to each coronary territory was calculated. The LV end-diastolic volume (EDV), end-systolic volume (ESV), and LV ejection fraction (LVEF) were automatically obtained in each isotope using QGS software. In both infarcted and non-infarcted coronary territories, when 123I-BMIPP defect score was higher than that of the 99mTc-sestamibi defect score, it was considered as a mismatched defect, and when 123I-BMIPP defect score was similar to the 99mTc-sestamibi defect score it was considered as a matched defect. Mismatched defects in non-infarcted coronary territory were regarded as myocardial ischemia. We assessed its diagnostic value for the detection of myocardial ischemia in the non-infarcted territory. A severe coronary stenosis in non-infarcted coronary territory was defined as $\geq 90\%$ diameter narrowing on visual estimation.

Results: The correlations of EDV, ESV and LVEF between 99mTc-sestamibi and 123I-BMIPP were excellent (r=0.98, 0.98, and 0.93, respectively). Bland-Altman limits of agreement were -16.9 to 21.3 for EDV, -20.0 to 14.9 for ESV, and -6.3 to 11.8 for LVEF. While 33 patients (75%) showed mismatched defect, the remaining 11 (25%) showed matched defect in infarcted coronary territory. In the non-infarcted territory, 32 vessels with severe residual coronary stenosis were found in 24 out of 44 patients. In the 84 out of 88 non-infarcted coronary territories except for 4 previous infarcted territories, mismatched defect was found in 30 coronary territories and matched defect in 54. To detect myocardial ischemia with severe residual coronary stenosis in the non-infarcted area, 99mTc-sestamibi/123I-BMIPP dual-isotope imaging showed sensitivity, specificity, and accuracy of 72%, 87%, and 81%, respectively.

Conclusions: Simultaneous 99mTc-sestamibi/123I-BMIPP dual-isotope imaging at rest with low radiation exposure using CZT SPECT system may be feasible not only for evaluation of infarcted myocardium but also for identification of severe residual stenosis in the non-infarcted area in patients with AMI without the need for the stress during the early phase.

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Direct comparison between invasive fractional flow reserve and dynamic single photon emission computed tomography in patients with coronary artery disease

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Background: The ability of positron emission tomography (PET) in myocardial blood flow (MBF) evaluation is well established. Conversely, the ability of myocardial perfusion imaging with solid state cameras equipped with Cadmium-Zinc-Telluride (CZT) in evaluating MBF and myocardial flow reserve (MFR) has been proposed only recently. However, the validation of these measures against the invasive gold standard represented by fractional flow reserve (FFR) is still awaited. Purpose: To evaluate the correlation of MBF and MFR quantified by CZT against invasive FFR values.

Methods: Twenty-three consecutive patients (61.2±6.8 years; 12 women, 11 men) with suspected and known stable CAD were enrolled. Within 7 days all patients underwent non-invasive assessment of MBF and CFR, obtained by a dynamic acquisition with CZT and Tc99-MIBI, and invasive assessment of coronary anatomy with the estimation of FFR values in at least one main coronary artery showing an intermediate coronary lesion (30–90% stenosis). The values of stress (adenosine pharmacological test) and rest MBF were calculated semi-automatically by a net retention model. The value of MFR was calculated as MBF ratio (MBF stress / MBF rest). FFR was calculated during the steady-state (60–90 sec) adenosine induced hyperemia as a ratio of the mean distal intracoronary pressure to the mean arterial pressure.

Results: The values of MBF in left anterior descending coronary artery (LAD), left circumflex (LCx) and right coronary artery (RCA) territories significantly differed at stress and rest study (table 1). There was a good correlation between dynamic SPECT MFR values and the results of invasive FFR (r=0.65; p=0.0001). The value of global MFR was 1.83 (interquartile range 1.35; 2.24) and FFR was 0.79±0.1. According to the ROC analysis dynamic CZT MFR value, less or equal to 1.49 (AUC 0.88; CI 0.7–0.97; p<0.0001) allows identifying ischemia with sensitivity 71.4% and specificity 93%.

