Research Article

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2025.62.009747

Hypothesis of the Mechanism of Hypertensive Crises of the Systemic and Pulmonary Circulations

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ARTICLE INFO

Received: June 15, 2025

Published: June 27, 2025

Citation: Vladimir Ivanovich Ermoshkin. Hypothesis of the Mechanism of Hypertensive Crises of the Systemic and Pulmonary Circulations. Biomed J Sci & Tech Res 62(3)-2025. BJSTR. MS.ID.009747.

ABSTRACT

The article is devoted to a very important topic in medicine: the cause of blood pressure (BP) surges and hypertensive crises in the systemic and pulmonary circulation, the role of atherosclerosis. An answer is given to the question: why are crises in the systemic circulation always sudden?

Keywords: Hypertensive Crises; Atherosclerosis; Arteriovenous Anastomoses; Blood Stasis; Breathing Exercises; Diaphragmatic Breathing

Objective

To find and substantiate the causes and mechanism of hypertensive crises, based on the known evidence of the cause of atherosclerosis [1-3]. From the mouths of the "giants" of medicine and ordinary cardiologists it is known (without evidence) that atherosclerosis is the cause of all cardiovascular diseases. The prediction is probably correct, but what is the mechanism, what is primary, what is secondary? Doctors do not yet have a single and correct opinion.

Results

First, let us remind the reader what is known about the causes of hypertensive crisis of the systemic circulation (SC) and pulmonary circulation (PC), what guidelines official medicine has made for itself and for patients (see points A and B).

A. The main reason responsible for the occurrence of hypertensive crises of the systemic circulation is "hypertension". However, crises can also be observed in secondary hypertension, infectious diseases, ischemic heart disease, kidney disease, traumatic brain injury, strokes, hyperthyroidism, alcoholism, hormonal imbalance, aortic atherosclerosis, diabetes mellitus, systemic lupus erythematosus, and nephropathy of pregnancy. Crises can be caused by: excessive physical exertion, stress factors, use of medications that increase blood pressure.

B. In cardiology practice, pulmonary edema and crises can complicate various diseases of the cardiovascular system: atherosclerotic and postinfarction cardiosclerosis, acute myocardial infarction, infective endocarditis, arrhythmia, hypertension, heart failure, aortitis, cardiomyopathy, myocarditis, atrial myxomas. Pulmonary edema often develops against the background of congenital and acquired heart defects - aortic insufficiency, mitral stenosis, aneurysm, coarctation of the aorta, patent ductus arteriosus, septal defects, etc.

And now let's move on to the author's opinion. It can be considered that medicine has not been able to find the correct and only cause of hypertension and atherosclerosis for many decades. In the author's article "Proof of the Cause and Mechanism of Atherosclerosis, "standing on the shoulders of the giants of medicine" [1,2] the proof itself is given, but there are no responses or criticism from the leaders of medicine yet. But there are many additional requests from various countries addressed to me for the publication of similar materials in

medical journals. Generally speaking, there are no articles with any theoretical evidence in medicine, or there are very few of them (the author did not find any). Medicine has "evidence-based medicine", but this is not the same thing. "Evidence-based medicine" is based on statistical confirmation that a given new drug or intervention should have a certain positive effect. In practice, it is customary to make major decisions by "voting" of a very narrow circle of people close to the "top". Any new recommendation on the interpretation and method of treatment very often has 10-30 authors! This is very similar to mutual responsibility. So, the true cause of many diseases and, most importantly, atherosclerosis is a periodically occurring imbalance in the volumes of arterial and venous blood due to uncontrolled leaks of arterial blood into the veins during stress [4].

It turns out that the main link in the problem is short-term critical losses in arterial blood volume, relative to some average volume for each individual. The normal balance of arterial and venous blood is approximately from 15:85 to 20:80. But during stress and increases in blood pressure, arteriovenous anastomoses can open, and uncontrolled leaks of blood from the arterial pool occur. For example, from the superior mesenteric artery to the portal vein [5]. Seconds after opening, the anastomoses usually close. And this can happen several times. Medicine has long known about this phenomenon, but apparently did not attach importance to it. Thus, the main link in the problems of hypertension and atherosclerosis of arteries near and above the heart was found in 2020-2024. The reports on this are in the articles [1-4]. The reason is the mysterious physical forces of endothelial detachment from the muscular layer of the arterial walls.

