99. Cardiovascular complications in obstructive sleep apnoea

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Cardiovascular risk factors and the metabolic syndrome in patients with obstructive sleep apnea

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Background: Obstructive sleep apnea (OSA) is an important cause of cardiovascular morbidity and mortality. The aim of this study was to assess cardiovascular risk factors and the prevalence of the metabolic syndrome in patients with OSA.
Methods: For this retrospective analysis we analyzed clinical and laboratory cardiovascular risk factors and the prevalence of the metabolic syndrome in 96 patients with OSA (AHI ≥ 15/h) and 36 snorers without OSA (AHI ≤ 5/h).
Results: Mean ± SD apnea-hypopnea-index was 35±27/h and 1.0±2.3/h in patients with OSA and snorers, respectively. Patients with OSA had a higher body-mass-index (33±7kg/m^2) than snorers (26±4kg/m^2) without OSA (p<0.01). There was a significantly higher prevalence of hypertension (73% vs. 39%, p<0.01) and metabolic syndrome (48% vs. 19%, p<0.01) in OSA patients than in snorers without OSA. There were no significant differences in the prevalence of hyperlipidemia, heart failure, chronic obstructive airway disease, heart rhythm disturbances or cerebrovascular disease between the groups. Fasting blood glucose levels (121±59mg% versus 99±41 mg%, p<0.02), C-reactive protein levels (2±2.8 mg/dl versus 1±1.9mg/dl, p=0.05), and leukocyte cell count (7.5±2.5G/l versus 7.2±2.1G/l, p<0.05) were significantly higher in OSA patients. Both the body-mass-index (r=0.396, p<0.01) and apnea-hypopnea index (r=0.312, p<0.01) were significantly correlated with C-reactive protein levels in the total sample.
Conclusions: We found a high prevalence of cardiovascular risk factors in patients with OSA. Both obesity and severity of OSA appear to be important determinants of subclinical systemic inflammation associated with the disease.

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Sympathetic activity at rest and during exercise in metabolic syndrome subjects with and without obstructive sleep apnea

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High muscle sympathetic nerve activity (MSNA) has been incriminated in the cardiovascular morbidity associated to either metabolic or obstructive sleep apnea (OSA) syndrome. We hypothesize that the highly prevalence of OSA in metabolic syndrome patients may have synergistic effect on MSNA.
Methods: Thirteen subjects with metabolic syndrome (IDF Consensus, 2005) were screened for their OSA status. We measured, along with anthropometric and metabolic variables, MSNA (microneurography) at rest and during isometric exercise. The isometric handgrips were followed by 3 min of post exercise forearm circulatory arrest in order to isolate the metaboreflex contribution to exercise responses.
Results: Eight patients were found to have OSA and 5 without OSA. Both groups were matched for BMI, waist circumference, blood pressure, triglycerides, insulin and HDL cholesterol values. Sympathetic nerve traffic was significantly greater in subjects with OSA than in subjects without OSA (45±10 vs 34±8 bursts/min, p<0.03). MSNA at rest correlated significantly with AHI (r= 0.62, p<0.02).The sympathetic response to isometric exercise was also higher in patients with OSA (55±10 vs 40±6 bursts/min, p<0.02). During postexercise circulatory arrest, MSNA remained higher in the OSA group (p<0.04). Moreover, the sympathetic response to exercise was also correlated to the apnea/hypopnea index (r=0.8; p<0.05).
Conclusions: In this study 61% of the patients exhibited OSA. OSA severity was significantly correlated to MSNA at rest and during exercise. This is likely to play a significant contribution in cardiovascular morbidity of patients suffering from both OSA and metabolic syndrome.

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Endothelial dysfunction in patients with obstructive sleep apnea independent of metabolic syndrome

