ROLE OF INCREASED PERIPHERAL VASCULAR RESISTANCE IN BURN SHOCK

L. L. Shik and N. A. Len'kova

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In intact rabbits and rabbits with exclusion of the aortic and carotid sinus reflexogenic zones the effects of lethal burn trauma were studied on indices of the hemodynamics and respiration. Similar changes were found in the cardiac output and total oxygen consumption in the rabbits of the two groups. By contrast with intact animals, in rabbits with exclusion of the reflexogenic zones burns did not lead to any sharp rise of peripheral vascular resistance. The systemic arterial pressure fell correspondingly in these animals by a much greater degree than in the intact rabbits. The survival period of the denervated rabbits after burns was shorter than that of the intact rabbits. It is concluded that the increase in the peripheral vascular resistance in burn shock is of a reflex compensatory nature.

KEY WORDS: burn shock; peripheral vascular resistance; hemodynamics.

In burn shock, a high total peripheral vascular resistance is observed [1, 3, 5, 6, 8, 9, 12, 17]. The increase in peripheral resistance is explained by the increased formation of vasoactive substances [6, 13] and aggregation of red cells in the microcirculatory system [14] observed in response to burn trauma. According to some workers [14, 16], a sharp increase in peripheral resistance is the essential cause of the fall in cardiac output in burn shock. Evidence has been obtained of the effectiveness of sympatholytics in the treatment of patients with severe burns [16] and in experimental animals [2, 4, 15]. Meanwhile other workers observed no change in the cardiac output after administration of a sympatholytic [11] and, on the other hand, after administration of vasopressin in association with infusion therapy the survival rate of the experimental animals was increased [18]. Burn trauma causes a fall in cardiac output which, for practical purposes, is rapid [3, 7, 10, 12] and it can be tentatively suggested that the observed increase in the peripheral vascular resistance is a compensatory reflex response to this fall.

The object of the present investigation was to test this hypothesis experimentally.

EXPERIMENTAL METHOD

Experiments were carried out on 30 rabbits under urethane anesthesia (1 g/kg, intravenously). Burn trauma was inflicted on the surface of the abdomen and side (30% of the body surface) by application of boiling water for 1 min. Burns were inflicted on 11 animals 20-40 min after division of the cardiac depressor and carotid sinus nerves.

Simultaneous determinations were made of the cardiac output (CO), systemic arterial pressure (BP), total peripheral vascular resistance (TPR), total oxygen consumption (TOC), and minute respiratory volume (MRV). The measurements were made 10 and 30 min after burning and every subsequent 30 min until death of the animals. CO was determined by the thermodilution method. Ringer's solution, cooled to 1-5°C, was injected in a volume of 1.5 ml into the right atrium and changes in the blood temperature were recorded in the arch of the aorta by means of an electrothermometer. A medical needle thermistor (980Ω at 20°C, $\alpha = -2.8\%$ at 1°C) was used as the temperature sensor. The thermodilution curve was recorded on an EPP-09M3 automatic self-recording potentiometer. BP was recorded in the femoral artery by means of electromanometer. TPR was calculated by the usual formula. TOC and MRV were determined continuously by means of a closed system with CO_2 absorption and automatic O_2 supply.

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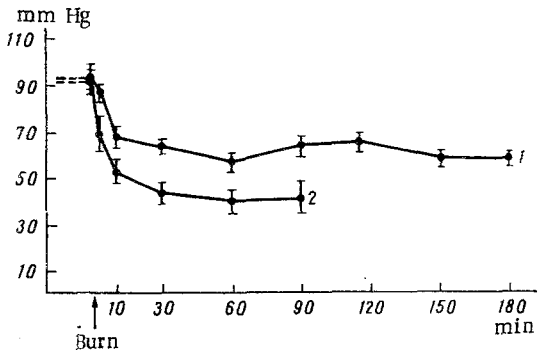

Fig. 1

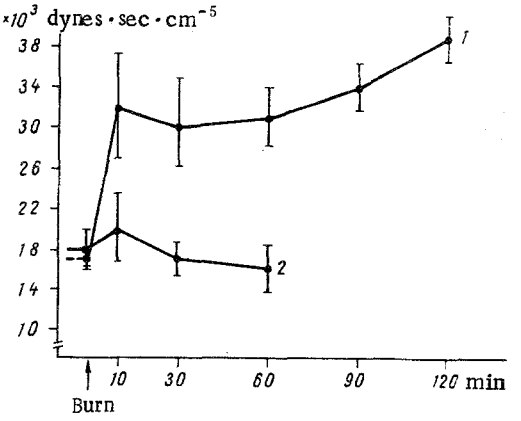

Fig. 2

Fig. 1. Changes in BP in response to burn trauma in rabbits with intact (1) and denervated (2) aortic and carotid sinus reflexogenic zones. Differences between corresponding points of curves 1 and 2 statistically significant ($P < 0.05$). Abscissa, time after burning (in min); ordinate, BP (in mm Hg).

