Impedance Changes during the Electrical and Mechanical Activities of a Bullfrog Heart

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Abstract Electrical impedance of a bullfrog ventricle was recorded epicardially by the V-I method and the bridge method together with an electrocardiogram (ECG) and a contraction curve, in terms of shortening. Impedance change during one cardiac cycle of a contracting ventricle corresponded completely to the contraction curve in its time course: impedance decreased during systole and attained minimum at the peak of contraction, and the durations of both curves were almost identical. The ratio of impedance just before T-wave to that just after R-wave ($|Z_T|/|Z_R|$) of an in situ heart was increased when the ventricle contracted isovolumetrically by means of mineral oil: the ratio was far less than 1.0 in control ventricle, while it was more than 1.0 in oil-filled one (2/3 cases). Upon isometric condition or treatment of cadmium the motion restricted ventricle caused an increase in the impedance ratio ($|Z_T|/|Z_R|$) to higher than 1.0, as in the case of oil-filled ventricle. The epicardial recording of ventricular impedance seems to consist of two components: contraction (shortening)-dependent impedance change and contraction (shortening)-independent impedance change, which reflects the membrane activity. Vigorous contraction will probably mask the latter and reveal only the former.

The electrical impedance change during a cardiac cycle of a whole heart has been recorded by many investigators with the bridge method. The results they observed, however, were quite contradictory: ROSENBLUETH and DEL POZO (1943), CRANEFIELD et al. (1951), RUSHMER et al. (1953), and MURATA (1956) observed an increase in the impedance during systole; conversely, RAPPORT and RAY (1927) and HAYASE (1949) recorded a decrease in the impedance during the same period.

We used a new V-I method by which impedance measurement could be made easily (see METHODS). Our results solved the above contradiction observed in the experiments on the impedance change in in situ ventricle by considering
ventricular motion. Furthermore, we discussed the influence of the ventricular shortening on the electrical impedance mathematically. Thus, it became clear that the impedance of the in situ heart had two components: contraction-dependent one, whose change corresponded to the shortening of the ventricle, and contraction-independent one. This will provide not only the basic knowledge to the impedance plethysmography of the chest, which aims to measure cardiac output or contraction (KUBICEK et al., 1970; LABABIDI et al., 1970), but also the nature of cardiac muscle membrane (DE MELLO, 1972).

Preliminary accounts of this work are given elsewhere (HAYASHI et al., 1978).

MATERIALS AND METHODS

Both the in situ and isolated ventricles of 41 bullfrogs (Rana catesbeiana) of either sex were used between May and July 1978. The experimental animals, which were captured in the Saitama district, were supplied by Nihon Dobutsu. The frogs were kept at room temperature (23-28°C) in a bath for a week or two without feeding. The water level was adjusted to just above the abdomen of the animals. They were anesthetized with intrathecal injection of 0.5 ml urethane solution (0.25 g/ml). The heart was exposed by opening the chest wall at the midline.

Arrangement of electrodes. The heart of a bullfrog was suspended vertically after the pericardium was removed. Figure 1A illustrates the schematic diagram of arrangement of electrodes on the in situ ventricle. Current or stimulating electrodes (S1 and S2), which were made of a Ag-AgCl plate (5 mm x 5 mm), tapered cotton, and Ringer’s solution, were attached to both the apex and the atrioventricular groove. The area of contact between the electrode and the ventricle was about 0.01 cm². The recording electrodes (R1 and R2), which were arranged vertically (7-8 mm apart) between current electrodes, were made

Fig. 1. Arrangement of electrodes on the surface of an in situ ventricle (A) and in an excised ventricle (B) of a bullfrog. V: ventricle. A: atrium. S: sinus. R1 and R2: recording electrodes for a V-I method (A) or a bridge method (A and B). S1 and S2: current electrodes for the V-I method (A) or stimulating electrodes for driving the ventricle (B).
of either the same type of current electrode (used in earlier experiments, Fig. 1A) or stainless steel wires of 0.2 mmϕ (later experiments); the former type of electrodes were attached on the ventricular surface, and the latter type of those were set in the ventricle (0.5 mm in depth). Both types of recording electrodes gave the similar results. The composition of the Ringer's solution used is as follows (mM): NaCl, 110; KCl, 2; CaCl₂, 1; glucose, 10; Tris/HCl buffer, 10 (pH 7.2) (HAYASHI and HORIUCHI, 1971).

