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Consensus Statement

Clinical and Research Considerations for Patients With Hypertensive Acute Heart Failure: A Consensus Statement from the Society of Academic Emergency Medicine and the Heart Failure Society of America Acute Heart Failure Working Group

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ABSTRACT

Management approaches for patients in the emergency department (ED) who present with acute heart failure (AHF) have largely focused on intravenous diuretics. Yet, the primary pathophysiologic derangement underlying AHF in many patients is not solely volume overload. Patients with hypertensive AHF (H-AHF) represent a clinical phenotype with distinct pathophysiologic mechanisms that result in elevated ventricular filling pressures. To optimize treatment response and minimize adverse events in this subgroup, we propose that clinical management be tailored to a conceptual model of disease based on these mechanisms. This consensus statement reviews the relevant pathophysiology, clinical characteristics, approach to therapy, and considerations for clinical trials in ED patients with H-AHF. (*J Cardiac Fail 2016;22:618–627*)
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Pathophysiology of Hypertension in Acute Heart Failure

Hypertensive acute heart failure (H-AHF) is defined as the rapid onset of pulmonary congestion in the setting of a systolic blood pressure >140 mm Hg, and often >160 mm Hg.[1] Many patients with H-AHF have a history of poorly controlled hypertension.[2] The consequences of longstanding hypertension include changes to both the vasculature as well as the left ventricle (LV), resulting in increased stiffness and reduced compliance across the cardiovascular system.[3,4] Such stiffening increases systolic load on the LV myocardium, triggering intra- and extracellular adaptations that tend to normalize systolic and diastolic sarcomere stress.[1] Many of these changes occur at the expense of LV compliance and ultimately lead to clinically significant diastolic dysfunction.[56] As the functional ventricular-vascular relationship becomes uncoupled, the LV has insufficient cardiac reserve to compensate for the increases in afterload and preload that accompany hypertensive episodes and physical exertion.[7] As a result, the poorly compliant cardiovascular system with chronic hypertension responds with larger changes in LV filling pressure for a given change in preload or afterload.[8] Therefore, it is important that therapy for H-AHF includes interventions to both improve vascular compliance and reduce intravascular filling pressure.

Vascular Dysfunction and Its Role in Acute Heart Failure

Normal cardiovascular function requires close integration between the heart and vasculature to provide adequate blood flow to the vital organs and periphery.[9] A compliant aorta acts as a capacitor, reducing the peak pressure generated during ventricular systole and promoting continuous forward flow throughout the cardiac cycle. When the force of this flow reaches the junction of medium and small resistance arterioles, vessel expansion and recoil occurs, generating both forward and backward waveforms. While the forward component provides perfusion to target organs through the microcirculation, the reflected wave propagates back up the vascular tree, augmenting central aortic pressure. The net effect is a dynamic process of ventricular-arteriolar coupling that serves as a major determinant of cardiac output, providing a mechanism for adaptive changes in response to metabolic needs.[9]

In patients with chronic hypertension the aorta and large arteries stiffen, enhancing the amplitude and velocity of the reflected pulse wave generated by resistance arterioles.[10] Arterioles also adapt to chronic increases in systemic blood pressure through smooth muscle hypertrophy, a process that normalizes end-arteriolar pressure at the expense of a further increase in large-artery pressure and increased pulse wave velocity. The reflected wave, which normally reaches the central aorta after aortic valve closure, can increase velocity enough to return to the proximal aorta in late systole. This results in increased load for ventricular contraction and may trigger early aortic valve closure. An immediate consequence of shortened LV systole due to premature aortic valve closure is compromised coronary artery perfusion, as well as increased diastolic volume and pressure, resulting in increased pulmonary venous pressures, predisposing to development of pulmonary congestion.[11,12] In the setting of compromised ventricular function, the effect of vasoconstriction, especially when abrupt, is harder to overcome, contributing to the onset of H-AHF.

Blood Pressure and Its Relationship to Acute Heart Failure

Large registries have shown that 50% of patients with AHF have elevated blood pressure at presentation to the ED.[13,14] Moreover, the initial systolic blood pressure is a strong predictor of outcomes, with higher blood pressures associated with lower rates of in-hospital mortality and 30-day myocardial infarction, death, or rehospitalization, as well as a greater likelihood of discharge within 24 hours.[15,16] These associations may have corresponding anatomic implications as a result of contractile reserve, because higher systolic blood pressure[15] is more often seen in the setting of preserved ejection fraction, with the likelihood of an ejection fraction >40% increasing 3% for every 1 mm Hg presenting systolic blood pressure >120 mm Hg (Table 1).[17] However, presenting blood pressure depends on multiple factors, including medication adherence, making it difficult to reliably predict underlying ejection fraction based on blood pressure alone.

Ultimately, blood pressure summarizes cardiac contractile force relative to the vascular resistance it encounters.[18,19] Maintenance of blood pressure is tightly regulated by baroreceptors, primarily in the aorta and carotid arteries although renal mechanisms are also involved. Changes in cardiac output and systemic vascular resistance are triggered by sympathetic nervous system and neurohormonal activation in response to baroreceptor-mediated detection of alterations in vascular pressure. Thus, when confronted with increased arterial resistance, a heart with normal contractile reserve is able to maintain cardiac output with a net increase in systolic blood

Table 1. Heart Failure With Preserved Ejection Fraction According to Presenting Blood Pressure

Systolic Blood Pressure (mm Hg)	Proportion With Heart Failure With Preserved Ejection Fraction (%)
110	43.5
120	46.6
130	47.8
140	48.0
160	58.7
180	63.6
200	73.3

From Styron et al.[17]