Published in final edited form as:
Am Heart J. 2010 July ; 160(1): 152–158. doi:10.1016/j.ahj.2010.04.010.

Race/Ethnic Disparities in Left Ventricular Diastolic Function in a Tri-Ethnic Community Cohort

Cesare Russo, MD[1], **Zhezhen Jin, PhD**[2], **Shunichi Homma, MD**[1], **Tatjana Rundek, MD, PhD**[4], **Mitchell S.V. Elkind, MD**[3], **Ralph L. Sacco, MD, MS**[4,5], and **Marco R. Di Tullio, MD**[1]

[1]Department of Medicine, Columbia University, New York, NY

[2]Department of Biostatistics, Columbia University, New York, NY

[3]Department of Neurology, Columbia University, New York, NY

[4]Department of Neurology, University of Miami, Miami, FL

[5]Department of Epidemiology and Human Genetics, University of Miami, Miami, FL

Abstract

Background—Racial-ethnic disparities exist in cardiovascular risk factors, morbidity and mortality. Left ventricular (LV) diastolic dysfunction is a predictor of mortality and of cardiovascular outcome including incident heart failure. We sought to assess whether race-ethnic differences in diastolic function exist. Such differences may contribute to the observed disparities in cardiovascular outcomes.

Methods—Two-dimensional echocardiography was performed in 760 participants (539 Hispanic, 117 non-Hispanic black, 104 non-Hispanic white) from the Cardiac Abnormalities and Brain Lesions (CABL) study. LV diastolic function was assessed by standard Doppler flow profile and tissue Doppler imaging (TDI). Early (E) and late (A) trans-mitral diastolic flow, and mitral annulus early diastolic velocities (E') were recorded and E/A and E/E' ratios were calculated.

Results—Blacks and Hispanics had higher body mass index ($p=0.04$, $p<0.01$), higher prevalence of hypertension (both $p\leq0.05$) and diabetes (both $p<0.01$), and lower level of education (both $p<0.01$) compared to whites. In age- and sex-adjusted analyses, Hispanics and blacks showed worse indices of diastolic function than whites. Hispanics had lower E/A ratio ($p=0.01$), lower E' and higher E/E' (both $p<0.01$) than whites, whereas blacks had lower E' ($p<0.05$) and a trend toward a higher E/E' ratio ($p=0.09$) compared with whites. These race-ethnic differences in diastolic function were attenuated in multivariate models adjusted for cardiovascular risk factors.

Conclusions—Differences in LV diastolic function exist between race-ethnic groups. However, modifiable cardiovascular risk factors and socio-demographic variables, rather than intrinsic race-ethnic heterogeneity, seem to explain most of the observed differences.

Corresponding author: Marco R. Di Tullio, MD, Division of Cardiology, Columbia University, College of Physicians & Surgeons, 630 West 168th Street, New York, N.Y. 10032, Phone: 212-305-8805, Fax: 212-342-6051, md42@columbia.edu.

Conflict of interest: None.

Keywords

Diastolic function; Race; Ethnicity; Risk factors; Echocardiography

Introduction

Despite a general improvement in life expectancy and health conditions in the past decades, strong health disparities by race-ethnicity still exist in the United States.[1-3] Several reports have shown that disparities in the incidence of cardiovascular diseases and outcome are largely explained by the uneven distribution of cardiovascular risk factors and by socio-economic variables. The strength of the association between individual cardiovascular risk factors and the development of cardiovascular disease does not seem to be affected by race-ethnicity per se.[2,4-9]

Heart failure is a leading cause of morbidity and mortality in the U.S., and its prevalence is steadily rising.10 Blacks and Hispanics show higher incidence rates of heart failure compared with whites.11-[14] An impairment of left ventricular (LV) diastolic function with preserved systolic function is the cause of heart failure in up to 50% of the patients.[15,]16 Diastolic dysfunction can be an asymptomatic, early precursor of heart failure,17 has been shown to have prognostic value in population settings,18-[21] and can be easily detected by transthoracic echocardiography using traditional Doppler flow velocity and tissue Doppler techniques.[22,23]

Inter-racial differences in LV diastolic function might be related to different risk of developing heart failure, which in turn could partially explain the differences in cardiovascular burden observed among different race-ethnic groups. Recent data suggest that, in patients with arterial hypertension, racial differences in diastolic function exist that may be independent of the presence of cardiovascular risk factors.[24] The aim of the present study was to evaluate possible differences in diastolic function parameters in a tri-ethnic community cohort, and their relationship with cardiovascular risk factors and other potentially associated variables.

