Non-invasive estimation of right atrial pressure by combined Doppler echocardiographic measurements of the inferior vena cava in patients with congestive heart failure

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Key words:
Congestive heart failure; Doppler echocardiography; Right atrial pressure.

Background. **In patients with congestive heart failure, evaluation of right atrial pressure (RAP) provides useful therapeutic, functional and prognostic information. The aim of this study was to investigate whether a combination of inferior vena cava variables measured by Doppler echocardiography could provide a reliable non-invasive estimate of RAP.**

Methods. **One hundred consecutive patients with severe congestive heart failure (ejection fraction 24 ± 6%) due to dilated cardiomyopathy were evaluated by simultaneous Doppler echocardiography and hemodynamic studies. RAP, end-expiratory (IVCDmax) and end-inspiratory (IVCDmin) diameters of the inferior vena cava, its collapse index [CIIVC = (IVCDmax - IVCDmin/IVCDmax)*100] and systolic fraction of forward inferior vena cava flow were measured and correlated by both single and multilinear regression analysis. The accuracy of generated equations was tested in a separate testing group of 61 patients at baseline and a subgroup of 20 patients after loading manipulations, prospectively studied in the same methodological setting.**

Results. **All Doppler echocardiographic variables were correlated with RAP. The IVCDmin showed the strongest correlation ($r = 0.84$, $p < 0.0001$). Stepwise regression analysis identified two equations for predicting RAP: 1) RAP = (6.4*IVCDmin + 0.04*CIIVC - 2) ($r = 0.82$, $p < 0.0001$, SEE 1.7 mmHg) in all patients, and 2) RAP = (4.9*IVCDmin + 0.01*CIIVC - 0.2) ($r = 0.92$, $p < 0.0001$, SEE 1.2 mmHg) in patients without tricuspid regurgitation. In the testing group estimated and measured RAP was strongly correlated at baseline ($r = 0.95$, SEE 1.3 mmHg, $p < 0.00001$) and after loading manipulations ($r = 0.96$, SEE 1.2 mmHg, $p < 0.00001$). The agreement between invasive and non-invasive measurements of RAP in identifying patients with normal (≤ 5 mmHg), moderately increased (< 5 RAP < 10 mmHg) and markedly increased (≥ 10 mmHg) RAP was 81 or 93% using equation 1 or 2, respectively.**

Conclusions. **Our results provide evidence that in patients with congestive heart failure indices derived from Doppler measurements of the inferior vena cava can be used to produce an accurate, strong and non-invasive estimate of RAP. This is another example of the usefulness of Doppler echocardiography in evaluating hemodynamic profile and its changes in patients with congestive heart failure. Echocardiographic assessment of the inferior vena cava should be included in the evaluation of patients with congestive heart failure.**

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Introduction

In patients with congestive heart failure evaluation of right atrial pressure (RAP) is an important clinical objective because this pressure reflects right cardiac hemodynamics and central blood volume[1]. RAP can be useful as a prognostic marker, and to guide "unloading therapy"[2-4]. Furthermore, determination of RAP can improve the accuracy of non-invasive assessment of pulmonary artery systolic pressure, which would improve diagnosis of pulmonary hypertension and clinical decision-making in patients evaluated for heart transplantation[5-8].

The inferior vena cava is a highly compliant vessel; its size and dynamics reflect the changes in RAP and central blood volume. The size and shape of this vessel change during physiological respiration. The entity of these variations is related not only to hemodynamic changes, but also to volume overload duration[9-11]. The dimension and measurements of the inferior vena cava are easy to study by M-mode and two-dimensional echocardiography[12].

However, limited data are available to define the relationship between vena cava dimension, its variation and RAP in patients with congestive heart failure.

We performed a prospective study to evaluate whether measurements of the diameter of the inferior vena cava and changes in that diameter during respiration could provide an accurate assessment of mean RAP.

