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Race and the Natural History of Chronic Heart Failure: A Propensity-Matched Study

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Abstract

Background—Racial differences in the epidemiology and outcomes of heart failure are well known. However, the association of race with the natural history of heart failure has not been previously studied in a propensity-matched population of chronic heart failure in which all measured baseline patient characteristics are well-balanced between the races.

Methods and Results—Of the 7788 patients with chronic systolic and diastolic heart failure in the Digitalis Investigation Group trial, 1128 were nonwhites. Propensity scores for being nonwhite were calculated for each patient and were used to match 1018 pairs of white and nonwhite patients. Matched Cox regression analyses were used to estimate associations of race with outcomes during 38 months of median follow-up. All-cause mortality occurred in 34% (rate, 1180/10000 person-years) of whites and 33% (rate, 1130/10000 person-years) of nonwhite patients (hazard ratio when nonwhite patients were compared with whites, 0.95, 95% confidence interval, 0.80–1.14; p=0.593). All-cause hospitalization occurred in 63% (rate, 3616/10000 person-years) of whites and 65% (rate, 3877/10000 person-years) of nonwhite patients (hazard ratio, 1.03, 95% confidence interval, 0.90–1.18; p =0.701). Respective hazard ratios (95% confidence intervals) for other outcomes were: 0.95 (0.75–1.12) for cardiovascular mortality, 0.82 (0.60–1.11) for heart failure mortality, 1.05 (0.91–1.22) for cardiovascular hospitalization and 1.17 (0.98–1.39) for heart failure hospitalization.

Conclusion—In a propensity-matched population of heart failure patients where whites and nonwhites were balanced in all measured baseline characteristics, there was no racial differences in major natural history end points.

Keywords

Heart failure; race; natural history; propensity scores

The natural history of heart failure has been reported to be worse among nonwhites, particularly among African Americans, compared to whites.[1–6] Most of these observational studies used traditional regression-based risk adjustment models. However, propensity score matching has emerged as a more efficient and transparent method to establish causal associations in

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observational studies.[7–15] As in randomized clinical trials, in propensity-matched studies, risk-adjusted balanced study populations can be assembled without access to outcomes data. Further, bias reduction using propensity matching can be objectively estimated and the covariate balance can be presented in a reader-friendly tabular format.[12–17] Therefore, the objective of this study was to evaluate whether race had an independent effect on major natural history endpoints in a population of propensity-matched ambulatory chronic heart failure patients who were well-balanced in all measured baseline covariates.

Methods

This is a post-hoc propensity-matched study of the Digoxin Investigation Group (DIG) trial (1991–1993), conducted in the United States and Canada. The rationale, design and results of the DIG trial have been published previously.[18, 19] Of the 7788 ambulatory patients with chronic heart failure and normal sinus rhythm, 6800 had ejection fraction ≤ 45%. Overall, 1128 (15.5%) patients were nonwhites, of whom 80% were African Americans and race was self-identified by patients.[6] Details of racial and ethnic information for nonwhite patients was not available for this analysis. We focus our current analysis on a subset of 2788 propensity-matched patients. The primary outcomes were mortality and hospitalizations due to all causes, cardiovascular causes and worsening heart failure. Data on vital status were 99% complete.[20]

Statistical Analysis

There were significant imbalances in baseline covariate distribution between nonwhite and white patients (Table 1). To account for this imbalance, we calculated propensity scores for being nonwhite for each patient, which was then used to match nonwhite and white patients using an SPSS macro.[12–15, 21] Of the several propensity score methods, matching has several advantages. It allows estimation of bias before and after matching and assembly of a post-match cohort in which two groups are well-balanced in all measured baseline covariates. Further, it allows display of that balance in visually pleasant graphic or tabular formats. Finally, it provides a rather conservative estimate of an association. Absolute standardized differences were used to estimate bias before and after matching.[12–17] Absolute standardized differences in propensity scores between white and nonwhite patients before and after matching were 84% and 0.1% respectively and those for all measured covariates were <5% (Figure 1). An absolute standardized difference of <10% is considered an acceptable reduction of bias.[12–17]

Baseline characteristics of nonwhite and white patients were compared using Pearson's chi-square and Wilcoxon's rank-sum tests and are displayed in Table 1. To avoid inflation of significance in the larger pre-match sample (n=7788), we assembled a pre-match sample size (n=2036) that was similar to the post-match group. This was done by selecting a random subset of 1018 white patients from the 6660 pre-match white patient population and then merged them with 1018 matched nonwhite patients. Kaplan-Meier and matched Cox regression analyses were used to determine the association of race with various outcomes. All statistical tests were done using SPSS for windows version 15 (SPSS Inc. Chicago, IL), with 2 tailed 95% confidence levels; a p value <0.05 was required to reject the null hypothesis.

