The Structural Implications of
the Linear Electrical Properties
of Cardiac Purkinje Strands

W. H. FREYGANG and W. TRAUTWEIN

From the Second Physiological Institute of the University of Heidelberg, Heidelberg, Germany. Dr. Freygang’s present address is National Institute of Mental Health, National Institutes of Health, Bethesda, Maryland 20014. Dr. Trautwein’s present address is II. Physiologisches Institut der Universität Heidelberg, Heidelberg, Germany.

ABSTRACT An attempt has been made to decide upon the most reasonable equivalent circuit that will describe the passive linear properties of the Purkinje strands of sheep heart muscle. In order to do this, measurements were made of the phase angle of the characteristic admittance as well as the longitudinal impedance, both as functions of frequency. The results of both types of experiments indicate the presence of a longitudinally oriented capacity with a time constant of about 60–70 μsec. It is suspected that this capacity and time constant represent the connection between the cells, both radially and longitudinally. The plasma membrane contains another capacity with a time constant of about 15 msec.

INTRODUCTION

The Purkinje strand (false tendon) of the sheep heart continues to serve as a particularly useful type of cardiac muscle for electrophysiological experimentation. Despite some structural differences between Purkinje strands and ventricular muscle, the coupling between the cells appears to be identical (Sommer and Johnson, 1968). Recently Fozzard (1966), Weidmann (1966), Johnson and Sommer (1967), Sommer and Johnson (1968), and Page, Power, Fozzard, and Meddoff (1969) have related physiological and microscopic studies in order to explain the electrophysiological findings. The view that we obtain from these studies is that the coupling between the cells is purely resistive and that two capacities separate the myoplasm from the extracellular space. These two capacities, $C_m$ and $C_v$, in Fig. 1 A, are at the surface of the cluster of cells that comprise the electrically excitable portion of the strand and are recognizable from each other because one has a resistive element, $R_e$, in series with it. The experiments reported in this paper, however, suggest an alternative interpretation. It is that the coupling between
the cells is in part through a capacity and that only one capacity is to be found at the surface of the cluster of cells.

At the outset of this project, we knew from the work of Fozzard (1966) and that of Dudel, Peper, Rüdel, and Trautwein (1966) that the equivalent circuit had at least two time constants. These authors assumed that one could ignore a possible capacitive component in the longitudinal flow of current and that the two capacities were for radial current. We shall make the same assumptions initially in order to simplify the presentation here, although later we shall describe a capacity that is encountered by the flow of current in the longitudinal direction. These assumptions lead to four possible, four element equivalent circuits for the radial flow of current that we should consider. Each of the circuits has two time constants. The canonical or simplest forms of these circuits are illustrated in Fig. 1. If one allows five elements (three resistances and two capacities) in the two time constant circuit, there are twelve canonical forms.\(^1\) None of the 12 circuits will behave as a pure capacity at very high frequencies. In general, biological membranes do behave as pure capacities at very high frequencies and we therefore make the assumption that Purkinje strands are not exceptional in this regard. Therefore we have not added a resistance in series to each circuit in Fig. 1.

Fozzard (1966) chose to interpret his data by assuming that circuit A in Fig. 1 represented the structure of a Purkinje strand. The resistance, \(R_m\), and capacity, \(C_m\), in parallel were taken to represent the plasma membrane that separates the inside of the cells from the outside. The term plasma membrane, as used here, refers to the electrically excitable membrane that produces the action potential. Capacity \(C_e\) in circuit A is in a membrane between the inside of the cells and a space that is connected to the external

\(^1\) Personal communication from Ronald M. Foster to Kenneth S. Cole who transmitted it to us.
solution through a resistance $R_s$. It must be assumed in this circuit that the membrane resistance in parallel with $C_s$ is either so large that it cannot be measured or, more likely, that it is distributed between $R_s$ and $R_m$. Resistance, $R_s$, could be in the clefts between the cells; it is probably not in a transverse tubular system because this system is poorly developed or absent in Purkinje cells (Sommer and Johnson, 1968).

Fig. 1 B is also a possibility for the likely equivalent circuit. Here one could assume that $R_p$ and $C_p$ represent the resistance and capacity of the plasma membrane. The resistance and capacity, $R_{nr}$ and $C_{nr}$, could represent another barrier in series with the plasma membrane. This other barrier could be the connections between the cells (nexus or tight junctions) or the basement membrane that surrounds the bundles of cells.

The circuits C and D in Fig. 1 do not seem to be likely possibilities. The

| TABLE I COMPOSITION OF SOLUTIONS |
|----------------------------------|
| Solution                      | K $^+$ | Na $^+$ | Choline | Ca $^{2+}$ | Mg $^{2+}$ | Cl$^-$ | HCO$^-$_3 | HPO$_{-}$_4 | Sucrose |
|-------------------------------|--------|---------|---------|------------|------------|--------|-------------|-------------|---------|
| Tyrode, mM                    | 5.4    | 148.3   | —       | 1.8        | 1.0        | 148    | 11.9       | 0.4         | —       |
| Choline tyrode, mM            | 5.4    | 12.3    | 137     | 1.8        | 1.0        | 148    | 11.9       | 0.4         | —       |
| 1 mM $K^+$ solution, mM       | 1.0    | 12.3    | 137     | 1.8        | 1.0        | 145.6  | 11.9       | 0.4         | —       |
| 0.5 mM $K^+$ solution, mM     | 0.5    | 12.3    | 137     | 1.8        | 1.0        | 143.1  | 11.9       | 0.4         | —       |
| 55.4 mM $K^+$ solution, mM    | 55.4   | 12.3    | 87      | 1.8        | 1.0        | 148    | 11.9       | 0.4         | —       |
| Sucrose solution, mM          | 5.4    | 12.3    | —       | 1.8        | 1.0        | 11.0   | 11.9       | 0.4         | 137     |

plasma membrane of electrically excitable cells must generally be represented by a resistance and a capacity in parallel. The plasma membrane would then be represented by $C_2$ and $R_i$ in these circuits. The structural nature of the pure capacity, $C_s$, in circuit C, or pure resistance, $R_i$, in circuit D, in series with the plasma membrane is difficult to imagine. Also, the nature of the components in parallel with the plasma membrane would have to remain mysterious. It seems to us, therefore, that the circuits A and B in Fig. 1 are the most likely canonical forms from which we have to choose. Of course it is possible, or likely, that the Purkinje strand is represented by a two time constant circuit that has more than four elements in it and is not a canonical form. In that case, all one can do is to choose the canonical form that is the closest approximation to the structure.

