Arterial stiffness is associated with left ventricular systolic and diastolic function in never-treated hypertensives
Dimitrios Terentes-Printzios, Charalampos Vlachopoulos, Gregory Vyssoulis, Panagiota Pietri, Panagiotis Xaplanteris, Nikolaos Ioakeimidis, Nikolaos Alexopoulos, Aikaterini Siama, Alexios Samentzas, Christodoulos Stefanadis
Peripheral Vessels Unit, 1st Department of Cardiology, Athens Medical School, Hippokration Hospital, Athens, Greece

Background
Hypertension is associated with increased arterial stiffness. Arterial stiffness, which is a predictor of cardiovascular risk, has been shown to correlate with left ventricular function in middle-aged and elderly patients. We assessed the hypothesis that arterial stiffness is associated with left ventricular systolic and diastolic function in never-treated hypertensive patients.

Methods
Study population and design
We enrolled 200 consecutive essential hypertensives (Table 1) with preserved left ventricular ejection fraction (LVEF>50%). Arterial stiffness was determined with carotid-femoral pulse wave velocity (PWV). In addition, echocardiography was performed and venous blood samples were drawn.

Evaluation of arterial stiffness
Carotid-femoral PWV, an established index of arterial stiffness, was calculated from measurements of pulse transit time and the distance traveled between 2 recording sites (PWV = distance in meters divided by transit time in seconds) with a validated noninvasive device (Complior).

Evaluation of systolic and diastolic function
Detailed two-dimensional and Doppler echocardiograms were obtained in all the patients by a single experienced observer, using a Philips Sonos 5500 machine (Philips, Eindhoven, Netherlands). (Table 2) The parasternal long-axis view that maximized LV cavity dimension was used to make wall thickness and chamber diameter measurements perpendicular to the walls. Transmural early (E) and late (A) diastolic velocities were recorded in the apical four-chamber view (no extensive wall motion abnormalities were present in our patients). Left ventricular mass index (LVMI) was calculated with the formula LVMI=LVM/Body surface area (BSA), using the Devereux method (LVM=1.04 [(LVEDD+PWT+SWT)−LVEDD])x0.8+0.6, where LVEDD: left ventricular end-diastolic diameter; PWT: posterior wall thickness; SWT: septal wall thickness; 1.04: specific gravity of the myocardium; 0.8: correction factor). LVEF was calculated using the Teichholz method as follows: Ejection fraction=(EDVI−ESVI)/ESVI x100, where EDVI=[(7 x EDD)]/[2.4 + EDD] / BSA and ESVI=[(7 x ESD)]/[2.4 + ESD] / BSA. (EDVI:End-diastolic volume index, ESVI:End-systolic volume index, EDD:End-diastolic diameter, ESD:End-systolic diameter)

Results
PWV was inversely correlated with LVEF (R=-0.34, p<0.001) and the ratio of peak early (E) to peak atrial (A) Doppler mitral valve flow velocity (E/A) (R=-0.31, p<0.001). PWV showed a positive correlation with LVMI (R=0.32, p<0.001), left atrial diameter (R=0.39, p<0.001) and A (R=0.27, p<0.001). (Figure) In multivariable regression analysis, all the aforementioned associations were independent of age, sex and diastolic blood pressure (p=0.028, adjusted R² of the model=0.21; p=0.034, adjusted R² of the model=0.21; p=0.003, adjusted R² of the model=0.23; p=0.015, adjusted R² of the model=0.20; p=0.044, adjusted R² of the model=0.21, respectively).

Conclusions
In never-treated hypertensives arterial stiffness is consistently and independently associated with impaired systolic and diastolic function. This finding provides further insights into the role of arterial stiffness in left ventricular function.