A Theoretical Examination of Ventricular Repolarization and the Secondary T Wave

LEO G. HORAN, R. CHRIS HAND, JENNIFER C. JOHNSON, MARANDAPALLI R. SRIDHARAN, THOMAS B. RANKIN, AND NANCY C. FLOWERS

SUMMARY Two theoretical waveforms appear in the study of the ventricular repolarization process: the primary T wave (T₁) derived from many local variations in repolarization properties alone, assuming a uniform starting time, and the secondary T wave (T₂) dictated by activation sequence alone, assuming uniform identical action potential durations throughout ("zero gradient"). Subtraction of the appropriate T₁ from the observed T wave (T₃) following any given sequence of ventricular activation should yield T₂. An experimental waveform, the intrinsic T wave (Tᵢ) following total simultaneous depolarization, has been proposed to be identical with T₂. This study uses a 1675-element computational model of the left ventricular myocardium to generate eight orthogonal electrocardiographic waveforms representative of left ventricular activation and repolarization. Normal propagation and ectopy were simulated realistically; sequence was based on the electrophysiological properties of the individual elements. The model was assigned experimentally derived recovery times to generate T₁ and uniform fixed recovery times to generate the true or reference Tᵢ. A systematic search was made for the unknown parameter for calculating the secondary T wave, and we found that scanning the normal QRS with an area-sampling shutter 56 msec wide reproduced Tᵢ (r = 0.9984). However, correlation between theoretical Tᵢ and experimental Tᵢ reached a maximum of 0.9856. The failure of absolute congruence between Tᵢ and Tᵢ, derives from the multiplicity of elements and the resulting frequency distribution of actual local recovery times instead of a single ideal instant to provide the basis for aligning related waves in time.

This is a report of a study that used a computer model of left ventricular myocardial activation and repolarization* to examine the relationships between the ventricular gradient, 1 the intrinsic T wave, 2 and the primary and secondary T waves. 3 The surface electrocardiographic sequels of the electrical activity in the model have been recorded compactly in multipolar scalar lead format 4 from which body surface maps and moving dipole displays or vectorcardiograms were obtained.

Three frontal plane vectors were constructed by Wilson from the net areas of the QRS complex and under the T wave in the Einthoven leads of the electrocardiogram: the QRS area vector, the T area vector, and their sum, the QRS+T area vector or ventricular gradient. The QRS vector thus defines the mean direction of spread of ventricular excitation, the T vector defines the inverse of the mean direction of spread of ventricular repolarization and the ventricular gradient points from the region of longest average duration of the excitatory process to that of the shortest. 1 Under conditions varying the order of excitation (QRS), the gradient vector (QRST) remains relatively constant. 5 Thus, as a crude model of ventricular repolarization properties, the gradient vector should "predict" the T vector given the QRS vector. Unfortunately, the ventricular gradient (G) remains a statistical summary, but its power derives from the fact that, in the relationship, G = QRS + T (whether scalar or vector), if one of the three terms goes to zero the other two become identical (although in the instance of G = 0, QRS and T become equal but of opposite sign).

Thus, in the occasion of uniform instantaneous depolarization of all ventricular muscle, the QRS area vector becomes zero and the gradient and the T area vector are equal. Reasoning from this, von Dam and Durrer 6 recorded the T wave following total instantaneous depolarization. Such an "intrinsic" T wave should depend on the intrinsic local electrical properties of action potentials distributed throughout the myocardium and not on any variation in starting times. 7 Local, artificial electrical stimuli alter local repolarization times somewhat, 8 but since the total instantaneous depolarization led to a T wave with the same gradient as with the normally conducted QRS plus T, 4 it is reasonable to assume that the effect of the general depolarizing shock is evenly distributed and therefore does not cause relative changes in local duration of recovery.

Ablidkov et al. 9 proposed deriving a "primary" T wave by subtracting a calculated "secondary" T wave from the actually recorded T wave. The secondary T wave is constructed by assuming that all ventricular action potentials are of uniform configuration and duration. This is tantamount to a ventricular gradient of zero. In that case the T for any lead should be oppositely directed from the QRS for that lead and proportional in amplitude for successive instants to increasing and decreasing increments.
of the appropriate QRS area. (This follows from the postulate of uniformity: the secondary T spread would follow the same path through the ventricle as the QRS.) However, because of the sharp upstroke, boundaries of activation separate all-or-none regions—resting or depolarized. During the T wave, the same geographic boundaries as for the earlier QRS may exist simultaneously separating now the minimally repolarized region from the intermediate repolarized region, and the completely repolarized region, and so on.

