429

Early impairment of coronary reserve in men with essential hypertension: a quantitative myocardial contrast echocardiographic study.

V. Di Bello[1], D. Giorgi[1], R. Pedrinelli[1], C. Palagi[1], M.G. Delle Donne[1], E. Talini[1], M. Paterni[2], M. Mariani[1]. *[1]Dipartimento Cardio Toracico, Pisa, Italy; [2]Instituto Fisiologia Clinica, CNR, Pisa, Italy*

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 dipyridamole; 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 (ß) increased after dipyridamole of 270% in healthy ($p<0.01$), while in hypertensive this parameter 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 arterioles under dipyridamole 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".

E. Abergel[1], G. Chatellier[2], A.A. Hagege[1], A. Oblak[1], A. Linhart[1], A. Ducardonnet[3], J. Menard[4]. *[1]Hôpital Européen Georges Pompidou, Cardiologie, Paris cedex 15, France; [2]Hôpital Européen Georges Pompidou, Informatique médicale, Paris, France; [3]Institut coeur effort santé, Cardiologie, Paris, France; [4]université paris VI, Santé publique, Paris, France*

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 adaptations remain largely unknown. We performed serial echocardiography in very high level professional bicyclists participating to two races (1995 and 1998) of the "Tour de France".
Methods and Results: 286 athletes (Gr. A) [148 cyclists in 1995 (Gr. A1) and 138 cyclists in 1998 (Gr. A2)] were included; 37 participated to both races (Gr. B); 52 matched sedentary volunteers served as controls (Gr. C). Diastolic LV diameters (LVIDd, mm) were 60.1±3.9, 59.2±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±1.3, 11.6±1.3, 10.6±1.1 and 8.6 ±1.0 in groups A, A1, A2 and C respectively (A1 vs. A1 and A vs. C, $p<0.0001$). Among athletes, 147 (51%) had LVIDd $>$ 60 mm (usual threshold for pathologic enlargement in athletes), 17 (11.6%) along with LV ejection fraction (EF) $<$ 52% (2 SD below the mean of controls). Applying a threshold of 30.6 mm/m^2 for indexed LVIDd (2SD 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 LVIDd $<$ 55mm.In group B, serial examinations showed increased LVIDd (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, $p<0.001$) WTs. Conclusions: 1) More than 50% of these very high level athletes demonstrates 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.

L. Faber[1], D. Welge[1], H. Seggewiss[2], D. Fassbender[1], H.K. Schmidt[1], U. Gleichmann[1], D. Horstkotte[1]. *[1]Heart Center North Rhine-Westphalia, Cardiology Dept., Bad Oeynhausen, Germany; [2]Leopoldina Hospital, Department of Internal Medicine, Schweinfurt, Germany*

Background and Introduction: Percutaneous septal ablation (PTSMA) for hypertrophic obstructive cardiomyopathy (HOCM) requires the exact definition of the septal myocardium to be attacked. We report on our cumulative experience with PTSMA guided by intra-procedural contrast echo (MCE) on an intention-to-treat basis in 344 patients (pts.) from 1/1996 – 4/2003.
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 free of cardiac symptoms ($p<0.0001$). A satisfactory mid-term reduction of the left ventricular outflow gradient (LVOTG) was achieved in 261 pts. (83%; from 60±33 to 14±20 Hg at rest and 121±41 to 40±37 mm Hg with provocation; $p<0.0001$). 128 pts. (41%) were free from outflow obstruction. LVOTG with probatory balloon occlusion (PBO) during the intervention was 39±31 mm Hg ($p<0.001$). There was a weak correlation between the LVOTG with PBO and the residual LVOTG after 3 months (0.3; $p<0.01$).
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 interventional strategy of PTSMA is about 15-20% in accordance with the reported pathoanatomic 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.

G. Beer, C. Strunk-Müller, B. Stolle, H. Kuhn. *Städtische Kliniken Bielefeld - Mitte, Klinik für Kardiologie, Bielefeld, Germany*

Introduction: Generally, diagnosis of typical HOCM with subaortic obstruction and dynamic pressure gradient accross the left ventricular outflow tract is made by non invasive diagnostic procedures with great certainty by employing transthoracic (TTE) and/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 bicycle exercise Doppler echocardiography were performed. Additionally in most pts multiplane TEE was performed.
Results: In 7 of 350 pts, (2%) subvalvular aortic stenosis (5 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; Sam-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 poststenotic 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 therapy, because in pts with subvalvular aortic stenosis surgical treatment is mandatory.