[273] Obstructive sleep apnoea and risk of cardiovascular disease in men. A follow-up study

Joaquín Durán-Cantolla,[1] Felipe Aizpuru,[2] Ramón Rubio,[1] Lourdes Guerra,[1] Germán De La Torre,[1] Renny Nuñez,[1] Enma Socarrás.[1] [1]Sleep Unit. Respiratory Division, Hospital Txagorritxu., Vitoria., Álava., Spain; [2]Research Unit., Osakidetza., Vitoria., Álava, Spain

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Our group *(AJRCCM 2001; 163:1-5)* studied a cohort of general population men, from 30-70, by sleep tests to estimate the prevalence of obstructive sleep apnoea (OSA). This cohort has been follow-up to know if OSA is an independent risk factor of cardio-vascular disease (CVD).

Our town has around 250,000 habitants (99.9% in the National Health Service) and we have two hospitals. All of diagnoses are codified by specialists. After we obtained the approval by the Health Authorities, we consulted the CVD (ICD-9 390-459).

A total of 958 valuables subjects have been follow-up an average of 7.8 years (SD 0.8). We categorized the severity of OSA according of respiratory disturbance index (RDI) at base line, by quartiles, and we studied the incidence of CVD through the follow-up period. The table shows the prevalence, incidence and OR for CVD, depending of severity of OSA.

OSA and risk of CVD

RDI	Incidence of CVD (p < 0.0005)	OR (CI 95%)* for prevalence of CVD	OR (CI 95%)* for incidence of CVD
0-2 (n=216)	3.6%	1	1
3-6 (n=296)	6.2%	1.4 (0.6-2.9)	1.2 (0.5-2.9)
7-13 (n=232)	7.8%	1.7 (0.8-3.7)	1.4 (0.6-3.5)
>14 (n=214)	12.6%	1.8 (0.9-3.9)	1.9 (0.8-4.6)

*Adjusted by age, body mass index and alcohol and tobacco consumption

Conclusions: There is a dose-response relationship between OSA and CVD. However, the magnitude of the risk is not enough to be definitive, and more follow-up is necessary.

Funded by: FIS (01/157); Department of Health of Basque Govern 2001 and FEPAR 2001.

Supported by an unrestricted educational grant from Novartis.

[274] Association between obstructive sleep apnoea (OSA) and risk of systemic hypertension in men. A prospective study

Joaquín Durán-Cantolla,[1] Felipe Aizpuru,[2] Ramón Rubio,[1] Germán De La Torre,[1] Renny Nuñez,[1] Enma Socarrás,[1] Lourdes Guerra.[1] [1]Sleep Unit. Respiratory Division, Hospital Txagorritxu., Vitoria., Álava., Spain; [2]Resarch Unit, Osakidetza., Vitoria., Álava., Spain

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

We studied a cohort of general population, from 30-70, by sleep tests and blood pressure (BP), and OSA was an independent risk factor for SH in a cross sectional study *(AJRCCM 2001; 163:1-5).* The aim was to confirm these results in a prospective study. From 1,050 men studied at baseline, to date we contacted 851 (623 fully valuables) by questionnaires and three BP measures (age 57.0 ± 10.0). The mean of follow-up has been 7.8 ± 0.8 years. We categorized the severity of OSA according of respiratory disturbance index (RDI) at base line, by quartiles, and SH en two groups: stage 1: BP ≥ 140/90; and stage 2: BP ≥ 160/100. The prevalence of SH for stage 1 and 2 at baseline were 22.6% and 9.8%, and at follow-up were 45.8% and 27.2%, respectively. Among those free of SH at baseline, the incidence of SH was 34.1 % (stage 1) and 17.8% (stage 2). The table shows the OR for SH, depending of severity of OSA.

Association between OSA and the incidence of SH

RDI	0-2 (n=102)	3-6 (n=150)	7-13 (n=115)	> 13 (n=105)	P trend
Stage 1 (OR and CI 95%)*	1	0.8 (0.5-1.5)	1.0 (0.6-1.8)	1.3 (0.7-2.3)	0.014
Stage 2 (OR and CI 95%)*	1	1.1 (0.5-2.3)	1.2 (0.5-2.6)	2.0 (0.9-4.4)	0.003

*Adjusted by age, BMI, alcohol, tobacco and fitness

Conclusions: OSA is an independent risk factor for more severe etages of SH and there is a dose-response relationship.

Funded by: FIS (01/157); Department of Health of Basque Govern 2001 and FEPAR 2001.

Supported by an unrestricted educational grant from Novartis.

[275] Diastolic diurnal hypertension is underestimated in patients with unknown hypertension at time of sleep apnea diagnosis

J.L. Pepin,[1] J.P. Baguet,[2] L. Hammer,[2] H. Pierre,[2] P. Levy,[1] J.M. Mallion.[2] [1]Sleep Lab, Grenoble Univ Hosp, France; [2] Cardiology and Hypertension, Grenoble Univ Hosp - BP 217, Grenoble Cedex 09, France

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Objective: To characterize clinical and ambulatory blood pressure (BP) in newly diagnosed apneic patients (SAS) without known hypertension (HT).

Design and Methods: 41 unselected apneic patients (34 men), mean age = 48±12 years, mean BMI = 28.6±5.7 kg/m², mean AHI = 43±21/h. were included. BP was considered as normal by their general practioner and all of them were free of any medication for HT. Clinical BP was measured following the WHO recommendations and 24 hours ambulatory blood pressure monitoring (ABPM) was assessed using the Spacelabs ® monitor. Clinical HT was defined as a clinical systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg and ambulatory HT as a daytime SBP ≥ 135 mmHg and/or DBP ≥ 85 mmHg.

Results : Mean clinical BP was 133/86 mmHg and mean daytime ambulatory BP was 128/86 mmHg. 16/41 (39%) of the patients exhibited a clinical HT, and 62.5% (25/40) demonstrated a diurnal HT on ABPM. Isolated diastolic HT was found in 25% (4/16) of the patients with clinical HT and in 52% (13/25) of the patients with diurnal ambulatory HT. AHI tended to be higher in patients with isolated diastolic HT than in patients with systolo-diastolic HT (50±25 vs 38±23/h).

Conclusions: HT is largely under-diagnosed particularly in apneic patients unknown to be hypertensive. Isolated diastolic HT is more prevalent in SAS than in essential HT. Using 24 hour ABPM allows to identify one third of HT undiagnosed by clinical measurement. Conversely, SAS should be evoked in patients with isolated diastolic HT.

Supported by an unrestricted educational grant from Novartis.

