SIMULATION STUDY OF QRS-T WAVES BASED ON AN ECCENTRIC SPHERICAL MODEL OF THE HEART

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The relationship between the difference in the action potential duration in the myocardium and the T wave polarity was investigated by a mathematical model of the electrical activity of the heart. In this study we constructed an eccentric spherical model which duplicates the ventricles except for the interventricular septum. Our model is assumed to be composed of the working myocardium and the excitation conduction system which is uniformly distributed on the endocardial surface. The ventricular gradient was defined as a linear decrease (β msec/cm) of the duration of the action potential from the endocardium to the epicardium according to the concept proposed by Wilson.

Theoretical analysis based on this model strongly suggests that the ventricular gradient of 10–40 msec/cm accounts for the normal QRS-T angle since vectorcardiographic analysis have revealed that the QRS-T angle closely correlates with the angle (θ) between the directions of the propagation waves of depolarization and repolarization. The QRS-T waves in the standard limb and chest leads were calculated from the sum of the time-varying dipoles derived from the action potential, assuming that the heart was in a homogeneous conducting medium. Simulated QRS-T waves were compatible with the clinically observed electrocardiograms not only under normal conditions but also with changes in the spacial position of the heart and the wall thickness.

Positive T waves were obtained in the left precordial leads when the ventricular gradient was more than 20 msec/cm and when the amplitude of T wave increased with the transmural gradient. Our model also indicates that differences in the velocities of the excitation wave in the conduction system and the working myocardium may affect the polarity of the T wave.

THE clinical significance of the polarity and the amplitude of the T wave has been well recognized, but the electrophysiological principle accounting for the polarity of the T wave has not been studied in detail sufficiently.^{1,2} Wilson et

al³ have demonstrated theoretically that the QRS-T area does not reach zero due to differences in the duration of the action potential in the myocardium and thus, has introduced a concept of the ventricular gradient as a cause of the upright T wave in the left precordial leads.

Reynolds et al⁴, Burgess et al⁵ and VanDam et al⁶ have shown that the action potential duration in the subendocardial layer is longer than that in the epicardial layer by measuring the refractory period in the dog's myocardium, based on the

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Fig.1. The eccentric spherical model in the orthogonal coordinates.

Fig.2. The propagation process of excitation in an eccentric spherical model. The figure shows a part of the frontal section of the model.

close correlation between the duration of the action potential and the refractory period being observed. Recently, Spach and Barr^{7,8} recorded the spread of the waves of depolarization and of repolarization through the ventricular wall in anesthetized dogs and found that the repolarization wave travels from the subepicardium to the subendocardial layer.

Although these electrophysiological findings

were adequate to support the concept of the transmural gradient of the duration of action potential, the quantitative relationship between the T wave polarity and the excitation process has not been studied extensively. McLeod et al.¹ have presented the electrophysiological interpretation of the upright T wave in a muscle-strip model and Harumi et al.⁹ reported the theoretical investigation of the T loop in the vectorcardiogram. These studies, however, were within the limits of the conceptual study and the analyses were not based on the time-dependent electrical motive force and its propagation. Although we reported a more sophisticated rectangular solid model of the heart and investigated the factors affecting the T wave polarity based on the transmembrane action potential of the myocardial cells and the propagation velocities of the conduction system and the working myocardium, our previous model did not represent a whole heart but only a part of the left ventricular free wall^{10,11}

In this study we have constructed a new eccentric spherical model of the heart which could be similar to the whole heart and have investigated the relationship between the transmural gradient of the action potential duration and the polarity of the T wave, as well as the effect of the cardiac position in the thorax on the QRS-T wave in standard limb leads and precordial leads.

METHODS

In order to simulate the electrical potential of the heart, a mathematical model of the electrical activity of the ventricles was developed with the assumption that the ventricles, except for the interventricular septum, have essentially an eccentric spherical configuration. We divided it into small blocks and assigned to each a time-varying dipole derived from the transmembrane action potential of the myocardial cells. In order to calculate the time of depolarization of each block, the propagation process of excitation was also assumed based on the electrophysiological concept.

