Proceedings of I. Vekua Institute
of Applied Mathematics
Vol. 75, 2025

STUDY OF ELECTRICAL CONDUCTION PROCESSES IN BLOOD VESSELS USING CABLE THEORY

Natela Zirakashvili, Teona Zirakashvili

Abstract. Traditionally, the electrical conduction processes in blood vessels are explained using cable theory where locally generated signals (membrane potential V_m) passively spread along the arteriolar wall. As a rule, decomposition is quantified, with a constant λ of length derived from cable theory. Using cable theory on blood vessels depends on assumptions that may not be necessarily performed for small arteries and arterioles. It is known, that arterioles are composed of at least two layers of cells: endothelial cells (EC) and one or more layers of smooth muscle cells (SMC), which are connected by myoendothelial gap junctions (MEGJ). In this study, arterioles composed of two cell layers are studied, focusing on changes in membrane potential within both EC and SMC layers. A suitable stationary problem is formulated and solved analytically using the method of separation of variables. Additionally, numerical modelling of membrane potential propagation is conducted using MATLAB software. Isopotential contours of the membrane, as well as two-dimensional and three-dimensional graphs depicting the numerical results, are presented in the study.

Keywords and phrases: Cable equation, membrane potential, arteriole, endothelial cell layers, smooth muscle cell layers, method of separation of variables.

AMS subject classification (2020): 35Q92, 35J25, 35J05, 92F05.

1 Introduction

The membrane potential V_m plays a critical role in vascular compressibility, regardless of mechanical or chemical stimuli. V_m is determined in each individual cell by the activity of ion channels and each cell in turn shares information with neighboring cells through gap junctions (channels). This direct exchange of information presumably enables multiple branches of the blood vessel to coordinate their actions. Such coordination would, in theory, allow arterial structures to effectively regulate blood flow [1-3].

It is believed that the passive electrotonic propagation of electrical signals is the primary mode of intercellular communication in blood vessels [4, 5]. This process is characterized by the propagation of current along the vessel wall and is traditionally explained using cable theory [6,7]. The accepted length-constant λ, has been widely used as a convenient measure of conduction length [7–10]. The physiological interpretation of the length constant derived from cable theory has its limitations. Therefore, cable theory should be viewed primarily as a simplified model for comprehending conduction processes rather than as an exact mechanistic model.

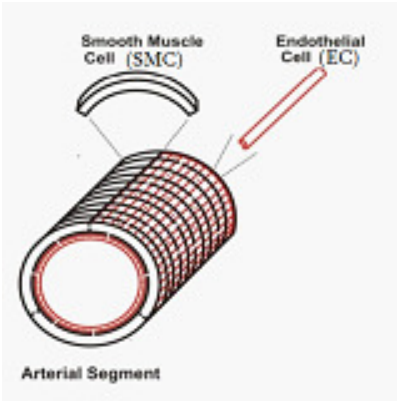

Figure 1: Schematic drawing of an arterial segment.

Length constant λ, derived from cable theory, is widely used as a measure of conduction because of its relatively simple implementation and theoretical simplicity.

It is noteworthy that arterioles consist of at least two cell layers: an endothelial cell (EC) layer and one or more smooth muscle cell (SMC) layers. These layers are connected by myoendothelial gap junctions (MEGJ). ECs are long and flat and oriented along the long axis of the arteriole. SMCs are spindle shaped cells and wrap around ECs in a perpendicular arrangement. ECs and SMCs are modeled by an overlapping scheme, as shown in figure 1 [11].

This paper studies two cell-layered (EC and SMC cell layers) arterioles and membrane potential changes in these layers. Numerical modelling is conducted using MATLAB software for a one-dimensional model of a two-cell-layered cylindrical arteriole in the absence of current at the beginning and end of the cable (arteriole). Numerical results are obtained and corresponding membrane isopotential contours, 2D and 3D graphs are presented.

The paper consists of five sections.

After the introduction, the second section presents the main theoretical issues.

In the third section, the problem is set and solved, focusing on the change in membrane potential within a finite-length cylindrical arteriole composed of two cell layers in the absence of current at the beginning and end of the cable.

