Clinical electrocardiographic studies of bifid T waves

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SUMMARY In 129 electrocardiograms from 129 patients showing bifid T waves as well as U waves the intervals from the beginning of the QRS complex to the two T wave apices (QaT_1 , QaT_2), to the end of the T wave (QeT), and to the apex of the U wave (QaU) were measured. Eighty additional electrocardiograms from matched control subjects showing single peaked T waves were also studied. The precordial distribution of bifid T waves was assessed by calculating lead prevalence indices. This index progressively increased from 2.15 in the age range 20-29 years to 3.72 in the age range 60-69 years, and was significantly higher in patients with left ventricular hypertrophy and ischaemia (4.04) than in those with otherwise normal electrocardiograms (2.35). Thus older age and left ventricular pathology were accompanied by a more leftward location of bifid T waves. Exercise accentuated the bifid nature of the T wave in 12 of 18 patients with otherwise normal electrocardiograms, and diminished it in 11 of 19 cases with left ventricular hypertrophy and ischaemia. When 41 otherwise normal tracings showing bifid T waves were compared with those of 42 matched controls showing single peaked T waves, the QTc was longer and the eTaU interval shorter in the group with bifid T waves. Similarly, 40 patients with left ventricular hypertrophy and ischaemia showing bifid T waves had longer QTc and shorter eTaU intervals than 38 patients with the same diagnosis with single peaked T waves.

These findings suggest that right precordial bifid T waves in younger patients with otherwise normal electrocardiograms probably result from delayed right ventricular repolarisation, whereas left precordial bifid T waves in older patients with left ventricular hypertrophy and ischaemia may indicate repolarisation delay in the ischaemic left ventricle.

Although the genesis of any electrocardiographic abnormalities should ultimately be confirmed by experimental studies, theoretical deductions and various clinical observations provide useful data. Bifid (notched or split) T waves are frequently seen in healthy children and young adults^{1,2} and hence are often regarded as an electrocardiographic normal variant. Nevertheless, they also occur in certain pathological conditions, including organic heart diseases,^{3,4} disorders of the central nervous system,⁵ and alcoholism.^{6,7} These findings suggest that the electrophysiological mechanisms responsible for the production of bifid T waves are not necessarily identical in individual cases. Thus the clinical significance of bifid T waves has not

been established, and the genesis of this waveform remains unknown except that a delayed right ventricular repolarisation may be a possible explanation in children.^{1,2} Our experimental study using isolated perfused rabbit hearts showing bifid T waves appeared to suggest that asynchronous repolarisation of different regions of the ventricles—particularly the right after the left ventricle—was the mechanism of such a waveform,⁸ which supports the above clinical concept.

On the other hand, the previous studies of the clinical features of bifid T waves did not report the precordial distribution of bifid T waves in detail and made no attempt to correlate their presence with the QTc or predicted QT interval. Hence, the present study was carried out to determine further the electrocardiographic characteristics of bifid T waves by paying special attention to the precordial localisation of this waveform in different patient groups and to the effects of bifid T waves on ventricular repolarisation time (QT interval).

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Accepted for publication 13 March 1984

Patients and methods

We reviewed a total of 4000 consecutive electrocardiograms and selected those tracings in which the T wave had two peaks in addition to an identifiable U wave. Tracings showing atrial fibrillation were excluded. The electrocardiograms of 113 patients (26 men, 78 women, nine children) were thus obtained for initial study, and only one representative tracing being selected for each patient. For each tracing the precordial leads showing bifid T waves were noted and the following intervals measured to the nearest 1/100 s with a magnifying glass and a pair of calipers: basic cycle length (RR interval) and QaT_1 , QaT_2 , aT_1aT_2 , QeT and QaU intervals (Fig. 1). Care was taken to select portions of the tracings showing a stable RR interval. Since the apparent end of the T wave shows a slight variation in individual leads, the QeT interval was determined by the longest interval in the three to six simultaneously recorded precordial leads. QaT_1 , QaT_2 , and QaU intervals were measured in leads showing the most prominent bifid T and U waves respectively. Other electrocardiographic abnormalities were also identified. The precordial distribution of bifid T waves was quantitatively assessed by calculating the lead prevalence index (see results).

