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Continuous positive airway pressure delivered by oronasal mask may not be effective for obstructive sleep apnoea

To the Editors:

Continuous positive airway pressure (CPAP) is considered the gold standard treatment for patients with moderate to severe obstructive sleep apnoea (OSA). The treatment of OSA with CPAP was first conceptualised using a nasal-only interface because the pressure delivered through the nose would be transmitted to the back of the upper airway and would push the palate anteriorly [1]. Since the first description, the CPAP industry has developed a large number of different interfaces in order to improve patient comfort and adherence to treatment. Patients with OSA frequently present nasal obstruction and oronasal interfaces may be used to deliver CPAP. Nasal and oronasal masks are often used interchangeably and the choice of CPAP delivery interface for OSA therapy remains largely based on clinical experience. However, patients with OSA on oronasal mask are less adherent to CPAP for reasons that are not completely understood [2]. One recent randomised trial [3] and a preliminary report [4] suggest that the effectiveness of CPAP for treating OSA is variable when delivered by an oronasal interface. We describe a well-documented patient in whom CPAP was not effective when an oronasal mask was used due to the posterior displacement of the tongue.

A 69-yr-old Japanese–Brazilian, body mass index 26.1 $kg \cdot m^{-2}$, presented to the outpatient sleep clinic complaining of typical symptoms suggestive of OSA, including loud snoring, witnessed apnoeas and excessive daytime sleepiness. The patient had a positive medical history of systemic hypertension and diabetes mellitus. A standard overnight polysomnography (Alice 5; Philips Respironics, Murrysville, PA, USA) confirmed severe OSA, with apnoea-hypopnoea index (AHI) 76 events per h and lowest oxygen saturation 58%. An in-laboratory manual CPAP titration study was performed with an oronasal mask because of reported oral breathing during sleep. CPAP was gradually increased up to 16 cmH_2O with no clear elimination of OSA at any single CPAP. The overall AHI during the CPAP titration with oronasal mask was 32 events per h and the lowest oxygen saturation was 78%. The patient was then scheduled for a new CPAP titration study that was initiated with a nasal mask, with elimination of OSA at CPAP of 7 cmH_2O. The mask was changed to an oronasal mask during the second half of the study. In contrast to the first half and similar to the first titration study, OSA was not abolished and obstructive hypopnoeas persisted despite a progressive raise of CPAP up to 16 cmH_2O (fig. 1). We therefore hypothesised that CPAP delivered by an oronasal interface was not effective due to posterior displacement of the tongue caused by oral pressure. The patient was submitted to a sleep endoscopy study in the early morning using an intravenous infusion of midazolam that was slowly titrated until initiation of sleep, as previous described [5]. The endoscope was inserted through a latex-sealed hole in the mask to directly visualise the upper airway. The oropharyngeal region was

