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RESEARCH ARTICLE

Hydrodynamic theory of atherosclerosis formation in humans - «Reaction to spasm». Cylindrical cholesterol plaque is the cause of heart attack and stroke.

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Conflicts of Interest Statement: I, Rusanov Sergey, am the sole author of the manuscript.

ABSTRACT

An analysis of scientific literature, photo and video materials showed that plaques in humans are completely different from plaques in animals and the «contemporary official description» of AS. They differ in appearance, lack of inflammation of the walls of the artery, clinical manifestations, complications. It has also been found that «true» plaque has nothing to do with the first four types of atherosclerosis in humans. It was necessary to understand how in the lumen of a healthy artery a strong, elastic, yellow, homogeneous plaque in the form of a hollow cylinder - a «cylindrical plaque» - appears very quickly? As a result, the «Hydrodynamic Theory» was proposed, which answers all the complex questions related to the appearance, aging and destruction of «true» plaques in humans. It describes the etiology, pathogenesis, clinical manifestations, classification, complications, methods of treatment and prevention of «cylindrical» plaques that cause heart attacks and strokes in humans.

keywords: Atherosclerosis; LDL; Heart attack; Stroke; Hypertension; Thrombus; Embolus; Endothelial dysfunction; Dissection

Abbreviations

AS - atherosclerosis;
ASL - atherosclerotic lesions;
ASL I - type I atherosclerotic lesions;
ASL IV - type IV atherosclerotic lesions;
ASL I-IV - types I-IV atherosclerotic lesions;
ASL V - type V atherosclerotic lesions;
ASL VI - type VI atherosclerotic lesions;
ASL V-VI -types V and VI atherosclerotic lesions;
ED - endothelial dysfunction;
FC - fibrous cap;
LC - lipid core with molten lipids;
LDL - low density lipoproteins;
MFC - macrophage foam cells;
VV - vasa vasorum;
CCP - cylindrical cholesterol plaque;
FCCP - friable cylindrical cholesterol plaque;
SCCP - soft cylindrical cholesterol plaque;
DCCP - dense cylindrical cholesterol plaque;
OCCP - old cylindrical cholesterol plaque;

Introduction

Scientists began to study atherosclerosis over 100 years ago. Currently, about 20 million people die each year from vascular diseases. From the very beginnings of the study of atherosclerosis (AS), doctors and scientists have taken as a basis the opinion that vascular lesions in animals and people are completely identical.[1] Therefore, the data obtained in the process of studying of AS on animal models were used to ensure prevention and treatment of AS in humans. «Contemporary official description» of AS is taught in medical institutes, after a long study: its appearance, pathogenesis, etiology, clinical manifestations, complications, methods of prevention and treatment. It is believed that «contemporary official description» of AS completely and absolutely accurately describes all atherosclerotic processes occurring in the vessels of animals and humans.[2-6]

In the process of studying of AS and monitoring of patients, had a lot of questions about the accuracy of this claim. In practice, many patients, before the onset of acute problems, faced no problems with the cardiovascular system, which rejected the view of the preliminary, perennial development of atherosclerotic lesions (ASL) inside human vessels. Many patients before going to the doctor led a healthy lifestyle and did not have «bad habits», which may indicate the absence of a significant influence of many generally accepted «factors» and «risks» on the development of acute processes in the vessels. The emergence of acute problems in previously healthy people leading a healthy lifestyle was confirmed by scientists from the University of Sydney.[7] Many people smoke and suffer from obesity for decades and do not have vascular problems, which indicates that these factors do not have significant impact on the development of acute vascular problems. The report «The Health Consequences of Smoking: A Report of the Surgeon General» - does not mention the effect of smoking on the occurrence of atherosclerosis at all.[8] Obesity and overweight have a «big impact» on statistics only when calculating the number of obese people among those with atherosclerosis. If we calculate differently and compare the dependence of the appearance of atherosclerosis in all obese people, the «dependence» is completely absent. Two billion people worldwide over the age of 18 are overweight or obese. Approximately 3,4 million obese or overweight patients die from atherosclerosis every year.[9] This is only 0.17% of all people with such a disorder.

Has been seen a significant dependence of acute vascular problems on nerve overload that occurs in people who previously had no problems with blood vessels. Dependence of acute problems from severe nervous overloads in previously completely healthy people can only say that the problem in the vascular system arises acutely, is created easily and is not connected with the long-term development of atherosclerotic plaque inside the intima. Numerous studies have shown that there is a dependence of vascular problems from changes occurring in the central and autonomic nervous system.[10-26]

During an operation to remove a cholesterol plaque in humans, in several places of the aorta and in several vessels of the heart, yellow, homogeneous, soft, strong, elastic formations in the form of a «tube» are visible, completely repeating the contours of the vessel. These cholesterol plaques were easily cut and easily removed from the vessel. The wall, in the area where the cholesterol plaque was located, was not damaged and was identical to those places where the plaque was not. The plaque walls, located in close proximity to the artery wall, was smooth and followed the contours of the vessel. The exact same cholesterol plaque can be observed in a contemporary video made during «Carotid endarterectomy».[27-37] The appearance in the form of a «tube» («hollow cylinder»), the presence of a healthy artery wall under the plaque, the absence of a wound surface on the inner wall of the artery after the removal of the plaque, the presence of a smooth outer wall of the plaque, the homogeneous structure and absence of necrotic nuclei in the structure of the plaque itself, said that this plaque is an independent structure, which is located inside the lumen of an artery and is not associated with inflammation of the artery wall.

As a result of the analysis, it was hypothesized that ASL appear in human vessels due to severe nervous overload. ASL should be inside the lumen of an artery, should be able to appear very quickly and has nothing to do with perennial inflammation and damage caused to the intima.[38-41]

According to the current view, ASL in humans and animals should be completely identical to each other. The analysis revealed facts that showed a 100% difference between these lesions.[40] ASL in animals is provided by inflammation and swelling of the inner wall of the artery (intima). With the current level of photo and video technology, it is easy to see that humans in a cholesterol plaque does not have several important signs of inflammation - there is no liquid inside the “body” of a cholesterol plaque (there is no plaque edema), there is no local temperature increase (due to increased blood supply), and also no reddening of the «body» of the plaque itself. At the same time, many factors are visible that show that a plaque in humans is in no way connected with the wall of the

artery, it has a uniform, strong, elastic yellow structure.[27-37] The application of the results of studies obtained in animal models are ineffective for the prevention and treatment of «true» cholesterol plaques in humans.

According to modern concepts, in the arterial wall of a humans, at first, an atherosclerotic lesion of type I (ASL I), consisting of a single necrotic nucleus, appears. With an increase in the size of the nucleus, an atherosclerotic lesion of type IV (ASL IV) is formed. The lumen of the artery is blocked on one side. It is believed that over the course of decades many ASL IVs will appear, which merge and form an "altered intima". It is believed that the «true» cholesterol plaque is the "altered intima", consisting of a large number of necrotic nuclei. The analysis showed that in human arteries there are two completely different forms of ASL that are not interconnected.[39] Only ASL I-IV are located in the intima and stretch the middle and outer layer of the artery. «True» atherosclerotic lesions of type V-VI (ASL V-VI) are located only in the lumen of the artery and are in no way associated with inflammation of the intima. The wall of the artery shows no signs of damage. In the area of the presence of a «true» cholesterol plaque, there are no signs of the presence of ASL I-IV, and even more so, there are no signs of their fusion with each other. All these data show that «true» cholesterol plaques in humans have their own characteristics, their own etiology and pathogenesis.

According to the current view, the «contemporary official description» of atherosclerosis should fully coincide with ASL in humans and with ASL in animals. Since ASL in humans and ASL in animals differ by 100%, the possibility of matching characteristics with either ASL in humans or ASL in animals was analyzed.[41] Analysis showed that this type of ASL is absent from both human and animal vessels. As a result of the analysis, it can be assumed that this form of ASL was artificially synthesized to simplify the understanding of the atherosclerotic process and mimic the similarities between processes in animals and two completely different processes in humans.[41] The «Inflammatory theory» that underlies the understanding of the «contemporary official description» of atherosclerosis cannot possibly be used to study «true» cholesterol plaque in humans.

The complete individuality of the «true» cholesterol plaque in humans, the absence of inflammation at the time of the appearance of the plaque, requires a revision of the theory of the occurrence of cholesterol plaque in humans.[38-41]

For effective prevention and treatment of «true» cholesterol plaque, it is necessary to determine the real cause of its appearance, to understand the mechanism of accumulation of low-density lipoproteins (LDL) in the lumen of the arteries, as well as find answers to other difficult questions (table No. 1).

The difference between atherosclerotic lesions in animals, two types of lesions in humans, and the «contemporary official description» of AS is reflected in table №2.

In this article presents the «Hydrodynamic theory» of the occurrence of «true» plaque in humans (APL V-VI) in the lumen of an artery, which directly depends on a strong narrowing of the arterial wall («Reaction to spasm»). This theory helps to understand the etiology, pathogenesis, clinical manifestations, complications, as well as the impact of ASL V-VI on persistent increase in the blood pressure (Fig. 3) and the pathological increase in LDL levels in blood (Fig. 4). On the basis of this theory offers methods of prevention and treatment of the cholesterol plaque that creates real problems in human vessels.

Since, when appearing, ASL V-VI looks like a hollow cylinder, and there are no areas of inflammation, necrosis and sclerosis in the arterial wall - suggests to call such a plaque - «Cylindrical cholesterol plaque» (CCP) (Fig. 1-5).[39-41]

The application of the knowledge set forth in the «Hydrodynamic Theory» can save the lives and health of more than 20 million people a year, who currently die and become disabled due to improper prevention and treatment of «true» plaque in humans.