[276] Influence of obstructive sleep apnea in coronary artery disease: an 8-years-follow-up

Gerrit C. Hagenah,[1] Elif Gueven,[1] Stefan Andreas.[1] [1]Cardiology and Pneumology, Georg-August-University, Goettingen, Germany

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Mortality from coronary artery disease (CAD) shows a decline attributed to improvements in treatment of CAD. Still the mortality rate remains high in this group of patients. Obstructive sleep apnea (OSA) was found to be an independent risk factor for arterial hypertension and myocardial hypertrophy. Little is known about prevalence and prognostic significance of OSA in CAD. Therefore cardiovascular complications (CVC) in 50 patients with CAD were investigated in patients with versus without OSA in an 8-years-follow-up. The 25 patients in the OSA-group had a RDI of 19.4 ± 6.7/h compared to a RDI of 5.1 ± 2.3/h in the nonOSA-group ($p< 0.00001$). Body-mass index was also higher in the OSA-group (27.8 ± 4.2 vs 25.7 ± 3.0 kg/m²; $p<0.05$). After a time intervall of 8 years 5 patients died in the OSA-group due to CVC and 2 in the nonOSA-group ($p<0.1$). The number of patients having CVC regarding myocardial infarction and cerebral insult were 21 in the initial OSA-group versus 18 in the nonOSA-group ($p<0.1$). The proportion of the OSA-group to the nonOSA-group, regarding mortality and CVC and a shift in the surviving patients from nonOSA-group to OSA-group and the other way around, shows a significant increased risk (logrank test) of CVC in the OSA-patients ($p<0.05$). Allthough the number of patients included in this study is small the influence of OSA appears to be a possible factor explaining an increased mortality in CAD.

Supported by an unrestricted educational grant from Novartis.

[277] Circadian prevalence of Cheyne-stokes respiration in patients with congestive heart failure

Thomas Brack,[1] Irene Thuer,[1] Christian F. Clarenbach,[1] Olivier Senn,[1] Georg Noll,[2] Erich W. Russi,[1] Konrad E. Bloch.[1 1] Division of Pulmonary Medicine, University Hospital, Zurich, Switzerland; [2]Division of Cardiology, University Hospital, Zurich, Switzerland

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Cheyne-Stokes respiration (CSR) is not only a symptom of severe congestive heart failure (CHF), but may also deteriorate CHF through sympathetic over-stimulation. Nevertheless, the circadian prevalence of CSR in patients with CHF is not well known. Thus, we recorded the breathing pattern in patients with CHF unobtrusively with a portable monitoring system. 10 outpatients with CHF (left ventricular ejection fraction 28 ± 8 %) were studied during 24 h of their usual activities with a light-weight monitoring system (LifeShirt, Vivometrics, USA). The elastic shirt incorporates an integrated respiratory inductive plethysmograph, a pulse-oximeter, an ECG and an accelerometer. Recordings were analyzed for CSR during day and night, separately. The absolute (min) and relative (%) times of CSR, the duration of a CSR cycle (cycle time) and the apnea-hypopnea index (AHI) are summarized in the **Table**. All patients displayed CSR during more than 10% of the nighttime, and 2 and 5 patients had CSR during more than 10% and 5% of the daytime, respectively. **In summary**, we found a considerably higher prevalence of circadian Cheyne-Stokes respiration than previously reported in patients with CHF. Improved recognition of CSR by novel monitoring techniques may allow to better assess the prognosis and optimize the therapy of CHF.

	CSR (min)	CSR (%)	Cycle time (sec)	AHI (cycles/h)
Daytime	45 (11 - 179)	6 (1 - 19)	58 (54 - 60)	4 (1 - 9)
Nighttime	183 (103 - 263)	25 (18 - 47)	54 (52 - 60)	17 (11 - 42)

Values are medians (25% to 75% quartile range)

Supported by an unrestricted educational grant from Novartis.

[278] Left ventricle structure and function in patients with obstructive sleep apnoea (OSA)

Jaroslaw Kober,[1] Robert Plywaczewski,[2] Witold Z. Tomkowski,[1] Anna Fijalkowska,[1] Jan Zielinski.[2] [1]Internal Medicine, Institute of TB and Lung Diseases, Warsaw, Poland; [2]II Department of Respiratory Medicine, Institute of TB and Lung Diseases, Warsaw, Poland

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

The aim of this study was to investigate: 1. left ventricle (LV) structure and function and, 2. influence of obesity (OB) and systemic hypertension (HT) on changes in LV in the pts with OSA. 118 pts were involved in the study (98M and 20 F, mean age 48.5 ± 8.4 yr). The pts were divided into following groups: 1.obese with OSA and HT (22 pts), 2.obese with OSA, without HT (20pts), 3.pts with OSA without OB and HT (21pts), 4.obese with HT but without OSA (18pts), 5.obese without OSA as well as HT (17pts), 6.control group - healthy subjects without OSA, OB and HT (20). Echocardiography was used to assess: LV diameter (LVD), intraventricular septum (IVS), LV mass index (LVMI), ejection fraction (EF), mitral early diastolic velocity to atrial velocity ratio (Ev/Av). Results:

	IVS(mm)	LVD(mm)	LVMI(g/m²)	Ev/Av	EF(%)
OH	11.9±1.8	64.6±5.9	173.8±43	0.89±0.2	49.0±5.2
ON	10.8±1.1	57.9±6.4	120.8±33	0.87±0.2	50.2±5.7
OC	9.0±1.1	55.2±4.6	117.5±19	1.0±0.24	58.2±6.7
GH	10.4±1.2	59.2±5.7	138.9±26	0.9±0.18	55.7±5.0
GN	9.5±1.1	57.6±6.7	121.4±34	1.15±0.35	54.5±6.4
ZZ	8.2±0.7	48.0±3.4	79.2±12	1.76±0.13	63.1±4.2
p<0.05	1,3,4,5	1,2	1,2	2,6	2,6,7,8

Sign.changes between: 1.OH/all groups, 2.ZZ/all groups, 3.ZZ/GH, 4.ZZ/ON,

5.GH/OC, 6.GN/OH, GN/OH, GN/GH, 7.OC/OH, OC/ON, 8.OH/GH, ON/GN, ON/GH, OC/GN

Conclusions: OSA seems to be associated with enlarement and failure of LV esspecially in pts with obesity and systemic hypertension.

Supported by an unrestricted educational grant from Novartis.

