# EFFECT OF BURNS IN A RECIPIENT DOG ON FUNCTION OF AN EXTRACORPOREALLY PERFUSED ISOLATED HEART

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Extensive burns lead to a decrease in cardiac output (CO) [2, 5, 6, 10, 12]. The cardiac ejection begins to diminish during the first 2 min after infliction of the burn, and after 5 min it is reduced to 50% of its initial value [4]. The causes of such a rapid fall in CO are not sufficiently clear. This phenomenon has been explained by heart failure [3, 7, 10], by a decrease in the venous return, and by a combination of both [3]. Direct measurements of myocardial contractility after burns have not been undertaken on the intact organism, and conclusions regarding heart failure are based on indirect evidence. Perfusion of isolated papillary muscles of the cardiac ventricles with serum from a burned rabbit has a negative inotropic action [1, 8].

In the present investigation the effect of humoral factors of burn trauma on the contractile function of the dog's heart was studied at the organ level.

## EXPERIMENTAL METHOD

Nine experiments were carried out on mongrel dogs. The donors of the hearts weighed 5.5-11.5 kg and the recipients 19-29 kg. Under pentobarbital anesthesia (30 mg/kg) and with artificial ventilation of the lungs with a mixture of 50% oxygen and 50% air, the heart donor's chest was opened, heparin was injected in a dose of 3 mg/kg, and a cannula was introduced into the left subclavian artery. After clamping of the descending aorta and brachiocephalic artery, some blood found its way into a receiver located at a height of 100 cm. The functioning heart-lung preparation was isolated. The left lung was removed. Catheters were introduced into the descending aorta, the common trunk of the pulmonary artery, and the left atrium (Fig. 1A). The heart was connected by these catheters to a perfusion system. During the first 10 min blood was directed into the aorta. The right lung was then removed and an adequate blood supply preserved to the isolated heart. The heart was then perfused for 1 h, blood being directed into the left atrium under constant pressure (Fig. 1B). The volume velocity of inflow was 50-60 ml/min/kg body weight of the heart donor. A burn affecting 30-40% of the body surface of the recipient dog was inflicted with boiling water for 2 min. Observations continued for 1 h after infliction of the burn. Before and after burning, volume loading tests of the isolated heart were carried out at the 5th, 15th, 30th, 45th, and 60th minutes. For this purpose the vessel from which blood entered the left atrium was raised for 10-15 sec, thus increasing the end-diastolic pressure in the left ventricle from 0 to 15 mm Hg. At the height of loading, the following parameters were recorded for 10-15 cycles: the ECG, the pressure in the aorta (AP), the pressure in the left ventricle (LVP) by means of EMT-34 transducers, the inflow pressure into the left atrium, visually, the volume velocity of inflow (VVI) into the left atrium, the cardiac ejection (CE), and the coronary blood flow (CBF) from the ejection from the right ventricle by means of flow detectors of the Nycotron-372 and 376 electromagnetic flowmeter. The ECG, AP, LVP, VVI, CE, and CBF of the isolated heart were recorded on an 8-channel Mingograph-800 instrument (from Elema Schönander, Sweden), and the temperature of the inflowing blood was maintained at 36 ± 0.5°C. The heart rate (HR), systolic and diastolic pressure in the aorta (APS and APD respectively), CBF (per 100 g weight of the heart), the systolic LVP ($LVP_S$), and the end-diastolic LVP (EDLVP), the maximal rate of rise of LVP ($dP/dt_{max}$), the index of myocardial contractility (IC, after Veragut), the specific cardiac output (CSO), and the stroke work ($A_{str}$) were calculated. The arterial and venous blood gas composition was determined by the IL-213 apparatus (Italy). The oxygen demand (OD) was calculated by Fick's equation.

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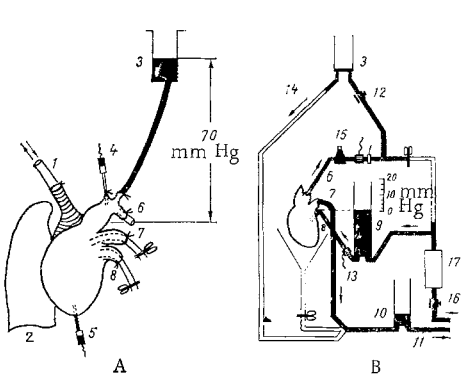


