J. Physiol. (1952) 118, 348–360

THE ELECTRICAL CONSTANTS OF PURKINJE FIBRES

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(*Received* 24 *April* 1952)

The aim of the present work was to measure the myoplasm resistance and the membrane resistance and capacity in Purkinje fibres of the mammalian heart. A knowledge of these characteristics is important in any discussion of cardiac excitation and conduction, and may also be of interest to those concerned with the movement of ions through cell membranes.

The theoretical method employed was similar in principle to that introduced by Hodgkin & Rushton (1946). This is based on the assumption that a single fibre can be regarded as a cable-like structure with a core of well-conducting protoplasm and a thin surface membrane having a high resistance and a large capacity per unit area. In the present case the analysis was simplified by keeping the fibre in a large volume of saline and using internal electrodes to apply current and to record potential. This has the advantage that fewer independent measurements are needed in order to determine the basic constants of the fibre.

The equations of cable theory are usually applied to a uniform fibre of infinite length, while the fibres of the cardiac syncytium fuse with one another at distances of less than 1 mm. The difficulties arising from this situation were reduced by using single fibres in the Purkinje system of the kid which were found to be free from interconnexions for a length of a few mm and by making use of a phenomenon characteristic for heart muscle, the 'healing-over' of cut surfaces.

THEORY

This section gives the results of applying the standard equations of cable theory (Cole & Curtis, 1938; Hodgkin & Rushton, 1946) to three special cases. In each case a current I_0 is applied to the interior of a fibre at a sealed end ($x=0$). The total resistance across the seal is assumed to be infinite, for reasons which are discussed on p. 350. At the other end, the cable is supposed to extend to infinity (case 1), or it is terminated at $x=L$ by a short circuit (case 2), or an infinite resistance (case 3). Case 3 applies to fibres which have been separated from the syncytium at both ends, while cases 1 and 2 are useful in considering fibres which have been left in connexion with the syncytium at one end.

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In the steady state the distribution of the electrotonic potential V is given by the following equations:

case 1 $$V = V_0 \exp(-x/\lambda), \quad (1)$$

case 2 $$V = V_0 \frac{\sinh[(L-x)/\lambda]}{\sinh[L/\lambda]}, \quad (2)$$

case 3 $$V = V_0 \frac{\cosh[(L-x)/\lambda]}{\cosh[L/\lambda]}, \quad (3)$$

where V_0 is the electrotonic potential at $x=0$ which is given by

case 1 $$\frac{V_0}{I_0} = r_i \lambda, \quad (4)$$

case 2 $$\frac{V_0}{I_0} = r_i \lambda \tanh(L/\lambda), \quad (5)$$

case 3 $$\frac{V_0}{I_0} = r_i \lambda \coth(L/\lambda). \quad (6)$$

λ is the space constant which is equal to $\sqrt{(r_m/r_i)}$ where r_m is the membrane resistance × unit length and r_i is the resistance of the core per unit length.

For each theoretical case there is now a pair of equations which can be solved for the two unknowns, r_i and r_m.

The specific resistance of the myoplasm (R_i) and the resistance × unit area of membrane (R_m) are then calculated by the equations

$$R_i = \pi a^2 r_i, \quad (7)$$

$$R_m = 2\pi a r_m, \quad (8)$$

where a is the radius of the fibre.

METHOD

Excised strands of the ventricular conductive system of the kid ('false tendons') were studied in a bath of Tyrode solution as described in an earlier paper (Draper & Weidmann, 1951). Single Purkinje fibres could sometimes be found in the finest arborizations of the false tendons, immediately before their entry into the ventricular wall. These had a thickness of 40–100 μ and measured a few mm between their peripheral cut end and the point of fusion with a thicker branch of the false tendons. The preparation was mounted as seen in Fig. 1 with the single fibre underneath