2-1. Morphological basis of the model

An eccentric spherical model of the electrical activity of the heart was constructed and its geometry was described in the orthogonal (XYZ) coordinates; Y axis is a long axis and X-Z plane represents a horizontal plane (see, Fig. 1). The center of the inner sphere (the ventricular cavity) is not identical with that of the outer sphere; the

Fig.3. The relationship between the ventricular gradient (β) and the angle (θ) between the directions of depolarization and repolarization propagation when the ratio of the inner radius (r) to the distance of the point (a) from the center of the inner sphere is 0.6 and the velocity of the conduction system (v_1) is 200 cm/sec. The solid circles represent the relationship between the ventricular gradient (β) and the QRS-T angle obtained from the simulated QRS and T vectors.

latter is located away from the former with a distance d along the direction of the Y axis so that the ventricular cavity is open at the atrio-ventricular groove. It is assumed that the excitation propagation system which is composed of Purkinje fibers covers the inner surface and the remaining part of the working myocardium. Although the interventricular septum is not considered in our model, the contribution of the electromotive force of the septum is minor except for the initial part of QRS complex and thus, presumably would not affect the polarity of T wave.

Our eccentric model simulates the thick left ventricular wall and the relatively thin right ventricular wall if we assumed that the initial excitation point (Q' in Fig. 1) is shifted to the right along the X axis from the under-pole of the model (Q), since the initial excitation point cor-

Fig.4. Simulated QRS and T loops in the horizontal plane with various ventricular gradients (β : 0, 20, 40 and 60 msec/cm).

responds to the root of the interventricular septum; the right and left side of this point (Q') should be included in the right and left ventricles, respectively, see Fig. 1.

The size, the wall thickness of the model and the diameter of inlet of the model (the atrio-ventricular groove) are based on the anatomical findings of the heart.

2-2. Excitation propagation process

It has been assumed that an excitation wave is propagated uniformly in all directions in both of the excitation conduction systems and the working myocardium, each with their own respective velocities. Therefore, each block is excited when the initial excitation wave arrives at that particular block from any of the neighboring blocks. The duration of the excitation caused by the transmembrane action potential is assumed to be shorter from the endocardium to the epicardium

Fig.5. Simulated electrocardiograms (QRS-T waves) in standard 12 leads of a normal subject.

according to the concept introduced originally by Wilson.³ In this study we shall refer a “ventricular gradient” to the transmural gradient of the duration of excitation and not to the QRS-T vector which is often used as a “ventricular gradient”. The extent of the transmural gradient was expressed by β msec/cm (wall thickness). Thus, the action potential duration is longest in the subendocardial layer and declines linearly toward the epicardium, while it is assumed that there is no difference in the action potential duration between the apex and the base.

When the initial excitation of the model begins at point Q (the under-pole), the propagation pathway to any point P (a, ξ, δ) in the model should be on the plane including the points Q, P and the center (O) of the inner sphere, see Fig. 2. If we assume that the exit point from the inner surface to point P be P_1 (r, ξ, δ_1); then the length of the pathway (L) can be expressed as follows:

$$L = \overline{QP_1} + \overline{P_1P} \\ = r(\delta_1 + \pi/2) + \sqrt{r^2 + a^2 - 2ar \cos(\delta - \delta_1)} \dots (1)$$

Accordingly, the propagation time from point Q to P is obtained as the minimum value of t in the following formula:

$$t = r/v_1 \cdot (\delta_1 + \pi/2) + \\ 1/v_2 \sqrt{r^2 + a^2 - 2ar \cos(\delta - \delta_1)} \dots (2)$$

Where, v_1 and v_2 represent the propagation velocities of the Purkinje fibers and the working myocardium, respectively.

Therefore, the initial time of depolarization at point P can be represented as $t_{\min}$, where α represents the ratio of the propagation velocities of the conduction system and the working myocardium ($\alpha = v_1/v_2$);

Fig.6. The rotation of the cardiac model around the three axes (X, Y, Z). The normal spacial position of the heart was simulated by the rotation of 30°, 45° and 45° around X, Y and Z axes, respectively.