The 113 patients were grouped according to the electrocardiographic diagnosis using the Minnesota code.⁹

Group 1—41 patients had electrocardiograms which were considered to be within the normal limits except for the presence of bifid T waves.

Group 2—Nine patients satisfied voltage criteria for left ventricular hypertrophy but had no associated ischaemic ST-T changes.

Group 3—17 patients showed signs of myocardial

Fig. 1 Diagram showing the various intervals measured.

ischaemia either in the resting electrocardiogram or during exercise.

Group 4—24 patients showed evidence of left ventricular hypertrophy and ischaemia. Since our preliminary study of these 113 cases showed the most pronounced differences in measurements to be between those with otherwise normal electrocardiograms (group 1) and those with evidence of left ventricular hypertrophy and ischaemia (group 4) 16 additional cases were added to the latter group, giving a total of 40 cases.

Group 5—22 patients had miscellaneous electrocardiographic abnormalities such as evidence of atrial disease, right or left axis deviation, right bundle branch block, combined ventricular hypertrophy, and the Wolff-Parkinson-White syndrome.

Control groups

To determine whether or not the presence of bifid T waves actually delays ventricular repolarisation, 42 age and sex matched control subjects with normal electrocardiograms showing only single peaked T waves in all 12 leads (group 1a) were selected for comparison with group 1. Similarly, 38 cases of left ventricular hypertrophy and ischaemia having only single peaked T waves (group 4a) were chosen as a matched control for group 4. Thus a total of 209 electrocardiograms were analysed.

CLINICAL FEATURES OF PATIENT GROUPS

Group 1—Of the 41 patients in group 1 with otherwise normal electrocardiograms two had mild hypertension and one each atrial septal defect, ventricular septal defect, and mitral regurgitation. All these five patients were aged <30 years and were female except for a 6 year old boy with atrial septal defect. The remaining 36 cases had miscellaneous, non-cardiac problems such as gastrointestinal disorders, obesity, pleurisy, tonsillitis, etc. Most patients underwent the electrocardiographic examination on their first visit to hospital and hence were taking no medication except for six patients (three taking diuretics, two minor tranquillisers, and one antihypertensive agents). The mean age was 31.1 years.

Group 1a, the age and sex matched normal control for group 1 (mean age 34.1 years), had various non-cardiac diagnoses and were not receiving any cardioactive drugs.

Group 2—Of the nine patients in group 2, six had essential hypertension, two hyperthyroidism, and one gastrointestinal disturbances. Their mean age was 53.0 years. At the time of the electrocardiographic recording three of the six patients with hypertension were taking antihypertensive or diuretic drugs and three were not. No echocardiographic or angiographic evidence for ventricular hypertrophy was available in

these patients, however.

Group 3—Of the 17 patients, eight had hypertension, five ischaemic heart disease, two severe anaemia, and one each cor pulmonale resulting from pulmonary tuberculosis and autonomic imbalance. Their mean age was 46.1 years.

Group 4—Of the 40 patients in group 4, 36 had hypertension, of which seven were associated with ischaemic heart disease, three with congestive heart failure, two with diabetes mellitus, and two with cerebrovascular accidents. Of these 36 patients, 25 were taking various antihypertensive or beta or alpha adrenergic blocking agents, diuretics, or calcium antagonists, either singly or in combination, when the recordings were made; 11 patients were taking no medication at the time of the recording. The remaining four patients had miscellaneous non-cardiac diseases.