Preliminary reports of this work have already appeared (Freygang and Trautwein, 1969; Trautwein and Freygang, 1969).

**METHODS**

The experiments were performed on Purkinje strands from sheep at temperatures between $35^\circ$ and $37^\circ$C. The preparations used for the phase angle measurements
were 1 cm long, or longer, and relatively thin without branches. The composition of
the solutions in which the preparations were immersed is listed in Table I. A mixture
of 95% O₂ and 5% CO₂ was bubbled through the solutions. At least a half-hour of
immersion in each solution was allowed before measurements were made.

Measurements of the Phase Angle of the Characteristic Admittance, \( \theta \)

The arrangement of the apparatus for this type of experiment is illustrated in Fig. 2.
The method is essentially the same as the one described by Freygang, Rapoport, and
Peachey (1967). The crux of the method is avoidance of the complications that arise
from the capacity across the wall of the micropipette with which membrane potential,

\[ E_m, \]
can be measured (the right-hand one). This was accomplished by holding the
voltage inside this pipette at the resting potential of the impaled cell. The amplifier,
\( A_2 \), applied the negative feedback to do this. The pipette on the left side received
the alternating current from the oscillator. The grounded shield between the pipettes
reduced the capacitive coupling between them so that no cross-talk was seen except
at the highest frequencies (3 kHz and higher). The amount of cross-talk could be
observed when the left pipette was withdrawn so that its tip was just outside the cell
while the right pipette's tip remained in the cell. The phase angle between the signals,
\( E_s \) and \( E_n \), was measured with a AD-YU Electronics Inc. (Passaic, N. J.) Type
405L-3 precision phase meter. Further details and an analysis of the circuit can be
found in the paper of Freygang et al. (1967).

In order to record a curve that had a well-defined hump and did not exceed 45°
we attempted to have the tips of the pipettes as close together as possible in the prep-
aration (Falk and Fatt, 1964). Since one cannot see the tips after they have been
inserted in the preparation, we adjusted their relative positions to one another so that
the phase angles were a minimum at high frequencies. Despite these efforts, the phase angles did exceed 45° at high frequencies. The sinusoidal component of $E_m$ was less than 5 mV peak to peak.

Measurements of the Longitudinal Impedance

The chamber that we employed for the measurements of the longitudinal impedance is illustrated in Fig. 3. One of the platinum black electrodes was connected to a grounded point in the Wheatstone bridge. The output of the bridge was amplified and observed on an oscilloscope. The source of the alternating current to the bridge was a Princeton Applied Research Corporation (Princeton, N. J.) Model JB 6 two phase lock-in amplifier. The amplified output of the bridge was also fed to the lock-in amplifier in order to achieve a balance of the bridge with very little current flowing in the preparation. The most current applied to the preparation was less than 1 µAmp. At low frequencies and with much higher currents than this, it was possible to see action potentials and nonlinear behavior in the amplified output from the bridge. The capacity of the chamber without a preparation in it was subtracted from the measured capacities.

Drift in the bridge balance was generally severe enough to require compensation. This was done by referring the readings back to those measured at a fixed frequency. It did not matter if the fixed frequency was high or low; the same corrected values were obtained in both cases. The source of the drift seemed to be the amount of fluid that surrounded the portion of the strand that was immersed in paraffin oil.

The readings from the bridge for the parallel conductance, $G$, and parallel capacitance, $C$, for each frequency were converted to the equivalent series resistance, $R_s$, and series reactance, $X_s$, by means of the formulae

$$ R_s = \frac{G}{G^2 + (\omega C)^2} \quad \text{and} \quad X_s = \frac{-\omega C}{G^2 + (\omega C)^2} $$

in which $\omega = 2\pi$ frequency. The values of $R_s$ were corrected for the resistance of the chamber by subtracting this value (generally 300Ω) from the value of $R_s$. One can calculate that this resistance was the resistance of the fluid in the holes in the parti-
tions. The frequency-dependent series reactance of the junction between the electrolyte and the platinum black electrodes was too small to require subtraction of it from the values obtained when a preparation was in the chamber.

After the impedance measurements had been made, the Purkinje strands were frozen, sectioned, and stained. The cross-sectional areas occupied by the Purkinje cells and by the connective tissue were sketched from optical projections of the histological sections. The areas of 5–10 sections of each preparation were measured with a planimeter. The mean areas of each Purkinje strand and the mean percentages of the area occupied by the Purkinje cells in the sections are listed in Table VIII. In most preparations these percentages were rather constant, except in a few preparations in which side branches were cut. The data from those preparations with cut branches were discarded.

**Theory**

The Phase Angle of the Characteristic Admittance, $\theta$

A general circuit that describes a Purkinje strand is drawn in Fig. 4. Any of the circuits for the radial flow of current in Fig. 1 could have been chosen since they have the same characteristics. The choice of the circuit for the path for longitudinal flow of current is also arbitrary. It could be the circuit drawn in Fig. 5 B rather than the one in Fig. 5 A; both circuits are equivalent to each other. We chose the circuit in Fig. 5 A because the structural significance could be that $r_m$ is the myoplasmic resistance and $r_{nl}$ and $c_{nl}$ could be the longitudinally oriented junctions between the cells. The circuit in Fig. 5 B has no significance according to present knowledge of the ultrastructure of Purkinje fibers.

The characteristic admittance, $A_\theta$, is the complex ratio of the current applied to a point in an infinite cable, such as the one drawn in Fig. 4, divided by the voltage induced at that same point. For any such infinite cable with distributed parameters it follows from the cable equations that the

![Figure 4. The general circuit. All the parameters are for a unit length of the strand. Thus, $r_p$ and $r_{mr}$ have the dimensions of ohm·cm, $C_p$ and $C_{mr}$ are $\mu$F/cm, $r_m$ and $r_{nl}$ are ohm/cm, and $C_{nl}$ is $\mu$F·cm.](image-url)
characteristic admittance is given by equation 1

\[ A_0 = 2(Z_l \cdot Z_r)^{-1/2} \]  

(1)

in which \( Z_l \) is the impedance in the longitudinal direction and \( Z_r \) is the impedance in the radial direction. Equation 1 can be easily obtained from the equations of Falk and Fatt (1964). For the circuit in Fig. 4, \( Z_l \) is given by equation 2

\[ Z_l = r_{nl} + r_m(1 + \omega^2 T_{nl}^2) - j\omega T_{nl} r_{nl} \]  

(2)

in which \( T_{nl} = c_{nl} \cdot r_{nl} \).

