427 Endothelial function in normotensive healthy siblings of hypertensive patients.
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**Background:** Hypertension is a multifaceted, multifactorial disease. It has a poly-genetic bases. Intact endothelium plays a central role in vasomotion and blood pressure control.

**Aim of the work:** was to assess endothelial mediated vasodilatation as an indicator of endothelial function in normotensive healthy siblings of hypertensive patients.

**Methods:** Forty healthy normotensive siblings of hypertensive patients with mean age 29.4±4.3, and no other risk factors. Twenty age and sex comparable healthy control individuals were selected. After full history, clinical examination, and laboratory investigation echocardiographic examination was done and left ventricular mass index was calculated (LVMI). Duplex ultrasonography of the right brachial artery was done through a previously well defined segment. Peak flow velocity (PFV), and brachial artery diameter were measured by pulsed wave and B-mode ultrasonography 30 sec before inflation (D1), of a pneumatic tourniquet to a pressure of 250 mmHg for 3-5 minutes at the forearm proximal to the site of arterial scanning and 90 sec after deflation (D2). After 15 minutes for vessel recovery a second resting scan was taken and the diameter of the brachial artery (D3), was measured then nitroglycerin spray 400 ?g was given sublingually and the measurements were repeated after 3-5 minutes (D4). Simultaneous electrocardiographic recording was done for timing. Percentage flow mediated dilatation (FMD) and nitroglycerine mediated dilatation (NMD), were calculated as D2-D1/D1X100 and D4-D3/D3X100.

**Results:** normotensive healthy siblings of hypertensive patients had a significantly higher LVMI (P<0.05) and a significantly lower percentage FMD compared to normotensive healthy controls (P<0.05). There was no significant difference as regard to percentage NMD between siblings and controls (P>0.05). Because FMD is endothelial dependent response, the impaired percentage FMD means that normotensive, risk factors free siblings of hypertensive patients had endothelial dysfunction.

**Conclusion:** healthy normotensive siblings of hypertensive patients had endothelial dysfunction. It may play a role in the pathogenesis of hypertension and may have a genetic bases.

428 Peripheral endothelial function and left ventricular hypertrophy in patients with essential hypertension.
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Left ventricular hypertrophy (LVH) is an important cardiovascular risk factor. Still less is known about the association between left ventricular mass (LVM) and endothelial function in peripheral circulation.

The aim of the study was to investigate the relation of LVM and plasma levels of soluble intercellular cell adhesion molecule (s-ICAM), soluble vascular cell adhesion molecule (s-VCAM) and endothelium-dependent flow-mediated dilatation in brachial artery (FMD) in hypertensive patients (pts).

**Material and methods:** Studied group consisted of 57 pts (26 males, 31 females) mean age 53.5±11.7 with essential hypertension and without coronary artery disease. LVM was assessed echocardiographically according to the formula: LVM = 0.8[(LVIDd + PWTd + VSTd) 3 - LVIDd 3] + 0.6. Left ventricular mass index (LVMI) was calculated as LVM divided by body surface area. Cut-off value for LVH was LVM > 134 g/m² for males and 110 g/m² for females. Plasma level of s-ICAM and s-VCAM was estimated by ELISA method. FMD was measured as the change of brachial artery diameter during reactive hyperemia by use of high-resolution ultrasound.

**Results:** LVH was found out in 29 pts (51%). Groups of patients with and without LVH did not differ with respect to plasma level of s-ICAM and s-VCAM. Patients with LVH showed significantly lower values of FMD. No significant correlations between left ventricular mass parameters (LVM and LVMI) and FMD or plasma level of s-ICAM and s-VCAM were noted.

<table>
<thead>
<tr>
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<th>no LVH (n=28)</th>
<th>LVH (n=29)</th>
<th>p</th>
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<tbody>
<tr>
<td>FMD [mm]</td>
<td>0.31 ± 0.10</td>
<td>0.22 ± 0.12</td>
<td>0.05</td>
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<tr>
<td>s-ICAM [ng/mL]</td>
<td>208.7 ± 46.3</td>
<td>340.8 ± 90.7</td>
<td>NS</td>
</tr>
<tr>
<td>s-VCAM [ng/mL]</td>
<td>1156.6 ± 537.2</td>
<td>1113.0 ± 544.8</td>
<td>NS</td>
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**Conclusion:** In conclusion, endothelium-dependent flow-mediated dilatation in brachial artery is more impaired in hypertensive pts with left ventricular hypertrophy than in those without. There are no differences in neurohormonal parameters of endothelial function - s-ICAM and s-VCAM - between hypertensive pts with and without left ventricular hypertrophy.
429 Early impairment of coronary reserve in men with essential hypertension: a quantitative myocardial contrast echocardiographic study.