Methods

Study population

The study cohort was derived from the Northern Manhattan Study (NOMAS), an epidemiological study evaluating the incidence and risk factors for stroke in the population of Northern Manhattan. The study design and recruitment details regarding NOMAS have been described previously.[25] From September 2005 through December 2008, NOMAS subjects over age 50 that voluntarily agreed to undergo a brain MRI study and a more extensive echocardiographic evaluation including diastolic function assessment were included in the Cardiac Abnormalities and Brain Lesion (CABL) study. This subset of individuals constitutes the study population of the present report. Informed consent was obtained from all study participants. The study was approved by the Institutional Review Board of Columbia University Medical Center.

Risk Factors Assessment

Hypertension was defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg at the time of the visit (mean of two readings), or patient's self-reported history of hypertension or of anti-hypertensive medications. Diabetes mellitus was defined as fasting blood glucose ≥126 mg/dL or patient's self-reported history of diabetes or of diabetes medications. Hypercholesterolemia was defined as total serum cholesterol >240 mg/dL, a patient's self-report of hypercholesterolemia or of use of lipid-lowering treatment. Coronary

artery disease was defined as a history of myocardial infarction, coronary artery bypass grafting, or percutaneous coronary intervention. Race-ethnicity was determined by self-report using a questionnaire modeled after the U.S. Census Bureau. Individuals were divided into three groups: non-Hispanic whites (whites), non-Hispanic blacks (blacks), and Hispanics.

Echocardiographic Assessment

Transthoracic echocardiography was performed using a commercially available system (iE 33, Philips, Andover, MA) by a trained, registered sonographer according to a standardized protocol. LV end-diastolic diameter (LVDD), inter-ventricular septum thickness and posterior wall thickness (PWT) were measured from a parasternal long-axis view according to the recommendations of the American Society of Echocardiography.26 Left ventricular mass, calculated with the Devereaux formula,27 and left atrial antero-posterior diameter, were indexed by body surface area (BSA) to account for the effect of body size. Left ventricular relative wall thickness, an index of LV geometry, was calculated with the following formula: 2×PWT/LVDD.28 LV ejection fraction was calculated using the biplane modified Simpson's rule.

Trans-mitral diastolic flow was obtained from an apical 4-chamber view. Color Doppler was used to visualize the trans-mitral flow; the pulsed Doppler sample volume was placed perpendicular to the inflow jet, at the level of mitral valve leaflet tips. Doppler baseline and velocity scale were adjusted to obtain optimal visualization of the inflow spectrum. At least four cardiac cycles were recorded during patient apnea and the images were stored in digital format for off-line analysis.

LV myocardial velocities were evaluated by tissue Doppler imaging (TDI) and sampled on the longitudinal axis from the apical 4-chamber view. Two-dimensionally guided pulsed TDI sample volume was placed at the level of the lateral and septal mitral valve annulus; Doppler gain and wall filter were adjusted to reduce artifacts and the velocity scale was set to ±20 cm/s. Four consecutive beats were recorded during patient apnea and stored in digital format for off-line analysis.

Peak velocities of the early (E wave) and late (A wave) phase of the mitral inflow pattern from Doppler recordings were measured and their ratio (E/A) was calculated; peak early diastolic velocity of the lateral (E' lat) and the septal (E'sep) mitral annulus by pulsed-TDI were measured. The ratio between the E and the E' waves (E/E'sep, E/E' lat) was calculated as an index of LV filling pressures.

Statistical Analysis

Data are presented as mean ± standard deviation for continuous variables and as proportions for categorical variables. Differences between race-ethnic groups were assessed by one-way analysis of variance (ANOVA) for continuous variables and by chi-square test for proportions. The Kruskal-Wallis non-parametric test was performed when the dependent variable was not normally distributed. Analysis of covariance (ANCOVA) was performed separately for each diastolic function parameter to assess differences between race-ethnic groups after adjustment for covariates. The effect of demographic and clinical variables on diastolic parameters was tested in univariate analysis, and the threshold for entry in the multivariate models was set at a p-value <0.1. Differences among race-ethnic groups were assessed in progressive multivariate models. Estimated marginal means adjusted for covariates and 95% confidence intervals (CI) were derived.

For all statistical analyses, a 2-tailed $p<0.05$ was considered significant. Statistical analyses were performed using SAS software version 9.1 (SAS Institute Inc., Cary, NC).