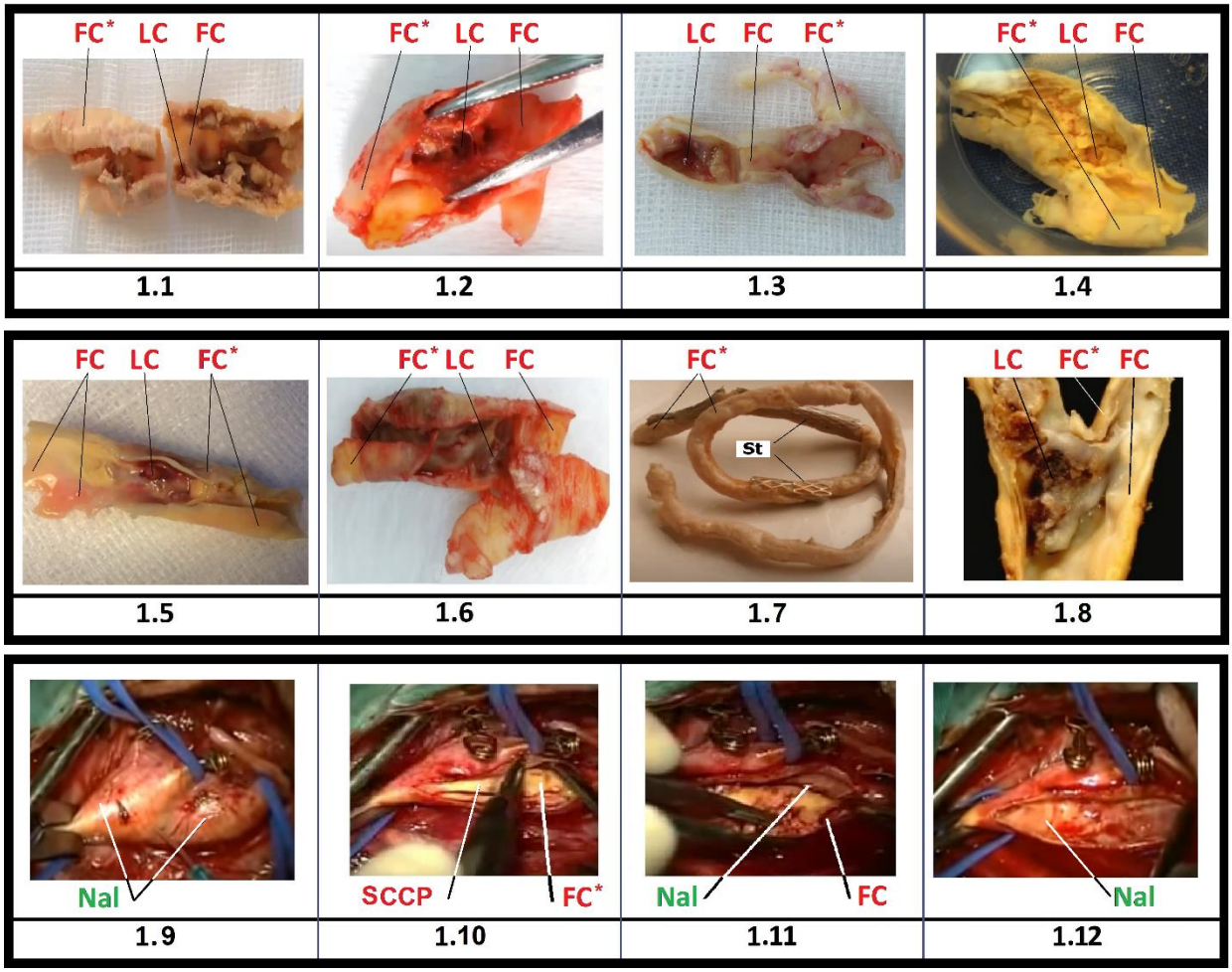

Figure 1: The types V and VI atherosclerotic lesions (ASL V-VI) in humans - «Concentric cylindrical cholesterol plaque» (CCP) after removal of from the artery retains its shape and looks like a «solidified silicone». (The description of the figure No. 1 is in Appendix No. 1)

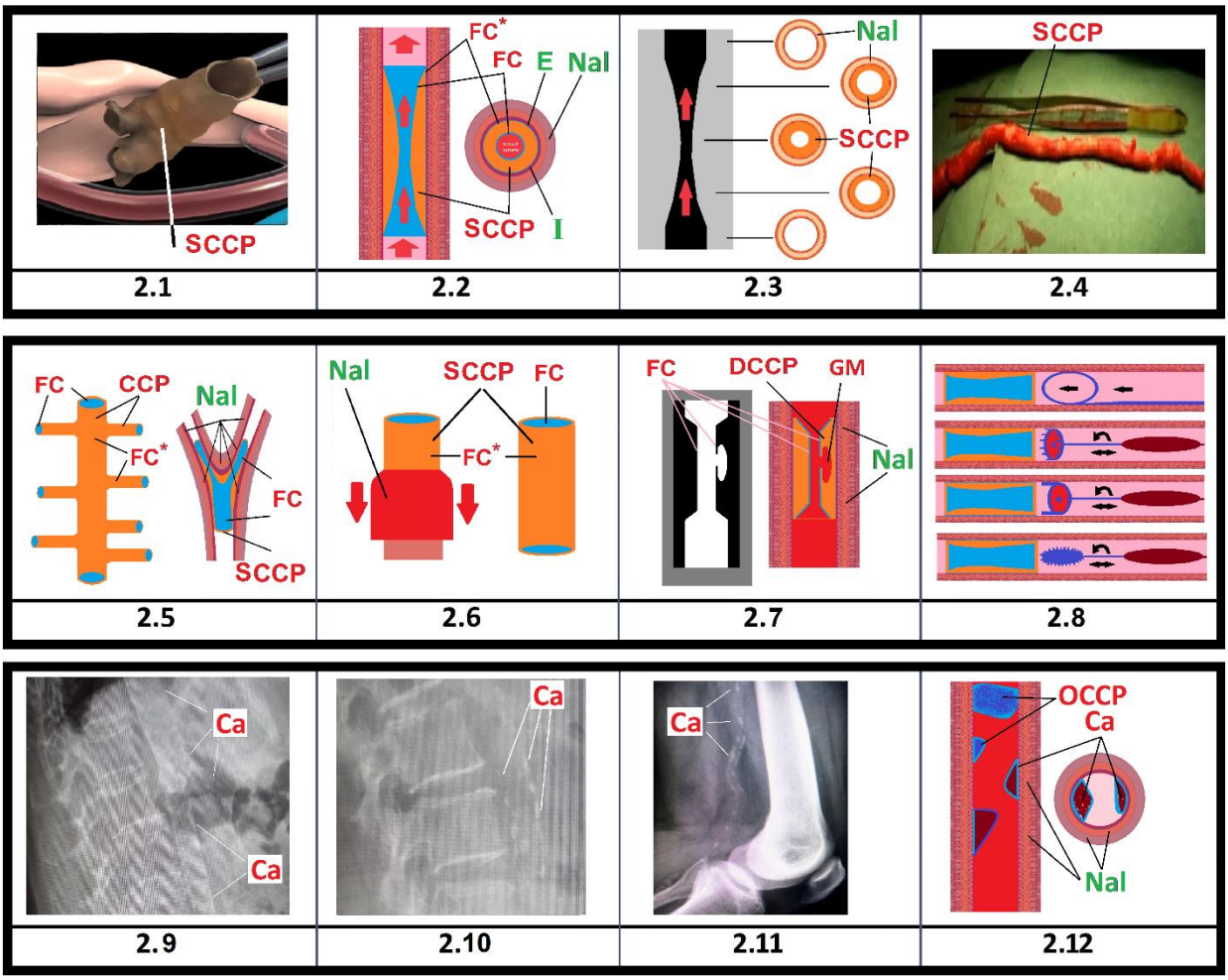

Figure 2: The types V and VI atherosclerotic lesions (ASL V-VI) in humans - «Concentric cylindrical cholesterol plaque» (CCP). CCP is an independent pathological structure in the lumen of a healthy artery, consists entirely of LDL. (The description of the figure No. 2 is in Appendix No. 2)

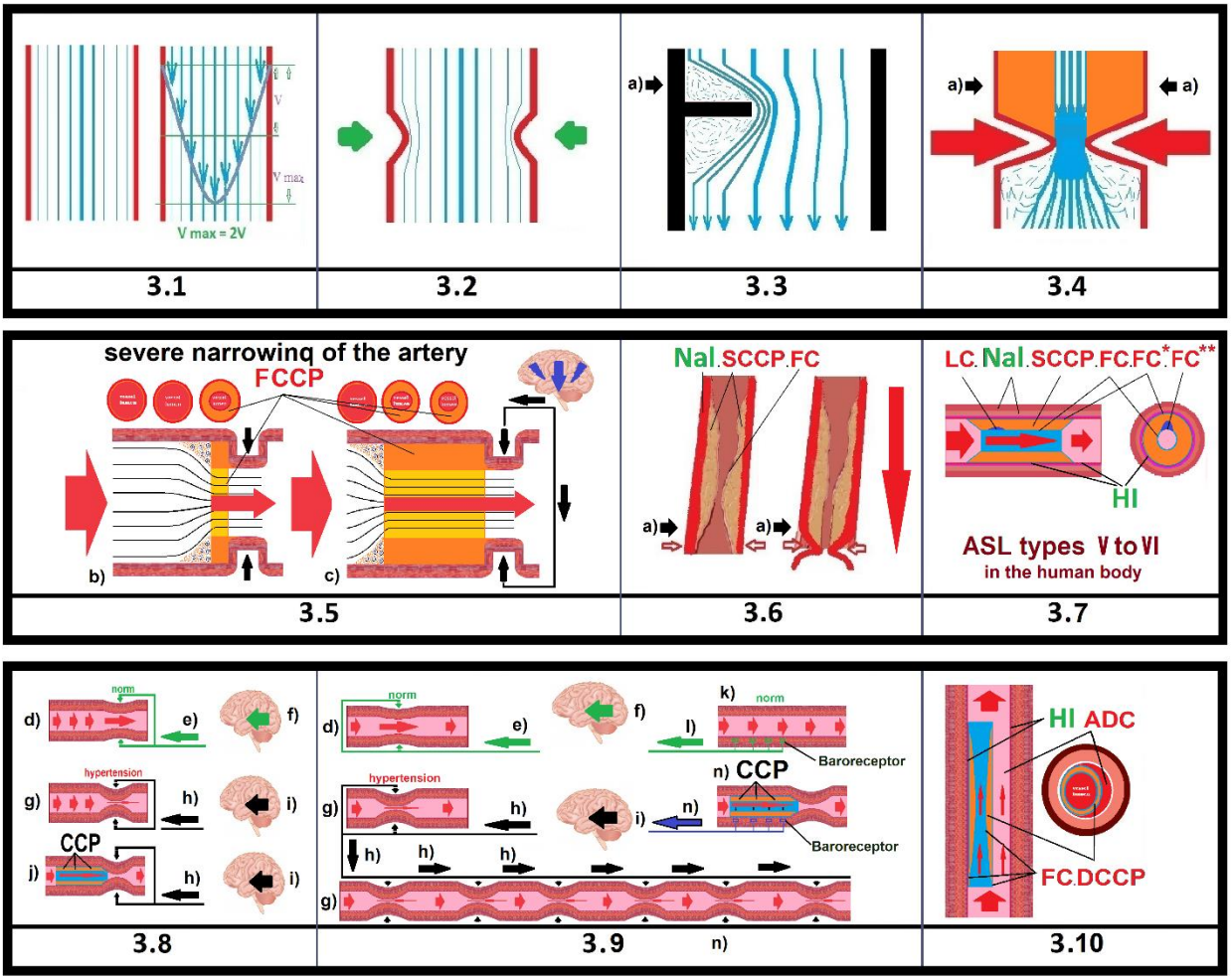

Figure 3: The formation of «Concentric cylindrical cholesterol plaque» (CCP) in the lumen of the artery. Effect of CCP on blood pressure. Arterial dissection. (The description of the figure No. 3 is in Appendix No. 3)

