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*Advan in Physiol Edu* 272:S26, 1997.



***Advances in Physiology Education*** is dedicated to the improvement of teaching and learning physiology, both in specialized courses and in the broader context of general biology education. It is published four times a year in March, June, September and December by the American Physiological Society, 9650 Rockville Pike, Bethesda MD 20814-3991.

ISSN: 1043-4046, ESSN: 1522-1229. Visit our website at http://www.the-aps.org/.

# MODELING BLOOD FLOW IN VESSELS WITH CHANGEABLE CALIBER FOR PHYSIOLOGY AND BIOPHYSICS COURSES

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A model based on elementary principles of hydrodynamics and mathematics is proposed for classroom research on concepts related to blood flow physiology. This is an analog model of the vascular system in which blood flow is represented by electrical current flowing in a resistance circuit. The model permits analysis of the change in hemodynamics with local stenosis of both large and peripheral vessels.
*AM. J. PHYSIOL. 272 (ADV. PHYSIOL. EDUC. 17): S26–S30, 1997.*

*Key words:* hemodynamic parameters; analog model; classroom physiology-physics-mathematics modeling



Study of characteristics of blood flow in the vascular system has an important place in physiology and pathophysiology courses. One of the main topics is analysis of hemodynamics in sites where there is a change in vessel caliber. Such changes may be observed at the boundaries of vessels with different calibers, in sections of vessels with thrombi, when local spasms of vessels occur, in progressive aneurism, and with a number of other pathologies. In these cases, the question arises: how will pressure (P) and blood flow (Q) change locally and in the entire vascular system? Which characteristics of vessels will most influence P and Q?

For better teaching and learning about this problem, with the purpose of going deep into this theme in a biophysics course preceding a physiology course, students are encouraged to research the problem of changing P and Q under the following conditions: *1*) stenosis of a large vessel (the aorta or an artery), for example as when a thrombus forms in it; and *2*) stenosis of a peripheral vessel (a capillary or an arteriole) in a multibranched section of the vascular system.

For solving this problem we propose the use of an electrical resistance model of the vascular system. This approach permits results to be obtained using simple algebraic equations and does not require knowledge of differential equations [as in the resistive-capacity Frank model (1)].

The suggested model can be used to simulate Q and P changes, allowing students to alter diameters and observe changes when vessels are connected in parallel or in series. Students must show the principal differences in hemodynamic changes during stenosis in large vessels and in vessels of the peripheral system. One of the main aspects of modeling is for students to learn how to make assumptions correctly and to gain an understanding of physiological processes.

## RESISTANCE MODEL

This model operates under the following assumptions: *1*) vessel elasticity is not taken into account; *2*) only changes in mean pressure are investigated, i.e., pressure pulsations during the cardiac cycle are not taken into consideration; and *3*) flow is laminar.

In this model, blood flow in vessels is analogous to the electrical current in the circuit of active resistances. The central equation governing the relationships among Q, pressure drop ($\Delta$P), and hydraulic resis-

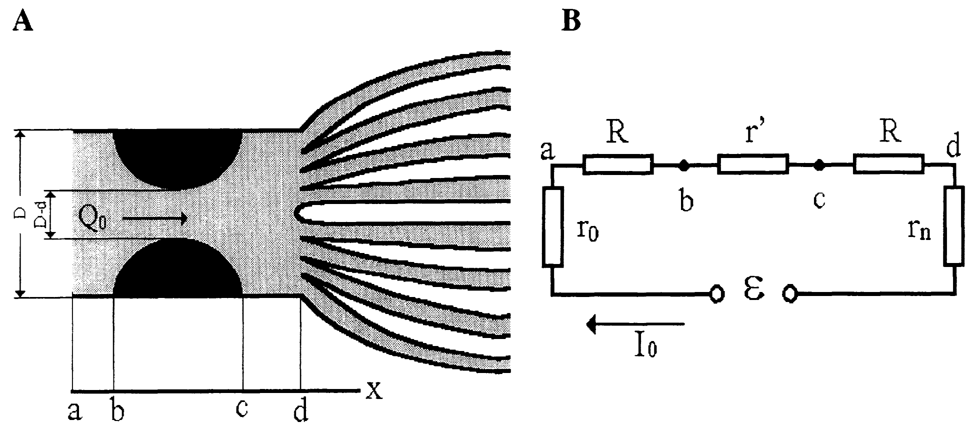


**FIG. 1.**
**A large vessel with a localized stenosis at *bc* (*A*) and equivalent electrical scheme (*B*). D, caliber of the vessel at *points a* and *d*; d, change of the vessel caliber along section *bc* with stenosis; D − d, resulting caliber of the vessel in section *bc* with stenosis; *R* is equivalent to the hydraulic resistance of sections *ab* and *cd* of the vessel; *r′* is equivalent to the hydraulic resistance of section *bc*; $r_0$ is equivalent to the hydraulic resistance of the vascular system preceding *point a*; $r_n$ is equivalent to the hydraulic resistance of the vessel after *point d*; ε is equivalent to the mean pressure ($P_0$) at the beginning of the aorta; $I_0$ is equivalent to blood flow ($Q_0$).**

