CHANGES IN THE CIRCULATION AND RESPIRATION IN LETHAL BURN SHOCK

N. A. Len'kova

UDC 617-001.36-02:617-001.17-036.88-07:616.12-008.1+616.24-008.4]-072.7

Changes in various parameters of the hemodynamics and gas exchange taking place during lethal shock caused by burns covering 30% of the body surface were recorded simultaneously in experiments on rabbits. The sharp fall in the minute volume of the heart during the first minutes after burn trauma was shown to depend both on a decrease in the venous return and on cardiac failure. In most cases the arterial pressure falls sharply only in the terminal state. It is postulated that the oxygen consumption falls because of a decrease in the minute volume of the heart. The activity of the vasomotor and respiratory centers is not diminished until the terminal state.

Both in animals [4, 6, 7, 9-11] and in man [12, 13], burn shock is accompanied by a sharp decrease in the minute volume of the heart (MVH) and the oxygen consumption [4, 7] and a decrease in the circulating volumes of plasma (CPV) and blood (CBV). According to some workers, CBV and MVH decrease simultaneously [13]; according to others, the decrease in MVH is not due to the fall in CBV [6, 8-10]. In burn shock the arterial pressure (AP) falls and they regard this as an indicator of the severity of the shock [2, 3], whereas other workers do not consider that lowering of the AP is characteristic of burn shock [1, 6, 8, 10].

By measuring various parameters of the hemodynamics and gas exchange simultaneously, the writer attempted to establish the presence or absence of correlation between their changes during the development of burn shock.

EXPERIMENTAL METHOD

The experimental animals were 29 rabbits anesthetized with urethane (1 g/kg, intravenously) and 10 rabbits served as the control group. Tracheotomy was performed and catheters inserted through the jugular vein into the right atrium, into the femoral vein, and the femoral artery. A "thermoprobe" (a catheter carrying a thermistor) was introduced via the carotid artery into the aorta. Simultaneous measurements of MVH, AP, the central venous pressure (CVP), the O_2 consumption, the minute volume of respiration (MVR), CPV, the hematocrit index and hemoglobin (Hb) concentration in the arterial blood, and the temperature of the blood in the aorta began 10-15 min later. The ECG was recorded at the same time. Measurements were made every 30 min (CPV, hematocrit index, and Hb every hour) for 5 h.

After the original measurements the surface of the abdominal wall and thighs of 19 rabbits was burned with boiling water for 1 min. Measurements were made immediately after (1-3 min) burning, again after 10-15 and 30 min, and thereafter hourly until the animals died.

MVH and the central blood volume were calculated from the temperature dilution curve, for which purpose 1.5 ml of Ringer's solution, cooled to 1-5° C, was injected into the right atrium. The CVP was recorded in the right atrium by means of a water manometer. AP was recorded in the femoral artery with an electromanometer. The CPV was determined by the dye (Evans' Blue) dilution method, 10 min after

Laboratory of Experimental and Clinical Physiology, A. A. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. A. Vishnevskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 77, No. 5, pp. 14-17, May, 1974. Original article submitted July 10, 1973.

TABLE 1. Changes in Parameters of Hemodynamics and Respiration in Rabbits with Burn Shock (M ± m)

Parameter determined	Initial data	After burning (in % of initial)						
		immediately	10–15 min	30 min	1 h	2 h	3 h	4 h
MVH (in ml/min/kg body weight)	165±7	60±8	45±5	38±3	37±3	30±3	25±2	—
Systolic volume (in ml/kg body weight)	0,63±0,04	70±13	48±8	44±5	39±4	36±4	32±0,7	—
AP (in mm Hg)	92±3	95±3	69±4	70±3	63±4	72±4	65±4	51±9
Peripheral resistance (in dynes·sec·cm^{-5})	17·10^3±0,9·10^3	166±21	180±26	174±16	182±15	266±26	280±30	—
O_2 consumption (in ml/min)	22,4±1	88±3	73±5	77±4	59±4	53±5	38±4	33±3
MVR (in liters/min)	0,83±0,05	136±10	124±10	133±9	116±12	107±11	67±7	49±11
Central blood volume	52±4	86±4	86±6	75±4	70±5	67±12	75±11	—
CPV	124±9	—	90±9	77±7	71±5	79±10	72±11	78±18
CBV (in ml)	208±11	—	94±9	77±9	69±5	77±11	64±13	63±6