Methods

Study population. *Learning group.* One hundred nine consecutive patients with severe congestive heart failure due to ischemic or idiopathic dilated cardiomyopathy, admitted to our Heart Failure Unit for evaluation and treatment of advanced heart failure, were considered.

The clinical diagnosis of congestive heart failure was confirmed by a documented history of cardiac decompensation requiring hospitalization, by the presence of symptoms (orthopnea, dyspnea, asthenia, palpitations) and by physical examination (S3, rales, elevated jugular venous pressure, hepato-jugular reflux, ascites, edema). Dilated cardiomyopathy was defined by two-dimensional echocardiographic demonstration of a dilated left ventricle (left ventricular end-diastolic volume index > 78 ml/m^2) with severe left ventricular systolic dysfunction (ejection fraction < 35%).

Patients with technically inadequate Doppler echocardiographic recordings (2 patients), and those with atrial fibrillation (7 patients) and/or prosthetic valves were excluded. Clinical, hemodynamic and Doppler echocardiographic characteristics of the patients are reported in table I.

Testing group. A prospectively enrolled group of 61 patients was used to evaluate the reliability of the equations to estimate RAP generated in the learning group. Right heart catheterization and the Doppler echocardiographic study were performed simultaneously in these patients at baseline and in a subgroup of 20 patients after left ventricular loading manipulation by nitroprusside infusion (10 patients) or passive leg-lifting (10 patients). These patients had similar characteristics to those of the learning group (Table I).

Table I. Clinical, Doppler echocardiographic and hemodynamic characteristics at baseline.

	Learning group (n=100)	Testing group (n=61)	p
Age (years)	54 ± 8	53 ± 9	NS
Etiology (ischemic/non-ischemic)	42/58	31/30	NS
Heart rate (b/min)	78 ± 15	78 ± 14	NS
Systolic blood pressure (mmHg)	115 ± 23	108 ± 23	NS
NYHA class I/II/III (n=)	8/52/40	5/30/26	NS
LVEDVI (ml/m^2)	141 ± 60	150 ± 55	NS
LVESVI (ml/m^2)	107 ± 45	110 ± 50	NS
LVEF (%)	24 ± 6	25 ± 5	NS
Inferior vena cava			
X (cm/s)	33 ± 19	27 ± 19	NS
Y (cm/s)	41 ± 15	39 ± 20	NS
SF (%)	38 ± 20	33 ± 22	NS
IVCDmin (mm)	10 ± 9.7	10 ± 8.6	NS
IVCDmax (mm)	15 ± 6.3	16 ± 6.1	NS
CIIVC (%)	55 ± 29	46 ± 28	NS
Tricuspid regurgitation (n=)	34 (34%)	18 (30%)	NS
Tricuspid regurgitant jet area (cm^2)	6.2 ± 4.8	6.2 ± 4.4	NS
Cardiac index (l/min/m^2)	2.1 ± 0.6	2.0 ± 0.5	NS
Pulmonary wedge pressure (mmHg)	20 ± 10	21 ± 9	NS
Right atrial pressure (mmHg)	7.8 ± 4	7.5 ± 4	NS
Therapy			
Diuretics	84 (84%)	50 (82%)	NS
Digitalis	69 (69%)	41 (67%)	NS
ACE-inhibitors	81 (81%)	55 (90%)	NS
Nitrates	56 (56%)	35 (57%)	NS

CIIVC = collapse index of the inferior vena cava; IVCDmax = inferior vena cava maximum end-expiratory diameter; IVCDmin = inferior vena cava minimum end-inspiratory diameter; LVEDVI = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVESVI = left ventricular end-systolic volume index; SF = systolic fraction of forward filling flow of the inferior vena cava; X = peak velocity of systolic forward filling wave of inferior vena cava flow; Y = peak velocity of diastolic forward filling wave of inferior vena cava flow.

