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HEAD AND NECK

Myoelectric characteristics of tensor palatini and collapsibility of upper airway in OSA patients with different phenotypes under DISE

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Abstract

Objective This study aims to evaluate the combination of myoelectric characteristics of tensor palatini muscle (TP) and collapsibility of upper airway in obstructive sleep apnea (OSA) patients with different external phenotypes of collapse pattern at velum level under drug-induced sleep endoscopy (DISE).

Study design Case series with planned data collection.

Setting Operation room.

Subjects and methods 36 mainly collapse pattern at velum level OSA subjects underwent DISE with synchronous tensor palatini electromyograms (TP EMG), and polysomnography (ALICE 6). According to the phenotype of collapse pattern at velum level in DISE, the subjects were divided into group 1 (concentric collapse), group 2 (anteroposterior collapse), and group 3 (lateral collapse). Each group consisted of 13, 14, and 9 subjects, respectively, and was observed the electromyographic indexes at awake, sleep onset, during apnea and the third respiratory cycle after apnea. The active and passive upper airway critical closing pressure (Pcrit) of each group were measured at the same time, and the difference of neuromuscular response between different groups was evaluated.

Results In tonic TPEMG, group 1 showed the highest value during awake and sleep onset, while group 2 was the highest during apnea and after apnea. In peak TPEMG, group 1 showed the highest value during awake. Group 2 showed the highest value during other states. In passive Pcrit and *D* value (difference between passive Pcrit and active Pcrit), group 2 was the highest, while group 1 was the highest in active Pcrit. Difference was statistically significant.

Conclusions Under different states of awake, sleep onset, apnea and after apnea, the response force of tensor palatini muscle of OSA subjects with different phenotypes under DISE was different. Group 1 showed the highest EMG values only when awake and sleep onset, and it was most prone to collapse. Group 2 had the highest anatomical load (passive Pcrit) and the highest neuromuscular compensatory effect (*D* value).

Keywords Obstructive sleep apnea · Drug-induced sleep endoscopy (DISE) · Tensor palatini electromyograms (TPEMG) · Upper airway critical closing pressure (Pcrit)

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Introduction

OSA is a heterogeneous disorder in which upper airway (UA) collapses repeatedly during sleep, resulting in intermittent low oxygen saturation and sleep structure disorder, and then, a series of physiological changes, cardiovascular, cerebrovascular and endocrine functions are damaged.

The main causes of UA collapse are passive critical closing pressure of the upper airway, arousal threshold, loop gain, and muscle responsiveness (PALM) mechanism. 69% of patients with OSA have one or more nonanatomic pathophysiologic traits [1].

In the past, otolaryngologists which devoted to the improvement of craniofacial bone frame and soft-tissue stenosis in OSA patients paid more attention to passive critical closing pressure of the upper airway (passive Pcrit), i.e., resistive loading. Recently, the potential role of inadequate upper-airway dilator muscle function during sleep plus an anatomically collapsible upper airway has been widely concerned. However, much of the study was focused on the genioglossus (GG) of the tongue base region, while the tensor palatini muscle (TP) of the palatopharyngeal region was less studied.

TP is the main dilator that affects the collapse of velum level in OSA patients by stiffen the UA walls, not enlarge the UA lumen. It starts from the lateral plate of eustachian tube cartilage, eustachian tube membrane, sphenoid spine, root of pterygoid process, posterior margin of greater pterygoid, scaphoid fossa of sphenoid bone, hooks over the pterygoid hamulus, and ends at midline aponeurosis of the palate. It contracts through the pterygoid hamulus to tenses the soft palate outwards. Being tonic muscles, its tonic activity (expiratory phase) is considered to be an important determinant of basic airway size and stiffness. The major input to it appears to be the wakefulness stimulus [2], less response to CO_2 or inspiratory resistive loading [3–5].

In this study, we aimed (1) to reveal the characteristics of TP EMG activation in different external phenotypes (concentric, anteroposterior and lateral collapse [6]) under DISE in collapse pattern at velum level OSA patients and (2) to known apart from the central control, whether the neuromuscular response had differences in three types of phenotypes OSA patients or not?

Patients and methods

Ethical considerations

This study received institutional review board approval at the Beijing Friendship Hospital of the Capital Medical University, Beijing, China. Ethics: 2018-p2-159-01. Informed written consent was obtained from all participants.