tance (*W*) is Poiseuille's law (2)

$$\Delta P = Q \cdot W$$

The electrical analogy is Ohm's law

$$V = IR$$

The electrical flow (current; *I*) is equivalent to Q the voltage drop (V) is equivalent to ΔP, and electrical resistance (*R*) is equivalent to *W*.

Under steady-state conditions the flow ($Q_0$) through any given cross section must equal Q through any other cross section. In an equivalent electrical system the total current ($I_0$) is also constant.

### Model of Blood Flow in a Large Vessel With Local Stenosis

When local stenosis of a large vessel takes place, for example by spasm or thrombus formation (Fig. 1*A*), Fig. 1*B* shows the equivalent electrical circuit. Because $Q_0$ is constant, the $I_0$ in the equivalent electrical circuit also does not change. This may be realized only by an increase of electrical potential at *point a* at the expense of an electromotive force (ε) increase. This is analogous to an increase of mean pressure ($P_0$) in the arterial entrance.

With the use of the equivalent electrical scheme, it is not difficult to calculate that the pressure in the vessel at *point a* is increased to the value

$$P_0' = P_0 \frac{2 + w'/W}{2 + w/W}$$

where $P_0$ is the mean pressure at *point a*, when stenosis is absent; *w* is the hydraulic resistance of *bc*, when stenosis is absent; *w′* is the hydraulic resistance of section *bc* with stenosis; *W* is the hydraulic resistance of regions *ab* and *cd*.

At sections *ab*, *bc*, and *cd* the values of the ΔP (pressure difference) are calculated by the formulas

$$\Delta P_{ab} = \Delta P_{cd} = \frac{P_0}{2 + w/W}$$

$$\Delta P_{bc} = \frac{P_0 \cdot w'/W}{2 + w/W}$$

The sections' resistances are calculated by the formulas

$$w = \frac{8\eta l}{\pi(D/2)^4}$$

$$w' = \frac{8\eta l}{\pi[(D - d)/2]^4}$$

$$W = \frac{8\eta L}{\pi(D/2)^4}$$

where $\eta$ is the coefficient of blood viscosity, $l$ is the length of region *bc*, $L$ is the length of regions *ab* and *cd*, D is the caliber of the undamaged vessel, and d is the change of the vessel caliber along the sections with stenosis; D − d is the resulting caliber of the vessel in this section.

The calculated pressure distribution along the vessel is presented in Fig. 2. Each line corresponds to the ratio d/D.

Thus, on the basis of the represented pure resistance model, the increase of the blood pressure in the cardiac left ventricle may be estimated when stenosis appears in a large artery if Q is constant.

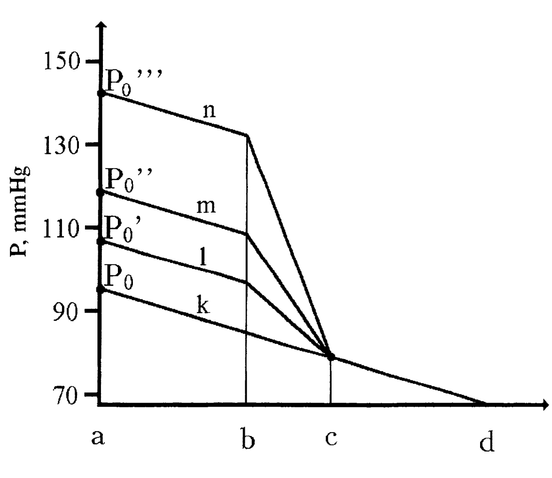


**FIG. 2.**
**Pressure distribution along a large vessel with localized stenosis with the ratio d/D (for *lines k, l, m,* and *n*, ratio d/D is equal to 0, 0.1, 0.25, and 0.4, respectively). Different primes indicate different initial pressures.**

