COMPARATIVE STEREOMETRIC INVESTIGATION
OF THE INTRAMURAL ARTERIAL BED OF THE
HUMAN MYOCARDIUM BY INJECTION AND
NONINJECTION METHODS

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A comparative stereometric investigation was made of the intramural arterial bed of the wall of the left ventricle in 20 injected and 30 noninjected hearts from clinically healthy persons aged 24-87 years dying as a result of trauma. The volume density of the intramural arterial bed of the myocardium  $(V_{\mathbf{v}})$  of the wall of the left ventricle was found to be on average 9 times greater in the injected than in the noninjected hearts. The value of  $V_{\mathbf{v}}$  was highest with an average type of coronary blood supply. A nonlinear relationship was found between  $V_{\mathbf{v}}$  and age. With an increase in the weight of the heart,  $V_{\mathbf{v}}$  fell. In hearts of equal weight from women and men,  $V_{\mathbf{v}}$  was significantly higher in women. The noninjection stereometric method of investigation of the intramural arterial bed of the heart simplifies the evaluation of the state of the myocardial blood supply, makes it more objective, and can be used in morbid anatomical practice.

KEY WORDS: heart; intramural arterial bed; stereometry.

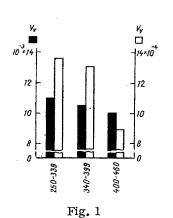
Great importance in the pathogenesis of ischemic heart disease is attached to the state and function of the microcirculatory bed of the heart [8, 10-13]. Among investigations by means of injection methods [2, 4-7, 14, 15], there have been virtually no stereometric studies of the microcirculatory bed of the myocardium. In ordinary autopsy practice it is very important to assess the state of the microcirculation in the myocardium in ordinary histological preparations.

This paper describes a comparative stereometric study by injection and noninjection methods of the intramural arterial bed of the myocardium of the left ventricle of clinically healthy persons of both sexes, aged 24-87 years, dying as a result of violence.

### EXPERIMENTAL METHOD

Fifty hearts were studied: 20 of them were injected with a suspension of finely divided lead carbonate (300 g in 500 ml of 4% gelatin solution) for 45 min under a pulsating pressure of 80-160 mm Hg [3, 9]. Thirty hearts were studied stereologically in ordinary histological preparations stained with hematoxylin-eosin and picrofuchsin-fuchselin. From six regions of the wall of the left ventricle (upper, middle, and lower thirds of the anterior and posterior regions) one horizontally, one vertically, and two frontally oriented pieces were cut in each case. Two histological sections were obtained from each piece. To determine the volume fraction of the intramural arteries of the myocardium, the histological sections were examined planimetrically in strips from the epicardium to the endocardium by means of a modified Avtandilov's ocular grid [2]. Only arteries divided at an angle of not less than 45° were counted [1]. On the basis of the combined planimetric data obtained from three planes of the myocardium the volume density of the intramural arterial bed of the myocardium ( $V_v$ ) was calculated. The results of the measurements were subjected to statistical analysis, with a 95% level of significance.

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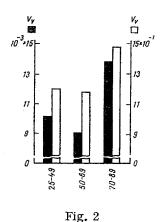


Fig. 1. Relationship between volume density of intramural arterial bed  $(V_{\mathbf{v}})$  of myocardium and weight of heart. Abscissa, weight of heart (in g); ordinate, value of  $V_{\mathbf{v}}$  (in conventional units). Black columns represent injected, white, columns noninjected hearts.

Fig. 2. Dynamics of changes in  $V_{\rm V}$  with age. Abscissa, age (in years). Remainder of legend as in Fig. 1.

### EXPERIMENTAL RESULTS

The value of  $V_{v}$  of the injected hearts of persons of the same age groups was found to be on average 9 times greater than that of the noninjected hearts. However, the dynamics of the results of the measurements of the volume density was the same by the two methods.  $V_{v}$  for the myocardium was found to be greatest for an average type of coronary blood supply:  $0.0112 \pm 0.0001$  (for injected) and  $0.00135 \pm 0.00002$  (for noninjected hearts). With a left type of blood supply,  $V_{v}$  was significantly lower:  $0.0096 \pm 0.0001$  and  $0.00084 \pm 0.00001$ , and with the right type the values were  $0.0102 \pm 0.0001$  and  $0.00107 \pm 0.0001$  respectively.