Cardiac catheterization. Right cardiac catheterization was performed by a 7F Swan-Ganz balloon tipped catheter inserted into the right internal jugular vein and advanced through the right heart cavities into the pulmonary artery. The pulmonary capillary wedge position was confirmed by the appearance of a typical wedge pressure tracing and/or by the fluoroscopic observation of the catheter tip during balloon inflation. Fifteen minutes after the catheter insertion, measurements were obtained with the patients in a supine position, using an HP transducer connected to a 7005 Marquette polygraph (Marquette, Waukesha, WI, USA). Pressure tracings were recorded at a speed of 50 cm/s on a scale calibrated from 0 to 60 mmHg.

Cardiac output was determined by averaging three thermal dilution curves obtained by injecting 10 ml of saline solution at 0°C into the right atrium.

RAP, pulmonary artery pressures and mean pulmonary wedge pressure were recorded. Arterial systolic and diastolic blood pressures were measured with a noninvasive method by a calibrated v-look-cuff connected to a 7005 Marquette system. Heart rate, derived from a standard ECG lead, was monitored continuously.

Echocardiography. Echocardiographic studies were performed using an HP Sonos 1000 ultrasound system (Hewlett Packard, Palo Alto, CA, USA) with 2.5 and 3.5 MHz transducers. Combined two-dimensional and M-mode echocardiographic examinations were performed during right heart catheterization with the patient lying in a supine position. A two-dimensional echocardiogram of the inferior vena cava was obtained from a subxiphoid view. The transducer was held and translated to obtain clear inferior vena cava images; in this setting, the track-ball to obtain M-mode scanning was positioned perpendicularly to the long axis of the inferior vena cava within 2 cm of its entrance into the right atrium[12]. All images were recorded on a videotape and subsequently analyzed using the software packages built into the ultrasound system. Using the respiratory trace, which mirrors the movement of the diaphragm during respiration, and the leading edge method, the maximal end-expiratory and minimal end-inspiratory diameters of the inferior vena cava were measured. These measurements using the QRS as a marker, were obtained in the diastolic phase. Collapse index of the inferior vena cava, which identifies the percent decrease in the diameter of the inferior vena cava with quiet inspiration, was calculated as the difference of diameters to maximal diameter ratio percent. Inferior vena cava flow was obtained from a subxiphoid view. After identification of the inferior vena cava flow by B-mode, a sample volume was set about 1 cm from the entry into the right atrium. Peak velocities of forward systolic and diastolic inferior vena cava flow were measured from averaging five consecutive cardiac cycles. Systolic forward inferior vena cava flow was calculated as the peak velocity of the systolic wave divided by the sum of the peak systolic and diastolic velocity percent.

Reproducibility. Intra and interobserver reproducibility of Doppler echocardiography measurements was assessed in 20 consecutive patients. Measurements were repeated at least 1 week apart by the same observer and by a second independent observer. The variability of the parameters considered was then evaluated calculating the mean relative difference between paired measurements and its standard deviation. There were close intra and interobserver agreements for the measurements evaluated. The mean differences between measurements were close to zero (end-inspiratory and end-expiratory inferior vena cava diameter - 0.02) indicating that there was no systematic difference among the examinations; the standard deviations of the differences were small, between 6-10% of the mean values for the two variables.

Statistical analysis. Statistical analysis was performed using the SAS statistical package[13]. To compare clinical, hemodynamic and echocardiographic variables between the groups a Student's t test for independent samples and two-way tables with Pearson χ^2 test were used. Linear regression analysis was carried out to correlate different indices derived from measurements of the inferior vena cava and RAP in patients with and without tricuspid regurgitation. In the learning group all indices were submitted to multiple forward linear regression analysis to generate the predicting equations. Two separate multiple forward linear regression analyses were performed. A first multiple forward linear regression analysis was carried out considering echo-Doppler variables measured in all patients in the learning group. Since tricuspid regurgitation can influence Doppler echocardiographic measurements, a second multiple forward linear regression analysis was carried out evaluating the same echo-Doppler variables in the subgroup without tricuspid regurgitation. The Bland-Altman method was used to evaluate the agreement between the invasive and noninvasive methods used to estimate RAP[14]. A probability value of < 0.05 was considered statistically significant. All values are expressed as mean value ± SD.

