

Dynamic Extension of Starling Resistor Model

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Introduction

Bionics technology is widely used for the investigation and management of biological systems. The main objectives for such systems were formulated in [1]. Medical electronics evolves toward intelligent interactive systems [2], it is necessary to ensure the effectiveness of such systems [3]. This requires adequate models of physiological processes. These models of the cardiovascular system are based on lumped parameter description using the electrical analogue of cardiovascular systems [4]. In computational models voltage corresponds to pressure, current to flow, capacitance to elastic properties and inductance to inertial properties. The resistance simulates the resistance of flow encountered by the blood as it flows from the major arteries to minor arteries and capillaries due to decreasing vessel diameter. Particular segments of vessels are described by Windkessel models. These models are linear – values of resistance, capacitance and inductance are constant.

Tissue pressure increases in the case of a stroke or trauma. In areas with increased tissue pressure the linear models are unsuitable.

Static Starling resistor model

The Starling resistor describes vascular beds with increased compartment pressure [4, 5]. The blood flow Q in the vessel is determined by the relationships between inflow pressure P_I , external pressure P_E and outflow pressure P_V . There are three West zones [6] where these relationships are defined. In zone 1 ($P_E > P_I > P_V$) blood flow Q equals zero. In zone 2 ($P_I > P_E > P_V$) Q is proportional to $(P_I - P_E)$. In zone 3 ($P_I > P_V > P_E$) Q is proportional to $(P_I - P_V)$.

In the case of compartment injury, there are tissue pressure gradients from the centre to the periphery of focal injury. As a result, different West zones exist in the same

vascular network. This will cause blood flow diversion via the pathway of least resistance. This blood flow redistribution in veins is called “cerebral venous steal” [7]. Starling’s model is a model for the investigation of the effects of tissue pressure differences on blood flow. It is considered that increased venous pressure may increase blood flow to regions with increased tissue pressure and prevent secondary injury from the venous steal.

The concept of blood flow redistribution with increased venous pressure was verified by the static Starling resistor model. A constant blood pressure is used in this model; this may cause errors in estimations.

A segment of vascular system with increased tissue pressure is depicted (Fig 1).

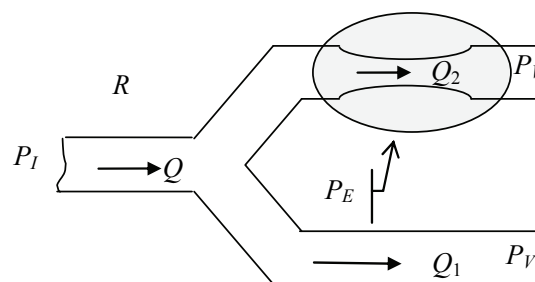


Fig 1. A segment of vascular system

The blood flow in the vessel Q_1 is dependent on inflow pressure P_I , external pressure P_E and outflow pressure P_V . The blood flow in the vessel Q_2 is dependent on inflow pressure P_I , and outflow pressure P_V . (There is no tissue pressure on the vessel Q_2). R – resistance to flow.

The lumped parameter circuit may be used to model a segment of the cardiovascular system [8, 9, 10]. The electrical analogue of this segment is depicted in Fig. 2.

I_1 corresponds to blood flow in vessel Q_1 , I_2 corresponds to blood flow in vessel Q_2 , and U corresponds to input pressure P_I .