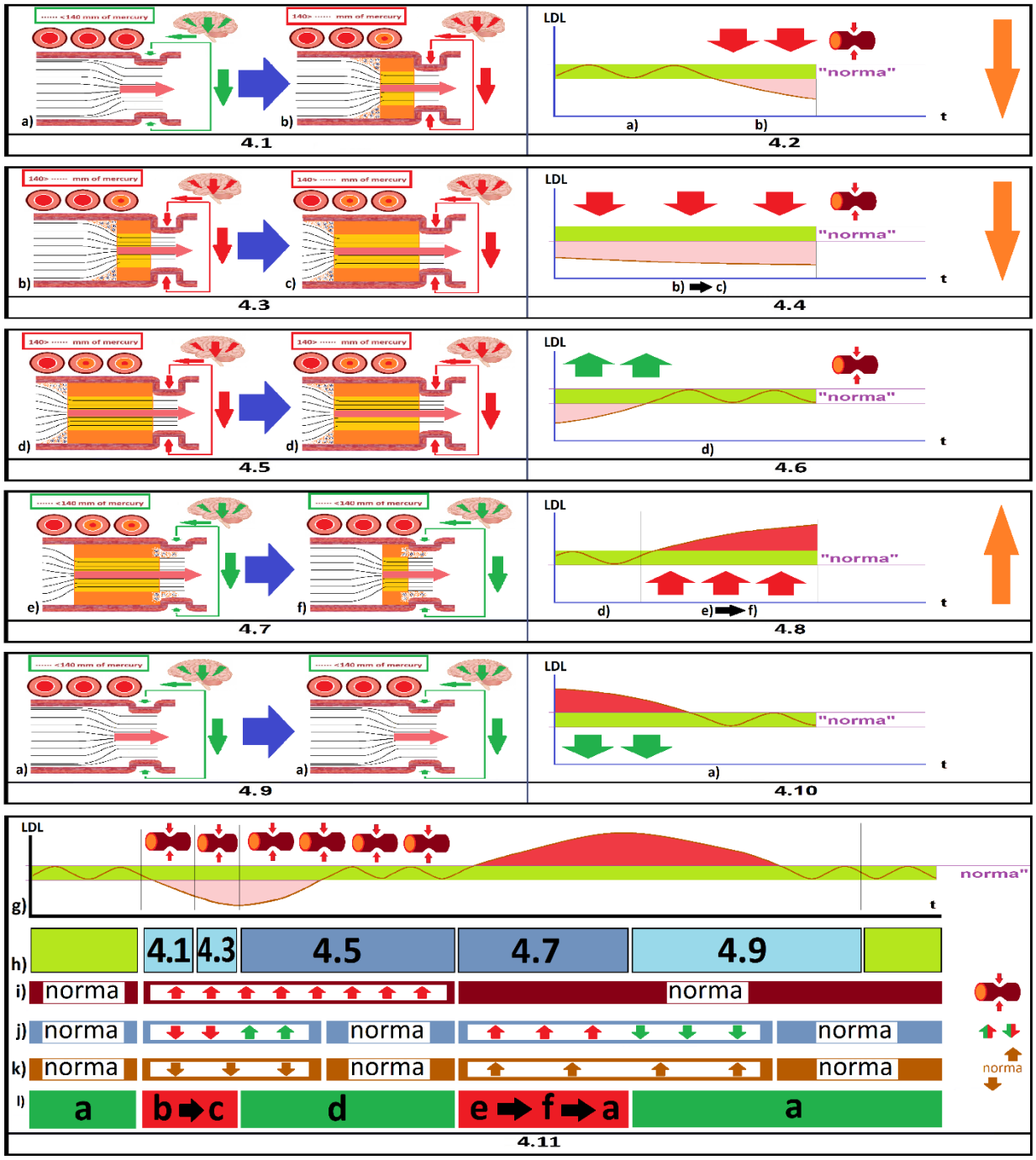

Figure 4: The mechanism of the pathological increase in the level of LDL in the blood depends on the formation and destruction of cylindrical cholesterol plaques (CCP) in the lumen of the artery. (The description of the figure No. 4 is in Appendix No. 4)

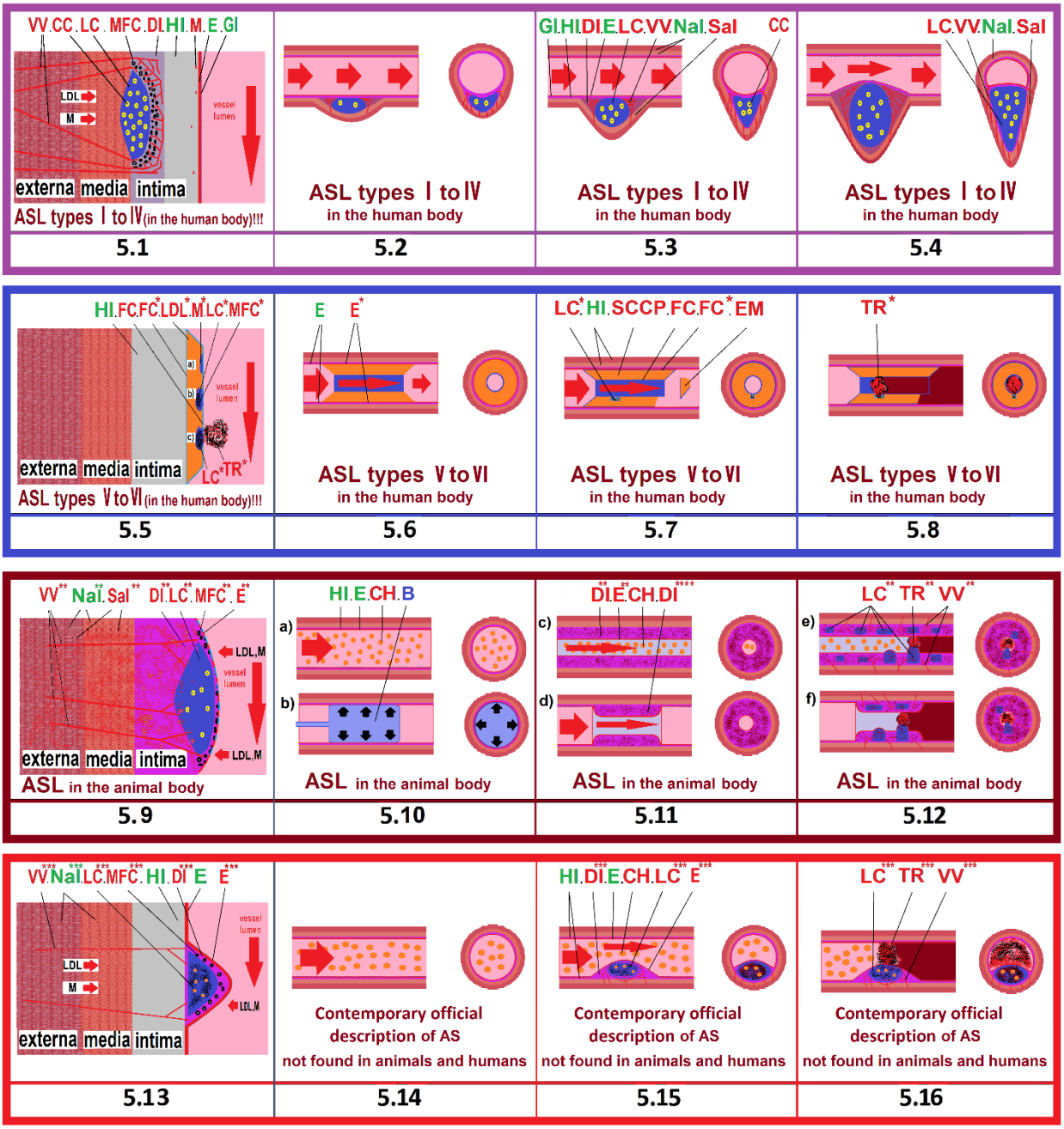

Figure 5: Four types of atherosclerotic lesions. Two types of ASL in human arteries, one type of ASL in arteries and veins in animals, and a «contemporary official description» of AS, which in reality is absent in the vessels of humans and animals. (The description of the figure No. 5 is in Appendix No. 5)

A «Hydrodynamic Theory» of appearance of cylindrical cholesterol plaque - «Reaction to spasm»

ASL V-VI is independent structure that is located in the lumen of the artery and has no pathological connection with the artery wall. ASL V-VI is «true» plaque in humans and has no connection with ASL I-IV in humans, with «true» plaque in animals and with «contemporary official description» of ASL.[38-41] ASL V-VI has its own characteristics: etiology, pathogenesis, appearance, clinical manifestations, complications, prevention and treatment. Taking into account it has a form of the hollow cylinder, and the absence of sclerotic damage to the artery wall, suggests calling ASL V-VI as «Cylindrical cholesterol plaque» (CCP) (Fig. 1.1-1.11, 2.1-2.12, 3.1-3.10,5.5-5.8).[38-41]

<u>The etiology of the appearance of cylindrical cholesterol plaque</u>

The main cause of CCP is anything that can lead to strong compression of artery wall. The

contraction of the vascular wall occurs after the supply of nerve impulse from the vasomotor center to the muscles of artery. Due to the fact that such compression can occur simultaneously in several arteries, it is possible to claim that the reason is the over excitation of the vasomotor center, located in the vegetative nervous system (ANS). Such «over excitation» in ANS can occur independently, when there are problems inside ANS itself, and as a result of the influence on it from the central nervous system (CNS). Over excitation in CNS is possible in case of occurrence of global cataclysms and reasons of a personal nature, such as a loss of a loved one, war, earthquake, tsunami, family and work problems, watching football matches, depression, aggression, constant pain factor, experiences about failed attempts to eliminate harmful «factors» and «risks» (failed attempts to «quit smoking» or «lose weight»), as well as other factors that can lead to intense feelings in a human. In case of a long and strong over excitation in CNS, there is pathological influence on ANS, which can cause a persistent strong over excitation in ANS. All «factors» and «risks» that have systemic «toxic» effects on the artery walls have no influence on the appearance of CCP, but can influence on further aging and destruction of CCP.

Thus - CCP can appear because of any external and internal «factors» and «risks» that can cause a strong and long-term over excitation in CNS and ANS, over excitation of vasomotor center and strong compression of the artery wall with the formation of «a big obstruction» to the blood flow in the arterial lumen (Fig. 3.1-3.9).[38-41]

The pathogenesis of cylindrical cholesterol plaque

Under normal conditions, systemic arterial pressure (AD) is regulated within the «physiological norm». This is ensured by reflex connections and baroreceptors located in the carotid sinus and aortic arch. A short-term increase in AD above the «norm» can occur when certain conditions associated with the normal response of the body to external and internal stimuli occur. The AD should be restored immediately after the termination of the stimulating factors and further, remains within the «norm». In case of the appearance of factors described in «etiology» - a strong long-term over excitation occurs in ANS, which, in the long run, can lead to a strong long-term over excitation in ANS, including in the vasomotor center. Increased tone of vasomotor center leads to «severe contraction» which occurs in arteries with muscle fibers. Since the center consists of smaller sections and has control of many arteries - such «strong contractions» can occur both separately in different arteries and in several arteries simultaneously. The degree of narrowing of the lumen in different arteries may be different.

When blood pressure is «below the norm» and within the «norm» - the blood in the vessels of a human flows in a laminar type. To ensure normal blood pressure, a slight narrowing of the artery wall occurs. In places where narrowing occurs and in places with a slight narrowing - the entire mass of blood evenly passes through the lumen of an artery (Fig. 3.1, 3.2). Increased tonus in the vasomotor center leads to a local «very strong contraction» of the artery lumen. The lumen of an artery narrows much more than it needs to ensure a normal physiological level of AD. With such a strong narrowing of the lumen of an artery conditions arise that are called in the «Hydrodynamics» - as «sudden contraction». This narrowing creates an obstacle in the form of a ring with a narrow opening. The mass of the blood is stratified. The main elements of the blood tend to pass through a narrow opening at the same time as the blood flow. Due to the appearance of a «strong obstacle, places of vortices appear from the side of the artery walls, in which speed of blood flow is greatly reduced.

