ELASTICITY OF THE LOW-PRESSURE SYSTEM IN BURN SHOCK

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The relationship between the central venous pressure (CVP) and the circulating blood volume (CBV) during an artificial increase in the CBV by injection of dextran to the amount of 2% of the body weight was investigated in experiments on rabbits with burn shock and the corresponding control. In the control animals an increase in the CVP was always observed in response to injection of dextran. Animals with burn shock were divided into three groups depending on changes in the CVP in response to dextran injection: the animals of group 1 were indistinguishable from the control, in the animals of group 2 the CVP rose much higher than in the control, while in the animals of group 3 the CVP was unchanged.

The circulatory system is divided into high- and low-pressure systems [12], the latter including the veins, the right heart, and the pulmonary circulation. It has been shown that the veins of the systemic circulation and the whole of the pulmonary circulation constitute a single system [12, 16, 18]. The low-pressure system has incomparably greater elasticity than the arterial system and contains 80-85% of the total blood volume [13, 20, 25]. The properties of this elastic system are reflected most fully by the P–V diagram [12], i. e., the curve showing the relationship between the venous pressure (P) and blood volume (V). The P–V diagram of the vascular system as a whole can therefore be considered to be determined chiefly by the properties of the low-pressure system [12, 13]. Elevation of the central venous pressure (CVP) with an increase in the circulating blood volume (CBV) has been demonstrated [1, 2, 8, 14, 16, 17, 19, 22, 23, 26]. According to some of these investigators, the relationship between changes in the blood volume and pressure is linear [14, 16, 19, 22, 23], while according to others it is more complex [8, 17, 21]. It has also been shown that any change in venous tone leads to changes in the P–V relationship [10, 11, 13, 24, 25].

Absolute values of the venous pressure in animals and man are very variable under normal [2, 13, 14, 20, 21] and pathological conditions, for example in shock [3, 15]. One reason for this variability is that the CVP is determined by three factors: the tone of the low-pressure system, the CBV, and the cardiac activity. Many workers have accordingly concluded that the most valuable information is given, not by isolated measurements of the CVP, but by serial measurements especially in response to an increase in the blood volume, i. e., the P–V curves, which give an idea not only of the state of the venous system and the CBV deficiency, but also of cardiac insufficiency [7, 9, 20, 21, 25].

The object of this present investigation was to compare the P–V curves in rabbits under normal conditions and in burn shock.

EXPERIMENTAL METHOD

Experiments were carried out on rabbits anesthetized with urethane (1 g/kg). The P–V ratio in burn shock was studied in 32 animals, and 37 animals constituted the control group. A burn was inflicted on the surface of the abdomen and thighs (30% of the body surface) with boiling water for 1 min. The arterial pres-

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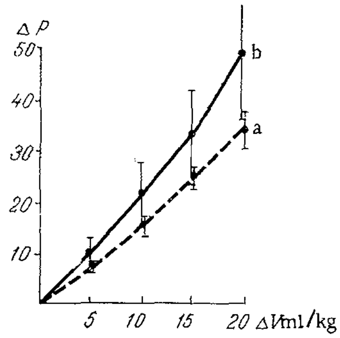

Fig. 1

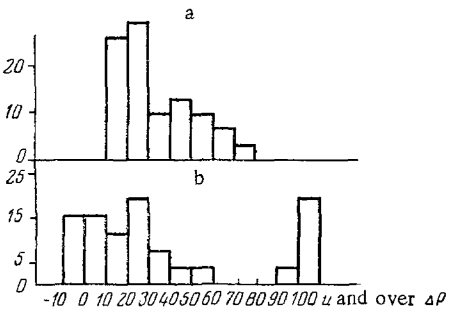

Fig. 2

Fib. 1. Changes in CVP as a function of changes in blood volume. Abscissa, increase in blood volume (in ml/kg); ordinate, increase in CVP (in mm water): a) control animals; b) burned animals, vertical lines indicate mean error.

Fig. 2. Histogram of distribution of increase in CVP in response to injection of dextran (20 ml/kg). Abscissa, increase in CVP (in mm water); ordinate, number of experiments (in %): a) control animals; b) animals with burns.

