A Propensity-Matched Study of Elevated Jugular Venous Pressure and Outcomes in Chronic Heart Failure

Philippe Meyer, MD[a], O. James Ekundayo, MD, DrPH[b], Chris Adamopoulos, MD, PhD[c],
Marjan Mujib, MBBS[b], Inmaculada Aban, PhD[b], Michel White, MD[a], Wilbert S. Aronow, MD[d], and
Ali Ahmed, MD, MPH[b,e,]*

The independence of association between elevated jugular venous pressure (JVP) and outcomes in heart failure (HF) has not been well studied. The objective of propensity-matched study was to determine if an elevated JVP had intrinsic associations with outcomes in chronic systolic and diastolic HF. Of the 7,788 participants in the Digitalis Investigation Group trial, 1,020 (13%) had elevated JVP at baseline. Propensity scores for elevated JVP were estimated for all patients based on 32 baseline characteristics and were used to match 827 pairs of patients with normal and elevated JVP. Hazard ratios (HR) and 95% confidence intervals (CI) were estimated to compare outcomes associated with elevated versus normal JVP during 34 months of median follow-up. Before matching, all-cause mortality occurred in 31% and 47% (unadjusted HR 1.70, 95% CI 1.54 to 1.88, p <0.0001), and all-cause hospitalization occurred in 60% and 71% (unadjusted HR 1.35, 95% CI 1.25 to 1.47, p <0.0001) of patients with normal and elevated JVP, respectively. After matching, all-cause mortality occurred in 48% and 45% (matched HR 0.95, 95% CI 0.80 to 1.12, p = 0.521), and all-cause hospitalization occurred in 70% and 70% (matched HR 0.97, 95% CI 0.87 to 1.09, p = 0.613) of patients with normal and elevated JVP, respectively. Elevated JVP had no intrinsic associations with cardiovascular mortality (matched HR 0.93, 95% CI 0.77 to 1.12, p = 0.440) or hospitalization for HF (matched HR 0.94, 95% CI 0.78 to 1.14, p = 0.532). In conclusion, an elevated JVP is a marker of higher burden of sickness and poor outcomes. However, elevated JVP had no intrinsic association with mortality or hospitalization in chronic HF.   © 2009 Published by Elsevier Inc. (Am J Cardiol 2009;103:839–844)

The assessment of fluid volume status is of crucial importance in patients with chronic heart failure (HF), and estimation of jugular venous pressure (JVP) is one of the most reliable means of assessing fluid volume.[1] However, little is known about the association of elevated JVP and outcomes in chronic HF. In one study, elevated JVP was independently associated with adverse outcomes in chronic systolic HF.[2] However, this association has not been validated in other similar populations. The objective of the present study was to determine whether baseline elevated JVP was associated with poor HF outcomes in a propensity-matched population of ambulatory chronic systolic and diastolic HF in which patients with normal and elevated JVP would be well-balanced in all measured baseline covariates.

Methods

We used a public-use copy of the Digitalis Investigation Group (DIG) dataset obtained from the National Heart Lung and Blood Institute. The rationale, design, and results of the DIG trial have been previously reported.[3–6] Briefly, 7,788 ambulatory patients with chronic HF in normal sinus rhythm were randomly assigned to receive digoxin or placebo. These patients were recruited from 302 clinical centers in the United States (186) and Canada (116) between 1991 and 1993 and followed for a mean length of 37 months. Most patients were receiving diuretics and angiotensin-converting enzyme inhibitors, and 6,800 (87%) had left ventricular ejection fraction <45%. Elevated JVP was present in 1,020 patients (13%) at the time of randomization or within the previous 30 days. Elevated JVP was estimated by study investigators by physical examination and was described as jugular venous distension. In this report, we use the term *elevated JVP*, and data on elevated JVP were available from all 7,788 patients. The primary outcomes for the current analysis were mortality and hospitalizations due to all causes; other outcomes studied included mortality and hospitalizations due to cardiovascular causes and HF. Data on vital status were 99% complete.[7]

[a]Montreal Heart Institute, Montreal, Canada; [b]University of Alabama at Birmingham, Birmingham, Alabama; [c]Papageorgiou General Hospital, Thessaloniki, Greece; [d]New York Medical College, Valhalla, New York; and [e]VA Medical Center, Birmingham, Alabama. Manuscript received August 27, 2008; revised manuscript received and accepted November 18, 2008.

