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Short Communication

Hypothesis Formation. Is there a Connection between the Venous Fullness of the Pelvic Organs and the Brain insufficiency?

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INTRODUCTION

First of all some introductory words, taken from the manual for trouble-free operation of machines and mechanisms with hydraulic control. Problems arising from the operation of hydraulic systems are usually caused by the following factors: violation of the oil change period, irregularity in cleaning of the hydraulic system, in replacement of filter elements, in elimination of leaks, in checking of pressure, flow rate and oil level. And now let's turn our attention to the cardiovascular system and to its large circle of blood circulation, in which arterial blood is pumped instead of oil. It works mostly like the hydraulic systems doesn't it? But there is one fundamental difference: researchers of pathologies of the cardiovascular system have overlooked the elimination of leaks and, naturally, the effect of leaks on the entire system as a whole. It is the complex effect of blood leakage that can explain many Cardio Vascular Diseases (CVD). Let's emphasize: leaks from the arterial system and, accordingly, overflow of the venous system, with a simultaneous increase in the amount of intercellular fluid. Almost all my previous works [1-16] are just related to the effect of blood leakage through large Arteria Venous Anastomoses (AVA).Based on simple logic, the meaningful meaning of AVA is an emergency discharge of high blood pressure in the system. While AVA is opened – the Blood Pressure (BP) falls rapidly, while it's closed - the pressure again rises. Thus, there are so-called "jumps of blood pressure", or syndrome of "Ermoshkina-Lukyanchenko". But sometimes, in an unhealthy way of life, the AVA's wrong movings are possible: after opening, they can close with more than necessary delays. Over time, leakage of arterial blood into the venous system through AVA can overflow the venous bed. According to the New Arrhythmia Theory, overcrowded hollow veins can serve as a conductor of mechanical impulses that suddenly begin to run many times in the same circle: the left ventricle, the aorta, the AVA, the liver, the hollow vein, the heart and trigger mechanically induced cardiomyocyte excitations or from the atria, either from the ventricles. As a result, either atrial or ventricular paroxysmal tachycardia are possible with equal QQ intervals on the ECG, respectively with narrow or wide QRS dents. The intervals are equal to adjacent intervals because the traveling times of the mechanical wave along the same vessels are almost the same. Note that under other conditions, there are only single extra systoles, or other types of arrhythmia. Uncontrolled leakage of blood from the arteries leads to damage to the venous valves, to blockage of the capillary circulation, especially in so-called gravitational traps: in the legs and in the pelvic area. With lived years with sedentary lifestyle, blood stasis in these organs can lead to big problems: to atherosclerosis and to other CVD, to systemic inflammation and cancer [11,15,16].A few words about the problem of chronic cerebrospinal venous insufficiency (English CCSVI or CCVI) in humans. It turned out, that this problem is very topical in clinical medicine in recent years. Previously, traditionally in medicine, the focus was on the arterial blood circulation, which was studied much better than the venous blood circulation. But in recent years 20-30 there has been a significant increase in the number of works devoted to violations of venous outflow from the brain. Let's consider different points of view of official medicine on the causes of chronic cerebrospinal venous insufficiency and phlebectasia. Phlebectasia is a medical name that indicates the expansion of jugular veins. For a long time it is believed that this state occurs as a consequence of violations in the work of valves and vessels. There are a number of reasons why blood cannot normally circulate through the veins, it's accumulated and stretches the