difference in Ach-induced significant CAS and associated parameters when compared with those of patients without documented anemia.

Table. Acetylcholine Provocation Test and Associated Parameters

Variables, n (%)	Anemia group (n=62)	Non-Anemia group (n=388)	P Value
Hb (g/dL)	10.9±0.69	15.7±20.64	0.28
Spasm at baseline angiography	16 (34.8)	171 (44.1)	0.24
Ach Provocation (+)	21 (50.0)	191 (50.8)	1.00
ST change	2 (4.8)	8 (2.1)	0.64
Chest pain	12 (26.1)	136 (35.1)	0.05*
Myocardial Bridge	7 (15.2)	64 (16.5)	1.00
(+) Provocation to Ach dose			
A1 (20μg)	0 (0.0)	14 (3.6)	0.38
A2 (50μg)	7 (16.7)	93 (24.7)	0.26
A3 (100μg)	35 (83.3)	270 (71.6)	0.14
Spasm after Ach injection			
Focal	5 (23.8)	38 (19.8)	0.77
Diffuse	16 (34.8)	154 (39.7)	0.53
Severe spasm (>70%)	9 (19.6)	92 (23.7)	0.50

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Impact of Trimetazidine on Drug-Eluting Stent-Associated Acetylcholine-Induced Coronary Artery Spasm and 12-Month Clinical Outcomes

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Introduction: Trimetazidine (Vastinan®) has been shown to have anti-ischemic properties. However, there is no previous data regarding its impact impact on drug-eluting stent (DES) associated coronary artery spasm (CAS). We investigated the impact of Trimetazidine on acetylcholine (Ach)-induced coronary artery spasm (CAS) and 12-month clinical outcomes in patients (pts) underwent percutaneous coronary intervention (PCI) with DES. **Methods:** Among 1862 consecutive pts treated with DES, 1010 pts underwent repeat angiogram within 12 months either because of routine follow up or recurrent chest pain suspicious of DES associated spasm. Among them, 160 pts underwent the acetylcholine (Ach) provocation test by injecting incremental doses of 20, 50, 100 ug into the left coronary artery between March 2004 and April 2009. The Ach provocation test results, its associated parameters, and 12-month clinical outcomes of pts treated with DESs were compared between the trimetazidine group (n=66) and non- trimetazidine group (n=94). **Results:** The baseline characteristics were similar between the two groups. All the pts who received the trimetazidine as an angina medication after the stenting, Vastinan SR® 35mg bid daily were administered for more than 6 months. The rate of positive Ach provocation test results after the stenting and CAS associated parameters were not different between the two groups. Further, at 12 months, overall clinical outcomes including the rate of death, myocardial infarction (MI), repeat PCI, major adverse cardiac events (MACE) were similar between the two groups. (Table1) **Conclusion:** In our study, trimetazidine administration after the DES implantation did not exert any impact on the Ach provocation test results and at least one-year major clinical outcomes.

Table 1. Clinical Outcomes at 12 months

Variables, n (%)	rimetazidine (n=66 pts)	Non-rimetazidine (n=94 pts)	P Value
Total death	0(0.0)	1(1.1)	1.00
Cardiac death	0(0.0)	1 (1.1)	1.00
MI	0 (0.0)	0 (0.0)	0.40
TVR	2 (3.0)	4 (4.3)	0.82
Total MACE	2 (3.0)	5 (5.3)	0.70
Stenting of de novo lesion	4 (6.1)	5 (5.4)	0.26
Spasm FU d/t recurrent chest pain	8 (12.1)	4 (4.3)	0.07*

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Endothelial Function After VEGF 165 Gene Therapy in End-Stage Ischemic Cardiac Disease: Preliminary Results

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Introduction: Endothelium is the main regulator of vascular wall homeostasis and maintain a relaxed vascular tone mostly by releasing mediators including nitric oxide (NO). Cardiac patients experience endothelial dysfunction manifested by abnormalities in vasoreactivity and inflammatory process. VEGF stimulates production of NO by enzyme nitric oxide synthase (eNOS) and induces proliferation and migration of endothelial progenitors cells (EPC) optimizing angiogenesis process. This study aimed assess endothelial function by flow mediated-dilation (FMD) technique in patients with end-stage ischemic cardiac disease undergoing direct myocardial injections of VEGF 165 plasmid. **Methods:** Clinical trial by temporal series. Were included 11 patients (10 men). Inclusion criteria: age less 75 years, left ventricular ejection fraction (LVEF)>25%, presence of symptoms of angina and/or heart failure, despite treatment optimized and absence of clinical diagnosis of tumors. VEGF 165 plasmid DNA (2000μg) was administered by direct myocardial injections by infra-mammary left thoracotomy. Vascular function was assessed by FMD technique before intervention, 10 and 30 days after procedure. Data were expressed as percentage of the baseline diameter after reactive hiperemia (%FMD) and sublingual nitroglycerine spray (%NTG). Data are presented as mean±SD, statistical analysis were performed by Friedman test and statistical significance was set at $p<0.05$. **Results:** Baseline characteristics: age 58.6±6.2 yr; LVEF 61.1±5.9%; angina class (CCSA) 2 (n=1), 3 (n=5) and 4 (n=3); heart failure class (NYHA) III (n=7) and IV (n=2).%FMD was: baseline (6.9±5.5%), 10 days (8.9±3.4%) and 30 days (9.9±6.7%); p=0.76.%NTG was: baseline (5.9±4.3%), 10 days (8.9±6.4%) and 30 days (8.3±4.3%); p=0.18. **Conclusion:** Our data demonstrate that VEGF 165 gene therapy was unable to improve endothelial function after one month.

