Electrocardiograms of 25 Marathon Runners before and after 100 Meter Dash*

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ECG characteristics of well-trained endurance athletes who are in a state of training vagotonia were investigated in comparison with male and female controls of the same age group. The effects of sympathicotonia on these ECG characteristics of training vagotonia were also investigated by recording ECG immediately after 100 meter dash in endurance athletes. Thus, ECG characteristics of endurance athletes could be divided into 2 groups, an acute and a chronic effect of maximal exercise. On the basis of these observations and inferences, the athletic heart was classified into 5 stages, and their probable ECG manifestations were reasoned.

GELLHORN has shown that exercise activity of one side of the autonomic nervous system is accompanied by an increase in the activity of the opposing system¹⁻³. In this manner, stimulation of the sympathetic nervous system will tend to increase the activity of the parasympathetic nervous system. It is generally accepted that characteristics of the circulatory system of well-trained athletes are manifestations of the so-called training vagotonia⁴. The effects of moderate to maximal exercise on the electrocardiogram of non-athletes have been studied by many investigators by means of the MASTER, treadmill or postural test⁵⁻¹⁷. However, the study of the effects of the maximal exercise on the electrocardiogram of well-trained endurance athletes is rare.

This investigation was carried out to see the effects of maximal exercise on the electrocardiogram of well-trained endurance athletes and, if possible, to differentiate the electrocardiographic changes due to an imbalance in the autonomic nervous system from those due to anatomical or histopathological changes of the heart.

SUBJECTS AND METHODS

I. Subjects

Among 25 marathon runners of the Self Defence Forces (endurance athletes), all males aged 19 to 31, electrocardiograms were obtained before 100 meter dash from 19 cases and after 100 meter dash from 23 cases. In only 18 cases, electrocardiograms could be recorded before and immediately after 100 meter dash. As controls, electrocardiograms were obtained from 20 ordinary members of the Self Defence Forces (male controls), all males aged 19 to 32 and from 27 students of Yamaguchi Red Cross Nursery College (female controls), all females aged 18 to 20. In endurance athletes, the effect of an altered heart position after dash was made minimum by recording all electrocardiograms in a recumbent position. In order to minimize "shyness response" with a rise in the heart rate, electrocardiograms of student nurses were recorded in a closed room by a classmate.

II. Procedures of measurements

1) Heart rate

The heart rate was calculated from the electrocardiogram in all cases except in cases of Wenckebach's period and marked respiratory arrhythmia which were excluded from the statistic analysis of the heart rate.

2) P wave in the standard limb leads

The P wave in the standard limb leads was measured under magnification with a rollfilm viewer for chest x-ray. One of the major difficulties was the small size of the measurements. Because of this, it was difficult to recognize variations in voltage of less

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than ca. 0.25 mm, which in many cases represent a change in amplitude of the order of ca. 25 per cent. Moreover, the amplitude of each P wave somewhat varied in the same lead. In this investigation, the highest P wave was employed for the measurements in each lead.

3) P wave axis

The P wave axis was determined by means of the triaxial reference system derived from the Einthoven triangle.

4) Sum of SV1 + RV5

Both SV1 and RV5 were measured with the naked eye to the nearest 0.5 mm and added arithmetically. As in the P wave, the largest SV1 and RV5 were selected for the measurements.

5) Incomplete RBBB patterns in V1

After REINDELL and KLEPZIG^{18,19}, incomplete RBBB patterns in which QRS duration is longer than 0.08 sec. were divided into two types. A physiological pattern is an Rsr', rSr'' or rsr' pattern in which R or r is higher than r' ("physiologisch" type of REINDELL and KLEPZIG). A pathological pattern is an rsR' pattern in which R' is higher than r ("unvollständig" type of REINDELL and KLEPZIG).

6) Notched or slurred QRS complexes in V1

A notched or slurred QRS complex in V1 in which QRS duration is shorter than or equal to 0.08 sec. is a QRS complex which has a notching or slurring somewhere in up- or downstrokes of the R or the S wave.

7) TV5/RV5 ratio

The TV5 and RV5 were measured with the naked eye, and the TV5/RV5 ratio was calculated.

8) ST segment in V6

The ST segment in V6 was measured under magnification with a rollfilm viewer for chest x-ray only in endurance athletes before and after 100 meter dash. In this group, the ST segment in V6 was concave or horizontal in most cases, so an ST segment elevation or depression was measured from the level of the PR segment to the lowest portion of the concave or horizontal ST segment. The Ta wave was not observed in the left precordial leads in this survey. In male and female controls the ST segment in V6 was junctional in most cases, and an accurate measurement of an ST segment elevation or depression was almost impossible.

9) Negative, diphasic and bifurcated T waves in right to mid-precordial leads

A negative T wave is a T wave which is completely inverted without any portion of positivity. A diphasic T wave observed in the right precordial leads is a T wave with an initial positivity followed by a terminal negativity. A T wave with an initial negativity followed by a terminal positivity (the so-called strain pattern) was not observed in the right precordial leads in this survey. A bifurcated T wave

(die 2-gipflige T-Welle²¹ or a notched T wave²²) is a T wave which has two peaks which can be separated from the U and P waves. Namely, a bifurcated triphasic T wave is positive-negative-positive in polarity. When the negative portion of a bifurcated T wave was large and the second peak was small and overlapped with the U wave, it was classified into the diphasic T wave. In most cases, a diphasic T wave with a terminal negativity is followed by an apparently heightened positive U wave.

10) U waves in unipolar precordial leads

The U wave was scored as 1 when it showed a distinct peak which could be separated from the downstroke of the T wave. The U wave was scored as 0.5 when it fused stepwise with a downstroke of the T wave without forming its own peak. The U wave was scored as 0 when there was no visible U wave. No negative U wave was observed in this survey.

RESULTS

I. Electrocardiograms at rest in endurance athletes, male controls and female controls

1) Heart rate

An average heart rate was 55.5/min in endurance athletes (48 to 68/min), 65.2/min in male controls (49 to 85/min) and 63.5/min in female controls (52 to 77/min).

2) P wave amplitude

The amplitude of the P wave in lead I averaged +0.6 mm in endurance athletes (0 to +1.1 mm), +0.4 mm in male controls (+0.2 to +0.8 mm) and +0.6 mm in female controls (+0.3 to +1.0 mm). That in lead II averaged +1.1 mm in endurance athletes (+0.1 to +1.8 mm), +0.6 mm in male controls (+0.1 to +1.0 mm) and +0.7 mm in female controls (-0.3 to +1.1 mm). That in lead III averaged +0.4 mm in endurance athletes (-0.2 to +1.2 mm), +0.2 mm in male controls (-0.3 to +1.0 mm) and +0.2 mm in female controls (-0.6 to +1.0 mm).

3) P wave axis

The P wave axis averaged +48.9 degrees in endurance athletes, +40.2 degrees in male controls and +35.2 degrees in female controls. A P wave axis of more than +30 degrees was found in 77.7 per cent of endurance athletes, 63.2 per cent of male controls and 63.0 per cent of female controls (Table I).

4) Notched P waves

A notched, M-shaped P wave in one or more

of the standard and unipolar extremity leads was observed in 72.2 per cent of endurance athletes, 11.1 per cent of male controls and 0 per cent of female controls.

5) Respiratory arrhythmia

Respiratory arrhythmia was observed in 10.0 per cent of endurance athletes, 0 per cent of male controls and 18.5 per cent of female controls (Table II).

6) Nodal rhythm

Nodal rhythm was observed in one case of female controls. This might be a manifestation of "shyness response" (Table II).

7) First degree A-V block

First degree A-V block was observed in 10.0 per cent of endurance athletes, 0 per cent of male controls and 7.4 per cent of female controls (Table II).