The 38 patients in group 4a included 22 patients with hypertension, four with ischaemic heart disease, and five with cardiomyopathy. Seven patients were being treated for non-cardiac illnesses at the time of the recording. Their mean age was 57.4 years. No cases of significant electrolyte imbalance were noted in any of the four groups in whom such data were available.

TIMING OF APICES OF BIFID T WAVES

The relative timing of the two apices of the bifid T wave was analysed. Firstly, the predicted timing of the apex of a single peaked T wave (predicted QaT interval) (Fig. 1) was determined in each tracing, based on the sex and RR interval of the basic sinus rhythm, using the values for normal adults.¹⁰ The difference between this predicted QaT and the measured QaT₁ interval (Fig. 1) was calculated as a percentage of the predicted QaT and expressed as

$\Delta 1 = [((Q - aT_1) - (\text{predicted } QaT)) / (\text{predicted } QaT)] \times 100$. A similar measurement for the second apex of the bifid T wave was expressed as $\Delta 2$.

Results

PRECORDIAL DISTRIBUTION OF BIFID T WAVES

According to age

It is well known that bifid T waves are most commonly seen in precordial leads, and the present study confirmed this finding. The precordial distribution of bifid T waves showed that they became more prevalent in the lateral precordial leads with increasing age (Table 1). The different age groups were compared by calculating the lead prevalence index in the following manner. The lead number (for example, 1 for lead V1) was multiplied by the number of cases showing bifid T waves in that lead, for all six leads; the product was then divided by the total number of leads in which bifid T waves were seen to give the lead prevalence index (Table 1). Thus this index represents, in the simplest way, the "average" location of bifid T waves in precordial leads (for instance, a value of 2.50 would indicate a site exactly midway between leads V2 and V3), and enabled us quantitatively to compare the precordial distribution between different patient groups.

Table 1 shows that in the younger age groups bifid T waves were most often seen in lead V2 and were seldom found in V4–V6. In the older age groups more patients showed bifid T waves in the lateral precordial leads, occurring in V6 only in the age groups 50–59 and 60–69 years. The lead prevalence indices for the age groups 30–39, 40–49, 50–59, and 60–69 years were 2.37, 3.20, 3.38, and 3.94 respectively, with the last three values being significantly higher ($p < 0.01$) than the index for the age group 20–29 years.

Table 1 Number of patients with bifid T waves in precordial leads according to age and to electrocardiographic diagnosis. Lead prevalence index was calculated by multiplying the lead number by the number of leads showing bifid T waves for that lead and dividing the product by the total number of leads in which bifid T waves were seen

	<i>No of patients</i>	<i>Precordial leads</i>						<i>Product/ No of leads</i>	<i>Lead prevalence index</i>
		<i>V1</i>	<i>V2</i>	<i>V3</i>	<i>V4</i>	<i>V5</i>	<i>V6</i>		
		<i>Age group (yr)</i>							
20-29	12	3	10	4	1	0	0	39/18	2.17
30-39	21	1	20	7	1	1	0	71/30	2.37
40-49	29	0	18	15	13	8	0	173/54	3.20
50-59	25	0	11	18	15	5	3	176/52	3.38
60-69	17	0	2	11	11	9	2	138/35	3.94
		<i>Electrocardiographic diagnosis*</i>							
Group 1	41	3	38	19	3	0	0	148/63	2.35
Group 2	9	0	5	4	3	3	0	49/15	3.27
Group 3	17	0	10	16	10	3	0	123/39	3.15
Group 4	40	0	1	17	26	14	4	251/62	4.05

*Group 1, otherwise normal ECG; group 2, voltage criteria for left ventricular hypertrophy (LVH) alone; group 3, ST-T changes suggesting myocardial ischaemia; group 4, ECG evidence of LVH and ischaemia.

