Noninvasive Evaluation of Right Atrial Pressure

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In current practice, right atrial pressure (RAP) is an essential component in the hemodynamic assessment of patients and a requisite for the noninvasive estimation of the pulmonary artery pressures. RAP provides an estimation of intravascular volume, which is a critical component for optimal patient care and management. Increased RAP is associated with adverse outcomes and is independently related to all-cause mortality in patients with cardiovascular disease. Although the gold standard for RAP evaluation is invasive monitoring, various techniques are available for the noninvasive evaluation of RAP. Various echocardiographic methods have been suggested for the evaluation of RAP, consisting of indices obtained from the inferior vena cava, systemic and hepatic veins, tissue Doppler parameters, and right atrial dimensions. Because the noninvasive evaluation of RAP involves indirect measurements, multiple factors must be taken into account to provide the most accurate estimate of RAP. The authors review the data supporting current guidelines, identifying areas of agreement, conflict, limitation, and uncertainty. (J Am Soc Echocardiogr 2013;26:1033-42.)

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The terms "central venous pressure" (CVP) and "right atrial pressure" (RAP) are synonymous as long as no obstruction of the vena cava is present. The gold standard for the evaluation of RAP is invasive monitoring using a central venous catheter. Yet this is an invasive method not without risks[1-3] and is thus not practical for widespread appli-cation. The normal range for RAP is between 1 and 7 mm Hg.[4] Elevated values have prognostic implications for both morbidity and mortality,[5-8] making the accurate assessment of RAP a determining factor in patient assessment, management, and outcomes.[9,10] The noninvasive evaluation of RAP, also a crucial component of the noninvasive estimation of the pulmonary artery pressures, includes the physical examination along with Doppler echocardiographic indices.

PHYSICAL EXAMINATION

Jugular Venous Pressure (JVP)

Sir Thomas Lewis, in 1930, first proposed the determination of a patient's venous pressure during the physical examination.[11] Lewis observed that the top of the jugular veins of normal individuals and the vertical distance from the top of the fluid in the manometer (which was used at that time to measure CVP) always came to lie within 1 to 2 cm of the sternal angle. Currently, examination of JVP is the mainstay of the bedside estimation of CVP.[5,12,13]

Evaluation of CVP using the jugular veins is preferably done using the right-sided jugular vein, which is in direct line with the right atrium, and clinical assessment of CVP on the left may be marginally higher than that on the right.[14] Both the external jugular vein and the internal jugular vein (IJV) can be used for evaluation. Although the external jugular vein is easier to visualize, the IJV is preferred because it does not have valves and is in line with the superior vena cava (SVC) and the right atrium.[15] The patient should be recumbent with the head elevated at 30° to 45°. The level of venous pressure is estimated by identifying the highest point of oscillation of the jugular vein (which occurs during the expiratory phase of respiration). This level is then related to the middle of the right atrium, where venous pressure is, by convention, zero. A reliable substitute is the angle of Louis at the junction of the manubrium and the body of the sternum, located 5 cm above the middle of the right atrium (in approximation for practical purposes). The vertical distance (in centimeters) from the sternal angle to the top of the jugular venous wave represents the JVP (Figure 1); thus, CVP equals JVP + 5 cm.[14] Because the clavicle lies vertically 2 cm above the sternal angle, only a CVP of 7 cm will be observed. JVP estimation may be inaccurate if the jugular vein is constricted or torturous, and it might be difficult to assess in patients with short necks, with prior neck surgery, or with prior catheter placement in the jugular vein.

The Abdominojugular Reflux

The test consists of assessing JVP before, during, and after abdominal compression. It is performed with firm pressure of 20 to 30 mm Hg applied to the midabdomen for 10 to 30 sec.[16] The CVP of normal individuals usually remains unchanged or does not increase by >4 cm for a beat or two (usually for <10 sec), or it may fall slightly.[16,17] If the CVP rises by >4 cm and stays elevated throughout the maneuver, this correlates with elevated RAP.[18] Reports suggest that a positive result is indicative of right ventricular (RV) failure or

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Abbreviations

ASE = American Society of Echocardiography

BSA = Body surface area

CVP = Central venous pressure

DTI = Doppler tissue imaging

IJV = Internal jugular vein

IVC = Inferior vena cava

JVP = Jugular venous pressure

RA = Right atrial

RAP = Right atrial pressure

RV = Right ventricular

SVC = Superior vena cava

3DE = Three-dimensional echocardiography

2DE = Two-dimensional echocardiography

VTI = Velocity-time integral

elevated pulmonary capillary wedge pressure.[16] However, if the patient strains (Valsalva maneuver) during the test, it may cause a false-positive result.

In general, the physical examination for the assessment of RAP has limited accuracy compared with invasive[19-24] or echocardiographic[25,26] studies, most commonly underestimating JVP[20-23] in the setting of elevated CVP.[23] Eisenberg *et al.*[19] found that after a baseline physical examination and assessment of CVP, catheter-derived CVP measurements differed significantly from those obtained on clinical examination in 50% of patients, and in 58% of these, the treatment plan was changed on the basis of the invasive assessment of CVP. Consequently the use of the physical examination might be best for categorizing CVP as either low to normal or elevated.

Doppler Echocardiography

Doppler echocardiography is routinely used to noninvasively estimate RAP. The Doppler echocardiographic methods to estimate RAP include assessment of the inferior vena cava (IVC), SVC, and hepatic vein indices of size and flow; Doppler systemic venous flow; tricuspid valve Doppler inflow; tricuspid valve tissue Doppler; and evaluation of right atrial (RA) dimensions. However, in uncommon circumstances, the flow into the right atrium and consequently the Doppler velocities may be affected by RA compression or distortion, as well as in the setting of RV inflow obstruction due to tumor or tricuspid stenosis. In these situations, assessing the tricuspid gradient can be useful for the estimation of RAP.

IVC Indices

The IVC is a highly compliant vessel, and consequently its size and dynamics vary with changes in CVP and volume. Blood flow from the superior and IVC into the right atrium is biphasic, with the largest forward flow occurring during ventricular systole. In general, there is a reciprocal relationship between pressure and flow; when flow increases (in the IVC as well as the right atrium), pressure decreases and vice versa. During inspiration (which produces negative intrathoracic pressure), vena cava pressure decreases and flow increases[27,28] (Figures 2A–2C). Because the vena cava acts as a capacitance reservoir, phasic increases in forward flow from the cava to the right atrium are accompanied by decreases in vena cava size. The smallest IVC dimension is seen during ventricular systole. At low or normal RAPs, there is systolic predominance in IVC flow, such that the systolic flow is greater than the diastolic flow. As RAP increases, it is transmitted to the IVC, resulting in blunting of the forward systolic flow, reduced IVC collapse with inspiration, and eventually IVC dilatation (Figures 2D–2F). The size and area of the IVC are affected by position

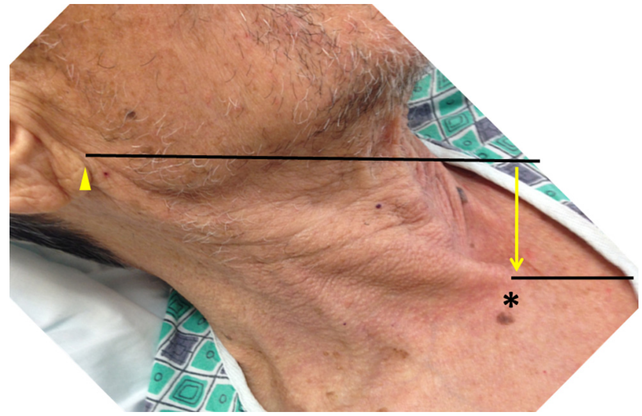

Figure 1 Evaluation of JVP. With the patient lying at about 45°, the highest point of pulsation of the jugular vein is identified (*arrowhead*). This is then related to the angle of Louis, found at the junction of the manubrium with the body of the sternum (*asterisk*). The vertical distance to the top of the jugular venous wave (*arrow*) can be determined and reported, in centimeters, as the JVP.

as well: IVC diameter and area are consistently largest when the patient is evaluated in the right lateral position, intermediate in the supine position, and smallest in the left lateral position.[29] Patient position, therefore, is an important factor to consider when correlating IVC size and shape with hemodynamic variables.