**Model of Blood Flow in a Peripheral Vessel with Local Stenosis**

In the case of stenosis of one peripheral vessel in a multibranched section of the vascular system (Fig. 3*A*), let the number of the parallel vessels be $n > 10$, because when $n > 10$ it may be considered that the total resistance of the electrical system (Fig. 3*B*) is not changed; the main circuit current flow and potential difference across *points a* and *d* remain the same. Here a redistribution of current flow has occurred between resistances (and accordingly a redistribution of the Q between vessels: the greater part flows to undamaged vessels). Referring to the equivalent electrical circuit, it may be calculated

$$\Delta P_{ab} = \Delta P_{cd} = \frac{P_a - P_d}{2 + w'/W}$$

$$\Delta P_{bc} = \frac{(P_a - P_d) \times w'/W}{2 + w'/W}$$

Figure 4 shows the pressure distribution along a vessel in which local stenosis has taken place.

The basic clinical parameter of cardiovascular system function is Q. With the use of this resistance model it may be investigated how the characteristics of the section with local stenosis (its length and the change of vessel caliber) influence Q in a given vessel. Referring to the equivalent electrical circuit (Fig. 3*B*) it is not difficult to produce the equation

$$q' = q_0 \frac{2 + w/W}{2 + w'/W}$$

where $q_0$ is the blood flow in each vessel when stenosis is absent, $q'$ is the blood flow in a vessel with a change in caliber.

The ratio $q'/q_0$ does not vary linearly with thrombus size. Along the *x*-axis there is the ratio d/D. If vessel stenosis is absent (d = 0), the flow is not changed ($q'/q_0 = 1$ as is shown in Fig. 5). When the caliber of this vessel is decreased to zero (the thrombus completely occludes this vessel; d = D), the flow $q' = 0$. Moreover, the flow in this vessel depends also on the thrombus length. It is known that thrombi may stretch

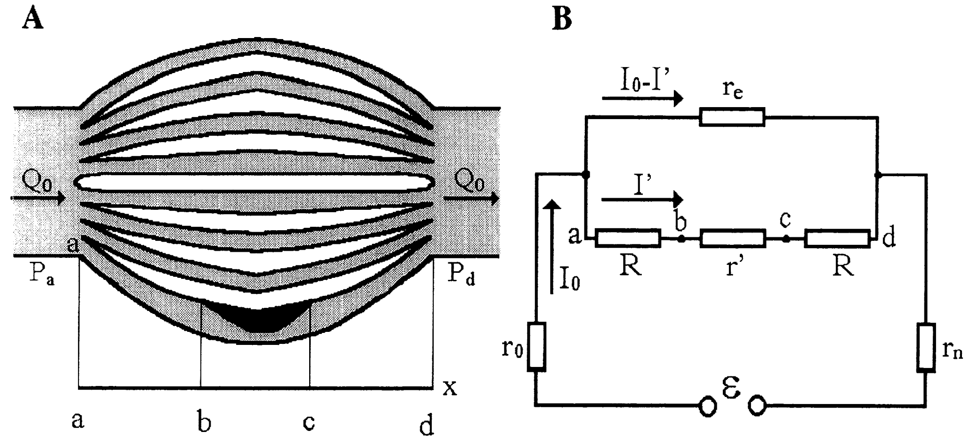


**FIG. 3.**
**A multibranched vessel system with localized stenosis in one of the peripheral vessels (*A*) and the equivalent electrical scheme (*B*). $r_e$, Equivalent shunt resistance, corresponding to the total hydraulic resistance of all undamaged vessels aligned in parallel; $I'$ is equivalent to flow (q) in the peripheral vessel with stenosis; $I_0 - I'$ is equivalent to flow in all other vessels of the multibranched system.**

along the entire length of a vessel. In Fig. 5, this possibility is also taken into account: in the model, the section *bc* may be increased to the length of the entire vessel. In Fig. 5, *right*, three variants of the relative length of the stenosis (section *bc*) and three curves of flow corresponding to these different lengths are represented.

The decrease of Q in the damaged vessel may lead to the lowering of the intensity of the metabolism between the blood and tissue and may cause hypoxia of the neighboring tissue and perhaps even its necrosis. The effects may be associated with such conditions as infarct and insult.