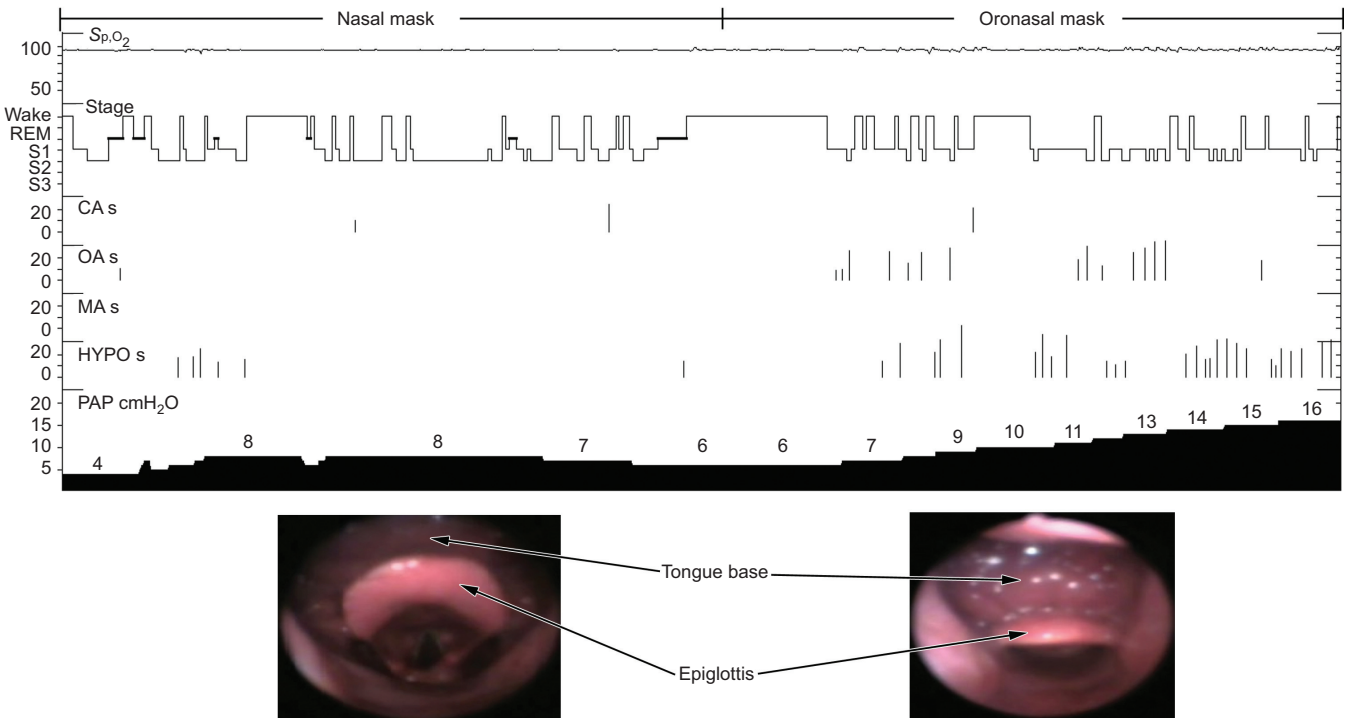

FIGURE 1. Polysomnography summary of a natural sleep continuous positive airway pressure (CPAP) titration study (top) and sleep endoscopy images (bottom) using both nasal and oronasal masks. During the first part of the natural sleep CPAP titration, a nasal CPAP of 7 cmH_2O adequately controlled obstructive events. During the second part, an oronasal mask was used. Observe the persistence of obstructive respiratory events even at pressures up to 16 cmH_2O. The endoscopic image taken with nasal CPAP of 7 cmH_2O shows a wide open oropharynx. In contrast, the image taken with oronasal CPAP of 16 cmH_2O shows the tongue base posteriorly displaced, pushing the epiglottis and significantly narrowing the airway. S_{p,O_2}: arterial oxygen saturation measured by pulse oximetry; CA: central apnoea; OA: obstructive apnoea; MA: mixed apnoea; HYPO: hypopnoea; PAP: pulmonary artery pressure; REM: rapid eye movement. Respiratory event time is shown in seconds.

observed while the patient used both nasal and oronasal mask. The oropharynx was open while the patient was on nasal mask at CPAP of 7 cmH_2O but was partially obstructed while on oronasal mask at CPAP of 16 cmH_2O due to a posterior displacement of the tongue. The images were reviewed off line, frame by frame, and pictures were taken at the smallest upper airway dimension during each condition, corresponding to early inspiration (fig. 1). Air leak with oronasal mask at CPAP of 16 cmH_2O was 57 and 30 $L \cdot min^{-1}$ during the CPAP titration night and induced sleep, respectively. During the latter condition pressure at the mask and was equal to the set by the equipment. A nasal CPAP of 7 cmH_2O was prescribed and the patient reported significant improvement of symptoms after 3 months of follow-up.

The present case report conveys two important messages that may be relevant to some patients regarding the choice of CPAP interfaces for the treatment of OSA. First, in contrast to nasal interface, CPAP delivered by an oronasal interface was not completely effective in treating OSA. In our patient, a relatively low nasal CPAP (7 cmH_2O) was able to abolish OSA. In contrast, three independent sleep studies (including two natural sleep studies and one induced sleep study with midazolam) showed that CPAP titrated up to 16 cmH_2O using an oronasal interface did not adequately treat OSA. Secondly, the direct visualisation of the upper airway during CPAP titration provided a mechanism to explain this apparent paradox. We showed that, as expected, the upper airway was open at nasal CPAP of 7 cmH_2O. In contrast, the oropharyngeal airway was partially obstructed while on oronasal CPAP of 16 cmH_2O mask due to a posterior displacement of the tongue. The study therefore suggests that the pressure delivered through the mouth may push the tongue backwards. This hypothesis is consistent with a previous report showing that CPAP through an oronasal mask was not able to open the upper airway even at pressures well above the calculated critical closing pressure obtained with a nasal mask [6]. A significant air leak on oronasal mask could lead to insufficient mask pressure and explain the lack of treatment effect. However, the pressure delivered at the mask during induced sleep was equal to that displayed by the equipment. Because obstruction occurred immediately after the interface was changed, it is unlikely that obstruction was caused by an increase in surface tension due to dryness of the upper airways associated with oral breathing. One alternative explanation is that the oronasal mask pushed the mandible backwards, causing a false retrognathism. Although we did not use any imaging method to detect changes in mandibular position in our patient, one recent preliminary study that compared nasal and oronasal interface with and without a mandibular advancement device (MAD) in a group of OSA patients with a history of difficult CPAP titration showed no impact of MAD on the titrated oronasal CPAP in most cases. Interestingly enough, in this preliminary study, four out of eight patients had obstructive events even after a CPAP of 20 cmH_2O was reached while using an oronasal mask [4]. One limitation of our case report is that we were not able to quantify the proportion of