8) Second degree A-V block of Wenckebach type

Second degree A-V block of Wenckebach type was observed in 10.0 per cent of endurance athletes and 0 per cent of both male and female controls (Table II).

9) Premature beats

Ventricular premature beats were observed in one case of each group. These might be induced reflexly by mental tension during ECG recording (Table II).

10) Notched or slurred QRS complexes in V1

A notched or slurred QRS complex in V1 (QRS duration ≤ 0.08 sec) was observed in 40.0 per cent of endurance athletes, 52.6 per cent of male controls and 33.3 per cent of female controls. As shown in Fig. 1, some of the cases with a notched QRS complex in V1 showed a pathological incomplete RPBB pattern (rsR') in V4R. However, because of small size of the sample of notching in V1 and of absence of V4R recording in most of these cases, no statistical approach was made (Table III).

11) Incomplete RBBB patterns in V1

Incomplete RBBB patterns in V1 (QRS duration > 0.08 sec) were observed in 35.0 per cent of endurance athletes, 21.0 per cent of male controls and 3.7 per cent of female controls. A pathological rsR' pattern was found in 15.0 per cent of endurance athletes and 0 per cent of both male and female controls. A physiological RSr', rSr' or rsr' pattern was found in 20.0 per cent of endurance athletes, 21.0 per cent of male controls and 3.7 per cent of female controls (Table III).

12) Negative and diphasic T waves in right to mid-precordial leads

The percentage occurrence of negative and diphasic T waves in V1 was 95.0 per cent in endurance athletes, 73.6 per cent in male controls and 81.6 per cent in female controls. That in V2 was 36.8 per cent in endurance athletes,

TABLE I P WAVE AXIS AT REST

	<i>endurance athletes</i>	<i>male controls</i>	<i>female controls</i>
<i>less than -30 to 0 degrees (%)</i>	0	5.3	14.8
<i>0 to +30 degrees (%)</i>	22.2	31.6	22.2
<i>+30 to +60 degrees (%)</i>	44.4	42.1	44.5
<i>+61 to more than +90 degrees (%)</i>	33.3	21.1	18.5

TABLE II OCCURRENCE OF ARRHYTHMIAS AT REST

	<i>endurance athletes</i>	<i>male controls</i>	<i>female controls</i>
<i>respiratory arrhythmia (%)</i>	10.0	0	18.5
<i>nodal rhythm (%)</i>	0	0	3.7
<i>first degree A-V block (%)</i>	10.0	0	7.4
<i>Wenckebach A-V block (%)</i>	10.0	0	0
<i>premature beats (%)</i>	5.0	5.3	3.7

5.3 per cent in male controls and 0 per cent in female controls. That in V₃ was 15.8 per cent in endurance athletes and 0 per cent in both

male and female controls (Table IV).

13) Bifurcated T waves in mid-precordial leads

A) Y. T., a 75-year-old male

B) T. K., a 69-year-old female

Fig. 1. Coexistence of notching and incomplete RBBB in the right precordial leads. A) In V_{4R}, a QRS complex shows a pathological pattern of incomplete RBBB. In V₁, an upstroke of the S wave is notched, and the QRS complex resembles a physiological pattern of incomplete RBBB. B) In V₁, the P and T waves are both inverted, and the QRS complex shows an rS pattern with a small notching in the upstroke of the S wave. Namely, the QRS complex in V₁ resembles that in aVR and shows a cavity potential of the right ventricle. In V₂, the P wave is diphasic, and the QRS complex shows a pathological pattern of incomplete RBBB. In general, the location of an incomplete RBBB pattern in the unipolar precordial leads depends upon the interrelation between the heart position and the electrode.

TABLE III OCCURRENCE OF A NOTCHED QRS COMPLEX AND INCOMPLETE RBBB PATTERNS IN V₁

	endurance athletes	male controls	female controls
a notched QRS complex in V ₁ (%) (QRS ≤ 0.08 sec)	40.0	52.6	33.3
incomplete RBBB patterns (%) (QRS > 0.08 sec)	35.0	21.0	3.7
a physiological RSr' pattern (%)	20.0	21.0	3.7
a pathological rsR' pattern (%)	15.0	0	0

TABLE IV OCCURRENCE OF NEGATIVE AND DIPHASIC T WAVES IN THE RIGHT TO MID-PRECORDIAL LEADS

	endurance athletes	male controls	female controls
V ₁ (%)	95.0	73.6	81.6
V ₂ (%)	36.8	5.3	0
V ₃ (%)	15.8	0	0
V ₄ (%)	0	0	0

A bifurcated T wave which appears to the left of a negative or a diphasic T wave was observed in 26.3 per cent of endurance athletes, 0 per cent of male controls and 7.4 per cent of female controls (Table V). When the negativity between the two peaks of a bifurcated T wave was small, the first peak was merged into the second one, and a T wave with a blunt peak was formed in the next lead to the left of the distinctly bifurcated T wave as shown in Fig. 7.

14) U waves in unipolar precordial leads

The percentage occurrence of the U wave in V₁ was 5.3 per cent in endurance athletes, 36.9 per cent in male controls and 43.2 per cent in female controls, that in V₂ 79.0 per cent in endurance athletes, 100 per cent in male controls and 96.5 per cent in female controls and that in V₃ 63.2 per cent in endurance athletes, 92.0 per cent in male controls and 85.1 per cent in female controls. The percentage appearance of the U wave in V₄ was 28.9 per cent in endurance athletes, 76.3 per cent in male controls and 64.8 per cent in female controls, that in V₅ 10.5 per cent in endurance athletes, 44.7 per cent in male controls and 44.5 per cent in female controls and that in V₆ 5.3 per cent in endurance athletes, 13.2 per cent in male controls and 14.8 per cent in female controls (Table

VI).

15) Concave RS-T elevation in left precordial leads

A concave RS-T elevation in the left precordial leads was found in 80.0 per cent of endurance athletes, 31.6 per cent of male controls and 11.1 per cent of female controls. Namely, the percentage occurrence of a concave RS-T elevation was inversely proportional to that of the U wave in the left precordial leads (Table VII). The Cornell Medical Index of 16.6 per cent of endurance athletes with this concave RS-T elevation was included in the area III (provisionally neurotic) of FUKAMACHI's discriminative chart²⁰, and that of 83.6 per cent of endurance athletes in the areas I and II (normal and provisionally normal).

16) Amplitudes of SV₁ and RV₅ and sum of SV₁ + RV₅

The amplitude of SV₁ averaged 12.9 mm in endurance athletes (6 to 29 mm), 9.1 mm in male controls (3 to 23 mm) and 6.6 mm in female controls (2 to 21 mm). That of RV₅ averaged 37.6 mm in endurance athletes (16 to 48 mm), 18.4 mm in male controls (10 to 28 mm) and 13.3 mm in female controls (5 to 24 mm). Namely, SV₁ in endurance athletes was 142 per cent of that in male controls and 196 per

TABLE V OCCURRENCE OF BIFURCATED T WAVES IN THE MID-PRECORDIAL LEADS

	<i>endurance athletes</i>	<i>male controls</i>	<i>female controls</i>
V ₂ to V ₄ (%)	26.3	0	7.4

TABLE VI OCCURRENCE OF U WAVES IN UNIPOLAR PRECORDIAL LEADS

	<i>endurance athletes</i>	<i>male controls</i>	<i>female controls</i>
V ₁ (%)	5.3	36.9	43.2
V ₂ (%)	79.0	100.0	96.5
V ₃ (%)	63.2	92.0	85.1
V ₄ (%)	28.9	76.3	64.8
V ₅ (%)	10.5	44.7	44.5
V ₆ (%)	5.3	13.2	14.8

TABLE VII OCCURRENCE OF CONCAVE RS-T ELEVATIONS IN THE MID-TO LEFT PRECORDIAL LEADS

	<i>endurance athletes</i>	<i>male controls</i>	<i>female controls</i>
V ₄ to V ₆ (%)	80.0	31.6	11.1

cent of that in female controls. RV₅ in endurance athletes was 204 per cent of that in male controls and 281 per cent of that in female controls. The sum of SV₁+RV₅ averaged 50.3 mm in endurance athletes (43 to 67 mm), 27.4 mm in male controls (13 to 41 mm) and 19.8 mm in female controls (11 to 37 mm). Namely, the sum of SV₁+RV₅ in endurance athletes was 184 per cent of that in male controls and 254 per cent of that in female controls (Table VIII).