Two kinds of arrangement of electrodes were made on excised ventricles. The first kind is shown in Fig. 1B. The excised ventricle was cut at atrioventricular groove, and was fixed to a wax plate with its apex upward by means of 8 stainless steel pins; two recording electrodes (Ag-AgCl wire, 0.5 mmϕ), which were 8 mm apart, were almost vertically pierced through the wall of the ventricle; two stimulating electrodes, which were 3 mm apart, were arranged in a similar way.

The second kind (not shown in a figure) was to record isometric or isotonic contraction of a ventricular strip with impedance; ventricular strip (10 mm in length, 1–2 mm in width) together with endocardium and epicardium was suspended from a tension transducer (strain gauge, Nihon Kohden SB-1T) in the air. After 1 or 5 g weight was loaded to the preparation, a displacement transducer (strain gauge, Nihon Kohden SB-1TH) was attached to the lower end of the ventricular strip. The types of recording and current electrodes were the same as those used in in situ ventricle (i.e., stainless wire, and Ag-AgCl, cotton and Ringer's solution, respectively). The current electrodes were attached to the ends of the ventricular strip, and the recording electrodes were placed 5 mm apart between the current electrodes. In case of isometric contraction, the lower end of the strip was clamped. The error on recordings of both isometric and isotonic contraction were very small: 4 g in tension development caused 0.0057 mm in length change (0.1%) for isometric contraction, and 2 mm in length change brought about 0.07 g in tension (1.4%) for isotonic contraction.

Impedance measurement by V-I method. A newly devised technique was used to determine the impedance of a ventricle by a voltage-current (V-I) method. This technique enabled us to measure impedance not only on a quiescent preparation but an electrically active one. Figure 2A illustrates the block diagram of the V-I method for impedance measurement. The constant current of a sinusoidal wave (OSC: Kikusui 417A), whose intensity was monitored by voltage difference across R₁, was supplied from current electrodes (S₁ and S₂), and the voltage difference between two recording electrodes (R₁ and R₂) was recorded (Preamp: Nihon Kohden AVB-8). The impedance (|Z|) was calculated from the ratio of voltage amplitude to current amplitude. When the heart was excited spontaneously or by electrical stimulation (Nihon Kohden MSE-40), an electrocardiogram (ECG) made the impedance measurement (V-I method).
difficult because the ECG showed much larger amplitude than did the sinusoidal voltage signal. This difficulty was eliminated in the following way. Sinusoidal voltage signal (100 or 1,000 Hz) was first superimposed on ECG, and was stored in channel A of a digital wave memory (NF SID-2961). Then, only the ECG was stored in channel B of the wave memory. The sinusoidal voltage signal was obtained by subtracting the signal of Ch. B from that of Ch. A, and was displayed on a cathode-ray oscilloscope (Tektronix 564B-3A3-2B67) and on a pen writer chart (YEW 3078).

The features of this new V-I method are as follows: (1) Impedance changes for one cardiac cycle are obtained at one time. This requires much shorter time than the bridge method. (2) Phase shift, which is inevitably
accompanied by a V-I method using a filter, never occurred in the new V-I method.

**Impedance measurement by bridge method.** The widely used bridge method for impedance measurement was compared with the V-I method. Figure 2B shows the block diagram of the parallel resistance bridge for impedance measurement. Two arms of the bridge were fixed resistances \( R_b: 207 \, \text{k}\Omega \), and the other arm consisted of a decade resistance \( R_x: \text{YEW 2786} \) and a decade capacitance \( C_x: \text{YHP 4440B} \) connected in parallel. The rest of 4 arms was a preparation with electrodes attached. The output of the bridge was fed to a preamplifier (Nihon Kohden AVB-8), and was separated into ECG and a sinusoidal signal (less than 4% of the amplitude of ECG) by a high-pass filter (Deitel FLT-U2). The bridge balance around T-wave or R-wave was detected by a cathode-ray oscilloscope at 1,000 Hz. When the preparation was not excited spontaneously, it was driven by an isolated square pulse of 10 V, 3 msec at 1.6 sec intervals.

