Ultrasonographic examination of the venae cavae

There are two venae cavae in humans. The superior vena cava (SVC) comprises the connection of the left and right brachiocephalic veins and ends on the top of the right atrium, after entering the pericardium. The inferior vena cava (IVC) comprises the connection of the left and right iliac veins and ends on the floor of right atrium, after crossing the diaphragm. Whereas the SVC is an intrathoracic vessel, the IVC is an intraabdominal one, its short intrathoracic part being purely virtual. Both venae cavae provide venous return to the right heart, approx. 25% via the SVC and 75% via IVC [1, 2].

Ultrasonographic examination of the SVC can be performed by a transesophageal approach [3]. To remain open this collapsible vessel requires a distending pressure greater than the critical pressure producing collapse, i.e. its closing pressure. Because lung inflation increases pleural pressure more than right atrial pressure, the distending pressure of the SVC, i.e. right atrial pressure minus pleural pressure, is reduced by lung inflation, and may become insufficient to maintain the vessel open in a hypovolemic patient (Fig. 1). This collapsible vessel can be compared to a “Starling resistor.” The influence of SVC zone conditions on respiratory changes in SVC diameter is illustrated with clinical examples in Fig. 2.

We have thus proposed to use the SVC collapsibility index, calculated as maximal expiratory diameter minus minimal inspiratory diameter, divided by maximal expiratory diameter, as an index of fluid responsiveness in mechanically ventilated patients exhibiting circulatory failure [4]. This requires recording of a long-axis view of the vessel using a multiplane transesophageal probe, by coupling motion mode with two-dimensional mode. Our measurements in a group of 66 patients with septic shock as reported in a previous issue, demonstrated that a SVC collapsibility index higher than 36% predicts a positive response to volume expansion, marked by a significant increase in Doppler cardiac output, with 90% sensitivity and 100% specificity [4]. We also found a bimodal distribution for the SVC collapsibility index: most patients exhibited either a partial or complete collapse of the vessel or the absence of significant change in its diameter during inflation. This confirms our hypothesis that the SVC can be compared to a “Starling resistor” which obeys the all-or-nothing law.

Ultrasonographic examination of the IVC can be performed by a transthoracic, subcostal approach [5, 6]. Mea-
Fig. 2  Left Schematic representation of the superior vena cava (SVC) as a Starling resistor, with an inflow pressure (the upper body mean systemic pressure, MSP), an outflow pressure (the central venous pressure, CVP), and an external pressure (the pleural pressure, Ppl). Right panel Clinical examples illustrating the three zone conditions. Top panel (condition 1) Inflow pressure becomes lower than the pleural pressure during lung inflation, which produces a complete collapse of the whole vessel. This setting is illustrated by ultrasonographic examination of the venae cavae in a hypovolemic patient exhibiting low MSP. Middle panel (condition 2) the outflow pressure is reduced, and lung inflation produces a localized collapse at entry into the right atrium. This setting is illustrated (right) by ultrasonographic examination after clamping of the inferior vena cava during a surgical procedure, a maneuver which suddenly decreases CVP but does not change MSP. Bottom panel (condition 3) Outflow pressure is much greater than the external pressure, and the SVC remains fully open during lung inflation. This setting is illustrated (right) by ultrasonographic examination of the venae cavae after volume expansion.

measurement of IVC diameter in different positions has proven useful in separating normal subjects from patients with elevated right atrial pressure [7]. In their famous study of venous return Guyton et al. [8] observed in dogs that negative right atrial pressure from 0 down to –4 mm Hg increases venous return, but then beyond –4 mm Hg, further increase in the negative pressure causes no more increase in the venous return. Guyton et al. explained this failure by the collapse of the IVC when entering the thoracic cavity, illustrating the inability of a collapsible vessel to transmit a negative pressure. To our knowledge, the first demonstration of the reality of this phenomenon in humans was provided by our group in asthmatic patients [9] (Fig. 3).

