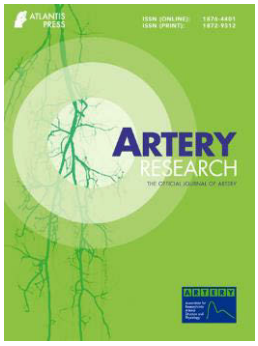

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1. ACUTE INFLAMMATION INCREASES ARTERIAL STIFFNESS, CENTRAL SYSTOLIC BLOOD PRESSURE, AND ENDOTHELIAL DYSFUNCTION IN HEALTHY SUBJECTS

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1.
ACUTE INFLAMMATION INCREASES ARTERIAL STIFFNESS, CENTRAL SYSTOLIC BLOOD PRESSURE, AND ENDOTHELIAL DYSFUNCTION IN HEALTHY SUBJECTS

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Background: Acute systemic inflammation or infection transiently increases risk of cardiovascular events, but the underlying mechanisms are not fully understood. Although observational studies suggest that inflammation is positively associated with arterial stiffness and blood pressure, the causal relationship of this is not clear. Because increased inflammation could be related to vascular dysfunction, we tested the hypothesis that an acute inflammation causes an increase in arterial stiffness and blood pressure.
Methods: Using a randomized double blind sham placebo-controlled cross over design, 19 healthy subjects (male 10, female 9; age 24±4 yrs) were injected with an influenza vaccine (0.5 m/L) as a model to generate systemic inflammation, and a sham vaccine (normal saline). C-reactive protein (CRP), interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-a) were measured as a markers of inflammation. Brachial artery flow mediated vasodilatation (FMD) was measured using B-mode ultrasound. Aortic augmentation index (AIx), carotid-radial pulse wave velocity (PWV), and central blood pressure were measured using applanation tonometry. These variables measured at baseline before each vaccination, 24 hours and 48 hours after each vaccination.
Results: Compared with sham placebo, the influenza vaccination caused a significant increase in CRP (1.42±0.6 at baseline, 2.81±1.0 after 24 hours, 5.0±1.3 mg/L after 48 hours, $p<0.05$) and IL-6 (1.12±0.3, 2.56±0.4, 2.26±0.6 pg/mL, $p<0.05$). Central systolic blood pressure (98.0±7.4, 104.5±10.8, 100.7±8.4 mmHg, $p<0.05$) and PWV (7.8±0.9, 8.6±1.5, 8.7±1.3 m/s, $p<0.05$) were significantly increased after an influenza vaccination but not sham vaccination. FMD was significantly decreased after acute inflammation (6.91±2.5, 3.22±2.8, 3.30±2.5%, $p<0.05$).
Conclusion: These findings show that acute inflammation caused a temporary increase in arterial stiffness and central blood pressure, and acutely reduced endothelial function. This offers insight into the noted increased risk of cardiovascular events associated with acute inflammation.

2.
DECELERATION TIME OF THE LEFT VENTRICULAR OUTFLOW TRACT FLOW VELOCITY: A SURROGATE DOPPLER ECHOCARDIOGRAPHIC PARAMETER FOR CENTRAL PULSE PRESSURE

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Background: Central pulse pressure (PP) is an independent predictor of cardiovascular outcomes. However, a Doppler echocardiographic parameter reflecting central PP and arterial stiffness is not known yet. The aim of this study was to evaluate whether the left ventricular outflow tract (LVOT) flow deceleration is correlated with central PP and parameters of arterial stiffness.
Methods: In 175 subjects (65 men, mean age 57 years), transthoracic echocardiogram and radial artery tonometry were simultaneously performed. The patients who had left ventricular (LV) systolic dysfunction, significant arrhythmia, valvular disease, coronary artery disease and renal insufficiency were excluded. The blood flow velocities through the LVOT were recorded using conventional pulsed Doppler and deceleration time (DT) of LVOT flow was measured. Central hemodynamics including central PP, pressure augmentation and augmentation index were noninvasively measured using radial artery tonometry. PP amplification was defined as the ratio of peripheral to central PP.
Results: DT was significantly correlated with peripheral PP ($r=0.23$, $p=0.002$). However, it showed stronger positive correlation with central PP ($r=0.44$, $p<0.001$), pressure augmentation ($r=0.50$, $p<0.001$) and negative correlation with PP amplification ($r=-0.33$, $p<0.001$). Multiple linear regression analysis, controlled for variables such as age, gender, height, peripheral diastolic blood pressure, heart rate and LV ejection fraction, revealed an independent and significant strong correlation between LVOT DT and central PP ($\beta = 0.34$, $p=0.001$).
Conclusion: DT of the LVOT flow velocity is a surrogate Doppler echocardiographic parameter for central PP. Prolonged LVOT DT would be a useful parameter to detect reduced compliance of a central artery.

3.
ESTIMATION OF CORONARY FLOW RESERVE CAPACITY USING TRANSTHORACIC DOPPLER ECHOCARDIOGRAPHY AND THE COLD PRESSOR TEST IS USEFUL FOR DIAGNOSING VARIANT ANGINA

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Background: The cold pressor test (CPT) has been used to detect vasospastic angina, but its sensitivity for predicting vasospasm is low. The aim of this study was to determine if the estimation of coronary flow reserve capacity in the left anterior descending coronary artery using transthoracic Doppler echocardiography (TTE) and CPT can predict coronary artery spasm.
Methods: A provocation test of coronary artery spasm using acetylcholine (50 and 80 μg) was performed in 65 subjects (52±10 yrs, M:F=41:24) with chest pain and normal coronary angiograms. Before and during CPT, the peak (PDV) and mean diastolic flow velocity (MDV) of the distal left anterior descending coronary artery were estimated using high frequency TTE (Figure), and ECG, blood pressure, and symptoms were monitored every minute. CPT%PDV and CPT%MDV were defined as the % change of PDV and MDV during CPT, respectively. The subjects were divided into two groups??(Spasm+group, 31) and (Spasm-group, 34)]?? and compared.
Results: 1. The CPT%PDV was 52.75±24% in the Spasm-group and 4.99±23.62% in the Spasm + group ($p<0.001$). 2. The CPT%MDV was 50.22±27.83% in the Spasm-group and 6.83±23.81 in the Spasm+group ($p<0.001$). 3. The CPT%PDV<31.1% had a sensitivity of 93.5% and a specificity of 90% for predicting coronary artery spasm (95% CI:0.939-0.979, $p<0.001$). 4. The CPT%MDV<30.55% had a sensitivity of 90% and a specificity of 76.5% for predicting coronary artery spasm (95% CI:0.884-0.950, $p<0.001$).
Conclusion: Estimation of coronary flow reserve capacity using TTE and CPT is a promising noninvasive test for predicting variant angina.