Fig.7. The changes of T wave polarity in lead V₅ when the ventricular gradient (β) varies from 0 to 60 msec/cm.

$$t_{\min}(a, \xi, \delta) = 1/v_1 \cdot \left[r(\pi/2 + \delta - \cos^{-1} \frac{r + \sqrt{(1-\alpha^2)(r^2 - a^2\alpha^2)}}{a\alpha^2}) + \sqrt{a^2\alpha^2 + (\alpha^2 - 2)r^2 - 2r\sqrt{(r^2 - \alpha^2)(r^2 - a^2\alpha^2)}} \right] \dots (3)$$

where $\delta < \cos^{-1} \frac{r + \sqrt{(1-\alpha^2)(r^2 - a^2\alpha^2)}}{a\alpha^2}$,

$t_{\min}(a, \xi, \delta)$ is expressed as follows;

$$t_{\min}(a, \xi, \delta) = 1/v_1 \cdot \sqrt{r^2 + a^2 + 2ar \sin \delta} \dots (4)$$

Since the action potential at point P is represented by $D_0 - (a-r)\beta$ where the action potential duration on the subendocardium is D_0 , the time t' at which the repolarization terminates is obtained from Eq. (5):

$$t'(a, \xi, \delta) = t(a, \xi, \delta) + D_0 - (a-r)\beta \dots (5)$$

Thus, the directions of the spread of the depolarization and repolarization are obtained as the gradient of $t(a, \xi, \delta)$ and $t'(a, \xi, \delta)$, respectively as follows:

$$\text{grad}(t(a, \xi, \delta)) = \left(\frac{\sqrt{a^2\alpha^2 - r^2}}{av_1}, 0, \frac{r}{av_1} \right) \dots (6)$$

$$\text{grad}(t'(a, \xi, \delta)) = \left(\frac{\sqrt{a^2\alpha^2 - r^2}}{av_1} - \beta, 0, \frac{r}{av_1} \right) \dots (7)$$

Accordingly, the angle (θ) between the directions of depolarization and repolarization at point P can be represented as follows;

$$\tan \theta = \frac{v_1\beta(r/a)}{v_1\beta\sqrt{\alpha^2 - (r/a)^2} - \alpha^2} \dots (8)$$

This Eq(8) shows that the angle (θ) is the function of the ventricular gradient (β) and the propagation velocities of the excitation of the

Fig.8. Simulated QRS-T waves when the velocity of the excitation in the conduction system (v_1) was 100, 200 and 300 cm/sec, while the velocity of the excitation in the working myocardium (v_2) was unchanged (40 cm/sec).

Fig.9. Simulated QRS-T waves when the velocity of the excitation in the working myocardium (v_2) was 20, 40 and 80 cm/sec, while the velocity of the excitation in the conduction system (v_1) remained unchanged (160 cm/sec).

conduction system and the working myocardium (v_1 and $\alpha(v_1/v_2)$).

2-3 Simulation of QRS-T wave

In order to simulate the electrical potential (QRS-T wave), the following pertinent parameters were included in our model.

- (i) Size of the model (r, R, d)
- (ii) Spacial position of the model in the thorax
- (iii) Transmural action potential of myocardial cells
- (iv) Ventricular gradient (β)
- (v) Propagation velocities of excitation of the Purkinje fibers and the working myocardium (v_1, v_2)
- (vi) Position of the initial excitation point (Q')
- (vii) Position of the observation point

The spacial position of the heart in the thorax was simulated by the rotation of the model around the three axes (X, Y, Z) and the transmembrane action potentials were given by the functions which represent their configuration as

previously reported (see, Appendix)¹⁰ The position of the observation points on the chest surface was given based on the geometry of the precordial leads (V_1-V_6), and the center of the heart was estimated from the biplane chest X-ray films obtained from a healthy adult.

In this study, with previously mentioned assumptions, the heart was assumed to be in a homogenous conducting medium. The electrical potential (V) generated at the observation point was obtained by the solid angle theory¹² with a dipole moment (ϕ) in the small exciting area (s) and the solid angle (ω) subtended by the small area at the observation point as follows;

$$V = 1/4\pi\epsilon \int \phi(t, s) d\omega \dots\dots\dots (9)$$

where, ϵ represents the conductivity of the volume conductor. The electrical potential of the whole heart was calculated as a sum of the

Fig.10. The effect of the thickening of the ventricular wall on the QRS and T waves.

instantaneous potentials generated from the boundary of the heart, i.e. the surface of the cardiac model including the endocardium and the epicardium.