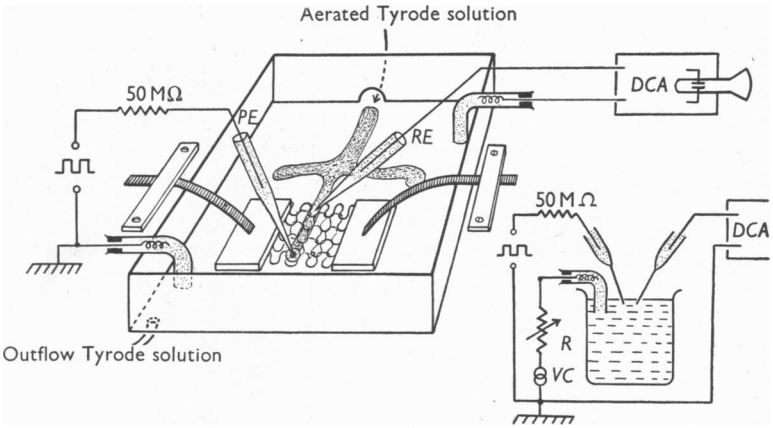

Fig. 1. Diagram of experimental arrangement (not to scale). *PE* = polarizing microelectrode, *RE* = recording microelectrode. Two indifferent electrodes (Ag-AgCl, Agar-Ringer) in contact with pool of Tyrode solution. *DCA* = balanced d.c. amplifier preceded by push-pull cathode follower. Inset: circuit used to monitor strength of polarizing current. *R* = step resistor of 10 × 100,000 Ω, *VC* = voltage calibrator.

a layer of ladies' stocking. The latter was held in place by Perspex blocks and pieces of watch spring. Friction at the meshes prevented the branch from rolling under the pressure of the penetrating electrode. No microdissection was required. On two occasions experiments were performed on single fibres which had been found in tissue bundles that cross the valleys between muscular trabeculae of the kid ventricles. About 50% of all hearts dissected contained a suitable preparation for the purpose of the present investigation.

A square wave of polarizing current was introduced into the fibre as shown in Fig. 1. The square pulses and their intervals were chosen sufficiently long (about 100 msec) for the process of charging and discharging the membrane to be practically complete at the end of each half-cycle. Anodal pulses were used, i.e. current flow increased the resting potential. The strength of the current was adjusted so as to produce potential changes of 5–10 mV at the site of the leading-in electrode. At the end of each experiment the current strength was monitored by a circuit as shown in the inset of Fig. 1.

The microelectrodes were of the type described by Ling & Gerard (1949), and had an external tip diameter of 0·5 μ or less. They were filled with a 3 M-KCl solution (Nastuk & Hodgkin, 1950).

A method described by Beutler & Stämpfli (1948) was used to estimate the specific resistance of Tyrode solution. The reference electrolyte was N/10-KCl.

RESULTS

The spread of the electrotonic potential

In most experiments the polarizing electrode was introduced into the fibre at a distance of about 50 μ from the cut end. A second microelectrode was moved along the fibre and inserted at various distances in order to record the amplitude and time course of the change in membrane potential produced by current flow. Fig. 2 illustrates the spatial decrement and time course of the electrotonic potential in a quiescent preparation.

As a rule the specimens were spontaneously active, and the value of their membrane resistance underwent cyclic changes. With such fibres all measurements were made at the beginning of diastole or, to be more precise, at 100 msec after the moment of lowest internal potential (cf. Fig. 2 of Weidmann, 1951*b*).

The properties of the sealed end

Fig. 3 shows the variation of resting potential along a fibre which had been cut at one end and allowed to seal over. It will be seen that the potential is practically constant and shows no decline near the cut end. This suggests that a new membrane has formed at the cut end and agrees with the well-known observation that the cardiac injury potential declines rapidly but can be restored by a new injury close to the old one (Engelmann, 1877, and others).

Evidence that the sealed end may be regarded as a high resistance is provided by the three sets of experimental points in Fig. 4. There the crosses give the variation in electrotonic potential when the current is applied at the sealed end. The two sets of circles give the effect of applying the same current at different distances from the sealed end and show that the electrotonic potential is practically constant in the region of $x=0$. This indicates that there is no

longitudinal current at $x = 0$, which means that the sealed end may be regarded as an infinite resistance. It must be emphasized that this result does not imply that the new membrane is especially effective as an insulator, or even that it is as good an insulator as a normal membrane. The cross-sectional area of the