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Background: Obstructive sleep apnea syndrome (OSAS), characterized by intermittent hypoxia/reoxygenation, has been identified as an independent risk factor for cardiovascular diseases.
Our aim was to assess the endothelial function in the patients with different OSAS activity and to hypothesize about the relationship of endothelial function to severity of sleep apnea.
Methods: Fifty-two subjects with OSAS recently diagnosed by polysomnography were classified into two groups according to presence and absence of metabolic syndrome and also according to the severity of apnea: mild to moderate OSAS group and severe OSAS group.
Endothelial functions of the brachial artery were evaluated by using high-resolution vascular ultrasound. Endothelial-dependent dilatation (EDD) was assessed by establishing reactive hyperemia and endothelial-independent dilatation (EID) was determined by using sublingual isosorbide dinitrate. Spearman correlation and regression analysis were performed.
Results: EDD was not significantly worse in patients with OSAS and metabolic syndrome as compared with OSAS without metabolic syndrome (4.53±0.68 versus 4.29±0.90, P > 0.05). EDD was significantly worse in patients with severe OSAS compared with patients with mild to moderate OSAS (4.98±0.87 versus 4.28±0.74, P < 0.05).
Conclusions: Endothelial dysfunction in obstructive sleep apnea may be independent of metabolic syndrome. Increased severity of OSAS is associated with significant endothelial dysfunction, which may relate to the pathophysiology of athrosclerosis of OSAS.

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Sleep-related breathing disorders in patients under cardiac resynchronization therapy with ventricular pacing

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Patients with heart failure present with central sleep apnea syndrome and Cheyne Stokes respiration (which is associated with increased mortality). The prevalence of sleep-related breathing disorders in a cohort of 15 patients (12 M; 41-73 years) affected by idiopathic or post-ischemic dilatative cardiomiopathy (III-IV NYHA functional class) was evaluated. They undergoing to cardiac resynchronization therapy with the application of biventricular pacing. Patients in responders and non-responders to resynchronization therapy according to strain and strain rate values performed with ecocolordoppler TDI (7 patients were responders and 8 non-responders) were classified. All patients underwent ambulatory overnight cardiorespiratory polygraphy. Moreover, anthropometric data and daytime sleepiness with Epworth scale were collect. 4 patients had a BMI > 30 kg/m^2 (average BMI 27.4, range 22.6-33.9), while a daytime hypersomina was present in 66% of patients (ESS average 11.5, range 0-18). Polygraphics alterations were present in 66% of patients with high prevalence of central apnea associated with Cheyne-Stokes respiration (7/10 patients). Only 2 patients responder (28%) had sleep-related breathing disorders characterized by mild obstructive sleep apnea (OSAS) with an AHI index, respectively of 5.8 and 11.6. All non-responders patients had sleep-related breathing disorders with an average AHI index of 22.1 (range 8-29.7) and 7 of them had central apnea associated with Cheyne-Stokes respiration (87.5%). Patients non responder to resynchronization therapy show greater frequency and gravity of sleep-related breathing disorders which should require further studies.

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High prevalence of obstructive sleep apnoea in revascularized patients with coronary artery disease – RICCADSA trial

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One third of the patients with coronary artery disease (CAD) have been suggested to suffer from obstructive sleep apnoea (OSA) compared with 13% of healthy controls matched by age, gender and Body-Mass-Index. The current study (RICCADSA) is an on-going randomized controlled trial addressing the impact of continuous positive airway pressure (CPAP) in revascularized CAD patients and concomitant OSA (Apnoea-Hypopnoea-Index [AHI] ≥ 15/h) without daytime sleepiness (Epworth Sleepiness Scale [ESS] <10). The primary end-point is the combined rate of new revascularization, myocardial infarction, stroke and cardiovascular mortality over a 3-year period. The trial was started December 1, 2005 to comprise 400 participants including 200 non-sleepy OSA patients (randomized to CPAP or non-CPAP), 100 sleepy (ESS≥ 10) OSA patients on CPAP and 100 non-OSA subjects (AHI<5/h). Patients with dominantly Cheyne-Stokes respiration and/or with AHI 5.0-14.9 (borderline OSA) are not included in the trial. Out of 560 revascularized CAD patients, 286 (51.1%) agreed to participate in the study by January 31, 2008. OSA was prevalent in 176 (61.6%), of whom 108 (61.4%) were regarded as non-sleepy. Borderline OSA was observed in 51 (17.8%) whereas 59 (20.6%) had no OSA. We conclude that more than half of the revascularized CAD patients have OSA, which is much higher than previously reported. When completed, the current trial will contribute to define the impact of CPAP as a non-pharmacological intervention for CAD patients with OSA regardless daytime sleepiness.