Patients

Inclusion criteria included moderate and severe mainly collapse pattern at velum level male OSA subjects, aged between 20 and 60 years which were evaluated by overnight polysomnography (PSG) at Beijing friendship Hospital, China, with typical symptoms including snoring, witnessed apneas, and daytime sleepiness Patients were treated between May 2018 and February 2019.

Exclusion criteria were as follows: subjects with dominantly tongue base collapse, previous history of soft palate area surgery, and previous or current neuromuscular disease.

All subjects underwent complete head and neck physical examination.

Polysomnography study

PSG studies all performed before DISE at the Sleep Center of Beijing Friendship Hospital, China. PSG recordings were collected and analyzed offline (Alice 6, Philips) during light-off to light-on. Sleep stages and arousals were scored using standard criteria according to the American Academy of Sleep Medicine (AASM), and recordings included electroencephalography, electrooculography, chin electromyography, and electrocardiography. Transcutaneous pulse oximetry was used, with respiratory effort recorded using inductance plethysmography.

DISE with synchronous PSG and TP electromyography (TPEMG)

Moderate-to-severe male OSA subjects were recruited after the first night PSG study. In the next day, they underwent DISE with synchronous TPEMG (tensor palatini electromyograms) using intraoral fine-wire electrodes and polysomnography. Subjects were brought to the operating room and placed supine on the table with a pillow of an approximate thickness of 15 cm. Continuous vital sign monitoring and bispectral index (BIS) monitoring was initiated, besides PSG. Oral and nasal mucous membrane anesthesia was achieved with 1% tetracaine, and two intraoral fine-wire electrodes were inserted 1–1.5 cm into the palate at a 45° angle along the lateral surface of the pterygoid plate using a 23-gauge needle [7].

The anesthesiologist began sedation used propofol. When BIS values reached 70, OSA subjects went into simulated N2 sleep [8]. BIS was maintained between 60 and 70 for 15 min, and then, DISE was performed. Using the VOTE scoring system [9–11], at least 3 respiratory cycles were

observed at each stenosis plane. According to the three forms of velum obstruction, the subjects were divided into group 1 (concentric collapse), group 2 (anteroposterior collapse), and group 3 (lateral collapse). 13, 14, and 9 mainly collapse pattern at velum level subjects in each group were selected, respectively.

Active and passive Pcrit

OSA subjects wore a modified oral–nasal mask which had two added holes, one was at the superior lateral and the other at lower medial with an air leakage valve (Fig. 1c) and the mask connected to a bilevel positive airway ventilation (BiPAP, ST25, Philips Respironics). When OSA subjects went into the simulated N2 sleep state, the electronic laryngoscope went from the lower medial hole of the mask into the subjects' upper airway. The superior lateral hole was sealed. (Fig. 1a). BiPAP adjustment settings: the pressure of exhalation and inhalation was the same each time. From 4 cm H_2O pressure, increased pressure by 1 cm H_2O continuously every one minute interval, to activate UA neuromuscular responses, until the respiratory event disappears, the synchronous image of graphic workstation showed that the airway was completely open, and then, step-down 1 cm H_2O continuously every 1-min interval until the airway was occluded, recorded the pressure of the BiPAP at the moment as approximate active Pcrit.

The anesthesiologist increased the blood concentration of propofol, added muscle relaxant, inhibited central drive to the respiratory pump muscles and UA muscle peripheral reflexes, moved the subjects from sedation to anesthesia, and intubated the patients' trachea. The external end of intubation was led out from the superior lateral hole of the mask and connected to the anesthesia machine. (Fig. 1b) Other steps were the same as above. BiPAP adjustment settings: from 4 cm H_2O pressure, increased pressure by 1 cm H_2O continuously every 5 s interval, because there was no need to induce neuromuscular reflexes, it could rapidly pressurize until the airway was fully opened, then gradually reduced the pressure until the airway was closed, and recorded the pressure of the BiPAP at the moment as approximate passive Pcrit.

