ELECTRONICS AND ELECTRICAL ENGINEERING
ISSN 1392 – 1215 2009. No. 6(94)
ELEKTRONIKA IR ELEKTROTECHNIKA

MEDICINE TECHNOLOGY
T115
MEDICINOS TECHNOLOGIJA

Theoretical Aspects of Measuring Input Resistance in Syncytial Tissue by Means of Double-Barreled Electrode

A. Grigaliūnas, R. Veteikis
Institute for Biomedical Research, Kaunas University of Medicine
Eivenių st. 4, LT-50161 Kaunas, Lithuania, phone: +370 37 302966, e-mail: romualdas.veteikis@kmu.lt
K. Lukoševičius, K. Muckus
Kaunas Academy of Physical Education
Sporto st. 6, LT-44221 Kaunas, Lithuania, phone: +370 37 302648, e-mail: lukaskaz@yahoo.com

Introduction

Investigation of the intercellular electric communication is of utmost importance for the explanation of the mechanisms of excitation wave spread in the cardiac tissue. Measurement of the input resistance presents one of possibilities to evaluate the intercellular electric coupling. In the syncytial structure, the input resistance can be measured by using double-microelectrode technique [1-3], when one barrel of the microelectrode is used for delivering a rectangular jump of current, and the other one is used for recording the intracellular potential. However, when introduced to cell, such microelectrodes, because of their large dimensions, may damage the membrane to a greater extent than single-barrel microelectrodes used exclusively for recording the intracellular potential or for delivering the current [4,5]. Also, the possibility that the current leakage via the damaged membrane may exert a considerable effect during the measurement of the input resistance by means of a double-barreled microelectrode cannot be rejected.

In the present work, the results of input resistance measurement in the rabbit's right auricle endocardium by means of a double-barreled microelectrode, under normal conditions and under heptanol-induced blockade, are presented. Mathematical model for determination of input resistance was constructed and experimental data were analysed on theoretical basis.

Methods

Measurements were performed on rabbit's right atrium preparations in vitro. Preparation involving crista terminalis and trabecular area were perfused by Tyrode solution of the following composition (in mM): NaCl – 136; KCl – 2.7; $CaCl_2$ – 1.8; $MgSO_4$ – 0.5; NaH_2PO_4 – 4.6; $NaHCO_3$ – up to pH=7.35; glucose – 2 g/l; temperature – 37 ± 0.5^0C. The input resistance measurements were performed in the trabecular area of isolated preparations from the rabbit right atrium. The preparation was stimulated at the rate of 3 Hz, with the bipolar electrode placed on crista terminalis. A double microelectrode was used for recording the input resistance, one barrel serving for delivering the current jump I_o, and the other one for recording the jump of the intracellular potential (ΔV_i) and extracellular potential (ΔV_e). The input resistance (R_{in}) was calculated according to Ohm's law:

$$R_{in} = \frac{\Delta V_i - \Delta V_e}{I_o}. \quad (1)$$

The measurement was first performed under normal conditions, then 3.5 mM heptanol, an efficient inhibitor of electric coupling [6], was added to the normal Tyrode. The electric coupling used to deteriorate (while the input resistance increased) within 10 – 15 min, a complete blockade being reached, as indicated by the absence of action potentials at the recording site, with the ensuring cessation of the input resistance increase.

Simulating measurement of the input resistance

When the input resistance is measured by double-barreled microelectrode, current I_o flows through resistance of the myoplasm r_{pl}, resistance of intercellular contacts r_k and resistance of the electrogenic membrane r_m (electric diagram in Fig.1a). In reality the cell is injured when microelectrode is inserted and leakage current flows through the leakage resistance r_d at the damaged site (Fig.1b).

According to theoretical models [7], the input resistance (R_{in}) in three dimensional isotropic syncytial tissue, which consists of spherical tightly packed cells with intercellular contacts of high conductivity, depends on resistances of intercellular contacts and myoplasm:

$$R_{in} = \frac{(\rho_{pl} - \rho_T)}{4\pi r_o} + \frac{\pi R_k}{32 r_c^2}, \quad (2)$$

where ρ_{pl} – resistivity of myoplasm, ρ_T – resistivity of Tyrode solution, r_o – distance between tips of double-barreled microelectrode, R_k – resistivity of intercellular contacts, r_c – radius of cell.