According to electrocardiographic diagnosis

The precordial distribution of bifid T waves in the various diagnostic groups is shown in Table 1, which excludes the 24 cases in group 5 with miscellaneous electrocardiographic abnormalities. Patients in group 1 with otherwise normal electrocardiograms showed a precordial distribution of bifid T similar to that in the age groups 20–29 and 30–39, with a lead prevalence index of 2.35. On the other hand, patients in both group 2 and group 3 had indices which were significantly higher than that in group 1 ($p<0.01$). The highest lead prevalence index of 4.05 was obtained in group 4.

TIMING OF APICES OF T WAVES

In the 41 patients in group 1 with otherwise normal electrocardiograms, $\Delta 1$ measured -14.3 (0.8)% (mean (SD)), indicating that the first peak of the bifid T wave (aT_1) occurred 14.3% earlier than the expected T wave apex (Fig. 2). $\Delta 2$ was 11.3 (1.0)% and hence the second peak of the T (aT_2) was delayed from the expected T wave apex by an amount similar to that by which aT_1 was premature. As a result $\Delta 1+\Delta 2$, or the ratio of the aT_1aT_2 interval to the predicted QaT interval, was 25.6%. In the patients in group 2 satisfying voltage criteria for left ventricular hypertrophy alone and those in group 3 with signs of ischaemia the aT_1 appeared less prematurely and the aT_2 occurred after greater delays than in those in group 1, although the differences were statistically not significant. $\Delta 1+\Delta 2$ again corresponded to 26.9 and 26.6% of the predicted QaT interval in these two groups. On the other hand, the patients in group 4 with signs of left ventricular hypertrophy and ischaemia had a significantly smaller $\Delta 1$, and a significantly greater $\Delta 2$ than those in group 1. This implies that in this group the appearance of aT_1 was less premature (-7.0 (1.1)%), whereas that of aT_2 was greatly delayed ($+24.7$ (1.5)% in relation to the tim-

Fig. 2 Comparison of the timing of two T wave apices between the four groups of patients with otherwise normal electrocardiograms (group 1), those with voltage criteria for left ventricular hypertrophy (group 2), those with ischaemic changes at rest or during exercise (group 3), and those with left ventricular hypertrophy and ischaemia. $\Delta 1 = [((QaT_1) - (Pred QaT)) / (Pred QaT)] \times 100$; $\Delta 2 = [((QaT_2) - (Pred QaT)) / (Pred QaT)] \times 100$ (Pred QaT = interval from the beginning of the QRS complex to the predicted apex of a single-peaked T wave in normal subjects.)

ing of a single peaked T wave. $\Delta 1+\Delta 2$ in this group was 31.7% of the predicted QaT interval indicating a wider separation of the two apices. In view of these findings various measurements were compared between groups 1 and 4 (Table 2). These two groups showed significantly different values for most indices.

CORRELATION WITH INVERTED T WAVES IN ADJACENT LEADS

The correlation between bifid T waves and inverted T

Table 2 Comparison of various measurements between patients in group 4 with left ventricular hypertrophy and ischaemia and those in group 1 with otherwise normal electrocardiograms both showing bifid T waves. Values are means (SD)

	Group 4 (n=40)	Group 1 (n=41)	Difference	p value
Age (yr)	55.4(7.4)	31.8(13.7)	+23.8	<0.001
Intervals:				
RR (0.01 s)	97.4(16.7)	90.7(13.9)	+6.7	<0.05
QeT (0.01 s)	45.9(4.2)	41.1(3.2)	+4.8	<0.001
QTc (0.01 s)	46.6(3.0)	43.4(2.2)	+3.2	<0.001
QaU (0.01 s)	55.1(4.1)	46.6(3.0)	+8.5	<0.001
eTaU (0.01 s)	9.2(1.8)	8.2(1.5)	+1.0	<0.01
Change from predicted QeT (%)	+13.6(6.7)	+2.8(5.4)	+10.8	<0.001
Change from predicted QaU (%)	+10.8(5.7)	+0.3(5.1)	+10.5	<0.001
QaT ₁ (0.01 s)	29.0(2.7)	26.7(2.2)	+2.3	<0.001
QaT ₂ (0.01 s)	39.0(4.1)	34.7(3.1)	+4.3	<0.001
aT ₁ aT ₂ (0.01 s)	10.1(2.7)	8.0(2.0)	+2.1	<0.001
$\Delta 1$ for aT ₁ (%)	7.0(1.1)	14.3(0.8)	-7.3	<0.001
$\Delta 2$ for aT ₂ (%)	24.7(1.5)	11.3(1.0)	+13.4	<0.001