Table 1. The values of Stress/Rest MBF

Myocardial blood flow (ml/min/g)	Stress†	Rest [†]	Wilcoxon test p-value
LAD MBF	0.62 (0.42; 0.90)	0.37 (0.2; 0.6)	0.0003
LCX MBF	0.84 (0.53; 0.98)	0.38 (0.2; 0.5)	0.00002
RCA MBF	0.63 (0.48; 0.91)	0.36 (0.31; 0.52)	0.0002
Total MBF	0.67 (0.55; 0.81)	0.36 (0.3; 0.5)	0.00008

†Median and interquartile range. MBF: myocardial blood flow; LAD: left anterior descending artery territory; LCx: circumflex artery territory; RCA: right coronary artery territory.

Conclusion: There is a good relationship between regional MFR and invasive FFR. In selected patients it could be of help in order to assess the hemodynamic significance of coronary artery stenosis non-invasively and to better stratify the risk of CAD.

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Global myocardial blood flow and coronary flow reserve quantification in patients with and without relative regional perfusion defects using dynamic solid-state detector SPECT and Tc-99m sestamibi

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Background/Introduction: In PET, myocardial blood flow (MBF) provides incremental diagnostic and prognostic value for the workup of coronary artery disease. Novel fast solid-state detector equipped SPECT systems are capable of dynamic imaging and may enable MBF quantification, too.

Purpose: Our aim was to determine feasibility of MBF results derived from SPECT in patients with and without scintigraphic hypoperfusion at rest and stress. Methods: Using a GE Discovery NM530c system, 58 patients underwent dynamic cardiac SPECT-imaging at rest and 50 patients at stress. After careful patient positioning using a test dose, 300–350 MBq Tc-99m Sestamibi were injected by bolus pump (Braun) and a 10min listmode acquisition was started simultaneously. Static relative perfusion images were acquired subsequently according to standard protocol. Regadenoson was used for vasodilator stress. Using INV1A software, dynamic SPECT data (21 frames) were generated with (AC) and without (NC) attenuation correction using an external CT. Time-activity curves were generated with frame-based manual motion correction, and fitted using a two-compartment-model. MBF was extrapolated from K1 using previously established equations for sestamibi (Leppo et al.), and goodness of fitting was determined by χ². Coronary flow reserve (CFR) was calculated as the ratio of stress-MBF and rest-MBF.

Results: 31 (53,4%) patients presented with a regional perfusion defect on static rest images. Global rest-MBF was significantly reduced in these patients in NC (1.30±0.44 vs. 1.99±1.05 ml/min/g; p=0.004) and in AC (0.43±0.07 vs. 0.53±0.16 ml/min/g; p=0.004), and CFR was lower (NC: 1.08±1.30 vs. 1.88±1.25; p=0.018 NC (20.92±0.97 vs. 1.69±1.05; p=0.028). χ^2 values were significantly higher in NC (265±229 vs. 146±45; p=0.006) but not in AC (234±166 vs. 184±71; p=0.421), implying a worse goodness of fit in cases with rest perfusion defects without AC. 4 (8,0%) patients showed a regional stress-induced, but reversible perfusion defect. No significant differences were found for global MBF, CFR and χ^2 comparing patients with and without stress hypoperfusion in NC (MBF 3.79±0.79 vs. 3.13±1.89 ml/min/g, p=0.15; CFR 2.23±2.13 vs. 1.90±1.14, p=0.59; χ^2 126±61 vs. 232±181, p=0.24) and AC (MBF 3.01±0.94 vs. 2.23±1.41 ml/min/g, p=0.13; CFR 1.65±1.07 vs. 1.66±0.81, p=0.57; χ^2 159±69 vs. 254±177, p=0.427.

Conclusions: Quantification of global MBF and CFR using dynamic solid-state detector SPECT is feasible, yielding reduced values in patients with regional rest perfusion defects. However, patients with stress-induced regional ischemia showed a wider variability of global MBF and CFR. Subsequent studies will need to determine whether this reflects heterogeneous disease severity or methodological scatter.

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PET/CT with 18F-sodium fluoride in patients with aortic stenosis and different aortic valve morphology

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Introduction: Aortic stenosis (AS) is the most frequent valvular heart disease. The mechanisms underlying calcification process are incompletely understood. Purpose: The aim of this study was to access valve calcification activity in pts with tricuspid aortic valve (TAV) and bicuspid aortic valve (BAV) using 18F-sodium fluoride (18F-NaF) uptake and calcium scoring.