\( Z_r \) is given by equation 3

\[ Z_r = \frac{r_p \left( r_{nr} + \frac{1 + \omega^2 T_{nr}^2}{1 + \omega^2 T_p^2} \right) - r_m \left( 1 + \frac{r_m}{r_{nl}} (1 + \omega^2 T_{nl}^2) \right)}{1 + \omega^2 T_{nr}^2} \]  

(3)

\[ \tan 2\theta = \frac{r_{nr} \left( T_{nl} + T_{nr} \right) + \left( T_{nl} + T_p \right) \left( 1 + \frac{r_m}{r_{nl}} (1 + \omega^2 T_{nl}^2) \right)}{r_p \left[ 1 - \omega^2 T_{nl} T_{nr} + \frac{r_m}{r_{nl}} (1 + \omega^2 T_{nl}^2) \right]} \]  

(4)

When \( r_m \) is zero, equation 4 simplifies to equation 4a.

\[ \tan 2\theta = \frac{r_{nr} \left( T_{nl} + T_{nr} \right) + \left( T_{nl} + T_p \right) \left( 1 + \frac{\omega^2 T_{nr}^2}{1 + \omega^2 T_p^2} \right)}{r_p \left[ 1 - \omega^2 T_{nl} T_{nr} \right] + \left[ 1 - \omega^2 T_{nl} T_p \right] \left( 1 + \frac{\omega^2 T_{nr}^2}{1 + \omega^2 T_p^2} \right)} \]  

(4a)
When $c_{nl}$ is zero, equation 4 simplifies to equation 4 b.

$$\tan 2\theta = \frac{\omega \left( \frac{r_{nr}}{r_p} T_{nr} + T_p \left( 1 + \frac{\omega^2 T_{nr}^2}{1 + \omega^2 T_p^2} \right) \right)}{r_{nr} + \frac{1 + \omega^2 T_{nr}^2}{1 + \omega^2 T_p^2}}$$  \hspace{1cm} (4b)

When $r_{nr}$ is zero, equation 4 reduces to equation 4 c.

$$\tan 2\theta = \frac{\omega \left[ 1 + \frac{r_m}{r_{nl}} + \omega^2 T_{nl} \left( 1 - \frac{r_m}{r_{nl}} \right) \right]}{1 + \frac{r_m}{r_{nl}} + \omega^2 T_{nl} \left( 1 - \frac{r_m}{r_{nl}} \right) - T_p}$$  \hspace{1cm} (4c)

**The Longitudinal Impedance**

As we have already discussed, the circuit in Fig. 5 A seems a reasonable circuit for the longitudinal flow of current. Unfortunately the whole strand of Purkinje cells requires a more complicated circuit because one needs a path for the flow of current in the connective tissue and Tyrode solution that surround the Purkinje cells. We have chosen the circuit in Fig. 6 to represent the whole Purkinje strand. The circuit in Fig. 5 A is part of Fig. 6, but we have added the elements $r_o$, $r_e$, and $c_e$ to account for the flow of current through the connective tissue and Tyrode solution. The configuration of the part of the circuit that includes these elements is arbitrary. Although the significance of the values of $r_o$, $r_e$, and $c_e$ is not at all clear, we need to calculate them from the data in order to calculate $r_{nl}$, $r_m$, and $c_{nl}$. It is not necessary to have a general equation for the impedance of the circuit in Fig. 6 because it is possible to calculate the parameters from the plot of $X$, vs. $R$, the impedance locus. The method of calculation is described in a subsequent section of this paper.

![Figure 6. Circuit for longitudinal current in a Purkinje strand. $r_o$, $r_e$, $r_m$, and $r_{nl}$ have the dimensions of ohm/cm and $C_{nl}$ and $C_e$ are $\mu$F.cm.](image-url)
RESULTS

The Phase Angle of the Characteristic Admittance, $\theta$

The result of an experiment is illustrated in Fig. 7. The sodium concentration was low (12.3 mM) in the Tyrode solution, most of it having been replaced by choline in order to avoid nonlinear behavior. The two curves in Fig. 7 are attempts to fit the data with equations 4a and 4b. The same values for the parameters were used in both equations. It was assumed that $T_{\tau r}$ equalled $T_{n1}$ in the calculation with equation 4a. If these time constants represent the connections between cells, one might expect that the time constant for the radial connections should be the same as that for the longitudinally oriented connections. As will be described later, we did find that the two time constants were about equal. Clearly the better fit was obtained with equation 4a at the higher frequencies. The significance of the last two points at high frequencies is dubious because of the cross-talk between the pipettes. At frequencies below 100 Hz, however, both equations fit the data equally well with the same values for the parameters. This part of the curve depends upon the values of $T_p$ and $r_{\tau r}/r_p$ and is not sensitive to the value of $T_{n1}$. When equation 4b is employed, $\theta$ cannot be greater than 45°. Values of $\theta$ greater than 45° are possible with equations 4 and 4a because the denomina-

![Figure 7](image-url)
tors of these equations can have a negative sign. At high frequencies, and with the same values for $T_p$, $r_{nr}/r_p$ as well as for $T_{nr}$ and $T_{ni}$, which are the same, the values of $\theta$ will be greater when equation 4 $a$ is employed than when equation 4 is used. The difference between the values of $\theta$ from equations 4 and 4 $a$ at high frequencies depends upon the value of $r_m/r_{ni}$. Generally the curves that we obtained experimentally were between the two extremes of the calculated curves in Fig. 7. Our conclusion from this is that a longitudinally oriented capacity, such as $c_{ni}$ in Fig. 4, plays a role in determining $\theta$ and must be included in the equivalent circuit because values of $\theta$ greater than 45° appear at high frequencies. The cross-talk between the pipettes is too small to explain the angles greater than 45°. As we shall describe later, the measurements of the longitudinal impedance also showed a capacity in the path of the longitudinal flow of current.

In order to fit the theory to the experimental data, then, equation 4 should be used. Unfortunately, that requires that $r_m/r_{ni}$ be evaluated. This greatly increases the amount of labor in the calculations. However, since both equations 4 $a$ and 4 $b$ fit the experimental data at low and intermediate frequencies as well as equation 4, we chose equation 4 $b$ for this purpose because it is the simpler. We were therefore unable to evaluate $T_{nr}$, $r_m/r_{ni}$, and $T_{ni}$ from measurements of the phase angle $\theta$. We could only estimate an upper limit for $T_{nr}$ from equation 4 $b$, in addition to $T_p$ and $r_{nr}/r_p$, of course. We were, however, able to estimate $r_m/r_{ni}$ and $T_{ni}$ from the longitudinal measurements of impedance.

