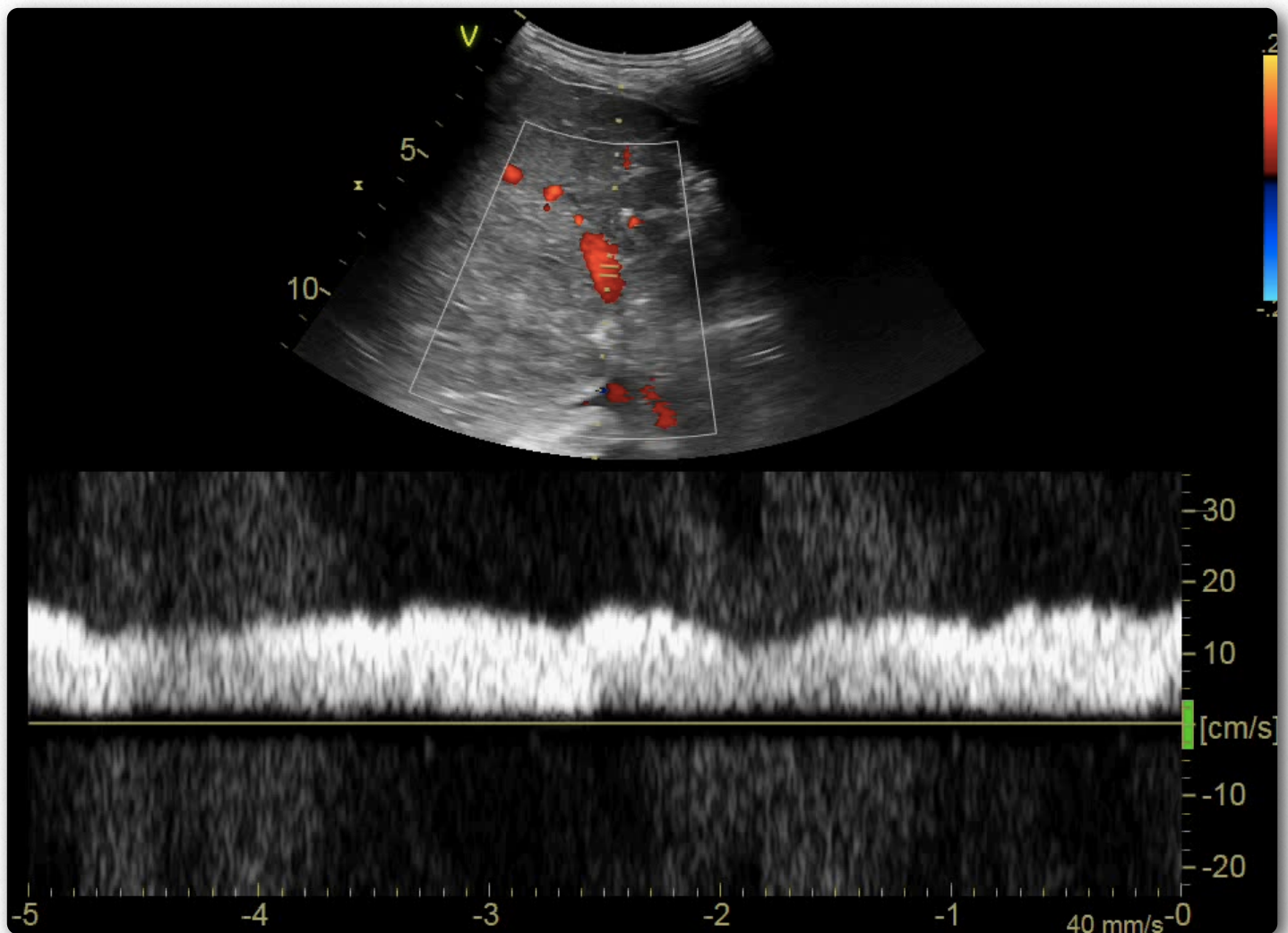# 6

Special Skills: Venous Congestion

with co-authors Dr. Korbin Haycock & Dr. Rory Spiegel

The set of ultrasonographic parameters described here are by no means new, having been described in the literature decades ago. However, they have never been routinely used by front-line clinicians in the management of disorders where venous congestion is critically important. It is our group's feeling that, being often under-recognized and under-diagnosed, this is an area of POCUS that can provide significant value in fluid management.