TPEMG activity measurement and parameters

Raw TPEMG signals were band-pass filtered (30–1000 Hz), and a 50-Hz notch filter was applied. Raw and moving time average signals of TG were recorded and analyzed offline (software Spike 2, CED, Cambridge, UK.). It calculated on the root mean square (RMS) amplitude. All TPEMG signals were expressed as percentage of the maximal value (%max). Maximal TPEMG activity was defined as the largest several large swallow tasks in quiet breathing [12]. The following TPEMG parameters were measured according to each respiratory cycle, including tonic TPEMG (lowest value during expiration) and peak phasic TPEMG (highest value during inspiration) at awake, sleep-onset, during apnea and the third respiratory cycle after apnea respectively. (Figs. 2, 3, 4) Given the potential delay of apnea on TPEMG activity, we chose the third respiratory cycle after apnea to measure the maximum response of TPEMG after apnea. At the end of apnea, it sometimes caused an arousal. (Fig. 3).

Statistical procedures

Statistical analysis was performed using SPSS 22.0 (SPSS, Inc., an IBM Company, Chicago, IL). Data are reported as means ± SD. Shapiro–Wilk normality test was used to test normal distribution of data. Levene's test was used for homogeneity of variance. For normally distributed and equal variances assumed data, one-way ANOVA was used, Bonferroni used for comparison of each two groups; for

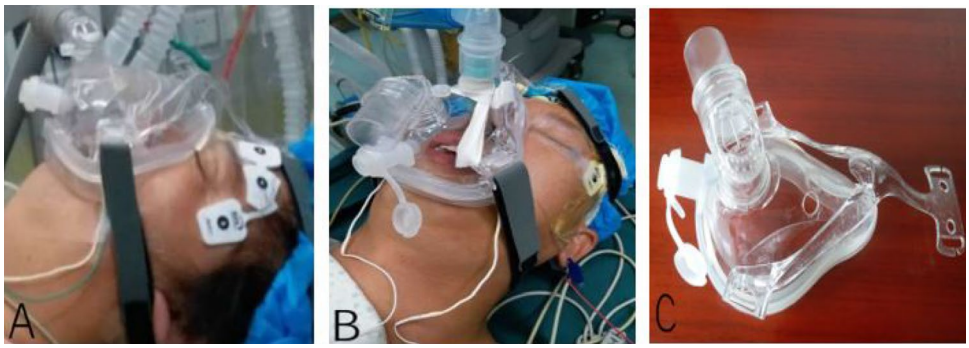

Fig. 1 connection diagram of active and passive Pcrit. **a** Active Pcrit: a modified oral–nasal mask connected to a BiPAP. The electronic laryngoscope went through the lower medial hole of the mask into the patient's upper airway. The superior lateral hole was sealed by a sealing film. **b** Passive Pcrit: the anesthesiologist intubated the patients' trachea in the state of muscle relaxation. The intubation is led out from the superior lateral hole of the mask and connected to the anesthesia machine. Other items are the same as active Pcrit. **c** Modified oral–nasal mask which had two added holes, at the superior lateral and lower medial with an air leakage valve, and connected to a BiPAP

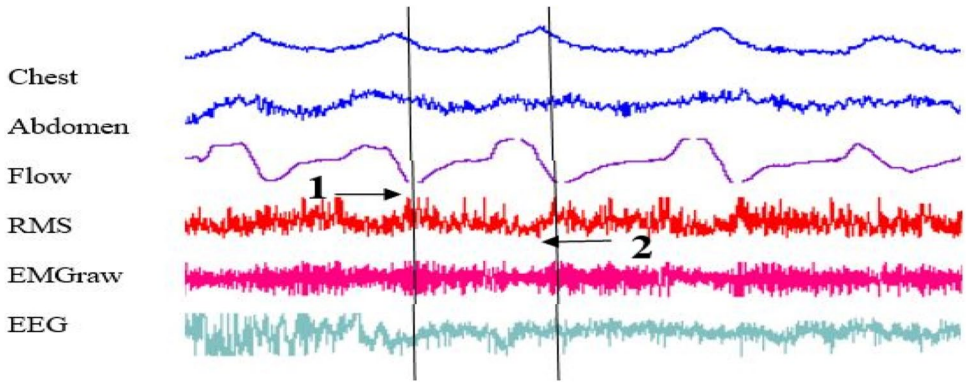

Fig.2 TPEMG in single respiratory cycle of awake period. Example of TPEMG measurement during wakefulness in OSA patients. **a** Peak phasic TPEMG: peak RMS amplitude during inspiration. **b** Tonic TPEMG: lowest RMS amplitude during inspiration *Chest* movement of chest, *Abdomen* movement of abdomen, *Flow* nasal pressure, *RMS* the root mean square (RMS) amplitude of TPEMG, *EMGraw* raw TPEMG signal recording by intraoral electrodes. *EEG* electroencephalogram