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The relation between obstructive apneas in OSA patients and autonomic activity

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Obstructive sleep apnea (OSA) is characterized by episodes of upper airway obstruction which produces acute autonomic changes. Spectral components of heart rate variability of low frequency (LF) and high frequency (HF) provide measurement of sympathetic and parasympathetic activity. In this study we evaluated LF, HF and LF/HF in OSA patinets, in 2NREM sleep during 1 min of basal sleep, 20 sec before an obstructive apnea (OA), during and 20 sec after this. We analysed OA followed (not pure) and not followed (pure) by a cortical arousal. So 10 patients were studied: 8 were male, mean age: 58,50 y, mean BMI: 30,60 kg/m^2, mean AHI: 18,45 events/h and all of them had excessive daytime sleepiness. The difference between LF during 20 sec period before OA (pure and not pure) and that during the same OA was statistic significant. The difference between LF during pure OA and that during 20 sec period after the same OA was statistic significant, but there was no statistic difference in not pure OA for this period. The difference between LF during basal period and that during 20 sec after pure and not pure OA was statistic significant, and also was the difference between LF and LF/HF during 20 sec period after pure and not pure OA. There was no statistic difference of HF among these different periods.

Conclusion: sympathetic activation occurs during pure OA and persists for a little while after its cessation, and then returns to the level which is the basal of that patient. Sympathetic activation persists after the end of not pure OA, which is related with arousal. So, sympathetic activation occurs even in the absence of an arousal, but in its presence is more intense.

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Prediction of circadian changes in time domain (TD) heart rate variability (HRV); use of short term HRV monitoring

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Introduction: The relatively sympathetic diurnal & parasympathetic nocturnal tone has been measured by HRV derived from 24hr Holter(HM)recordings-an encumbrance to the elderly.Analysis of short-term HRV may prove an alternate to HM in these patients.15.5min HRV monitoring(using ANX-3.0) involves various autonomic challenges.Parasympathetic reserve is tested in deep breathing(DB) while sympathetic tone is measured during the Valsalva maneuver.The ability of ANX-3 to predict circadian changes in HRV has not yet been tested

Methods: Simultaneous 24hr HM &15.5min ANX-3 recordings were obtained from 20 ambulatory 70-90 y/o(18M, 2F) patients(8 records excluded by arrythmia/ectopy).HRV was computed for each 24hr period of HM using 3 standard TD measures(rMSSD, pNN50 & SDNN).TD HRV determined from the ANX phase of DB was compared with nightime HRV HM data while the TD during Valsalva was compared with daytime HRV from HM data using SPSS 11.0. Correlations were determined using Pearson's coefficient.

Correlation between HM data & ANX recordings per subject

Patient ID	Holter HRV (Daytime) vs. ANX HRV during Valsalva	Holter HRV (Daytime) vs.whole 15.5 min ANX HRV	Holter HRV (Nighttime) vs.ANX HRV during deep breathing
1	0.98	1.00	0.79
2	0.92	0.89	0.32
3	0.98	0.93	0.95
4	0.98	0.83	0.98
5	0.90	0.91	0.79
6	0.83	0.97	0.62
7	0.96	0.91	0.89
8	0.94	0.96	0.97
9	0.89	1.00	0.93
10	0.87	0.87	0.91
11	0.73	1.00	1.00
12	0.79	0.78	0.98
Average	0.90	0.92	0.84

Results: Comparisons between diurnal TD HRV(HM) was correlated with ANX measures during Valsalva (90%,p=0.12-0.4) &whole 15.5min ANX testing(92%,p=0.0-0.3).The TD HRV from the deep breathing phase of ANX was also comparable to nocturnal HRV from HM (84%,p=0.0-0.4).

Conclusion: The data supports the hypothesis that TD HRV values computed from various phases of short term ANX recordings predict circadian changes in HRV.

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Autonomic responses to apneas in relation with the severity of sleep apnea syndrome

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Introduction: Autonomic dysfunction has been demonstrated in patients with obstructive sleep apnea syndrome (OSAS). Pulse transit time variations during sleep (PTTv) reflect the autonomic response to apneas.

Aim: To study the hypothesis that autonomic responses to apneas are blunted in patients with severe OSAS.

Methods: 12 patients with mild OSAS (apnea-hypopnea index AHI = 8.06±1.56, mean age 52.67), 10 with moderate OSAS (AHI = 21.2±5.8, mean age 56.9), and 9 with severe OSAS (AHI = 54.78±7.91, mean age 54.68) who had underwent full polysomnography were selected, after the exclusion of major confounders for dysautonomy (like diabetes and ethylism). The coefficient of variation of the heart rate was calculated automatically during total sleep time(CVcalc) and manually on sampled apnea-free epochs in all sleep stages (CVman) by dividing the standard deviation by the mean. PTT was the interval between the R wave on ECG and the pulse wave. The heart rate response to apneas (ΔHR) was measured manually on sampled epochs.

