Open Problem: How to Compel Arteriovenous Anastomoses Work Normally?

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The main problem facing modern medicine is the high mortality of people from cardiovascular diseases (CVD).

Another difficult problem is kidney injury. As I am a physicist by training, I solved the problem differently and found that arrhythmia, heart failure, metabolic syndrome, chronic kidney injuries and others are interrelated [1-4].

I have spent over 6 years for discovering these connections. In fact, I was able to prove a new mechanism of cardiac arrhythmia and the new mechanism of most CVD.

Initially, I preceded from the fact arterial vessels need valves to relieve peak blood pressure values for safe operation. The physical and psychological load people face is huge, and blood vessels need to be able to adapt to rapid changes. When arteriovenous anastomoses (AVAs) are open, a sharp drop in arterial blood pressure occurs, when the AVAs are close within 2-5 seconds, the blood pressure increases again. This is because of the notorious jumps in blood pressure, named later syndrome “Ermoshkin-Lukyanchenko”. Jumps of arterial pressure are detected in many people, but the real reason for this no one knew.

Open AVAs leads to a blood transfusion from the arteries into the veins and then to an excessive influx to the vena cava. According to the New Theory of Arrhythmia, the overcrowded hollow veins (vena cava) can lead to the fullness of the liver and spleen. In addition, overcrowded hollow veins can serve as a “condutor” of mechanical impulses that suddenly begin to run many times in the same circle: the left ventricle, the aorta, artery, AVA, the liver, vein, the hollow vein, the heart and trigger the mechano-induced excitation of the cardiomyocytes either from the atria or from the ventricles. As a result, either atrial or ventricular paroxysmal tachycardias are possible with equal QP intervals on the ECG, respectively with narrow or wide QRS waves. Intervals are equal to each other because the traveling times of a 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. Further study of the effect of open AVA or AVA with a violation of their functionality showed that these small connective vessels may be involved not only in arrhythmia, but in other diseases: diseases of pelvic organs, metabolic disorders, systemic inflammation, brain's venous failure, heart failure and many others.

So, let is consider the mechanism of chronic and acute kidney injury. It is believed that the mechanism of kidney injury is currently unknown. The AKI is a state that results from a disorder of the kidney blood flow, damage of the membranes of the nephron, sudden obstruction of the ureters, while the levels of urea and creatinine in blood plasma are rapidly increasing. The CKI are the same disorders that pass a little noticeably and at a much slower pace.

Annually about 150 people out of 1 million need urgent help for acute renal insufficiency. As a rule, two-thirds of them need hemodialysis and hemosorption. Even against the background of treatment, mortality in all forms of acute renal failure reaches 20-50%. The usual recommendation of doctors: we must seek and treat the underlying disease, then treat renal failure. But there is a new theory, a new mechanism for the emergence of cardiovascular and other human diseases is proposed.

The present article proposes the selection and consideration of arguments in favor of the new mechanism of renal failure in the form of questions and my new answers.

1) Renal insufficiency can be acute and chronic. Why?

**Cause:** If, after the opening of the large AVA, venous pressure increases in the entire venous pool and increases rapidly in the renal vein, the AKI may occur. If the leakage of arterial blood through AVA is small, but prolonged with periods of remission, then the CKI arises. The open AVA creates an increased venous pressure in large veins. This pressure, spreading through the renal veins, reaches the renal venules, partially or completely blocks the capillary circulation in the kidneys. There is a violation of the formation of urine, necrosis of kidney cells and KI is gradually developing.

2) In renal failure, the pressure in the renal veins, as well as in other veins of the great circle of blood circulation increases. In this connection, the question arises whether renal venous hypertension is associated with arises venous hypertension other organs?

**Cause:** Yes, venous pressure increases not only in the renal veins, but also in those organs in which the venous valves are either broken or these organs are subject to additional hydrostatic pressure, for example, in pelvic area and legs. There is venous plethora.

3) The AKI often occurs in patients in hospital who have already been hospitalized with severe illness or injury. The hospital mortality rate may exceed 50%.

**Cause:** In the supine position with open AVA, venous blood with increased pressure can freely flow into any organ. This blood can flow into the organs of the middle and upper parts of the body, including the kidneys and renal veins, especially because there are no valves in these veins. Suddenly, due to a decrease in the pressure gradient between the arteries and venules, blockage of capillary circulation in the kidneys and death of the patient may occur.

