HOSPITAL PHYSICIAN®

CRITICAL CARE MEDICINE BOARD REVIEW MANUAL

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The Hospital Physician Critical Care Medicine Board Review Manual is a study guide for fellows and practicing physicians preparing for board examinations in critical care medicine. Each quarterly manual reviews a topic essential to the current practice of critical care medicine.

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Hypertensive Crises

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Hypertensive Crises

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INTRODUCTION

Hypertensive emergencies and hypertensive urgencies are commonly encountered in many clinical settings, including the intensive care unit (ICU). Prompt pharmacotherapy tailored to the patient's presentation is the most important intervention for limiting morbidity and mortality in the ICU. Many physicians-in-training have the "reflex" of rapidly lowering an elevated blood pressure, an action that may be associated with significant morbidity and death. The purpose of this manual is to review common concepts, misconceptions, and pitfalls in the diagnosis and management of patients with hypertensive crises. (Pregnancy-induced hypertension, including pre-eclampsia and eclampsia, is not included in this review.)

EPIDEMIOLOGY OF HYPERTENSIVE CRISES

Hypertension affects more than 50 million Americans and approximately 1 billion individuals worldwide.[1–3] Most of these patients have essential hypertension, and approximately 30% are undiagnosed.[2] Unfortunately, a large number of Americans have hypertension that has never been diagnosed or treated. Indeed, only 24% of Americans overall with hypertension have adequate blood pressure control, and this figure drops to only 14% among Mexican Americans.[2]

It has been estimated that approximately 1% to 2% of patients with hypertension develop a hypertensive crisis at some point during their lives.[4,5] The epidemiology of a hypertensive crisis parallels that of essential hypertension; it occurs more frequently among African Americans and the elderly, and men are affected twice as frequently as women.[6,7] The true incidence of hypertensive crisis is variable and uncertain, however, and depends on a number of factors, including the prevalence of treated hypertension, socioeconomic factors, and the availability of antihypertensive medication. Most patients that present with a hypertensive crisis have previously been diagnosed as hypertensive, and many have been prescribed antihypertensive therapy but have inadequate blood pressure control.[6] Despite the development of increasingly effective antihypertensive treatments, the incidence of hypertensive crisis has increased. Hospital admissions for hypertensive emergencies more than tripled between 1983 and 1990, from 23,000 to 73,000 per year in the United States.[8] Hypertension-related deaths have increased in several patient populations in recent years.[9–11] The reported incidence of postoperative hypertensive crisis varies depending on the population examined, with most studies reporting an incidence of between 4% and 35%.[12–14]

PATHOPHYSIOLOGY OF ACUTE ELEVATIONS OF BLOOD PRESSURE

The factors leading to the severe and rapid elevation of blood pressure are poorly understood. The release of humoral vasoconstrictor substances from the stressed vessel wall is thought to be responsible for the initiation and perpetuation of the hypertensive crisis.[15,16] Increased blood pressure results in endothelial damage with local intravascular activation of the clotting cascade, fibrinoid necrosis of small blood vessels, and release of vasoconstrictor substances. This leads to a vicious cycle of further vascular injury, tissue ischemia, and release of vasoconstrictors. Activation of the renin-angiotensin system has been strongly implicated in the initiation and ongoing vascular injury of severe uncontrolled hypertension.[17–20]

In patients with hypertensive crisis there may be an alteration in the autoregulation of the renal or the cerebral vascular beds. Autoregulation implies the ability of blood vessels to dilate or constrict to maintain normal organ perfusion. Healthy arteries in normotensive individuals can maintain blood flow over a wide range of mean arterial blood pressures, usually ranging between 60 and 150 mm Hg. In patients with chronic elevation of blood pressure, however, changes in the arterial circulation occur, with a shift of autoregulation toward increased constriction. Therefore, when acute blood pressure elevations increase above the autoregulatory range, tissue damage occurs as a result of either excessive vasoconstriction and consequent ischemia or of complete loss of autoregulation and loss of vascular integrity.

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