This work then responds to the following question: Are the primary T wave and the intrinsic T wave the same? If the answer is that they may be—then what are the conditions necessary for them to be the same?

**Methods**

**Construction of the Ventricular Model**

The chambers of the heart of a previously healthy 19-year-old man who had died an accidental death were filled (but not distended) with cotton prior to brief fixation in formaldehyde solution. Proper consent for this study had been obtained at the time of autopsy according to our usual institutional rules. Then, with the aid of a 1-cm deep well, serial horizontal slices were made of the heart held in the upright position corresponding to the standing position in life. These slices were traced onto ruled centimeter paper. From these tracings, serial slices of left ventricular muscle then were interpolated and approximated by assays of 3.2 mm-edged cubes (Fig. 1). The coordinate positions of the centroids of these cubes with reference to a cubical volume 6.4 cm on each edge were tabulated.

**Computation of Activation Sequence**

Each cube was assumed to contain myocardium with electrical properties paralleling those of membrane action potentials but simplified to trapezoidal shape. Relative durations according to anatomic region were estimated from the data of Burgess et al. and Autenrieth et al. Time was specified in units equivalent to 1.6 msec. A three-digit statement of these repolarization properties was entered for each cube containing muscle. The first two digits specified the expected duration of the plateau (phase 2) and the third digit the duration of downstroke (phase 3) to the nearest 10 time units (see Fig. 2). Blocks on the endocardial or cavitary surface were distinguished by negative signs to indicate the presence of Purkinje fiber connections. When we designated a number of initial firing sites, the digital computer then assigned an activation sequence based on a conduction time through each block of five time units (8 msec) unless the block also contained Purkinje connections, in which case, conduction required only one time unit (1.6 msec). These assignments corresponded to assumed conduction velocities of 2 m/sec for specialized fibers and 0.4 m/sec for nonspecialized fibers. The time of excitation for each block then became designated by a two-digit prefix to the original specification of repolarization properties. After several trials, we found subendocardial excitation sites which produced serial isochronous wavefronts corresponding to those reported by Durrer et al.

**Computation of the Generator Effect**

Any contiguous cluster of eight cubes was considered an effective local current generator; the magnitude of current flow was estimated to be directly proportional to potential differences between facing pairs. The potential difference for any instant was calculated from the difference in membrane action potentials as specified by the five-digit characteristic. Thus, Cartesian vector sources were computed for each successive cluster as shown in Figure 3. The net effect for the whole myocardium was obtained by summing the individual effects of all these dipole sources. Computation time was reduced by an order of 10 by estimating the equivalent multipolar generator effect from the eccentric dipolar shift equations rather than by directly computing surface potential from the dipoles.

**Simulation of Simultaneous Excitation**

Multipolar electrocardiographic scalar lead waveforms were derived by allowing an activation sequence to proceed in the computational model and to be followed by a recovery sequence based on the assigned properties (Fig. 4). Except where the higher order waveforms were needed for map display, observations were confined to the dipolar (x, y, z) and quadripolar waveforms. The sampling interval during the QRS complex was taken at every 3.2 msec and during the S-T segment and T wave at every 12.8 msec. Then the activation order was altered in one of two ways: (1) to simulate ectopic beats, a selected site of epicardial excitation was chosen and a new activation sequence computed, or (2) to simulate total depolarization, a discharge was postulated to be of sufficient energy to depolarize instantly all muscle elements not currently in phase 1 or 2 of the membrane action potential. The scalar
electrocardiographic waveforms resulting from total depolarization were labeled intrinsic T waves.

Results

Once familiarity with the model had been gained, the two following hypotheses were examined:

Hypothesis I

The secondary T wave generated by the ventricular myocardium (i.e., as modeled) under zero-gradient conditions is the same as that calculated from increasing and decreasing increments of the “surface” QRS complexes produced by that order of activation (Abildskov’s method). This straightforward statement raised several questions which we dealt with either by testing or by assumption. The first question concerns zero-gradient or uniform repolarization: it specifies neither the duration nor shape (slope) of the repolarization process; it merely states that repolarization is the same throughout the ventricular

Figure 2 Diagram representing average local myocardial action potential simplified to trapezoidal form. The upstroke (a composite of local phases 0) requires 8 msec equaling the transit time through a 3.2-mm cube of ventricular myocardium. The overshoot (phase 1) is assumed to be averaged out and the plateau (phase 2) and descent (phase 3) are specified by local recovery code as described in the text. Bottom: An evolving sequence of depolarization and repolarization as seen in the ninth horizontal ventricular slice of the model. A “normal” set of left sepal sites of Purkinje fiber initiation has been postulated, a “normal” set of repolarization characteristics programmed into the myocardial elements, and the sequence allowed to run its course according to conduction velocities and repolarization durations. The numbers indicate the msec after onset and the local shading increases from blank at -100 mv level of potential to most intense (#) as shown in upper diagram.

