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Identifying Discordance of Right- and Left-Ventricular Filling Pressures in Patients with Heart Failure by the Clinical Examination

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Abstract

Background: In approximately 25% of patients with heart failure and reduced left-ventricular ejection fraction (HFrEF), right-ventricular (RV) and left-ventricular (LV) filling pressures are discordant (i.e., one is elevated while the other is not). Whether clinical assessment allows detection of this discordance is unknown. We sought to determine the agreement of clinically- versus invasively-determined patterns of ventricular congestion.

Methods: In 156 HFrEF subjects undergoing invasive hemodynamic assessment, we categorized patterns of ventricular congestion (no congestion, RV only, LV only, or both) based on clinical findings of RV (jugular venous distention, JVD) or LV (hepatojugular reflux, orthopnea, or bendopnea) congestion. Agreement between clinically and invasively determined [RV congestion if right atrial pressure (RAP) ≥10 mmHg and LV congestion if pulmonary capillary wedge pressure (PCWP) ≥22 mmHg)] categorizations was the primary endpoint.

Results: The frequency of clinical patterns of congestion was: 51% no congestion, 24% both RV and LV, 21% LV only, and 4% RV only. JVD had excellent discrimination for elevated RAP (C=0.88). However, agreement between clinical and invasive congestion patterns was poor, κ=0.44 (95% CI 0.34–0.55). While those with no clinical congestion usually had low RAP and PCWP (67/79, 85%), over one-half (24/38, 64%) with isolated LV clinical congestion had PCWP <22 mmHg, most (5/7, 71%) with isolated RV clinical congestion had PCWP ≥22 mmHg, and õne-third (10/32, 31%) with both RV and LV clinical congestion had elevated RAP but PCWP <22 mmHg.

Conclusions: While clinical examination allows accurate detection of elevated RAP, it does not allow accurate detection of discordant RV and LV filling pressures.

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Keywords

clinical examination; congestion profiles; bendopnea; jugular venous pressure; hemodynamics

Clinical assessment of congestion in patients with heart failure (HF) is largely dependent upon manifestations of elevated right-ventricular filling pressures (i.e. right atrial pressure, RAP) including elevated jugular venous pressure (JVP), edema, or ascites. A series of studies over many years have characterized the relationship of the right ventricular filling pressure, and the left ventricular filling pressure, (i.e. pulmonary capillary wedge pressure, PCWP), in patients with heart failure. A concordant relationship of the RAP and PCWP is seen in most patients (~75%) and forms the rationale for the use of the JVP to estimate volume status in patients with heart failure.[1–4] However, it is important to recognize the significant minority of individuals (~25%) with discordant ventricular filling pressures, both because such patterns portend a worse outcome,[1, 4–7] but also to form the basis of future studies which are needed to determine how best to treat patients with these less common, but challenging, hemodynamic patterns. Currently, it is unknown whether clinical assessment by history and physical examination allows accurate detection of isolated elevated right- or left-sided ventricular filling pressures. Thus, we performed a prospective, cross-sectional study to establish the utility of the clinical examination to categorize patterns of ventricular congestion in a group of patients undergoing invasive hemodynamic assessment.

METHODS

Study design and subject selection

We conducted a single-center prospective, cross-sectional study of a convenience sample (based on investigator availability to enroll) of 156 subjects with heart failure and reduced ejection fraction (HFrEF) who were referred for a clinically indicated invasive hemodynamic assessment by right heart catheterization at the University of Texas Southwestern Medical Center between May 2016 and June 2018. Inpatient or outpatient subjects 18 years of age or older with HFrEF, defined as a left ventricular ejection fraction (LVEF) ≤ 40% within 6 months of enrollment, were eligible for participation. Subjects were excluded if they had undergone cardiac transplantation or were on mechanical circulatory support. The study protocol was approved by the University of Texas Southwestern Institutional Review Board. All subjects provided written informed consent. The authors declare that all supporting data are available within the article.

Data collection

A history and physical examination for each subject was completed by an investigator (DP) within 6 hours prior to the right-heart catheterization. Data collected included demographics, past medical history, cardiac medications, etiology of cardiomyopathy, left ventricular ejection fraction (LVEF), and New York Heart Association (NYHA) classification as determined by the Specific Activity Scale.[8, 9] Laboratory values were abstracted from the clinical chart if they were within one week of enrollment for outpatients, and the same day as enrollment for inpatients. Estimated glomerular filtration rate (eGFR) was determined by the Modification of Diet in Renal Disease (MDRD) equation.[10]

Clinical assessment

At the time of enrollment and within 6 hours prior to the right-heart catheterization, subjects were assessed for clinical congestion with the following protocol: JVP was assessed by determining the height of the venous pulsation above the angle of Louis and adding 5cm to account for the distance to the right atrium; hepatojugular reflux (HJR) was present if there was a sustained increase in JVP during ten seconds of continuous pressure on the abdomen; bendopnea was present if the patient developed dyspnea within 30 seconds of bending at the waist while sitting in a chair as if tying their shoes and ensuring that the patient was not holding their breath; and orthopnea was present if the supine patient developed dyspnea within 2 minutes of the bed being lowered to 0 degrees. Orthopnea, bendopnea and HJR were selected given their associations with elevated PCWP.[11–13] Jugular venous distention (JVD) *apriori* was considered present if the JVP was estimated to be ≥12 cm. Subjects were classified into 4 patterns of ventricular congestion based on findings from their clinical assessment: 1) no congestion: if no JVD, HJR, orthopnea, or bendopnea were present; 2) isolated left-sided congestion: if any of HJR, orthopnea, or bendopnea were present in the absence of JVD; 3) isolated right-sided congestion: if JVD was present without HJR, orthopnea, or bendopnea 4) both left- and right-sided congestion: if JVD was present with any of HJR, orthopnea, or bendopnea.