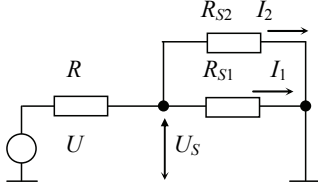


Fig. 2. The electrical analogue of vascular system segment

In this model resistors R_{S1} and R_{S2} are Starling resistors which value depend on pressures P_I , P_E and P_V

$$R_S = \begin{cases} \frac{R_0}{1 - \frac{P_E}{P_I - P_V}}, & P_I - P_V > P_E, \\ \infty, & P_I - P_V \leq P_E. \end{cases} \quad (1)$$

In the electrical analogue of vascular system segment U corresponds to pressure P_I , U_S corresponds to pressure difference ($P_I - P_V$). The values of resistors R_{S1} and R_{S2} may be different tissue values E_1 and E_2 (E in electrical analogue of vascular system corresponds to tissue pressure P_E)

$$R_{Sj} = \begin{cases} \frac{R_0 j U_S}{U_S - E_j}, & U_S > E_j, \\ \infty, & U_S \leq E_j, \quad j=1,2. \end{cases} \quad (2)$$

When the blood vessel bifurcates two cases are possible:

- $E_1 \gg E_2$, or $E_1 \ll E_2$. In this case there is no blood flow in the vessel with a higher tissue pressure;
- $E_1 \approx E_2$. In this case:

$$U_S = \frac{U(t)R_0R_0^1 + R(R_0E_1 + R_0^1E_2)}{R_0R_0^1 + R_0R + R_0^1R}, \quad (3)$$

$$I_{S1} = \begin{cases} \frac{U(t)R_0 - E_1(R_0 + R) + E_2R}{(R_0 + R)R_0}, & U(t) > E_1, \\ 0, & U(t) \leq E_1, \end{cases} \quad (4)$$

$$I_{S2} = \begin{cases} \frac{U(t)R_0 - E_2(R_0 + R) + E_1R}{(R_0 + R)R_0}, & U(t) > E_2, \\ 0, & U(t) \leq E_2. \end{cases} \quad (5)$$

Experiments

We have built two static models in MATLAB and its supplement SIMULINK. The input signal $U(t)$ for the first model is blood pulse. This curve can be divided to two parts; the first part represents cardiac tissue in systole and can be approximated by sine wave and the second part of the input curve is equal to zero

$$U(t) = \begin{cases} U \sin^2\left(\frac{\pi \cdot t}{T_s}\right), & t \in (0, T_s), \\ 0, & t \in (T_s, T). \end{cases} \quad (6)$$

The input signal is depicted in Fig. 3.

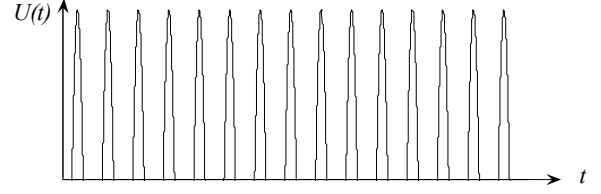


Fig. 3. The input signal of model

The input signal for the second model is constant voltage U (corresponds to constant pressure P_I). The second model corresponds to the model represented in [5]. Other parameters in both models: $R_0 = 30$, $R = 85$, $U = 1$.

These values of parameters are chosen in order to compare modelling results with results represented in [5]. The modelling results are depicted in Fig. 4 and Fig. 5, where

$$S1 = \frac{I_1}{I_1 + I_2}, \quad S2 = \frac{I_2}{I_1 + I_2}. \quad (7)$$

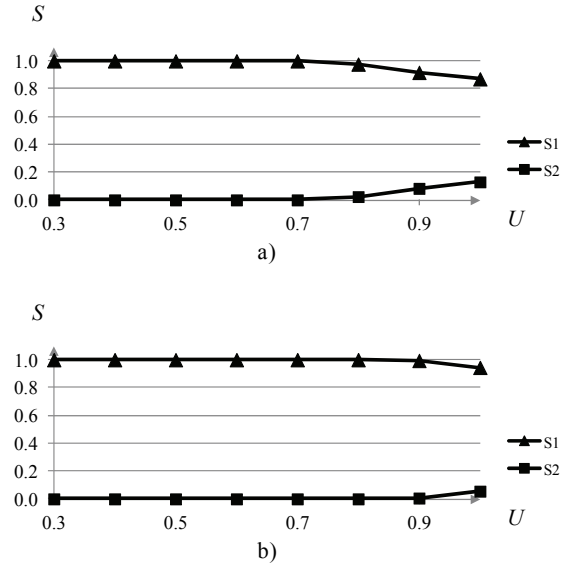
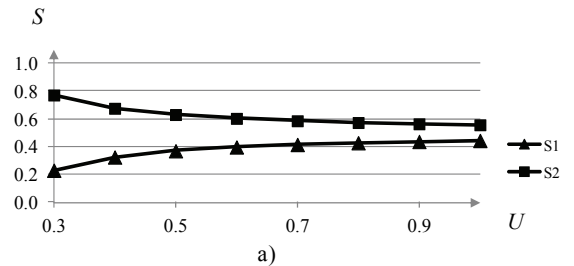


