Numerical Simulation of Conduction Delay in Blocked Purkinje Tissue

By Fred H. Terry, James R. Wennemark, and Daniel A. Brody

ABSTRACT

The model used for passive conduction in muscle fibers consisted of a region of nonpropagating fiber of variable length, bounded proximally and distally by normal active tissue. The voltage variation on this fiber was governed by the passive wave equation. This system was analyzed by Fourier series techniques on the digital computer, using a cardiac action potential as the input. With a fixed distal threshold, delays up to 45 msec were simulated by varying the length of nonconducting fiber. Delay time was very sensitive to the extent of the inactive region. Inactive tissue lengths of 1.6 mm and 2.0 mm caused delays of 6.1 msec and 11.7 msec, respectively, but lengths of 2.35 mm and 2.36 mm caused delays of 31.7 msec and 40.0 msec, respectively. Inactive areas longer than 2.37 mm caused complete block. With inactive regions 2.35 mm long, a 5% reduction in distal threshold reduced the delay time 35% and a 5% increase caused complete block. Delay time was also sensitive to variations in the membrane parameters. A 10% reduction in membrane capacitance produced approximately a 10% decrease in delay, but such a reduction in membrane resistance caused approximately a 10% increase in delay. Curves recorded in blocked regions of canine Purkinje fibers were simulated by superimposing nonpropagating potentials from proximal and distal sites. The familiar two-component wave forms and other contour variations were quite realistic in the simulation, demonstrating its effectiveness in predicting voltage variations in nonpropagating Purkinje tissue.

KEY WORDS computer simulation electrotonus in heart
conduction block mathematical model simple delay
passive membrane parameters

Previous studies (1, 2) have suggested that transmission through a section of conduction block produced by electrical depolarization in canine specialized conduction fibers may be explained by electrotonic interaction. Electrical depolarization, although not a physiological condition, is used to model physiological block because its effect is reversible and relatively easy to control. The two-component wave forms characteristic of simple delay are routinely produced in electrical depolarization block. This paper reports the behavior of a passive cable model of electrotonic interaction, using physiological parameters and signals. Conduction delay in the model is related to the time required for the attenuated signal from the proximal boundary of the block to reach threshold and initiate a regenerative response at the distal boundary of the block. The characteristic two-component wave forms associated with simple delay may result from interaction of electrotonic phenomena initiated at the proximal and distal active sites. Failure of the attenuated signal to reach threshold at the distal boundary of the block causes complete block. Intermediate forms of block may be explained by the introduction of slow phase-4 diastolic depolarization (2). Although it has not been proven that these assumptions form a valid model of physiological block, the wave forms

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reported here exhibit significant similarities with physiological data.

**Model**

Central to the model chosen to represent simple delay is a length of excitable tissue with an elevated threshold for initiation of regenerative impulses. It is well known that membrane behavior may be approximated by linear equivalent electrical networks if time-varying signals are applied below the level necessary for impulse generation.

The inactive fiber is modeled by cable theory; that is, an analogy is drawn between the fiber and a length of submarine cable. The resistance of the inner conductor of the cable represents the internal resistance of the fiber, and the outer conductor models the resistance of the surrounding fluid. The dielectric separating the two conductors is analogous to the passive membrane. Proximal and distal to this length of inactive fiber we assumed that the transmembrane potential was governed by a regenerative impulse modified by electrotonic interaction.

Several investigators have measured the passive cable properties of excitable tissue, using a variety of experimental preparations and techniques. Many different models have been used to represent the shunting of the inner and outer conductors through the membrane. The study reported in this paper was restricted to muscle fiber models, but the technique presented is independent of the distributed parameter system used.

In 1952, Weidmann (3) reported measurements of the electrical constants of Purkinje fibers based on the two-path transmembrane model shown in Figure 1B. Measurements were made using techniques of square wave testing introduced by Hodgkin and Rushton (4). Falk and Fatt (5) reported measurements on frog sartorius muscle fibers based on a three-path membrane model shown in Figure 1C. In contrast to the work of Weidmann, they used alternating current for their measurements (1 Hz to 10 kHz). Both experimenters analyzed their observations on the basis of cable theory, assuming the fibers to be linear and time invariant over the range of voltages used. Differences in results appeared because square wave testing obscured the high-frequency response of the cable system and masked the existence of a distinct third pathway.