17) Amplitude of TV₅ and TV₅/RV₅ ratio

The amplitude of TV₅ averaged 12.4 mm in endurance athletes (7.5 to 20.0 mm), 5.0 mm in male controls (1.5 to 11.0 mm) and 5.8 mm in female controls (3.0 to 10.0 mm). Namely, TV₅ in endurance athletes was 248 per cent of that in male controls and 214 per cent of that in female controls. The TV₅/RV₅ ratio averaged 0.33 in endurance athletes (0.20 to 0.56), 0.30 in male controls (0.16 to 0.45) and 0.45 in female controls (0.21 to 0.75). Namely, the TV₅/RV₅ ratio, which should approach zero or be negative in coronary insufficiency with or without T wave inversion, was greater in endurance athletes than in ordinary members of the Self Defence Forces. In two control groups, the ratio was greater in student nurses than in ordinary members of the Self Defence Force (Table VIII).

II. Electrocardiographic changes after 100 meter dash in endurance athletes

1) Heart rate

An average heart rate increased from 55.5/min (48 to 68/min) to 73.0/min (48 to 102/min) after dash. Namely, the heart rate increased by 21.4/min (−17 to +51/min) on an average. In one case, it was decreased by

17/min after dash. This may indicate an earlier onset of a rebound vagotonia.

2) P wave amplitude

After dash, an average amplitude of the P wave in lead I changed from +0.6 mm (0 to +1.1 mm) to +0.5 mm (0 to 1.0 mm), that in lead II from +1.1 mm (+0.1 to +1.8 mm) to +1.4 mm (+0.1 to +2.1 mm) and that in lead III from +0.4 mm (−0.2 to +1.2 mm) to +0.7 mm (−0.5 to +2.0 mm). The percentage occurrence of a notched P wave which was 72.2 per cent before dash remained unaltered after dash.

3) P wave axis

An average P wave axis increased from +48.9 (0 to +80) to 62.0 (0 to +105) degrees after dash. While the percentage occurrence of P wave axis between 0 and +30 degrees decreased from 22.2 to 5.6 per cent and that between +31 to +60 degrees from 44.4 to 33.4 per cent, the P wave axis between +61 to more than 90 degrees increased markedly from 33.3 to 61.2 per cent. The P wave axis deviated to the right by more than 10 degrees in 50.0 per cent, to the left by more than 10 degrees in 11.1 per cent and either to the right or to the left by less than 10 degrees in 38.9 per cent (Table IX and Fig. 2).

4) Respiratory arrhythmia

The percentage of respiratory arrhythmia increased from 10.0 to 35.0 per cent. This might be due to a vigorous thoracic breathing after dash. No S-A block was observed before and after dash (Table X).

5) Nodal rhythm

In one case, nodal rhythm was observed immediately after dash and returned to sinus rhythm during postexercise ECG recording. This

TABLE VIII AMPLITUDES OF SV₁, RV₅ AND TV₅, SUM OF SV₁+RV₅ AND TV₅/RV₅ RATIO

	<i>endurance athletes</i>		<i>male controls</i>		<i>female controls</i>	
	<i>average</i>	<i>range</i>	<i>average</i>	<i>range</i>	<i>average</i>	<i>range</i>
SV ₁ (mm)	12.9	6-29	9.1	3-23	6.6	2-21
RV ₅ (mm)	37.6	16-48	18.4	10-28	13.3	5-24
TV ₅ (mm)	12.4	7.5-20.0	5.0	1.5-11.0	5.8	3.0-10.0
SV ₁ +RV ₅ (mm)	50.3	43-67	27.4	13-41	19.8	11-37
TV ₅ /RV ₅	0.33	0.20-0.56	0.30	0.16-0.45	0.45	0.21-0.75

may indicate that the first pacemaker in the S-A node which has been accustomed to bradycardia is unable to increase the heart rate beyond a certain critical point, and the second pacemaker in the A-V node relieves the first pacemaker in the S-A node when an increase in the heart rate is required (Table X and Fig. 3).

6) A-V blocks

Both first degree A-V block and second degree A-V block of Wenckebach type disappeared after dash. This indicates that these two types of A-V block observed in endurance athletes are the most important electrocardiographic manifestations of the training vagotonia (Table X and Figs. 4 and 5).

7) Notched QRS complexes and incomplete RBBB patterns in V₁

In one case, a physiological incomplete RBBB pattern changed to a pathological one after dash.

A notched QRS complex in V₁ in two cases changed to a physiological incomplete RBBB pattern after dash. Accordingly, the percentage occurrence of physiological and pathological incomplete RBBB patterns increased from 35.0 to 45.0 per cent, and the percentage occurrence of a notched QRS complex in V₁ decreased from 40.0 to 30.0 per cent. In no case of a normal QRS complex in V₁, a distinct notching was formed after dash. Namely, the cause of notching and incomplete RBBB in endurance athletes seems to be mostly of anatomical nature (regulative dilatation and adaptive hypertrophy) and partly of functional (Table XI and Fig. 6).

8) Negative and diphasic T waves

The percentage occurrence of negative and diphasic T waves increased from 95.0 to 100.0 per cent in V₁, from 36.8 to 65.3 per cent in V₂, from 15.8 to 30.2 per cent in V₃ and from 0 to 13.1 per cent in V₄. This seems to indicate

TABLE IX P WAVE AXIS AFTER DASH

	<i>before dash</i>	<i>after dash</i>
<i>less than -30 to 0 degrees (%)</i>	0	0
<i>0 to +30 degrees (%)</i>	22.2	5.6
<i>+31 to +60 degrees (%)</i>	44.4	33.4
<i>+61 to more than +90 degrees (%)</i>	33.3	61.2

Fig. 2. Rightward deviation of the P wave axis after dash.
Y. N., a 20-year-old male
The P wave axis was ca. +70 degrees before dash and ca. +90 degrees after dash. A marked Ta wave was observed in leads II and III after dash.

T. H., a 19-year-old male

Fig. 3. Nodal rhythm occurred immediately after dash.

In lead II, all P waves are negative with a PQ interval of 0.12 sec., indicating that the pacemaker is present in the A-V node at this moment. In lead aVR, all P waves are normally inverted with a PQ interval of 0.16 sec., indicating that pacemaker is present in the S-A node at this moment. In lead aVF, the 1st and 6th to 9th P waves are positive with a PQ interval of 0.16 sec., and the 2nd to 5th P waves negative with a PQ interval of 0.12 sec. This indicates that the former P waves are of sinus origin, and the latter P waves of A-V nodal origin. The coupling interval of a QRS complex of sinus origin with a QRS complex of A-V nodal origin (0.72 sec.) is much shorter than that of a QRS complex of A-V nodal origin with a QRS complex of sinus origin (1.14 sec.).

Y. N., a 23-year-old male
before dash (PQ interval; 0.24 sec.)

after dash (PQ interval: 0.18 sec.)