Invasive hemodynamic assessment

Interventional cardiologists or advanced heart failure cardiologists who were blinded to the clinical assessment of congestion performed routine supine hemodynamic measurement via right heart catheterization. All pressures were obtained at end-expiration in steady state. The mean pressure of the a-wave was used for determining the RAP and PCWP. Subjects were classified into 4 invasively-determined patterns of ventricular congestion: 1) no hemodynamic congestion: if RAP < 10 mmHg and PCWP < 22 mm Hg; 2) isolated left-sided ventricular congestion: if RAP < 10 mmHg and PCWP ≥ 22 mmHg; 3) isolated right-sided ventricular congestion: if RAP ≥ 10 mmHg and PCWP < 22 mmHg; 4) both left- and right-sided ventricular congestion: if RAP ≥ 10 mmHg and PCWP ≥ 22 mmHg. These cutoffs were chosen based on previously accepted values indicating congestion.[2, 4, 11] Cardiac index was measured either by thermodilution or the indirect Fick methods.

Statistical analysis

Descriptive statistics were reported as mean (SD), median (25^{th}, 75^{th} percentiles), or number (%), where appropriate. Categorical variables were compared among the four patterns of ventricular congestion by the Fisher's exact test and continuous variables by the Kruskal Wallis test. We used the Bonferroni correction to account for multiple testing when assessing the significance of the differences of hemodynamic variables between various combinations of two specific patterns of ventricular congestion. Receiver operating curves were constructed and the area under the curve (AUC) was determined. Univariable and multivariable models were developed to demonstrate the association of physical examination findings with elevated PCWP. The variables included in the multivariable regression models were those that were used to determine the different clinical patterns of ventricular congestion. The prespecified primary endpoint of this study was the kappa coefficient of the

agreement between the clinical and invasively-determined patterns of ventricular congestion. Three exploratory analyses were performed. First, we classified left-sided congestion solely on the basis of bendopnea (not incorporating HJR or orthopnea). Second, left-sided congestion was defined on the basis of orthopnea (not incorporating bendopnea or HJR). Lastly, we recalculated the Kappa statistic in assessing agreement of the clinical assessment with the invasive measurement using ≥20 mm Hg as the threshold to define an elevated PCWP. Statistical significance was set at $p \leq 0.05$. Statistical analyses were conducted using SAS version 9.4 software (SAS Institute, Inc., Cary, North Carolina).

RESULTS

Baseline clinical characteristics of patients by clinical pattern of ventricular congestion are summarized in Table 1. Half of the patients had no exam findings of congestion (n=79, 51%), nearly one-quarter of the patients (n=38, 24%) had both left- and right-sided clinical congestion, approximately one-fifth (n=29, 21%) left-sided clinical congestion, and only 7 (4%) had isolated right-sided clinical congestion. Across all groups, the majority were middle-aged men. The physical exam findings of each group are also shown in Table 1. Of those with isolated left-sided clinical congestion, bendopnea was present in most, and either HJR or orthopnea present in approximately one-half. Rales and a third heart sound were infrequent but most commonly present in those with both right- and left-sided clinical congestion. Body mass index was similar across groups. Those with versus without right-sided congestion (either JVD alone or with markers of left-sided congestion) had worse renal function as evidenced by higher BUN and creatinine and lower GFR and higher use of inotropes.

The right heart catheterizations were typically performed in the context of worsening heart failure (51%) and/or evaluation for heart transplant or LVAD (34%). Of these, 2 had suspicion for suspicion for infiltrative cardiomyopathy and also underwent endomyocardial biopsy. Less frequent indications included: weaning of inotropes, evaluation for mitral or aortic valve repair, or pulmonary hypertension. Most patients (66%) were inpatients. Table 2 illustrates the frequency of the invasive hemodynamic profiles. The majority (59%) had concordant low RAP and low PCWP, and 20% had concordant high RAP and high PCWP. Twenty-one percent had discordant right- and left-sided filling pressures, with 10% having a low RAP but elevated PCWP, and 11.5% with an elevated RAP and low PCWP.