Fig. 2. Changes in TPR in response to burn trauma in rabbits with intact (1) and denervated (2) aortic and carotid sinus reflexogenic zones. Differences between corresponding points on curves 1 and 2 statistically significant ($P < 0.01$). Abscissa, time after burning (in min); ordinate, TPR (in dynes · sec · cm^{-5}).

EXPERIMENTAL RESULTS

Before burn trauma, in animals with normal innervation of the aortic and carotid sinus reflexogenic zones (Group 1), and in animals with divided depressor and carotid sinus nerves (Group 2) CO was 165 ± 7.03 and 178 ± 15.7 ml/min · kg respectively. Burns caused an instantaneous sharp decrease in CO in both groups of animals: During 10 min CO fell to 45 ± 5.2% from its initial level in the rabbits of group 1 and to 50 ± 9.1% in the rabbits of group 2. A subsequent gradual fall of cardiac output was observed in both groups of animals until they died. TOC fell at the same time: 10 min after burning it was 72 ± 5.4% of its initial level (7.9 ± 0.3 ml/min · kg) in the rabbits of group 1 and 72 ± 6.6% (8.6 ± 0.5 ml/min · kg) in the rabbits of group 2. After 1 h TOC was 59 ± 3.9 and 56 ± 7.4% of its initial value respectively. Meanwhile MRV increased to 133 ± 8.2% of the initial value (0.30 ± 0.02 liter/min · kg) in the animals of group 1 and to 131 ± 12.0% (initial value 0.25 ± 0.01 liter/min · kg) in the animals of group 2. Because of the increase in pulmonary ventilation and the high O_2 concentration in the arterial blood, respiratory failure cannot be considered as the cause of the decrease in TOC. The high degree of correlation between the changes in TOC and MRV ($r = + 0.89$ and $+ 0.8$ respectively for Groups 1 and 2) suggests that changes in TOC were due to changes in the cardiac output of the burned animals.

In the animals with an intact innervation of the aortic and carotid sinus, reflexogenic zones burn trauma did not cause BP to fall sharply. In the rabbits of Group 1, it fell on average by 30% in the course of 10-15 min after trauma and remained close to that level practically until the onset of the terminal state (Fig. 1). Division of the depressor and carotid sinus nerves before burning led to a rise in BP on average by 14% (from 98 ± 6.5 to 112 ± 7.8 mmHg). The increase lasted for only a comparatively short time, and after 20-40 min, approaching the time of burning, the mean BP was virtually back to its initial level. By contrast with the animals of Group 1, in those of Group 2 burn trauma caused a rapid and considerable fall in BP (Fig. 1). From 10 to 15 min after trauma BP in the rabbits of Group 2 was 59% of its initial level and it continued to decline thereafter. The differences between changes in BP in response to burn trauma in the rabbits with intact and denervated aortic and carotid sinus reflexogenic zones were statistically significant (Fig. 1).

In the rabbits with intact innervation of the reflexogenic zones, simultaneously with the sharp decrease in cardiac output and maintenance of BP at a relatively high level, TPR rose very sharply immediately after burning and continued to rise progressively until the

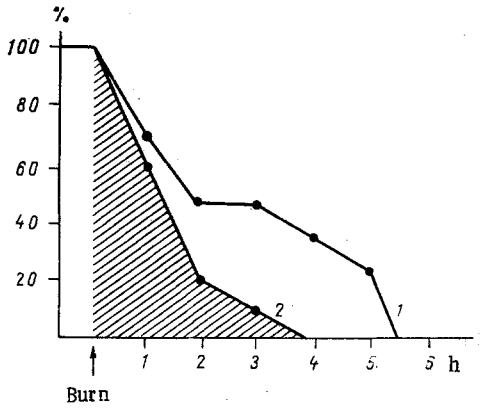

Fig. 3. Survival rate of rabbits after burn trauma. 1) Rabbits with intact, 2) rabbits with denervated aortic and carotid sinus reflexogenic zones. Differences statistically significant. Significance calculated for 3 h by χ^2 method. Abscissa, time after burning (in h); ordinate, percentage of animals surviving at the given time.

terminal state (Fig. 2). From a value of $17 \times 10^3 \pm 0.9 \times 10^3$ dynes · sec · cm^{-5} before burning, TPR virtually doubled in the course of 10 min after trauma, to $32 \cdot 10^3 \pm 5.1 \cdot 10^3$ dynes · sec · cm^{-5}; after 1.5 h it was 246 ± 12.0%, and after 3 h it was 302 ± 28.6% of its initial level.