waves was also studied in groups 1 and 4. Of the 41 patients in group 1 three had bifid T waves in V1 alone. In the remaining 38 patients with bifid T waves in V2 (alone or also in V3–V4) T waves in V1 were inverted in 33 and positive in only five. Of the 40 patients with left ventricular hypertrophy and ischaemia (group 4), four had bifid T waves in V6. In the remaining 36 cases with bifid T waves in V5 or more rightward leads T waves in leads further to the left of those showing bifid T waves were inverted in nine, diphasic in 11, and flattened in 11, whereas five patients had ST depression without these T wave changes.

EFFECTS OF EXERCISE ON BIFID T WAVES

The effects of exercise on bifid T waves were studied in 56 patients who underwent double Master's exercise tests and had satisfactorily stable electrocardiograms immediately after exercise. With exercise the RR interval was shortened by 13.6%. As expected, the QeT, QaT₁, QaT₂, and QaU intervals were all significantly shortened, by 8.3, 8.7, 10.9, and 8.7% respectively. Although the ratios of QaT₁ to QeT and of QaT₂ to QeT were essentially unchanged, a somewhat greater shortening of QaT₂ than QaT₁ resulted in a slight (16.8%) but significant ($p < 0.01$) decrease in the aT₁aT₂ interval.

In addition to these interval changes exercise often altered the T wave amplitude (Fig. 3). In case 1 (a 32 year old woman with an otherwise normal electrocardiogram) the control tracing showed a barely perceptible bifid T wave in lead V2. After exercise the bifid nature became more prominent in V2, and a notch on the downstroke of the T wave also appeared in V3. In case 2 (a 58 year old man with signs of left ventricular hypertrophy and anterolateral wall ischaemia) a bifid T wave was noted in V5 (and probably also V6) in the control tracing. The electrocardiogram recorded immediately after exercise showed a greater depression of ST segment and increased T wave amplitude in V5 and V6 with a disappearance of the bifid nature. In 18 patients in group 1 undergoing exercise test bifid T waves became more prominent in 12, were unchanged in two, and became less prominent in four. In contrast, exercise in 19 patients in group 4 made the bifid configuration more evident in five and less evident in 11, with essentially no change in the remaining three. The difference between these two groups was statistically significant by Fisher's exact test ($p < 0.02$), possibly suggesting different mechanisms involved in producing bifid T waves.

COMPARISON WITH PATIENTS SHOWING SINGLE PEAKED T WAVES

The various interval measurements in the 42 age and

sex matched controls with normal electrocardiograms showing single peaked T waves (group 1a) were com-

Fig. 3 Electrocardiograms showing changes in bifid T waves with exercise (a) in a patient with an otherwise normal electrocardiogram and (b) in one with signs of left ventricular hypertrophy and ischaemia.