Results

Correlation between inferior vena cava and right atrial pressure measurements. *Linear regression analysis.* In all 100 patients M-mode and two-dimensional Doppler echocardiography analysis gave high quality recordings of the inferior vena cava. Figure 1 shows the temporal relation between inferior vena cava diameter, electrocardiographic and respiratory phases. Inferior vena cava diameters, their collapse indexes and systolic forward inferior vena cava flow correlated with RAP (Table II; Fig. 2). The inspiratory inferior vena cava diameters provided the strongest correlation. The correlations be-

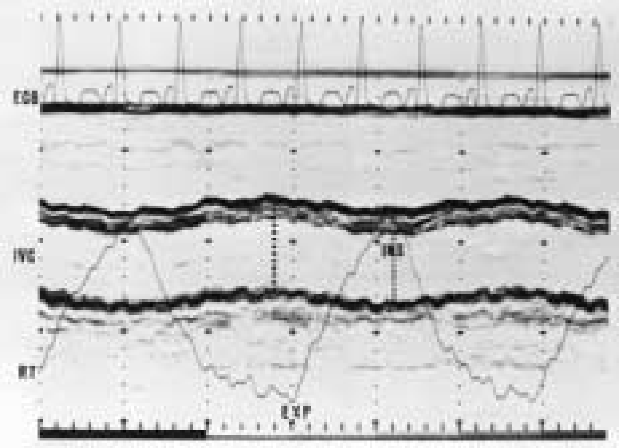


Figure 1. *Relationship between electrocardiogram (ECG), end-expiratory (EXP) and end-inspiratory (INS) phases and M-mode inferior vena cava (IVC) diameters. RT = respiratory trace.*

tween inferior vena cava parameters and RAP did not improve considering only patients without tricuspid regurgitation (Table II).

Multiple forward linear regression analysis. Using RAP as the dependent variable and inferior vena cava and Doppler echocardiographic measurements as independent variables, a first multivariate equation was produced (equation 1) from the multiple forward regression analysis. The most important predictor was the inspiratory inferior vena cava diameter, whereas the index of its collapse added a smaller contribution. The cumulative correlation coefficient was 0.82. This procedure was repeated and similar results produced in a subgroup of patients without tricuspid regurgitation (equation 2) (Table III). In patients without tricuspid regurgitation the standard deviation of mean differences between estimated and measured RAP was less than that observed in patients with tricuspid regurgitation (1.2 vs 1.8 mmHg, p < 0.001) (Fig. 3).