The localization of these concentrated detachment forces and the growth of the first "plaques" are always in favorite (obviously non-random, but known in advance) places, where physical forces act and concentrate, aimed at compression (reduction) the lumen of the aorta or arteries. Compression forces are not produced by the muscular layer of the arteries or the elastic layer of the aorta, these forces act from inside the arteries and can lead to dysfunction of the endothelium, the forces act directly from the flow, the reason is the lack of arterial blood volume in the system. Mysterious forces arise at the "points" where these forces are maximum with a vertical spine. And these forces are located in the upper part of the cardiovascular system, near the myocardium. This includes the aortic arch, coronary arteries, the bifurcation zone of the carotid arteries, the aortic valve, and even the endothelium inside the left ventricle. It is here that the first plaques most often grow, their calcification, and the degree of atherosclerosis increases. This is how heart failure gradually develops. As heart failure develops, human health steadily deteriorates. Simultaneously with the emergence of forces of endothelial detachment and stretching of the middle layer of arteries in the same layer [1], as compensation for stretching, suction forces arise that force LDL and any other blood fragments from the flow to penetrate through the intima into the muscle layer, giving rise to endothelial dysfunction, atherosclerosis, and plaques. On the other hand, the cause of atherosclerosis of arteries and plaques located significantly below the heart have their own peculiarity - they usually occur later, after the first signs of the formation of heart failure.

In addition, endothelial damage in the leg arteries has more extensive atherosclerosis zones, there is no concentration of physical tearing forces. Here, endothelial dysfunction and atherosclerosis occur for another reason: due to slowing down of blood flow, increased pressure in the veins and venules of the lower half of the body, damage to the venous valves. As a result, plasma can leak into the intercellular space, weight gain, edema, varicose veins, vasodilation, thrombosis, delayed glucose turnover, which can lead to type 2 diabetes due to the high level of glycated hemoglobin HbA1C.Thus, the physics of atherosclerosis in large arteries near the heart indicates the negative role of pulsed pressure affecting the walls of the arteries, especially during diastole. And in the lower half of the body, on the contrary, positive excess pressure acts and the main factor is congestion, leading to thrombosis of both veins and arteries. And congestion in turn gradually increases due to blood leaks through anastomoses during periods of stress, especially during physical inactivity.

It can be considered that the main global mystery of medicine, the cause and mechanism of atherosclerosis of the arteries, has been solved: due to periodically occurring imbalances in the volumes of arterial and venous blood. According to researchers, on average, about 15-20% of the total blood volume is in the arteries [3], i.e. under high pressure, and about 85-80% is in the veins under low pressure. The sum of these volumes is called the volume of total circulating blood; this volume can lie in a wide range: from 4 to 6 liters or more. A simple estimate shows that the volume of arterial blood in humans is still not very large, but at the same time, it should be almost constant. This is the main parameter of the biology of a healthy person, for each individual this volume has its own average value with some deviations from it in emergency cases. This parameter (the value of arterial blood volume) is in the range of 600-900 ml, and depends on the size of the person, the training of his muscles, commitment to a healthy lifestyle and, apparently, has some weak dependence on age. This value has not yet been studied, being a blank spot in medicine. Blood under high pressure is cyclically ejected into the aorta, which is accompanied by pulse waves in all arteries. The stroke volume of blood in one ejection is from 60-70 ml to 120-190 ml in men and from 40-50 ml to 90-150 ml in women [6]. These estimates are true for both the small and large circles of blood circulation. Thus, in a person in a calm state, during one period between contractions of the heart, the volume of arterial blood of the large circle can increase (and simultaneously decrease in arterioles and capillaries) by approximately 10% or more, i.e. by a significant amount. On the other hand, in the veins of the large circle, with each stroke, replenishment (and expenditure) occurs by only 1-1.5%. Feel the difference! And all this is subject to Harvey's law on the equality of "ejection volumes". Monitoring the

constant (on average) volume of arterial blood occurs at the organism level, a person does not think about it. But in vain, breathing exercises for 2-3 minutes every, say, 2 hours of wakefulness with an emphasis on sharp and consecutive 5-10 exhalations with sharp rises of the diaphragm (and with pauses between series of 10-15 seconds) can regularly replenish, possibly, the lost volume due to stress. Daily moderate physical activity is also useful, and physical activity does not happen without breathing exercises! According to many well-known statements of doctors, during periods of stress, anxiety, stress, a person's blood pressure (BP) rises due to psychological problems.

At the same time, the content of stress hormones in the blood increases, such as cortisol, adrenaline, etc. Due to frequent increases in blood pressure, sooner or later large arteriovenous anastomoses may open in a person for an emergency decrease in blood pressure at a local level (for example, between the mesenteric artery and the portal vein) [6], while blood from the arteries may flow into the veins, leaving some of the working cells without sufficient nutrition. Fluctuations in blood pressure, blood volumes, and hormones in a person can last for decades - this is human nature, and a person can remain practically healthy for a long time. It is appropriate to recall here that athletes' blood pressure can rise up to 300/160 mmHg. But in cases of frequent and significant leaks of arterial blood into the veins, mainly due to stress, especially with a sedentary lifestyle, naturally, critical deficiencies in the total volume of arterial blood and excess venous blood are created.