In 1979, Natori *et al.*[30] first described measuring IVC diameter and its change during respiration. Several studies have evaluated the correlation between RAP and different IVC parameters[30-41] (Table 1). However, fewer studies have evaluated the validity of the suggested IVC parameters for the accuracy of the estimation of RAP.[35,39,41] Most, but not all,[29,31] studies have demonstrated good correlations between the IVC collapsibility index ($[IVC_{max} - IVC_{min}]/IVC_{max}$) and RAP ($0.57 < r \leq 0.76$).[30,33-35,39] Although there is a correlation between IVC diameter and RAP ($0.72 < r \leq 0.86$),[29,31,35,40] some reports suggest that the correlation found between IVC diameter and RAP does not permit it to be used for the reliable estimation of RAP,[31,33] because of the variability and overlap between patients with normal RAPs and those with elevated RAPs. It is hypothesized that an increase of RAP beyond a certain level may cause only minimal increases in IVC diameter and the degree of IVC collapsibility with inspiration. Thus, IVC dimensions and collapsibility can be used to detect elevated CVP, but they have limited utility in identifying the magnitude of CVP elevation.

On the basis of several studies,[33,35,37] the American Society of Echocardiography (ASE) in 2005[42] recommended using maximal IVC diameter 1 to 2 cm from the junction of the right atrium and the IVC at end-expiration and the IVC collapsibility index to give an estimate of RAP. This measurement is best obtained from the subcostal view, with the IVC viewed in its long axis (Figure 2). Measurements should be made with the patient lying supine (because the left lateral position may underestimate maximal IVC diameter).[29] However, Brennan *et al.*[41] evaluated the 2005 ASE recommendations for assessing RAP[42] and found that only 43% of patients were correctly classified. On the basis of the results of their study, they suggested a new cutoff range that gives an estimation with range limits of 5–10 mm Hg (Table 2). The newer 2010 ASE guidelines[43] have been revised as noted in Table 2. These parameters yield more accurate results when estimating low or high RAP: IVC diameter < 2.1 cm and collapse > 50% correlates with a normal RAP of 0 to 5 mm Hg.

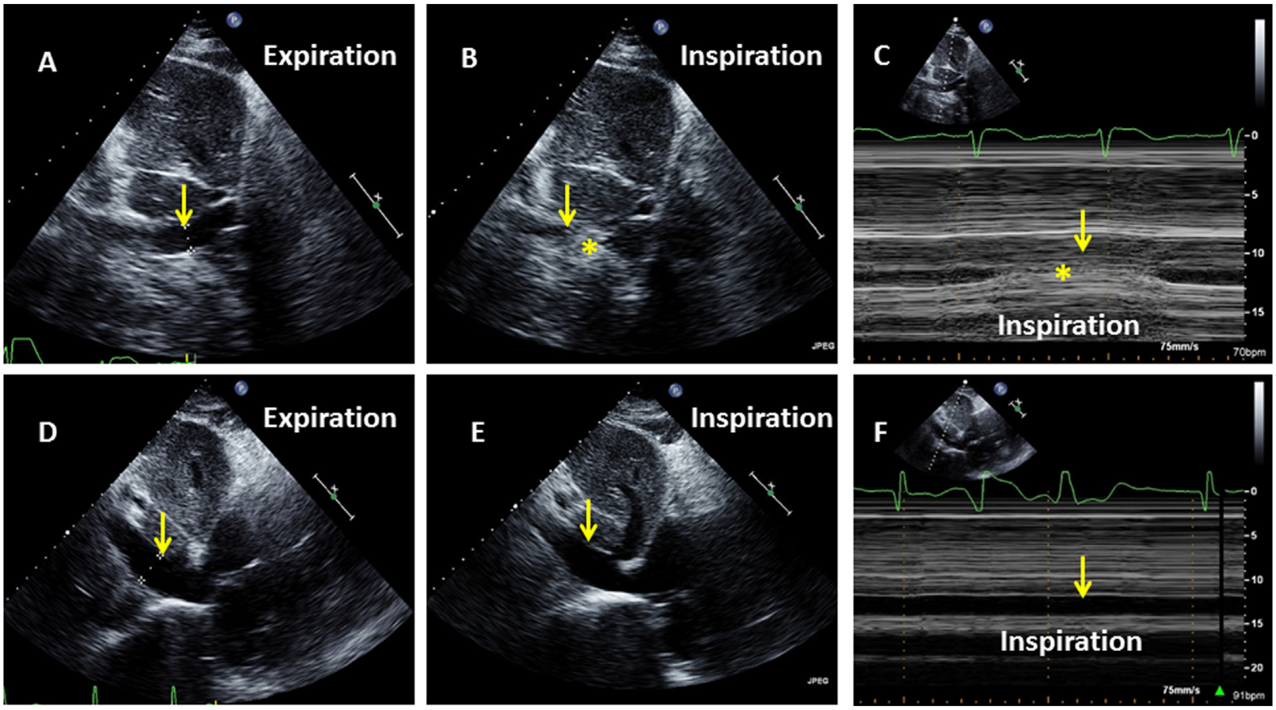

Figure 2 Echocardiographic evaluation of RAP using IVC dimension and collapsibility. Subcostal 2DE during expiration **(A)** and inspiration **(B)** and M-mode echocardiography **(C)** demonstrating good inspiratory collapse (*asterisk*) of the IVC (*arrow*) in a patient with normal RAP and 2DE during expiration **(D)** and inspiration **(E)** and M-mode echocardiography **(F)** demonstrating no inspiratory collapse of the IVC in a patient with elevated RAP.

IVC diameter $<$ 2.1 cm with $<$ 50% collapse and IVC diameter $>$ 2.1 cm with $>$ 50% collapse correspond to an intermediate RAP of 5 to 10 mm Hg. IVC diameter $>$ 2.1 cm with $<$ 50% collapse suggests a high RAP of 15 mm Hg. The guidelines recommend using mid-range values of 3 mm Hg for normal and 8 mm Hg for intermediate RAP. If there is minimal collapse of the IVC (<35%) and/or secondary indices of elevated RAP are present (Table 3, discussed next), the guidelines recommend upgrading to the higher pressure limit (i.e., 5 and 10 mm Hg in the cases of normal and intermediate RAPs, respectively). Patients with low compliance with deep inspirations may have diminished IVC collapse, and a "sniff" maneuver causing a sudden decrease in intrathoracic pressure and by that accentuating the normal inspiratory response might be required to differentiate those with true diminished IVC collapsibility from those with normal collapsibility.