Fig. 1

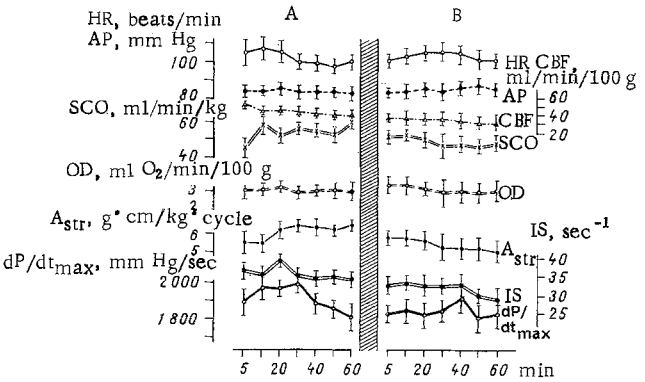


Fig. 2

Fig. 1. Scheme of removal (A) and perfusion (B) of isolated heart. 1) Tube to apparatus for artificial ventilation of lung; 2) right lung; 3) vessel containing autologous blood; 4) catheter for measuring pressure in aorta; 5) catheter for measuring pressure in left ventricle; 6) tube in descending aorta; 7) tube in common trunk of pulmonary artery; 8) tube in left atrium; 9) vessel for regulating velocity of inflow; 10) venous reservoir; 11) venous trunk to donor; 12) regulator of resistance to cardiac ejection; 13) detector of electromagnetic flowmeter; 14) trunk for shunting blood from pressure stabilizer; 15) air bubble trap, detectors of volume velocity of blood flow and temperature; 16) regulator of lumen of arterial trunk; 17) heat exchanger.

Fig. 2. Indices of function of isolated heart before (A) and after (B) infliction of burn.

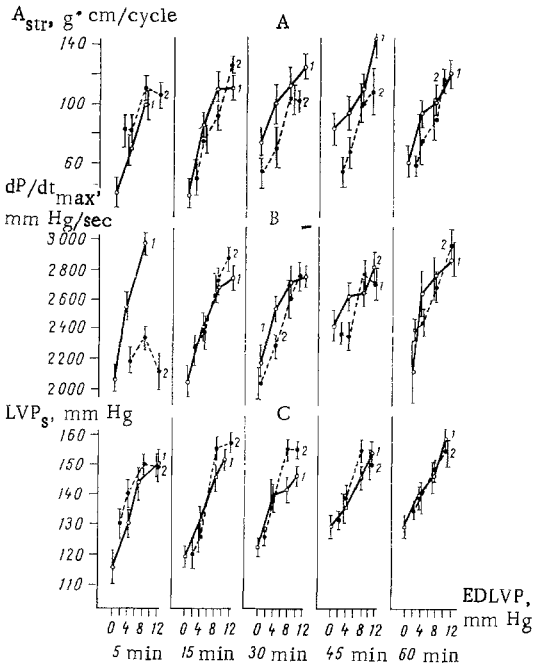


Fig. 3. Dependence of $A_{str}$ (A), $dP/dt_{max}$ (B), and $LVP_S$ (C) on EDLVP before (1) and after (2) burning during loading tests.

## EXPERIMENTAL RESULTS

The conditions of perfusion, the coronary hemodynamics, and OD of the isolated heart were the same before and after burning (Fig. 2). HR, $LVP_S$, $dP/dt_{max}$, and IS were unchanged after burning, and although a tendency for IS to fall was observed it was not statistically significant. By the end of the first hour after burning, against the background of an increase in EDLVP ($P < 0.02$) there was a significant fall in the stroke volume. This led to a reduction in SCO ($P < 0.02$) and $A_{str}$ ($P < 0.05$) at the 60th minute after burning.

The loading test showed that, except displacement of the curve of $dP/dt_{max}$ as a function of EDLVP downward and to the right at the 5th minute after burning, no inhibition of cardiac function was found in any other observation (Fig. 3). This does not agree with the result of the writers' previous experiments on dogs to study cardiac function in the intact animal [4]. Using the same model of burns, a rapid and significant worsening of myocardial function was obtained. This suggests the possibility of a direct negative inotropic effect of the nervous system on the heart of the burned animal in conjunction with the unfavorable role of the hemodynamic factor. The possibility cannot be ruled out that the action of humoral burn depressants was masked by increased sensitivity of the denervated heart to catecholamines. Proof of the existence of such a mechanism is required. Meanwhile evidence has been obtained of the direct inhibitory influence of the serum of burned rabbits on contractility of the papillary muscles [1]. This would appear to contradict the attempt to explain the "insensitivity" of the isolated heart to burn-induced myocardial depressants. It is not known, however, whether the law of denervation is expressed equally on the whole organ, in which the intracardial nervous system is completely intact, thus maintaining self-regulation of the heart, and on a strip of tissue. It is likewise not clear whether the experimental results depend on the species of experimental animal used.

After burns the contractile and pumping function of the isolated dog's heart, by contrast with that of the heart in the intact organism, is not inhibited although in both cases the two hearts are supplied with blood containing the same humoral agents.

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