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RESEARCH ARTICLE

Refutation of the Identity of the Contemporary Official Description of Atherosclerosis with the Real Atherosclerotic Lesions in Humans and Animals

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ABSTRACT

The "contemporary official" description of AS, according to the generally accepted theory, should completely coincide with vascular lesions in an animal and vascular lesions in humans. A study of AS in humans and animals has shown that humans have two separate types of lesions that are completely unrelated. Both species have their own characteristics (etiology, pathogenesis, appearance), which are completely different from each other. Lesions in humans also do not coincide with lesions in animals. The "contemporary" type of AS differs both from AS in animals and AS in humans, but at the same time creates the illusion of "identity" of these processes. In practice, the "contemporary" look cannot be found in the vessels of animals and humans. This article analyzes how the "contemporary" type of atherosclerotic lesion appeared, and how, for 100 years, the "contemporary" type could create an imitation of the "similarity" of completely different processes.

The key words

Atherosclerosis; LDL; Atherosclerotic plaque; Heart attack; Stroke; Pathogenesis of atherosclerosis; Stress, Atherosclerosis in animals; Atherosclerosis in humans.

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

According to the contemporary official theory of atherosclerosis, lesions in vessels of animals are completely identical to lesions in humans.[1] A contemporary official description of atherosclerosis was presented after conducting a long study of atherosclerosis, which sheds light on the appearance, etiology, pathogenesis, clinical manifestations, consequences for the organism, methods of prevention and treatment.[2-6] Scientists and doctors have been preventing and treating atherosclerosis in humans for 100 years with the help of this description. Author's previous articles[7-9] revealed that 2 separate types of atherosclerotic lesions that have nothing in common can be detected in humans.[8] Each type of lesion develops independently and is not converted into another. Each specie has its own characteristics that are completely different from each other. The first type of lesion according to the classification - types I–IV atherosclerotic lesions (ASL I–IV) look like a single small tubercle (containing one lipid core), which grows for several decades on the outer border of the intima, first strongly stretches the middle and outer lining of the artery and only then it blocks the arterial lumen from one side (eccentrically)[10]. The first type of lesion is so rare that it never occurs during surgery aimed at removal of "real" human atherosclerotic plaques.

The second type of lesion according to the classification - types V and VI atherosclerotic lesions (ASL V–VI)[10] ("real" human plaque) - a yellow, soft, homogeneous, strong, elastic "tube" located inside the lumen of the artery,[11-36] which, according to the theory proposed by the author, appears in the lumen in just a few minutes. The accumulation of low density lipoproteins (LDL) in front of the place of severe narrowing of the artery causes the formation of the "tube". The author suggests calling such a plaque "Cylindrical cholesterol plaque" (CCP).[7-9] Such a plaque is not connected to the arterial wall, it can be easily removed from the artery, easily cut and scraped off the wall. The wall of the artery, located near such a plaque, is absolutely healthy. A plaque does not have necrotic cores just after it's formation and it occludes the lumen of the artery so that the blood evenly departs from the walls of the artery (concentrically) and flows through the

middle of the plaque itself. Then, over time, it has necrotic cores. The destruction of such a plaque causes the formation of areas of eccentric arrangement, and the LDL remaining in the plaque are replaced by calcium. "Cylindrical cholesterol plaque" is involved in the increasing of the level of LDL in the blood, creates a persistent reflex increase in blood pressure due to improper operation of baroreceptors, and also creates an imitation of endothelial dysfunction.[7-9]

The study of atherosclerotic lesions (ASL) in animals showed that problems arise when it comes to the "identity" between ASL in animals and two types of ASL in humans. All lesions are 100% different from each other.[9] Traumatization of the endothelium in animals by various "factors" causes endothelial dysfunction and the subsequent appearance of inflammation in the intima of vessels. The intima swells, becomes loose, edematous, increases in volume, occludes the lumen of vessels concentrically, forcing blood to squeeze into a narrow opening, creating problems for the nutrition of organs. At the same time, the endothelial layer is never stretched, even in case of the formation of a large number of lipid core (LC) in the intima.[37] Also, the endothelium in animals rarely ruptures on its own.[38]

Traumatic factors affect not only the arteries of animals, but also cause damage to the intima of the veins, which also contain endothelium and intima. After injury of the endothelium of the veins, endothelial dysfunction appears in them. Just like it does in the arteries.[39-43] Elevated levels of LDL in the blood in genetically modified animals[5] cannot selectively affect the endothelium of the arteries and the endothelium of the veins. Swelling of the intima of arteries results in ischemia of organs, and swelling of intima of the veins leads to the stagnation of blood in the organs. Accumulation of mast cells and the appearance of a large number LC occurs in case of endothelial dysfunction.[44-47] The presence of lesions in the veins in experimental animals has not been studied. It was unable to find any studies for this request. Thus, there are a number of significant differences between lesions in humans and lesions in animals. The lesion in animals is systemic in nature, characterized by concentric occlusion of the lumen of all arteries and veins due to swollen, loose intima of all vessels. In humans, in the majority of cases, the lumen of the artery is occluded because of the accumulation of LDL. Such accumulation can be found only in arteries that have a muscular layer. Such plaques are never formed in the veins of humans.

The main objective of this article was to study and compare the contemporary official description of atherosclerotic lesions with real atherosclerotic manifestations in humans and animals and to determine whether the contemporary official description is reliable or not. Also, the study can help us to determine how the contemporary official description was able to create the appearance of "identity" between completely different vascular damages in humans and animals.

Initially, two completely different processes in human vessels were modified. Two different types of ASL were combined into one sequential ASL. Thus, 6 types of

lesions appeared in the classification, which was developed step by step, starting with 1 and ending with 6.[10] Such a combination allowed to easily transfer the "Inflammatory theory" to ASL in humans – "…long-term inflammation of the endothelium and intima in humans caused, first, the appearance of one LC, and then, over many decades, caused the appearance of a large number of LCs that "merged" into one concentric lesion..." Therefore, a soft, elastic, hard "real" plaque that vascular surgeons operate on is called "altered intima" by surgeons.

In order to create the appearance of "identity" between a combined lesion in humans and a lesion in an animal, it was necessary to address some more questions:

- initially, the location of the lesion in the artery must be eccentric (as the first type and the destroyed second type in humans);

- ASL in the beginning should be located only in one single place, the surrounding intima should be completely healthy (as the first type in a person);

- the development of the lesion should be long, take decades (as the first type in humans);

- subsequent development of the lesion results in the formation of a concentric artery occlusion (as the second type in a person after formation);

- the lesions should merge and form a large array of pathogens of plaque due to the "altered intima" (as the second type in a person after formation);

- inflammation inside the intima can occur only after damage to the endothelium caused by various traumatic factors and the appearance of endothelial dysfunction (as in animals);

- there should be no lesion in the veins (systemic manifestation) (as in case of animals);

- there should be no initial swelling of the intima of all vessels and concentric occlusion of all vessels (as in animals);

- there should be no development of a large number of LC in the loose, swollen intima without stretching the endothelium in the direction of the arterial lumen (as in animals);

- a fibrous layer should cover the inner layer of the plaque and rupture together with the formation of blood clots (like the second type in humans).

The contemporary official description was able to address all issues and create the appearance of "identity" between the lesions in animals and the two types of lesions in humans, but at the same time a completely new structure was formed. The contemporary official structure of ASL is not present in the vessels of animals and humans. How the contemporary official type of atherosclerosis (AS) was formed and how it was able to solve complex issues when it comes to "identity" is the subject of this study.

Original description of AS in animals

After injury to the arteries and veins walls, due to feeding with pure cholesterol, as well as a large amount of LDL in the blood of genetically modified rats, the endothelial cells become inflamed[1] throughout all the arteries

and veins of animals. Under the inflamed endothelium, the intima becomes swollen and inflamed. The damage to intima and endothelium occurs in veins and muscular arteries. The swollen intima causes a concentric occlusion of arteries and veins. The blood to and from the organs is disturbed. The increase of intima in volume occurs within a few weeks. Organs suffer from chronic ischemia. LDL and macrophages penetrate the inflamed, swollen, loose intima through the damaged endothelium. Macrophages take in LDL and evolve into macrophage foam cells (MFC). The subsequent MFC destruction causes formation of a LC, which contains liquid lipids and cholesterol crystals. A large amount of LC occupies all areas of the inflamed intima of arteries and veins. The LC increases in size and further occludes the lumen of the artery. When increasing LC occludes the lumen of the artery from one side. Each LC is several mm long. Due to inflammation - vasa vasorum (VV) begin to sprout from the side of the outer layer.[7-9] Muscle fibers migrate inside the intima. Also, fibrin strands are formed inside the intima. In case of rupture of the inner surface of the artery above the LC a thrombus is formed.[49-52] Calcium deposition leads to ASL calcification.[53-55] The color of the lesion at autopsy is white (Fig. 1.1–1.4).[56]