Results

Population characteristics

The characteristics of the study cohort (N=760) are shown in Table I. Mean age was 70.8±9.6 years, and 479 (63.0%) were women. Pair-wise comparisons among the race-ethnic groups are also shown in Table I. Hispanics were significantly younger than blacks and whites (both $p<0.0001$), and whites had significantly lower body mass index than Hispanics and blacks ($p<0.05$ and $p<0.0001$). Among cardiovascular risk factors, hypertension was significantly more prevalent in blacks than in whites (76.9% vs. 62.5%, $p<0.05$), and the prevalence of diabetes was greater in Hispanics (34.5%) and blacks (25.6%) than in whites (7.7%, both $p<0.001$). Frequency of high school or higher level of education was significantly lower in Hispanics and blacks compared to whites (all $p<0.0001$). LV ejection fraction, LV mass, and relative wall thickness were not significantly different between the three groups. Differences in heart rate were found, with Hispanics showing higher heart rate than whites ($p<0.01$).

Relationship between cardiovascular risk factors and diastolic function parameters

The association between cardiovascular risk factors and parameters of diastolic function is shown in Table II. Age, gender, diabetes, body mass index, hypertension, hypercholesterolemia, relative wall thickness, LV ejection fraction and heart rate were significantly associated with at least one mitral flow parameter of diastolic function (E, E/A). All the risk factors, except for CAD, were significantly associated with at least one TDI-based parameter of diastolic function.

Diastolic function parameters and race-ethnicity

A first multivariate model was constructed to test differences in diastolic function parameters among the three race-ethnic groups after adjustment for age and sex. In this model, significant differences between race-ethnic groups were observed in the E/A ratio (overall $p<0.05$), lateral and septal E' velocity (overall $p<0.05$ and <0.01), and septal E/E' ratio (overall $p<0.05$). Trans-mitral E wave did not show significant differences between the groups ($p=0.89$). Results of pair-wise comparisons between race-ethnic groups are shown in Table III, model 1. The E/A ratio was significantly lower in Hispanics compared with whites ($p=0.01$). Blacks and Hispanics showed significantly lower E' velocities than whites ($p=0.04$ and $p=0.001$ for E' sep; $p=0.06$ and $p=0.007$ for E' lat). E/E' ratio was significantly higher in Hispanics than whites ($p=0.03$ for E/E' lat; $p=0.006$ for E/E' sep), and it showed a trend to be higher in blacks than whites ($p=0.09$).

In a multivariate model adjusted for demographics, cardiovascular risk factors, and anti-hypertensive treatment, none of the diastolic function parameters (E wave, E/A ratio, E' lat, E' sep, E/E' lat and E/E' sep) showed significant differences between race-ethnic groups (overall p values: 0.62, 0.32, 0.72, 0.57, 0.54 and 0.26 respectively; Table III, model 2). The further inclusion into the model of echocardiographic variables of LV structure and function did not affect the results (Table III, model 3).

Discussion

The present study is the first to investigate differences in diastolic function among race-ethnic groups in an unselected community-based cohort. We showed that, after adjusting for age and sex, blacks and Hispanics had worse diastolic function than whites. Hispanics showed lower trans-mitral E/A ratio, lower E' velocity and higher E/E' ratio compared to whites, whereas blacks showed significantly lower E' septal velocity, a trend towards higher E/E' ratio, but no differences in the E/A ratio compared with whites. In the multivariate analysis, adjusting for major cardiovascular risk factors and other potential confounders, we no longer found

significant differences in diastolic function parameters among the three race-ethnic groups, suggesting that most of the differences observed were due to the significant imbalance in risk factors, rather than to intrinsic race-ethnic heterogeneity.

These findings must be interpreted in the pathophysiologic paradigm of LV diastolic dysfunction. With the impairment of LV relaxation, the early component of the mitral inflow shows a reduction in its peak velocity causing a reduction in the E/A ratio. As the diastolic dysfunction progresses, the increase in left atrial pressure causes an increase of the E/A ratio (pseudo-normalization of the mitral inflow; stage II of diastolic dysfunction).22,23 The mitral annulus early velocity (E') and the E/E' ratio are less pre-load dependent than the trans-mitral flow, allowing the distinction between a normal and a pseudo-normal mitral flow.29,30 In particular, the differences found in the E/E' ratio, especially in Hispanics and despite their younger age, suggest a higher LV stiffness in this group compared with whites.