Physical exam findings of Right- and Left-sided congestion and invasive hemodynamics

The invasive hemodynamics, stratified by clinical patterns of ventricular congestion, are shown in Table 3. Subjects without clinical congestion had a lower RAP, PCWP, and mean PA than either those with isolated RV congestion or those with both RV and LV congestion. PVR also differed among groups though once multiple testing was considered, only the indirect Fick estimate of PVR between subjects without congestion and those with both RV and LV congestion remained significant. Subjects with isolated LV congestion had a lower RAP than those with isolated RV congestion and those with both RV and LV congestion. They also had a lower PCWP and mean PA than those with both RV and LV congestion. Cardiac index was similar among all groups. Both JVD and JVP had excellent

discrimination for detection of an elevated RAP (Figure 1). The discrimination of JVD for detection of an elevated PCWP was good (Figure 2, C=0.73) with bendopnea providing additional utility, increasing the c-statistic to 0.79 (p=0.005 for the comparison).

Univariable models of physical exam findings and elevated PCWP are summarized in Table 4. Each physical examination finding was associated with an elevated PCWP. In multivariable logistic regression models, once JVD or JVP were entered, only bendopnea and the presence of an S3 remained associated with elevated PCWP (Table 5).

There was only modest agreement between the clinical and invasive hemodynamic patterns of ventricular congestion κ=0.44 (95% CI 0.34–0.55) (Table 6). While those with no clinical congestion usually (67/79, 85%) had a low measured RAP and PCWP, the limited agreement resulted from several key discrepancies between the clinical and invasive patterns. Specifically, over one-half (24/38, 64%) of those with clinical LV congestion had a PCWP < 22 mmHg, the majority (5/7, 71%) of those with isolated clinical RV congestion had a PCWP ≥ 22 mmHg, and approximately one-third (10/32, 31%) of those with both clinical RV and LV congestion actually had an elevated RAP but a PCWP < 22 mmHg. Agreement was similarly poor when left-sided congestion was defined as PCWP ≥ 20 mmHg rather than 22 mmHg: κ=0.46 (95% CI 0.35–0.57). The discrepancy between clinical and hemodynamic patterns of ventricular congestion was also evident when comparing the PCWP in the following subgroups of patients: the PCWP in those with JVD but no orthopnea/bendopnea/HJR (n=7) was higher than those with bendopnea without JVD (n=25): [24 (18, 32) mmHg vs. 19 (10, 24) mmHg, p=0.04, respectively] or those with either orthopnea or bendopnea or HJR but no JVD (n=38): [the latter 18 (10, 24) mmHg, p=0.02]. In exploratory analyses, a comparison between the pattern of ventricular congestion based only on bendopnea as a marker of LV congestion and JVD as a marker of RV congestion and invasively defined profiles was undertaken (Supplemental table 1). Agreement remained modest with this approach. (κ=0.53 (95% CI 0.42–0.64). Similarly, when orthopnea was substituted for bendopnea in this approach, agreement was modest (κ=0.45, 95% CI 0.34–0.56).

DISCUSSION

This is the first study, to our knowledge, to assess the utility of the clinical examination in identifying patients with heart failure who have discordant patterns of ventricular congestion. We found that while estimation of JVP had excellent discrimination for an elevated RAP and moderate discrimination for an elevated PCWP, the clinical examination was not able to accurately identify discordance of right- and left-sided ventricular filling pressures, in other words an elevated PCWP when the RAP was low or a low PCWP when the RAP was high.

Previous studies have assessed the accuracy of the clinical examination for estimation of ventricular filling pressures. Several studies have shown that the clinical examination allows accurate estimation of the jugular venous pressure, and hence the RAP, in patients with HF.[14, 15] The current study confirms those findings. Other studies have assessed the utility of the clinical examination to detect an elevated PCWP. In an analysis of the ESCAPE trial,

once estimated RAP by JVP was included, no other clinical examination parameter was associated with a PCWP > 22 mmHg.[11] Our study likewise found the JVP was strongly associated with an PCWP in multivariable logistic regression modeling and had good discrimination for an elevated PCWP by ROC analysis. While bendopnea was associated with elevated PCWP in multivariable models, and did provide added discrimination to the JVD to detect an elevated PCWP, it did not perform well as a diagnostic test to detect either an elevated PCWP when there was no JVD, nor did its absence exclude an elevated PCWP when JVD was present. The same finding was true of orthopnea and HJR. Together, these findings reinforce that estimation of the JVP remains the key component of the clinical examination when estimating whether LV filling pressures are elevated or not.

The utility of the JVP to estimated LV filling pressures is based on the finding that most patients with HF have concordant low or high RAP and PCWP, meaning that the RV and LV filling pressures mirror one another. However, ~25% of patients have discordant pressures, where the RAP may be low and the PCWP high (compensated RV pattern) or the RAP may be high and the PCWP may be low (right-left equalizer pattern).[1–4] Our study confirmed this finding, with 21% of patients having discordant right- and left-sided filling pressures.