The fourth section presents graphs of the numerical results obtained by solving the given problem with appropriate physical interpretation.

The fifth section provides the conclusion.

2 Theoretical aspects

Propagation of V_m changes in biological cable-like structures is usually described using cable theory. A primary challenge of cable theory lies in computing the membrane potential. Cable theory, which was developed in recent decades, is older than the cable equation itself, it is a variation of the equations developed by Lord Kelvin to model the propagation of electrical signals in underwater telegraphs. Cable theory was initially used on the conduction of potentials in the axon by Hodgkin and Rushton [6], it is applied to neurons, cell arrays [12] and blood vessels [13,14].

The passive cable equation is written as follows (see Appendix A):

$$\tau_m \frac{\partial V(x,t)}{\partial t} = \lambda^2 \frac{\partial^2 V(x,t)}{\partial x^2} - V(x,t), \tag{1}$$

where $V := V_m$ is membrane potential, $\lambda = \sqrt{\frac{r_m}{r_l}}$ is the length (space) constant of the passive cable equation, while $\tau = r_m c_m$ is time constant, r_m is membrane resistance, c_m is the membrane capacitance, and r_l cell cytoplasm resistance per unit length. Space and time constants are parameters, which are used to measure and characterize specific properties of excitable tissues.

When applying cable theory, the following basic assumptions are usually made for small blood vessels [15]:

1. Intracellular and extracellular spaces are one-dimensional and homogeneous.
2. Electrotonic conduction is ohmic.
3. The membrane resistance is independent of the resting membrane potential.
4. Ion channel conductance does not depend on time (or voltage).
5. Intracellular and extracellular ion concentrations are constant.

3 Formulation and solution of the problem

Let's consider a cylindrical two celled-layered arteriole of length L. Let's solve the passive cable equation with the following boundary and initial conditions: 1) there is no current flowing input (at the beginning of the cable) and output (at the end of the cable) in the cable (in arteriole) and 2) at the beginning of time, the current is a function only of the spatial coordinate x of the cable. The boundary condition represents a problem similar to the heat conduction problem of an insulated end pole. Thus, the boundary and initial conditions are written as follows:

$$\begin{aligned} &\text{For} \quad x = 0: \quad V_{,x}(x,t) = 0, \\ &\text{For} \quad x = L: \quad V_{,x}(x,t) = 0, \end{aligned} \tag{2}$$

$$\text{For} \quad t = 0: \quad V(x,0) = f(x). \tag{3}$$

Using the method of separation of variables and taking into account boundary (2) and initial (3) conditions, the following image is obtained [16]:

$$V(x,t) = \frac{2}{L}\frac{L^2}{2} + \frac{2}{L}\sum_{n=1}^{\infty}\left[\frac{L^2}{\pi n}\sin(\pi n) + \left(\frac{L}{\pi n}\right)^2(\cos\pi n - 1)\right] \times \cos\left(\frac{\pi n x}{L}\right) e^{\left(1-(\lambda\pi n/L)^2\right)t/\tau_m}$$
$$= L + 2L\sum_{n=1}^{\infty}\left(\frac{1}{\pi n}\sin(\pi n) + \frac{1}{(\pi n)^2}(\cos\pi n - 1)\right) \times \cos\left(\frac{\pi n x}{L}\right) e^{\left(1-(\lambda\pi n/L)^2\right)t/\tau_m}.$$

$$V_{,x}(x,t) = -2\sum_{n=1}^{\infty}\left[\pi n\left(\frac{1}{\pi n}\sin(\pi n) + \frac{1}{(\pi n)^2}(\cos\pi n - 1)\right) \times \sin\left(\frac{\pi n x}{L}\right) e^{\left(1-(\lambda\pi n/L)^2\right)t/\tau_m}\right]. \tag{4}$$

