LITERATURE CITED

1. N. N. Gorev, I. M. Kozhura, L. V. Kostyuk, et al., Experimental Atherosclerosis and Age [in Russian], Moscow (1972).
2. E. I. Chazov and A. N. Klimov (editors), Dyslipoproteinemias and Ischemic Heart Disease [in Russian], Moscow (1980).
3. World Health Organization Annual Report of World Health Statistics [in Russian], Moscow (1980).
4. A. N. Klimov and I. E. Ganelina, The Phenotyping of Hyperlipoporteinemias (Technical Recommendations) [in Russian], Moscow (1975).
5. E. V. Montsevichyute-Eringene, Patol. Fiziol., No. 4, 71 (1964).
6. A. V. Tokar', P. P. Chayalo, and E. S. Rudaya, Ter. Arkh., No. 8, 11 (1980).
7. L. K. Finagin, I. M. Kozhura, and M. U. Zaika, Byull. Éksp. Biol. Med., No. 2, 155 (1978).
8. I. K. Shkhvatsabaya et al., (editors), The Epidemiology of Cardiovascular Diseases [in Russian], Moscow (1977).
9. J. L. Beaumont, L. A. Carlson, G. R. Cooper, et al., Bull World Health Org., 43, 891 (1970).
10. J. Folch, M. Lees, and G. H. Sloan-Stanley, J. Biol. Chem., 226, 497 (1957).
11. H. Haller, M. Hanefeld, and W. Jaross, Lipidstoffwechselstorungen. Diagnostik, Klinik und Therapie, Jena, Gustav Fischer Verlag (1975).
12. L. N. Katz and J. Stamler, Experimental Atherosclerosis, Springfield (1953).
13. W. Reuter, J. Gerontol., 24, 669 (1976).

PERICARDIAL RESORPTION IN THE GROWING ORGANISM UNDER NORMAL CONDITIONS AND IN EXPERIMENTAL PERICARDITIS

O. V. Romashin and A. A. Men'shikov

UDC 616.11-002-092.9-033.1-073.916

KEY WORDS: pericardial cavity; isotope resorption; experimental pericarditis.

Inflammatory diseases of the pericardium stem from a wide range of etiologic causes [1]. Recently, with the development of cardiac surgery, the incidence of postoperative pericarditis has increased [5]. In pediatric practice pericarditis is rare and, as a rule, it accompanies diseases associated with septicemia and the acute pneumonias, and it considerably aggravates the course of the underlying disease.

There are no data in the literature on the resorptive capacity of the peri-epicardial system in the young growing organism under normal and pathological conditions. The object of this investigation was to determine any differences which may exist in resorption from the peri-epicardial cavity of young animals under normal conditions and in experimental pericarditis [7].

EXPERIMENTAL METHOD

Experiments were carried out on 26 mongrel puppies 1-3 months, using a technique specially developed by ourselves. Under ether-thiopental anesthesia with controlled respiration a microirrigator was sutured into the pericardial cavity, and one end of it was brought out through a subcutaneous channel in the posterior surface of the animal's neck. Through this irrigator radioactive ^{131}I-hippuran (M-315), with a short half-life (5-6 days) and half-elimination time (3-4 min), and which is not cumulative in organs or tissues, could be injected into the pericardial cavity [6]. The isotope is resorbed by the terminal portions of the blood and lympatic systems [4], is excreted from the blood stream by the kidneys, and accumulates in the bladder. The animal was fixed to the operating table in the supine position and the radioactive background was recorded from the region of the heart (the lower border of the counter was at the level of the costal angle) and the region of the urinary bladder (the level of the pubic symphysis) by means of STO-5 and SBT-7 recording counters and a B-3 computer. Both counters were placed 1 cm from the skin surface. To reduce the external background radiation the counters were placed under lead covers. The dose of isotope, with a radioactivity of 0.5 mCi/ml, injected into the animal was 0.5 ml/kg body weight. This dose had no pathogenic action on the tissues, for it was 10-15 times smaller than the minimal dose.

Department of Operative Surgery, Altai Medical Institute, Barnaul. Department of Human Anatomy, Novosibirsk Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR Yu. I. Borodin.)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 92, No. 11, pp. 535-537, November, 1981. Original article submitted March 26, 1981.

0007-4888/81/9211-1486$07.50

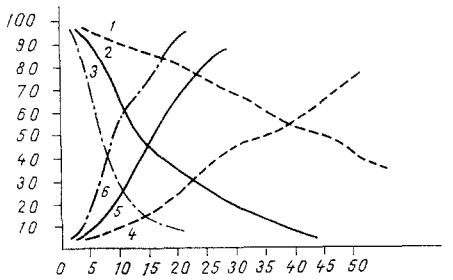

Fig. 1. Evacuation of isotope from the pericardial cavity and its accumulation in the bladder. 2) Curve of elimination of isotope from normal pericardial cavity, 5) accumulation of isotope in normal urinary bladder. 1, 4) The same, on 5th day of experiment; 3, 6) the same, on 28th day of experiment. Abscissa, time (in min); ordinate, intensity of radioactivity (in % of control).

