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

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Abstract

The independence of association between elevated jugular venous pressure (JVP) and outcomes in heart failure (HF) has not been well studied. The objective of this propensity-matched study was to determine if an elevated JVP had intrinsic associations with outcomes in chronic systolic and diastolic HF. Of the 7788 Digitalis Investigation Group trial participants 1020 (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, 1.54–1.88; P<0.0001) and all-cause hospitalization occurred in 60% and 71% (unadjusted HR, 1.35; 95% CI, 1.25–1.47; P<0.0001) of normal and elevated JVP patients respectively. After matching, all-cause mortality occurred in 48% and 45% (matched HR, 0.95; 95% CI, 0.80–1.12; P=0.521) and all-cause hospitalization occurred in 70% and 70% (matched HR, 0.97; 95% CI, 0.87–1.09; P=0.613) of normal and elevated JVP patients respectively. Elevated JVP had no intrinsic associations with cardiovascular mortality (matched HR, 0.93; 95% CI, 0.77-1.12; P=0.440) or HF hospitalization (matched HR, 0.94; 95% CI, 0.78-1.14; P=0.532). In conclusion, an elevated JVP is a marker of higher burden of sickness and poor outcomes. However, elevated JVP had not intrinsic association with mortality or hospitalization in chronic HF.

Keywords

Heart failure; Jugular venous pressure; Mortality; Morbidity; Outcomes

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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 this 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, 7788 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 US (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 6800 (87%) had left ventricular ejection fraction <45%. Elevated JVP was present in 1020 (13%) patients 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 manuscript we will use the term elevated JVP and data on elevated JVP were available from all 7788 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]

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 7788 patients using a non-parsimonious, 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 post-match 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 Chi square and Wilcoxon rank-sum tests for pre-match, and McNemar's test and paired sample t-test for post-match comparisons, as appropriate. Kaplan-Meier and matched Cox regression analyses were used to determine the association of elevated JVP (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-15 for Windows.[16]

Results

Pre-match 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 higher burden of symptoms and comorbidities 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 pre-match cohort, all-cause mortality occurred in 31% (rate, 1054/10000 person-years) and 47% (rate, 1789/10000 person-years) of patients with normal and elevated JVP respectively (unadjusted hazard ratio {HR} when elevated JVP is compared with normal JVP, 1.70; 95% confidence interval {CI}, 1.54–1.88; P<0.0001; Table 2). This association lost significance when adjusted for propensity score (propensity-adjusted HR, 1.00; 95% CI, 0.88–1.14; P=0.963). The association of elevated JVP with cardiovascular and HF mortalities are displayed in Table 2. All-cause hospitalization occurred in 60% (rate, 3664/10000 person-years) and 71% (rate, 5186/10000 person-years) of patients with normal and elevated JVP respectively (unadjusted HR, 1.35; 95% CI, 1.25–1.47; P<0.0001; Table 3). This association lost significance when adjusted for propensity score (propensity-adjusted HR, 1.02; 95% CI, 0.93–1.12; P=0.701). The association of elevated JVP with cardiovascular and HF hospitalizations are displayed in Table 3.

In the post-match cohort, all-cause mortality occurred in 48% (rate, 1866/10000 person-years) and 45% (rate, 1699/10000 person-years) of patients with normal and elevated JVP respectively (matched HR, 0.95; 95% CI, 0.80–1.12; P=0.521; Table 2 and Figure 2a). The association of elevated JVP with cardiovascular and HF mortalities are displayed in Table 2. The association between elevated JVP and all-cause mortality was homogeneous across a wide spectrum of subgroups except for the one by gender (Figure 3). All-cause hospitalization occurred in 70% (rate, 5056/10000 person-years) and 70% (rate, 4882/10000 person-years) of patients with normal and elevated JVP respectively (matched HR, 0.97; 95% CI, 0.87–1.09; P=0.613; Table 3 and Figure 2b). The association of elevated JVP with cardiovascular and HF hospitalizations are displayed 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 HF patients who are at risk for poor outcomes.

Unadjusted associations between elevated JVP and outcomes are likely due to many pre-match 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 symptom, and receive diuretics, all of which are markers of poor prognosis in these patients.[10-12, 15, 17-19]

This is further confirmed when this association completely disappeared in the propensity-matched cohort and also when adjusted for propensity scores in the pre-matched cohort, which suggest 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 parameters have been shown to be associated with poor prognosis,[20-22] these studies were based on small number of systolic HF patients 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 (SOLVD) treatment trial compared the outcomes of 280 chronic systolic HF patients with elevated JVP with those of 2199 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 both systolic and diastolic HF patients distinguish our study from that study.