[279] Severe bradyarrhythmias in pts with sleep apnea hypopnea syndrome (OSAHS) and the effect of CPAP treatment. A long-term study with the use of implantable loop recorders

Sophia E. Schiza,[1] Emmanouel N. Simantirakis,[2] Maria E. Marketou,[2] Stavros I. Chrysostomakis,[2] Eirene Lampiri,[1] Panayiotis E. Vardas,[2] Nikos M. Siafakas.[1] [1]Dept. of Pneumonology, Sleep Disorders Unit, Heraklion, Greece; [2]Dept. of Cardiology, University General Hospital, Heraklion, Greece

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Several studies have described severe bradyarrhythmias in pts with OSAHS. There is great variation in their incidence, mainly because they were evaluated by means of short-term Holter ECG recordings. The aim was to clarify the incidence of extreme bradyarrhythmias in such pts and to evaluate the effect of long-term CPAP treatment.
Methods: We prospectively enrolled 23 pts (16M, 7 W) with the diagnosis of OSAHS. In all of them an implantable loop recorder (ILR) with the ability to record the heart rhythm for 18 months was implanted. In this part of the study, we looked for cardiac pauses > 3 sec, two months before the treatment of CPAP was initiated and two months after. During the same time interval, the pts underwent two 48 hour Holter recordings.
Results: During the 2 month period before CPAP, the ILR revealed that 35 % of pts demonstrated extreme bradyarrhythmias. The percentage by 48 hour Holter was only 9%. In the two months following the initiation of CPAP, the incidence of pauses recorded by the ILR was reduced to 13% (p=0.06) and by 48 hour Holter, to 4% (p=NS). The Holter recorded pauses only in pts with very frequent episodes recorded by the ILR.
Conclusion: About one third of pts with OSAHS evidence long periods of asystole, which tend to reduce by CPAP treatment. Holter used until now seem unable to precisely record the incidence of severe bradyarrhythmias and the effect of CPAP treatment.

Supported by an unrestricted educational grant from Novartis.

[280] No benefit of overdrive atrial pacing for treatment of obstructive sleep apnoea syndrome (OSAS)

J.L. Pepin,[1] P. Defaye,[2] C. Deschaux,[1] Y. Poezevara,[1] P. Levy.[1] [1]Sleep Laboratory, Univ Hosp, Grenoble, France; [2]Cardiology, University Hospital, Grenoble, France

Sunday, September 28, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders and cardiovascular disease (10:45 AM-12:45 PM) Hall N

Background: Garrigues et al., overdriving atrial pacing (15 beats per minute faster than mean baseline nocturnal heart rate), reported a half reduction in sleep apnoea severity (N Engl J Med 2002, 34:404-12).

Design and Methods: We studied 15 unselected patients without cardiac failure (11 men; mean 71± 10 years) who had received permanent atrial-synchronous ventricular pacemakers for symptomatic brady-arythmias and unknown to have OSAS. All patients underwent three polysomnographic evaluations one month apart. The first night was performed for baseline evaluation and then, in random order, one night in spontaneous rhythm and one in pacing mode with atrial overdrive (15 beats per minute faster than mean baseline nocturnal heart rate).

Results: OSAS was highly prevalent in this population as 13/15 (87%) and 10/15 (67%) of the patients exhibited an AHI more than 15 and more than 30/hour respectively (mean apnoea + hypopnoea index (AHI) = 48±28/h, mean BMI = 27.5±3.2 kg/m²). The nocturnal spontaneous rhythm was 58± 6 beats per minute at baseline as compared with 73±7 beats per minute with atrial overdrive pacing (P< 0.002). AHI was 49±26 in spontaneous rhythm, as compared with 52±24 with atrial overdrive pacing (P=NS). Overdrive pacing changed none of the respiratory indices, sleep fragmentation or sleep structure parameters.

Conclusions: Patients treated by pacemakers for symptomatic brady-arythmias should be screened for sleep apnoea. We failed to confirm the data of Garrigues et al. (N Engl J Med 2002, 346:404-12) regarding the efficacy of overdrive atrial pacing in OSAS.

Supported by an unrestricted educational grant from Novartis.

[P647] Relation between heart rate variability (HRV) and sleep stages

Hacer Gundogdu,[1] Caglar Cuhadaroglu,[1] Erdem Kasikcioglu,[2] Ebru Durgun,[1] Turhan Ece.[1] [1]Chest Department, Medicine Faculty of Istanbul, Istanbul University, Istanbul, Turkey; [2]Sport Medicine, Cardiologist, Medicine Faculty of Istanbul, Istanbul University, Istanbul, Turkey

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Clinical aspects and consequences of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/15

Recently heart rate variability (HRV) has been used to asses the changes in the autonomic nervous system. It has two parts named as low frequency (LF) and high frequency (HF). LF indicates the sympathetic nervous system dominance and HF does the parasympathetic nervous system. To show the differences of HRV in between the sleep stages will aid the assessment of sleep studies. In our study the relationship between the sleep stages and HRV has been evaluated. Sixteen cases who were performed full polysomnography at our laboratory due to snoring and found to have no sleep apnoea or upper airway resistance syndrome (RDI:<5) were included in the study. HRV measurements were made by using Somnologica software. LF and HF parts of HRV were measured separately for every sleep stage and LF/HF ratios between stages were compared. LF/HF ratio is 0, 9 in wakefulness and REM sleep but it is 0, 2 in slow wave sleep (stage3/4).There have been found statistically significant difference in LF/HF ratio among wakefulness and all sleep stages. The same difference has also been found between the REM and NREM sleep. (p<0, 01) In our study we showed that while there is dominance of parasympathetic system activity in slow wave sleep, in REM sleep just like in wakefulness sympathetic nervous system activity is dominant. We concluded also, the HRV difference could be used to separate wakefulness, REM and NREM sleep stages.

_____

Supported by an unrestricted educational grant from Novartis.

[P657] Hypertension, sex and OSAS

Michal Bednarek,[1] Robert Plywaczewski,[1] Luiza Jonczak,[1] Jan Zielinski.[1] [1]Department of Respiratory Medicine, National Research Institute of TB and Lung Diseases, Warsaw, Poland

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Clinical aspects and consequences of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/15

Objectives: To assess relations between OSAS, sex and arterial hypertension in a random population sample. *Materials and methods:* Study population, 356 males (53%) and 320 females (47%), mean age 56.6 ±8.2 (range 41-72) years, was selected from voting list for parliamentary election in Warsaw. History of arterial hypertension (AH) and drugs usage was taken. Overnight polysomnography and repeated blood pressure measurements were recorded. *Results:* OSAS was diagnosed in 59 (8.8%) males and 17 (2.5%) females. Mean AHI was 25.3, $SaO_{2\ mean}$92.1%, $SaO_{2\ min}$ 76.9%, and T90 of 18.9% did not differ between males and females. Arterial hypertension was diagnosed in 173 (48.6%) males and 135 (42.2%) females (NS). 55% of males and 76% of females were treated for AH ($p<0.001$), using 1.7 ±0.8 drugs. Odds ratio for coincidence of OSAS and AH in males was 1.9 (CI 1.3-2.9), in females 6.9 (CI 1.9-24.7). Arterial hypertension was present in 59% of males and 82% of females with OSAS. Multivariate regression analysis showed significant role of sex, age, body composition, and snoring, but not of AHI or oxygenation in the development of AH. *Conclusions:* Arterial hypertension was more frequent in OSAS population, especially in women. It was not related to severity of OSAS.

