

HYPOXEMIA DURING BILEVEL POSITIVE AIRWAY PRESSURE TREATMENT IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME AND CHRONIC RESPIRATORY INSUFFICIENCY

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INTRODUCTION AND THE AIM OF THE STUDY: In a minority of patients with obstructive sleep apnea (OSA) syndrome chronic respiratory insufficiency develops, usually as a consequence of chronic alveolar hypoventilation. One of the options of the treatment of the patients with OSA syndrome and chronic alveolar hypoventilation is bilevel positive airway pressure (BPAP) during sleep with or without additional oxygen. It is not known, however, which patients benefit from this treatment. Thus, the aim of the study was to find out which factors influence the results of BPAP treatment in the patients with OSA syndrome and chronic alveolar hypoventilation. **MATERIAL AND METHODS:** Material of the study consisted of 55 adult patients (12 women) with obesity (body mass index >30 kg/m², mean 46 ± 7 kg/m²) and OSA syndrome (apnea/hypopnea index >30 , mean 62 ± 18), and chronic respiratory insufficiency (PaCO₂ >45 mmHg, mean $54\pm 5,6$ mmHg). In all patients polysomnography was performed with BPAP treatment and additional oxygen – when indicated (sustained SaO₂ $<88\%$). The aim of adequate BPAP titration was to eliminate all the obstructive apneas and hypopneas and to obtain maximal possible SaO₂. **RESULTS:** The mean SaO₂ during BPAP treatment was $87\pm 5\%$ in NREM sleep and $85\pm 8\%$ in REM sleep. In 31 patients (56%) the mean SaO₂ during sleep was $<88\%$ despite optimal BPAP and oxygen titration: $83\pm 4\%$ during NREM sleep and $81\pm 7\%$ during REM sleep vs $91\pm 2\%$ and $90\pm 3\%$, respectively, in the remaining patients ($p<0,001$). In 18 patients (33%) there was sustained severe hypoxemia (mean SaO₂ $<85\%$) during BPAP treatment: $81\pm 4\%$ in NREM sleep and $72\pm 10\%$ in REM sleep. All but one women belonged to this group. The patients with severe hypoxemia during sleep and BPAP treatment had lower vital capacity (VC), lower diurnal PaO₂ and higher PaCO₂ as compared with remaining patients ($2,0\pm 0,8$ l vs $2,8\pm 0,9$ l, $p<0,05$; 49 ± 7 mmHg vs 54 ± 8 mmHg, and 57 ± 5 mmHg vs 52 ± 6 mmHg, $p<0,01$, respectively). **CONCLUSION:** In approximately 1/3 of the obese patients with severe OSA syndrome and chronic respiratory insufficiency severe sleep hypoxemia persists during optimally titrated BPAP machine and with additional oxygen delivery. The female patients and the patients with low VC and severe chronic alveolar hypoventilation in the course of OSA syndrome are at risk of sustained and severe hypoxemia during sleep despite optimal BPAP titration.