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CARDIOLOGY 2001, OCTOBER 18–20, VIENNA, AUSTRIA: ABSTRACTS

ENDOTHELIN-1 IS INVOLVED IN CORONARY NO-REFLOW

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Background: No-reflow (NR) is a multifactorial condition comprising an acute reduction in coronary flow (TIMI grade 0–1) in the absence of epicardial vessel obstruction. Previous data have demonstrated that tissue factor that is shed from the atherosclerotic plaque, causes NR by inducing immediate and widespread coronary microvascular fibrin deposition. However, current concepts invoke vasoconstriction as an additional factor in NR. Therefore, we investigated the role of endothelin (ET-1), a potent vasoconstrictor peptide that is present in atherosclerotic lesions.

Methods and Results: Initial experiments utilized homogenates from atherosclerotic arteries and confirmed the presence of ET-1 antigen at levels tenfold above normal plasma concentrations (0.4 ± 0.07 fmol/ml). In the next step, right coronary arteries (RCAs) from 6 domestic pigs were selectively injected with 2 ml each of homogenized atherosclerotic human plaque material containing 6.9 ± 4 fmol/ml ET-1 per g of tissue. In subsequent angiograms, flow was compromised in 5 of 6 animals. Fractional flow reserve dropped from 2.0 ± 1 to 0.8 ± 0.2 with a marked decrease of baseline flow immediately after the injection. To examine the role of ET-1 in human acute coronary NR, coronary blood was drawn from patients undergoing percutaneous interventions using the distal protection devices PercuSurge GuardWire (n = 10), the Exciser Catheter System Thrombus Removal Device (n = 9) and Angioguard (n = 2). Fine particulate material that was recovered from each of those devices contained 0.13 ± 0.18 fmol ET-1 antigen/40ml whole blood (Percu Surge), 0.11 ± 0.12 fmol ET-1 antigen/80ml whole blood (Exciser), and 0.005 ± 0.02 fmol ET-1 antigen/40000 ml (Angioguard).

Conclusions: The data suggest that ET-1 is released into the coronary circulation from ruptured atherosclerotic plaque and contributes to the no-reflow phenomenon, possibly by inducing spasm in the depending coronary microvasculature.

SYMPTOMATIC IMPROVEMENT AFTER TRANSCATHETER ATRIAL SEPTAL DEFECT CLOSURE IN ADULTS

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Background: Transcatheter atrial septal defect (ASD) closure has been shown to be feasible and safe. However, little is known about the clinical outcome of adult pts, particularly those of advanced age.

Methods: We performed transcatheter ASD closure with the Amplatzer Septal Occluder in 55 adults (mean age 47 ± 17 years, 35 female) of whom 35 were older than 40 years (up to 82 yrs). Patients were followed for up to 2 years.

Results: ASD was successfully closed in all pts (device size 23 ± 6 mm, range 10–34 mm). No major complications occurred. Minor complications were atrial fibrillation (2), transient AV-block (1) and transient ST-elevation (1). At follow-up, a mild residual left-to-right shunt was found in 2 pts.

Prior to intervention, 24 pts were symptomatic of whom 19 were older than 40 years. Most frequent symptoms were limited exercise capacity and shortness of breath (NYHA class 2–3 or 3 in 13 pts). At follow-up, all but one pt improved. This patient remained in NYHA class 3 but had marked pulmonary hypertension. All other patients were asymptomatic or had only mild shortness of breath. All of the 10 pts who were 65 yrs or older and who were treated because of significant symptoms markedly improved.

Conclusion: Transcatheter atrial septal defect closure can safely and successfully be performed in adults. Symptomatic improvement can generally be expected even in patients of advanced age.

CAN TRANSOESOPHAGEAL ECHOCARDIOGRAPHY ACCURATELY PREDICT THE IMPLANT SIZE FOR TRANSCATHETER ATRIAL SEPTAL DEFECT CLOSURE?

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Background: Successful transcatheter atrial septal defect (ASD) closure requires careful selection of the appropriate device size. Undersizing may result in a residual shunt and even device embolization. Balloon sizing is used to determine the "stretched" defect diameter but it is time consuming. The purpose of this study was to evaluate whether transoesophageal echo (TEE) can predict the stretched defect diameter and, therefore, the required device size.

Methods: Transcatheter ASD closure with the Amplatzer ASD Occluder was performed in 55 adults (mean age 47 ± 17 years, 35 female). The size of the device was determined by balloon sizing. The stretched defect diameter was compared with the native diameter measured by TEE considering morphologic characteristics of the interatrial septum.

Results: The native defect diameter (TEE) was 15 ± 4 mm (6–22 mm). The stretched diameter (balloon sizing) was 23 ± 6 mm (10–34 mm). Thus, the size of the device exceeded the native defect diameter by 65 ± 47 % (9 ± 5 mm) with a wide variation ranging from 6 to 200 % (1 to 16 mm). The relation between morphologic characteristics of the interatrial septum (thickness, aneurysm, mobility, rim to the aorta) and the distensibility of the defect was studied. Although differences between stretched and native diameters tended to be greater in pts with a thin and/or aneurysmatic interatrial septum, prediction of defect distensibility in individual pts was impossible. Even for those pts in whom a high defect distensibility was expected from the morphologic appearance of the interatrial septum, differences between native and stretched diameters varied from 2 to 16 mm (10 to 200 %).

Conclusion: TEE is crucial for selection of patients eligible for transcatheter ASD closure and for the monitoring of the

procedure. However, the stretched defect diameter and thus the size of the device cannot be reliably predicted from the TEE determined native defect diameter even if the morphology of the residual interatrial septum is considered.

Intracoronary thrombectomy in acute coronary syndrome with a new percutaneous device: the Vienna X-Sizer study

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Background: Thrombus formation following plaque rupture is responsible for acute vessel occlusion in acute myocardial infarction (AMI) and contributes to compromised flow in unstable angina (UA). The X-Sizer catheter is a new device for percutaneous coronary thrombectomy. This prospective single-center controlled randomised study investigated the effects of the X-Sizer system with regard to epicardial flow and microcirculatory function in pts with acute coronary syndrome (ACS).

Methods: So far 44 patients (34 male; 55.3 ± 9.6 yrs) with ACS (31 AMI, 13 UA) in native vessels were randomised to treatment with the X-Sizer followed by PTCA and/or stenting (n = 22) or to PTCA and/or stenting alone. Quantitative coronary angiographic (QCA) and Doppler flow measurements, TIMI Frame Count, ST-segment score (= sum of ST-elevation in all 12 standard leads) and CK values were obtained. Major Adverse Cardiac Events (MACE) were determined after 30 days.

Results: In all pts treated with X-Sizer the lesion could be successfully crossed. No device related complications occurred. MACE after 30 days was observed in one pt in the X-Sizer and in two pts in the control group. Acute lumen gain, corrected TIMI Frame Count (cTFC), CK peak and Coronary Flow Reserve (CFR) were not significantly different between the two groups. However, ST-segment scores immediately after, and 6 h post procedure were significantly lower in the X-Sizer group **(Table 1).**

Conclusion: The initial results of this ongoing first randomised trial demonstrate safety and efficacy of the X-Sizer catheter for thrombus-removal in ACS. A significantly faster normalization of ST-segment elevation was observed in pts treated with X-Sizer. This possibly indicates improved myocardial salvage by additional use of the X-Sizer system.

Table 1: G. Beran et al.

	Acute lumen gain (mm)	cTFC pre	cTFC post	CFR post	ST-score pre (mm)*	ST-score post	ST-score 6 h post
X-Sizer	2.03 ± 0.9	77 ± 30	19 ± 11	1.54 ± 0.6	10.8 ± 15.6	2.5 ± 3.5**	1.6 ± 2.2**
Control	1.94 ± 0.6	73 ± 37	24 ± 14	11.55 ± 0.7	11.7 ± 7.1	5.5 ± 4.8**	4.0 ± 3.5*

Data are given as mean values ± SD, * in pts with AMI, ** p < 0. 05 (t-test)

Experience with prostaglandin E1 bridging to high dose beta-blocker therapy in advanced heart failure patients — different outcome in patients with and without chronic low dose beta-blocker pretreatment

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Background: PGE1 improves clinical symptoms, neurohumoral status and haemodynamics and reduces the risk of heart failure (HF) worsening. Thus it is used to initiate or increase β-blocker therapy in patients (pts) with HF worsening. We compared the outcome of pts with and without chronic low dose β-blocker pretreatment before HF worsening.

