ANALYSIS OF THE CAUSES OF THE CIRCULATORY DISTURBANCE IN BURN SHOCK

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Changes in the parameters of the hemodynamics and gas exchange during an artificial increase in the circulating blood volume produced by intravenous injection of dextran (2% of the body weight) were studied in experiments on rabbits with lethal burn shock. Depending on the changes in the parameters studied following injection of dextran the burned animals can be divided into three groups: in group 1 the decrease in the minute volume of the heart was due mainly to cardiac failure, in group 2 to a lowering of the tone of the low pressure system, and in group 3 to a combination of both factors.

KEY WORDS: burn shock; hemodynamics; gas exchange; response of the cardiovascular system to volume loading.

A characteristic feature of burn shock is the sharp decrease in the minute volume of the heart (MVH) [1, 3-8, 10, 11, 13, 14]. This decrease may be caused by a decrease in the circulating blood volume (CBV), regarded by supporters of the plasma loss theory [9, 10, 12, 14] as the cause of burn shock. However, according to other observations, the sharp decrease in MVH occurs before the decrease in CBV [6-8]. Some workers [4, 7] consider that the cause of the sharp decrease in MVH is cardiac failure developing in response to burn trauma. Others [11, 13] see the cause of the decrease in MVH in the sharp increase in peripheral resistance accompanied by a decrease in the effective CBV.

The role of the decrease in CBV in the disturbances of the hemodynamics and gas exchange in burn shock was studied in response to an artificial increase in CBV.

EXPERIMENTAL METHOD

Experiments were carried out on 70 rabbits anesthetized with urethane (1 g/kg). The control group consisted of 37 animals. In 33 animals a burn covering 30% of the body surface was inflicted [3]. To increase the CBV, dextran (6% solution) was injected into the femoral vein of the animals in a dose of 2% of the body weight at the rate of 5 ml/min. Dextran was injected into the burned animals 15-30 min after burning. The MVH was determined by the thermal dilution method. The central blood volume (CV) also was calculated from the thermal dilution curve. The circulating plasma volume (CPV) was found by the dye dilution method, using Evans' Blue. The hematocrit number (HN) in arterial blood was determined with the aid of a spiral centrifuge.

The CBV was calculated. The hemoglobin concentration in the arterial blood was determined by a photoelectric-colorimetric method. The arterial pressure (AP) in the femoral artery was recorded by means of an electromanometer. The central venous pressure (CVP) was measured in the right atrium by means of a water manometer. The systolic volume (SV) of the heart and the peripheral vascular resistance (R) were calculated. The oxygen consumption (pO_2) was tested continuously by means of a closed system with CO_2 absorption and automatic O_2 supply. The minute volume of respiration (MVR) was de-

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TABLE 1. Effect of Dextran on Hemodynamic Parameters in Rabbits

Parameter studied	Control animals						
	Initial data	after injection of dextran (% of initial values)					
		immediately	10 min	30 min	1 h	2 h	3 h
MVH (in ml/min/kg)	160 ± 12	198 ± 11	164 ± 8	143 ± 10	124 ± 7	115 ± 7	97 ± 5
AP (in mm Hg)	88 ± 2	114 ± 3	110 ± 2	105 ± 3	100 ± 3	112 ± 4	117 ± 7
SV (in ml)	0,60 ± 0,07	222 ± 15	179 ± 11	158 ± 11	146 ± 7	127 ± 7	114 ± 7
R (in dynes·sec·cm^{-5})	17·10^3 ± 2·10^3	60 ± 2	72 ± 2	82 ± 4	93 ± 5	104 ± 6	121 ± 9
pO_2 (in ml/min)	21 ± 0,8	110 ± 3	103 ± 3	98 ± 3	93 ± 3	90 ± 4	88 ± 5
MVR (in liters/min)	0,75 ± 0,02	109 ± 4	108 ± 3	106 ± 4	105 ± 4	101 ± 3	102 ± 4
CPV (in ml)	107 ± 5	148 ± 5	132 ± 4	128 ± 6	138 ± 8	143 ± 7	144 ± 8
CBV (in ml)	170 ± 6	122 ± 3	114 ± 3	112 ± 7	121 ± 7	125 ± 5	130 ± 11
CV (in ml)	58 ± 3	148 ± 4	132 ± 4	121 ± 6	114 ± 5	109 ± 4	101 ± 4
	37 ± 2	67 ± 3	72 ± 3	77 ± 2	80 ± 2	79 ± 2	82 ± 4