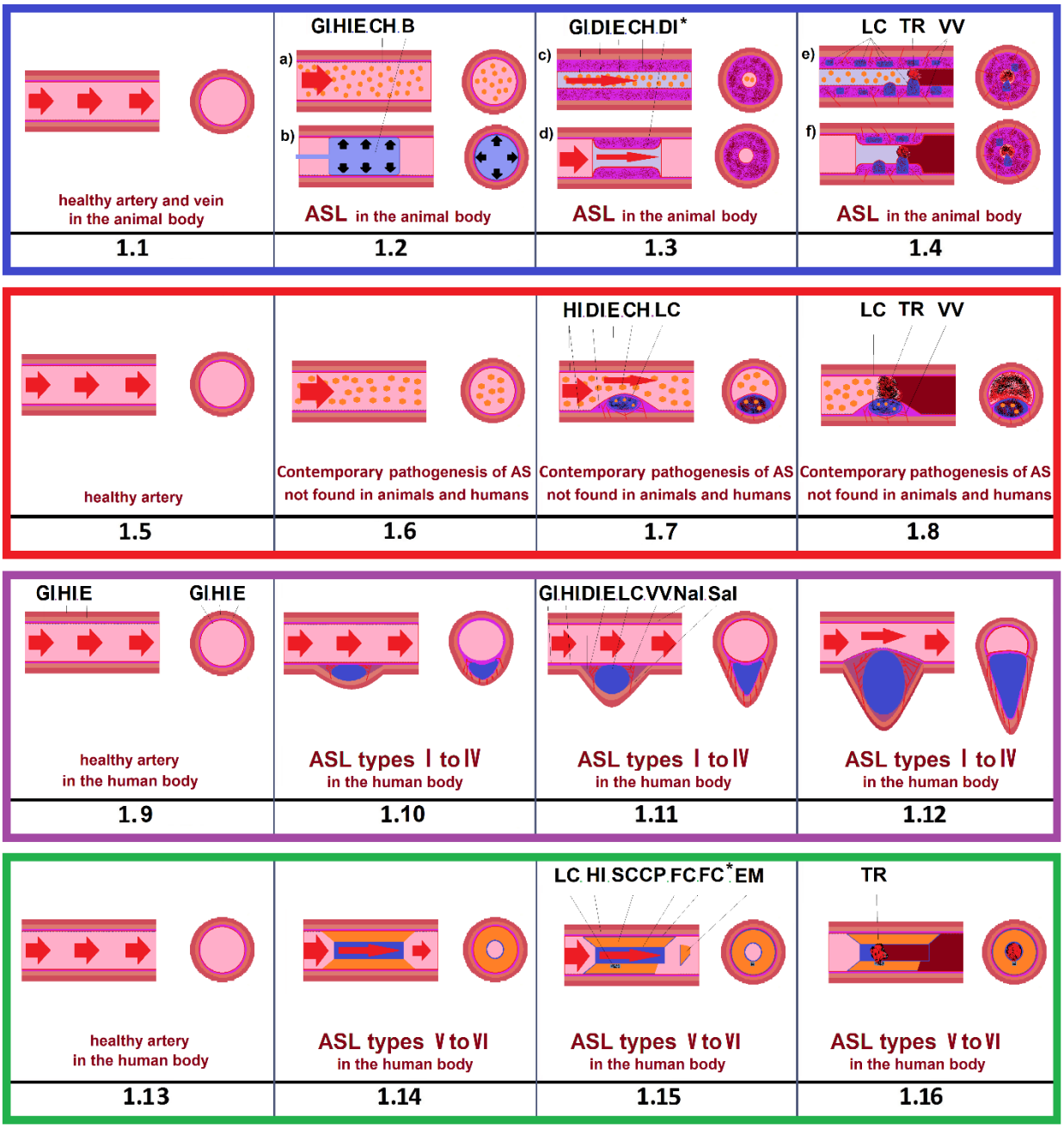

Figure 1.

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.

1.1–1.4) 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. 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. 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; 1.4) When the inner surface of the intima (endothelium) is ruptured above one of the numerous LCs, a thrombus is formed in the arterial lumen. Emboli are absent;

a,c,e – injury to the arterial walls and veins 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). b,d,f – local injury of the artery walls by the balloon.

1.5–1.8) The contemporary official description of AS, which describes precisely how AS "should look like in animals" to be "similar" to AS in humans. 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 - muscular arteries are inflamed. 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. There is an absolutely healthy intima around the injury site; 1.7) 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; 1.8) In case of rupture of the inner surface of the artery above the LC (endothelium) a thrombus is formed. Emboli are never present.

1.9–1.12) ASL I–IV – never seen on video, but described as the type of AS in humans. A focus of inflammation is formed in one single place of the muscular artery, inside the arterial wall, in the intima, near the inner layer. The endothelium covers the intima from the side of the arterial lumen. VV begin to sprout from the side of the outer layer due to inflammation, LDL and macrophages enter the intima through them. Macrophages absorb LDL and become MFC. The MFC destruction causes the formation of lipid core with molten lipids containing liquid lipids and cholesterol crystals floating in them. Growth is very long. The middle and outer shell becomes stretched. The dimensions of the LC must be very large before occluding the arterial lumen. Only after that the organs suffer from chronic ischemia. The endothelium is always healthy, never ruptures, a blood clot in the lumen of the artery is never formed. Emboli never happen;

1.13–1.16) 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. I suggest calling ASL V–VI "cylindrical cholesterol plaque" (CCP);

1.14–1.16) 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 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 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. The CCP may contain an LC located near the arterial lumen; 1.15) LC enzymes destroy the fibrous capsule – a thin fibrous wall is formed; 1.15) The destruction of the CCP wall causes the formation of an embolus 1.16) When the thin fibrous wall over the LC is ruptured, a thrombus may form.

B – balloon; CH – cholesterol; DI – damaged artery intima; DI* – damaged artery intima after balloon injury; E – endothelium; EM – embolus; FC – fibrous cap (in the lumen of the artery); FC – internal fibrous capsule; FC*– outer fibrous capsule; GI –glycocalyx; HI –healthy intima of the artery; LC – lipid core; NaI – normal artery layers; SaI – stretched artery layers; SCCP– soft cylindrical cholesterol plaque; TR – thrombus; VV – vasa vasorum.

The description of ASL I–IV in human

There is a separate type of AS in human vessels, which is defined as ASL I–IV in the classification. A focus of inflammation is formed in one single place of the muscular artery, inside the wall of the artery, in the intima, near the inner layer. The endothelium covers the intima from the side of the lumen of the artery. VV begin to sprout from the side of outer layer due to inflammation. LDL and macrophages enter the intima through them. Macrophages absorb LDL and become MFC. Macrophages take in LDL and become MFC. The MFC destruction causes a LC formation, containing liquid lipids and cholesterol crystals floating in them. Growth is long–term. Before closing the lumen of the artery, the dimensions of the LC must be very large. Only then do the organs suffer from chronic ischemia (Figures 1.9–1.12).[7-9,10]

The description of ASL V–VI in humans

There is a separate type of AS in human vessels, which in the classification is defined as ASL V–VI. Absolutely all ASL persisting in humans that pose real problems for the nutrition of the heart and brain, and which are operated on by vascular surgeons, belong to ASL V–VI. In order to distinguish this cholesterol plaque from the "classic" ASL in the animal and the very rare ASL I–IV in humans. I propose to name the main plaque which creates significant health problems in humans as ASL V–VI (the "cylindrical cholesterol plaque" - CCP).

CCP has a uniform color and structure, occludes the lumen of the artery from all sides along the perimeter of the artery, and may contain LC located near the lumen of the artery, which, unlike the LC in the animal, never protrudes into the lumen of the artery.[11-36] CCP may not contain any LC at all[7-9,12-15]; CCP is never present in human veins. It is located only in arteries that have muscle fibers; The CCP has a specific length and has a start and end location. The length can vary from a few centimeters (in the vessels of the heart)[17] to several tens of centimeters (in the lower extremities)[14]; CCP has 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[11-36]; When the CCP is exposed to various instruments, it can easily be separated from the arterial wall without the formation of wound surfaces and without bleeding. CCP can be cut with burs and cutting instruments without causing a bleeding and preserves a dense, uniform structure[15,24-28]; CCP can be easily "stretched" (crushed) with a help of balloon.[11,22,27] A healthy arterial wall is visible after removal of the plaque.[13,14,20,21,31,33-36] CCP can have branches that completely repeat the anatomical features of the arteries in which it is located.[11-14,17-19,57] The artery around the CCP may be turned inside out and be removed from the CCP like a stocking from a leg.[12,13] The outer and inner layers of the CCP have the same, uniform yellow color, smooth shiny appearance, completely following the contours of the artery. The outer surface that touched the wall of the artery has a flat, smooth, shiny surface. The inner surface of the CCP may be smooth or pitted and damaged.[11-36] The CCP is covered by a fibrous capsule both from the side of the arterial lumen and from the side of the artery wall.[56] Endothelium is never present in CCP from the side of the arterial lumen, and never has VV from the side of the arterial wall. CCP can "age", become destroyed, form emboli. The rupture of the thin fibrous cap over the LC may cause a formation of thrombus. Calcium deposition occurs in the remaining places after partial destruction of the CC.[56] The dissection of aorta causes a detachment of the CCP from the arterial wall, creating a path for blood flow between the outer wall of the CCP and the artery wall. CCP is composed of dead cells and an abundance of oxidized forms of low–density lipoprotein (oxLDL). It also contains immunocompetent cells in lesions producing mainly pro–inflammatory cytokines (Figures 1.13–1.16).[57] CCP goes through several stages of development: friable cylindrical cholesterol (FCCP) plaque, soft cylindrical cholesterol plaque (SCCP), dense cylindrical cholesterol plaque (DCCP), old cylindrical cholesterol plaque (OCCP).[7-9]