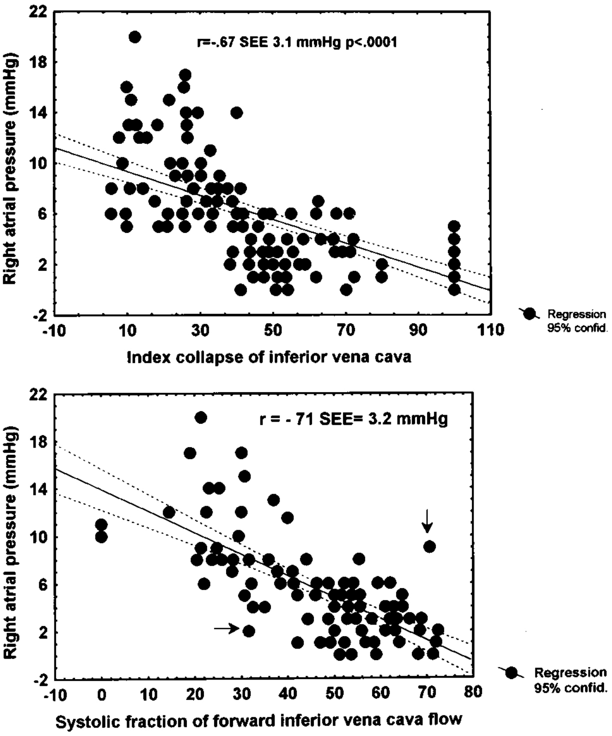


Figure 2. *Scatterplots show relation between right atrial pressure and collapse index of the inferior vena cava* (top) *and systolic fraction of forward inferior vena cava flow* (bottom). *The* horizontal arrow *indicates a patient with severe tricuspid regurgitation, normal right atrial pressure and a decrease in systolic fraction of forward inferior vena cava flow. The* vertical arrow *points to data from a patient with increased right atrial pressure, severe tricuspid regurgitation, and normal systolic fraction on forward inferior vena cava flow.*

Estimation of right atrial pressure in a prospective testing group. In a prospectively enrolled testing group of patients with congestive heart failure, the equations derived were applied to determine the estimate of RAP. At baseline and after loading manipulations, a strong correlation between estimated and measured RAP was obtained in patients with and without tricuspid regurgitation (Fig. 4).

Table II. Correlations between right atrial pressure (RAP) and echo-Doppler variables in all patients and in those without tricuspid regurgitation (TR).

Doppler and B-mode echocardiographic indices	All patients (n=100)		Patients without TR (n=61)	
	r	SEE	r	SEE
Inferior vena cava diameters				
IVCDmin	0.84	2*	0.87	1.2*
IVCDmax	0.76	2.7*	0.75	1.9*
CIIVC	-0.67	3.1*	-0.65	2.3*
Inferior vena cava venous flow				
X	-0.72	3.6*	-0.73	2.5*
Y	0.37	3.1*	0.40	2.5
SF	-0.71	3.5*	-0.77	2.7*

Abbreviations as in table I. * $p < 0.05$.

Table III. Multivariate forward analysis.

Models	Cumulative r	SEE
1. M-mode measurements of the inferior vena cava in all learning groups		
IVCDmin	0.72	2.8
IVCDmin + CIIVC	0.82	1.7
Final equation: RAP = (6.4*IVCDmin + 0.04*CIIVC) - 2		
2. M-mode measurements of the inferior vena cava in learning patients without TR		
IVCDmin	0.84	1.8
IVCDmin + CIIVC	0.92	1.2
Final equation: RAP = (4.9*IVCDmin + 0.01*CIIVC) - 0.2		

Abbreviations as in tables I and II.

The agreement between invasive and non-invasive measurements of RAP in identifying patients with normal (≤ 5 mmHg), moderately increased (< 5 RAP < 10 mmHg) or markedly increased (≥ 10 mmHg) RAP was 81 or 93% using equation 1 or 2 respectively (Fig. 5). Changes in RAP induced by preload manipulations were correctly predicted using both equations.