**Recording of contraction and ECG by motion picture.** The movement of a ventricle was photographed together with ECG on the screen of a cathode-ray oscilloscope by an 8 mm cinecamera (Beaulieu 4008 ZM2) at a speed of 70 frames/sec. This method was used to avoid the influence of unnecessary loads to the preparation. Immediately before taking the motion picture, simultaneous recording of ECG and impedance by V-I method was carried out, enabling us to compare the time course of impedance and that of contraction curve by using ECG as a time reference.

**RESULTS**

**Impedance of in situ normal ventricle**

Figure 3 represents a typical example of an electrocardiogram (ECG, upper trace) and an impedance curve recorded by the V-I method at 100 Hz (lower trace). Because the current through the preparation between recording electrodes was constant, the amplitude of impedance curve was directly proportional to the impedance. The surface of a ventricle was so dried up that the amplitude of epicardial ECG attained up to 50 mV. Because the amplitude of R-wave was much larger than that of the sinusoidal wave, the R-wave could not completely be cancelled by the subtraction procedure, which in turn made it difficult to see impedance changes during the R-wave in most experiments.

The amplitude of the impedance curve during a cardiac cycle took its maximum value immediately after the R-wave, and then gradually decreased to its minimum value just before the T-wave. The ratio of the impedance just before the T-wave \(|Z_T|\) to that immediately after the R-wave \(|Z_R|\) was \(0.733 \pm 0.037\), mean \(\pm 1\) S.E.M. Then it increased to the maximum immediately before the R-wave again.

The tendency of this impedance change was confirmed by the bridge method.
Fig. 3. Electrocardiogram (upper trace) and impedance measured by the V-I method (lower trace) of an in situ frog ventricle. The interruption of the lower trace around QRS-wave is due to the large QRS-wave which remains uncanceled. Note a remarkable impedance decrease during systole.

Fig. 4. Measurement of contraction of an in situ frog ventricle by using a cinecamera (70 frames/sec). Upper trace: bipolar electrocardiogram. Middle trace ($ΔL$): distance between recording electrodes. Lower trace ($ΔZ$): impedance (V-I method). Middle and lower traces are standardized with those just after the R-wave.

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(1,000 Hz) on in situ ventricle. When the bridge was balanced just after the R-wave, $R_x$ was 10.50 kΩ and $C_x$ was 0.00064 μF in one case. When the bridge was balanced just before the T-wave, $R_x$ decreased to 7.50 kΩ (71% of that of just after the R-wave) and $C_x$ was unchanged (0.00064 μF). The same tendency was observed in two other preparations.

We used the cinecamera to analyze the relationship between contraction and impedance curve. Figure 4 illustrates the simultaneous recording of ECG (upper trace), change in distance of recording electrodes ($\Delta L$, middle trace), and change in impedance of the ventricle measured by the $V-I$ method ($\Delta |Z|$), lower trace) during a cardiac cycle in an in situ normal ventricle. Even though the measured points of ECG were discrete, the persistence of spots on the screen of a cathode-ray oscilloscope enabled us to trace the whole course of ECG. The data of $\Delta L$ and $\Delta |Z|$ were expressed as the percentage of those around R-wave, and the curves were fitted by eye. The time course of the contraction ($\Delta L$) coincides with that of the impedance curve ($\Delta |Z|$) during a cardiac cycle with respect to the duration and the cardiac phase of the maximal decrease. The same results were obtained in 3 other preparations. We will discuss later the following two phenomena: (1) the duration of contraction is much longer than that of ECG (QT-time), (2) percentage of maximal decrease in impedance ($\Delta |Z|$) is slightly larger than that in contraction ($\Delta L$).

![Figure 5](image)

**Fig. 5.** Relationship between impedance and electrode distance of an in situ frog ventricle. Impedance was measured by the $V-I$ method and expressed as the ratio of that around T-wave to that just after the R-wave. Electrode distance was measured from a cinefilm and expressed as the ratio in the similar manner to the impedance. Closed circle: control heart. Open circle: heart filled with mineral oil. Linear regression line (solid line) for control hearts is drawn by using the least square method. Hypothetical regression line ($y=x$) is represented by a broken line.