Diastolic dysfunction is a common finding in hypertensive31 and diabetic patients.32 Both these conditions are associated to LV hypertrophy and obesity, which in turn are strong determinants of LV diastolic function.[33] In our study cohort, hypertension was more prevalent in blacks and Hispanics, and diabetes was far more prevalent in Hispanics (35.3%) than in blacks (22.9%) and whites (7.0%), differences that could reasonably explain the greater impairment in diastolic function observed in Hispanics and blacks. However, we found no differences in LV mass and geometry (measured as the relative wall thickness) between the three groups, implying that the observed differences in diastolic function were possibly mediated by factors other than the simple increase in LV mass. In fact, diabetes is associated with changes in myocardial metabolism and structure that contribute to diastolic abnormalities. In diabetic hearts, glucose metabolism is reduced in favor of energy production from beta-oxidation of free fatty acids[34,35] leading to the accumulation of toxic intermediates that affect calcium handling, promote apoptosis, and affect myocardial mechanics.36 Hyperglycemia is also associated with alterations in the renin-angiotensin system, which can cause oxidative damage, cell apoptosis and increased interstitial fibrosis.37 In addition, an increased resting tension of the cardiomyocytes as been described as a prominent cause of increased LV stiffness in diabetes.[38]

Our findings do not confirm those from the only other study that investigated racial differences in LV diastolic function. In that report on hypertensive participants from the ASCOT (Anglo-Scandinavian Cardiac Outcomes Trial) study,[24] authors concluded that African-Caribbeans had worse diastolic function than white Europeans even after adjusting for confounding covariates, hypothesizing that unknown race-related factors may have played a role in determining differences in diastolic function. That study, however, was conducted in hypertensive patients who had been treated for one year with one of two combinations of two anti-hypertensive drugs. Therefore, the study population may have had more advanced hypertensive heart disease, and the results cannot therefore be extrapolated to the general population. Our study was conducted in an unselected sample of the population living in Northern Manhattan, that might be at a stage in which the early prediction of an increased risk of future heart failure and cardiovascular events is more relevant.

In a population context and from a preventive standpoint, our study provides valuable insight into the subject of race/ethnic disparities in cardiovascular diseases. Diastolic dysfunction is associated with worse cardiovascular outcome and with increased risk of incident heart failure, 17,18,[39] and may represent an early asymptomatic marker of target organ damage in the progression to overt heart failure. The greater impairment of diastolic function that we observed in blacks and Hispanics might be an early occurrence in a chain of events leading to the greater cardiovascular burden that characterizes these race-ethnic groups. Recent data from the VALIDD (Valsartan in Diastolic Dysfunction) study showed that, in hypertensive patients,

blood pressure control improved diastolic function, regardless of the type of anti-hypertensive medication used,[40] underscoring the role of modifiable risk factors in the pathogenesis of diastolic dysfunction. Our study supports the concept that an earlier and better control of traditional risk factors in high risk populations could possibly narrow the gap in cardiovascular burden between races, by preventing or delaying the transition from asymptomatic abnormalities to overt disease.

This study has some limitations. Our Doppler flow analysis was limited to the peak flow velocities; however, the use of tissue Doppler parameters helped to detect abnormalities in LV diastole even when pseudo-normalized flow pattern was present. The proportion of Hispanics in our study group was very high, and this may affect the ability to detect significant differences with the other smaller race-ethnic groups. However, this race-ethnic distribution is representative of the community living in Northern Manhattan,[41] and this offered us the opportunity of studying different race-ethnic groups living in the same urban environment. Additionally, the study population was primarily elderly; therefore, our results should not be directly applied to younger populations with lower cardiovascular risk profiles. Despite study participants were selected using a generator of random telephone numbers, the voluntary individual participation may have resulted in a self-selection of the study participants, a circumstance that is common to studies of this type. Finally, due to the cross-sectional design of our study, we cannot draw any conclusion about the prognostic value of our findings, and therefore our study must be seen as hypothesis-generating.

In conclusion, our study shows that, in a community-based tri-ethnic cohort, black and Hispanic subjects have worse diastolic function parameters compared with whites, an observation that may portend the well-documented disparities in the future development of heart failure and in cardiac morbidity and mortality. However, disparities in modifiable cardiovascular risk factors and socio-demographic variables, rather than intrinsic race-ethnic differences, seem to account for most of the difference that we observed, suggesting a role for risk factor control in reducing race-ethnic disparities in diastolic function and their sequelae.

Acknowledgments

The authors wish to thank Janet De Rosa, MPH, for the coordination of the study activities; Rui Liu, M.D. and Michele Alegre, R.D.C.S., for the performance and preliminary interpretation of the echocardiographic studies; Rafi Cabral, M.D. and Palma Gervasi-Franklin for their help in the collection and management of the data.