The strong bivariate associations of elevated JVP with major natural history endpoints in chronic systolic and diastolic HF in our study suggest that an elevated JVP is an excellent marker of poor outcomes in these patients. Further, 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 HF patients with acute dyspnea in the emergency department or in the hospital.[23, 24] This low prevalence of elevated JVP may be due to the fact that the internal jugular vein is behind the sternocleidomastoid muscle in the neck and may not be clearly visible in chronic HF.[25] An alternative approach may be to use the external jugular vein, keeping in mind its limitation as a superficial vein.[26] 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 while estimating JVP.[27]

Several limitations of our study must be acknowledged. DIG participants were predominantly young men in normal sinus rhythm from the pre-beta-blocker era of HF therapy which may limit generalizability. The low prevalence of elevated JVP at baseline indicate 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 SOLVD participants.[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 all patients with HF

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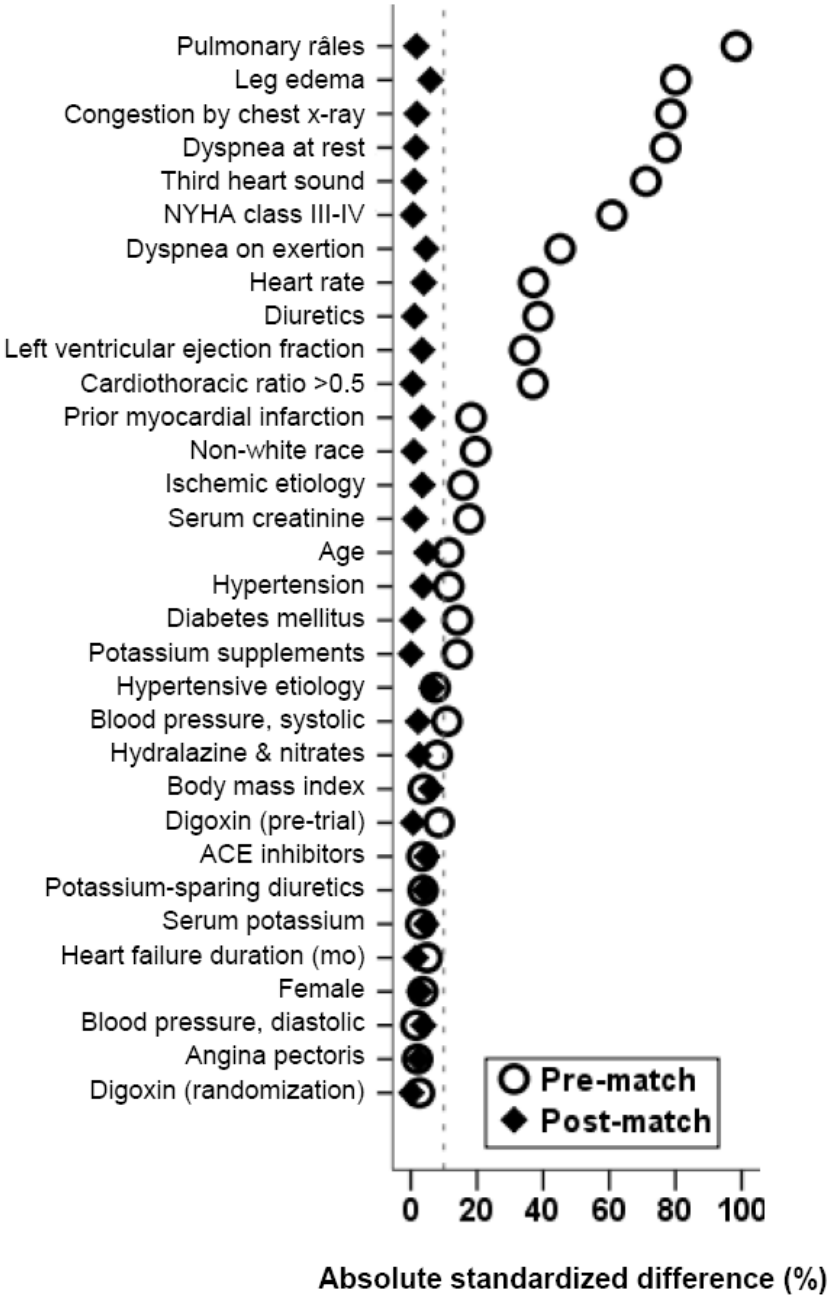

