ROLE OF REACTIONS OF CAPACITIVE VESSELS IN THE PATHOGENESIS OF EXPERIMENTAL CARDIOGENIC SHOCK

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Reactions of the system of capacitive vessels of the abdominal and pelvic organs were investigated in dogs with cardiocytotoxic necrosis of the myocardium complicated by cardiogenic shock. During the first minutes of development of acute myocardial necrosis a considerable volume of circulating blood was observed to be retained in the venous part of the vascular reservoir of the abdominal and pelvic organs. Limitation of the venous return to the heart was shown to play an essential role in the pathogenesis of the hemodynamic disorders accompanying the development of cardiogenic shock.

Analysis of data on the pathogenesis of acute hemodynamic disorders accompanying the development of postinfarction cardiogenic shock demonstrates the important role of circulatory changes in the system of capacitive vessels [10, 14]. In the initial period of development of cardiogenic shock the central venous pressure (CVP) frequently falls indicating limitation of the flow of blood toward the heart [9].

In the investigation described below, the state of the capacitive vessels of the abdominal and pelvic organs was studied during the development of experimental cardiogenic shock.

TABLE 1. Changes in Indices of Cardio- and Hemodynamics in Dogs with Experimental Cardiogenic Shock (M ± m)

Index	Control	Time after injection of anticardiac serum, min			
		5	15	30	60
Volume of blood retained, ml	0	77,3±28	157,0±50,4	208,2±57,5	351,2±111,7
P		<0,05	<0,05	<0,05	<0,05
SAP, mm Hg	161±7,9	124,6±10,5	131,8±10,3	138,5±10,4	123,5±9,4
P		<0,05	>0,05	>0,05	<0,05
CVP, mm Hg	14,7±4,25	13,4±3,8	18,5±2,6	26,3±5,8	43,0±14,5
P		>0,05	>0,05	>0,05	>0,05
Perfusion pressure in resistive vessels of abdominal organs, mm Hg	119,4±8,3	111,2±9,8	114,5±9,9	114,2±9,9	114,5±6,4
P		>0,05	>0,05	>0,05	>0,05
Pressure in left ventricle, mm Hg	162,2±2,8	125,6±3,6	133,7±3,5	138,5±3,7	123,8±4,2
P		<0,01	<0,01	<0,01	<0,01
dp/dt_{max} for left ventricle, mm Hg/sec	3826±614	3008±709	3090±672	3010±681	3244±991
P*		<0,05	<0,02	<0,01	<0,02
FDV_{lv}, mm Hg	2,3±1,1	2,6±0,9	3,6±1,6	4,8±1,7	2,3±1,6
P		>0,05	>0,05	>0,05	>0,05

*P calculated by difference method.

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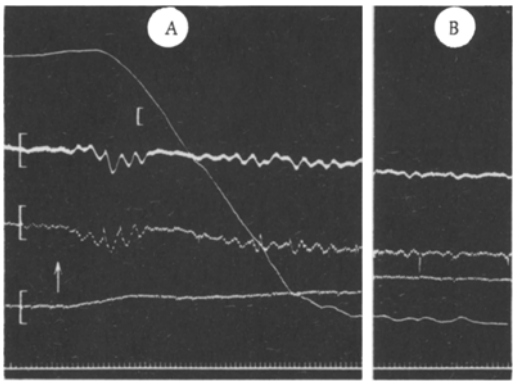

Fig. 1. Changes in hemodynamics of a dog after intracoronary injection of anticardiac serum. From top to bottom: changes in blood volume in extracorporeal reservoir (ml), perfusion pressure in vessles of abdominal and pelvic organs (mm Hg), SAP (mm Hg), CVP (mm water), time marker (10 sec). A) Intracoronary injection of anticardiac serum (time of injection indicated by arrow); B) 30 min after injection of serum. Calibration from bottom to top: from 0 to 20; from 110 to 130; from 120 to 140.