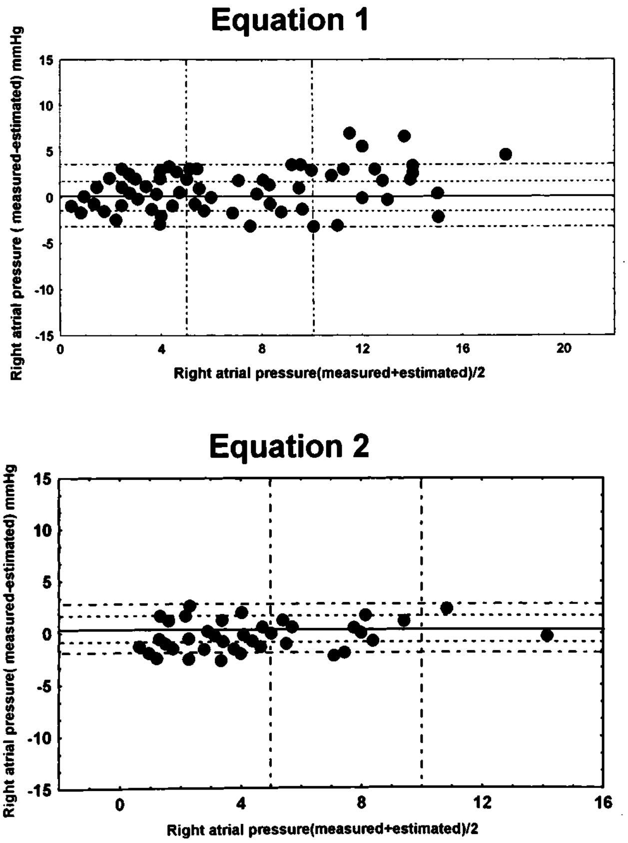


Figure 3. *Bland-Altman plots showing the differences between measured and Doppler echocardiography estimated right atrial pressures* (on the ordinate) *plotted against their mean baseline values 0* (on the abscissa). *The* horizontal lines in the center *represent the mean differences between two measurements; the* upper *and* lower horizontal lines *represent 1 and 2 standard deviations of these means. The* top *and* bottom plots *are related to equation 1 and 2 respectively.*

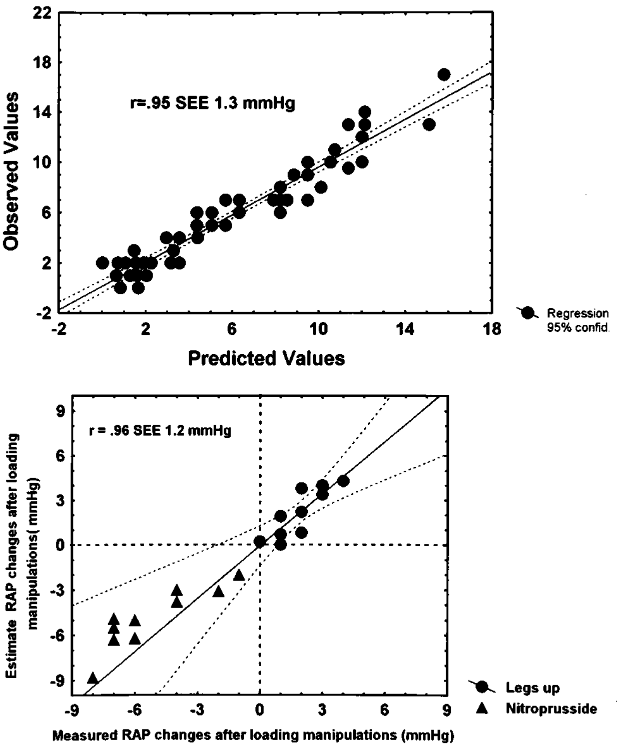
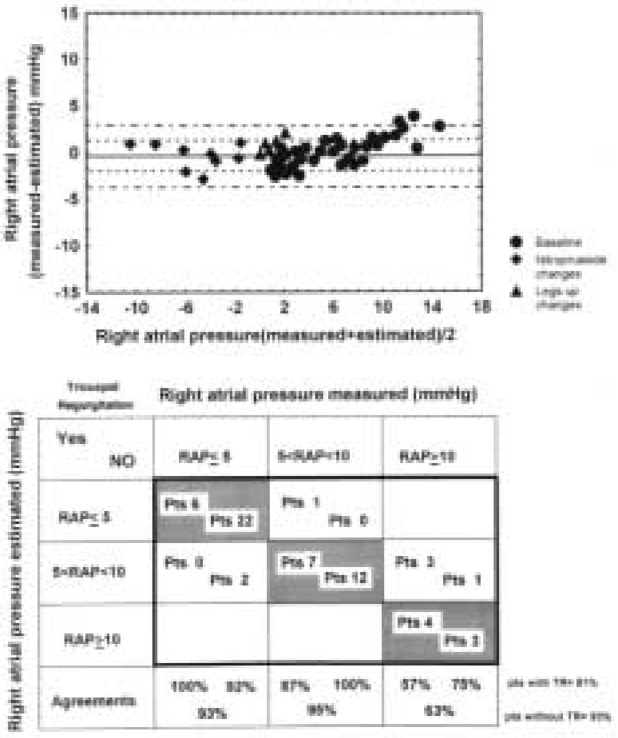