nasal and oral breathing while the oronasal mask was used. It is possible that this is a major determinant of how the oronasal interface will impact on the distribution of pressures in the nasal and oral segments. There is evidence that upper airway resistance during sleep and the propensity to obstructive sleep apnoea are significantly lower while breathing nasally rather than orally [7].

TEO *et al*. [3] have recently reported the first randomised trial that compared CPAP titration with nasal and oronasal masks in patients with newly diagnosed OSA. While the CPAP pressures achieved by nasal and oronasal interfaces was similar (~11 cmH_2O) the AHI was significantly lower on nasal mask (5.3 ± 3.4 *versus* 11.0 ± 10.4 events per h, respectively; $p=0.01$). Moreover, the high AHI standard deviation while on oronasal mask indicates a wide effectiveness variability. In the study of TEO *et al*. [3] the use of oronasal mask was also associated with worse sleep quality, higher air leak and poorer patient satisfaction. We speculate that the observed mechanisms in our patient may help to explain why several patients are not effectively treated while on oronasal CPAP. These mechanisms may contribute to higher air leak and poorer treatment satisfaction.

In conclusion, our case report is in line with recent studies suggesting that CPAP delivered by oronasal interface may not be effective in a subgroup of patients with OSA [3, 4]. Our case report suggests that CPAP delivered through an oronasal interface may push the tongue backwards in some patients. Therefore, a nasal mask should be preferred as the first option as a CPAP interface. Patients on an oronasal mask should be carefully followed. The low compliance to CPAP when an oronasal interface is used may be partially explained by suboptimal therapeutic effectiveness.

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Combined pulmonary fibrosis and emphysema associated with microscopic polyangiitis

To the Editors:

Microscopic polyangiitis (MPA) is a necrotising multiorgan vasculitis associated with a variety of circulating autoantibodies, such as anti-neutrophil cytoplasm antibodies (ANCAs) against myeloperoxidase (MPO) [1]. Typical and most common pulmonary involvement comprises of alveolar haemorrhage secondary to pulmonary capillaritis as well as interstitial lung fibrosis [2] and progressive obstructive lung disease [1, 3, 4].

The combination of pulmonary fibrosis and emphysema (CPFE) is a recently defined syndrome, encompassing a distinct radiology, revealing both upper-lobe emphysema and lower-lobe fibrosis on high-resolution computed tomography (HRCT) of the chest, as well as lung function profile, with apparently preserved lung volumes contrasting with disproportionally impaired gas exchange, as assessed by reduced diffusing capacity of the lung for carbon monoxide [5, 6]. CPFE has been recently described in the context of connective tissue diseases [7]. Nevertheless, it is still debatable whether CPFE represents a distinct syndrome or it is just a phenotype of pulmonary fibrosis with coincidental emphysema. Here, we describe for the first time, in a male patient, a novel type of pulmonary manifestation of MPA, the combination of pulmonary fibrosis and emphysema.

In 2008, an 80-yr-old Greek-Caucasian male, heavy ex-smoker (80 pack-yrs), ex-farmer and coal worker with a history of idiopathic pulmonary fibrosis, based on the absence of other underlying conditions and the presence of a histopathological and radiological pattern of usual interstitial pneumonia combined with upper-lobe emphysema (fig. 1a and b) since 2005, was referred to the emergency department of the University Hospital of Alexandroupolis (Alexandroupolis, Greece) due to