Fozzard (6) reported measurements on cardiac Purkinje fibers which were consistent with a three-path model. In addition to square wave measurements similar to those of Weidmann, he measured the capacitive filling at the beginning of a voltage clamp. Comparison of electrophysiological and anatomical studies suggested that the pathways consisted of: (1) membrane resistance, rm, (2) membrane capacitance, cm, and (3) a series combination of tubule resistance, rt, and capacitance, ct.

In this study we compared the responses of passive two-path and three-path models to an applied cardiac action potential. Although actual resistances and capacitances have been reported in the literature, only certain ratios (space constants and time constants) were

\[ A: \text{Section of coaxial cable. B and C are alternative models of a small segment of this cable used to model muscle fiber. In the model, the cable is assumed to be made up of a chain of such segments connected end to end. B: Two-path membrane model. C: Three-path system. The parameters represent internal resistance, } r_i, \text{ external resistance, } r_e, \text{ transmembrane resistance, } r_m, \text{ transmembrane capacitance, } c_m, \text{ tubule resistance, } r_t, \text{ and tubule capacitance, } c_t.\]
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necessary to describe the cable response. The membrane space constant, \( \lambda_m \), is the square root of the ratio of the membrane resistance to the sum of the internal and external resistances 

\[
\lambda_m = \sqrt{\frac{r_m}{r_i + r_e}}.
\]

For the two-path model, the maximum voltage attained along the line in response to a subthreshold voltage step falls to 1/e times the input step in a distance equal to one space constant. The membrane time constant, \( \tau_m \), is the product of the membrane resistance and the membrane capacitance (\( \tau_m = r_m c_m \)). In the two-path model, the voltage across the membrane at the site of an applied current step comes within 1/e of its maximum value in one time constant. Similar parameters may be obtained for the third pathway. These parameters do not have the same physical significance as their two-path analogues but appear in the same way in the voltage equations summarized in the Appendix (\( \lambda_i = \sqrt{\frac{r_i}{r_i + r_e}} \) and \( \tau_i = r_i c_i \)). The temporal and spatial distribution of voltage on the linear cable may be completely described using these parameters.

The three-path data from Falk and Fatt (5) were used as the primary model and compared to a two-path system which had the same low-frequency response (such as would be measured by techniques of square wave testing). The equivalent parameters for this two-path system fell within the range measured by Weidmann (3) for Purkinje tissue. The appropriate time constants and space constants are summarized in Table 1.

Solutions to the cable equations exist for a step current applied to the two-path model (4) and the three-path model (5), but these solutions are difficult to evaluate and apply to a model of conduction block. Since we were interested in physiological signals rather than square waves, the equations were solved numerically on the digital computer, using the cardiac action potential as the input. The action potential used in this study had a repetition frequency of 2 Hz with duration of 250 msec, resting potential of -90 mv, overshoot of 32 mv, and upstroke velocity of 500 v/sec. This signal was not in a form that could be used directly with the cable equations, since only a limited number of solutions have been found for these equations. The steady-state sinusoidal response is one solution that has been characterized. This type of signal is attractive for two reasons: (1) the time response of the system is easy to determine at any distance from the input and (2) a repetitive signal may be represented as a sum of sinusoids by use of Fourier techniques.

