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ATHEROSCLEROTIC CHANGES OF VESSELS CAUSED BY RESTRICTION OF MOVEMENT

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Atherosclerotic Changes of Vessels Caused by Restriction of Movement

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Disorders of lipoprotein metabolism regulation and the development of arterial lipidosis in various animals may be achieved as a result not only of excessive and prolonged intake of products rich in fat and fatty substances, but also by extensive and protracted functional stress on the central nervous system without prior introduction of cholesterol or substances similar to it [1-3]. Structural changes and lipidosis of arteries also develop during acute restriction of movement in rabbits [4].

In this work we will cite data obtained during a study of some biochemical indices and aortic wall structures in rabbits during limitation of their movement. The experiments were conducted on 16 rabbits using a method developed by V. V. Zyavokin.

In order to restrict the movement of the animals, they were placed in specially constructed narrow cages for 10, 20, and 30 days.

Before movement restriction and at the end of the experiment the animals' blood was studied for content of cholesterol, of beta-lipoproteins, and catecholamines. Also studied were some blood coagulation system indices, and EKGs were taken from the second standard lead. When the observations were completed, the animals were sacrificed and their aortas were morphologically studied.

Restriction of movement had a definite effect on blood lipoproteins. 10 days following the beginning of movement restriction, the quantity of beta-lipoproteins increased (P<0.001), and following 20-30 days the general cholesterol content also increased (P<0.001), and following

*Numbers in the margin indicate pagination in the foreign text.
Catecholamine content in the blood also increased, free adrenaline content increased in the first few days of the experiment, and after 20-30 days following the beginning of movement restriction, noradrenaline content also increased. Shifts in the blood coagulation system occurred simultaneously with the changes in blood lipid and catecholamine content: recalcification time decreased, and the prothrombin index increased.

Obvious shifts in EKG also were obtained. Deviation in the ST interval from the isoelectric line and deformations in the T wave were most striking. These changes were observed on the 20th and 30th days of the experiment.

When the observations were concluded, the animals were sacrificed and macro- and micromorphologic studies of the aorta were conducted.

Macroscopic study on the intima of the aorta quite clearly revealed lipidosis in some rabbits. In rabbit number 1, fatty maculae and atherosclerotic plaques were discovered in the intima of the descending aorta and its abdominal portion; in rabbit number 7, fatty striations and plaques were noted primarily in the abdominal portion of the aorta and in the region of the valves; these same changes could be noted also in rabbits number 22, 3, 4, 9, and 16; no changes were detected in aortic intima of the remaining animals, although plication of the intima and fatty plaques were present everywhere.

Microscopic study of the damaged portions of these aortas showed that deposition of sudano- and scarletophilic lipids, in the form of droplets of varying sizes and rhomboid crystals, was detected in those areas containing lipid maculae. Fatty inclusions occupied the entire thickness of the intima, in which collagen fibre swelling and impregnation of the intermediate matter with acid mucopolysaccharides were noted. Accumulation of histiocytes containing droplets of fat in the cytoplasm were also found here and there. Where atherosclerotic plaques occurred, permeation by droplets and crystals of sudano- and scarletophilic lipids into all layers of the intima was detected, in places also from the media to the adventitia. Between the fatty
inclusions and around them there were accumulations of "xanthomatous" and lymphoid cells, as well as histiocytes containing fatty inclusions in their cytoplasm, but which had not yet become xanthomatous. Such histiocytes were discovered alongside free fat droplets in various levels of the media width. The spread of fibrous connective tissue, in places undergoing hyalinization, was noted on the surface of the atherosclerotic plaques. Where plaques occurred, the intima was smoothed over, the internal elastic membrane thickening, and distension and unevenness of elastic membrane thicknesses were observed in the media (figure 1). Loose restriction of the rabbits' movement for 10 days, and particularly alternation of movement restriction with rest also for 10 days (in the course of 120 days), did not lead to the development of atherosclerotic changes in the aorta.

Fig. 1. An atherosclerotic plaque covered with hyalinized fibrillar connective tissue. Infiltration of the intima and media by sudanophilic lipids. Fatty inclusions also penetrate the deep layers of the media (below left). Sudan III staining, 7x20 mag., no. 54-6

In summarizing the studies that were conducted, we may include the following:

Restriction of movement in rabbits causes atherosclerotic changes in the walls of the aorta and shifts in EKG characteristic of coronary atherosclerosis. This fact, in our opinion, is worthy of attention,
and further in-depth study of it should contribute greatly toward an understanding of the etiopathogenesis of atherosclerosis and the development of measures for preventing and treating this disease.

The sclerotic changes in the walls of the aorta described by V. V. Tyavokin and confirmed by us, of course, are the result of movement restriction in rabbits, but it is still difficult to say what is the mechanism of their development.

We consider the statements of V. V. Tyavokin [4,5] on this question to be correct. Attention should, however, be focussed on the effect of centrogenic disturbance of lipoprotein and catecholamine metabolism. Constant and prolonged restriction of movement, of course, alters the functional condition of the higher central nervous system, and through changes in the hypothalamic centres may apparently become the source of disruption of the normal course of lipoprotein and monoamine metabolism. This is all the more probable, since in our experiments the content of lipoproteins and catecholamines in the blood increased, and participation of central nervous system mechanism in this was convincingly shown [2,3,6].
REFERENCES


