Prof. Dr. Vincze János

1.

In my opinion, IVT shows great similarity and emphasizes similarity.

In the living organism, it never happens that two different effects are identical.

So IVT ~ NO, but not IVT ≈ NO

This explanation has to be mathematically understood and transposed into biophysical biology.

It follows that the answer to the first question should be that:

IVT is likely to result in NO due to its mechanism of action.

(a) Take account of the shearing forces:

The angle of rotation of the shear φ is proportional to the distance d from the fixing and the torque (M) that creates the rotation:

$$\varphi = \frac{2}{\pi \cdot G} \cdot \frac{d}{r^4} \cdot M$$

where: G - shear modulus; r - the radius of the circle measured in the transverse direction of the system.

(b) Nerve stimulation should be considered:

The action potential of the nervous system is described as follows:

$$\mathbf{A(t) = K \,.\, \ln s(t) + m}$$

where K - is a constant, m is the resting potential, s (t) h, where the excitation value of the membrane.

So describing the action potential with a functor connection:

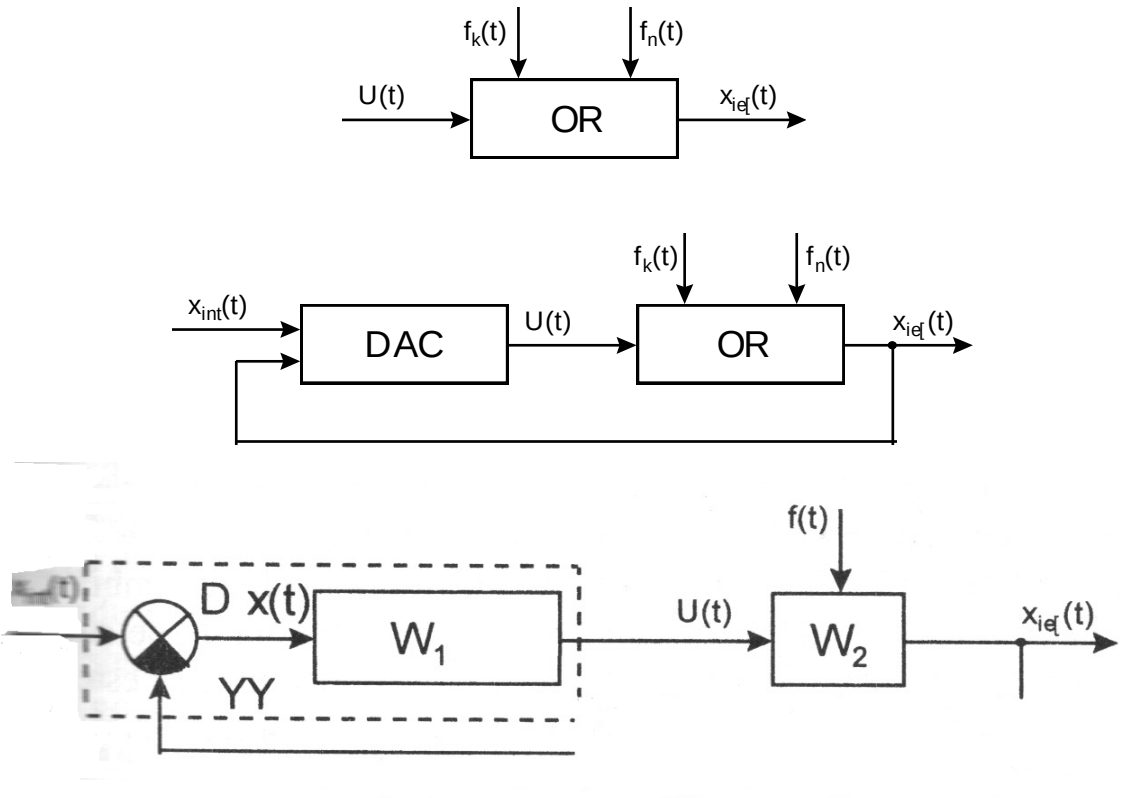
$$\mathbf{A_M(t) =} \begin{cases} \mathbf{m} & \mathbf{t \le t_0 + \eta;\ t > t_2} \\ \mathbf{K \,.\, \ln s(t) + m} & \mathbf{t_0 + \eta < t \le t_1 + \eta} \\ \mathbf{A_M \exp(-t/\tau) + m} & \mathbf{t_1 + \eta < t \le t_2} \end{cases}$$