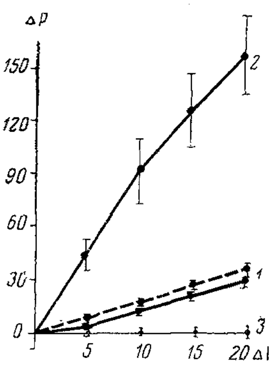

Fig. 3. Changes in CVP as a function of changes in blood volume. Abscissa, increase in blood volume (in ml/kg); ordinate, increase in CVP (in mm water). Broken line represents control animals; 1,2, and 3) animals with burns; vertical line represents mean error.

sure (AP) was measured in the carotid or femoral artery by an electromanometer and the CVP in the right atrium was measured by a water manometer. The catheter for measuring CVP was inserted into the right atrium via the jugular vein. To produce an artificial increase in the CBV, dextran (6% solution) was injected into the femoral vein at a rate of 5 ml/min. The total volume of dextran injected was 2% of the body weight. Dextran was injected into the experimental animals 15-30 min after burning.

EXPERIMENTAL RESULTS AND DISCUSSION

In normal rabbits the CVP varied from -15 to +54 mm water with a mean value of 16.1 ± 1.91 mm water. The mean AP for these animals was 91 ± 1.65 mm Hg. The AP 10-15 min after burning was reduced by 3-65% (mean 30%) of its initial value. In six of the 32 burned rabbits the CVP was increased 15-30 min after burning on the average by 21.7 mm water (from 7 to 57 mm water), in 13 animals the CVP was reduced on the average by 28.6 mm water (from 6 to 65 mm water), and in 13 animals the CVP was unchanged for 15-30 min after burning.

After injection of dextran into the control rabbits the CVP was increased in all the animals. Injection of dextran into the burned rabbits led to a similar rise of CVP in some animals, to an even sharper increase in the CVP in others and, finally, to little or no change in CVP in the animals of a third group. The mean increase in CVP after injection of dextran was the same for the burned rabbits as a whole as for the control animals. The P—V diagram based on the mean data after injection of dextran into the control and experimental rabbits is illustrated in Fig. 1. The differences (ΔP) between them were not statistically significant at any values of ΔV.

The histograms of distribution of changes in CVP in the control and burned rabbits in response to injection of dextran in a dose of 20 ml/kg are shown in Fig. 2. It will be seen that the increase in CVP in most of the control animals was 20-30 mm water, with a scatter on the low side to 10 mm water and on the high side to 60 mm water, or very rarely to 80 mm water. In some of the burned rabbits the increase in CVP also varied from 10 to 60 mm water. However, groups of animals could be distinguished for which CVP in response to injection of dextran either remained virtually unchanged (P from −10 to +10 mm water) or increased very considerably (from 90 to 100 mm water or more), which was not observed in the control animals. Burned rabbits could thus be divided into three groups: 1) animals whose CVP increased in response to injection of dextran within the same limits as the control animals, 2) animals with a particularly marked reaction of the CVP, and 3) animals for which the CVP did not react to an increase in the blood volume. These rabbits accounted for 44, 19, and 37%, respectively, of the total number of burned animals.

The $\dot{P}-V$ curves plotted from mean data for these groups of rabbits and for the control animals are illustrated in Fig. 3. The difference between the data for the experimental and control rabbits are statistically significant (P for all corresponding points < 0.01; see curves 2 and 3) while for the points in curve 1 and the control they are not significant.

In the burned rabbits of group 1, for which the increase in CVP under the influence of dextran was the same as in the control, the decrease in CVP to its initial level occured on the average in a much shorter period (30 min) than in the control animals (60 min). Among the rabbits of group 2, in which a sharp increase in CVP was observed after dextran injection, in three animals it was considerably increased 1 h after the injection, two rabbits died within 30 min after dextran injection when the CVP was high, and only in one animal did the CVP return to normal 1 h after the injection. In the rate of restoration of their normal CVP the animals of group 2 thus differed significantly from the rabbits of group 1.

During injection of dextran the AP of the control animals increased on the average by 11 ± 1.71 mm Hg and in the burned rabbits on the average by 28 ± 2.33 mm Hg. The differences between the increases in AP in the burned rabbits and control animals were statistically significant. This was connected with the fact that after burning the AP was lowered and the injection of dextran thus helped to restore it to normal, whereas in the control animals the injection of dextran caused the AP to rise above normal.