Considering (1) in future we will assume that

$$r_{pl} = (\rho_{pl} - \rho_T)/4\pi r_o, \quad (3)$$

where $\rho_T/4\pi r_o$ – the input resistance of Tyrode solution.

According to [8], the resistance r_m has no influence on the input resistance of the tissue. Therefore the resistance of the electrogenic membrane r_m may be removed out of the equivalent diagram (Fig. 1c).

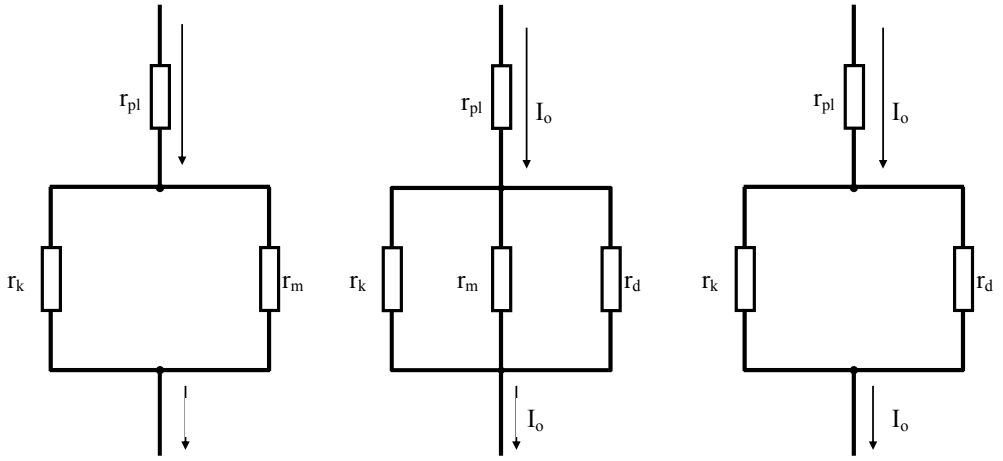


Fig. 1. Equivalent diagram of input resistance for one cell: r_{pl}, resistance of the myoplasm; r_k, resistance of intercellular contacts; r_m, resistance of the electrogenic membrane; r_d, leakage resistance during the damage

In the ideal case if the cells membrane is not injured by microelectrode impalement, there is no current flow through the site of impalement ($r_d \to \infty$). If the r_m is neglected, then the input resistance of the tissue R_{in} is

$$R_{in} = r_{pl} + r_k. \quad (4)$$

However in reality the insertion of the microelectrode into the cell causes the leakage current through the site of impalement [8–11]. Therefore the measured input resistance R_{im} in that case is

$$R_{im} = r_{pl} + r_k \cdot r_d / (r_k + r_d) \quad (5)$$

If the intercellular contacts are blocked (by the heptanol) the resistance of the intercellular contacts $r_k \to \infty$, and experimentally obtained input resistance (R_H) is described by (6)

$$R_H = r_{pl} + r'_d, \quad (6)$$

where r'_d – the leakage resistance of the damaged tissue by microelectrode impalement.

Eq. (2–6) form the system of equations where some quantities or their range are known (ρ_{pl}, ρ_T, r_o, R_{im}, R_H), and some are unknown (R_{in}, r_{pl}, r_k, r_d, r'_d).

Supposing the leakage resistance of the damaged tissue does not changes after impalement of the microelectrode under control conditions and under influence of heptanol, i.e. $r'_d = r_d$, the solution of equations (2–6) leads to:

$$R_{in} = \frac{(\rho_{pl} - \rho_T)^2 - 8\pi r_o R_{im}(\rho_{pl} - \rho_T) + 16\pi^2 r_o^2 R_{im} R_H}{16\pi^2 r_o^2 (R_H - R_{im})} \quad (7)$$

The results of mathematical simulation will be used analyzing measurements of input resistance in the rabbit's right atrium under control and influence of heptanol.