Table 3 Comparison of various measurements between group 1 with normal electrocardiograms showing bifid T waves and group 1a with normal electrocardiograms showing single peaked T waves and between group 4 with left ventricular hypertrophy showing bifid T waves and group 4a with left ventricular hypertrophy showing single peaked T waves. Values are means (SD)

	Group 1 (n=41)	Group 1a (n=42)	Difference	p value	Group 4 (n=40)	Group 4a (n=38)	Difference	p value
Age (yr)	31.8(13.7)	34.1(13.2)	-2.3	NS	55.4(7.4)	57.4(11.3)	-2.0	NS
Intervals:								
RR (0.01 s)	90.7(13.9)	88.2(15.6)	+2.5	NS	97.4(16.7)	83.1(22.1)	+14.3	<0.005
QeT (0.01 s)	41.1(3.2)	38.3(4.1)	+2.8	<0.001	45.9(4.2)	39.6(4.3)	+6.3	<0.001
QTc (0.01 s)	43.4(2.2)	40.6(0.7)	+2.8	<0.001	46.6(3.0)	43.4(2.5)	+3.2	<0.001
QaU (0.01 s)	49.2(3.0)	49.0(4.1)	+0.2	NS	55.1(4.1)	51.6(5.1)	+3.5	<0.001
eTaU (0.01 s)	8.2(1.5)	10.6(2.0)	-2.4	<0.001	9.2(1.8)	12.0(2.3)	-2.8	<0.001
Change from predicted QeT (%)	+2.8(5.4)	-1.4(5.6)	+4.2	<0.001	+13.6(6.7)	+5.2(6.0)	+8.4	<0.001
Change from predicted QaU (%)	+0.3(5.1)	0.0(5.4)	+0.3	NS	+10.8(5.7)	+6.6(7.2)	+4.0	<0.01

pared with those in the 41 patients in group 1 (Table 3). There were no significant differences between these two groups in age and RR interval. The QeT as well as the QTc interval and the difference from the predicted QeT interval were, however, all significantly longer in the patients in group 1 with bifid T waves than in those in group 1a with single peaked T waves. The QaU intervals, on the other hand, were almost identical in these two groups, and their values corresponded with the predicted values.

The results of the 40 patients with evidence of left ventricular hypertrophy and ischaemia with bifid T waves (group 4) were compared with those of 38 matched controls with single peaked T waves (group 4a) (Table 3). The QTc interval was significantly longer ($p<0.01$) in patients with bifid T waves than in those without. In both groups, the QeT as well as QaU interval were greater than their predicted values. The degree of prolongation of the QaU interval was, however, less than that of QeT in the group with bifid

T waves, whereas the opposite was true in the group with single peaked T waves. As a result, the eTaU interval was shorter in the former than in the latter.

Since the mean RR interval in group 4 was significantly longer than in group 4a, and since the calculations of QTc as well as the difference from the predicted QeT may not completely eliminate the effects of the cycle length on ventricular repolarisation time, the QeT intervals in individual patients in both groups were plotted against their respective RR intervals (Fig. 4). Although there was some overlapping of values between these two groups, most patients in group 4 showed longer QeT intervals than those in group 4a for given RR intervals. Hence, the use of the QTc interval and the change from the predicted QeT interval in comparing these groups appears warranted. As the percentage of patients with hypertension was lower in group 4a than in group 4, a similar comparison was made between the 36 patients with hypertension in group 4 and in 22 such cases in group 4a. Significant differences in QeT, QTc, and change from predicted QeT interval were again noted between these two groups (not shown), a result similar to that shown in Table 3.

Discussion

In our initial population of 113 patients with bifid T waves selected from 4000 consecutive electrocardiograms, about one third of all the adult patients (33) were aged <40 years. This agrees with the finding that in the absence of demonstrable heart disease bifid T waves are most commonly seen in the first decade of life (20.5%) becoming less frequent in higher age groups.² Furthermore, right bundle branch block significantly increased the incidence of this waveform regardless of age.² Since the left ventricle is not sufficiently developed to overshadow the right ventricular events in children as well as in young women, and since right bundle branch block delays depolar-

Fig. 4 Correlation of QeT intervals and RR intervals in individual patients in group 4 (○) (left ventricular hypertrophy and ischaemia showing bifid T waves) and in group 4a (●) (left ventricular hypertrophy and ischaemia showing single peaked T waves).

isation (and repolarisation) of the right ventricle, these findings may suggest that a delayed right ventricular repolarisation is a possible cause of the second T wave apex in those patients. The present finding that the bifid T waves seen in younger (predominantly female) subjects with otherwise normal electrocardiograms (group 1) were recorded mostly around lead V2 appears to support such a hypothesis. The significant prolongation of QeT, QTc as well as the change from the predicted QeT interval in the patients in group 1 compared with a control group (group 1a) with single peaked T waves (Table 3) indicates that bifid T waves definitely result from a delayed repolarisation in some areas of the ventricles.