The electrotonic spread on the cable model was determined in the following way. First, the periodic input signal was reduced to its harmonic sinusoidal components by Fourier analysis. This procedure is explained by Defares and Sneddon (7). Each harmonic was then attenuated and shifted in phase by an amount dependent on the distance between the observation point and the input. The attenuation and phase-shift constants have been derived by Tasaki and Hagiwara (8) for the two-path model and by Falk and Fatt (5) for more general membrane systems including the three-path model. Finally, these modified sinusoids were summed to represent the waveform at the observation point. By calculating the response at various distances from the input, we could observe the degradation of the transmembrane action potential on a nonpropagating fiber model. The integrals and summations inherent in Fourier analysis require large amounts of computer time, and as a result this technique has been neglected until recently. Cooley and Tukey (9) introduced an algorithm in 1965 which performs these operations much more rapidly. As a result the Fourier transform has become a practical technique in digital computer solution of differential equations and simulation of

### Table 1

<table>
<thead>
<tr>
<th>Electrophysiological Constants for Two-Path and Three-Path Models</th>
<th>Space constant (mm)</th>
<th>Time constant (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two-path model</td>
<td>2.0</td>
<td>20.3</td>
</tr>
<tr>
<td>Three-path model</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Membrane</td>
<td>2.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Third path</td>
<td>0.65</td>
<td>1.3</td>
</tr>
</tbody>
</table>

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linear systems. The application of Fourier series analysis to this cable problem is outlined in more detail in the Appendix.

Simulation Results

**Passive Cable Response**

A distributed parameter system is characterized by complex interrelationships between distance and time. The time response changes as the observation point moves from the stimulus site. We have chosen to present the data graphically to demonstrate changes in time response as the wave is observed at different points along the passive cable. These data represent a very long cable so that electrotonic spread may be demonstrated explicitly without complications introduced by reflections.

Figure 2 shows the time response of long two-path and three-path cables to an applied cardiac action potential. The general characteristics of the two sets of curves are the same. As distance from the input increases, the wave shape is smoothed and the amplitude decreases. The phase-1 response of the transmembrane potential disappears within 0.8 mm of the input, and we see a progressive degradation of the upstroke velocity. As the observation point moves away from the input, more time is required for the voltage to reach a given value. For instance, as the observation point moves from 0.8 mm away from the stimulus site to 2.0 mm, the time required for the transmembrane potential to reach $-60$ mv increases from 1.22 msec to 11.7 msec. In Figure 2, the voltage never rises to $-60$ mv for the curves representing distances greater than 2.0 mm. The differences between the two models is not apparent in Figure 2, but there is some difference during the first 10 msec. Upstroke velocity degrades more quickly for the two-path model with values of 50 v/sec and 26 v/sec at 0.6 mm and 0.8 mm, respectively. At these same distances, the three-path model has velocities of 129 v/sec and 43 v/sec. Another manifestation of this relationship is the time the response remains at base line. On the two-path model, the voltage stays within 0.06 mv of the resting potential for 2.5 msec and 5.0 msec at distances of 3.4 mm and 4.8 mm, respectively. The three-path model is at base line for only 1.0 msec and 2.3 msec at these distances.

**Two-Component Wave Forms**

When a signal is delayed in traversing a region of blocked tissue, two-component wave forms are characteristically recorded. There is a first deflection temporally associated with the proximal transmembrane action potential and a second related to the onset of the transmembrane action potential distal to the block. The model presented in the preceding section is inadequate to describe simple delay, since it assumes an active response only at the proximal boundary.

The model for simple delay consists of a region of nonpropagating fiber, bounded proximally and distally by normal active tissue. A constant threshold of $-60$ mv (30 mv from the resting level) was assumed distal to the block. Hoffman and Cranefield (10) list thresholds between $-65$ mv and $-55$ mv for Purkinje tissue. When the transmembrane potential at the distal boundary reached this threshold by electrotonic spread from the proximal boundary, an action potential was initiated in fibers beyond the passive region. Since no active process is involved in the region between the two boundaries, both action potentials cause voltage deflections in the passive region and their effects must be superimposed. The delay is the time between onset of the transmembrane action potential...
just proximal to the passive region and onset of the active response in the distal fibers. By varying the length of passive tissue, we could vary the time necessary to reach threshold at the distal boundary.

The relationship between the length of block segment and delay time is summarized in Figure 3. Delay increases quite slowly with distance for a short block, but delay changes rapidly with length as the segment becomes longer. For short delays, the voltage is rising rapidly at threshold, and delay time is related to the initial upstroke velocity. For long delays, the maximum voltage is only slightly above threshold, and it changes slowly with time. The initial upstroke is completed, and these long delays are related to the time necessary to reach maximum voltage at a given distance.

