Journal of the American Heart Association

ORIGINAL RESEARCH

Racial Differences in the Use of Aortic Valve Replacement for Treatment of Symptomatic Severe Aortic Valve Stenosis in the Transcatheter Aortic Valve Replacement Era

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BACKGROUND: Aortic valve replacement (AVR) is a life-saving treatment for patients with symptomatic severe aortic valve stenosis. We sought to determine whether transcatheter AVR has resulted in a more equitable treatment rate by race in the United States.

METHODS AND RESULTS: A total of 32 853 patients with symptomatic severe aortic valve stenosis were retrospectively identified via Optum's deidentified electronic health records database (2007–2017). AVR rates in non-Hispanic Black and White patients were assessed in the year after diagnosis. Multivariate Fine-Gray hazards models were used to evaluate the likelihood of AVR by race, with adjustment for patient factors and the managing cardiologist. Time-trend and 1-year symptomatic severe aortic valve stenosis survival analyses were also performed. From 2011 to 2016, the rate of AVR increased from 20.1% to 37.1%. Overall, Black individuals were less likely than Whites to receive AVR (22.9% versus 31.0%; unadjusted hazard ratio [HR], 0.70; 95% CI, 0.62–0.79; fully adjusted HR, 0.76; 95% CI, 0.67–0.85). Yet, during 2015 to 2016, AVR racial differences were attenuated (29.5% versus 35.2%; adjusted HR, 0.86; 95% CI, 0.74–1.02) because of greater uptake of transcatheter AVR in Blacks than Whites (53.4% of AVRs versus 47.3%; *P*=0.128). Untreated patients had significantly higher 1-year mortality than those treated (adjusted HR, 0.57; 95% CI, 0.53–0.61), which was consistent by race (interaction *P* value=0.52).

CONCLUSIONS: Although transcatheter AVR has increased the use of AVR in the United States, treatment rates remain low. Black patients with symptomatic severe aortic valve stenosis were less likely than White patients to receive AVR, yet these differences have recently narrowed.

Key Words: racial differences in care ■ symptomatic severe aortic valve stenosis ■ transcatheter aortic valve replacement

Symptomatic severe aortic valve stenosis (ssAS) is a deadly, but curable, condition. Left untreated, up to half of all patients with ssAS will die within 2 years[1] of symptom onset; however, if treated with aortic valve replacement (AVR) in an appropriate time frame, patients with ssAS can experience alleviation of symptoms and return to a normal life trajectory. Consequently, AVR for the treatment of ssAS has received a class I recommendation from both European and American valvular heart disease guideline committees.[2,3] Treatment of ssAS was previously limited to surgical intervention, resulting in a large share of patients being placed on medical therapy,[4] but the development of a less invasive transcatheter AVR (TAVR) alternative has disrupted these traditional treatment paradigms and referral patterns across the

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Supplementary material for this article is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.119.015879

For Sources of Funding and Disclosures, see page 9.

JAHA is available at: www.ahajournals.org/journal/jaha

CLINICAL PERSPECTIVE

What Is New?

- In the United States, rates of aortic valve replacement (AVR) for symptomatic severe aortic valve stenosis (ssAS) were low (36% in 2016), and Black individuals were significantly less likely to undergo AVR than White individuals with ssAS, despite similar 1-year survival for White and Black individuals when stratified by treatment status.
- In recent years, racial differences in AVR treatment rates have narrowed, with a greater rate of increase in the uptake of transcatheter AVR in the Black community.

What Are the Clinical Implications?

- Clinicians should examine their local ssAS treatment rates and redouble efforts to ensure that all appropriate patients with ssAS undergo treatment with AVR (whether surgical or transcatheter).
- Given the meaningful reduction in racial differences in ssAS treatment with transcatheter AVR availability in the United States, efforts should be made to ensure access to this technology for clinically appropriate indications in the Black community.
- Recognizing that Black individuals with ssAS are less likely to undergo treatment, further research is needed to understand and address the underlying causes of these differences.

Nonstandard Abbreviations and Acronyms

AS	aortic stenosis
AVR	aortic valve replacement
HR	hazard ratio
ICD-9-CM	*International Classification of Diseases, Ninth Revision, Clinical Modification*
ICD-10-CM	*International Classification of Diseases, Tenth Revision, Clinical Modification*
SAVR	surgical aortic valve replacement
ssAS	symptomatic severe aortic valve stenosis
TAVR	transcatheter aortic valve replacement

globe.[5,6] Since the US Food and Drug Administration approved TAVR technology in 2011, the use of TAVR has dramatically increased, and since 2017, TAVR has been the dominant treatment for ssAS.[7] Despite these improvements, it remains unclear whether access to treatment has been equitable across different patient groups.

In many areas of medicine, patient access to treatment has not been equally distributed.[8,9] Specifically, prior studies in the United States have found that Black individuals are less likely than their White counterparts to receive access to costly cardiovascular therapies, such as percutaneous coronary intervention, coronary artery bypass grafting, cardiac transplantation, ventricular assist devices, or automatic implantable cardioverters-defibrillators. Before the introduction of TAVR, Black individuals were significantly less likely to receive AVR compared with Whites.[10] A recent review of the STS TVT (Society of Thoracic Surgeons Transcatheter Valve Therapy) Registry covering the post-TAVR introduction period 2011 to 2016 showed an underrepresentation of Black individuals among those receiving transcatheter therapies.[11] Other studies covering the post-TAVR introduction period, described in a recent review by Wilson et al,[12] have also shown that Black patients are underrepresented and less likely to receive AVR. Nevertheless, most studies were either single-center studies, focused only on TAVR, or they did not measure long-term outcomes or treatment trends over time.[12] In addition, none of these studies focused on treatment rates for all patients diagnosed with ssAS. To date, there have been limited new investigations into whether the evolution of AVR practice patterns has affected the overall ssAS treatment rate in the Black community, as well as whether TAVR is driving any such change.

The goal of our study was to conduct a population-based analysis to better understand race-based treatment differences among patients with ssAS in the United States and the implications of these treatment differences on the health of older individuals in the Black community. Specifically, we investigated the following 3 issues: (1) the race-related use of AVR for ssAS; (2) time trends in AVR use by race; and (3) race-related 1-year survival with ssAS, according to treatment status.

METHODS

Data are available through license with Optum, and further details of the methods will be made available on request.

Data Source

This observational cohort study was conducted using Optum deidentified electronic health records,[13] which is a patient-level database that standardizes and

integrates multiple US-based electronic health record data systems. The longitudinal clinical repository, containing data from 2007 to 2018, is one of the largest in the United States and is derived from >50 healthcare provider organizations, including >2000 hospitals and 7000 clinics. The database tracks the clinical progress of patients across different providers, allowing for the longitudinal evaluation of outcomes over time, with a subset of patients having linked Optum claims records. The data are sourced from both the ambulatory and inpatient setting, which is similar to claims-based data sets covering diagnosis and procedure codes, laboratory results, clinical observations, medications, and structured data on patient status and basic laboratory values. In terms of echocardiographic readings, the data are primarily limited to ejection fraction, although a select group of records have aortic valve measurements available. Several studies have previously been published using this database, including work in diabetes mellitus, neurology, and heart failure.[14–22] Because no identifiable protected health information was leveraged for this study, institutional review board approval was not required.

Study Population

This study included patients newly diagnosed with ssAS between 2011 and 2016 who were part of an Optum-integrated delivery network, which is a formal network of healthcare providers and organizations that offers care services and insurance plans, and is less likely to contain missing data[23]. Valve disease was captured from a structured table of physician findings indexed on the words "aortic stenosis."[14] Aortic stenosis (AS) severity was defined if the following terms ("severe," "critical," or a combination of these terms) were positively linked to AS.[24–26] We excluded patients with neutral or negative terms, such as "negative," "deny," "not," "suspect," "potential," "rule out," or a combination of these terms, in association with their AS diagnosis. Given that there were limited primary echocardiographic records available to confirm the diagnosis, Kaplan-Meier analysis was performed to confirm the expected stratification of patient survival based on mild, moderate, and severe disease (Figure S1). Further validation of the severe AS assessment was performed by cross referencing echocardiography report data with reported severity from the structured physician notes among the subgroup of patients with available echocardiography reports, as described below (Table S1). The echocardiographic severity of AS was based on American Heart Association guidelines and included velocity, aortic valve area, mean gradient, and left ventricular ejection fraction.[2] Given variability in the use of echocardiography parameters to evaluate ssAS, patients were graded as severe if they had at least one measure meeting criteria.[27]

Patients were classified as symptomatic if there were structured reports of angina, dyspnea on exertion, dyspnea, presyncope, or syncope in the 6 months before diagnosis or a diagnosis of heart failure. The objective approach to determining ssAS (namely, the presence or absence of symptoms in a patient's history, without attribution to AS specifically) is similar to previously described methods.[25,28,29]

Patients who had at least 1 year of history in the electronic health record before ssAS diagnosis, and at least 1 year of follow-up or a record of death in the year after date of ssAS, were included in the base cohort to allow for appropriate risk adjustment and complete patient follow-up. Using these methods, a base cohort of 37 910 integrated delivery network patients with ssAS between 2011 and 2016 was identified. Of these patients, 11 were excluded because they had a left ventricular assist device before diagnosis and, therefore, a poor prognosis. Because the study was focused on evaluating treatment rates and outcomes in the non-Hispanic Black ("Black") and non-Hispanic White ("White") communities, 5046 patients who identified another racial or ethnic group were excluded. The final cohort was composed of 32 853 patients. All patients had 1 year of history before diagnosis to increase the likelihood of a first diagnosis of ssAS and have a baseline period to evaluate comorbidities. To internally validate our data on treatment rate, we repeated our analyses in the Optum claims-linked patient set. This cohort includes a subset of patients in the electronic health record who have continuous insurance claims data during the study period, reducing the risk of missing records. After applying our inclusion/exclusion criteria, a subset of 1919 patients was identified within the integrated patient set. Given the small sample size of Black patients in the integrated patient set (n=57), we only repeated analysis to validate the distribution of race and treatments for all patients, including both White and Black.