RESULTS

3-1. Theoretical relationship between the ventricular gradient (β) and the angle (θ) between the directions of depolarization and repolarization propagation

Since Eq. (8) in Methods gives a theoretical relationship between the ventricular gradient (β) and the angle (θ) which is obtained from the proceeding directions of depolarization and repolarization, this relation was depicted when the pertinent parameters are within the physiological range as follows:¹³⁻¹⁷

$v_1 = 100 - 300$ cm/sec (the velocity of the excitation propagation in the Purkinje fiber)

$v_2 = 30 - 100$ cm/sec (the velocity of the excitation propagation in the working myocardium)

$r = 3.0 - 5.0$ cm (the radius of the inner sphere)

$R = 7.0 - 8.0$ cm (the radius of the outer sphere)

Figure 3 shows the β - θ relation with the fol-

lowing parameter values; $v_1 = 200$ cm/sec, $\alpha = v_1/v_2 = 3 - 6$, $r/a = 0.6$. This demonstrates that the angle (θ) which corresponds to the QRS-T angle (see below) increases with the decrease of the ventricular gradient (β) and remains within the normal range ($20 - 140^\circ$)^{18,19} when β is $10 - 40$ msec/cm. In these range β - θ curve was nearly horizontal, indicating a great change of angle (θ) with a little change of the ventricular gradient (β). The ratio of the propagation velocities of the conduction system to that of the working myocardium ($\alpha = v_1/v_2$) also affects the β - θ relation. Figure 3 shows that the smaller ventricular gradient accounts for the normal angle (θ) when the ratio α is small.

In our model the angle (θ) is not identical with the QRS-T angle since the angle (θ) depends on the r/a (the ratio of the radius (r) of the inner sphere to the distance (a) of the point from the center of the sphere) as well as the ventricular gradient (β) (see, Eq. (8)), while the QRS-T angle represents the angle between the mean QRS and T vectors. Figure 4 shows the change of T vector loops in horizontal plane with the various ventricular gradient (β). The directions of maximum QRS and T vectors become closer with the increase of the ventricular gradient. The solid circles in Fig. 3 represent the relationship be-

Fig.12. Simulated QRS-T waves in the limb leads in the horizontal position of the heart.

axis (transverse axis) based on the physiological arrangement of the heart in the thorax, see Fig. 6.

The QRS complexes and T waves duplicated the characteristic clinical electrocardiograms of 12 standard leads except for the initial part of QRS complexes at leads aVL, V_1 and V_2 . T waves at leads I, II, III, aVF and $V_4 - V_6$ were positive. The polarity of T waves at leads aVR, V_1 and V_2 was the same as that of the QRS complexes in these leads and T wave at V_3 of the transitional zone was flat.

(ii) Ventricular gradient (β) and the polarity of T waves

Figure 7 shows the T wave changes in lead V_5 when the ventricular gradient increased from 0 to 60 msec/cm. The positive T wave was obtained when the ventricular gradient was more than 20 msec/cm and the amplitude of T waves increased with the ventricular gradient (β). This is con-

sistent with the decrease of angle (θ) when the ventricular gradient (β) increased in the β - θ relation described previously.

(iii) The effect of the propagation velocity on the T waves

When the ratio of the velocities of the conduction system to the working myocardium was constant ($\alpha = v_1/v_2 = 4$), the T waves became higher with the increase of the velocity of the conduction system (v_1) from 100 to 300 cm/sec (Fig. 8). Conversely, when the velocity of the conduction system was constant ($v_1 = 160$ cm/sec), the T wave became higher with the increase of the velocity of the working myocardium from 20 to 80 cm/sec (Fig. 9). These indicate that the amplitude of the T waves is affected by the velocity of the conduction system and the working myocardium as well as the ventricular gradient.