Figure 3 Diagram of a representative cluster of eight cubes whose relative local potentials determined an effective local current generator. The potential difference across the interface between each adjacent pair of cubes contributes to the equivalent local heart vector placed at the central common junction between all eight cubes. The effect for the whole myocardium was obtained by scanning the total array of cubes. For the 1675 cubes, a total of 2536 common junctions were examined.
VENTRICULAR REPOLARIZATION AND SECONDARY T WAVE/Horan et al.

myocardium. We therefore initially assumed a fixed duration equivalent to the mean of the durations assigned throughout the ventricular myocardium corresponding to "normal" conditions.

The second uncertainty arises from lack of knowledge of how much of the QRS duration corresponds to any given instant in the secondary T wave. Abildskov et al. postulated that, under conditions of uniform recovery, successive wavefront boundaries corresponding to degrees of repolarization would appear (and subsequently disappear) in the ventricular myocardium in the same order as during activation and with the same contours. However, because of the difference in the duration of the upstroke of activation (phase 0) and the downslope of repolarization (phase 3), several such "boundaries" could be expected to be found simultaneously during repolarization. If the surface QRS could be computed at any given moment from the contour of the boundary of the underlying ventricular wavefront, the secondary T wave amplitude should be computed by adding up the effects of exactly these same boundaries which still happen to be present, this time marking the steps down the slow slope of repolarization rather than up the fast slope of activation. Since the QRS already summarized serial wavefront boundary effects, successive instants in the secondary T wave should correspond to the successive sums of serial instants in the QRS complex. This can be visualized as moving a piece of paper in which there is a vertical slit somewhat narrower than the QRS complex—from left to right across the QRS complex. As the opening moves, the viewer sees first successively increasing areas of QRS, then successively changing areas, and, finally, successively decreasing areas. These areas predict successive elements of amplitude of the secondary T wave.

Unfortunately, the duration of the slit or incremental QRS sample-scanner from which to make the secondary T wave calculation is not known. We therefore progressively increased the duration of the increment with which we scanned the QRS area to obtain a family of predicted secondary T waves. The final unknown was the duration of the secondary T wave. In a practical situation, its duration would have to bear some predictable relationship to that of the observed T wave if it were to be useful. We therefore concentrated our attention on comparison with the "true" secondary T wave because it was generated by the model from a set of uniform repolarization characteristics. This uniform pattern was the mean of the normal variations in repolarization time throughout the model. Our plan was to make further adjustment by stretching or compressing the time base, but that turned out to be unnecessary in practice.

Thus the hypothesis was tested in the following fashion: two sequences of ventricular activation and repolarization (one normal, one left ventricular ectopic) were permitted to produce orthogonal QRS and T waveforms as described earlier. Next the duration and repolarization slope of all the elements in the model were averaged. Then all elements were reassigned this average set of repolarization properties, a condition satisfying the requirement of zero gradient (Fig. 4A). The model then was permitted to generate "true" secondary T waves for both the normal and ectopic sequence. The QRS complexes for these two activation sequences were examined by scanning with an area-sampling gate which varied from 1 time unit up to the total width of the QRS complex. As the gate moved into, through, and out of the QRS complex at 1.6-msec increments, the area of QRS within the gate was converted to an amplitude estimate for the calculated secondary T wave. This produced a family of predicted secondary T waves. Table 1 and Figure 5 summarize our findings as to the correlation between the generated and the calculated secondary T waves. In each instance, the eight simultaneous T waves for each set all were included in the comparative determination of correlation coefficients. Adjustment of time base was not performed because the calculated secondary T wave approached the true secondary T wave both in form and duration as the calculating aperture widened. The best correlation resulted from using a sampling increment or gate 56 msec wide to scan both the 93-msec-wide normal QRS complex \( (r = 0.9984) \) and the 106-msec-wide ectopic QRS complex \( (r = 0.9930) \).

Hypothesis 2

The intrinsic T wave following total simultaneous depolarization and the calculated primary T wave are the same.