EXPERIMENTAL METHOD

Cardiogenic shock was produced in dogs by the method described earlier [1] involving intracoronary injection of an anticardiac cytotoxic serum obtained from rabbits immunized with a saline extract of dog myocardium. In acute experiments on dogs weighing 16-21 kg, anesthetized with pentobarbital intravenously, the systemic arterial pressure (SAP), central venous pressure (CVP), resistance to the blood flow in the vessels of the abdominal and pelvic organs (by resistography), changes in the capacity of this vascular region (by the extracorporeal venous reservoir method [10]), the pressure in the left ventricle and its first derivative (dp/dt_{max}), and also the final diastolic pressure of the left ventricle (FDP_{lv}) were recorded. The various parameters were recorded before and during the course of 1-2 h after injection of the serum.

EXPERIMENTAL RESULTS AND DISCUSSION

In most experimental animals marked retention of blood in the system of capacitive vessels of the abdominal and pelvic organs was observed during the first few minutes after intracoronary injection of the anticardiac serum (Table 1). Retention after 5 min averaged 77.3 ml, rising after 1 h to 351.2 ml. The rate of deposition of blood differed significantly in the different experiments. In some experiments it was already intensive during the first 2-3 min after injection of the serum, whereas in others it started later (after 5-10 min). Later the rate of deposition of blood slowed down considerably; in some cases retention ceased and then was resumed. Frequently the total volume of circulating blood "excluded" from the system was 500-600 ml. Under conditions of stabilization of the venous return to the heart the degree of lowering of SAP, the pressure in the left ventricle, and dp/dt_{max} was less than in experiments in which an extracorporeal circulation was not used. For instance, whereas in experiments without stabilization of the venous return to the heart SAS fell after intracoronary injection of anticardiac serum by an average of 47%, to reach 70 ± 5 mm Hg, in the experiments with stabilization of the venous return to the heart via the system of the inferior vena cava it fell only by 22.6%. Systemic arterial pressure in the left ventricle (by 22.6%) and dp/dt_{max} for the left ventricle (by 21.4%) fell within the same limits.

Analysis of the results shows that during the development of acute myocardial degeneration when the venous return to the heart was stabilized the fall in SAP was due chiefly to weakening of the contractile function of the heart. In most experiments an increase in CVP and FDP_{lv} was observed (Figs. 1 and 2), pointing to the development of congestive cardiac failure [11]. No such increase in CVP or FDP_{lv} was observed as a rule in the experiments without stabilization of the venous return.

In most investigations on animals with experimental cardiogenic shock, changes of considerable magnitude but of varied direction were observed in the tone of the resistive vessels in different vascular fields [4, 7, 8]. In the present experiments, when the total resistance of the resistive vessels of the abdominal and pelvic organs

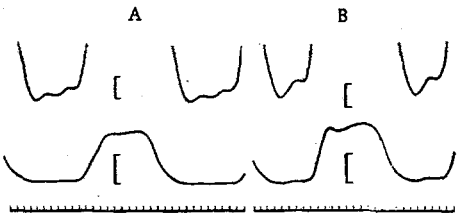

Fig. 2. Changes in pressure in left ventricle of dog with experimental cardiogenic shock. A) Initial data; B) 30 min after intracoronary injection of anticardiac serum. Pressure in left ventricle (mm Hg) below; diastolic component of left ventricular pressure (mm Hg) above. Tape winding speed 250 mm/sec. Calibration: from 0 to 10 above, from 0 to 100 below.

were studied, no significant changes were found after intracoronary injection of anticardiac serum (Table 1), except in the first 3-6 min. This confirms observations showing that changes in the tone of the resistive vessels are not always consistent in the different vascular regions and it is evidence that changes in the capacity of the resistive vessels of the abdominal and pelvic organs do not play an essential role in the deposition of blood in cardiogenic shock. This process takes place mainly, it is considered, through changes in the capacity of the venous reservoir of the systemic circulation. According to some investigators [12, 13], one possible mechanism of the deposition of blood in cardiogenic shock is reflex dilatation of capacitive vessels. The possibility of such effects has been proved experimentally [2, 6, 10]. At the same time, various biologically active substances (serotonin, histamine, prostaglandins, kinins, and so on), whose concentration in the circulating blood changes considerably in myocardial infarction and cardiogenic shock [3, 5], may have an important action on the changes in capacity of the vascular system and on the hemodynamics as a whole.

During the development of experimental cardiogenic shock caused by intracoronary injection of anticardiac cytotoxic serum, considerable retention of blood in the abdominal and pelvic organs thus takes place, with the result that efficiency of the pumping function of the heart is reduced and the systemic perfusion pressure falls considerably.

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