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Review Article

The Role of Cervical Vertebral Arteries Blood Flow in Centralized Aerobic-Anaerobic Energy Balance Compensation: When Hypothesis Becomes a Theory

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Submitted: 06 October 2021

Accepted: 23 October 2021

Published: 27 October 2021

ISSN: 2641-7731

Abstract

If we consider the functioning human body as a dissipative structure, then we need to assume the existence of a regulative system, that collects and analyses data related to the structure functioning, and, eventually, sends signals across the body to adjust homeostasis. If the entrance of information is blocked, the system will start to distribute wrong signals leading to dysfunction of the structure. This approach was applied to Arterial Hypertension (AHT) caused by the blocking of vertebral arteries. The preliminary data so far support the hypothesis. We proposed for the discussion that was to collect the additional data set, required to consider hypothesis proven.

INTRODUCTION

Let's consider the vertebral creature as a dissipative structure. Then it should be commonly accepted, that the structure is balancing between two ways of glucose oxidation - Aerobic (AE) and Anaerobic (AN) to cover its energetic needs [1]. A century ago Ervin Bauer summarized that - "All living organisms are characterized by being a system that is not in equilibrium in its environment and is so organized that it transforms the sources and forms of energy taken up from its environment into such state that acts against the establishment of equilibrium in the given environment. All the energy taken up by the organism from the environment must be fully used to deviate from the equilibrium state" [2,3]. According to his theory, the organism within itself must have some energy potential to work against the equilibrium; otherwise, it will be unable to maintain the necessary order and will be eventually "dissolved" by the external environment. The interpretation of this law in physics is nothing but the first law of thermodynamics and it can be reflected using the following equations:

$$Q = U + W \quad (1)$$

Where Q is the heat, U is the internal energy of the system, and W is the work carried out by the system.

Since there must be a constant level of the internal energy ECONST in the living body, we produced a variation of the formula applicable to living systems:

$$E = E_{CONST} + W \quad (2)$$

Where E is the sum of all energies penetrating the biosystem, E_{CONST} is the constant total internal energy of the biosystem, and W is the sum of all the work done by the biosystem in the external environment. It is obvious, that to maintain a structure through which the constantly changing energy flow is passing on a constantly adjusted level of energy, we need feedback, which adjusts the consumption level.

HYPOTHESIS FORMULATION

A hypothesis of centralized AE and AN energy balance compensation CAAEBC of the human body proposed, that to maintain a constant level of (E_{CONST}) energy metabolism in the brainstem, the microcirculatory and cellular levels of the AE (oxygen-E_{AE}) and AN (glucose, lipoproteins, etc.-E_{AN}) molecular components of the metabolism are constantly monitored to fulfill

$$E_{CONST} = E_{AE} + E_{AN} \quad (3)$$

The decrease of E_{AE}, due to particular reasons (reduction in oxygen content in the microcirculatory bed and brain stem

cells), two types of centralized adaptation reactions take place in order to maintain the overall unchanged E_{CONST} value. These are reactions of CAAEBC in order to maintain the unchanged level of E_{CONST}. Moreover, the reactions of AN compensation, as less energy-efficient, are triggered only with the complete depletion of the reserves of AE compensation reactions. Therefore, if the access of the blood flow to the rhomboid fossa is jammed by the bulges on cervical discs cartilage (Figure 1), then the cerebellum starts to lift up BP, causing AHT. The inability to get access leads to the list of chronic diseases (see below). The recovery of the access leads vice versa to the stepwise healing of these diseases.

HYPOTHESIS EXPLANATION

AE compensation reactions are neurogenic cardiovascular reactions, which are expressed in a steady rise in Blood Pressure (BP) (an increase in the cardiac output force), a narrowing of the peripheral capillaries at rest, and an increase in cardiac rate. The goal of the AE compensation reaction is to increase the brain stem blood perfusion and hence restoration of the E_{AE} level.

AN compensation reactions are neurohumoral metabolic reactions that lead to an increase in the AN metabolism of sugars, phospholipids, and other energetically rich biochemical compounds. The purpose of this reaction is to increase E_{AN}, to maintain the balance of E_{CONST}, with a reduced E_{AE}.

