Reports of Scientific Meetings

International Symposium on Metabolic Changes in Shock

An International Symposium on Metabolic Changes in Shock was held in Freiberg, West Germany, October 30–November 1, 1969. The meeting, not limited to metabolic changes in shock, covered many aspects of this complex condition, and one of the basic concepts discussed before the large American and German audience was the unitarian concept of shock. Presented by J. Fine, this theory suggests that the basic lesion in "refractory shock" is alteration of the reticuloendothelial system in the spleen and liver. However, most of the presentations indicated that whatever the terminal lesion of irreversible shock might be, many factors contribute to its development, including organ failure, and, furthermore, the evolution of shock depends upon whether the initial lesion is in heart, lung, liver or kidney, as well as upon the functional reserve present in these organs.

Shoemaker and Ulmer described increased pulmonary arterial pressure and pulmonary vascular resistance in patients with hemorrhagic and traumatic shock. They emphasized that these changes are enhanced during transfusion and that because the right side of the heart is normally not well adapted to increased pressure loads transfusion should be performed cautiously to avoid overloading the right ventricle and precipitating pulmonary edema. This would be especially true when large volumes are infused rapidly, as, for example, use of large volumes of colloid-free balanced salt solutions advocated by some in the management of shock.

Alteration of pulmonary function in shock was described by Kinney, who emphasized the increase in deadspace and alveolar ventilation, the decrease in pulmonary compliance with increased work of breathing, and the increase in alveolar–arterial O_2 gradient so frequently observed. Atelectasis, alteration of the ventilation:perfusion ratio, platelet aggregation, fat embolism, and thromboembolism may all contribute to these alterations in pulmonary function in shock. Kinney also described the increase in O_2 requirement present in such conditions and emphasized that it occurred at a time when O_2 transport capacity is impaired.

Alteration in hepatic function in shock was described by several authors. Kessler reported a decrease in hepatic perfusion during hemorrhagic shock, resulting in a decreased O_2 supply and severe tissue hypoxia even in the early stages. Eisele reported that in man septic shock is associated with a decrease in pressure gradient between portal vein and central vein, low portal vein P_o_2, and portal vein lactate concentrations higher than those in arterial blood. Other signs of alteration in hepatic function are the decrease in hepatic thermogenesis reported by Stoner and the decrease in reticuloendothelial system (R.E.S.) activity reported by Fine. The lowering of hepatic activity in shock is, however, accompanied by an increase in fibrinogen synthesis.

Changes in the microcirculation of the skin of human volunteers were illustrated by a strikingly descriptive motion picture shown by Brannemark. Low-flow states were accompanied by granulocytic rigidity blocking the entrance to precapillary sphincters, as well as by erythrocyte diapedesis, plasma leakage, and formation of periendothelial microthrombi which are released into the circulation following restoration of flow. Lewis indicated that these alterations in the microcirculation are accompanied by a significant defect in capillary transport which can be defined as the product (P X S) of permeability and available capillary surface area. The defect in capillary transport which occurs during shock is out of proportion to the changes in blood flow.

Kidney function is also altered in shock, and the changes emphasized during the symposium included a 50 per cent decrease in blood flow (mostly to the cortex), lower O_2 utilization, and Na reabsorption. The severe limitation of
diuresis which occurs during shock may persist for two or three weeks afterwards and is often followed by excessive urinary output.

Of the many endocrine disturbances present in shock, the increase in aldosterone activity was the only one mentioned: there is an increase in secretion and a decrease in metabolic inactivation of this corticoid.

All these alterations of organ function contribute to the development of the metabolic changes present in shock. Of these, increased blood lactate levels are most pronounced. Hyperlactatemia and its prognostic value were discussed by Weil, who reported that in 42 patients with circulatory shock the best prognosis was provided by measurements of lactate levels, not by lactate-pyruvate ratios or levels of either pyruvate or excess lactate. As lactate increased from 2.1 to 8.0 mM/l, the estimated probability of survival decreased from 90 to 10 per cent. Other changes in carbohydrate metabolism present in shock received little mention, including the initial hyperglycemia followed by hypoglycemia and other alterations in metabolic pathways.

Alterations in coagulation in shock were discussed by Hardaway and Bergentz. They described the appearance during refractory shock of a consumption coagulopathy characterized by a progressive reduction in coagulation factors sensitive to the action of thrombin, such as fibrinogen, platelets, Factors V, VIII and XIII, and by an increased synthesis and turnover of fibrinogen by the liver and an increased consumption of platelets. Initially, hypercoagulability of the blood is present, but if shock persists a state of hypoagulability develops. The disseminated intravascular clotting so frequently observed is often associated with other contributory factors such as: appearance of microthrombi and erythrocyte aggregation; the presence of thromboplastic material such as fat emboli, intravascular catheters; factors inhibiting fibrinolysis (state of hypercoagulability is usually balanced by an increased fibrinolytic activity); blockage of the reticuloendothelial system. It is now widely recognized that blockage of the R.E.S. plays a significant role in the course of experimental shock, but the significance of this dysfunction in man remains difficult to assess.

A large number of papers were devoted to septic and endotoxin shock, in both of which the primary lesion appears to be metabolic. MacLean described a type of bacteremic shock characterized by normal or increased flow rates and cardiac output, hypotension, decreased oxygen consumption and arteriovenous oxygen difference, and elevated blood lactate, which occurs in man. Dibman emphasized the discrepancy between the observed large increase in cardiac output and relatively small increment in \( O_2 \) consumption. Shoemaker reported similar observations.

Vasoactive intermediates responsible for circulatory changes in septic shock were discussed by Jacobson, who eliminated the possible roles of histamine, catecholamine and 5-hydroxytryptamine. Habermann similarly doubted that the kinins had a role in the pathogenesis of septic shock.

In view of the complex picture of shock, it was not surprising that no single method of antishock therapy could be defined. The routine use of large doses of corticosteroids in endotoxin shock was not recommended until "an objective study confirmed their clinical effectiveness." The use of beta- and alpha-blocking agents was not strongly or systematically advocated, no more than that of proteinase inhibitors. The value of symptomatic therapy aimed at restoring homeostasis appeared clearly: volume replacement controlled by central venous pressure measurement, inhalation of \( O_2 \)-enriched gas mixtures, mechanical ventilation, and control of acid-base factors and of \( P_{O_2} \) in arterial blood.

The last session devoted to use of plasma substitutes (gelatin, dextran, hydroxyethyl starch) made it evident that no ideal substitute for whole blood is as yet available.

Gabriel G. Nahas, M.D.
Professor of Anesthesiology
Columbia College of Physicians and Surgeons
New York, New York