Both intrinsic and primary T waves are considered to be estimates of the effect generated by the intrinsic repolarization properties of the underlying myocardium. The intrinsic T wave is "experimental" in nature in that it is postulated to be observed as the immediate sequel of simultaneous depolarization of all elements of the ventricular myocardium. The primary T wave was calculated by
TABLE 1  Correlation coefficients between True and Calculated Secondary T Waves

<table>
<thead>
<tr>
<th>msec*</th>
<th>&quot;Normal&quot;</th>
<th>&quot;Ectopic&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>0.7350</td>
<td>0.7779</td>
</tr>
<tr>
<td>24</td>
<td>0.8606</td>
<td>0.8879</td>
</tr>
<tr>
<td>40</td>
<td>0.9554</td>
<td>0.9661</td>
</tr>
<tr>
<td>56</td>
<td>0.9984</td>
<td>0.9930</td>
</tr>
<tr>
<td>72</td>
<td>0.9660</td>
<td>0.9715</td>
</tr>
<tr>
<td>88</td>
<td>0.9209</td>
<td>0.9321</td>
</tr>
</tbody>
</table>

* Width of aperture of QRS scanning gate.

subtracting the true secondary T wave from the observed T wave. (The secondary T wave calculated from the observed QRS complex with the proper gate could have been used as easily.)

Testing this hypothesis, as shown in Figure 6, brought to light another uncertainty. What is the fiducial mark by which to align the two T waves (observed and secondary) for subtraction? We tested serial shifts in alignment. Corresponding instants of the secondary T wave were subtracted from the observed T wave to produce the primary T wave. The best correlation occurred when the onset of the secondary T wave was closely aligned with the onset of the observed T wave. However, the correlation value was distinctly less ($r = 0.9456$) than the correlation values found between the true and calculated secondary T waves (0.9984 and 0.9930). The latter values indicate that the intermediate operation of area sampling of the QRS does not produce a serious loss when the signal information of the 1675 elements is transformed into the eight waveforms. Were the intrinsic and primary T waves truly the same, the corresponding arithmetic operations should have also produced little discrepancy.

Thus we considered the attempt to confirm the identity of the intrinsic and the primary T wave to be incompletely successful. We perceived that, out of a large number of possible time durations of the action potentials between the shortest and the longest, we originally chose the mean to generate the secondary T and had set the alignment for subtraction accordingly. We then went back to a simple three-element model of excitation and repolarization, as shown diagrammatically in Figure 7, in which the QRS

Figure 5. Comparison of the true T waves with calculated T waves (using Abildskov's method) for both the normal (A) and ectopic (B) sequence. In both instances, the area-sampling shutter was set at 24, 56, and 88 msec, as indicated in Table 1. Only the three dipolar leads and the rms waveform are repeated. The rms waveform includes the values of both the dipolar and quadripolar leads. The true secondary is completely superimposed by the 56-msec gate calculation.

Figure 6. Comparison of intrinsic T waves to primary T waves. The intrinsic T waves from the second column of Figure 4 are repeated on the left. Only the dipolar leads and the rms waveform are shown; the rms waveform, however, includes the values from the five quadripolar leads also. Primary T waves (on the right) were derived by subtracting generated secondary T waves (third column of Figure 4) from observed T waves (first column of Figure 4). Alignment of offset of secondary T wave with offset of the observed T produced a primary T (broken line) with relatively low correlation with the intrinsic T wave ($r = 0.8748$). When the onset of the secondary was moved to coincide with that of the observed, the primary T (bold line) more closely approached the intrinsic T ($r = 0.9456$).
VENTRICULAR REPOLARIZATION AND SECONDARY T WAVE/Horan et al. 755

UNIFORM DURATION: SHORT
Normal
Cardiomyocyte
Ectopic

UNIFORM DURATION: MEAN
Normal
Cardiomyocyte
Ectopic

UNIFORM DURATION: LONG
Normal
Cardiomyocyte
Ectopic

Figure 7 Diagrams of the relationships between representative action potentials and the observed, primary, intrinsic and secondary T waves. In each instance the conditions are simplified to consider the effect of potentials in three muscle blocks on an axial T wave. The relative potential difference at each instant is considered to produce a proportional positive ion-current flow toward the direction of greater negativity; for practical purposes the (negative) potential of the epicardial trapezoid subtracted from the (negative) potential of the endocardial trapezoid predicts the shape of the axial "surface" T waveform. In each diagram, on the left is the "normal" sequence, in the middle is the total instantaneous depolarization effect, and on the right is the "ectopic" sequence and T are derived from the relationship between subendocardial and subepicardial action potentials. In contrast to the simple outline of relationships shown in Figure 7, A or C, we actually were frequently operating more as shown in Figure 7B. The simple model predicted that either early or late alignment of the secondary T for subtraction should yield a primary T wave near-coincident with the intrinsic T wave. By contrast, intermediate positioning of the secondary T wave should produce a phase change which destroys the identity. Figure 7 illustrates the closer approach to identity between intrinsic and primary T; provided the primary T is derived from appropriate alignment of the secondary T.