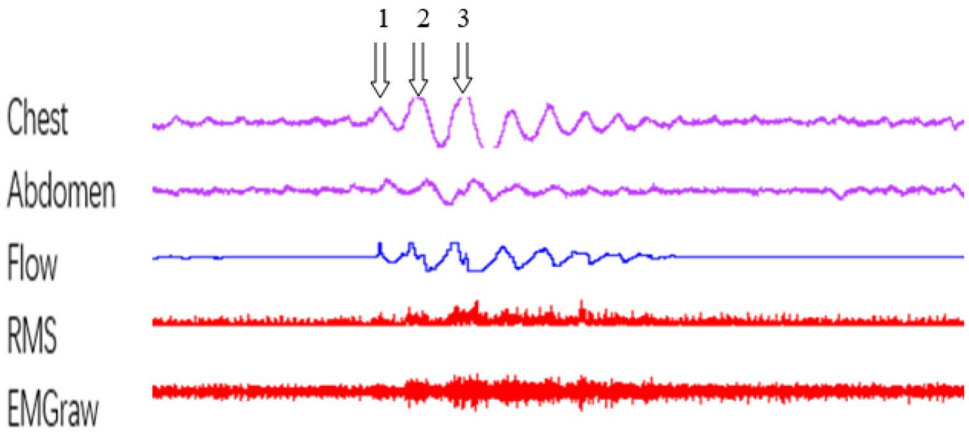

Fig. 3 Changes of nasal airflow, thoracoabdominal movement and TPEMG after apnea in a patient. Patient had the recovery of respiratory movement and the increase of muscle activity compensation after apnea. **a, b,** and **c** Represent the first, second and third respiratory cycles after apnea. It can be seen that the amplitude of the third respiratory cycle is the largest, and the corresponding EMG amplitude is also the largest. Considering the potential delay of apnea on TPEMG activity, we chose the third respiratory cycle to measure the maximum response of TPEMG after apnea. *Chest* movement of chest, *Abdomen* movement of abdomen, *Flow* nasal pressure, *RMS* the root mean square (RMS) amplitude of TPEMG, *EMGraw* raw TPEMG signal recording by intraoral electrodes

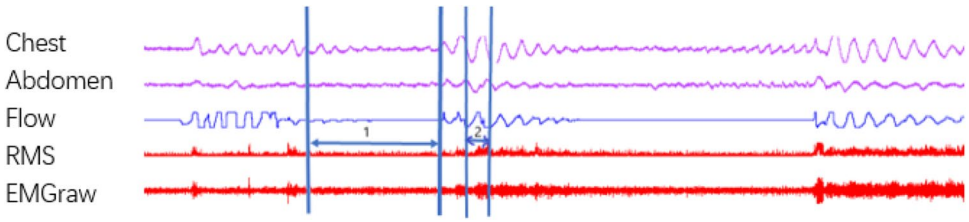

Fig.4 Example of TPEMG measurements during obstructive apnea events in OSA patients during synchronous DISE (**a**) for the minimum activation of TP activity monitored at the onset of the collapse of the pharyngeal cavity, which we considered to be a good period in which to measure the lowest TPEMG in sleep for OSA patients. Apnea TPEMG was defined during each period of disappearance of nasal pressure. **b** For the third respiratory cycle after apnea. Given the potential for the large changes in TPEMG, we analyzed the third respiratory cycle after apnea to quantify TPGME. *Chest* movement of chest, *Abdomen* movement of abdomen, *Flow* nasal pressure, *RMS* the root mean square (RMS) amplitude of TPEMG, *EMGraw* raw TPEMG signal recording by intraoral fine-wire electrodes

equal variances not matching data, Welch's ANOVA was used, and Games–Howell used for comparison of each two groups; for nonnormal distribution data, Kruskal–Wallis H and Mann–Whitney tests were used. Statistical significance was defined as $P < 0.05$. Bonferroni corrected the pair comparison, $P_{adj} < 0.017$.