So what are the clinical conditions in which it is important to consider venous congestion as a potentially significant factor?

a. congestive heart failure - this is the most obvious one.

b. shock & de-resuscitation- as discussed previously, avoiding volume overload can diminish a significant amount of complications.

c. acute renal failure - contrary to common practice, fluid administration only benefits the subset of patients with pre-renal failure, and either is the cause or can worsen the other subsets.

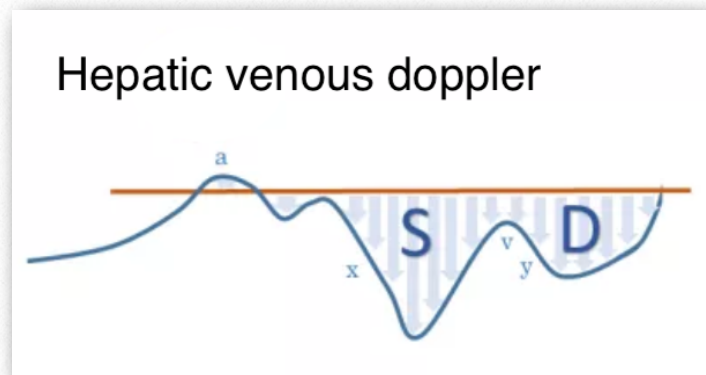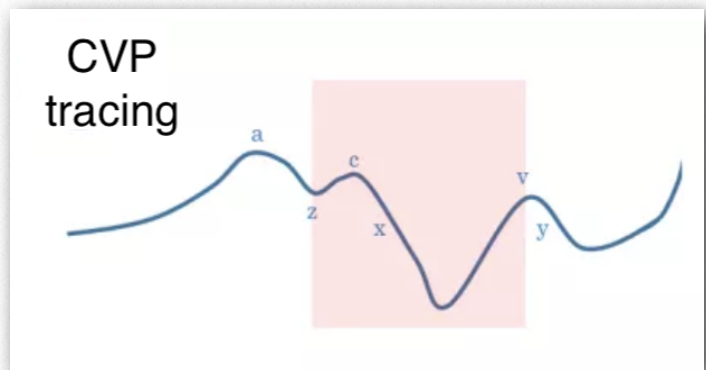d. any patient where you are considering giving fluids.

Looking at that list, it becomes rather apparent that you should probably be looking at this in most of your patients. Of course, without realizing it, you likely are, if you have started to apply the concepts in the earlier sections of this handbook.

The Physiology of Venous Congestion

The IVC is the initial screen for venous congestion. Whether due to increased stressed venous volume or an increased right atrial pressure (for any given reason), the first venous compartment where congestion becomes apparent is the IVC. When the IVC reaches the flat part of it's compliance, the pressure begins to be transmitted in a retrograde fashion and dilation of the hepatic venous tree is seen (rabbit ears or staghorn appearance in short axis), and so on for the renal veins. The normal flow pattern see in the hepatic veins closely mirror the CVP tracing from which they essentially origi-

Full IVC in SAX with "Staghorn" appearance

nate, and are described as having three waves, a retrograde A wave, followed by antegrade S and D waves. This can be well seen in the excellent diagrammatic representation below (adapted from Kenny, with permission).