Finally, Figure 8 shows in three-dimensional vectorcardiographic format the dipolar lead data during normal activation and normal recovery, during recovery following total simultaneous activation, and during a secondary T process. Study of these displays coupled with reasonable expectation of the probable normal spread of activation permitted visual "recognition" of relatively early recovery times in the subepicardium, especially at the posterior base and midwall, which indeed were the built-in characteristics assigned originally.

Discussion

Use of the Multipolar Format

The confirmation in the computational model of these hypotheses suggests the near identity of the primary and intrinsic T wave and the identity of the two forms of secondary T wave. It further suggests that experimental assay of repolarization properties in the intact animals is a reasonable expectation for sufficiently detailed surface potential recordings which would permit equivalent multipolar reduction and display. Implicit is the power of the multipolar expansion to express in exactly the same format both the forward prediction of any model of ventricular activation and the inverse prediction of any comprehensive set of body surface potentials. The chief strength of the common format is that it is comprehensive, nonredundant, and a convenient medium of exchange between the outcoming and ingoing transfers of information.

Limitations and Advantages of the Mosaic Model

The multiple-element computational model of ventricular myocardium has three major disadvantages in predictive power. (1) It is not muscle but only certain properties of muscle which are known or estimated. (2) It is composed of 1675 blocks to which the properties are attributed, rather than a greater number of electrically connected cells or fibers. (3) Purkinje cell contributions are restricted to specifying faster cell-to-cell transmission rates reversed from the normal. Dotted lines = the action potentials of uniform duration necessary to generate a secondary T wave (i.e., one dependent on activation sequence alone). Panel A and C illustrate the predicted effects of early and late alignment of the secondary T waves with consequent sameness of intrinsic and primary T waves. Panel B illustrates intermediate alignment and the disappearance of similarity.
The correlation value indicates the time at which "inter-...timewaves exactly the same? Had the intrinsic T wave been...tuated contributions, or (3) Purkinje recovery times may...intrinsic and primary T waves, they are not the same,...hypothesis regarding the possible benefit of attempting to...the model includes this property.

This particular isolated left ventricular model should be...tional interaction among cells which could allow for the possibility of propagated repolarization. We believe, however, that the first two limitations are not severe, in that the major determinants of T wave morphology probably are the variations in local recovery times and that these are represented satisfactorily by local averages. If discrete generator contributions to the electrocardiographic waveforms arise from Purkinje fibers (as has been suggested for the U wave), this model will fail to include them. If, on the other hand, the Purkinje contribution is strictly that of faster cell-to-cell transmission along its endocardial network, the model includes this property.

The "vectorcardiogram" of the left ventricular model during "normal" activation and repolarization (row 1); during uniform repolarization following normal activation (row 2); recovery following total simultaneous depolarization (i.e., the intrinsic recovery process) (row 3); during ectopic activation and repolarization (row 4); and during uniform repolarization following ectopic activation (row 5). The respective plane views are right sagittal (RS), frontal (F), and horizontal (T). See text for discussion.

FIGURE 8

The Relationship of Primary to Intrinsic T Wave

Despite the practical identity between true and calculated secondary T waves, the intrinsic T waves closely approached but did not coincide exactly with the primary T waves. Why weren't the intrinsic and the primary T waves exactly the same? Had the intrinsic T wave been obtained directly following a depolarization shock to an experimental animal, a number of explanations would have been available: (1) the experimental shock failed to excite a portion of the myocardium, (2) hyperpolarization of some or all of the muscle introduced new and undetermined contributions, or (3) Purkinje recovery times may contrast markedly in duration from those of working myocardium. However, live data were not employed at this stage, and the question remained, what then was the small difference due to? The discrepancy lay not in the normal activation sequence, not in the secondary T being dependent directly upon that sequence, and not in the intrinsic T. Rather, it depended on lack of an exactly appropriate basis for aligning the related waves in time. Very early assignment of position for the secondary T improved correlation. The three-element models (Fig. 7) provided an intuitive appreciation for the possible benefit of attempting to line up repolarization events near either the common beginning or the common end of the phase 3 downslopes of the regional action potentials. However, when a very great number of elements are included (as in either the ventricular model or in life), the relationships are no longer a simple choice of early, intermediate, and late. In this event, an instant of greatest incidence of useful alignment is sought for but not readily predicted. The correlation value indicates the time at which "intermediate" alignments were at a minimum and "early" or late alignments at a maximum. We are forced to conclude that while it was appealing intuitively to equate the intrinsic and primary T waves, they are not the same, principally because the primary T wave cannot be defined precisely.