Fig. 4. The blood flow distribution when blood vessel bifurcates: a – model with constant pressure; b – model with blood pulse. Tissue pressure $E_1 = 0$, $E_2 = 0.2$

The blood flow improvement in blood vessels with increased tissue pressure is less noticeable when the first model is used.

The case where tissue pressure gradient is small is depicted in Fig. 5.



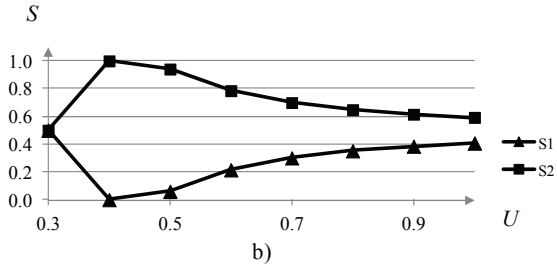


Fig. 5. The blood flow distribution when blood vessel bifurcates: a – model with constant pressure; b – model with blood pulse. Tissue pressure $E_1 = 0.1, E_2 = 0.13$

In the case where the difference of tissue pressure value is not large, modelling results differ significantly. The elevation of pressure in a certain range does not restore blood flow.

The results of the model with blood pulse are more precise because the blood flow redistribution occurs only during the time period when pressure of blood pulse exceeds some threshold.

Dynamic model

The electrical analogue of a cardiovascular segment is depicted in Fig. 6. The model includes two Starling resistors with common inflow and a capacitance. Starling resistors evaluate tissue pressure and capacitance evaluates blood vessel elastic properties.

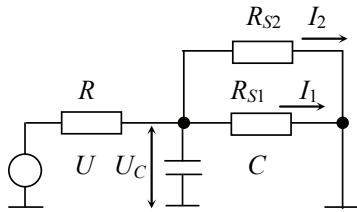


Fig. 6. The electrical analogue of a vascular system segment evaluating blood vessel elastic properties

In this model we must solve the following equations:

$$\frac{dU_C}{dt} = \frac{U_C}{C} \left(\frac{1}{R} + \frac{1}{R_{S1}} + \frac{1}{R_{S2}} \right) - \frac{U}{RC}, \quad (8)$$

$$R_{S1} = \begin{cases} \frac{R_0 U_C}{U_C - E_1}, & U_C > E_1, \\ \infty, & U_C \leq E_1, \end{cases} \quad (9)$$

$$R_{S2} = \begin{cases} \frac{R_0 U_C}{U_C - E_2}, & U_C > E_2, \\ \infty, & U_C \leq E_2. \end{cases} \quad (10)$$

We have built a dynamic model in MATLAB and its supplement SIMULINK. The main subsystem of this model is depicted in Fig. 7.

The input signal is the same as in the first static model. It is possible to change tissue pressure values E_1, E_2 and to use various resistors R_0 for different blood vessels.

This possibility is very important for modelling more complex cardiovascular subsystems.

The results of a modelling are depicted in Fig. 8.

