td>0.21</td>
<td>30</td>
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<td>QRS-7/8 X</td>
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<td>-90</td>
<td>162</td>
<td>200</td>
<td>-2.35</td>
<td>0.49</td>
<td>142</td>
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<tr>
<td>Y</td>
<td>405</td>
<td>-22</td>
<td>106</td>
<td>106</td>
<td>-1.03</td>
<td>0.33</td>
<td>100</td>
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<tr>
<td>Z</td>
<td>405</td>
<td>395</td>
<td>183</td>
<td>331</td>
<td>0.52</td>
<td>0.10</td>
<td>182</td>
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<td>ST -1/8 X</td>
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<td>-30</td>
<td>51</td>
<td>108</td>
<td>-1.12</td>
<td>0.74</td>
<td>34**</td>
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<td>ST -2/8 X</td>
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<td>0.65</td>
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<tr>
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<td>52</td>
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<tr>
<td>Y</td>
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<td>81</td>
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<tr>
<td>Z</td>
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<td>63</td>
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<td>142</td>
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<td>0.28</td>
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<td>Z</td>
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<td>-277</td>
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<td>43</td>
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<td>0.43</td>
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<tr>
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<td>58</td>
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<td>-145</td>
<td>124</td>
<td>6</td>
<td>-1.22</td>
<td>0.33</td>
<td>117</td>
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</table>

*S < sd (P ≤ 0.05).

**See < sd (P ≤ 0.01).**

Regression analysis of 32 selected ECG parameters with heart rate as independent variable (Y = A + B · X) in rest and exercise records. List of parameters in table 2. P-3/8 and ST-8/8 correspond to the maximum spatial magnitude of the P and T wave.

Results of regression analysis of all ECG measurements during exercise and recovery can be obtained from the authors.

Abbreviations N = number of records used; sd = standard deviation; A, B = regression parameters—intercept and slope; r = correlation coefficient; see = standard error of estimate.

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The P wave, the QRS complex, and the T wave during exercise include different positions of the recording electrodes relative to the heart at rest and during exercise; alterations of the action potentials or the atrial and ventricular activation patterns; augmentation of the atrial repolarization wave (Tα); temporally increased serum potassium levels, increased hematocrit, and changes in the intracardiac blood volume. However, none of these can account for all ECG changes during exercise, as will be shown below, and little is known about the combined effect of these factors.

It is unlikely that the ECG changes are caused largely by differences in the position of the heart because the spatial orientation of the maximum P, QRS and T vectors remained constant (fig. 2). Alterations in the shape of the action potentials occur when the heart rate increases, and these may contribute to the ECG changes during exercise.

It has been suggested that the augmentation of the P wave is caused by a more synchronous activation of the right and left atrium during exercise. However, the duration of the QRS complex did not change during or after exercise, and we observed no shortening of the P wave at heart rates from 60 to 120 beats/min, which makes this mechanism unlikely.

It is striking that the changes of the QRS complex and the ST-T segment during exercise have similar spatial orientations and magnitudes. This suggests that one mechanism is responsible for both the QRS and the ST-T changes.

Lepeschkin indicated that the lowering of the R wave as well as a depression of the initial part of the ST segment may be caused by an augmented atrial repolarization wave. This Tα wave usually is opposite in direction to the P wave and becomes more negative when the P wave increases. During exercise, however, the most striking changes of the P amplitude were seen in the Y lead, while the largest changes of the ST vectors occurred in lead X. Also the ST-T
changes were continuous from the terminal part of QRS until the peak of the T wave, which is far beyond the end of the T wave (fig. 5). Consequently, this mechanism can explain only part of the observations.

Lepeschkin also suggested that temporarily increased serum potassium levels may be responsible for the T wave changes during exercise. However, Simonson did not find a correlation between the serum potassium levels and T wave amplitudes during strenuous work.

The P waves observed during exercise in the present study correspond to the pattern of predominant right atrial overload which has been described recently by Ishikawa et al. It is generally accepted that changes in the intracardiac blood volume, as well as hematocrit changes, influence the apparent electric forces of the heart according to the Brody effect. Since the atrial depolarization fronts are largely tangentially oriented, an increased right atrial volume should cause a decrease in the right atrial forces, which is contradictory with the P wave pattern in right atrial overload. It is not clear how this dilemma can be explained.

The study of Braunwald et al. of the left ventricular volume in man during mild supine exercise, as well as recent data obtained during isometric exercise and in animal experiments, shows a reduction of the left ventricular end-systolic volume during exercise. This may contribute to the lowering of the T wave during exercise, because the repolarization vectors are radially oriented. The augmentation of the T wave after exercise may then be explained by a return of the end-systolic volume to resting levels, or even by a temporary overshoot of this volume immediately after exercise.

The increased hematocrit caused by fluid loss during exercise may contribute to the augmentation of the P wave magnitude, as shown in animal experiments. However, as hematocrit increases only 10% during strenuous exercise, this factor may account for only 5–20% augmentation of the P wave. Nelson et al. have shown that the magnitude and orientation of the QRS vectors are altered by increased hematocrit. According to the Brody effect, the tangentially-oriented depolarization fronts in the right ventricle would be enhanced by this factor while the radially-oriented left ventricular forces would be reduced. This may explain the shift toward the right and superiorly of the QRS complex during exercise.

While all these mechanisms need further analysis, it is attractive to consider the concept that the ECG changes during exercise are, in part, dependent on the hemodynamic performance of the heart. This concept is supported by the recent observation that patients with a manifest reduction of the R amplitude during exercise have benefited more from physical training than others who do not show such ECG changes.

Clinical Implications

The data presented in this study show that the P, QRS, and T waves in the ECG change gradually, in a predictable manner, during a multistage exercise procedure. In particular, the amplitudes of the P wave and the first half of the ST segment appeared to be linearly related to heart rate during exercise. The detailed analysis of the exercise ECG which permitted these observations was only possible after noise reduction by computer processing of the ECG signal.

These findings imply that the diagnostic value of exercise ECGs may be improved when ST-segment measurements, obtained by digital computer techniques in a given subject, are compared with those in a control group at the same heart rate. As the measurements in the recovery period were different from those at corresponding heart rates during exercise, separate criteria should be used for analysis of exercise and recovery records. Preliminary results from our laboratory indicate that the sensitivity of the exercise ECG for detection of ischemic heart disease is indeed considerably enhanced when these principles are employed for interpretation of ECG measurements during exercise.

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