Results of experimentally measured input resistance

In all 15 experiments were carried out. In different experiments, the measured input resistance values, R_{im}, ranged, under normal conditions from 0.47 MOhms to 0.95 MOhms. Under heptanol-induced blockade of the intercellular electric coupling, the input resistance values, R_H, ranged from 1.17 to 2.59 MOhms, i.e. they were 1.5 to 4.5 times higher than those under normal conditions (Table 1).

Table 1. Experimentally obtained values of the input resistance (MOhm) in the rabbit's right atrium. R_{im} – input resistance under normal conditions (averages of all measurements in individual experiments), R_H – input resistance measured in the presence of heptanol (averages of all measurements in individual experiments); Av – average of all experiments

Expt No	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Av.
R_{im}	0.85	0.71	0.47	0.51	0.65	0.74	0.55	0.73	0.59	0.68	0.44	0.8	0.95	0.85	0.83	0.69
R_H	1.29	1.17	1.69	2.24	1.63	2.41	1.51	2.07	1.71	2.12	1.33	1.51	2.5	2.59	1.85	1.84

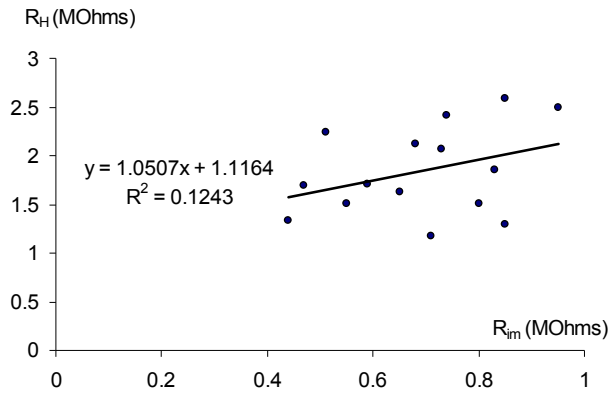


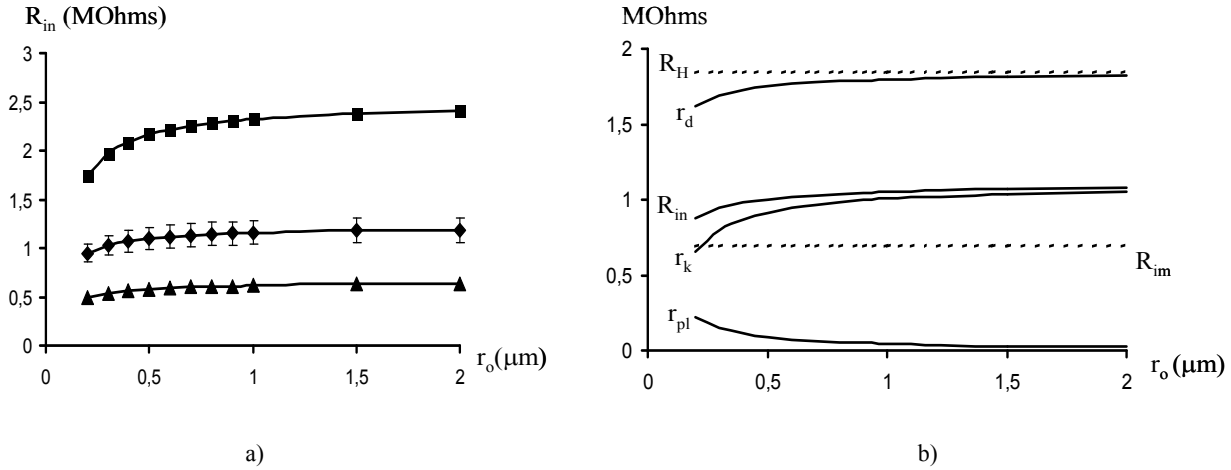
Fig 2. Relation between the input resistance measured under influence of heptanol (R_H) and the input resistance under normal conditions (R_{im}). Equation of the linear regression and the coefficient of determination are presented in the picture

The analysis of R_H dependency on R_{im} (Fig. 2) shows there is a weak relation between these quantities: the coefficient of determination R^2 is 0.1243.

Results and Discussion

It is known that heptanol blocks the intercellular electric communication in myocardium without exerting any effect on the resistance of the electrogenic membrane [6, 12]. Therefore, the input resistance of the tissue would be the same as the input resistance of isolated cell.