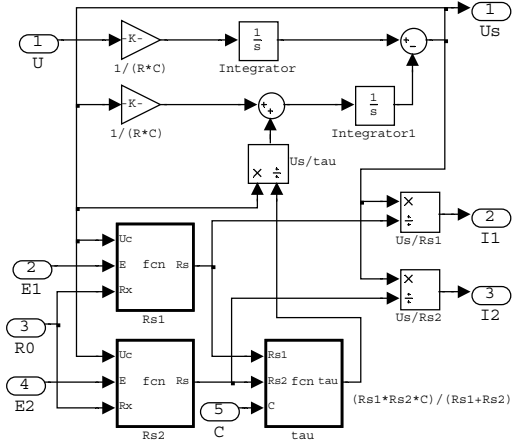


Fig. 7. The main subsystem of model

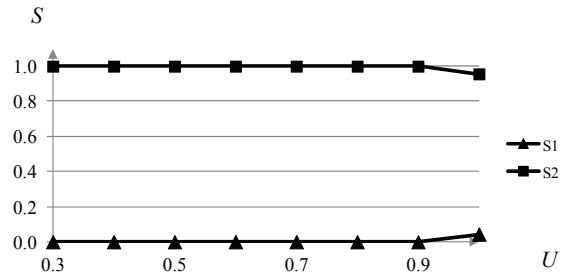


Fig. 8. The blood flow distribution. Tissue pressure $E_1 = 0, E_2 = 0.2$

In this case parameters S_1 and S_2 are

$$S_1 = \frac{I_{11}}{I_{11} + I_{21}}, \quad S_2 = \frac{I_{21}}{I_{11} + I_{21}}, \quad (11)$$

where

$$I_{11} = \frac{1}{T} \int_0^T I_1(t) dt, \quad I_{21} = \frac{1}{T} \int_0^T I_2(t) dt. \quad (12)$$

The modelling results in Fig. 8 show that increased blood pressure does not restore blood flow misdistribution efficiently when tissue pressure gradient is high.

The case when tissue pressure gradient is low depicted in Fig. 9.

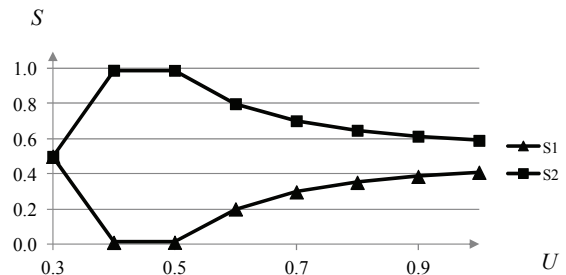


Fig. 9. The blood flow distribution. Tissue pressure $E_1 = 0.1, E_2 = 0.13$

When the tissue pressure difference is low, elevated pressure restores the blood flow more effectively. In some range of blood pressure the ratio of the flow in the vessels 1 and 2 increases. When blood pressure exceeds a certain threshold (depending on tissue pressure) then blood flow ratio decreases.

Conclusions

The development of intelligent medical systems, electronic monitoring systems required for physiological models. The blood pressure support biomedical devices need a model of the cardiovascular system.

The Starling resistor static model does not evaluate blood vessels elastic properties, so blood flow redistribution with elevation of pressure is overrated.

The Starling resistor dynamic model is more precise and allows fixing the blood pressure threshold when the blood flow improvement in a blood vessel with increased tissue pressure occurs. This threshold is less expressed in static model. The threshold fixing is very important by creating the real-time blood pressure support biomedical devices, because too high pressure may be harmful.