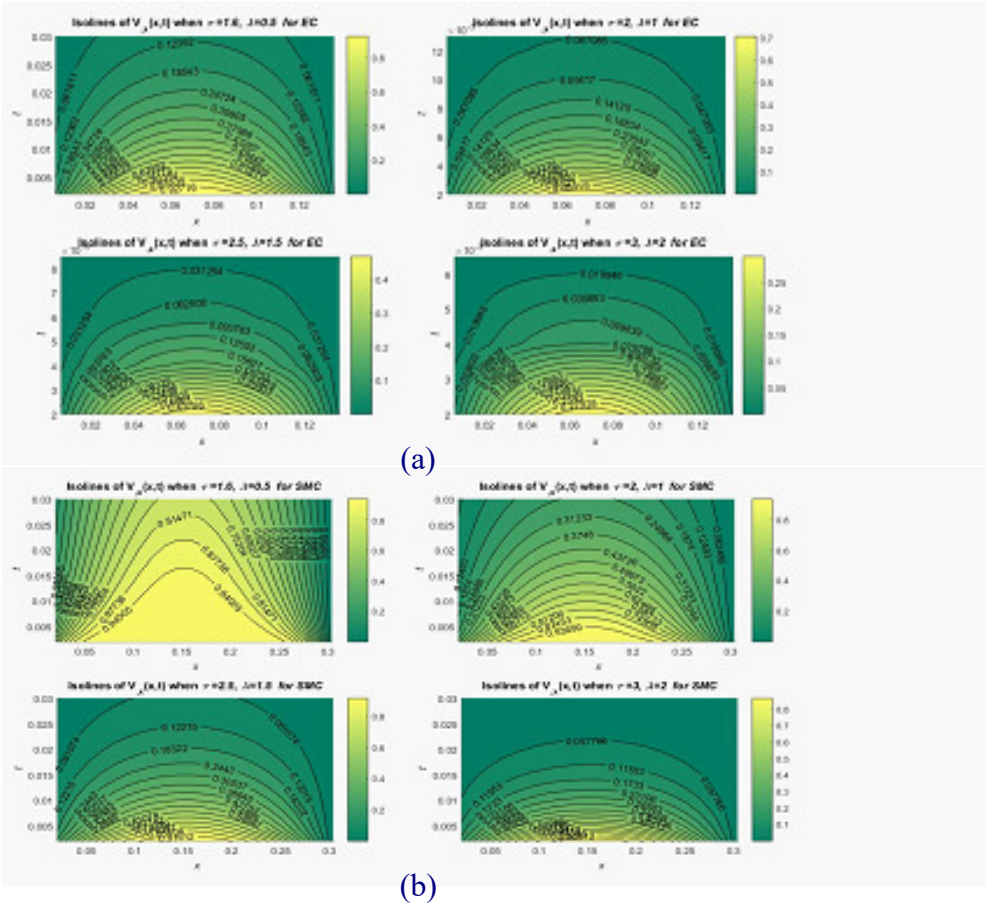

Figure 2: Membrane isopotential contours for different fixed values of time and spatial constants (a) for the endothelial cell (EC) layer, (b) for the smooth muscle cell (SMC) layer.

4 Numerical realization

For a two-cell-layered (EC and SMC cell layers) one-dimensional model of a cylindrical arteriole, in the absence of current at the beginning and end of the cable (arteriole), numerical modeling is carried out using MATLAB software for the following data: for four pairs of time and length constants 1) $\tau = 1.6ms, \quad \lambda = 0.5mm$; 2) $\tau = 2ms, \quad \lambda = 1mm$; 3) $\tau = 2.5ms, \quad \lambda = 1.5mm$; 4) $\tau = 3ms, \quad \lambda = 2mm$. The pitch of spatial (length) discretization is $\Delta x = 6\mu m = 0.006mm$, and of time discretization is $\Delta t = 0.005ms$. The four pairs of numerical values of $V_{,x}$ of the membrane potential change are received for time τ and spatial λ constants and while x (from $0.006mm$ to $0.136mm$, for EC and to $0.304mm$, for SMC) and t (from $0.01ms$ to $0.03ms$) changes.