For instance, the input resistance obtained by the *whole cell patch clamp* method in the isolated ventricular cell of guinea-pig was 25.9 MOhms [13]. The input resistance measured by the single-barrel microelectrode in the same object was 12.4 MOhms [14]. Therefore the total block of the intercellular electric communication in the rabbit's atrium may lead to an increase of the input resistance by many times. In reality the input resistance of myocardial tissue under influence of heptanol on the average increases only by 2.67 times and reaches 1.84 MOhms. This inadequacy between theoretical hypothesis and our experimental data may be explained by the emerging of the leakage current through the resistance r_d at the injured site of the cell (Fig.1c). The degree of injury depends on the method the input resistance is measured: the membrane is injured more when the input resistance is measured by double-barreled microelectrode than by *whole cell patch clamp* method. On the base of our model and experimentally obtained values of the input resistance under control (R_{im}) and under heptanol (R_H) we can assess the values of the real input resistance (R_{in}), the leakage resistance of the damaged site (r_d), contribution of the intercellular contacts (r_k) and myoplasm (r_{pl}) into the input resistance of myocardial tissue when microelectrodes of different dimensions are used for measurements. Minimum, maximum and average values of the real input resistance are presented on the Fig. 3a.



a) b)

Fig. 3. Estimated values of input resistance (R_{in}) at different sizes of microelectrode's tip (r_o): a – min (▲), max (■) and averaged (●) R_{in} values of all experiments; b – averaged values R_{in}, r_{pl}, r_k, and r_d of all experiments; dashed lines – averaged values of input resistance at control and under heptanol influence

All calculations were made assuming that the resistivities of the intercellular medium of the myocardial tissue (r_{pl}) and Tyrode solution (ρ_T) are respectively 120 Ohms·cm [15] and 64 Ohms·cm [16], also R_{im} and R_H do not depend on the distance r_o between the tips of a double-barreled microelectrode. We obtained that the resistance of the myoplasm (r_{pl}) increases and the resistance of intercellular contacts (r_k) decreases considerably when the dimensions of the microelectrode r_o are smaller. In our experiments the input resistance measured in Tyrode solution was 50-100 KOhm. According to equation (3) the dimensions of such microelectrode's tip are 0.5 ÷ 1.0 μm and therefore have a small influence on the R_{in} (Fig. 3b).

Positive correlation exists between experimentally obtained input resistance R_{im} and R_{in} derived from equation (7): R_{im} increases with the increase of R_{in} (Fig. 4a). For instance, the coefficient of determination (R^2) is 0.654, when $r_o = 0.5$ μm.

However, the relation between the input resistance (R_{im}) measured in control and the leakage resistance r_d (Fig. 4b) is weak: the coefficient of determination (R^2) is 0.1243, when $r_o = 0.5$ μm.