vessel. There are protracted headaches. Through the valves of internal jugular veins, the greatest amount of blood is pumped, which is the basis of this system. Jugular veins are designed to drain blood from the cervical parts of the cerebral cortex towards the brachiocephalic and then to the hollow vein. Phlebectasia does not have age limits, it can appear absolutely at any age. From the point of view of official medicine, this pathology has its own causes. Here is an incomplete list of reasons: various injuries of the cortex and cervical spine; strokes, fractures of the spinal sections, collarbones and ribs; a serious disruption in the operation of venous valves; stretching the walls of blood vessels; diseases of the cardiovascular system; osteochondrosis; problems with normal blood circulation; malignant neoplasms; diseases of the endocrine system; defects in the muscles of the back; the influence of working muscles on the course of venous blood. Separately, it is necessary to note other factors negatively affecting the jugular veins: a constant and prolonged sitting in a rather uncomfortable position; genetic predisposition of blood vessels to diseases. Symptoms of the disease manifest not immediately, sometimes for several years. Signs that the neck is not in order is that there is a significant increase in the veins on the neck - the vessels on top acquire a blue color (venous stagnation), and the lower ones clearly appear under the skin (high venous pressure). It is indicated that reflux (blood flow in the opposite direction) of venous blood through the jugular veins is not always a pathology; reflux takes place when lifting the bar by weightlifters, when playing on the pipe, when coughing, sneezing, defecating, during intercourse. That's basically all that has been worked out for several decades. So, as before, there is no satisfactory answer to the main question. What are the mechanism and the dominant cause of the violation of venous outflow from the brain? What is the cause of concomitant CNS disorders? Why is there periodically a pathological venous reflux [17] in the internal jugular vein?. Most recently, since 2013, articles [18-22] appeared, in which the idea was expressed that in the pathology of increased venous pressure of the brain, in the disorders of the central nervous system, some elements may play a role in human aging extracranial venous system. It is recognized that the development of the human venous system is subject to many variations and that these variations do not necessarily represent pathological variants. The authors of this idea point out that one of the central questions that need to be further investigated is to determine the cause of a significant narrowing of the extracranial venous system with hemodynamic consequences for intracranial venous drainage. They report that the narrowing occurs with respect to the proximal adjacent segment of the veins, i.e., closer to the chest, closer to the brachiocephalic and hollow veins. In addition to what has been said, there are neuropathological observations, which are convincing arguments in relation to the "vascular origin" of Multiple Sclerosis (MS). Observations show that plaques of MS are exclusively perivenular, i.e., are located around the venules, and the dimensions of the veins determine the shape, direction and size of lesions [23]. This is also confirmed by recent studies of images, which show that most lesions in MS are associated with major veins [24]. Such data raises fundamental questions about the nature of this disease, that is, why their pathognomonic damages do not develop around the arteries and what is the role of cerebral venous inflammation in the pathogenesis of MS. Another article [25] states about the possible consequences of vascular problems and cerebral ischemia. Peri Ventricular Leukomalacia (PVL) is a form of lesions of the white matter of the cerebral hemispheres in children, discovered by morphologists, one of the causes of cerebral palsy in children. The PVL is characterized by the appearance of foci of necrosis, mainly coagulative, in the periventricular zones of the white matter of the