Results

Patients had a mean age of 61 years and 31 % were women. Significant imbalance in several baseline characteristics before matching and the balance achieved after matching are displayed in Table 1 and Figure 1. During the median follow-up period of 38 months, 681 (33%) patients died from all causes, 521 due to cardiovascular causes and 230 due to progressive heart failure, and 1304 patients were hospitalized for all causes, 1048 from cardiovascular causes and 665 due to worsening heart failure.

Kaplan-Meier plots for all-cause mortality are displayed in Figure 2a. All-cause mortality occurred among 347 white patients (rate, 1180/10000 person-years) during 2941 person-years of follow-up and 334 nonwhite patients (rate, 1130/10000 person years) during 2955 person-years of follow-up (hazard ratio {HR} when nonwhite patients were compared with whites, 0.95, 95% confidence interval {CI}, 0.80–1.14, p =0.593; Table 2). Race was not associated with cause-specific mortalities (Table 2).

Kaplan Meier plots for all-cause hospitalization are displayed in Figure 2b. All-cause hospitalization occurred in 642 white patients (rate, 3616/10000 person-years) during 1776 person-years of follow-up and 662 nonwhite patients (rate, 3877/10000 person-years) during 1708 person-years of follow-up (HR when nonwhite patients were compared with whites, 1.03, 95% CI, 0.90–1.18, p =0.701; Table 3). There was a trend toward increased heart failure hospitalization among nonwhites (HR when nonwhite patients were compared with whites, 1.17, 95% CI, 0.98–1.39, p =0.093; Table 3). In the pre-match cohort (n=2036), there was a significant association between race and heart failure hospitalization (unadjusted HR, 1.44, 95% CI, 1.23–1.69, p <0.0001), which remained significant after adjustment for propensity scores (adjusted HR, 1.34, 95% CI, 1.12–1.59, p <0.0001). Post-match associations of race and other cause-specific hospitalizations are displayed in Table 3.

Discussion

The findings from this propensity-matched study, in which patients were balanced in all measured baseline covariates, suggest that race per se was not associated with any major natural history endpoints including mortality and hospitalization due to all causes and cardiovascular causes. This is the first report of a propensity-matched study of the effect of race on outcomes in chronic heart failure. However, there was a trend toward increased risk of hospitalization due to worsening heart failure among nonwhites.

These results are not surprising as the pathophysiological basis of heart failure and response to pharmacotherapy among white and nonwhite heart failure patients seem rather similar than dissimilar.[2, 22] African Americans heart failure patients seem to respond to angiotensin-converting enzyme (ACE) inhibitors and beta-blockers in a way similar to white patients.[2, 22] Data from the African-American Heart Failure Trial indicated a unique survival benefit from a combination of isosorbide dinitrate and hydralazine among African American heart failure patients.[23] However, the mechanism of such benefit has not yet been established and there is no evidence to suggest that combined vasodilator therapy would not be beneficial among some whites or other ethnic groups.[24–26]

Our finding of an increased risk of hospitalization due to worsening heart failure among nonwhite heart failure patients is consistent with similar finding by other investigators.[4, 6, 27] This is unlikely to be explained by differences in measured baseline characteristics as patients were well balanced in all measured baseline characteristics. However, it is possible that there were imbalances in unmeasured covariates such as patient and/or family preferences, and changes in baseline covariates during follow up. For example, data from general population and hospitalized heart failure patients suggest that African Americans with chronic kidney disease are more likely to advance to kidney failure requiring dialysis.[28, 29]

We observed that before matching there were significant racial differences in prognostically important baseline characteristics. However, after propensity matching, all measured baseline characteristics were well-balanced between white and nonwhite patients, and race per se was not significantly associated with any major natural history end points. However, pre-match differences in baseline characteristics indicate that race may be a marker of other prognostic covariates. For example, nonwhite heart failure patients were much younger than their white

counterparts. While this may suggest an apparently favorable prognostic association, these data also imply that nonwhite patients may have developed their heart failure at an earlier age. Compared to white patients, nonwhites were also more likely to be women, have a history of hypertension and diabetes, and have cardiomegaly. These underscore the need and the importance of optimum management of hypertension and diabetes, two of the most common causes of heart failure. Suboptimal treatment of these conditions in nonwhite patients is well documented in the literature.[30–32]

