TREATMENT OF SHOCK

Principles and Practice

JOHN BARRETT • LLOYD M. NYHUS

Second Edition

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Preface

In the 12 years that have elapsed since the first edition of this book was published, we have seen enormous strides in our understanding of the pathophysiology of the shock state. This new edition has been extensively rewritten to reflect this new understanding of shock and the methods necessary for its treatment. Part I of the book consists of an update of the effects of shock on the microcirculation and reflects our current understanding of the pathophysiology and treatment of these lesions. Part II is a much expanded view of the effects of shock on the vital organs and includes completely new sections on the brain and the liver as well as expanded concepts of the effects of shock on the lungs, heart, and kidney. The treatment of the underlying cause of shock is found in Part III, which includes a new section on the initial management of the patient in traumatic shock as well as new sections on the pharmacologic treatment of shock and the management of shock in infants and children.

Throughout the work, we have attempted to maintain a practical orientation for the physician who is called on to manage patients in shock. We have attempted to provide an understanding of the pathophysiology of shock as a background for the modern concepts of therapy.

With the increasing sophistication of modern medicine, and the rapid and rational delivery of patients in shock to medical facilities, the shock patient is now a more common sight in the emergency department, operating room, and intensive care sections of our hospitals. Many patients who formerly would have died are now surviving to present the clinician with the challenge of management of the shock state.

In 1872, Gross defined the shock state as "the rude unhinging of the machinery of life." Although modern understanding of shock now permits a more precise definition, shock continues to exert profound effects on the entire organism and on all organ systems. It is only by constantly updating our understanding of what shock is and what it does that we can plan a rational treatment approach. We have provided in this volume a current view of the state of the art of shock management by renowned national and international experts. We wish to thank each of them individually for their contribution to this work, as well as Ms. Catherine Judge in the publications office of the Department of Surgery at the University of Illinois, and the many people at Lea & Febiger, especially Raymond Kersey, without whose help this work would not have been possible.

Chicago

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Contents

Part I
TREATING THE MICROCIRCULATORY LESIONS

Chapter 1
MICROCIRCULATION IN SHOCK

Allan M. Lefer • Stuart K. Williams

Shock is a multifaceted disorder that has a number of causes and a variety of pathophysiologic mechanisms. Our discussion is concerned with "circulatory shock" to differentiate it from other types of shock including anaphylactic shock, neurogenic shock, and electric shock, all of which are quite different from circulatory shock and involve primary disturbances in organ systems other than the circulatory system, e.g., respiratory, nervous, etc.

Circulatory shock can be defined as a sustained reduction in blood flow perfusing the vital tissues and cells of the body to an extent that results in significant tissue damage, and if uncorrected, can lead to death. There are a variety of types of circulatory shock including hemorrhagic (oligemic, hypovolemic), septic (endotoxic, bacteremic), cardiogenic (cardiac), traumatic, intestinal ischemic (splanchnic ischemic), acute pancreatitis (pancreatic), and burn (thermal) shock. Although these types of shock all have characteristic hemodynamic features and somewhat different time courses,[1] all have several common features. Nevertheless, all types of circulatory shock result in a severe hypotensive state at some stage in their development, and virtually all involve an early splanchnic vascular hypoperfusion and a later impairment of cardiac function. One important and consistent feature of circulatory shock is a fundamental insufficiency of microcirculatory flow leading to inadequate perfusion of the somatic cells of many of the important organs of the body. These alterations in the microcirculation, the effects of the resultant ischemia of the cells, and the modification of the components of the microcirculation are the major themes of this chapter.

FLUID AND ELECTROLYTE SHIFTS IN SHOCK

The sequelae of microvascular complications can be described simply as an insufficiency of blood perfusion to peripheral tissues resulting in cellular hypoxia and death. Concurrent with this insufficiency of flow is microcirculatory failure resulting from a chain of metabolic events. In the past, investigations into the effects of shock have focused on the macrocirculation since it was assumed that the microvascular bed suffers little damage until the shock process has become irreversible. However, a new understanding of the importance of the microcirculation in disease states has led to the belief that early in the shock state, damage occurs in the intrinsic regulatory processes that influence microvascular blood flow and exchange.

After a fall in mean arterial blood pressure, at least four major changes occur in the microcirculation. These include 1. disturbed diffusional transport, 2. loss of

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myogenic adjustments, 3. nutritional shunting, and 4. rheological changes. All of these conditions can be summarized as the inability of the terminal vascular bed to regulate the orderly distribution of blood flow.

Severe circulatory shock can be considered the net result of several contributing factors. All of these factors can be linked directly to the deterioration of microcirculatory function and can be considered as altered performance of either blood flow, vascular exchange, or both. Alterations in blood flow are thus related to the suppression of autoregulation,[2] the occurrence of spontaneous vasomotion,[2] and diminished smooth muscle cell reactivity.[3] Changes in microcirculatory exchange function are related to an imbalance of hydraulic pressure and osmotic forces, a change from net fluid filtration to absorption resulting in hemodilution followed secondarily by the loss of fluid, the occurrence of plasma protein leakage, and later to impaired lymphatic drainage.