In practice, a system of contour lines (isolines) is often used to analyze the results of measurements. For an arteriole, some contours of the membrane potential change $V_{,x}$ for a flat area in the vector field of length x and time t are presented. Some contours for a flat area in a vector field of length x and time t. (We can find such a system of points in which the numerical values of changes in membrane potentials are equal - by connecting them, lines of equal membrane potentials are obtained, the so-called membrane isopotential contours). Figure 2 presents membrane isopotential contours, for four fixed values of time τ and spatial λ constants, fig. 2a for the endothelial cell (EC) layer, and fig. 2b for the smooth muscle cell (SMC) layer.

As can be seen from Figure 2, the membrane isopotential contours in all four cases visually look the same, in particular, they are all approximately in the shape of an elliptic circle, only their numerical values slightly differ from each other.

Figure 3 presents for an arteriole, three-dimensional graphs of the propagation of the membrane potential change $V_{,x}$ for four values of τ and λ in a space-time vector field. Figure 3a presents for the endothelial cell (EC) layer, while figure 2b, for the smooth muscle cell (SMC) layer. While increasing t in both layers $V_{,x}$ tends to zero, herewith $V_{,x}$ becomes equal to zero (i.e. $V_{,x}$ disappears sooner) in the (EC) layer for a smaller value of t, than in the (SMC) layer. As well as when τ and λ increase, $V_{,x}$ disappears sooner (i.e. for smaller values of t,$V_{,x} \to 0$). $V_{,x} = 0$ at the beginning and end of the cable, and $V_{,x}$ takes the maximum value approximately in the middle of the cable, which can be seen from formula (4) and figure 3.

Figure 4 presents the graphs of $V_{,x}$ along x for four pairs of time τ and spatial λ constants and for four fixed values of t. Figure 4a presents the graphs of $V_{,x}$in the endothelial cell (EC) layer, in particular, in the upper left figure $t = 0.01ms$, in the upper right figure $t = 0.02ms$, in the lower left figure $t = 0.05ms$, in the lower right figure $t = 0.09ms$. $V_{,x}$-graphs are presented in figure 4b in the smooth muscle cell (SMC) layer, namely, in the upper left figure $t = 0.01ms$, in the upper right figure $t = 0.02ms$, in the

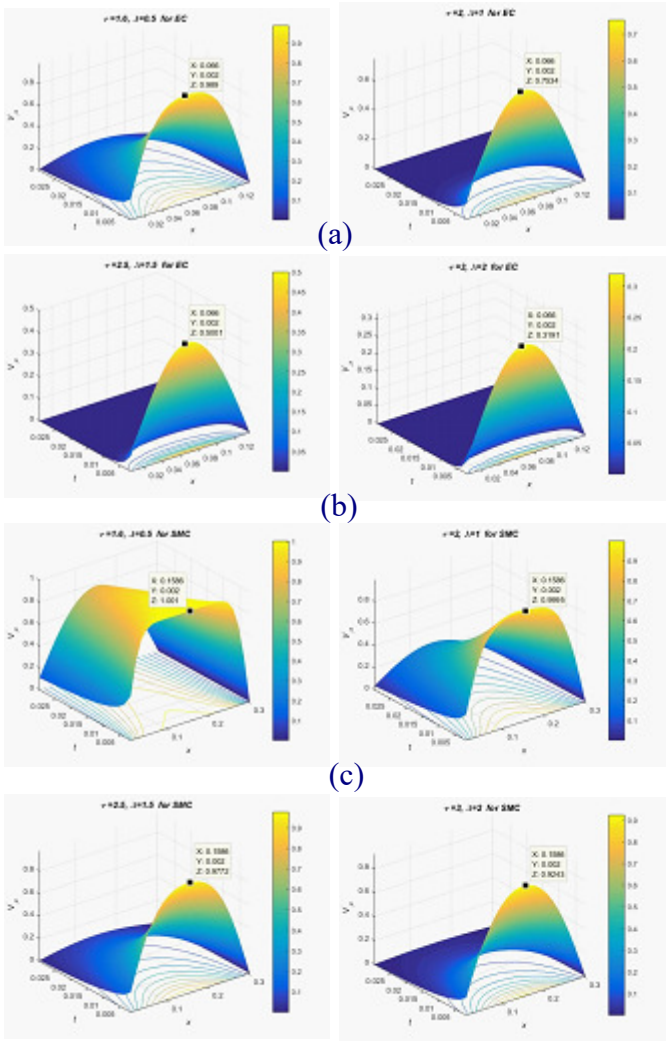

Figure 3: 3D graphs of the distribution of $V_{,x}$ of the membrane potential change in the x and t vector field for different fixed values of the time and space constants (a), (b) for the endothelial cell (EC) layer, (c), (d) for the smooth muscle cell (SMC) layer.

