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EDITORIAL COMMENT

# Intrarenal Venous Flow

## A Window Into the Congestive Kidney Failure Phenotype of Heart Failure?*

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Although the importance of congestion in the disease progression of heart failure has long been recognized (1), much has focused on how cardiac impairment can lead to renal dysfunction. Over the past decade, there is increasing recognition that the ability of the kidneys to compensate for fluid overload can be influenced not only by reduced arterial perfusion or underlying intrinsic renal abnormalities, but also by increased venous pressure (2–4). However, unlike the heart, the lack of reliable bedside tools that can provide insights into real-time renal physiology has somewhat limited our abilities to better understand the factors contributing to cardio-renal syndrome. To date, clinicians rely on surrogates of venous congestion that are primarily inferred by structural and vascular flow abnormalities in the right heart, inferior vena cava, or hepatic veins (**Figure 1**) (5–7). Although there have been promises of directly quantifying renal perfusion using ultrasound contrast, they are yet to be clinically applicable (8).

Being a versatile, noninvasive tool for evaluating abnormal renal artery stenosis or obstructive uropathy, renal Doppler ultrasonography has rarely been investigated for profiling renal hemodynamics in the setting of heart failure. Recent reports have suggested that estimating the degree of arterial renal blood flow with the renal resistive index may identify higher-risk patients with heart failure (9,10). Meanwhile, a low intrarenal venous impedance index (VII) calculated from the renal venous waveforms has been associated with raised renal interstitial pressure caused by non-heart failure conditions such as acute ureteral obstruction (11,12) or diabetic nephropathy (13). In this issue of *JACC: Heart Failure*, Iida et al. (14) systematically examined distinct renal Doppler flow patterns in patients with largely nonischemic heart failure sta-

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bilized either following hospital admission or in the outpatient setting. Interestingly, this patient cohort included ~40% patients with either heart failure with preserved ejection fraction or pulmonary arterial hypertension. Despite relatively preserved cardiac and renal function, 43% had advanced (New York Heart Association functional class III to IV) symptoms and yet only 28% demonstrated an increase in right atrial pressures. The authors first observed that VII was difficult to quantify in this population, in part because there were disruptions of the normal continuous intrarenal venous flow (IRVF) pattern. They therefore speculated that the IRVF patterns were largely attributable to altered renal interstitial pressures in the setting of increased venous congestion (as reflected by estimated right atrial pressure). Indeed, venous congestion can produce elevated right-side pressures that transmit backward into the renal parenchyma similar to that observed in hepatic venous flow patterns (**Figure 1**), leading to increased pulsatility of the IRVF signal. This pulsatility (so-called "discontinuous IRVF pattern") may reflect increased compliance of renal parenchyma and its venous vessels in response

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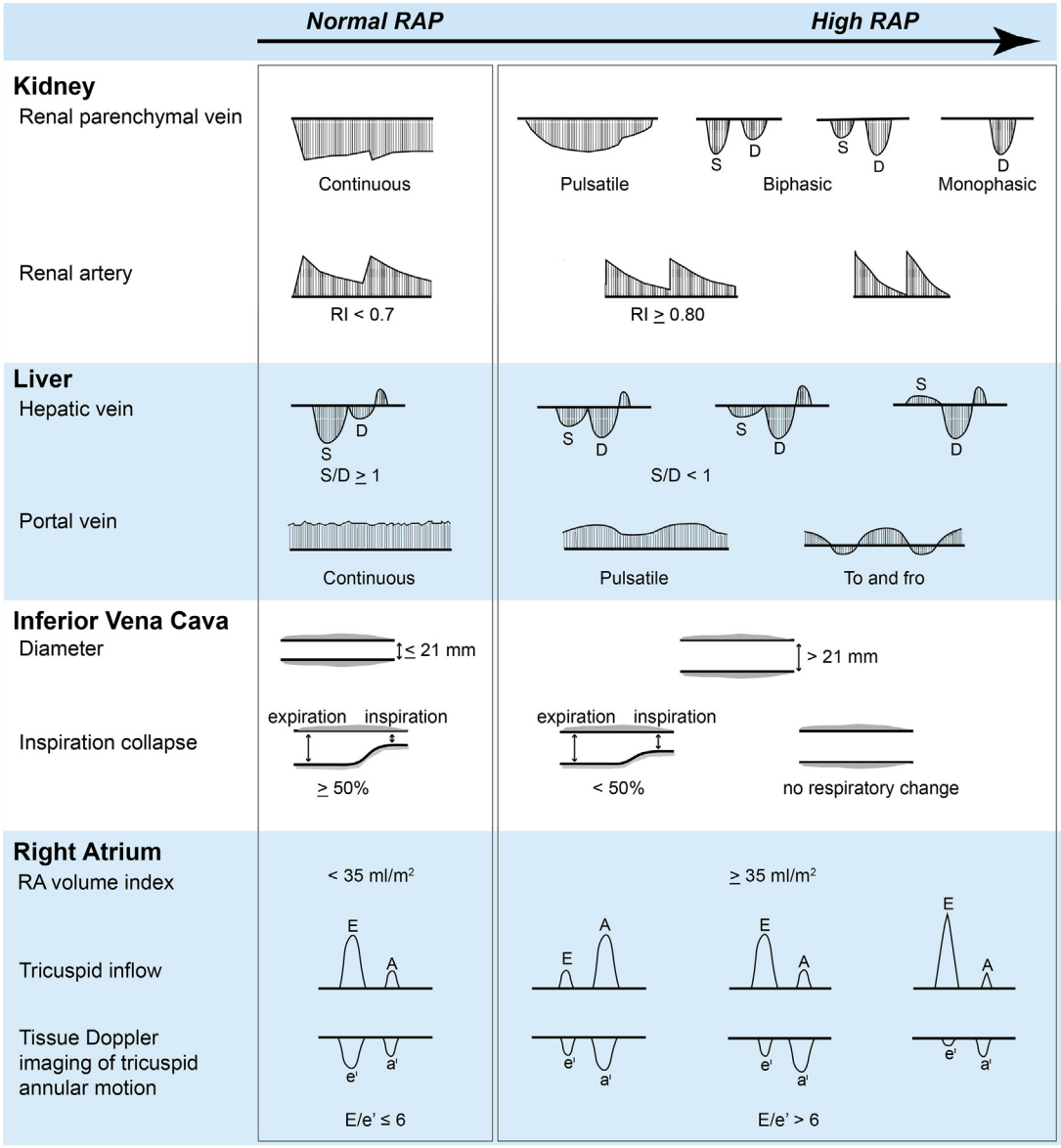
**FIGURE 1** Ultrasound Patterns Across the Spectrum of RAP




RA = right atrium; RAP = right atrial pressure; RI = resistive index.

to increasing venous pressure within the encapsulated kidneys. It can manifest by biphasic forward velocity that peaks during each cardiac cycle (biphasic pattern), or a gradual diminution of velocity throughout systole that evolves into a diastolic-only flow pattern (monophasic pattern). Because patients in this study were relatively lean (mean body mass index of 23 kg/m$^2$) and the fact that all IRVF measurements were performed by a single individual, the technical feasibility and consistency of Doppler waveform sampling of interlobular vessels in a more diverse group of patients and sonographer should be investigated. It is also important to note that discontinuous or biphasic patterns of IRVF and lower VII are not specific to heart failure, and have been described in normal pregnancies to at least in part be explained by reduced vascular compliance from increased renal