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In Fig. 5, the horizontal and vertical axes illustrate the impedance and the distance between recording electrodes of an in situ normal ventricle (closed circle), respectively, and the relationship between them is summarized. The data of both impedance and distance are standardized (namely, the data just before the T-wave is divided by those immediately after the R-wave of the same cycle). The regression line obtained by the least square method is $y = 0.408x + 0.558$ (correlation coefficient, 0.86; standard error of correlation coefficient, 0.10; significant correlation). As this regression line does not coincide with a hypothetical line ($y = x$), it is estimated that even though there is a relationship between impedance and distance, at least one more factor is also involved in this relation (see DISCUSSION).

**Impedance curve of motionless ventricle**

If impedance change during a cardiac cycle in moving ventricle is mainly caused by its shortening, the pattern of impedance change should be changed by forced restriction of shortening. This type of experiment was performed first by replacing intraventricular Ringer's solution for mineral oil under the Stannius' second ligature condition. The ventricle was electrically stimulated, and motion of ventricle was almost ceased (isovolumetric contraction) though ECG still existed.

![Impedance curve of motionless ventricle](image)

Fig. 6. Electrocardiogram (upper trace) and impedance measured by the V-I method (lower trace) of an in situ frog ventricle filled with mineral oil. The heart was ligated at both aorta and atrioventricular groove. The ventricle was driven at 1.6 sec interval. The same preparation as that appeared in Fig. 3. Note no appreciable change in impedance during a cardiac cycle.

* The slope of the actual regression line is different from that of the hypothetical one ($|Z_i| = 9.39 > |Z_{i-1}| = 3.71$, $p < 0.01$). The point on the actual regression line ($\bar{x}, \bar{y}$) is different from that on the hypothetical line ($\bar{x}, \bar{y}$) ($Z_i = 9.39$, $p < 0.01$) (KAWADA and MARUYAMA, 1963).

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Figure 6 shows an example of the impedance curve of such a mineral oil treated ventricle. The preparation was the same as that appeared in Fig. 3. The time course of impedance during a cardiac cycle was quite different from that of control: it became minimum immediately after R-wave, and maximum just before T-wave (\(|Z_T|/|Z_R|\) was 105% in this case). In all 6 cases of mineral oil injection, \(|Z_T|/|Z_R|\) was larger than that of control; \(|Z_T|/|Z_R|\) was 0.79±0.04 in control ventricle, but it was 1.01±0.03 in oil injected ventricle (significant, \(p<0.01\)).

Open circles in Fig. 5 represent the relationship between impedance and distance which is measured between recording electrodes in an oil-injected ventricle. The regression line is \(y=-0.025x+1.026\) (crossing the point (1.0,1.0) and is almost in parallel to abscissa. No significant correlation). It is concluded that impedance change of in situ ventricle may be caused by at least two factors, i.e., contraction (shortening)-dependent and independent ones.

Cadmium of about 10 μM has been known to weaken contraction of frog cardiac muscle remarkably without appreciable change in its action potential (Hayashi and Horiuchi, 1971). Figure 7 shows the effect of cadmium on impedance of in situ ventricle. The impedance change during a cardiac cycle in control heart (Fig. 7A) was essentially the same as that in Fig. 3. The amplitude of impedance curve took its maximum immediately after the R-wave, and its minimum just before the T-wave (\(|Z_T|/|Z_R|\), 0.59). By slow injection of CdCl₂ (1 mM)-containing Ringer’s solution of 4 ml into a posterior caval vein, the heart became almost motionless in spite of existence of ECG. The final concentration of cadmium was estimated approximately 0.2 mM under the assumption of an even distribution of Cd into blood, and blood volume was about 1/13 of body weight in the animal (unpublished observation).