Figure 1.
Love plot displaying absolute standardized differences for covariates between chronic heart failure patients with and without elevated jugular venous pressure, before and after propensity score matching (ACE=angiotensin-converting enzyme; NYHA=New York Heart Association)

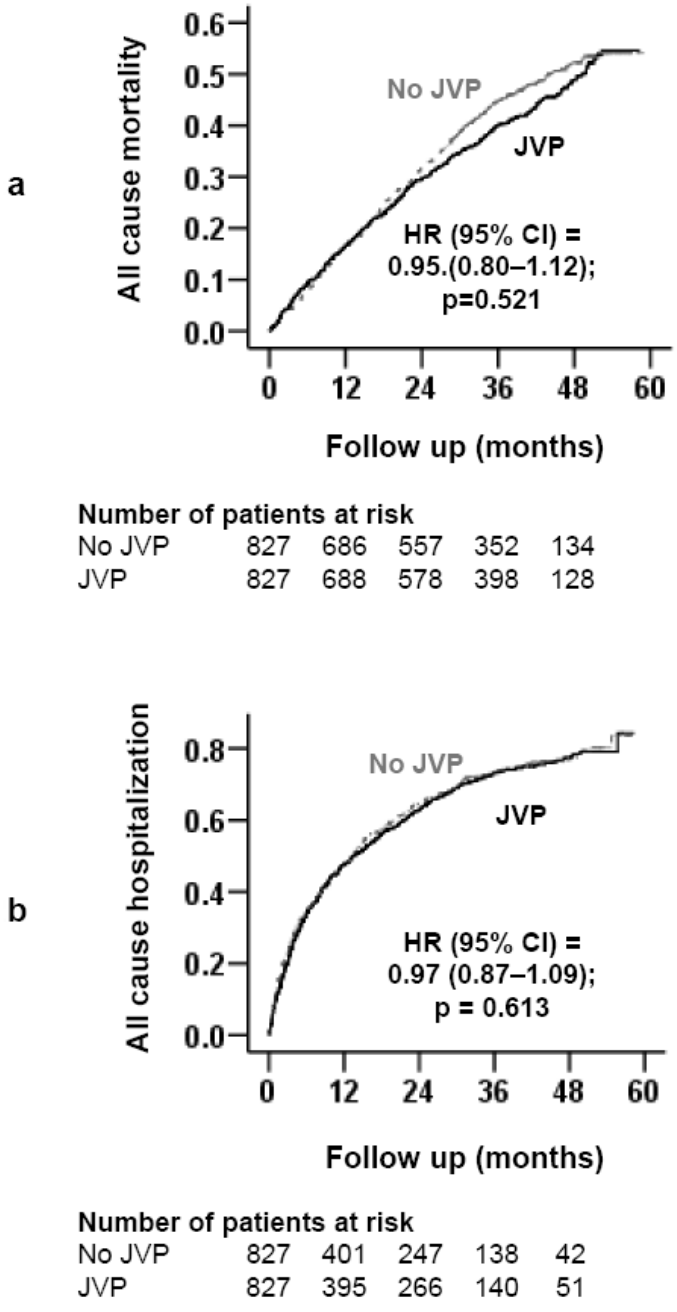

Figure 2.
Kaplan-Meier plots for (a) all-cause mortality, and (b) all-cause hospitalization (CI=confidence interval; HR=hazard ratio; JVP=jugular venous pressure)