The contemporary official description of AS

After injury to the arteries walls, due to a large amount of LDL in the blood the endothelial cells become inflamed. Under the inflamed endothelium, the intima becomes swollen and inflamed. The damage to intima and endothelium occurs in muscular arteries. LDL and macrophages penetrate the inflamed, swollen, loose intima through the damaged endothelium. Macrophages take in LDL and evolve into MFC. The subsequent MFC destruction causes formation of a LC, which contains liquid lipids and cholesterol crystals. The LC increases in size and occludes the lumen of the artery. When increasing LC occludes the lumen of the artery from one side. LC is several mm long. Due to inflammation -VV begin to sprout from the side of the outer layer. Muscle fibers migrate inside the intima. Also, fibrin strands are formed inside the intima. In case of rupture of the inner surface of the artery above the LC a thrombus is formed. Calcium deposition leads to ASL calcification (Figures 1.5–1.8).

Questions to the contemporary official description of AS

After getting acquainted with the contemporary official description of AS, a number of questions arise:

– why is the contemporary official pathogenesis of AS described as a local malnutrition of the organ? In animals, the intima of the arteries and veins becomes inflamed, swollen, and the nutrition of all organs and systems is disturbed – all organs suffer immediately after injury. In humans, in case of ASL I–IV, only one organ suffers; in case of ASL V–VI, only one organ suffers;

– why is the contemporary official pathogenesis of AS described only as a malnutrition of organs and not described as a violation of outflow from organs? In animals, the intima of all veins swells and creates a violation of the outflow of blood from the organs. In humans, in case of ASL V–VI and ASL I–IV, there is no violation of the outflow of blood from the organs;

– why is the contemporary official pathogenesis of AS described as local damage to the endothelium with the presence of an absolutely healthy intima around the lesion in all other arteries and veins? In animals, AS is a systemic lesion affecting the intima and endothelium of absolutely all arteries and veins. In case of a person with ASL I–IV a local intima injury with healthy endothelium over the site of the lesion can be observed. In case of ASL V–VI, the intima and endothelium are always healthy;

– why is the contemporary official pathogenesis of AS described as an only one single LC? In animals, a large amount of LC is created in the intima of all arteries and veins in the inflamed, swollen intima. A person with ASL I–IV has only one single LC. With ASL V–VI, there may not be a single LC, or there may be several;

– why is the contemporary official pathogenesis of AS described as eccentric arterial occlusion? In animals, the inflamed intima of all arteries and veins immediately occludes the lumen of the vessel concentrically. In the process of development and increase in the size of the LC inside the inflamed intima it can additionally occlude eccentrically. In humans, in case of ASL the only one, single LC always occludes eccentrically. In ASL V–VI, the plaque always occludes concentrically; if destroyed, it may occlude eccentrically;

– why is the contemporary official pathogenesis of AS described as the penetration of macrophages and LDL through the damaged endothelium of arteries? In animals, they penetrate through the endothelium of all veins and arteries. In humans, in case of ASL I–IV they penetrate through the VV from the side of the outer layer of the artery. The endothelium is not damaged and nothing penetrates through it. In ASL V–VI, the entire plaque consists of LDL, which has entered the plaque directly from the blood. Macrophages enter the plaque only at the sites of rupture of the fibrous capsule. The endothelium is not damaged and nothing penetrates through it;

– why is the contemporary official pathogenesis of AS described as the result of

exposure to the increased levels of LDL in the blood (only on arteries). In animals with elevated LDL levels in the blood all veins and arteries are damaged (systemic manifestation). In a person with ASL I–IV the level may be normal. In ASL V–VI, the level may be normal, even in the presence of an increased level of LDL in the blood, the plaque forms only in the artery and has a certain length (no systemic damage);

– why is the contemporary official pathogenesis of AS described as the result of high cholesterol exposure? Animals being fed with high amounts of cholesterol were exposed to systemic damage to all arteries and veins (systemic damage). In case of a person with ASL I–IV blood cholesterol levels may be normal. In case of ASL V–VI the level may be normal. Even in case of an elevated level of cholesterol in the blood a person does not have a systemic lesion;

– why is the contemporary official pathogenesis of AS described as a process that develops over several decades? In animals, the intima was swelling a few weeks after the consumption of cholesterol, in case of being injured by a balloon – after a few days. In humans, ASL I–IV damage develops over several decades. In ASL V–VI, the plaque appears within a few days;

– why is the complication of "contemporary official pathogenesis of AS" causing thrombus formation described as endothelial rupture? In animals, endothelial rupture may occur causing thrombus formation. In humans with ASL I–IV the endothelium is never damaged, never torn. A thrombus is never formed. In ASL V–VI, the endothelium is located between the plaque and the arterial wall and is always intact. A thrombus is formed only in case of the fibrous capsule rupture from the side of arterial lumen;

– why, when describing the contemporary official pathogenesis of AS, such a complication as embolism is not described? In animals, the endothelium rupture doesn't cause the formation of embolus. In humans, with ASL I–IV, the endothelium is never damaged, and an embolus never forms. In ASL V–VI, the plaque may rupture causing the formation of an embolus;

– why do the contemporary official pathogenesis of AS use anti–inflammatory therapy as a preventive measure? In animals, such prevention works effectively. In humans, in case of ASL I–IV and ASL V–VI doesn't yield results;

– why in the process of treatment of contemporary official description of AS it is customary to use instruments that cut off the plaque itself and instruments in the form of a ring that separate the plaque from the wall. In animals, it is impossible to remove swollen intima in parts, and it is also impossible to remove it from the middle layer of the vessel.[56] In humans, ASL I–IV cannot be separated from the wall. In case of ASL V–VI, any instruments are suitable. The plaque is easily cut, peeled off, and easily moves away from the arterial wall.

Such questions arise at the present stage of medicine development because

doctors and scientists around the world unconditionally recognized the "similarity» between AS in an animal and AS in humans.[58] In all the following years, no one has verified the "similarity" between AS in an animal and AS in humans, and also no one has verified the "continuity of development" of the two types of AS in humans.

"Coincidences" between AS in an animal and true cholesterol plaque in humans

The discoverers of atherosclerosis (including Anichkov N.) can be easily understood. They saw that when studying true "original" AS in an animal and true cholesterol plaque in humans (ASL V–VI), there are many "coincidences":

Coincidence 1. Similar appearance. When it comes to an animal, an inflamed intima looks like a "tube" inside a vessel. In humans, the accumulation of LDL also looks like a tube inside a vessel (ASL V–VI);

Coincidence 2. Similar clinical picture - nutrition of organs is disturbed. In animals - by increasing the size of the intima in the form of a "tube". In humans - due to the accumulation of LDL in the form of a "tube";

Coincidence 3. The same place of development of LC - inside "tube". In animals - LC development in the inflamed intima (tube inside vessel). In humans – LC development in cholesterol plaque (tube inside vessel);

Coincidence 4. Similar rate of occurrence of ASL. In animals, the intima becomes inflamed in a few weeks. In humans, LDL also stick together within a week;

Coincidence 5. The same pathological composition of ASL. Macrophages, LDL, MFC and LC are found in ASL in animals and humans.

Theories of AS appearance

The study of animal models on rabbits, rats and other animals gave rise to the emergence of to a large number of theories concerning the appearance of AS. One of the main theories is called the "Inflammatory Theory", which fully explains the appearance of the inflammatory process inside the intima in animals under the influence of various "factors" and "risks".[57]

Questions about the possibility of using "inflammatory" and other similar theories in humans arise together with a more detailed and careful consideration of ASL in humans.

Researchers note that for "some strange reasons" anti–inflammatory therapy has no impact on the appearance of AS in humans[59,60] and, at the same time, ASL in humans appear under conditions that don't exist in experimental animals – strong emotional experience.[61-77] ASL are located differently in the vascular bed, have different appearance, different causes of appearance, and complications occur differently.[7-9] A large number of inconsistencies requires a comparison between ASL in animals and ASL in humans.

At the present stage, the characteristics of AS, which are described as "the current understanding of the pathogenesis of AS", have changed greatly compared to AS in animals. Systematic inflammation, swelling of

all arteries and veins, swelling of the intima around the ASL disappeared, but a long–term developing local lesion appeared with a unilateral location of the ASL in the arterial lumen.

At the same time, the characteristics of AS in humans also changed a lot. To date, only a solitary ASL located from one side of the arterial lumen is described (ASL I–IV), and absolutely nothing is mentioned about tubular plaque (ASL V–VI – CCP).