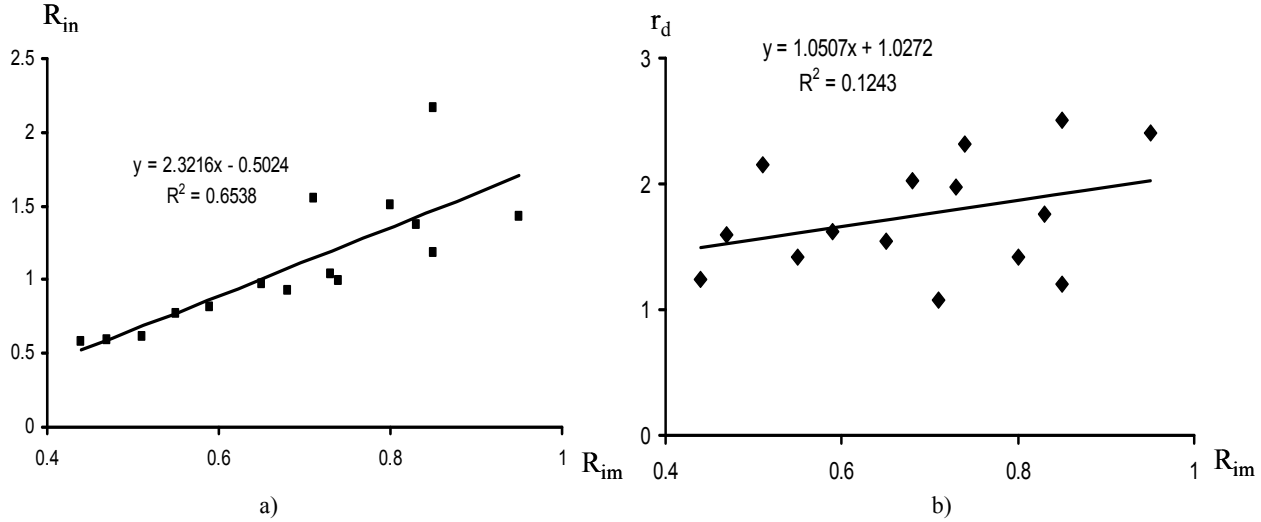


Fig. 4. R_{in} and r_d dependence on values of myocardial tissue input resistance R_{im} measured under control conditions

There are reports on the input resistance values ranging from tens of kOhms to ten megohms in the cardiac tissue [17-21]. The value range is very wide and depends on the measurement technique, the object of investigation, and the animal species. For example, when the input resistance was measured in the trabecular area of the rabbit's right atrium using the double-barreled microelectrode technique, the obtained values ranged from 180 to 500 kOhms [22], but when the single-barreled microelectrode was used the average value of R_{in} was 320 kOhms [23]. The investigation of intercellular communication in the mouse right atrium by means of a double-barreled microelectrode yielded the input resistance values from 300 kOhms to 600 kOhms [24]. The input resistance, when measured at the same site using two microelectrodes, was found to be 35 kOhms [25]. Yet it is known that, in the rabbit's right atrium, deterioration of the electric communication induced by increasing the intracellular concentration of Ca^{2+} ions markedly increases the input resistance, which is also true of the Purkinje fibers in the dog's heart [17]. An increase in the input resistance of the cardiac tissue may be also induced by ouabain [26]. An increase in the input resistance is also noted when the preparation of the rabbit's right atrium was placed in the hypertonic solution [27]. However, in none of the above cases the intracellular communication was completely blocked and therefore they cannot be used for the assessment of the influence of membrane damage on the measurement results. Furthermore, all measurements have been performed by using different techniques, and the causes of the deterioration of the intercellular electric communication are very different. However, no data on measurement of the input resistance under influence of heptanol was found. In the ideal case, the measured input resistance consists of the resistance of the closest cellular myoplasm, the resistance of contact membrane and the resistance of cellular membrane connected in parallel (Fig. 1a). If the cellular membrane is damaged, the resistance of the damaged site is parallel to the resistance of the contact membrane (Fig. 1b).

As has been shown in the two-dimensional and three-dimensional models of syncytial media, the input resistance is but slightly dependent on the resistance of the electrogenic membrane [28]. Besides, heptanol blocks the intercellular electric communication almost without exerting any effect on the resistance of the electrogenic membrane [6]. Therefore, it may be assumed that during the experiment the resistance of the electrogenic membrane remains the same both under normal conditions and under heptanol-induced intercellular decoupling. The input resistance of the tissue which consists of tightly packed cells slightly depends on the resistance of the membrane and is mainly defined by the resistance of intercellular contacts [22], and therefore it is possible that upon the total blockade of the intercellular electric communication, input resistance would increase a few dozens of times. For a cylindrical cell, 50 μm long and 15 μm in diameter, whose membrane resistivity is 3 to 4 kOhm·cm^2 and is equal to the resistivity in the trabecular cells of the rabbit's right atrium [23], the input resistance should increase by about 100 times. Our results show that, with heptanol-induced blockade of the intercellular electric communication, the input resistance increases only by 1.52 to 4.51 times. Since the heptanol of 3.5 mM concentration blocks intercellular contacts and have no marked influence on the resistance of cellular membrane this inadequacy between theoretical calculation and experimental data can be explained by the emerging of the leakage current through the damaged site where the microelectrode is inserted and therefore an increment of the input resistance is significantly less than is predicted by the model. So the leakage resistance r_d should be added to the equivalent diagram for the input resistance (Fig. 1b).