The portal system may show abnormalities as the congestion becomes more severe, making it potentially a more specific marker of
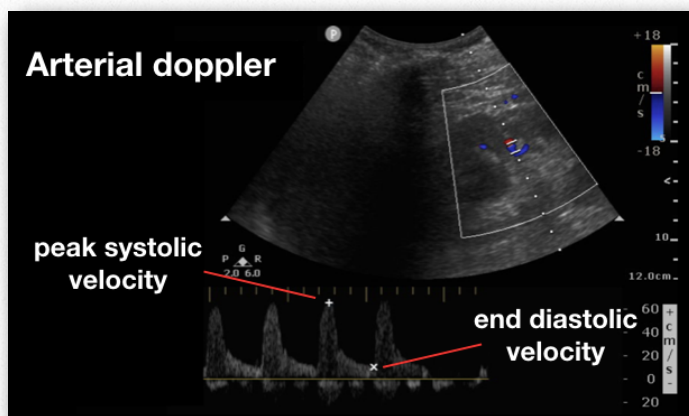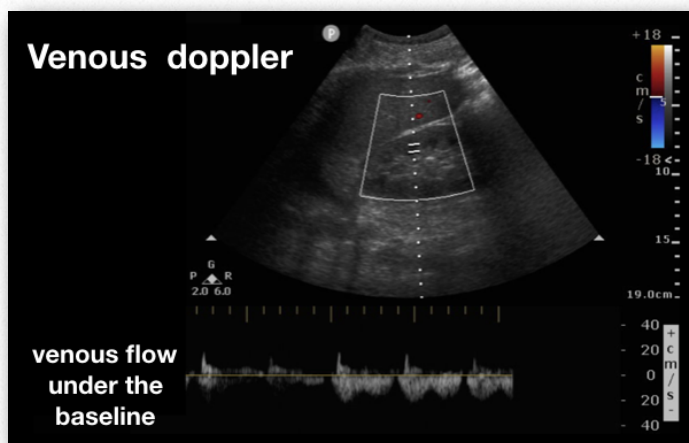
an injurious level of congestion. This likely takes place because the pressure has to transmit across the hepatic sinusoids which buffer the hepatic venous pressure. Eventually, the portal venous flow pattern changes, going from a mono-phasic signal to a progressively more pulsatile pattern that can eventually become interrupted (100% pulsatility).

A fairly linear relationship between right atrial pressure and portal vein pulsatility index has been described in CHF patients in acute exacerbation (Shih et al, Catalano et al).
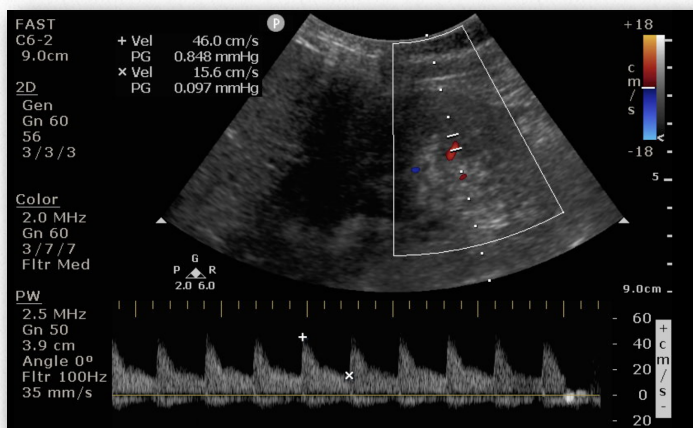
Renovascular Doppler

Doppler interrogation of the renal interlobar or arcuate blood vessels can be used to assess for renal venous congestion. There are two components of the intra-renal Doppler exam: the renal resistive index (RRI) and the intra-renal venous flow. Because the arteries and veins travel in an anti-parallel manner along the same anatomic path, it is possible to assess both the arterial and venous flow patterns within a single PW Doppler gate. When assessing the kidney for venous congestion, the renal artery (RA) flow is seen as the Doppler waveform above the baseline, whilst the venous Doppler is seen below the baseline. Iida et al have an excellent review article on this.