An effort to identify patients into discordant categories by physical examination findings has clinical importance, as often these discordant groups, in particular, those who are “right-left equalizers” may have worse clinical outcomes, including and mortality.[1, 4–6] A recent study of patients with hospitalized patients with heart failure supported the hypothesis that clinical outcomes can be related to patterns of congestion,[16] though the classification in that study incorporated rales, a finding which does not appear to be a reliable marker of left-ventricular filling pressure in patients with advanced heart failure.[17] Accurate identification of patients with discordant filling pressures via clinical evaluation would also facilitate future studies to determine how best to treat patients with these discordant hemodynamic patterns, a currently unanswered question.[7] However, the data reported herein demonstrated that the clinical examination is not able to differentiate patients into discordant right- and left-sided congestion categories, in large part because of the strong association of the JVP with PCWP, such that when JVD is present, the PCWP is usually (71%) elevated even if bendopnea, orthopnea, or HJR are not present. Similarly, when JVD is not present, the PCWP is not elevated even if bendopnea, orthopnea, or HJR are present (64%) (Figure 3). As such, these observations do not support the utility of the clinical examination to detect discordance between right- and left- sided ventricular filling pressures. Thus, if there is concern regarding discordant filling pressures, invasive hemodynamic measurement may be particularly useful.

Our study has limitations. This is a single center study performed by advanced heart failure cardiologists with an interest in the relationship between the physical examination and hemodynamic assessments. Furthermore, most subjects were white men with NYHA class II-III symptoms. Thus, the accuracy of the clinical examination findings may not be generalizable to other practice settings or all patients. The physical examination was performed by one provider so agreement could not be determined. We did not assess severity of tricuspid regurgitation at the time of the clinical examination, and thus cannot evaluate the potential impact on JVP or RAP assessment. We additionally did not incorporate other

echocardiographic parameters (such as inferior vena cava diameter or E/e' ratio) since an echocardiogram was not performed routinely on the same day as the physical examination and right heart catheterization. Due to the high concordance of RAP and PCWP, there was a relatively small sample size of patients with discordant hemodynamic profiles, which may have hindered our ability to discriminate these congestion profiles. Nevertheless, the low agreement, and its confidence intervals, suggests the utility of the clinical examination for this purpose would still be suboptimal even with a larger sample size. We enrolled a convenience sample of patients based on the availability of the investigator and the cardiac catheterization laboratory to enroll research patients. However, the convenience sampling was due to neither the severity of illness nor the presence or absence of clinical exam findings of congestion. These data were acquired in patients with HFrEF and should not be extrapolated to those with LVEF > 40%, though we previously demonstrated that there is high concordance of RAP and PCWP in those with heart failure with preserved ejection fraction.[3] Finally, most subjects were not taking angiotensin receptor-neprolysin inhibitors (ARNIs) or sodium/glucose cotransporter 2 inhibitors (SGLT2i). However, we do not believe that such medical therapy would alter the correlation of the clinical examination with invasive hemodynamics.

In conclusion, while the clinical examination has excellent utility for detection of an elevated RAP, and moderate utility for an elevated PCWP, it does not allow for accurate determination of whether there is concordance or discordance of RV and LV filling pressures. Thus, if there is clinical concern that a patient may have discordant pressures, invasive hemodynamic assessment is required.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Nonstandard Abbreviations and Acronyms

eGFR	Estimated glomerular filtration rate
HF	heart failure
HJR	hepatojugular reflux
HFrEF	heart failure with reduced ejection fraction
JVD	jugular venous distension

JVP	jugular venous pressure
LVEF	left ventricular ejection fraction
MDRD	modification of diet in renal disease
PCWP	pulmonary capillary wedge pressure
RAP	right atrial pressure

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What Is New?

- There was a strong association of the jugular venous pressure (JVP) with pulmonary capillary wedge pressure (PCWP) such that when jugular venous distension (JVD) was present, the PCWP was usually elevated even if bendopnea, orthopnea, or hepatojugular reflux were not detected.
- When JVD was absent, the PCWP often was not elevated even if bendopnea, orthopnea, or HJR were present.

What are the Clinical Implications?

- The clinical examination is not able to differentiate patients into discordant right- and left-sided congestion profiles.
- If there is concern regarding discordant filling pressures, invasive hemodynamic measurement may be indicated.