Fig. 4. Effect of dash on first degree A-V block

The PQ interval was 0.24 sec. before dash and 0.18 sec. after dash. The RS-T segment in lead II was slightly depressed, and a diphasic T wave in lead III became upright after dash. The QRS amplitude was little affected by dash.

that negative and diphasic T waves in the right precordial leads are mostly anatomical and are

also partly functional in nature and that the electromotive forces of the area responsible for

TABLE X OCCURRENCE OF ARRHYTHMIAS AFTER DASH

	<i>before dash</i>	<i>after dash</i>
<i>respiratory arrhythmia (%)</i>	10.0	35.0
<i>nodal rhythm (%)</i>	0	5.0
<i>first degree A-V block (%)</i>	10.0	0
<i>Wenckebach A-V block (%)</i>	10.0	0
<i>premature beats (%)</i>	5.0	0

M. K., a 21-year-old male
before dash (Wenckebach phenomenon)

after dash (regular sinus rhythm)

Fig. 5. Effect of dash on second degree A-V block of Wenckebach type. Second degree A-V block of Wenckebach type disappeared after dash and became a regular sinus rhythm. Before dash, a QRS complex in V1 showed a pathological pattern of incomplete RBBB, and that in V2 a physiological one. The pathological pattern of incomplete RBBB in V1 remained unaltered after dash. The r' in V2 before dash became a small notching in the upstroke of the S wave after dash. The negativity of the T wave in V2 was rather decreased after dash.

the T wave negativity are increased after dash (Table XII).

9) Bifurcated T waves

The bifurcated T wave in V2 to V4 increased from 26.3 to 43.5 per cent after dash. The significance of this observation seems to be the same as that of negative and diphasic T waves. Namely, an increase in the percentage occurrence of bifurcated T waves is due to an increase in the electromotive forces of the area responsible for the T wave negativity (Table XIII and Fig. 7).

10) U waves in unipolar precordial leads

After dash, the percentage occurrence of the U wave in all precordial leads also increased except in V1 and V6 in which it remained nearly unaltered. Namely, the percentage U wave appearance increased from 79.0 to 84.7 per cent in V2, from 63.2 to 76.0 per cent in V3, from 28.9 to 45.7 per cent in V4 and from 10.5 to 17.4 per cent in V5. Although the increase in percentage occurrence of the U wave was not so marked as that of the negative, diphasic and bifurcated T waves, the susceptibility of the U wave to the maximal exercise seems to indicate that the causation of the U

TABLE XI OCCURRENCE OF A NOTCHED QRS COMPLEX AND INCOMPLETE RBBB PATTERNS IN V₁ AFTER DASH

	before dash	after dash
a notched QRS complex in V ₁ (QRS=0.08 sec) (%)	40.0	30.0
incomplete RBBB patterns in V ₁ (QRS>0.08 sec) (%)	35.0	45.0
a physiological Rsr' pattern (%)	20.0	25.0
a pathological rsR' pattern (%)	15.0	20.0

Fig. 6. Interchangeability of physiological and pathological patterns of incomplete RBBB. A QRS complex in V₁ showed a physiological Rsr' pattern before dash and remained unaltered after the 1st dash. After the 2nd dash, however, a QRS complex in V₁ showed a pathological rsR' pattern with an increase in the negativity of the T wave in V₁ and V₂.

TABLE XII OCCURRENCE OF NEGATIVE AND DIPHASIC T WAVES IN THE RIGHT TO MID-PRECORDIAL LEADS AFTER DASH

	before dash	after dash
V ₁ (%)	95.0	100.0
V ₂ (%)	36.8	65.3
V ₃ (%)	15.8	30.2
V ₄ (%)	0	13.1

wave is mostly anatomical and is also partly functional in nature, i.e., the electromotive forces and duration of repolarization of the area responsible for the U wave is increased after exercise (Table XIV and Fig. 8).

11) Concave RS-T elevations in mid- to left

precordial leads

The percentage occurrence of the concave RS-T elevation in the mid- to left precordial leads was decreased from 80.0 to 43.5 per cent after dash because of an ST segment depression, but the configuration of the concave RS-T seg-

TABLE XIII OCCURRENCE OF BIFURCATED T WAVES IN MID-PRECORDIAL LEADS AFTER DASH

	before dash	after dash
V_2 to V_4 (%)	26.3	43.5

Fig. 7. Appearance of a bifurcated T wave after dash.

In V2 before dash, the downstroke of the T wave can be separated into three portions, the 1st and 2nd peaks of the T wave and the U wave. The peak of the T wave in V3 before dash is blunt because of a fusion of the 1st and 2nd peaks of the bifurcated T wave. After dash, the negativity of the T wave in V2 was increased, and the 2nd peak of the bifurcated T wave was erased out by this negativity. In consequence, a positive-negative diphasic T wave was formed in V2 after dash. In V3 after dash, the fusion of the 1st and 2nd peaks of the bifurcated T wave was separated by an increase in the negativity between the two peaks, and a completely bifurcated T wave was formed in consequence. The T waves in V4 to V6 are high and sharply peaked because of a TU fusion.

TABLE XIV OCCURRENCE OF U WAVES IN UNIPOLAR PRECORDIAL LEADS AFTER DASH

	before dash	after dash
V_1 (%)	5.3	4.4
V_2 (%)	79.0	84.7
V_3 (%)	63.2	76.0
V_4 (%)	28.9	45.7
V_5 (%)	10.5	17.4
V_6 (%)	5.3	4.4

ment was retained in all cases. This seems to indicate that the concave RS-T elevation is to a greater extent chronically and adaptively conditioned and to a lesser extent acutely and functionally conditioned.

12) ST segment in V6

An average deviation of the ST segment from the isoelectric line in V6 changed from +0.52 mm (−0.1 to +1.1 mm) to +0.10 mm (−0.7 to +0.8) after dash. Namely, the ST segment was depressed by 0.48 mm on an average (+0.2 to −0.9 mm). In no case, it was depressed more than 1.0 mm in this survey. The ST segment seems to be also mostly anatomically conditioned with some susceptibility to an acute overload.

13) SV1 and RV5

In general, the amplitude of SV1 and RV5 remained almost unaltered after dash, and slight changes in them showed no definite tendency. This seems to indicate that SV1 and RV5 are

adaptively and anatomically conditioned and affected little by an acute ventricular load.

14) TV5 and TV5/RV5 ratio

An average TV5 was lowered from +12.4 mm (+7.5 to +20.0 mm) to +8.3 mm (+1.5 to +15.5 mm) after dash in amplitude. Namely, TV5 was lowered by 4.1 mm (−10.8 to +4.0 mm) on an average. In two cases, TV5 was heightened by 4.0 mm and 1.0 mm, respectively. Consequently, the TV5/RV5 ratio was decreased from 0.33 to 0.22 after dash. This indicates that although TV5 is also anatomically conditioned, it has a greater susceptibility to an acute ventricular load than SV1, RV5 and ST segment in V6 (Table XV).

DISCUSSION

1) Heart rate

It is believed that alterations in heart rate are brought about by simultaneous reciprocal changes in the sympathetic and parasympathet-

Fig. 8. Exaggeration of the U wave after dash.

In V3 to V5 before dash, the U wave is merged stepwise into the downstroke of the T wave without forming its own peak. In V3 to V4 after dash, the terminal negativity of the T wave was increased, and the U wave was separated from the downstroke of the T wave by an incisura caused by the terminal negativity of the T wave.

TABLE XV AVERAGE TV₅ AND TV₅/RV₅ RATIO AFTER DASH

	before dash	after dash
TV ₅ (mm)	12.3	8.3
TV ₅ /RV ₅ ratio	0.33	0.22

ic influences on the heart²³⁻²⁵. As is generally accepted, a slow heart rate at rest in endurance athletes might be a manifestation of a training vagotonia⁴. In one case in this investigation, the heart rate was found decreased in a post-exercise electrocardiogram. This can be interpreted that a rebound vagotonia sets in earlier in this case than in other cases because of a higher degree of a training vagotonia.