Funding: The study was supported by grants from the National Institute of Neurological Disorders and Stroke (NINDS) R01 NS36286 (P.I.: Dr. Marco R. Di Tullio) and NS29993 (P.I.'s: Drs. Ralph L. Sacco/Mitchell S. V. Elkind).

This study was supported by grants R01 NS36286 (P.I.: Dr. Marco R. Di Tullio) and NS29993 (P.I.'s: Drs. Ralph L. Sacco/Mitchell S. V. Elkind) from the National Institute of Neurological Disorders and Stroke.

The authors are solely responsible for the design and conduct of this study, all study analyses, the drafting and editing of the paper and its final contents.

References

1. Mensah GA, Mokdad AH, Ford ES, et al. State of disparities in cardiovascular health in the United States. Circulation 2005;111:1233–41. [PubMed: 15769763]
2. Thomas AJ, Eberly LE, Davey SG, et al. Race/ethnicity, income, major risk factors, and cardiovascular disease mortality. Am J Public Health 2005;95:1417–23. [PubMed: 16006418]
3. Hunt KJ, Resendez RG, Williams K, et al. All-cause and cardiovascular mortality among Mexican-American and non-Hispanic White older participants in the San Antonio Heart Study- evidence against the "Hispanic paradox". Am J Epidemiol 2003;158:1048–57. [PubMed: 14630600]
4. Carnethon MR, Lynch EB, Dyer AR, et al. Comparison of risk factors for cardiovascular mortality in black and white adults. Arch Intern Med 2006;166:1196–202. [PubMed: 16772247]

5. Gillum RF, Mussolino ME, Madans JH. Coronary heart disease risk factors and attributable risks in African-American women and men: NHANES I epidemiologic follow-up study. Am J Public Health 1998;88:913–7. [PubMed: 9618619]
6. Chambless LE, Folsom AR, Sharrett AR, et al. Coronary heart disease risk prediction in the Atherosclerosis Risk in Communities (ARIC) study. J Clin Epidemiol 2003;56:880–90. [PubMed: 14505774]
7. Liao Y, McGee DL, Cooper RS. Prediction of coronary heart disease mortality in blacks and whites: pooled data from two national cohorts. Am J Cardiol 1999;84:31–6. [PubMed: 10404847]
8. Hozawa A, Folsom AR, Sharrett AR, et al. Absolute and attributable risks of cardiovascular disease incidence in relation to optimal and borderline risk factors: comparison of African American with white subjects--Atherosclerosis Risk in Communities Study. Arch Intern Med 2007;167:573–9. [PubMed: 17389288]
9. Alexander M, Grumbach K, Selby J, et al. Hospitalization for congestive heart failure. Explaining racial differences. JAMA 1995;274:1037–42. [PubMed: 7563454]
10. Rosamond W, Flegal K, Furie K, et al. Heart disease and stroke statistics--2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2008;117:e25–146. [PubMed: 18086926]
11. Brown DW, Haldeman GA, Croft JB, et al. Racial or ethnic differences in hospitalization for heart failure among elderly adults: Medicare, 1990 to 2000. Am Heart J 2005;150:448–54. [PubMed: 16169322]
12. Bahrami H, Kronmal R, Bluemke DA, et al. Differences in the incidence of congestive heart failure by ethnicity: the multi-ethnic study of atherosclerosis. Arch Intern Med 2008;168:2138–45. [PubMed: 18955644]
13. Loehr LR, Rosamond WD, Chang PP, et al. Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). Am J Cardiol 2008;101:1016–22. [PubMed: 18359324]
14. Bibbins-Domingo K, Pletcher MJ, Lin F, et al. Racial differences in incident heart failure among young adults. N Engl J Med 2009;360:1179–90. [PubMed: 19297571]
15. Zile MR, Nappi J. Diastolic Heart Failure. Curr Treat Options Cardiovasc Med 2000;2:439–50. [PubMed: 11096548]
16. Vasan RS, Larson MG, Benjamin EJ, et al. Congestive heart failure in subjects with normal versus reduced left ventricular ejection fraction: prevalence and mortality in a population- based cohort. J Am Coll Cardiol 1999;33:1948–55. [PubMed: 10362198]
17. Aurigemma GP, Gottdiener JS, Shemanski L, et al. Predictive value of systolic and diastolic function for incident congestive heart failure in the elderly: the Cardiovascular Health Study. J Am Coll Cardiol 2001;37:1042–8. [PubMed: 11263606]
18. Redfield MM, Jacobsen SJ, Burnett JC Jr. et al. Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. JAMA 2003;289:194–202. [PubMed: 12517230]
19. Bella JN, Palmieri V, Roman MJ, et al. Mitral ratio of peak early to late diastolic filling velocity as a predictor of mortality in middle-aged and elderly adults: the Strong Heart Study. Circulation 2002;105:1928–33. [PubMed: 11997279]
20. Fox ER, Han H, Taylor HA, et al. The prognostic value of the mitral diastolic filling velocity ratio for all-cause mortality and cardiovascular morbidity in African Americans: the Atherosclerotic Risks in Communities (ARIC) study. Am Heart J 2006;152:749–55. [PubMed: 16996852]
21. Schillaci G, Pasqualini M, Verdecchia P, et al. Prognostic significance of left ventricular diastolic dysfunction in essential hypertension. J Am Coll Cardiol 2002;39:2005–11. [PubMed: 12084601]
22. Nishimura RA, Tajik AJ. Evaluation of diastolic filling of left ventricle in health and disease: Doppler echocardiography is the clinician's Rosetta Stone. J Am Coll Cardiol 1997;30:8–18. [PubMed: 9207615]
23. Ommen SR, Nishimura RA. A clinical approach to the assessment of left ventricular diastolic function by Doppler echocardiography: update 2003. Heart 2003;89(Suppl 3):iii18–iii23. [PubMed: 14594871]