Methods and Results: 66 HF pts in NYHA class IV refractory to oral medical treatment received continuous iv PGE1. 34 pts (LVEF 15 ± 5 %, RR mean 77 ± 14 mmHg, cardiac index 1.9 + 0.5 L/min/m², plasma big endothelin [ET] 7.4 ± 3.5 fmol/ml) were pretreated with 36 ± 30 % of the maximum β-blocker dose for 4 ± 3 months (group A), 32 pts (LVEF 16 ± 5 %, RR mean 78 ± 14 mmHg, cardiac index 1.8 ± 0.5 L/min/m², plasma big ET 7.1 ± 4.0 fmol/ml) were not (group B). After 4.5 + 7.2 months β-blocker dose was increased to 80 ± 40 % of the maximum in group A (p = 0.0001), after 4.3 + 5.6 months group B received 64 ± 39 % of the maximum β-blocker dose. During this treatment period plasma big ET decreased significantly compared to baseline (5.9 ± 4.2 fmol/ml in group A, p = 0.01 vs baseline; 3.2 ± 2.1 fmol/ml in group B, p = 0.0001 vs baseline; p = 0.003 between groups), plasma big ET did not decrease in group A, but decreased in group B below 4.3 fmol/ml (a cutpoint repeatedly shown to indicate very poor prognosis). In group A 7 pts (21 %) could be weaned from PGE1, 6 pts (18 %) received a left ventricular assist device, 16 pts (47 %) were transplanted and 5 pts (15 %) died. In group B 19 pts (59 %) could be weaned from PGE1 (p = 0.001 vs group A), 1 pt (3 %) received a left ventricular assist device, 12 pts (38 %) were transplanted and no pt died (p = 0.02 vs group A).

Conclusion: Despite similar baseline characteristics pts with chronic low dose β-blocker pretreatment before HF worsening have a worse outcome compared to pts without β-blocker pretreatment before HF worsening when they are treated with PGE1 as bridging to high dose β-blocker therapy.

Prognostic power of neurohumoral parameters in chronic heart failure depends on the clinical stage and on the observation period

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Background: Endothelin (ET) and natriuretic peptides have prognostic significance in chronic heart failure (CHF). As the stimuli for formation of these neuro-

hormones are different, we investigated whether their prognostic power depends on the clinical stage and on the length of the observation period.

Methods and Results: Plasma levels of big ET, BNP, N-terminal BNP (N-BNP) and N-terminal ANP (N-ANP) in addition to 11 clinical and haemodynamic variables were obtained from 453 patients with LVEF ≤ 35 %. According to their NYHA class and LVEF, patients were stratified into group A – mild CHF (n = 114), group B – moderate CHF (n = 210) and group C – severe CHF (n = 128). For prediction of the combined endpoint death or urgent HTX a multivariate analysis was performed after an observation period up to 1, 2 and 3 years in all patients and in each subgroup. Best independent predictors were as follows:
All patients: Up to 1 year – big ET ($p = 0.0001$, $x^2 = 59$), 2 and 3 years – N-ANP ($p = 0.0001$, $x^2 = 68$; $p = 0.0001$, $x^2 = 89$).
Group A: Up to 2 and 3 years – N-ANP ($p < 0.001$, $x^2 = 12$; $p = 0.0001$, $x^2 = 25$).
Group B: Up to 1 and 3 years – N-ANP ($p = 0.0001$, $x^2 = 16$; $p = 0.0001$, $x^2 = 22$), 2 years – N-BNP ($p = 0.0001$, $x^2 = 19$).
Group C: Up to 1, 2 and 3 years – big ET ($p = 0.0001$, $x^2 = 23$; $p = 0.0001$, $x^2 = 22$; $p = 0.0001$, $x^2 = 20$).

Conclusion: Big ET was the best independent marker for short-term prognosis and in severe CHF, natriuretic peptides (especially N-ANP) were better markers for long-term prognosis and in mild and moderate CHF.

Elevation of serum big endothelin is associated with impairment of endothelium mediated vasodilatation in patients with chronic heart failure

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Background: We have previously reported that ETA receptor blockade improves impaired endothelium-dependent flow-mediated vasodilatation (FMD) in CHF patients. In the present study we investigated the relationship between big ET plasma levels (by radioimmunoassay) and FMD (of the brachial artery by high resolution ultrasound).

Methods: 44 CHF patients (NYHA class I/II/III/IV–10/7/22/5 patients; LVEF < 30 %) and 13 age, sex and risk factor-matched controls have been studied. Patients were stratified into group A, big ET below the upper normal range of 1.8 fmol/ml, group B, big ET between 1.8 and 4.3 fmol/ml (the latter value being a cutpoint repeatedly shown to indicate very poor prognosis) and group C, big ET above 4.3 fmol/ml.

Results: Big ET plasma levels were 3.5 ± 2.2 fmol/ml in CHF patients and 2.1 ± 0.6 fmol/ml in controls ($p < 0.05$). FMD was 6.8 ± 5.2 % in CHF patients and 10.6 ± 5.1 % in controls ($p < 0.05$). Comparing the control group and the CHF subgroups (group A – n = 14, LVEF 19 ± 4 %, group B – n = 16, LVEF 20 ± 5 % and group C – n = 14, LVEF 19 ± 6 %) with each other, FMD was similar between controls and group A (10.5 ± 3.7 %), but differed significantly between controls and group B (5.8 ± 6.1 %, $p < 0.05$), between controls and group C (4.2 ± 2.8 %, $p < 0.01$), between group A and group B $p < 0.05$), and between group A and group C ($p < 0.01$). The difference between groups B and C was not statistically significant. In a multivariate analysis including age, gender, underlying heart disease, smoking, diabetes, hypercholesterolaemia, hypertension and big ET, big ET was the strongest independent predictor of endothelial dysfunction ($R = –0.53$, $p = 0.0002$) with only diabetes mellitus providing additional information ($R = 0.33$, $p = 0.002$).

Conclusion: Patients with normal big ET plasma levels have a similar FMD compared to controls, whereas patients with elevated big ET plasma levels have an impaired FMD. Big ET is a strong independent predictor of endothelial dysfunction.

Increased prevalence of elevated plasma factor VIII in chronic thromboembolic pulmonary hypertension

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Background: Chronic thromboembolic pulmonary hypertension (CTEPH) is the result of non-resolving pulmonary thromboembolism, eventually leading to right heart failure and death. In accordance with the absence of deep vein thrombosis (DVT) in over 60 % of CTEPH patients, and a lack of risk factor sharing with DVT, there are no known abnormalities of coagulation and fibrinolysis that could explain the clinically progressive thrombosis in the pulmonary vasculature of these patients.

Methods and Results: Because plasma factor VIII above 150 IU/dl has been shown to confer a 5-fold increased risk for DVT, we measured plasma factor VIII and levels in CTEPH patients (n = 87) and compared them with age and sex matched healthy controls (n = 82). To rule out dysfunctional endothelium of pulmonary hypertension as a source for elevated plasma factor VIII, the data were also compared with matched samples from patients with non-thromboembolic pulmonary arterial hypertension (PAH, n = 68). In CTEPH patients factor VIII above 150 IU/dl was more prevalent than in controls (85.7 % versus 18.2 %, $p < 0.0001$) and PAH patients (55.5 %, $p = 0.002$). More-

Table 2: D. Bonderman et al. Haemodynamic parameters and plasma factor VIII in 18 CTEPH patients before and after successful PTE

Parameter	Baseline	Postoperative	P-value
mPAP (mmHg)	54 ± 14	32 ± 12	< 0.05
CO (L/min)	4.4 ± 1.1	5.5 ± 1.1	< 0.05
PVR (dynes x s x cm^{-5})	943 ± 435	317 ± 213	< 0.05
MVS (%)	59 ± 11	67 ± 7	< 0.05
Factor VIII (IU/dl)	224.4 ± 97.3	211.1 ± 97.2	0.71

mPAP = mean pulmonary arterial pressure, CO = cardiac output,
PVR = pulmonary vascular resistance, MVS = mixed venous saturation

over, CTEPH patients had higher levels of factor VIII than controls (232.9 ± 103.2 IU/dl versus 93.0 ± 23.3 IU/dl, $p < 0.001$) and PAH patients (168.2 ± 81.2 IU/dl, $p = 0.009$). In addition, plasma von Willebrand factor (vWF), a factor VIII-dependent risk factor for DVT, was significantly increased in CTEPH (267.9 ± 137.5 %) versus PAH (208.1 ± 112.7 %, $p = 0.043$). Haemodynamic improvement after pulmonary thromboendarterectomy (PTE) did not normalize plasma factor VIII levels (224.4 ± 97.3 IU/dl versus 211.1 ± 97.2 IU/dl, $p = 0.71$, $n = 18$) in CTEPH patients **(Table 2).**

Conclusions: The data support the concept of a thromboembolic origin of CTEPH.