In rabbits with denervated aortic and carotid sinus reflexogenic zones TPR did not increase in response to burn trauma (Fig. 2). Relative to its initial level ($18 \cdot 10^3 \pm 1.8 \cdot 10^3$ dynes · sec · cm^{-5}) it was 110% 10 min, 99% 30 min, and 98% 1 h after burning. The differences between the changes in TPR in response to burn trauma in the rabbits of groups 1 and 2 were statistically significant. It can be concluded from these differences that the peripheral vascular resistance increases after burning as a reflex response to the sharp fall in cardiac output and the commencing fall in BP. The reflex increase in TPR maintains BP at a relatively high level until the onset of the terminal state.

A statistically significant difference was found in the survival rate of the animals of groups 1 and 2 at different times after trauma (Fig. 3).

Although the compensatory reaction of an increase in TPR could not save the animals from death under the conditions of burn trauma used, it nevertheless prolonged their life and thus played an adaptive role.

The results of these experiments undoubtedly indicate that the rise in TPR is not the cause of the reduction in cardiac output. It will be recalled that the work of the heart increases with an increase in TPR only if it leads to an increase in the systematic BP. In burn shock the increase in TPR is a compensatory reflex response, preventing a sharp decline in BP, which is nevertheless maintained at a lower level than initially. There are therefore no grounds for regarding the rise in TPR as an unfavorable response or for counting on a favorable effect from measures selectively aimed at reducing TPR. On the contrary, measures promoting an improvement in the hemodynamics (such as infusion therapy, leading to an increase in cardiac output) make the compensatory vaseconstriction superfluous and lead to normalization of TPR [4].

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CHANGES IN CONTRACTILE ACTIVITY OF THE RABBIT MYOCARDIUM AS A RESULT OF BURN SHOCK OF VARIED DURATION

L. A. Vasilets, E. G. Vornovitskii, and B. I. Khodorov

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Experiments were carried out on papillary muscles isolated from the rabbit heart 10, 60, or 180 min after thermal injury to the animal. Isometric contractions were recorded during stimulation of the preparation at changing frequencies (within the range from 0.1 to 2 Hz) and during poststimulation potentiation. The degree of disturbance of myocardial contractility as a result of burns was found to increase with an increase in the duration of burn shock: In all the papillary muscles isolated 3 h after burning and in 50% of those isolated 1 h after burning the "biphasic" frequency—strength (f—P) relationship characteristic of the normal myocardium was converted into "monophasic" (the amplitude of the contractions decreased progressively with an increase in frequency) and poststimulation potentiation, normally absent, appeared. After shock lasting 10 min, poststimulation potentiation was observed only in some preparations and no change in f—P was present. The normal inotropic relationships of the myocardial rhythm were restored after a twofold increase in $[Ca^{++}]_o$ or after prolonged (3-4 h) perfusion of the preparation with normal Tyrode solution. Changes in inotropic relationships of the myocardial rhythm in burn shock were similar to the changes in f—P observed after blockade of the calcium channels with compound D-600.

KEY WORDS: burn shock; contractile activity of the myocardium; frequency—strength dependence.

The study of the causes of the decrease in cardiac output in response to thermal injury has recently attracted increasingly wide attention. By studying changes in the contractility of the heart in the intact organism several workers have obtained indirect evidence of the development of insufficiency of the heart muscles during burns [2-5]. Myocardial depressants have been found in some investigations in the blood serum of burned animals and patients [6, 8, 11]. To obtain direct proof of the effect of thermal trauma on the myocardium, the present writers previously studied the contractile activity of isolated fragments of myocardium taken from the heart of rabbits 1 h after burn trauma [1]. These experiments showed that myocardial contractility was disturbed in 50% of the preparations. The disturbance was of the type that the normal "biphasic" dependence of the strength of myocardial contractions on the frequency of stimulation was converted into "monophasic" (within the frequency range 0.1 to 2 Hz).

Laboratory of Physiology and Laboratory of Biophysical Research, A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Sciences of the USSR V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 87, No. 5, pp. 402-405, May, 1979. Original article submitted July 14, 1978.