Outcomes

The primary outcome of interest in this study was treatment of ssAS by any AVR (TAVR or surgical AVR [SAVR]), in the year after ssAS diagnosis. Patients undergoing AVR were identified using *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*), and *International Classification of Diseases, Tenth Revision, Clinical Modification* (*ICD-10-CM*), codes (*ICD-CM-9*: 35.05, 35.06, 35.21, and 35.22; *ICD-CM-10*: 02RFxxx) and Current Procedural Terminology codes (33405-6, 33410-13, and 33361-33366). The last year evaluated for treatment in the study was 2017, allowing

for follow-up of 1 year after diagnosis for patients diagnosed in 2016. The secondary outcome of interest was all-cause mortality by 1 year after diagnosis. Date of death was captured in Optum based on the Social Security Death Masterfile, and follow-up time was calculated from date of diagnosis to date of death.

Covariates

Patient history was evaluated in the year before diagnosis. Race and ethnicity were extracted from physician notes in Optum. Comorbidities were identified using *ICD-9-CM* and *ICD-10-CM* codes supplemented with physician notes; these comorbidities included atrial fibrillation, cancer, conduction disorders, chronic obstructive pulmonary disease, dementia, diabetes mellitus with and without complications, myocardial infarction, osteoarthritis, peripheral vascular disease, heart failure, moderate and severe renal disease, and the use of supplemental oxygen. Patient status was also assessed using the Deyo modification of the Charlson Comorbidity Index.[30] Records were reviewed to determine hospitalization in the year before diagnosis and in the diagnostic setting. *ICD-9-CM*, *ICD-10-CM*, and Current Procedural Terminology codes were used to assess percutaneous coronary intervention, implantable cardioverter defibrillator and/or pacemaker implantation, and dialysis. Prior cardiac surgeries were evaluated, but not included in the models because the sample size was too small. Left ventricular ejection fraction, creatinine, and body mass index were obtained from structured laboratory or observational data. Other covariates, including age, year of diagnosis, sex, area income and education level, census region/division, insurance, and smoking status, were directly captured by Optum from patient records. Imputation of missing variables with <10% of missing data was accomplished via multivariate imputation by chained equations (mice) using the mice version 2.9 package.[31] Imputation replaced 8.8% for insurance, 5.8% for smoking, 2.6% for income, 2.5% for education, 0.2% for sex, and 0.15% for age. The reported rate of missing data is within previously reported ranges.[32] Missing data for variables with >10% missing, including left ventricular ejection fraction (30.8%), creatinine (19.9%), and body mass index (11.3%), were coded as "unknown." The full list of *ICD-9-CM* and *ICD-10-CM* codes is presented in Table S2.

A managing cardiologist was identified in a subset of 24 775 patients in the Optum records using unique identification numbers and the reported physician specialty. The primary cardiologist was determined as the most frequent managing cardiologist in the 3 months before and after a diagnosis of ssAS. To control for the impact of the provider, the treatment rate of the primary cardiologist (when present) in tertiles was also included as a covariate in a subset analysis. The patient characteristics for those with an identifiable cardiologist are presented in Table S3.

Statistical Analysis

Characteristics of Black and White individuals were compared using χ^2 tests. Continuous variables (income level, percentage college educated, and median age) were compared using the Kruskal-Wallis test. Treatment penetration was evaluated over time for all patients and then for White and Black patients. We assessed any potential change in racial disparities in treatment over time by using an interaction term between race and year of ssAS diagnosis.

The primary objective of this study was to understand the association between race and the likelihood of AVR. Fine-Gray subdistribution hazards models[33] were used to assess the association between race (non-Hispanic White and non-Hispanic Black) and receipt of AVR in the year after AS diagnosis, where AVR was the outcome of interest and death from any cause was a competing event. Sequential model building was used to assess control for patient- and physician-related factors, including a subset analysis with patients who had an identifiable primary cardiologist associated with AS diagnosis and follow-up. This latter analysis included an additional variable for percentage of severe AS patients who a provider referred for AVR; this percentage was transformed into a tertile rank across all providers. The aim of this analysis was to control for the relative extent to which provider behavior affects subsequent receipt of AVR. In addition, further sensitivity analysis was performed among patients with available ejection fraction, creatinine, and body mass index by repeating our models in these cohorts. The sequential models and corresponding subdistribution hazard ratios (HRs) are presented in Table S4. To assess treatment patterns over time, the likelihood of AVR was stratified by 2-year intervals (2011–2012, 2013–2014, and 2015–2016). The sample size of Black patients was too small to analyze the association between race and the use of TAVR versus SAVR approaches.

A second analysis was performed to assess the association between race and 1-year all-cause mortality using multivariable Cox proportional hazards model. The proportional hazard assumption was evaluated by plotting Schoenfeld residuals for the outcome of AVR treatment within 1 year after diagnosis by race (Figure S2). Residual plots did not show large deviation from a horizontal line, indicating the proportional hazard assumption was met. Sensitivity analyses were performed to evaluate the potential impact of immortal time bias by using multivariable time-dependent Cox regression models. Receipt of AVR was treated as a time-dependent variable to account for the immortal

time bias. We also evaluated any differential benefit of treatment for survival by race by using an interaction term between race and treatment status. Covariates for all models were defined a priori and sequentially modeled. All analyses were conducted using SAS version 9.4 and R version 3.5.2 with a $P \leq 0.05$ considered significant.

RESULTS

Between 2011 and 2016, we identified 32 853 patients with ssAS in the Optum cohort. Of these patients, 31 593 (96.2%) were identified as White and 1260 were identified as Black (3.8%), which is consistent with the results in the integrated set (White versus Black, 1862 [97.0%] versus 59 [3.0%]). Among the study population, the median age was 80 years (25th–75th percentile, 71–85 years), 47.5% were women, and 18.3% had a reduced left ventricular ejection fraction. Most patients reported dyspnea as a primary symptom (n=29 589 [90.1%]), with angina reported in 9263 (28.2%) and syncope in 9825 (29.9%).

Compared with White patients with ssAS, Black patients were younger (median age, 74 versus 80 years; $P<0.001$), were more likely to be women (56.7% versus 47.1%; $P<0.001$), and had a greater burden of comorbidities (median Charlson Comorbidity Index, 2 versus 1; $P<0.001$) (Table). Black patients were more likely to live in lower-income communities (income level, $39 005 versus $41 792; $P<0.001$) and have Medicaid health insurance (6.7% versus 2.2%). Also, Black patients were more likely than White patients to have an initial diagnosis during an acute-care hospitalization (rather than outpatient clinic) (60.7% versus 41.7%).

Among patients with ssAS, AVR was performed in 30.7% (n=10 068) in the first year following diagnosis. The median (25th–75th percentile) time between AVR and date of ssAS diagnosis was 39 days (15–86 days). The results stayed consistent in the integrated claims set, with 29.4% of patients with ssAS (n=565) undergoing AVR in the first year after diagnosis (median time between AVR and date of ssAS diagnosis, 48 days; 25th–75th percentile, 22–98 days). Compared with White patients, Black patients with ssAS were less likely to receive AVR by 1 year (22.9% versus 31.0%; unadjusted HR, 0.70; 95% CI, 0.62–0.79). These racial differences in AVR use persisted after adjustment for demographics, comorbidities, geographic region, socioeconomic status, and insurance status (Table S4) (adjusted HR, 0.76; 95% CI, 0.67–0.85). Similar results were found when the analyses were repeated in patients who had available ejection fraction, creatinine, and body mass index (Table S4).