For example, a delay of 1.95 msec occurs when the passive tissue extends 1.0 mm. A 20% increase in length increases the delay by 50%. On the other hand, a delay of 19.5 msec corresponds to a block length of 2.25 mm and a 5% increase in length increases delay by 60%. The maximum voltage reached at 2.25 mm is only 1.91 mv above threshold and occurs 47.6 msec after onset of the proximal transmembrane action potential.

Estimated maximum delay for a threshold of —60 mv is 50.6 msec, and it occurs at a passive tissue length of 2.372 mm. This estimated maximum delay is extrapolated from data on the maximum voltage attained at various distances and the time necessary to reach this maximum. The curves dealing with time to threshold are all carried to an estimated maximum delay. We assume that complete block occurs for lengths greater than those associated with this delay, since the transmembrane potential never reaches threshold.

There are several parameters in this simulation which can affect the delay associated with a given length of nonconducting fiber. The effect of the first of these, threshold potential at the distal boundary, is also summarized in Figure 3. If the threshold at the distal

![Figure 3](https://example.com/figure3.png)

**FIGURE 3**

*Delay vs. distance from the input.* A: Time necessary for electrotonic potential to reach threshold as the observation point moves from the proximal site of stimulus application. The central curve is for a threshold of —60 mv. The 10% curve is for a threshold of —57 mv and the —10% curve is for the threshold of —63 mv. The end-points (X) represent extrapolated maximum delay. B: Comparison of delay time for the two-path system (dashed lines) and three-path model (solid lines). Thresholds of —60 and —57 mv have been plotted. A logarithmic scale was chosen to demonstrate the difference between the two models, which is most apparent for very short delays (< 5 msec).
boundary of the passive fiber is increased, the delay associated with the block length is increased, since it takes longer to reach this higher voltage. Delay times associated with thresholds of $-57$ mV and $-63$ mV (±10% at a threshold 30 mV above resting potential) are shown in Figure 3. At 1 mm, delay varies from 1.71 msec to 2.20 msec as threshold increases through this range, representing a 29% increase. However, at 2.25 mm (delay of 19.5 msec) a decrease in threshold from $-60$ mV to $-63$ mV reduces delay to 14.2 msec, but an increase to $-57$ mV produces complete block. The extreme sensitivity of delay to threshold is important since slow phase-4 depolarization and membrane accommodation at the distal active site will surely change threshold. These data suggest, on a quasi-static level, some of the effects of these changes.

The remaining parameters investigated are passive properties of the nonconducting fiber itself. The broadest comparison involves the two-path and three-path models. The differences between the two-path and three-path systems are only evident at very short delays. The differences, which are masked in the presentation of Figure 3A, are emphasized in Figure 3B where a logarithmic scale was used for delay time. The three-path model reaches threshold more rapidly for delay times less than 5 msec. The curves are very similar for longer delays so that in the remaining study only the two-path model was used.

The passive membrane parameters, $r_m$ and $c_m$, may be changed in Purkinje fiber preparations (11). The membrane capacitance, $c_m$, affects only the membrane time constant. A 10% decrease in $c_m$ causes a 10% reduction in time constant. At a given distance, this 10% decrease in capacitance would cause approximately a 10% reduction in time to threshold. The effect of changing membrane capacitance is shown in Figure 4A. At each distance shown, delay decreases with decreasing capacitance.

The effect of changing resistance is more complicated, since $r_m$ occurs in the expressions for both time and space constants. A 10% reduction in $r_m$ produces a 10% reduction in time constant, decreasing delay time. On the other hand, this reduction in membrane resistance produces approximately a 5% reduction in space constant. For example, the response of a blocked segment 2.0 mm long with reduced space constant should have the same delay as the response of a 2.1 mm segment without such reduction. The two variations associated with a change in $r_m$ act

![Figure 4](http://circres.ahajournals.org/)

Delay vs. distance for variations in membrane parameters in the two-path system. A: Effect of variations in membrane capacitance (±10% around the control). B: Effect of varying membrane resistance. All curves are carried to the estimated maximum delay.
in opposite directions on delay. The effect on the space constant dominates over the effect on the time constant, and a reduction in \( r_m \) increases time to threshold at a given observation point by approximately 10%. This is shown in Figure 4B.