If you look closely at the description of the contemporary official description of AS, you can see that this kind of AS has characteristics that could make the description of AS in an animal a little more similar to ASL I–IV in humans. There is a single plaque with one LC, the intima near the plaque is normal, the endothelium is damaged only above the plaque, and is located only in the muscular arteries. LDL are absorbed by macrophages. MFC and LC are formed in the intima. The inner layer destruction causes thrombus formation.

The problem is that it is not possible to find a contemporary official description of AS in vessels in animals or in arteries in humans – it does not correspond to either real AS in an animal or two real types of AS in humans.

Where did this type of AS come from? In the process of comparing of the "original" AS in animals and the contemporary official description of AS, a lot of common characteristics can be observed. In animals, in case of removal of unnecessary elements that "create problems" for the identity of animal AS and human AS, a contemporary official description of AS appears.

How did the contemporary official description of atherosclerosis come about?

To create a "contemporary official" description of AS - "wrong" words and sentences were removed from the text of the "original" description of AS.

"After injury to the arteries ~~and veins~~ walls, due to ~~feeding with pure cholesterol, as well as~~ a large amount of LDL in the blood ~~of genetically modified rats,~~ the endothelial cells become inflamed ~~throughout all the arteries and veins of animals.~~ Under the inflamed endothelium, the intima becomes swollen and inflamed. The damage to intima and endothelium occurs ~~in veins and~~ muscular arteries. ~~The swollen intima causes a concentric occlusion of arteries and veins. The blood to and from the organs is disturbed. The increase of intima in volume occurs within a few weeks. Organs suffer from chronic ischemia.~~ LDL and macrophages penetrate the inflamed, swollen, loose intima through the damaged endothelium. Macrophages take in LDL and evolve into MFC. The subsequent MFC destruction causes formation of a LC, which contains liquid lipids and cholesterol crystals. ~~A large amount of LC occupies all areas of the inflamed intima of arteries and veins.~~ The LC increases in size and ~~further~~ occludes the lumen of the artery. When increasing LC occludes the lumen of the artery from one side. ~~Each~~ LC is several mm long. Due to inflammation - VV begin to sprout from the side of the outer layer. Muscle fibers migrate

inside the intima. Also, fibrin strands are formed inside the intima. In case of rupture of the inner surface of the artery above the LC a thrombus is formed. Calcium deposition leads to ASL calcification. ~~The color of the lesion at autopsy is white.~~"

As a result, the remnants of the edited "original" description match the contemporary official description of atherosclerosis.

"After injury to the arteries walls, due to a large amount of LDL in the blood the endothelial cells become inflamed. Under the inflamed endothelium, the intima becomes swollen and inflamed. The damage to intima and endothelium occurs in muscular arteries. LDL and macrophages penetrate the inflamed, swollen, loose intima through the damaged endothelium. Macrophages take in LDL and evolve into MFC. The subsequent MFC destruction causes formation of a LC, which contains liquid lipids and cholesterol crystals. The LC increases in size and occludes the lumen of the artery. When increasing LC occludes the lumen of the artery from one side. LC is several mm long. Due to inflammation -VV begin to sprout from the side of the outer layer. Muscle fibers migrate inside the intima. Also, fibrin strands are formed inside the intima. In case of rupture of the inner surface of the artery above the LC a thrombus is formed. Calcium deposition leads to ASL calcification."

The removal of "unnecessary" elements completely changed the meaning of the contemporary official theory of AS and made it more adapted and universal, giving answers to difficult questions:

– instead of a systemic process accompanied with swelling of the entire intima of all arteries and veins in an animal, AS became localized, no more than a few mm. in length;

– instead of damage to all veins and arteries in the animal, only damage to the muscular arteries appeared;

– in animals, instead of chronic ischemia of all organs, local ischemia appeared at the site of the plaque's appearance;

– instead of stagnation of blood caused by the swelling of the intima of the veins in the animal, a normal outflow from the organs appeared;

– instead of systemic inflammation of the intima of arteries and veins, impregnated with MFC and LC in the animal, one tubercle appeared with one LC, surrounded by a healthy intima;

– instead of concentric occlusion of the artery in case of inflammation of the entire intima of the animal, an eccentric occlusion of the artery appeared at the site of plaque localization;

– instead of the rapid appearance of swelling and inflammation of the intima of all vessels in the animal, a prolonged infiltration growth of the plaque into the lumen of the artery appeared.

Unfortunately, such a "stripped–down copy" is nothing more than a "fantasy on the topic of medicine" and has no scientific and practical basis. Thus, we can consider the contemporary official description of AS as a separate type of AS, which is not "identical"

with any of the previously described types of AS and is an artificially synthesized type of AS, which is necessary to simulate the "identity" between processes in organisms of animals and persons.

As it was described in the previous article, even the "original" description of AS in animals cannot in any way be used to study AS in humans. The same principle applies to the artificially created, contemporary official description of AS. The knowledge gained from these types of AS cannot in any way be used for the prevention, treatment and study of AS in humans.

Try to compare ASL I–IV in human with AS contemporary official description and "original" description AS in animals.

There are some properties of ASL I–IV that coincide with both the contemporary official description and the "original" description of AS in animals:

– ASL I–IV appears due to the inflammatory process;

– ASL I–IV is located in the intima;

– ASL I–IV has an endothelium in the arterial lumen.

There are properties of ASL I–IV that only coincide with the contemporary official description of AS:

– ASL I–IV looks like the only place in the artery where macrophages and MFC have been accumulated;

– ASL I–IV has one single LC;

– there is a healthy intima around ASL I–IV;

– ASL I–IV has a small length (a few mm.);

– ASL I–IV is located only in the muscular arteries;

– ASL I–IV grows for a very long time (tens of years).

There are some properties of ASL I–IV that don't coincide with both the contemporary official description and the "original" description of AS in animals:

– ASL I–IV stretches the middle and outer layer of the artery;

– ASL I–IV begins to occlude the arterial lumen only when it is greatly enlarged in size;

– ASL I–IV is replenished with LDL and Macrophages only through VV;

– ASL I–IV never has damaged endothelium;

– ASL I–IV never ruptures into the arterial lumen and does not form blood clots.

Thus, we can conclude that the contemporary official ("shortened") description of AS does not have a complete resemblance to ASL I–IV.

Let's compare ASL V–VI in humans with AS contemporary official description and "original" description AS in animals.

On numerous videos you can see the main "real" ASL in humans, which is operated both inside the artery with the help of special instruments and open surgical operations – ASL V–VI.[11-36] In previous articles, the author

has described in detail the appearance and characteristics of ASL V–VI. Let us compare them with AS with the contemporary official and "original" descriptions of AS in animals.[7-9]

There are properties characteristic of ASL V–VI that coincide both with the contemporary official description and the "original" description of AS in animals:

– ASL V–VI may contain macrophages, MFC, LC.

There are properties characteristic of ASL V–VI that only match the contemporary official description of AS:

– ASL V–VI is located only in the arteries of the muscular type;

– ASL V–VI when the inner layer is ruptured, a thrombus forms in the lumen of the artery.

There are properties of ASL V–VI that only coincide with the "original" description of AS in animals:

– ASL V–VI looks like a tube;

– ASL V–VI blocks the arterial lumen from all sides;

– ASL V–VI appears within a few days and weeks.

There are properties characteristic of ASL V–VI that don't coincide with both the contemporary official description and the "original" description of AS in animals:

– ASL V–VI consists of LDL;

– ASL V–VI may delaminate;

– ASL V–VI has a uniform yellow color;

– ASL V–VI appears after a nervous strain;

– ASL V–VI may not have LC;

– ASL V–VI is easily separated from the wall;

– ASL V–VI does not have VV;

– ASL V–VI does not cause bleeding when detached from the wall;

– ASL V–VI may retain its shape when removed from the artery;

– ASL V–VI has a smooth outer layer;

– ASL V–VI has a fibrous capsule from the side of the arterial lumen and from the side of the arterial wall;

– ASL V–VI in case of aging can be destroyed and form emboli;

– ASL V–VI has damage only from the side of the arterial lumen;

– ASL V–VI has branches that repeat the branches of the arteries;

– ASL V–VI is associated with high blood pressure;

– ASL V–VI has a different length ranging from 1 cm. to tens of centimeters.

Thus, it can be concluded that ASL V–VI share several similar characteristics with both the contemporary official description of AS and the "original" description of AS in animals. On closer examination, we can see that ASL V–VI has a lot of individual properties, and single coincidences are of a formal nature and do not confirm the identity of these processes in any way.