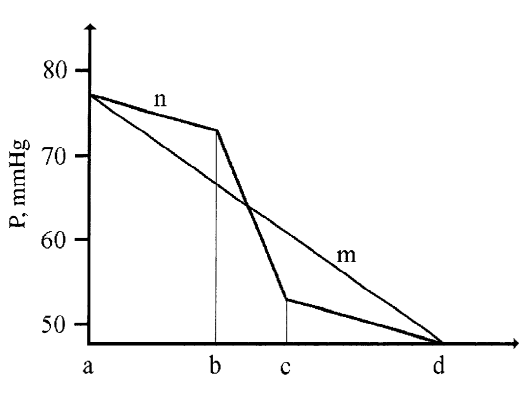


**FIG. 4.**
**Pressure distribution along one of the peripheral vessels in a multibranched system. *m*, Pressure drop in a peripheral vessel without stenosis; *n*, pressure changes in different section of the peripheral vessel with stenosis; *ab* and *cd* are the sections without stenosis, *bc* is the section with stenosis.**

### Use of Resistance Models in the Educational Process

The resistance models described above for large vessels (Fig. 1) and for small peripheral vessels (Fig. 3) are carried out with the use of personal computers

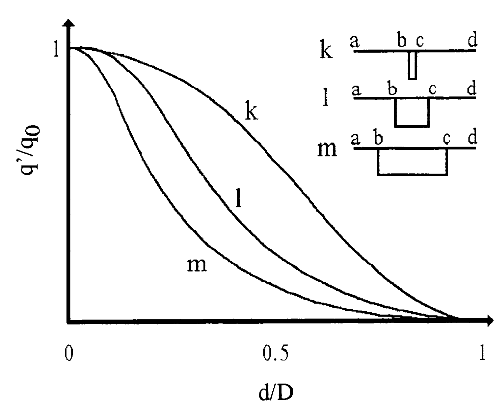


**FIG. 5.**
**Influence of change of vessel caliber on blood flow in a peripheral vessel; curves correspond to the different lengths of the stenosed section [for *curves k*, *l*, and *m*, ratios of length of region *bc* to length of regions *ab* and *cd* (*l/L*) are equal to 0.04, 0.2, 0.5, respectively, and relative lengths of the stenosed sections *bc* in vessel *ad* are shown (*right*)]. Values D and d are illustrated in Fig. 1.**

with Pentium processors using specially developed computer programs. During the lessons each student works individually. On the monitor, blood flow in different parts of the vascular system (Fig. 1*A* and 3*A*) is presented in color with the respective equivalent electrical schemes (Figs. 1*B* and 3*B*). The professor suggests that the students solve 10 tasks. In particular the students can alter the caliber of vessel (D) and its length ($L$), the parameters of the vessel section with stenosis: the resulting caliber (D − d) and the length ($l$), the total hydraulic peripheral resistance of the multibranched vascular system ($r_e$), the mean pressure ($P_0$), the blood flow ($Q_0$), and the blood viscosity ($\eta$).

In accordance with the model postulates these changes will cause a pressure drop ($\Delta P$) and changes in blood flow (Q) in the vessel sections. The computer solves the equations and families of graphs for the changes in $\Delta P$ and Q are plotted on the basis of these calculations. Examples of such graphs are illustrated in Figs. 2, 4, and 5. The color of each curve corresponds to the color of the task. For example, if the *task k* (in Fig. 5, *top right*) is colored blue then the *curve k* corresponding to this task will also be blue. This is true for all graphs and tasks in the model analysis.

This model is studied by the students in the Sechenov Moscow Medical Academy in their courses in biophysics and physiology (10 tasks) and more deeply in their course in pathophysiology (5 additional tasks). Moreover, the resistance model is studied by physicians who wish to improve their specialist qualifications. Before graduation, during final examinations, both the students and the physicians improving their qualifications are given the questions and the tasks on this subject. Practically all of them answer the questions and analyze the suggested situations correctly. This result confirms the high efficiency of education using the resistance model.

## CONCLUSION

This suggested lesson has the following simultaneous effects: *1*) gives the students concrete knowledge of the important physiological and pathophysiological topic of blood flow hemodynamics; *2*) gives the students experience of physics, analogs, and mathematical modeling of physiological processes; and *3*) develops a scientific research approach to the correct formulation of physiological problems and their solutions.

The authors thank Professor of Biological Science V. F. Antonov for constructive discussion and N. E. Bogatyreva for taking part in the creation of the computer program.

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Received 19 September 1996; accepted in final form 5 March 1997

## References

1. **Caro, C. G., T. J. Pedley, R. C. Schroter, and W. A. Seed.** *The Mechanics of the Circulation*. Oxford, UK: Oxford University Press, 1978.
2. **Landau, L. D., and E. M. Lifshits.** *The Theoretical Physics. The Hydrodynamics*. Moscow, Russia: Nauka, 1986.