Table 2, taken from Figure 4, summarizes the effect of changing membrane parameters. The control data represent the system with a space constant of 2.0 mm and a time constant of 20.3 msec. The delay with \( c_m \) reduced by 10% is approximately 10% less at each distance.

The delay with a 10% reduction in \( r_m \) at 2.00 mm is 10% shorter than the control at 2.10 mm and the delay at 2.10 mm (reduced \( r_m \)) is 10% shorter than the delay at 2.20 mm (control).

Finally, note in Figure 4 that varying capacitance does not change the maximum passive length tolerated before onset of complete block. Only the delay time is decreased in direct relationship to \( c_m \). However, reduction of \( r_m \) decreases maximum block length as well as increasing delay time at a given length.

The maximum delays estimated in this study are shorter than some recorded in the laboratory. Two possible explanations may be suggested for this variance. First, the membrane parameters in blocked tissue may be different from those assumed here for normal tissue. It is possible that time and space constants may be quite different in blocked tissue, altering maximum delay. Second, slow phase-4 diastolic depolarization may lower the electrotonic potential required to initiate a

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**TABLE 2**

<table>
<thead>
<tr>
<th>Block length (mm)</th>
<th>Delay (msec)</th>
<th>Reduced capacitance (10%)</th>
<th>Reduced resistance (10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.00</td>
<td>11.7</td>
<td>10.5</td>
<td>12.7</td>
</tr>
<tr>
<td>2.10</td>
<td>14.2</td>
<td>12.5</td>
<td>15.6</td>
</tr>
<tr>
<td>2.20</td>
<td>17.3</td>
<td>15.1</td>
<td>20.8</td>
</tr>
</tbody>
</table>

**FIGURE 5**

Characteristic two-component wave forms for a delay of 11.7 msec. Transmembrane potential (TMP) vs. time for different distances away from the site of the proximal transmembrane action potential. The curve labeled Proximal is representative of waves in the proximal active region, recorded 0.5 mm ahead of the block. Numbers above the curves represent distance in millimeters from the proximal boundary. The curves recorded at 0.05 to 1.55 mm are in the passive region. The block region extends for 2.0 mm so the last four curves are recorded in the distal active region to show decay of the prepotential.
regenerative response at distal sites. The electrotonic potential from the proximal boundary superimposed on the diastolic depolarization may reach threshold after delays longer than those predicted by our model. Experimental measurements are needed to distinguish between these alternative explanations.

Figures 5 and 6 show characteristic two-component wave forms generated by the superposition of voltage from proximal and distal action potentials. The first component of this wave form is caused by voltage spreading from the proximal action potential and the second component by spread from the distal action potential. The degradation of upstroke velocity and double crestedness are quite prominent in these figures. The duration of the transmembrane potential near the proximal boundary is increased by the amount of delay (measured at 50% of peak amplitude). This prolongation is caused by the second deflection which appears on the repolarization limb coincident with the upstroke of the distal action potential. In the model, this deflection on the proximal repolarization phase is caused by electrotonic spread from the distal action potential. The prepotential recorded at distal sites represents electrotonic spread from the proximal active site. The disappearance of this prepotential with distance is shown in Figure 5.

Figure 6 shows the response at fixed positions within the region of electrotonic interaction for three different simulated delays: 19.0 msec, 31.7 msec, and 40.0 msec. Figures 5 and 6 suggest the variety of forms the characteristic two-component response may take.

The 11.7-msec delay of Figure 5 has been expanded in Figure 7. The first action potential conducted distally (recorded at 2.05 mm) has been superimposed to emphasize the timing relationships discussed above. A great deal of similarity exists between these curves and those recorded for canine Purkinje fibers (1). The first component is largest near the proximal portion of the block but decays as the observation site moves distally. The second component, rising with the onset of the
distal active response, increases as the test site approaches this distal edge. Likewise, its upstroke becomes sharper. The onset of the second component of the passive response comes closer to the onset of the distal transmembrane action potential as the distal boundary is approached.