According to Dobroborskiy, such reactions of the organism are manifestations of Phenotypic Adaptation (PA) [4]. The PA of a living organism to any changes in the environment first takes place by small forces along a simpler path. First of all, oxygen saturation of the brain occurs to a certain level, after which the reflex mechanism causing compensatory arterial AHT (AHT) is disconnected. If the brain encounters oxygen starvation for an extended period, then according to PA theory, changes occur at the biochemical level, namely, the balance of biochemical process shifts, that is, the contribution to the energy balance of AN processes increases and the contribution of AE processes decreases. The brain, lacking oxygen, determines its decrease, as a decrease in the level of oxygen in the atmosphere and thus, tries to adapt the work of the organism under an AE conditions [5,6]. In other words, the brain tries to adapt to the already changed, according to the information from the oxygen detector, external environment, which has remained the same. Since the brain in such a situation begins to receive signals about the critical conditions of the heart, then, as a control center, to save the cardiac resource, it rebuilds the biochemical processes under conditions of a reduced partial pressure of oxygen. There is a shift in AE⇄AN equilibrium towards AN, thus preserving the overall balance of energy that is necessary to fulfill Bauer's universal principle of biology and to balance the effect on the body of the second law of thermodynamics.

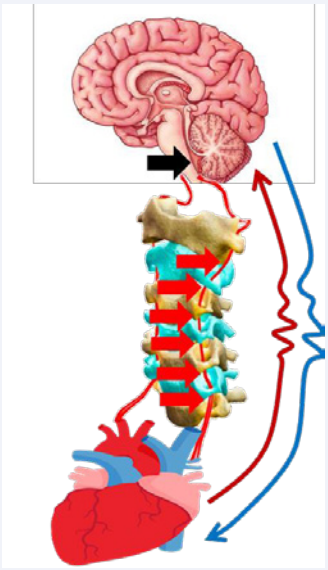

Figure 1 Schematic representation of hypothesis. Red arrows indicate parts of cervical vertebra arteries where jam with cervical discs' bulges usually take place. Black arrow indicates rhomboid fossa - the place of acquisition of the oxygen availability data.

Considering the work of these compensatory mechanisms- "fast" and "slow", we could present a remarkable clinical example- squeezing the vessels of the neck to cause a person short-term hypoxia (oxygen deprivation) or tough physical exercise (sudden increment of oxygen consumption) will immediately result in the reflexive increase of his BP and heart rate [5,6]. When the squeezing or exercise ends, all vital signs will quickly recover back to normal. This is an example of "quick" adaptation. If a person already has a long-term occlusion of the vessels due to cervical osteochondrosis or there is a narrowing of the lumen of the vessels due to the atherosclerotic process, we will see the manifestations of the action of the "slow" adaptation with a shift in the AE⇄AN balance, namely, the development of the metabolic syndrome in general, and the type 2 diabetes, in particular.

Although the theory of energy balance is coherent with Dobroborskiy's theory of PA, the CAAEBC theory is an improved theory, due to the introduction of such concepts as:

- E_{CONST}, which allows us to structure the balance of body energies.
- E_{AE} and E_{AN} of AE and AN contributions to the overall energy flow of the body.
- In theory, we link the regulation of these energy processes to the partial pressure of oxygen in the brain stem as anatomical positioning.

Let's see how such a hypothesis allows predicting the work of the organism in certain conditions and explains the causes and mechanisms of the development of metabolic disorders. In the final paragraph we will conclude, what measurements should be presented to justify our hypothesis. Let's continue with AHT, caused by intervertebral discs compression with hernias and protrusions of the cervical spine. The anatomical features of the cervical vertebrae are such that veins and arteries pass through the holes in their transverse processes (*arteria vertebralis*, *venae vertebralis*). Due to the displacement of the vertebrae and the constant deep muscle spasm around them, the vessels are subsequently clamped, their lumen is narrowed (the arteries are thin and convoluted), and the blood flow is reduced up to ten times or may even be stopped at all and, as a result, the amount of delivered oxygen to the oxygen detectors in the brain is dramatically reduced. Since the brain through detectors' signal

observes the lacks of oxygen, it takes emergency measures and commands the heart to increase the strength and/or heart rate so that blood, through all the blocks and obstacles, is still able to reach the brain and provide much-needed oxygen. A stable compensatory increase in pressure and/or heart rate is being developed-thus the brain is protected from hypoxia. Therefore, as soon as it is possible to unlock the vertebral arteries and veins, the pressure and heart rate should return to normal. In this case, if we are able to heal the patients from AHT through the restoration of vertebral arteries patency, then our hypothesis should be considered as confirmed. The normalization of AHT through the restoration of the blood flow of the brain stem is easy to register by measurement of BP and arterial linear blood flow (V_A). In this case, the decompression of the vertebral arteries and veins during the correction of deep neck muscles leads to measurable restoration of V_A of *sinistra* and *dextra arteria vertebralis* to the values of *arteria carotis communis*. The fact is that with age, there is a progression of osteochondrosis and complications arise that are associated with the gradual displacement of the cervical vertebrae. To prove this hypothesis, we will need to compare hemodynamic and biochemical data before and after treatment, before and after the restoration of the access of the blood flow to the rhomboid fossa of the cerebellum [7,8].

