

HOSPITAL PHYSICIAN®

CARDIOLOGY BOARD REVIEW MANUAL

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The *Hospital Physician Cardiology Board Review Manual* is a peer-reviewed study guide for fellows and practicing physicians preparing for board examinations in cardiology. Each bimonthly manual reviews a topic essential to the current practice of cardiology.

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Cardiac Tamopnade: Diagnosis and Treatment

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Table of Contents

Introduction.	2
Patient Presentation.	2
Physiology and Pathology	2
Approach to Diagnosis.	3
Treatment	7
Summary Points	9
References.	9

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Cardiac Tamponade: Diagnosis and Treatment

Fadi M.F. Alameddine, MD, and Andro G. Kacharava, MD, PhD

INTRODUCTION

Cardiac tamponade is a compression of the heart caused by an excessive accumulation of contents in the pericardium. These contents can include fluid, blood, pus, or gas resulting from inflammation, trauma, or rupture of the heart. The 3 principal features of cardiac tamponade are elevation of intracardiac pressures, limitation of ventricular filling, and reduction of cardiac output. Severe cardiac tamponade can lead to hemodynamic deterioration and shock. The quantity of fluid necessary to produce this critical condition may be as small as 150 mL if the effusion develops rapidly, or more than 2000 mL if the effusion develops slowly and the pericardium has time to stretch and adjust to the increasing volume.¹

Multiple medical conditions can lead to cardiac tamponade. The 4 most common causes of accumulation of fluid in the pericardium in an amount sufficient to cause serious obstruction to the cardiac inflow of blood are neoplastic and infectious diseases, idiopathic pericarditis, and uremia. Cardiac surgery and percutaneous coronary interventions are frequent iatrogenic causes of cardiac tamponade.

PATIENT PRESENTATION

A 57-year-old man with a 3-hour history of worsening shortness of breath is brought by ambulance to the emergency department. The patient reports a 1-month history of chest discomfort and mild shortness of breath that has become progressively worse within the past several days. His last general medical examination was performed 3 months earlier. At that time, he was in good health, and his primary care physician reemphasized the need for him to comply with his diabetes, hypertension, and hyperlipidemia medications.

On initial physical examination in the emergency department, the patient is in respiratory distress, tachy-

cardic, and hypotensive with peripheral cyanosis. Cardiovascular examination is remarkable for attenuated heart sounds and extreme elevation of venous pressures. Breathing sounds are normal. An electrocardiogram (ECG) shows decreased amplitude of QRS complexes but otherwise is unremarkable. Echocardiography is performed and confirms the diagnosis of cardiac tamponade. Emergent needle pericardiocentesis is performed, and 1.5 L of serosanguinous fluid are drained. Immediately following pericardiocentesis, the blood pressure and heart rate normalize.

A catheter is left in the pericardial space, and over the next 48 hours an insignificant amount of fluid (< 80 mL) is drained. The catheter is removed. Further workup to determine the cause of the pericardial effusion does not identify a specific cause. The patient is treated with nonsteroidal anti-inflammatory medications and is discharged from the hospital. Predischarge echocardiogram does not reveal further accumulation of pericardial fluid.

PHYSIOLOGY AND PATHOLOGY

The pericardium is an elastic, closed fibroserous sac that envelops the entire heart and much of the ascending aorta, the main pulmonary artery, all pulmonary veins, and portions of the inferior and superior venae cavae. It consists of an inner serous layer and an external layer composed of fibrous tissue. The pericardial space between the 2 layers contains a small amount of ultrafiltrate of plasma that acts as a lubricant and normally drains through the right lymphatic duct via the right pleural space and through the thoracic duct via the parietal pericardium.^{2,3}

The pericardium is relatively inextensible and has a limited ability to expand acutely in the face of rapidly increasing pericardial contents. The exponential volume–pressure relationship for the pericardium is shown in **Figure 1**. The flat portion of the J-shaped volume–pressure curve represents intrapericardial