After making such an assumption, the damage resulting from the insertion of a double microelectrode may be assessed. It may be concluded that the leakage via the damaged area has a large effect on the results of input resistance measurement. Besides, as the resistance of the damaged site is connected in parallel to the membrane and the intercellular contacts resistances, the bigger the value of input resistance measured during the experiment, the larger the effect of the damaged site resistance on the accuracy of measurements.

Conclusions

The double-barreled electrode is difficult to insert into the cell due to a relative large diameter of the tip. New technologies should provide for decreasing the later, thereby facilitating the microelectrode insertion and decreasing the membrane damage. Such an improved microelectrode would make it possible to reduce the dependency of the input resistance values measured on inhomogeneities of the membrane. On the other hand, due to the increased value of the measured input resistance, the resistance of the damaged site would have a bigger effect on the accuracy of measurements. Therefore, the double-barreled microelectrode technique should only be used for a qualitative assessment of intercellular communication.

References

1. **Johnson E. A., Tille J.** Investigation of the electrical properties of cardiac muscle fibres with the aid of intracellular double-barrelled electrodes // J. Gen. Physiol. – 1961. – Vol. 44. – P. 443-67.
2. **Duivenvoorden J. J., Bouman L. N., Bukauskas F. F., et al**. Phase dependency of electrotonic spread of hyperpolarizing current pulses in the rabbit sinoatrial node // J. Mol. Cell. Cardiol. – 1990. – Vol. 22. – P. 415–27.
3. **Duivenvoorden J. J., Bouman L. N., Opthof T., et al**. Effect of transmural vagal stimulation on electrotonic current spread in the rabbit sinoatrial node // Cardiovasc. Res. – 1992. – Vol. 26. – P. 678–86.
4. **Davis R. E., Stretton A. O**. Passive membrane properties of motorneurons and their role in long–distance signaling in the nematode Ascaris // J Neurosci. – 1989. – Vol. 9(2). – P. 403–14.
5. **Rink T. J**. Measurement of free calcium concentration in living cells // Pure & Appi. Chem. – 1983. – Vol. 55. – No. 12. – P. 1977–1988.
6. **Jongsma H. J., Rook M. B.** Morphology and electrophysiology of cardiac gap junction channels // In: D.P. Zipes, J. Jalife, editors / Cardiac electrophysiology: from cell to bedside. WB Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo. – 1995. – P. 115–26.
7. **Veteikis R.** Theoretical and experimental investigations of passive electric properties of cardiac tissue. Research report presented for habilitation // Biomedical sciences, biophysics (02B). Kaunas University of Medicine, Kaunas. – 2001. – P. 108.
8. **Fedida D., Sethi S., Mulder B. J, ter Keurs H. E.** An ultracompliant glass microelectrode for intracellular recording // Am J Physiol. – 1990, Jan; 258 (1 Pt 1). – P.164–170.
9. **Macefield V. G.** Spontaneous and evoked ectopic discharges recorded from single human axons // Muscle Nerve. – 1998. – No. 21(4). – P. 461–468.
10. **Svirskis G, Gutman A, Hounsgaard J.** Detection of a membrane shunt by DC field polarization during intracellular and whole cell recording // J Neurophysiol. – 1997 – Vol. 77(2). – P. 579-86.
11. **Svirskis G, Hounsgaard J, Gutman A.** Detection of inhomogeneities in membrane ohmic resistance in geometrically complex systems // Membr Cell Biol. – 2000. – Vol. 14(3):413-20.
12. **Bastiaanse E. M., Jongsma H. J., van der Laarse A., Takens–Kwak B. R.** Heptanol–induced decrease in cardiac gap junctional conductance is mediated by a decrease in the fluidity of membranous cholesterol–rich domains // J Membr Biol. – 1993. – No. 136(2). – P.135–45.