Results: CVcalc, CVman in non-REM sleep and ΔHR were lower in the severe OSAS group than in the mild OSAS group ($p < 0.01$ for CVcalc and for ΔHR, p = 0.02 for CVman) and correlated inversely with the AHI (r = -0.81 for CVcalc, -0.72 for CVman, -0.83 for ΔHR). The amplitude of PTT variations during apneas (PTTv) also showed an inverse correlation with the AHI (r = -0.65). PTTv did not differ between groups ($p > 0.05$).

Conclusions: Heart rate response to apneas was diminished in patients with severe OSAS, more evidently than the PTT response. Moreover, heart rate variability in apnea-free periods was impaired in patients with severe OSAS, which could be proven in non REM sleep.

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Obstructive sleep apnea syndrome – the evaluation of right ventricular function in moderate – severe patients

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Objectives: Obstructive sleep apnea (OSA) may influence the cardiac function by several mechanisms. OSA is associated with significant increased morbidity and mortality. Little is known about OSA influence on right ventricular (RV) function.

Methods: We studied the RV function in symptomatic OSA patients (AHI≥15). 75 pts (65 males) (aged 52,3±10,00) with moderate OSA (group A-17 pts (15 males), aged 54,6±9,2, body mass index (BMI) 33,6±6,3) and severe OSA (group B 58 pacients (50 males), aged 51,7±10,3, BMI 35,1±5,9) were studied. The patients had no known pulmonary or trombo- embolic diseases and they had normal left ventricular systolic function. The echocardiographic evaluation was made by VIVID 7 GE (two dimensional, pulse and continous wave Doppler evaluation, tissue Doppler imaging (TDI) evaluation). TEI index (index of global cardiac function) was determined by the ratio of the sum of the isovolumic times divided by the ejection time.

Results: There were no differences between A group and B group in gender (p=0,829), age (p=0,283), concomitant arterial hypertension (p= 0,741) and BMI value (p=0,387). RV TEI index was pathological in 31 patients (41,33%) and there was statistically significant difference between groups (11,76%, 50,0%, p= 0,016). The average value of RV TEI index was 0,31±0,23 and there was statistically significant difference between groups (0,19±0,13, 0,356±0,24, p=0,011).

Conclusions: The severe OSA patients had worse RV diastolic function than the patients with moderate OSA.

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Association of vascular endothelial dysfunction with disease severity in obstructive sleep apnoea

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Objective: Obstructive sleep apnoea (OSA) has been recently associated with features of the metabolic syndrome. Vascular endothelial dysfunction (VED) represents a characteristic finding that significantly associates with many of the metabolic syndrome functional correlates. We sought to determine the degree of VED in OSA patients and assess its relationship with diease severity.

Methods: We recruited 64 patients of those reffered to the clinic that were confirmed to have OSA diagnosis (45 men, 19 women; aged: 54±12 years) with Body Mass Index (BMI) 32±1 kg/m^2, and no concurrent disease. They all underwent

nocturnal polysomnography, measurement of flow-mediated dilation (FMD) as an idex of endothelial function, by means of high-resolution ultrasound and pulmonary function testing, including spirometry, flow-volumes curves, lung volume and diffusion lung capacity assessment.
Results: There was a significant negative correlation between FMD (3.42±0.45) and apnea-hypopnoea index (AHI; 51.2±27.1) (p= 0.045, r^2 =0.085), as well as a significant positive correlation between FMD and TLC (p= 0.042, r^2 = 0.094), even after adjustment for BMI and tobacco use. Furthermore, there was a trend of positive correlation, albeit not statistically significant, between FMD and FVC, FEV_1 and FEF_{25-75}.
Conclusions: In obese (class I) OSA patients disease severity significantly associates with gradual FMD impairment. Early investigation of endothelial function in such patients might lead to timely therapeutic intervention and prevent many of the consequences of the metabolic syndrome. Further studies should investigate underlying pathophysiological mechanisms.

