Coronary artery stenosis quantification with 256-slice computed tomography

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Background: Multidetector row computed tomography (MDCT) allows noninvasive visualization of the coronary arteries. We sought to determine the accuracy of 256-slice coronary CT angiography (256-CCTA) stenosis quantification compared to the gold standard invasive quantitative coronary angiography (QCA). In addition, we investigated the reproducibility of 256-CCTA plaque characterization.
Method: A total of 71 atherosclerotic lesions (in 32 patients) were analyzed with CCTA and QCA. Cross-sectional images, perpendicular to the axis of the coronary artery, were created by multi-planar reformation, on which we measured the minimum lumen area (MLA), the reference diameter (RD) and the length of the atherosclerotic plaque. We calculated cross-sectional area (CSA) stenosis, the sensitivity, specificity and accuracy of 256-CCTA to identify lesions with 50% and 70% luminal narrowing as compared to QCA. The reproducibility of plaque characterization was examined by calculating interobserver agreement and kappa value.
Results: Mean MLA was 1.7 mm^2 (IQR: 1.1 to 2.6) measured with CCTA; 1.6 mm^2 (IQR: 0.6 to 2.5) with QCA. A strong correlation was observed between the CCTA and QCA MLA values ($\rho = 0.69$, $p < 0.0001$). In addition, strong correlation was detected between CCTA and QCA regarding CSA values ($\rho = 0.73$, $p < 0.0001$). The CCTA slightly underestimated the area stenosis (-0.3 mm^2; -20.9%). Regarding the plaque length we found a significant difference between the CCTA (18.1 mm, IQR: 10.7 to 26.9) and the QCA (12.1 mm, IQR: 9.9 to 18.0) measurements ($p = 0.0006$). The 256-CCTA showed high sensitivity, moderate specificity and high accuracy (>50% and >70% stenosis; sens.: 95.5% and 84.1%: spec.: 66.7% and 63.0%: accuracy: 93.0% and 76.1%, respectively). Among 71 plaques, 27 calcified, 31 mixed and 13 non-calcified were identified. The inter-reader agreement was 82% (kappa = 0.68).
***Summary:* For stenosis quantification and plaque analysis 256-CCTA proved to be a reliable tool. A strong correlation was detected between the 256-CCTA and QCA measurements regarding the percentage of CSA stenosis. The plaque length was significantly higher according to 256-CCTA as compared to the QCA measurements. The plaque characterization showed a good reproducibility.**
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Does increased aortic stiffness predict reduced coronary flow velocity reserve in patients with suspected coronary artery disease?

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Introduction: In recent studies, reduction in coronary flow velocity reserve (CFR) has been demonstrated in patients with increased aortic stiffness. Stress transoesophageal echocardiography (TEE) has been found to be a suitable method for the simultaneous evaluation of CFR and aortic stiffness parameters. The present study was designed to test whether increased echocardiography-derived aortic elastic modulus [E(p)] predicts impaired CFR in patients with suspected coronary artery disease (CAD).
Methods: The present study comprised 158 patients with suspected CAD. Stress TEE was used in all cases to measure CFR and E(p). CFR was calculated as the ratio of posthyperaemic to basal peak diastolic coronary flow velocities. E(p) was calculated by using the following formula: SBP-DBP/[(DS-DD)/DD], where SBP and DBP are the systolic and diastolic blood pressures, and DS and DD are the systolic and diastolic aortic diameters. A CFR value <2 was considered abnormal.
Results: Patients with CFR <2 had higher resting and lower posthyperaemic diastolic coronary flow velocities. Both mean aortic atherosclerosis grade (as a morphologic characteristic) (1.31 ± 0.68 vs. 1.02 ± 0.89, $p < 0.05$) and aortic distensibility (E(p) as a functional characteristic) (892 ± 584 mm Hg vs. 723 ± 495 mm Hg, $p < 0.05$) were increased in subjects with CFR <2. In ROC analysis, the cut-off value for E(p) to predict impaired CFR was ≥670 mm Hg, with 61% sensitivity and 61% specificity (ROC area 0.60, $p = 0.026$). The logistic regression model identified higher grade of aortic atherosclerosis (hazard ratio (HR) 2.01, $p < 0.05$) and increased E(p) as independent predictors of reduced CFR (HR 1.10, $p < 0.05$).
***Conclusion:* Increased aortic stiffness predicts reduced CFR in patients with suspected CAD.**
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Stenting as an effective treatment of superior vena cava syndrome: review of 280 cases – single centre experience