Supported by an unrestricted educational grant from Novartis.

[P662] Obstructive sleep apnoea and risk of systemic hypertension in the elderly. A cross sectional study

Joaquín Durán-Cantolla,[1] Felipe Aizpuru,[2] Ramón Rubio,[1] Germán De La Torre,[1] Renny Nuñez,[1] Enma Socarrás,[1] Joan Sallés.[3]
[1]Sleep Unit. Respiratory Division, Hospital Txagorritxu, Vitoria, Álava, Spain; [2]Resarch Unit, Osakidetza, Vitoria, Álava, Spain; [3]School of Pharmacology, Basque Country University, Vitoria, Álava, Spain

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Clinical aspects and consequences of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/15

It has been proved an association between obstructive sleep apnoea (OSA) and systemic hypertension (SH) in the middle-age people. However, this association has not been appropriately studied in the elderly.

We present the results of a cross sectional study of the general population [405 subjects from 71 to 100 years (232 men and 173 women)]; mean 81 yr (SD 7), designed to determine SH and OSA by standard polysomnography. The prevalence of SH was 60% of men and 77% of women, and 16% of men and 34% of women had and BMI > 30mg/Kg². The table shows the association between de apnoea-hypopnoea index (AHI) and SH, controlled by confounding variables.

Conclusions: There is an association between OSA and SH in the elderly. However, the magnitude of this association is less than in the middle-aged people, and it is only present for high levels of AHI. These results suggest that OSAH in the elderly might have less cardiovascular consequences than in the middle-ages.

Funded by: FIS (93/0053), (95/1176), (97/0844; FEDES 2FD97/0766-C03-33; Department of Health. Basque Govern 1992 and 1996 and FEPAR 1996,1997.

Association between OSAH and SH

AHI	n	Crude OR (CI 95%)	Adjusted OR* (CI 95%)
< 4.9	75	1.0	1.0
5 - 14.9	111	1.0 (0.6-1.9)	0.9 (0.4-1.8)
15 - 29.9	119	0.8 (0.4-1.4)	0.6 (0.3-1.2)
> 30	100	2.0 (1.0-3.9)	1.8 (0.8-4.0)

*By age, BMI, neck circunference, alcohol and tobacco consumption

Supported by an unrestricted educational grant from Novartis.

[P663] Hypertension in patients with sleep disordered breathing

Igor Jurikovic, Zuzana Friedbergová. Clinic of Respiratory Medicine and Chest Surgery, Teaching Hospital Bulovka, Prague 8, Czech Republic, Czech Republic; Department of Ear, Throut and Nose, Teaching Hospital Bulovka, Prague 8, Czech Republic, Czech Republic

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Clinical aspects and consequences of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/15

Aims: To find out the occurrence of hypertension in different groups of patients with sleep disordered breathing . Further we wanted to know if it is influeced by straight relations of sleep diserdered of breathing or confounding factors.

Materials and methods: We investigated 87 adults patients, aged 23- 64 years, referred to the sleep laboratory with suspected sleep apnoea syndrome and snoring. Patients were divided in 3 same groups with 29 patients. Group A patients mostly with upper airway resistence syndrome and mild sleep apnoe syndrome, Group B with severe sleep apnoe syndrome and Group C with sleep apnoe syndrome and daytime hypoxaemia and hypercapnia.

We took medical history, provide measuring of blood pressure and sleep monitoring with screening device MESAM4. Next day we provide lung function testing and investigating of blood for biochemical analysing (blood glucose, cholesterol, high density lipoprotein, low density lipoprotein, triglycerides).

Results: Patients in Group C had the highes occurrence of hypertension (75%) than patients in Group A and B (11% resp.38%). The confounding factors were also much abnormal in patients in Group C. The biggest differences in following factors were in parameters of sleep breathing.

Conclusion: The occurrence of hypertension is depending on confounding factors. We assume this relation is supported by contemporary presens of sleep disordered breathing.

Supported by an unrestricted educational grant from Novartis.

[P665] Obstructive sleep apnea and cardiovascular disease- A retrospective study

Marta Drummond, J. Carlos Winck, Sofia Pereira, Augusta Machado, Adelina Amorim, Joao Almeida, J. Agostinho Marques.
Pulmonology Department, Hospital Sao Joao, Porto, Portugal; Epidemiology Department, Hospital Sao Joao, Porto, Portugal

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Clinical aspects and consequences of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/15

Introduction- Obstructive Sleep Apnea (OSA) is associated with effects in cardiovascular physiology. Some studies have connected this pathology with Cardiovascular Disease (CVD).
Aim- Determine the prevalence of CVD in patients with different severity degrees of OSA and verify the association between CVD and OSA.
Materials and Methods- Retrospective study based on data from 155 consecutive patients with OSA referred to a Sleep Disordered Breathing Unit in a teaching Hospital during the year of 2001.
Results- The majority of patients were male (83,2%), mean age was 53,6 ± 11,9 years. Severe OSA was observed in 52,9% patients, moderate in 20% and mild in 27,1% (mean AHI of 35,2 ± 23,8/hour). The majority of patients (67,1%) were obese (Body Mass Index- BMI>30). The mean BMI was 33,1 ± 6,34. CVD was present in 52,3% cases, being Arterial Hypertension (AH) the most common disease (45,8%). Acute Myocardial Infarction appeared in 6,5% and Angina in 3,9% cases. Lipid Disorders were reported by 31% patients and 11% reported Diabetes mellitus. The majority (51,3%) of patients smoked. The severity of OSA was significantly higher in patients with AH (p= 0,0496) and significantly lower in patients who developed Cerebrovascular Disease (p= 0,0369).
Conclusions- The studied population presented a high prevalence of CVD, being the severity of OSA significantly higher in patients with AH. The increased prevalence of CVD in patients with OSA has been documented in epidemiologic recent studies but the mechanisms that lays beneath keep uncertain.

Supported by an unrestricted educational grant from Novartis.

