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RACE/ETHNIC AND SEX DIFFERENCES IN LARGE AND SMALL ARTERY ELASTICITY – RESULTS OF THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS (MESA)

Objective: Reduction in arterial elasticity marks progression toward cardiovascular morbidity and mortality. Variability in arterial elasticity may help account for race/ethnic and gender differences in cardiovascular risk.

Design: Cross-sectional study.

Setting: Whites, African Americans, Hispanics and Chinese aged 45–84 years free of clinically recognized cardiovascular disease were recruited in six US communities.

Participants: We examined 3,316 women and 3,020 men according to race/ethnicity and sex.

Main Outcome Measures: Large (LAE) and small artery (SAE) elasticity, derived from radial artery diastolic pulse wave contour registration in all subjects in a supine position using tonometry. LAE and SAE were adjusted for ethnicity, age, clinical site, height, heart rate, blood pressure, antihypertensive medication and body mass index, diabetes, smoking, and circulating lipids.

Results: Much of the sex difference in arterial elasticity was explained by height. After adjustment, LAE did not differ by race/ethnicity, but mean SAE in African Americans was 4.2 mL/mm Hg \times 100 and 4.4 mL/mm Hg \times 100 in Hispanics compared to means of 4.6 mL/mm Hg \times 100 in Whites, and 4.8 mL/mm Hg \times 100 in Chinese.

Conclusions: Reduced SAE may indicate earlier vascular disease in African Americans and Hispanics than other groups. (*Ethn Dis.* 2009;19:243–250)

Key Words: Blood Pressure, Arterial Elasticity, MESA Study

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INTRODUCTION

A diagnostic challenge is to detect abnormal structure and function in the vascular system before the development of symptoms or signs of cardiovascular disease (CVD).^{1,2} Variability in arterial elasticity may help account for race/ethnic and sex differences in cardiovascular risk; and knowledge of arterial elasticity might improve risk stratification, and identify individuals with early vascular damage who are predisposed to future vascular events, by providing a direct assessment of abnormal structure or tone in the arterial vasculature.^{3,4} Arterial elasticity is related to, but conceptually broader than, systemic blood pressure. However, the physiologic link between vascular elasticity and arterial pressure makes it difficult to separate the adverse effects of pressure from those of the vascular functional and structural alterations that characterize loss of elasticity. Since endothelial dysfunction and nitric oxide deficiency are characteristic features of hypertension and of other risk factors for morbid events, it has been proposed that blood pressure elevation may be viewed, in part, as a complication of functional and structural changes in the microcirculation, and that structural changes in

the large arteries leading to morbid events may be viewed as a complication of both pressure elevation and endothelial dysfunction.⁵

Current evidence indicates that arterial stiffness is a predictor for CVD events in the general population,^{6–9} in patients with hypertension,¹⁰ end-stage renal disease,¹¹ impaired glucose intolerance,¹² and coronary artery disease.¹³ Several noninvasive techniques are available to assess arterial elasticity based on analysis of the arterial pulse wave regarding either the systolic or the diastolic part.¹⁴ The CR-2000 (HDI, Inc., Eagan, MN) provides information about the pools of large and small arteries derived from the radial artery pulse waveform using a mathematical model based on a modified Windkessel model.¹⁵ The Multi-Ethnic Study of Atherosclerosis (MESA), an epidemiologic cohort study that aimed to study a variety of subclinical CVD measures, elected to include the CR-2000 device to obtain information about arterial elasticity. The Windkessel model leads to two measures, namely large artery elasticity (LAE), pertaining to the pool of large arteries, and small artery elasticity (SAE), pertaining to the pool of small arteries, and was validated in an animal model.¹⁶ Besides the finding that SAE was inversely associated with incident CVD,⁹ randomized trials have shown that valsartan^{17,18} as well as a combination of amlodipine and atorvastatin increased both LAE and SAE.¹⁹ Aging,²⁰ smoking,²¹ diabetes,²² and

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