The validity of adjusting the evaluation of IVC size to body surface area (BSA) is controversial. Data correlating IVC size indexed mainly to BSA are inconsistent and limited to only a few studies.[31,33,41,44,45] Mintz *et al.*[31] indexed IVC size measurements to patients' BSAs and found a correlation between IVC index and RAP, but they still concluded that the degree of correlation did not permit the use of the IVC for an accurate estimation of RAP. Moreno *et al.*[33] found no correlation between RAP and IVC parameters, whether indexed to BSA or not ($r < 0.25$), whereas Cheriex *et al.*[44] showed a good correlation ($r = 0.92$) between IVC index and RAP for evaluating hydration status in hemodialysis patients. Kosiak *et al.*[45] developed an alternative index consisting of the IVC/aorta ratio for evaluating patients' volume status in the emergency department. They found in 52 healthy volunteers that IVC and aorta diameters significantly increased after fluid intake and proposed an IVC/aorta index of 1.2 $\pm$ 2 SD for SD = 0.17 as a reference value for the healthy population aged 20 to 30 years; however, this finding needs confirmation. Brennan *et al.*[41] did not find that indexing the IVC to BSA improved accuracy over the standard IVC measurement for RAP estimation, and the ASE 2010 recommendations[43] do not recommend an IVC index related to BSA.

The published data indicate that IVC size and collapsibility indices are appropriate to define RAP as high or low and are not a method for providing a precise numeric value. It should be noted that the IVC can be dilated in individuals with normal RAPs; Table 4 lists the common causes of a dilated IVC in the setting of normal RAP.[37,46] To overcome some of the limitations of RAP estimation through IVC indices, additional Doppler echocardiographic parameters have been evaluated and proposed to better quantify RAP (Table 5), which are further discussed.

Systemic Venous Indices

The central venous flow pattern seen in the vena cava, jugular, and hepatic veins is characterized as seen in Figure 3A by three distinct waveforms when evaluated by Doppler. The first is the systolic wave (Vs), caused by RA relaxation and descent of the tricuspid ring associated with RV systole. The second is the diastolic wave (Vd), which occurs during rapid ventricular filling when the tricuspid valve is open. The third is a positive A wave, which occurs with RA contraction and represents reverse flow. The A wave is small and might not be present in normal individuals.[47] In the majority of normal adults, inspiration increases the magnitude of Vs and Vd, whereas the A wave, if present, decreases in size.[47] At low or normal RAPs, there is systolic predominant venous flow, such that the velocity of Vs is greater than the velocity of Vd (Figure 3A). As demonstrated in Figure 3B, with elevation of RAP, the systolic flow predominance is lost, such that Vs is substantially decreased, and Vs/Vd is <1. The

Table 1 Findings from major studies evaluating the correlation between IVC and RAP

Study	Number of patients	Timing of invasive and noninvasive evaluation	Major findings
Natori *et al.*[30]	14	NA	IVCCI was inversely proportional to CVP when >10 cm H_2O
Mintz *et al.*[31]	111	NA	IVC EDD normalized to BSA was correlated with RAP (r = 0.72)
			IVC EDD > 10 mm/m^2 predicted elevated RAP (predictive accuracy, 94%)
			IVCCI was not correlated with RAP
Moreno *et al.*[33]	175	<24 h	IVC diameter was poorly correlated with RAP ($r \leq 0.4$)
			IVCCI was correlated with RAP (r = 0.71)
Nakao *et al.*[29]	83	<24 h	IVC diameter (r = 0.85) and area (r = 0.89) were correlated with RAP
			IVC diameter > 10 mm predicted elevated RAP (>8 mm Hg) (sensitivity, 84%; specificity, 96%; predictive accuracy, 95%) when measured in the left lateral position
			IVCCI was not correlated well with RAP and was more affected by IVC size than by RAP
Simonson *et al.*[34]	27	Simultaneous	Poor association between maximal IVC diameter and RAP (r = 0.35)
			Good association between minimal IVC diameter and RAP (r = 0.56)
			IVCCI was inversely correlated with RAP ($r = -0.57$)
Kircher *et al.*[35]	83*	<24 h	IVCCI was correlated with RAP (r = 0.75); best sensitivity, specificity, and predictive accuracy for RAP < 10 and > 10 mm Hg when IVCCI was ≥50%
			IVC EDD was correlated with RAP (r = 0.71)
Jue *et al.*[37]	49†	Simultaneous	IVC diameter at expiration was poorly correlated with RAP (r = 0.58)
			No correlation between IVC change and RAP (r = 0.13)
			IVC diameter ≤ 12 mm predicted RAP ≤ 10 mm Hg (sensitivity, 25%; specificity, 100%)
Nagueh *et al.*[39]	35	Simultaneous	IVCCI was correlated with RAP (r = 0.76)
			No good correlation for IVCCI with mechanically ventilated patients (r = 0.4)
Ommen *et al.*[40]	71	Simultaneous	IVC dimension was correlated directly with RAP (r = 0.74)
Brennan *et al.*[41]	102‡	±1 h of invasive evaluation	Five different classifications on the basis of IVC size and collapsibility (see Table 2)
			Using traditional ASE 2005 criteria,[42] only 43% of patients were classified adequately
			Indexing IVC to BSA did not improve accuracy

EDD, End-diastolic dimension; *IVCCI*, IVC collapsibility index; *NA*, not available.
*Patients on mechanical ventilation excluded; retrospective analysis.
†All patients on mechanical ventilation.
‡Thirty percent of patients had undergone cardiac transplantation.

Table 2 Estimation of RAP using IVC parameters

ASE 2010 recommendations[43]		Brennan *et al.*[41]	
IVC diameter (cm) and collapse (%)	RAP range (mean) (mm Hg)	IVC diameter (cm) and collapse (%)	RAP range (mm Hg)
Normal: ≤2.1 and >50	0–5 (3)	≤2.1 and >55	<5
Indeterminate*	5–10 (8)	≤2.1 and 35–55 or >2.1 and >55	0–10
High: >2.1 and <50	10–20 (15)		
		>2.1 and 35–55	10–15
		>2.1 and <35	10–20
		≤2.1 and <35	Undetermined

*In cases which the IVC diameter and collapse do not fit the normal or high criteria. Preferably secondary indices of elevated RAP should be integrated (see Table 3).

higher the RAP, the lower the pressure gradient between these veins and the right atrium, causing diminished forward systolic flow. This blunted gradient is present in patients with restrictive heart disease and elevated right-sided filling pressures.[48-51] Sivaciyan and Ranganathan[48] demonstrated that the jugular venous flow pattern and its variation are closely related to RAP. Although Vs/Vd > 1 was associated with normal RAP, those who demonstrated ratios ≤ 1 had elevated RAPs.

Appleton *et al.*[49] found in healthy individuals that SVC flow was obtainable in all subjects, but hepatic vein flow imaging was adequate in only 40% of patients. A subsequent study by the same group in 14 patients with restrictive ventricular physiology[50] also showed that in patients with elevated RAP, Vs/Vd was ≤1. A study by Ghio *et al.*[51] also confirmed that SVC flow was closely related to RAP, with a normal Doppler pattern identifying patients with normal RAPs, a predominant Vs pattern identifying those with slightly elevated RAPs (≤8 mm Hg) (sensitivity, 69%; specificity, 81%), and a predominant Vd identifying those with elevated RAPs (>8 mm Hg) (sensitivity, 52%; specificity, 95%). Consistent with the initial study by Appleton *et al.*,[50] the success rate reported in obtaining flow velocity curves from the SVC was

Table 3 Indices of elevated RAP

• Dilated IVC with diminished respiratory collapse
• Tricuspid E/e′ ratio > 6
• Diastolic flow predominance in the SVC, jugular vein, or hepatic veins
• Bulging interatrial septum to the left atrium
• Dilated right atrium

Table 4 Causes for IVC enlargement in the presence of normal RAP

• Prominent Eustachian valve
• Athletic training
• Large BSA
• Mechanical ventilation
• Narrowing of the IVC-RA junction
• Web or tissue present in the IVC

100% compared with 76% in the hepatic veins. Patients with severe tricuspid regurgitation were excluded from this study because this may cause altered flow patterns in the systemic veins.

