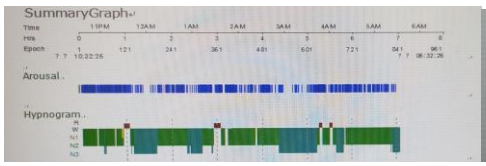
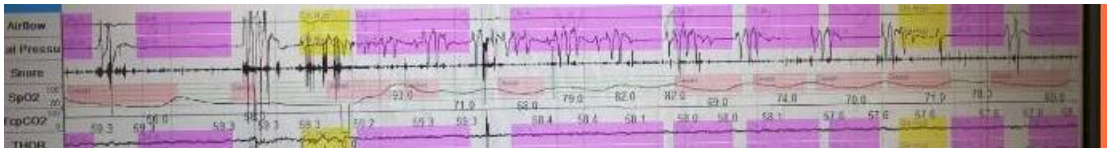
This 46 years old man with underlying hypertension history visited to sleep medicine out-patient department for chief complaint of non-refreshing sleep with excessive daytime sleepiness, morning headaches and memory lapses over last 1-2 years. He had also been facing with troublesome symptoms like loud snoring, apneic episodes with gagging and choking sense on nocturnal sleep, witnessed by his bedpartner, and sense of breathlessness on climbing up above second floor. He does not smoke nor drinking alcohol. On physical examination, his body weight was 102 kg and height was 154 cm, BMI was 43, neck circumference was 46 cm, macroglossia and excessive throat tissue density and lateral peri-tonsillar narrowing, Mallampati score of 3 and grade 3 tonsillar hypertrophy were noted. His palate was not highly arched. ESS score was 15. Then we arranged him nighttime polysomnogram (PSG) including TCO2 monitoring as we suspected him of sleep disordered breathing. Upon his initial PSG data, TST was 6 hours 47 mins, AHI was 157/h, Obstructive Apnea of 127.2/h, Mixed Apnea of 23/h, Obstructive Hypopnea of 5.9/h, 3%ODI of 148.6/h and snore index of 216.7/h. Mean SpO2 was 73% and minimal SpO2 was 34%. Total arousal index was 72.4/h and respiratory arousal index was 52.5/h. Mean awake TCO2 was 32.6 mmHg. TCO2 monitoring showed TCO2 of more than 50 mmHg on sleep for 41.1 min, the highest one to 58.4 mmHg. So, his awake and sleep TCO2 difference was 18. His highest heart rate was 415 bpm and average heart rate during sleep was 96 bpm. So, the patient was diagnosed as obesity-associated sleep hypoventilation with hypoxemia and concomitant severe obstructive sleep apnea and hypopnea, snoring according to 2020 AASM criteria.





Then we arranged split night PSG study for titrating his CPAP level, and found it was 10 cmH2O at which AHI residue of 10 /hr. We recommended him to use CPAP therapy and to adopt constant regular healthy lifestyle modification, regular physical activity for body weight reduction. He lost BW of 6 kg over 6 months but BMI still 40. PSG follow up after 3 months of CPAP therapy showed residual AHI of 10/h and highest TCO2 of 43 mmHg. His sleep-disordered related symptoms got significantly improved much with CPAP therapy. His spirometry results showed mild degree of restrictive airway dysfunction of FVC in 2.73 L (65%), FEV1/FVC ratio is 0.71 and

FEV1 is 3.0 L (72%). We will continue assess his BW changes, sleep-related symptoms signs, daytime sleepiness score, and regular pulmonary function test, 6-MWT, any emergence of other comorbid disease, follow up polysomnography and serum bicarbonate level (if needed) on upcoming visits. We will introduce him other possible intensive weight management strategies to him if needed.

Analyses: That patient is morbidly obese, and he has nocturnal hypercapnia by nocturnal hypoventilation although he has no daytime hypercapnic evidence, so he belongs to stage I of Obesity Hypoventilation Syndrome (OHS) (that is Obesity-Associated Sleep Hypoventilation) which means intermittent nocturnal hypercapnia get fully recovery on daytime. However, we had not checked his daytime arterial serum bicarbonate level then. Concurrently, he had also had severe obstructive sleep apnea with predominant OSA symptoms and higher AHI of more than 30/hr. Therefore, our treatment plan is initially aimed at splinting of his upper airway where frequent complete or partial collapse of his upper airway on sleep due to excessive local fat mass around his throat and Oro-pharyngeal tissue. So, we started to treat him with CPAP therapy to minimize those. Finally, we found appropriate CPAP level for him is 10 cmH2O. Till so far, he has being on the way of much improved in obstructive sleep sign and symptoms under regular CPAP therapy at 10 cmH2O during nighttime sleep. His nocturnal hypercapnia is due to pathophysiology of morbid obesity and excessive fat depositions surrounding upper airway, around the chest and abdominal walls cast which led him vulnerable to increased airway luminal collapsibility and concomitant impaired ventilatory mechanical constraints affecting the chest wall and diaphragmatic mechanics, and also reduced central respiratory drive by impaired leptin level and sensitivity secreted from plentiful adipose tissue and cells to stimulate neural ventilatory center in addition to prolong sleep apnea hypopnea and inter-apneic hypoventilation and diurnal hypoventilation. So, he exhibits marked positive response to CPAP therapy. Although effective weight reduction is the essence for both OHS and OSA therapy, but patient can’t achieve it very well that he needs to put more effort on to decrease his BMI down to below 35.

Conclusion: We should keep mindful of obesity-associated sleep hypoventilation (that is Stage I and II of hypoventilation in obesity) in grossly morbid obese patient though patient does not have criteria of obesity hypoventilation syndrome that means persistent daytime awake hypercapnia of more than 45 mmHg or more than 50 mmHg for 10 minutes with cardiometabolic abnormalities. In such patients, measuring of daytime serum bicarbonate level is also needed to detect whether it is less than 27 mmol/L (i.e. complete wash out of nocturnally accumulated CO2) or more than 27mmol/L (i.e. incomplete wash out of CO2). Anyhow, healthy dietary and lifestyle

adoption with regular physical activity is important adjunctive therapeutic modality as positive airway pressure therapy (either CPAP or bi-level PAP therapy) and pulmonary rehabilitation play critical roles in such patient.

中文題目：**肥胖相關之睡眠通氣不足和嚴重阻塞性睡眠呼吸終止症併低血氧症**

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