*Corresponding author: Tel: 205-934-9632; fax: 205-975-7099.

E-mail address: aahmed@uab.edu (A. Ahmed).

Table 1
Baseline patient characteristics by elevated JVP status before and after propensity matching

Variable	Before Matching			After Matching		
	Normal JVP (n = 6,768)	Elevated JVP (n = 1,020)	p Value	Normal JVP (n = 827)	Elevated JVP (n = 827)	p Value
Age (yr)	64 ± 11	65 ± 11	0.001	65 ± 12	65 ± 11	0.335
Women	1,660 (25%)	266 (26%)	0.284	229 (28%)	219 (27%)	0.629
Nonwhite	915 (14%)	213 (21%)	<0.0001	147 (18%)	150 (18%)	0.900
Body mass index (kg/m²)	27 ± 5	27 ± 5	0.258	28 ± 6	27 ± 5	0.195
Duration of HF (months)	30 ± 36	31 ± 39	0.130	32 ± 39	33 ± 40	0.761
Primary cause of HF						
Ischemic	4,724 (70%)	636 (62%)		511 (62%)	525 (64%)	
Hypertensive	679 (10%)	126 (12%)	<0.0001	119 (14%)	101 (12%)	0.723
Idiopathic	953 (14%)	158 (16%)		135 (16%)	126 (15%)	
Others	412 (6%)	100 (10%)		62 (8%)	75 (9%)	
Previous myocardial infarction	4,344 (64%)	564 (55%)	<0.0001	458 (55%)	472 (57%)	0.523
Current angina pectoris	1,846 (27%)	269 (26%)	0.546	239 (29%)	230 (28%)	0.659
Hypertension	3,142 (46%)	532 (52%)	0.001	434 (53%)	419 (51%)	0.486
Diabetes mellitus	1,870 (28%)	348 (34%)	<0.0001	268 (32%)	270 (33%)	0.958
Medications						
Pretrial digoxin use	2,887 (43%)	478 (47%)	0.011	387 (47%)	390 (47%)	0.921
Trial use of digoxin	3,391 (50%)	498 (49%)	0.446	408 (49%)	407 (49%)	1.000
ACE inhibitors	6,314 (93%)	960 (94%)	0.322	769 (93%)	779 (94%)	0.363
Diuretics	5,155 (76%)	921 (90%)	<0.0001	729 (88%)	732 (89%)	0.873
Potassium-sparing diuretics	509 (8%)	87 (9%)	0.259	80 (10%)	70 (9%)	0.447
Potassium supplement	1,854 (27%)	345 (34%)	<0.0001	281 (34%)	281 (34%)	1.000
Symptoms and signs of HF						
Dyspnea at rest	1,179 (17%)	526 (52%)	<0.0001	343 (42%)	349 (42%)	0.785
Dyspnea on exertion	4,943 (73%)	919 (90%)	<0.0001	739 (89%)	727 (88%)	0.372
Third heart sound	1,320 (20%)	526 (52%)	<0.0001	373 (45%)	369 (45%)	0.871
Pulmonary rales	766 (11%)	535 (53%)	<0.0001	355 (43%)	348 (42%)	0.689
Lower extremity edema	1,107 (16%)	526 (52%)	<0.0001	374 (45%)	350 (42%)	0.203
NYHA class						
Class I	1,045 (15%)	58 (6%)		53 (6%)	55 (7%)	
Class II	3,856 (57%)	388 (38%)	<0.0001	342 (41%)	337 (41%)	0.889
Class III	1,787 (26%)	500 (49%)		396 (48%)	389 (47%)	
Class IV	80 (1%)	74 (7%)		36 (4%)	46 (6%)	
Heart rate (beats/min)	78 ± 12	83 ± 14	<0.0001	82 ± 13	82 ± 14	0.417
Blood pressure (mm Hg)						
Systolic	128 ± 20	125 ± 21	0.001	126 ± 21	126 ± 21	0.651
Diastolic	75 ± 11	75 ± 12	0.622	75 ± 12	75 ± 12	0.449
Chest radiograph findings						
Pulmonary congestion	679 (10%)	430 (42%)	<0.0001	258 (31%)	265 (32%)	0.709
Cardiothoracic ratio >0.5	3,924 (58%)	766 (75%)	<0.0001	606 (73%)	604 (73%)	0.954
Serum creatinine (mg/dl)	1.27 ± 0.36	1.34 ± 0.41	<0.0001	1.33 ± 0.40	1.33 ± 0.41	0.793
Serum potassium (mEq/L)	4.34 ± 0.44	4.33 ± 0.45	0.366	4.34 ± 0.45	4.32 ± 0.45	0.351
Ejection fraction (%)	33 ± 13	28 ± 12	<0.0001	30 ± 13	29 ± 12	0.491