Results

For the 36 subjects who completed the protocol, the mean age and BMI were 44.81 ± 7.72 years and 27.16 ± 2.95 kg/m^2, respectively. The mean AHI was 31.00 ± 7.72 events/hour of sleep. The mean min SaO_2 and mean SaO_2 during sleep were 76.63 ± 8.51% and 93.40 ± 1.89%, respectively. Subjects' demographics and polysomnography parameters had no statistical difference among the three groups. (Table 1).

Tonic TPEMG

In tonic TPEMG, group 1 showed the highest value during awake and sleep onset, while group 2 was the highest during apnea and after apnea. There was significant difference between group 1 and group 2 ($P = 0.01$) and group 1 and group 3 ($P = 0.00$) during awake; group 1 and group 3 (P = 0.00) during sleep-onset; group 1 and group 2 ($P = 0.01$, $P = 0.00$); and group 2 and group 3 ($P = 0.00$, $P = 0.00$) during apnea and after apnea, respectively. (Table 2, Fig. 5).

Peak TPEMG

In peak TPEMG, group 1 showed the highest value only during awake. Group 2 showed the highest value during other states. There was significant difference between group 1 and group 3 ($P = 0.00$) and group 2 and group 3 ($P = 0.01$) during awake, group 1 and group 2 ($P = 0.01$), and group 2 and group 3 ($P = 0.00$) during after apnea. (Table 2, Fig. 6).

Table 1 Patient demographics, perioperative polysomnography parameters

parameters	Group 1 ($n = 13$)	Group 2 ($n = 14$)	Group 3 ($n = 9$)	Total ($n = 36$)
Age (year)	44.56 ± 7.76	45.00 ± 9.15	44.89 ± 7.04	44.81 ± 7.72
BMI (kg/m^2)	28.96 ± 2.87	26.47 ± 3.04	26.07 ± 2.30	27.16 ± 2.95
AHI (events/h)	31.53 ± 7.96	29.93 ± 7.62	31.54 ± 8.40	31.00 ± 7.72
SupineAHI (events/h)	40.33 ± 3.47	37.57 ± 9.01	33.87 ± 5.13	37.58 ± 4.23
Mean SaO_2 (%)	92.40 ± 2.56	93.84 ± 1.53	93.94 ± 0.99	93.40 ± 1.89
Min SaO_2(%)	74.11 ± 7.27	77.44 ± 11.45	76.63 ± 8.513	76.63 ± 8.51

Patient demographics and polysomnography parameters were no statistical difference among the three groups

Group1 concentric collapse, group 2 anteroposterior collapse, group 3 lateral collapse

BMI body mass index, *AHI* apnea–hypopnea index, *min* SaO_2 lowest oxygen saturation, *mean* SaO_2 mean oxygen saturation during sleep

Table 2 Tensor palatini muscle (TP) EMG data of OSA subjects at awake, sleep onset, apnea, and the third respiratory cycle after apnea

EMG	Groups			*P**	*P***	*P****
	Group 1	Group 2	Group 3			
Tonic TPEMG, (%max)						
Awake	0.05 ± 0.05	0.04 ± 0.01	0.02 ± 0.00	0.01*	0.00**	0.07
Sleep onset	0.03 ± 0.00	0.02 ± 0.00	0.01 ± 0.00	0.02	0.00**	0.10
Apnea	0.01 ± 0.00	0.02 ± 0.00	0.01 ± 0.00	0.01*	0.19	0.00***
After apnea	0.02 ± 0.00	0.04 ± 0.04	0.02 ± 0.00	0.00*	0.65	0.00***
Peak TPEMG, (%max)						
Awake	0.78 ± 0.07	0.65 ± 0.05	0.42 ± 0.07	0.14	0.00**	0.01***
Sleep-onset	0.41 ± 0.06	0.45 ± 0.07	0.39 ± 0.07	0.91	0.74	0.78
Apnea	0.44 ± 0.17	0.53 ± 0.05	0.34 ± 0.05	0.28	0.36	0.02
After apnea	0.62 ± 0.09	0.98 ± 0.06	0.52 ± 0.09	0.01*	0.56	0.00***