Formed blood elements are accumulated in these places, having certain characteristics. LDL have such characteristics. Since the obstruction of blood flow looks like a «ring» - LDL accumulate before the obstacle in the form of a «ring». The created «ring» from the LDL creates a new obstacle in the form of a «ring». As a result, a «tube is formed in the lumen of an artery («hollow cylinder») from accumulated «rings». The whole «tube» consists of LDL. In case of very long contraction of the arteries - LDL start to stick together. A soft, yellow, elastic, firm, strong like frozen silicone, plaque in the form of a «tube» or «hollow cylinder» consisting only of LDL with small inclusions of other formed blood elements caught in a vortex. That is why suggests calling such a plaque - «Cylindrical cholesterol plaque» (CCP). After the formation of the CCP, fibrous layers are formed from the side of lumen of an artery, and from the outer side of the CCP, near the artery wall. The inner layer has its own name - «fibrous cap» (FC). FC does not allow macrophages in blood to absorb the LDL that CCP consists of.

Thus, the formation of CCP is caused by a strong compression of the lumen of an artery and the appearance of an obstruction to the blood flow. A «plaque» in the form of a «tube» is formed in front of this obstacle, consisting only of LDL. Gluing LDL to each other and the appearance of fibrous layers ensures the formation of long, concentric, soft, strong, elastic, yellow, uniform structure, in the form

of a tube with a hole in the middle in the lumen of an artery (Fig. 3.1-3.9).[38-41]

Appearance of a cylindrical cholesterol plaque

The appearance of the CCP changes with aging and deterioration of «body» of CCP.

After gluing the LDL together in the form of «tube» and formation of fibrous layers from the wall's side and from the side of artery lumen - CCP is formed, which can be observed on multiple videos made during the operation. CCP is long, concentric, soft, strong, elastic, yellow, uniform structure, in the form of a tube with a hole in the middle, has a firm, durable, elastic structure, is located in the artery lumen, has a length of several centimeters, up to a few tens of centimeters, completely repeats the internal artery lumen, continues in the branch of the arteries. Once formed, CCP occludes the artery lumen from all sides (concentrically). The entire CCP consists of a uniform yellow material. The outer side of the CCP always remains smooth and shiny. The CCP is easily removed from the artery without forming a wound surface on the artery wall. The inner surface of the CCP, after formation, has a smooth surface covered with a fibrous layer. In case of aging, the inner layer has caverns formed after breaking necrotic nuclei. On the inner side, under FC, there may be lipid core (LC) with liquid content, as well as cavities after dissolution or destruction of the inner surface of CCP. As the CCP ages - some parts come off. Parts of CCP which remained in the lumen of aorta or lumen of an artery can occlude the vascular lumen only from one side (eccentrically). In very old CCP - LDL can be completely replaced by calcium. CCP is located only in arteries which have a muscle layer.[38-41]

At the transverse cut, it can be seen that the CCP occludes he lumen of the artery in a form of a ring. There are fibrous layers from the side of artery and from the side of artery wall. The damage of FC causes the appearance of LC, which is located directly under the FC, grows inside of the «body» of plaque. There are no muscle cells and vasa vasorum (VV) inside the CCP. Endothelium of artery is located between CCP and artery wall (Fig. 1, 2).[38-41]

Clinical manifestations of cylindrical cholesterol plaque

The rapid formation of CCP leads to decrease in the lumen of the artery and insufficient blood flow to the organ at high load. Thus, CCP located in the vessels of the heart, manifests itself as acute ischemic heart disease. Disorders in the cerebral and neck vessels lead to signs of chronic or acute circulatory disturbance in the brain. When the «thin fibrous cap» is ruptured and a clot is formed in the lumen of the artery, acute ischemia appears behind the CCP localization site. In case of formation of emboli, an acute ischemia appears at the place of overlap of the vessel lumen ин embolus.[38-41]

Classification of types of cylindrical cholesterol plaque

- The FCCP (friable cylindrical cholesterol plaque) is the original plaque formed from accumulated LDL in front of a site of artery narrowing. Until the LDL are glued together - such a plaque can easily crumble and return to the vascular bed all the LDL that were in it;
- The SCCP (soft cylindrical cholesterol plaque) - when LDL are glued with each other, the plaque is formed in the form of a firm, uniform, yellow, elastic «tube» or «cylinder» in the lumen of artery. Fibrous layers are formed from the side of the lumen of the artery and from the artery wall;
- The DCCP (dense cylindrical cholesterol plaque) - when FC is damaged, macrophages penetrate the plaque, absorb LDL and produce mast cells and one NYA. The NYA develops immediately behind the FC and destroys it. A fibroadenoma with a thin lid is formed. When the thin FC is ruptured, a clot may appear in the lumen of the artery. The photographs show that the inner surface of each plaque had several NYA;
- The OCCP (old cylindrical cholesterol plaque) - destruction of CCP with the formation of eccentric lesions, formation of emboli, and substitution of LDL with calcium (Fig. 1, 2).[38-41]

Complications

Pathological increase in level of low density lipoproteins in blood

Short cycles of increase and decrease in blood pressure (vegetative-vascular dystonia, neuro-circulatory dystonia) lead to higher levels of LDL in the blood. After a strong constriction of the arteries - LDL from the blood are accumulated in the CCP. The LDL level decreases dramatically in blood and is much lower than the «norm». Immediately after that, mechanisms are launched that ensure the restoration of level of LDL in the blood is to the normal level. When the compression of the artery walls stops, the geometry of the vessel wall is restored. If the artery was extended before the LDL is glued together - the LDL, from the CCP, are easily returned to the vascular bed. The vortex zones that appear behind the plaque promote the breakdown of accumulated LDL and the return of LDL to the blood stream. Taking into account that during this time the LDL level has been restored to the normal level- the LDL level in the blood becomes above

«normal». Each cycle of plaque formation and destruction leads to an increase in LDL levels in blood. Between cycles - the LDL level in the blood tends to return to the «norm» level due to the consumption of «excess» LDL - so the measurement of LDL level in the blood gives no practical data, as it is constantly changing from the maximum digits to the «norm» level. If the compression and expansion cycle occurs before the LDL level is restored to the «normal level» - the final» LDL level will be even higher. The indicator of recovery can only be a constant level of LDL in the blood within the limits of physiological norm (Fig. 4).[38-41]

Persistent increase in blood pressure in case of cylindrical cholesterol plaque

The constant presence of CCP in the area of baroreceptors - causes persistent increase in blood pressure. The presence of an additional «tube» in the lumen of the artery causes decrease in diameter of internal lumen of an artery. According to «Hydrodynamics» (Bernoulli's law), a decrease in the inner diameter of the vessel causes a decrease in the pressure of liquid on the artery walls. If the CCP is located at the locations of the baroreceptors, the pressure on the baroreceptors in this location will be consistently lower than the «true» arterial pressure in vessels without CCP. At continuously reduced pressure on the baroreceptors - the reflex connection is activated, which leads to a constant reflex increase in the systemic AP. The solution to this problem is to remove the CCP from the localization site of baroreceptors (Fig. 3.9).[38-41]

Persistent dysregulation of oxygen level in case of cylindrical cholesterol plaque

The constant presence of CCP in the localization site of chemoreceptors - causes constant violation of reflex regulation of chemicals in the blood. The presence of tube in the artery lumen that is dense, no longer pervious to the blood causes the permanent overlap of the receptors and the inability to correctly assess the level of chemical elements in the blood - the normal operation of chemoreceptors is disturbed. Chemoreceptors are primarily sensitive to changes in partial pressure (P) O_2, and secondary to pH reduction and CO2 increase. It has been suggested that imitation of hypoxia may lead to an increase in the number of active oxygen species, such as peroxides, ions, and free radicals, but, at the same time, there is no reliable data regarding this issue. The solution to this problem is to remove the CCP from the localization site of chemoreceptors.[38-41]

The formation of fibroatheroma with thin lid

In case of little damage caused to the integrity of FC - macrophages penetrate the CCP that absorb the LDL that the entire CCP consists of. Mast cells and LC are formed immediately under the FC. LC enzymes destroy fibrin strands of FC and make it thinner - a «fibroatheroma with thin lid is formed» (Fig. 5.5-5.8).[38-41]

The formation of a clot in the lumen of the artery

Rupture of the « thin fibrous cap» can cause a clot in the lumen of the artery.

The presence of a large number of lesions on the inner surface of the CCP can indicate that in case of rupture of the «thin fibrous cap», there may be such a situation that a thrombus may not appear. The formation of a thrombus in case of the rupture of the «thin fibrous cap» is not a prerequisite. Patients do not observe any clinical manifestations associated with the destruction of the CCP for a long time. That is why, in the process of aging of the CCP, only separate parts from the original cylindrical shape of the CCP remain (Fig. 5.5-5.8).

The formation of an embolus in the artery

Destruction of CCP can lead to the appearance of emboli. In the process of «aging» CCP its partial destruction occurs. Taking into account that the CCP is located in the lumen of an artery and can be damaged by LC enzymes - part of the CCP can tear off and penetrate into the blood flow - therefore, the embolus can lead to the appearance of an obstruction to the blood flow in places located very far from the location of the plaque itself (CCP).[38-41]

Endothelial dysfunction

The presence of CCP in the artery - causes the disruption of the normal functioning of endothelium as CCP is located in the lumen of an artery and is tightly pressed top its wall. CCP blocks the access of blood to the endothelial receptors by mimicking «endothelial dysfunction» when injected, at a given location, with chemical irritants.[38-41]

Artery calcification

Aging and destruction of CCP - causes artery calcification. CCP aging causes destruction, oxidation and replacement of LDL by calcium crystals which penetrate into remnants of CCP.[38-41]

Dissection of arteries, aorta

A dense, strong, elastic cholesterol plaque contains a strong fibrous layer both on the side of the lumen of the vessel and on the side of the artery wall. Such a plaque can partially break away from the inner wall of the artery with the formation of

severe pathology - dissection of the artery or aorta. The strong structure of the plaque allows it to withstand blood pressure for a long time without destruction.[38-41]

Diagnostics and treatment

Cylindrical cholesterol plaque - diagnostics

Contemporary methods of ASL research in humans are carefully developed and successfully applied, as they were developed and implemented based on observations of the current CCP. Practicing vascular doctor didn't include the generally accepted «Inflammatory theory» in these methods of research.

Cylindrical cholesterol plaque - surgical treatment

Contemporary methods of ASL surgical treatment in humans have also been developed and successfully applied, because vascular surgeons have used these methods on the basis of CCP operations. Practicing doctors didn't take into account the generally accepted «Inflammatory theory» in these methods of research.

Drug treatment of cylindrical cholesterol plaque

It is necessary to use all methods that can reduce or even completely limit the overstrain in the CNS and VNS in case of cataclysms of global scale or personal problems, stop pain, use methods to restore the psychological state of people who experienced complex stressful situation (war, flood, earthquake, death or serious illness of a loved one). It is necessary to use drugs that, on the one hand, reduce nervous excitability and, on the other hand, strongly block arterial spasm, both at the level of the Central Nervous System and at the level of arterial receptors.

The use of anti-inflammatory drugs does not affect the appearance of a «true plaque» in humans, but it can reduce the rate of damage and destruction of an already formed plaque. Anti-inflammatory drugs help reduce the complications of «true» plaque.

The use of statins for the prevention and treatment of CCP (to detect LDL in the blood) does not make sense, since the appearance of CCP in the vessels of a human does not depend on the level of LDL in the blood. [38-41]

Prevention of the appearance of cylindrical cholesterol plaque

Taking into account the etiology and pathogenesis of CCP, for prevention, it is necessary to use methods that allow a human to prevent CNS and ANS overstrain, including cataclysms of global scale or personal problems, in case of constant pain in the body. Prevention involves careful monitoring of a state of human who is in a difficult life situation (war, natural disasters, death or serious illness of a loved one, as well as other factors that can cause a human to have a persistent strong nervous strain). For prevention, drug therapy can be used - «sedative» drugs for the CNS and ANS, as well as drugs that block the strong compression of the arteries, and alternative methods can also be used to «reduce excitement in the nervous system». It is necessary to strictly control the regular intake by patients with hypertension - drugs that block vasoconstriction and drugs that have a sedative effect on the nervous system.