![Fig. 7. Electrocardiogram (upper trace) and impedance measured by the V-I method (lower trace) of a normal in situ frog ventricle (A) and a Cd-treated one (B). The contraction of the ventricle was almost ceased by the application of 0.2 mM Cd for 1 min. Note the disappearance of the impedance change in B.](image-url)
Fig. 8. Electrocardiogram (upper trace) and impedance measured by the bridge method (lower trace) of a normal excised frog ventricle. A: the bridge balanced around the T-wave. B: the bridge balanced around QRS-wave. Small pips in the lower trace at R-wave are due to the large R-wave which pass through the highpass filter.

Table 1. $R_x$, $C_x$, $|Z_t|$, and $|Z_T|/|Z_R|$ in excised ventricles.

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* $|Z_T|$, $|Z_R| = R_x/\sqrt{1+(\omega C_x R_x)^2}$, $\omega = 2\pi f$, $f = 1$ kHz in this experiment.

The injection of cadmium caused the disappearance of impedance change of ventricle during a cardiac cycle: the $|Z_T|/|Z_R|$ became 0.99 in this case. The other two cases showed the similar results.

Figure 8 represents the electrocardiogram (upper trace) and impedance (lower trace) of an excised fixed ventricle. Ventricular movement was restricted by fixing with many pins. The ventricle was driven at about 1.6 sec intervals and impedance of ventricle was measured with an arrangement demonstrated in Fig. 2B. The bridge was balanced around the R-wave ($R_x$: 10.5 k$\Omega$, $C_x$: 0.00345 $\mu$F, Fig. 8A) or just before the T-wave ($R_x$: 11.3 k$\Omega$, $C_x$: 0.00325 $\mu$F, Fig. 8B). $|Z_T|/|Z_R|$ was 1.08 in this case. The other cases are summarized in Table 1. These results also showed that $|Z_T|/|Z_R|$ was more than 1 in a motion-restricted ventricle.

Figure 9 shows impedance change of an excised ventricular strip which contracts isotonically (9A) or isometrically (9B). Impedance curve (upper trace, by $V$-$I$ method), ECG (middle trace), and contraction (lower trace) were simultaneously recorded. In the isotonic contraction, the envelope of impedance change coincided with contraction curve. The impedance ratio of the minimum
Fig. 9. Simultaneous recording of impedance (upper trace), bipolar electrocardiogram (middle trace), and contraction (lower trace) of an excised ventricular strip. The preparation was stimulated at the start of the sweep of CRO (0.4 Hz). A: isotonic contraction. B: isometric contraction. Note the disappearance of the impedance change in B.

to the maximum was 0.77 in this case. In contrast, in the isometric contraction, which was forced to be motionless, no appreciable change in impedance was observed during one cardiac cycle. The impedance ratio under isometric contraction measured by the same manner to the isotonic contraction was 0.97.

DISCUSSION

The impedance of a nervous cell or tissue decreased remarkably during its action potential (COLE and CURTIS, 1938; TASAKI and HAGIWARA, 1957). This was due to the transient increase in permeability of sodium ion and delayed increase of potassium ion (HODGKIN and HUXLEY, 1952). On the other hand, WEIDMANN (1951) observed an increase in impedance during the plateau phase of a sheep heart by the intracellular electrode technique. We expected from the analogy of the experiment of Weidmann that the extracellularly recorded impedance of the in situ heart might also increase during QT-interval. However, our results on in situ moving heart was quite opposite to this expectation: the impedance just before T-wave was the minimum during the cardiac cycle.

The results of impedance measurement of previous investigators were contradictory; the results of ROSENBLUETH and DEL POZO (1943) agreed with ours on the impedance increase during systole on a motionless ventricle. CRANEFIELD et al. (1951), RUSHMER et al. (1953), and Murata (1956) observed increase in impedance during systole of moving hearts. Cranefield et al. obtained their results by using a suction electrode in order to reduce the influence of extracellular pathway. The results of Rushmer et al. seems to be chiefly based on the volume decrease in intraventricular cavity. The reason for the discrepancy between Murata’s results and ours may be that while our electrodes were always
attached to the same spots of a ventricle, his electrodes were arranged in the solutions separated by the ventricular wall so that the distance of electrodes was constant regardless of the ventricular contraction. RAPPORT and RAY (1927) observed a decrease in impedance during isometric contraction. This did not agree with ours; probably their isometric condition was not complete so that relative position of electrodes to the heart was changed. HAYASE (1949) obtained qualitatively similar results to ours on moving ventricle, but his results on the peak of impedance were variable.