The renal resistive index can be calculated from the RA waveform, and is determined by multiple factors and may be affected by renal venous congestion as well (Iida et al). RRI is defined as systolic peak velocity minus end-diastolic velocity/peak systolic velocity. The figure below thus shows an RRI of 0.66 (46-15.6/46) A RRI of less than 0.6 is consid-

ered normal, whereas and value of greater than 0.7 is pathological. An advantage of having both arterial and venous waveforms in the same gate is that capacity to identify systole and diastole accurately, and therefore the corresponding venous phases. RRI abnormalities should prompt consideration of venous congestion, but due to its multi-factorial nature, an abnormal

finding in isolation should not be taken as definitive evidence of volume overload.

Conversely, the intra-renal venous Doppler pattern is a more reliable indication of reno-sarca and venous congestion. As right atrial pressures rise, the intra-renal venous Doppler pattern transitions from a normal phasic pattern to first an uninterrupted pulsatile waveform, followed by an interrupted bi-phasic pattern (with one waveform happening during systole and the second during diastole), then finally to a diastolic mono-phasic Doppler pattern. In the bi-phasic pattern, the systolic component corresponds to the S wave of the hepatic vein Doppler (or the x descent of the CVP waveform) and the diastolic component corresponds to the D wave (or the y decent of the CVP waveform). Just as increases in right atrial pressure result in a relative decrease in absolute magnitude of the hepatic S/D wave ratios, the systolic component of the bi-phasic intra-renal venous Doppler signal gradually decreases in velocity as the right atrial pressure rises, until the pattern progresses to a diastolic only mono-phasic pattern and the systolic wave eventually decreases and disappears (Iida et al, Tang et al).

To perform the intra-renal Doppler exam, place the color flow box over the kidney to find the interlobar vessels residing between any two medullary pyramids. Fanning the probe to achieve a different US beam alignment may facilitate the acquisition of a better Doppler waveform in some cases where only a weak signal or no signal can be identified. Next, place the PW Doppler gate over the interlobar vessels and activate the Doppler. The PW Doppler scale can be decreased for more accurate measurement of RRI if deemed necessary. If determination of

Technique: To perform the intra-renal Doppler exam, first place the color flow (CF) box over the kidney to find the interlobar vessels residing between any two medullary pyramids. Fanning the probe to achieve a different US beam alignment may facilitate the acquisition of a better doppler waveform in some cases where only a weak signal or no signal can be identified. Next, place the PW doppler gate over the interlobar vessels and activate the doppler. The PW Doppler scale can be decreased for more accurate measurement of RRI if deemed necessary. If determination of the RRI is desired, it is best to sample at least three different vessels to calculate a mean RRI if possible.

the RRI is desired, it is best to sample at least three different vessels to calculate a mean RRI if possible.

An interrupted bi-phasic to mono-phasic venous Doppler pattern very likely is an indicator of increasingly severe congestion, and should be of concern. Occasionally, an uninterrupted pulsatile venous waveform (not to be confused with the bi-phasic or mono-phasic patterns) can be found in hyperdynamic flow states, and does not necessarily represent elevated right atrial pressures if no other evidence of venous congestion is found on the IVC, hepatic, or portal vein evaluation. However, when other evidence of elevated right atrial pressures exists, this pulsatile intra-renal venous Doppler pattern would be significant.

Classification

Our group uses the following system to examine the venous circulation:
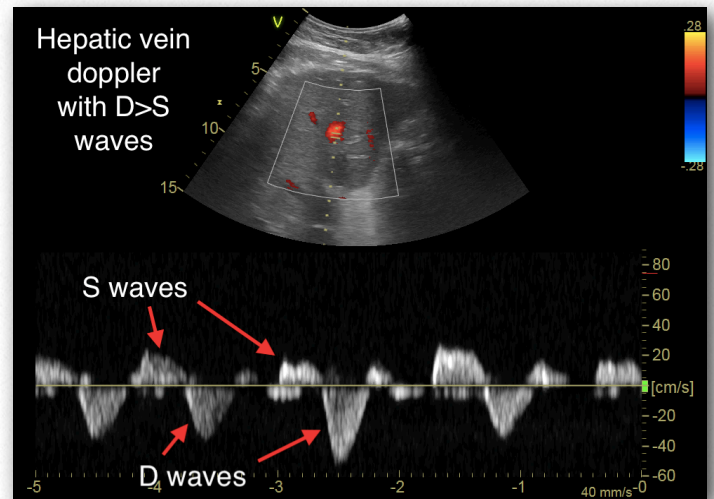
Inferior Vena Cava (IVC)