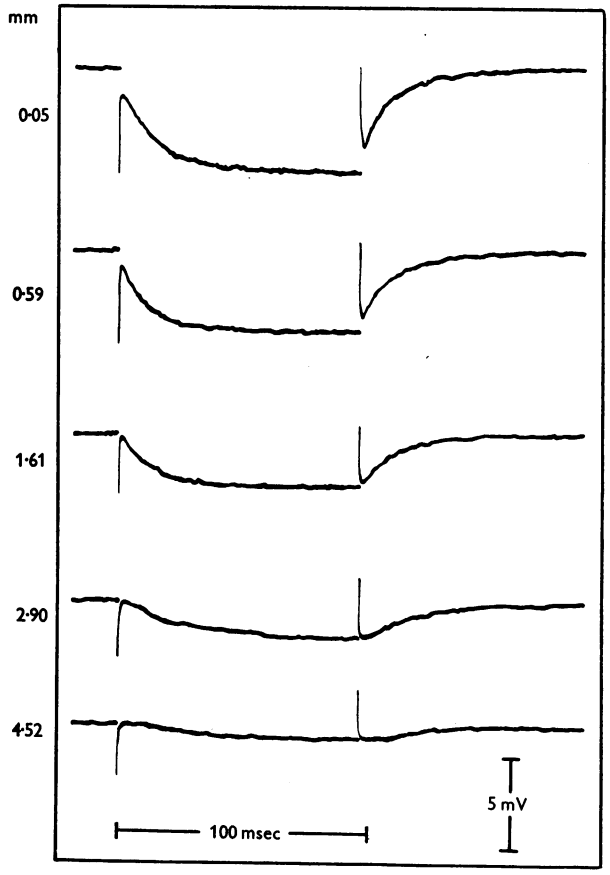

Fig. 2. Changes in membrane potential at various distances from polarizing electrode. Current flow caused downward deflexion. Make and break of each current pulse is marked by short spike (capacity artifact). From quiescent fibre no. 10.

fibre is about 5×10^{-5} cm^2 so that the terminal resistance would be of the order of 20 MΩ if the new membrane had a resistance of 1000 Ωcm^2, which is only half the average obtained in Table 1. 20 MΩ is effectively infinite since the characteristic resistance $[\sqrt{(r_m r_i)}]$ was about 0·5 MΩ.

The properties of the syncytial end

After entering the syncytium the Purkinje fibre branched repeatedly, so that one would expect the terminal resistance at the syncytial end to be lower than that which would exist if the fibre continued to infinity. A convenient

approximation is to assume that the inside and outside of the fibre are effectively short-circuited at the point where it enters the syncytium. This assumption, together with that discussed in the preceding paragraph, was

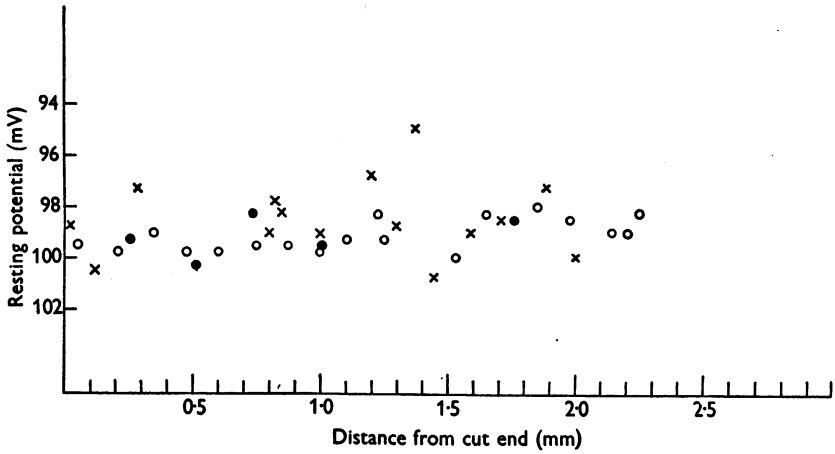

Fig. 3. Values of resting potential as measured at various distances from the cut end of a single fibre (no. 6).

