DR. EDOARDO BERNKOPF
Medico chirurgo Specialista in Odontoiatria e protesi dentaria e-mail: edber@studiober.com
VICENZA 36100- Via Garofolino, 1 - Tel. 0444/545509 **Sito internet: www.studiober.com**
PARMA 43100 - Via Petrarca, 3 - Tel. e Fax 0521/236426
ROMA 00100 - Via Massaciuccoli, 19 (P.zza Annibaliano) Tel. 06/86382917

CHEST Original Research

Full text: http://www.chestjournal.org/cgi/content/full/130/5/1362

SLEEP MEDICINE

Il ruolo patogenetico dell'Apnea nel Sonno nell'ipertensione e nel rischio di incidente cardiocircolatorio é comprovato da numerosi studi scientifici. Questo lavoro prova l'efficacia del nostro dispositivo intraorale nell'Apnea nel Sonno e , di conseguenza, nelle complicanze cardiocircolatorie . E.B.

Autonomic Cardiac Modulation in Obstructive Sleep Apnea*

Effect of an Oral Jaw-Positioning Appliance

Paolo Coruzzi, MD; Massimo Gualerzi, MD; Edoardo Bernkopf, MD; Lorenzo Brambilla, MD; Valerio Brambilla, MD; Vanna Broia, MD; Carolina Lombardi, MD; and Gianfranco Parati, MD

Background: **Patients with obstructive sleep apnea (OSA) are characterized by deranged cardiovascular variability, a well-established marker of cardiovascular risk. While long-term treatment with continuous positive airway pressure leads to a significant improvement of cardiovascular variability, little is known of the possibility of achieving the same results with other therapeutic approaches. The aim of our study was to investigate the responses of autonomic indexes of neural cardiac control to another type of OSA treatment based on an oral jaw-positioning appliance.**
Methods: **In 10 otherwise healthy subjects with OSA (OSA+) and in 10 subjects without OSA (OSA−) we measured heart rate, BP, and indices of autonomic cardiac regulation derived from time-domain and spectral analysis of R-R interval (RRI), before and after 3 months of treatment with the oral device. High-frequency (HF) power of RRI was taken as an index of parasympathetic cardiac modulation, and the ratio between low-frequency (LF) and HF RRI powers as an indirect marker of the balance between sympathetic and parasympathetic cardiac modulation.**
Results: **At baseline, in comparison with OSA− subjects, OSA+ subjects displayed a significantly lower RRI variance ($p < 0.02$) and reduced HF RRI powers ($p < 0.001$). After 3 months of treatment with the oral device, the OSA+ group showed a marked reduction in apnea-hypopnea index ($p < 0.001$), a lengthening in RRI and a significant increase in its variance ($p < 0,02$), an increased HF RRI power (from 134 ± 26 to 502 ± 48 ms^2, $p < 0.001$), and a reduction in LF/HF RRI power ratio (from 3.11 ± 0.8 to 1.5 ± 0.5). As a result of these changes, after the 3-month treatment there were no more significant differences between the two groups in these parameters. In both OSA+ and OSA− groups, body weight, heart rate, and BP did not change over time.**
Conclusions: **Three months of treatment with a specific oral jaw-positioning appliance improves cardiac autonomic modulation in otherwise healthy patients with OSA of mild degree.**
(CHEST 2006; 130:1362–1368)

Key words: autonomic nervous system; cardiovascular prevention; heart rate variability; obstructive sleep apnea; oral device

Abbreviations: AHI = apnea-hypopnea index; CPAP = continuous positive airway pressure; HF = high frequency; LF = low frequency; low O_2 = lowest oxygen saturation; OSA = obstructive sleep apnea; OSA+ = with obstructive sleep apnea; OSA− = without obstructive sleep apnea; OSAS = obstructive sleep apnea syndrome; RRI = R-R interval; Sao_2 = arterial oxygen saturation

Obstructive sleep apnea (OSA) is a common and often neglected disorder[1] that is strongly associated with known cardiovascular risk factors, including obesity, insulin resistance, and dyslipidemia. OSA itself may represent an independent risk factor for hypertension,[2] heart failure,[3] myocardial infarction,[4] and stroke.[5] The mechanisms underlying the association between OSA and cardiovascular disease are not well defined, even though it has been suggested that a deranged cardiovascular variability may represent a pivotal link in this context.[6] An altered cardiovascular variability, reflecting a deranged autonomic cardiovascular regulation, may not only predict morbidity and mortality in patients with

Ipertensione-Apnea nel sonno

BMJ 2000 Feb 19;320(7233):479-82 **Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study.**
Lavie P, Herer P, Hoffstein V Sleep Laboratory, Bruce Rappaport Faculty of Medicine, Israel Institute of Technology, Haifa, Israel. [Medline record in process]