4 Separate types of AS

At the moment, using the obtained data, we can briefly describe 4 separate types of AS:

Type of AS №1: The "original" description of AS in animals, which describes the real processes occurring in the vessels in experimental animals when the walls of the vessels are injured in various ways (Figures 1.1–1.4);

Type of AS №2: The contemporary official description of AS, which describes how "AS should look like in animals" in order to "look like" AS in human AS. It is a completely fictional description of AS, which is designed to "combine" the characteristics of AS in an animal and a person (Figures 1.5–1.8);

Type of AS № 3: ASL I–IV – a type of AS in humans that is never seen on video, which is described only in the literature and illustrates a "similarity" of AS in humans and AS in animals. In theory, ASL I–IV is the predecessor of ASL V–VI (Figures 1.9–1.12);

Type of AS № 4: ASL V–VI – a real, separate and most dangerous for humans, type of AS that causes heart attacks and strokes in humans, which has nothing to do with AS in animals, with the contemporary official description of AS, and also has nothing to do with ASL I–IV. Described in the classification as a continuation of the development of AS in humans after ASL I–IV. There is no description in the literature of an "early" plaque (ASL V) in the form of a soft elastic tube, in which LCs are completely absent. It is an independent structure with its own etiology, pathogenesis, appearance, clinical manifestations, complications, prevention and treatment options (Figures 1.13–1.16). ASL V–VI – described in detail in previous articles by the author.[7-9]

Conclusion

The "contemporary understanding of the pathogenesis of AS" is based on the believe that the pathological processes in animal arteries occur in exactly the same way in human arteries. Over the past 100 years, no one has been able to verify this statement. As it was shown in previous articles, animals and humans have three completely different types of AS (one in animals and two in humans). This article has addressed the question of why the contemporary official description of AS is entirely differs from the description of the three independent types of atherosclerosis that actually occurs in animals and humans. As follows from the result of the analysis in the article, the "contemporary official" description of atherosclerosis is a modified (shortened) description of AS in animals, which, as a result of such a change completely changed the meaning and the nature of the "contemporary pathogenesis of AS". Such a "stripped–down copy" has no scientific and practical basis. We can consider the contemporary official description of AS a separate type of AS, which is not "identical" with any of the previously described types of AS and is an artificially synthesized type of AS, which is necessary to simulate the "identity" between processes in organisms of animals and persons. The contemporary official description of atherosclerosis is a description

that is completely divorced from the reality. Such artificial syndrome does not exist neither in animals, nor in humans. The "original" description of AS in animals cannot in any way be used to study AS in humans. The same principle applies to the artificially created, contemporary official description of AS. The knowledge gained from these types of AS cannot in any way be used for the prevention, treatment and study of AS in humans. For the treatment and prevention of atherosclerosis in humans, it is necessary to study the really dangerous to the human health atherosclerotic lesions of type V and VI, which we call "cylindrical cholesterol plaque".[7-9]

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Additional materials

All four types of AS are different from each other. The contemporary official description of AS sometimes "coincides" with AS (italics) in animals (highlighted in bold), sometimes "coincides" with one type of AS in humans (ASL I–IV) (highlighted in bold). But it is also can be seen that the contemporary official description has almost no "common properties" with the real type of human AS (ASL V–VI), which creates the main problems, and which is the main cause of heart attacks and strokes in the human arteries.

Table 1. Etiology and pathogenesis of the four types of AS

	"Original" model of AS in animals (Figures 1.1–1.4)	***Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)***	**Types ASL I–IV in humans (Figures 1.9–1.12)**	**Types ASL V–VI in humans (Figures 1.13–1.16)**
The cause of ASL	Injury with the help of a balloon, a great amount of cholesterol, LDL in the blood	*"Factors" and "risks", "elevated blood pressure", "adverse hemodynamic conditions", elevated LDL level in the blood*	Prolonged spasm of the artery wall, which caused inflammation inside the arterial wall	Strong nervous overstimulation. Catastrophes of local and global significance, unsuccessful "prevention" of the "contemporary" type of AS – "quitting smoking" and "weight loss"
The influence of elevated level of LDL on appearance of ASL	Yes. Only in genetically modified rats	*Yes.*	No.	No.
The influence of elevated pressure of appearance of ASL	No.	*Yes. The appearance of swirl zones causes "damage to the endothelium" and the appearance of inflammation in one place*	Yes. Prolonged compression causes inflammation in the artery wall	Compression of the artery much higher than "normal" causes accumulation of LDL and the appearance of plaque
The influence of "factors" and "risks" on the appearance of ASL	**Yes. Only artificial chemical and physical irritants**	***Yes. Only artificial chemical and physical irritants***	None. It doesn't have impact on appearance	No. It doesn't have impact on appearance
The impact of nervous overstimulation on the appearance of ASL	**No.**	***No.***	**No.**	Yes.

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
Mechanism of the appearance of ASL	In case of inflammation of the intima of all arteries and veins, the intima becomes loose, swollen. Macrophages and LDL penetrate through the endothelium, a lot of LC is formed	*Inflammation of the endothelium of the arteries. Macrophages and LDL penetrate through the endothelium, and one LC is formed. Sprouts into the arterial lumen. the "intima" is healthy around*	Local inflammation of all layers of the artery (intima, middle and outer layer). VV germinates from the outer layer. Elements penetrate through VV, causing the formation of one LC. Grows outward, stretches the outer and middle layer	LDL are accumulated in the form of a ring in front of the place of strong narrowing. Further increases in length in the form of a tube. Gradually, LDL stick together, forming a soft, strong, elastic tube in the arterial lumen
The rate of appearance and development of ASL	Several weeks (due to inflammation and swelling of the intima and the development of numerous LCs within the intima)	***Several decades***	**Several decades**	A loose, weak plaque forms in a few minutes, (due to the accumulation of LDL in front of the obstruction). After a few days, a dense, strong, elastic plaque is formed (due to LDL gluing together). After LDL gluing and FC formation, the plaque structure is only destroyed
How does the ASL influence the blood flow	Initially, it blocks the lumen only concentrically (due to intimal swelling), later eccentrically (due to the growth of LC)	***Always eccentrically. Grows long***	**Always eccentrically. Grows long**	Initially occludes the lumen only concentrically (due to adhering LDL inside the artery). In the future, eccentrically (due to the dissolution of the plaque). Growth of LC within the plaque does not affect the lumen of the artery

Table 2. Structure of the four types of AS

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
The appearance of ASL	Swollen, inflamed, loose vascular intima, impregnated with MFC and numerous LCs, with a large amount of fibrin and smooth muscle cells. The color of the lesion at autopsy is white	*Single tubercle towards the arterial lumen, covered with "damaged" endothelium containing one LC*	Single tubercle towards the outer and middle shell. These shells are stretched. Protrudes beyond the outer anatomical dimensions	Elastic, yellow, soft, strong, homogeneous tube inside the lumen of the artery. Follows the contours and branches of an artery
Systematic processes	Yes.	*No.*	No.	No.
The place of appearance of ASL	Systemically. All veins and arteries in an animal. Locally – only when injured by a balloon	*Locally. Only in arteries with a muscular layer*	Locally. Only in arteries with a muscular layer	Locally. Only in arteries with a muscular layer
What does the ASL consist of	Consists of swollen, loose, inflamed intima. It is impregnated with MFC and LC, muscle cells, calcium crystals, fibrin filaments. There are no free LDL, as they become absorbed immediately	*There is one LC There are many MFC around it. There are no free LDL, as they become absorbed*	There is one LC There are many MFC around it. There are no free LDL, as they become absorbed	Only from LDL, which are glued together with fibrous threads Inside and outside covered with a fibrin layer
The presence of inflamation	Yes. (only after injury caused to walls) (in veins and arteries)	*Yes. (only after injury caused to walls)*	Yes.	Firstly no. After the formation of plaque (inside a plaque)
The presence of elements - LDL	There are few free (are absorbed immediately). They come through endothelium	*There are few free (are absorbed immediately). They come through endothelium*	There are few free (are absorbed immediately). They come from the outer layer of an artery through VV	There are a lot of free LDL. The whole plaque fully consists of LDL
Where is LDL located	Only in macrophages and MFC (in veins and arteries)	*Only in macrophages and MFC in arteries*	Only in macrophages and MFC in artery	The whole plaque consists of LDL
The presence of elements - macrophages	There are a lot of them. They come through endothelium	*There are a lot of them. They come through endothelium*	There are a lot of Free MFC. The come from the	There are few of them. They come through the

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
			outer layer of artery through VV	damaged FC, stay near the site of damage of FC
The presence of elements – MFC	**A lot of them**	*A lot of them*	**A lot of them**	A few – near the place of rupture of FC
Where are macrophages and MFC located	Along the entire length of inflamed intima (in veins and arteries)	*In single LC in artery*	**In single LC in artery**	In places of rupture of FC, under FC
The presence of elements – LC	A lot of them	*One.*	**One.**	Near the damage of FC
Where the LC is located	Along the entire length of inflamed intima (in veins and arteries)	*In one single place in arterial wall*	**In one single place in arterial wall**	In places of rupture of FC, under FC
The presence of elements – calcium	**Yes.**	*Yes.*	No.	**Yes.**
The presence of elements – VV	Yes. (in veins and arteries)	*Yes. (in arteries)*	**Yes. (in arteries)**	No.
What is covered ASL from the side of the lumen of the vessel	**The intima is covered with damaged endothelium**	*The intima is covered with damaged endothelium*	**The intima is covered with damaged endothelium**	The plaque is covered with a fibrous cap. Intima is healthy. The endothelium is located between the plaque and the intima
The surface of ASL from the side of vessel's wall	Normal junction of the intima with the inner wall of veins and arteries	*Common junction of the intima with the inner wall of the artery*	**Common junction of the intima with the inner wall of the artery**	The plaque is externally covered with a fibrous layer, adjacent to the endothelium and intima of the artery
Stretching of vessel membranes	**No.**	*No.*	Yes. (middle and outer membranes)	**No.**
The length of ASL	Over the whole surface of all veins and arteries. Each LC within the inflamed intima is limited to a few mm in length	*Only a few mm.*	**Only a few mm.**	Only a few mm. From a few millimeters to tens of centimeters