What do we know from currently available data?

1. The treatment according to[8] leads to the dramatic increment in VA with the simultaneous return of BP to normal level (Table 1) [7].

2. As a rule for elderly people, the diagnosis of "essential" or "idiopathic" AHT simply implies nothing more than a compensatory increase in BP due to circulatory disturbances in the brain stem due to compression of the vessels at the level of the cervical spine [9-14].

This brings us to AHT. Over time a person develops an osteochondrosis process in the cervical region, and since this is where the vertebral arteries pass through the transverse processes of the vertebrae, their lumen naturally narrows blood flow into the brain stem and into the rhomboid fossa where the vascular center is located. Osteochondrosis is a disease that is directly related to the psychological state of the individual, namely, the accumulation of stress factors in the body. This happened evolutionarily so that any nervous shock manifests itself in the tension of the neck muscles. Therefore, it is necessary to "squeeze the head in the shoulders, hide it" to protect the cervical arteries, because the predator, attacks the neck first. At its core, this reaction is atavism. Evolution should continue and similar atavisms will eventually disappear. But while there is such an effect, any stress, social or any other kind, produces an automatic reaction-the neck strains, and the head is drawn into the shoulders. Most people have special target muscles that harden when stressed. This is a kind of vicious circle: stress causes the release of adrenaline, which in turn strains the muscles of the neck and upper back, making even more adrenaline, etc.

Table 1: Effect of treatment sample of 100 patients according to on BP and VA [7,8].

Parameter	Male	Female
Sample size	50	50
Age, years	63.2±7.1	63.1±6.8
Systolic P_A, torr before treatment*	160±10.9	162±11.7
Systolic P_A, torr after treatment*	126±13.7	127±13.2
Changes after treatment in systolic V_{AV}, cm/s	20.8±6.9	21.9±8.3
Changes after treatment in systolic V_{AC}, cm/s	1.8±1.5	1.8±1.2

Over time, this condition becomes habitual. There is a chronic spasm of deep muscles and in general a spasm of all muscles of the cervical and thoracic sections. Against this background the microcirculation and nutrition of the intervertebral discs and ligaments are disturbed, their weakening occurs, and the vertebrae begin to shift and clamp the vessels. The partial pressure of oxygen in the brain stem decreases because the flow of blood decreases. Due to the activation of the vascular center in the rhomboid fossa, the brain stem delivers an efferent signal to the heart and it starts increasing the pressure by increasing the heart force and the heart rate. According to Dobroborskiy's theory, this is the first stage of PA. According to the hypothesis of CAAEBC, it is an AE compensation of the energy balance. First, there are short-term episodes of increased blood pressure to normalize the perfusion of the brain. With great efforts, through all these obstacles, blood is continually being pushed around the body. As a result of this mechanism, the partial saturation of oxygen in the brain tissues increases and blood pressure decreases. This is a kind of "biological relay". You can draw an analogy with an overheated internal combustion engine: the fan turns on, the engine cools, the fan turns off. If at this stage treatment isn't started, the muscle spasm does not go anywhere by itself. As a rule, symptomatic treatment is appointed, although it is not related to the elimination of the underlying problem. The patient thinks that he is being treated by taking antihypertensive drugs, but the original cause develops further. Moreover, the following occurs: after the continual abrupt rise in blood pressure, the body begins to perceive this state as being in an environment with a small amount of oxygen.

That is to say that the central apparatus-the brain-regularly receives less oxygen than it should have received by the parameters that are laid in the "BIOS". Accordingly, the control center proceeds to the processes of additional energy release from processes not related to breathing. The 'constant', that is, the total energy consumption of the body, must be preserved, otherwise, the destruction of the system (organism) will inevitably begin. The brain marks regular overloads and strains on the heart muscle and then activates the mechanism of spasm of the peripheral vessels, thus reducing the burden on the heart. Gradually, a slow centralization of blood circulation takes place. Therefore, the brain temporarily compensates for the state of hypoxia. With the unjustified administration of beta-blockers and vasodilators acting on the renin-angiotensin-aldosterone system, the brain receives an additional toxic effect that nullifies all its adaptive responses. The control center persistently continues the process of compensating the energy balance, due to which, over time, these drugs cease to function properly.