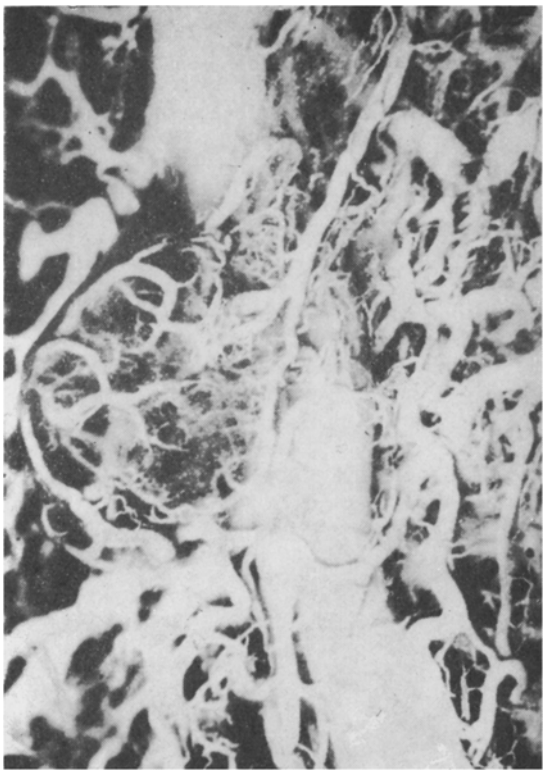

Fig. 2. Development of abundant subepicardial venous network and invasion of fibrin deposit by veins. Twentieth day of pericarditis. Combined photomacro-micrograph, 50 X.

Acute pericarditis was induced by injecting a 24-h culture of *Staphylococcus aureus* (strain No. 209) into the pericardial cavity in a dose of 2×10^8 bacterial cells/kg body weight. To prevent cardiac tamponade after the development of pericarditis, 2-3 ml of seropurulent fluid was withdrawn daily from the pericardial cavity. Resorption during pericarditis was studied daily, after the first day of the disease, for 1 month. In the course of the experiment, and also after the end of 1 month, the animals were removed from the experiment. Heart preparations were studied by histological methods (van Gieson, Weigert), and the epicardial blood vessels were injected with polychromic media and the lymphatics with Gerota's medium, followed by clearing. In the control, autopsy specimens of the hearts of infants under 1 year old, dying from pericarditis, were studied in the same way [2].

EXPERIMENTAL RESULTS

Under normal conditions evacuation of the isotope from the pericardial cavity took place most rapidly during the first 20 min and was complete after 45-50 min; accumulation of the isotope in the blood and bladder followed a parallel course.

Following injection of the isotope together with the staphylococcal culture resorption was retarded on average by 6%. The half-elimination time was 17 min (normal 12 min), and elimination of the remainder took place more slowly during the next 10-15 min. This was evidently attributable to the local vascular response to injection of the staphylococcal culture [3]. On the 3rd day, the half-elimination time of the isotope was 24 min, twice the normal period, and complete elimination occurred at the beginning of the 2nd hour. Maximal delay in absorption of the isotope was observed on the 5th-6th day of the disease. The half-elimination time was 45 min, and elimination was complete after 2 h, i.e., it took place almost 6 times more slowly than normally (Fig. 1).

Examination of the heart preparations showed thickening of the serous cover on account of deposition of fibrin and suppurative infiltration of the epicardium of the heart down to the surface layers of the myocardium. The venules were dilated and distended by blood cells, some of which escaped into the paravasal cellular tissue.

Later the resorptive function was gradually restored and returned to normal values on the 14th day. The resorption time then gradually diminished. On the 21st day of the disease 50% of injected isotope was absorbed after 8 min, a rate 1.5 times faster than normal. Elimination of the isotope took place at a constant rate until the process was complete. On the 28th day of the disease the resorption time was even longer, 3-4 times the normal values.

Examination of the heart preparations showed the development of an abundant subepicardial venous network. Up to 60 venous vessels with a diameter of 0.007-0.05 mm or more could be counted in an area of 1 mm^2 of the epicardial surface, compared with the normal figure of 8-12 vessels. Organization of the venous vessels and areas of fibrin deposition also were observed (Fig. 2). Probably this intensive development of the epicardial venous network was responsible for the increase in the resorptive capacity of the peri-epicardial system.

Experimental pericarditis thus delays resorption of isotope from the pericardial cavity with a maximum on the 5th-6th days of the disease, thus favoring accumulation of metabolic products and toxins in the organ. On the 14th day of the disease the level of isotope resorption returns to normal values. On the 28th day of the experiment resorption is increased by 3-4 times, evidently on account of the development of an abundant subepicardial venous network.

LITERATURE CITED

1. Z. M. Volynskii and E. E. Gogin, Diseases of the Pericardium [in Russian], Leningrad (1964).
2. B. A. Dolgo-Saburov, Outlines of Functional Anatomy of the Blood Vessels [in Russian], Leningrad (1961).
3. I. K. Esipova, O. Ya. Kaufman, G. S. Kryuchkova, et al., Outlines of Hemodynamic Changes in the Structure of the Vessel Wall [in Russian], Moscow (1971).
4. V. V. Kupriyanov and Ya. R. Karaganov, Kardiologiya, No. 6, 3 (1969).
5. E. N. Meshalkin, T. F. Arkhipova, and G. G. Chasovskikh, Metabolism and Structure of the Myocardium in Congenital Heart Defects [in Russian], Novosibirsk (1978).
6. A. Ya. Prokopchuk, Tr. Minsk. Med. Inst., 20, 140 (1957).
7. A. M. Khil'kin and V. A. Svetlov, Experimental Models of Lesions of the Heart and Vessels [in Russian], Moscow (1979).