I'm not going to detail it further:

2. Conduct IVT

The IVT effect is a regulatory mechanism in the living organism, and therefore the feedback procedure should be applied.

So, so called block schemes must be written::



This is a more complete feedback box point

So the terms and formulas for negative feedback apply:

Here, o (t) is the output output value,

o * (t) is the average value

o (t1), o (t2) or (t3), ..., o (tn) = o * (t); the consecutive values as a function of time

t1 <t2 <t3 <... <tn is the time series

then only feed back negative if the following two conditions are met::

$$|o^*(t) - o(t_1)| > |o^*(t) - o(t_2)| > |o^*(t) - o(t_3)| > \ldots > |o^*(t) - o(t_n)|$$

$$\frac{d\left|o(t_i) - o^*(t)\right|}{dt} \leq 0.$$

then only feed back positive if the following two conditions are met::

$$|o^*(t) - o(t_1)| < |o^*(t) - o(t_2)| < |o^*(t) - o(t_3)| < \ldots < |o^*(t) - o(t_n)|$$

$$\frac{d\left|o(t_i) - o^*(t)\right|}{dt} \geq 0$$

3.

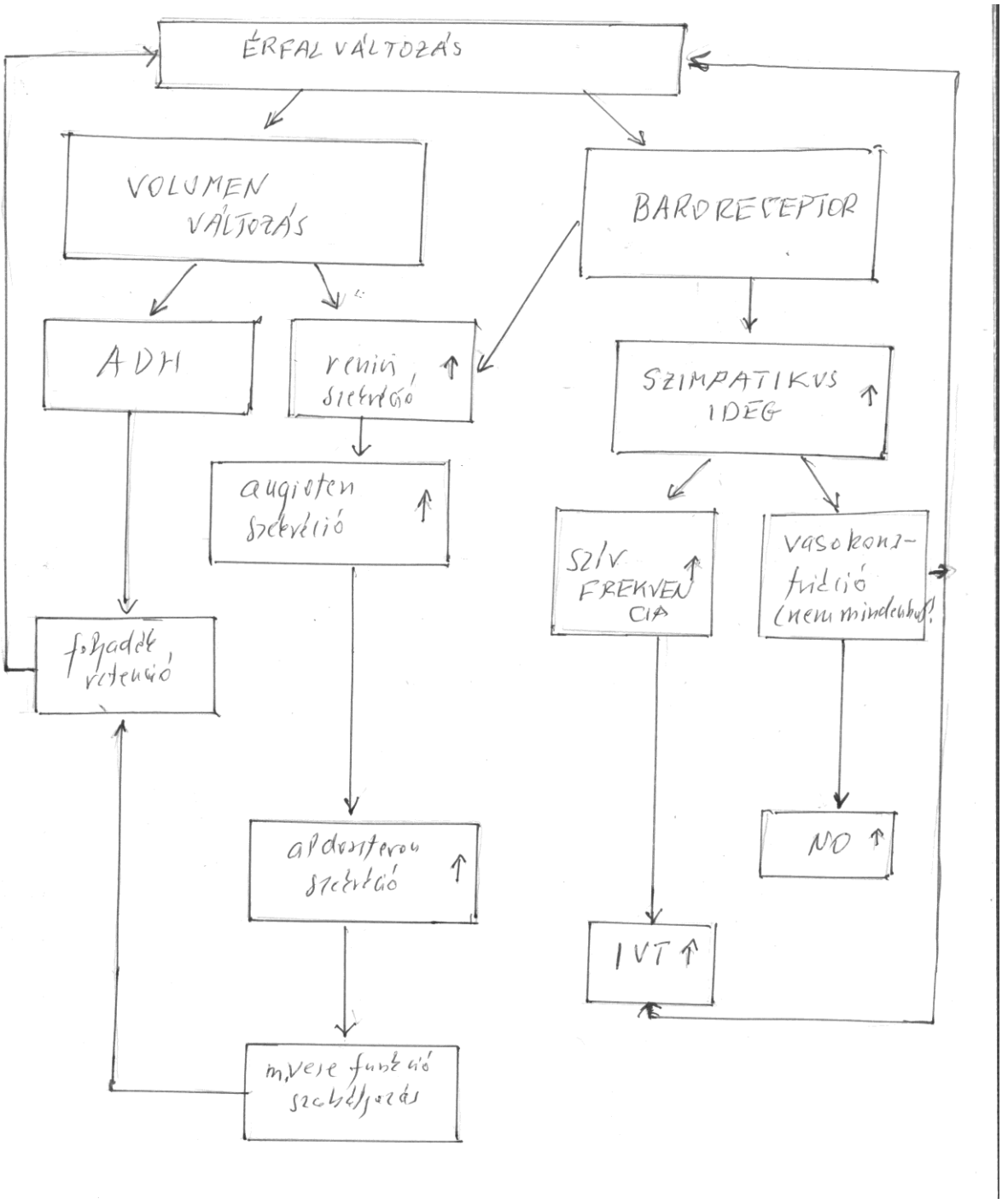
The processes can be most clearly observed through the example of hypovolaemic shock. As a result of the loss of a significant amount of blood (more than 25% of the circulating blood volume), saturation pressure decreases in the bloodstream. Arterial blood pressure decreases and at the same time decreases in venous insufficiency. As less blood flow to the right heart part, the volume of the volume will also be smaller and this will further reduce arterial blood pressure. The circulatory system of high pressure (sinus caroticus) and low pressure (large veins, right atrium) baroreceptors respond to decreased wall stress with increased pulse response. Due to a significant reduction in blood pressure, brain hypoxia also activates the "cerebral ischemic" reflex, which