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positive T wave in the left precordial leads. Although Wilson³ presented a theory in 1934 that the positive T wave is due to the differences in the duration of excitation in the myocardium and called it a "ventricular gradient", direct evidence to clarify the quantitative relationship between the differences in the action potential duration and the polarity of T wave has not been obtained since the local differences on the duration of excitation was difficult to detect owing to the slow and nonuniform repolarization process. McLeod¹ and Lepeschkin²⁰ studied the theoretical cause of the positive T wave by using a strip myocardial model and thereafter, Harumi⁹ and Burgess²¹ revealed the significance of the differences in the duration of excitation from the vectorcardiographic study. However, no previous studies have clarified as to why such a small amount of the transmural gradient in the action potential duration seen by several investigators causes the positive T wave. Although a cardiac model recently reported by Thiry et al.²² is more sophisticated, it was not satisfactory to explain the quantitative relationship between the ventricular gradient and the amplitude of T wave.

We have demonstrated in a previous report that the ventricular gradient of more than 30 msec/cm accounts for the positive T wave in a rectangular solid model which represents a part of the left ventricular wall, assuming that the same propagation velocities exists as in the present study.^{10,11} In the present model which represents a three dimensional architecture of the ventricles except for the interventricular septum, the transmural gradient of the duration of the action potential of more than 20 msec/cm was shown to contribute to the positive T wave. Although this critical value of the ventricular gradient depends on the observation point, our model has confirmed that the small differences in the action potential duration of excitation between the endocardium and epicardium as much as 30 msec/cm can cause a positive T wave.

Such an amount of the transmural gradient has been observed indirectly in the dog's heart by Reynolds et al.⁴ and VanDam et al.⁶ Burgess et al.⁵ have also reported a gradient of between 15–25 msec across the ventricular wall. Taking the wall thickness of the dog's ventricular wall into account, these results indicate that the endocardial-to-epicardial gradient of excitation is 15–50 msec/cm of wall thickness. These findings are in agreement with our results estimated from the β - θ relation in the normal range of QRS-T angle

(20–140°, see Fig. 3).

Our results also suggest that the propagation velocities of the conduction system and the working myocardium can affect the polarity of T wave as well as the ventricular gradient (Fig. 8, 9). If the velocities of the conduction system and the working myocardium are 300 and 100 cm/sec, respectively, the T wave at lead V₅ became positive with the ventricular gradient of only 10 msec/cm (Fig. 13). Although direct evidences that the propagation velocities affect the T wave polarity have not been presented, the decrease of the propagation velocity of the working myocardium may be related to the T wave changes in the patients with myocardial damage.

The present study confirms Wilson's "ventricular gradient" as a cause of the positive T wave and demonstrates that theoretically the smaller amount of the ventricular gradient than that previously accepted accounts for the positive T wave in the left chest surface leads.

4-2. *Validity of the model and its limitation*

In the present study we constructed the sophisticated model which simulates the electrical activity of the both ventricles. Although the size and the wall thickness were based on anatomical findings, the following assumptions made in this study may invalidate the results.

- 1) the interventricular septum was absent in our model
- 2) the eccentric spherical model may not be a satisfactory duplication of the configuration of the ventricles
- 3) the propagation of the excitation is assumed to be uniform in all directions with a respective constant velocity in the conduction system and the working myocardium

The excitation in the interventricular septum propagates through the His bundle and the electromotive force is directed to both left and right sides. Therefore, a substantial part of the electromotive forces in the septum seems to be dissipated except for the initial propagation wave from the left to right in the upper septum. This indicates that the absence of the septum has little electrical effect on the QRS-T wave except for the initial component of the QRS complex. However, this septal component which contributes to the initial Q wave in the left precordial leads and the initial R wave in the right precordial leads was not included in this study. Indeed, this absence of the septal force reflects the abnormal Q wave in the simulated electrocardiograms in

leads V_1 and V_2 (Fig. 5).

The shift of the initial excitation point on the right side from the under-pole of the model augmented the abnormal Q waves. The contribution of the shift of the initial excitation point was evaluated by changing the extent of the shift. Figure 14 shows the QRS-T wave at the lead V_1 when the shift was only 25° to the right, showing the decrease of abnormal Q wave when compared with the control wave (QRS-T wave at lead V_1 in Fig. 5). The shift of the initial excitation wave was done to simulate the thicker wall of the left ventricle than the right ventricle in spite of the foregoing faults. When we shift the initial excitation point clockwise 30° from the under-pole of the model, the wall thickness at that point (point Q' in Fig. 1) is approximately 0.9 cm and the majority of the thick part of the model is included in the left ventricle. However, this modification was performed to simulate the anatomically thicker wall of the left ventricle than that of right ventricle.

