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The acute effects of mental arithmetic, cold pressor and maximal voluntary contraction on arterial stiffness in young healthy subjects

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Abstract *Background:* Mental stress, dynamic exercise and cold pressor stimulation all increase blood pressure (BP); however, their effects on arterial stiffness are not well described.
Methods: Twenty-three young healthy subjects (14 female/9 males), aged 18—30 years (23 ± 3 years), underwent mental arithmetic stress (mental arithmetic test [MAT]), cold presser test (CPT) and dynamic exercise (30% of maximal voluntary contraction [30% MVC]). Blood pressure and indices of arterial stiffness, pulse wave velocity (PWV; m/sec) and augmentation index (AIx; %) were measured at baseline, during the intervention (MAT, CPT and 30% MVC), and at 5 min and 10 min after the end of the intervention on separate days within a 3-week period. All values are given as means and standard deviations; statistical analysis was carried out using JMP software (Version 7).
Results: During MAT, CPT and 30% MVC there were respective increases in heart rate (HR/min) 27%, 16% and 10% ($P < 0.001$); systolic BP 16%, 17% and 12% ($P < 0.01$); diastolic BP 15%, 23% and 15% ($P < 0.01$); AIx 13%, 29% and 30% ($P < 0.05$); and PWV 14%, 12% and 16% ($P < 0.01$). When the model was corrected for HR, systolic BP and diastolic BP the changes both in PWV and AIx remained significant ($P < 0.01$).

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Conclusions: MAT, CPT and 30% MVC each increase the indices of arterial stiffness independently of HR and baseline blood pressure levels.

Introduction

Aortic stiffness and wave reflections affect left ventricular function, coronary blood flow and the mechanical integrity of arteries.[1] Arterial stiffness and its hemodynamic consequences are now established as predictors of adverse cardiovascular outcome.[2] Pulse wave velocity and augmentation index are increasingly used in the clinical assessment of patients with hypertension and various cardiovascular (CV) risk factors.[3] Arterial elastic properties and wave reflections are altered in the presence of cardiovascular risk factors such as smoking,[3] diabetes mellitus,[5] hypertension,[6] obesity,[7] hypercholesterolemia,[8] and a family history of premature coronary artery disease.[9] Also, they are modified by several pharmacologic and non-pharmacologic means such as nutritional products and lifestyle habits, including smoking cessation,[4] caffeine intake,[10] weight reduction and regular exercise.[11]

Measurement of arterial stiffness provides direct evidence of target organ damage and is now commonly made in assessing cardiovascular risk and is recommended in current ESH hypertension guidelines.[12] Given the prognostic importance and therapeutic implications of a 'single measure' it is essential that such measurements are accurate. It is important to understand how common experiences e.g. stress, may influence such measurements. Furthermore, as with other hemodynamic responses, it is important to know if arterial stiffness changes acutely in response to physiological stimuli.

Mental stress, physical activity and environmental temperature adjustment are common in everyday life. Stress has been shown recently to be an important factor in predicting various physical and mental health outcomes, including cardiovascular morbidity. Atherosclerosis and coronary artery disease are associated with chronic mental stress. Acute stress may induce myocardial ischemia,[13] left ventricular dysfunction[14] and may even lead to sudden cardiac death.[15] Both mental and physical stress release catecholamines[16] they may also contribute to endothelial dysfunction.[17] Both catecholamine levels and endothelial function are regulators of arterial stiffness and wave reflections.[18]

Arterial responses to the cold pressor test (CPT) are due to sympathetic activation and the CPT is widely used for the assessment of cardiovascular responses, particularly vasoconstriction.[19] Our bodies respond to cold temperature by vasoconstriction to reduce heat loss to the surrounding environment. Being modulated by activation of the sympathetic nervous system, vasoconstriction is thus also related to the level of catecholamines in the blood.[20]

Constantly maintained hand-grip tension will also activate the sympathetic nervous system and increase blood pressure. The cortical impulses that initiate voluntary muscle contractions (the so-called central command) contribute to the increase in sympathetic tone. Local mechanisms also play a pivotal role in the triggering of this reflex. Muscle contraction activates afferent nerves sensitive to mechanical deformation (mechanoreceptors). The increase in muscle metabolism and the relative ischemia resulting from compression of blood vessels by the contracting muscle generates metabolic products that then activate chemo-sensitive afferent nerves (chemoreceptor or metaboreceptor). These chemoreceptors constitute the afferent limb of a reflex that results in sympathetic activation and increased blood pressure.[21]

We therefore examined hemodynamic and arterial responses to mental stress, cold pressor and isometric hand grip exercise (maximal voluntary contraction) in a group of healthy volunteers.

Materials and methods

The study included 23 young, healthy normotensive subjects who were not on any medication or agents that influence blood pressure such as oral contraceptives, steroids, or hormone replacement therapy. None of them had a history of high blood pressure, evidence of any vascular disease, cerebrovascular accident, coronary artery disease, valvular heart disease, dysrhythmias, diabetes mellitus, heart failure or any other significant medical condition.

All measurements were made in the same temperature-controlled room (22 °C). Subjects gave informed consent and the study had institutional ethics committee permission.

Study protocol

All subjects were studied on 4 occasions on separate days within a 3-week period. Subjects were asked to avoid smoking, caffeine-containing drinks, chocolate/sweets and heavy exercise for 24 h preceding each study. The subjects rested, supine, for 10 min before baseline measurements of heart rate (HR/min), systolic blood pressure (BP, mm Hg), diastolic BP (mm Hg), pulse wave velocity (PWV, m/sec) and augmentation index (AIx, %) were made. The arterial stiffness indices were measured by The Arteriograph (TensioMed Ltd), a recently developed and validated computerized device using an oscillometric method to determine PWV and AIx.[22,23] Oscillometric pressure curves (pulsatile pressure changes in the brachial artery) registered in the upper arm, are detected by plethysmography. Pulsatile changes in pressure in the artery beneath an inflated pressure cuff induce pressure changes in the cuff, which become an indirect measure for the pulsatile pressure changes in the artery beneath.[22] The Arteriograph, which yields a simultaneous measure of brachial blood pressure