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Purpose: VCSS constitutes a severe clinical manifestation of central venous outflow obstruction mostly caused by external compression by malignancies or intraluminal thrombus formation due to indwelling medical devices. This potentially fatal clinical entity with increasing prevalence requires prompt effective solution restoring upper body venous outflow in order to relieve symptoms and enable diagnostic and therapeutical procedures ameliorating and prolonging patients' life. VCS stenting represents an effective, low-morbidity and low-mortality alternative to insufficient medical treatment, irradiation, chemotherapy or high-risk open surgery. We describe and evaluate its technical success, clinical effectivity, primary and secondary patency and reasoning in reinterventions in a large group of patients.
Material and Methods: 280 patients (age range 23-87 years) underwent endovascular treatment for SVCS in our center between November 2002 and December 2011 (malignant etiology in 260 cases, benign in 20). SVC was primarily stented in 263, balloon dilated in 2, interventional attempt was unsuccessful in 15 cases. Patients were invited back immediately upon recurrence of symptoms.
Results: All successful interventions lead to immediate relief of symptoms. No peri-procedural mortality occurred. Complications included 2 cases of SVC rupture, 1 stent fracture, 3 cases of hemopericardium, 1 pericardial puncture, 3 cases of stent migration into atrium, 1 stent migration into ventricle. All complications were solved conservatively or percutaneously. Reocclusion occurred in 12 cases (including 5 times in a single patient), 2 days-2.5 months after the procedure. Restenosis was reported in 10 cases (including 5 times in two patient), 2 weeks-9 months following the procedure). Reinterventions included selective thrombolysis in case of reoclussion, re-PTA in restenosis and another stent placement.
***Conclusion:* Stenting has in our center proved to be a highly technically successful, clinically effective, low-morbidity, low-mortality, low-complication, low-restenosis rate treatment option for VCSS.**
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Detailed hemodynamic characterization of athlete's heart using left ventricular pressure-volume analysis in a rat model

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The development of professional sport and sudden cardiac death cases among athletes aroused emerging interest in sports cardiology. Several research groups investigated exercise training induced left ventricular (LV) hypertrophy in animal models, however only sporadic data exists about detailed hemodynamic measurements. We aimed to establish and validate the rat model of athlete's heart and provide a detailed functional characterization using the modern sophisticated method of pressure-volume (PV) analysis.
LV hypertrophy was induced by swimming training (200min/d for 12 weeks). Sedentary rats were placed in the swimming apparatus for 5min/d. After completion of the swimming portocol we performed echocardiographic measurements and LV PV analysis using a microtip pressure-conductance catheter to investigate the morphology and function of the LV, respectively.
Echocardiographic examinations showed LV concentric hypertrophy according to the wall-thickness values (LV mass index: 2.41±0.08 vs. 2.03±0.08g/kgBW, $p<0.05$), which was confirmed by post-mortem measured heart weight and histological morphometry.
Invasive hemodynamic measurements showed unchanged heart rate, arterial pressure and LV end-diastolic volume along with decreased end-systolic volume, increased stroke volume (175±8 vs. 145±8µl) and ejection fraction (73±1 vs. 64±2%) in trained rats compared to sedentary controls. The PV loop-derived sensitive, load-independent contractility indexes were found to be significantly increased (preload recruitable stroke work: 77±7 vs. 54±5mmHg). We observed increased LV stroke work (15±1 vs. 11±1mmHg*ml) and maximal power (92±9 vs. 60±6mW) in athlete's heart. Despite the significant hypertrophy, the LV stiffness was not increased, while there was an improvement in active relaxation (Tau: 9.6±0.3 vs. 10.9±0.3ms).
According to our results, we established a rat model of physiologic LV hypertrophy. It is the first study, which provides a detailed characterization of functional changes and hemodynamic relations in athlete's heart.
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Resistance to antiplatelet therapy in patients with acute coronary syndrom

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Background: Antiplatelet therapy does not provide sufficient laboratory inhibition of platelet activity for all patients. The aim of this study was to find down the presence of aspirin and clopidogrel resistance in patients with acute coronary syndrom and determine relationship between the resistance, angiographic severity of coronary artery disease and recurrence of acute coronary syndrom (ACS).
Methods: Aspirin and clopidogrel resistance was evaluated with optical transmission aggregometry after stimulation by arachidonic acid (detection of aspirin resistance) and ADP with prostaglandin E1 (detection of thienopyridin resistance) among patients with ACS hospitalized in Department of Internal Medicine I, University