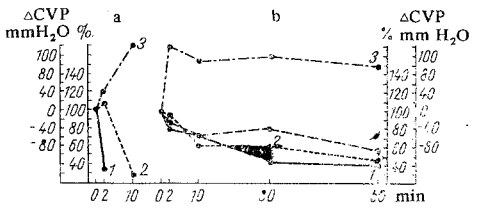

Fig. 1. Changes in parameters of hemodynamics in response to lethal burn trauma in two (a and b) different experiments. Abscissa, time after burning (in min); ordinate, changes in MVH, CBV, and AP (in % of initial values) and CVP (in mm water): 1) MVH; 2) AP; 3) CVP; 4) CBV.

injection. CBV was calculated. The O_2 consumption was determined continuously by means of a closed system with absorption of CO_2 and an automatic supply of O_2. The MVR was determined by a gas meter. The coefficient of oxygen utilization (COU) was calculated. The peripheral vascular resistance was calculated from the values of MVH and AP. The systolic value of the heart also was calculated.

EXPERIMENTAL RESULTS

During the first few hours of the experiment the parameters investigated changed only negligibly in the control animals.

Of the 19 animals receiving burns three died the first 30 min. The longest period of survival after burning was 5 h (3 rabbits).

The mean changes in the parameters of the hemodynamics and gas exchange arising after burning are given in Table 1. Changes in the parameters of the hemodynamics in response to burn trauma in particular experiments are illustrated in Figs. 1-3. It will be clear from Table 1 that MVH fell sharply during the first few minutes after burning and thereafter it continued to fall gradually. The systolic volume fell almost as sharply. Meanwhile CPV and CBV 10-15 min after burning on the average were still close to normal. They fell later, but by a much lesser degree than MVH. In certain experiments, however, there was a marked decrease in CPV and CBV during the first minutes after burning (Figs. 1b and 2). The central blood volume fell parallel with CPV.

Of the 11 cases in which the CVP was measured, in 4 it was unchanged or changed only very slightly after burning, in 3 it fell, and in 4 rabbits it rose sharply, but 3 of them died during the 30 min after burning. One of these experiments is illustrated in Fig. 1a. Together with the sharp rise in CVP there was also a very marked decrease in MVH, the AP fell rapidly, and the animal died. This suggests the rapid development of heart failure as a result of burn trauma. Another case, also with a sharp increase in CVP immediately after burning is illustrated in Fig. 1b. In this case the increase in CVP was observed despite a marked fall in CBV during the first minutes after burning. Meanwhile AP fell fairly sharply. These findings suggest that this rabbit also developed heart failure which was followed by a decrease in CBV and also by a decrease in MVH immediately after burn trauma.

An experiment in which a parallel decrease in CBV, CVP, and MVH was observed immediately after burning. Presumably the main cause of the decrease in MVH in this rabbit was the decrease in CBV. An experiment in which CPV and CVP did not fall after burning and AP was maintained at a fairly high level is shown in Fig. 3; despite these facts, MVH fell sharply after burning. As the writer showed previously, in such cases special measures must be taken to detect the decrease in venous tone responsible for the diminution of the venous return. This explains the absence of an increase in CVP and it masked the heart failure. In such cases the fall in MVH was due to the simultaneous effect of several different factors.