Thus, to analyze the effect of the wall thickness to the T wave polarity, we simulated the QRS-T waves in the model with a wall thickness twice that of the control by increasing the radius (R) of the outer sphere and the distance (d) between the two centers of the sphere. Our results have demonstrated no changes in the T wave polarity when the ventricular gradient remains constant, although the increase of the amplitude

of T wave is associated with the increase in the amplitude of QRS complex (Fig. 10).

These strongly suggest that our model is acceptable for the analysis of T wave polarity in relation to the ventricular gradient of the action potential duration through the ventricular wall. However, some discrepancies are seen in the T wave polarity at leads V_2 , V_3 between the simulated electrocardiogram and those observed clinically. In our model, the T wave is inverted at leads V_2 and is flat at lead V_3 , while the normal electrocardiograms usually show positive T waves in these chest leads. Although oversimplification of the model and the differences in the excitation wave may be a factor, our model of the ventricles has confirmed the previous results obtained from the rectangular solid model which simulated only a part of the ventricular wall; only a small amount of the gradient of the action potential duration through the ventricular wall between 20–30 msec/cm contributes to the positive T wave.

Appendix

Approximation of the transmembrane action potential

The transmembrane action potential of myocardial cells is due to a periodic ion exchange across the cell boundary and consists of four phases. Each phase was fitted by the function as follows;

1. phase 0: ($0 < t < t_1$)

$$f(t) = (h_1/t_1) \cdot t$$

2. phases 1 and 2: ($t_1 < t < t_2$)

$$f(t) = (h_1 - h_2) e^{-h_3(t-t_1)} + h_2 - h_4(t-t_1)$$

3. phase 3: ($t_2 < t < t_3$)

$$f(t) = f(t_2) \left[\frac{-\cos(\pi - \omega_2) + \cos(\omega_1 + \frac{(\pi - \omega_1 - \omega_2)(t-t_2)}{t_3-t_2})}{\cos \omega_1 - \cos(\pi - \omega_2)} \right] \dots \dots \dots (10)$$

where t_1 , t_2 and t_3 represent the terminal time of phase 0, 2 and 3, respectively and h_1 and h_2 are the maximal potential values in phases 1 and 2. The graph of $f(t)$ is shown in Fig. 15. Phase 0 was represented by a steep up-slope line and phase 1 was duplicated by a decaying exponential (h_3 is a rate constant decaying) superimposed on a down-slope line representing phase 2, a slope of which was given by h_4 . Phase 3 was simulated by a part of a cosine curve to duplicate using a relatively simple function. $f(t_2)$ represents the

amplitude of an action potential at time t_2 . The parameters, ω_1 and ω_2 indicate the angle range in a cosine curve as shown in Fig. 15.

The numerical values which appear in Eq. (10) have been chosen to give a realistic transmembrane action potential with $f(t)$ in millivolts (mV) and t in milliseconds (msec). Since the parameters in Eq. (10) are all variable in our model, various shapes of the transmembrane action potential can be produced. The ventricular gradient was given with shortening t_2 in Eq. (10)

from endocardium to epicardium by β msec/cm.