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Aims of present study were: a) to demonstrate whether Quantitative Myocardial Contrast Echocardiography (QMCE) could detect coronary flow reserve through the analysis of "refilling curves" generated by micro bubbles transit into myocardium both at rest and after vasodilatation induced by diprydamol; b) to explore with this method coronary microcirculatory function in two different models: essential hypertension and healthy controls.

Methods and Results: Two groups of strictly age-matched males were studied (case-control study): twelve, young, a symptomatic and never treated essential hypertensive patients with a mild degree of left ventricular hypertrophy with a normal left ventricular function and eleven healthy controls. QMCE was performed in all study subjects. We used as echocardiography contrast agent the Sonovue TM, a second generation ultrasound micro bubbles. Real-time Color-coded Power Modulation was performed with a phased-array system interfaced to a S3 transducer (1.3 -3.6 MHz). In healthy subjects there was a little increase in Myocardial Blood Volume (30%) between basal and hyperemic status (p<0.05); so in hypertensives this parameter increases of 22% (p<0.05). Coronary blood velocity (8) increased after diprydamol of 270% in healthy (p<0.01), while in hypertensive this parameter increased increased only of 150% (p<0.02). Coronary Blood Flow Reserve was significantly lower in hypertensive (C: 4.4±0.3; H: 3.3±0.3; p<0.01).

Conclusion: Results of our study documented that coronary microcirculation in young adult hypertensive patients, showed an early impairment both in the vasodilatation capacity of the resistance arteries under diprydamole induced hyperemia, as demonstrated by a reduction of Coronary Reserve.

430 Dilated cardiomyopathy patterns in very high level endurance athletes: serial evaluation of 286 professional bicyclists of the "Tour de France".

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Knowledge of left ventricular (LV) structural modifications associated to athlete's heart is mandatory for disease screening. However, cardiac changes in very high level endurance athletes have been scarcely reported in large cohorts and long-term evaluations. Therefore, we investigated for the first time in a systematic study 350 consecutive patients of patients with hypertrophic obstructive cardiomyopathy (HOCM) referred for catheter interventional therapy of HOCM.

Methods and Results: 286 athletes (Gr. A) [148 cyclists in 1995 (Gr. A1) and 138 of the "Tour de France". Diastolic LV diameters matched sedentary volunteers served as controls (Gr. C). Diastolic LV diameters in 286 athletes (Gr. A) [148 cyclists in 1995 (Gr. A1) and 138 of the "Tour de France". Diastolic LV diameters matched sedentary volunteers served as controls (Gr. C). LV diameters (WT, mm) were 11.1±3.9, 52.9±3.8, 61.0±3.9 and 49.0±4.3 in groups A, A1, A2 and C respectively (A1 vs. A2 and A vs. C, p<0.0001). Maximal LV wall thicknesses (WT, mm) were 11.1±3.1, 11.6±3.1, 10.6±3.1 and 8.6±1.0 in groups A, A1, A2 and C respectively (A1 vs. A2 and A vs. C, p<0.0001). Among athletes, 147 (51%) had LVOTG > 60 mm (usual threshold for pathologic enlargement in athletes), 17 (11.6%) along with LV ejecution fraction (EF) <52% (2 SD below the mean of controls). Applying a threshold of 30.6 mm²/m² for indexed UVID (25% above the mean of controls), 205 (71%) athletes had LV dilatation. Among athletes, 25 (8.7%) had septal WT > 13 mm but only 2 along with UVID < 55mm.In group B, serial examinations showed increased UVID from 58.3±4.8 in 1995 to 60.3±4.2 mm in 1998, p<0.001) but decreased septal (from 11.8±1.2 mm in 1995 to 10.8±1.2 mm in 1998, p<0.001) and posterior (from 10.6±1.0 mm in 1995 to 9.9±0.8 mm in 1998, p<0.001) WT. Conclusions: 1) More than 50% of these very high level athletes demonstrated marked LV dilatation along with decreased EF in more than one case out of ten, arising the problem of the presence of a dilated cardiomyopathy (DCM); 2) Further unexpected LV dilatation and wall thinning may occur with time; 3) Hypertrophic cardiomyopathy (HCM) is less often evoked, as increased WT is less common (9%) and scarcely occurred in the absence of LV dilatation (<1%). These results have important implications for screening (DCM, HCM) in these populations.