24. Sharp A, Tapp R, Francis DP, et al. Ethnicity and left ventricular diastolic function in hypertension an ASCOT (Anglo-Scandinavian Cardiac Outcomes Trial) substudy. J Am Coll Cardiol 2008;52:1015–21. [PubMed: 18786484]
25. Sacco RL, Roberts JK, Boden-Albala B, et al. Race-ethnicity and determinants of carotid atherosclerosis in a multiethnic population. The Northern Manhattan Stroke Study. Stroke 1997;28:929–35. [PubMed: 9158627]
26. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63. [PubMed: 16376782]
27. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450–8. [PubMed: 2936235]
28. Ganau A, Devereux RB, Roman MJ, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol 1992;19:1550–8. [PubMed: 1534335]
29. Nagueh SF, Middleton KJ, Kopelen HA, et al. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol 1997;30:1527–33. [PubMed: 9362412]
30. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation 2000;102:1788–94. [PubMed: 11023933]
31. Zanchetti A, Cuspidi C, Comarella L, et al. Left ventricular diastolic dysfunction in elderly hypertensives: results of the APROS-diadys study. J Hypertens 2007;25:2158–67. [PubMed: 17885561]
32. Boyer JK, Thanigaraj S, Schechtman KB, et al. Prevalence of ventricular diastolic dysfunction in asymptomatic, normotensive patients with diabetes mellitus. Am J Cardiol 2004;93:870–5. [PubMed: 15050491]
33. Mureddu GF, de SG, Greco R, et al. Left ventricular filling in arterial hypertension. Influence of obesity and hemodynamic and structural confounders. Hypertension 1997;29:544–50. [PubMed: 9040436]
34. Rodrigues B, Cam MC, McNeill JH. Metabolic disturbances in diabetic cardiomyopathy. Mol Cell Biochem 1998;180:53–7. [PubMed: 9546630]
35. Diamant M, Lamb HJ, Groeneveld Y, et al. Diastolic dysfunction is associated with altered myocardial metabolism in asymptomatic normotensive patients with well-controlled type 2 diabetes mellitus. J Am Coll Cardiol 2003;42:328–35. [PubMed: 12875772]
36. Young ME, Guthrie PH, Razeghi P, et al. Impaired long-chain fatty acid oxidation and contractile dysfunction in the obese Zucker rat heart. Diabetes 2002;51:2587–95. [PubMed: 12145175]
37. Fiordaliso F, Li B, Latini R, et al. Myocyte death in streptozotocin-induced diabetes in rats in angiotensin II- dependent. Lab Invest 2000;80:513–27. [PubMed: 10780668]
38. van Heerebeek L, Hamdani N, Handoko ML, et al. Diastolic stiffness of the failing diabetic heart: importance of fibrosis, advanced glycation end products, and myocyte resting tension. Circulation 2008;117:43–51. [PubMed: 18071071]
39. Sharp AS, Tapp RJ, Thom SA, et al. Tissue Doppler E/E' ratio is a powerful predictor of primary cardiac events in a hypertensive population: an ASCOT substudy. Eur Heart J. 2009
40. Solomon SD, Janardhanan R, Verma A, et al. Effect of angiotensin receptor blockade and antihypertensive drugs on diastolic function in patients with hypertension and diastolic dysfunction: a randomised trial. Lancet 2007;369:2079–87. [PubMed: 17586303]
41. U.S. Census Bureau. American Factfinder. 2000

Table I

Characteristics of the study cohort by race-ethnicity.