References

1. **Balaisis P., Eidukas D., Keras E., Valinevicius A.** The Selection of Biotronics Measures // *Electronics and Electrical Engineering*. – Kaunas: Technologija, 2010. – No. 1(97). – P. 9–14.
2. **Dubauskiene N., Markevicius V., Faktorovicius A.** Challenges of Close Loop Electronic Medical Systems // *Electronics and Electrical Engineering*. – Kaunas: Technologija, 2010. – No. 5(101). – P. 95–98.
3. **Guzauskas R., Dubauskiene N., Navikas D., Valinevicius A.** Ensuring the Efficiency of the Hazardous Biotronics Technologies // *Electronics and Electrical Engineering*. – Kaunas: Technologija, 2009. – No. 3(91). – P. 107–112.
4. **Abdolrazaghi M., Navidbakhsh M., Hassani K.** Mathematical Modelling and Electrical Analog Equivalent of the Human Cardiovascular System. // *Cardiovascular Engineering*. – Springer Netherlands, 2010. – Vol. 10. – No. 2. – P. 45–51. DOI: 10.1007/s10558-010-9093-0.
5. **Lakin D. W., Stevens A. S., Thakore J. N.** On the Pressure Dependence of a Starling-Like Resistor. // *International Journal of Pure and Applied Mathematics*, 2006. – Vol. 32 – No. 1 – P. 23–32.
6. **Pranevicius M., Pranevicius O.** Cerebral venous steal: Blood flow diversion with increased tissue pressure. // *Neurosurgery*, 2002. – Vol. 51. – No. 5. – P. 1267–1274.
7. **Brienza N., Ayuse T., O'Donnell C. P., Permutt S., Robotham J. L.** Regional control of venous return: Liver blood flow // *American Journal of Respiratory and Critical Care Medicine*, 1995. – Vol. 152. – No. 2. – P. 511–518.
8. **Formaggia L., Lamponi D., Tuveri M., Veneziani A.** Numerical modeling of 1-D arterial networks coupled with a lumped parameters description of the heart. // *Computer Methods in Biomechanics and Biomedical Engineering*, 2006. – Vol. 9. – No. 5. – P. 273–288.
9. **Liang F., Takagi S., Himeno R., Liu H.** Multi-scale modeling of the human cardiovascular system with applications to aortic valvular and arterial stenosis. // *Medical and Biological Engineering and Computing*, 2009. – Vol. 47. – No. 7. – P. 743–745.
10. **Wang J. J., Parker, K. H.** Wave propagation in a model of the arterial circulation. // *Journal of Biomechanics*, 2004. – Vol. 37. – No. 4. – P. 457–470.

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The development of intelligent medical systems, electronic monitoring systems required for physiological models. The proposed model of venous blood flow realized on the electric circuit basis can be used to support the development of blood pressure, real-time devices. Designed for venous blood nonlinear dynamic model is an extension of Starling resistor model. When the tissue pressure increased, static models are not suitable. Suggested non-linear dynamic model allows determining the threshold pressure, when blood flow returns to normal in damaged blood vessels. The integrated model can be used for blood pressure support biomedical devices. III. 9, bibl. 10 (in English; abstracts in English and Lithuanian).

A. Mikuckas, A. Venčkauskas, I. Mikuckienė. Dinaminis Starlingo rezistoriaus modelio tobulinimas // Elektronika ir elektrotechnika. – Kaunas: Technologija, 2012. – Nr. 6(122). – P. 7–10.

Kuriant medicininės intelektualios elektronines monitoringo sistemas reikalingi fiziologinių sistemų modeliai. Pasiūlytas elektros grandinių pagrindu sukurtas veninės kraujotakos modelis gali būti panaudotas kuriant kraujo spaudimo palaikymo realaus laiko įrenginius. Sukurtas veninės kraujotakos dinaminis netiesinis modelis yra Starlingo rezistoriaus modelio patobulinimas. Esami statiniai modeliai netinka, kai audinių spaudimas padidėjęs. Sukurtas dinaminis netiesinis modelis leidžia nustatyti spaudimo slenkstį, kai kraujotaka pažeistose kraujagyslėse atsinaujina. Integruotą modelį galima naudoti biomediciniuose kraujo spaudimo palaikymo įrenginiuose. II. 9, bibl. 10 (anglų kalba; santraukos anglų ir lietuvių k.).