2) P wave and P wave axis

Rightward deviation of the P wave axis is one of the criteria of SCOTT^{26,27} and SELVESTER and RUBIN²⁸ for pulmonary emphysema and also one of the criteria of GROSS²⁹ and FOWLER and associates³⁰ for the P pulmonale. According to CAIRD and STANFIELD^{31,32}, a P wave axis of $+70$ degrees or more is common in acute respiratory failure. It was also reported that arterial oxygen desaturation increases the height of the P wave^{29,33} and oxygen inhalation decreases it^{29,34}. According to OHMURA³⁵, PII and PIII are heightened during the Valsalva maneuver and resemble the P pulmonale with a more vertical P wave axis. According to GORDON et al.³⁶, however, P wave amplitude furnishes little information about the right atrial weight or the right atrial volume, but provides informations about the left atrial volume. While P wave duration shows no correlation with atrial weights, it displays a significant positive correlation with both atrial volumes. CHOU and HELM³⁷ theorized that left atrial enlargement might increase the voltage of the mid- and late portion of the P wave, and in the absence of intra-atrial conduction defect, can produce a pattern of the P pulmonale in the limb leads. It seems to be likely that the deviation of the P wave axis to the right and an increase in the amplitude of PII and PIII in endurance athletes might be due to dilatation and hypertrophy of the right atrium. A more rightward deviation of the P wave axis after dash in the athletic heart can be ascribed to an acute effect of asphyxial state immediately after dash. A higher incidence of diphasic and notched P waves in one or more of the standard limb and unipolar extremity leads in endurance athletes might be due to a simultaneous presence of right and left atrial dilatations.

3) Respiratory arrhythmia

A higher incidence of respiratory arrhythmia at rest in female controls might be due to a more thoracic than abdominal breathing in student nurses. An increase in the percentage occurrence of respiratory arrhythmia in endurance athletes after dash might be also due to a vigorous thoracic breathing immediately after dash. No S-A block was observed in this investigation.

4) Nodal rhythm

Nodal rhythm observed in a student nurse at rest was transient. This might be due to a "shyness response" with a reflex increase in the automaticity of the second pacemaker in the A-V node and suppression of the first pacemaker in the S-A node. As reported previously³⁸, nodal rhythm occurs reflexly in nervous individuals through the autonomic nervous system and disappears after sedation. In endurance athletes in this investigation, nodal rhythm was observed immediately after dash and returned to sinus rhythm during postexercise ECG recording. It seems to be likely that because the first pacemaker in the S-A node which has been accustomed to bradycardia due to a training vagotonia can not increase the heart rate beyond a certain critical level, the second pacemaker in the A-V node takes over the S-A node during strenuous exercise. After exercise, a rebound vagotonia sets in, and the A-V node is again relieved by the S-A node which is more accustomed to bradycardia than the A-V node.

5) A-V blocks

MARRIOT and MENENDEZ³⁹ reported a Japanese wrestler with a normal athlete's heart showing how the bradycrotic phase of healthy sinus arrhythmia can lead to nodal escape and A-V dissociation. KITAMURA⁴ reported second degree A-V block of Wenckebach type observed in a champion of swimming. He reported also first degree A-V block in 36 per cent of swimmers, 18 per cent of players of a bicycle race and 42 per cent of wrestlers. REINDELL found first degree A-V block in 35 per cent of champions of a bicycle race, 30 per cent of marathon runners, 25 per cent of skiers and 25 per cent of amateur boxers⁴. Both first degree A-V block and second degree A-V block of Wenckebach type occur also in patients with sustained mental tension through the autonomic nervous

system and disappear after tranquilization⁴⁰. In all cases in this investigation, both first degree A-V block and second degree A-V block of Wenckebach type disappeared after dash. This indicates that these first and second degree A-V blocks in well-trained endurance athletes are due to a rebound vagotonia and can be abolished by stimulation of the sympathetic nervous system through the maximal exercise. These A-V blocks are the most important and reliable electrocardiographic signs of the training vagotonia of the athletic heart.

6) Notched QRS complexes in V1

One of the characteristic electrocardiographic findings of emphysema with superimposed right ventricular hypertrophy is an rSR' pattern in V1 or a predominant R wave in the right precordial leads²⁶. In 36 of 89 cases of pulmonary emphysema, SELVESTER and RUBIN²⁸ found slurred S waves or rSR' patterns in the right precordial leads, suggesting either early right ventricular hypertrophy or conduction defects of the right bundle branch. OHMURA³⁵ observed a transient occurrence of an rSR' pattern in V1 after the Valsalva maneuver in some of patients with chronic pulmonary diseases. As shown in Fig. 1, a notched or slurred QRS complex in V1 is in some cases an indication of a possible presence of a physiological or a pathological pattern of incomplete RBBB in V4R. Moreover, as observed in endurance athletes in this investigation, a notched QRS complex in V1 changed to a physiological pattern of incomplete RBBB in 2 of 8 cases after dash. It is probable that not only an rSR' pattern but also a notched QRS complex in V1 represents a delay in the right ventricular conduction and a superimposed early right ventricular hypertrophy in some cases.

7) Incomplete RBBB

It has been demonstrated in congenital and acquired heart diseases that the so-called incomplete RBBB pattern is not infrequently associated with mild or moderate right ventricular hypertrophy and is ascribed to diastolic overloading⁴¹. Namely, retardation of right ventricular conduction of the type of physiological and pathological patterns of incomplete RBBB is found in overloading of the right ventricle such as in mitral stenosis, pulmonary

stenosis and atrial septal defect¹⁹. REINDELL et al.⁴⁴ ascribed the incomplete RBBB in athletes to a physiological hypertrophy and dilatation especially of the right ventricle. In fact, incomplete RBBB is found most frequently in endurance athletes such as long-distance runners or champions of a bicycle race^{4,19}, and there was no organic lesion in 25 to 50 per cent of incomplete RBBB⁴². In Japanese athletes, incomplete RBBB occurred in 26 per cent of swimmers, 24 per cent of players of a bicycle race and 25 per cent of champions of weight lifting⁴. According to ROSKAMM et al.⁴, the percentage occurrence of incomplete RBBB is directly proportional to the volume of the heart, i.e., it was 45 per cent when the cardiac volume was between 1200 and 1300 ml and only 14.3 per cent when the heart volume was between 800 and 900 ml. In another report, ROSKAMM et al.¹⁹, observing a transformation of a pathological incomplete RBBB to a physiological one, a decrease in the percentage occurrence of a physiological incomplete RBBB and a restoration of normal electrocardiograms several years after the retirement of endurance athletes, indicated that incomplete RBBB observed in the athletic heart is a manifestation of an adaptation and neither prodromes nor signs of organic cardiac lesions. However, KITAMURA⁴ is of the opinion that the factor which determines the reversibility is the degree of cardiac hypertrophy and dilatation. Irreversible cardiac damages occur when the cardiac hypertrophy and dilatation exceed a certain critical degree and a transition occurs from adaptation to exhaustion. In this investigation, the percentage occurrence of incomplete RBBB increased from 35.0 to 45.0 per cent immediately after dash.