An increase in the weight of the heart was accompanied by a decrease in the volume density (Fig. 1). For instance, within the weight range of 280-339 g,  $V_v$  for injected hearts was 0.0110  $\pm$  0.0001 and for noninjected hearts 0.00136  $\pm$  0.00002 respectively. In hearts weighing 400-459 g  $V_v$  fell to 0.0100  $\pm$  0.0001 and 0.00089  $\pm$  0.00001 respectively.

The changes in  $V_v$  of the heart in different age groups were nonlinear in character (Fig. 2). Between the ages of 25 and 49 years,  $V_v$  for the injected and noninjected hearts was  $0.0101\pm0.0001$  and  $0.00120\pm0.00002$  respectively, whereas between the ages of 50 and 69 years a small decrease in the parameter to  $0.0091\pm0.000$  and  $0.00118\pm0.00002$  was observed. In persons over 70 years of age the value of  $V_v$  rose sharply:  $0.0138\pm0.0002$  and  $0.00148\pm0.00002$ .

To examine more closely the influence of the weight and age factors on changes in  $V_v$  each age group was divided into three subgroups based on weight (Fig. 3). As Fig. 3 shows, for hearts of the same weight the dependence of this parameter on age is the same as that described above.

 $V_{V}$  was found to be higher for the hearts of women than of men. To rule out the effect of the weight factor on changes in  $V_{V}$ , an analysis was made of subgroups in which the weight of the heart ranged from 290 to 380 g for injected hearts (the mean weight for men  $M_{M}=373\pm17$  g, for women  $M_{W}=355\pm15$  g) and 230-380 g for noninjected hearts ( $M_{M}=333\pm15$  g,  $M_{W}=327\pm13$  g).  $V_{V}$  for women was 0.0147  $\pm$  0.0002 for injected hearts and 0.00157  $\pm$  0.00002 for noninjected hearts, whereas in men the corresponding values were 0.0090  $\pm$  0.0001 and 0.00120  $\pm$  0.00002.

The reason why  $V_v$  was 9 times greater for the injected hearts was evidently postmortem contraction of the intramural arteries of the myocardium and the absence of intra-arterial pressure. Compression of the vessels by the muscle mass is important, especially if the heart stops in systole. During postmortem coronary transfusion with various solutions, in this case a solution of gelatin containing a suspension of lead carbonate, even if "intravital" conditions were created (a pulsating pressure of between 80 and 160 mm Hg), passive dilatation of the arteries took place to a greater degree than the maximal intravital dilatation.

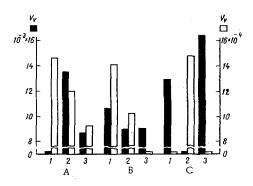


Fig. 3. Effect of age and weight of heart on value of  $V_V$ . Abscissa, weight of heart (in g): 1) 240-315, 2) 316-390, 3) 391-460, and age (in years): A) 25-49, B) 50-69, C) 70-89. Remainder of legend as in Fig. 1. Only one case is represented by black column A (3).

The opposite relationship between  $V_{v}$  and the weight of the heart can be explained by the ratio between the mass of the myocardium and the total volume of the vessels. For instance, with an increase in the weight of the heart the absolute volume of the intramural arterial bed of the myocardium either remained unchanged or it increased more slowly than did the absolute weight of the myocardium.

Age changes in  $V_{\rm V}$  of the heart are evidently connected with the following factors: 1) a change in the absolute weight of the heart with age — physiological hypertrophy of the heart between the ages of 50 and 69 years and a decrease in weight through atrophy in old age; 2) thickening of the walls of the intramural arteries of the myocardium through proliferation of the intima and sclerosis of the media in old age; 3) dilatation of the wall of the arteries of the heart in old age on account of degenerative changes in the elastic membranes; 4) tortuosity of the arteries in old people; 5) individual differences in the architectonics of the intramural arterial bed (the type of branching of the intramural arteries, the angles of origin of the branches, the number and diameter of the branches at different levels of ramification). This can evidently explain the individual observations in which a high value of  $V_{\rm V}$  was found in the young, middle-aged, and old heart, or when this parameter still remained high despite considerable hypertrophy of the myocardium.

A high value of the parameter  $V_v$  associated with the middle type of coronary blood supply does not correlate separately with either a small weight of the heart or with the female sex. Most frequently in this case extreme variations with a high value of  $V_v$  are found. It can tentatively be suggested that this is due to optimal characteristics of the architectonics of the intramural arterial bed for the supply of blood that is associated with this type of coronary circulation.

The noninjection stereometric method of investigation of the state of the intramural arterial bed of the heart simplifies the assessment of the state of the myocardial blood supply, makes it more objective, and can be used in morbid anatomical practice.