Data presented as n (%) or mean ± SD.

ACE = angiotensin-converting enzyme; NYHA = New York Heart Association.

Because of significant imbalances in baseline covariates between patients with and without elevated JVP (Table 1), we used propensity score matching to assemble a cohort of patients who would be well-balanced in all measured baseline covariates.[3–6] We estimated propensity scores for elevated JVP for each of the 7,788 patients using a nonparsimonious, multivariate logistic regression model, adjusting for all available baseline covariates presented in Figure 1. Propensity score models are sample-specific adjusters and are not intended to be used for out-of-sample prediction or estimation of coefficients. Therefore, instead of fitness and discrimination, a propensity model's effectiveness is better assessed by its ability to reduce bias after matching. Using a greedy matching protocol, we matched 827 pairs of patients with and without elevated JVP who had similar propensity scores.[8] The details of the matching protocol have been described elsewhere.[9–12] We then objectively estimated postmatch bias reduction using absolute standardized differences (<10% being inconsequential bias and 0% indicating no residual bias) and presented them as a Love plot.[12–15]

For descriptive analyses, we used Pearson's chi-square and Wilcoxon rank-sum tests for prematch and McNemar's test and paired sample *t* test for postmatch comparisons, as appropriate. Kaplan-Meier and matched Cox regression analyses were used to determine the association of elevated JVP
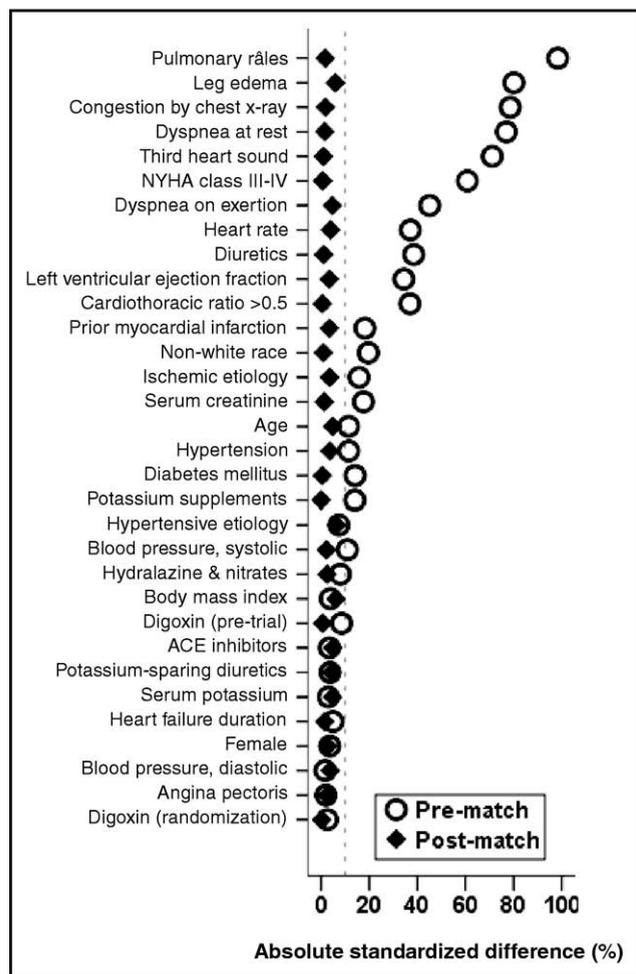
Figure 1. Love plot displaying absolute standardized differences for co-variates between chronic HF patients with and without elevated JVP before and after propensity score matching. ACE = angiotensin-converting enzyme; NYHA = New York Heart Association.

