PARTIAL PRESSURE OF OXYGEN IN DIFFERENT ORGANS AND TISSUES AND SOME HEMODYNAMIC PARAMETERS IN TOURNIQUET SHOCK

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UDC 616-005.4-06:616-001.36]-092.9-07:616-008.922.1

KEY WORDS: oxygen; tourniquet shock; generalized hypoxia; muscle; skin; liver; kidney; small intestine; cerebral cortex.

Pathological states arising after restoration of the circulation in the limb after a long period of ischemia are characterized by complex disturbances in various systems of the body and by high mortality [2, 3, 6, 7, 10, 13, 15].

In the opinion of specialists in various fields one of the most important pathogenetic factors lying at the basis of irreversible disturbances of homeostasis in these states is generalized hypoxia, predominantly of circulatory origin, which develops as a rule against the background of disorders of the central and systemic hemodynamics [2, 4, 8, 9, 15].

The aim of the present investigation was to study the character of changes in the partial pressure of oxygen (pO_2) in muscles, skin, liver, kidney, small intestine, and cerebral cortex and to compare them with changes in the hemodynamics during the development of the reaction of the body to tourniquet ischemia and to removal of the tourniquet.

EXPERIMENTAL METHODS

Experiments were carried out on 160 noninbred albino rats weighing 350-400 g. Tourniquet shock was produced by applying a tourniquet consisting of eight circular turns of model airplane rubber to the upper third of both hind limbs for 6 h. At the end of this time the tourniquets were removed and the circulation in the limbs restored. To study pO_2 a platinum electrode enclosed in a hollow needle with an external diameter of 0.3 mm was used [1]. Polarograms were recorded on the LP-7e polarograph (Czechoslovakia). To determine pO_2 the electrode was inserted into the skin for 1-2 mm and into muscle for 4 mm; to investigate the liver an incision was made in the anterior abdominal wall parallel to the costal margin, 1 cm long, the peritoneal cavity was opened (under aseptic conditions), and the electrode was inserted into the right lobe to a depth of 3 mm. The electrode was inserted into the kidney to a depth of 1.5 mm (into the cortex) through a lumbar approach. To investigate the small intestine the electrode tip was inserted tangentially into the intestinal wall for 1.5 mm; to investigate the brain, a burr-hole 1.5 mm in diameter was drilled and, by means of a micrometer, the electrode was inserted into the cortex for 100-150 μ. To study pO_2 in the intact muscles and skin, the forelimbs were used. The position of the electrode in each experiment was verified by the oxygen inhalation test.

The cardiac output (CO) was determined by the thermodilution method [12]. A type MT-54 thermistor was introduced through the left carotid artery as far as the aortic orifice. Physiological saline at room temperature was injected into the left jugular vein in a volume of 0.1 ml for one determination. The thermodilution curves were recorded on a TZ-21S potentiometric automatic writer (Czechoslovakia).

The arterial pressure (BP) was measured in the left carotid artery by means of an electromanometer. In experiments in which CO was determined, BP was recorded in the caudal artery by Korotkov's bloodless method, using a special attachment (Harvard Apparatus, USA). The frequency and amplitude of the respiratory movements were recorded by an impedance method,

Department of Operative Surgery and Topographic Anatomy, Department of Surgery, and Faculty of Preventive Medicine, I. M. Sechenov First Moscow Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR V. V. Kovanov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 94, No. 7, pp. 19-22, July, 1982. Original article submitted October 18, 1981.

 0007-4888/82/9407-0872$07.50

TABLE 1. Time Course of pO_2 (in mm Hg) in Some Organs and Tissues of Rats during Tourniquet Shock (M ± m)