8) Concave RS-T elevations

Many investigators accepted this pattern as a normal variant. As to the cause of this concave RS-T elevation, however, there is no unanimous opinion. Some ascribed it to a racial difference, and others to a nutritional difference⁴⁵⁻⁴⁷. MELLEROWICZ⁴⁸ ascribed an ST segment elevation in athletes to alterations in the tension of the autonomic nervous system. GOLDMAN⁴⁹ is of the opinion that the electromotive forces generated during repolarization have reached sufficient magnitude prior to the

termination of depolarization, resulting in an upward displacement of the RS-T junction. NAKAMOTO⁵⁰⁻⁵⁴ reported a higher incidence of this concave RS-T elevation in mid- and left precordial leads in individuals exposed to mental stresses for more than 2 to 3 months and ascribed this pattern to a vegetative imbalance caused by persistent or intermittent mental tension⁵⁵. Physiologically, the heart is influenced by both mental tension and physical overload through the autonomic nervous systems. Accordingly, there must be a common factor in the cause of the concave RS-T elevation due to mental tension and that due to physical overload, and sympathicotonia induced by mental tension and that by physical exercise must be followed in a similar fashion by a rebound vagotonia. Thus, the amplitude between the sympathicotonia and the vagotonia is much greater in individuals with mental tension and in those with physical exercise than in normal subjects without these mental and physical stresses. This greater amplitude between the sympathicotonia and the vagotonia might be at first adaptive and shows even a greater tolerance to mental and bodily stresses, but later it becomes exhausted and pathological, and various signs of an imbalance of the autonomic nervous system appear⁵⁵. Namely, the concave RS-T elevation is reversible while it is in an adaptive stage but becomes irreversible when anatomical changes have occurred in an exhaustive stage. In 76.5 per cent of patients with the concave RS-T elevation of mental origin, the scores of the Cornell Medical Index was included in the areas III (provisionally neurotic) and IV (neurotic) of FUKAMACHI's discriminative chart^{20, 55}. In only 16.6 per cent of those with the concave RS-T elevation of physical origin in this investigation, it was included in the area III. Therefore, the concave RS-T elevation is not pathognomonic for actual neurosis but characteristic for an imbalance in the autonomic nervous system with a greater amplitude between sympathicotonia and vagotonia.

9) ST segment in V6

According to CASE et al.⁵⁶, an ST depression of the electrocardiogram results from reduction of left main coronary flow in the dog. Inti-

mately associated with this electrocardiographic changes are metabolic, ionic and hemodynamic alterations. All of these alterations are reversible following restoration of coronary flow. According to BERKSON et al.⁹, there was no clearcut relation between the age of men and the ST segment response to strenuous exercise under the age of 40. All men exhibited some degree of junctional ST depression in the precordial leads during strenuous exercise. The ST depression during strenuous exercise was less than 1 mm in 10 per cent, between 1 and 2 mm in 65 per cent and from 2 to 3 mm in 24 per cent of the cases studied. This junctional ST depression returned to the baseline within the first few minutes of the postexercise period in most cases. They stated further that T wave alterations took longer to return to the resting tracing level than junctional ST depression. In this investigation, although the athletes were subjected to postexercise ECG recording immediately after dash, an actual ECG recording was started a few minutes after the end of exercise. Therefore, as indicated by BERKSON et al.⁹, the ST segment depression by exercise has to some extent returned to the pre-exercise level before the postexercise ECG recording is started. In fact, alterations in the ST segment in V6 was much less than those in the T wave in V5 in postexercise electrocardiogram.

10) Negative and diphasic T waves

It is generally accepted that in adults the T wave in leads derived from the left side of the precordium is normally upright, while in children they are frequently inverted⁵⁷. The theories advanced in explanation of this phenomenon are concerned largely with the difference between adult and juvenile hearts and chests, and the resultant changes in relationship of the heart to the chest wall and exploring electrode. It is also reported that negative T waves in the right precordial leads of juveniles can be made to disappear by potassium administration⁵⁸. This indicates that the negative T waves in the right precordial leads of juveniles are not caused by organic lesions and that they are electrophysiological characteristics of juvenile hearts. According to SERIKI and SMITH⁵⁹, inverted or diphasic T waves are commonly seen in precordial leads on the right side but becomes less

prominent with increasing age after 11 years. LITTMANN⁶⁰ suggested that the presence of diphasic or inverted T waves in the precordial leads constitutes a persistence of the juvenile pattern and is not a manifestation of organic heart diseases. MIZUNO⁶¹ gave the percentage occurrence of the negative and diphasic T waves in young Japanese aged 18 to 29. According to him, the percentage occurrence of negative or diphasic T waves in V1 was 19.8 per cent in males and 51.5 per cent in females, that in V2 0 per cent in males and 2.9 per cent in females and that in V3 0 per cent in both males and females. SHANNO⁶² observed T wave negativity in 6 per cent of 100 student nurses in CF2, 1 per cent in CF3 and 0 per cent in CF4. DUPUY⁶³ considered T wave abnormalities in apparently normal soldiers to be functional variants because they were reversible following rest and sedation and suggested that these variants might be due to cardiac rotation or to anxiety associated with a rapid cardiac rate. THOMPSON⁶⁴ described interesting electrocardiographic changes which occurred during hyperventilation in individuals with anxiety neurosis. In a similar manner, KATZ⁶⁵ have observed reversible T wave changes in nervous individuals. GRAYBIEL, STARR and WHITE⁶⁶ have also noted ST interval and T wave changes resulting from the inhalation of tobacco smoke. WIENER, RIOS and MASSUMI⁶⁷ emphasized the racial predilection of the Negro for a benign type of T wave inversion with elevated RS-T segment which represents an aberration of the repolarization process. WASSERBURGER and associates⁶ have shown that a majority of patients with early repolarization develop precordial T wave inversion with brief hyperventilation. However, one's mental and emotional experiences in society by which his heart action is sensitively affected differ markedly by the race to which he belongs, and in addition anxiety state and hyperventilation are intimately related through the autonomic nervous system. Therefore, the magnitude of the problem and the difficulty for a precise differentiation from the pathologic mimics are great. These T wave abnormalities might be also the result of associated alkalosis or might be due to coronary vasoconstriction. As is well known, T wave inversion occurs also

during central nervous system diseases⁶⁸⁻⁷¹. Observations in this investigation rather indicate the presence of separate cardiac muscle masses for the negative and positive T waves, respectively. An increase in the negativity of the T wave in the right precordial leads after dash seems to indicate that this is due more to right ventricular strain than to alterations of the heart position. In other words, sympathicotonia induced by dash increases the electromotive forces of the muscle mass responsible for the T wave negativity in the right precordial leads.

11) Bifurcated T waves

According to FLEISCH²¹, the bifurcated T wave (die 2-gipflige T-Welle) can be differentiated from the TU fusion wave (die TU Verschmelzungswelle^{72,73}) because the bifurcated T wave is followed by separate U and P waves. He divided the bifurcated T wave into three types: the first type with two peaks of the same amplitude, the second type with the first peak higher than the second and the third type with the second peak higher than the first. However, these three types are mutually interchangeable. He stated further that the bifurcated T wave corresponds to a tendency to negativity of the T wave, often being a transitional stage between an upright and a diphasic or inverted T wave. The bifurcated T wave is found in subjects with a normal circulatory system as well as those with coronary insufficiency, but their incidence is highest in young patients with pronounced imbalance of the autonomic nervous system. FLEISCH²¹ who recorded leads I, II, V2, V4 and V6 before, during and after exercise in 630 normal and abnormal subjects observed a bifurcated T wave in 14.5 per cent. Its percentage occurrence was 27.8 per cent in a group of "vegetative Dystonien", 13.6 per cent in a group of "Coronarinsuffizienzen", 6.5 per cent in a group of "Kreislaufgesunde" and 7.3 per cent in a group of "fragliche Herzbeteiligung". The occurrence of bifurcated T waves was most frequent in V4 (10.5 per cent) and then in V2 (6.3 per cent). According to FLEISCH²¹, the percentage occurrence of bifurcated T waves was decreased during exercise and increased one minute after exercise. KIESSLING, SCHAAF and LYLE²² who studied T wave

changes in the electrocardiograms of normal individuals considered a notched (bifurcated) T wave in V2 as normal because, as they stated, this is a transitional notching from a negative to a positive T wave. However, they considered a notched T wave in V5 as abnormal. FRIESE⁷⁴ considered all bifurcated T waves as abnormal. According to HOLZMANN⁷⁵, however, the negative portion that lies between the two peaks is due to a small preterminal negativity of the T wave. On account of the presence of a larger portion of the T wave positivity, a usual nega-

tive or diphasic T wave can not be formed. As shown in Fig. 9, a bifurcated T wave in the right precordial leads appears to the left of a negative or a positive-negative diphasic T wave and to the right of a positive T wave. Ordinarily, the order of transition of the T wave from the right to the left in the right precordial leads is as follows: a completely negative, a positive-negative diphasic, a bifurcated and a completely positive T wave. Also in the left precordial leads, a bifurcated T wave is found in a small number of patients with left ventric-

Fig. 9. Bifurcated T waves in the right to mid-precordial leads.