Methods: 58 pts with asymptomatic AS were divided into 2 groups: 31pts with TAV and 27 pts with BAV. Pts with infective endocarditis and rheumatic heart disease were excluded. The aortic jet velocity (Vmax) was chosen as ECHO marker of AS severity. PET/CT (Discovery 710, GE) was performed in 60 minutes after intravenous administration of 5 mCi 18F-NaF. The circular region of interest (ROI) of 3.52 square millimeters was drawn around areas of maximal 18F-NaF uptake in the valve and the mean level of standardized uptake value (SUV) was measured within ROI. Blood-pool activity as a background was estimated using SUV mean within the same ROI, which was drawn on the left atrium. Ratio between SUV mean tissue and SUV mean background (TBR) was calculated. CT calcium scoring was performed using dedicated software and recorded in Agatston units. Results: Patients in TAV group were significantly older then in BAV group (63.1±6.13 yrs vs 58.22±8.04 yrs, p=0.011). TAV and BAV groups were comparable in AS severity assessed by ECHO (Vmax: 2.99±0.57 m/s vs 3.02±0.68m/s, p=0.87). Both groups did not differ in valvular calcification degree (Agatston score: 1496.03±1346.42 vs 1612.96±1482.08, p=0.76) and 18F-NaF uptake level (TBR: 1.43±0.26 vs 1.47±0.35, p=0.62). We noted strong positive correlation between TBR and Agatston score for TAV and BAV groups (r=0.8, p<0.001 and r=0.68, $p{<}0.001)$ as well. Interestingly, we found only the correlation between Vmax and Agatston score (r=0.52, p=0.002), but didn't observe significant relationship between Vmax and TBR (r=0.29, p=0.12) in TAV group. Whereas, positive correlation between Vmax and TBR and Agatston score was obtained for BAV group (r=0.72, p<0.001 and r=0.69, p<0.001).

Conclusions: Only in BAV pts we revealed the relationship between aortic stenosis severity and valve calcification activity. Absence of the association between 18F-NaF uptake level and ECHO markers of AS severity possibly may be explained by slower rates of calcification process in patients with TAV.

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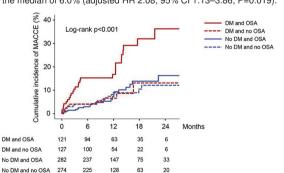
Clinical significance of obstructive sleep apnea in patients with acute coronary syndrome in relation to diabetes status: insights from the OSA-ACS project

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Background: OSA is highly prevalent in patients with acute coronary syndrome (ACS) and is associated with increased risk of recurrent cardiovascular events. Studies have indicated possible interaction between OSA and diabetes mellitus (DM) in glycemic control. Whether the impact of OSA on cardiovascular outcomes after ACS may be modified by DM status remains unclear.

Purpose: This study sought to investigate the association of OSA with subsequent cardiovascular events in ACS patients with or without DM.

Methods: The OSA-ACS project is a large-scale, prospective, observational study to evaluate the effects of OSA on cardiovascular outcomes in patients presenting with ACS in the contemporary era. From June 2015 to May 2017, consecutive eligible patients admitted for ACS underwent overnight cardiorespiratory polygraphy during hospitalization. Recruited patients were categorized into OSA (AHI \geq 15 events h⁻¹) and non-OSA (AHI < 15 events h⁻¹) groups. The primary endpoint was major adverse cardiovascular and cerebrovascular event (MACCE), defined as a composite of cardiovascular death, myocardial infarction, stroke, ischemia-driven revascularization, or hospitalization for unstable angina or heart failure. This study conformed to the principles of the Declaration of Helsinki. Results: Among 804 patients, 248 (30.8%) had DM. The rate of MACCE was significantly higher in patients with versus without DM (14.5% vs. 8.1%, HR 1.88, 95% CI 1.21-2.92, P=0.005) during a median follow-up of 1 year (0.7-1.7). The figure showed the Kaplan-Meier curves for MACCE according to presence of DM and OSA. OSA was associated with 2.5 times the risk of MACCE in patients with DM (22.3% vs. 7.1% in non-OSA group, adjusted HR 2.49, 95% CI 1.16-5.35, P=0.019) but not in those without DM (8.5% vs. 7.7% in non-OSA group, adjusted HR 0.94, 95% CI 0.51-1.75, P=0.848). DM patients without OSA had a similar 1year rate of MACCE as patients without DM (7.1% vs. 8.1%, adjusted HR 0.94, 95% CI 0.46-1.95, P=0.876). Moreover, for patients with baseline glucose levels above the median of 5.9 mmol/L, presence of OSA increased the risk of incurring a MACCE (adjusted HR 2.25, 95% CI 1.17-4.32, P=0.015), but no increased risk was found in patients with glucose levels <5.9 mmol/L (adjusted HR 0.78, 95% CI 0.36-1.65, P=0.510). Similarly, the increased risk of MACCE in OSA patients was only observed in patients with admission levels of haemoglobin A1c above the median of 6.0% (adjusted HR 2.08, 95% CI 1.13-3.86, P=0.019).