relative to normal JVP with various outcomes. Subgroup analyses and first-order interactions were used to test the heterogeneity of the association between elevated JVP and all-cause mortality. Chronic kidney disease was defined as an estimated glomerular filtration rate <60 ml/min per 1.73 m^2 of body surface area. All statistical tests were done using SPSS for Windows, version 15 (SPSS, Chicago, Illinois).

Results

Prematch imbalances in baseline covariates and balances achieved after matching are displayed in Table 1 and Figure 1. Patients with elevated JVP were older, more likely to be nonwhite, and generally had a higher burden of symptoms and co-morbidities, all of which were balanced after matching (Table 1). Values of absolute standardized differences for all covariates after matching between patients with normal and elevated JVP were <10% (Figure 1).

In the prematch cohort, all-cause mortality occurred in 31% (rate 1,054/10,000 person-years) and 47% (rate 1,789/10,000 person-years) of patients with normal and elevated JVP, respectively (when elevated JVP is compared with

normal JVP, unadjusted hazard ratio [HR] 1.70, 95% confidence interval [CI] 1.54 to 1.88, p <0.0001; Table 2). This association lost significance when adjusted for propensity score (propensity-adjusted HR 1.00, 95% CI 0.88 to 1.14, p = 0.963). The association of elevated JVP with cardiovascular and HF mortalities are listed in Table 2. All-cause hospitalization occurred in 60% (rate 3,664/10,000 person-years) and 71% (rate 5,186/10,000 person-years) of patients with normal and elevated JVP, respectively (unadjusted HR 1.35, 95% CI 1.25 to 1.47, p <0.0001; Table 3). This association lost significance when adjusted for propensity score (propensity-adjusted HR 1.02, 95% CI 0.93 to 1.12, p = 0.701). The association of elevated JVP with cardiovascular and HF hospitalizations are listed in Table 3.

In the postmatch cohort, all-cause mortality occurred in 48% (rate 1,866/10,000 person-years) and 45% (rate 1,699/10,000 person-years) of patients with normal and elevated JVP, respectively (matched HR 0.95, 95% CI 0.80 to 1.12, p = 0.521; Table 2 and Figure 2). The association of elevated JVP with cardiovascular and HF mortalities are listed in Table 2. The association between elevated JVP and all-cause mortality was homogeneous across a wide spectrum of subgroups except for gender (Figure 3). All-cause hospitalization occurred in 70% (rate 5,056/10,000 person-years) and 70% (rate 4,882/10,000 person-years) of patients with normal and elevated JVP, respectively (matched HR 0.97, 95% CI 0.87 to 1.09, p = 0.613; Table 3 and Figure 2). The association of elevated JVP with cardiovascular and HF hospitalizations are listed in Table 3.

Discussion

The findings from the current analysis suggest that elevated JVP was a marker of increased mortality and morbidity in ambulatory patients with chronic HF. However, data from our propensity-matched population in which patients with and without elevated JVP were well balanced in all measured baseline characteristics suggest that elevated JVP had no intrinsic association with outcomes in these patients. These findings are important as elevated JVP is the most reliable sign of fluid overload and can be used to identify patients with HF who are at risk for poor outcomes.