cerebral hemispheres in newborn infants (rarely in the stillborn). Etiologically, PVL is a hypoxic-ischemic lesion of white matter in the brain. Pathogenetic factors: hypoxia, acidosis, hypocapnia, toxins, etc. Foci of necrosis (infarcts) occur in the border zone between ventriculofugal and ventriculopetic arterial branches, localized in periventricular white matter of the brain. Around lesions of PVL, other lesions can also be detected, for example, as a "diffuse component". So, the problems of chronic cerebrospinal venous insufficiency and cerebral blood flow disorders are very serious, it was suggested that the cause of serious cerebral disorders may be an extracranial venous network, but the pathological mechanism and ways of solving this problem have not yet been determined.

A new vision of the problem (hypothesis)

Let's now consider the author's point of view on this problem. Let's see if there is an effect of open anastomoses located in the liver, intestine on the chronic cerebrospinal venous insufficiency, on the violation of venous outflow from the brain? In particular, there is a report on the existence of large AVA between the superior mesenteric artery and portal vein [26]. Assuming that long-term open AVA can raise venous pressure to a value commensurate with arterial pressure, it can be assumed that in the AVA region on the artery side, the average pressure is about 70 - 80 mm Hg, and on the side of the vein, the pressure can be about 30 - 60 mm Hg, or 410 - 820 mm H2O. Such a pressure of 41- 82 cm H2O, taking into account the losses on the conversion of part of the potential energy into kinetic energy, is in principle capable of lifting venous blood from AVA to neck level and above in some periods of time. This means that open AVA can block the circulation of the brain in a standing or sitting position, not to mention the lying position. It is known that the cardiovascular system can adapt with increasing mean and / or systolic pressures. At first the vessels simply stretch, increasing their lumen. With prolonged increase in pressure, there is an excessive crimp of the vessels, lengthening of the vessels, and an increase in the thickness of their walls. Then the growth of anastomoses and collaterals and an increase in the volume of blood vessels begin. Downstream, narrowing of the main vessels may occur. The narrowed sections of the vessels begin to strengthen, i.e., there is their sclerosis, increased stiffness. Open AVA and increased pressure in the vena cava are forced to increase the stiffness of the vein walls near the right atrium and to narrow the right atrioventricular orifice. The purpose of narrowing the jugular and other veins of the neck is to counteract the increase in the limiting venous pressure that propagates from the bottom upward, in order to preserve the performance of the smallest tender vessels that ensure the removal of the products of vital activity of the brain cells. Apparently, it is necessary to agree that these simple rules are valid both for the arterial and for the venous system. Constriction of the vessel reduces the kinetic energy of the blood, reduces blood pressure. The constriction protects the organs located "in front" downstream from overloads. "Ahead" for the physiologically correct course, but for the pathological course "in front" is in the opposite direction. But if this is so, open large AVA located near the liver or elsewhere can lead to a pathological increase in pressure in the hollow veins and then in all veins (including the functioning of the valves), including in the jugular veins. A particularly significant increase in venous pressure and a slowing of the capillary circulation in the brain can occur in the lying position, for example when sleeping. So, in the jugular veins, the blood should flow towards the hollow veins from top to bottom, but in fact it can flow backwards to the head for some periods of time. The ultimate possible point of the pathological movement of the blood, or rather of the spread of increased pressure – are the small delicate veins, brain venules, neurons. That is why the central nervous system beforehand develops protection against pressure: narrowing of large veins on the approaches to the venules of the brain. The ultimate case of effective protection: "corrugated vein". Such veins, for example, can be observed in the esophagus or larynx. And all these pathological changes are the result of the action of open AVA. If all types of protection do not help, then due to a further increase in venous pressure there is a blockage, a stasis of capillary circulation, seepage of venous blood into the intercellular space, edema. Pathological changes begin in the most vulnerable places of the venous system of the head, so it is in the venules of the brain that micro thrombi, plaques, necrosis, and cicatricle changes in surrounding tissues are formed. In fact, there is a picture of pathologies, symmetrical picture of the destruction of venules in organs in the pelvic area and legs. Differences in pathological changes in the blood circulation of the brain and neck from blood circulation pathologies of the lower half of the body, as a rule, only in the later time of the onset of diseases due to the action of gravity forces. Those organs that are located above the heart are more safe, the pathology of venous plethora develops later. Studying problems of venous insufficiency of the brain, I managed to find a direct confirmation of the proposed hypothesis of the onset of chronic cerebrospinal venous insufficiency, the role of open AVA anastomoses. The article [27] states: "The fact that venous insufficiency can cause acute neurologic disorders has been convincingly demonstrated in the report on a patient with an artificial arteriovenous shunt. This patient had growing headaches, gait disturbance and cognitive dysfunction. The patient's condition improved significantly after the elimination of this shunt (type AVA). There are other confirmations of the New Hypothesis. In my opinion, Vegetative Vascular Dystonia (VVD) is a disease that characterizes the first stage of the onset of vascular changes with the periodic overflow of the hollow veins due to open AVA, this is the stage of adjusting many vessels to counter the spread of increased venous pressure upwards, into the brain, this is the first stage of cerebrospinal venous insufficiency. Here is the usual point of view of doctors involved in VVD [28]. "Unfortunately, the problems of prevention and treatment of VVD often become relevant not at the appearance of the first symptoms, but already in connection with the questions of how to treat an impaired cerebral circulatory insufficiency (up to a stroke) and neurocardiological disorders leading to disability. It is difficult to single out a separate target organ of therapy, responsible for the clinical picture in such a disease as VVD: treatment is aimed at removing the complex of manifestations in a number of systems. "One can conclude: the cause of the VSD medicine is still unknown.