Subacute stent thrombosis and severe gastrointestinal disorders

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Background: Subacute thrombosis is a potentially lethal complication after coronary stent implantation. Initial attempts to reduce its occurrence with heparin and/or warfarin were not satisfactory. To date, a combination antiplatelet therapy with aspirin plus ADP inhibitors (ticlopidine, clopidogrel) has emerged as the standard treatment for prevention of subacute stent thrombosis with a remaining incidence between 0.5 % and 0.8 %.

Methods: To examine whether incomplete drug absorption due to severe gastrointestinal (GI) disorders might account for subacute stent thrombosis we retrospectively analysed the medical histories of all patients with angiographically documented subacute stent thrombosis (> 72 hours after stent implantation) at our centre between November 1995 and July 2001. Patients receiving warfarin were excluded from the analysis.

Results: Fourteen patients (0.5 %) had experienced subacute stent thrombosis. Of these patients 3 (21.4 %) had a short bowel syndrome after gastrojejunostomy due to gastric ulcer, and one (7.1 %) suffered from diffuse erosive gastritis. Another 4 (28.6 %) patients had discontinued antiplatelet drug therapy.

Conclusions: Subacute stent thrombosis is associated with GI conditions where a reduced surface area for drug absorption may lead to decreased plasma levels of ADP inhibitors. Pharmacokinetic studies and, subsequently, different dosing regimes are required for patients with the short bowel syndrome/erosive gastritis to prevent the occurrence of subacute stent thrombosis.

Inhibition of smooth muscle proliferation by GPIIb-IIIa antagonists in-vitro: eptifibatide more effective than abciximab or tirofiban

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Background: Recent data suggest that among the platelet glycoprotein (GP) IIb-IIIa ($\alpha_{IIb}\beta_3$) integrin antagonists not only abciximab (ReoPro®, a chimeric monoclonal antibody), but also eptifibatide (Integrilin®, a cyclic heptapeptide) is able to bind to integrin-receptors of the α_v subunit family, whereas the affinity of tirofiban (Aggrastat®, a peptidomimetic) is restricted to $\alpha_{IIb}\beta_3$. We were interested whether these differences in integrin specificity might influence the effect on human smooth muscle cell (SMC) migration and/or proliferation *in-vitro*.

Methods: A two-dimensional assay system for determination of SMC migration/proliferation was used. Sterilized steel supports were inserted into gelatine coated six well plates. Human umbilical artery SMC were seeded into the middle hole of each support. At confluency, inserts were removed and after 24 hours of serum deprivation, GP IIb-IIIa antagonists were added in culture media with 10 % calf serum. After 20 days cell-layers were stained, digitally quantified (Scion Image) and in parallel cells counted.

Results: No GP IIb-IIIa antagonist influenced SMC area significantly at therapeutic plasma concentrations (abciximab: 200 ng/ml; eptifibatide: 2 µg/ml, tirofiban: 40 ng/ml). At 10× conc. eptifibatide reduced SMC area significantly by 30 % ($p = 0.01$) and at 30× conc. by 41 % ($p = 0.001$), whereas abciximab and tirofiban showed no significant influence. This effect was paralleled by a decrease in cell counts (30× conc.: 43 % reduction, $p = 0.005$), reflecting an inhibition of cell proliferation.

Conclusions: Among the currently available GP IIb-IIIa antagonists, only eptifibatide showed a dose-dependent inhibition of SMC proliferation in our *in-vitro* assay system. Abciximab and tirofiban exerted no significant effects. As these findings might be due to eptifibatide's broader range of integrin specificity ($\alpha_{IIb}\beta_3$ and various a_v subunits of integrin), compared to abciximab ($\alpha_{IIb}\beta_3$ and $\alpha_v\beta_3$) or tirofiban (only $\alpha_{IIb}\beta_3$), we hypothesize that inhibition of SMC proliferation requires simultaneous blockade of several integrin receptors. The clinical relevance of this phenomenon, with reduction of late restenosis after percutaneous coronary interventions needs to be proven.

Long-term outcome of patients With ventricular septal defect considered not to require surgical closure during childhood

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Aim of the Study: The purpose of the study was to assess the long-term outcome of patients with small ventricular septal defects (VSD) considered not to require surgical closure during childhood. Although patients with small VSD have generally been considered not to require surgery, more recent data suggest that a significant percentage of these patients develop serious problems during adult life.

Methods: 229 consecutive pts (115 females) with a VSD considered too small to require surgery during childhood as defined by normal pulmonary artery pressure (PAP), < 50 % shunt, pulmonary vascular resistance ≤ 200 dynes × sec × cm^{-5}, no VSD related aortic regurgitation, and no symptoms who had no additional haemodynamically relevant heart defect were followed: Physical examination, ECG, and echocardiography were per-

formed in all pts in 1 to 3 year intervals; exercise test and Holter monitoring could be performed in 140 and 127 pts, respectively.

Results: Follow-up could be completed in 222 pts (97 %). Mean age at last visit was 30 ± 10 years. Spontaneous VSD closure was observed in 14 pts (6 %). No pt died, 4 pts (1.8 %) had an episode of endocarditis of whom 2 required aortic valve replacement, one additional pt (0.4 %) had surgical closure for haemodynamic reasons. For 118 pts who entered the study between 1993–1996 and were prospectively followed for 7.4 ± 1.2 years, event free survival was 99.1 ± 0.8 % at 3 years, 96.5 ± 1.7 % at 6 years, 95.5 ± 1.9 % at 8 years. At last visit, 94.6 % of all pts studied were free of symptoms. LV size by echocardiography was normal in 198 (89 %) patients, borderline in 23 patients and definitely enlarged in only one patient. None had systolic LV dysfunction and PAP was normal in all except one. Mean exercise capacity was 92 ± 21 % of expected and 87 % of pts had no arrhythmias on Holter monitoring with the rest showing benign rhythm disorders.

Conclusions: Outcome in well-selected pts with a small VSD is good. Surgical closure does not appear to be required during childhood as long as left-to-right shunt is less than 50 % and signs of left ventricular volume overload are absent, when PAP is not elevated, and no VSD related aortic regurgitation or symptoms are present.

Analysis and study of sudden cardiac deaths in central Greece (retrospective study)

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Purpose of study: The distribution of sudden cardiac deaths by cause, gender, age, year and place of living has been studied all over the world. There are only a few data analysis studies in Greece, so far. The purpose of this study is to analyze and study sudden cardiac deaths, which occurred in the county of Trikala during the period from 1990–2000.

Material and Methods: We wrote down 268 cases of out-of-hospital sudden cardiac deaths, which occurred in the adult population in the last 11 years. Necrotomy took place in the General Hospital of Trikala. Age and place of living were unknown in 12 and 14 cases, respectively. (Population of the county of Trikala: 139,500, population of the city of Trikala: 51,670). The sudden cardiac death is defined, in accordance to the international standards, as the natural, unexpected, cardiac death occurring within one hour of the onset of acute symptoms. Chi^2 and t-test were performed for statistical analysis.

Results: Sudden cardiac deaths: A: By year: 1990: 24, 1991: 31, 1992: 36, 1993: 11, 1994: 26, 1995: 25, 1996: 21, 1997: 16, 1998: 26, 1999: 26, 2000: 26. B: By gender: 214 male (m) (80 %), mean age 60.5 years old and 54 female (f) (20 %), mean age 67 years old. C: By age: 15–24: 1 (1 m, 0 f), 25–34: 9 (8 m, 1 f), 35–44: 18 (17 m, 1 f), 45–54: 47 (43 m, 4 f), 55–64: 58 (46 m, 12 f), > 64: 123 (92 m, 31 f). D: By place of living: city of Trikala: 73, rest county: 157, other places: 24. E: By cause: acute myocardial infarction: 242 (90.2 %), acute cardiac failure: 19 (7 %), heart failure: 3 (1.2 %), acute cardiogenic pulmonary oedema: 1 (0.4 %), unexplained: 3 (1.2 %).

Conclusions: 1. The incidence of sudden cardiac death has a significant preponderance in males compared with females and increases according to age. 2. Sudden cardiac deaths occur more often out of the city of Trikala. 3. The major cause of sudden cardiac deaths is acute myocardial infarction.

Pulmonary toxicity of amiodarone – case report

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Introduction: Amiodarone is an antiarrhythmic agent with significant benefit for ventricular dysrhythmias in patients who do not respond to most other antiarrhythmic drugs. One of its side effects is pulmonary toxicity.