431 Intra-procedural myocardial contrast echocardiography in septal ablation for symptomatic hypertrophic obstructive cardiomyopathy.

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Background and Introduction: Percutaneous septal ablation (PTMSA) for hypertrophic obstructive cardiomyopathy (HOCM) requires the exact definition of the septal myocardium to be attacked. We report on our cumulative experience with PTMSA guided by intra-procedural contrast echo (MCE) on an intention-to-treat basis in 344 patients (pts.) from 1/1996 – 4/2002.

Results: Ethanol injection was withheld in 28 pts. (8%), predominantly due to an unwanted extension of the region at risk as documented by MCE in 20 pts. (6%). Furthermore, in 40 pts. (12%) a target vessel (TV) change was necessary for the same reason. In-hospital mortality in the 316 pts. who received a mean dose of 2.7±1.2 ml of ethanol was 1.2% (4 pts.). After 3 months, symptoms had improved in 262 pts. (90%) from NYHA class 2.9±0.4 to 1.5±0.7, 157 pts. (54%) reported to be symptom free. After 1 year, 222 pts. (85%) maintained 1.5±0.5 NYHA class. Asymptomatic patients (pts.) from 1/1996 – 4/2003.

Conclusion: An unwanted extension of the area at risk for the ethanol-induced necrosis as shown by intra-procedural MCE is the main reason to stop an attempted septal ablation. The cumulative impact of intra-procedural MCE on the interven- tional strategy of PTMSA is about 15-20% in accordance with the reported patho- anatomic finding of irregular perfusion areas of septal perforator arteries in about the same percentage.

432 Discrete subvalvular aortic stenosis - pitfall in the echocardiographic diagnosis of patients with hypertrophic obstructive cardiomyopathy (HOCM) referred for catheter interventional treatment.

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Introduction: Generally, diagnosis of typical HOCM with subaortic obstruction and dynamic pressure gradient across the left ventricular outflow tract is made by non invasive diagnostic procedures with great certainty by employing transtho- racic exercise Doppler echocardiography or transoesophageal echocardiography (TEE). However, in pts with asymmetric septal hypertrophy additional discrete subvalvular membrane may be a potential pitfall in the diagnosis of HOCM as described in several case reports in the literature. The exclusion of discrete subvalvular aortic stenosis (DSAS) is of special importance in pts referred for catheter interventional therapy. To date, systematic investigations concerning the frequency of DSAS in symptomatic pts, referred for catheter interventional therapy of HOCM are lacking.

Methods: Therefore, we investigated for the first time in a systematic study 350 consecutive symptomatic (functional class 3 or 4 according to NYHA) pts with HOCM who were referred for this new catheter interventional therapy. In all pts TTE and bi- cycle exercise Doppler echocardiography were performed. Additionally in most pts multiplane TEE was performed.

Results: In 7 of 350 pts (2%) subvalvular aortic stenosis (female pts and 2 male pts; age 16 to 63 years; functional class 3 according to NYHA; mean septal diameter 19 mm; mean diameter of the posterior wall 13 mm; Sarn-like motion in all pts) non compatible with HOCM was found. 6 of these pts belonged to the membranous type of DSAS; in one pt a tunnel-form of subvalvular aortic stenosis was present. In all cases the diagnosis could be confirmed by surgical treatment. In most pts TEE evaluation was of crucial importance with demonstration of a typical subvalvular membrane (in 6 pts) which was situated a few millimeters below the aortic valve. In all cases asymmetric septal hypertrophy mimicking HOCM was seen. In all pts there were small changes only seen at echocardiography, however a very pronounced intraoperative finding was present.

Conclusion: The frequency of discrete subvalvular aortic stenosis in symptomatic pts referred for catheter based treatment of HOCM is unexpectedly high (2%). Especially in pts in whom TTE evaluation is of insufficient quality, the use of multiplane TEE with careful evaluation of the small poststenotitic subvalvular area in HOCM is of importance in diagnosing and classifying DSAS (membranous type, fibromuscular ring, tunnel type). This is of special significance prior to catheter interventional ther- apy, because in pts with subvalvular aortic stenosis surgical treatment is mandatory.