A) The T wave is inverted in V1, diphasic (positive-negative) in V2 and bifurcated in V3. The height of the 1st and 2nd peaks of this bifurcated T wave is nearly the same.

B) The T wave is negative in V1, diphasic (positive-negative) in V2 and bifurcated in V3. The 1st peak of this bifurcated T wave is higher than the 2nd one.

C) The T wave is inverted in V1, bifurcated in V2 and positive in V3. The 2nd peak of this bifurcated T wave is higher than the 1st one. In V2, the 2nd peak of this bifurcated T wave simulates the U wave. However, the QT interval in V4 is 0.42 sec., and the interval between the R wave to the 2nd peak of the T wave in V2 0.38 sec. Therefore, the 2nd peak is well within the QT interval and not the U wave. In all cases, a bifurcated T wave appears to the left of a negative or a diphasic (positive-negative) T wave and to the right of a positive T wave.

A) T. K., a 65-year-old male with essential hypertension

B) H. A., a 56-year-old male with essential hypertension

Fig. 10. Bifurcated T waves in coronary insufficiency of the left ventricle. A bifurcated T wave is observed in V4 and V5 in case A and in V2 in case B. In both cases, a bifurcated T wave is observed to the left of a positive T wave and to the right of a diphasic (negative-positive) T wave.

Fig. 11. Synthesis of a bifurcated T wave by summation of negative and positive T waves. A summation of a positive T wave with a blunt peak and a negative T wave with a sharply peaked nadir forms a bifurcated (positive-negative-positive) T wave.

ular hypertrophy and coronary insufficiency. FLEISCH²¹ found a bifurcated T wave in only 0.6 per cent of a total of 633 electrocardiograms in V6. As shown in Fig. 10, when the ST segment in the left precordial leads is simultaneously depressed with an initial negativity of the T wave, as observed in the so-called strain pat-

tern, a bifurcated T wave occurs to the left of a positive T wave and to the right of a negative-positive diphasic or a completely inverted T wave. Usually, the order of transition of the T wave from the right to the left in the left precordial leads is as follows: a completely positive, a bifurcated, a negative-positive diphasic and a completely negative T wave. It seems to be likely that a bifurcated T wave is a manifestation of an increased non-uniformity of repolarization process in the heart muscle. As illustrated in Fig. 11, a bifurcated T wave can be explained as a fusion wave caused by summation of opposing electricities of a negative and a positive T wave.

12) U wave and TU fusion

SCHMID and ENGLER⁷⁶ studied the U wave in athletes. They observed an increase in the height of the U wave and a more frequent occurrence of the U wave in a group of cardiovascular diseases than in a group of athletes. With these observations, they concluded that the U wave can not be employed as the criterion for the degree of training. BLUMBERGER⁷⁷ also observed an increase in the height of the U wave and a more frequent appearance of the U wave in those with hypertension and myocardial lesions than in normal controls. According to

HOLZMANN⁷⁸, the U wave is probably produced by a re-entrance of the K ion which has emerged out of the cell at the end of a systole. In overloading of the left ventricle or in left ventricular hypertrophy, the U wave appears in lead I and in the left precordial leads. In most cases, a heightening of the U wave is associated with other signs of hypertrophy and left axis deviation, and not infrequently an appearance or a heightening of the U wave is the first sign of abnormal electrocardiograms. According to GOLDBERGER⁷⁹, the U wave indicates the return of the stimulated ventricles to the resting state. Therefore, the complete return of the heart to the resting state occurs at the end of the U wave. In many cases, a positive U wave which follows an isoelectric T wave can mimic a positive T wave with an increase in the QT interval. The end of the T wave and the beginning of the U wave can overlap and become a TU fusion wave. HOLZMANN⁷⁸ classified a TU fusion wave into three types: a TU fusion wave with a summa-

Fig. 12. Synthesis of a TU fusion wave by summation of T and U waves.

A summation of a positive U wave and a downstroke of a positive T wave prolongs the duration of the upstroke and shortens that of the downstroke of the T wave with a consequent delay and heightening of the peak of the T wave. However, a concave RS-T elevation with a sharply peaked T wave is occasionally followed by another U wave. This may indicate that the cardiac muscle mass responsible for the U wave can be more than one, i.e., the posterobasal portion of the left ventricle, papillary muscles, the interventricular septum etc.

tion peak, a TU fusion wave with a dominant T component and a TU fusion wave with a dominant U component. In a given heart, there may be separate muscle masses which are responsible for a negative T wave, a positive T wave and a U wave, respectively. A shortening of the QU interval with an unchanged QT interval causes a later onset of the peak of the T wave with a gentle upstroke and a steep downstroke of a TU fusion wave as illustrated in Fig. 12. An increase or a decrease in the electromotive forces of the heart muscles which are responsible for these waves causes an increase or a decrease in the amplitude of these waves, and an accelerated or delayed completion of repolarization of these heart muscles may cause a fusion or a separation of these waves. Occasionally, a concave RS-T elevation with a sharply peaked TU fusion wave is followed by another U wave. This may indicate that the muscle mass responsible for the U wave can be more than one. It may be the posterobasal portion of the left ventricle, papillary muscles⁸³, the interventricular septum⁷⁹ etc. These changes can occur through an imbalance in the autonomic nervous system caused by persistent mental tension, continuous physical training or by sedentary daily life^{55,80}.

13) SV1 and RV5

Among many sports, the loading of the cardiovascular system is undoubtedly greatest in endurance athletics. As a result of intensive training over many years, the circulatory adaptation of a champion-class endurance athlete approaches its upper limit. The circulatory adaptation is characterized by an increase of the roentgenologic heart volume, by increased QRS and T amplitudes and by an increase of the mean spatial QRS and T vectors¹⁶. An increased roentgenologic heart volume of endurance athletes has been ascribed to an increase in the systolic residual blood volume, and a decreased roentgenologic heart volume after strenuous exercise to a decrease in the diastolic heart volume by a complete ejection of the residual blood volume. According to KITAMURA⁴, the roentgenologic heart volume of the Japanese athletes was largest in marathon runners, followed by champions of a bicycle race and wrestlers. These are manifestations of phy-

siological adaptation with an increased distensibility of the heart muscle caused by a long-term training through the autonomic nervous system. This increase in the distensibility of the heart muscle with an increase in the electrical activity may be the cause of a marked increase in the amplitude of SV1 and RV5 observed in endurance athletes in this investigation. The fact that the amplitude of SV1 and RV5 was little affected by maximal exercise seems to indicate that the increases in SV1 and RV5 are due to chronic effects of exercise such as regulative dilatation and adaptive hypertrophy. According to MIZUNO⁶¹, SV1 averaged 9.36 mm in young non-athletic males and 8.36 mm in young non-athletic females. RV5 averaged 16.61 mm in young non-athletic males and 12.90 mm in young non-athletic females. Also in this investigation, RV5 was significantly higher in male controls (18.4 mm) than in female (13.3 mm).