Conclusion: ACS Patients with DM and OSA had a greater risk of MACCE at 1 year. In contrast, the prognosis of ACS patients with DM but without OSA is favorable and similar to patients without DM. Further randomized trials to explore the efficacy of OSA treatment as secondary prevention after ACS in patients with DM are highly warranted.

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Middle age men with short sleep duration have two times higher risk of cardiovascular events than those with normal sleep duration, a cohort study with 21 years follow-up

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Background: Self-assessed short sleep duration have previously been reported to be associated with increased risk of cardiovascular disease. However, other studies do not support this finding.

Purpose: To investigate whether short sleep duration at fifty years of age is associated with increased risk of major adverse cardiovascular events (MACE) during a 21-year follow-up period.

Methods: In 1993 50% (n=1463) of all men living in Gothenburg, born in 1943, were invited to participate. Of those 798 (54%) accepted and filled out questionnaires (including questions about average duration of sleep per day), underwent physical examination, ECG and blood pressure monitoring and anthropometric data were measured. Re-examinations were conducted in 2003 and in 2014. MACE was assessed by a combination of review of medical records of all participants, the Swedish Hospital Discharge registry and the Swedish Death Cause registry. MACE were defined as: acute coronary syndrome, stroke, TIA or heart failure.

Results: Nineteen subjects were excluded because of inadequate sleep questionnaires, and 19 were excluded since they had MACE before baseline. Thus, 760 individuals were included in the final analysis, in total 14,848 person-years at risk. Men with short sleep time had more hypertension and more diabetes, were more obese, were to a larger extent smokers/former smokers, were less physically active and had worse sleep quality. Kaplan-Meier analyses showed an increased risk of MACE in those with sleep less than five hours per night compared to the group set as reference (7–8 hours), p=0.003. Patients with a sleep time of <5 hours had an almost two time higher risk of developing MACE during the follow-up period. The risk increase remained almost the same after adjustment for confounders (Table 2). If, in addition to those with MACE, all those with hypertension at baseline also were excluded, the hazard ratio (HR) for developing MACE remained significant with HR 2.45 (95% confidence interval [CI]1.34 - 4.46) in univariate and HR; 2.39 (95% CI 1.31 - 4.35) in multivariate analysis.

Table 2. COX regression

	≤5 hours HR (95% CI)	6 hours HR (95% CI)	7–8 hours reference	>8 hours HR (95% CI)
Model 1*	2.15 (1.28-3.61)	0.89 (0.62-1.29)	1	0.75 (0.28–2.04)
Model 2 [†]	1.97 (1.17-3.31)	0.89 (0.62-1.29)	1	0.77 (0.29-2.09)
Model 3 [‡]	1.93 (1.15-3.25)	0.88 (0.61-1.27)	1	0.79 (0.29-2.14)

 * Unadjusted. † Adjusting for BMI >30, diabetes. ‡ Adjusting for BMI >30, diabetes, smoking status.

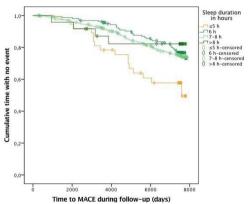


Figure 1. Kaplan-Meier survival curve

Conclusion: Sleep duration of five hours or less per night in those middle-aged men was associated with a two-fold higher risk of developing cardiovascular disease 2 decades beyond.

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Association of actigraphy-measured sleep parameters and subclinical atherosclerotic burden: the PESA study

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