Time of determination of parameter	Number of determinations	Intact skin	Intact muscle	Liver	Kidney	Intestinal wall	Cerebral cortex
Normal	37	28,2±2,6 (100%)	24,3±2,5 (100%)	35,4±3,7 (100%)	30,4±2,6 (100%)	15,2±3,0 (100%)	39,6±2,4 (100%)
1 h before removal of tourniquets	98	22,3±1,5 (79,1%)	22,2±1,2 (91,3%)	25,8±1,3 (72,8%)	23,3±1,1 (76,6%)	15,1±1,3 (99,3%)	40,2±1,4 (102,8%)
15 min after removal of tourniquets	98	18,0±2,2 (63,8%)	20,3±2,4 (83,5%)	20,4±2,0 (57,6%)	18,4±1,5 (60,5%)	14,7±2,4 (96,7%)	40,5±2,3 (103,6%)
30 min after removal of tourniquets	96	19,4±1,8 (69,4%)	19,6±1,8 (80,6%)	15,4±1,5 (43,5%)	16,7±2,4 (54,9%)	12,5±2,6 (82,2%)	39,7±2,0 (101,5%)
1 h after removal of tourniquets	90	21,0±3,3 (74,4%)	20,5±2,4 (84,3%)	11,7±2,8 (33,0%)	15,7±1,2 (51,6%)	14,3±1,5 (94,1%)	41,6±2,4 (106,4%)
2 h after removal of tourniquets	86	22,4±3,0 (79,4%)	19,8±2,0 (81,4%)	11,5±1,8 (32,4%)	14,0±2,7 (46,0%)	14,8±2,4 (97,3%)	42,9±3,7 (109,7%)
4 h after removal of tourniquets	70	22,8±2,6 (80,8%)	19,3±1,7 (79,4%)	9,5±2,7 (26,8%)	14,8±1,7 (48,6%)	14,8±3,2 (97,3%)	42,4±2,6 (108,4%)
Control (after 5 h of observation)	25	24,7±1,7 (87,5%)	23,8±2,4 (97,9%)	37,2±2,9 (105,0%)	33,6±3,4 (110,5%)	14,8±1,2 (97,3%)	37,2±3,1 (95,1%)

Legend. $P < 0.05$; figures in parentheses show percent of normal.

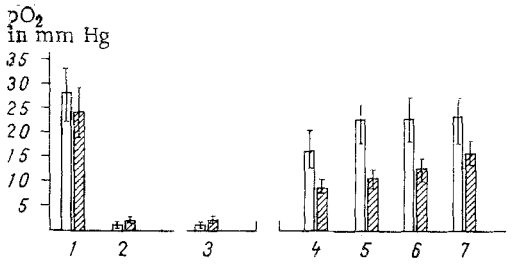

Fig. 1. pO_2 in limb tissues during ischemia and after restoration of their blood flow. Unshaded columns — skin, shaded — muscles. 1) Normal, 2) beginning of ischemia, 3) after 6 h of ischemia; 4-7) 1, 2, 3, and 4 h respectively after removal of tourniquets.

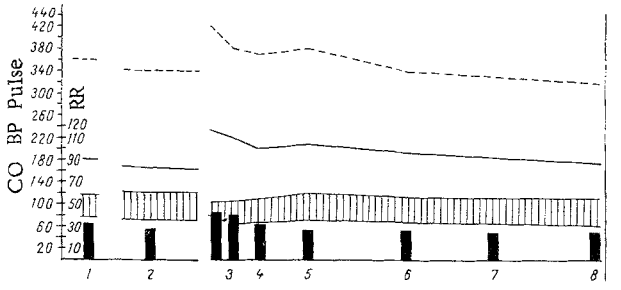

Fig. 2. Effect of tourniquet shock on some parameters of hemodynamics and respiration in principal group of animals (average data). Columns show CO (in percent of normal); shaded area denotes BP (in mm Hg); continuous line shows respiration rate (RR) (in cycles/min); broken line shows pulse rats (beats/min). 1) normal, 2) 1 h before removal of tourniquets; 3-8) 15 and 30 min, 1, 2, 3, and 4 h respectively after removal of tourniquets.

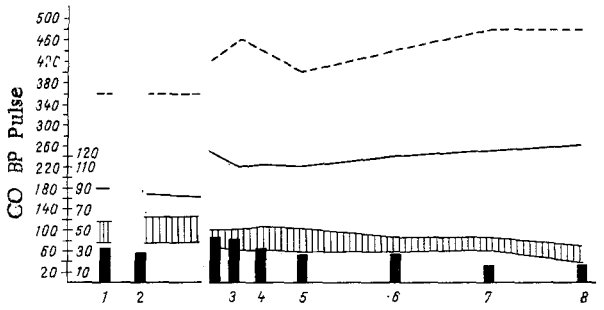

Fig. 3. Effect of tourniquet shock on some parameters of hemodynamics of respiration in group of rats dying 5-6 h after removal of tourniquets (mean data). Legend as to Fig. 2.

using needle electrodes. The ECG was recorded in standard lead II. All parameters were recorded synchronously on a polygraph (Biograph) during 1 h before removal of the tourniquets and for 4-5 h after restoration of the circulation in the limbs. The animals were anesthetized with urethane (600 mg/kg, intraperitoneally). Intact rats, in which similar tests were carried out during a period of 6 h, under the same type of anesthesia, served as the control. Normal values of CO_2 in the corresponding tissues and organs of noninbred albino rats were determined in a separate series of experiments (Table 1).