Case Presentation: A 54 y.o. man was admitted to the hospital with fever, malaise, dyspnoea and non-productive cough. Three months before admission the patient underwent a coronary-artery bypass grafting because of three-vessels disease. Postoperatively, an episode of symptomatic atrial fibrillation was diagnosed and amiodarone was added at a dose of 200 mg/d (without loading dose) to his basic medication. Four days before admission a fever up to 39.2 °C and dyspnoea developed.

He appeared tachypnoeic the breath sounds were accentuated, but no crackles were heard. The heart rate was 100, rhythmic. His blood gases revealed a hyperventilation with moderate hypoxaemia. In chest x-ray bilateral interstitial infiltration was seen, a high resolution CT showed ground-glass opacities in both lungs with reticular abnormalities, septal thickening. Laboratory studies disclosed a mild leucocytosis, elevated sedimentation rate and acute phase reaction. The thyroid hormones were within normal range. Pulmonary function tests were abnormal with severe decrease in total lung capacity and carbon diffusing capacity. In bronchoalveolar lavage there was an increased number of neutrophils, lymphocytes and eosinophils, CD8+ cells were also increased with a low CD4/CD8 ratio. Foamy macrophages were shown and the histology revealed widened septa and mixed infiltrates. The diagnosis of hypersensitivity pneumonitis due to amiodarone was made and the patient was started on corticosteroids and oxygen. Amiodarone has been discontinued. His condition stabilized within 10 days and he was discharged.

Discussion: Pulmonary toxicity is the feared side effect of amiodarone. In 20 % of the patients the pneumonitis developes acute, mimicking pneumonia. Differential diagnosis includes any type of interstitial pneumonia.

Conclusion: The diagnosis of amiodarone pneumonitis is one of exclusion. There is no threshold dose for developing pulmonary complications. In case of our patient it was a maintenance dose of 200 mg for 2 months.

Atorvastatin or simvastatin concomitant to LDL-apheresis treatment in patients with homozygous and heterozygous familial hypercholesterolaemia: a prospective crossover study

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Background: To enable further reduction in LDL-cholesterol (LDL-C) in patients with homozygous (n = 4) and severe heterozygous (n = 10) familial hypercholesterolaemia (FH) undergoing LDL-immunoapheresis we altered the concomitant drug treatment from simvastatin to atorvastatin following a prospective designed crossover study protocol.

Methods: While LDL-apheresis was performed at weekly intervals, lipoprotein values were compared under 40 mg of simvastatin followed by a wash-out period of 4 weeks and when patients received atorvastatin therapy, starting with a 10 mg dose and escalating every 4 weeks up to 80 mg.

Results: Comparable levels of LDL-C were obtained when 10 mg of atorvastatin (206 ± 63 mg/dl) were administered as during treatment with 40 mg of simvastatin (212 ± 38 mg/dl). When 4 weeks on 80 mg of atorvastatin a further 26 % reduction in LDL-C values (157 ± 34 mg/dl) was obtained, and kinetic analysis demonstrated that LDL-C levels increased from a post-treatment value of 28.8 ± 14.2 mg/dl in a first order kinetic to 156.6 ± 25.5 mg/dl at day 7. When these kinetic results were compared to those without concomitant lipid lowering drug treatment patients presented LDL-C values below the recommended target range for an extended duration of almost 48 hours (until 72 hours after LDL-apheresis). The levels of HDL-cholesterol and plasma fibrinogen remained comparable during the entire study period. The change in lipid lowering drugs resulted in a 40 % reduction in LDL-apheresis treatments despite improved LDL-C levels prior to and following LDL-apheresis treatment.

Conclusion: Our results show that aggressive LDL-C reduction by atorvastatin combined with LDL-apheresis is a save and efficient choice in lipoprotein therapy in patients with heterozygous and even homozygous FH.

Right ventricular metastatic choriocarcinoma obstructing in- and outflow tract

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Objective: We wanted to demonstrate that fast diagnosis and immediate treatment of intracavitary myocardial neoplasms as soon as they present themselves with symptoms, is essential for accurate treatment of these patients.

Methods: Surgical procedure was performed on a 34 year old man in whom ECHO heart examination was revealing a metastatic tumour in the enlarged right ventricle (RV) obstructing the RV in- and outflow tract and extending from the tricuspid valve to the pulmonary valve, filling the entire ventricle and outflow tract. The tumour was removed on arrested heart, the method enabling preservation of the tricuspid valve and it's supporting structures. The mass could not be removed from the RV free wall, since the tumour invaded it completely and an attempt at removal would result in complete absence of contractile force of the RV. An additional chemotherapy was carried out with four cycles according to the BEPO scheme.

Results: The results of histologic investigation of removed tissue revealed a metastasis of choriocarcinoma with components of immature teratoma, originating from right testis.

Control CT of the thorax was performed after six months, detecting no metastases in the lung parenchyma. MRI of the heart, however, still showed a thickened RV free wall and apex, mass vascularity equalled that of the myocardium – compared to postoperative heart US recordings, there was no regression of the mass in the RV wall. The patient is doing well one year postoperatively.

Conclusions: The diagnostic procedures for detecting the tumours should be non-invasive (ultrasound, CT, MRI) since in our opinion invasive methods, such as ventriculography, do not give a better insight into the lesion, but may endanger the patient with the risk of distal embolization of the tumour. Immediate operation should be practised for accurate treatment of these patients.

Intraaortic balloon pump use in medical intensive care unit in medical center ljubljana. Results of 10 years of use

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Aim of the study: Aim of our study was to examine the use of an intraaortic balloon pump (IABP) in our ICU. We were interested in indications, complications and patients outcome.

Patients: From January 1, 1990 through December 31, 2000 we inserted IABP to 187 of our patients. We managed to retrieve 164 of their medical records. The average age of our patients was 64.1 years (21–83 years), 106 were males.

Results: From 1990 through 1996 there were 10 patients with IABP support per year on average. With the introduction of percutaneous coronary interventions in acute coronary syndromes in 1997 the number of patients with IABP support gradually rose up to 55 in 2000. Most of the patients required IABP support due to cardiogenic shock in the setting of acute myocardial infarction (AMI). Other indications were: unstable angina pectoris, mechanical complications and rhythm disturbances during AMI, PTCA complications and myocarditis. The average time of IABP support was 4.1 days (few hours to 15 days). Complications were rare (total 8 %; 7 % vascular, 1 % infectious). Outcome was as follows: successful weaning and discharge from ICU – 45 patients, surgery – 66 patients, death – 58 patients.

Conclusions: Use of IABP in our unit is increasing especially since introducing

percutaneous intervention techniques in acute coronary syndromes. The increase is also related to widening of indications for IABP support (bridging to surgery). Complications and outcome are comparable with results achieved in other centers.

Improvement of cardiac output in severe chronic heart failure by combining ACE-inhibitors and either eprosartan or telmisartan

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Background: The importance of ACE-inhibitors in the therapy of chronic heart failure is established. Angiotensin II receptor type 1 antagonists act according to a different pharmacological mechanism by additionally blocking the locally and not ACE-generated angiotensin II in the vessel wall and the myocardium. Furthermore, the AT-1-receptor antagonists block the prejunctional AT-1-receptors and reduce the sympathetic outflow. We examined the hypothesis that an additional effect on cardioprotection can be achieved by the combination of common ACE- and bradykininase-inhibitor action and the specific effects of sartans.

Methods: 51 patients (mean age 68.8 ± 7.8 years) with severe chronic heart failure receiving long-term medication with digitalis, diuretics, and ACE-inhibitors were randomised after clinical recompensation in a blind fashion to either Eprosartan (n = 17; 508.8 ± 118.0 mg/d) or placebo (n = 17). Additionally a prospective study by using Telmisartan (n = 17; 66.1 ± 15.4 mg) was performed. Haemodynamic measurements were made by impedance-cardiography at baseline and after 9.9 ± 2.3 days.

Results: Additional treatment with sartans resulted in an improvement of cardiac output. There was an increase in cardiac output from 2.38 ± 0.58 to 3.19 ± 1.0 l/min (p = 0.009) in the Eprosartan-group and from 2.24 ± 0.48 to 2.80 ± 0.75 l/min (p = 0.0013) in the Telmisartan-group while there was no increase of cardiac output in the control group.

Conclusions: The additional treatment of severe heart failure patients who received digitalis, diuretics, and ACE-inhibitors with the AT 1-receptor antagonist Eprosartan or Telmisartan shows a beneficial effect by increasing cardiac output. This effect may be due to the sartans additional property of blocking the autocrine interaction of locally and not ACE-generated angiotensin II with their respective vascular and myocardial AT 1-receptors as well as the influence on prejunctional AT 1-receptors located on sympathetic nerve terminals.