lower left figure $t = 0.05ms$, in the lower right figure $t = 0.09ms$. Figure 4 shows that in both layers (EC and SMC layers)1) when t increases $V_{,x}$, decreases 2) $V_{,x}$ takes maximum value in the middle of the cable. Moreover in the (EC) layer for one and the same value of t, $V_{,x}$ is lower than in (SMC) layer.

The 5th figure presents the graphs of $V_{,x}$ while t change, for four pair values of time τ and spatial λ constants and for four fixed values of x. Figure 5a presents $V_{,x}$ graphs in the endothelial cell (EC) layer, namely, in the upper left figure $x = 0.006mm$, in the upper right figure $x = 0.012mm$, in the lower left figure $x = 0.03mm$, in the lower right figure $x = 0.054mm$.

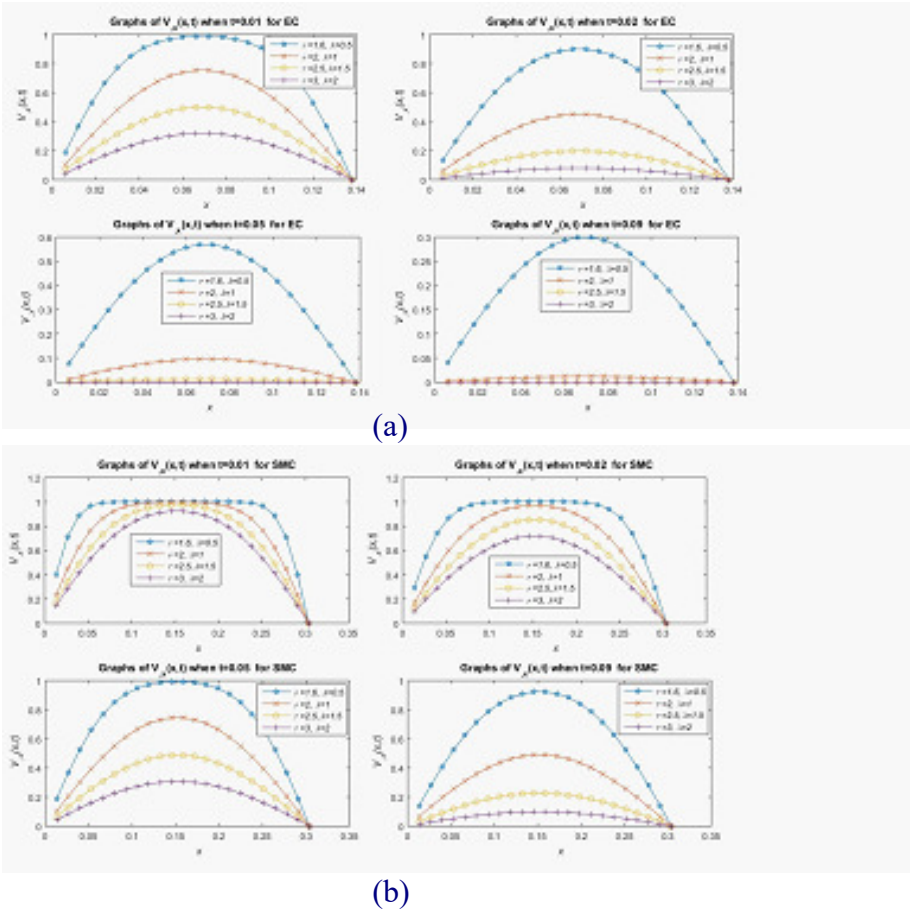

Figure 4: $V_{,x}$graphs of membrane potential change along x, for different fixed values of time τ and spatialλ constants and for four fixed values of t, (a) for the endothelial cell (EC) layer, (b) for the smooth muscle cell (SMC) layer.