It is not difficult to evaluate $T_p$, $r_{nr}/r_p$, and the upper limit of $T_{nr}$ when equation 4 $b$ is employed. Estimates can be made from approximate equations:

$$T_p \approx \frac{\tan 2\theta}{\omega} \quad \text{at the low frequencies},$$

$$T_{nr} \quad \text{(upper limit)} \approx \frac{\tan 2\theta}{\omega} \quad \text{at the high frequencies},$$

$$r_{nr} = \frac{\omega T_p - \tan 2\theta}{\tan 2\theta - \omega T_{nr}}(1 + \omega^2 T_{nr}^2)$$

$$\frac{r_m}{r_p} = \frac{\omega T_p - \tan 2\theta}{\tan 2\theta - \omega T_{nr}}(1 + \omega^2 T_{nr}^2)$$

at the intermediate frequencies.

All the results given in Tables II through VII were obtained by using these approximate values as guides and then more accurate values were obtained by automatic digital computation using the method of least squares.

In experiments with a few Purkinje strands, the phase angles went almost to 45° at the intermediate frequencies and then gradually to larger angles at higher frequencies. Such a result can be approximated by equation 4 $c$. This equation applied when $r_{nr}$ in Fig. 1 B is zero. Our suspicion is that $r_{nr}$ was
small in these preparations because there were many radial connections between the cells.

The values of the parameters in the other likely equivalent circuit, Fig. 1A, can be obtained from the values of $\frac{T_{nr}}{r_p}$, $\frac{T_{nr}}{r_m}$, and $T_{nr}$. The equations that relate these two circuits are as follows:

$$T_s = \frac{T_{nr} + \frac{T_{nr}}{r_p}}{1 + \frac{T_{nr}}{r_p}}$$

in which $T_s = c_s r_s$. Knowing $T_s$, we calculate $T_m = c_m r_m$

$$T_m = \frac{T_{nr} T_p}{T_s}.$$ 

Next we calculate $\frac{1}{c_{m'r_s}}$ from the equation

$$\frac{1}{c_{m'r_s}} = \frac{1}{T_{nr'}} + \frac{1}{T_p} - \frac{1}{T_s} - \frac{1}{T_m}.$$ 

$$\frac{r_s}{r_m}$$ is then $\frac{c_m r_s}{T_m}$ and $\frac{c_m}{c_s} = \frac{T_m}{T_{nr'} + T_p + T_m - T_s}$.

Values for both sets of parameters are given in Tables II through VII. It should be remembered that the values of $T_{nr}$ are upper limits in these tables.

The Effect of Changing the Potassium Concentration

In an attempt to identify the components in the circuits that determine the relation between phase angle and frequency, we altered the composition of the external solution. Fig. 8 illustrates the results of an experiment in which the concentration of potassium was changed from 5.4 to 55.4 mM. The effect was reversible when the strand was returned to the 5.4 mM potassium solution. The position of the micropipettes in the preparation was not altered during the change of solutions. Only the low frequency portion of this curve was altered by the change of solution. $T_p$ was reduced from 11 to 4.2 msec and $R_{nr}/R_p$ was increased from 0.073 to 0.190 by the 55.4 mM potassium solution, if the data in Fig. 8 are treated as if Fig. 1B were the equivalent circuit. This experiment shows the well-recognized effect of high potassium solutions in lowering the resistance of the plasma membrane (Noble, 1966; Dudel, Peper, Rüdel, and Trautwein, 1967). It seems reasonable that $R_p$, $C_p$, and $T_p$ represent the plasma membrane in the circuit in Fig. 1B. Since the high frequency portions of these curves were not altered by the raised potassium...
concentration, it seems likely that $T_r$, $r_m/r_{nl}$, and $T_n$ were not greatly altered by the change in potassium concentration.

If we treat the data in Fig. 8 as if the circuit in Fig. 1 A applied, we find that the high potassium solution lowered $T_m$ from 2.80 to 1.23 msec and raised $R_p/R_m$ from 0.131 to 0.380. The change in $T_r$ was small (0.981–0.889). Therefore we should represent the plasma membrane by $R_m$, $C_m$, and $T_m$ in Fig. 1 A.

Results of other experiments in which the potassium concentration of the Tyrode solution was lowered from 5.4 to 1.0 mm are listed in Tables II and III.

III. Lowering the potassium concentration increased $T_p$ and $R_p$ indicating the long recognized effects of low potassium on the resistance of the plasma membrane if Fig. 1 B is used as the model circuit. On the other hand, lowering the potassium concentration increased $R_m$ and $T_m$ if the circuit in Fig. 1 A is assumed. $R_m$ is then the resistance of the plasma membrane in the circuit of Fig. 1 A. The capacities in both circuits (Figs. 1 A and B) were not significantly altered by the changes in potassium concentration, nor were the other parameters significantly altered.

With respect to the significance of these results in terms of histological structures, the following views seem reasonable. If $R_{nr}$ of Fig. 1 B was the resistance of the connections between cells, the insensitivity of $R_{nr}$ to changes in the potassium concentration of the external solution seems a reasonable result. One would not expect changes in the extracellular potassium concentra-
tion to change the intracellular potassium concentration of the cells very much if the distribution of potassium is a Donnan equilibrium. The junctions between the cells are in contact with the internal potassium concentration. If \( R_n \) were in the basement membrane, we might not expect it to be altered because the conductivity of the external solution would not be greatly altered by the changes in potassium concentration. Therefore both of these possibilities remain for the circuit in Fig. 1 B.

For the circuit in Fig. 1 A, the results fit with the view that \( R_s \) could be in the clefts between the cells because the conductivity of the Tyrode solution was not greatly altered by the changes in potassium concentration. If the membrane containing \( C_s \) has a resistance, as it probably does, this resistance must be much greater than \( R_s \). This requires some explanation. If there were a resistance, \( R_{se} \), in parallel with \( C_s \) included in the circuit in Fig. 1 A, the apparent values that we would measure would be the quantities marked with an asterisk.

\[
T_m^* = \frac{C_m R_m (R_s + R_{se})}{R_m + R_s + R_{se}}
\]

\[
T_e^* = R_e C_e \left( \frac{R_{se}}{R_s + R_{se}} \right)
\]

\[
\left( \frac{C_s}{C_m} \right)^* = \frac{C_s}{C_m} \left( \frac{R_{se}}{R_s + R_{se}} \right)^2
\]
These values would not be sensitive to changes in $R_c$ if $R_{cs}$ were much greater than $R_e$. Thus $R_e$ could be changed by the changes in potassium concentration without changing $(C_e/C_m)^*$ and $T_e^*$ significantly.