Different origin of elevated hepatocyte- and vascular endothelial growth factor serum levels in patients after myocardial infarction. Role of leukocyte activation

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Abstract withdrawn.

Association of coronary calcium with the GNB3 polymorphism in early coronary artery disease

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Abstract withdrawn.

Changes in myocardial electrical and mechanical function assessed by endocardial mapping after percutaneous transluminal coronary angioplasty

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Background: Nonfluoroscopic electroanatomical mapping displays simultaneously the myocardial viability and the regional mechanical function of the heart, which might be improved after angioplasty of the narrowed coronary artery.

Purpose: We compared the pre-percutaneous transluminal coronary angioplasty (PTCA) and follow-up (FUP) endocardial voltage and local linear shortening (LLS) values in order to investigate the ability of NOGA endocardial mapping for detection of hibernating myocardium.

Methods: The left ventricular unipolar voltage and LLS values were analysed in the first 10 patients (8 male, 61 ± 12 yrs, all with stable angina pectoris, three-vessel disease and previous myocardial infarction) before and 6 months after PTCA. The NOGA segmental quantitative analysis was performed after transformation of the 3-dimensional endocardial map into polar map, containing 12 segments (anterior, lateral, posterior and septum, each of them with apical, mid and basal subsegments). 201-Thallium rest and late-rest images were performed in all patients, and the quantitative 201-Thallium uptake data were analysed by polar map analysis by division into 12 comparable myocardial segments.

Results: On the basis of the PTCA localization, the myocardium was divided into “treated” and “untreated” regions. In the treated myocardial areas, the unipolar voltage values (from 7.71 ± 3.37 to 9.74 ± 4.50 mV, $p < 0.05$; normal value > 15 mV) and the LLS values (from 6.81 ± 5.70 % to 9.24 ± 6.12 %, $p < 0.05$, normal value > 11 %) increased significantly. In contrast, in the untreated

region, neither the unipolar voltage nor the LLS values changed. A trend to increase in 201-Thallium resting uptake (64.2 ± 14.8 % vs 69.3 ± 15.6 %, n.s.) and a significant improvement in 201-Thallium late resting uptake (index of myocardial viability) (55.4 ± 17.3 % vs 68.3 ± 16.9 %, $p < 0.05$) could be detected in the treated myocardial segments. The 201-Thallium uptake did not change in the non-treated areas.

Conclusion: Myocardial regions with parallel decreased endocardial voltage and LLS values of NOGA endocardial mapping might present hibernating myocardium, as a significant improvement of both the voltage and LLS values have been detected after restoration of myocardial blood flow.

Acute stent thrombosis after intracoronary implantation of RG-13577 coated stents in pigs

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Background: RG-13577 is a non-toxic synthetic heparin-mimicking polyanionic compound (polymer of 4-hydroxyphonoxy acetic acid) which has been shown to inhibit *in-vitro* vascular smooth muscle cell proliferation induced by basic fibroblast growth factor and to block cell division in the G1/S-phase transition. The aim of the present study was to reduce neointimal hyperplasia using RG-13577-coated stents after intracoronary stent implantation in pigs.

Methods: In general anaesthesia, the left coronary artery was cannulated in 14 domestic pigs. After administration of 200 IU/kg heparin, 8 pigs received bare (non-coated native) Genius stents (EuroCor GmbH, Bonn, Germany) either in the left anterior or left circumflex coronary arteries. After 4 weeks follow-up (250 mg acetylic salicylic acid administered daily *per os* to prevent stent thrombosis), repeat angiography and intravascular ultrasound (IVUS) using automatic pullback were performed. Six RG-13577-coated Genius stents were implanted in 6 more pigs in one left coronary artery branch.

Results: Five of 6 pigs receiving RG-13577-coated Genius stents died within 1 hour after stent implantation due to ventricular fibrillation. In 2 pigs, acute stent occlusion was documented angiographically *in-vivo*, while *post mortem* angiography proved acute stent thrombosis in the hearts of 2 other pigs. In one pig, no visual documentation of stent occlusion was done. Coronary angiography revealed also acute stent thrombosis in the 6th (survived) pig with RG-13577-coated Genius stent, which experienced a small myocardial infarction. In the other group (bare stent group), no acute or subacute complication occurred and all pigs survived the 4-week follow-up. Volume measurements by 3D-reconstruction of IVUS (EchoPlaque, Indec Systems, CA) revealed an intimal volume of 20.31 ± 5.76 mm^3 within the stents. IVUS showed a minimum lumen area of 3.48 ± 0.68 mm^2, a maximum vessel area of 8.50 ± 0.79 mm^2 and a maximal intimal area of 1.91 ± 0.53 mm^2.

Conclusion: Even though RG-13577 has heparin-like capabilities, intracoronary implantation of RG-13577 coated stents leads to acute in-stent thromboses in pigs. Bare Genius stent implantation shows favourable results.

External electrical cardioversion of persistent atrial fibrillation (AF)

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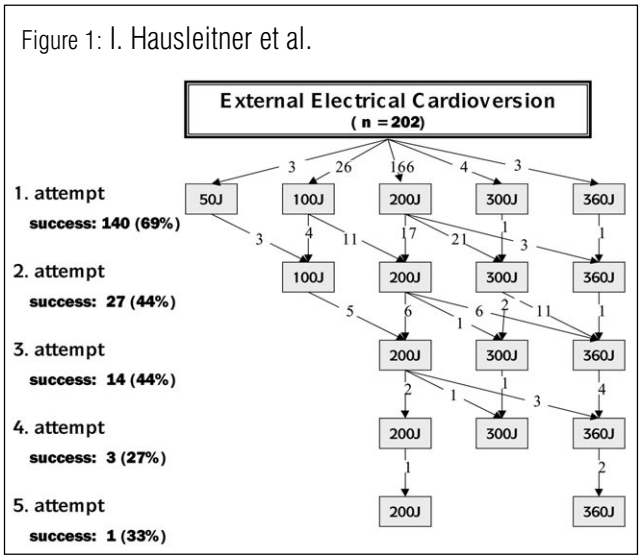
Background: Restoration of sinus rhythm (SR) is the primary goal in patients with persistent AF. Direct-current electrical cardioversion (DCCV) can be performed primary or after pharmacological failure.

Methods: Within 6 years DCCV was applied to 162 patients with 202 episodes of persistent AF. Response to electrical therapy was assessed.

Figure 1: I. Hausleitner et al.



Results: SR was restored by DCCV in 185 episodes of persistent AF (92 %). In 34 out of the 202 episodes preceding pharmacologic therapy had failed. Non-responders to electrical therapy had more often enlarged left atria. Choice of the initial energy level was free. 200 Joule were applied for first shock in 165 cases and succeeded in 75 % (124 episodes). Overall for successful termination a median of 1.4 shocks was necessary. No serious adverse events occurred during observation period prior to hospital discharge **(Figure 1).**

Conclusion: External electrical cardioversion is safe and effective for termination of persistent AF. As initial shock energy we recommend 200 J.

Prediction of outcome by neurohumoral activation, six minute walk test and the Minnesota living with heart failure questionnaire in an outpatient cohort with congestive heart failure

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Introduction: Neurohumoral activation is a well-known phenomenon in congestive heart failure. Due to the central mechanism it is a value tool as a prognostic marker. Nevertheless no data exist comparing the most powerful markers as N-ANP, BNP, N-BNP and big-endothelin 1 in one model in a widespread population of heart failure patients. The six-minute walk test is very controversial discussed in respect to its predictive power and the Minnesota living with heart failure questionnaire has never been evaluated, whether it is related to outcome.

Aim of the study: To compare these parameters in respect to short-term outcome in an ambulatory heart failure population.

Patients and methods: Ninety-six patients (57 ± 8 years, 82 % male, left ventricular ejection fraction of 26 ± 10 %) who were treated as out-patients in the heart failure unit were included. Within one day blood samples of N-ANP, N-BNP, BNP and big-endothelin 1 were obtained as well as six-minute walk test and Minnesota living with heart failure questionnaire were measured. The predictive power of these variables in respect to one-year event free survival was calculated by a Cox regression analysis.

Results: All variables investigated had the power to predict outcome in an univariate analysis. Multivariate analysis revealed that only N-ANP ($x^2 = 66$, $p < 0.0001$) beside Minnesota living with heart failure questionnaire ($x^2 = 11$, $p < 0.001$) and BNP ($x^2 = 7$, $p < 0.01$) are independent predictors. Kaplan-Meier analysis revealed a significant difference in outcome after three months and one year at a cutpoint of 5000 fmol/ml ($p < 0.0001$).