13. **Wilders R., Kumar R., Joyner R. W., Jongsma H. J., Verheijck E. E., Golod D., van Ginneken A. C., Goolsby W. N.** Action potential conduction between a ventricular cell model and an isolated ventricular cell // Biophys J. –1996. – No. 70(1). – P. 281–95.
14. **Mèszàros J.** Effect of palmitoylcarnitine on the passive electrical properties of isolated guinea pig ventricular myocytes // Eur J Pharmacol.—1991. – No. 194(1). – P. 107–10.
15. **Schanne O. F.** Measurement of cytoplasmic resistivity by means of the glass microelectrodes // In: Lavallee M, Schanne OF, Herbert NC, eds. / Glass microelectrodes. New York, London, Sydney, Toronto: Wiley & Sons Publishers. – 1969. – P. 299–231.
16. **Sperelakis N., Macdonald R. L.** Ratio of transverse to longitudinal resistivities of isolated cardiac muscle fiber bundles // J electrocardiology. – 1974. – Vol. 7(4). – P. 301–314.
17. **Kiss T.** Electrical properties of the cardiac muscle cell membrane and its role in the excitation-contraction coupling // Acta Biochim. Biophys. Acad. Sci. Hung. – 1977. – Vol. 12. – P. 291-302.
18. **De Mello W. C.** Interaction of cyclic AMP and Ca^{2+} in the control of electrical coupling in heart fibers // Biochim. Biophys. Acta. – 1986. – Vol. 888. – P. 91–9.
19. **Pressler M. L.** Intracellular pH and cell–to–cell transmission in sheep Purkinje fibers // Biophys. J. – 1989. – Vol. 55. – P. 53–65.
20. **Pressler M.L., Elharrar V., Bailey J.C.** Effects of intracellular calcium ions, verapamil, and lanthanum on active and passive properties of canine cardiac Purkinje fibers. Circ. Res. – 1982. – Vol. 51. – P. 637-51.
21. **Tuganowski W., Korczynska I., Wąsik K., et al**. Effects of calmidazolium and dibutyril cyclic AMP on the longitudinal internal resistance in sinus node strips // Pflüg. Arch. – 1989, vol. 414. – P. 351–3.
22. **Bukauskas F., Gutman A., Kišūnas K. et al**. Electrical cell coupling in rabbit sinoatrial node and atrium: experimental and theoretical evaluation // In: L.N.Bouman, H.J.Jongsma, editors / Cardiac Rate and Rhythm. Physiological, morphological and developmental aspects. Hague, Boston, London: Martinus Nijhof. – 1982. – P. 195–216.
23. **De Mello W.C.** Passive electrical properties of the atrioventricular node // Pflüg. Arch. – 1977, vol. 371. – P. 135–9.
24. **Tanaka I., Sasaki Y.** On the electrotonic spread in cardiac muscle of the mouse // J. Gen. Physiol. – 1966, vol. 49. – P. 1089–1110.
25. **Kuriyama H. A., Goto M., Maeno T. et al**. Comparative studies on transmembrane potentials and electrical characteristics of cardiac muscles // In: Y. Katsuki, editor / Electrical activity of single cells. Tokyo: Igaku Shoin. – 1960. – P. 243–60.
26. **Ando S., Kodama I., Ikeda N., Toyama J., et al.** Effects of ouabain on electrical coupling of rabbit atrial muscle fibers // Jap. Circul. J. – 1981. – Vol. 45. – P. 1050–5.
27. **Bukauskas F., Veteikis R.** Vlijanije gipertoničeskogo I beskalcievogo rastvorov na mežkletočnuju električeskuju sviaz v uzlovoj tkani serdca // In: M.P. Roščevskij editor/ Sravnitelnaja elektrokardiologija, Leningrad: Nauka Publ. – 1981. – P. 52–4.
28. **Bukauskas F., Bytautas A., Veteikis R., et al**. Teoretičeskij analiz raspredelenija elektrotoničeskogo potenciala v nepreryvnych anizotropnych sincitialnych sredach // In: F.Bukauskas editor // Elektrofiziologija serdca. Vilnius: Mokslas Publ. – 1992. – P. 5–21.

Received 2009 02 13

A. Grigaliūnas, R. Veteikis, K. Lukoševičius, K. Muckus. Theoretical Aspects of Measuring Input Resistance in Syncytial Tissue by Means of Double-Barreled Electrode // Electronics and Electrical Engineering. – Kaunas: Technologija, 2009. – No. 6(94). – P. 93–98.