The main points of the technical improvement to measure impedance in our experiments were as follows. (1) By using the wave memory, it became possible to separate small sinusoidal voltage from the complex wave-form of ECG and sinusoidal voltage. (2) The V-I method using 4 electrodes shortened the time for impedance measurement during the cardiac cycle, and nullified the polarization and resistance of electrodes observed in case of 2-electrodes-V-I method. (3) We applied the electrodes to the same spots of the ventricular wall in spite of its motion. The impedance measurement performed as stated above revealed that, in a normal contracting ventricle, time course of impedance and contraction curves were almost identical: impedance decreased during systole and attained to the minimum at the peak of shortening (cf., Fig. 4, Fig. 9A). $|Z_T|/|Z_R|$ attained to about 0.7. On the other hand, in the ventricle whose motion was restricted by the treatment of filling mineral oil (cf., Fig. 6), cadmium (cf., Fig. 7), or stretch (cf., Fig. 8, Fig. 9B), $|Z_T|/|Z_R|$ increased to approximately 1 or more.

The capacitive component of impedance was so small that the impedance was nearly equal to the resistance in this experiment (see Table 1). The extracellularly recorded resistance ($R$) consisted of extracellular component ($R_o$) and membrane component ($R_i$) in parallel. $R = R_oR_i/(R_o+R_i)$. $R$ decreased during shortening of the ventricle. $R_i$ became maximum during electrical activity (WEIDMANN, 1956), while $R_o$ decreased as the heart shortened. The influence of decrease in $R_o$ on the combined resistance ($R$) was much larger than that of increase in $R_i$, especially $R_o < R_i$. This is the reason why increase in $R_i$ was masked by decrease in $R_o$.

The hypothesis ventured in the RESULTS (cf., Fig. 5) was that the relative impedance ($Δ|Z|$), which reflected the superficial tissue, including wetting of the ventricular surface, was directly proportional to the relative distance between electrodes ($ΔL$). This hypothesis, however, could not possibly be approved because the hypothetical line and the experimental regression line was statistically different. For this problem, we propose in this paper a model of a ventricular strip under resting (a) and isotonically contracting conditions (b) (Fig. 10A). We postulate the following 4 assumptions: (1) the shape of the strip is cylindrical regardless of shortening; (2) the total volume of the strip is not altered by shortening; (3) shortening is uniform in any part of the strip, and the ratio of

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cross-sectional area of intracellular space to extracellular one is constant; and (4) resistivity is constant whatever part of the strip we take as an example and whether resting or shortening occurs. The results of ventricular strip will probably be extended to the whole ventricle because the whole ventricle is considered to consist of many rows of ventricular strips in parallel. Furthermore, as we will see later, the point which shows the relation between impedance and distance of ventricular strip (cf., Fig. 9) is on the curve of experimental equation of the whole heart (cf., Fig. 10). Let the impedance of the resting strip and that of contracting strip be \( |Z| \) and \( |Z'| \), respectively. Other symbols are represented in Fig. 10A. \( |Z| = \rho L/(\pi r^2) \), \( |Z'| = \rho L'/(\pi r'^2) \), where \( \rho \) is resistivity. According to the assumption, \( r^2L = r'^2L' \). Then \( d|Z| = |Z'|/|Z| = dL' \). Therefore, the relationship between \( x \), relative impedance \( (T/R) \) and \( y \), relative distance \( (T/R) \) is \( y^2 = x \). This theoretical line is shown as a broken line in Fig. 10B. The solid line of Fig. 10B is fitted for the data of normal and oil-filled conditions in Fig. 5. Relative impedance of normal contracting ventricle is assumed to be a function of contraction (relative distance). Since the state of oil-filled motionless ventricle is an extreme case of normal contracting one, both data are included in a same experimental equation. The experimental equation \( y = a \cdot x^b \) is obtained by determining \( "a" \) and \( "b" \). By the least square method, \( a = 0.984 \) and \( b = 0.428 \). These values agree well with the coefficients of theoretical equation, \( a = 1 \) and \( b = 0.5 \) \( (p<0.01) \). From this test, it becomes clear that the impedance change during a cardiac cycle is mainly due to the movement (shortening) of the whole ventricle.