| Fiber | $T_n'/T_n$ | $T_p'/T_p$ | $(R_{m}/R_p)'$ | $(C_p/C_m)'$ | $T_s/T_s$ | $(R_d/R_m)'$ | $(R_d/R_m)'$ | $T_m'/T_m$ |
|-------|-------------|-------------|----------------|--------------|------------|--------------|--------------|------------|
| B     | 1.02        | 1.77        | 1.67           | 1.04         | 1.13       | 1.23         | 1.74         | 1.60       |
| C     | 1.00        | 1.62        | 1.69           | 0.955        | 1.04       | 1.13         | 1.70         | 1.96       |
| D     | 0.98        | 6.97        | 7.96           | 0.995        | 0.995      | 1.15         | 7.85         | 6.87       |
| E     | 1.04        | 2.60        | 2.11           | 0.18         | 1.31       | 1.44         | 2.27         | 2.06       |
| F     | 0.92        | 4.07        | 5.60           | 0.788        | 0.825      | 0.820        | 4.53         | 4.56       |
| G     | 1.06        | 2.96        | 3.92           | 0.712        | 0.999      | 0.798        | 3.04         | 3.44       |
| Mean  | 1.00        | 3.33        | 3.81           | 0.930        | 1.04       | 1.10         | 3.52         | 3.35       |
| ±SE   | ±0.020      | ±0.813      | ±1.03          | ±0.069       | ±0.070     | ±0.101       | ±0.967       | ±0.852     |

In the box heading primed parameters refer to those in the 1.0 mm potassium solution.

| Fiber | Resting potential | Solution | $T_n$ | $T_p$ | $R_{m}$ | $R_{p}$ | $C_p/C_{m}$ | $T_s$ | $C_d/C_m$ | $R_d/R_m$ | $T_m$ |
|-------|-------------------|----------|-------|-------|---------|---------|--------------|-------|------------|-----------|-------|
| N     | 70                | Choline  | 0.33  | 15.0  | 0.207   | 9.41    | 2.85         | 6.18  | 0.265      | 1.74      |       |
| O     | 70                | Choline  | 0.37  | 75.5  | 0.070   | 14.28   | 5.29         | 12.35 | 0.081      | 5.29      |       |
| P     | 80                | Choline  | 0.30  | 42.5  | 0.094   | 13.32   | 3.33         | 10.97 | 0.110      | 3.25      |       |
| Q     | 80                | Choline  | 0.30  | 23.5  | 0.113   | 8.85    | 2.66         | 6.96  | 0.144      | 2.66      |       |

Effects of Cocaine

The effects of adding 0.1% cocaine to the choline Tyrode solution were the same as those obtained when the potassium concentration was lowered. These results are summarized in Tables IV and V. Somewhat more pronounced effects were obtained with a 0.25% cocaine solution. Only $R_p$ in the circuit of Fig. 1 B or $R_m$ in the circuit of Fig. 1 A was altered by cocaine. Again, it seems that these elements represent the resistance of the plasma membrane.

With respect to $R_e$, in Fig. 1 B, if it represents the resistance of the basement...
membrane, then this membrane is not sensitive to cocaine. This is perhaps unusual. If \( R_{sr} \) represents the junctions between the cells, it would indicate that cocaine does not enter the cells or does not come in contact with the junctional membrane. If the effects of cocaine are considered in respect to the circuit in Fig. 1 A, we must conclude that the material in the clefts between the cells is not sensitive to cocaine. This material is probably a mucopolysaccharide having a composition like that of basement membrane. The resistance

\[
\text{TABLE V}
\]

**EFFECT OF 0.1% COCAINE CHOLINE TYRODE**

| Fiber | \( \frac{T_{nr}}{T_{nr}} \) | \( \frac{T_{pr}}{T_{pr}} \) | \( \frac{(R_{nr}/R_{p})}{(R_{nr}/R_{p})} \) | \( \frac{(C_p/C_{nr})}{(C_p/C_{nr})} \) | \( \frac{T_{4}/T_{p}}{(C_p/C_{nr})} \) | \( \frac{(R_{sr}/R_{m})}{(R_{sr}/R_{m})} \) | \( T_{m}/T_{m} \) |
|-------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| N     | 1.12            | 5.03            | 2.96            | 1.52            | 1.86            | 2.00            | 3.27            | 3.04            |
| O     | 1.07            | 1.57            | 2.32            | 0.634           | 0.777           | 0.790           | 2.22            | 2.18            |
| P     | 1.25            | 2.94            | 2.66            | 0.885           | 1.31            | 1.25            | 2.68            | 2.81            |
| Q     | 1.31            | 2.61            | 3.13            | 0.637           | 1.05            | 0.801           | 2.50            | 3.26            |
| Mean  | 1.19            | 3.04            | 2.77            | 0.919           | 1.25            | 1.21            | 2.67            | 2.82            |
| ±SE   | ±0.056          | ±0.726          | ±0.178          | ±0.209          | ±0.231          | ±0.284          | ±0.222          | ±0.233          |

In the box heading primed parameters refer to those in cocaine solution.

\[
\text{TABLE VI}
\]

**EFFECT OF SUCROSE SOLUTION**

| Fiber | Resting potential | Solution | \( T_{nr} \) | \( T_{pr} \) | \( \frac{R_{nr}}{R_{p}} \) | \( \frac{C_p}{C_{nr}} \) | \( T_{4} \) | \( \frac{C_d/C_{m}}{R_{sr}/R_{m}} \) | \( T_{m} \) |
|-------|-----------------|----------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| I     | —               | Choline  | 0.28            | 12.5            | 0.190           | 8.48            | 2.23            | 5.72            | 0.249           | 1.57            |
| J     | 90              | Choline  | 0.40            | 8.00            | 0.100           | 2.60            | 1.09            | 1.49            | 0.249           | 2.53            |
| K     | 80              | Choline  | 0.50            | 7.25            | 0.270           | 5.92            | 1.94            | 2.10            | 0.491           | 1.87            |
| M     | 85              | Choline  | 0.32            | 11.5            | 0.076           | 2.73            | 1.11            | 2.23            | 0.150           | 3.22            |
|       | —               | Choline  | 0.33            | 8.50            | 0.340           | 8.76            | 2.40            | 4.51            | 0.457           | 1.17            |

in parallel with \( C_s \) must be much greater than \( r_s \) because \( R_s \) and \( C_s \) were not significantly altered. This conclusion is the result of the same argument that we used with respect to \( R_{sr} \) for the potassium experiments.

