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## **New Ways to Visualize and Combat Congestion in Heart Failure**

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Congestion is at the heart of heart failure (Figure 1; 1, 2). Indeed, congestion and arrhythmias, which may not be unrelated, can be considered the two most important, therapeutic targets in patients with heart failure. Congestion is a key cause of exercise intolerance and symptoms and the most important cause of admissions for heart failure (3). Accordingly, managing congestion is a key task of the heart failure physician; it is also one of the most difficult. Guidelines on heart failure provide a plethora of advice on the use of neuro-endocrine antagonists but virtually no advice on how to use diuretics, which are probably the most used, abused and effective agents for managing congestion (4).

In patients admitted with symptoms of worsening heart failure, evidence of congestion on clinical examination is often obvious, but not always (5). There can be considerable diagnostic uncertainty; there are many reasons for breathlessness and peripheral oedema other than heart failure. Moreover, peripheral congestion (oedema) may occur without cardiac congestion (an increase in atrial pressures) even in patients who have heart failure, especially during a diuresis, because of delays in repatriating fluid from tissues into the circulation. Aggressive diuresis in such patients may be deleterious, causing hypotension and renal dysfunction (6). Patients may also have cardiac congestion without peripheral oedema, because it takes time for raised pressures to translate into oedema, especially when lymphatic drainage is good or the patient is on bed rest and gravity has not taken its toll. Clinical inexperience, patient obesity and respiratory disease and the difficulty in eliciting clinical signs such as jugular venous distension add further confusion and complexity.

A raised jugular venous pressure is probably the most reliable clinical sign of fluid overload and, when its assessment is performed by expert clinicians, it identifies patients with a worse outcome (7, 8). Recently, the increasing availability of portable, or hand-held, ultrasound

machines has stimulated interest in a more technologically sophisticated approach to the assessment of fluid status and congestion. The diameter of the inferior vena cava (IVC) can easily be measured with a modicum of training and offers a fairly reliable estimate of right atrial pressure. Measuring the IVC or the internal jugular vein diameter, with their responses to respiratory manoeuvres, allows an even more precise, reproducible and objective measure of the severity of congestion (9, 10). Thus, imaging the IVC is a quick and convenient method for routinely assessing cardiac congestion at the bedside in patients with suspected heart failure that not only helps confirm the diagnosis but might also guide diuretic therapy and the timing of discharge. It can also provide prognostic information complementary to that obtained clinically or by measuring plasma concentrations of natriuretic peptides and independent from other measures of cardiac function such as left ventricular ejection fraction; congestion is similarly bad news whether the patients has HFrEF or HFpEF (11,12). Algorithms for the assessment of congestion have been proposed, but do not yet include venous ultrasound. Some variables used in these models, such as the severity of dyspnoea or fatigue, are subjective and open to assessment bias by the patient and clinician, and others, such as the clinical assessment of JVP, also require considerable clinical skill. Using such criteria, up to 40% of patients admitted with worsening heart failure will still have at least moderate congestion at discharge (13). Failure to control congestion places the patient at high risk of recurrent hospitalization and death (1, 14).

In the current issue of this journal, Dr. Josa-Laorden and colleagues provide interesting insight into these issues. They prospectively followed 85 patients for six months after an admission for worsening heart failure; two-thirds of patients had an LVEF  $\geq 50\%$ . IVC diameter was measured within 24h of admission using a portable hand-held device. Most patients had a dilated ( $>2.0\text{cm}$ ) IVC (55%) or attenuation of the normal inspiratory IVC

collapse (70%); of those with a dilated IVC, only 5 (11%) had a normal (>50%) inspiratory response. Patients with a dilated IVC were more likely to have a distended JVP and hepatomegaly on physical examination, and higher plasma concentrations of biomarkers reflecting volume overload, such as NTproBNP and CA125. Patients who had a dilated IVC that failed to collapse appropriately during inspiration had the worst outcome. A few patients had an apparently dilated IVC but with appropriate collapse on inspiration and appeared to have less advanced heart failure. These might simply have been larger patients with a naturally larger IVC; alternatively, restoration of the inspiratory collapse may precede reduction in IVC diameter in patients responding to diuretic therapy or patients who are less sick may be able to generate a greater inspiratory pressure. Conversely, patients whose IVC was not dilated but failed to collapse appropriately appeared sicker; perhaps IVC dilatation depends on the chronicity as well as severity of congestion. As highlighted by the authors, the studied sample-size was small, and some mismeasurement and misclassification is inevitable, especially when the result is binary – a 49% or 51% IVC collapse are unlikely to carry different biological meaning. Using two or more threshold criteria (eg:- >60%; 30-60% and <30% collapse) to classify people into high, low and intermediate groups would likely improve the clinical and prognostic utility of the measurement. Other factors might have influenced these results: we do not know the time interval between the onset of symptoms that prompted the hospital admission and the ultrasound assessment or the total amount of diuretics administered. It would have been useful to have had measurements before and after treatment. The acute effects of diuretic treatment may have a more marked effect on IVC collapsibility than on its diameter (15): whether this reflects an acute haemodynamic response to diuretics, or improved inspiratory performance, or both is not clear. Finally, it is unknown in how many patients a correct IVC visualisation was possible.

Importantly, discrepancies might exist between clinical and echocardiographic assessment of fluid overload. Of those with a non-dilated IVC, 42% were considered to have a distended JVP on clinical examination, whilst 35% of those with a dilated IVC were reported not to have a distended JVP. This highlights the possible subjectivity of the clinical examination, some measurement error during imaging and the need for more objective and standardised methods for assessing congestion. However, as noted above, there may be other factors that affect the relationship between right atrial pressure and IVC diameter, such as disease chronicity and the acute response to therapy.

In summary, ultrasound assessment of the IVC in patients admitted with suspected heart failure appears helpful in assessing congestion and stratifying risk; whether it adds useful information to the clinical examination, which may depend on the clinician's expertise, and whether it can be used to guide therapy or the timing of discharge awaits further investigation. Alternative invasive and non-invasive devices for measuring atrial pressure and congestion should also be considered for the acute and long-term management of fluid balance. If congestion can be mastered the war on heart failure is at least half-won (1, 14).

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**Legend to figure 1.** Schematic diagram showing factors important to the development of moderate, or more severe congestion in patients with heart failure, reflected by a dilated but still collapsible inferior vena cava (IVC; a), or a dilated and stiff IVC at echocardiography (b), respectively. Cardiac dysfunction leads to a rise in atrial pressures and/or a fall in systemic arterial pressure. The fall in net renal perfusion pressure triggers salt and water retention causing an expansion in circulatory volume and a further rise in atrial pressure. This may lead to peripheral (including renal parenchymal) oedema (6). Rising venous pressure and the inability of plasma volume expansion to restore systemic arterial pressure triggers further salt and water retention. The increase in the diameter of the IVC may reflect the chronicity of the elevation in RA pressure; the failure of the IVC to collapse on inspiration could reflect an acute on chronic component. Response to diuretics will likely restore the inspiratory response before an effect is observed on IVC diameter.



Heart Failure

↑ intravascular hydrostatic pressure (chronic)

Overwhelmed lymphatics



Renal Parenchymal Oedema

Pre-existing glomerular damage  
Hypertension/T2DM



↑ Diuretic resistance  
↑ Anaemia

Diuretic



Renal Dysfunction

Renal artery atherosclerosis

↓ Systemic Arterial Pressure

↑ Salt and water retention

↑ Intra vascular volume

↑ RA pressure

