The Mechanism of Bronchial Asthma. Why Do the Most Serious Asthma Attacks Occur at Night?

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Objective

An attempt to study the mechanism of arrhythmia and other cardiovascular disease (CVD) in humans.

Methods

On the basis of new assumptions about the role of abnormal arteriovenous anastomoses (AVA) [1], taking into account the new theory of arrhythmia [2], which I persistently move forward from 2011, opened were some prospects in unraveling the secrets of cardiovascular disease. I participated several times in medical conferences at the People’s Friendship University (Moscow) [3], in S-Petersburg (Cardio-stim-2016) [4], in Brisbane (Arrhythmia-2016) [5]. I had several discussions with the famous cardiologists.

Results

The populations of many countries feel that a standstill in all theoretical cardiology was formed in the last few decades. The mortality caused by cardiovascular diseases in most developed countries is unacceptably high.

There is still a lot of unknown or unproven in the causes of chronic obstructive pulmonary disease (COPD), in understanding the causes of bronchial asthma (BA).

There are many hypotheses, but they are only partially supported by some experts [6,7,8]. Effective treatment does still not exist. It is believed that these diseases with an unknown etiology are incurable. This situation happens because the true mechanism remains unknown for doctors and patients.

There are a number of clinical signs and effects of the presence of asthma in humans. But the official medical studies consider such causes as inflammation of the respiratory tract, disorders of circadian rhythms in contact with allergens and others. Standstill is formed for many decades! And medical practice confirms this.

What shall we do? I do not agree with it.

I developed “New Theory of arrhythmias and cardiovascular disease” in 2011 and continue developing it today. Maybe somebody would pay attention to it? As the author of this new theory, I see that the interest in research and hypotheses is increasing every year day by day.

On the basis of the new theory, it turns out that almost all the cardiovascular diseases (CVD) with unknown etiology are caused by anastomoses AVA (maybe groups of AVA), which increase the venous pressure on the system and local levels and block the capillary circulation. Generally speaking, in a healthy person the AVAs can be opened only under significant physical or psychological stress in order to

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sharply decrease the peak arterial pressure. The main difference between healthy and unhealthy person is a process of closing the AVA, it closes in a healthy person completely and in time.

Let me suppose, furthermore, that in all the organs, portal system, liver, spleen and other places just the own heart increases the detrimental for a human health venous pressure. In all the organs, where venous pressure is increased, the own heart is “guilty” in it! Only the main?? heart with the use of AVA can increase the pressure in veins beyond the norm, and no other more powerful mechanism exists! Apparently, a person can be considered as healthy only until any direct uncontrolled leakage of arterial blood into the veins or intercellular fluid occurs. Otherwise, at least the weight gain and edema appear.

Official medicine mistakenly believes that the venous pressure may increase due to the presence of some “obstacles” such as a blood clot or a tumor. But the “obstacle” is a passive element, it has to increase the pressure in the veins for a short time, it cannot do it for a long time. The pressure increase in veins and their extension occur only due to their direct communication with the arteries. These phenomena are proven using the Cardio code device [9].

Open and not closing in time AVAs lead to long-term discharge of arterial blood into the veins. Capacitive veins increase their volume and pressure. Vena cava is significantly expanded. Depending on the body position the blood may retrogradely enter the venous system of any organ from the vena cava. Mixed blood goes backwards to the small veins and venules trying to stay as low as possible, i.e. firstly it goes to the lower extremities. Edema appear, first on legs, close to feet, then higher and higher. Over the years, taking into account the modern lifestyle and hypodynamia, edema cannot “disappear” during sleep in a horizontal position, i.e. 8 hours.

Over the years, edemata occur in the pelvic area (say goodbye to a healthy sex!), then in the area of liver and spleen (say goodbye to merry feasts with alcohol!), then ascites appear (say goodbye to brisk walking and jogging!), and finally an increased level of venous pressure reaches the lungs (that is really serious!).

Of course, the sequence of edemata and stagnations varies from person to person, but the following rule remains: the direction is bottom-up. The mechanism and sequence of edemata, starting from feet and finishing with lungs, is the same. According to the conventional medicine terminology, most often the right-sided heart failure appears first, then the left-sided one.

It is interesting that both failures sometimes occur in a healthy heart. And it becomes clear due to the new theory.

In this article we do not speak about the circulatory system of the head and conditions of cerebral edema, as there are big differences in hydrostatics. This is a topic for another article. But the mechanism of edemata is the same, they are caused by opened AVAs!

Let me emphasize that lungs have a mixed circulatory system. Vessels of large and small circles of blood are located parallel to each other in the lung tissues. And this feature is a key to understanding the mechanism of BA.