Because there may be differential access to specialty care, we repeated these analyses after limiting the cohort to those who were seen at least once by a cardiologist. The distribution of patient characteristics is demonstrated in Table S3. Overall, 72.6% of the cohort (n=23 839) had an identifiable cardiology specialist, but this number was slightly lower among Black versus White patients (68.6% versus 72.7%; $P<0.001$). Yet, among those who saw a cardiologist, treatment with AVR within 1 year remained lower among Black versus White patients (27.3% versus 36.6%; $P<0.001$; Table S4; adjusted HR, 0.74; 95% CI, 0.66–0.85; for the 9014 patients who were not managed by a cardiologist, the HR of AVR Black versus White, 0.787 [95% CI, 0.587–1.056]; $P=0.780$).

To evaluate the potential impact of TAVR dispersion on race-based differences in AVR use, we evaluated trends in ssAS treatment from 2011 to 2016. Rates of AVR by 1 year increased over time for both Blacks and Whites with ssAS (Figure 1). Compared with SAVR use, the racial gap in TAVR use improved over time. Because of the more rapid increase in TAVR use among Black patients, there was no significant difference in the overall use of AVR by race after controlling for risk factors in the last years of the study, 2015 to 2016 (29.5% versus 35.2%; adjusted HR, 0.86; 95% CI, 0.75–1.01; Figure 2). The interaction between race and year of ssAS diagnosis was not statistically significant (P-interaction=0.095).

Overall, in this ssAS cohort, 1-year survival was higher among treated patients than untreated patients (90.5% versus 69.1% 1-year survival; $P<0.01$; adjusted HR in Cox proportional hazards model, 0.36; 95% CI, 0.34–0.39; adjusted HR in time-dependent Cox proportional hazards model, 0.57; 95% CI, 0.53–0.61), and a similar benefit of treatment was observed among Black and White patients (P-interaction=0.52) after controlling for treatment received within 1 year. Among treated patients, survival was similar by race after adjustment for comorbidities and demographics (HR, 0.96; 95% CI, 0.66–1.38), although untreated Black patients experienced significantly better survival than untreated White patients after risk adjustment (HR, 0.85; 95% CI, 0.75–0.95; Figure 3). Similar results were obtained when the analyses were repeated in patients who had available ejection fraction, creatinine, and body mass index (treated HR, 0.98; 95% CI, 0.62–1.55; untreated HR, 0.79; 95% CI, 0.69–0.91).

DISCUSSION

In the United States, we found that nearly two thirds of patients with ssAS remained untreated 1 year after diagnosis. Furthermore, we found that Black patients

Table 1. Patient Characteristics, Overall and Stratified by Race

Patient Characteristics	Overall (n=32 853)	Non-Hispanic Whites (n=31 593)	Non-Hispanic Blacks (n=1260)	*P* Value
Age, median (25th–75th percentile), y	80 (71–85)	80 (71–85)	74 (64–83)	<0.001
<64	4529 (13.79)	4203 (13.30)	326 (25.87)	
65–79	11 668 (35.52)	11 179 (35.38)	489 (38.81)	
≥80	16 630 (50.62)	16 185 (51.23)	445 (35.32)	
Unknown	26 (0.08)	26 (0.08)	0 (0.0)	
Women	15 591 (47.46)	14 877 (47.09)	714 (56.67)	<0.001
Charlson Comorbidity Index				
0	8542 (26.00)	8302 (26.28)	240 (19.05)	<0.001
1	6948 (21.15)	6740 (21.33)	208 (16.51)	
2	5135 (15.63)	4964 (15.71)	171 (13.57)	
3	4094 (12.46)	3899 (12.34)	195 (15.48)	
≥4	8134 (24.76)	7688 (24.34)	446 (35.40)	
Atrial fibrillation	9067 (27.60)	8851 (28.02)	216 (17.14)	<0.001
Cancer	4206 (12.80)	4033 (12.77)	173 (13.73)	0.31
Cardiac conduction disorders	3114 (9.48)	3008 (9.52)	106 (8.41)	0.19
CAD	12 652 (38.51)	12 223 (38.69)	429 (34.05)	<0.001
CVA	4129 (12.57)	3965 (12.55)	164 (13.02)	0.12
COPD	3390 (10.32)	3264 (10.33)	126 (10.00)	0.70
Dementia	959 (2.92)	912 (2.89)	47 (3.73)	0.08
Diabetes mellitus without complications	1925 (5.86)	1805 (5.71)	120 (9.52)	<0.001
Diabetes mellitus with complications	8907 (27.11)	8432 (26.69)	475 (37.70)	<0.001
Prior myocardial infarction	3779 (11.50)	3592 (11.37)	187 (14.84)	<0.001
Osteoarthritis	4826 (14.69)	4703 (14.89)	201 (15.95)	0.41
Peripheral vascular disease	4904 (14.93)	4703 (14.89)	201 (15.95)	0.30
Heart failure	6691 (20.37)	6368 (20.16)	323 (25.63)	<0.001
Moderate-to-severe renal disease	7076 (21.54)	6612 (20.93)	464 (36.83)	<0.001
Current smoking	3753 (11.81)	3556 (11.64)	197 (15.93)	<0.001
Supplemental oxygen	1445 (4.40)	1386 (4.39)	59 (4.68)	0.62
Percutaneous cardiac procedures	695 (2.12)	673 (2.13)	22 (1.75)	0.35
Pacemaker	400 (1.22)	383 (1.21)	17 (1.35)	0.66
Hemodialysis	488 (1.49)	388 (1.23)	100 (7.94)	<0.001
Dyspnea	29 589 (90.06)	28 426 (89.98)	1163 (92.30)	0.01
Dyspnea on exertion	4659 (14.18)	4468 (14.14)	191 (15.16)	0.31
Angina	9263 (28.20)	8901 (28.17)	362 (28.73)	0.67
Syncope	9825 (29.91)	9464 (29.96)	361 (28.65)	0.32
Ejection fraction, %				
≤34	2624 (7.99)	2494 (7.89)	130 (10.32)	0.02
35–49	3370 (10.26)	3232 (10.23)	138 (10.95)	
≥50	16 881 (51.38)	16 242 (51.41)	639 (50.71)	
Unknown	9978 (30.37)	9625 (30.47)	353 (28.02)	
Creatinine, mg/dL				
<0.9	10 759 (32.75)	10 438 (33.04)	321 (25.48)	<0.001
1.0–1.4	10 133 (30.84)	9776 (30.94)	357 (28.33)	
1.5–1.9	3068 (9.34)	2957 (9.36)	111 (8.81)	
≥2.0	2649 (8.06)	2360 (7.47)	289 (22.94)	
Unknown	6244 (19.01)	6062 (19.19)	182 (14.44)	

(*Continued*)

Table 1. Continued

Patient Characteristics	Overall (n=32 853)	Non-Hispanic Whites (n=31 593)	Non-Hispanic Blacks (n=1260)	*P* Value
BMI, kg/m^2				
<20.0	1800 (5.48)	1714 (5.43)	86 (6.83)	0.01
20.1–25.0	7583 (23.08)	7319 (23.17)	264 (20.95)	
25.1–30.0	10 009 (30.47)	9648 (30.54)	361 (28.65)	
≥30.1	10 992 (33.46)	10 529 (33.03)	463 (36.75)	
Unknown	2469 (7.52)	2383 (7.54)	86 (6.83)	
Diagnosed in inpatient	13 952 (42.47)	13 187 (41.74)	765 (60.71)	<0.001
Hospitalized in year prior	16 611 (50.56)	15 809 (50.04)	802 (63.65)	<0.001
Region				
Midwest	16 687 (50.79)	16 148 (51.11)	539 (42.78)	<0.001
Northeast	3954 (12.04)	3848 (12.18)	106 (8.41)	
South	7502 (22.84)	7016 (22.21)	486 (38.57)	
West	3933 (11.97)	3840 (12.15)	93 (7.38)	
Other/unknown	777 (2.37)	741 (2.35)	36 (2.86)	
Year of diagnosis				
2011–2012	7348 (22.37)	7106 (22.49)	242 (19.21)	0.001
2013–2014	10 919 (33.24)	10 446 (33.06)	473 (37.54)	
2015–2016	14 586 (44.40)	14 041 (44.44)	545 (43.25)	
Insurance				
Commercial	5861 (17.84)	5629 (17.82)	232 (18.41)	<0.001
Medicaid	786 (2.39)	702 (2.22)	84 (6.67)	
Medicare	16 468 (50.13)	15 808 (50.04)	660 (52.38)	
Other	9137 (27.81)	8876 (28.09)	261 (20.71)	
Uninsured	601 (1.83)	578 (1.83)	23 (1.83)	
Income level, median (25th–75th percentile), $*	40 125 (35 814–46 866)	41 792 (35 814–46 866)	39 005 (35 020–43 755)	<0.001
College educated, median (25th–75th percentile), %*	22 (18–28)	22 (18–27)	25 (18–29)	<0.001

Data are given as number (percentage), unless otherwise indicated. BMI indicates body mass index; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; and CVA, cerebrovascular accident.