Figure 4. *The scatterplots show the relation between observed and predicted baseline values of right atrial pressure (RAP)* (top) *and their changes* (bottom) *after loading manipulations in a prospective study of a group of patients with congestive heart failure.*

Discussion

The non-invasive determination of RAP in patients with congestive heart failure is an important goal for evaluating concomitant right hemodynamic alterations and effects of unloading therapy. Our results showed that a combination of indices derived from M-mode inferior vena cava measurements could provide a realistic estimate of RAP at baseline and after unloading manipulations.

In several studies Doppler echocardiographic measurements of the inferior vena cava have been shown to be useful in estimating right hemodynamics in normal subjects and in patients with a variety of cardiac diseases[15-17]. Measurements of the diameter of the inferior vena cava, in M- and B-mode echographic evaluations, showed different degrees of correlation with RAP, which were determined by several causes: methods, etiology, and indices considered. All these studies agreed that the end-inspiratory diameter and inspiratory collapse of the inferior vena cava could provide useful information for non-invasive evaluation of RAP. Other studies have also shown that forward systolic fraction of inferior vena



Yes NO	RAP≤5	5<RAP<10	RAP≥10
RAP≤5	Pts 6 Pts 22	Pts 1 Pts 0	
5<RAP<10	Pts 0 Pts 2	Pts 7 Pts 12	Pts 3 Pts 1
RAP≥10			Pts 4 Pts 3
Agreements	100% 92% 93%	87% 100% 95%	57% 75% 63%

Figure 5. *Bland-Altman plots* (top) *showing the differences between measured and Doppler echocardiography estimated right atrial pressures (RAP)* (on the ordinate) *at baseline and after loading manipulations plotted against their mean values* (on the abscissa). *The* horizontal lines in the center *represent the mean differences between two measurements; the* upper *and* lower horizontal lines *represent 1 and 2 standard deviations of these means. The graph* (bottom) *shows the agreements between classifications of patients with normal, moderate or large increase of RAP measured and estimated by equations 1 and 2. TR = tricuspid regurgitation.*

cava flow can provide an estimate of RAP[18]. In our study, forward systolic fraction of inferior vena cava flow showed an inverse relationship with RAP. However the correlation coefficient was similar to those of indices derived from measurements of the diameter of the inferior vena cava with a wide standard error of estimate which prevented a realistic non-invasive evaluation of RAP. On the other hand, inferior vena cava flow is determined by the instantaneous pressure gradient which is the result of several complex mechanisms involving right atrial and ventricular functions, tricuspid regurgitation and mean circulatory filling pressure. These factors can affect the instantaneous a-x and v-y gradients and concomitant forward inferior vena cava flow. Multivariate stepwise forward regression analysis, including different indices derived from vena cava measurements, identified the independent contribution of each variable to the definition of the best model for estimating RAP. Both equations provided useful estimates of RAP at baseline and after loading manipulations. Minimum inferior vena cava diameter and collapse index were the only variables which were included in these models. The inferior vena cava is a capacitance vessel connected to the right atrium; during inspiration, negative intrathoracic pressure induces a decrease in RAP which generates an increase of forward positive gradient pressure in the inferior vena cava which accelerates venous return to a maximum *plateau* with a sigmoid shape[19]. This amount of venous blood restores the system to a static condition. Thus, the minimum diameter of the inferior vena cava so induced, can be expected to provide an indirect estimate of filling pressure related to “unstressed volume” which represents the fraction of blood volume needed for basal filling of the vascular bed[9]. This filling pressure of the vascular bed is also strongly correlated with mean pressure of the right atrium. In contrast, the “stressed volume”, which coincides with the ramp of the sigmoid shape, corresponds to the “dynamic” fraction of blood volume returned to the right atrium as “venous return” after inspiration. The forward gradient pressure that moves this quantum of blood volume depends on the baseline RAP and on the variations induced with inspiration[20]. The complementary contributions of these two indices act to reduce the estimate error to a minimum. Surprisingly, the systolic fraction of forward inferior vena cava flow was not included in the final model. This variable is related to instantaneous a-x and v-y gradients in the right atrium, to the vascular capacitance of the inferior vena cava, the blood volume and tricuspid regurgitation. Thus, a normal systolic fraction of inferior vena cava flow can be determined in concomitance with a good vascular capacitance, severe tricuspid regurgitation and increased mean RAP (Fig. 3, vertical arrow); in contrast, a decrease in systolic fraction of inferior vena cava flow can occur in the presence of normal RAP and mild tricuspid regurgitation but with the exhaustion of vascular capacitance (Fig. 3, horizontal arrow). Inspiration introduces an external perturbation (negative intrathoracic pressure) into this inertial system whose response – collapse and minimum diameter of the inferior vena cava – is a function of RAP. These physiologic considerations explain why a combination of the two indices described above can produce an accurate estimate of RAP.