The 5b figure presents $V_{,x}$ graphs in the smooth muscle cell (SMC) layer, namely, in the upper left figure $x = 0.006mm$, in the upper right figure $x = 0.012mm$, in the lower left figure $x = 0.03mm$, in the lower right figure $x = 0.054mm$. When t increases in both EC and SMC layer $V_{,x}$ tends to zero, moreover in layer EC , $V_{,x}$ becomes equal to zero for the smaller value of t (i.e. $V_{,x}$ will disappear sooner) than in SMC layer.

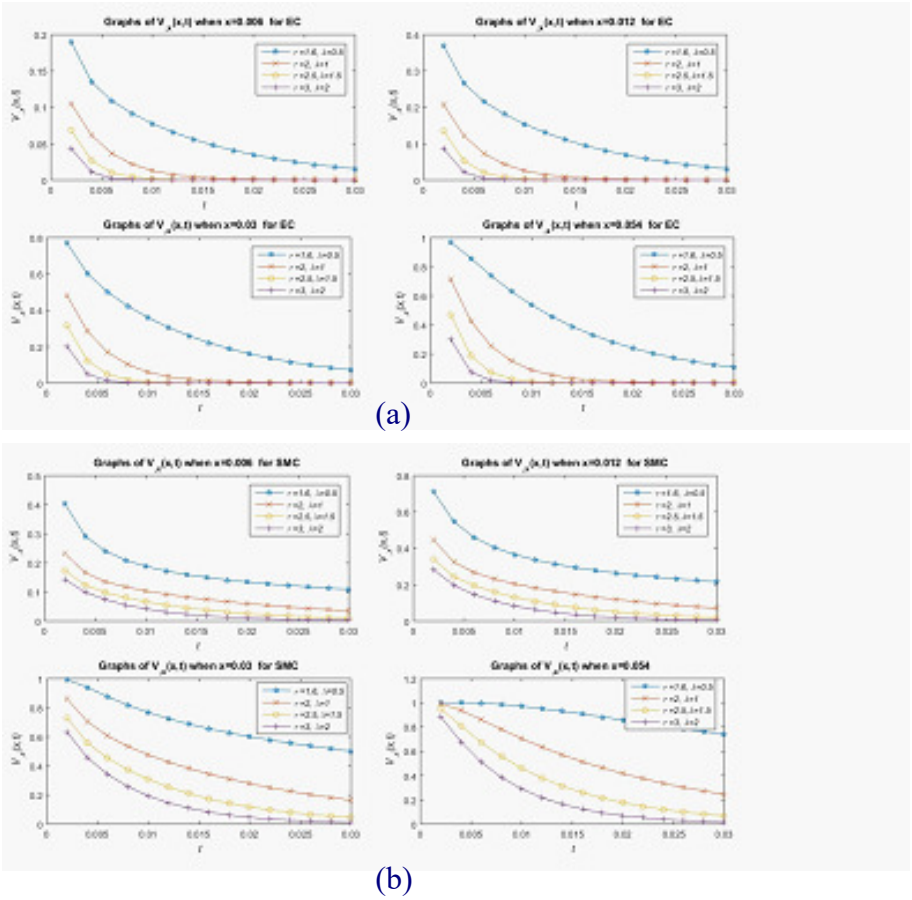

Figure 5: $V_{,x}$graphs of membrane potential change along t for different fixed values of time τ and spatialλ constants and for four fixed values of x, (a) for the endothelial cell (EC) layer, (b) for the smooth muscle cell (SMC) layer.

5 Conclusion

The main results presented in this paper can be formulated as follows:

1. The change in membrane potential in the endothelial cell layer and the smooth muscle cell layer is studied using the passive one-dimensional cable equation (endothelial cell (EC) and smooth muscle cell (SMC) layers), for a two-cell-layered arteriole.