Conclusions: We conclude that in an open clinical cohort of patients with big differences in the progression of the disease. N-ANP is the most reliable predictor of worsening heart failure in short-term. Beside BNP, especially LHFQ is an excellent substitute in case of lacking of specialized laboratories.

Circulating levels of N-terminal atrial natriuretic peptides and N-terminal brain natriuretic peptide in acute and chronic coronary heart disease

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Objectives: The peptides derived from the atrial natriuretic peptide (ANP) prohormone, proANP (1–30), proANP (31–67) and the N-terminal proANP (1–98) hormone have natriuretic, diuretic as well as vasodilatatory effects and antagonize the renin-angiotensin-aldosterone system. The N-terminal brain natriuretic peptide (Nt-proBNP) shares the same cardioprotective properties. ProANP(1–98) and Nt-proBNP were found to be elevated after acute myocardial infarction. It is also known that ischaemia and hypoxia may enhance the secretion of the proANP (1–98) hormone by atrial cells. Up to date no data are available about the plasma levels of the different N-terminal ANPs in patients with stable and unstable angina.

Methods: Plasma levels of proANP (1–30), proANP (31–67), proANP (1–98) and Nt-proBNP hormones were determined in 115 patients with different clinical stages of coronary artery disease, according to the Canadian Cardiac Society classification (I–IV).

Results: ProANP (1–30) and proANP (31–67) levels were found to be higher in patients with stable angina (CCS I, II) and effort angina (CCS III) compared to patients with angina at rest (CCS IV, $p = 0.014$, $p = 0.02$ and $p = 0.003$, respectively). The levels of proANP (1–98) hormone were higher in the CCS III and IV groups compared to chronic stable patients (CCS I–II, $p = 0.006$). In contrast, Nt-proBNP levels were significantly lower in CCS III–IV than in CCS I–II ($p = 0.003$). Patients, with "b" and "c" angiographic lesion type in the culprit lesion have significantly higher levels of both proANP(1–98) and Nt-proBNP. This tendency was also found with mid-proBNP, but it did not reach statistical significance.

Conclusions: Acute ischaemia seems to have inhibiting effects on the cleavage of the proANP (1–98) hormone leading to decreased levels of the potentially cardioprotective proANP (1–30) and (31–67). In contrast to other studies, our results suggest, that in patients with effort angina and angina at rest, the Nt-proBNP and mid-proBNP levels are lower than in chronic stable patients.

The quality of the lesion type in the coronary vessel seems to affect the plasma levels of proANP (1–98) and Nt-proBNP hormones.

Plasma levels of cardiac troponin T, troponin I, creatinkinase MB fraction, C-reactive protein and fibrinogen correlate with TIMI risk score and long-term follow-up in patients with angina pectoris

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Background: The aim of the present prospective study on 155 consecutive patients (110 male, 63 ± 11 y) with typical chest pain was to measure fibrinogen, C-reactive protein (CRP) and cardiac troponin T (cTnT) at basal conditions, as well as troponin I (cTnI), myoglobin, and creatine phosphokinase myocardial fraction (CKMB) at baseline and after 6 hours in order to prove the accuracy of these parameters as markers of different risk in accordance with the TIMI risk score for subsequent major adverse cardiac events (MACE).

Methods: The TIMI risk score was calculated and the patients were classified into high (score 6–7, n = 27 patients, 17.4 %), medium (score 4–5, n = 64 patients, 41.3 %) and low (score 0–3, 64 patients, 41.3 %) risk groups.

Results: High risk patients experienced significantly more frequently acute myocardial infarction (22.2 vs 6.3 and 3.1 %, $p < 0.05$), revascularization (37 vs 10.9 and 10.9 %, $p < 0.005$), death (29.6 vs 10.9 and 1.6 %, $p < 0.05$) and composite MACE (63 vs 20.3 and 14.1 %, $p < 0.001$) than medium and low risk patients. Significantly elevated plasma levels were found for TnI, CRP, CKMB, cTnT and fibrinogen in patients of the high risk group as compared to patients with medium or low risk **(Table 3).** Plasma levels of myoglobin did not identify the patients with medium or high risk.

Receiver operator characteristic (ROC) analyses revealed the following cut-off points for high risk groups: for cTnI at baseline 0.3 U/l (predictive accuracy [p.a.] = 0.837), for cTnI after 6 h 0.64 U/l (p.a. = 0.908); for CRP at baseline 0.34 mg/dl (p.a. = 0.734), for CRP after 6 h 0.732 mg/dl

Table 3: N. Jordanova et al.

	High risk n = 27	Medium risk n = 64	Low risk n = 64	p-value
cTnI baseline [U/l]	5.57 ± 8.27	0.32 ± 0.61	0.16 ± 0.31	< 0.001
cTnI 6 h [U/l]	8.34 ± 9.32	0.78 ± 1.72	0.29 ± 0.63	< 0.001
CRP baseline [mg/dl]	1.45 ± 1.91	0.49 ± 1.00	0.81 ± 1.59	0.019
CRP 6 h [mg/dl]	1.95 ± 2.09	0.83 ± 1.34	0.62 ± 0.94	< 0.001
Myoglobin baseline [U/l]	46.7 ± 46.9	28.9 ± 26.2	34.6 ± 33.6	0.055
Myoglobin 6 h [U/l]	50.1 ± 42.5	40 ± 55.2	32.5 ± 27	0.127
CKMB baseline [U/l]	14.9 ± 20.7	2.9 ± 4.9	2.6 ± 2.6	< 0.001
CKMB 6 h [U/l]	9.8 ± 11.5	3.7 ± 5.4	4.5 ± 8.5	0.004
CTnT baseline [U/l]	0.57 ± 1.1	0.03 ± 0.16	0.02 ± 0.09	< 0.001
Fibrinogen [mg/ml]	385 ± 84	324 ± 63	322 ± 67	< 0.001

(p.a. = 0.784), for CKMB at baseline 2.25 U/l (p.a. = 0.761), for CKMB after 6 h 2.9 (p.a. = 0.742) and for fibrinogen at baseline 367 mg/ml (p.a. = 0.737).

Conclusion: In our patients with typical chest pain, the cut-off levels for determination of a high-risk group according to the TIMI risk score differ from those used in general and are either lower (CRP and CKMB at baseline and CKMB after 6 h) or higher (cTnI at baseline and after 6 h and CRP after 6 h) for the respective parameters.

The On-X® heart valve: echocardiographic characteristics

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Background: The On-X® is a new mechanical heart valve with a special valve design. An On-X® has a flared orifice inlet, an elongated orifice and thin leaflets that are positioned parallel when the valve is open. The position of the valve is supra-annulary.

Patients and Methods: 154 On-X® prostheses were implanted, 73 aortic and 81 mitral valves. Mean age was 61.3 ± 10.3 a (range 30–79) for implantation in mitral and 59.2 ± 10.1 a (range 25–79) for aortic position. Echocardiograms were performed including complete Doppler examination at discharge, 3 to 6 months after implantation and after one year. Appearance of the valve in echocardiography was observed.

Results: The On-X® showed a better haemodynamic as compared to Carbomedics® and Carbomedics SAV® heart valves. In both positions movements of the leaflets could just poorly be seen. In contrast to all other prostheses only a very minimal closing volume was found. Closing volumes were more often detected in aortic positioned valves as in mitral positioned valves. The size of this closing volume did not correlate with valve size **(Table 4).**

Conclusion: This new valve has superior haemodynamics and demonstrates significant improvement in regard to closing volumes. Due to design features the leaflets can be seen poorly on echo.

Platelet-monocyte cross-talk and tissue factor expression in stable and unstable angina

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Background: Tissue factor (TF) is the major procoagulant *in-vivo* and usually absent from blood cells. However, monocyte tissue factor (TF) expression and platelet activation are both features of unstable angina.

Methods and Results: Monocyte-TF and procoagulant activity (PCA), monocyte platelet load (CD42+) and adherent platelet activation were determined by flow cytometry and chromogenic assay from 57 coronary artery disease patients with stable (SA, n = 42) and unstable angina (UA, n = 15). Monocyte TF levels and PCA were significantly higher in UA (16.4 ± 3.3 MFI, 68.5 ± 38.2 units) compared with SA (14.1 ± 3.1 MFI, p = 0.03; 47.9 ± 18.1 units; p = 0.04). Both platelet load (r = 0.69, p < 0.0001) and platelet activation (r = 0.47; p = 0.002) as determined by CD42 and CD62P expression on CD14+ monocytes positively correlated with monocyte-TF expression. TF messenger RNA was expressed in 26 % of SA and in 37.5 % of UA.