Grade 0 - < 5 mm with respiratory variation

Grade 1 - 5-9 mm with respiratory variation

Grade 2 - 10-19 mm with respiratory variation

Grade 3 - > 20 mm with respiratory variation

Grade 4 - > 20 mm with minimal or no respiratory variation

Note: The IVC is interrogated in long and short axis along the intra-hepatic segment and a visual average is done. Respiratory variation is defined as a 20% or more change in surface area in short axis.

Hepatic Vein (HD) - interrogation by pulsed wave doppler, identification and analysis of A, S and D waves:

Grade 0 normal S > D

Grade 1 S < D with antegrade S

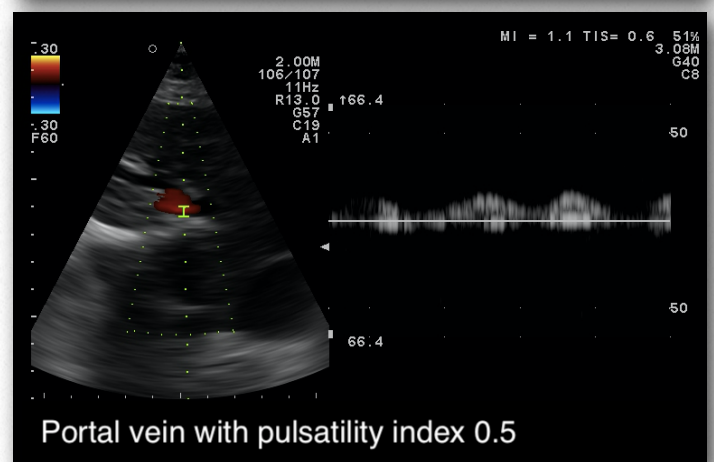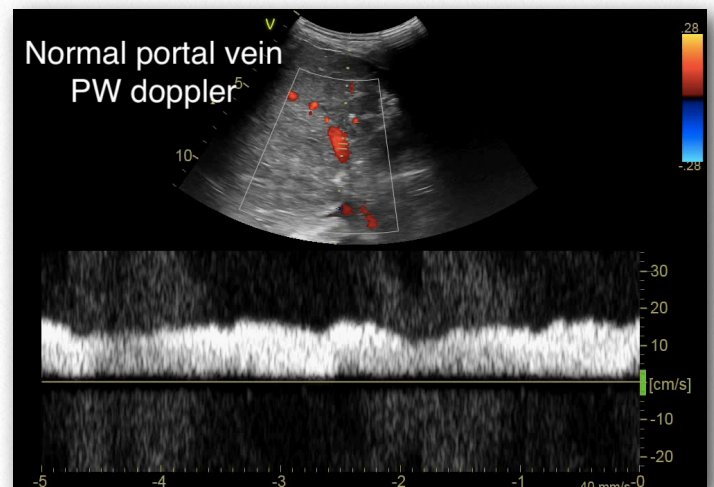Grade 2 S flat or inverted or biphasic trace.

Note - there is some confusion about the nomenclature of hepatic doppler waves in the literature, with some authors describing the waves in terms of absolute size in the physiological direction of flow (the approach we have chosen where the S wave is normally the larger of the two negative deflections), and others using a positive vs negative deflection interpretation of greater or lesser (making a smaller amplitude S wave 'greater' than the D wave in mathematical +/- terms). Also, some authors will illustrate the A wave after the D wave, others before the S wave. We have chosen the latter as it matches with the traditional a-c-x-v-y of the CVP tracing description.