EXPERIMENTAL RESULTS

Time Course of pO_2 in Ischemized Limbs. After application of the tourniquet pO_2 in muscles and skin fell in the course of 2-5 min to 1-2 mm Hg and remained at that level throughout the period of tourniquet ischemia. After resumption of the circulation in the limb pO_2 began to rise. As Fig. 1 shows, pO_2 rose faster in the skin, and after 2 h it had already reached 80% of normal. In muscle tissue pO_2 rose slowly, and after 4 h it was still 32-40% below normal.

Time Course of pO_2 in Nonischemized Tissues and Organs. A fall in pO_2 was observed in the skin, liver, and kidney during the period of 1 h before removal of the tourniquet, but in intact muscle, small intestine, and the cerebral cortex pO_2 was almost unchanged. After removal of the tourniquets pO_2 continued to fall steadily for 4 h in the liver and kidney to 26.8 and 48.6% of normal respectively. During the first 60 min after removal of the tourniquets pO_2 in intact skin fell from 79.1 to 74.4%, but after 1.5-2 h it rose again to 80.8%, but did not reach the normal level. During the first 60 min after removal of the tourniquets pO_2 in the small intestine and intact muscles also feel to 94.1% and 83.4% respectively, but later it remained at the same level. In the cerebral cortex pO_2 as a rule remained high at all times of observation.

No significant changes were found in BP and at no time of observation did the blood pressure fall below 100 mm Hg (Fig. 2). The heart rate increased at the time of resumption of the blood flow in the limbs, but was restored in the course of the next 15-25 min.

CO fell during the hour before removal of the tourniquets, but rose again during the first 15-25 min after removal. During the next 4 h of observation this parameter fell steadily to 70.6% of normal (Fig. 2).

After removal of the tourniquets dyspnea developed with an increase in amptlitude of the respiratory movements, which continued for 1.5 h (Fig. 2). Next followed a gradual slowing of the respiratory movements and their amplitude returned closer to normal.

In the course of the investigation one group of animals (about 30%) was discovered in which development of the pathological process during the first few hours of observation was characterized by rapid decompensation, leading to death 5-6 h after removal of the tourniquests (Fig. 3). In these animals pO_2 fell progressively in the tissues and organs studied, especially in the liver and kidney. The only exception was pO_2 in the cerebral cortex, which fell only in the period of agony, i.e., when the hemodynamics was severely disturbed. In the animals which died quickly CO was reduced, the tachycardia was increased, the systolic pressure fell in the course of 2-3 h to 60 mm Hg or lower, and dyspnea increased. Characteristically the rats always died after respiratory arrest, which was followed by cessation of cardiac activity.

The results of these experiments showed that in tourniquet shock a significant fall of pO_2 in the liver and kidneys is observed even before the tourniquets are removed and the circulation restored to the ischemized limbs. The basic feature of this phenomenon is that the fall of pO_2 in these organs takes place before development of negative shifts in the central and systemic hemodynamics and external respiration. The mechanism of this phenomenon is not specially examined in this communication, but it can be tentatively suggested that after application of tourniquets disturbances of the microcirculation take place in certain organs and tissues, due to nociceptive stimulation and humoral factors. Other workers also have reported similar disturbances [4, 5], for example, in the acute period of the long-term crush syndrome, and in conjunction with the results of the present experiments this may perhaps be evidence that the pO_2 changes discovered are specific for pathological states that are based on prolonged acute ischemia of the limbs.

We know from the literature that in the post-tourniquet period a reaction develops to the inclusion of the ischemized limbs in the circulation, when various products of abnormal metabolism and tissue destruction, biologically active substances, increased numbers of certain ions, etc., pass from the limb into the rest of the body [3, 6, 7, 9, 10]. Changes also develop in the blood clotting system, together with acidosis and a deficiency of the circulating plasma volume on account of edema of the ischemized limbs [3, 6, 8, 10, 11, 14]. These factors lead to pathological disturbances of the central and systemic hemodynamics, and ultimately to generalized hypoxia.

However, the present experiments showed that in the postischemic period of tourniquet shock disturbances of the oxygen balance in certain organs and tissues (with the exception of the cerebral cortex) are observed sooner than disturbances of the central and systemic hemodynamics.

Bearing in mind the probable hypoxic changes in the liver and kidneys, which evidently develop before removal of the tourniquets and, consequently, before the disturbances of the central and systemic hemodynamics, it can be tentatively concluded that the disturbance of the detoxicating function of these organs facilitates the development of tourniquet shock. The mechanism of this phenomenon requires further study.

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