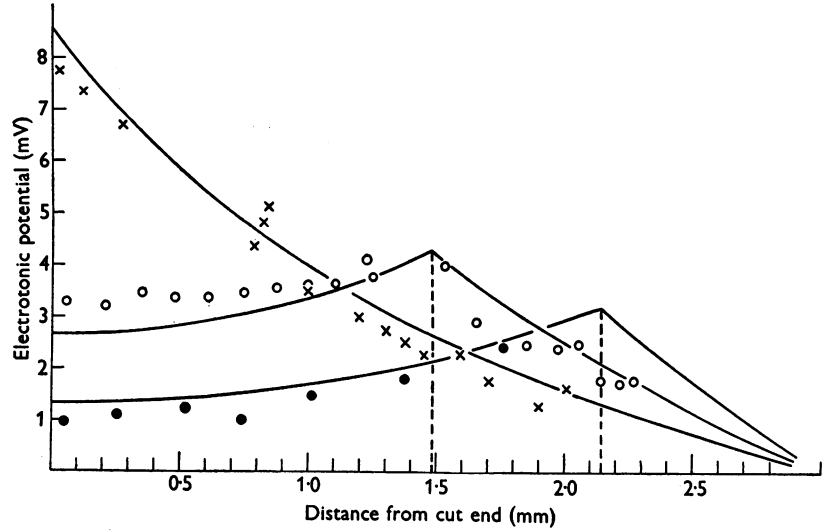

Fig. 4. Spatial distribution of electrotonic potential near cut end of fibre 6. Position of polarizing electrode at 0·02 mm (crosses), 1·48 mm (open circles) and 2·14 mm (full circles). Solid lines are theoretical curves; their parameters have been chosen by trial and error to give best overall fit.

used in calculating the three continuous curves in Fig. 4 from equations similar to those in the theoretical section. The same values of r_i and r_m were used for all three curves, so that the equations are subjected to a severe test. Thus if r_i and r_m are chosen to fit one set of points no further adjustments can

be made before drawing the other two curves. In spite of this there is reasonable agreement between theory and experiment in all three cases. It therefore appears that the assumptions of an infinite resistance at a sealed end and a short circuit at the syncytial end are reasonable approximations. An upper limit to the error introduced by the second assumption was obtained by recalculating the data using equations (1) and (4) instead of equations (2) and (5) (p. 354).

The basic constants

Table 1 summarizes the results obtained with eight different preparations; it also contains the primary data on which the calculations were based. R_m and R_i were obtained by inserting the values found for the space constant λ, for the polarization resistance V_0/I_0 and that for the fibre radius a into the

TABLE 1. Electrical constants of Purkinje fibres

Expt. No.	Theoretical case	Fibre diam. (μ)	L (mm)	λ (mm)	V_0/I_0 (kΩ)	R_i (Ωcm)	R_m (Ωcm^2)	τ_m (msec)	C_m (μF/cm^2)
3	3	94	4·0	2·3	375	106	2400	(39)	(16)
4	2	88	3·2	1·2	227	116	760	(22)	(29)
5	2	56	4·5	2·4	484	52	2140	(20)	(9)
6	2	53	3·0	1·4	487	79	1170	(18)	(15)
7	2	66	5·2	1·6	390	84	1300	18·5	14·2
8	2	90	4·3	2·0	580	190	3380	—	—
9	3	49	3·9	1·5	1050	130	2400	(22)	(9)
10	2	104	6·4	2·5	234	80	1930	20·5	10·6
Mean		75	4·3	1·9	478	105	1940	19·5	12·4

R_i = specific resistance of myoplasm (Ωcm); R_m = membrane d.c. resistance (Ωcm^2); τ_m = membrane time constant (msec); C_m = membrane capacity (μF/cm^2). For significance of other symbols see theoretical section.