	Overall N=760	Black N=117	Hispanic N=539	White N=104	P B vs. W	P H vs. W	P B vs. H
Age, years	70.8±9.6	74.2±10.2	69.3±8.9	74.3±10.3	0.93	<0.0001	<0.0001
Women, n (%)	479 (63.0)	80 (68.4)	340 (63.1)	59 (56.3)	0.07	0.22	0.28
Body mass index, kg/m^2	28.2±4.8	27.8±4.7	28.6±4.7	26.5±5.0	0.04	<0.0001	0.11
Hypertension, n (%)	543 (71.4)	90 (76.9)	388 (72.0)	65 (62.5)	0.02	0.05	0.28
Diabetes, n (%)	224 (29.5)	30 (25.6)	186 (34.5)	8 (7.7)	0.0004	<0.0001	0.06
Hypercholesterolemia, n (%)	462 (60.9)	62 (53.5)	334 (62.0)	66 (63.5)	0.13	0.77	0.09
Coronary artery disease, n (%)	45 (5.9)	8 (6.8)	31 (5.8)	6 (5.8)	0.74	0.99	0.65
Anti-hypertensive treatment, n (%)	128 (16.8)	29 (24.8)	84 (15.6)	15 (14.4)	0.05	0.76	0.02
Education, high school or higher, n (%)	218 (28.7)	58 (49.6)	80 (14.9)	80 (76.9)	<0.0001	<0.0001	<0.0001
Echocardiographic variables							
LV ejection fraction, %	63.3±7.2	62.6±8.4	63.5±7.0	63.5±6.4	0.39	0.99	0.26
LVMI, g/m^2	103.8±26.2	103.4±29.3	104.3±25.4	101.4±26.8	0.61	0.29	0.74
Relative wall thickness	0.50±0.09	0.51±0.10	0.50±0.08	0.50±0.10	0.29	0.81	0.09
Left atrial diameter/BSA, mm/m2	22.2±3.1	21.7±3.3	22.4±3.0	22.1±3.2	0.36	0.38	0.03
Heart rate, bpm	69.9±11.3	68.4±11.8	70.8±11.3	67.4±10.5	0.48	0.004	0.05

LVMI: Left Ventricular Mass Index. BSA: Body surface area. B: Black. H: Hispanic. W: White. Anti-hypertensive treatment includes beta-blockers and/or ace-inhibitors and/or diuretics

Table II

Univariate association of demographics, cardiovascular risk factors and echocardiographic variables with diastolic function parameters.

	E, cm/s	E/A	E' lat, cm/s	E' sep, cm/s	E/E' lat	E/E' Sep
Age, per year	−0.12 (0.06)	−0.007‡ (0.001)	−0.11‡ (0.008)	−0.07‡ (0.006)	0.11‡ (0.01)	0.12‡ (0.01)
Male sex	−4.95‡ (1.27)	0.01 (0.02)	0.09 (0.17)	0.11 (0.12)	−0.90‡ (0.25)	−1.16‡ (0.28)
Diabetes	5.36‡ (1.35)	−0.02 (0.02)	−0.50† (0.18)	−0.34† (0.13)	1.38‡ (0.26)	1.68‡ (0.30)
Body mass index, per unit	0.52‡ (0.13)	−0.003 (0.002)	−0.04* (0.02)	−0.01 (0.01)	0.11‡ (0.02)	0.10‡ (0.03)
Hypertension	0.42 (1.37)	−0.06* (0.02)	−1.29‡ (0.18)	−1.00‡ (0.12)	1.53‡ (0.26)	1.92‡ (0.30)
Hypercholesterolemia	1.12 (1.27)	−0.05* (0.02)	−0.25 (0.17)	−0.20 (0.12)	0.38 (0.25)	0.60* (0.28)
Coronary artery disease	0.33 (2.63)	−0.06 (0.05)	−0.42 (0.36)	−0.41 (0.24)	0.31 (0.51)	0.81 (0.59)
LVMI, per g/m^2	−0.03 (0.02)	−0.001 (0.000)	−0.02‡ (0.003)	−0.02‡ (0.002)	0.02‡ (0.004)	0.04‡ (0.005)
Relative wall thickness, per unit	−21.0† (7.12)	−0.67‡ (0.12)	−6.33‡ (0.95)	−5.19‡ (0.63)	4.73‡ (1.37)	7.12‡ (1.58)
LV Ejection Fraction, per unit %	0.33‡ (0.09)	0.001 (0.002)	0.01 (0.01)	0.02† (0.008)	0.01 (0.02)	0.003 (0.02)
Heart rate, per bpm	−0.23‡ (0.05)	−0.006‡ (0.001)	−0.01 (0.07)	0.001 (0.005)	−0.02 (0.01)	−0.04† (0.01)