*Based on zip 3 area.

with ssAS were significantly less likely than their White counterparts to receive treatment. These racial differences remained significant after adjusting for a wide range of patient and provider characteristics, and after limiting the analysis to those who had access to subspecialty care. Nevertheless, we found that the racial gap in AVR use declined over time and became marginal by 2015 to 2016; this finding was partially attributable to more rapid adoption of TAVR among Black versus White patients. Our study confirms that differential use of AVR is important because untreated patients with ssAS continue to experience high rates of mortality.

One of the key findings from our study was that AVR rates among those with ssAS remained alarmingly low, as recently as 2016. Despite the growing use of transcatheter options, only 1 in 3 patients with ssAS receives AVR in the United States.[34,35] Interestingly, only at the level of the cardiologist was there observed to be a significant difference in the likelihood of AVR; for patients not managed by a cardiologist, the likelihood of treatment was similar across races. These results suggest that conditions of the level of the cardiologist are driving the observed gaps in care.

Reasons for undertreatment are complex and include both anatomic and personal circumstances. In a recent analysis of patients from 5 large TAVR centers, anatomic complexity and medical futility each account for roughly 20% of nontreatment, but “patient preference” was cited as the primary reason for nontreatment in 31%.[36] Notably, nearly 15% of patients in the study did not feel that they were adequately engaged in the decision process, and almost one third of medically managed patients questioned whether theirs was the most appropriate treatment strategy. Because our study used an objective assessment of symptoms from the electronic health record (per previously described methods),[28,29] a portion of the treatment gap may have

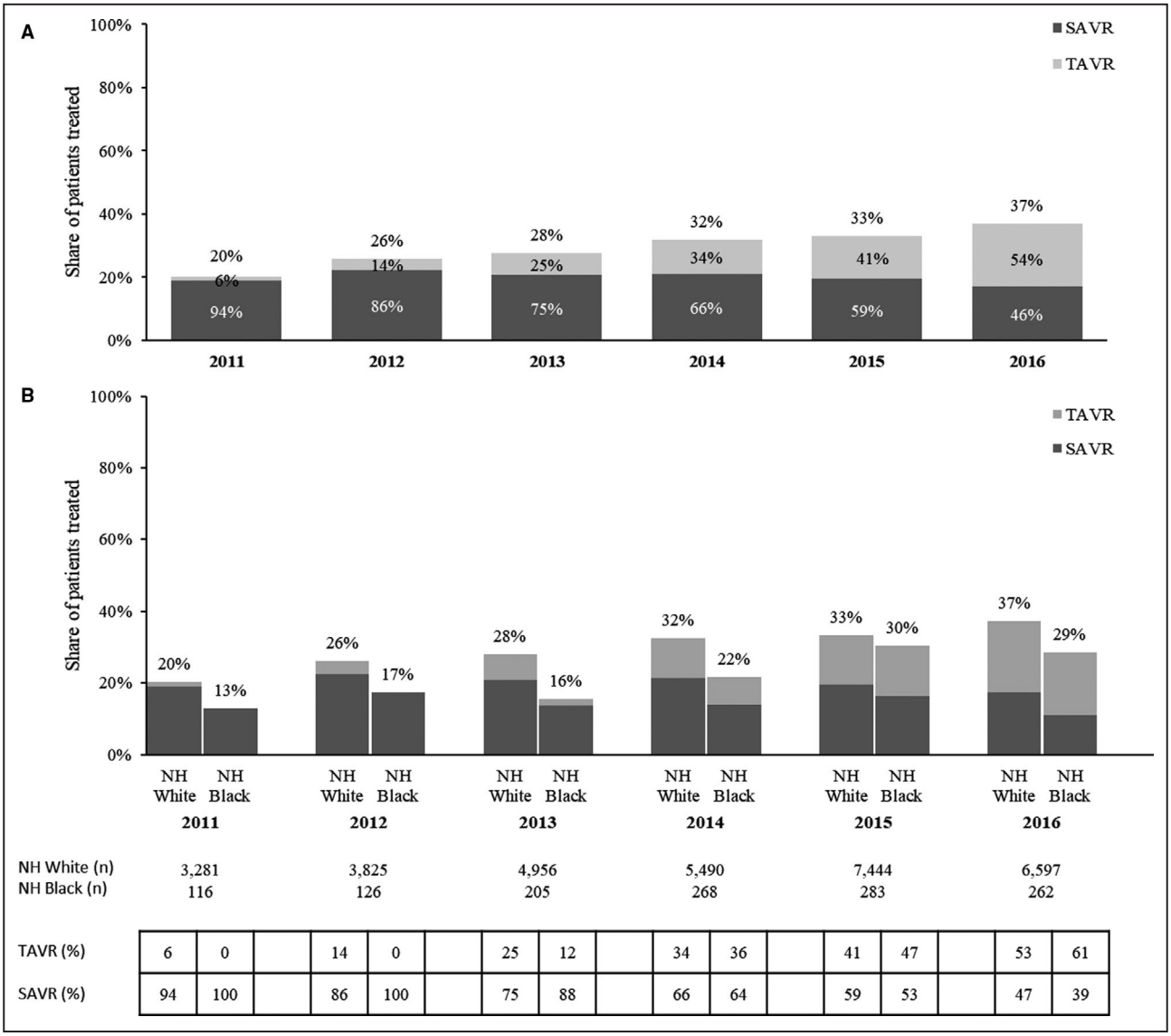

Figure 1. Overall treatment rate and share of transcatheter aortic valve replacement (TAVR) and surgical aortic valve replacement (SAVR).

Treatment rate and share of TAVR and SAVR among all aortic valve replacements over time for patients with symptomatic severe aortic valve stenosis: overall (**A**) and stratified by race (**B**). NH indicates non-Hispanic.

been driven by nonrecognition of symptoms by either the patient or the physician. Prior reviews of ssAS management have highlighted the difficulty in assessing the often nonspecific associated symptoms, particularly in an elderly comorbid population.[37] Further research is needed to fully understand the complex set of circumstances leading to the undertreatment of these patients.

Race-related disparities in the treatment of ssAS were well chronicled before the introduction of TAVR in the United States, but conflicting reports were published by others early after the introduction of TAVR.[38,39] Two recent large cohort studies by Alkhouli et al, both covering the period 2011 to 2016 and using the National Inpatient Sample database and TVT Registry, showed lower rates of TAVR utilization among Black patients.[11,38] Yet, compared with our study, these studies focused on procedure utilization and did not address treatment of the disease process (eg, SAVR/TAVR treatment rates). In addition, these studies did not specifically focus on patients diagnosed with ssAS, included patients without ssAS, and excluded patients undergoing SAVR. During the initial 4 years of our study, we observed that Black individuals with ssAS were significantly less likely than White individuals to receive AVR. Despite adjustment by sequential modeling of patient-level, census-based, and socioeconomic factors, there was no observed improvement in the likelihood of receiving AVR in Black patients. Previously published studies examining 1-year mortality after AVR also found no difference in mortality between Black and White patients, which corresponds

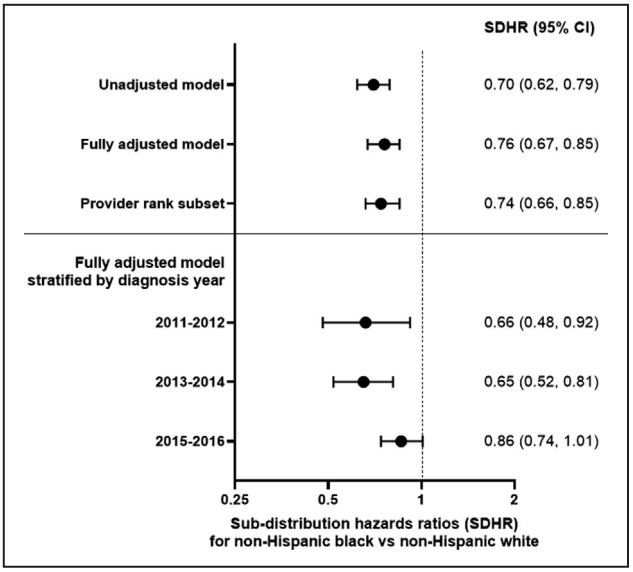

Figure 2. Likelihood of aortic valve replacement (AVR) among Blacks and Whites.
Likelihood of AVR among non-Hispanic Blacks vs non-Hispanic Whites by univariate and multivariate analysis, including a subset analysis controlling for provider behavior by including provider referral rank. The fully adjusted model was then stratified by year of diagnosis to understand changes over time in the race-associated likelihood of AVR. Note: hazard ratio<1 suggests Black patients are less likely to receive treatment. SDHR indicates subdistribution hazards ratio.

with our findings,[11,38–43] thereby emphasizing the need for future studies that will focus on factors not measured in the current study.