Several limitations of our study must be acknowledged. This is not a population-based study, which limits its generalizability. In clinical trials patients receive additional care from experienced research personnel (for example over 90% were receiving ACE inhibitors) and are followed up more rigorously than in routine clinical practice. Further, patients in this study were younger, predominantly male, had normal sinus rhythm, and are from the pre-beta-blocker era of heart failure therapy, which may further limit generalizability. Finally, like any non-randomized study, propensity matching cannot account for unmeasured covariates, which may explain away our key findings. However, for any hidden covariate to function as a confounder, it must be strongly associated with both race and outcomes, and not strongly related to any of the covariates displayed in Table 1.

In conclusion, in a propensity-matched population of heart failure patients in which whites and nonwhites were well balanced in all measured baseline covariates, despite a trend for increased heart failure hospitalization among nonwhites, race by itself was not associated with all-cause and cardiovascular mortality or hospitalization. However, the higher prevalence of hypertension, diabetes and cardiomegaly, and the development of heart failure at a younger age among nonwhites highlight the need for early detection and optimal treatment of hypertension and diabetes and primary prevention of heart failure at an earlier age in these patients.

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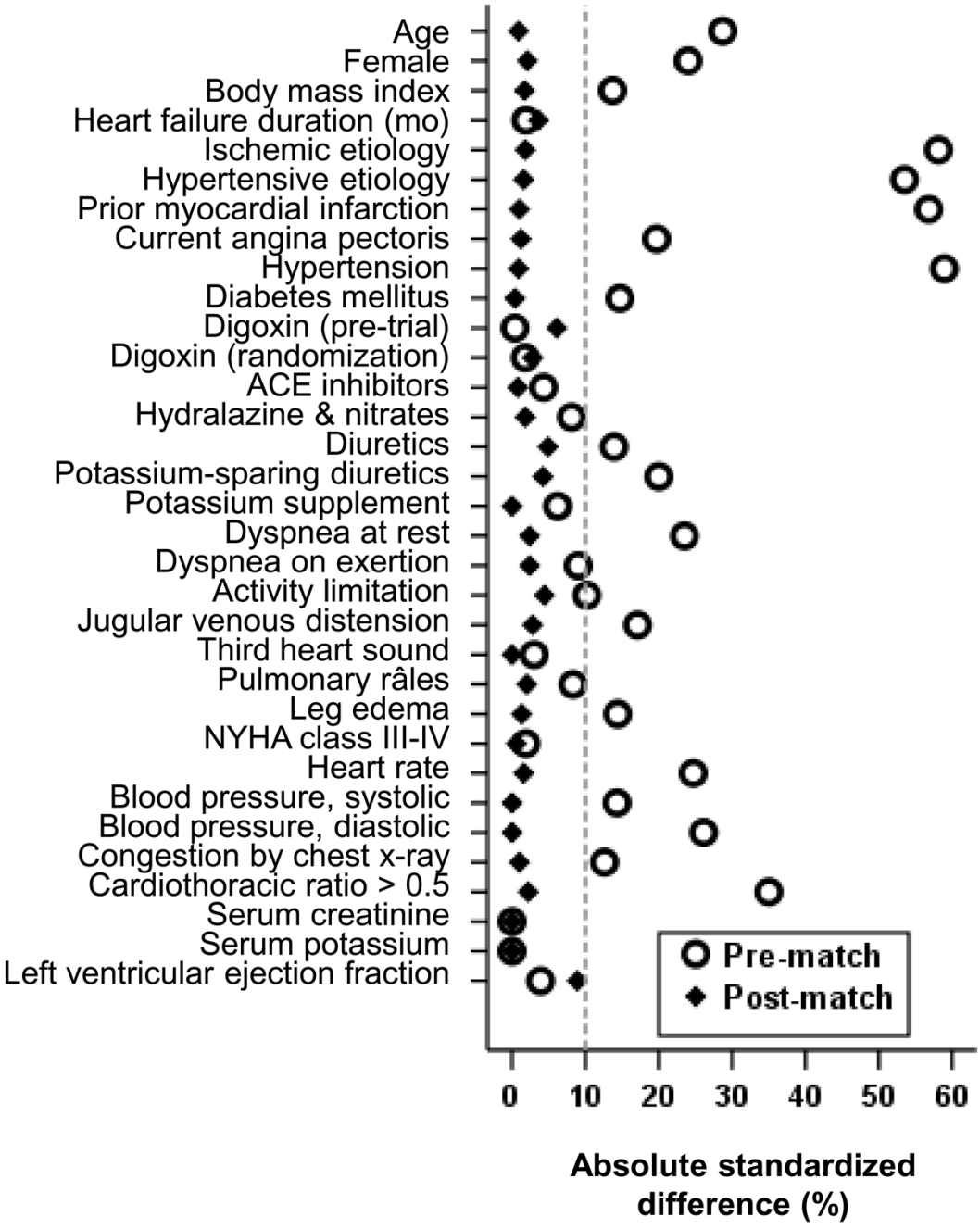
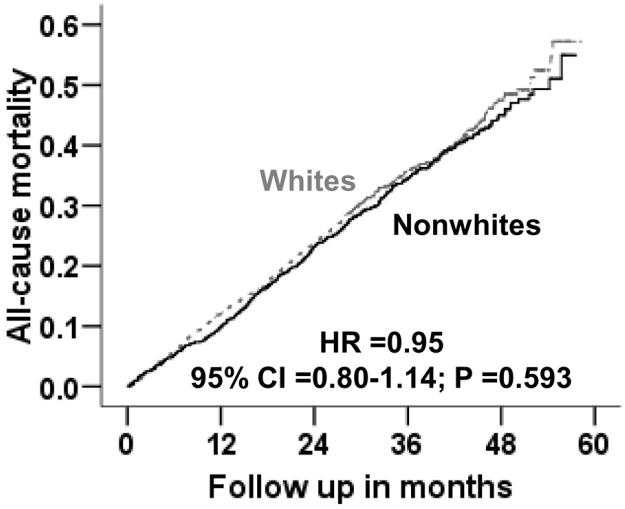