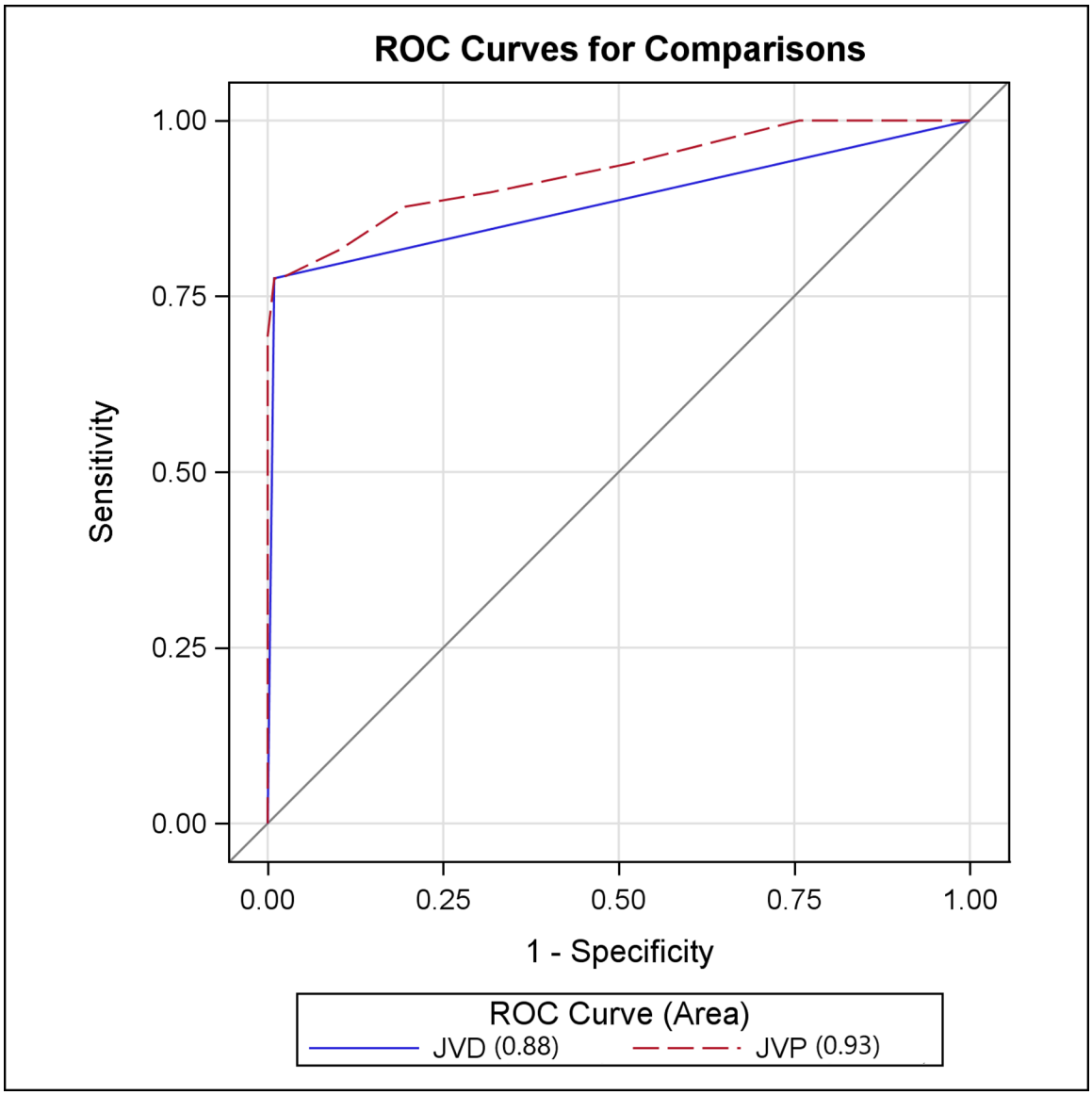

Figure 1.

ROC curve with elevated right atrial pressure (≥ 10mmHg) as the outcome variable and jugular venous distension or jugular venous pressure as the predictors. Estimated jugular venous distension is strongly associated with a right atrial pressure ≥10 mmHg with AUC = 0.88. Jugular venous pressure is strongly associated with a right atrial pressure ≥10 mmHg with AUC = 0.93.

