

# THE SIGNIFICANCE OF SHOCK IN THE PATHOGENESIS OF EXPERIMENTAL SEPSIS

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G. M. Gurevich [1] showed that complications of anaerobic infection and sepsis are observed in the majority of wounded patients who suffered third-degree shock.

It is known that during an operation of the Connors type, carried out under local anesthesia, in a number of cases when the surgeon carries out various manipulations in the region of the pleura and, especially, in the region of the hilus of the lung, symptoms of suboperative shock arise. G. M. Gurevich observed that the postoperative period of these patients has a difficult course on account of exacerbation of the infectious process. By changing the method of anesthetization and changing to hexenal anesthesia, the author eliminated the possibility of having symptoms of suboperative shock develop, and the postoperative course of these patients improved.

I. E. Minkevich talks of the presence of a condition favorable to the development of gas gangrene in wounded persons after shock or large blood loss.

A. Z. Chernov [2] observed that in individual cases, the state of shock is a predisposing factor to the development of sepsis.

In order to confirm the indicated data experimentally, we set up experiments on rabbits.

First series of experiments. In this series, a dose of streptococcus culture which did not lead to the development of sepsis was administered into the peripheral vein of the ear of 8 animals. During the period of observation (from 3 to 6 weeks) no changes whatsoever were evident in the behavior of the animals, their temperature and weight.

Second series of experiments. As a control, a one-day-old culture of the same strain of streptococcus on 0.25% agar was injected into 7 rabbits by two methods: intravenously in 5 animals, subcutaneously in 2. Five rabbits died in the course of the first 24 hours, including the 2 rabbits which received streptococcus subcutaneously, and 2 rabbits were sluggish, refused food, lost weight, and were feverish during the first days after the injection. Later these rabbits recovered and no pathological symptoms were evident in them after prolonged observation.

On autopsy of the 5 dead rabbits, petechial hemorrhages in the subcutaneous cellular tissue, a large quantity of serosanguinous exudate in the thoracic cavity, a marked enlargement of the heart, and a considerable enlargement of the liver, which was congested with blood and flabby, were found. Blood removed from the heart by sterile means during the autopsy and seeded in serum broth grew the initial strain of streptococcus.

Third series of experiments. In this series of experiments, a one-day-old streptococcus culture of the same dosage as in the first series of experiments was administered twice to 9 rabbits at an interval of 10 days. As a result of these experiments, 2 rabbits died on the third and eighth day after the second administration of the culture. The remaining animals remained healthy during prolonged observation. Even fracture of a hip without

anesthesia after intravenous infection did not cause the development of sepsis, a matter which can be explained, we believe, by the high resistance of rabbits to trauma (the difficulty of producing shock experimentally in rabbits is well-known).

Thus, in our preliminary experiments, development of sepsis was not observed in rabbits infected with streptococcus.

Fourth series of experiments. We did not succeed in producing constant traumatic shock in rabbits by crushing an extremity, by traumatizing the mesentery, by prolonged stimulation of the sciatic nerve. So we kept the method of producing shock by means of insulin.

A dose (proportionate to their weight) on one-day-old streptococcus culture which had not caused the development of sepsis in control experiments, was injected in the peripheral vein of the rabbits. Following this, 2 units of insulin per 1 kg of the animal's weight were injected subcutaneously. In the first minutes following this, a growing sluggishness, shallow, rapid respiration, and tachycardia could be observed. Usually the rabbits kept the same pose and, when their quiet was disturbed, they moved with difficulty and again took the same pose. In this period it was possible to observe various signs of restlessness in the animals: quick, uncoordinated movements of the head or ears.

All of the above signs increased noticeably for an average of 1-1/2 hours after the administration of insulin. At the end of this period, part of the rabbits suddenly suffered intense spasms of a clonic type; the others, weaker, younger, animals weighing up to 1500 g, developed spasms of a tonic nature at the moment of greatest weakness and apathy: the rabbit lay on its belly, stretched out full length, with the paws extended, the back bent, and head down. Some of the animals developed a complete absence of reaction: the rabbit fell on its side and lay motionless in this position with the head lowered and the ears down; the heartbeat was barely audible, the respiration shallow. This condition soon ended in death, if suitable measures were not taken.

As soon as the first signs of shock appeared, either as motor stimulation (spasms) or in the form of extreme sluggishness, we administered from 10 to 20 ml, depending on the weight of the animal, of 40% glucose solution intravenously and 0.5 ml of adrenalin (1:1000) subcutaneously. Such measures cured the described condition of the animals and the rabbit soon became active. When there was a delay in applying these measures, the animal died.

In all, we observed and investigated 16 rabbits which had been infected earlier and which had suffered pronounced signs of insulin shock for a short time.

Three rabbits died 2 days after infection. These animals were in a serious condition which grew progressively worse from the moment of infection until their death; they did not accept food, lost weight sharply and were feverish. At autopsy, serosanguinous exudate in the thoracic and pericardial cavities and a marked enlargement of the heart were found; the lungs were collapsed, often with pneumonic areas; the liver was enlarged, flabby; the spleen was of ordinary size, dark in color, cloudy in section, easily scraped. In the abdominal cavity, the exudate was of a serous or serosanguinous nature. Blood from the heart and tissue from internal organs, taken under sterile conditions and seeded in serum and sugar broth, grew the initial strain of streptococcus.

The course of the process was more prolonged in the remaining 13 rabbits after they underwent shock and infection. Adynamia and apathy, marked loss of weight (450-850 g) and a high temperature (39.1-41°) were observed in them.

This group of animals was killed from 10 to 15 days after infection.

On autopsy, an exudate of a serosanguinous nature was usually observed in the pericardial cavity of the animals, marked enlargement of the heart, especially of the right side; the lungs were collapsed, pale pink in color with a small area of paler color; the liver greatly enlarged, congested, with a pale, often quite white, edge; the spleen was large in size, dark in color, cloudy on section, easily scraped. In individual cases, firm whitish-colored nodes could be observed on the heart valves. Heart blood and lung, liver, and spleen tissue removed under sterile conditions and seeded in serum and sugar broth grew the initial strain of streptococcus.

Investigation of the blood of the rabbits during the period of their illness showed a regular increase in the number of leucocytes (10,000-15,500). An increased number of pseudoeosinophils and a decrease in

lymphocytes was observed. The more leucocytes there were, the more marked the discrepancy between them. No significant changes at all were observed in the number of erythrocytes.

Thus, our experiments showed the pathogenetic relationship between shock and sepsis. This relationship is the result, apparently, of changes in the reactivity of the organism following shock suffered by the animal.

Observations showed that the more severe the signs of shock were in the animal, the more serious was the septic process which developed and the more difficult its clinical course.

The facts which are presented permit consideration of the fact that even after patients are brought out of shock and its clinical signs have been liquidated, it is necessary to carry out rational prophylactic therapy in order to avoid subsequent reactions, raising the reactivity of the organism and, consequently, improving the course of the basic process and the prophylaxis of complications.

#### LITERATURE CITED

[1] Gurevich, G. M. in the book: Materials of the 1st Inter-Republic Conference of the Learned Medical Councils of the People's Commissars for Health of the Baltic Soviet Socialist Republics, Riga, 1946.

[2] Chernov, A. Z. Wound Sepsis, in the book: Experience of Soviet Medicine in World War II, v. XXIX, p. 192 (1951).