Table 3. Clinical manifestations and implications of the four types of AS

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
Clinical manifestations	Chronic ischemia of all organs, impaired blood flow from all organs	*Chronic ischemia of one organ*	Asymptomatic for a long time, then chronic ischemia of one organ	Acute organ ischemia after a few days
Time of appearance of Chronic ischemia	Immediately after intima's swelling Increase with LC growth inside the lumen	***During the last 10–20 years***	**During the last 10–20 years**	In a few days after the beginning of nervous overstimulation
Symptoms of acute ischemia	**Thrombus. After endothelial rupture over LC**	***Thrombus. After endothelial rupture over LC***	None. The endothelium above the plaque is always intact	At the beginning – a few days after the beginning of nervous overstrain and the appearance of the plaque. During plaque aging: when FC ruptures above LC – thrombus; when part of the plaque is torn
The impact of "factors" and "risks" on development and appearance of implications	It is claimed that yes. Such factors are not present in animals. Only artificial irritants	*It is claimed that yes*	Yes. They strengthen the inflammatory process	Yes. It has impact on aging and destruction of plaque
Implication - embolism	**No.**	***No.***	**No.**	Yes.
Implication – thrombus	**Yes.**	***Yes.***	No.	**Yes.**
Implication - aortic dissection	**No.**	***No.***	**No.**	Yes.
Implication - unstable atheroma with thin lid	**No.**	***No.***	**No.**	Yes.
Implication - the damage to fibrous cap	**Absense of FC**	***Absense of FC***	**Absense of FC**	Yes.

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
Implication - endothelial damage	**Often.**	*Often.*	No.	No. Endothelium is located between the plaque and arterial wall
Implication - the malfunctions of chemoreceptors	**No.**	*No.*	**No.**	Yes.
Implication - the influence of the plaque itself on a persistent increase in systemic arterial pressure – a violation of work of the baroreceptors	**No.**	*No.*	**No.**	Yes.
Implication - increase of level of LDL	**No.**	*No.*	**No.**	Yes.
Implication - calcification	Along the entire length of intima (in veins and arteries)	*Along the entire length of intima*	No.	In places of destruction and dissolution of LDL. Repeats the contours of ASL

Table. 4 The prevention and treatment of the four types of AS

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
Treatment	Anti– inflammatory therapy	*Anti– inflammatory therapy*	Anti–inflammatory therapy	Drugs for increased nervous excitability and severe compression of the artery, removal of plaque from the artery
Prevention	Anti– inflammatory therapy	*Anti– inflammatory therapy. Elimination of factors and risks "quitting smoking", "weight loss", "treatment of type–2 diabetes"*	Anti–inflammatory therapy	Drugs for increased nervous excitability and strong compression of artery, stopping attempts to "quit smoking" and "lose weight", restoring the normal functioning of the nervous system in various ways
Treatment - the impact of statins on appearance of ASL	Affects in genetically modified rats	*Claimed to be yes*	No impact at all	Does not affect the appearance, growth and destruction of the plaque
Treatment - surgical treatment	No.	*No.*	No.	Yes.
Treatment - can be removed from an artery	Cannot be removed from vessels without damaging the artery wall	*Cannot be removed from vessels without damaging the artery wall*	Cannot be removed from vessels without damaging the artery wall	Easy to remove without damaging the artery wall
Treatment - tools to remove	No tools	*No tools*	No tools	A lot. Various: scrapers, cutters, rings, etc.
Treatment - possibility of removal while maintaining the shape	Cannot be removed	*Cannot be removed*	Cannot be removed	Easily removed. Retains shape
Treatment - ability to gradually remove (in layers)	No.	*No.*	No.	Yes, easily separated in pieces or whole without damage to the artery and the person

	"Original" model of AS in animals (Figures 1.1–1.4)	*Contemporary official description of AS. Is not present in humans, not in animals (Figures 1.5–1.8)*	Types ASL I–IV in humans (Figures 1.9–1.12)	Types ASL V–VI in humans (Figures 1.13–1.16)
Treatment - what does the inner wall of the "plaque" look like after removal?	The "plaque" cannot be removed. The inner layer consists of endothelium	*The "plaque" cannot be removed. The inner layer consists of endothelium*	The "plaque" cannot be removed. The inner layer consists of endothelium	The inner layer consists of endothelium. The inner wall of the plaque can be completely healthy – smooth, shiny, without bleeding and necrosis. With aging, there are places of damage from the lumen of the artery
Treatment - what does the outer wall of the "plaque" look like after removal?	The "plaque" cannot be removed	*The "plaque" cannot be removed.*	The "plaque" cannot be removed	The outer wall of the plaque can be completely healthy – smooth, shiny, without bleeding and necrosis
Treatment - what does the arterial wall look like after removal of plaque?	Wound surface with bleeding from the VV	*Wound surface with bleeding from the VV*	Wound surface with bleeding from the VV Cavity in the wall of the artery from the stretched middle and outer membranes from which liquid contents leaked	After removal of the plaque, the artery wall is completely healthy – smooth, shiny
Treatment - balloon expansion and stent fixation	No.	*No.*	No.	Yes.

References

1. Buja LM, Nikolai N. Anitschkow and the lipid hypothesis of atherosclerosis. *Cardiovasc. Pathol.* 2014. vol. 23. no. 3. P. 183–184. PMID: 24484612. DOI: 10.1016/j.carpath.2013.12.004.

2. Atherosclerosis. Wikipedia. [Internet] [reviewed 2022 Sept. 27]. Link

3. What Is Atherosclerosis? – NHLBI, IH. [Internet] [reviewed 2022 Sept. 27]. Link

4. Bergheanu SC, Bodde MC, Jukema JW. Pathophysiology and treatment of atherosclerosis : Current view and future perspective on lipoprotein modification treatment. *Neth Heart J.* 2017 Apr;25(4): 231–242. PMID: 28194698 PMCID: PMC5355390 DOI: 10.1007/s12471–017–0959–2.

5. Crowther MA. Pathogenesis of atherosclerosis. *Hematology Am Soc Hematol Educ Program.* 2005:436–41. PMID: 16304416. DOI: 10.1182/asheducation–2005.1.436.

6. Zhao Y, Qu H, Wang Y, et al. Small rodent models of atherosclerosis. *Biomed Pharmacother.* 2020 Sep;129:110426. Epub 2020 Jun 20. PMID: 32574973. DOI: 10.1016/j.biopha.2020.110426.

7. Rusanov S. The affection of the disturbance of the hydrodynamics of blood in case of stress on pathological increase of level of low density lipoproteins in blood. The formation of cylindrical plaques, and their participation in the development of acute ischemic disorders of heart and brain. *Med Hypotheses.* 2017 Sep;106:61–70. Epub 2017 Jul 3. PMID: 28818274. DOI: 10.1016/j.mehy.2017.07.001.

8. Rusanov S. New in the etiology, pathogenesis, prevention and treatment of atherosclerosis. The two different types of cholesterol plaques have nothing to do with each other. *Ann Circ.* 2021 6(1): 004–011. DOI: 10.17352/ac.000018.

9. Rusanov S. Atherosclerosis in Animals is a Separate Type of Atherosclerosis that has Nothing to do with the Two Types of Atherosclerosis in Humans. *Medical Research Archives* [S.l.]. v. 10, n. 4, apr. 2022. Date accessed: 07 Sept. 2022. DOI: https://esmed.org/MRA/mra/article/view/2760. Link

10. Stary H, Chandler A Dinsmore R, et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. *Arterioscler Thromb Vasc Biol.* 1995; 15: 1512– 1531 PMID: 7670967. DOI: 10.1161/01.atv.15.9.1512.

