Phagocytosis and Bactericidal Activity of Leukocytes in Hemorrhagic Shock

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Polymorphonuclear leukocytes from dogs in hemorrhagic shock did not demonstrate impairment in phagocytosis or killing of Staphylococcus aureus or Escherichia coli in vitro.

Bacteria and endotoxin are found consistently in the blood of animals in shock (2). Moreover, an impairment in reticuloendothelial clearance (1, 2), a decrease in the adherence of leukocytes to the endothelial lining of capillaries, and diminished emigration of leukocytes (3) occur in shock. To determine whether impairment of polymorphonuclear neutrophil (PMN) function contributed to the bacteremia seen in shock, we investigated the bactericidal and phagocytic properties of PMN from the blood of dogs in hemorrhagic shock.

Hemorrhagic shock was induced in five female dogs (mean weight of 9.6 kg) by the method of Lefer and Martin (4). Irreversible hemorrhagic shock was produced in all animals as evidenced by a mean bleedout volume of 53.2 ± 5.6 (standard error of mean) ml/kg, a mean duration of oligemia of 198 ± 13 min, and a mean survival time of 93 ± 6 min. Myocardial depressant factor (MDF) concentration assayed from plasma obtained shortly before death exhibited a mean MDF concentration of 53.9 ± 2.0 MDF units, a value typical for animals in lethal shock (4). These animals died too rapidly to manifest overt signs of sepsis.

Before and after (within 30 min of death) the induction of shock, 20 ml of blood was drawn from each animal, heparinized (10 units/ml), and sedimented for 60 min in 3% dextran. The supernatant fluid, containing plasma, leukocytes, and platelets, was divided into two samples and centrifuged at 200 × g for 12 min. The white blood cell buttons were then resuspended in 3.4 ml of Hanks balanced salt solution without bicarbonate and 0.4 ml of heterologous plasma from a shocked or control dog. To this was added washed Escherichia coli (0111 B4) or coagulase-positive virulent Staphylococcus aureus to give a bacteria: white blood cell (WBC) ratio of 1:1. The suspensions were mixed, samples were drawn, and the rest of the mixture was tumbled at 20 rev/min at 37 C. Samples were later drawn at 15, 30, 60, and 120 min. The total number of bacteria remaining and the fate of the cell-associated and cell-free bacteria

![FIG. 1. Representative total, sediment (PMN-associated), and supernatant (PMN-free) bacterial counts in systems with control and postshock blood. E. coli proved equally susceptible to phagocytosis (supernatant curve) and killing (total curve) in the presence of control (A) and shock (B) leukocytes and serum. Virulent S. aureus was phagocytized but resisted killing equally in control (C) and shock (D) leukocytes and serum. Crossover tests, using control leukocytes with shock serum and shock leukocytes with control serum, also demonstrated normal PMN function.](http://iai.asm.org/)
NOTES were determined at each interval by differential centrifugation and subsequent dilution and plating of appropriate fractions by previously described procedures (5). WBC and differential counts obtained from shocked dogs were not significantly different from counts determined before the induction of shock.

PMN function was tested in the following systems: control leukocytes with control plasma; control leukocytes with shock plasma; shock leukocytes with control plasma; shock leukocytes with shock plasma (all tests were duplicated in different animals).

Both the phagocytic and bactericidal ability of PMN were essentially unaffected by the induction of shock in these animals (Fig. 1). Thus, the susceptibility to infection and the decreased clearance of bacteria seen in hemorrhagic shock are not the result of changes in the function of polymorphonuclear neutrophils.

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LITERATURE CITED