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REFERENCE

1. MACLEOD, A. G.: The electrogram of cardiac muscle: An analysis which explains the regression of T deflection. *Am. Heart J.* 15: 165-186, 1938.
2. TOYAMA, J., KOBAYASHI, T., MURAKI, H., FUJINO, T., HORI, K., OKAJIMA, M. & YAMADA, K.: A method to construct a T-wave of the electrocardiogram. *Jap. Circ. J.* 31: 313-318, 1967.
3. WILSON, F. N., MACLEOD, A. G., BARKER, P. S., JOHNSTON, F. D. & ARBOR, A.: The distribution and the significance of the areas of the ventricular deflections of the electrocardiogram. *Am. Heart J.* 10: 46-61, 1934.
4. REYNOLDS, Jr., E. W. & VANDER ARK, C. R.: An experimental study on the origin of T-waves based on determinations of effective refractory period from epicardial and endocardial aspects of the ventricle. *Circ. Res.* 7: 943-949, 1959.
5. BURGESS, M. J., GREEN, L. S., MILLAR, K., WYATT, R. & ABILDSKOV, J. A.: The sequence of normal ventricular recovery. *Am. Heart J.* 84: 660-669, 1972.
6. VANDAM, R. TH. & DURRER, D.: Experimental study on the intramural distribution of the excitability cycle and on the form of the epicardial T wave in the dog heart in situ. *Am. Heart J.* 61: 537-542, 1961.
7. SPACH, M. S. & BARR, R. C.: Analysis of ventricular activation and repolarization from intramural and epicardial potential distributions for ectopic beats in the intact dog. *Circ. Res.* 37: 830-943, 1975.
8. SPACH, M. S. & BARR, R. C.: Ventricular intramural and epicardial potential distributions during ventricular activation and repolarization in the intact dog. *Circ. Res.* 37: 243-257, 1975.
9. HARUMI, D., BURGESS, M. J. & ABILDSKOV, J. A.: A theoretical model of the T wave. *Circulation* 34: 657-668, 1966.
10. INOUE, M., HORI, M., KAJIYA, F., KUSUOKA, H., ABE, H., FURUKAWA, T. & TAKASUGI, S.: Theoretic analysis of T-wave polarity based on the cardiac model of the electrical activity. *J. Electrocardiol.* (in press)
11. INOUE, M., INADA, H., HOKI, N., FUKUSHIMA, M., HORI, M., KUSUOKA, H., ABE, H., KAJIYA, F., FURUKAWA, T. & TAKASUGI, S.: A simulation of QRS-T wave based on the transmembrane action potential. *Jap. J. Med. Electr. Biol. Eng.* 15: 121-128, 1977.
12. SWIHART, J. C.: Numerical methods for solving the forward problem in electrocardiography, ed. C. V. Nelson and D. B. Geselowitz, Oxford pp. 257-293, London 1976.
13. TRAUTWEIN, W., GOTTSTEIN, U. & FEDERSCHMITT, K.: Der Einfluss der Temperatur auf den Aktionsstrom des excidierten Purkinje-Fadens, gemessen mit einer intrazellulären Elektrode. *Pflug. Arch. ges. Physiol.* 258: 243-260, 1953.
14. DRAPER, M. H. & WIEDMANN, S.: Cardiac resting and action potentials recorded with an intracellular electrode. *J. Physiol.* 115: 74-94, 1951.
15. TRAUTWEIN, W.: Elektrophysiologie der Herzmuskelfaser. *Ergebn. Physiol.* 51: 131-198, 1961.
16. SCHAEFER, H.: The general order of excitation and of recovery. *Am. New York Acad. Sci.* 65: 743-767, 1957.
17. SCHER, A. M. & YOUNG, A. C.: The pathway of ventricular depolarization in the dog. *Circ. Res.* 4: 461-469, 1956.
18. DRAPER, H. W., PEPPER, C. J., STALLMAN, F. W., LITTMANN, D. & PIPBERGER, H. V.: The corrected orthogonal electrocardiogram and vectorcardiogram in 510 normal men (Frank lead system) *Circulation* 30: 853-864, 1964.
19. SCHAU, F.: Zur Klinischen Anwendung des korrigierten orthogonalen Vkg-Ableitungssystems nach Frank. I. Untersuchungen bei Normalen. *Cardiologia* 40: 1, 1962.
20. LEPESCHKIN, E.: Modern Electrocardiography. The P-Q-R-S-T-U Complex, Williams & Wilkins Co., Baltimore, 1951.
21. BURGESS, M. J., HARUMI, K. & ABILDSKOV, J. A.: Application of a theoretic T-wave model to experimentally induced T-wave abnormalities. *Circulation* 34: 669, 1966.
22. THIRY, P. S., ROSENBERG, R. M. & ABBOTT, J. A.: A mechanism for the electrocardiogram response to left ventricular hypertrophy and acute ischemia. *Circ. Res.* 36: 92-104, 1975.
23. HOFFMANN, B. F., KAO, C. Y. & SUCKLING, E. E.: Refractoriness in cardiac muscle. *Am. J. Physiol.* 190: 473-482, 1957.