[P671] Blood pressure and apnea induced skin circulatory response after two antihypertensive agents in OSA patients

Ding Zou,[1] Ludger B. Grote,[1] Derek N. Eder,[2] Yuksel Peker,[1] Jan A. Hedner.[1] [1]Sleep Laboratory, Department of Pulmonary Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden; [2]Institute of Clinical Neuroscience, Sahlgrenska University Hospital, Gothenburg, Sweden

Sunday, September 28, 2003, 1:30 PM, Thematic Poster Session: Management of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/16

Objective: To compare the effect of Doxazosin (DOXA, an α-receptor inhibitor) and Enalapril (ENAL, an ACE inhibitor) on blood pressure and apnea induced digital skin vasoconstriction in hypertensive obstructive sleep apnea (OSA) patients.
Methods: Double-blind cross-over evaluation of DOXA and ENAL (target dosage 8 and 20mg o.d., respectively) in 14 hypertensive OSA patients (age 55±1.7 yrs, BMI 30.1±0.95kg/m²). Two-week treatment periods were separated by three weeks wash-out. Monitoring included 24-hour blood pressure (BP), continuous nocturnal non-invasive BP, peripheral arterial tonometry (PAT) and polysomnography at the end of each treatment period. Skin circulatory changes during and after apnea were expressed as PAT ratio (%) between 3 nadir pulse amplitudes during 21 sec after apnea and 3 peak pulse amplitudes during apnea. Ten events (highest systolic pressures) were selected during nREM and REM sleep.
Results: Office systolic BP (p=0.047) but not diastolic BP was lower after ENAL at the end of treatment while 24-hour BP (or day/night portions) did not differ. The PAT ratio after DOXA was 40.1± 9.8% and after ENAL 32.0±11.2% (p=0.006). Differences encompassed both nREM and REM sleep. There was no difference in OSA severity between treatments.
Conclusion: Skin vascular responses following apnea are at least in part α-receptor mediated. The antihypertensive effect of DOXA was proportionally weak in hypertensive OSA patients and the mechanism behind this phenomenon will need further analysis.

Supported by an unrestricted educational grant from Novartis.

[P1181] The effect of hypoxia and hypercapnia on chemoreceptor and baroreceptor reflexes - a mechanism for hypertension in obstructive sleep apnoea

Claire M. Bowker,[1] Victoria Cooper,[2] Stanley B. Pearson,[1] Roger Hainsworth,[2] Mark W. Elliott.[1] [1]Department of Respiratory Medicine, Leeds Teaching Hospitals NHS Trust, Leeds, West Yorkshire, United Kingdom; [2]Academic Unit of Cardiovascular Medicine, University of Leeds, Leeds, West Yorkshire, United Kingdom

Sunday, September 28, 2003, 2:45 PM, Poster Discussion: Sleep related breathing disorders and control of breathing (2:45 PM-4:45 PM) Hall L

We have reported that asphyxia resets the baroreceptor - vascular resistance reflex towards higher blood pressures. This study was designed to determine whether this effect was due to the action of hypoxia on peripheral chemoreceptors or hypercapnia on central chemoreceptors.

11 controls (aged 20 - 55, 6 male) were studied. Stimuli of -40 to +60 mmHg were applied to the carotid baroreceptors using a neck chamber device. Vascular resistance responses were assessed in the forearm (change in BP / change in brachial flow velocity) and cardiac responses measured (changes in RR interval). Stimulus response curves were defined during (i) breathing air, (ii) hypoxia and (iii) hypercapnia. Sigmoid functions were applied to the curves and the maximum differential (equivalent to peak gain) and the corresponding carotid pressure (equivalent to 'set point') were determined.

Hypoxia resulted in an increase in heart rate but no change in mean BP. Peak gain response to baroreceptor stimulation was significantly reduced ($p<0.05$) but there was no effect on 'set point'. Hypercapnia resulted in a decrease in heart rate and a significant increase in BP. There was no change in the gain of the vascular responses but 'set point' was significantly increased ($p<0.02$). Neither hypoxia or hypercapnia effected the cardiac responses.

These results suggest that the interaction of both peripheral and chemoreceptors on baroreceptor function may contribute to promoting hypertension in patients with OSA.

Supported by an unrestricted educational grant from Novartis.

[P1775] Spectral analysis and detrended fluctuation analysis on heart rate variability recognises sleep stages and sleep apnea

Thomas Penzel,[1] Jan W. Kantelhardt,[2] Armin Bunde,[2] Volker Gross,[1] Karl Kesper,[1] Heinrich F. Becker,[1] Claus Vogelmeier.[1] [1] Division of Pulmonary Diseases, Depart. of Internal Medicine, Hospital of Philipps-University, Marburg, Germany; [2]Institute for Theoretical Physics, Justus-Liebig University, Giessen, Germany

Monday, September 29, 2003, 1:30 PM, Thematic Poster Session: Diagnosis of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/13

Sleep can be regarded as a standardised test condition for the autonomous nervous system because extrinsic factors are reduced. Heart rate is a marker for the autonomous nervous system and is modulated by sleep stages and by sleep disordered breathing.We applied two different and complementary methods for the analysis of heart rate variability in 14 healthy volunteers, 33 patients with moderate and 31 patients with severe sleep apnea. Sleep stages were reduced to wake, light sleep, deep sleep and REM sleep. The spectral parameters VLF, LF, HF, LF/HF were computed using fourier analysis. Scaling parameters alpha1 and alpha2 were derived by applying detrended fluctuation analysis (DFA) in order to detect long range correlations within R-R interval series. Both parameter sets were evaluated using discriminant analysis on the basis of one value per person and per sleep stage. Spectral analysis could correctly identify 69.7% of apnea classification and 54.6% of sleep stage classification. DFA could correctly identify 74.4% resp. 85.0% of classifications. In conclusion, spectral analysis can detect sleep apnea with a moderate accuracy, but it is not convincing in detecting sleep stages. DFA can detect sleep stages much better than spectral analysis and is also better in detecting sleep apnea. This reflects that sleep apnea causes more cyclical changes in heart rate whereas sleep stages cause different beat-to-beat regulations of heart rate.

Supported by an unrestricted educational grant from Novartis.