14) TV5 and TV5/RV5 ratio

According to GROSS⁸¹, a negative correlation was proved to exist between the length of the horizontal ST segment and the height, duration and surface area of the T wave and a positive correlation between the length of the horizontal ST segment and QT interval. According to LINZBACH⁸², a critical weight of the heart (kritisches Herzgewicht) is 500 g. In the athletic heart of less than 500 g, an increase in weight

is due to a growth with physiological harmony (eutrophy) and there are no signs of relative coronary insufficiency. According to MIZUNO⁶¹, TV5 averaged 5.74 mm in young males and 4.17 mm in young females. These are nearly the same as those observed in control groups in this investigation. A high T wave in endurance athletes observed in this investigation may partly be due to a TU fusion which is usually associated with the concave RS-T elevation. The electrocardiographic characteristics of eutrophy of the athletic heart is an increase in the sum of SV1+RV5 with a normal TV5/RV5 ratio. An average TV5/RV5 ratio was 0.33 in endurance athletes, 0.30 in male controls and 0.45 in female controls. In left ventricular hypertrophy with an isoelectric T wave, it approaches zero and even becomes negative when the T wave is inverted. Therefore, the heightening of TV5 as well as SV1 and RV5 is due to a growth of the heart with physiological harmony (eutrophy).

15) Stages of athletic hearts and their electrocardiographic manifestations

Intensive training may first cause alterations in the equilibrium of the autonomic nervous system. Further load to the cardiovascular system may then cause regulative dilatation and adaptive hypertrophy (eutrophy). Further overload to this state of eutrophy may cause abnormal dilatation and hypertrophy which is

Stages	I	II	III	IV	V
Equilibrium in the autonomic nervous system	Normal	Altered	Altered	Altered	Altered
Cardiac size	Normal	Normal	Eutrophic and regulatively dilated	Hypertrophic and abnormally dilated	Hypertrophic and abnormally dilated
Organic lesions	Absent	Absent	Absent	Absent	Present
ECG manifestations		Bradycardia	Concave RS-T elevation increased (SV1+RV5) First degree A-V block Second degree A-V block of Wenckebach type Right axis deviation of the P wave Notched P waves in the limb leads Incomplete RBBB Lowered TV5 Decreased TV5/RV5 ratio Ischemic ST depression		

Fig. 13. Stages of athletic hearts and probable ECG manifestations

to some extent irreversible. Continuation of this state of abnormal dilatation and hypertrophy may eventually cause histopathological changes which are irreversible. A diagrammatic representation of stages of athletic hearts and their probable electrocardiographic manifestations are given in Fig. 13.

SUMMARY

Electrocardiograms were taken before and after 100 meter dash from 25 endurance athletes of the Self Defence Forces, all males aged 19 to 31. As controls, electrocardiograms were recorded also from 20 ordinary members of the Self Defence Forces, all males aged 19 to 32 and from 27 student nurses, all females aged 18 to 20.

I. Electrocardiograms at rest

1) An average heart rate was 55.5/min in endurance athletes, 65.2/min in male controls and 63.5/min in female controls.

2) PII averaged +1.1mm in height in endurance athletes, +0.6 mm in male controls and +0.7 mm in female controls.

3) An average P wave axis was +48.9 degrees in endurance athletes, +40.2 degrees in male controls and +35.2 degrees in female controls.

4) A notched P wave in one or more of the standard and unipolar extremity leads was observed in 72.2 per cent of endurance athletes, 11.1 per cent of male controls and 0 per cent of female controls.

5) Respiratory arrhythmia was observed in 10.0 per cent of endurance athletes, 0 per cent of male controls and 18.5 per cent of female controls.

6) Nodal rhythm was observed in one case of female controls.

7) First degree A-V block was observed in 10.0 per cent of endurance athletes, 0 per cent of male controls and 7.4 per cent of female controls.

8) Second degree A-V block of Wenckebach type was observed in 10.0 per cent of endurance athletes and 0 per cent of both male and female controls.

9) Ventricular premature beats were observed in one case of each group.

10) A notched QRS complex in V1 was

found in 40.0 per cent of endurance athletes, 52.6 per cent of male controls and 33.3 per cent of female controls.

11) Incomplete RBBB patterns in V1 were observed in 35.0 per cent of endurance athletes, 21.0 per cent of male controls and 3.7 per cent of female controls.

12) A negative or diphasic T wave in V1 was observed in 95.0 per cent of endurance athletes, 73.6 per cent of male controls and 81.6 per cent of female controls, that in V2 36.8 per cent of endurance athletes, 5.3 per cent of male controls and 0 per cent of female controls and that in V3 15.8 per cent in endurance athletes and 0 per cent in both male and female controls.

13) A bifurcated (notched) T wave in the mid-precordial leads was found in 26.3 per cent of endurance athletes, 0 per cent of male controls and 7.4 per cent of female controls.

14) The percentage occurrence of the U wave in V4 was 28.9 per cent in endurance athletes, 76.3 per cent in male controls and 64.8 per cent in female controls, that in V5 10.5 per cent in endurance athletes, 44.7 per cent in male controls and 44.5 per cent in female controls and that in V6 5.3 per cent in endurance athletes, 13.2 per cent in male controls and 14.8 per cent in female controls.

15) A concave RS-T elevation in the left precordial leads was found in 80.0 per cent of endurance athletes, 31.6 per cent of male controls and 11.1 per cent of female controls.

16) The sum of SV1+RV5 averaged 50.3 mm in endurance athletes, 27.4 mm in male controls and 19.8 mm in female controls.

17) TV5 averaged 12.4 mm in height in endurance athletes, 5.0 mm in male controls and 5.8 mm in female controls. The TV5/RV5 ratio averaged 0.33 in endurance athletes, 0.30 in male controls and 0.45 in female controls.

II. Electrocardiographic changes after 100 meter dash in endurance athletes

1) An average heart rate increased from 55.5/min to 73.0/min. In one case, it was decreased by 17/min.

2) PII increased from +1.1 mm to +1.4 mm and PIII from +0.4 mm to +0.7 mm in height. The percentage occurrence of a notched P wave remained unaltered.

3) An average P wave axis increased from +48.9 to +52.0 degrees. The percentage occurrence of the P wave axis between +61 to more than 90 degrees increased from 33.3 per cent to 61.2 per cent. The P wave axis deviated to the right by more than 10 degrees in 50.0 per cent.

4) Respiratory arrhythmia increased from 10.0 to 35.0 per cent.

5) In one case, nodal rhythm appeared immediately after dash and returned to sinus rhythm during postexercise ECG recording.

6) Both first degree A-V block and second degree A-V block of Wenckebach type disappeared after 100 meter dash.

7) The percentage occurrence of a notched QRS complex in V1 decreased from 40.0 to 30.0 per cent, and that of incomplete RBBB pattern in V1 increased from 35.0 to 45.0 per cent.

8) The percentage occurrence of a negative or diphasic T wave increased from 95.0 to 100.0 per cent in V1, from 36.8 to 65.3 per cent in V2, from 15.8 to 30.2 per cent in V3 and from 0 to 13.1 per cent in V4.

9) A bifurcated T wave in mid-precordial leads increased from 26.3 to 43.5 per cent.

10) The percentage occurrence of the U wave increased from 79.0 to 84.7 per cent in V2, from 63.2 to 76.0 per cent in V3, from 28.9 to 45.7 per cent in V4 and from 10.5 to 17.4 per cent in V5.

11) The percentage occurrence of a concave RS-T elevation in the left precordial leads was decreased from 80.0 to 43.5 per cent because of ST segment depression, but the configuration of the concave RS-T segment was retained in all cases.

12) The ST segment in V6 was depressed from +0.52 to +0.10 mm on an average.

13) The sum of SV1+RV5 remained almost unaltered.

14) An average TV5 was lowered from +12.4 to +8.3 mm. Consequently, the TV5/RV5 ratio was decreased from 0.33 to 0.22 on an average.

15) Stages of athletic hearts and their electrocardiographic manifestations were discussed.

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