**Effects of the Sucrose Solution**

This solution (Table I) was employed because of its low conductivity. The results obtained with the sucrose solution are summarized in Tables VI and VII.

If the circuit in Fig. 1 B is assumed, the value of \( T_{nr} \) is seen not to change
significantly. More important, the data at high frequencies were not altered by the change of solution. Thus it seems likely that $T_{nr}$, $T_{nt}$, and $r_m/r_{nt}$ were not altered by the sucrose solution. This result would seem reasonable if $T_{nr}$ is located at the junctions between the cells because they would not come in contact with the external solution. If $T_{nr}$ were located in the basement membrane, we would certainly expect its value to rise in this low conductivity solution. This can be regarded as a strong argument against the basement membrane being the location of $R_{nr}$ and $C_{nr}$. If $R_{nr}$ and $C_{nr}$ were not altered much, we can see from Table VII that $R_p$ was reduced and $C_p$ was increased by the sucrose solution. One might expect on physical grounds that the capacity would decrease in a solution of low ionic strength (Everitt and Haydon, 1968). It may be that solutions of low ionic strength produce an increase in membrane capacity in the squid giant axon because when Guttman (1966) employed a sucrose gap she found the capacity to be four to eight times larger than the value she calculated from the measured surface area. The increase in capacity may also be a specific effect of sucrose. Measurements with direct current pulses also showed that the characteristic resistance of the Purkinje strands was lowered by the sucrose solution. The lowered $R_p$ did not return completely to its original value when the strand was returned to Tyrode solution. It would seem that the lowered $R_p$ is the result of permanent damage to the plasma membrane by the sucrose solution. Other studies with sucrose solutions have shown a rise in the resistance of the plasma membrane (Trautwein, Dudel, and Peper, 1965). Probably the discrepancy can be explained by the duration of the exposure to this solution.

When the circuit in Fig. 1 A is assumed, a lowering of $T_m$ also seems to be an effect of the sucrose solution. The cause is probably the result of the lowered resistance of the plasma membrane, as noted in the previous paragraph. $T_s$ was almost doubled. If $C_m$ is assumed not to change, this effect seems to come from an increase in both $C_s$ and $R_s$. Fozzard (1966) also found $T_s$ to be

| Fiber | $T_{nr}'$ | $T_p'$ | $(R_{nr}/R_p)'$ | $(C_p/C_{nr})'$ | $T_m'/T_m$ | $(C_s/C_m)'$ | $(R_s/R_m)'$ | $T_s'/T_s$ |
|-------|-----------|--------|----------------|-----------------|------------|----------------|----------------|------------|
| I     | 1.00      | 0.760  | 0.487          | 1.56            | 1.13       | 0.520          | 0.356          | 0.447       |
| J     | 1.25      | 0.906  | 0.370          | 1.96            | 1.78       | 0.507          | 0.368          | 0.372       |
| K     | 1.03      | 0.739  | 0.224          | 3.21            | 2.16       | 0.328          | 0.325          | 0.368       |
| M     | 1.89      | 0.703  | 0.206          | 2.02            | 2.46       | 0.392          | 0.595          | 0.455       |
| Mean  | 1.29      | 0.797  | 0.322          | 2.19            | 1.54       | 0.434          | 0.391          | 0.545       |
| ±SE   | ±0.207    | ±0.037 | ±0.066         | ±0.356          | ±0.253     | ±0.186         | ±0.047         | ±0.065      |
increased in preparations immersed in this sucrose solution. If $R_s$ represents the resistance in the clefts between the cells, it is very reasonable that it should be increased by the solution of low conductivity.

In an attempt to find an easy explanation for the increases in $G_p$ and $G_r$, we examined the cross-sections of cells in frozen histological sections. No gross alterations in surface area of the cells could be found as a consequence of immersion in the sucrose solution. Apparently the structural changes responsible for the increase in capacity are beyond the resolution of light microscopy.

The Longitudinal Impedance

The measurements of the longitudinal impedance, after conversion into the series resistance, $R_s$, and the series reactance, $X_s$, and correction for electrode resistance and stray capacity, yielded curves like the one in Fig. 9. Two distributions of data are apparent, a high frequency distribution and a low frequency distribution. In other experiments the dip in the values between the two distributions was deeper than the dip in this figure, but the curve in this figure is more typical of our results. When a Purkinje strand that had had its myoplasm squeezed out was used as the preparation, the high frequency distribution almost vanished. It would seem from this experiment that the low frequency distribution is a contribution of the connective tissue, although the presence of a reactance in the connective tissue was not expected. In order to confirm this, we used a piece of tendon from a heart valve to see whether it too had a reactance. Although the connective tissue of a tendon and a Purkinje strand may not be entirely the same structurally, we did find a reactance in the longitudinal impedance of the tendon that gave a low to intermediate frequency distribution.

EGTA (ethyleneglycol-bis($\beta$-aminoethylether)-$N,N'$-tetraacetic acid) makes the connective tissue very soft and spongy while the Purkinje cells appear to
be intact. The effect of EGTA is to remove the low frequency distribution. It seems likely, therefore, that the low frequency distribution is a contribution of the connective tissue and that the high frequency distribution arises from the Purkinje cells. The electrical constants for the circuit in Fig. 6 that describes the Purkinje strand were calculated in the following manner. The value of $r_m + r_n$ was calculated from the specific resistance for direct current of the core of a bundle of Purkinje cells, 110 $\Omega$cm (Weidmann, 1952; Fozzard, 1966), the cross-sectional area of the bundle of Purkinje cells, and the length of the strand. The cross-sectional area of the Purkinje cells and of the connective tissue was obtained from histological sections, as is described in the Methods section. Other constants were calculated from the following equations. $R_1$, $R_2$, and $R_3$ are described in Fig. 9, as well as $f_1$ and $f_2$.

\[
\begin{align*}
  r_e &= \left( \frac{1}{R_0} - \frac{1}{R_1} \right)^{-1} \\
  r_0 &= \left( \frac{1}{R_1} - \frac{1}{r_m + r_{nl}} \right)^{-1} \\
  r_m &= \left( \frac{1}{R_3} - \frac{1}{r_0 - \frac{1}{r_e}} \right)^{-1} \\
  r_{nl} &= (r_m + r_{nl}) - r_m \\
  C_e &= \frac{1}{\omega_1 \left[ \frac{1}{r_e + \left( \frac{1}{r_0 + \frac{1}{r_m + r_{nl}}} \right)^{-1}} \right]} \\
  C_{nl} &= \frac{1}{\omega r_{nl} \left[ \frac{1}{r_m + \left( \frac{1}{r_0 + \frac{1}{r_e}} \right)^{-1}} \right]} 
\end{align*}
\]