[P1777] Obstructive sleep apnea syndrome (OSAS). Value of cardiovascular markers (CM) to detect arousals

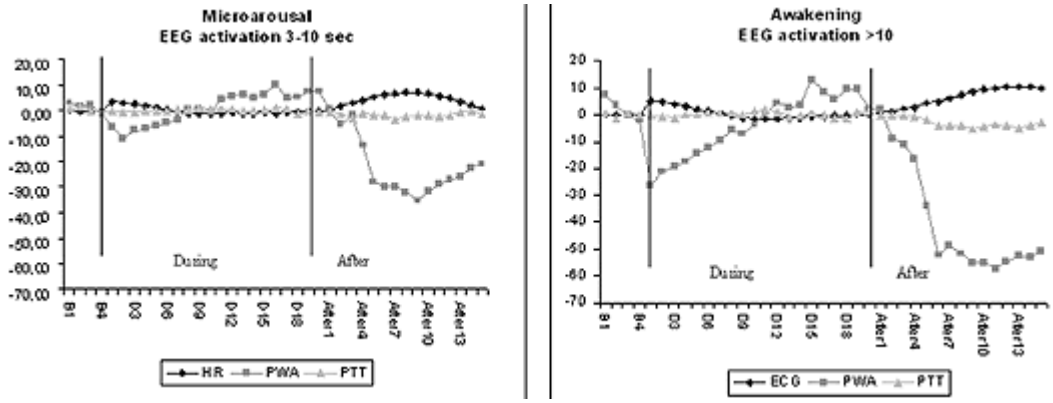
José Haba,[1] Jean G. Frey,[1] Georges Darbellay,[2] Jean M. Tschopp.[1] [1]Sleep Lab, CVP, Montana; [2]EPFL, Lausanne, Switzerland

Monday, September 29, 2003, 1:30 PM, Thematic Poster Session: Diagnosis of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/13

We analysed the relationship between CM, such as heart rate (HR), pulse transit time (PTT) and pulse wave amplitude (PWA), and sleep related respiratory events (RE) in 50 randomly chosen RE from 5 patients. These 3 CM were measured 15 sec pre-, per and 15 sec post- RE such as apnea (A), hypopnea (H) and respiratory effort-related arousal (RERA). To describe the CM evolution pattern around event, data were analysed as follows: The mean of the 5 first CM values prior to the onset of RE was substracted from all data points. Then we tried to known wheter these CM could discriminate EEG microarousals (Ma, 3-10 sec) or awakenings (Aw, > 10 sec).

RE were associated with relative bradycardia and increased PTT and PWA. After events there were always transient tachycardia and drops in PTT and PWA. There were no significant differencies in the variation of each CM regarding to A, H or RERA. Changes in PWA were the greatest of the 3 CM.

The CM variations were significantly greater when the respiratory event was associated with Aw than Ma for HR and PWA (p= .026 and p= .029, **Fig**). In conclusion HR and PWA seems better discriminants of arousals than PTT.Variations of cardiovascular markers around respiratory events might contribute to quantify sleep fragmentation provided developement of automated algorythms. (Click to see figure 1)



Figure

_____

Supported by an unrestricted educational grant from Novartis.

[P1788] The correlation between sleepiness, disease-specific quality of life and the prevalence of sleep related disorder in patients with congestive heart failure

Sylvia L. Montalvan Dobmayr,[1] Gabriele Gloger,[1] Guenther Riegger,[1] Friedrich C. Blumberg,[1] Michael Pfeifer.[1] [1]Innere Medizin II, Universitätsklinik Regensburg, Regensburg, Germany

Monday, September 29, 2003, 1:30 PM, Thematic Poster Session: Diagnosis of sleep related breathing disorders (1:30 PM-3:30 PM) Hall Z/13

Objective: Sleep-related breathing disorders [SRBD] are common in patients with chronic heart failure [CHF]. The purpose of this study is to determine the possibility of predicting the existence of an SRBD in CHF patients by using self-administered questionnaires about sleep propensity and disease-specific quality of life.

Methods: Settings: Clinic of cardiology of the University Hospital in Regensburg, Germany. 107 ambulatory CHF-patients (ejection fraction <40% (mean 28%, range 10-39%), age 62.6±10.5, 84.1% men, body mass index [BMI] 28.0±4.8 kg/m²) were examined by an apnoe screening measuring nasal airflow by a canula, thorax excursions by an effort belt, oxygen saturation and pulse by a flexible finger oximeter (Stardust®). The sleep propensity was assessed by a simple questionnaire measuring the general level of daytime sleepiness (Epworth Sleepiness Scale [ESS]). To evaluate, the disease-specific quality of life the Minnesota Living with Heart Failure questionnaire [M-LWHF] was used.

Results: 78 (72.9%) individuals had positive screening results (apnoe hypopnoe index [AHI] or oxygen desaturation index [ODI] >15/h). The scoring in the ESS (mean 6.8±3.7) and M-LWHF (mean 34.8±23.1) correlated significantly ($p<0.02$). However neither ESS nor M-LWHF correlated with AHI or ODI ($p>0.05$).

Conclusions: The questionnaires ESS and M-LWHF are not sufficiently suitable for detecting sleep-related breathing disorders in CHF patients.

Supported by an unrestricted educational grant from Novartis.

[2246] Theophylline increases muscle sympathetic activity in patients with congestive heart failure

Stefan Andreas, Andre Delekat, Lars Lütje, Gerrit Hagenah, Hartwig Reiter. Cardiology and Pneumology, Universitätsklinikum Göttingen, Göttingen, Germany

Monday, September 29, 2003, 2:45 PM, Oral Presentation: Management of sleep related breathing disorders (2:45 PM-4:45 PM) Hall N

Cheyne-Stokes respiration (CSR) in patients with chronic heart failure (CHF) causes oxygen desaturation, impaired sleep, sympathetic activation and thereby likely increased mortality. Therefore treatment of CSR with oxygen, CPAP or theophylline gains growing interest (Javaheri New Engl J Med 1996; 335: 562-7). However, theophylline as an adenosine receptor antagonist might increase sympathetic activity.

Therefore we aimed to investigate whether theophylline i.v. increases sympathetic activity in patients with CHF and matched controls in a placebo controlled setting. Muscle sympathetic nerve activity (MSNA) was evaluated by microneurography of the peroneal nerve in 22 patients and 22 controls (age 49±2 (mean±SEM) years; BMI 25±1, ejection fraction 29±1% (patients only)). MSNA was 37±3 burst/min in the patients and 26±3 burst/min in the control group ($p< 0.01$).

Theophylline i.v. over 30 min resulted in a plasma concentration of 53±2 umol/l, an increase in MSNA (35±4 vs 41±4%; $p< 0.0001$) an increase in heart rate (65±2 vs 68±2 /min; $p< 0.001$), in mean blood pressure (83±3 vs 87±3 /min; $p< 0.0001$), in minute ventilation (5,1±0,4 vs 5,8±0,4 l/min; $p< 0.001$) and concomitant reductions in arterial pCO_2. Furthermore there was an increase in plasma renin but not in norepinephrine concentration. There were no significant effects of placebo and no significant between-group-interactions (i.e. CHF versus controls) in ANOVA.