In brief, we have confirmed the identity between true and calculated secondary T waves and a lack of identity between primary and intrinsic T waves despite a high degree of relatedness. In so doing we have examined some of the uncertainties which must be removed to obtain primary and secondary T waves from observed wave-
forms. These steps may make it easier to estimate alterations in myocardial repolarization properties during drug, electrolyte, and ischemic interventions in experimental and clinical studies.

Acknowledgments

We gratefully acknowledge the assistance of Victoria Gohmann in preparation of the manuscript and of Melissa Vogt in preparation of the illustrations.

References

5. Wilson FN, Macleod AG, Barker PS, Johnston FD: The determination and the significance of the areas of the ventricular deflections of the electrocardiogram. Am Heart J 10: 46-61, 1934

The Magnitude of the Electromotive Force of Canine Ventricular Myocardium

SABURO MASHIMA, KENICHI HARUMI, AND SATORU MURAO

SUMMARY The isolated and perfused dog heart was placed in a cubic container filled with Tyrode's solution. Ventricular ectopic beats were produced by electrical stimulation of the left ventricular wall, and initial QRS vectors of these beats were determined with orthogonal leads from the surface of the container. At the same instants, the activated area on the epicardial surface was measured by means of a large number of contiguous bipolar leads from the epicardial surface. The QRS vector and the activated epicardial area were found to be nearly proportional. By use of these results and a calibration system with artificial dipoles, the double layer moment of the ventricular activation wave was calculated as 0.13 mA cm per unit area. This value corresponds to 60% of the maximal possible value but not as much as the potential caused by a constant-current source within the solution. The relationship between the QRS voltage and the conductivity of the medium was analyzed by a simplified model of the system and was found to correspond approximately to that of a constant-current source within a spherical heart with a resistivity 2 to 3 times that of Tyrode's solution.

Acknowledgments

We gratefully acknowledge the assistance of Victoria Gohmann in preparation of the manuscript and of Melissa Vogt in preparation of the illustrations.

References

5. Wilson FN, Macleod AG, Barker PS, Johnston FD: The determination and the significance of the areas of the ventricular deflections of the electrocardiogram. Am Heart J 10: 46-61, 1934

from The Second Department of Internal Medicine, University of Tokyo, Tokyo, Japan.
Address for reprints: Saburo Mashima, M.D., The Second Department of Internal Medicine, University of Tokyo, Tokyo, Japan.
Received August 29, 1977; accepted for publication January 31, 1978.

6. Van Dam R Th, Durrer D: The T wave and ventricular repolarization. Am J Cardiol 14: 294-301, 1964
7. Lepeschkin E: Modern Electrocardiography, vol 1. The P-Q-R-S-T-U Complex, Baltimore, Williams & Wilkins, 1951, pp 118-120
8. Han J, Garcia de Julon P, Moe GK: Fibrillation threshold of prema-
13. Autenrieth G, Saruwitz B, Kuo CS, Arita M: Primary T wave abnormalities caused by uniform and regional shortening of ventricu-
lar monophasic action potential in dog. Circulation 51: 668-676, 1975
14. Durrer D, Van Dam KTH, Freud GE, Janse MJ, Meijler FL, and Arzbach C: Total excitation of the isolated human heart. Circu-
lation 41: 899-912, 1970
17. Watanabe Y: Purkinje repolarization as a possible cause of the U wave in the electrocardiogram. Circulation 51: 1030-1037, 1975
A theoretical examination of ventricular repolarization and the secondary T wave.
L G Horan, R C Hand, J C Johnson, M R Sridharan, T B Rankin and N C Flowers

doi: 10.1161/01.RES.42.6.750

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/42/6/750.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation Research_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation Research_ is online at:
http://circres.ahajournals.org/subscriptions/