On the other hand, Fleisch ascribed the high incidence of bifid T waves in younger individuals to a pronounced autonomic imbalance.⁴ Development of bifid T waves under certain conditions, apparently as functional changes of ventricular repolarisation, may indirectly support such a concept. For instance, this waveform frequently accompanies disorders of the central nervous system—regardless of the presence or absence of other electrocardiographic abnormalities⁵—and also alcoholism especially during a period of withdrawal.^{6,7} Phenothiazine treatment of psychotic patients often produces bifid T waves, which disappear after propranolol administration.¹¹ Certain experimental studies also appear to support this view as Yanowitz *et al* found that a stimulation of the cervical sympathetic nerves in dogs shortened, whereas surgical sections of these nerves prolonged, the ventricular refractory period.¹² Hence uneven sympathetic discharges in the right compared with the left cardiac nerves would result in regional differences of repolarisation and possibly bifid T waves.

In this respect, it must be pointed out that the right and the left sympathetic nerves are said to innervate the anterior and posterior walls of the ventricles respectively and not the right and the left ventricle.¹² Theoretically, asynchronous repolarisation of the anterior and posterior ventricular walls would produce diphasic (rather than bifid) T waves in precordial leads. Nevertheless, the occurrence of bifid T waves in precordial leads next to a lead showing either a negative or diphasic T wave is well known,³ and has been reconfirmed in the present study. The possibility of asynchronous repolarisation of the anterior and posterior walls contributing to the genesis of bifid T waves in certain instances thus cannot be ruled out. Indeed, in our experimental study, we noted one heart in which bifid T waves were produced apparently by such an asynchrony of repolarisation between the anterior and posterior walls of the ventricles.⁸

Nevertheless, most isolated rabbit hearts showing this waveform had a delayed right ventricular repolarisation,⁸ apparently supporting our hypothesis. A

further indication of the role of asynchronous repolarisation of the right and the left ventricles in producing bifid T waves is their response to exercise. Exercise tended to exaggerate the bifid nature of T waves in the patients in group 1 with otherwise normal electrocardiograms. It is conceivable that an increased overall sympathetic tone associated with exercise causes dissimilar repolarisation changes in the right versus the left ventricle rather than in the anterior versus the posterior wall of both ventricles. More specifically, by postulating that exercise produces a greater acceleration of left ventricular repolarisation than of right ventricular repolarisation, one can expect an increased asynchrony between the two ventricles when a delayed right ventricular repolarisation was initially responsible for the second T wave apex in patients in group 1.

One criticism of the above explanation is that the vectorial force resulting from a delayed right ventricular repolarisation may not attain a sufficient magnitude to produce bifid T waves, if the much smaller right ventricular mass is taken into consideration. Nevertheless, when the vectorial force of right ventricular repolarisation sustains little cancellation by the already diminishing left ventricular repolarisation vector the former could modify the T wave configuration. Our experimental studies as mentioned above⁸ indeed supported this theory. The following observations in the present study appear to indirectly support this concept: (a) the amplitude of the second T wave apex was usually smaller than that of the first apex; and (b) bifid T waves in patients in group 1 were predominantly seen in V2, a lead positioned in close proximity to the right ventricle (Table 1). If asynchronous repolarisation of the anterior versus the posterior wall of both ventricles caused by non-uniform discharges of the right and left sympathetic nerves were responsible for the production of this waveform, these findings may not be readily explained.