Figure 1.
Absolute standardized differences before and after propensity score matching comparing covariate values for white and nonwhite patients

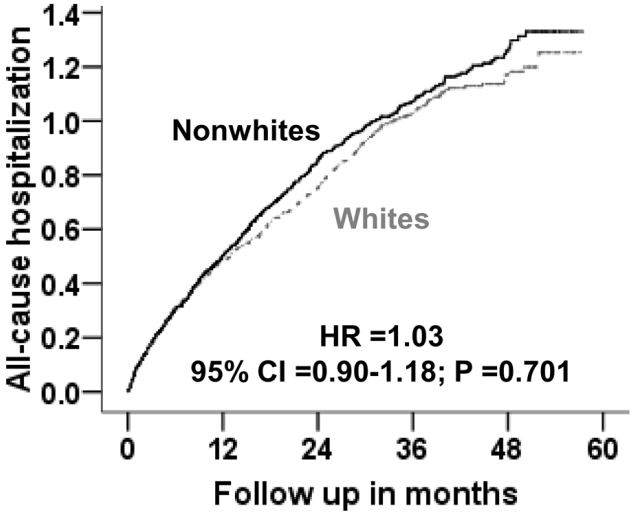
Figure 2a



Number of patients at risk

Whites	1018	897	798	544	202
Nonwhites	1018	911	787	525	219

Figure 2b



Number of patients at risk

White	1018	592	427	241	83
Nonwhite	1018	580	382	225	79

Figure 2.
Kaplan-Meier plots for all-causes mortality (2a) and all-causes hospitalizations (2b).

Table 1

Baseline patient characteristics by race, before and after propensity score matching