The spread of electric excitation in the cardiac tissue is possible via local currents due to intercellular electric coupling. The measurements of input resistance were made, by using a double-barreled microelectrode technique, in order to evaluate the intercellular electric coupling between the myocardial cells in the trabecular area of the right atrium. Reliability of this technique was assessed by using a heptanol-induced blockade of intercellular electric communication. The baseline values of input resistance varied from 0.47 to 0.95 Mohms. Under the heptanol-induced decoupling, the propagation of excitation ceased and the input resistance value ranged from 1.17 to 2.59 MOhms, i.e. was smaller than the resistance of electrogenic membrane of one isolated atrial cell. A mathematical model of tissue input resistance was developed for analysis of experimental data. The results obtained can be explained by eventual injury of electrogenic membrane due to the double-barreled microelectrode penetration with ensuring effect upon the measurement accuracy largely dependent upon the resistance of damaged site. Ill. 4, bibl. 23 (in English; abstracts in English, Russian and Lithuanian).

А. Григалюнас, Р. Ветейкис, К. Лукошявичюс, К . Муцкус. Теоретические аспекты измерения входного сопротивления в синцитиальной ткани при помощи двухканального микроэлектрода // Электроника и электротехника. – Каунас: Технология, 2009. – № 6(94). – С. 93–98.

Распространение возбуждения в миокардиальной ткани осуществляется при помощи локальных токов через межклеточные контакты. При помощи двухканальных микроэлектродов измеряли входное сопротивление в трабекулах правого предсердия сердца кролика с целью оценки межклеточной электрической связи. Достоверность этого метода была оценена с использованием блокады межклеточной электрической связи при помощи гептанола. Контрольные значения входного сопротивления были в интервале от 0,47 до 0,95 МОм. В условиях полного межклеточного электрического разобщения (при воздействии гептанолом) прекращалось распространение возбуждения, а величина входного сопротивления выросла и была в интервале от 1,17 до 2,59 МОм, т.е. была значительно меньше сопротивления электрогенной мембраны одной изолированной предсердной клетки. Разработана математическая модель входного сопротивления ткани, при помощи которой проанализированы экспериментальные данные. Полученные результаты могут быть объяснены повреждением электрогенной мембраны на месте вхождения двухканального микроэлектрода, а сопротивление повреждения может иметь большое влияние на точность измерения входного сопротивления. Ил. 4, библ. 23 (на английском языке; рефераты на английском, русском и литовском яз.).

A. Grigaliūnas, R. Veteikis, K. Lukoševičius, K. Muckus. Įėjimo varžos matavimo sincitiniame audinyje dvikanaliu elektrodu teoriniai aspektai // Elektronika ir elektrotechnika. – Kaunas: Technologija, 2009. – Nr. 6(94). – P. 93–98.

Elektrinė sujaudinimo banga širdies audinyje sklinda veikiant vietinėms srovėms per tarpląstelinius kontaktus. Viena iš pagrindinių metodikų, įvertinančių tarpląstelinio elektrinio ryšio dydį, yra įėjimo varžos matavimas. Šios metodikos patikimumas buvo įvertintas matuojant įėjimo varžą triušio dešiniajame prieširdyje. In vitro perfuzuojant širdies gabalėlį normaliu Tirodės tirpalu įėjimo varžos dydžiai buvo nuo 0,47 iki 0,95 megomo. Heptanolio poveikis užblokuodavo sujaudinimo bangos sklidimą, o įėjimo varža buvo 1,17 – 2,56 megomo intervale, t. y. gerokai mažesnė, negu vienos izoliuotos širdies ląstelės elektrogeninės membranos varža. Eksperimentų duomenims analizuoti pateikiamas įėjimo varžos matavimo matematinis modelis. Gauti rezultatai paaiškinami tuo, kad dvikanalis mikroelektrodas įdūrimo metu gali pažeisti ląstelės elektrogeninę membraną ir pažeidimo vietos varža gali turėti didelę įtaką matavimo tikslumui. Il. 4, bibl. 23 (anglų kalba; santraukos anglų, rusų ir lietuvių k.).