## LITERATURE CITED

- 1. G. G. Avtandilov, N. I. Yabluchanskii, T. A. Gevondyan, et al., Byull. Éksp. Biol. Med., No. 2, 250 (1977).
- 2. V. D. Arutyunov, "The blood vessels in myocardial infarction and the dynamics of vascularization of the heart scar," Candidate's Dissertation, Moscow (1961).
- 3. V. D. Arutyunov, Arkh. Pat., No. 8, 87 (1964).
- 4. N. A. Dzhavakhishvili and M. E. Komakhidze, Vessels of the Heart [in Russian], Tbilisi (1963).
- 5. S. P. Il'inskii, Ter. Arkh., No. 6, 33 (1954).
- 6. B. A. Lapin, "Special features of the circulation in the heart in angina pectoris," Candidate's Dissertation, Moscow (1952).
- 7. A. V. Smol'yannikov and T. A. Naddachina, Problems in Pathological Anatomy and Pathogenesis of Coronary Insufficiency [in Russian], Moscow (1963).
- 8. A. I. Strukov, Kardiologiya, No. 10, 5 (1973).
- 9. M. S. Tolgskaya, Arkh. Patol. Anat., No. 1, 138 (1941).
- 10. E. I. Chazov (Editor), Myocardial Infarction [in Russian], Moscow (1971).
- 11. E. I. Chazov and O. M. Eliseev, Kardiologiya, No. 12, 10 (1976).
- 12. A. M. Chernukh, P. N. Aleksandrov, and O. V. Alekseev, The Microcirculation [in Russian], Moscow (1975), p. 402.

- 13. I. K. Shkhvatsabaya, Kardiologiya, No. 7, 5 (1975).
- 14. G. Farrer-Brown, Br. Heart J., 30, 527 (1968).
- 15. V. Russanen, Angiology, 24, 345 (1973).

# CHANGES IN DENDRITES OF CORTICAL NEURONS IN EXPERIMENTAL ALCOHOL INTOXICATION

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Dendrites of cortical neurons were studied in rats in the different stages of alcohol intoxication. Two categories of changes in the dendrites develop under these circumstances: destructive and compensatory. The dynamics of these changes depends on the periods of alcohol intoxication and individual differences in the central nervous system of the animals.

KEY WORDS: cerebral cortex; action of alcohol; dendrites.

Many clinical and experimental investigations have shown that prolonged alcohol intoxication leads to disturbances of higher nervous activity, changes in autonomic, somatic, emotional, and mental functions, depression of cortical excitability, phasic changes in the EEG, and so on [1, 3, 9, 12].

Changes in nerve cells, fibers, glia, and blood vessels during prolonged alcohol intoxication in man and experimental animals have been studied in detail by the methods of light microscopy. An initial lesion of cortical structures followed by the spread of the process to the subcortex, brain stem, and spinal cord is generally accepted. Some investigators have stated that the changes in the nerve cells are reversible during the first 20 days of chronic alcohol poisoning [4, 11]. It has recently been shown that the structure not only of nerve and glial cells, but also of interneuronal connections, is changed in alcohol intoxication [2, 11].

Considering the important role of dendrites in the mechanisms of brain activity and the absence of data of the dynamics of changes in the dendritic apparatus of neurons in alcohol intoxication, the investigation described below was undertaken in order to study changes in the dendrites and their spines—the specific post-synaptic receptor structures—at different times during experimental alcohol intoxication.

#### EXPERIMENTAL METHOD

Experiments were carried out on albino rats aged 3 months which were given a 35% solution of ethyl alcohol instead of water. In the initial period (10-20 days) they were given 2-3 ml alcohol daily, for 2-2.5 months the mean daily dose of alcohol was increased to 8-10 ml, and starting from the third month the dose given was 14-17 ml. The animals were killed 10 and 20 days and 2, 4, 8, and 12-15 months after the beginning of the experiment. Golgi's silver impregnation method was used.

# EXPERIMENTAL RESULTS

After the first 10-20 days of the experiment neurons whose dendrites, especially the basal, contained few spines and sometimes had irregular outlines and no spines whatsoever, were found in the lower layers of the cortex. The dendrites of most neurons of the upper layers of the cortex, on the other hand, were covered by many spines. Whether there were few or many spines, some of them showed hypertrophy. Contacts of varicose type between processes of the swollen oligodendrocytes and the main trunks and the infrequent spines of the dendrites could be seen (Fig. 1).

After 2 months of alcohol intoxication the irregularity of the outlines of the dendrites of many small and medium-sized neurons in the lower layers of the cortex increased. In most cells the basal dendrites had no

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