Jugular Venous Flow and JVP

Several investigators have used ultrasonography of the jugular veins to predict CVP. Although the external jugular vein is easier to visualize, its tortuous course, competent valves, and small size might not accurately reflect the transmission of pressure from the right atrium. The right IJV is a large vessel that directly connects to the SVC, but when evaluated clinically, the IJV may be visible only 20% of the time.[23] Lipton[52] observed and described sonographic patterns within the IJV determining venous collapse between the supine, semiupright, and upright positions and concluded that patients can be differentiated as those with low (<10 mm Hg), high (>10 mm Hg), and extremely high (>20 mm Hg) CVPs. Donahue *et al.*[53] demonstrated a good correlation ($r = 0.82$) between IJV end-expiratory diameter and CVP in 34 supine patients. They determined with good accuracy whether a patient had low or high CVP. Simon *et al.*[54] assessed right IJV vascular compliance and concluded that an increase in the cross-sectional area of the right IJV of >17% during the Valsalva maneuver ruled out elevated RAP. Deol *et al.*[55] compared ultrasound collapse pressure with JVP obtained clinically and with CVP in a group of 38 patients, of whom 11 (29%) were mechanically ventilated. Although the ultrasound collapse pressure was capable of accurately measuring JVP, it underestimated CVP, especially when CVP levels were high, concluding that measuring the ultrasound collapse point (which reflects JVP) does not reflect true CVP.

Hepatic Vein Dimensions, Flow Patterns, and Collapsibility

The right and left hepatic veins empty into the IVC at the level of the diaphragm and are best imaged by echocardiography from the subcostal view (Figure 4). The hepatic venous flow pattern is closely related to the central venous flow pattern[47,51,53] (Figure 3B). Like the IVC, as RAP rises, the hepatic veins dilate and collapse less with inspiration. In one study, left hepatic vein diameter correlated well ($r = 0.81$) with the percentage increase in CVP.[56] Imaging was obtained in 90% of patients, yet underestimation occurred when RAP values exceeded 12 mm Hg.

Using Doppler, the hepatic vein systolic filling fraction, which is the ratio of the velocity-time integrals (VTIs) (Vs VTI/[Vs VTI + Vd VTI]), can be obtained. A value < 55% was found to be the most sensitive (86%) and specific (90%) sign of RAP > 8 mm Hg. With higher RAP, there was a decrease in systolic filling fraction.[39] In this study, the best model for the prediction of mean RAP was $21.6 - 24 \times$ hepatic vein systolic filling fraction. This was further validated in the same analysis on a prospective population of 50 patients, with a good correlation ($r = 0.89$), but reproducibility was only moderate ($r = -0.5$) in another study.[40] Importantly, hepatic vein flow velocities have been validated in mechanically ventilated patients (sensitivity, 86%; specificity, 90%),[39] provided that the velocities are averaged over five or more consecutive beats and constitute at least one respiratory cycle.[43]

Thus, the use of systemic and hepatic vein flow parameters can serve as an alternative to IVC parameters in individuals in whom the IVC may appear enlarged despite normal systemic venous pressure, or when subcostal views may not be optimal. It should be remembered that atrial compliance and relaxation, severe tricuspid regurgitation, and tricuspid annular descent affect flow patterns and make them less reliable. In addition, the presence of atrial fibrillation or past cardiac surgery can cause the hepatic vein systolic flow to be diminished regardless of RAP.[40,57]

Tricuspid Doppler and Doppler Tissue Imaging (DTI)

Maximum velocities of the tricuspid E and A waves are significantly higher during inspiration than during expiration when measured from the atrial side of the tricuspid valve. However, when measured from the ventricular side in the apical four-chamber view, respiration affects only the maximum velocity of the E wave, without greatly affecting the E/A ratio.[58] In contrast to mitral flow, on which age has an important effect, Pye *et al.*[59] found that there was no significant correlation between any tricuspid flow parameter and age, although two smaller studies did find weak correlations.[60,61] Scapellato *et al.*[62] used tricuspid Doppler to estimate RAP, finding a significant correlation with RAP ($r = 0.98$). The highest positive correlation was found between RV filling acceleration rate and RAP. An acceleration rate > 560 cm/sec^2 predicted RAP > 5 mm Hg with sensitivity of 100% and specificity of 99%. On the basis of the data in this study, the authors developed a detailed formula for the calculation of RAP: $RAP = -1.263 + 0.01116 \times$ acceleration rate, and they confirmed this finding in another study.[63]

Tissue Doppler allows the recording of myocardial and annular velocities. The maximal early filling velocity through the tricuspid valve during diastole (E wave) increases with increasing RAP. Evaluation using DTI can measure the velocity of tissue relaxation of the lateral tricuspid annulus in diastole (e′ wave; Figure 5). Nagueh *et al.*[64] found a strong relation between RAP and the E/e′ ratio ($r = 0.75$). They found that a high E velocity combined with a low e′, resulting in an E/e′ ratio > 6, signals an RAP > 10 mm Hg (Figure 5B). This correlation was also accurate in patients with mechanical ventilation. Sade *et al.*[65] reproduced these findings, showing a good correlation between E/e′ ratio and RAP ($r = 0.7$), giving the formulation of $RAP = 1.62\ E/e' + 2.13$, which was also applicable and a good indicator of RAP in mechanically ventilated patients. In 12 patients who underwent several simultaneous measurements, this ratio was also a valid indicator of RAP change, with an increase of >2 in the E/e′ ratio associated with an increase of ≥5 mm Hg in RAP. In patients who have undergone cardiac surgery, tricuspid E/e′ ratio might not be as accurate for the estimation of RAP. A subsequent study by Patel