As highlighted in a comprehensive review by Batchelor et al, it is important for healthcare systems to identify and review treatment bias in underserved minorities with ssAS when considering their performance in treating such patients.[44] Our study identified disparities in treatment of ssAS in Black patients compared with White patients, which highlights that gaps in treatment likely still exist, even with the upwards trend in the number of TAVR procedures performed.

Although race-related differences were observed in the first 4 years of our analysis, in more recent years, these differences are no longer present. The dissipation of this effect has tracked a greater uptake of less-invasive TAVR technology in the Black community, a finding that is not easily explained, but may indicate a lower barrier to treatment with TAVR (versus SAVR) procedures in the Black community. Prior studies have suggested that Black individuals are more likely than White individuals to refuse invasive cardiovascular procedures when such interventions are indicated and recommended by healthcare providers.[10,45] The reasons for Black patients' higher refusal rates have yet to be fully elucidated, but may be attributable to differences in cultural preferences for procedural risk aversion or inability of providers to communicate risk

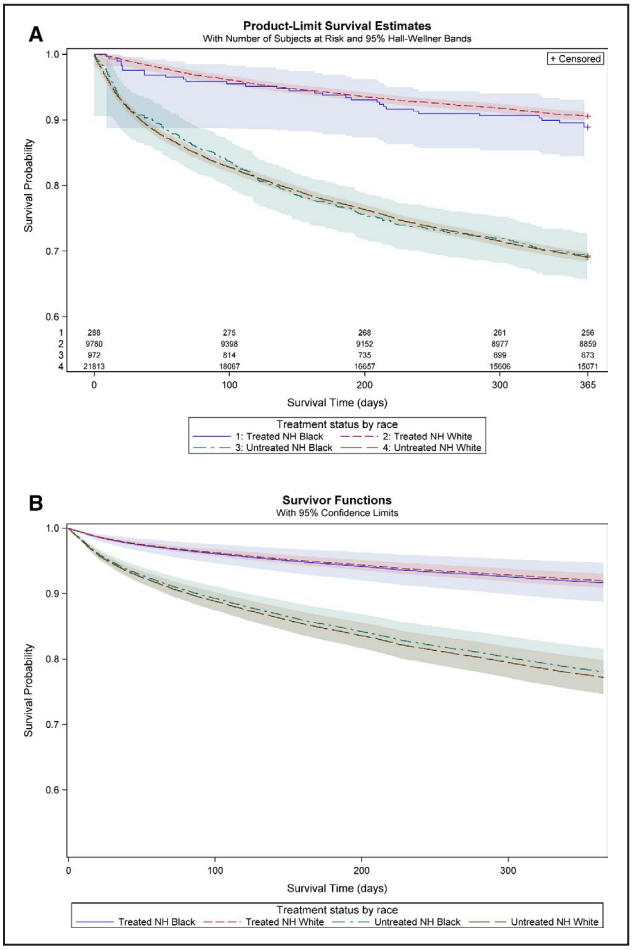

Figure 3. Overall survival by treatment status and race.
Kaplan-Meier (KM) curves for overall survival stratified by treatment and stratified by race: unadjusted KM curve (**A**) and adjusted KM curve (**B**). NH indicates non-Hispanic.

to underrepresented racial and ethnic groups. Other barriers specifically related to the healthcare system include referral and treatment bias, subconscious physician bias, and lack of culturally appropriate communication.[44] Poor awareness about treatment bias, as well as lack of cultural awareness and sensitivity in physician-patient interactions, may result in additional treatment barriers.[44] Targeted efforts may help address issues of undertreatment for all populations, but most notably, underrepresented racial and ethnic groups. Improvements in screening protocols that encourage earlier diagnosis, as well as the implementation of patient and provider educational programs, such as widespread use of shared decision making, direct-to-patient educational efforts, and referral protocols to improve diagnosis-to-referral times and reduce loss-to-follow-up rates, may all increase the uptake of this life-saving therapy.

Our study had several limitations. First, we identified severe AS based on physician records, without reference to an underlying confirmatory echocardiogram;

therefore, there is a risk of misdiagnosis. To address this potential limitation, we evaluated survival between patients identified as having mild, moderate, and severe AS; this analysis demonstrated the expected survival decrement, adding credence to the severity recorded in the notes. We also used a process of automated abstraction of the medical record to identify symptoms within 6 months of severe AS diagnosis. This is consistent with other seminal reports[28]; however, it may have resulted in earlier detection of ssAS than in other studies, perhaps explaining the lower than expected 1-year mortality rates observed in this cohort. Second, we relied on the medical record for identification of racial background, which may or may not correspond to a patient's self-designation. Nonetheless, misclassification of race would have been expected to blunt racial differences observed in our analysis. Third, this data set included a proportionately smaller cohort of Black patients, limiting our ability to evaluate other questions, such as race-related differences in TAVR versus SAVR outcomes. Fourth, reasons for undertreatment are complex, and the low rates of treatment reported herein may have been impacted by patient preferences for a medical management strategy; however, the extent to which these decisions are truly informed has been questioned.[36] Fifth, this analysis included observational data, prone to unmeasured biases, which should be considered when interpreting comparative results. As for unmeasured confounders, our analyses were based on the assumption of random distribution. Sixth, this analysis does not address race-based differences in rates of ssAS diagnosis. In fact, we have shown herein that a substantially greater proportion of Blacks versus Whites receive an initial diagnosis of ssAS during acute-care hospitalizations; such a finding may suggest an outpatient setting diagnosis failure in the Black community. Seventh, analyses using the integrated data set validated the rates of race and treatments in the final cohort of this study; however, given the small sample size of Black patients in the integrated set, no further sensitivity analyses were applied herein. Finally, the treatment of AVR is a rapidly evolving field, and our results point to incremental improvements in both overall treatment rates and racial disparities over time. Although we used the latest available data for this analysis (through 2016, allowing at least 12 months for treatment and follow-up), it is likely that progress has occurred since the accrual of these data; the extent of such improvement cannot be verified in this analysis.

In conclusion, treatment rates for ssAS remained low in the United States through the year 2016, despite availability of the less invasive TAVR alternative to SAVR. Overall, Black patients with ssAS were less likely than White patients to receive AVR; however, we also found that there has been a more rapid adoption of TAVR by Black patients than White patients, which appears to have contributed to narrowing the racial gap in AVR use in recent years. Because AVR is associated with improved outcomes in appropriately selected patients with ssAS, these findings demonstrate that more should be done to increase rates of AVR for clinically appropriate patients with ssAS in both White and Black communities.

ARTICLE INFORMATION

Received February 12, 2020; accepted June 19, 2020.

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Acknowledgments

The authors would like to thank Erin Campbell, MS, for her editorial contributions to this manuscript; Stuart Gander, Jason Jager, and Otto Liska, of Boston Consulting Group, for their support of this project; and Boye Gricar, PhD, Sibyl Munson, PhD, and Laxmi Gannu, of Boston Strategic Partners, Inc, for assistance with manuscript preparation.

Sources of Funding

The study was funded by Edwards Lifesciences; a portion of Dr Brennan's research effort was funded by the Burroughs Wellcome Fund.

Disclosures

Dr Brennan reports consulting and speaking funds from Edwards Lifesciences and AtriCure. Dr Boero reports consulting for Edwards Lifesciences. Dr Lowenstern reports funding through National Institutes of Health T-32 training grant 5 T32 HL069749-14. Dr Thourani reports research and advising for Edwards Lifesciences. Dr Vemulapalli reports grants/contracts: American College of Cardiology, Society of Thoracic Surgeons, Abbott Vascular, Boston Scientific, National Institutes of Health, and Patient-Centered Outcomes Research Institute; and advisory board/consulting: Boston Scientific, Janssen, and Premiere. Dr Wang reports research grants to the Duke Clinical Research Institute from AstraZeneca, Bristol Myers Squibb, Cryolife, Portola, and Regeneron; and consulting honoraria from AstraZeneca. Dr Peterson reports being the coinvestigator for the ACC STS TAVR (American College of Cardiology Society of Thoracic Surgeons Transcatheter Aortic Valve Replacement) Registry. The remaining authors have no disclosures to report.

Supplementary Materials

Tables S1–S4
Figures S1–S2

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Supplemental Material

Table S1. Validation of Optum Physician's Report of Aortic Stenosis.