(continued)

	Burned animals									
	Initial data	after burning (% of initial value)			after injection of dextran (% of initial values)					
		immediately	10–15 min	30 min	immediately	10 min	30 min	1 h	2 h	3 h
MVH (in ml/min/kg)	171 ± 10	66 ± 4	51 ± 4	39 ± 4	119 ± 15	101 ± 11	76 ± 8	56 ± 7	46 ± 5	37 ± 5
AP (in mm Hg)	92 ± 2	89 ± 3	72 ± 3	69 ± 4	97 ± 3	94 ± 3	91 ± 3	81 ± 4	81 ± 4	70 ± 6
SV (in ml)	0,62 ± 0,04	67 ± 4	50 ± 5	42 ± 4	140 ± 19	115 ± 13	86 ± 10	76 ± 10	62 ± 6	56 ± 8
R (in dynes·sec·cm^{-5})	15·10^3 ± 10^3	136 ± 9	152 ± 13	191 ± 22	122 ± 22	114 ± 16	140 ± 17	172 ± 19	214 ± 31	246 ± 50
pO_2 (in ml/min)	21,9 ± 1	86 ± 3	80 ± 4	69 ± 5	85 ± 7	82 ± 7	79 ± 4	66 ± 4	54 ± 4	43 ± 3
MVR (in liters/min)	0,74 ± 0,05	155 ± 14	135 ± 14	140 ± 16	137 ± 14	123 ± 12	116 ± 10	109 ± 10	91 ± 10	77 ± 8
CPV (in ml)	119 ± 10	111 ± 6	87 ± 4		139 ± 11	115 ± 5	120 ± 5	114 ± 7	102 ± 8	91 ± 7
CBV (in ml)	190 ± 17	111 ± 7	87 ± 4		116 ± 8	106 ± 7	103 ± 4	103 ± 6	90 ± 7	82 ± 6
CV (in ml)	58 ± 5	86 ± 4	86 ± 7	82 ± 7	125 ± 8	110 ± 8	100 ± 8	85 ± 6	88 ± 7	92 ± 10
	38 ± 1	99 ± 2	100 ± 2		61 ± 3	64 ± 3	71 ± 3	75 ± 4	76 ± 3	73 ± 3

TABLE 2. Hemodynamic Indices in Burned Rabbits of Three Groups

Group of animals	Parameter studied	Initial data	After burning (% of initial value)			After injection of dextran (% of initial values)			
			immediately	10-15 min	30 min	immediately	10 min	30 min	1 h
1) Sharp increase in CVP	MVH (in ml/min/kg)	206±20	55±2	37±3	25±4	54±17	64±21	47±11	41±17
	AP (in mm Hg)	87±5	101±5	76±9	73±10	93±14	81±15	94±4	79±13
	R (in dynes · sec · cm^{-5})	$14 \cdot 10^3 \pm 10^3$	172±4	203±20	277±18	231±53	182±58	266±61	241±67
	pO_2 (in ml/min)	21,2±3	80±8	76±8	60±12	50±13	51±14	64±18	43±12
	MVR (in liters/min)	0,74±0,1	147±30	122±29	130±37	128±32	110±30	97±36	83±38
	CPV (in ml)	132±32	121±10	90±9	—	124±5	119±8	123±6	—
	CBV (in ml)	210±55	124±9	93±11	—	101±6	96±2	105±2	—
2) No Increase in CVP	MVH (in ml/min/kg)	180±14	63±5	42±7	36±7	133±25	89±7	66±6	42±6
	AP (in mm Hg)	97±7	78±8	52±9	52±6	89±8	89±6	76±6	65±7
	R (in dynes · sec · cm^{-5})	$16 \cdot 10^3 \pm 10^{-3}$	122±12	143±26	157±36	80±15	100±16	125±20	167±28
	pO_2 (in ml/min)	22,4±2	80±4	70±12	63±10	96±15	91±9	81±6	68±4
	MVR (in liters/min)	0,73±0,06	146±12	114±11	117±22	140±11	127±16	113±9	118±12
	CPV (in ml)	107±20	—	79±5	—	142±26	113±18	112±15	107±23
	CBV (in ml)	179±32	—	80±4	—	112±18	129±21	96±12	97±12
3) Increase in CVP the same as in the control animals	MVH (in ml/min/kg)	140±6	84±4	66±4	54±4	165±17	115±15	83±14	66±11
	AP (in mm Hg)	79±4	83±6	70±6	72±4	109±5	106±5	97±6	88±6
	R (in dynes · sec · cm^{-5})	$15 \cdot 10^3 \pm 10^{-3}$	107±5	109±5	144±13	70±6	95±10	125±12	149±20
	pO_2 (in ml/min)	23,8±1	87±5	86±4	76±5	95±7	94±6	83±2	66±5
	MVR (in liters/min)	0,87±0,05	136±8	130±10	140±18	128±13	120±10	107±8	104±7
	CPV (in ml)	123±6	—	89±7	—	145±26	114±8	120±8	114±7
	CBV (in ml)	197±6	—	86±8	—	123±23	99±6	105±5	110±11