These findings suggest that theophylline increases sympathetic nervous activity and therefore should be used with great caution in patients with CHF and CSR.

Supported by an unrestricted educational grant from Novartis.

[2247] Effects of bi-level therapy in heart failure patients with Cheyne-Stokes respiration

Ingo U. Fietze, Alexander Blau, Claudia Moeller, Martin Glos, Heinz Theres, Christian Witt. Pulmology, Medical Sleep Center, University Hospital Charite, CCM, Berlin, Germany

Monday, September 29, 2003, 2:45 PM, Oral Presentation: Management of sleep related breathing disorders (2:45 PM-4:45 PM) Hall N

Patients with reduced left ventricular function (LVEF) of the heart associated with Cheyne-Stokes respiration (CSR) during sleep have an increased mortality risk. Aim of this study was to analyse the effects of a bi-level therapy in relation to LVEF, sleep macrostructure and daytime sleepiness.

Method:

38 patients (mean age 58 ± 10.8 years, mean BMI 27.6 ± 3.6 kg/m²) with previously known CSR were included. The mean AHI detected in one diagnostic polysomnography (PSG) was 30.5 ± 18.4/h. Following a randomised application of bi-level-therapy either in spontan/timed (S/T) or in AutoSetCS® mode 31 patients underwent a control PSG and cardiac and clinical investigations 6 weeks after.

Results:

In 31 patients the AHI decreased up to 8.6 ± 9.6 in patients using the AutoSetCS® mode and up to 9 ± 7.8 /h in patients using the S/T mode. The mean LVEF increased from 24.9 ± 7.9 to 28.3 ± 9.8 % for the whole bi-level group ($p<0.01$). The increase in the AutoSetCS® group was from 24.3 ± 6.7 to 26.3 ± 8.7, and in the S/T group from 25.3 ± 9 to 29.8 ± 10.6 %.

The score from the Epworth Sleepiness Scale decreased during therapy from 9 ± 4 to 7 ± 3 (AutoSetCS®) and from 11 ± 4 to 9 ± 4 (S/T), respectively. Sleep macrostructure did not change significantly.

Conclusion:

In patients with reduced LVEF, the bi-level therapy, both in S/T- and AutoSetCS mode, is effective with regard to treatment of CSR, improvement of LVEF, and reduction of daytime sleepiness.

Supported by an unrestricted educational grant from Novartis.

[2530] Body movements and heart rate in habitual snoring children

Luana Nosetti, Luigi Ferrini Strambi, Vincenza Castronovo, Carlo Marazzini, Luigi Nespoli. Pediatric Department, Insubria University, Varese, Varese, Italy; Sleep Disorders Center, HRCCSH San Raffaele, Milano, Milano, Italy

Tuesday, September 30, 2003, 10:45 AM, Oral Presentation: Sleep related breathing disorders in children (10:45 AM-12:45 PM) Hall H

Aim of our study was to evaluate in habitual snoring children heart rate (HR) variability and changes of body position during sleep in relation to respiratory parameters and age. 743 children 450 M,293 F, mean 4.9+-2.2 yrs were evaluated by 1-nught ambulatory recording (MESAM IV) that monitors heart rate, body position, snoring and oxygen saturation. Snoring %=/>15 was considered pathological: 211 children were snorers (SN) while 532 non-snorers (non-SN). Mean HR was significantly higher in SN compared to non-SN 96.2+-72.4 vs 85.9+-12.4; p=.002 as well as the HR variation index in relation to O2 desaturations (HRVI) 41.1+-17.5 vs 36.2+-20.2; p=.002 Min SaO2% was 84.4+-8.0 in SN and 88.1+-6.0 in non-SN p=.001 Mobility index resulted 7.2+-4.3 in SN and 5.9+-4.1 in non-SN p=.001. Linear regression analysis shows that there is a decrease of mean HR as well as of the mobility index with increasing age. Using the two-way analysis of variance the effect of age, pathological snoring and their interaction were tested: Min SaO2% was the only parameter influenced by pathological snoring F=13.6; p=001, whereas HRVI,HR and mobility index were significantly affected by age HR: F=17.6, p=.001; HRVI: F=7.2, p=.008; mobility index: F=47.5,p=.001. The impact of snoring on HR is lower in children compared to adults. Age resulted to be the strongest factor to explain cardiac changes and mobility during sleep and should always be considered when evaluating snoring effect in children.

Supported by an unrestricted educational grant from Novartis.

[P2944] Respiratory mechanics study; breathing pattern; and sleep study in patients with congestive heart failure and nocturnal cheyne-stokes respiration

Luis V.F. Oliveira,[1] Alberto Braghiroli,[2] Lorenzo Apendini,[2] Paola A. Lanfranchi,[2] C.F. Donner.[2] [1]Instituto de Pesquisa & Desenvolvimento - IP&D, Universidade do Vale do Paraíba - UNIVAP, São José dos Campos, São Paulo, Brazil; [2]Salvatore Maugeri Foundation, Medical Centre of Veruno, Veruno, Novara, Italy

Tuesday, September 30, 2003, 1:30 PM, Thematic Poster Session: Sleep related breathing disorders and other diseases (1:30 PM-3:30 PM) Hall Z/16

It is well-established that in patients with congestive heart failure (CHF), respiratory muscle strenght is reduced. The aim of the present study was to describe the respiratory mechanics and the muscle strength in patients with congestive heart failure in association with Cheyne Stokes respiration; to verify if there is an association between the shortness of breath with changes in lung function. In a sample of 33 patients with stable post-ischemic CHF NYHA class II to III, mean LVEF 23,5 ± 7,7%, 17 patients (51%) an IAH upper 30, the others (49%) presented AHI lower 30. Cardiac function assessed by echocardiography and functional status did not differ between the two groups, but patients with IAH upper 30 had a lower exercise capacity (Vo2max p lower 0,05). The respiratory mechanical value of Ti/Ttot (p lower 0,024), P0,1 (p lower 0,04), P0,1/(Vt/Ti) (p lower 0,034) showed significant diference, indicating a increased stimulus to the respiratoy muscles due to an increment on the inspiratory effective impedance, suggesting that mechanical load to the respiratory muscles were increased determining a different strategy to recruit the muscles with a shift of the respiratory pattern. Studies have demonstrated diaphragmatic myopathy and atrophy similar, in part, to the changes in peripheral skeletal muscles. The mechanism of these alterations remains to be elucidated.

Supported by an unrestricted educational grant from Novartis.