Unadjusted associations between elevated JVP and outcomes are likely due to many prematch imbalances on key prognostic variables between patients with normal and elevated JVP. Patients with elevated JVP were more likely to be older, have diabetes mellitus, renal insufficiency, cardiomegaly, lower mean left ventricular ejection fraction, higher New York Heart Association class symptoms, and receive diuretics, all of which are markers of poor prognosis in these patients.[10–12,15,16–18] This is further confirmed as this association completely disappeared in the propensity-matched cohort and also when adjusted for propensity scores in the prematch cohort, suggesting that an elevated JVP is a marker of poor prognosis and does not have any intrinsic prognostic value of its own. This lack of an independent association of elevated JVP with outcomes in chronic HF is mechanistically plausible. The JVP is an indirect clinical measure of right atrial pressure and may reflect left ventricular filling pressure. Although these hemodynamic characteristics have been shown to be associated with poor prog-

Table 2
Association between elevated JVP and mortality in chronic HF before and after propensity matching

Outcomes	Rate Per 10,000 Person-Years (Events/Total Follow-Up Years)		Absolute Rate Difference* (Per 10,000 Person-Years)	HR (95% CI)	p Value
Prematch	Normal JVP (n = 6,768)	Elevated JVP (n = 1,020)			
All-cause	1,054 (2,131/20,219)	1,789 (475/2,655)	+735	1.70 (1.54–1.88)	<0.0001
Cardiovascular	829 (1,677/20,219)	1,412 (375/2,655)	+583	1.70 (1.52–1.90)	<0.0001
Progressive HF	353 (714/20,219)	727 (193/2,655)	+374	2.07 (1.76–2.43)	<0.0001
Postmatch	Normal JVP (n = 827)	Elevated JVP (n = 827)			
All-cause	1,866 (398/2,133)	1,699 (373/2,196)	−167	0.95 (0.80–1.12)	0.521
Cardiovascular	1,467 (313/2,133)	1,321 (290/2,196)	−147	0.93 (0.77–1.12)	0.440
Progressive HF	727 (155/2,133)	660 (145/2,196)	−66	0.94 (0.71–1.23)	0.628

* Absolute differences in rates of events per 10,000 person-years of follow-up were calculated by subtracting the event rates in the normal JVP group from the event rates in the elevated JVP group (before values were rounded).

Table 3
Association between elevated JVP and hospitalizations* in chronic HF before and after propensity matching

Outcomes	Rate Per 10,000 Person-Years (Events/Total Follow-Up Years)		Absolute Rate Difference† (Per 10,000 Person-Years)	HR (95% CI)	p Value
Prematch	Normal JVP (n = 6,768)	Elevated JVP (n = 1,020)			
All-cause	3,664 (4,044/12,019)	5,186 (724/1,396)	+1,522	1.35 (1.25–1.47)	<0.0001
Cardiovascular	2,402 (3,416/14,221)	3,576 (594/1,661)	+1,174	1.42 (1.31–1.55)	<0.0001
Worsening HF	1,086 (1,888/17,387)	1,931 (399/2,066)	+845	1.71 (1.53–1.90)	<0.0001
Postmatch	Normal JVP (n = 827)	Elevated JVP (n = 827)			
All-cause	5,056 (583/1,153)	4,882 (578/1,184)	−175	0.97 (0.87–1.09)	0.613
Cardiovascular	3,508 (483/1,377)	3,338 (470/1,408)	−170	1.02 (0.87–1.19)	0.841
Worsening HF	1,890 (319/1,688)	1,813 (314/1,732)	−77	0.94 (0.78–1.14)	0.532

* Data shown include the first hospitalization of each patient for each cause.

† Absolute differences in rates of events per 10,000 person-years of follow-up were calculated by subtracting the event rates in the normal JVP group from the event rates in the elevated JVP group (before values were rounded).

nosis,[19–21] these studies were based on small number of patients with systolic HF with short follow-up and did not adjust for key prognostically important covariates.