What do these deficiencies in the volume of arterial blood and excess venous blood in the systemic circulation lead to? Answer: long periods when the heart works with a lack of arterial blood gradually lead to the growth of endothelial damage zones in the arteries near the heart, and then to the growth of atherosclerosis and plaques [6], and over the years to heart failure! Gradually, there is a "strengthening" of the walls of the aortic arch and aortic valve (calcification), since with a lack of blood, mysterious physical (mechanical) forces act to tear the endothelium from the muscular layers of the arteries [7]. By the way, these mysterious forces continue to act even with already formed plaques, and the tearing forces are concentrated precisely on the tops of the plaques, causing repeated destruction there. Installing a stent in the plaque area can only help for a while, because physical forces do not disappear anywhere: a new plaque may form next to the stent. That is why calcification (strengthening of the surface) of plaques is so important for arteries! After all, if the artery does not heal, then repeated tearing of the upper (former endothelial) layer can lead to the formation of a large or small thrombus, which, in turn, can lead to a stroke or heart attack. This is the physics of first harmless damage, and then dysfunction of the endothelium and plaque growth for a person with a vertical spine! Constant damage to the endothelium leads to inflammation of the vessel walls, which is confirmed by an increase in the concentration of C-reactive protein [8]. The final result for many patients by the age of 50-60: cardiac output gradually decreases, and all this is due to spasm and decreased elasticity of the valve apparatus, the aortic arch and the left ventricle, in other words, due to increasing heart failure. Atrial fibrillation (fibrillation) may begin, which occurs due to changes in the mechanical properties of the tissues of the atria, ventricles and valves. Medicine introduced the concept of "rientry", but from the point of view of physics, the source of fibrillation lies in the piezoelectric effect that occurs in the mouths of the veins and atria [8], which periodically "knocks down" the work of the conducting system. At a young age, these recurring events with blood leaks during stress and arterial blood volume deficiencies are compensated by organismic adjustments: for example, some increase in the capacity of the pulmonary circulation, random physical and respiratory loads, frequent changes in body position, fluctuations in blood pressure and heart rate.

The main regulator is the vasomotor center, located in the medulla oblongata, but having its own receptors for pressure and blood volume in the heart chambers, aorta and large arteries! A deficiency of arterial blood in the arteries signals this by increasing blood pressure, and excess - by decreasing blood pressure. But the fact that organismic blood volume adjustments exist and they try to "work" under any conditions and at any age is proven by observations of patients with heart failure. Patients with a history of hypertension, atherosclerosis and heart failure very often experience shortness of breath, breathing difficulties, asthma, pulmonary edema.

And all this is due to the body's adjustments with attempts to raise blood pressure and pump additional volumes of blood through the pulmonary circulation in order to replenish the blood volume in the arterial bed to the "norm" (norm from 600 to 900 ml)! As a result of these adjustments, "blood pressure jumps" may appear in the arteries of both the pulmonary and systemic circulations. In cases where the output in the systemic circulation is limited, mainly due to calcification of the aortic valve and atherosclerosis of the aortic arch, and in the pulmonary circulation the stenosis of the pulmonary trunk is less significant (or does not exist at all), an overflow of venous blood in the pulmonary vessels occurs. All this, as a rule, is characterized by additional perfusion of fluid from the pulmonary vessels into the interstitial space and alveoli. Dyspnea and a serious crisis in the pulmonary circulation may occur. It turns out that the "primary" blood deficit occurred in the arterial bed of the large circulatory system, and the volume is replenished through the pulmonary arteries and veins of the small circulatory system!

This is how a "hypertensive crisis" of the pulmonary circulation occurs, although the regulation of volumes and pressure has been disrupted due to a gradual decrease in the throughput of the systemic circulation: the aortic valve and the aortic arch! In cases of relatively large leaks of arterial blood of the systemic circulation, replenishment of the blood volume in the arterial bed to the "norm" becomes increasingly difficult. A crisis is approaching, since a smaller total volume of arterial blood with a fixed volume of the arterial bed (the