Conclusions: We show elevated monocyte-TF expression and TF-dependent PCA in UA compared with SA patients. Monocyte-TF activity correlating with platelet load and platelet activation may be triggered by platelet leucocyte cross-talk *in-vivo*.

Relationship between blood pressure, lipids and therapy in patients in secondary prevention of IHD

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Background: Hypertension is often associated with hyperlipidaemia increasing the global atherosclerotic risk in a multiplicative way. The effect of the therapy in secondary prevention has not been studied at Slovakia. This study evaluates occurrence of hypertension and effect of the hypotensive therapy in relationship to lipid profile in high risk patients with symptomatic IHD.

Table 4: M. T. Kasimir et al. Data of last timepoint of examination

On-X® aortic valve				On-X® mitral valve		
Diameter	**n**	**Mean gradient**	**Velocity**	**Diameter**	**n**	**MVA**
19 mm	5	19.85 mmHg	2.4 m/s	23 mm	1	
21 mm	22	11.5 mmHg	2.1 m/s	25 mm	24	2.9 cm²
23 mm	22	11.1 mmHg	2.08 m/s	27 mm	41	2.6 cm²
25 mm	17	9.8 mmHg	1.95 m/s	29 mm	2	
27 mm	7	9.8 mmHg	2.0 m/s	31 mm	17	2.8 cm²

Table 5: J. Lietava et al.

	Mono [%]	Two drugs [%]	Three drugs [%]	≥ Four drugs [%]	Together [%]
SBP > 139 mmHg	66.5	73.2	79.4	90.9	64.2
DBP > 89 mmHg	44.9	53.4	54.4	63.6	35.8
SBP + DBP normal	29.5	23.8	15.7	9.1	26.6
	1556	895	159	12	2622

Patients and method: In a cross-sectional multicentre trial 5062 patients with symptomatic IHD were examined and hyperlipidaemia (total cholesterol > 5.2 mmol/l) screened for statin therapy (2237 M/2825 F). Both sexes were in middle age (56.6 ± 10.37 vs 60.1 ± 9.51 yrs; $p < 0.001$), mildly obese (BMI 28.3 ± 3.96 vs 28.3 ± 4.30 kg/m^2; NS) with mild hypertension (SBP 139.7 ± 18.28 vs 144.6 ± 18.47 mmHg; $p < 0.001$) (DBP 85.5 ± 9.30 vs 86.1 ± 9.36 mmHg; $p < 0.001$). Males suffered more MIs (36.0 % vs 10.5 %; $p < 0.001$) and were more often smokers (16.4 % vs 4.9 %; 0.001). Diabetes mellitus was similarly distributed in both sexes (26.7 % vs 26.9 %; NS).

Results: Out of 5062 patients with symptomatic IHD 2622 (51.8 %) were treated hypertensives. **Table 5** compiles percentages of hypertensives with controlled blood pressure during mono- and combined therapy.

Conclusion: Patients with symptomatic IHD and hyperlipidaemia exhibit 51.8 % occurrence of treated hypertension, which is controlled in 26.6 % of them. Paradoxically, with increasing complexity of the therapy decreases its effectiveness, probably conditioned by increased severity of hypertension.

Doppler gradients across Sorin tilting disc valves accurately reflect catheter gradients in-vitro

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Background: Significant discrepancies between Doppler and catheter gradients have been reported for bileaflet prosthetic heart valves. They are caused by localized gradients and pressure recovery. The occurrence of this phenomenon appears to be independent of the particular design of bileaflet valves. Tilting disc valves have been insufficiently studied so far. While good agreement between Doppler and catheter gradients was found for Medtronic Hall valves, discrepancies have been reported for Björk-Shiley valves.

Methods: In a well-controlled pulsatile flow model we studied various sizes of Sorin tilting disc prostheses (19 to 25 mm) using continuous-wave and pulsed-wave Doppler as well as direct measurements of pressure and flow rate. At each of 8 different flow rates (cardiac output 2.0 to 8.0 l/min) peak and mean Doppler and catheter gradients were simultaneously measured. Valve areas were calculated using the continuity equation and the Gorlin formula.

Results: Excellent correlation and agreement between Doppler and catheter gradients were found (r = .95–.99 for peak gradients and r = .98–.99 for mean gradients). For small valve sizes (19 and 21 mm) the mean difference between Doppler and catheter gradients was 4.2 ± 3.3 mmHg for peak and 0.5 ± 1.25 mmHg for mean gradients, respectively. Orifice areas as calculated by the Doppler continuity equation did not show flow dependent changes. In contrast, orifice areas calculated by the Gorlin formula increased slightly by 10 to 15 % in small valves.

Conclusion: Peak and mean Doppler gradients across Sorin tilting disc valves accurately reflect the pressure drop as determined by catheter measurements. Prosthetic valve areas calculated with the Gorlin formula increase with increasing flow in small valve sizes whereas no flow dependent changes were observed in continuity equation valve areas.

Polymorphic membrane proteins (PMPs) of *Chlamydia pneumoniae* induce the chemokines interleukin-8 (IL-8) and macrophage chemotactic protein-1 (MCP-1) and the pro-inflammatory cytokine interleukin-6 (IL-6) in human umbilical vein endothelial cells (HUVECs) in-vitro

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Background: IL-6, IL-8 and MCP-1 are present in atherosclerotic lesions of the vessel wall. Based on clinical studies a role for *C. pneumoniae* in the development of atherosclerosis is currently discussed. Recent *in-vitro* studies have shown that *C. pneumoniae* can activate cultured endothelial cells thereby inducing among other effects also IL-6, IL-8 and MCP-1 expression in these cells.

Aim: It was the aim of the present study to investigate whether purified isolated components of *C. pneumoniae*, namely polymorphic membrane proteins (PMPs) could affect production of IL-6, IL-8 and MCP-1 in human endothelial cells *in-vitro.*

Methods: HUVECs were incubated with purified PMPs. In order to exclude possible LPS-mediated effects aliquots of the respective PMPs were boiled for 5 minutes prior to addition to HUVECs. Plasminogen activator inhibitor-1 (PAI-1), IL-6, IL-8 and MCP-1 were quantified by specific ELISAs.

Results:
1. Out of 15 PMPs tested PMP 20 and PMP 21 were the strongest inducers of IL-6, IL-8 and MCP-1 production in HUVECs. The effects were dose and time dependent.

2. The effect of PMP 20 and PMP 21 on IL-6, IL-8 and MCP-1 was abolished by heat-treatment.

3. None of the PMPs tested had an effect on PAI-1 production in HUVECs.

Conclusions: We conclude from our data that specific PMPs of *C. pneumoniae* induce the expression of pro-inflammatory cytokines in HUVECs *in-vitro*. If such a mechanism is also operative *in-vivo*, *C. pneumoniae* could by specific interactions of its PMPs with the endothelium contribute to the process of vascular injury during the development of an atherosclerotic lesion.

Seldom fibrotic band in the aortic arch as a cause of an innocent murmur

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Background: Children with innocent murmurs are often referred to a paediatric cardiologist for diagnosis. The most common murmurs of early childhood are the so called Still's murmurs followed by pulmonary or aortic ejection murmur and venous hum. There also exists a high coincidence with false tendinous structures within the left ventricle. We found another seldom reason for an innocent murmur.

Methods: We report 6 patients (age 5 to 22 years) presented to our outpatient pediatric cardiology clinic for workup of heart murmur. None of them had abnormalities on clinical exam, ECG or echocardiography. They presented a similar murmur that in contrast to innocent murmurs was also audible over the back. The echocardiography was done on two different echo-machines with cross-sectional and colour-Doppler and the string was reproducible from every examiner.

Results: With closer examination of the aorta with cross-sectional echo we revealed an echogenic, tendinous structure in all 6 patients crossing either the lumen of the descending aorta or aortic arch as another cause for innocent heart murmur. The characterization was made by auscultation, but localization and proof was made by echocardiography of existing intraaortic structures.

Conclusions: We suggest that if intraventricular echo is normal, and the murmur is not consistent with a Still's murmur or aortic/pulmonic ejection murmur, that echo exam should include a thorough evaluation for seldom tendinous structures in the aortic arch. More invasive diagnostic testing does not appear to be indicated, if the diagnosis is made by an experienced cardiologist.

Electrophysiologically guided risk stratification and ICD therapy in patients with unexplained syncope, no documented ventricular arrhythmias and structural heart disease

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Aims: To evaluate electrophysiologically guided implantable cardioverter defibrillator (ICD) therapy in patients with syncope, no documented ventricular tachycardia (VT) and structural heart disease.