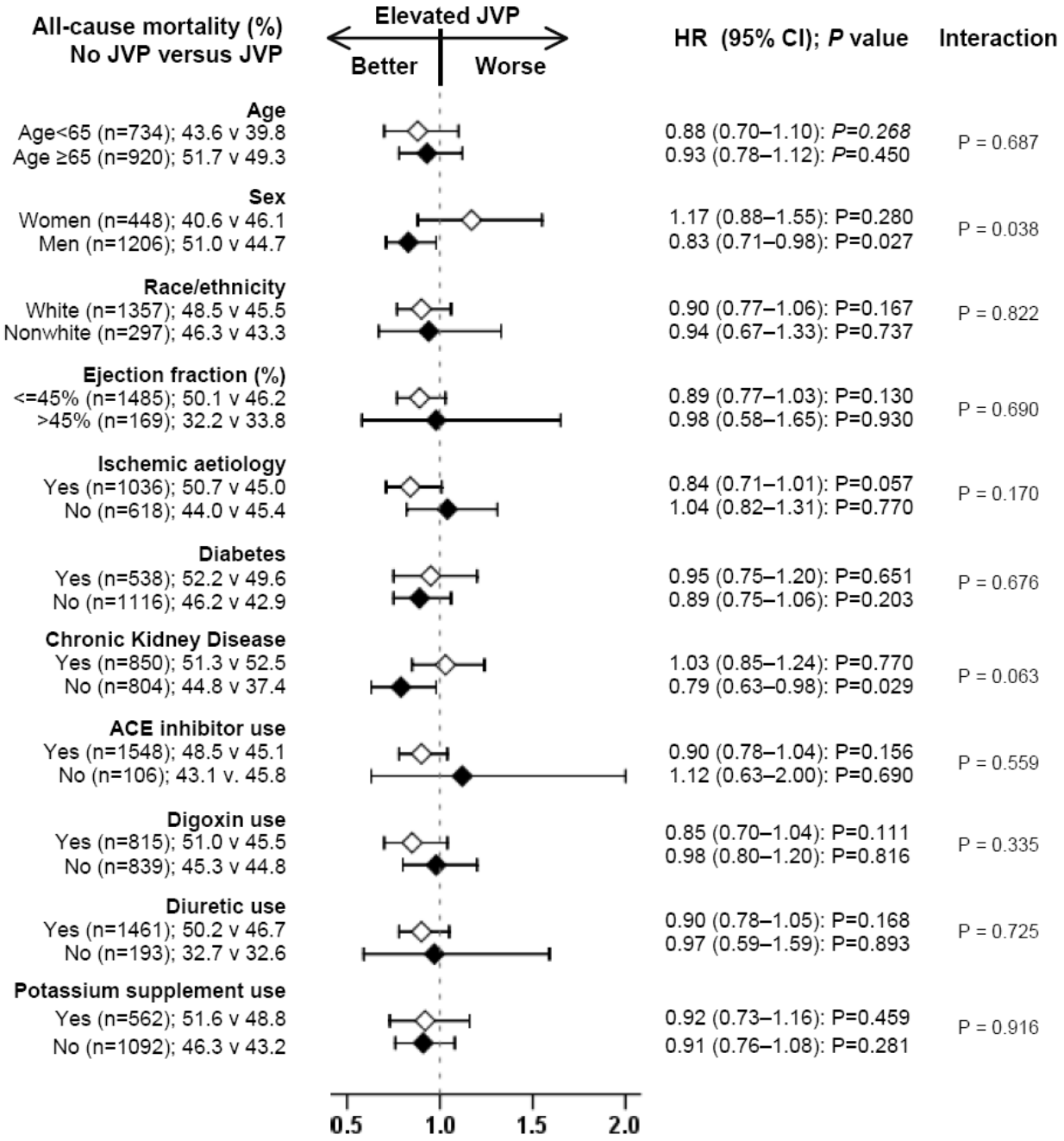

Figure 3.

Association of elevated jugular venous pressure (JVP) with all-cause mortality in subgroups of propensity-matched chronic heart failure patients (ACE=angiotensin-converting enzyme; CI=confidence interval; HR=hazard ratio)

Table 1

Baseline patient characteristics, by elevated jugular venous pressure (JVP) status, before and after propensity matching

