property of TT may be important when it is used for the creation of an experimental model of neuropathological syndromes. The results also confirm the theory of generator mechanisms of neuropathological syndromes [2], according to which specific manifestations of the corresponding syndrome are determined by the localization of a GPEE in a certain brain structure.

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STATE OF THE VASCULAR TONE AND SOME BLOOD BIOCHEMICAL INDICES IN EXPERIMENTAL ATHEROSCLEROSIS AND SENSITIZATION

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The study of the state of tone of the hind limb vessels and some blood indices of lipid metabolism in rabbits with experimental atherosclerosis and sensitization revealed an increase in vascular resistance in the region studied and a disturbance of lipid metabolism in animals of the two experimental groups. The results confirm previous observations that the allergic component is a factor which complicates the course of atherosclerosis.

KEY WORDS: atherosclerosis; sensitization; vascular tone.

The role of the allergic factor in the pathogenesis of atherosclerosis is well known [4-6,13]. Since one of the manifestations of atherosclerosis is a change in vascular reactivity [2,7], the role of allergy in the changes in the state of vascular tone is of great importance. Data in the literature on this matter are highly contradictory. For instance, the possibility of protracted hypertension in sensitized animals has been demonstrated [10]. At the same time, other work has shown that the allergic component lowers the rise in arterial blood pressure produced experimentally [9] or leaves it unchanged [3]. The contradiction between these results, it can tentatively be suggested, is due to some extent to the fact that the vascular tone was assessed by these workers indirectly.

It was accordingly decided to undertake the present investigation in order to compare the level of vascular tone under conditions of sensitization and alimentary atherosclerosis, with the aim of elucidating the role of the allergic component in the changes in vascular reactions in atherosclerosis.

Some indices of lipid metabolism were determined in parallel tests on the animals of all groups.

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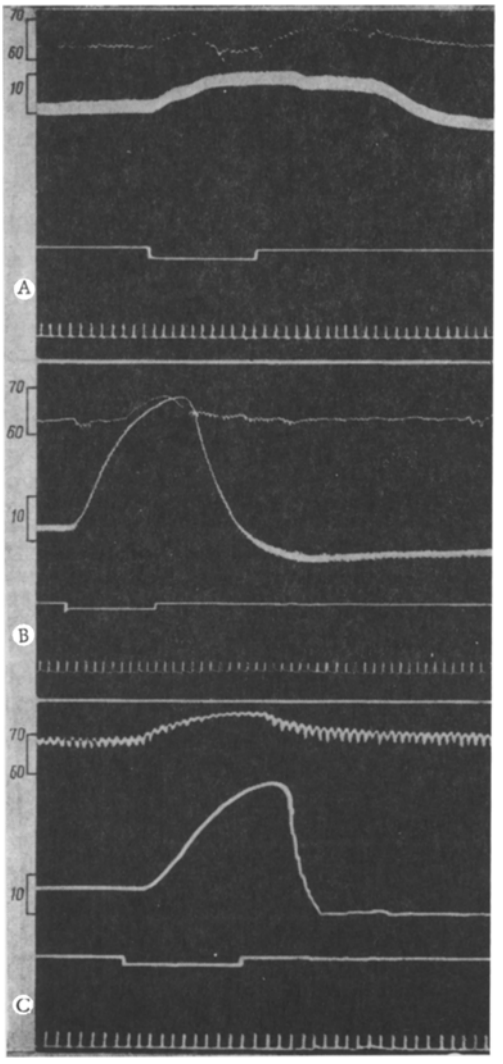

Fig. 1. Character of changes in blood pressure (in carotid artery) and perfusion pressure (in femoral artery) in response to stimulation of sympathetic nerves in control rabbits (A), rabbits with experimental atherosclerosis (B), and sensitized rabbits (C). From top to bottom: blood pressure, perfusion pressure, stimulus marker, time marker, 2 sec. Calibration of pressure in mm Hg.

EXPERIMENTAL METHOD

Experiments were carried out on 36 rabbits weighing 2-2.3 kg. The rabbits of group 1 (15) served as the control. The rabbits of group 2 (12) were kept on an atherogenic diet as in N. N. Anichkov's model of cholesterol atherosclerosis [1]. Cholesterol was given to the animals in an oily solution in a dose of 0.2 g/kg body weight daily for 90 days. The rabbits of group 3 (nine) were sensitized by subcutaneous injection of normal horse serum in a dose of 0.2 ml/kg body weight every third day, for a total of six injections.

Every fifth day the antibody (precipitin) titer was determined in each animal's serum and it varied during the course of sensitization from 1 : 8000 to 1 : 32,000. Vascular responses to stimulation of the lateral sympathetic chains at the level of L_4-L_6 were studied in the femoral artery by the method of resistography [12]. Sympathetic fibers were stimulated with pulses from an ISE-01 electronic stimulator. The strength of stimulation was twice the threshold level and its frequency 15 pulses/sec; the duration of each pulse was 4 msec.

The serum cholesterol concentration was determined by Elek's method, lecithin from the lipoid phosphorus level, and α- and β-lipoprotein fractions by electrophoresis on paper. The experimental results were subjected to statistical analysis [11].

EXPERIMENTAL RESULTS

Stimulation of the lateral sympathetic chains in the animals of all three groups led to biphasic effects: a well-marked vasoconstrictor response, followed by vasodilatation (Fig. 1).

In the animals of group 1 (control) the pressor component of vascular tone amounted to 12.4 ± 1.72 mm Hg. In rabbits kept on an atherogenic diet (group 2), the magnitude of the vasoconstrictor response to stimulation increased by 28.3 ± 2.91 mm Hg ($P < 0.001$). For animals sensitized with protein antigen (group 3) the vasoconstrictor response also was stronger (19.8 ± 2.15 mm Hg; $P < 0.05$) than in the control rabbits.

The cholesterol concentration in the control animals was 53 ± 2.7 mg %. In the rabbits with experimental atherosclerosis it was much higher (959 ± 196.98 mg %; $P < 0.001$). Sensitization was accompanied by some increase in the blood cholesterol (58 ± 2.4 mg %; $P < 0.05$). The lecithin concentration, which was 142 ± 13.0 mg % in the control animals, was changed in the rabbits of the two experimental groups. In the sensitized group the changes were not significant (144 ± 9.8 mg %; $P > 0.05$), whereas atherosclerosis led to a more marked increase in its blood concentration (211.61 ± 39.5 mg %; $P < 0.02$). The β-lipoprotein level also was raised in the animals of both experimental groups: from 69.5 ± 1.8% in the control of 85.7 ± 3.22% in group 2 ($P < 0.001$) and to 72.1 ± 2.3% ($P < 0.02$) in group 3.

The results of this investigation thus show that experimental atherosclerosis is accompanied by a marked increased in vascular tone and by characteristic humoral disturbances (an increase in the blood concentrations of cholesterol, lecithin, and the β-lipoprotein fraction). Sensitization also led to an increase in peripheral vascular tone, although fluctuations in the indices of lipid metabolism were not significant. The dilatation arising after the end of the vasopressor phase can be regarded as a passive vascular reaction [8].

It can be postulated on the basis of the results of the present investigation that the allergic component, which is always present in atherosclerosis, is probably one of the factors disturbing vascular reactions in this disease.

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