Table 5 Methods for evaluation of RAP besides IVC evaluation

Method	Number of patients	Timing of invasive and noninvasive evaluation	Major findings
Systemic veins			
Jugular vein pattern and collapsibility	34[53]	Simultaneous	CVP categorized as high (≥10 mm Hg) or low (<10 mm Hg) was correlated with IJV diameter (r = 0.82)*
	67[54]	Within 1 h	An increase of >17% IJV CSA during Valsalva maneuver ruled out elevated RAP (>12 mm Hg) (sensitivity, 90%; specificity, 74%; NPV, 90%; PPV, 60%)
	38[†,55]	Simultaneous	Ultrasound compression was correlated with JVP but underestimated CVP (r = 0.81–0.91 for JVP, r = 0.62–0.5 for CVP)
Jugular venous flow[‡]	82[48]	Within 1 d	Vs > Vd was associated with normal RAP
			Vs ≤ Vd was associated with elevated RAP
			Single diastolic flow was associated with elevated RAP
SVC flow	40[49]	NA	Defined the normal pattern of SVC flow (Vs > Vd)
SVC flow[§]	120[51]	Within 1 h	2 ≥ Vs/Vd ≥ 1 (normal) = RAP ≤ 5 mm Hg (sensitivity, 86%; specificity, 78%)
			Vs/Vd >2 (predominant systolic) = RAP ≤ 8 mm Hg (sensitivity, 69%; specificity, 81%)
			Vs/Vd < 1 (predominant diastolic) = RAP > 8 mm Hg (sensitivity, 52%; specificity, 95%)
Hepatic veins			
Hepatic venous flow	40[49]	Within 12 hours	Defined normal hepatic venous flow (Vs > Vd)
	14[50]		Vs/Vd ≤ 1 was associated with elevated RAP
Left hepatic vein diameter[‖]	32[56]	Simultaneous	Left hepatic vein diameter was correlated well with RAP (r = 0.81)
HVFF	35[39]	Simultaneous	>55% suggested RAP > 8 mm Hg (sensitivity, 86%; specificity, 90%)[¶]
	50[39]	Simultaneous	RAP = 21–24 × HVFF (r = 0.89)
	71[40]	Simultaneous	Modest correlation with RAP (r = −0.5)[#]
DTI			
Acceleration rate of RV early filling[62]	77	Simultaneous	Acceleration rate was a predictor of RAP (r = 0.98)
			Acceleration rate > 560 cm/sec^2 predicted RAP > 5 mm Hg (sensitivity, 100%; specificity, 99%)
Tricuspid E/e′ ratio	62[64]	Simultaneous	E/e′ ratio > 6 suggested RAP > 10 mm Hg (r = 0.75)**
	89[65]	Simultaneous	RAP = 1.62 E/e′ +2.13 (r = 0.7)[††]
	40[66]	Immediately before obtaining invasive data	No good correlation (r = 0.09)[‡‡]
Tricuspid E/A ratio	40[‡‡,66]	Immediately before obtaining invasive data	Good correlation with RAP (r = 0.57)[§§]
RV rIVRT[57]	45	Within 45 min	RV rIVRT< 59 ms was a predictor of RAP > 8 mm Hg (sensitivity, 80%; specificity, 88%; AUC, 0.994)
RA dimensions			
3D RAVi[66]	80[‡‡]	Immediately before obtaining invasive data	3D RAVi ≥ 35 mL/m^2 + IVC ≥ 2 cm was accurate for identifying RAP > 10 mm Hg (sensitivity, 89%; specificity, 92%; accuracy, 88%)[‖‖]

AUC, Area under the curve; *HVFF*, hepatic vein systolic filling fraction; *NA*, not applicable; *NPV*, negative predictive value; *PPV*, positive predictive value; *RAVi*, RA volume index; *rIVRT*, regional isovolumic relaxation time.
*Not applicable to mechanically ventilated patients.
†Twenty-nine percent of patients were mechanically ventilated.
‡Not applicable to patients who had undergone cardiac surgery and patients with severe left ventricular volume overload.
§Only patients with ejection fractions < 35% were included. Patients with severe tricuspid regurgitation were excluded.
‖Imaging was obtainable in 90% of patients. Underestimation occurred when RAP was >12 mm Hg.
¶Also validated for mechanically ventilated patients. Patients with severe tricuspid regurgitation were excluded.
#Patients with mechanical ventilation or severe tricuspid regurgitation were not included.
**Also validated for mechanically ventilated patients.
††Not applicable to patients who had undergone cardiac surgery.
‡‡Only patients with acute decompensated heart failure were included.
§§Inflow parameters could be determined in only 43% of patients.
‖‖Analysis was not feasible in 18% of patients.

et al.[66] in patients with heart failure did not find a good correlation between RAP and E/e′ ratio but did find a modest but significant correlation with tricuspid E/A ratio (r = 0.57, P = .02).

RV regional isovolumic relaxation time refers to DTI of the lateral tricuspid annulus in the apical four-chamber view and is measured as the time period between the end of systolic annular motion and the

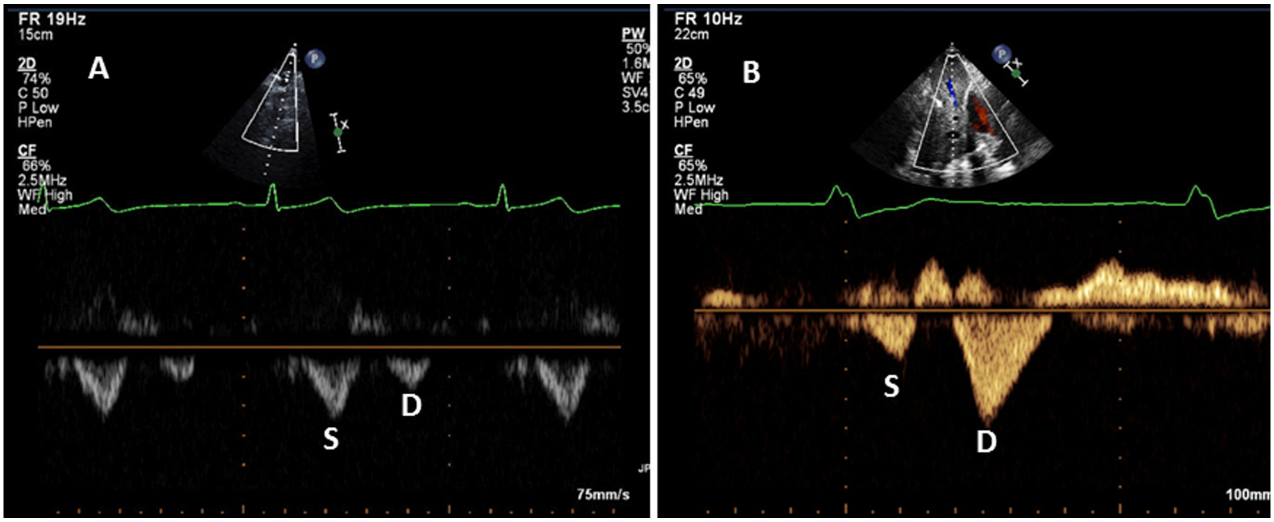

Figure 3 Doppler imaging of flow in systemic veins. Pulsed-wave Doppler echocardiography showing **(A)** flow velocity in the SVC of a normal adult with normal biphasic pattern of forward flow, with systolic flow velocity (S) greater than diastolic flow velocity (D) and S/D > 1 and **(B)** flow velocity in the hepatic vein in a patient with elevated RAP showing diminished S, increased D, and S/D < 1.

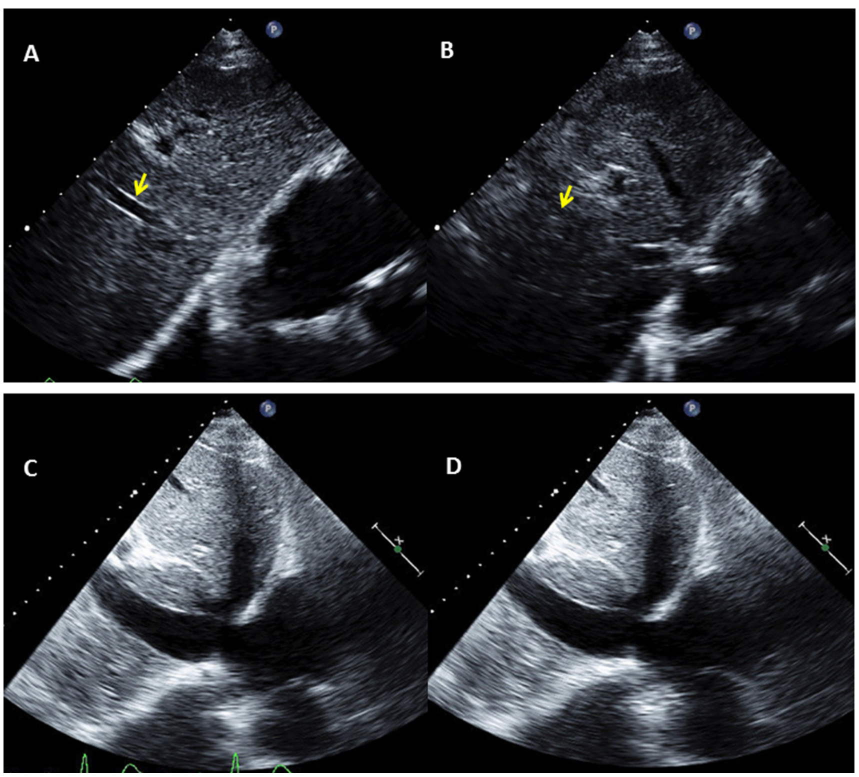

Figure 4 Evaluation of hepatic vein collapsibility. Subcostal view during **(A)** expiration and **(B)** inspiration showing inspiratory collapse of the hepatic vein (*arrow*) in a patient with normal RAP and no collapse of the hepatic vein during **(C)** expiration and **(D)** inspiration in a patient with elevated RAP.