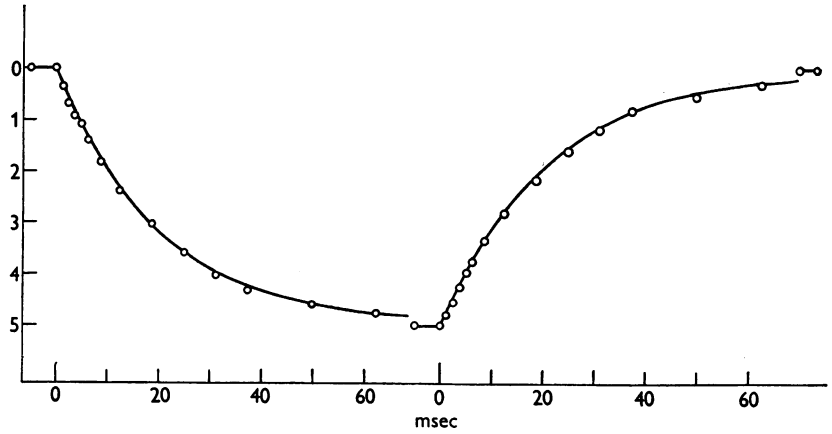

Fig. 5. Time course of the rise and fall of total membrane charge (relative units). The circles are experimental points obtained by graphical integration of the tracings shown in Fig. 2. The solid lines are exponentials with time constants of 20 and 21 msec for the rise and fall of charge respectively. The rise of membrane charge is plotted downwards.

equations in the theoretical section. Some comment seems necessary on the method by which the membrane time constant τ_m was determined. In the case of two quiescent preparations (nos. 7 and 10) a relatively laborious procedure was applied, namely that of computing the time course of the rise and fall of the total charge on the fibre membrane. This can be done by graphical integration of the potential-time curves as recorded at various distances from the polarizing electrode (method suggested by Hodgkin & Rushton, 1946). According to theory the rise and fall of the total membrane charge ought to follow exponential laws, if the fibre is sealed at one end and extends to infinity in the other direction. Fig. 5 shows that this expectation was fulfilled to a satisfactory degree in a fibre in which the total length was about 2·5 times the space constant. The remaining six preparations were spontaneously active which means that their membrane resistance was continuously changing. No theoretical method is available for calculating the time constant under these conditions, but an approximate estimate could be obtained by neglecting the resistance change and by finding the time necessary for the electrotonic potential to reach 84% of its 'final' value (cf. Table 1 of Hodgkin & Rushton, 1946). This was done on a charging or discharging curve which had been recorded in early diastole and from as close as possible to the site of the polarizing electrode. While there is no theoretical justification for such a procedure, it may be expected that the answer will not be radically wrong since over a period comparable to the membrane time constant (20 msec) the d.c. resistance of the plasma membrane increased by less than 10%. In Table 1 the values obtained with rhythmically active preparations are listed in brackets and they have not been considered in calculating the average.

Sources of error

(1) Most fibres fused with a syncytium of conductive elements at a distance L from their cut end. Beyond $x=L$ there was an increase in cross-sectional area and consequently a stronger flow of membrane current per unit distance than would be expected if the cable continued uniformly for an infinite length. However, there was no direct contact between the fibre core and the bathing solution as assumed in calculating the electrical constants. The maximal possible error arising from this simplification could be assessed by re-evaluating the experimental data on the assumption that the single fibre continued for an infinite length. The average space constant came out 6% lower, the specific core resistance 4% higher and the membrane resistance 2% lower than suggested by Table 1. The single fibre was generally longer than twice the space constant, which accounts for the relatively small amount of uncertainty.

(2) There may be alternative paths for the polarizing current than that through the fibre membrane. The possibility of current flow through cut surfaces was dealt with earlier in this paper, while evidence was given previously (Draper & Weidmann, 1951) that the hole in the surface membrane around the tip of the microelectrode does not represent any appreciable leak.

(3) It may be questioned whether the endocardial layer surrounding the tissue bundle did not represent a considerable resistance to current flow and thus was the seat of part of the total voltage drop. This possibility seems to be excluded by the following experiment. A continuous square wave of polarizing current was caused to flow into the core of the fibre through a first microelectrode. Potential was recorded continuously from the tip of a second capillary while this

was slowly lowered towards the bundle and finally inserted into the fibre. The record showed no potential changes synchronous with the flow of polarizing current up to the moment when action potentials appear which are typical for Purkinje tissue.