When the process of oxygen starvation is aggravated (no one treats osteochondrosis!), the following adaptive mechanism is activated: the biochemical component. There is a need for

extracting energy from AN source. There is a shift in AE⇄AN energy balance, and then a shift in the acid-base balance, resulting in the body's "acidification". Thus, the picture of PA related to functioning in a medium without oxygen is traced. Thus, diabetes mellitus type 2 develops in the form of impaired insulin metabolism and disruption of the normal functioning of the pancreas. That is, the organism tries to preserve the energy in the body on the constant level with the help of these reactions. However, modern medicine perceives the adaptive reactions of the organism as pathological states and orders them to be blocked by medicinal means, so that there is a fairly crude intrusion into the chain of cascades of fine biochemical processes. Without a common understanding of the mechanisms of central regulation, this is not only inefficient but also harmful. Against the background of constant imbalance, not only glucose but also the body's proteins begin to be consumed in the form of an energy subject. Furthermore, the function of the liver and cholesterol carriers in the blood is disrupted. Lipoproteins of high and low density and protein somas start being used for biochemical decomposition. Accordingly, their quality drops sharply and the process of delivery of secondary bile acids returning to the liver for the production of quality bile is disrupted. Bile, not having a sufficient amount of secondary bile acids, begins to stagnate and separates poorly, resulting in a toxic effect on the liver. The liver begins to reduce the production of somatomedin as it is a high-energy production process, this is a very complex hormone, and it is demanded in the same quantities as insulin. We see the so-called "vicious circle" developing.

The efforts of science are mainly aimed at finding more and more complex and selective biochemical agents capable of rigidly blocking the chemical cascades described above. That is to say, a search for the cause of essential AHT at the molecular level. However, the true reason is at the level of regulation, and more specifically, at the level of correct and harmonious work of the first sphere. The second sphere, in this case, already suffers from this cycle as a substrate, from which the control center extracts energy by any means, that is, our body suffers directly as a result of a prolonged disruption of energy exchange processes. Here is a general outline of the chain of immediate causes and pathophysiological links in the development of essential AHT. To stop these adaptive reactions, reverse biochemical processes, and return the body to a normal way of functioning, it is necessary to act directly on the cause. First of all, it is necessary to urgently restore the basilar blood flow and maintain it for a long time at a normal level, because according to the theory of PA (the hypothesis of CAAEBC), the organism, as a complex open and nonequilibrium system, cannot readjust at once. If the brain stem has already reorganized the regulation of biochemical processes due to a lack of oxygen, then it must make sure that we regularly replenish the oxygen level, and that this process will keep going. Only then the biochemical mechanisms of adaptation will weaken, and homeostasis will return to normal. In our practice, we apply our corrections, as a method of releasing vertebral arteries from osteochondrosis-driven spasms and compulsory exercises in the rehabilitation gym to saturate the blood with oxygen and produce additional ATP, which is necessary for the body for recovery processes.

How is it possible to prove the effectiveness of rehabilitation from the point of restoration of the informational flow to the brain with objective methods? First of all, it can be done by the analysis of the collected data on BP and VA before and after sessions. Some data is already presented in Table 1. So far after the first correction, we see an immediate improvement in blood flow. On the second step the data on biochemical and physical measurements which prove the recovery of overall well-being, strength, and endurance should be collected. It is currently on the way. The third step is to measure and monitor the pH level of the blood. We separate it from the rest of the biochemical parameters because of its instability, after the blood's separation from the body.

We hope that steps 2 and 3 will be able to reveal the right of our hypothesis to become a theory.

CONSEQUENCES OF THE HYPOTHESIS

If confirmed, this hypothesis could lead to a novel approach in AHT and related diseases treatment from the very anamnesis.

FUNDING

This paper has been supported by the RUDN University Strategic Academic Leadership Program (recipient A.A.V.).

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Cite this article

Vetcher AA, Zhukov KV, Gasparuan BA, Shishonin AY (2021) The Role of Cervical Vertebral Arteries Blood Flow in Centralized Aerobic-Anaerobic Energy Balance Compensation: When Hypothesis Becomes a Theory. Ann Cardiovasc Dis 5(1): 1027.