Numbers in table are parameter estimates (standard errors in parentheses). LVMI: Left ventricular mass index.

*p<0.05;

†p<0.01;

‡p<0.001

Table III

Adjusted mean values and 95% confidence intervals of diastolic function parameters by race-ethnicity

	Race-Ethnicity			P values		
Model 1	**Black**	**Hispanic**	**White**	**B vs. W**	**H vs. W**	**B vs. H**
E, cm/s	69.5 (66.4-72.6)	70.2 (68.8-71.7)	70.4 (67.1-73.7)	0.68	0.92	0.67
E/A	0.87 (0.81-0.92)	0.81 (0.78-0.83)	0.89 (0.83-0.94)	0.57	0.01	0.05
E' lat, cm/s	8.17 (7.79-8.55)	8.08 (7.90-8.26)	8.69 (8.28-9.09)	0.06	0.007	0.69
E' sep, cm/s	6.22 (5.95-6.48)	6.09 (5.97-6.21)	6.61 (6.33-6.89)	0.04	0.001	0.40
E/E' lat	9.12 (8.55-9.69)	9.32 (9.05-9.58)	8.60 (8.00-9.21)	0.22	0.03	0.54
E/E' sep	11.85 (11.19-12.51)	12.12 (11.82-12.43)	11.03 (10.33-11.74)	0.09	0.006	0.47
Model 2	**Black**	**Hispanic**	**White**	**B vs. W**	**H vs. W**	**B vs. H**
E, cm/s	68.9 (65.7-72.0)	70.6 (69.1-72.1)	69.5 (65.9-73.2)	0.78	0.60	0.34
E/A	0.85 (0.80-0.91)	0.82 (0.79-0.84)	0.86 (0.80-0.93)	0.82	0.21	0.24
E' lat, cm/s	8.12 (7.74-8.51)	8.17 (7.99-8.35)	8.34 (7.90-8.79)	0.43	0.50	0.83
E' sep, cm/s	6.18 (5.91-6.44)	6.16 (6.03-6.29)	6.34 (6.03-6.65)	0.39	0.31	0.92
E/E' lat	9.07 (8.50-9.64)	9.27 (9.00-9.54)	8.86 (8.21-9.51)	0.61	0.28	0.55
E/E' sep	11.81 (11.14-12.47)	12.06 (11.75-12.38)	11.33 (10.56-12.10)	0.32	0.10	0.51
Model 3	**Black**	**Hispanic**	**White**	**B vs. W**	**H vs. W**	**B vs. H**
E, cm/s	69.0 (66.0-72.1)	70.6 (69.2-72.1)	69.1 (65.6-72.7)	0.97	0.47	0.38
E/A	0.85 (0.80-0.91)	0.82 (0.79-0.84)	0.86 (0.79-0.92)	0.94	0.26	0.23
E' lat, cm/s	8.12 (7.73-8.50)	8.18 (8.00-8.36)	8.30 (7.86-8.74)	0.50	0.63	0.77
E' sep, cm/s	6.18 (5.93-6.43)	6.17 (6.05-6.29)	6.30 (6.00-6.59)	0.51	0.44	0.95
E/E' lat	9.10 (8.54-9.66)	9.25 (8.99-9.52)	8.87 (8.22-9.52)	0.58	0.31	0.64
E/E' sep	11.83 (11.19-12.47)	12.04 (11.74-12.34)	11.36 (10.63-12.10)	0.32	0.11	0.57

B: Blacks. H: Hispanics. W: Whites. Covariates for model 1: age and sex. Model 2: model 1 + body mass index, hypertension, diabetes, hypercholesterolemia, coronary artery disease, education (high school or higher), antihypertensive treatment. Model 3: model 2 + LV mass index, relative wall thickness, LV ejection fraction, heart rate.