Hepatic vein doppler with D>S waves

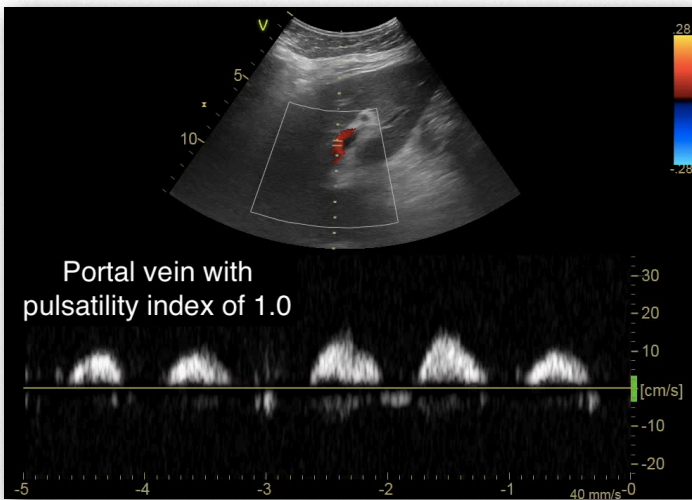### Portal Vein (PV) interrogation (PW)

Grade 0 - < 0.3 pulsatility index

Grade 1 - 0.3-0.49 pulsatility index

Grade 2 - 0.5-1.0 pulsatility index

Note - Pulsatility index is calculated as (Vmax-Vmin)/Vmax

Normal portal vein PW doppler

Portal vein with pulsatility index 0.5

Portal vein with pulsatility index of 1.0

Renal Doppler (RD)- interrogation by pulsed wave doppler and waveform analysis.

Venous:

Grade 0 continuous monophasic/pulsatile flow

Grade 1 dis-continuous biphasic flow

Grade 2 dis-continuous monophasic flow (diastole only)

Arterial: renal resistive index is measured (peak systolic-end-diastolic/peak systolic, with normal range below 0.7).

Generating the views:

Hepatic doppler is relatively straightforward and given the three large hepatic veins, relatively simple. Either the middle hepatic vein in the subxiphoid area or the right hepatic vein from a lateral angle are usually accessible echographically. Similarly, the portal vein can be seen dividing into left and right, such that the right can be interrogated from a lateral approach, which is the most commonly used. As for all doppler examinations, the most parallel angle is best. One must be careful to analyze the waveform during a respiratory pause, as the vessel phasing in and out with respiratory liver movement may prevent proper tracing generation, particularly with the portal vein. Time does have to be spent getting used to this, and building the confidence to know that you are getting an appropriate sample of the venous flow.

Venous Assessment Concordance

It is important to note the importance of concordance between these different viewpoints - as there are some false-positives - making the accuracy of the congestion analysis dependent on this. The IVC congestion is primordial in the development of organ congestion, hence must be present. Portal vein pulsatility can occur in young patients with hyperdynamic circulation as well as in patients with cirrhosis, whether cardiac or otherwise. As noted earlier, renal venous doppler may also show some anomalies with hyperdynamic states. Hepatic venous flow pattern can be clearly abnormal with tricuspid regurgitation diminishing the S wave, but this may not reflect clinically significant organ congestion. In the authors' opinion, IVC congestion coupled with one or more find-

ings of significant doppler findings in the other should make the syndrome of pathologic venous congestion more likely.

Clinical implications

So what is the clinical significance of these findings? It is important to realize that venous congestion is invariably the result of right sided failure (tension pneumothorax and tamponade excepted), be it primary, secondary, systolic, diastolic or valvular. The most common cause remains secondary, due to left-sided failure. Hence right sided failure can occur in the absence of systolic RV dysfunction or even RV/LV ratio or TAPSE abnormalities. A common clinical lapse here is to think that the cause of the elevated atrial pressure somehow mitigates against its effects, which it does not. The splanchnic organs do not care why the elevated downstream pressure is congesting them. The only mitigating factor seems to be time, as some patients do seem to adapt very well to the chronically elevated pressures. One theory is progressive adaptation with increased lymphatic drainage preventing intra-organ congestion, though this has its limits as the thoracic duct, draining into the right atrium, will also be subject to congestive dynamics.