11. YouTube [Internet]. [place unknown]: Surg Spot. [Video], Karotidnaya endarterektomiya: hirurgicheskoe lechenie kak profilaktika ishemicheskogo insulta; 2020 Oct 11

[reviewed 2022 Sept. 27]; [7:51, 7,58,10:23, 11:04, 11:15, 19,50, 20:39, 21:02, 23:21] Available from: https://www.youtube.com/watch?v=e8–mohC5ChQ&ab_channel=SurgSpot Russian. Youtube

12. YouTube [Internet]. [place unknown]: Surgery Department. [Video], 1 Angioplastika: stentirovanie podklûčičnoj arterii 2. Karotidnaâ èndarterèktomiâ; [reviewed 2022 Sept. 27]; [2:21]. Available from: https://www.youtube.com/watch?v=2QeQD1iLAvI&ab_channel=SurgeryDepartment English. Youtube
13. YouTube [Internet]. [place unknown]: Nejrokhirurgiya. [Video], Operatsiya – karotidnaya ehndarterehktomiya 2; [reviewed 2022 Sept. 27]; [1:21–1:36] Available from: https://www.youtube.com/watch?v=1WSyddN4HpA English. Youtube
14. YouTube [Internet]. [place unknown]: Galina Skokova. [Video], Endarterectomy; 2012 Jul 2 [reviewed 2022 Sept. 27]; [1:57, 9:19, 13:50]. Available from: https://youtu.be/R8dVYf–LV44 English. Youtube
15. YouTube [Internet]. [place unknown]: Tech Insider. [Video], Devices 'eat' bad cholesterol; [reviewed 2022 Sept. 27]; [0:40, 0:54] Available from: https://www.youtube.com/watch?v=–1ybFwlbtDI English. Youtube
16. YouTube [Internet]. [place unknown]: TAUVOD. [Video], Eradication of Atherosclerosis;[reviewed 2022 Sept. 27]; [8:40] https://www.youtube.com/watch?v=OA17PM4KBuM&ab_channel=TAUVOD English. Youtube
17. YouTube [Internet]. [place unknown]: CTSNetVideo. [Video], Coronary Endarterectomy of the Left Anterior Descending Artery; [reviewed 2022 Sept. 27]; [2:50, 4:56] Available from: https://www.youtube.com/watch?v=4A9C5fnaRho&t=8s English. Youtube
18. File: Carotid Plaque.jpg [Internet] [reviewed 2022 Sept. 27]. Link
19. YouTube [Internet]. [place unknown]: CTSNetVideo. [Video], Triple Coronary Endarterectomy in a Patient Undergoing CABG; 2019 Jan 29 [reviewed 2022 Sept. 27]; [5:11] Available from: https://www.youtube.com/watch?v=tol4O8U9Tsc&t=305s English. Youtube
20. YouTube [Internet]. [place unknown]: Alvin Wang. [Video], Carotid endarterectomy; 2007 Jul 17 [reviewed 2022 Sept. 27]; [01:40–03:22]. Available from: https://www.youtube.com/watch?v=ImHe–7RoqVc English. Youtube
21. YouTube [Internet]. [place unknown]: NSPC Brain & Spine Surgery. [Video], CAROTID SURGERY; 2020 May 26 [reviewed 2022 Sept. 27]; [3:47–7:05] Available from: https://www.youtube.com/watch?v=_qPtAWXJEs8 English. Youtube
22. YouTube [Internet]. [place unknown]: Dr. Sandeep Burathoki. [Video], Carotid

Artery Stenting video 2012 Nov 16 [reviewed 2022 Sept. 27]; [2:00–2:30] Available from: https://www.youtube.com/watch?v=83uSylrTEHg English. Youtube

23. YouTube [Internet]. [place unknown]: UCLA Health. [Video], Carotid Artery Disease & Stroke Angioplasty – Dr. May Nour | UCLAMDChat; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=b03GSL13QWs&t=369s English. Youtube
24. YouTube [Internet]. [place unknown]: BusinessWire. [Video], Covidien Announces Final Results from Landmark DEFINITIVE LE Study; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=LJuNgeisRas English. Youtube
25. YouTube [Internet]. [place unknown]:BD. [Video], Rotarex™ Rotational Excisional Atherectomy System; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=AwvdlJE6zRE English. Youtube
26. YouTube [Internet]. [place unknown]: Philips Image Guided Therapy Devices. [Video], Atherectomy; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=Vjno6WbBD_c English. Youtube
27. YouTube [Internet]. [place unknown]: Intact Vascular. [Video], Tack Endovascular System® mechanism of action (MOA); 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=tvcKFWXdvr0 English. Youtube
28. YouTube [Internet]. [place unknown]: ciro. [Video], rotablador; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/results?search_query=rotablador English. Youtube
29. YouTube [Internet]. [place unknown]: Navigating Radiology. [Video], Doppler Ultrasound Part 1 – Principles (w/ focus on Spectral Waveforms); 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=4pipx9hhl9g English. Youtube
30. YouTube [Internet]. [place unknown]: Icahn School of Medicine. [Video], The Mount Sinai Surgical Film Atlas: Carotid Endarterectomy; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=Oa8XV27KNAY English. Youtube
31. YouTube [Internet]. [place unknown]: Houston Methodist DeBakey CV Education. [Video], Carotid Endarterectomy – CEA (Maham Rahimi, MD, Travis Vowels, MD, Thomas Loh, MD); 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=AWXCpRJAN8M&t=425s English. Youtube
32. YouTube [Internet]. [place unknown]: Houston Methodist DeBakey CV Education. [Video], Carotid Endarterectomy (CEA) Part 2 (ALAN B. LUMSDEN, MD); 2018 July 2 [reviewed 2022 Sept. 27]; Available from:

https://www.youtube.com/watch?v=E_wWpRKBy4w&t=205s English. Youtube

33. YouTube [Internet]. [place unknown]: Barrow Neurological Institute. [Video], Carotid Endarterectomy Requiring Intra–arterial Shunting; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=AUa–NdnszXQ English. Youtube

34. YouTube [Internet]. [place unknown]: Icahn School of Medicine. [Video], The Mount Sinai Surgical Film Atlas: Carotid Endarterectomy; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=Oa8XV27KNAY English. Youtube

35. YouTube [Internet]. [place unknown]: MMCTS. [Video], Pulmonary endarterectomy – an example of treatment of right ventricular after load failure; 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=BYpk_AqzBxs&t=131s English. Youtube

36. YouTube [Internet]. [place unknown]: SurgMedia. [Video], Carotid Endarterectomy (CEA); 2018 July 2 [reviewed 2022 Sept. 27]; Available from: https://www.youtube.com/watch?v=De8WjNR6aJs English. Youtube

37. Qi J, Ping R, Zhang S, Xu Y, Wu K, Li Y. Effects of Cervical Rotatory Manipulation (CRM) on Carotid Atherosclerosis Plaque in Vulnerability: A Histological and Immunohistochemical Study Using Animal Model. *Biomed Res Int.* 2019 Feb 4;2019:3793840. doi: 10.1155/2019/3793840. PMID: 30863777; PMCID: PMC6378770.

38. Shiomi M, Fan J. Unstable coronary plaques and cardiac events in myocardial infarction-prone Watanabe heritable hyperlipidemic rabbits: questions and quandaries. *Curr Opin Lipidol.* 2008 Dec;19(6):631-6. doi: 10.1097/MOL.0b013e3283189c18. PMID: 18957890

39. Lopes RD, Mehta RH, Hafley GE, Williams JB, Mack MJ, Peterson ED, Allen KB, Harrington RA, Gibson CM, Califf RM, Kouchoukos NT, Ferguson TB Jr, Alexander JH; Project of Ex Vivo Vein Graft Engineering via Transfection IV (PREVENT IV) Investigators. Relationship between vein graft failure and subsequent clinical outcomes after coronary artery bypass surgery. *Circulation.* 2012 Feb 14;125(6):749-56. doi: 10.1161/CIRCULATIONAHA.111.040311. Epub 2012 Jan 11. PMID: 22238227; PMCID: PMC3699199.

40. Ward AO, Caputo M, Angelini GD, George SJ, Zakkar M. Activation and inflammation of the venous endothelium in vein graft disease. *Atherosclerosis.* 2017 Oct;265:266-274. doi: 10.1016/j.atherosclerosis.2017.08.023. Epub 2017 Aug 24. PMID: 28865843.

41. Kim FY, Marhefka G, Ruggiero NJ, Adams S, Whellan DJ. Saphenous vein graft disease: review of pathophysiology, prevention, and treatment. *Cardiol Rev.* 2013 Mar-Apr;21(2):101-9.

doi: 10.1097/CRD.0b013e3182736190. PMID: 22968180.

42. Yamawaki H, Iwai N. Mechanisms underlying nano-sized air-pollution-mediated progression of atherosclerosis: carbon black causes cytotoxic injury/inflammation and inhibits cell growth in vascular endothelial cells. *Circ J.* 2006 Jan;70(1):129-40. doi: 10.1253/circj.70.129. PMID: 16377937.

43. Qi J, Ping R, Zhang S, Xu Y, Wu K, Li Y. Effects of Cervical Rotatory Manipulation (CRM) on Carotid Atherosclerosis Plaque in Vulnerability: A Histological and Immunohistochemical Study Using Animal Model. *Biomed Res Int.* 2019 Feb 4;2019:3793840. doi: 10.1155/2019/3793840. PMID: 30863777; PMCID: PMC6378770.

44. Yuan W, Xu C, Li B, Xia H, Pan Y, Zhong W, Xu L, Chen R, Wang B. Contributions of Costimulatory Molecule CD137 in Endothelial Cells. *J Am Heart Assoc.* 2021 Jun;10(11):e020721. doi: 10.1161/JAHA.120.020721. Epub 2021 May 22. PMID: 34027676; PMCID: PMC8483511.