It will be clear from Table 1 that the AP, after falling 10-15 min after burning, remained approximately constant until the terminal state. The peripheral resistance rose sharply. This points to

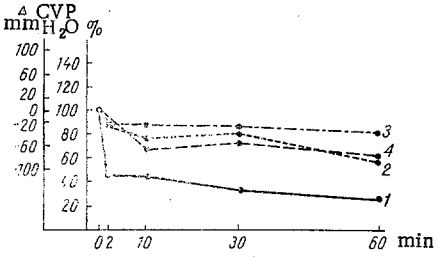

Fig. 2

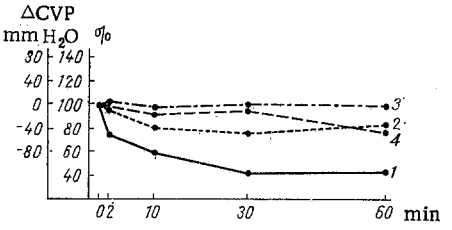

Fig. 3

Fig. 2. Changes in parameters of hemodynamics in response to lethal burn trauma. Legend as in Fig. 1.

Fig. 3. Changes in parameters of hemodynamics in response to lethal burn trauma: 4) CVP remainder of legend as in Fig. 1.

preservation of the activity of the vasomotor center until the animals died. The O_2 consumption fell parallel with the decrease in MVH. MVR, which was increased immediately after burning, remained high for 1 h and then started to fall. Against the background of the increased MVR, COU fell by a greater degree than the O_2 consumption, evidence of relative hyperventilation and maintenance of the excitability of the respiratory center. The hematocrit index and Hb concentration in the arterial blood remained on the average close to normal. There was a mean rise of 0.6° C in the blood temperature immediately after burning followed by a fall of 4° C 3 h after burning.

It can be concluded from these results that in most cases AP does not reflect the severity of burn shock, for it does not fall sharply until the terminal state. The sharp fall in MVH during the first few minutes after burning is a characteristic feature of burn shock. The causes of the fall in MVH may be the reduced venous return as a result of the decrease in CBV or in the venous tone and also the heart failure. All these factors probably contribute but their role in each individual case may differ. In some cases the fall in MVH can be clearly picked out as the main cause.

LITERATURE CITED

1. T. Ya. Ar'ev, Klin. Med., No. 3, 7 (1962).
2. P. N. Gol'dberg, in: The Physiology of Threatening States [in Russian], No. 1, Frunze (1963), p. 53.
3. D. M. Grozdov, in: Shock and Terminal States [in Russian], Leningrad (1960), p. 290.
4. A. N. Kuznetsova and B. I. Lektorskii, Abstracts of Proceedings of the 1st Republican Conference on Burns [in Russian], Kiev (1964), p. 35.
5. V. G. Trubachev, Changes in the Circulating Blood Volume after Extensive Burns Combined with Blood Loss, Author's Abstract of Candidate's Dissertation [in Russian], Leningrad (1967).
6. N. A. Fedorov, V. B. Troitskii, and S. A. Lazarevskii, Abstracts of Proceedings of the 1st Republican Conference on Burns [in Russian], Kiev (1964), p. 36.
7. L. L. Shik, A. N. Kuznetsova, and B. I. Lektorskii, in: Proceedings of the 4th Scientific Conference on the Problem of Burns [in Russian], Leningrad (1965), p. 276.
8. E. L. Dobson and G. E. Warner, Circulat. Res., 5, 69 (1957).
9. H. A. Fozzard, in: Research in Burns, Philadelphia (1962), p. 109.
10. I. P. Gilmore and S. W. Handford, J. Appl. Physiol., 8, 393 (1956).
11. D. D. Michie, R. S. Goldsmith, and A. D. Mason, Circulat. Res., 13, 468 (1963).
12. B. A. Pruitt, A. D. Mason, and J. A. Mongrief, J. Trauma, 11, 36 (1971).
13. D. W. Richards, Harvey Lectures (1943-1944), 39, 217 (1944).