Fig. 3 An example of M mode echocardiography of the inferior vena cava (IVC) in spontaneously breathing asthmatic patients. Note the short duration of inspiration, accompanied by a collapse of the vessel, and the increased duration of the expiration (compare Fig. 4, above)

Fig. 4 M mode echocardiography of the inferior vena cava (IVC) in a spontaneously breathing healthy volunteer (above) and in a mechanically ventilated patient (below). Cyclic changes in IVC diameters are opposite, the largest value being observed during expiration in spontaneous breathing, and during inspiration in positive pressure breathing.
Fig. 5  Simultaneous measurement of central venous pressure (CVP) and inferior vena cava diameter (IVC diam) recorded at end-expiration in 108 mechanically ventilated patients. The pressure/diameter relationship for the vessel is characterized by an initial ascending part (arrow 1), where the index of compliance (slope of diameter/pressure curve) does not change, and a final horizontal part (arrow 2), where the index of compliance progressively decreases, reflecting distension.

In a healthy subject breathing spontaneously, cyclic changes in pleural pressure, which are transmitted to the right atrial pressure, produce cyclic changes in venous return, with an inspiratory acceleration, inducing an inspiratory decrease in IVC diameter of approx. 50% (Fig. 4, above) [5]. This cyclic change in vena cava diameter is abolished, however, when the vessel is dilated because, although some inspiratory increase in venous return persists, the vessel actually stays on the horizontal part of its pressure-diameter relationship (Fig. 5). This is the case when cardiac tamponade [10] or severe right ventricular failure is present [6].

In a mechanically ventilated patient, the inspiratory phase produces an increase in pleural pressure, which is transmitted to the right atrial pressure, thus reducing the venous return. As a result respiratory changes in IVC diameter are reversed, compared with those observed during spontaneous breathing, with an inspiratory increase, and an expiratory decrease (Fig. 4, below). However, regarding spontaneous breathing these changes are abolished by vena cava dilatation produced by a high volume status, and/or a high right atrial pressure, the inferior vena cava staying on the horizontal part of its pressure-diameter relationship (Fig. 5). Cyclic respiratory changes in IVC diameter can thus be observed only with a normal or low volume status in a mechanically ventilated patient. In the past, these changes were poorly correlated with atrial pressure during mechanical ventilation [11]. Lack of IVC diameter variation in a mechanically ventilated patient exhibiting circulatory failure rules out the patient’s ability to respond fluid in more than 90% of cases [12].

Feissel et al. [12] first proposed the use of cyclic respiratory changes in IVC diameter to detect fluid responsiveness in a mechanically ventilated patient, and their original findings are reported in a recent issue. Expressing respiratory variability in IVC diameter as maximal inspiratory diameter minus minimal expiratory diameter, divided by the average value of the two diameters, they found that a 12% increase in inferior vena cava diameter during lung inflation allowed discrimination between responders and non-responders to volume loading, with a positive predictive value of 93% and a negative predictive value of 92% [12]. The great merit of this work is to propose a noninvasive parameter to evaluate volume loading. Moreover, this echocardiographic measurement is very easy at the bedside, and requires only minimal experience in echocardiography (Fig. 6). The findings of Feissel et al. are confirmed in an identical study by Barbier et al. [13], which appears in the same issue. It remains to be seen whether this index is still reliable in patients with a significant increase in intra-abdominal pressure, which could limit IVC diameter variations.

Another phenomenon occasionally observed in the IVC during mechanical ventilation is backward flow, which is not caused by tricuspid regurgitation but by cyclic compression of the right atrium by lung inflation. Such a sudden compression boosts blood backward from the right atrium to the IVC and does not concern tricuspid valve competency [14]. This backward flow might explain in part the inaccuracy of the thermodilution method in...
measuring cardiac output in mechanically ventilated patients [14].

In conclusion, ultrasonographic examination of the vena cavae provides new and accurate indices of fluid responsiveness in mechanically ventilated patients exhibiting circulatory failure. In our opinion, a complete evaluation of volume status in these patients should include both IVC and SVC examination.

References