(4) The result of measuring the polarizing current may be falsified (i) by the presence of an appreciable resistance in the indifferent electrode (Fig. 1, inset), (ii) by a leak between the electrode pool and earth, and (iii) by possible resistance changes in the polarizing microelectrode. The first two possibilities could be excluded by the finding that the amplitude of the recorded square wave was proportional to the value given to the monitoring resistor. The third possibility cannot be dismissed entirely since the resistance of the polarizing microelectrode was not checked regularly. Occasional measurements indicated, however, that over a period of hours this varied by a few MΩ at most. Such variations would not have affected the results by more than a small percentage since the circuit contained a series resistor of 50 MΩ.

(5) A certain amount of arbitrariness was involved in fitting r_i and r_m to a plot of V_m against x. The scatter seen in Fig. 4 is typical for results obtained with spontaneously active preparations. In most experiments the estimate of r_i and r_m could be based on at least five determinations of V_m along the fibre. On two occasions, however, only two measurements were available (nos. 5 and 9), and it seems possible that under these conditions the values may be out by as much as 30%.

(6) It was shown in an earlier paper (Weidmann, 1951*b*) that the 'apparent d.c. resistance' of the surface membrane decreases under an anode and increases under a cathode. The effect can be demonstrated particularly well towards the end of diastole but is not entirely absent in the beginning of diastole when the present measurements were taken. For an increase in membrane potential of 7 mV (typical amplitude of V_0) the resistance of the surface membrane was found to drop by about 10%. This non-linear behaviour of the fibre membrane must have introduced some systematic errors, the characteristic length being slightly overestimated and the core resistance underestimated.

(7) Variations in fibre thickness along each single fibre are thought to have introduced considerable errors. The diameters listed in Table 1 are mean values obtained from a number of estimates along each particular fibre; variations of ±20% about this mean were not uncommon. Serial sections of one preparation suggested that the cross-sectional area varied by as much as 1:1·8.

In conclusion it may be said that the more important sources of error should have affected the results in a random way; it is thus expected that the correct means lie within the range of values determined in individual experiments.

DISCUSSION

Organization of the myoplasm

The specific resistance of the intracellular phase was found to be 105 Ωcm while the resistivity of the bathing fluid (Tyrode solution at 37° C) was determined as 51 Ωcm. Similar ratios have previously been reported for nerve fibres of different invertebrates and for nerve and muscle fibres of the frog (see Katz, 1948).

Purkinje fibres are histologically made up of sub-elements. These were described as 'granules' ('Körner') by Purkinje (1845) and are to-day known as 'Purkinje cells'. Histologists describe them as short, thick cylinders with peripheral myofibrils, two to five of them in cross-section making up a fibre (Wahlin, 1935; Glomset & Glomset, 1940; Truex & Copenhaver, 1947). It may be asked whether the boundaries of these cells are comparable to ordinary cell membranes in the sense that they represent effective barriers to the movement

of ions. This question was recently answered by Curtis & Travis (1951) who found that false tendons of the ox responded to electrical stimulation in an all-or-nothing manner which seemed only to be compatible with the assumption of protoplasmatic continuity. The relatively low value determined for the specific core resistance seems to add further evidence in favour of the view expressed by Curtis & Travis.

In 1877 Engelmann concluded, on the ground of electrophysiological and histological arguments, that 'cardiac cells are in direct contact during life (leitend miteinander verbunden) but become independent as they die'. According to Engelmann the phenomenon of 'healing over' would depend on ionic barriers being newly formed at the boundaries of non-injured cells. The present results are in full agreement with this view. A different opinion was expressed in recent papers by Rothschuh (1950, 1951). He pointed to the fact that with heart muscle the depolarization resulting from injury is reversed in a few minutes, while with damaged skeletal muscle a process of membrane discharge slowly spreads over the whole length of the fibres. From this observation Rothschuh postulated the existence of transverse electrical barriers along the core of living heart-muscle fibres. These membranes, spaced at distances of less than 1 mm, are supposed to be responsible for holding up the process of depolarization. The present results may have no bearing on the situation in ordinary cardiac muscle. But they strongly suggest that the hypothesis of Rothschuh is not applicable to Purkinje fibres. For it was found that a single fibre heals over wherever it is cut, although its longitudinal core resistance is so low that the presence of transverse barriers which are effective *in vivo* must be denied.