It was first noted in the cardiology literature that the degree of portal vein pulsatility is related to RAP and severity of symptoms (Catalano et al, Goncalvesova et al, Ikeda et al). However, no studies were undertaken to assess whether tailoring treatment to target pulsatility seem to have been performed. Recently, the group of Denault et al. have produced a number of studies in cardiac surgery patients, recently

showing the association between portal vein pulsatility, abnormal intra-renal flow patterns and the development of acute kidney injury (Beaubien-Souligny et al). That group has also observed association between venous congestion and not only weaning from mechanical ventilation, as can be expected to some degree, but also with post-operative delirium (*Denault, personal communication*). It is not surprising that the brain and kidneys be particularly sensitive to elevations in venous pressure, since they are both encapsulated organs.

The concept of perfusion pressure is critical to keep in mind when it comes to organ dysfunction, as the delta between MAP and the downstream organ venous pressure is equally affected by a drop in MAP or a rise in venous pressure. The additional realization that proximal capillary pressure - the true upstream perfusion pressure to the tissue - is substantially lower than measured arterial pressure, and may be in the range of 10-20 mmhg (Shore), makes the importance of elevated venous downstream pressures even more striking. Understanding this makes POCUS assessment of venous flow logical and important.

Hence, the findings of pathological venous congestion should prompt decompression. Depending on the pathophysiology, the intervention may vary, hence further assessment is needed looking at RV function, LV function and the presence of reversible causes. Let's look at the common clinical syndromes mentioned earlier:

1. "simple" CHF due to LV dysfunction - if RV function is relatively intact, then fluid removal by diuresis should be the next step,

likely until markers of severity such as PV PI > 0.50 resolve. In pure RV failure, strategies to decrease pulmonary vascular resistance should be considered.

2. Shock and De-resuscitation: if you are resuscitating with fluids, and signs of venous congestion appear, it likely should represent a "stop point" and, even in vasopressor-dependent shock, consideration should be made for diuresis or fluid removal. There is often concern about worsening hemodynamics by removing intravascular volume in a patient in shock, however the LV is usually on the flat part of Starling's curve, and, contrary to common concerns, decongesting the RV usually results in hemodynamic improvement.

3. In acute renal failure, despite common practice, fluids should not be blindly administered and, in the authors' opinion, should likely not be given past a mid-sized IVC, except perhaps in the presence of significant pulmonary hypertension. By the same token, patients in acute renal failure with signs of significant venous congestion - particularly in renal venous Doppler - should have fluid removed in order to decrease intra-renal edema and improve renal perfusion pressure, as this suggests the presence of a functional cardio-renal syndrome. This should be of even more importance when there is AKI in conjunction with CHF, as common practice involves withholding diuretics or even administering fluids which, though well-intentioned, compounds the pathology.

4. Fluid therapy - fluids, including blood products, are often given liberally for a number of reasons. Most experienced sonographer-physicians prefer to have an idea of the venous system prior to administering significant amounts. Certainly signs of venous congestion would preclude the administration of fluids for the sake of volume. The author's general preference is to aim for a mid-sized IVC with preserved respiratory variation, however this may vary depending on clinical considerations.

Conclusion

This is a space to watch. Further studies are needed and several are underway which will help delineate the approaches and thresholds for therapy. In the meantime, in the absence of evidence for any other meaningful or measurable management strategies in this field, it seems reasonable to extrapolate tailoring therapy to prevent or steer the patient away from the known pathological states of venous congestion which have been clearly associated with renal failure, delirium and prolonged mechanical ventilation.