The regression curve of experimental data crosses the abscissa at the right of 1.0, but hypothetical curve does so at the point of just 1.0 (Fig. 10B).

![Fig. 10. Curve fitting of the data of Fig. 5. A: model of ventricular strip under resting (a) and isotonically contracting condition (b). Assumption: no change in volume and in resistivity of the preparation in spite of shortening. B: relationship between impedance and electrode distance of a ventricle. Experimental curve is drawn by using a parabola (solid line). Broken line is drawn from a calculated one on the model \( y^2 = x \), see text for further explanation).](image-url)
The difference may be ascribed to the increase in membrane impedance at terminal phase of plateau.

The ratio of the minimum membrane resistance (around rising phase) to the maximum one (around terminal phase of plateau) during a cardiac cycle in a sheep Purkinje fiber was approximately 300 (WEIDMANN, 1956). However, $|Z_T|/|Z_R|$ in our experiment was 1.1 at most (Fig. 5, Fig. 8, and Table 1). This discrepancy could be ascribed to the following reasons. First, as bipolar ECG represents the electrical activities of many cells which did not excite at the same time because of conduction, the impedance decrease around R-wave was suppressed. Thus, we measured the impedance 90 msec after the onset of R-wave (just after R-wave). Secondly, as our impedance measurement was made extracellularly most current flowed through $R_e$, and a small amount of current flowed through $R_i$. The ratio of extracellular potential $\xi V_o$ to membrane potential $\xi V_m$ is as follows: $\xi V_o/\xi V_m = -R_o/(R_o + R_{IR})$, where $R_{IR}$ is $R_i$ just after R-wave (KATZ, 1966). The membrane action potential of toad ventricle was about 100 mV (HAYASHI and AZUMA, 1962), and QRS-wave in Fig. 8 was approximately 16 mV. Then, $R_o/R_{IR} = 1/5$. $|Z_R| = R_{IR} \cdot R_o/(R_{IR} + R_o)$, $|Z_R|$: the impedance measured around R-wave extracellularly. $|Z_T| = R_T \cdot R_o/(R_T + R_o)$, $|Z_T|$: the impedance measured around T-wave extracellularly. Thus, $|Z| \equiv |Z_T|/|Z_R| = (R_{IR}/R_o) \cdot (1 + R_o/R_{IR})/(R_{IR} + R_o/R_{IR})$. The impedance ratio of the terminal phase of plateau (350 msec after the onset of action potential) to the initial phase of plateau (90 msec after the onset of action potential) was 3.35 (WEIDMANN, 1956). Then $|Z| = 1.13$. The experimental value (1.1) in this experiment is very close to the calculated one. Thirdly, the impedance change of frog ventricle might be somewhat smaller than that of a sheep Purkinje fiber. Intracellular recording of impedance in a frog ventricle will be reported elsewhere.

The ionic conductance during the action potential of Purkinje fiber of a sheep was already reported (MCALLISTER et al., 1975). The similar analysis will be reported elsewhere in relation to impedance.

It is generally considered that the duration of an action potential of mammalian cardiac muscle is almost equal to that of the contraction. Impedance curve and contraction curve of a bullfrog ventricle, however, lasted longer than ECG by 400–900 msec in this experiment: the impedance curve was still near its peak when the ECG terminated. This phenomenon was confirmed by an experiment using the suction electrode (HAYASHI and ARITA, 1979). The difference of duration of contraction curve and that of action potential in mammalian heart became larger as temperature decreased, and finally at room temperature of about 25°C the duration of the former became twice as long as that of the latter (TRAUTWEIN and DUDER, 1954; MARSHALL, 1957). Therefore, it might be merely due to temperature effects which made the duration of contraction much longer than that of ECG in a bullfrog ventricle. The impedance of ventricle at various temperature remains to be investigated.
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