Harmful and dangerous methods of "prevention" of atherosclerosis currently used

Promotion of a «healthy lifestyle» aimed at decrease in number of «factors» and «risks» that are not associated with impact on the nervous system can do more harm than good. Failure to comply with doctors' demands regarding constant «maintenance of a healthy lifestyle», «quick weight loss» and «compulsory quitting smoking» can cause a constant nervous experience in a human, create nervous tension in the CNS and ANS, and result in the appearance of CCP in the vessels. Ineffective and harmful methods of «prevention» of atherosclerosis include all methods and ways that do not correlate with the etiology and pathogenesis of CCP, such as the active use of Omega 3 and Omega 6, the intake of a large amount of fruits and vegetables, foods containing antioxidants and L-arginine, the use of the «Mediterranean» diet with olive oil. It is necessary to cancel the unjustified restriction of the consumption of salt, sugar, saturated fats, flour, fatty foods, fatty meat, foods containing cholesterol. It is necessary to stop active actions aimed at the «prevention» of atherosclerosis, such as active weight loss, sudden quitting of smoking, active physical activity, if they cause strong nervous experience due to the absence of a pronounced changes.

Conclusion

For more than 100 years, there has been an opinion that atherosclerotic processes in animals and humans are absolutely identical. Data obtained from animals are used for the prevention and treatment of humans. One of the leading theories of atherosclerosis is the «Inflammatory Theory». Thanks to modern technologies for photo and video recording of atherosclerotic plaques, it can be seen that humans has a plaque in the vessels that does not fit the description of inflamed intima. There are no systemic inflammatory processes in human vessels, which must necessarily be under the influence of systemic «factors» and «risks» and

elevated levels of LDL in the blood. The rate of plaque appearance and clinical manifestations do not fit the description of a plaque that grows over several decades inside an inflamed intima.

As a result of the study and further analysis, it was shown that in the vessels of humans there are two types of plaques that are absolutely unrelated. When comparing the «true» plaque and plaques in the animal, a 100% difference was also found between them. When studying the «contemporary official description» of AS it was found that this type does not exist either in the vessels of an animal or in the vessels of a humans. Thus, we can conclude that the «Inflammatory Theory» describes only the formation of plaques in animals and cannot be used in any way for the prevention and treatment of plaques in humans.

The analysis of the features characteristic only for the «true» plaque made it possible to develop the «Hydrodynamic Theory». This theory describes the etiology, pathogenesis, clinical picture, complications, methods of prevention and treatment of the «true» cholesterol plaque in humans, shows the mechanism of the pathological increase in LDL levels in the blood, and explains the appearance of a persistent increase in systemic blood pressure. The application of the knowledge set forth in the «Hydrodynamic theory» can save the lives and health of more than 20 million people a year, who currently die and become disabled due to improper prevention and treatment of «true» plaque in humans («Cylindrical cholesterol plaque»).

Additional materials

Annex 1. Name and description «Figure № 1». The types V and VI atherosclerotic lesions (ASL V-VI) in humans - «Concentric cylindrical cholesterol plaque» (CCP) after removal of from the artery retains its shape and looks like a «solidified silicone»

1.1-1.8, 1.10, 1.11) There is a long, soft, elastic, strong, yellow tube in the form of a hollow cylinder located in the arterial lumen, have a concentric arrangement. The blood passes through a narrow passage in the center of CCP. CCP follows all the contours of the artery. Always has FC. CCP can have any length - from a centimeter to tens of centimeters. The outer wall of the CCP is always smooth and does not depend on the presence of an LC inside the wall of the CCP. On the inner surface of the plaque, areas of damage to the fibrous cap, foci of necrosis are visible. After removal of from the artery retains its shape and looks like a «solidified silicone»; 1.7) The length of the types V and VI atherosclerotic lesions in the arteries of the lower extremities more than 10 cm. CCP may appear after stent placement. The CCP was removed from the superficial femoral artery 2 weeks after stent implantation; 1.9-1.12) The length of CCP in the carotid artery is more than 3 cm. CCP is located both in the central artery and continues in its branch; 1.9) The outer artery wall near CCP has no tubercles and thickening, does not have VV; 1.12) The wall of the artery that surrounds the CCP has a normal structure, without damages, inflammation, sprain, and without necrotic nuclei.

FC - inner fibrous cap (between plaque and the lumen of the artery); FC*- outer fibrous cap (between plaque and artery wall); LC - the lipid core is located not within the arterial wall, but within the wall of the CCP itself; Nal - normal artery layers; SCCP- soft cylindrical cholesterol plaque; St -stent.

Images 1.1-1.7 courtesy of Dr. Mikhaylov I. Ph.D. in medicine. Leading vascular surgeon. Clinical Hospital of the Academy of Sciences in St.Petersburg.

Annex 2. Name and description «Figure № 2». The types V and VI atherosclerotic lesions (ASL V-VI) in humans - «Concentric cylindrical cholesterol plaque» (CCP). CCP is an independent pathological structure in the lumen of a healthy artery, consists entirely of LDL.

2.1) CCP on the training video follows the contours of the vessel. Branching of the CCP in the lateral branches in the arteries; 2.2) There is a long, soft, elastic, strong, yellow tube in the form of a hollow cylinder located in the arterial lumen, have a concentric arrangement. Endothelium is located between «intima» and the external wall CCP. The blood passes through a narrow passage in the center of CCP. Always has FC; 2.3) X-ray with contrast agent. The anatomical dimensions of the artery do not change. When examining CCP it can be seen that CCP pinches the artery evenly on all sides of the artery. It can be seen that the blood passes through a narrow passage in the center of CCP; 2.4) There is a long, elastic, strong, yellow tube in the form of a hollow cylinder; 2.5) Branching of the CCP in the lateral branches in the arteries of the heart. The anatomical dimensions of the artery do not change. Branching of the CCP in the lateral branches of the artery in the carotid sinus. CCP follows the contours of the artery, is located on all sides, close to the artery wall. Blood flows inside the CCP and moves away from the artery walls. The anatomical dimensions of the artery do not change; 2.6) The artery wall that is intact, without inflammation, without tubercles and necrosis, can be

easily turned inside out; 2.7) X-ray with contrast agent and longitudinal section of the artery with CCP. Bleeding in the LC. LC located in the CCP wall itself, immediately under the FC. The artery wall remains intact. The anatomical dimensions of the artery do not change; 2.8) Use of instruments having a form of ring and cylinder to remove CCP; 2.9-2.11) X-ray - calcium crystals deposition in the CCP; 2.12) calcium crystals deposition in the CCP wall. All subsequent forms of types V and VI atherosclerotic lesions, concentric and eccentric, are the result of the destruction of the original concentric structure of the CCP. The artery wall near CCP does not have inflammation, VV and LC, bleeding sites, protruding parts, sprain of the artery membranes, has a normal appearance of the inner and outer surfaces.

Ca - calcium crystals deposition; CCP-Cylindrical Cholesterol Plaque; DCCP-Dense Cylindrical Cholesterol Plaque; E- endothelium between plaque and arterial wall in humans; FC - inner fibrous cap (between plaque and the lumen of the artery); FC*- outer fibrous cap (between plaque and artery wall); GM - hemorrhage; I - intima; Nal -normal artery layers; OCCP- Old cylindrical cholesterol plaque; SCCP- soft cylindrical cholesterol plaque.

Annex 3. Name and description «Figure № 3». The formation of «Concentric cylindrical cholesterol plaque» (CCP) in the lumen of the artery. Effect of CCP on blood pressure. Arterial dissection.

3.1) The flow with parallel walls. Fluid movement is laminar; 3.2) the weak cross-clamping of the vessel doesn't create obstacles to the blood flow. The movement of blood in the artery is laminar; 3.3) A strong narrowing of the lumen of the vessel creates a strong obstacle to the passage of fluid. In front of the narrowing of the vessel - swirl zones are formed (a). In this swirl zone, elements with certain properties accumulate (LDL); 3.4) the strong cross-clamping of the vessel creates a strong obstacle to the blood flow. LDL accumulate in front of the obstacle (a); 3.5) Formation of CCP when a strong narrowing of the arterial lumen occurs. All blood cells easily pass into formed narrow opening, and only LDL are retained within the wall, in front of the site of arterial narrowing, and quickly create a CCP in the form of a hollow cylinder; b) the first ring, which consists of LDL, which lingered in the zone of turbulence in front of the place of severe narrowing of the artery; c) many adherent rings of LDL, which form a plaque in the form of a tube - CCP. The entire plaque consists of only LDL; 3.6) the sticking of LDL occurs in a view of cylinder with a hole in the middle, and it begins from the place of cross-clamping of vessel. In the case of imitation of compression of vessel, appears a typical form of closing zone (a); 3,7) CCP is long, concentric, soft, strong, elastic, yellow, uniform structure, in the form of a tube with a hole in the middle, has a strong, elastic structure. CCP is covered by a fibrous capsule both on the side of the lumen of the artery and on the side of the wall of the artery. CCP overlaps the lumen of the muscular and muscular-elastic artery along the perimeter (concentric). Endothelium is located between Intima and the external wall CCP. The blood passes through a narrow passage in the center of CCP. Always has FC. CCP may contain LC located near the arterial lumen. The normal fibrous capsule is damaged by LC ferments. Because of this, a «thin fibrous cap» forms over the LC; 3.8) d) «normotonus» - the walls of the artery are slightly narrowed to maintain normal blood pressure; e) normal signal from the nervous system; f) normal functioning of the nervous system; g) «hypertonus» - a strong narrowing of the lumen of the artery. The appearance of a strong obstruction to the movement of blood; h) increased signal level from the nervous system; i) over excitation of the central nervous system leads to over excitation of the autonomic nervous system and leads to narrowing of the lumen of the artery; j) accumulation of LDL in the form of a tube, in front of the site of narrowing of the artery; 3.9) Signal from baroreceptors (l) shows the nervous system what level of blood pressure on the walls of the artery(k). In a thinner tube, the pressure on the wall is lower (m). Baroreceptors transmit incorrect information (n), which leads to an increase in systemic blood pressure (h, g); 3.10) Dissection of the artery. The strong outer wall of the CCP can break away from the inner artery wall so that blood can flow between the CCP and the artery wall, forming a pathology - an arterial dissection.

ADS - arterial dissection; CCP- Cylindrical Cholesterol Plaque; DCCP-Dense Cylindrical Cholesterol Plaque; E -whole endothelium in a human artery; FC - inner fibrous cap (between plaque and the lumen of the artery); FC*- outer fibrous cap (between plaque and artery wall); FC**- «thin fibrous cap» (damaged by LC enzymes); FCCP-friable cylindrical cholesterol plaque; HI -healthy intima of the artery; Nal - normal artery layers; LC -lipid core with molten lipids in CCP; SCCP- soft cylindrical cholesterol plaque.

Annex 4. Name and description «Figure № 4». The mechanism of the pathological increase in the level of LDL in the blood depends on the formation and destruction of cylindrical cholesterol plaques (CCP) in the lumen of the artery.