**Discussion**

The passive model presented in this paper exhibits many similarities with data recorded in heart muscle tissue. Degradation of upstroke velocity and characteristic double cresting quite naturally result in the passive segment of the model, assuming regenerative response only at the proximal and distal boundaries. This model makes no attempt to describe the electrochemical changes present in blocked tissue but strongly suggests that curves recorded in such regions may result from electrotonic spread.

Several refinements could be made to extend the applicability of this model. We have assumed the electrically passive region to be a uniform linear cable bounded by normal active regions having constant threshold. This is often not the case in experimental chemical or electrical block. In both systems, the blocking agent may diffuse from a site of maximum depression with no clear demarcation between active and passive tissue. To model this effect, we could include a cable with high threshold at the center, decreasing to both sides. This would add to the complexity of the analysis, but it would not significantly alter the curves recorded. Only the distances required for the various delays would change.

A more serious limitation is imposed by our uniform cable assumption. The membrane parameters (resistances and capacitances) may also vary with the strength of the blocking agent. This variation could change the wave shape recorded in the blocked segment. Solution of systems with parameters which change with distance requires use of techniques employed in the analysis of tapered wave-guide sections in microwave theory. This also seems to be an unnecessary complication in view of the results shown.

The final limitations deal with the assumptions at the distal active site. Two competing effects may be important at this boundary. The active membrane exhibits slow phase-4 depolarization, lowering the voltage requirements for excitation which must be met by electrotonic spread. This depolarization could play a part in higher degree block (2) or could lead to increased maximum simple delay, depending on its time course. In addition, since the electrotonic potential is changing slowly at the distal active site, some membrane accommodation must surely occur (12), increasing the threshold for activation. Both limitations could be overcome by assuming active membrane kinetics at the distal boundary (13), but more information concerning transthreshold events is necessary to evaluate these two effects. The data concerning different thresholds is an attempt to
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demonstrate, on a quasi-static basis, the importance of these two effects.

In spite of the limitations suggested above, the model does predict voltage variations in simple delay quite adequately. Some observations from the simulation are summarized below. (1) The two-path and three-path models behave in a similar fashion, differing only in the first 15 msec. (The equivalent single time constant of the system is 20.3 msec and its exponential half-time is 14.1 msec.) (2) Short delays may be simulated quite readily, with a passive length of 2.0 mm producing a delay of 11.7 msec. (The assumed membrane space constant is 2.0 mm.) (3) A maximum delay of 50.6 msec is predicted for a passive length of 2.372 mm (18.63! longer than the membrane space constant). (4) Decreasing distal threshold decreases delay and increases maximum length tolerated before complete block. (5) Decreasing capacitance decreases delay and has no effect on maximum block length. (6) Decreasing membrane resistance increases delay and decreases maximum block length. (7) Long delays may be quite difficult to repeat experimentally because of the extreme sensitivity of long delay times to system parameters.

This model study suggests that the electrotonic interaction between areas proximal and distal to a blocked region is sufficient to cause the conspicuous changes in duration and configuration of the transmembrane potentials recorded in physiological systems.

**Appendix**

**FOURIER TECHNIQUES IN CABLE THEORY**

The voltage variation with time and distance for steady-state sinusoidal excitation has been published by Tasaki and Hagiwara (8) for the two-path model and Falk and Fatt (5) for more general transmembrane models. They were using sinusoidal analysis to determine passive membrane parameters, but the solutions they found are also useful for determining the response of the linear cable to nonsinusoidal, periodic excitation.