With the use of the measured cross-sectional areas for the bundles of Purkinje cells and for the connective tissue, as well as the length of the strand that was in oil, the constants for the circuit in Fig. 6 were converted into specific resistances and capacities. These values are listed in Table VIII. From Table VIII it can be seen that about 17% of the core resistance of the bundles of Purkinje cells is in parallel with a capacity. This specific resistance, $\rho_{nl}$, presumably represents the resistance of the junction between the cells for the longitudinal flow of current. The value for $\rho_{nl}$, which would represent the resistance of the junctional structures contained in a cubic centimeter of myoplasm, is 18 ohm cm. The specific resistance of the myoplasm alone was 92 ohm cm. The mean time constant for the junctions between the cells amounted
to 64 μsec. The physical significance of the other values, \( r_0 \), \( \rho_c \), and \( k_c \), which presumably represent the connective tissue and the Tyrode solution that surrounded the preparations, is not clear.

**DISCUSSION**

*The Likely Equivalent Circuit*

The measurements of \( \theta \), the phase angle of the characteristic admittance, strongly suggest that there is a capacity that is encountered by the longitudinal flow of current as well as by radial flow of current. The evidence for this view

| Strand No. | Area  | Myoplasm | \( r_0 \) | \( \rho_c \) | \( k_c \) | \( \rho_{in} \) | \( \rho_{ad} \) | \( k_{ad} \) | \( T_d \) |
|------------|-------|----------|----------|-----------|----------|-----------|-----------|----------|--------|
| 1          | 112.6 | 35.6     | 50.5     | 2095      | 4.5      | 99.9      | 10.1      | 3.3      | 33     |
| 2          | 30.9  | 31.4     | 18.0     | 424       | 22.9     | 73.3      | 36.7      | 4.0      | 148    |
| 3          | 24.9  | 20.8     | 19.5     | 712       | 6.0      | 82.2      | 27.8      | 2.5      | 68     |
| 4          | 70.8  | 15.5     | 22.3     | 1504      | 6.5      | 92.2      | 17.8      | 5.1      | 92     |
| 5          | 69.1  | 35.0     | 48.8     | 940       | 7.5      | 93.9      | 16.1      | 2.1      | 34     |
| 6          | 64.0  | 13.0     | 25.2     | 828       | 18.2     | 79.1      | 30.9      | 2.7      | 83     |
| 7          | 18.0  | 31.6     | 42.0     | 2128      | 1.5      | 99.3      | 10.7      | 0.2      | 2      |
| 8          | 32.3  | 18.5     | 22.2     | 3109      | 1.0      | 98.2      | 11.8      | 5.9      | 70     |
| 9          | 55.4  | 16.6     | 11.0     | 2301      | 1.4      | 95.2      | 14.8      | 4.9      | 73     |
| 10         | 75.0  | 21.3     | 13.9     | 1800      | 2.8      | 98.8      | 11.2      | 3.9      | 44     |
| 11         | 19.8  | 15.1     | 26.0     | 4091      | 0.38     | 97.8      | 12.2      | 4.8      | 59     |
| Mean       | 52.1  | 23.1     | 27.2     | 1812      | 6.6      | 91.8      | 18.2      | 3.6      | 64.2   |

is that values of \( \theta \) that were greater than 45° were encountered when higher frequencies were employed. Perhaps there are other explanations for values of \( \theta \) greater than 45°, but we cannot think of any. Attempts to explain angles greater than 45° as a result of branching in the preparation were not successful. Unfortunately we were unable to estimate the time constant of the longitudinally oriented capacity from the measurements of \( \theta \). From results like those shown in Fig. 7, however, one can suspect that the time constant of the radial capacity is less than 100 μsec. Measurements of the longitudinal impedance indicate the presence of a longitudinally oriented capacity with a time constant of about 64 μsec. Since we did find a longitudinally oriented capacity that one might expect to be located in the junctions between the cells, as predicted by the measurements of \( \theta \), it seems reasonable to believe that the circuit in Fig. 1 B is the likely circuit and that

\[ T_{nr} = T_{ad} \approx 64 \text{ μsec}. \]

Other evidence for this view is the insensitivity of the measurements of \( \theta \) at
high frequencies to changes in the external potassium concentration, cocaine, and the sucrose solution. These findings suggest that the structures that are primarily responsible for the values of θ at high frequencies are located within the Purkinje cells and are therefore isolated from the extracellular solution. On the other hand, our results with changes in potassium concentration, cocaine, or the sucrose solution support the circuit in Fig. 1 A very well. One can imagine that it represents the circuit for radial flow of current without there being a radially oriented capacity in the junctions between the cells.

We calculate the following values for a Purkinje strand in 5.4 mM potassium, Tyrode solution. These values are based on the assumption that the membrane is smooth; i.e., without folding. Such an assumption is far from the truth because Page et al. (1969) have shown that the membranes are quite extensively folded. Therefore the physical significance of these values is not at all clear. Their value lies in the use of them in generally describing the electrical properties of the strands used in rough calculations. We assume that $R_p$ has the generally encountered value of 1700 ohm cm$^2$. Since $r_{nr}/r_p$ is 0.14, $R_{nr} = 240$ ohm cm$^2$. $T_p$ is about 15 msec, so $C_p$ is about 9 μF/cm$^2$. $T_{nr} = T_{ni} = 64 \times 10^{-6}$ μsec, so $C_{nr} = \frac{64 \times 10^{-6}}{240} = 0.3 \mu F/cm^2$. These values represent the elements in Fig. 4. The values for $R_{nr}$ and $C_{nr}$ represent the resistance and capacity from the cell in which the two pipettes are located in the other cells radially adjacent to this cell. Since Purkinje cells are about 100 μ in length, $R_{nr}$ is higher and $C_{nr}$ is lower than they would be if the cells were infinitely long.