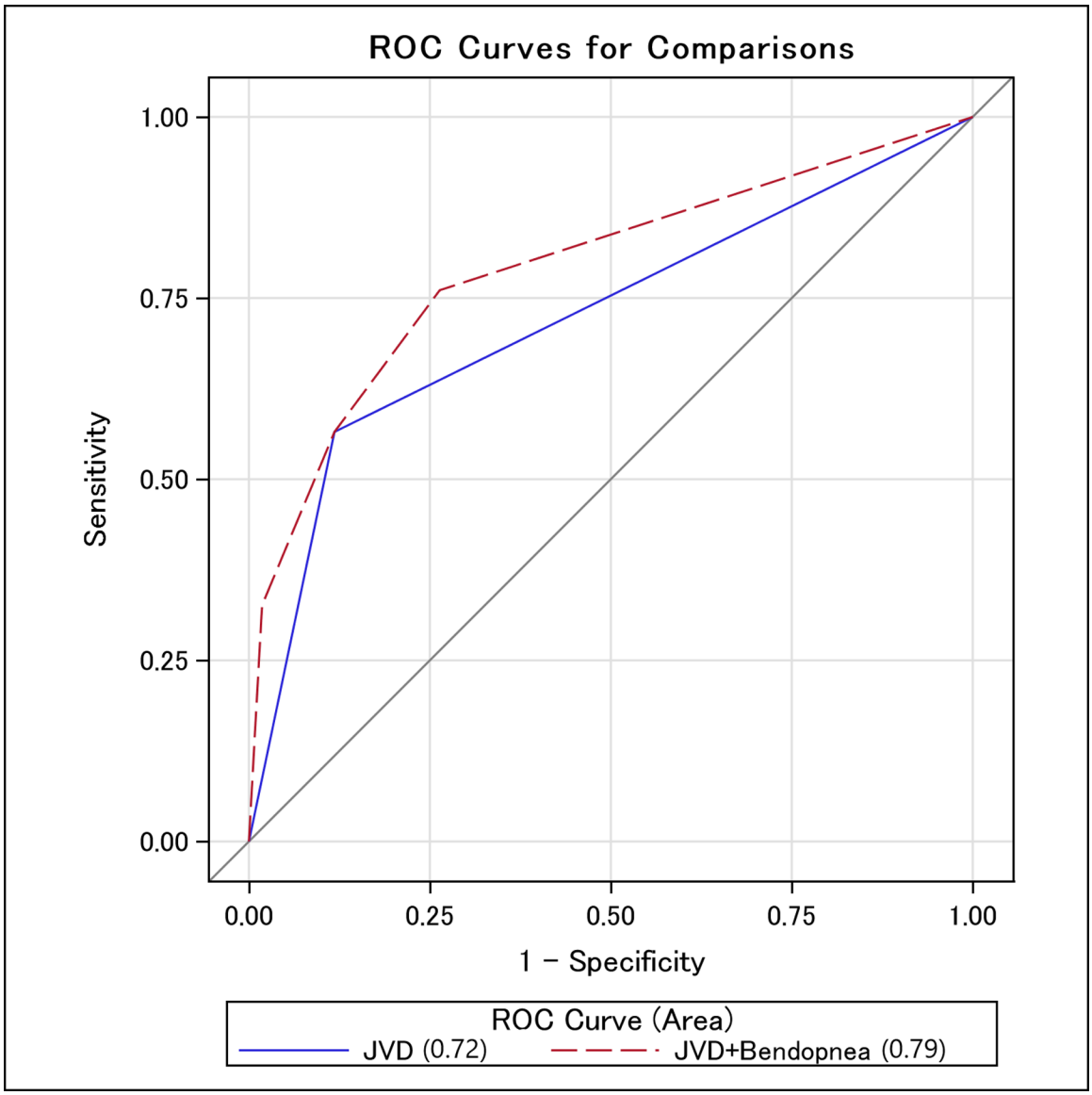

Figure 2.

ROC curves with elevated pulmonary capillary wedge pressure (≥ 22 mmHg) as the outcome variable and jugular venous distension (blue line) or jugular venous distension and bendopnea (red line) as the predictor. Bendopnea improved the AUC for detecting elevated pulmonary capillary wedge pressure compared with jugular venous distension alone (AUC 0.79 vs. 0.73, respectively; p=0.005).

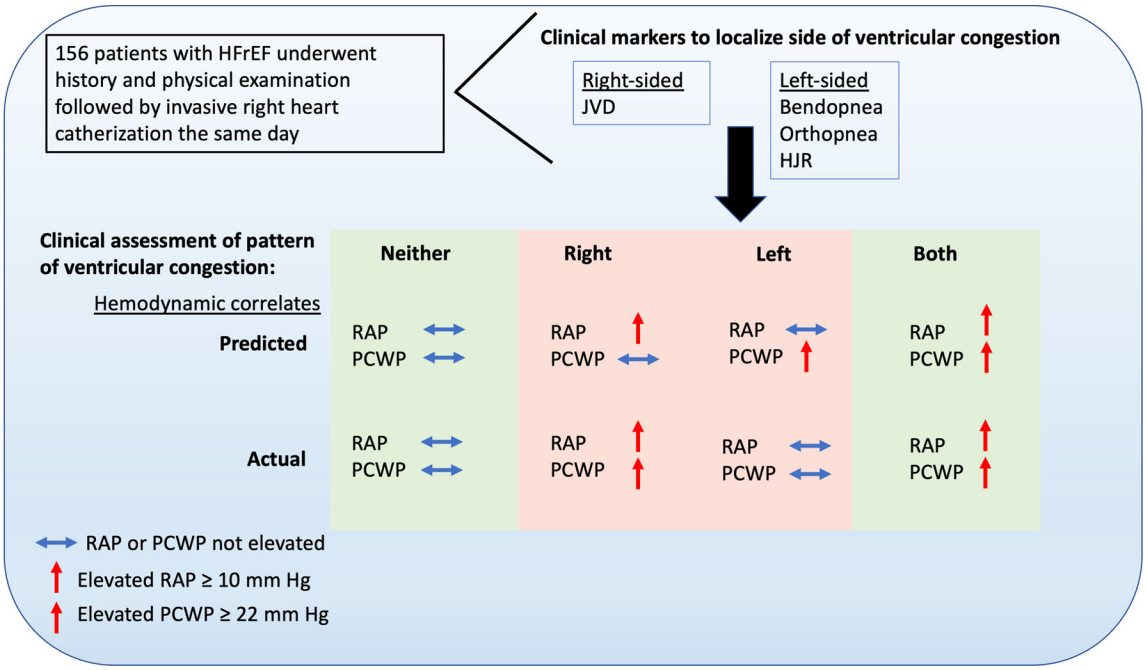

Figure 3.

The clinical examination was not able to accurately identify discordance of right- and left-sided ventricular filling pressures largely because of the strong association of the JVD with the PCWP. Specifically, when JVD was present, the PCWP was usually elevated even if bendopnea, orthopnea, or HJR were not present. Similarly, when JVD was not present, the PCWP was not elevated even if bendopnea, orthopnea, or HJR were present.