2. For a one-dimensional model of a two-cell-layered cylindrical arteriole, in the absence of current at the beginning and end of the cable, numerical modeling is carried out using the software MATLAB. Numerical results of the change in membrane potential are obtained, based on which membrane isopotential contours are constructed for a flat area

in the vector field of length x and time t. Some 2D and 3D graphs are also built.

3. From the obtained numerical results and corresponding graphs, it can be seen that the change in membrane potential $V_{,x}$ is smaller in the EC layer, than in the SMC layer, which is caused by poor current propagation in the SMC layer.

Cardiovascular diseases continue to be the foremost cause of mortality globally. It should be noted, that despite of extensive research over many years, the propagation of membrane potential in blood vessels has not been comprehensively explored, therefore, its study remains an actual topic of much modern scientific research. This paper aims to study changes in membrane potential in blood vessels using the cable equation. Finally, we are continuing our research and will study and solve some of the problems in this field to the fullest extent possible.

6 Appendices

Appendix A. Solution of passive one-dimensional cable equation
Let's consider a sufficiently long thin cylindrical excitable cell. We consider the cell as a long cylindrical body (cable) of membrane that surrounds the inner part of the cytoplasm. We assume that everywhere along its length, the membrane potential depends only on the length variable and not on the radial or angular variables, so that the cable can be treated as one-dimensional. The change of membrane potential V in small Δx interval is given in the form of $\Delta V := V(x+\Delta x,t) - V(x,t) = -i_l r_l \Delta x$, where r_l is the resistance of the cell cytoplasm per unit length, i_l is a current along the membrane. Let's say $\Delta x \to 0$,

$$\frac{\partial V}{\partial x} = -i_l r_l \qquad (A1)$$

A change in current along the membrane in the small Δx interval is given by the formula $\Delta i_l = -i_m \Delta x$, which i_m is membrane current per unit length. Also, let's say$\Delta x \to 0$,

$$\frac{\partial i_l}{\partial x} = -i_m \qquad (A2)$$

Membrane current, in each x point, represents leakage (lost) current i_r sum, due to volumetric i_c current and membrane resistance. Capacity (volumetric) i_c currenton membrane area with a given c_m capacity is shown with the following formula:

$$i_c = c_m \frac{\partial V}{\partial t},$$

and the current due to the membrane resistance is given by Ohm's law:

$$i_r = \frac{V}{r_m},$$

wherer_m is the resistance of the membrane. By adding these two members, we receive

$$i_m = c_m \frac{\partial V}{\partial t} + \frac{V}{r_m}.$$

By inserting these two members into (A2) and considering (A1), we receive

$$\frac{\partial i_l}{\partial x} = \frac{\partial}{\partial x}\left(-\frac{1}{r_l}\frac{\partial V}{\partial x}\right) = -\frac{1}{r_l}\frac{\partial^2 V}{\partial x^2} = -\left(c_m \frac{\partial V}{\partial t} + \frac{V}{r_m}\right).$$

From all the abovementioned, the passive cable equation is written as follows

$$\sigma_i \frac{\partial^2 V}{\partial x^2} = c_m \frac{\partial V}{\partial t} + \frac{V}{r_m}, \qquad (A3)$$

which $\sigma_i = \frac{1}{r_l}$ represents intracellular conductance.

By algebraically transforming the passive cable equation, namely by multiplying equation (A3) by r_m, the number of parameters is reduced to two main parameters. Thus, the passive cable equation is written in the following form

$$\tau_m \frac{\partial V}{\partial t} = \lambda^2 \frac{\partial^2 V}{\partial x^2} - V,$$

where, λ is the space (length) constant of the passive cable equation and is given by the following formula:

$$\lambda = \sqrt{r_m \sigma_i} = \sqrt{\frac{r_m}{r_l}},$$

which defines the distance along the cell over which the injected potential is reduced by a coefficient e. τ is a time constant and is given by the following formula

$$\tau = r_m c_m,$$

It determines the length of time during which the injected potential is reduced by the coefficient e. Space and time constants are useful parameters used to measure and characterize specific excitable tissue properties.

Acknowledgments. This work was presented at the Seminar of the I. Vekua Institute of Applied Mathematics of Iv. Javakhishvili Tbilisi State University. I would like to thank my colleagues for their valuable discussions.