Variable	Before matching			After matching		
	Normal JVP (n = 6768)	Elevated JVP (n = 1020)	P Value	Normal JVP (n = 827)	Elevated JVP (n = 827)	P Value
Age (year)	64 ± 11	65 ± 11	0.001	65 ± 12	65 ± 11	0.335
Female	1660 (25%)	266 (26%)	0.284	229 (28%)	219 (27%)	0.629
Non-white	915 (14%)	213 (21%)	<0.0001	147 (18%)	150 (18%)	0.900
Body mass index (kg/m^2)	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	4724 (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%)	
Prior myocardial infarction	4344 (64%)	564 (55%)	<0.0001	458 (55%)	472 (57%)	0.523
Current angina pectoris	1846 (27%)	269 (26%)	0.546	239 (29%)	230 (28%)	0.659
Hypertension	3142 (46%)	532 (52%)	0.001	434 (53%)	419 (51%)	0.486
Diabetes mellitus	1870 (28%)	348 (34%)	<0.0001	268 (32%)	270 (33%)	0.958
Medications						
Pre-trial digoxin use	2887 (43%)	478 (47%)	0.011	387 (47%)	390 (47%)	0.921
Trial use of digoxin	3391 (50%)	498 (49%)	0.446	408 (49%)	407 (49%)	1.000
ACE inhibitors	6314 (93%)	960 (94%)	0.322	769 (93%)	779 (94%)	0.363
Diuretics	5155 (76%)	921 (90%)	<0.0001	729 (88%)	732 (89%)	0.873
PS diuretics	509 (8%)	87 (9%)	0.259	80 (10%)	70 (9%)	0.447
Potassium supplement	1854 (27%)	345 (34%)	<0.0001	281 (34%)	281 (34%)	1.000
Symptoms and signs of HF						
Dyspnea at rest	1179 (17%)	526 (52%)	<0.0001	343 (42%)	349 (42%)	0.785
Dyspnea on exertion	4943 (73%)	919 (90%)	<0.0001	739 (89%)	727 (88%)	0.372
Third heart sound	1320 (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	1107 (16%)	526 (52%)	<0.0001	374 (45%)	350 (42%)	0.203

Variable	Before matching			After matching		
	Normal JVP (n = 6768)	Elevated JVP (n = 1020)	P Value	Normal JVP (n = 827)	Elevated JVP (n = 827)	P Value
NYHA class, %						
Class I	1045 (15%)	58 (6%)	<0.0001	53 (6%)	55 (7%)	0.889
Class II	3856 (57%)	388 (38%)		342 (41%)	337 (41%)	
Class III	1787 (26%)	500 (49%)		396 (48%)	389 (47%)	
Class IV	80 (1%)	74 (7%)		36 (4%)	46 (6%)	
Heart rate (beats /minute)	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	3924 (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

ACE=angiotensin-converting enzyme; HF=heart failure; NYHA=New York Heart Association; PS=potassium sparing

Table 2

Association between elevated jugular venous pressure (JVP) and mortality in chronic heart failure, before and after propensity matching

Outcomes	Rate, per 10000 person-years (Events / total follow up years)		Absolute rate difference* (per 10000 person-years)	Hazard ratio (95% confidence interval)	P value
Pre-match	Normal JVP (n = 6768)	Elevated JVP (n = 1020)			
All-cause	1054 (2131 / 20219)	1789 (475 / 2655)	+ 735	1.70 (1.54–1.88)	<0.0001
Cardiovascular	829 (1677 / 20219)	1412 (375 / 2655)	+ 583	1.70 (1.52–1.90)	<0.0001
Progressive heart failure	353 (714 / 20219)	727 (193 / 2655)	+ 374	2.07 (1.76–2.43)	<0.0001
Post-match	Normal JVP (n = 827)	Elevated JVP (n = 827)			
All-cause	1866 (398 / 2133)	1699 (373 / 2196)	- 167	0.95.(0.80–1.12)	0.521
Cardiovascular	1467 (313 / 2133)	1321 (290 / 2196)	- 147	0.93 (0.77–1.12)	0.440
Progressive heart failure	727 (155 / 2133)	660 (145 / 2196)	- 66	0.94 (0.71–1.23)	0.628

*Absolute differences in rates of events per 10,000 person-year 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 jugular venous pressure (JVP) and hospitalizations* in chronic heart failure, before and after propensity matching

Outcomes	Rate, per 10000 person-years (Events / total follow up years)		Absolute rate difference† (per 10000 person-years)	Hazard ratio (95% confidence interval)	P value
Pre-match	Normal JVP (n = 6768)	Elevated JVP (n = 1020)			
All-cause	3664 (4044 / 12019)	5186 (724 / 1396)	+ 1522	1.35 (1.25–1.47)	<0.0001
Cardiovascular	2402 (3416 / 14221)	3576 (594 / 1661)	+ 1174	1.42 (1.31–1.55)	<0.0001
Worsening heart failure	1086 (1888 / 17387)	1931 (399 / 2066)	+ 845	1.71 (1.53–1.90)	<0.0001
Post-match	Normal JVP (n = 827)	Elevated JVP (n = 827)			
All-cause	5056 (583 / 1153)	4882 (578 / 1184)	- 175	0.97 (0.87–1.09)	0.613
Cardiovascular	3508 (483 / 1377)	3338 (470 / 1408)	- 170	1.02 (0.87-1.19)	0.841
Worsening heart failure	1890 (319 / 1688)	1813 (314 / 1732)	- 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-year 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).