Table 1.

Clinical characteristics of subjects by clinical assessment congestion profiles

	No exam findings of congestion n=79	Left-sided congestion (Bendopnea or Orthopnea or HJR) n=38	Right-sided congestion (JVD only) n=7	Both left- and right-sided congestion (JVD and Bendopnea or Orthopnea or HJR) n=32	P value
Male; n (%)	60 (76)	24 (63)	6 (86)	25 (78)	0.41
Age, years; mean (SD)	58 (14)	58 (14)	55 (20)	60 (14)	0.82
Race; n (%)					0.10
Caucasian	51(65)	18 (47)	3 (43)	19 (59)	
Black	25 (32)	16 (42)	3 (43)	7 (22)	
Hispanic	3 (4)	4 (11)	1 (14)	5 (16)	
Asian	0	0	0	1 (3)	
LVEF; mean (SD)	25 (8)	24 (9)	16 (7)	22 (9)	0.0
Ischemic CM; n (%)	25 (32)	14 (37)	2 (29)	10 (31)	0.72
NYHA class; n (%)					0.003
1	3 (4)	1 (3)	0	0	
2	28 (35)	5(13)	4 (57)	3 (9)	
3	45 (57)	26 (68)	2 (29)	26 (81)	
4	3 (4)	6 (16)	1 (14)	3 (9)	
Past medical history; n (%)					
Diabetes	24 (30)	18 (47)	4 (57)	19 (59)	0.02
Hypertension	54 (77)	24 (63)	3 (42)	25 (78)	0.27
Smoker	35 (44)	16(42)	2(29)	15 (47)	0.87
Medications; n (%)					
ACE-I	31 (39)	14 (37)	1 (14)	9 (28)	0.48
ARB	29 (37)	13 (34)	0	6 (19)	0.08
BB	59 (75)	28 (74)	2 (29)	14 (44)	0.002
MRA	36 (46)	20 (53)	1 (14)	13 (41)	0.29
Digoxin	28 (35)	15 (39)	4 (57)	11 (34)	0.70
Diuretic	66 (84)	36 (95)	7 (100)	31 (97)	0.12
Inotrope	16 (20)	5 (13)	4 (57)	10 (31)	0.04
Physical exam findings					
JVP, cm; mean (SD)	7 (2)	8 (2)	14 (1)	14 (2)	N/A
JVD; n (%)	0	0	7(100)	32 (100)	N/A
HJR; n (%)	0	20 (45)	0	24 (75)	N/A
Orthopnea; n (%)	0	19 (50)	0	14 (44)	N/A
Bendopnea; n (%)	0	25 (66)	0	17 (53)	N/A
S3; n (%)	9 (11)	8 (21)	1 (14)	11 (34)	0.04
Rales; n (%)	0	1 (3)	0	3 (9)	0.04

	No exam findings of congestion n=79	Left-sided congestion (Bendopnea or Orthopnea or HJR) n=38	Right-sided congestion (JVD only) n=7	Both left- and right-sided congestion (JVD and Bendopnea or Orthopnea or HJR) n=32	P value
Edema; n (%)	8 (10)	13 (34)	4 (57)	18 (56)	<0.0001
BMI, kg/m^2; n (%)	28.9 (10.9)	26.6 (7.9)	27.2 (5.0)	30.9 (7.2)	0.13
Laboratory data					
Na, mmol/L; mean (SD)	139 (3)	138 (4)	133 (5)	136 (6)	0.007
Hgb, g/dL; mean (SD)	12.8 (2.20)	11.8 (2.2)	10.1 (1.3)	11.5 (2.1)	0.0006
BUN, mg/dL; median (IQR)	23 (17, 30)	25 (18, 34)	38 (20, 88)	40 (26, 69)	0.0001
Cr, mg/dL	1.3 (1.0, 1.8)	1.4 (1.1, 1.7)	1.8 (1.6, 2.7)	1.9 (1.3, 2.5)	0.01
eGFR, mL/min/1.73m^2; median (IQR)	60 (43, 74)	58 (44, 75)	35 (26, 52)	40 (27, 61)	0.01

Data are presented as n (%), mean (SD), or median (IQR).

ACE-I = angiotensin converting enzyme inhibitor; ARB = aldosterone receptor blocker; BB = beta blocker; BMI = body mass index; BUN = blood urea nitrogen; CM = cardiomyopathy; Cr = creatinine; eGFR = estimated glomerular filtration rate; Hgb = hemoglobin; HJR = hepatojugular reflux; JVD = jugular venous distension; JVP = jugular venous pressure; LVEF = left ventricular ejection fraction; Na = sodium; MRA = mineralocorticoid receptor blocker; NTproBNP = N-terminal pro B-type natriuretic peptide; NYHA = New York heart association; S3 = 3rd heart sound N/A Not applicable as definitional

Table 2.

Frequency of hemodynamic profiles of heart failure subjects; n (%).