4.1,4.2) a) the pressure is within the physiological standard. A slight compression of the vessel doesn't lead to a disturbance of flow of blood. The level of LDL is within the physiological standard; b) the pressure is above the physiological standard. The formation of strong obstacle to the flow of blood. The abrupt cross clamping creates an obstacle in the form of a ring; The accumulation of LDL in the vessel's walls in the places of cross-clamping and the abrupt reduction of level of free LDL in the blood; 4.3,4.4) b, c)During an increase in cholesterol plaques in length, the level of LDL in the blood decreases all the time; 4.5-4.6) d)The homeostasis system restores LDL levels in the blood to the level of physiological standard when plaque growth ceases in length; 4.7-4.8) e, f)The stoppage of spasm. The relaxation of vessels. The destruction of friable cylindrical plaque. The receipt of LDL from the plaques to blood. The Level of free LDL becomes higher the physiological standard. The complete dissolution of FCCP maximizes the level of LDL in blood. The homeostasis system does not synthesize LDL until its level in the blood is restored within the physiological range; 4.9,4.10) With prolonged relaxation of the vessel (a-a) - the level of LDL in the blood returns to the physiological norm; 4.11) Graph of the dependence of the level of LDL on the stages of contraction and relaxation of the vessel; g) Graph of changes in LDL levels in the blood depending on the state of contraction (spasm) or relaxation of the artery; h) Numbers of cells with figures; i) Condition of the vessel wall (compressed or normal, relaxed); J) Direction of change in the level of LDL in the blood (increasing or decreasing); k) The state of the level of LDL in the blood compared to the level of the physiological norm (above the level or below the level); l) the position of the figure in the table, in accordance with the schedule.

Annex 5. Name and description «Figure № 5». Four types of atherosclerotic lesions. Two types of ASL in human arteries, one type of ASL in arteries and veins in animals, and a «contemporary official description» of AS, which in reality is absent in the vessels of humans and animals.

5.1-5.4) Types I-IV atherosclerotic lesions in human arteries. Very rare atherosclerotic disease of the arteries. The LC is located far from the endothelium, close to the middle layer of artery. The MFCs are located close to the LC. From the lumen of the artery - there is an area of healthy intimate region without inflammation, without MFC, LDL, with a very small number of macrophages. LDL and macrophages enter the lipid nucleus only from the outer layer of the artery, through the «vasa vasorum»; 5.2-5.4) The longitudinal and transverse section of the types I-IV atherosclerotic lesions, looks like a tubercle that protrudes beyond the anatomical dimensions of the artery. Also looks like a tubercle protruding inward (into the lumen) of the artery in type IV atherosclerotic lesions. Always contains liquid lipids in the lipid core. Always limited to a few millimeters LC length. There is always an area of healthy intima near the endothelium. Always has a whole endothelium. Never has a fibrous cap. Always stretches the middle and outer layer of the artery. It always has blood vessels that start from the outer layer of the artery; 5.5-5.8) The types V-VI atherosclerotic lesions in human arteries (ASL V-VI). ASL V-VI - is a separate, main and most dangerous type of AS in humans, which poses real problems for the nutrition of the heart and brain, and which is operated on by vascular surgeons. ASL V-VI is an independent pathological structure that appears in a short period of time in the lumen of a healthy artery, consists entirely of LDL. The ASL V-VI is covered by a fibrous capsule both on the side of the lumen of the artery and on the side of the wall of the artery; 5.5) The wall of the artery that surrounds the ASL V-VI has a normal structure, without damages, inflammation, sprain and without necrotic nuclei. From the side of the blood, the ASL V-VI is gradually covered with a fibrous cap (FC). On the outside of the ASL V-VI, a fibrous layer is also formed between the ASL V-VI and the artery wall. The LC is located only inside the ASL V-VI wall; When FC is damaged from the blood, macrophages penetrate into the ASL V-VI wall, MFC and LC are formed. The LC is located only inside the ASL V-VI wall (a, b); Blood clot formation at the site of FC damage(c); 5.6-5.8) The longitudinal and transverse section of ASL V-VI. ASL V-VI is long, concentric, soft, strong, elastic, yellow, uniform structure, in the form of a tube with a hole in the middle, has a strong, elastic structure. CCP has a uniform color and structure, overlaps the lumen of the muscular and muscular-elastic artery along the perimeter and may contain LC located near the arterial lumen. The ASL V-VI is covered by a fibrous capsule both on the side of the lumen of the artery and on the side of the wall of the artery. The ASL V-VI may contain an LC located near the arterial lumen; 5.7) LC enzymes destroy the fibrous capsule - a «thin fibrous cap» is formed; 5.7) The destruction of the ASL V-VI wall causes the formation of an

embolus; 5.8) When the thin fibrous cap over the LC is ruptured, a thrombus may form; 5.9-5.12) Atherosclerotic lesions in animals (ASL Animal). The «original» description of AS in animals which describes the real processes occurring in the vessels of experimental animals. Damage to the intima of the vessels and endothelium occurs at the site of injury to the wall. The intima becomes loose and edematous in all vessels in which there is an endothelium and intima. LDL and macrophages penetrate into the inflamed, swollen, loose intima through the damaged endothelium. Macrophages absorb LDL and become MFC. The destruction of MFC causes the formation of LC, which contains liquid lipids and cholesterol crystals floating in them. The lipid nucleus is located just behind the damaged endothelium. The middle and outer layers of the artery are never stretched. The LC size increases only in the lumen of the artery. Such LCs occupy all areas of the inflamed intima. The LC increases in size and further occludes the arterial lumen. LC occludes the arterial lumen from one side. Each LC is several mm long; VV begin to sprout from the side of outer layer due to the inflammation; 5.10) damage of the walls with pure cholesterol and a large amount of LDL in the blood of genetically modified rats. Damage to the intima and endothelium occurs in all vessels of the animal (arteries and veins) (a, c, e). Local damage to the walls of the artery with a balloon causes local damage to the endothelium and intima (b, d, f); 5,12) when the inner surface of the intima (endothelium) is ruptured above one of the numerous LCs, a thrombus is formed in the arterial lumen. The endothelium above the lipid core is torn only by mechanical action on it. Emboli are absent; 5.13-5.14) The «contemporary official description» of AS, which describes precisely how AS «should look like in animals» to be «similar» to AS in humans. This kind of ASL does not exist in nature. It is not in the vessels of man and is not in the vessels of animals; 5.13) It's believed that endothelial cells become inflamed at one single point in the artery due to the large amount of LDL in the blood. According to the «contemporary official description» of AS - only the muscular arteries are inflamed. It's believed that macrophages and LDL pass through the damaged endothelium. MFC are formed. A single LC appears. The LC increases in size and protrudes into the arterial lumen from one side of the artery. The LC is several mm. long and protrudes above the inner surface in the arterial lumen. Above the LC is a damaged, inflamed, distended endothelium. Around the site of damage are absolutely healthy intima and endothelium. VV begin to sprout inside the plaque from the side of the outer layer of the artery due to inflammation. Muscle fibers migrate inside the plaque; Fibrin strands appear; It is believed that this LC grows for a very long time - for several decades. It is believed that during this time, several more such LCs develop nearby, which merge with each other and form a «tube» of a large number of LCs in the lumen of the arteria. It is believed that this «tube», which consists only of small sections of LC with molten lipids, can break away from the inner wall of the artery and withstand the long time pressure of blood during dissection of the artery or aorta. It is believed that it may not manifest itself for many decades and manifest itself only under stress, due to rupture of the endothelium and the formation of a thrombus or embolus; 5.14) It is believed that when a damaged, inflamed, stretched endothelium ruptures above the LA, a thrombus forms in the lumen of the artery. It is believed that when the damaged, inflamed, stretched endothelium over the LC ruptures, an embolus can form from the LC substances.

B - balloon; CC - cholesterol crystals; CH - pure cholesterol in the blood; DI - damaged artery intima; DI** - damaged the intima of the artery in animals; DI*** - damaged the intima of the artery (in «contemporary official description» of AS); DI**** - damaged the intima of the artery in animals after balloon injury; E - whole endothelium in a human artery; E* -endothelium between plaque and arterial wall in humans; E** - damaged endothelium in animals; E*** - damaged endothelium over the lipid core; EM - embolus; FC - internal fibrous capsule (in the lumen of the artery); FC*- outer fibrous capsule (between + plaque and artery wall); GI -glycocalyx; HI -healthy intima; LC -lipid core with molten lipids in human intima; LC *- lipid core with molten lipids in Cylindrical Cholesterol Plaque; LC** -lipid core with molten lipids in animal intima; LC*** -lipid core with molten lipids in intima (in «contemporary official description» of AS); LDL -low density lipoprotein; LDL* -low density lipoprotein in Cylindrical Cholesterol Plaque; M -single macrophages; M* - macrophages; MFC -macrophage foam cells in human intima; MFC* -macrophage foam cells in Cylindrical Cholesterol Plaque; MFC** - macrophage foam cells in animal intima; MFC*** - macrophage foam cells in intima (in «contemporary official description» of AS); Nal - normal artery layers; Nal** - normal layers of arteries after injury to the artery wall by toxic substances; Nal*** - normal artery layers (in «contemporary official description» of AS); Sal - stretched artery layers; Sal** - stretched layers of arteries after trauma to the artery wall with a balloon; SCCP- soft cylindrical cholesterol plaque; TR*- thrombus in the lumen of an artery after rupture of the fibrous cap

in humans; TR** - thrombus in the lumen of an artery after rupture of the endothelia in animals; TR*** - thrombus in the lumen of an artery after rupture of the endothelia (in «contemporary official description» of AS); VV - vasa vasorum in the arterial wall in human; VV** - vasa vasorum in the arterial wall in animals; VV** - vasa vasorum in the arterial wall (in «contemporary official description» of AS).

Table № 1. Questions to the contemporary «inflammatory theory»

Questions	**Figures**
Why there is no description of a soft, elastic, yellow, homogeneous atherosclerotic lesions (ASL), without lesions, without lipid core with molten lipids, which looks like a tube or cylinder inside a completely healthy artery, which is clearly visible on contemporary videos made during carotid artery surgery, on the heart's arteries, lower extremity arteries? ASL V-VI have a concentric form and have a lumen in the center of the ASL.[27-37, 45-49]	Fig. 1.1- 1.12, 2.1-2.7
Why, when examining with contrast agent, does the blood move shift away from the artery walls and flow through the center of the «tube»?[30,33,36,48,50]	Fig. 2.3,2.7
How did type V atherosclerotic lesions (ASL V) take an ideal concentric shape?[27-37,45-49]	Fig. 1.1- 1.12, 2.1-2.8
How does the ASL V-VI size increase from a few millimeters to tens of centimeters in ASL V-VI, without LC in the artery wall?[27-37, 45-49]	Fig. 1.1-1.8, 2.1,2.4
How long does it take for ASL V-VI to increase to a few tens of centimeters?	Fig. 1.1-1.8, 2.1,2.4
Why, before being removed from the artery, ASL V looks like a tube (cylinder) with flat and smooth walls, and also retains this shape after removal from the artery?[27-37, 45-49]	Fig. 1.1-1.12, 2.1-2.8
Why are there multilayer fibroatheromas whose LCs are overlapped and are separated by fibrous layers?[52]	
How can ASL V-VI grow into arterial branching?[27-37,46,47]	Fig. 1.2,1.4,1.6,1.8,1.9-1.11,2.1,2.5,2.9
Why, during surgery, can the artery be turned inside out and removed from the ASL like the stocking is slipped off the leg?[28, 29]	Fig. 2.6
Why does the ASL V-VI have the same strong yellow color on both the inside and the outside?[27-37, 45-49]	Fig. 1.1-1.11, 2.1,2.4
Why is there no bleeding from the vasa vasorum (VV) in case when ASL V-VI are easily detached along the entire length from the artery wall and easily are removed from the artery?[27-37, 45-49]	Fig. 1.10-1.2,2.1
Why do LC and bleeding are formed inside the wall of ASL V-VI?[51]	Fig. 1.1-1.6,1.8, 2.7
Why does the patient, after putting the stent in the entire affected area, after a short period of time, again have a lesion area in the form of concentric ASL V-VI, next to the stented area?[30]	Fig. 1.7
How, in ASL V-VI, fibrous cap (FC) appeared on the inner surface of ASL V-VI, instead of the normal endothelium and part of the healthy intima? How did the replacement take place, and what happened to the endothelium - was it destructed, or did it remain under FC? What happened to glycocalyx?[40,52]	Fig. 1,2.2,5.5-5.8
Why, when removing the ASL, a healthy artery wall is visible in this place without damage, necrosis, stretching, bleeding and signs of inflammation?[27-37]	Fig. 1.12,2.1
Why, after a large increase in the length of ASL V-VI and overlap of the vessel lumen in the form of a tube, the appearance of the artery and its dimensions in width have not changed?[27-37]	Fig. 1.9,2.1
Why is there no LC in the artery wall along the perimeter of the ASL V-VI?[27-37]	Fig. 1.11,1.12,2.1
Why do the learning materials which give guidance on how to carry out the Carotid endarterectomy depict the ASL V-VI having the form of a tube that repeats the contour of a normal artery without stretched wall? The artery wall has a look as it does in healthy people?[29,36]	