Objective: To assess whether sleep apnoea syndrome is an independent risk factor for hypertension. Design: Population study. Setting: Sleep clinic in Toronto. Participants: 2677 adults, aged 20-85 years, referred to the sleep clinic with suspected sleep apnoea syndrome. Outcome measures: Medical history, demographic data, morning and evening blood pressure, and whole night polysomnography. Results: Blood pressure and number of patients with hypertension increased linearly with severity of sleep apnoea, as shown by the apnoea-hypopnoea index. Multiple regression analysis of blood pressure levels of all patients not taking antihypertensives showed that apnoea was a significant predictor of both systolic and diastolic blood pressure after adjustment for age, body mass index, and sex. Multiple logistic regression showed that each additional apnoeic event per hour of sleep increased the odds of hypertension by about 1%, whereas each 10% decrease in nocturnal oxygen saturation increased the odds by 13%. Conclusion: Sleep apnoea syndrome is profoundly associated with hypertension independent of all relevant risk factors.

J Hypertens 2001 Nov;19(11):2071-8

Blood pressure, cardiac structure and severity of obstructive sleep apnea in a sleep clinic population.

Kraiczi H, Peker Y, Caidahl K, Samuelsson A, Hedner J.

Departments of aClinical Pharmacology, bPulmonary Medicine and cClinical Physiology, Sahlgrenska University Hospital, Gothenburg, Sweden.

OBJECTIVES : We investigated whether the severity of obstructive sleep apnea (OSA) predicts blood pressure or cardiac left ventricular thickness in a clinical population of OSA patients, if adjustments are made for age, gender, use of antihypertensive agents, smoking, body mass index, history of coronary artery disease, hypercholesterolemia and circulating C-peptide concentrations. DESIGN : Relationships in this cross-sectional study were investigated with correlation analysis and multiple regression procedures. PATIENTS AND METHODS : Apnea-hypopnea index (AHI, polysomnography) and office systolic and diastolic blood pressures (SBP and DBP) were measured in 81 subjects referred to a university hospital sleep laboratory. Ambulatory blood pressures were recorded during one 24 h cycle. Left ventricular (LV) muscle size was quantified as two-dimensionally directed M-mode-derived end-diastolic thickness of interventricular septum and posterior chamber wall. RESULTS : After adjustment for separate or the entire set of covariates, AHI predicted office SBP and DBP as well as daytime ambulatory DBP and night-time ambulatory SBP and DBP, but not daytime ambulatory SBP. In contrast, associations between AHI and LV muscle thickness reflected complex inter-relationships with confounding variables. Smoking and age suppressed, whereas body mass index (BMI) and hypertension inflated the relationship between OSA severity and LV muscle thickness in this study. CONCLUSIONS : AHI is an independent predictor of several measures of blood pressure. OSA severity and LV muscle thickness appear to be primarily linked via increased blood pressure.

BMJ 2000 Feb 19;320(7233):479-82
Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study.
Lavie P, Herer P, Hoffstein V
Sleep Laboratory, Bruce Rappaport Faculty of Medicine, Israel Institute of Technology, Haifa, Israel.
[Medline record in process]
Objective: To assess whether sleep apnoea syndrome is an independent risk factor for hypertension. Design: Population study. Setting: Sleep clinic in Toronto. Participants: 2677 adults, aged 20-85 years, referred to the sleep clinic with suspected sleep apnoea syndrome. Outcome measures: Medical history, demographic data, morning and evening blood pressure, and whole night polysomnography. Results: Blood pressure and number of patients with hypertension increased linearly with severity of sleep apnoea, as shown by the apnoea-hypopnoea index. Multiple regression analysis of blood pressure levels of all patients not taking antihypertensives showed that apnoea was a significant predictor of both systolic and diastolic blood pressure after adjustment for age, body mass index, and sex. Multiple logistic regression showed that each additional apnoeic event per hour of sleep increased the odds of hypertension by about 1%, whereas each 10% decrease in nocturnal oxygen saturation increased the odds by 13%. Conclusion: Sleep apnoea syndrome is profoundly associated with hypertension independent of all relevant risk factors.

Curr Cardiol Rep 2005 7(3):211-5

The link between obstructive sleep apnea and heart failure: underappreciated opportunity for treatment.

Naughton M

Obstructive sleep apnea (OSA) is a newly recognized risk factor for the development of systemic hypertension, ischemic heart disease and congestive heart failure. Mechanisms responsible for these links include OSA-related hypoxemia and arousal from sleep-induced increased sympathetic activity, large negative intrathoracic pressure-induced increased left ventricular transmural pressure gradient, and impaired vagal activity plus oxygen radial formation. Secondary phenomena include increased platelet aggregability, insulin resistance, and endothelial dysfunction with reduced endogenous nitric oxide production. Safe nonpharmacologic, nonsurgical therapy, namely continuous positive airway pressure, can attenuate OSA, and improve cardiac function and quality of life. Searching for signs or symptoms of OSA from the patient (or bed partner), namely loud habitual snoring, apneas, nocturnal choking, orthopnea, paroxysmal nocturnal dyspnea, excessive daytime sleepiness, or cardiovascular disease, which is difficult to control, may reward the curious physician with another treatment avenue.