The circuit in Fig. 1 A was originally proposed for frog skeletal muscle by Falk and Fatt (1964). For this muscle, $C_e$ was taken to represent the capacity of the transverse tubular system, which is absent or sparse in Purkinje strands (Sommer and Johnson, 1968). In frog muscle, $R_e$ was thought to represent the resistance of some intracellular structure by Falk and Fatt (1964) and this view was supported by the experiments of Freygang et al. (1967). Since in Purkinje strands $R_e$ seems to be sensitive to solutions of low conductivity as was shown by Fozzard (1966) and as can be seen in this paper, an extracellular location for $R_e$ seems probable. We would guess that $C_e$ is the capacity of the membrane in the clefts in the cells and that $R_e$ is the resistance of the substance in the clefts. Since the membrane in the clefts seems to be a continuation of the surface membrane, one would expect it to have the same properties as the plasma membrane per unit area of its surface. We can then estimate that the resistance of the membrane in the clefts is about 2500 ohm cm$^2$ for a square centimeter at the surface of the cluster of cells that make up a Purkinje strand. The capacity would be about 7.5 μF/cm$^2$ for the membrane in the clefts. $R_e$ is estimated by Fozzard (1966) to be about 300 ohm cm$^2$. The membrane at the surface of the cluster would have a resistance of about 7500 ohm cm$^2$ and a capacity of 2.5 μF/cm$^2$. Such values yield the quantities...
$R_m = 2039 \text{ ohm cm}^2$, $C_m = 2.5 \mu F/cm^2$, $R_e = 336 \text{ ohm cm}^2$, and $C_e = 6.0 \mu F/cm^2$ for Fig. 1 A. These values are in accord with the estimates of Fozzard (1966).

If we consider that the longitudinal junctions between the cells are a flat surface, although we know that it is not a flat surface, we can calculate the longitudinal resistance between the cells. If we assume that the Purkinje cells are 100 μ in length, there are 100 cells per centimeter length. Therefore, since $\rho_{nl}$ was about 18 ohm cm, the resistance is about 0.18 ohm cm$^2$. It is of interest here to note that Weidmann (1966) estimated that the junctional resistance between the cells of calf myocardial tissue was 3 ohm cm$^2$ from his measurements of radiopotassium diffusion. The longitudinal junctional capacity of taenia coli has been estimated by Tomita (1969) to be 2 μF/cm, which is close to our estimate (3.6 μF/cm), but his estimate of $T_{nl}$ was much longer (0.5 msec) than ours (64 μsec). A capacity for the longitudinal junctions of 360 μF/cm$^2$ can also be calculated. These numbers suggest that the conductive and capacitive coupling between the cells is very large indeed. A decision about whether these numbers are reasonable on anatomical grounds must await an estimate of the junctional area between the cells at the intercalated discs.

The Foot of the Action Potential

One way to test whether the complete circuit is possible correct is to try to calculate the time constant of the foot of the action potential from it. The necessary equations were obtained in the following way. The circuit in Fig. 4 was assumed. We also assumed that $r_n$ and $c_n$ are absent in this circuit because all the cells should be conducting with the same velocity and there should be no flow of current between cells in the radial direction. If $V$ is the Laplace transform, with argument $p$, of the potential difference across the plasma membrane, $i_l$ is the longitudinal current, $x$ is distance along the length of the strand, $r_m$ is the resistance of a unit length of myoplasm, $r_{nl}$ is the resistance per unit length of the junctions between the cells, and $T_{nl}$ is the time constant of these junctions, we can write from the cable equations

$$\frac{\partial V(x, p)}{\partial x} = -i_l(x, p)r_m \left[ 1 + \frac{r_{nl}}{r_m} \left( \frac{1}{1 + pT_{nl}} \right) \right]$$

in which $i_l(x, p)$ is the Laplace transform of the longitudinal current.

Since the radial current, $i_r$, is given by the equation

$$-i_r(x, p) = \frac{\partial i_l(x, p)}{\partial x}$$

we obtain

$$\frac{\partial^2 V(x, p)}{\partial x^2} = i_r(x, p) \left[ r_m + \frac{r_{nl}}{1 + pT_{nl}} \right]$$
We can use the wave equation

$$p^2 V(x, p) = v^2 \frac{\partial^2 V(x, p)}{\partial x^2}$$  \hspace{1cm} (8)

in which $v$ is the constant conduction velocity in order to obtain

$$\frac{p^2}{v^2} V(x, p) = i_r(x, p) \left[ \frac{r_m}{1 + pT_{n1}} \right]$$  \hspace{1cm} (9)

The radial flow of current, $i_r$, is also

$$i_r(x, p) = \frac{1}{r_p} (1 + pT_p) V(x, p)$$  \hspace{1cm} (10)

in which $r_p$ is the resistance per unit length of the plasma membrane and $T_p$ is the time constant of the plasma membrane. Combining equations 9 and 10 and applying the inverse Laplace transform, we obtain the final equation.

$$V'' + AV' + BV + CV = 0$$  \hspace{1cm} (11)

in which the primes denote derivatives with respect to time and

$$A = \frac{1}{T_{n1}} - \frac{v^2 T_p r_m}{r_p}$$

$$B = - \frac{v^2 r_m}{r_p} \left( 1 + \frac{T_p}{T_{n1}} \left( 1 + \frac{r_{n1}}{r_m} \right) \right)$$

and

$$C = - \frac{v^2 r_m}{T_{n1} r_p} \left( 1 + \frac{r_{n1}}{r_m} \right)$$

Equation 11 was solved for the time constant of the foot of the action potential in the following manner

$$T_{n1} = 64.2 \mu\text{sec} \text{ (Table VIII)}$$

$$T_p = 15.2 \text{ msec} \text{ (mean from Tables II, IV, and VI)}$$

$$\rho_m = 91.8 \text{ ohm cm} \text{ (Table VIII)}$$

$$\frac{v^2 r_m}{r_p} = \frac{4v^2 \rho_m}{d R_p}$$

in which $d$ is the diameter, $v$ is the conduction velocity, and $R_p$ is the resistance (ohm cm²) of the plasma membrane. From Table I of Fozzard (1966), mean
values of these constants are

\[ R_p = 1714 \text{ ohm cm}^2 \]
\[ d = 78.3 \mu \]

therefore

\[ \frac{r^2}{r_m} = 1.95 \times 10^4 \]
\[ \frac{r_{nl}}{r_m} = \frac{\rho_{nl}}{\rho_m} = 0.198 \text{ (Table VIII)} \]

In equation 11 then

\[ A = -14.07 \times 10^3 \]
\[ B = -555.32 \times 10^6 \]
\[ C = -36.405 \times 10^9 \]

The reciprocal of the one positive root of the cubic equation is 0.03–0.04 msec, which is in the range of the average time constant of the foot of the action potential that Fozzard (1966) measured, which was 0.1 msec. This calculation should not be taken very seriously because it is very sensitive to the parameters that are chosen. Its value is only in showing that this test of the circuit does not rule it out.

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