To validate Optum physician's report of aortic stenosis, we leveraged the following approach:

- Among 32,853 patients with symptomatic severe aortic stenosis (ssAS) in the final cohort, we pulled the closest echo readings to severe aortic stenosis (sAS) diagnosis identified by physicians' notes
  - Echo readings included aortic valve area (AVA), velocity, mean gradient, and left ventricular ejection fraction (LVEF)
  - The closest echo readings in the seven days before sAS diagnosis from physicians' notes were pulled. The LVEF is included only if it is on the same day of AVA/velocity/mean gradient. The completeness of echo-readings is shown as below

Patients with available specific echo readings	Completeness of data
AVA	9,465 (28.8%)
Velocity	3,908 (11.9%)
Mean gradient	9,631 (29.3%)
LVEF	11,296 (34.4%)
AVA + Velocity	1,750 (5.3%)
AVA + Velocity + Mean gradient	1,394 (4.2%)
AVA + Velocity + Mean gradient + LVEF	1,219 (3.7%)

  - We restricted patients to those who had all the AVA, velocity, and mean gradient OR to those who had all the AVA, velocity, mean gradient and LVEF, then we compared the difference in sAS under the definition of AS echo readings and physicians' notes. The differences are listed as below.
    - AHA guidelines were applied here to define the severity of AS by using AVA, velocity, or mean gradient
    - If any of echo readings is identified as severe, then this patient is identified as severe AS

	Classification as severe by physicians' notes
Classification by Echo Tests	Patients with all available echo readings for AVA, velocity, mean gradient, and LVEF
Mild AS	14 (1.2%)
Moderate AS	218 (17.9%)
Severe AS	987 (81.0%)

  - The severity of AS by echo readings stratified by LVEF level is shown as below.

	Classification by Echo Tests		
Level of LVEF	Mild AS	Moderate AS	Severe AS
Patients with all available echo readings for AVA, velocity, mean gradient, and LVEF			
< 30%	0 (0.0%)	21 (9.6%)	76 (7.7%)
30% - 49%	4 (28.6%)	27 (12.4%)	169 (17.1%)
≥ 50%	10 (71.4%)	170 (78.0%)	742 (75.2%)

  - The severity of AS by echo readings stratified by left ventricular outflow tract velocity time integral (LVOT VTI) level is shown as below. Kappa statistics were calculated and are provided below the table.

	Classification by Echo Tests		
Level of LVOT VTI	Mild AS	Moderate AS	Severe AS
Patients with all available echo readings for AVA, velocity, mean gradient, and LVEF			
< 18	5 (83.3%)	8 (16.0%)	63 (25.3%)
18 - 22	0 (0.0%)	13 (26.0%)	67 (26.9%)
> 22	1 (16.7%)	29 (58.0%)	119 (47.8%)

Note: Kappa statistics (95% CI) for agreement for AS severity and LVOT (n=1,219): Simple Kappa 0.0018 -0.0512 (-0.1095 - 0.0071); Weighted Kappa: -0.0355 (-0.0902 - 0.0192)

- We also checked the distribution of AS severity classified by echo readings stratified by white and black patients among sAS patients from physician's notes to ensure that the racial disparities were not generated by sample selection. Kappa statistics were calculated and are presented below the table.

	Classification as severe by physicians' notes	
Classification by Echo Tests	Patients with all available echo readings for AVA, velocity, mean gradient, and LVEF	
	White	Black
Mild AS	14 (1.2%)	-
Moderate AS	207 (17.6%)	11 (25.6%)
Severe AS	955 (81.2%)	32 (74.4%)

Note: Kappa statistics (95% CI) for agreement for AS severity and LVEF (n=305): Simple Kappa 0.0018 (-0.0373 - 0.0410); Weighted Kappa: -0.009 (-0.0492 - 0.0311)

Interpretation:
81% of patients with severe AS by physician notes were also found to have severe AS by one or more echo criteria. An additional 17.9% of patients with severe AS by physician notes had moderate AS by echo criteria. Of these, 22% had a reduced LVEF and some proportion of those may reasonably be considered to represent low flow, low gradient patients. In this validation cohort, 1.2% of patients with severe AS by physician notes were classified as mild AS by echo criteria.

Table S2. ICD-9 and -10 Procedure and Diagnostic Codes and CPT Codes Used to Evaluate the Patient's Baseline Condition.

	ICD-9	ICD-10	CPT
Atrial fibrillation	42731	I480-I484, I489, I4891-I4892	
Cancer	140-172, 174-194, 196-198, 1990-1991, 200-208, 1950-1958	C0-C1, C20-C26, C30-C34, C37-C41, C43, C45-C58, C60-C85, C88, C90-C97	
Cardiac conduction disorders	4260, 4261, 42611, 42612, 42613, 4262, 4263, 4264, 42650, 42651, 42652, 42653, 42654, 4266	I440, I441, I442, I443, I4430, I4439, I450, I451, I4510, I4519, I452, I444, I445, I446, I4460, I4469, I447, I453	
COPD	49, 500, 501, 502, 503, 504, 505	I278, I279, J684, J701, J703, J40, J41, J42, J43, J44, J45, J46, J47, J60, J61, J62, J63, J64, J65, J66, J67	
Dementia	290	F051, G311, F00, F01, F02, F03, G30	
Diabetes without complications	2500, 2501, 2502, 2503, 2507	E100, E101, E106, E108, E109, E110, E111, E116, E118, E119, E120, E121, E126, E128, E129, E130, E131, E136, E138, E139, E140, E141, E146, E148, E149	
Diabetes with complications	2504, 2505, 2506	E102, E103, E104, E105, E107, E112, E113, E114, E115, E117, E122, E123, E124, E125, E127, E132, E133, E134, E135, E137, E142, E143, E144, E145, E147	
Prior myocardial infarction	410, 412	I21, I22, I252	
Osteoarthritis (OA)	715	M15-M19	
Peripheral vascular disease (PVD)	4439, 441, 7854, V434	I731, I738, I739, I771, I790, I792, K551, K558, K559, Z958, Z959, I70, I71	
Heart failure	4282, 42820, 42821, 42822, 42823, 4283, 42830, 42831, 42832, 42833, 4284, 42840, 42841, 42842, 42843	I502, I5020, I5021, I5022, I5023, I503, I5030, I5031, I5032, I504, I5040, I5041, I5042, I5043	
Moderate to severe renal disease	582, 5830, 5831, 5832, 5833, 5834, 5835, 5836, 5837, 585, 586, 588	I120, I131, N032, N033, N034, N035, N036, N037, N052, N053, N054, N055, N056, N057, N250, Z490, Z491, Z492, Z940, Z992, N18, N19	
Supplemental oxygen use	V462	Z9981	E1390, E1391, E0424, E0439, E1405, E1406, E0431, E0434, E1392, E0433, K0738, E0441, E0442, E0443,

			E0444, E0425, E0430, E0431, E0433, E0434, E0435, E0440, E0445, E0446
Percutaneous cardiac procedures (including PCI, ablation, and transcatheter mitral)	00.66, 36.06, 36.07, 37.26, 37.27, 37.33, 37.34, 35.97	270346, 027034Z, 02703D6, 02703DZ, 270446, 027044Z, 02704D6, 02704DZ, 271346, 027134Z, 02713D6, 02713DZ, 271446, 027144Z, 02714D6, 02714DZ, 272346, 027234Z, 02723D6, 02723DZ, 272446, 027244Z, 02724D6, 02724DZ, 273346, 027334Z, 02733D6, 02733DZ, 273446, 027344Z, 02734D6, 02734DZ, 02563ZZ, 02573ZZ, 025K3ZZ, 025L3ZZ, 02B63ZZ, 02B73ZZ, 02BK3ZZ, 02BL3ZZ, 02560ZZ, 02570ZZ, 025K0ZZ, 025L0ZZ, 02B60ZZ, 02B70ZZ, 02BK0ZZ, 02BL0ZZ, 02T80ZZ, 02K80ZZ, 02K83ZZ, 02K84ZZ, 4A023FZ, 02UG3JZ	92937, 92941, 92943, 92920, 92924, 92928, 92933, 33418, 33419
Pacemaker/ICD	00.51, 00.54, 37.94, 37.95, 37.96, 37.80, 37.81, 37.82, 37.83	02HK0KZ, 02HK3KZ, 02HK4KZ, 02HL0KZ, 02HL3KZ, 02HL4KZ, 0JH609Z, 0JH639Z, 0JH809Z, 0JH839Z, 0JH609Z, 0JH609Z, 0JH639Z, 0JH639Z, 0JH809Z, 0JH839Z, 0JPT0PZ, 0JPT3PZ, 02H60KZ, 02H60KZ, 02H63KZ, 02H63KZ, 02H64KZ, 02H64KZ, 02H70KZ, 02H70KZ, 02H73KZ, 02H73KZ, 02H74KZ, 02H74KZ, 02HK0KZ, 02HK0KZ, 02HK3KZ, 02HK3KZ, 02HK4KZ, 02HK4KZ, 02HL0KZ, 02HL0KZ, 02HL3KZ, 02HL3KZ, 02HL4KZ, 02HL4KZ, 02PA0MZ, 02PA3MZ, 02PA4MZ, 02PAXMZ, 0JH608Z, 0JH608Z, 0JH638Z, 0JH638Z, 0JH808Z, 0JH808Z, 0JH838Z, 0JH838Z, 0JPT0PZ, 0JPT3PZ, 02H63KZ, 02H73KZ, 02HK3KZ, 02HL3KZ, 02HN0KZ,	33202, 33203, 33216, 33217, 33224, 33230, 33231, 33240, 33249, 33270, 33271, 33202, 33203, 33206, 33207, 33208, 33212, 33213, 33221, 33216, 33217, 33224