When edema in the lower half of the body occur, the next free volume, where the excess venous blood can be placed, is the heart, lungs and upper extremities tissue. In the daytime the venous pressure is high in patients with pathologically opened AVAs in a standing or sitting position, but most often it is insufficient for the pulmonary edema formation. But when a patient takes a horizontal position the physical conditions for pulmonary edema may occur.

Due to the opened AVAs the venous blood with high pressure surges into the small venules and veins of the systemic circulation. As the pressure exceeds the threshold values, the fluid penetrates into the intercellular space. In the standing and sitting positions it hardly surges to the lungs, as they are located higher, on the same level with the heart, thus all this dirty mixture moves mainly to the legs due to gravity.

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But when a person is in a lying position the blood does not move to the legs, as in the horizontal position the venous pressure is almost everywhere the same, so the venous blood spreads in all directions, both to the head and legs. It can reach the lung tissue and cause edema, the same edema as in all organs below the diaphragm. The mechanism is the same. Then the excess intercellular fluid with increased pressure probably not immediately, but still penetrates into the alveoli, which belong to a small circle of circulation. Therefore the CO₂ and O₂ exchange is disordered in a person with BA, he begins to suffocate and choke with phlegm! The phlegm (fluid) is an excessive blood plasma passed through the opened AVAs along large veins to the lungs and then to alveoli.

Recently, appeared one more indirect proof of the plausibility of the theory proposed by me. To know more about testing of the “revolutionary” method of struggle against hypertension read the reports [10,11]. The device ROX AV Coupler was used in the testing, it is developed by the American company ROX Medical. Tests were carried out in England in 2014-2015. This device is essentially an artificial implant (arteriovenous shunt) with a 4 mm hole diameter and a continuous throughput of about 800 ml/min. The shunt was placed between the iliac artery and the adjacent vein. Tests were conducted on hypertensive patients resistant to treatment drugs. Due to this method of treatment of resistant forms of hypertension it was a success to show that a reduction in systemic blood pressure by an average of 27/20 mm Hg really occurred. Unfortunately, in 2-9 months over the distal shunt in 29% of cases appears a stagnation of venous blood and edemata, which are the same as in a natural permanently opened anastomosis [3,5]. One may ask why in the open natural arteriovenous anastomoses arrhythmias occur [2,3], and in permanently opened ROX-shunt do not, but on the contrary, as stated by researchers, the implant method contributes to treatment of not only arrhythmias, but some other CVDs [12].

In my opinion, it happens because the implant is made of solid material, which is significantly different from natural tissue of anastomoses, so the mechanical waves traveling along the walls of arteries are effectively reflected from foreign material and do not hit the vena cava wall. Thus, ROX Coupler reliably eliminates the cause of arrhythmias due to the impact of pulse waves. The ROX Coupler implant, struggling against the effects of hypertension, reduces the blood pressure and thereby eliminate the reason for the opening and closing of natural AVA. We can assume that after the installation of ROX-shunt the large natural AVA are forced to be in the closed state permanently, and the heart, lungs and some large vessels have to work with the increased load and it is useless to pump some extra volume of blood from major arteries into the veins. This excess value of pumping is 800 ml/min or 16% of the norm in 5000 ml/min. In my opinion, this solution is not optimal. It is obvious that for hypertensive patient in the future it is needed to develop the implants with a variable diameter of shunt lumen, down to zero value of lumen, depending on the current blood pressure in the arteries. But it is still better to “use” the natural anastomoses given to us by nature, and for that it is necessary to lead a healthy lifestyle with daily moderate physical loads on muscles and cardiovascular system. Another confirmation of the fact that the mechanical impulses is suppressed heterogeneity of tissue or a surgical suture is statistics on the results of greater efficiency bicaval (compared to biatrial) technique in heart transplant [13].

Conclusions

1. This theory should be still seen as a promising hypothesis, because it is not generally accepted yet. I believe and hope that the cause of many cardiovascular diseases with unknown etiology becomes clearer. At least the cause of heart premature beats and paroxysmal tachycardias looks quite reasonable in the framework of my new theory.

2. In accordance with the proposed new theory the methods of diagnostics and treatment of bronchial asthma and some other lung diseases should be reviewed.

3. A key element of the forthcoming research and experiments is a study of performance of the large arteriovenous anastomoses (AVAs) located mainly near the liver, portal and hepatic veins, liver and mesenteric arteries, vena cava. The key to treatment is apparently a thin adjustment of the AVAs performance, detection and tracking of the retrograde venous blood movement.

4. It is natural that a physical activity throughout the day, adequate sleeping time, special breathing exercises for involving the stagnant venous blood into the systemic circulation may be advised to prevent the cardiovascular disease.

5. The author is ready to cooperate with all interested parties.

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Bibliography


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