arteries are compressed to the limit) becomes more difficult to maintain and, when necessary, increase arterial blood pumping. The natural "myocardium" pump cannot cope with the task, at this point many long-acting drugs for hypertension can only harm, since they relax the muscles of the heart and blood vessels. By this time, due to the lack of arterial blood volume in the bloodstream, a natural spasm of small arteries and arterioles of the pelvic organs, arms and legs occurs (they become cold). But when an increase in blood volume due to a spasm of all peripheral arteries also cannot help solve the problem, when the lack of blood volume reaches a certain limit, a hypertensive crisis with a rapid increase in blood pressure suddenly begins, while the vasomotor center, increasing pressure due to the work of the myocardium, makes last attempts to supply the brain with a sufficient volume of arterial blood! At this time, the main human organ does not receive enough oxygen and nutrients, critical cerebral ischemia is approaching. For any person, these events are characterized by suddenness, because just an hour ago this person felt good or excellent. This is how an attack called a "hypertensive crisis" of the systemic circulation begins with an increase in blood pressure to 200 mm Hg. (The author of the article experienced such a crisis in 2018. The crisis was stopped by doctor with a magnesia injection, so the author can describe the crisis in the first person. Oddly enough, it was this crisis that accelerated the creation of a new theory of the cause and mechanism of atherosclerosis in 2020-2024.) Thus, in cases of the onset of signs of a crisis, it is necessary to call an ambulance.

But attacks can be prevented by oneself if a person begins to relieve the effects of stress in time and prophylactically replenishes the arterial pool, at least by adopting a horizontal body position, breathing exercises in the form of diaphragmatic breathing, with an emphasis on sharp exhalations and sharp upward movements of the diaphragm, or other well-known techniques! In no case should you lower blood pressure at such moments with blood pressure pills! Since taking pills can lead to a stroke! Yes, yes, yes! This has been proven by the long-term negative practice of lowering blood pressure using drugs with a rapid effect on the body during a crisis. Now let's return to the venous pool of the systemic circulation, because not everything is in order there either. Excess venous blood, including that which enters the veins through opening anastomoses, accumulates under the action of gravity in the lower half of the body under the action of the Earth's gravity. The veins expand, the blood flow rates decrease to the limit, the venous valves on the legs can be destroyed due to overload. With a sedentary lifestyle, excess venous blood leads to edema of the pelvic organs and legs. These constant excesses of blood and intercellular fluid contribute to weight gain, physical degradation of a person, the occurrence of varicose veins and thrombosis.

Venous blood that is retained for a long time loses its quality, it receives glucose (HbA1C) that has stagnated for 2-3 months. All this contributes to the development of type 2 diabetes and other diseases. The following fact is indicative. A jump in blood glucose in diabetics and pre-diabetics in the morning hours after sleep (or rest in a horizontal position) is precisely evidence of the flow of stagnant venous blood and intercellular fluid towards the upper half of the body and the head! This effect was called the "Somogyi effect" or the "dawn" effect. They came up with a beautiful name for diabetics, but it is not correct. Thus, the claims that all diseases are due to atherosclerosis, including atrial fibrillation, are most likely confirmed. And the "beginning of the beginnings" of all age-related diseases in humans is in mechanical damage to the aorta, aortic valve and large arteries near the heart due to uncontrolled leakage of arterial blood during moments of stress!

Conclusion

1. The mechanisms of occurrence of hypertensive crises of the large and small circles of blood circulation have been found!
2. A new theory of the cause and mechanism of atherosclerosis is confirmed!
3. It turns out that for Homo erectus, the main organismic parameter is the current (on average) volume of arterial blood. This parameter requires constant monitoring. The degree of atherosclerosis and the presence of a bouquet of cardiovascular diseases, and the life expectancy itself, depend on frequent deviations of this parameter from the optimal value (due to blood leaks during stress!)!
4. As a preventive measure against atherosclerosis, it is recommended to perform regular (say, every 2-3 hours) breathing exercises for 1-3 minutes, especially after stress.
5. Now it becomes obvious that numerous author's methods of promoting health through breathing exercises are useful. They replenish the arterial pool and prevent the development of atherosclerosis!
6. Ways to monitor arterial blood volume and ways to optimally replenish arterial blood volume in case of leaks are a large field for further research.

Turning to Big Medicine

Dear experts, academics, Doctors of Sciences! Indeed, for more than 100 years of joint research into the causes of atherosclerosis and crises, and the premature loss of parents, friends, and patients from hypertensive attacks, strokes, and myocardial infarctions, you have not been able to see a very simple, understandable, and transparent theory. You've just got it all mixed up. Why? My opinion is that you lack basic knowledge of physics and mathematics. Sorry. I had to say this, especially since the leaders of medicine have been trying to ignore this new theory for 5 years, apparently, the invention of new pills is more important to them. I'm sorry if there are any inconsistencies or inaccuracies in the presented theory... I'm not a doctor. I am a single

physicist who retired 15 years ago. I believe that doctors should take into account this new knowledge... Since the presented theory of the mechanism of atherosclerosis is "pioneer", there is no need to refer to the authors of most of the old theories about the cause of atherosclerosis. Most of the old theories are automatically included in the new theory as factors of atherosclerosis.

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ISSN: 2574-1241

DOI: 10.26717/BJSTR.2025.62.009747

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