onset of the e′ wave. Using this index, it was found that RV regional isovolumic relaxation time < 59 msec corresponds to RAP > 8 mm Hg (sensitivity, 80%; specificity, 88%).[57]

Doppler and DTI can provide an additional alternative for RAP evaluation when subcostal views cannot be obtained and there is inability to apply IVC and hepatic indices, as well as be used to corroborate the prediction of RAP using hepatic vein systolic filling fraction in patients on mechanical ventilation when the IVC collapsibility index is inaccurate.[64]

RA Dimensions

Elevated RAP can lead to enlargement of the right atrium and increases in RA dimensions. Using two-dimensional echocardiography

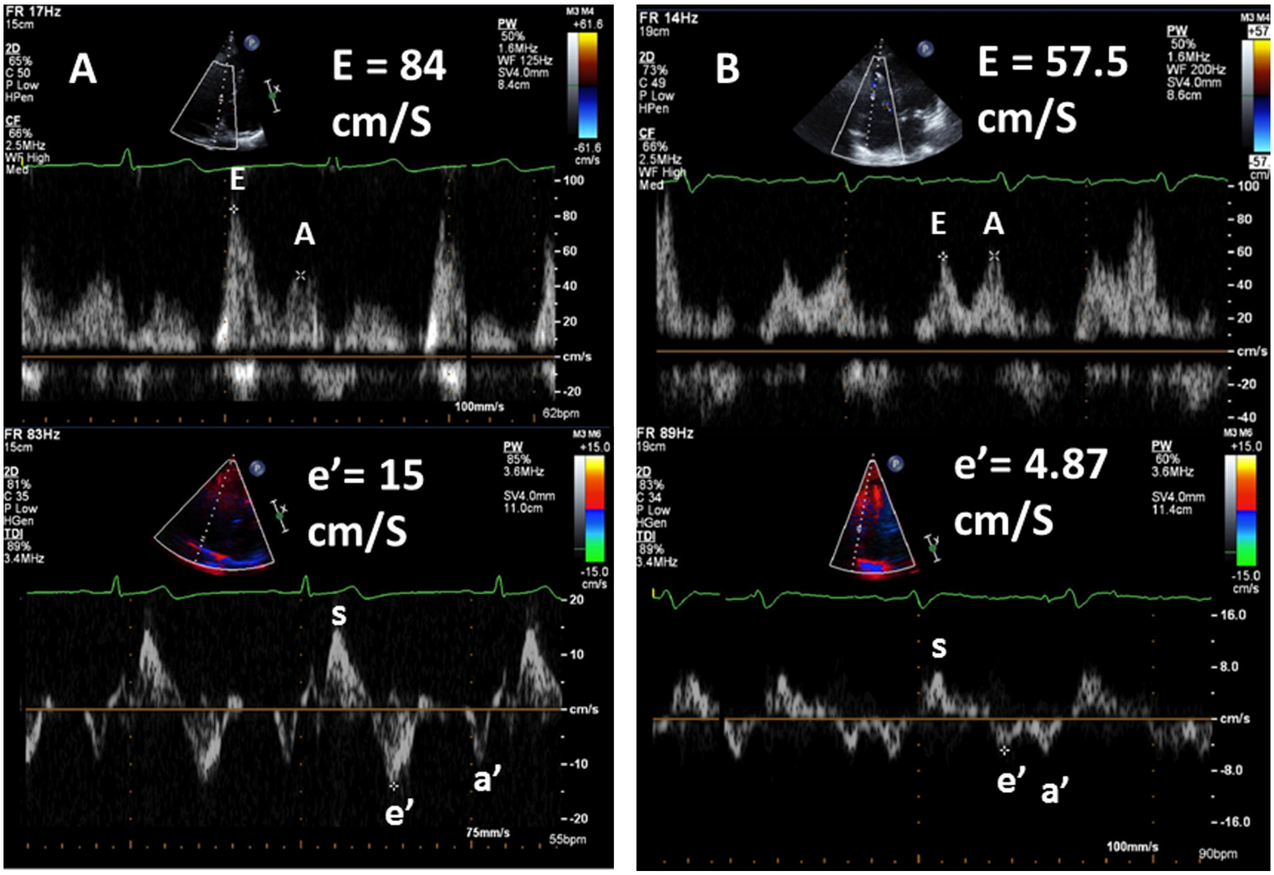

Figure 5 Doppler evaluation of tricuspid inflow. **(A)** (*Top*) Tricuspid inflow velocity Doppler recording (E = 84 cm/sec). (*Bottom*) Tricuspid annular velocity (e′) using DTI is 15 cm/sec in a patient with normal RAP. The E/e′ ratio is <6. **(B)** (*Top*) Tricuspid inflow velocity Doppler recording (E = 57.5 cm/sec). (*Bottom*) Tricuspid annular velocity (e′) is 4.87 cm/sec in a patient with elevated RAP. The E/e′ ratio is >6, which suggests an elevated RAP of >10 mm Hg.

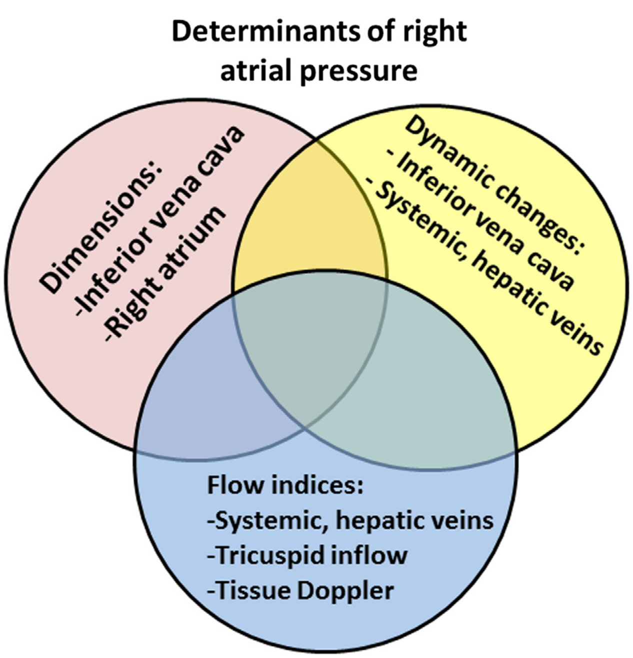

Figure 6 Schematic presentation of the interplay among the various parameters available for noninvasive evaluation and determination of RAP.