CONCLUSION

So, a plausible, logically grounded hypothesis partially confirmed in the experiment was stated that the main reason for chronic cerebrospinal venous insufficiency, venous congestion of the brain, venous reflux, pathological expansion of the jugular veins, cerebral circulatory disorders, VVD, disorders of certain functions of the central nervous system may be one and the same mechanism, the same underlying cause: the presence of relatively "large" periodically open Arterio Venous Anastomoses (AVA) that are in the extract the venous network, i.e. in the zone outside the brain. The work of large AVA is hidden from the eyes of doctors, basically it is also hidden from the eyes and sensations of patients. Only the most sensitive people can sometimes notice unusual pulsations in the abdomen, in the region of the liver, after which, due to overflow of hollow (cava) veins, naturally,

cardiac extra systoles are possible. I think that in the near future there will be a rethinking of the causes of many cardiovascular diseases and brain diseases, a serious study of the functioning of anastomoses, the study of measures to counter the pathological effect of abnormal AVA, the development of "smart" artificial arteriovenous shunts with a controlled lumen. All this is necessary to preserve human health and increase the duration of active life. An old proverb is confirmed: an old man is as old as his vessels. And the vessels are aging, as we see from the presented hypothesis, because of the abnormally working AVA anastomoses, which themselves are vessels that connect arteries and veins directly. For its part, the growth and discovery of AVA, apparently, are due to stress, hypodynamia, because of alcohol abuse, because of unhealthy lifestyles, because of poor heredity, etc. These are open AVA that lead to many diseases, including some diseases of the brain, cardiovascular and central nervous systems.