Why are only ASL V-VI depicted in all teaching materials on stenting of vessels?[29,36]	
Why there is no video dedicated to ASL I-IV?	
Why are machines for intra-arterial therapy designed in the form of cylinder or tube so that they can gradually cut layers of ASL V-VI without causing damage to the arterial wall?[53-56]	Fig. 2.8
Why are there no instruments and methods of surgery which can ensure the removal of ASL I-IV?	
Why, during surgery on the lower extremity arteries, the ring-shaped instrument can easily separate the long (about 15-20 cm) ASL V-VI from the artery wall and why can it be easily removed from the artery?[45]	
In what way the calcium is deposited within the healthy artery contour and not deposited in the «stretched tubercles» outside the contour of the artery that should be formed in ASL IV?[57]	Fig. 2.9-2.12
Why do people who lead healthy lifestyle and don't have bad habits suddenly experience problems with cardiovascular system and have heart attacks and strokes?[7]	
Why people who are at risk of AS development may not have any problems with malnutrition of the heart and brain for decades?[8,9]	
In what way does the stress affect the rapid development of ischemia and the rapid formation of ASL V-VI in absolutely healthy people, without bad habits with a healthy lifestyle? In accordance with the current theory, ASL IV develops for decades, slowly overlap the artery lumen and cannot rely on the stress factor?[10-26]	

Table № 2. Characteristics of atherosclerotic lesions of lesions in animals, humans, and «contemporary official description» of atherosclerosis

	Original version of AS in animals	***The description of ASL I-IV in human***	***The description of ASL V-VI in human (CCP)***	***«Contemporary official description» of AS (a form of ASL invented by someone, which is neither in the vessels of humans nor in the vessels of animals***
Appearance of each atherosclerotic lesions	*AS in animals* - («true» plaque in animals) is located in all vessels where there is endothelium and intima: In case of longitudinal section - in the case of toxic injury to the artery wall - the intima is edematous, friable, increased in size all over vessels. The endothelium	*The description of ASL I-IV in human* is located only in arteries with muscle fibers: In longitudinal section - no change is visible from the lumen of the artery during several decades, in the future, from the lumen of the artery appears a tubercle only a few millimeters long, which is always covered with a healthy endothelium. Did	*The description of ASL V-VI in human (CCP)* is located only in arteries with muscle fibers: In case of a longitudinal section, a soft, elastic, strong, homogeneous, yellow plaque in the form of a «tube» or «hollow cylinder» is observed inside the lumen of the artery. The plaque has a beginning and an end. Plaques can vary	*«Contemporary official description» of AS* - in a longitudinal section, according to the statement, there is one or more separate «tubercles» in the arterial lumen, that are covered with damaged endothelium. Each «tubercle» is a few millimeters in size. The endothelium and intima located near each «tubercle» are always intact and healthy. There are no changes outside the artery. Mergers «of several» tubercles

	is inflamed, not stretched, folded, remains in contact with blood. When the artery is injured by a balloon - the damage is located only at the point of injury by the balloon (Fig. 5.9-5.12)[40] In the transverse section - in case of injury to the artery wall - the external and middle layers of the artery remain unchanged. Intima is inflamed, swollen, loose, increased in size all around the perimeter of the vessel. The damaged endothelium is not stretched, is assembled into folds, is located in the lumen of the artery. There is a narrow opening for blood flow in the middle of the vessel. There are lesions with mast cells and LCs in the intima, from one or more sides. LCs and mast cells are located near the artery lumen just beyond the damaged endothelium. All the intima is filled with fluid, saturated with muscle cells and	not detect a single case which shows such a tubercle in the artery. Also, there are no photos of such a tubercle in the lumen of the artery. Outside the artery ASL IV should look like a tubercle protruding above the wall of the artery. Did not find a single photo or video showing such a tubercle outside the artery (Fig. 5.1-5.4)[39] In the transverse section - from one side of the artery, in the intima, on the boundary with the middle lining of the artery, mast cells accumulate and there is one single LC containing liquid lipids and cholesterol crystals There is always a healthy endothelium and a section of healthy intima from the side of lumen of an artery without mast cells and LC. In the process of development of the LC, which takes several decades, a stretching of the middle and outer layer of the artery takes place. VV sprout from the side of outer layer of artery. When the	in lengths starting from a few centimeters to several tens of centimeters. The plaque repeats the contours of the artery, and also completely repeats the contours of the branching of the arteries. Initially, immediately after the appearance, there are no areas of damage in the plaque, there are no LCs. The plaque is covered outside and inside with a fibrin layer. The inner fibrin layer located of the lumen of the artery has its own name - is called «fibrous cover» (FC). The outer layer of ASL is always smooth and follows the contours of the artery. The inner layer of ASL, immediately after formation, has a smooth surface and is not damaged. In case of FC damage macrophages enter the plaque. Mast cells and LC are formed below FC. LC enzymes dissolve FC - a «thin cap atheroma» is formed. When a thin FC is ruptured, a site of destruction is formed in the plaque, which can again be covered with a fibrous layer. The inner	lead to the formation of a strong, elastic, strong yellow «altered intima», which has a length of several centimeters to several tens of centimeters, repeats the contours of the vessels, and also repeats the branching of the vessels. How the loose, swollen, impregnated with fluid, muscle cells, fibrin fibers, mast cells, and LCs with fluid content, «altered intima» - became a homogeneous, strong, yellow, elastic plaque - no explanations in this regard. With aging, the «altered intima» is impregnated with calcium (Fig. 5.13-5.16)[41] In a transverse section, according to the statement, there is a «tubercle» covered with damaged, stretched endothelium from one side of the artery, stands to the side of the arterial lumen. The endothelium and intima located next to the «tubercle», along the perimeter of the artery and opposite to the LC, are always intact and healthy. There are mast cells and one LC inside the tubercle. The intima, within the «tubercle» next to the single LC, is impregnated with muscle cells and fibrous fibers. In the process of enlargement, LC grows only inside the lumen of an artery, and stretches only the

	fibrous fibers. When the artery is injured by a balloon, an additional inflammation of the middle and outer layer of the artery can occur, with the formation of VV, which begin in the outer layer of the artery and sprout into the intima (Fig. 5.9-5.12).[40]	LC increases in size, the LC grows towards the arterial lumen. Endothelium in the lumen of an artery, even in case of a strong overlap of the lumen of an artery, is always healthy (Fig. 5.1-5.4).[39]	surface of the plaque has many areas of damage. after repeated destruction of necrotic nuclei. In case of the accumulation of LDL in the areas of plaque destruction, and covering them again with a fibrous layer, multilayer fibroatheromas are formed. In case of prolonged destruction of the plaque, of the artery may appear in which the material of the plaque itself is partially absent. Such areas cause eccentric overlap of the artery due to the remaining pieces of the plaque. Replacement of LDL with calcium leads to plaque calcification (Fig. 1.1-1.12, 2.1-2.12, 5.5-5.8) [38-41] In case of transverse section, the plaque is located inside the arterial lumen. The outer wall of the plaque is tightly pressed to the inner surface of the artery and is in direct contact with the endothelium. The plaque has a thickness of several millimeters along the entire perimeter. The entire plaque consists of oxidized LDL.[57] All layers of	endothelium. The middle and outer shell of the artery isn't inflamed and damaged. According to the statement - VV sprout from the outer layer of the artery. Why VV appears in the absence of inflammation in the outer part of the intima, the middle and outer shell of the artery, no explanations in this regard. According to the statement, after a certain period of time, FC appears in the lumen of an artery instead of the endothelium, which completely covers the area above the LC. FC may become thin and rupture. How the whole replacement of endothelium for a fibrous cap occurs, no explanations in this regard? With the further development of the «tubercle» a thickening of the intima on the opposite arterial wall occurs. Why this happens- no explanations in this regard. With aging, the «altered intima» is impregnated with calcium (Fig. 5.13-5.16)[41]

			the artery are never damaged and retain their anatomical shape and size. The plaque is located along the entire perimeter of the artery and covers the lumen of the artery concentrically in the form of a ring immediately after formation. There is a hole for blood flow in the middle of the plaque. The plaque wall is homogeneous, has approximately the same thickness along the entire perimeter of the artery immediately after the appearance. With the «aging» of the plaque and the penetration of macrophages into the «body» of the plaque, mast cells and single LCs appear under the FC. LC always grows only inside the «body» of the plaque, and never increases into the lumen of the artery. When the thin FC is destroyed, the contents of the LC leak into the lumen of the artery. Rupture of a thin FC may cause thrombus formation in the lumen of the artery. In the place where there was LC, damage is formed in the form of a depression in	

			the «body» of the plaque. With the complete destruction of part of the «body» of the plaque, the formation of an eccentric overlap of the artery with the remains of the «body» of the plaque is possible. Replacement of LDL with calcium leads to plaque calcification (Fig. 1.1-1.12, 2.1-2.12, 5.5-5.8)[38-41]	
Entry of low density lipoproteins and macrophages into atherosclerotic lesions	*AS in animals* - when an artery is injured by toxic substances, macrophages and LDL enter the intima only through the damaged endothelium. In case of injury of artery by a balloon substances enter the intima through the endothelium and the VV (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - macrophages and LDL enter the intima only through the VV (Fig. 5.1-5.4)[39]	*The description of ASL V-VI in human (CCP)*- LDL don't enter the plaque, since the entire plaque consists of LDL. The entry of macrophages occurs only from the side of the lumen of an artery in case of the FC damage (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - the main entry of macrophages and LDL occurs only through the damaged endothelium. Additional intake of LDL and macrophages occurs through the VV (Fig. 5.13-5.16)[41]
Occlusion of lumen of vessel	*AS in animals* - at first concentric, later, with the growth of LC, eccentric (Fig. 5.9-5.12)[40]	*The description of ASL I-IV in human* is always eccentric (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - initially concentric, after destruction of ASL V-VI (CCP) - eccentric (Fig. 1-3,5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - at first eccentric, due to the development of one LC from one side of the artery wall, later, after edema of intima on the opposite side, it becomes concentric. How the thickening of the intima occurs on the opposite side of the artery, no explanations in this regard (Fig. 5.13-5.16)[41]