(2DE), RA size and volume can be assessed from many views but are most commonly determined using the apical four-chamber view.[43] Three-dimensional echocardiography (3DE) provides tomographic imaging of cardiac chambers and has the potential to be a more accurate modality for atrial volume quantification than 2DE and has been validated using magnetic resonance imaging.[67,68]

Although qualitative and two-dimensional echocardiographic measures of the right atrium (both size and volume) do not significantly correlate with RAP,[39] Patel *et al.*[66] hypothesized that 3DE might aid in the identification of patients with heart failure with elevated RAPs. In their study, assessing the utility of 3DE for the evaluation of elevated RAP, RA size by 2DE was only modestly correlated with RA volume by 3DE ($r = 0.58$, $P < .001$). However, right atrium volume by 3DE was found to correlate with RAP ($r = 0.51$, $P < .001$). Compared with the traditional assessment using an IVC diameter ≥ 2 cm and decreased respiratory collapse of <40%, 3DE-measured maximal RA volume ≥ 35 mL/m^2 combined with IVC diameter ≥ 2 cm resulted in improved sensitivity for the identification of RAP >10 mm Hg in the study validation group (sensitivity, 86%; specificity, 92%; accuracy, 88%).[66]

CONCLUSIONS

Currently, there is no single ideal parameter for noninvasive RAP estimation. Using the most recent ASE criteria[43] based on IVC parameters, it is most appropriate to categorize RAP into low (0–5 mm Hg), normal (6–10 mm Hg), or elevated (11–20 mm Hg). We believe additional information from some of the methods discussed in this review should aid in the better quantification of RAP. As noninvasive evaluation of RAP involves indirect measurements, multiple factors must be taken into account to provide an accurate estimate of RAP. It is the interplay among these echocardiographic findings measuring dynamic changes, flow, and dimensions that yields the best noninvasive assessment of RAP (Figure 6). In our view, the best approach would be to implement a scoring system using the various available indices of these parameters. Incorporating these variables into routine daily echocardiographic evaluation requires only additional minimal effort and has the potential to improve the hemodynamic profiling of patients, which can lead to better patient management and outcomes.

REFERENCES

1. Mansfield PF, Hohn DC, Fornage BD, Gregurich MA, Ota DM. Complications and failures of subclavian-vein catheterization. N Engl J Med 1994; 331:1735-8.
2. Merrer J, De Jonghe B, Golliot F, Lefrant JY, Raffy B, Barre E, et al. Complications of femoral and subclavian venous catheterization in critically ill patients: a randomized controlled trial. JAMA 2001;286:700-7.
3. Lorente L, Henry C, Jimenez A, Mora ML. Central venous catheter-related infection in a prospective and observational study of 2,595 catheters. Crit Care 2005;32:R631-5.
4. Davidson CJ, Bonow RO. Cardiac catheterization. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's heart disease: a textbook of cardiovascular medicine. 9th ed. New York: Elsevier; 2012. pp. 383-405.
5. Drazner MH, Rame JE, Stevenson LW, Dries DL. Prognostic importance of elevated jugular venous pressure and a third heart sound in patients with heart failure. N Engl J Med 2001;345:574-81.
6. Unverferth DV, Magorien RD, Moeschberger ML, Baker PB, Fetters JK, Leier CV. Factors influencing the one-year mortality of dilated cardiomyopathy. Am J Cardiol 1984;54:147-52.
7. Morely D, Brozena SC. Assessing risk by hemodynamic profile in patients awaiting cardiac transplantation. Am J Cardiol 1994;73:379-83.
8. Damman K, van Deursen VM, Navis G, Voors AA, van Veldhuisen DJ, Hillege HL. Increased central venous pressure is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. J Am Coll Cardiol 2009;53:582-8.
9. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med 2008;36:296-327.
10. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002;12:2000-8.
11. Lewis T. Remarks on early signs of cardiac failure of the congestive type. Br Med J 1930;1:849-52.
12. Stevenson LW, Tillisch JH, Hamilton M, Luu M, Chelimsky-Fallick C, Moriguchi J, et al. Importance of hemodynamic response to therapy in predicting survival with ejection fraction less than or equal to 20% secondary to ischemic or nonischemic dilated cardiomyopathy. Am J Cardiol 1990; 66:1348-54.
13. Steimle AE, Stevenson LW, Chelimsky-Fallick C, Fonarow GC, Hamilton MA, Moriguchi JD, et al. Sustained hemodynamic efficacy of therapy tailored to reduce filling pressures in survivors with advanced heart failure. Circulation 1997;96:1165-72.
14. Cook DJ, Simel DL. The rational clinical examination. Does this patient have abnormal central venous pressure? JAMA 1996;275:630-4.
15. Fang JC, O'Gara PT. The history and physical examination: an evidence-based approach. In: Bonow RO, Mann DL, Zippes DP, Libby P, eds. Braunwald's heart disease: a textbook of cardiovascular medicine. 9th ed. New York: Elsevier; 2012. pp. 107-25.
16. Ewy GA. The abdominojugular test: technique and hemodynamic correlates. Ann Intern Med 1988;109:456-60.
17. Matthews MB, Hampson J. Heaptojugular reflux. Lancet 1958;1:873-6.
18. Hultgren HN. The effect of increased venous return on the venous pressure in patients with congestive heart failure. Am Heart J 1950;39: 592-603.
19. Eisenberg PR, Jaffe AS, Schuster DP. Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients. Crit Care Med 1984;12:549-53.
20. Connors AF Jr., McCaffree DR, Gray BA. Evaluation of right-heart catheterization in the critically ill patient without acute myocardial infarction. N Engl J Med 1983;308:263-7.
21. Cook DJ. Clinical assessment of central venous pressure in the critically ill. Am J Med Sci 1990;299:175-8.
22. Stevenson LW. Are hemodynamic goals viable in tailoring heart failure therapy? Hemodynamic goals are relevant. Circulation 2006;113:1020-7.
23. Davidson R, Cannon R. Estimation of central venous pressure by examination of jugular veins. Am Heart J 1974;87:279-82.
24. Stein JH, Neumann A, Marcus RH. Comparison of estimates of right atrial pressure by physical examination and echocardiography in patients with congestive heart failure and reasons for discrepancies. Am J Cardiol 1997;80:1615-8.
25. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. Circulation 1984;70:657-62.
26. Brennan JM, Blair JE, Goonewardena S, Ronan A, Shad D, Vasalwala S, et al. A comparison by medicine residents of physical examination versus hand-carried ultrasound for estimation of right atrial pressure. Am J Cardiol 2007;99:1614-6.
27. Gabe IT, Gault JH, Ross J Jr., Mason DT, Mills CJ, Schillingford JP, et al. Measurement of instantaneous blood flow velocity and pressure in conscious man with a catheter-tip velocity probe. Circulation 1969;40: 603-14.
28. Wexler L, Bergel DH, Gabe IT, Makin GS, Mills CJ. Velocity of blood flow in normal human venae cavae. Circ Res 1968;23:349-59.
29. Nakao S, Come PC, McKay RG, Ransil BJ. Effects of positional changes on inferior vena caval size and dynamics and correlations with right-sided cardiac pressure. Am J Cardiol 1987;59:125-32.
30. Natori H, Tamaki S, Kira S. Ultrasonographic evaluation of ventilatory effect on inferior vena caval configuration. Am Rev Respir Dis 1979; 120:421-7.
31. Mintz GS, Kotler MN, Parry WR, Iskandrian AS, Kane SA. Real-time inferior vena caval ultrasonography: normal and abnormal findings and its use in assessing right-heart function. Circulation 1981;64:1018-25.
32. Rein AJ, Lewis N, Forst L, Gotsman MS, Lewis BS. Echocardiography of the inferior vena cava in healthy subjects and in patients with cardiac disease. Isr J Med Sci 1982;18:581-5.
33. Moreno FL, Hagan AD, Holmen JR, Pryor TA, Strickland RD, Castle CH. Evaluation of size and dynamics of the inferior vena cava as an index of right-sided cardiac function. Am J Cardiol 1984;53:579-85.
34. Simonson JS, Schiller NB. Sonospirometry: a new method for noninvasive estimation of mean right atrial pressure based on two-dimensional echocardiographic measurements of the inferior vena cava during measured inspiration. J Am Coll Cardiol 1988;11:557-64.
35. Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. Am J Cardiol 1990;66:493-6.
36. Goldberger JJ, Himelman RB, Wolfe CL, Schiller NB. Right ventricular infarction: recognition and assessment of its hemodynamic significance by two-dimensional echocardiography. J Am Soc Echocardiogr 1991;4: 140-6.
37. Jue J, Chung W, Schiller NB. Does inferior vena cava size predict right atrial pressures in patients receiving mechanical ventilation? J Am Soc Echocardiogr 1992;5:613-9.

