ROLE OF INCREASED PERIPHERAL VASCULAR RESISTANCE IN BURN SHOCK

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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, α = -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 an electromanometer. TPR was calculated by the usual formula. TOC and MRV were determined continuously by means of a closed system with CO₂ absorption and automatic O₂ supply.
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 O₂ 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