45. Swystun LL, Liaw PC. The role of leukocytes in thrombosis. *Blood.* 2016; 128:753–762. DOI: 10.1182/blood-2016-05-718114.

46. Slotta JE, Braun OO, Menger MD, Thorlacius H. Capture of platelets to the endothelium of the femoral vein is mediated by CD62P and CD162. *Platelets.* 2009 Nov;20(7):505-12. doi: 10.3109/09537100903215417. PMID: 19852690.

47. Castro-Ferreira R, Cardoso R, Leite-Moreira A, Mansilha A. The Role of Endothelial Dysfunction and Inflammation in Chronic Venous Disease. *Ann Vasc Surg.* 2018 Jan;46:380-393. doi: 10.1016/j.avsg.2017.06.131. Epub 2017 Jul 6. PMID: 28688874.

48. Getz GS, Reardon CA. Animal models of atherosclerosis. *Arterioscler Thromb Vasc Biol.* 2012 May;32(5):1104–15. Epub 2012 Mar 1. PMID: 22383700. PMCID: PMC3331926. DOI: 10.1161/ATVBAHA.111.237693.

49. Nakashima Y, Plump A, Raines E, et al. ApoE – deficient mice develop lesions of all phases of atherosclerosis throughout the arterial tree. *Arterioscler Thromb.* 1994 Jan;14(1):133–40. doi: 10.1161/01.atv.14.1.133. PMID: 8274468.

50. Orekhov A, Sobenin I. Modified lipoproteins as biomarkers of atherosclerosis. *Frontiers In Bioscience, Landmark.* 2018 23, 1422–1444, March 1, 2018. PMID: 29293443. DOI: 10.2741/4653

51. Ross R. Atherosclerosis – an inflammatory disease. *N Engl J Med.* 1999; 340:115–26. PMID: 9887164. DOI: 10.1056/NEJM199901143400207

52. Libby P, Ridker P, Hansson G. Inflammation in atherosclerosis from pathophysiology to practice. *J Am Coll Cardiol.* 2009 54: 2129–2138. PMID: 19942084. PMCID: PMC2834169.

DOI: 10.1016/j.jacc.2009.09.009.

53. Zhang X, Chen X, Liang Z, et al. Atorvastatin Promotes Macrocalcification, But Not Microcalcification in Atherosclerotic Rabbits: An 18F–NaF PET/CT Study. J *Cardiovasc Pharmacol.* 2021 Oct 1;78(4):544–550. PMID: 34651601. DOI: 10.1097/FJC.0000000000001088.

54. Aldrighi J, Lanz J, Mansur A, et al. Effect of sexual steroids on the calcium content of aortic atherosclerotic plaque of oophorectomized rabbits. *Braz J Med Biol Res.* 2005 May;38(5):705–11. Epub 2005 May 25. PMID: 15917951. DOI: 10.1590/s0100–79x2005000500007.

55. Aboshady I, Cody D, Johnson E, et al. Flat–panel versus 64–channel computed tomography for in vivo quantitative characterization of aortic atherosclerotic plaques. *Int J Cardiol.* 2012 May 3;156(3):295–302. PMID: 21185613 PMCID: PMC3074048. DOI: 10.1016/j.ijcard.2010.11.011.

56. Catanozi S, Rocha J, Passarelli M, et al. Pitfalls in the assessment of murine atherosclerosis. *Braz J Med Biol Res.* 2009 Jun;42(6):471–5. PMID: 19448893. DOI: 10.1590/s0100–879x2009000600001.

57. Frostegård J. Immunity, atherosclerosis and cardiovascular disease. *BMC Med.* 2013 May 1;11:117. PMID: 23635324. PMCID: PMC3658954. DOI: 10.1186/1741–7015–11–117.

58. Steinberg D. Thematic review series: the pathogenesis of atherosclerosis. An interpretive history of the cholesterol controversy: part *I. J. Lipid Res.* 2004. vol. 45. no. 9. P. 1583–1593. PMID: 15102877. DOI: 10.1194/jlr.R400003–JLR200.

59. Ota H, Omori H, Kawasaki M, et al. Clinical impact of PCSK9 inhibitor on stabilization and regression of lipid–rich coronary plaques: a near–infrared spectroscopy study. *Eur Heart J Cardiovasc Imaging.* 2022 Jan 24;23(2):217–228. PMID: 33637979. DOI: 10.1093/ehjci/jeab034.

60. Salvayre R, Negre–Salvayre A, Camaré C. Oxidative theory of atherosclerosis and antioxidants. *Biochimie.* 2016 Jun;125:281–96. Epub 2015 Dec 21. PMID: 26717905. DOI: 10.1016/j.biochi.2015.12.014.

61. Chevalier P, Dacosta A, Defaye P, et al. (1998) Arrythmic cardiac arrest due to isolated coronary artery spasm: Long–term outcome of seven resuscitated patients. *J Am Coll Cardiol.* 31:57–61 PMID: 9426018. DOI: 10.1016/s0735–1097(97)00442–7

62. Connelly K, MacIsaac A, Jelinek V. Stress, myocardial infarction, and the "tako–tsubo" phenomenon. *Heart.* 2004 Sep;90(9):e52. PMID: 15310721. PMCID: PMC1768425. DOI: 10.1136/hrt.2004.038851

63. Lowenstein C, Dinerman J, Snyder S . (1994) Nitric oxide: a physiologic messenger. *Ann Intern Med.* 120:227–237. PMID: 8273987. DOI: 10.7326/0003–4819–120–3–199402010–00009

64. Leor J, Poole W, Kloner R. Sudden cardiac death triggered by an earthquake. *N Engl J*

Med. 1996; 334:413– 419 PMID: 8552142. DOI: 10.1056/NEJM199602153340701

65. Kark JD, Goldman S, Epstein L. Iraqi missile attacks on Israel. *JAMA*.1995;273:1208–10. PMID: 7707629. DOI: 10.1001/jama.273.15.1208

66. Witte D, Bots M, Hoes A, et al. Cardiovascular mortality in Dutch men during 1996 European football championship: longitudinal population study. *BMJ.* 2000 Dec 23–30;321(7276):1552–4. PMID: 11124170. PMCID: PMC27557. DOI: 10.1136/bmj.321.7276.1552

67. Allegra J, Mostashari F, Rothman J, et al. Cardiac events in New Jersey after the September 11, 2001, terrorist attack. *J Urban Health.* 2005;82(3):358–63. PMID: 16000653. PMCID: PMC3456051. DOI: 10.1093/jurban/jti087

68. Feng J, Lenihan D, Johnson M, et al. Cardiac sequelae in Brooklyn after the September 11 terrorist attacks. *Clin Cardiol.* 2006;29 (1):13–7. PMID: 16477772. PMCID: PMC6654218. DOI: 10.1002/clc.4960290105

69. Rosengren A, Hawken S, Ounpuu S, et al. INTERHEART investigators. Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART study): case–control study. *Lancet.* 2004 Sep 11–17;364(9438):953–62. PMID: 15364186. DOI: 10.1016/S0140–6736(04)17019–0

70. Shen B, Avivi Y, Todaro J, et al. Anxiety characteristics independently and prospectively predict myocardial infarction in men: the unique contribution of anxiety among psychologic factors *J Am Coll Cardiol.* 51 (2008), pp. 113–119 PMID: 18191733. DOI: 10.1016/j.jacc.2007.09.033

71. Mittleman M, Maclure M, Sherwood J, et al. Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. *Circulation.* 1995 Oct 1;92(7):1720–5. PMID: 7671353. DOI: 10.1161/01.cir.92.7.1720

72. Allison T. Identification and treatment of psychosocial risk factors for coronary artery disease. *Mayo Clin Proc.* 1996 Aug;71(8):817–9. PMID: 8691905. DOI: 10.1016/S0025–6196(11)64849–0

73. Tsouna–Hadjis E, Mitsibounas D, Kallergis G, et al. Autonomic nervous system responses to personal stressful events in patients with acute myocardial infarction. Preliminary results. *Psychother Psychosom.* 1998;67(1):31–6. PMID: 9491438. DOI: 10.1159/000012256

74. Thombs B, Bass E, Ford D, et al. Prevalence of depression in survivors of acute myocardial infarction. *J Gen Intern Med.* 2006; 21:30–38. PMID: 16423120. PMCID: PMC1484630. DOI: 10.1111/j.1525–1497.2005.00269.x

75. Wells K, Rogers W, Burnam M, et al. Course of depression in patients with hypertension, myocardial infarction, or insulin–dependent

diabetes. *Am J Psychiatry.* 1993; 150:632–638. PMID: 8465882. DOI: 10.1176/ajp.150.4.632

76. Van Melle J, de Jonge P, Spijkerman T, et al. Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta–analysis. *Psychosom Med.* 2004; 66:814–822 PMID: 15564344. DOI: 10.1097/01.psy.0000146294.82810.9c

77. Kaptein K, de Jonge P, van den Brink R, et al. Course of depressive symptoms after myocardial infarction and cardiac prognosis: a latent class analysis. *Psychosom Med.* 2006; 68:662–668 PMID: 16987947. DOI: 10.1097/01.psy.0000233237.79085.57