Data on the electrolyte content suggest that the resistance of cardiac myoplasm ought to be somewhat higher than that of Tyrode solution. According to Lowry (1943), one-quarter of the intracellular phase in rat heart is taken up by solids; the most important cation fraction in fibre fluid is potassium while the nature of the anions is mainly unknown; it seems likely, however, that the anions are relatively large particles and have a lower mobility than the extracellular chloride (Lowry, 1943). The value of the core resistance, as determined in the present experiments, seems to be consistent with the view that all intracellular ions are free to move; but the possibility is not excluded that a minor fraction of the intracellular potassium is chemically bound (Krogh, Lindberg & Schmidt-Nielsen, 1944).

The membrane resistance

The membrane d.c. resistance of Purkinje fibres ($R_m = 2000\ \Omega cm^2$) is similar to that of frog skeletal muscle ($4000\ \Omega cm^2$; Fatt & Katz, 1951) and to that of different kinds of invertebrate nerve fibres ($1000–10{,}000\ \Omega cm^2$; see Katz, 1948, and Weidmann, 1951*a*). Caution seems to be necessary in interpreting the

significance of R_m, especially in the case of Purkinje tissue. From results reported by Draper & Weidmann (1951) it would appear possible that the inward flux of sodium ions decreases when the membrane potential is slightly increased above its resting value. If so, the current carried by sodium ions would contribute a 'negative component' to the total membrane conductance. It may thus happen that future measurements with radioactive tracers will reveal a permeability to a single ion (e.g. potassium) which is higher than would appear possible on the ground of a membrane resistance of 2000 Ωcm^2.

The membrane capacity

Kahn (1941), applying rectangular current steps to frog ventricle, obtained potential-time curves which indicated the presence of more than one characteristic time constant. This result was tentatively ascribed to the use of non-homogeneous material; but it might also have been due to an anomalous reactance of the type discussed by Cole (1949) and Teorell (1949). In records taken from Purkinje fibres in the phase of early diastole there was no sign of any 'anomalous reactance'. Furthermore, with two quiescent fibres, the time course of the rise and fall of the total membrane charge could be fitted by single exponentials (Fig. 5). This finding is consistent with the assumption that the measured membrane capacity has the nature of an electrical double layer with low dielectric loss.

The value of C_m in Purkinje fibres appears to be rather high: 12 μF as against 6–8 μF in frog skeletal muscle (Fatt & Katz, 1951) and about 1 μF in different kinds of non-medullated nerve (table 7 in Katz, 1948). Viewed under the microscope, a living Purkinje fibre appears to have irregular outlines suggesting that the fibre membrane is slightly folded. Measurements on stained serial sections indicated that folding on a microscopic scale is responsible for increasing the fibre circumference by a factor varying between 1·2 and 1·8. Estimates with fixed material may be subject to artifacts; but it seems reasonable to assume that the unfolded membrane of Purkinje tissue would have approximately the same basic constants as that of frog skeletal muscle.

In a rhythmically active tissue the presence of a surface membrane with a high capacity calls for an intense exchange of ions between the extracellular fluid and the intracellular phase. The following calculation is similar in principle to one made by Hodgkin & Huxley (1947); it is based on the assumption that the membrane capacity has the nature of an electrical double layer and that C_m does not change in the course of a cycle of activity. In order to displace the membrane potential by 135 mV (amplitude of the action potential in Purkinje fibres; Draper & Weidmann, 1951) a charge corresponding to $1{\cdot}7 \times 10^{-11}$ mole of monovalent ions has to be transferred across each sq.cm of surface membrane of the capacity 12 μF. In the rising phase of the action potential this charge is most probably carried by sodium ions (see Hodgkin,

Huxley & Katz, 1949; Keynes & Lewis, 1951; Draper & Weidmann, 1951). The content of sodium within a cardiac fibre may be assumed to be somewhat below 10 m.mole/kg of myoplasm (Lowry, 1943; Robertson & Peyser, 1951). An amount equivalent to at least 1/1000 of the intracellular sodium content would thus enter the core of a 75 μ fibre at the onset of each cycle of activity; in order to maintain the ionic order an equivalent amount would have to leave the fibre during the remaining part of the cycle. At the end of 10 min or less a kid Purkinje fibre working at 100 beats per min would have exchanged a quantity of sodium corresponding to at least the total intracellular content. This statement needs to be checked by a more direct method although some sporadic data on the exchange of ^{24}Na in frog heart (Krogh & Lindberg, 1944) indicate that such a situation is not impossible.