termined by means of a gas meter. All these measurements were made simultaneously in 11 control and 15 burned animals.

EXPERIMENTAL RESULTS

The results of the study of the various parameters after administration of dextran are given in Table 1. In the control rabbits the increase in CPV and CBV caused by injection of dextran continued for 3 h. The HN and hemoglobin values were correspondingly below normal for the same period. CV and MVH rose but returned to normal after 3 h. AP remained close to normal throughout the period of observation. R at first fell, but returned to normal after 2 h. CVP was increased by 34 ± 3 mm water and returned to its initial level on the average 2 h after the injection of dextran. pO_2 and MVR were virtually unchanged. After falling sharply after burning, the MVH rose at first approximately to its initial level under the influence of dextran administration, but it was reduced again after 30 min. The other parameters changed in the same way.

An adequate analysis of the results is possible only if allowance is made for the fact that the response of the CVP to dextran injection differed in principle in the different animals with burns [2]. The mean values of the parameters studied in the burned rabbits are therefore given in Table 2 combined into three groups depending on the character of the change in the CVP under the influence of dextran (a sharp rise in CVP in group 1, no rise in CVP in group 2, and increase in CVP similar to the control in group 3).

As Table 2 shows, a sharp decrease in MVH during the first few minutes after burn trauma was characteristic of the animals of all three groups before the injection of dextran, but the fall was sharpest in the rabbits of group 1, despite the fact that their CBV differed only a little from normal. Injection of dextran into these rabbits led to a sharp increase in CVP (by 90 mm water or more), but although MVH rose slightly it still remained far below its initial level. There was no corresponding fall in R. The CBV returned to normal after the injection of dextran. Two of the five rabbits in this group died a few minutes after the injection of dextran. These findings are evidence that the main cause of the decrease in MVH in the animals of this group is cardiac failure developing after burn trauma. Injection of dextran not only did not abolish this underlying cause of the decrease in MVH but, on the contrary, it aggravated the heart failure.

In the rabbits of group 2, in which the CVP did not rise in response to injection of dextran because of a decrease in venous tone, the greatest decrease in CBV after burning was observed. The main cause of the decrease in MVH in these animals was thus a decrease in the venous return. Injection of dextran abolished this cause for some time, and as a result the MVH rose to 33% above normal immediately after the injection of dextran. However, MVH began to decrease 10 min later despite the fact that the CBV 1 h after the injection of dextran was still practically equal to normal. This is in good agreement with the view that the decrease in MVH is connected with a decrease in the tone of the low pressure system.

In the animals of group 3 in which the increase in CVP after injection of dextran was similar to the control, injection of dextran caused neither heart failure nor a decrease in venous tone. In this group also the decrease in MVH after burning was less sharp, and the duration of its increase after the injection of dextran was more prolonged. In the animals of this group the decrease in MVH was possibly due both to a decrease in the venous return and to heart failure.

After burning pO_2 fell parallel with MVH. After the injection of dextran pO_2 rose almost to normal values in groups 2 and 3, in which MVH rose above the normal level; in group 1, in which MVH still remained much below normal even after the injection of dextran, pO_2 continued to decrease. This suggests that the decrease in pO_2 in burn shock was due to a decrease in MVH.

It can be concluded from these results that the cause of the decrease in MVH in burn shock varies. MVH may decrease as a result both of a decrease in the venous return and of cardiac failure. The relative importance of these two factors can be revealed by a rapid artificial increase in CBV.

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