To the best of our knowledge, this is the first study of associations of elevated JVP and outcomes in a propensity-matched population of chronic systolic and diastolic HF. An analysis of the participants in the Studies of Left Ventricular Dysfunction treatment trial compared the outcomes of 280 patients with chronic systolic HF and elevated JVP with those of 2,199 patients with normal JVP.[2] Although elevated JVP had no independent association with all-cause mortality in that study, it was associated with HF mortality and HF hospitalization. Despite many similarities in baseline characteristics between patients in that analysis and the current analysis, the use of propensity score matching design, the use of a more comprehensive list of variables, and the inclusion of patients with systolic and diastolic HF distinguish our study from that study.

The strong bivariate associations of elevated JVP with major natural history end points in chronic systolic and diastolic HF in our study suggest that an elevated JVP is an excellent marker of poor outcomes in these patients. Fur-

ther, an elevated JVP is the most reliable sign of fluid overload in HF. However, proper estimation of JVP remains a challenge, and an emphasis on the use of the internal jugular vein may likely underestimate elevated JVP in these patients, which was evident from the low prevalence of elevated JVP in our study. A similar low prevalence of elevated JVP has also been reported in patients with HF with acute dyspnea in the emergency department or in the hospital.[22,23] This low prevalence of elevated JVP may be because the internal jugular vein is behind the sternocleidomastoid muscle in the neck and may not be clearly visible in chronic HF.[24] An alternative approach may be to use the external jugular vein, keeping in mind its limitation as a superficial vein.[25] Therefore, a distended external jugular vein is unreliable unless the venous pulsation can be seen, the top of which should be used to estimate JVP. The distance between right atrium and sternal angle varies with body position and should be taken into account when estimating JVP.[26]

Several limitations of our study must be acknowledged. DIG participants were predominantly young men in normal sinus rhythm before the β-blocker era of HF therapy, which
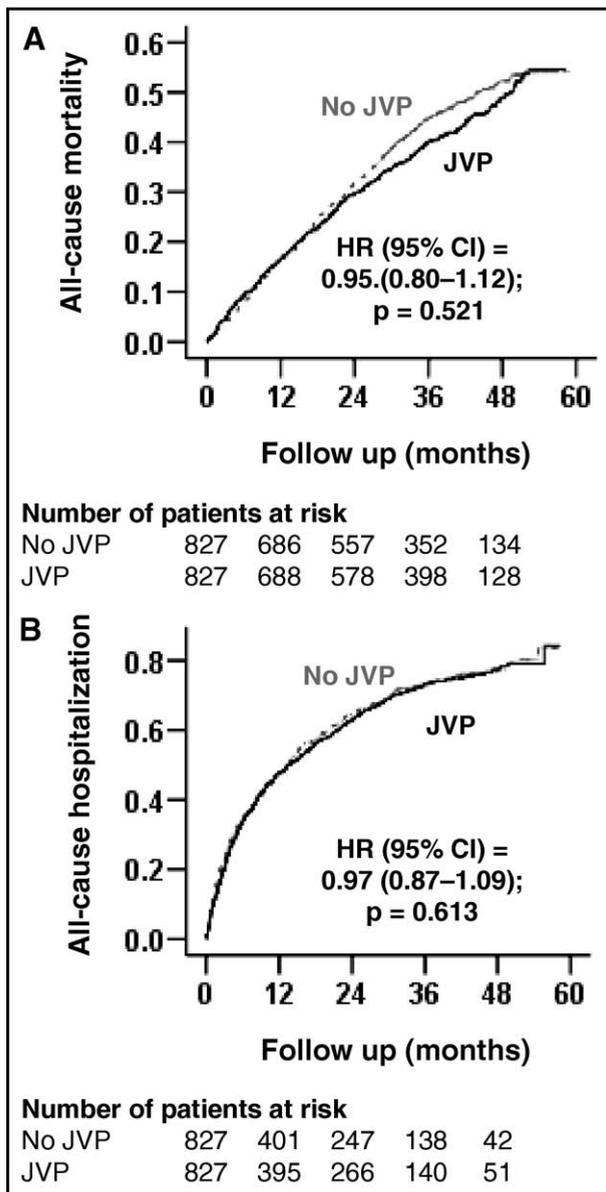
Figure 2. Kaplan-Meier plots for *(A)* all-cause mortality and *(B)* all-cause hospitalization.