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Is Cold Pressor Test Useful to Predict Cardiovascular Events in Patients with and without Known History of Coronary Artery Disease?

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Background: Endothelial dysfunction is not only the first known step in coronary artery disease (CAD) but also it can predict future cardiovascular events (CVE). Coronary arterial dysfunction can be tested with multiple methods, one of them is myocardial perfusion with single positron emission tomography (SPECT) with the cold pressor test (CPT). **Objective:** To determine the prevalence of CPT, in 803 consecutive patients (P), with and without known history of CAD, and analyze the incidence of CVE, in a follow up of 66 months. **Material and Methods:** 803 consecutive P that were tested in a Nuclear Cardiology Center with a normal exercise SPECT were included. We divided our P in two groups, Group I: 714 P, without history of known CAD, and Group II: 89 P with coronary artery revascularization (PTCA or CABG). With a time window of 2 to 5 days, a CPT was done. A positive (CPT) was defined as a new perfusion defect compared with the baseline SPECT and a negative one as no perfusion defects. The mean follow up was 30±11 months, having reached 95.5% of the P. The CVE we analyzed were: cardiac death, myocardial infarction, stroke and revascularization after an acute coronary syndrome. The mean age was 59±10 years, with a male prevalence of 56%. The risk factors of our P were: 12% diabetes, 55% family history of CAD, 74% dyslipidemia, 64% hypertension, 23% obese (BMI>30) and 20%¬ current smokers. **Results:** 41% of the P had a positive CPT. (Group I: 39%–Group II: 56%. P 0.003). We suspended the CPT in 7% of the P due to cold intolerance or vasovagal reaction. In Group II, we observed a higher prevalence of males, dislipedemia and positive CPT. There were 20 CVE during follow up, mainly in Group II (P 0,04). During the 66 month follow up, in Grupo I the survival free of events was 97% in the negative CPT and 91% in the positive CPT (P<0,007), and in Group II it was 85,7% and 83.6% respectively (P 0,59). **Conclusions:** 1. The CPT could predict CVE in P without known CAD. 2. In P with known history of CAD, although they had more CVE during follow up, the CPT wasn't able to predict them.

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Peripheral Artery Stiffening: A Pattern of Many Cardiovascular Disorders

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Introduction: We aimed to study the peripheral arteries elasticity indices in patients with diverse cardiovascular diseases. **Material and Methods:** The elasticity indices of the big, conduit (C1) and the small, resistive (C2) arteries as well as the vascular peripheral resistance (VPR) were assayed in patients with ischemic heart failure, arterial hypertension, pulmonary arterial hypertension, dislipidemias and deep venous thrombosis using the test method HLI/Pulse WaveTM CR-200. **Results:** The common event for all these disorders was the C2 index decrease by 42–55% from normal related to age and gender index values. This decline significantly correlated with NYHA class, duration of systemic or pulmonary hypertension, levels of the circulating LDL-cholesterol, TNF-alpha, IL-6, IL-1, RCP and AGEs (Advanced Glycated End products). The C1 index diminution was weaker, its significant reducing being evaluated especially in severe disorders (e.g. NYHA III-IV, pulmonary arterial hypertension more than 30 mm Hg). The VPR increased always when C2 index was decreasing and a strong correlation between them (r=-0,75–−0,79, p<0,01) has been established. Neurohumoral modulating therapy (e.g. ACE inhibitors and beta-adrenoblockers) markedly blunted the C2 index lowering. **Conclusions:** i) the peripheral arterial stiffening appears to be a predictor of many cardiovascular disorders, the elasticity loss being especially attributed to small arteries that results in the peripheral vascular resistance rise compromising the vessel-heart continuum; ii) the artery elasticity indices assessment can be a useful tool of the diagnosis and prognosis of the circulatory mismatch.

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The Effect of Adenovirus-Mediated Local Tissue Factor Pathway Inhibitor Gene Transfer on Thrombosis Formation in Rabbit Carotid Arteries

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Introduction: Thrombosis formation is the main cause of the acute coronary syndrome, which will even lead to sudden death. Tissue factor pathway inhibitor (TFPI) is an inhibitor of coagulation facor. **Aims:** To observe the effects TFPI on thrombosis formation in rabbit carotid