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Sleep patterns in patients with acute coronary syndromes

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Background: Critically ill patients are prone to sleep deprivation, poor sleep quality, and disturbed circadian patterns. There is little knowledge about sleep quality impairment in patients admitted in Coronary Care Unit (CCU) with acute coronary syndromes (ACS) and the underlying mechanisms.
Aim: The aim of this study was to assess nocturnal sleep quality in patients with ACS, during the acute event and after 1 and 6 months.
Methods: A total of 30 patients aged 42±8.8 years presenting with a first ever ACS and with preserved left ventricular function were studied. Patients underwent an overnight polysomnography (PSG) in our sleep disorders unit, within 3 days after the acute event, with a follow up PSG performed one and six months later, away from the CCU environment and without taken oral or intravenous sedation.
Results: Compared with the follow up, the initial PSG of the acute phase revealed difficulties in initiating and maintaining sleep, significant increases in arousal index (28.2±7 vs 16.4±5.7 vs 4.1±1.0, p<0.001) and significant reduction in Slow Wave Sleep (SWS) (10.6±4.9min vs 31.6±4.2min vs 50.1±3.8min, p<0.001) as well as in Rapid Eye movement (REM) sleep (5.4±4.3 vs 29.9±7.8 vs 47.8±8.3, p<0.001). Sleep architecture was less altered 1 month after the acute event, while 6 months later sleep duration as well as sleep stages were within normal range.
Conclusions: Patients with ACS have altered sleep architecture, in the form of increased arousals and decreased SWS and REM sleep, which tend to normalize after 6 months. The mechanisms of these alterations in sleep needs further investigation.

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Prevalence of obstructive sleep apnoea and its association with aortic root dilatation in patients with Marfan's syndrome

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Background: The high prevalence of craniofacial abnormalities, and possibly an increased upper airway collapsibility due to an abnormality of fibrillin, suggests there might be an increased prevalence of obstructive sleep apnoea (OSA) in patients with Marfan's syndrome, but there is very limited data on the actual prevalence. Aortic root dilatation and subsequent dissection are the most life-threatening manifestations of Marfan's syndrome and case reports suggested a possible association with OSA, but data from cohort studies are not available.
Methods: A sleep study was performed in 61 patients with Ghent-criteria positive Marfan's syndrome (mean age±SD, 38.3±12.9, 37 females) and in 26 healthy controls matched for age, gender, height and weight. In patients with Marfan's syndrome aortic root diameter was measured by two-dimensional, M-mode echocardiography.
Results: Mean±SD apnoea-hypopnoea index (AHI) and >4% O2 desaturation index (ODI) were significantly higher in patients with Marfan's syndrome (7.4±9.1 and 4.7±5.8 respectively) compared to controls (3.3±2.1 and 2.0±2.4 respectively) (p=0.02 for both comparisons). 20 patients with Marfan's syndrome had an AHI>5 (32.8%) compared to 3 patients in the control group (11.5%) (p=0.04). AHI was significantly correlated with aortic root diameter (r=0.50, p<0.001) and mean±SD aortic root diameter was significantly larger in patients with AHI>5 (4.5±0.6cm) than in patients with AHI<5 (3.7±0.6cm) (p<0.0001).
Conclusions: Among patients with Marfan's syndrome the prevalence of OSA is considerably higher than in the normal population. OSA may be a risk factor for aortic root dilatation in Marfan's syndrome.

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Daytime sleepiness and quality of life are not related to nocturnal Cheyne-Stokes respiration in heart failure

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The effects of central sleep apnea and Cheyne-Stokes respiration on sleep-related symptoms and quality of life in heart failure are poorly defined. We therefore aimed to investigate to what extent central sleep apnea and Cheyne-Stokes respiration are related to daytime sleepiness, nocturnal dyspnea and health related quality of life. Included were 203 consecutive patients with congestive heart failure stabilized before discharge from hospital. They underwent overnight cardiorespiratory sleep apnea recordings and answered nine specific questions on symptoms and two health related quality of life questionnaires (Nottingham health profile and Minnesota living with heart failure questionnaire). After exclusion of 7 patients with predominantly obstructive apneas and 14 with insufficient recordings, 182 patients (mean age 72 years, 57 women, 122 with >5 and 58 with > 30 apneas/hypopneas per hour) were included in the final analysis.
Falling asleep in front of the television was the only symptom related to apnea-hypopnea index. None of the other questions, nor generic quality of life or disease specific quality of life were related to apnea-hypopnea index or percent of time spent in Cheyne-Stokes respiration during the recording.
We conclude that Cheyne-Stokes respiration and central sleep apnea are not (or only weakly) related to daytime sleepiness, nocturnal dyspnea or generic or disease specific quality of life among patients with stable congestive heart failure. Worse quality of life and worse sleep quality was, on the other hand, related to clinical severity of heart failure according to the New York Heart Association classification.