The results of these experiments show that burns can cause changes in the elastic characteristics (tone) of the venous system: in 37% of cases no change or a very slight change in CVP was found when the volume of the low-pressure system was increased by injection of dextran after burn trauma. In these cases there is evidently a very large increase in the elasticity of the low-pressure system as a result of burn trauma.

Some workers regard the CVP as an indicator of the balance between the venous return and cardiac output. A considerable increase in the CVP in response to an increase in the blood volume and venous return under pathological conditions is regarded as a sign of myocardial insufficiency [9, 20, 25]. Most probably in the present experiments the very large increase in CVP in response to injection of dextran in some of the burned animals can be explained by the development of myocardial insufficiency after burning, a conclusion supported by evidence in the literature [4, 5, 6].

As was stated above, changes in the CVP after burns may take place in different directions, in agreement with data in the literature [15]. No connection could be found between the initial values of the CVP and its changes after burning, on the one hand, or the response of the CVP to injection of dextran, on the other hand.

The changes in CVP in response to burn trauma are very variable, evidently because burns can give rise simultaneously to changes in tone of the low-pressure system, to cardiac failure, and to a decrease in the CBV. The combination of these three factors probably determines the resultant level of the CVP in each concrete case.

LITERATURE CITED

1. Ya. M. Bass and M. A. Cherkasskii, in: The Venous Pressure [in Russian], Leningrad (1939), p. 27.
2. V. A. Val'dman, Vascular Tone [in Russian], Leningrad (1960).
3. A. N. Martynchev, I. I. Nikitina, and M. A. Trunin, The Venous Pressure in Clinical Surgery [in Russian], Leningrad (1963).
4. A. N. Medelyanovskii and V. B. Troitskii, Byull. Éksperim. Biol. i Med., No. 6, 32 (1965).
5. N. A. Fedorov, V. B. Troitskii, and S. A. Lazarevskii, Abstracts of Proceedings of the First Republican Conference on Burns [in Russian], Kiev (1964), p. 36.
6. L. L. Shik, G. A. Bykov, V. F. Gordeev, et al., Abstracts of Proceedings of the First All-Union Conference on Thermal Burns [in Russian], Moscow (1972), p. 87.
7. M. Atik, Am. Surg., 33, 118 (1967).
8. C. Burri and W. Müller, Anaesthetist, 15, 132 (1966).

9. C. E. Eastridge, F. A. Hughes, J. R. Prather, et al., Am. Surg., 32, 121 (1966).
10. J. W. Eckstein and W. K. Hamilton, J. Clin. Invest., 36, 1663 (1957).
11. J. W. Eckstein, W. K. Hamilton, and J. M. McCammond, Anesthesiology, 22, 525 (1961).
12. O. H. Gauer and J. P. Henry, Physiol. Rev., 43, 423 (1963).
13. O. H. Gauer and H. L. Thron, Physiol. Rev., 42, Suppl. 5, 282 (1962).
14. O. H. Gauer, J. P. Henry, and H. O. Sieker, Circulat. Res., 4, 79 (1956).
15. I. P. Gilmore and S. W. Hardford, J. Appl. Physiol., 8, 393 (1956).
16. J. P. Henry, O. H. Gauer, and H. O. Sieker, Circulat. Res., 4, 91 (1956).
17. H. Irisava, A. P. Greer, and R. F. Rushmer, Am. J. Physiol., 196, 741 (1959).
18. A. R. Kerr and J. W. Kirklin, Ann. Surg., 172, 278 (1970).
19. I. J. Lalich and P. H. Lorhan, J. Lab. Clin. Med., 27, 157 (1941).
20. E. M. Landis and J. C. Hortenstine, Physiol. Rev., 30, 1 (1950).
21. A. A. Lurie, Anesthesiology, 24, 368 (1963).
22. K. Pabst and O. H. Gauer, Arch. Ges. Physiol., 274, 213 (1961).
23. J. W. Pate, Am. Surg., 32, 117 (1966).
24. J. Ross, C. J. Frahm, and E. Braunwald, Circulat. Res., 9, 75 (1961).
25. M. K. Sykes, Ann. Roy. Coll. Surg. Eng., 33, 185 (1963).
26. I. V. Warren, E. S. Brannon, H. S. Weens, et al., Am. J. Med., 4, 193 (1948).