In interpreting the effect of different ions on the heart it is often assumed that the delay with which the test solutions act is entirely due to exchange diffusion in the extracellular space. This assumption was tacitly made by Draper & Weidmann (1951) when discussing the action of sodium-deficient solutions on the cardiac membrane potential. In this particular instance it seems unlikely that the main conclusions drawn from the experimental results are erroneous. But the possibility of a rapid exchange of ions across cardiac fibre membranes should be envisaged in interpreting similar experiments.

Conduction velocity

According to the local circuit theory the conduction velocity should depend on the fibre radius a and on electrical constants such as the membrane capacity C_m and the specific resistance of the core R_i. It was suggested by Katz (1948) that the data obtained with different kinds of fibres (skeletal muscle, non-medullated nerve) show a reasonable correlation if it is assumed that the conduction velocity is proportional to a factor $\sqrt{a}/C_m\sqrt{R_i}$. The data of Table 1 may be related to known characteristics of the frog sartorius muscle (Katz, 1948); it is then predicted that kid Purkinje fibres ought to conduct at 1·1 m/sec; actually a propagation rate of 2·2 m/sec was measured (Draper & Weidmann, 1951). The formula used by Katz (1948) certainly does not include all factors of importance. For example, it was pointed out by Trautwein (1950) and by Nastuk & Hodgkin (1950) that, for a given C_m, the rate of rise of the action potential must be regarded as a further factor affecting the conduction velocity. The upstroke of the action potential is about twice as fast in Purkinje tissue (37° C) as it is in frog sartorius muscle (17° C) (Nastuk & Hodgkin, 1950; Draper & Weidmann, 1951). Furthermore, the measured conduction velocity of Purkinje fibres may exceed the predicted rate because all parts of the preparation undergo some slow depolarization during diastole; thus when an action potential is propagated it meets a surface membrane with a potential close to the instability level. Such arguments should not be used

to explain away discrepancies before they can be put on a more quantitative basis. On the other hand, it seems clear that there is a rough agreement between predictions and experimental results; this is taken as a further argument in favour of the view that Purkinje fibres are electrically organized in the same general way as the fibres of skeletal muscle or non-medullated nerve.

SUMMARY

1. Cable analysis was performed on single Purkinje fibres in 'false tendons' of the kid heart. Current was caused to flow through the surface membrane by means of a micro-capillary electrode inserted into the myoplasm. Potential changes along the fibre were recorded from the tip of a second intracellular electrode.

2. The d.c. resistance of the surface membrane was 2000 Ωcm^2, the membrane capacity 12 $\mu F/cm^2$, the specific resistance of myoplasm 105 Ωcm and the characteristic length of the fibre 1·9 mm.

3. The spread of the electrotonic potential in the vicinity of a cut surface indicates that an injured Purkinje fibre heals over with a 'membrane' representing an effective barrier to current flow.

4. The relatively low value of the specific d.c. resistance of myoplasm (twice that of Tyrode solution) suggests (i) that the smaller units making up the Purkinje fibre, the Purkinje cells, are not surrounded by ionic barriers of any importance, (ii) that Purkinje fibres are not subdivided by transverse membranes (cf. Rothschuh, 1951) and (iii) that most of the intracellular ions must be free to move under the influence of an electric field.

I am highly indebted to Prof. A. L. Hodgkin and to Mr A. F. Huxley for suggesting to me the principle of the method and for providing the solution to the theoretical problem. For advice on histological questions I wish to thank Dr E. N. Willmer and Dr W. Jacobson. Kid hearts were given to me by Dr R. S. Comline and collaborators. The investigation was aided by an apparatus grant from the Rockefeller Foundation and it was carried out during the tenure of a scholarship awarded by the Stiftung für Biologisch-Medizinische Stipendien, Basel.

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