P839

Severity of nocturnal cardiac arrhythmias correlates with AHI in males with sleep apnoea

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Background: Sleep apnoea syndrome (SAS) is associated with high incidence of nocturnal cardiac arrhythmias (NCA).
Aim: To compare the severity of NCA in males with and without SAS and to assess the relationship between the severity of SAS and the severity of NCA.
Patients and methods: 33 males with mild to moderate SAS and 16 males without SAS, matched for cardiovascular co-morbidity (52±8 yrs, M±SD, versus 48±11 yrs) were studied by using whole-night polysomnography with simultaneous ECG analysis. Also echocardiography, carotid ultrasonography, blood pressure monitoring, baroreflex sensitivity and known risk factors of atherosclerosis (AS), were evaluated. Arrhythmia severity was scored using a special 7-degree scale.
Results: SAS patients had more severe NCA compared to controls (2,53±2,49, vs 1,13±1,64, p<0,05), much higher apnoea/hypopnoea index AHI=26,5±15,7, vs. 4,07±2,6/h, p<0,00000l), more severe hypoxemia ($MinSaO_2$=76,65±10,48, vs. 87±6,5%, p<0,0005), as well as greater maximal carotid wall thickness (IMT-max = 0,9l±0,21, vs. 0,77±0,18mm, p<0,05) and blunted baroreflex sensitivity (12,68±8,24, vs. 19,8±10,65 ms/mmHg, p<0,01). Higher age, pronounced structural changes of heart, decrease in left ventricular ejection fraction and signs of AS, were associated with increase in NCA severity. Multiple regression analysis revealed significant correlation of NCA severity with AHI (beta 0,458, p=0,0045).
Conclusion: Compared to controls, SAS patients had more severe NCA in correlation with severity of SAS, reflected by AHI. Already mild to moderate SAS promotes development of NCA.

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Does sleepiness have an impact on echocardiographic parameters in obstructive sleep apnea (OSA) after 6M CPAP therapy?

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Introduction: Recently, Robinson et al (ERJ 2006; 27: 1229-1235) reported that only sleepy hypertensive OSA patients have a reduction in 24-h blood pressure after 1M CPAP therapy. Therefore, we wanted to evaluate whether sleepiness [based on Epworth sleepiness scale (ESS) and subjective sleepiness] also has an impact on echocardiographic parameters in OSA after 6 M CPAP therapy.
Methods and Materials: We assessed 40 OSA patients [55 ± 10 y, M/F 33/7, BMI 30 ± 4] with moderate to severe OSA (AHI>20) (AHI 45 ± 22) and without cardiovascular history. We defined three groups [Gr 1: n=15, ESS<10, nonsleepy; Gr 2: n=13, ESS>10, nonsleepy; Gr 3: n=9, ESS>10, very sleepy]. In all patients polysomnography, physical examination and ECG as well as complete two-dimensional and Doppler echocardiography were performed. We evaluated heart rate (HR), blood pressure (BP), cardiac output (CO), stroke volume (SV), in-

terventricular septum thickness (IVS) and left ventricular ejection fraction (LVEF). After 6 M CPAP therapy, re-evaluation took place.
Results: After 6M CPAP therapy HR, BP, CO, SV, and LVEF were unchanged in each group. However, a trend to decease in BP, a trend to increase in EF and a normalisation in CO and SV could be observed. Moreover, IVS in Gr 1 [from 1.14 ± 0.26 cm to 0.96 ± 0.20 cm, p=0.022] and Gr 3 [from 1.29 ± 0.26 cm to 1.12 ± 0.20 cm, p=0.05] significantly decreased.
Conclusions: Sleepiness has no impact on improvement of echocardiographic parameters, except on the IVS, after 6M CPAP therapy. However, more patients have to be evaluated to make definite conclusions.