Previous comparative studies. Several studies using Doppler echocardiographic approaches have shown good correlations between Doppler echocardiography-derived indices and RAP[12,16,18]. Kircher et al.[16] identified how B-mode end-inspiratory diameter and collapse index of the inferior vena cava were correlated with atrial pressure. Our data revealed stronger correlations, probably because of the homogeneous population, and the fact that Doppler echocardiographic and hemodynamic evaluations were carried out simultaneously. From only one study was an equation derived for predicting RAP from stepwise regression analysis of Doppler echocardiographic variables[18]. In this model only systolic fraction of forward vena cava flow was included. In contrast, this variable was not included in our predictive model. The apparent discrepancy between these findings can be explained by important differences between the study groups: to determine the predictive equation, Nagueh et al.[18] used a small, heterogeneous group of patients with normal right atrial and ventricular functions and without tricuspid regurgitation. In contrast, our patients had significant right atrial and ventricular dysfunction and tricuspid regurgitation.

Clinical implications. Quantitative estimation of RAP is a useful goal for characterizing baseline hemodynamic profile and unloading therapy response in patients with congestive heart failure. Furthermore it helps to increase the accuracy of non-invasive determination of systolic pulmonary artery pressure. Assessment of simple measurements of the inferior vena cava provided an accurate estimate of RAP with a contained error (2SD of mean differences 3.6 mmHg) which improved in patients without tricuspid regurgitation (2SD of mean differences 2.4 mmHg). This accuracy was reproducible in a similar prospectively enrolled population at baseline (2SD of mean differences 3.4 mmHg) and after unloading manipulations (2SD of mean differences 2.2 mmHg). The variables included into our predictive model can be utilized routinely in echocardiography laboratories. This simple, non-invasive method may be a more comfortable and less expensive alternative to heart catheterization for evaluating pulmonary hemodynamic profile and its changes.

Study limitations. One of the aims of this study was to investigate, in patients with congestive heart failure, the reliability of indices derived from Doppler echocardiographic M-mode inferior vena cava measurements in estimating RAP; for this reason, our results cannot be generalized to other patients with normal or less compromised left ventricular function. Furthermore the equation derived can only be applied to patients for whom technically adequate M-mode inferior vena cava measurements are available. However, in our experience this was the case in all patients. Finally these results cannot be applied to patients with atrial arrhythmias, prosthetic valves, pericardial disease, and/or ventilation disorders.

In conclusion, our results provide evidence that in patients with congestive heart failure indices derived from the inferior vena cava can be used to provide an accurate, non-invasive estimate of RAP. This is another example of the usefulness of Doppler echocardiography, a simple, non-invasive technique, in evaluating hemodynamic profile and its changes in patients with congestive heart failure. Echocardiographic assessment of the inferior vena cava should be included in the evaluation of patients with congestive heart failure.

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