If a function $f(t)$ is repetitive with period $T$ and has only a finite number of discontinuities and turning points, it may be represented by a convergent Fourier series of the form

$$f(t) = a_0 + \sum_{n=1}^{\infty} \left( a_n \cos \frac{2\pi nt}{T} + b_n \sin \frac{2\pi nt}{T} \right).$$  \hspace{1cm} (1)

The constants $a_n$ and $b_n$ depend on the harmonic number, $n$, and the function $f(t)$. The d-c term $a_0$ is the average of the function $f(t)$, and $a_n$ and $b_n$ are averages over one period, $T$, of the fundamental frequency ($f_i = 1/T$) weighted by $\cos \frac{2\pi nt}{T}$ and $\sin \frac{2\pi nt}{T}$, respectively. The series is composed of sinusoids whose frequencies are integral multiples of the fundamental, $f_i$.

The input transmembrane action potential can be represented by a series of sinusoidal functions by computing the $a_n$'s and $b_n$'s from the appropriate averages. Because of its complicated shape, the transmembrane action potential was not put in closed mathematical form, but the averages were approximated by finite summations using potential values at regularly spaced points. The technique used for the evaluation is popularly known as the fast Fourier transform, developed by Cooley and Tukey (9). In the simulation it was necessary to use 2048 points equally spaced over one cycle (0.5 seconds) to reproduce an upstroke velocity of 490 v/sec. This resulted in 1024 harmonics in the series expansion ranging from 2 Hz to 2048 Hz.

Once the series representation of the transmembrane action potential input wave has been obtained, the output of any linear system can be calculated by use of a transfer function characteristic of that system. That is, if the input to a system is sinusoidal (given by Eq. 2), the output of the system will also be sinusoidal with the same frequency. Generally the output will be attenuated and shifted in phase compared to the input (Eq. 3).

$$f_i(t) = A \cos \omega t. \hspace{1cm} (2)$$

$$f_o(t) = H(\omega) A \cos(\omega t - \phi(\omega)). \hspace{1cm} (3)$$

The attenuation parameter [$H(\omega)$] and the phase shift [$\phi(\omega)$] usually depend on frequency.
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If, as in the present case, we have a Fourier series input rather than a single sinusoid, the output will be the sum of terms like Eq. 3. Each term of the input series is attenuated and shifted in phase by an amount dependent on the frequency of the harmonic and the characteristics of the system. The output is then the sum of this modified series.

For a linear transmission line the attenuation parameter and phase shift are dependent on the distance between the input and output as shown in Eq. 4a and 4b. That is, each different distance is considered the output of a different linear system.

\[ H(\omega) = e^{-\alpha x} \quad (4a) \]
\[ \phi(\omega) = -\beta x. \quad (4b) \]

The attenuation constant and phase constant are repeated here from the work of Tasaki and Hagiwara (8) for the two-path model:

\[ \alpha = \frac{1}{\lambda_m} \sqrt{1 + \frac{\sqrt{1 + 4\pi^2 f^2 \tau_m^2}}{2}}, \quad (5a) \]
\[ \beta = \frac{1}{\lambda_m} \sqrt{-1 + \frac{\sqrt{1 + 4\pi^2 f^2 \tau_m^2}}{2}}. \quad (5b) \]

The membrane and third-path constants are discussed in the text. These substitutions result in Eqs. 8a and 8b for \( \alpha \) and \( \beta \).

\[ \alpha = \frac{F}{\lambda_m} \sqrt{1 + \frac{\sqrt{1 + 4\pi^2 f^2 \tau_m^2}}{2}}, \quad (8a) \]
\[ \beta = \frac{F}{\lambda_m} \sqrt{-1 + \frac{\sqrt{1 + 4\pi^2 f^2 \tau_m^2}}{2}}. \quad (8b) \]

These two equations are similar to Eqs. 5a and 5b, but the detailed variation of these parameters with frequency is different.

These parameters along with Eqs. 4a and 4b completely define the cable system and allow us to determine the output transmembrane potential at any distance from an input potential. If the input to the system is given by Eq. 1, then the output, \( g(x, t) \) is

\[ g(x, t) = a_0 e^{-\alpha x} + \sum_{n=1}^{\infty} a_n \cos \left( \frac{2\pi n t}{T} - \beta_n x \right) + b_n \sin \left( \frac{2\pi n t}{T} - \beta_n x \right) \] (9)

The subscript \( n \)'s indicate that the parameters are frequency dependent.

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