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Peer-Reviewed Case Report

Intra-aortic Balloon Pump may Attenuate Adverse Hemodynamic Effects of Negative Intrathoracic Pressure in Cardiogenic Shock: Case Report

Faris G. Araj

Division of Cardiology, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, TX

Corresponding author: *faris.araj@utsouthwestern.edu*

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Abstract

High negative intrathoracic pressure (NIP), as occurs during obstructive sleep apnea or hiccups, results in adverse hemodynamic consequences, specifically decreased left ventricle (LV) performance. These untoward effects can potentially be catastrophic in the case of an already compromised LV in cardiogenic shock. The interplay of intra-aortic balloon pump counterpulsation during abrupt high NIP in cardiogenic shock is described.

Background

High negative intrathoracic pressure (NIP), as occurs during obstructive sleep apnea or hiccups, results in adverse hemodynamic consequences, specifically decreased left ventricle (LV) performance.[1,2] These untoward effects can potentially be catastrophic in the case of an already compromised LV in cardiogenic shock. The interplay of intra-aortic balloon pump (IABP) counterpulsation during abrupt high NIP in cardiogenic shock is described below.

Case Report

A 54-year-old man with end-stage nonischemic cardiomyopathy and prior cardioembolic stroke presented in cardiogenic shock and was urgently listed for heart transplantation. An IABP was used due to the presence of ventricular tachycardia limiting escalation of inotrope support. Frequent intense hiccups developed, and the associated hemodynamic changes were captured (Figure 1).

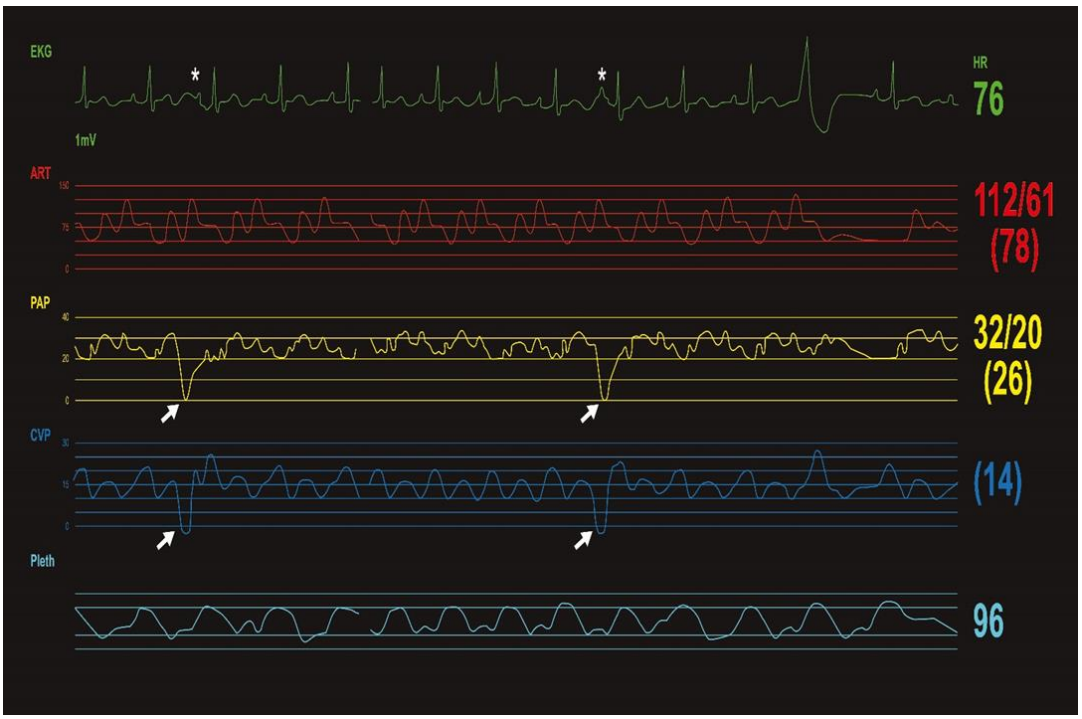

Figure 1: Hemodynamic effects of NIP during IABP support. *Hiccups result in EKG artifact (asterisks) and a decrease in PAP by 30 mm Hg and CVP by 16 mm Hg to a nadir of 0 mmHg and below 0 mmHg, respectively (arrows). A steady hemodynamic state is maintained by the IABP (red). Art indicates arterial line; CVP, central venous pressure; EKG, electrocardiogram; HR, heart rate; NIP, negative intrathoracic pressure; PAP, pulmonary artery pressure; Pleth, plethysmograph.*

Despite the high NIP generated, the IABP was able to maintain a steady hemodynamic state and counter the negative hemodynamic effects of NIP on the LV.

Discussion

Although seemingly benign, hiccups are an involuntary contraction of inspiratory muscles against a closed glottis. When intense, hiccups can result in high NIP.[3] A high NIP can increase sympathetic activation and adversely impact LV systolic function by increasing the transmural LV pressure (LV pressure-pleural pressure) and, hence, the LV afterload. This leads to a decrease in LV contractile force, stroke volume, cardiac output, and aortic pressure while increasing LV end-diastolic volume and pressure.[1, 2, 3]

Additionally, acute negative pressure pulmonary edema may develop due to a decrease in interstitial lung pressure, and an increase in venous return to the right heart during inspiration, in the presence of increased hydrostatic pressure from an elevated left heart pressure in the setting of cardiogenic shock.[4, 5]

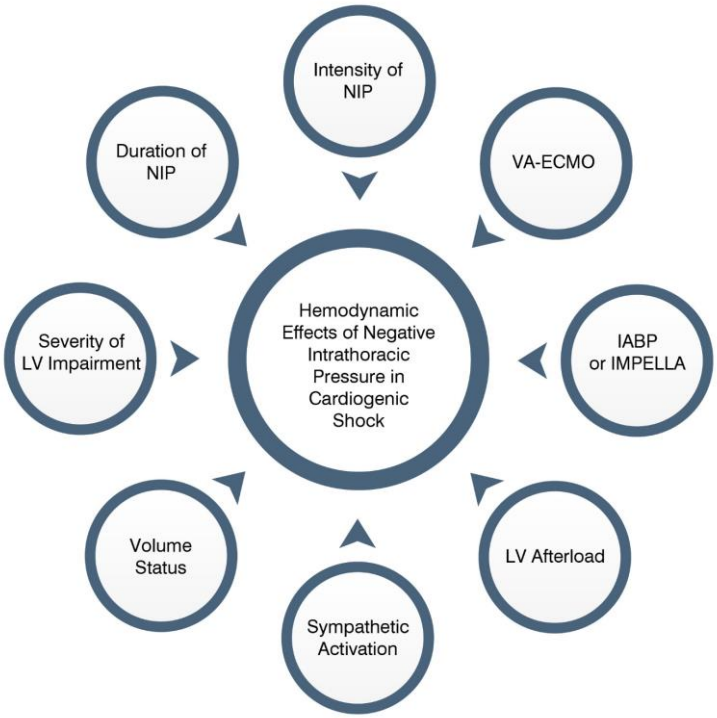

Figure 2: Complex interplay between negative intrathoracic pressure and hemodynamic changes during cardiogenic shock. *IABP indicates intra-aortic balloon pump; LV, left ventricular; NIP, negative intrathoracic pressure; VA-ECMO, venoarterial extracorporeal membrane oxygenation.*

The effects of NIP on LV performance are complex (Figure 2) and should be kept in mind when caring for patients in cardiogenic shock. By unloading the LV, the IABP appears to attenuate the hemodynamic impact of NIP.

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