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Antiarrhythmic effect of CPAP and uvulopalatopharyngoplasty (UPPP) in patients with severe OSAS
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Rationale: Cardiac arrhythmias (CA)are common in OSAS patients and may be clinically significant and life threatening. The purpose of this study was to evaluate the impact of CPAP and uvulopalatopharyngoplasty (UPPP) on the prevalence of CA in patients with severe OSAS.
Methods: We studied 100 consecutive OSAS patients, mean age was 55±16 years, 70 were males, mean BMI was 29±4. We excluded COPD subjects and patients with ischaemic heart disease and left ventricular failure (ejection fraction < 45%).We performed an eight channels polysomnography (Polymesam®) and Holter ECG overnight at day of enrolment, and after 30 and 90 days. Thirty patients had an Apnea/Hypopnoea (AHI) > 15/h + sleepiness: 20 were treated with CPAP (Group 1) and 10 with UPPP (Group 2).
Results: The AHI went from 59±23 to 15±8 and to 10±6 in Group 1, similarly, in Group 2 it went from 59.3±23 to 13±5 and to 9.7±5.The minum oxygen saturation increased equally in both groups.Heart rate decreased more rapidly in CPAP patients and went from 86±7 to 74±5, after 30 days (p=0.002) *vs* 82±8 to 75±6 (p=0.077).Atrial premature complexes went from 48±53 (for hour) to 17±21 and to 17±22, after 30 and 90 days, in Group 1 (p=0.014) *vs* 42±45 to 36±43 and to 35±42 in Group 2 (p=0.078).Ventricular premature complexes went from 46±60 to 22±36 and to 23±35 in Group 1 (p=0.007) *vs* 44±54 to 36±55 and to 35±54 in UPPP patients (p=0.026).
Conclusions: Cardiac arrhytmias and heart rate decreased in both groups, although data obtained from CPAP patients revealed a statistical significance.We suggest to adopt a"wait and see" policy in treating arrhytmias in OSAS patients and to delay the use of potentially dangerous antiarrhythmic drugs.

P843
Proinflammatory cytokines in patients with congestive heart failure and sleep disordered breathing
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Background: In previous studies, significantly elevated levels of proinflammatory cytokines and markers of inflammation have been reported in patients with sleep disordered breathing (SDB) and in patients with congestive heart failure (CHF). Furthermore, it is known that the prevalence of SDB is high in CHF patients.
Objectives and Methods: To asses the involvement of proinflammatory cytokines and markers of inflammation in the pathophysiology of SDB in CHF patients, serum levels of interleukin-6 (IL-6), interleukin-8 (IL-8) and C-reactive protein (CRP) were determined in patients with CHF (left ventricular ejection fraction<45%). In a prospective manner 24 CHF patients underwent polygraphy and measurement of IL-6, IL-8 and CRP. The hypothesis was that CHF patients with SDB have significantly higher levels of IL-6, IL-8 and CRP than CHF patients without SDB.
Results: Significantly higher levels of IL-6 were found in CHF patients with SDB than in CHF patients without SDB (23,7 pg/ml vs. 3,9 pg/ml; p<0.05). No significant difference between those two groups was seen in the levels of IL-8 (p=0.21) and CRP (p=0.27).
Conclusions: IL-6 may play a role in the development of SDB in CHF patients. Restrictions of this study arise from the small number of subjects and from the general limitation of polygraphy vs. polysomnography.

P844
Use of high frequency peak in spectral analysis of heart rate increment to improve screening of obstructive sleep apnoea syndrome
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Background: Several studies have associated the power ratio of the very low frequency (VLF) component of heart rate increment (HRI), %VLFI, with OSA syndrome. Quantification of this phenomena from RR variations has allowed to develop an useful screening tool but autonomic dysfunction may alter the observation of VLF rhythms.
Methods: to overcome this limitation this study uses the high-frequency increment (HFI) peak in the HRI spectrum which peak corresponds to a respiratory influence on RR variations over the frequency range 0.2 to 0.4 Hz. We evaluated 288 consecutive patients referred for snoring, observed breathing cessation and/or daytime sleepiness. Patients were classified as OSAS if AHI>15/h. Synchronized ECG Holter monitoring allowed HRI analysis.
Results: Using a %VLFI threshold of 2.4, sensitivity for OSAS was 74.9%, specificity 51%, positive predictive value 54.9% and negative predictive value 71.7% (33 false negative subjects). Using simultaneous thresholds of 2,4 for %VLFI and 0.4 Hz for HFI peak position increased negative predictive value to 78.2% while maintaining specificity at 50.6%. Among 11 subjects with %VLFI<2.4% and HFI peak >0.4 Hz, 9 manifested AHI >15, (all with AHI>45).
Conclusions: We conclude that concomitant use of %VLFI and HFI spectral analysis reduces the number of false negative subjects.