		02HN4KZ, 0JH608Z, 0JH638Z, 0JH808Z, 0JH838Z, 0JH60PZ, 0JH60PZ, 0JH63PZ, 0JH63PZ, 0JH80PZ, 0JH80PZ, 0JH83PZ, 0JH83PZ, 0JPT0PZ, 0JPT3PZ, 0JH604Z, 0JH634Z, 0JH804Z, 0JH834Z, 0JH605Z, 0JH635Z, 0JH805Z, 0JH835Z, 0JH606Z, 0JH636Z, 0JH806Z, 0JH836Z	
Hemodialysis	39.95, 54.98	5A1D00Z, 5A1D00Z, 3E1M39Z	90935, 90937, 90945, 90947, 4055F

CPT, Current Procedural Terminology; ICD-9, International Classification of Diseases, Ninth Revision; ICD-10, International Classification of Diseases, Tenth Revision; PCI, percutaneous coronary intervention; PVD, peripheral vascular disease; SDS, signs, diseases, and symptoms

Table S3. Patient Characteristics, Provider Subset.

	Overall	**Non-Hispanic White**	**Non-Hispanic Black**	**p-value**
	(n=23,839)	**(n=22,975)**	**(n=864)**	
Age				<0.001
≤64	3,312 (13.89%)	3,096 (13.48%)	216 (25%)	
65-79	8,944 (37.52%)	8,589 (37.38%)	355 (41.09%)	
80+	11,563 (48.5%)	11,270 (49.05%)	293 (33.91%)	
Unknown	0 (0%)	20 (0.09%)	20 (0.08%)	
Women	10,912 (45.77%)	10,450 (45.48%)	462 (53.47%)	<0.001
Charlson Comorbidity Index				<0.001
0	6,115 (25.65%)	5,960 (25.94%)	155 (17.94%)	
1	5,066 (21.25%)	4,925 (21.44%)	141 (16.32%)	
2	3,743 (15.7%)	3,624 (15.77%)	119 (13.77%)	
3	2,954 (12.39%)	2,831 (12.32%)	123 (14.24%)	
4+	5,961 (25.01%)	5,635 (24.53%)	326 (37.73%)	
Atrial fibrillation	6,883 (28.87%)	6,725 (29.27%)	158 (18.29%)	<0.001
Cancer	3,154 (13.23%)	3,025 (13.17%)	129 (14.93%)	0.13
Cardiac conduction disorders	2,430 (10.19%)	2,351 (10.23%)	79 (9.14%)	0.30
COPD	2,422 (10.16%)	2,338 (10.18%)	84 (9.72%)	0.66
Dementia	541 (2.27%)	514 (2.24%)	27 (3.13%)	0.09
Diabetes without complications	6,669 (27.98%)	6,328 (27.54%)	341 (39.47%)	<0.001
Diabetes with complications	1,450 (6.08%)	1,357 (5.91%)	93 (10.76%)	<0.001
Prior myocardial infarction	2,795 (11.72%)	2,666 (11.6%)	129 (14.93%)	0.03
Osteoarthritis	3,426 (14.37%)	3,301 (14.37%)	125 (14.47%)	0.93
Peripheral vascular disease	3,781 (15.86%)	3,629 (15.8%)	152 (17.59%)	0.16
Heart failure	5,008 (21.01%)	4,773 (20.77%)	235 (27.20%)	<0.001
Moderate to severe renal disease	5,078 (21.3%)	4,764 (20.74%)	314 (36.34%)	<0.001
Current smoking	2,632 (11.04%)	2,489 (10.83%)	143 (16.55%)	<0.001
Supplemental oxygen	986 (4.14%)	946 (4.12%)	40 (4.63%)	0.46
Percutaneous cardiac procedures	589 (2.47%)	573 (2.49%)	16 (1.85%)	0.23
Pacemaker	325 (1.36%)	313 (1.36%)	12 (1.39%)	0.95
Hemodialysis	332 (1.39%)	277 (1.21%)	55 (6.37%)	<0.001
Dyspnea	21,559 (90.44%)	20,758 (90.35%)	801 (92.71%)	0.02
Dyspnea on exertion	3,604 (15.12%)	3,471 (15.11%)	133 (15.39%)	0.82
Angina	7,074 (29.67%)	6,807 (29.63%)	267 (30.9%)	0.42

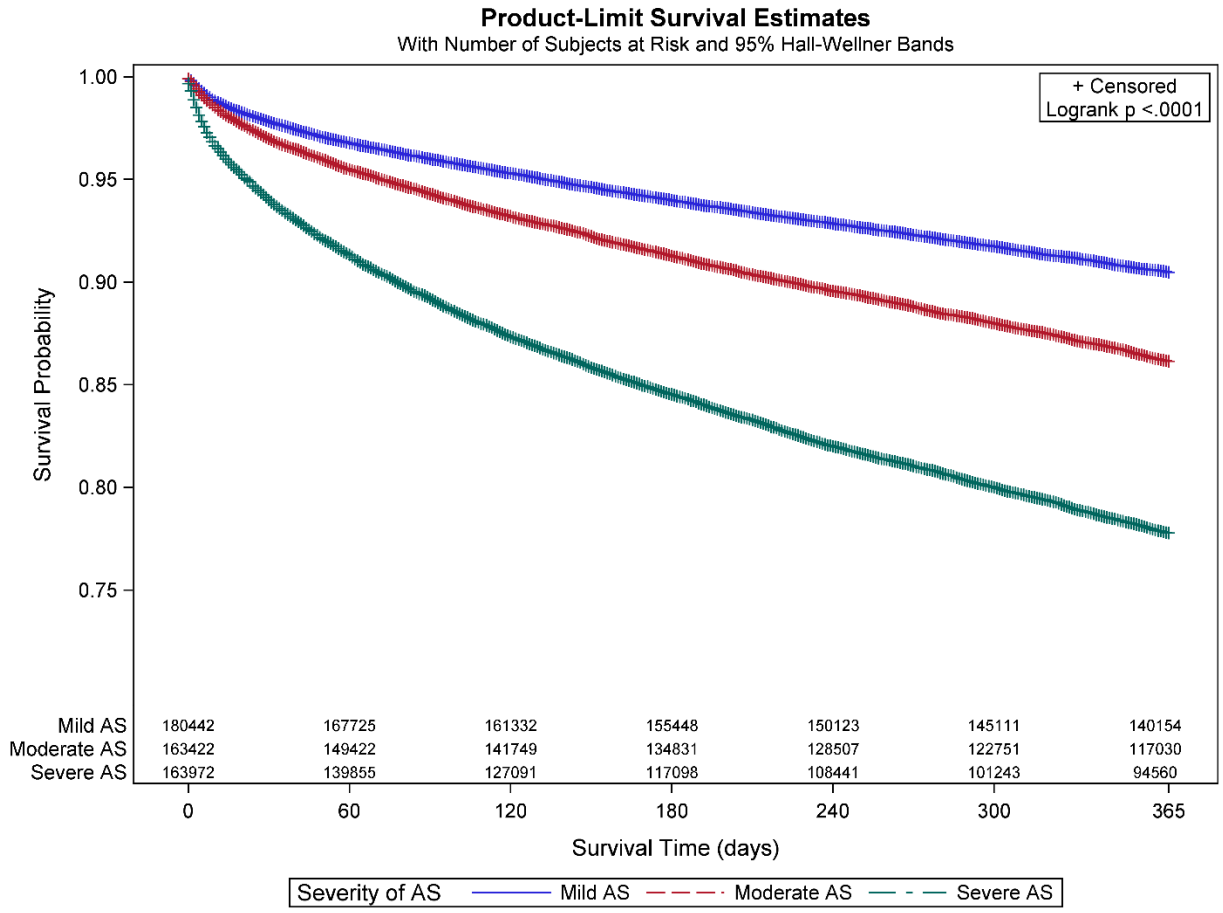
Syncope	7,260 (30.45%)	6,995 (30.45%)	265 (30.67%)	0.89
Ejection fraction				0.04
≤34	1,879 (7.88%)	1,791 (7.80%)	88 (10.19%)	
35-49	2,513 (10.54%)	2,415 (10.51%)	98 (11.34%)	
50+	12,795 (53.67%)	12,337 (53.7%)	458 (53.01%)	
Unknown	6,652 (27.9%)	6,432 (28%)	220 (25.46%)	
Creatinine				<0.001
<0.9	7,734 (32.44%)	7,529 (32.77%)	205 (23.73%)	
1.0-1.4	7,564 (31.73%)	7,308 (31.81%)	256 (29.63%)	
1.5-1.9	2,172 (9.11%)	2,088 (9.09%)	84 (9.72%)	
2.0+	1,818 (7.63%)	1,631 (7.1%)	187 (21.64%)	
Unknown	4,551 (19.09%)	4,419 (19.23%)	132 (15.28%)	
BMI				0.01
<20.0	1,137 (4.77%)	1,084 (4.72%)	53 (6.13%)	
20.1-25.0	5,361 (22.49%)	5,201 (22.64%)	160 (18.52%)	
25.1-30.0	7,502 (31.47%)	7,234 (31.49%)	268 (31.02%)	
30.1+	8,382 (35.16%)	8,046 (35.02%)	336 (38.89%)	
Unknown	1,457 (6.11%)	1,410 (6.14%)	47 (5.44%)	
Diagnosed in inpatient	9,057 (37.99%)	8,571 (37.31%)	486 (56.25%)	<0.001
% hospitalized in year prior	11,200 (46.98%)	10,680 (46.49%)	520 (60.19%)	<0.001
Region				<0.001
Midwest	12,075 (50.65%)	11,699 (50.92%)	376 (43.52%)	
Northeast	2,721 (11.41%)	2,654 (11.55%)	67 (7.75%)	
South	5,763 (24.17%)	5,415 (23.57%)	348 (40.28%)	
West	2,738 (11.49%)	2,685 (11.69%)	53 (6.13%)	
Other/unknown	542 (2.27%)	522 (2.27%)	20 (2.31%)	
Year of diagnosis				0.02
2011-2012	5,086 (21.33%)	4,933 (21.47%)	153 (17.71%)	
2013-2014	7,989 (33.51%)	7,673 (33.40%)	316 (36.57%)	
2015-2016	10,764 (45.15%)	10,369 (45.13%)	395 (45.72%)	
Insurance				<0.001
Commercial	4,409 (18.49%)	4,249 (18.49%)	160 (18.52%)	
Medicaid	577 (2.42%)	526 (2.29%)	51 (5.9%)	
Medicare	11,942 (50.09%)	11,495 (50.03%)	447 (51.74%)	
Other	6,458 (27.09%)	6,267 (27.28%)	191 (22.11%)	
Uninsured	453 (1.9%)	438 (1.91%)	15 (1.74%)	
Median area income level (25^{th}, 75^{th})	$40,125 ($35,814, $46,714)	$40,125 ($35,814, $46,866)	$39,005 ($35,020, $42,702)	<0.001
Median area share college educated (25^{th}, 75^{th})	22% (18%, 27%)	22% (18%, 27%)	25% (18%, 29%)	<0.001