[P2959] The impact of obstructive sleep apnea syndrome in asymptomatic patients to quality of life

Stavros M. Tryfon,[1] Ioannis Stanopoulos,[1] Efi Daskalopoulou,[2] Vasiliki Holeva,[3] Euklides Mavrofridis,[1] Elias Kakavelas.[1 1]
Respiratory Failure Unit, G. Hospital "G.Papanikolaou"Thessaloniki, Greece; [2]Sleep Laboratory,, G. Hospital "G.Papanikolaou"Thessaloniki, Greece; [3]Psychology Dpt, G.H. "Papageorgiou",, Thessaloniki, Greece

Tuesday, September 30, 2003, 1:30 PM, Thematic Poster Session: Sleep related breathing disorders and other diseases (1:30 PM-3:30 PM) Hall Z/16

The aim of this study is to investigate the relationship between diastolic blood pressure (DBP) response during incremental exercise test and severity of obstructive sleep apnea syndrome (OSAS) and the influence of this relationship to quality of life. We were studied 16 normotensive males (35,1±6,58y), who were admitted for the first time for OSAS (AHI=33,3±22,4), and 11 normal subjects (37,2±15,2y). All participants completed a quality of life questionnaire (Nottingham Health Profile-Part 1, NHP-Part1). The increase of DBP to 110mmHg in OSAS patients during ergometry, was achieved at SS lower VO2 compared to normal subjects (1881,6±703,5ml/min vs 1972,3±108,6 ml/min $p<0.05$). The diastolic hypertension was not correlated with the severity of OSAS. NHP-Part 1 results shown that patients had a mean score of <50 for both groups and there were no SS differences between them (Table I).

Dimensions	Normal	OSAS	Statistics
Emotional reactions	23.7 ± 24.7	31.9 ± 18.6	NS
Energy	46.1 ± 30	38.1 ± 34.8	NS
Pain	6.8 ± 4.5	12.1 ± 12	NS
Physical mobility	20.3 ± 15	21.8 ± 12.8	NS
Sleep	18.4 ± 19.8	21.1 ± 24.6	NS
Social isolation	21.5 ± 11.1	28.5 ± 11	NS

Normotensive OSAS patients seems to develop diastolic blood pressure elevation at an earlier stage during exercise. The analysis of the NHP-Part1 questionnaire shows that the dimensions "emotional reactions" and "energy" had the higher - but normal - values, which can be explained by the fact that these fields are heavily affected by OSAS.

Supported by an unrestricted educational grant from Novartis.

[3532] Prognostic value of periodic breathing in primary pulmonary hypertension (PPH)

C. Pison,[1] N. Chouri,[1] F. Felix,[1] B. Coltey,[1] R. Hamidfar,[1] J.L. Pepin.[1] [1]Département de Médecine Aiguë Spécialisée, CHU de Grenoble, Univ. J. Fourier, Grenoble, France

Wednesday, October 1, 2003, 8:30 AM, Oral Presentation: Pulmonary hypertension (8:30 AM-10:30 AM) Hall N

Periodic breathing or Cheyne-Stokes (CS) pattern during sleep is common during left ventricular failure and is associated with a poor prognosis independently of LVEF. In 23 consecutive patients with PPH, we prospectively looked at prevalence of CS pattern and its prognostic significance. Patients underwent pulmonary function tests (PFT), arterial blood gases, measurement of quality of life with Chronic Respiratory Questionnaire (CRQ), 6 min walking distance (6MWD), right heart catheterization and polygraphic recordings of sleep related respiratory disorders (SRRD). They were 56±15.1 yr. old, BMI: 24.6±6.1, FVC: 90.6±18.6%, FEV_1: 81.7±20.3%, TLC: 96.9±20.9% pred., PaO_2: 8.1±1.9, $PaCO_2$: 4.4±0.7 kPa, 6MWD: 295±120 meters, CRQ, dyspnea: 14.6±4.1 [5-35], fatigue: 13.9±6 [4-28], emotional function: 26.7±8 [7-49], symptoms: 16.1±5.2 [4-28], cardiac output: 4.2±1 l/min, pulmonary systolic pressure: 80±27 mm Hg, pulmonary arterial resistance: 925±490 $din.sec^{-1}.cm^{-5}$. Out of 23, 11 had no SRRD, 5 exhibited central sleep apnea syndrome (> 15 apnea-hypopnea / h) and 7 obstructive sleep apnea syndrome; 13 had no CS, 10 had a CS pattern during sleep. They were no significative difference regarding baseline PFTs, blood gases, 6MWD, CRQ scores or right hemodynamics between these subgroups. Our main results concern a significant difference in median survival of 70 months in patients without CS as opposed to 35 months in patients with a CS (p: 0.03). We conclude that SRRDs are common in patients with PPH and CS pattern should be systematically looked for in PPH patients considering its high prognostic value.

Supported by an unrestricted educational grant from Novartis.

[3572] Effect and significance of continuous positive airway pressure treatment on vascular endothelial function in patients with obstructive sleep apnea syndrome

Zhang Xilong,[1] Yin Kaisheng.[1] [1]Respiratory Medicine, The 1st Affiliated Hospital of Nanjing Medical University, Nanjing, Jiangsu, China

Wednesday, October 1, 2003, 10:45 AM, Oral Presentation: Systematic inflammation and metabolic disorders in sleep related breathing disorders (10:45 AM-12:45 PM) Hall E1

Objective: To investigate the changes of the vascular endothelial function index and myocardial ischemia in OSAHS patients with coronary heart disease (CHD) before and with continuous positive airway pressure (CPAP) treatment. Methods 18 patients with moderate-to-severe obstructive sleep apnea hypopnea syndrome (OSAHS) and CHD undergoing three months' CPAP treatment were recruited. The changes of their morning plasma nitric oxide (NO) and endothelin (ET), thr rate of NO/ET, total myocardial ischemia burden (TIB), apnea hypopnea index (AHI), minimal and mean pulse oxygen saturation (SPO_2) were compared and analysed before and with CPAP treatment. Results Compared with the parameters before treatment, there were significant increase in morning plasma NO and NO/ET($P < 0.01$), decrease in morning plasma ET($P < 0.05$), decrease in AHI, minimal SPO_2 ($P < 0.01$)as well as in mean SPO_2 and TIB($P < 0.05$)after three months' CPAP treatment. Conclusions CPAP treatment could play an important role in improvement and protection on vascular endothelial dysfunction and myocardial ischemia for OSAHS patients with CHD.

Key words Obstructive sleep apnea syndrome, continuous positive airway pressure, vascular endothelium, coronary heart disease

Supported by an unrestricted educational grant from Novartis.