Formation of emboli	*AS in animals* - not formed (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - not formed (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - formed by tearing off a piece of an elastic, strong piece from the «body» of the plaque (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - are formed in case of plaque's rupture. How the LC, which is filled with liquid lipids, can form a dense embolus in case of endothelium rupture, no explanations in this regard (Fig. 5.13-5.16)[41]
Formation of blood clots	*AS in animals* - with the application of strong mechanical forces and rupture of the endothelium over the LC, blood clots may appear. The endothelium does not rupture on its own in animals (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - do not form, the endothelium over the LC never ruptures (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - can be formed when the FC breaks over the LC. Since there many damages on the inner surface of the «old» plaques, as well as the presence in the arteries of «unaffected» people of large areas of completely destroyed plaques, including already calcified ones, it can be assumed that the appearance of a blood clot during the rupture of each LC is not a necessary condition (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - are formed in case of rupture of the endothelium or fibrous cap over the LC. The formation of a thrombus in case of the rupture of a fictitious plaque is a fictitious process and cannot be the subject of analysis (Fig. 5.13-5.16)[41]
Dependence on high, systemic blood pressure	*AS in animals* - depends (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - no data found (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - depends on the narrowing of the lumen of the artery with a strong increase in AD (Fig. 3,5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - depends. How, in case of an increase in systemic arterial pressure, only a single tubercle can appear in only one artery and why it can appear and grow many decades before an increase in arterial pressure is determined in a patient, no explanations are this

				regard (Fig. 5.13-5.16)[41]
Influence of atherosclerotic lesions on the formation of persistent elevated systemic blood pressure	*AS in animals* - no data found (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - local occlusion of the arterial lumen does not affect systemic arterial pressure (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - affects due to a violation of the reflex connection in the presence of an additional «tube» in the lumen of an artery near the baroreceptors (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - has impact. How a single tubercle can affect systemic arterial pressure, no explanations in this regards (Fig. 5.13-5.16)[41]
The impact of factors and risks that have a systemic impact on all vessels	*AS in animals* - systemic «factors» and «risks» cause systemic toxic damage to all vessels where endothelium and intima is present and lead to the appearance of «true» plaque in animals (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - systemic «factors» and «risks» cannot in any way affect the appearance of one single LC in one place within the artery, located deep within the intima, at the border with the middle layer of the artery (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - toxic systemic «factors» and «risks» cannot influence the appearance of ASL that is not associated with inflammation of the artery wall. Only «factors» and «risks» that cause increased excitability of the central and autonomic nervous system may have an impact on the appearance of ASL V-VI (CCP). Toxic» systemic «factors» and «risks» can influence FC damage and destruction of an already formed plaque (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to the statement - systemic and non-systemic «factors» and «risks», for some reason, firstly, affect only one small section of the artery, and later, affect areas located near the first lesion. At the same time, the endothelium of all other vessels remains healthy. Why they do not have a systemic effect on all human vessels that contain intima and endothelium, as it happens in animals, no explanations in this regard. Why people who lead a healthy lifestyle and do not have «factors» and «risks» have problems with AS, no explanations in this regard (Fig. 5.13-5.16)[41]
Effect of elevated levels of low density lipoproteins in blood on the occurrence of atherosclerotic lesions	*AS in animals* - an elevated level of LDL causes a toxic systemic impact on all vessels where an endothelium and intima is present. As a result,	*ASL I-IV in human* - no impact, as there is no systemic manifestation. ASL I-IV - is a local manifestation. If an increase of LDL in the blood	*ASL V-VI in human (CCP)* - does not impact as there is no systemic manifestation. ASL V-VI (CCP) is a local manifestation. If an increase in LDL in the blood of a human caused a	*«Contemporary official description» of AS* - according to the statement - affects the appearance of a single lesion, in one vessel, and only in the artery. How systemic injury to vessels by a large amount of LDL

	inflammation appears in all the vessels of the animal (Fig. 5.9-5.12)[40]	of a human caused a systemic toxic impact on all vessels, ASL would be present in all vessels that have endothelium and intima (Fig. 5.1-5.4)[39]	systemic toxic impact on all vessels, ASL would be present in all vessels that have endothelium and intima (Fig. 5.5-5.8)[38-41]	caused only local damage in only one vessel and did not have a systemic effect, no explanations in this regard (Fig. 5.13-5.16)[41].
The impact of atherosclerotic lesions being in the vessel on increase in low density lipoproteins levels in blood	*AS in animals* - no impact (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - no impact (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - there is an impact. When ASL V-VI (CCP) is formed and dissolved, LDL levels in blood become above normal (Fig. 4,5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to a statement - has impact. Damage to the endothelium causes an increase in the amount of LDL in the blood for «repair». Any increase in consumption of substances in the body causes an increase in the synthesis to recuperate losses, but the level of substance in the blood is never above normal. How the «increase in LDL consumption» caused a completely different situation - «increase in LDL levels» in the blood above physiological norms - no explanations in this regard (Fig. 5.13-5.16)[41]
Presence of damaged endothelium and «fibrous cap» in the lumen of an artery	*AS in animals* - a damaged endothelium is always present. Never a FC (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - a healthy endothelium is always present from the side of arterial lumen. Never a FC (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - a FC from the side of the artery is always present. There is always a fibrous layer from the side of the artery wall. On the inner surface of the ASL V-VI (CCP) there may be areas with endothelium deposited from the blood. he endothelium of wall of the artery is always located between the «body» of plaque	*«Contemporary official description» of AS* - according to a statement - at first, there is a damaged, stretched endothelium in the artery, in the future, the endothelium is replaced by FC. How and when the replacement of damaged endothelium on FC occurs, no explanations in this regard (Fig. 5.13-5.16)[41]

			and the intima of artery (Fig. 2,5.5-5.8)[38-41]	
The inflammation of intima	*AS in animals* - after injury to artery wall- the intima is inflamed in all vessels where there is endothelium and intima. Endothelium and intima in animals are present both in the arteries and in the veins. Intima which is swollen, loose, impregnated with fluid, muscle cells and fibrin strands, has a large number of lesions with mast cells and LCs (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - the intima is always damaged at the border with the middle layer of the artery, there a single LC is formed. The layer of intima from the side of lumen of an artery is always healthy, without change (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - the intima is always healthy, located between the «body» of the plaque and the endothelium layer from one side and the middle layer of the artery from the other side (Fig. 5.5-5.8)[38-41]	«*Contemporary official description*» *of AS* - according to a statement - intima is damaged in one single place of artery for several millimeters. LDL and macrophages penetrate into the intima in this place, mast cells and LC are formed. All the processes occur immediately under the endothelium. Enlargement of the LC occurs only in the lumen of an artery. The intima, which is located from the opposite side of the artery, is absolutely healthy. Somehow, after many years, the intima located from the opposite side of the artery (opposite the LC) also increases in size. For many years, several more LCs appear near the first LC, which merge into one plaque of a few centimeters to several tens of centimeters long - «changed intima» appears. «Altered intima» occludes the lumen of an artery concentrically, repeats the contours of the artery and its branching. How intima increases in size on the opposite wall, opposite the LC with the formation of concentric lesion - no explanations in this regard (Fig. 5.13-5.16)[41]

Rupture of the endothelium and «fibrous cap»	*AS in animals* - the endothelium is not ruptured on its own in animals - a mechanical force is required to cause a rupture (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - a healthy endothelium is never ruptured (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - in case of «thin fibrous cap» over the site of formation of LC - FC is easily ruptured (Fig. 2,5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to a statement - damaged, stretched endothelium can be ruptured on its own, in the future, for unknown reasons, ASL is covered by FC, which can also be ruptured (Fig. 5.13-5.16)[41]
The dissection of aorta	*The AS in animals* - is conditioned by decay of elastic fibers and the destruction of the entire wall of an artery - the vessel walls exfoliate (intima remains unchanged) (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - such processes don't occur (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - separation of a strong, elastic, durable, homogeneous ASL V-VI (CCP) from the inner healthy wall of a healthy vessel occurs (Fig. 3,10,5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to a statement - for several decades a «altered intima» is formed consisting of a large number of merged LC containing liquid lipids, and inflamed intima. Intima which is inflamed, loose, swollen, contains a lot of fluid, muscle cells and fibrin strands, mast cells, LCs with liquid content, VV residues. How loose, swollen, inflamed, saturated with mast cells and LCs with liquid lipids, «altered intima» - can have a strong, rigid, durable structure, which can easily tear itself from the middle wall of the artery and withstand the increasingly strong blood pressure without breaking for a long time - no explanations in this regard (Fig. 5.13-5.16)[41]
The possibility of surgical intervention on the affected area	*AS in animals* - it is impossible to operate, as all lesions are inflamed, loose, swollen intima with a huge amount of fluid, mast cells and LCs all over	*ASL I-IV in human* - it is impossible to operate because all lesions are the only LC with liquid contents inside the intima surrounded by mast cells and	*ASL V-VI in human (CCP)* - is easily cut into parts, exfoliated, peeled, trimmed with drills and separated from the wall of an artery with instruments having a form of a ring, is	*«Contemporary official description» of AS* - according to a statement - somehow surgery is carried out. In practice, there is not a single video and training manual regarding the operation of

	vessels. It is impossible to separate the entire inflamed intima from the inner layer of the vessel without forming a wound surface (Fig. 5.9-5.12)[40]	VV, which stretched the inner and outer walls of an artery. ASL I-IV is only a few millimeters long (Fig. 5.1-5.4)[39]	easily removed from the lumen of an artery when the artery is cut without causing damage to the wall of the artery. In textbooks, in educational films regarding the removal of plaque in humans - only ASL V-VI (CCP) is shown (Fig. 2,8,5.5-5.8)[38-41]	«contemporary» plaque (Fig. 5.13-5.16)[41]
«Endothelial dysfunction»	*AS in animals* - injury to wall caused by «toxic substances» cause damage to the endothelium of all vessels. Endothelium cannot perform its function due to damage. «Endothelial dysfunction» will manifest itself in all vessels with damaged endothelium (Fig. 5.9-5.12)[40]	*ASL I-IV in human* - never there. Endothelium located over ASL, as well as near ASL, is always healthy (Fig. 5.1-5.4)[39]	*ASL V-VI in human (CCP)* - a cholesterol plaque located in the lumen of an artery, doesn't allow the endothelium to contact with the blood. The endothelium of wall is located between the outer wall of the ASL and the inner wall of the artery. After the injection of irritant into the lumen of an artery in the ASL area, the artery wall cannot respond to the irritant, as the endothelium is covered by the «body» of plaque. This gives the impression that the endothelium is damaged and doesn't respond to irritant. Thus, the presence of ASL V-VI (CCP) in the lumen of an artery, makes it possible to imitate «endothelial dysfunction» (Fig. 5.5-5.8)[38-41]	*«Contemporary official description» of AS* - according to a statement - the injury to the artery wall caused by factors results in damage to the endothelium only in the area of the only LC. In this case, the endothelium is always present, but it is inflamed for only a few millimeters above the LC. A healthy endothelium is always present on the rest part of artery wall around ASL. Thus, when injecting chemical irritants into this area of the artery, a healthy endothelium should perform its function fully. That is why the «contemporary» kind of AS can never have «endothelial dysfunction» (Fig. 5.13-5.16)[41]

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