References

[1] D.T. Kurjiaka, S.S. Segal. Conducted vasodilation elevates flow in arteriole networks of hamster striated muscle. *Am. J. Physiol.*, 269:H1723-H1728, 1995

[2] S.S. Segal, T.L. Jacobs. Role for endothelial cell conduction in ascending vasodilatation and exercise hyperaemia in hamster skeletal muscle. *J. Physiol*, 536:937-946, 2001

[3] D.A. Williams, S.S. Segal. Feed artery role in blood flow control to rat hindlimb skeletal muscles. *J. Physiol*, 463:631-646, 1993

[4] K.A. Dora, S.L. Sandow, C.J. Garland. Myoendothelial gap junctions may provide the pathway for EDHF in mouse mesenteric arteries. *J. Vasc. Res.*, 40:480–490, 2003

[5] V.J. Schmidt, S.E. Wölfle, Cor de Wit: Gap junctions synchronize vascular tone within the microcirculation. *Pharmacol. Rep.*, 60:68–74, 2008

[6] A.L. Hodgkin, Rushton, WAH: The electrical constants of a crustacean nerve fiber. *Proc. R. Soc. Med.*, 134:444–479, 1946

[7] G.D. Hirst, T.O. Neild. An analysis of excitatory junctional potentials recorded from arterioles. *J. Physiol.*, 280:87–104, 1978

[8] G.J. Crane, T.O. Neild. An equation describing spread of membrane potential changes in a short segment of blood vessel. *Phys. Med. Biol.*, 44: N217–N221, 1999

[9] F. Gustafsson, N. Holstein-Rathlou. Conducted vasomotor responses in arterioles: characteristics, mechanisms and physiological significance. *Acta Physiol. Scand.*, 167:11–21, 1999

[10] G.G. Emerson, T.O. Neild, S.S. Segal. Conduction of hyperpolarization along hamster feed arteries: augmentation by acetylcholine. *Am. J. Physiol. Heart Circ. Physiol*, 283:H102–H109, 2002

[11] H.K. Diep, E.J. Vigmond, S.S. Segal, S.S. Welsh. Defining electrical communication in skeletal muscle resistance arteries: a computational approach. J. Physiol., 568(Pt 1):267-281, 2005, doi: 10.1113/jphysiol.2005.090233

[12] C.P. Fall, E.S. Marland, J.J. Tyson. editors. Computational Cell Biology. *pringer-Verlag, New York*, 2002

[13] A. Kapela, S. Nagaraja, N.M. Tsoukias. A mathematical model of vasoreactivity in rat mesenteric arterioles. II. Conducted vasoreactivity. *Am. J. Physiol. Heart Circ. Physiol.*, 298:H52–H65, 2010

[14] G.J. Crane, M.L. Hines, T.O. Neild. Simulating the spread of membrane potential changes in arteriolar networks. *Microcirculation*, 8(1):33-43, 2001

[15] G.R. Holt. A Critical Reexamination of Some Assumptions and Implications of Cable Theory in Neurobiology. PhD thesis. California Institute of Technology, Pasadena, California. doi:10.7907/HPPC-S237, 1998 https://resolver.caltech.edu/CaltechETD:etd-09122006-135415.

[16] N. Zirakashvili, T. Zirakashvili. Study of action potential propagation in cardiac tissues using cable theory. WSEAS TRANSACTIONS on BIOLOGY and BIOMEDICINE., 20:28-39, 2023, DOI: 10.37394/23208.2023.20.24.

Author's address:

Natela ZirakaSvili
I.Vekua Institute of Applied Mathematics of I. Javakhishvili Tbilisi State University
11, University St. 0186, Tbilisi, Georgia
E-mail: natela.zirakashvili@tsu.ge

Teona ZirakaSvili
Ilia State University, Kakutsa Cholokashvili Ave 3/5, 0162, Tbilisi, Georgia;
Tbilisi Heart and Vascular Clinic, LTD, 64 Lubliana St., 0159, Tbilisi, Georgia
E-mail: teona.zirakashvili.1@iliauni.edu.ge