causes the sympathetic nervous system to function as a generalized trigger. stimulation of α-receptors in all tissues where they occur in large numbers (kidneys, abdominal vascular veins, muscle and fat tissue) results in pronounced vasoconstriction, higher heart rate increases cardiac output, all of which are associated with arterial blood pressure In the first few hours after blood loss, the activity of the sympathetic nervous system is getting more and more intense.

4.

The reduction in peripheral resistance is caused by a body-wide vasodilation, although the mechanism is not yet fully known. Septic patients are mostly feverish, and this in itself increases tissue metabolism leading to local vasodilatation. Lactic acid liberated from the muscle tissue and consequent lactacidosis may cause ingestion. However, this alone is insufficient to explain the high degree of vasodilation.

Nitrogen monoxide (NO) plays an important role in the development of vasodilation. NO is normally produced in endothelial cells according to the need, and one of its most important functions is to create the proper vasodilation by regulating the smooth muscle tone. NO is identical to EDRF (endothelium-dependent relaxant / actor). From arginine, NO synthase (NOS) causes citrullin formation and NO is released. This form of NOS is called a constitutive form and its function is regulated by the intra-cellular Ca2 + level. Among the numerous physiological effects of NO, it is also worth mentioning the interfering effect of cells (immune cells, thrombocytes). In many cells, such as immune cells, there is another form of NOS in smooth muscle cells, the inducible form (iNOS). It is also quite similar to IVT's mechanism of action.

I tried to depict it in a block scheme.



May 2018