	PCWP <22mmHg	**PCWP ≥ 22 mmHg**
RAP <10 mmHg	92 (59)	15 (10)
RAP ≥10 mmHg	18 (12)	31 (20)

Table 3.

Invasive hemodynamics stratified by physical examination findings predictive of different congestion patterns

	No exam findings of congestion n=79	Left-sided congestion (HJR or Orthopnea or Bendopnea) n=38	Right-sided congestion (JVD only) n=7	Left- and right-sided congestion (JVD and HJR or Orthopnea or Bendopnea) n=32	p-value across groups
RAP, mmHg	4 (2, 6)	6 (3, 8) *	15 (10, 18) †	15 (12, 18) †	<0.0001
PCWP, mmHg	13 (8, 17)	18 (10, 24) ‡	24 (18, 32) †	24 (20, 30) †	<0.0001
Mean PAP, mmHg	23 (18, 30)	30 (17, 36) ‡	39 (32, 44) †	38 (31, 45) †	<0.0001
CI by TD, L/min/m^2	2.2 (1.8, 2.7)	2.2 (1.8, 2.7)	2.2 (1.8, 2.7)	1.9 (1.6, 2.2)	0.11
CI by indirect Fick, L/min/m^2	2.3 (1.9, 2.7)	2.2 (2.0, 2.6)	2.1 (1.5, 2.5)	2.1 (1.8, 2.4)	0.10
PVR by TD, wood units	2.2 (1.3, 3.0)	2.8 (1.6, 4.0)	2.9 (1.7, 5.1)	3.1 (1.8, 4.1)	0.03
PVR by indirect Fick, wood units	2.2 (1.2, 3.0)	2.8 (1.5, 3.6)	2.8 (2.5, 5.6)	3.0 (2.0, 3.7) †	0.02

Data are presented as median (IQR).

* P < 0.05 vs JVD only, and JVD with either HJR, orthopnea, or bendopnea.

† P<0.05 vs no congestion.

‡ P<0.05 vs. JVD with either HJR, orthopnea, or bendopnea. CI = cardiac index; JVD = jugular venous distension; HJR = hepatojugular reflux; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure; TD = thermodilution

Table 4.

Univariable models of physical examination findings and elevated pulmonary capillary wedge pressure (PCWP ≥ 22mmHg).

	Odds Ratio (95% CI)	P-value
JVD	9.7 (4.3–22.1)	<0.0001
JVP, cm H_2O	1.4 (1.2–1.5)	<0.0001
Orthopnea	3.4 (1.5–7.7)	0.002
Bendopnea	5.6 (2.6–12.0)	<0.0001
HJR	3.7 (1.7–7.7)	0.0006
S3	4.8 (2.1–11.2)	0.0003

HJR = hepatojugular reflux; JVD = jugular venous distension; JVP = jugular venous pressure; S3 = 3rd heart sound

Table 5.

Multivariable regression models of physical examination findings and elevated pulmonary capillary wedge pressure (PCWP ≥ 22mmHg) using jugular venous distension and jugular venous pressure.

	Model using JVD		Model using JVP	
	Odds Ratio (95% CI)	**P-value**	**Odds Ratio (95% CI)**	**P-value**
JVD	8.2 (3.1–22.1)	<0.0001	N/A	N/A
JVP, cm H_2O	N/A	N/A	1.3 (1.2–1.5)	<0.001
Orthopnea	0.9 (0.3–3.1)	0.88	0.8 (0.3–2.8)	0.78
Bendopnea	4.6 (1.5–14.6)	0.009	4.3 (1.4–13.3)	0.01
HJR	1.1 (0.4–2.9)	0.85	1.0 (0.4–2.6)	0.93
S3	3.7 (1.3–10.4)	0.01	3.3 (1.2–8.9)	0.02

HJR = hepatojugular reflux; JVD = jugular venous distension; JVP = jugular venous pressure

TABLE 6.

Agreement between the clinical and invasive hemodynamic congestion profiles.

		Physical examination derived congestion pattern			
		No exam findings of congestion (No congestion) N=79	**HJR or Orthopnea or Bendopnea (Left-sided congestion) N=38**	**JVD only (Right-sided congestion) N=7**	**JVD and HJR or Orthopnea or Bendopnea (Both right- and left-sided congestion) N=32**
Invasive hemodynamic derived congestion pattern	**RAP <10 mmHg, PCWP <22mmHg (No congestion)**	**67** *	24†	0	1
	RAP <10 mmHg, PCWP ≥22 mmHg (Left-sided congestion)	4	**11** *	0	0
	RA ≥ 10mmHg PCWP <22 mmHg (Right-sided congestion)	4	2	**2** *	10†
	RA ≥ 10mmHg PCWP ≥ 22mmHg (Both left and right sided congestion)	4	1	5†	**21** *

κ = 0.44 (95% CI 0.34–0.55)

HJR = hepatojugular reflux; JVD = jugular venous distension; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure

Number of subjects are shown.

*Represents agreement between the physical exam and invasive hemodynamic categorization.

†Highlights key discrepancies between the clinical and invasive hemodynamic categorizations of right and left sided ventricular filling pressures.