	Before matching*		Nonwhites (N=1018)	After matching†	
N (%) or mean (±SD)	**Whites (N=1018)**	**P**		**P**	**Whites (N=1018)**
Age (years)	64.5 (±10.2)	<0.0001	61.3 (±12.0)	0.961	61.2 (±11.5)
Age ≥65 years	533 (58.2%)	<0.0001	435 (42.7%)	0.858	431(42.3%)
Female	217 (21.3%)	<0.0001	324 (31.8%)	0.633	314 (30.8%)
Body mass index, kg/square meter	27.2 (±5.2)	0.001	28.0 (±6.4)	0.838	28.1 (±5.7)
Heart failure duration (mo)	29.4 (±36.5)	0.662	30.1 (±35.7)	0.448	28.8 (±37.5)
Primary cause of heart failure					
Ischemic	745 (73.2%)	<0.0001	466 (45.8%)	0.915	475 (46.7%)
Hypertensive	65 (6.4%)		257 (25.2%)		250 (24.6%)
Idiopathic	133 (13.1%)		187 (18.4%)		179 (17.6%)
Others	75 (7.4%)		108 (10.6%)		114 (11.2%)
Prior myocardial infarction	696 (68.4%)	<0.0001	419 (41.2%)	0.822	414 (40.7%)
Current angina pectoris	293 (28.8%)	<0.0001	207 (20.3%)	0.782	202 (19.8%)
Hypertension	421 (41.4%)	<0.0001	707 (69.4%)	0.847	711 (69.8%)
Diabetes mellitus	277 (27.2%)	0.001	346 (34.0%)	0.925	348 (34.2%)
Medications					
Pre-trial digoxin use	447 (43.9%)	0.929	449 (44.1%)	0.168	480 (47.2%)
Trial use of digoxin	515 (50.6%)	0.690	506 (49.7%)	0.535	492 (48.3%)
ACE inhibitors	944 (92.7%)	0.331	955 (93.8%)	0.855	953 (93.6%)
Hydralazine & nitrates	16 (1.6%)	0.067	28 (2.8%)	0.692	31 (3.0%)
Diuretics	792 (77.8%)	0.002	848 (83.3%)	0.269	829 (81.4%)
Potassium-sparing diuretics	95 (9.3%)	<0.0001	44 (4.3%)	0.349	53 (5.2%)
Potassium supplement	295 (29.0%)	0.162	324 (31.8%)	1.000	324 (31.8%)
Symptoms and signs of heart failure					
Dyspnea at rest	196 (19.3%)	<0.0001	298 (29.3%)	0.594	309 (30.4%)
Dyspnea on exertion	759 (74.6%)	0.042	718 (70.5%)	0.595	707 (69.4%)
Activity limitation	767 (75.3%)	0.022	721 (70.8%)	0.325	741 (72.8%)
Jugular venous distension	116 (11.4%)	<0.0001	177 (17.4%)	0.525	188 (18.5%)
Third heart sound	250 (24.6%)	0.499	237 (23.3%)	1.000	237 (23.3%)
Pulmonary râles	161 (15.8%)	0.061	193 (19.0%)	0.654	201 (19.7%)
Lower extremity edema	208 (20.4%)	0.001	270 (26.5%)	0.762	264 (25.9%)
NYHA functional class					
I	162 (15.9%)	0.937	159 (15.6%)	0.998	160 (15.7%)
II	539 (52.9%)		551 (54.1%)		547 (53.7%)
III	292 (28.7%)		286 (28.1%)		289 (28.4%)
IV	25 (2.5%)		22 (2.2%)		22 (2.2%)
Heart rate (/minute),	77.7 (±12.4)	<0.0001	80.8 (±12.7)	0.690	81.0 (±13.1)
Blood pressure (mm Hg)					
Systolic	127 (±20)	<0.0001	130 (±22)	0.768	130 (±22)
Diastolic	75 (±11)	<0.0001	78 (±12)	0.741	78 (±12)
Chest radiograph findings					
Pulmonary congestion	141 (13.9%)	0.005	188 (18.5%)	0.820	192 (18.9%)
Cardiothoracic ratio >0.5	592 (58.2%)	<0.0001	758 (74.5%)	0.614	748 (73.5%)
Serum potassium (mEq/L)	4.35 (±0.42)	0.001	4.28 (±0.46)	0.717	4.27 (±0.49)
Serum creatinine (mg/dL)	1.3 (±0.4)	0.686	1.3 (±0.4)	0.914	1.3 (±0.4)
Ejection fraction (%)	32.0 (±12.3)	0.381	31.5 (±13.2)	0.317	30.4 (±11.6)

ACE, Angiotensin-converting enzyme; NYHA, New York Heart Association.

[*] Of the 6660 white patients, a random subset of 1018 patients were selected to provide equal sample sizes for prematch and post match comparisons and to avoid significant P values in the prematch comparisons from a larger sample size.