REFERENCES

1. Ermoshkin VI. The new theory of heart failure. London. 15-17. 2017. https://goo.gl/CKpWsP
2. Ermoshkin VI. New theory of arrhythmia. Conceptual substantiation of arrhythmia mechanisms. Cardiometry. 2016; 8: 6-17. https://goo.gl/dKQHGQ
3. Ermoshkin VI. The mechanism of bronchial asthma. Why do the most serious asthma attacks occur at night?. EC Cardiology. 2016; 2. https://goo.gl/hxz5BZ
4. Ermoshkin VI. Arteriovenous anastomoses and cardiovascular diseases. 8th Cardiovascular Nursing & Nurse Practitioners Meeting. 2016 Las Vegas, USA. https://goo.gl/bNHKPM I
5. Ermoshkin VI. A new theory of certain cardiovascular diseases. EC Cardiology. 2016; 2. https://goo.gl/3u8qWW
6. ErmoshkinVI. Venous congestion due to large arteriovenous anastomoses. 566 Chiswick High Road, London, Greater London, W4 5YA, United Kingdom. https://goo.gl/DjsepA
7. Ermoshkin V. The cause of some cancers because of the open arteriovenous anastomoses. J Gastrointestinal Cancer Stromal Tumor. 2017; 2. https://goo.gl/F3tzBp
8. Ermoshkin VI. Problems of heart failure. Unexpected outcome. London. 2017; 15-17. https://goo.gl/C63jKX
9. Ermoshkin VI. Arrhythmia and Cardiac Surgery. 2016. Brisbane, Australia. https://goo.gl/pwyW4z
10. Ермошкин ВИ. НОВАЯ ГИПОТЕЗА АРИТМИИ СЕРДЦА У ЧЕЛОВЕКА. Стр 73. «Кардиостим-2016», Санкт-Петербург. 2016 ; 18 – 20.
11. Ermoshkin VI. The new theory of cancer complements ancient Chinese Qigong therapy. 2017. https://goo.gl/NDBkdL
12. Ermoshkin VI. Heart transplantation mysteriously eliminates arrhythmia. Cardiometry. 2016; 8: 18–21. https://goo.gl/geBAMe
13. Ermoshkin Vladimir. Commercial offer for cardiac centers and potential investors. 2017. https://goo.gl/iwvUAB
14. Ermoshkin VI. Pathological Role of the «Invisible» Anastomoses. J Bioengineer & Biomedical Sci. 2016; 6: 209. https://goo.gl/UW81yi
15. Vladimir Ermoshkin. Numerous studies show: not an excess of cholesterol, but first of all a systemic inflammation is the real reason of many cardiovascular diseases and of sudden death. Int J Car & Hear Heal. 2017; 1: 2: 22-24.
16. Vladimir Ermoshkin. The pathological mechanism of systemic inflammation in humans has opened. The reason is uncontrolled leakage of arterial blood through arteriovenous anastomoses. Int J Car & Hear Heal. 20-17; 1:2: 12-14.
17. Zamboni P, Galeotti R, Menegatti E, Malagoni AM, Tacconi G, Dall'Ara S, Bartolomei I, Salvi F. Chronic cerebrospinal venous insufficiency in patients with multiple sclerosis. J Neurol Neurosurg Psychiatry. 2009; 80: 392-399.
18. Magnano C, Belov P, Krawiecki J, Hagemeier J, Beggs C, Zivadinov R. Internal Jugular Vein Cross-Sectional Area Enlargement Is Associated with Aging in Healthy Individuals. PLoS ONE. 2016; 11: 0149532. https://goo.gl/Ej9prS
19. Zamboni P, Menegatti E, Galeotti R, Malagoni AM, Tacconi G, Dall'Ara S, et al. The value of cerebral Doppler venous haemodynamics in the assessment of multiple sclerosis. J Neurol Sci. 2009; 282: 21-7. https://goo.gl/rDTq3o
20. Zivadinov R, Marr K, Cutter G, Ramanathan M, Benedict RH, Kennedy C, et al. Prevalence, sensitivity, and specificity of chronic cerebrospinal venous insufficiency in MS. Neurology. 2011; 77: 138-44. https://goo.gl/z1jXvu
21. Zivadinov R. Is there a link between the extracranial venous system and central nervous system pathology?. BMC Medicine. 2013; 11: 259. https://goo.gl/Hy7cx4
22. Zivadinov R, Chung CP. Potential involvement of the extracranial venous system in central nervous system disorders and aging. BMC Medicine. 2013; 11: 260. https://goo.gl/rmgDMw
23. Adams CW. Perivascular iron deposition and other vascular damage in multiple sclerosis. J Neurol Neurosurg Psychiatry. 1988; 51: 260-265. https://goo.gl/rTTVkD
24. Tallantyre EC, Brookes MJ, Dixon JE, Morgan PS, Evangelou N, Morris PG. Demonstrating the perivascular distribution of MS lesions in vivo with 7-Tesla MRI.Neurology. 2008;70:2076-2078. https://goo.gl/Hnz993
25. Власюк В. В. Перивентрикулярная лейкомаляция у детей. — СПб.: Гликон Плюс, 2009. - 218 с. (Russian) ISBN 978-5-93682-540-8.
26. Internet resource. Clinical case. Russian. https://goo.gl/zJ494J
27. Salvadore JAS, Michael DD, Barry TK. Chronic Cerebrospinal Venous Insufficiency. A new paradigm and therapy for multiple sclerosis. 2010. https://goo.gl/DYggHp
28. Internet resource (Russian). http://venoz.ru/category/vsd