Elements of the Microcirculation

The major components of the microcirculation are arterioles, capillaries, and venules. The afferent vessels, the arterioles, as they approach the capillaries, are a single endothelial cell lining surrounded by layers of circular smooth muscle cells.[4] The smooth muscle layer becomes a single discontinuous layer at the level of the arteriolar-capillary junction. The arterioles are innervated primarily with adrenergic and a limited number of cholinergic fibers.[5] Capillaries consist of single endothelial cell tubes with no smooth muscle cell investment. The morphology of capillaries differs between tissues and generally relates to the permeability function in each tissue.[6] In the liver, spleen, and bone marrow, the capillaries are described as discontinuous, exhibiting gaps at interendothelial junctions.[7] Capillaries in the brain, heart, lungs, and muscle are characterized as continuous since the interendothelial junctions exhibit tight associations.[8] Venules are characterized by increasing diameter and increasing investment of smooth muscle cells.[6] There is sparse innervation of the small venules.

Changes in Microvascular Flow

During the initial stages of shock, as a direct result of lowered mean arterial blood pressure, the larger arterioles of the microvascular tree constrict.[9] This initial constriction can be terminated by treatment with sympatholytic drugs or α-adrenergic blockers indicating that this constriction is a result of compensatory neurogenic feedback.[10] The constriction of precapillary sphincters will subsequently lead to ischemia and a progression toward irreversibility. Further responses are the suppression of vasomotion,[9] a blunted response to constrictor stimuli,[3] and the inability to maintain intracapillary pressure.[11] In addition, because of the sustained arteriolar vasoconstriction, changes in precapillary sphincter activity have little effect on capillary blood flow. This results in the buildup of metabolic byproducts, and after several hours of oligemia, the terminal vascular bed becomes a passive structure incapable of compensatory adjustment.[10]

Microvascular Exchange in Shock

The maintenance of microcirculatory homeostasis exists as a balance between flow and capillary surface area, against hydraulic and osmotic pressure. The major determinant of fluid flux is capillary pressure (Pc) which is determined by 1. vol-

ume flow, 2. fluid exchange, and 3. autoregulation. The Starling equation permits the quantitative interpretation of the relationship between the major factors that influence fluid exchange in the microcirculation:

$$Q = k(Pc + \Pi i) - (Pi + \Pi p)$$

In this equation, Q represents net fluid flux (ml/min/100 g tissue), Pc is net capillary hydraulic pressure (mm Hg), Πi is interstitial or tissue pressure (mm Hg), Pi is interstitial hydraulic pressure (mm Hg), Πp is the capillary osmotic pressure (mm Hg), and k is the filtration constant of the endothelial cell membrane. Shock has an effect on all of these hemodynamic forces. The net effect of these forces on fluid flux is shown in Figure 1–1A. The major force of filtration is capillary hydraulic pressure (Pc) and the major force of absorption is the plasma oncotic pressure (Πp) whose major determinant is the concentration of plasma proteins.

Capillary pressure, Pc, is assumed to fall dramatically during initial shock, although this fall is somewhat offset by local regulation.[12] After prolonged hypotension and the onset of erratic microvascular flow, Pc drops precipitously and becomes variable throughout the microcirculation. Finally, with the drop of systemic pressure below 40 mm Hg, the observed Pc may be close to venous levels. This change is shown in Figure 1–1B. The final low Pc is mostly a result of the complete closure of terminal arterioles.[10] The net fluid movement during these stages can be seen as initial fluid absorption resulting in hemodilution, followed by a gradual changeover to a final small but persistent net filtration.[10]

With the fall in capillary pressure and subsequent hemodilution, the plasma colloid osmotic pressure (Πp) falls steadily from a level of approximately 25 mm Hg to values as low as 12 mm Hg. During the initial stages of shock, Πp remains low followed by a slow gradual increase. This reversal is most probably related to a rise in hematocrit.[10] Finally, the terminal phase of shock is characterized by an increase in hematocrit with concomitantly decreased Πp. The latter effect is exacerbated by the increased loss of plasma proteins during this stage because of increased permeability of the capillary endothelium.

To summarize, the shock state results in dramatic changes in both capillary pressure and plasma oncotic pressure. These changes result in marked shifts in the forces regulating fluid flux and in increased fluid levels in the extravascular space. Since many organs are relatively unaffected by changes in fluid permeability, it is unusual for peripheral edema to affect tissue survival. The lung, however, is extremely sensitive to increased extravasation of fluid resulting in the observed deleterious effects of pulmonary edema and other complications, i.e., adult respiratory distress syndrome (ARDS).

Blood Rheology and Shock

One problem often overlooked in discussions of hemodynamic changes observed during cardiovascular disease is the commonly observed changes in the rheologic properties of blood. Factors that govern rheologic properties are the concentration, shape, size, and deformability of suspended particles, the extent of cellular adhesion and aggregation, and finally the composition of the plasma.[13] With the occurrence of shock and associated changes in the plasma and blood components, we must pay closer attention to subsequent abnormalities in blood rheology. New interest has focused toward optimization of blood viscosity