Methods and Results: Programmed ventricular stimulation (PVS) was performed in 52 patients for risk stratification. The mean age was 62 ± 10 years; 40 patients had ischaemic and 12 patients had idiopathic cardiomyopathy. On PVS sustained VTs and ventricular fibrillation were induced in 7 and 4 patients, respectively. Two non-inducible patients spontaneously experienced symptomatic ventricular tachyarrhythmias shortly after PVS in hospital. These patients received an ICD (ICD group, n = 13). The remaining non-inducible patients were left on best conventional therapy (non ICD group, n = 39). During a follow-up period of 4.6 ± 2.7 years, 5 ICD patients received appropriate therapies. One and 6 patients died in the ICD and non-ICD group, respectively, and all deaths were non-sudden. Overall survival analysis revealed no significant difference. The positive and negative predictive values of PVS for tachyarrhythmias or sudden death were 36 % and 95 %, respectively.

Conclusions: In patients with structural heart disease, unexplained syncope and no documented sustained ventricular arrhythmias PVS reliably identifies patients at high risk for life-threatening tachyarrhythmias. Patients with severely depressed left ventricular function and bad clinical condition appear to benefit most. In this patient population primary ICD implantation appears the treatment of choice.

Mild and moderate aortic stenosis: a serious disease?

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Background: Rapid progression from mild and moderate aortic stenosis to severe disease has been observed. However, the natural history of this disease remains insufficiently determined.

Methods: Therefore, we prospectively followed 176 consecutive patients (73 female, age 58 ± 19 yrs) with mild to moderate aortic stenosis (jet velocity 2.5–4.0 m/s) from 1994 to 1999. The rate of progression of the peak aortic jet velocity was calculated, and a survival analysis was performed.

Results: Mean follow-up was 3.9 ± 1.6 yrs. Kaplan-Meier event-free survival for the entire patient group, with endpoints defined as death (n = 34) or aortic valve surgery (n = 33), was 95 ± 2 % at 1 yr, 75 ± 3 % at 3 yrs, and 60 ± 4 % at 5 yrs. Among 18 cardiac deaths, 6 were sudden, 1 patient had endocarditis and 11 congestive heart failure. Mean rate of progression of peak aortic jet velocity for the entire population was 0.24 ± 0.3 m/s/yr, but varied widely (–0.2 to 1.3 m/s/yr). It was significantly faster for patients with an endpoint (0.43 ± 0.04 m/s/yr), than for those without event (0.14 ± 0.02 m/s/yr; $p < 0.0001$). Patients younger than 50 years had a significantly better outcome with event-free survival rates of 100 % at 1 yr, 95 ± 3 % at 3 yrs, and 89 ± 5 % at 5 yrs compared with 93 ± 2 %, 68 ± 4 %, and 49 ± 5 % for patients older than 50 years ($p < 0.0001$). Peak aortic jet velocity at entry was also a significant predictor of outcome.

Of 129 patients with a follow-up echocardiographic exam, 59 (46 %) reached a peak aortic jet velocity of 4 m/s or greater during follow-up.

Conclusion: Rapid progression from mild or moderate to haemodynamically severe aortic stenosis is common. In particular, patients older than 50 years and patients in whom echocardiographic follow-up reveals a rapid increase of peak aortic jet velocity have a poor outcome. Thus, moderate and even mild aortic stenosis cannot be considered a benign disease and rapid progression is frequent.

The angiotensin-converting enzyme inhibitor lisinopril lowers plasma levels of C-reactive protein after myocardial infarction more efficiently than angiotensin II type 1 receptor antagonists

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Background: Recent studies have shown that atherosclerosis is prevented in animal models and humans by blockage of the renin-angiotensin system via angiotensin converting enzyme inhibitors (ACEI) or antagonists of the angiotensin II type 1 receptor (AT_1-antagonists), possibly by an antiinflammatory mechanism. We compared the antiinflammatory effects of ACEI and AT_1-antagonists after acute myocardial infarction (AMI) by measurement of high sensitivity C-reactive protein (CRP), a sensitive marker for ongoing inflammation and strong predictor of cardiovascular risk.

Methods: 63 patients with their first AMI and after fibrinolytic therapy were included in our study and randomized in a 2:1 ratio to receive either lisinopril 5 mg/d (n = 42) or an AT_1-antagonist (losartan 50 mg/d, n = 11; valsartan 80 mg/d, n = 10) starting 24 hours after the acute event. After 6 weeks of treatment the initial dose of lisinopril was increased during two weeks in steps of 5 mg to 40 mg/d whereas the initial dose of the AT_1-antagonists was not changed. Blood samplings were obtained after 10 days, 6 weeks and 6 months of treatment.

Results: At 6 weeks, CRP-levels decreased in both groups (lisinopril: $p < 0.001$, AT_1-antagonists: $p < 0.05$). A further decrease up to 6 months was only observed in the lisinopril group ($p < 0.05$) while no change was seen in the AT_1-antagonist group. After six months, median CRP-plasma levels in patients receiving lisinopril were 67.6 % lower than in patients receiving AT_1-antagonists (0.12 mg/dl vs 0.37 mg/dl, $p < 0.05$).

Conclusion: The ACEI lisinopril lowered plasma levels of C-reactive protein more efficiently than the AT_1-antagonists losartan and valsartan. ACE inhibition and the stabilization of bradykinin and subsequent increase in the production of the antioxidant nitric oxide (NO) may be responsible for the stronger antiinflammatory effect of high dose lisinopril. Whether an increase of dosage of AT_1-antagonists might have similar antiinflammatory effects via other mechanisms, eg increased NO production due to stronger stimulation of AT_2 receptors remains to be proven.

Intracoronary administration of a caspase-1-inhibitor (Ac-YVAD-cmk) reduces neointimal hyperplasia after stenting of porcine coronary arteries

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Background: The irreversible caspase-1 inhibitor Ac-YVAD-cmk inhibits apoptosis and proinflammatory cytokine release. In contrast to apoptosis inhibition, the chloromethylketone chain of Ac-YVAD-cmk increases smooth muscle cell apoptosis through elastase inhibition. The aim of the present study was to reduce neointimal proliferation after coronary stent implantation using Ac-YVAD-cmk.

Methods: After general anaesthesia, domestic pigs received intracoronary infusion of 50 mg Ac-YVAD-cmk (dissolved in 1 ml dimethylsulfoxid [DMSO] and 59 ml phosphate buffer [PBS], infusion rate 6 ml/min) into the left coronary artery (group 1, n =10), while 8 animals served as untreated controls (group 2) and 6 further pigs as vehicle controls (DMSO + PBS) (group 3). One coronary stent was then implanted in the left anterior descending or circumflex coronary artery. After 4 weeks, control angiography and intravascular ultrasound (IVUS) were performed. IVUS parameters were measured using a computer-assisted 3D analysis system.

Results: Ac-YVAD-cmk reduced in-stent neointimal volume (29.1 ± 12.5 vs 80.5 ± 24.9 and 67.4 ± 22.3 mm^3, $p < 0.005$) and maximal area stenosis (38.6 ± 8.0 vs 71.0 ± 6.4 and 67.0 ± 9.9 %, $p < 0.001$) assessed by IVUS in group 1 vs groups 2 and 3, but did not influence vessel remodeling (maximal vessel area 12.4 ± 3.3 vs 13.5 ± 2.1 and 12.3 ± 2.7 mm^3, non-significant). Smaller maximal neointimal thickness (0.38 ± 0.19 vs 0.94 ± 0.37 and 0.97 ± 0.44 mm, $p < 0.01$) and decreased maximal neointimal area (1.73 ± 1.53 vs 3.66 ± 1.54 and 4.03 ± 0.86 mm^2, $p < 0.01$) assessed by histology (computerized planimetry) were found in pigs in group 1 vs groups 2 and 3. IVUS results (maximal neointimal area and neointimal thickness) correlated significantly with histological data ($r = 0.774$, $p < 0.001$ and $r = 0.699$, $p < 0.001$, respectively). Injury score did not differ significantly between the groups.

Conclusion: Our data indicate that the caspase-1 inhibitor Ac-YVAD-cmk reduces in-stent neointimal proliferation. IVUS allows a quantitative 3D analysis of experimental neointimal proliferation and vessel remodeling and is able to guide targeted histological analysis.

Arterial compliance: a predictor of cardiovascular diseases?

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Background: Measures of the arterial compliance can be derived non-invasively from pressure pulse contour analysis of

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