[†] Of the 1128 nonwhite patients, 1018 (90%) were matched with 1018 white patients with similar propensity scores

Table 2

Cause-specific mortalities in heart failure patients by race

	Rate/10,000 person-years follow-up (Deaths/follow-up in years)*		Rate difference** (/10,000 person-years)	Matched hazard ratio† (95% confidence interval)	P value
	White (N=1,018)	Nonwhite (N=1,018)			
All-cause	1180 (347/2,941)	1130 (334/2,955)	− 50	0.95 (0.80–1.14)	0.593
Cardiovascular	911 (268/2,941)	856 (253/2,955)	− 55	0.95 (0.75–1.12)	0.388
Worsening heart failure‡	411 (121/2,941)	369 (109/2,955)	− 42	0.82 (0.60–1.11)	0.192
Other cardio-vascular§	500 (147/2,941)	487 (144/2,955)	− 13	1.00 (0.77–1.30)	1.000
Non-cardio-vascular	190 (56/2,941)	193 (57/2,955)	+ 3	1.02 (0.67–1.58)	0.913
Unknown	78 (23/2,941)	81 (24/2,955)	+ 3	1.27 (0.64–2.49)	0.494

*Total follow up period is same for all cause-specific mortalities as for all-cause mortality

**Absolute rate differences were calculated by subtracting the rates of death in the white group from the rates of death in the nonwhite group (before values were rounded).

†Hazard ratios and confidence intervals (CI) were estimated from matched Cox proportional-hazards models.

‡This category includes patients who died from worsening heart failure, even if the final event was an arrhythmia.

§This category include cardiac deaths presumed to result from arrhythmia without evidence of worsening heart failure and deaths due to atherosclerotic coronary disease, bradyarrhythmias, low-output states, and cardiac surgery and vascular deaths due to stroke, embolism, peripheral vascular disease, vascular surgery, and carotid endarterectomy.

Table 3

Cause-specific hospitalizations in heart failure patients by race

Cause for hospitalization*	Rate/10,000 person-years follow up (Hospitalizations/follow-up in years)		Rate Difference (/10,000 person-years)†	Matched hazard ratio (95% confidence interval)‡	P value
	White (N=1,018)	Nonwhite (N=1,018)			
All-cause	3616 (642/1776)	3877 (662/1708)	+ 261	1.03 (0.90–1.18)	0.701
Cardiovascular	2418 (500/2068)	2762 (548/1984)	+ 344	1.05 (0.91–1.22)	0.498
Worsening heart failure	1213 (301/2481)	1543 (364/2,359)	+ 330	1.17 (0.98–1.39)	0.093
Ventricular arrhythmia, cardiac arrest	97 (28/2899)	107 (31/2909)	+ 10	1.13 (0.65–1.95)	0.675
SV arrhythmias§	157 (22/2867)	128 (24/2901)	– 29	0.90 (0.54–1.51)	0.696
AV block, bradyarrhythmia	7 (2/2935)	3 (1/2954)	– 4	0.50 (0.05–5.51)	0.571
Suspected digoxin toxicity	69 (20/2906)	82 (24/2912)	+ 13	1.18 (0.62–2.25)	0.622
Myocardial infarction	159 (46/2893)	155 (45/2908)	– 4	1.06 (0.66–1.69)	0.811
Unstable angina	344 (95/2760)	386 (106/2747)	+ 42	1.07 (0.78–1.46)	0.690
Stroke	192 (55/2861)	205 (59/2874)	+ 13	1.02 (0.68–1.53)	0.917
Coronary revascularization¶	69 (20/2908)	38 (11/2924)	– 31	0.83 (0.36–1.93)	0.670
Cardiac transplantation	41 (12/2924)	7 (2/2949)	– 34	0.29 (0.06–1.38)	0.118
Other cardiovascular**	506 (137/2707)	398 (111/2786)	– 108	0.85 (0.64–1.12)	0.249
Respiratory infection	268 (76/2834)	259 (73/2821)	– 9	1.15 (0.80–1.65)	0.458
Other non-cardiovascular	1426 (339/2378)	1346 (326/2422)	– 80	0.85 (0.71–1.02)	0.074
Unspecified	20 (6/2935)	17 (5/2952)	– 3	0.33 (0.07–1.65)	0.178

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

†Absolute differences were calculated by subtracting the percentage of patients hospitalized in the white group from the percentage of patients hospitalized in the nonwhite group (before values were rounded).

‡Hazard ratios and confidence intervals (CI) were estimated from a Cox proportional-hazards models that used the first hospitalization of each patient for each reason.

§Supraventricular (SV) arrhythmias include Atrioventricular (AV) block and bradyarrhythmias

¶This category includes coronary-artery bypass grafting and percutaneous transluminal coronary angioplasty

**This category includes embolism, venous thrombosis, peripheral vascular disease, hypertension, other vascular surgery, cardiac catheterization, other types of catheterization, pacemaker implantation, installation of automatic implantable cardiac defibrillator, electrophysiologic testing, transplant-related evaluation, nonspecific chest pain, atherosclerotic heart disease, hypotension, orthostatic hypotension, and valve operation.