A Contemporary Picture of Congestion in Heart Failure: from Dropsy Impression to Multifaceted Reality

Anzhela Soloveva¹ • Marat Fudim²

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Abbreviations

ASCEND-The Acute Study of Clinical Effectiveness of HF	Nesiritide in Subjects With Decompensated Heart Failure trial
BP	Blood pressure
HF	Heart failure
GSN	Greater splanchnic nerve

Near universally, congestion in heart failure (HF) is considered to be the result of excessive water accumulation. Defined as increased cardiac filling pressures, congestion, in fact, is the result of a combination of volume expansion and cardiovascular changes. A number of the derivatives, such as composite clinical models, laboratory markers, impedance- and ultrasound-based tools, as well as direct invasive hemodynamics both at rest and exercise, have been used to assess and monitor congestion and prognosticate outcomes. However, the measures of congestion often do not correlate with each other and likely present different components of a patient's volume status. As such, an in-depth investigation of the mode of cardiac decompensation, tools to predict and measure it are needed to reframe the current decongestion strategy from mainly a diuretic-based to an individualized approach.

In this mini-focus issue entitled “Volume Management in Heart Failure”, Bapna et al. [1] present the unique effects of greater splanchnic nerve (GSN) stimulation via an implantable cuff system in healthy canines. The stimulation led to a rapid, adjustable, and reproducible increase of arterial, central

venous, and pulmonary arterial blood pressures (BP) up to 30 days. Conceptually, GSN stimulation could be the basis of a novel animal HF model, as the predominant sympathetic control of splanchnic vascular capacitance underlies the concept of fluid redistribution in HF. Sympathetic triggers (stress, exercise, infection, ischemia, etc.), via vessel contraction, lead to intravascular volume shifts, preload increase, and congestion-related symptoms in acute HF and chronic HF. Whether neurohormonal blockade can restore splanchnic capacitance in HF remains to be determined. Nijst et al. [2] studied the concept of cardiovascular volume reserve. In a contemporary cohort of 40 clinically euvoletic patients with HF and reduced ejection fraction, intravascular volume expansion with 0.7 L hydroxyl-ethyl-starch was safely tolerated and resulted in mostly transient intra-cardiac pressure increase. This supports the significance of vascular storage capacitance in cardiovascular conditions such as HF and hypertension. High filling pressures do not have to equate to high filling volumes. This was further confirmed by Fudim et al. [3] in patients with hypertension. The investigators found a wide range of intravascular blood volume parameters in a diverse patient cohort. This analysis underlined the potential utility of quantitated blood volume analysis to guide the volume management in cardiovascular diseases.

The provocative review by Morris et al. [4] explores the cardiovascular consequences of ilio caval venous obstruction. Central venous obstruction reduces cardiac preload reserve during physical activity and appears to be a possible cause of dyspnea and exercise intolerance. The authors describe when to suspect and how to diagnose, but also how to distinguish this disease presentation from HF with preserved ejection fraction. Both disease forms appear to have similar features.

Although the above-mentioned concepts enhance our perception of the low reliability of HF clinical signs and symptoms, clinically measured congestion is associated with outcomes and is a key driver of decision-making in clinical

✉ Marat Fudim
marat.fudim@duke.edu

¹ Division of Cardiology, Almazov National Medical Research Centre, Saint Petersburg, Russia

² Division of Cardiology, Duke University Medical Center, 2301 Erwin Road, Durham, NC 27715, USA

practice. In a post hoc analysis of the ASCEND-HF trial, Masson et al. [5] aimed to identify the prognostic role of a novel in-hospital score which combined orthopnea, edema, and NT-proBNP in acute HF. Residual congestion, present in 62% of patients, was characterized by a very high event rate after discharge. Though the risk prediction of the novel score did not outperform traditional risk factors in HF, the study reminds us of the remarkable contribution of congestion to adverse outcomes. As emphasized by the authors, it is difficult to generate a simple and universal congestion model that would apply to a heterogeneous HF population, with variable congestion mechanisms.

Concepts investigated in the Special Issue of the Journal uncover the complexity of congestion in HF and demand future investigations to explore complementary/alternative mechanisms of congestion in acute and chronic HF.

Compliance with Ethical Standards

Disclaimer The authors take responsibility for the manuscript's integrity and had complete control and authority over its preparation and the decision to publish.

Conflict of Interest Dr. Soloveva declares no conflict of interest. Dr. Fudim consults for AxonTherapies, Daxor and Galvani.

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