4) Usually, with the development of renal insufficiency (and with some other diseases too), doctors say: it is necessary to treat a “basic” disease.

**Cause:** This is a cunning medical step due to a lack of understanding of
the mechanism of almost all cardiovascular diseases (CVD). Doctors are looking for a “basic” disease and often “find” it. From the point of view of the New Theory of CVD, such a move can be justified by the fact that open AVAs are the cause of not just one single CVD, but many diseases at once!

5) First place in the frequency of development of the AKI occupy severe somatic (shock) conditions, accompanied by a decrease in cardiac output.

**Cause:** With open AVA, blood flows freely from the arterial basin into the venous pool. This leads to an imbalance in the volume of arterial and venous blood. And with a decrease in the volume of arterial blood, i.e. with hypovolemia, naturally, cardiac output is reduced.

The AKI and CKI are characterized by a slowing of blood flow in the kidneys, and the slowdown, according to medical observations due to significant blood loss, for example, during surgery or injury.

**Cause:** Not only in operations and injuries, arterial blood loss occurs. With open AVA, the blood flows freely from the arterial basin into the venous pool. This is also a loss of blood, or hypovolemia. And the hypovolemia is the cause of acute and chronic KI.

6) **The criterion for the presence of the KI is the total volume of urine released by the body, for example, per day.**

**Cause:** With open AVA the blood flows from the arterial basin to the venous pool. This can lead to an increase in venous pressure in the kidneys. There is a blockage of blood circulation. The excretion of urine in patients in comparison with healthy people, naturally, decreases.

7) As the liquids and the final metabolic products formed in the body accumulate in the blood, the acute renal insufficiency can lead to serious consequences, including the development of infection (sepsis).

**Cause:** In addition to what has been said, the cause of sepsis and systemic inflammation can be not only the kidneys in which necrosis due to stasis occurs, but also other organs that have been previously more affected by the pathological action of open AVA anastomoses, for example the organs of pelvic area or liver.

8) **The AKI may occur with malfunctions heart rhythm disturbances.**

**Cause:** There is such a connection as the only cause and mechanism of AKI and malfunctions cardiac rhythm disturbances are open AVA. The AKI occurs due to overflow of renal veins, and arrhythmia - because of the run of mechanical impulses over congested hollow veins.

**9)** Diuretics have no effect on prognosis and mortality from AKI.

**Cause:** According to the New Theory, diuretics can either worsen a patient's condition, or at best have no effect. The fact is that with open AVA there is either a constant or periodic leakage of arterial blood from the arterial pool into the venous bed. There may be acute arterial insufficiency.

10) Sometimes AKI develops with bilateral thrombosis of the renal arteries or veins.

**Cause:** When capillary renal circulation is blocked due to open AVA, the pressure gradient between arterioles and venules becomes critically small. The blood movement near the kidneys stops both in the arterial channel and in the venous vessels. Over time, there is stasis and thrombosis.

11) Sometimes there is an increase in venous pressure, there is a lack of capillary circulation (stasis and ischemia) in some organs, there is cardiac and / or renal insufficiency, but the arterial cardiac output is normal. **Why?**

**Cause:** Because a part of the blood is not sent to the organs, but passes directly to the large veins through the AVA. This is how partially "useless" heart work is carried out. That is why cardiac output can be normal, and capillary circulation in some organs is impaired.

**Conclusion**

**My opinion:** we cannot hush up the proposed theory. The mortality from CVD and cancer is very high and tends to increase. Modern treatment of almost all CVD is symptomatic. The results are weak. There is no single theory of CVD at all. This task is simply not solved, although, at least for more than 50-100 years, the CVD problem has been one of the major medicine problems.

Existing provisions in cardiology, methods of treatment, medicines-everything needs to be reviewed. If many human diseases are caused by open arteriovenous AVA anastomoses, then AVA should be treated.

An additional way of solving the problem is possible: to need to develop artificial AVA with adjustable diameter and to carry out the installation of AVA according to indications.

Who from serious medical structures will risk supporting the proposed New Theory and developing it? I have been waiting for more than 6 years. I'm waiting for the reaction from the leaders of cardiology right now. Sorry if I offended someone.

**Reminder:** Stand up from computer seats for exercises every 30 minutes!

**References**