38. Pepi M, Tamborini G, Galli C, Barbier P, Doria E, Berti M, et al. A new formula for echo-Doppler estimation of right ventricular systolic pressure. J Am Soc Echocardiogr 1994;7:20-6.
39. Nagueh SF, Kopelen HA, Zoghbi WA. Relation of mean right atrial pressure to echocardiographic and Doppler parameters of right atrial and right ventricular function. Circulation 1996;93:1160-9.
40. Ommen SR, Nishimura RA, Hurrell DG, Klarich KW. Assessment of right atrial pressure with 2-dimensional and Doppler echocardiography: a simultaneous catheterization and echocardiography study. Mayo Clin Proc 2000;75:24-9.
41. Brennan JM, Blair JE, Goonewardena S, Ronan A, Shad D, Vasslwala S, et al. Reappraisal of the use of inferior vena cava for estimating right atrial pressure. J Am Soc Echocardiogr 2007;20:857-61.
42. Lang RM, Bierig M, Devereaux RB, FlachskampF FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's guidelines and standards committee and the chamber quantification writing group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18: 1440-63.
43. Rudski LG, Lai WW, Afialo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23: 685-713.
44. Cheriex EC, Leunissen KM, Janssen JH, Mooy JM, van Hooff JP. Echography of the inferior vena cava is a simple and reliable tool for estimation of "dry weight" in haemodialysis patients. Nephrol Dial Transplant 1989;4: 563-8.
45. Kosiak W, Swieton D, Piskunowicz M. Sonographic inferior vena cava/ aorta diameter index, a new approach to the body fluid status assessment in children and young adults in emergency ultrasound—preliminary study. Am J Emerg Med 2008;26:320-5.
46. Goldhammer E, Mesnik N, Abinader EG, Sagiv M. Dilated inferior vena cava: a common echocardiographic finding in health trained elite athletes. J Am Soc Echocardiogr 1999;12:988-93.
47. Reynolds T, Appleton CP. Doppler flow velocity patterns of the superior vena cava, inferior vena cava, hepatic vein, coronary sinus, and atrial septal defect: a guide for the echocardiographer. J Am Soc Echocardiogr 1991;4: 503-12.
48. Sivaciyan V, Ranganathan N. Transcutaneous Doppler jugular venous flow velocity recording. Circulation 1978;57:930-9.
49. Appleton CP, Hatle LK, Popp RL. Superior vena cava and hepatic vein Doppler echocardiography in healthy adults. J Am Coll Cardiol 1987; 10:1032-9.
50. Appleton CP, Hatle LK, Popp RL. Demonstration of restrictive ventricular physiology by Doppler echocardiography. J Am Coll Cardiol 1988;11: 757-68.
51. Ghio S, Recusani F, Sebastiani R, Klersy C, Raineri C, Campana C, et al. Doppler velocimetry in superior vena cava provides useful information on the right circulatory function in patients with congestive heart failure. Echocardiography 2001;18:469-77.
52. Lipton B. Estimation of central venous pressure by ultrasound of the internal jugular vein. Am J Emerg Med 2000;18:432-4.
53. Donahue SP, Wood JP, Patel BM, Quinn JV. Correlation of sonographic measurements of the internal jugular vein with central venous pressure. Am J Emerg Med 2009;27:851-5.
54. Simon MA, Kliner DE, Girod JP, Moguillansky D, Villanueva FA, Pacella JJ. Detection of elevated right atrial pressure using a simple bedside ultrasound measure. Am Heart J 2010;159:421-7.
55. Deol GR, Collett N, Ashby A, Schmidt GA. Ultrasound accurately reflects the jugular venous examination but underestimates central venous pressure. Chest 2011;139:95-100.
56. Luca L, Mario P, Giansiro B, Maurizio F, Antonio M, Carlo M. Non invasive estimation of mean right atrial pressure utilizing the 2D-echo transverse diameter of the left hepatic vein. Int J Card Imaging 1992;8: 191-5.
57. Abbas A, Lester S, Moreno FC, Srivathsan K, Fortuin D, Appleton C. Noninvasive assessment of right atrial pressure using Doppler tissue imaging. J Am Soc Echocardiogr 2004;17:1155-60.
58. Uiterwaal C, Van Dam I, De Boo T, Van Keulen P, Folgering H, Hopman J, et al. The effect of respiration on diastolic blood flow velocities in the human heart. Eur Heart J 1989;10:108-12.
59. Pye MP, Pringle SD, Cobbe SM. Reference values and reproducibility of Doppler echocardiography in the assessment of the tricuspid valve and right ventricular diastolic function in normal subjects. Am J Cardiol 1991;67:269-73.
60. Iwase M, Nagata K, Izawa H, Yokota M, Kamihara S, Inagaski H, et al. Age related changes in left and right ventricular filling velocity profiles and their relationship in normal subjects. Am Heart J 1993;126:419-26.
61. Berman GO, Reichek N, Brownson D, Douglas PS. Effects of sample volume location, imaging view, heart rate and age on tricuspid velocimetry in normal subjects. Am J Cardiol 1990;65:1026-30.
62. Scapellato F, Eleuteri E, Temporelli PL, Imparato A, Corra U, Giannuzzi P. Doppler-derived acceleration rate of right ventricular early filling as a measurement of right atrial pressure in chronic heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 1998;81: 513-5.
63. Temporelli PL, Scapellato F, Eleuteri E, Imparato A, Giannuzzi P. Doppler echocardiography in advanced systolic heart failure: a noninvasive alternative to Swan-Ganz catheter. Circ Heart Fail 2010;3: 387-94.
64. Nagueh MF, Kopelen HA, Zoghbi WA, Quinones MA, Nagueh SF. Estimation of mean right atrial pressure using tissue Doppler imaging. Am J Cardiol 1999;84:1448-51.
65. Sade LE, Gulmez O, Eroglu S, Sezgin A, Muderrisoglu H. Noninvasive estimation of right ventricular filling pressure by ratio of early tricuspid inflow to annular diastolic velocity in patients with and without recent cardiac surgery. J Am Soc Echocardiogr 2007;20:982-8.
66. Patel AR, Alsheikh-Ali AA, Mukherjee J, Evangelista A, Quraini D, Ordway LJ, et al. 3D echocardiography to evaluate right atrial pressure in acutely decompensated heart failure correlation with invasive hemodynamics. J Am Coll Cardiol Img 2011;4:938-45.
67. Keller AM, Gopal AS, King DL. Left and right atrial volume by freehand three-dimensional echocardiography: in vivo validation using magnetic resonance imaging. Eur J Echocardiogr 2000;1:55-65.
68. Poutanen T, Ikonen A, Vainio P, Jokinen E, Tikanoja T. Left atrial volume assessed by transthoracic three dimensional echocardiography and magnetic resonance imaging: dynamic changes during the heart cycle in children. Heart 2000;83:537-42.