may limit generalizability. The low prevalence of elevated JVP at baseline indicates that many patients with elevated JVP may have been misclassified as having normal JVP, which may have underestimated the true association. However, the prevalence of elevated JVP in DIG participants was very similar to that of the participants in the Studies of Left Ventricular Dysfunction trial.[2] In conclusion, despite the lack of an intrinsic association between an elevated JVP and outcomes, because of its strong and significant bivariate association, an elevated JVP will remain a useful marker of prognosis in chronic systolic and diastolic HF. The usefulness of JVP may be enhanced by routine assessment of JVP in patients with HF.

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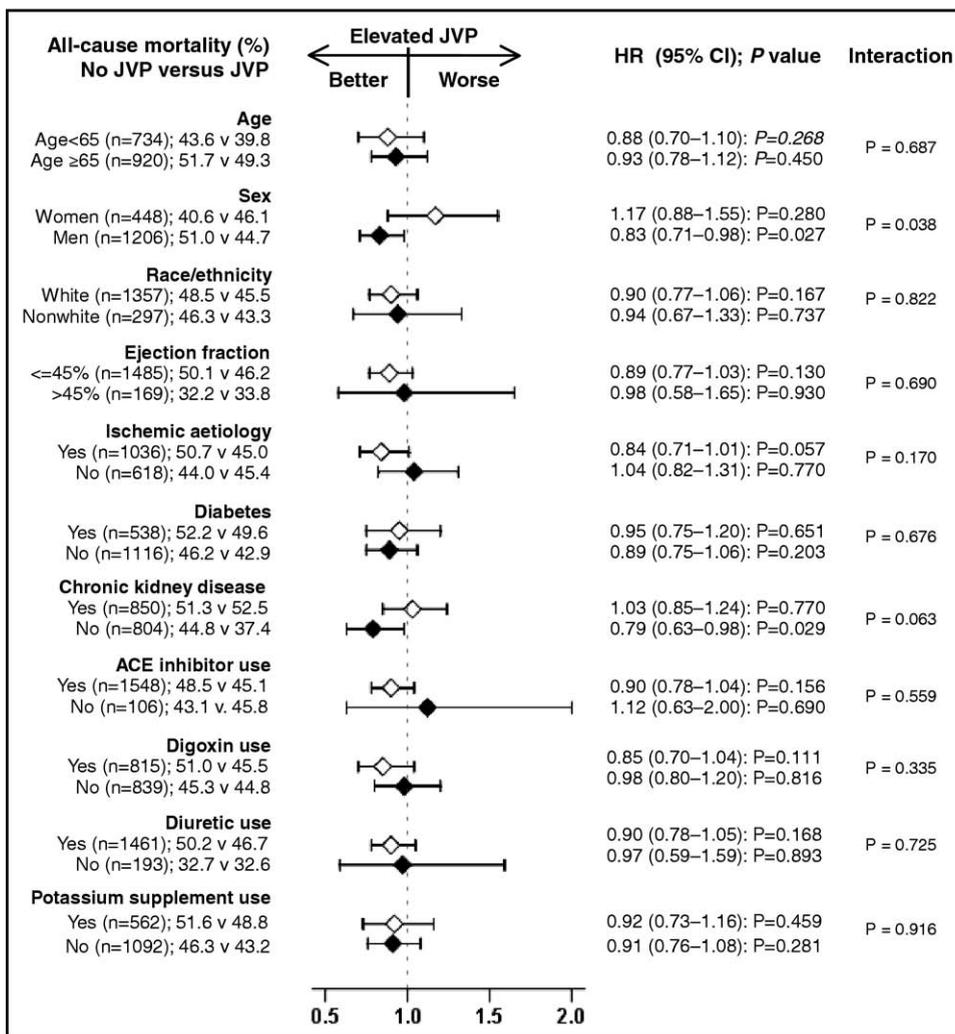
Figure 3. Association of elevated JVP with all-cause mortality in subgroups of propensity-matched patients with chronic HF. ACE = angiotensin-converting enzyme.

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