

HOSPITAL PHYSICIAN®

EMERGENCY MEDICINE BOARD REVIEW MANUAL

PUBLISHING STAFF

PRESIDENT, GROUP PUBLISHER

Bruce M. White

EDITORIAL DIRECTOR

Debra Dreger

SENIOR EDITOR

Bobbie Lewis

EDITOR

Robert Litchkofski

ASSISTANT EDITOR

Rita E. Gould

EDITORIAL ASSISTANT

Karen Meadows

EXECUTIVE VICE PRESIDENT

Barbara T. White, MBA

PRODUCTION DIRECTOR

Suzanne S. Banish

PRODUCTION ASSOCIATES

Tish Berchtold Klus

Mary Beth Cunney

PRODUCTION ASSISTANT

Stacey Caiazzo

ADVERTISING/PROJECT MANAGER

Patricia Payne Castle

NOTE FROM THE PUBLISHER:

This publication has been developed without involvement of or review by the American Board of Emergency Medicine.



The Association for Hospital Medical Education endorses HOSPITAL PHYSICIAN for the purpose of presenting the latest developments in medical education as they affect residency programs and clinical hospital practice.

Shock

Series Editor: Susan B. Promes, MD, FACEP

Residency Program Director, Division of Emergency Medicine, Associate Clinical Professor of Surgery, Duke University Medical Center, Durham, NC

Contributing Authors:

John Stein, MD

Attending Physician, Division of Emergency Medicine, Clinical Instructor of Medicine, University of California–San Francisco, San Francisco, CA

Eric Snoey, MD

Residency Program Director, Department of Emergency Medicine, Alameda County Medical Center, Highland General Hospital, Oakland, CA, Associate Clinical Professor of Medicine, University of California–San Francisco, San Francisco, CA

Table of Contents

Introduction	2
Definition	2
Etiology	2
Pathophysiology	2
Clinical Approach	4
Specific Causes of Shock	6
Summary	10
References	11

Cover Illustration by Christie Grams

Copyright 2001, Turner White Communications, Inc., 125 Stafford Avenue, Suite 220, Wayne, PA 19087-3391, www.turner-white.com. All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, mechanical, electronic, photocopying, recording, or otherwise, without the prior written permission of Turner White Communications, Inc. The editors are solely responsible for selecting content. Although the editors take great care to ensure accuracy, Turner White Communications, Inc., will not be liable for any errors of omission or inaccuracies in this publication. Opinions expressed are those of the authors and do not necessarily reflect those of Turner White Communications, Inc.

Shock

INTRODUCTION

Shock is the final common pathway for a number of illnesses frequently encountered in clinical practice. Unless promptly corrected, shock rapidly becomes irreversible. Although our understanding of shock pathophysiology has greatly increased over time, this condition continues to cause high morbidity and mortality. Because of the frequency and lethality of shock, clinicians must be able to assess and treat patients with this condition in a timely manner in order to correct the underlying disease process and abort the often terminal spiral toward death.

DEFINITION

A simple definition of shock is a state of poor tissue perfusion that results in inadequate delivery of essential substrates such as oxygen and glucose. In general, this condition may arise from either a quantitative deficiency of substrate delivery (as occurs during a state of low cardiac output) or a qualitative deficiency (as occurs in sepsis, where there is interference with substrate uptake). If the lack of these vital elements persists, normal metabolic processes invariably will cease, leading to cellular dysfunction and eventual organ failure.

ETIOLOGY

Four general categories of shock are recognized today: hypovolemic, cardiogenic, obstructive, and distributive (**Table 1**). Hypovolemic shock occurs in the setting of blood or fluid loss and is related to a decreased circulating blood volume. The decreased preload, ventricular filling pressures, and cardiac output lead to the final common pathway of decreased mean arterial pressure, poor tissue perfusion, and multiple organ dysfunction.

Cardiogenic shock is most commonly observed after a large myocardial infarction, but it can result from any direct injury to the myocardium and its structures or from pathologic bradycardias and tachycardias. Cardiac dysfunction in turn leads to decreased output and diminished tissue perfusion.

Obstructive shock occurs when the cardiovascular circuit is blocked either intravascularly (eg, large pulmo-

nary embolism, aortic dissection) or extravascularly (eg, tumors, tamponade, tension pneumothorax). There is a corresponding decrease in ventricular filling or an increase in afterload, which subsequently results in diminished cardiac output and shock.

Distributive shock, seen in sepsis, anaphylaxis, and spinal cord injuries, is unique in that diminished cardiac output is not usually the primary cause. One sees a profoundly abnormal dilation of vessels that simultaneously decreases preload and systemic vascular resistance. Left-ventricular end-diastolic filling may be reduced, resulting in decreased cardiac output despite an often hyperdynamic heart. The decreased systemic vascular resistance and increased vascular permeability result in a further lowering of mean arterial pressure and aggravation of shock.

PATHOPHYSIOLOGY

SYSTEMIC RESPONSE

Shock places significant stress on the body, triggering a response from multiple organ systems (**Figure 1**). These responses are aimed at maintaining homeostasis for the system in the early stages of the low perfusion state. However, if the underlying cause is not corrected, these systemic responses will eventually fail and irreversible deterioration will ensue.

The metabolic and circulatory derangements of shock trigger a complex array of homeostatic receptors in the body. Baroreceptors located throughout the cardiovascular system identify hypotension and respond by activating the sympathetic nervous system, with subsequent release of glucocorticoids and catecholamines from the adrenal medulla. At the same time, the juxtaglomerular apparatus in the kidney senses the decrease in perfusion and activates the renin-angiotensin-aldosterone system. These chemical changes result in the creation of a catabolic state. Hepatic glucose production and lipolysis of adipose tissue are activated, and the brain and heart increase their utilization of glucose. These homeostatic mechanisms initially aid in delaying the complications of shock.

Organ Injury

Central nervous system (CNS) injury is seen clinically as an alteration in mental status. Mental status can vary from normal in the early stages of shock to frank coma. Due to the autoregulation of regional blood flow, the