On the other hand, bifid T waves in certain instances appear to reflect organic heart diseases rather than functional changes. For instance, Suzuki considered that bifid T waves might represent the earliest sign of myocardial ischaemia in younger patients with anginal pain.¹³ Eisenberg and Simonson found that in anginal patients the bifid T wave appeared either before or after a period of more abnormal T wave changes such as diphasic or inverted T waves,³ thus likewise suggesting minor repolarisation abnormalities as a causative mechanism. In the present study, bifid T waves in older individuals, especially those with the electrocardiographic signs of left ventricular hypertrophy and ischaemia (group 4), were seen in the left precordial leads around V4 (Table 1). Such a significantly leftward distribution of bifid T

waves in the patients in group 4 suggests a different mechanism involved in this group compared with those in group 1. Firstly, ventricular repolarisation in patients in group 4 was completed later than those in group 1, as evidenced by the significantly longer QTc and greater change from the predicted QeT interval in the former group (Table 2). This was accompanied by a later appearance of both aT_1 and aT_2 but aT_2 showed a greater delay. Furthermore, patients in group 4 with left ventricular hypertrophy and ischaemia showing bifid T waves had significantly longer QTc as well as a greater change from the predicted QeT interval than even those in group 4a with left ventricular hypertrophy and ischaemia but only single peaked T waves (Table 3). Unfortunately, we still lack an experimental model of left ventricular hypertrophy and ischaemia that is suitable for a mapping study as reported separately.⁸ Nevertheless, certain experimental studies have shown the presence of longer functional refractory periods in chronically ischaemic myocardium,¹⁴ and prolonged action potential durations in Purkinje fibres surviving experimental myocardial infarction.¹⁵ Hence, in patients in group 4 a delayed repolarisation of the ischaemic left ventricular tissue may contribute to the second T wave apex (aT_2), whereas an earlier repolarisation of the remaining non-ischaemic myocardium, including the right ventricle, may be responsible for the first T wave apex (aT_1), provided that their vectors have appropriate relative orientations.

In relation to this hypothesis, the following two findings may possibly explain the difference between the patients in groups 4 and 4a: (a) localisation of the ischaemic areas was predominantly anterolateral in 32 of 40 (80%) patients in group 4 showing bifid T waves, and in only 19 of 38 (50%) patients in group 4a without this waveform, perhaps producing dissimilar vectorial orientations; and (b) the significantly longer RR intervals in group 4 favoured a wider separation of normal and delayed repolarisation vectors, although the QeT intervals in this group tended to be longer than in that in group 4a at individual ranges of RR interval (Fig. 4). If an exercise induced increased sympathetic tone accelerates repolarisation more appreciably in the left than in the right ventricle, as postulated above in group 1, the bifid T waves in group 4 would become less prominent as the second T wave apex is dependent on delayed repolarisation of the ischaemic left ventricle. Such a change was indeed observed in the present study.

As far as groups 2 and 3 are concerned, electrocardiographically these patients had lead prevalence indices (and hence precordial distribution of bifid T waves) between those of groups 1 and 4 (Table 1). Clinically, these groups included many hypertensive patients, who fulfilled either voltage criteria for left

ventricular hypertrophy or had ST-T changes suggesting myocardial ischaemia but not both abnormalities together. Thus these hypertensive patients in groups 2 and 3 may represent cases with milder electrophysiological abnormalities than those in group 4. Nevertheless the effect of including other non-cardiac patients may also explain this observation.

Finally, if the genesis of bifid T waves can be attributed to an asynchrony of repolarisation either between the right and the left ventricle or between the normal and abnormal areas of the ventricular myocardium their clinical significance would depend on whether this asynchrony is caused by functional alterations of repolarisation or by organic cardiac lesions. Further clinical and experimental studies on this waveform thus appear to be necessary.

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