Table S4. Sub-distribution Hazard Ratios for Sequential Model Building for the Association between Race and the Likelihood of AVR.

Model	Sub-distribution HR for Non-Hispanic Black vs. non-Hispanic White (95% CI)	Sub-distribution HR for Non-Hispanic Black vs. non-Hispanic White (95% CI) among patients with available left ventricular ejection fraction, creatinine, and body mass index.
Model 1: Unadjusted	0.70 (0.62, 0.79)	0.70 (0.62, 0.79)
Model 2: Adjusted by patient comorbidities	0.73 (0.65, 0.82)	0.76 (0.65, 0.88)
Model 3: Adjusted by census division	0.72 (0.64, 0.81)	0.76 (0.65, 0.88)
Model 4: Adjusted by insurance status	0.75 (0.66, 0.84)	0.78 (0.67, 0.90)
Model 5: Adjusted by socioeconomic status	0.74 (0.66, 0.83)	0.76 (0.65, 0.88)
Model 6: Adjusted by patient factors and the provider referral rank (subset analysis)	0.74 (0.66, 0.85)	0.77 (0.65, 0.91)
Model 7: Adjusted by all patient factors excluding provider rank	0.76 (0.67, 0.85)	0.79 (0.68, 0.92)

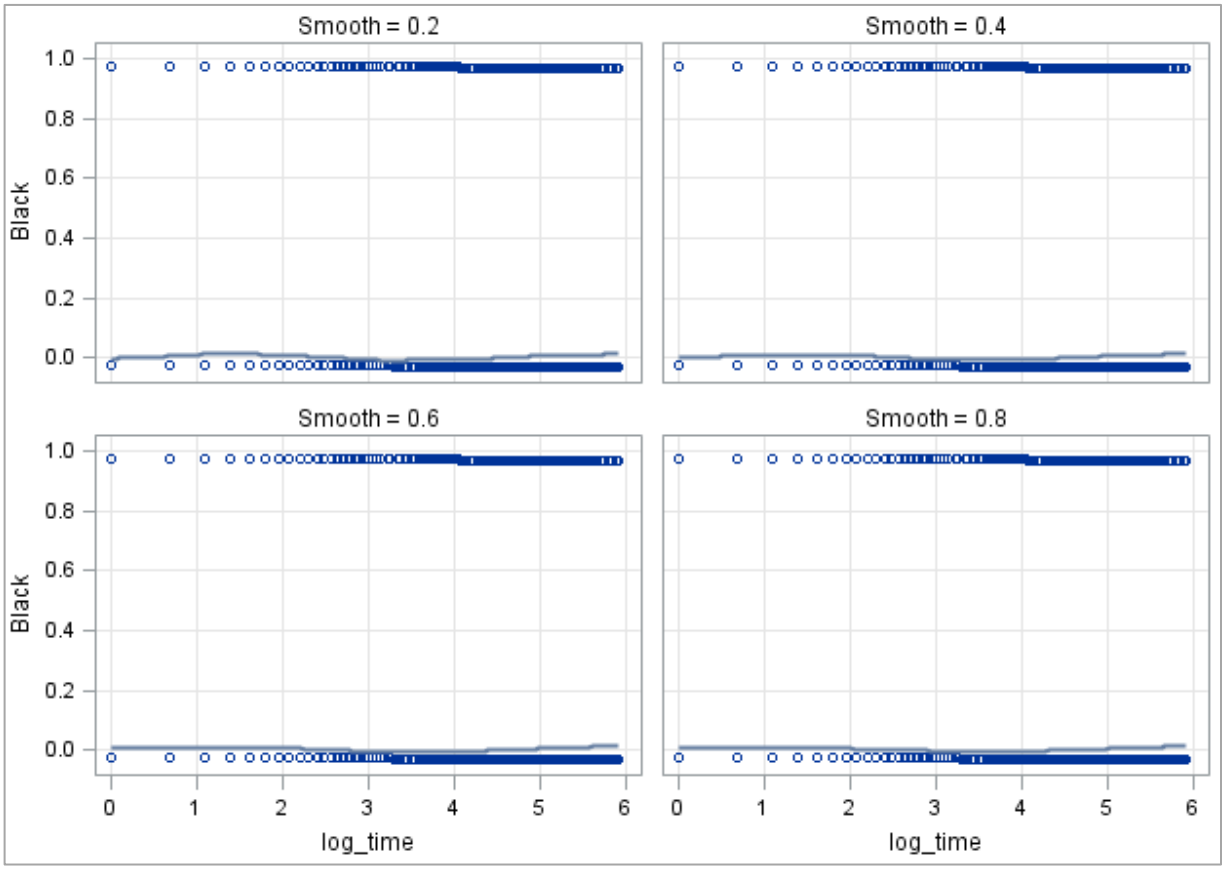
AVR, aortic valve replacement; CI, confidence interval; HR, hazard ratio

Figure S1. Unadjusted One-Year Mortality.



Kaplan Meier curves for the overall study cohort before inclusion and exclusion criteria were applied and with at least 1 year of patient history before their AS diagnosis. Survival was stratified by severity of aortic stenosis over a 1-year period post-diagnosis. Kaplan Meier analysis was performed to confirm the expected stratification of patient survival based on mild, moderate, and severe disease. Differences between the survival curves were tested using the log-rank test. The survival curves were significantly different from each other ($p < 0.001$). The number of patients with mild, moderate and severe AS are listed above each 60-day time interval.
AS: aortic stenosis.

Figure S2. Schoenfeld Residuals for Time versus Patient Race for the Outcome of AVR.



Schoenfeld residuals were used to assess that the Cox Proportional Hazards model assumptions were met. Time vs. patient race (black) is depicted for the outcome of AVR over the year following severe symptomatic aortic stenosis (ssAS) diagnosis. The top blue line represents black patients and the lower blue line represents white patients. A log transformation was applied to the time variable.
AVR, aortic valve replacement.