*Significant difference between group 1 and group 2

**Significant difference between group 1 and group 3

***Significant difference between group 2 and group 3

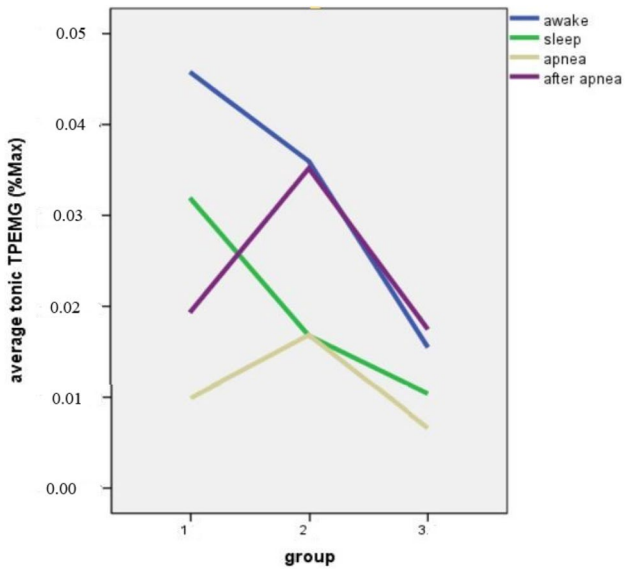

Fig.5 Comparison of groups tonic TPEMG (%max) during awake (quiet breathing), sleep onset, apnea TPEMG and after apnea TPEMG of OSA patients. There was significant difference between group 1 and group 2 ($P=0.01$) and group 1 and group 3 ($P=0.00$) during awake; group 1 and group 3 ($P=0.00$) during sleep onset; group 1 and group 2 ($P=0.01$, $P=0.00$); and group 2 and group 3 ($P=0.00$, $P=0.00$) during apnea and after apnea, respectively

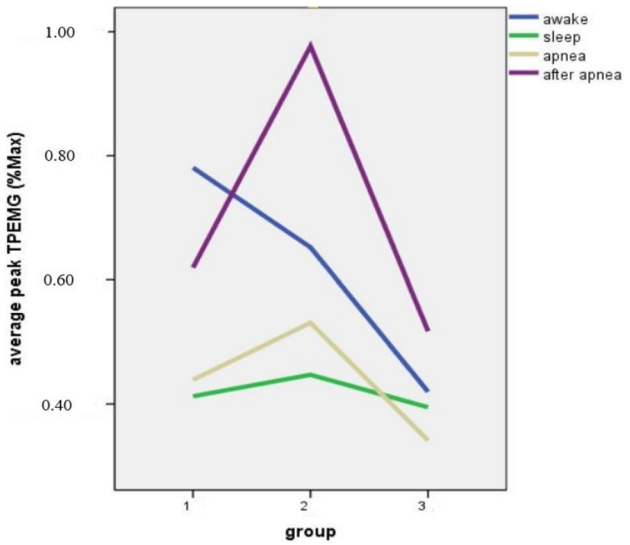

Fig.6 Comparison of groups peak TPEMG (%max) during awake (quiet breathing), sleep onset, apnea TPEMG, and after apnea TPEMG of OSA patients. There was significant difference between group 1 and group 3 ($P=0.00$) and group 2 and group 3 ($P=0.01$) during awake, group 1 and group 2 ($P=0.01$) and group 2 and group 3 ($P=0.00$) during after apnea

Active and passive Pcrit

Passive Pcrit and *D* value (difference between passive Pcrit and active Pcrit) of group 2 were the largest in groups, while group 1 was the highest in active Pcrit. There was statistical difference between group 2 and group 3 ($P=0.00$) in passive Pcrit, between group 1 and group 2 ($P=0.00$), and group 2 and group 3 ($P=0.01$) in *D* value. (Table 3, Fig. 7).

Discussion

The causes of repeated airway collapse in OSA patients during sleep include anatomic stenosis and functional stenosis. The detection of functional stenosis is mainly the detection of neuromuscular compensation function. There were two ways to measure neuromuscular response in this study. The first was the characteristics of TPEMG. TP is the main dilator in the palatopharyngeal region. Under DISE, OSA subjects with velopharyngeal plane stenosis have different external phenotypes—concentric, anteroposterior, and lateral collapse. TPEMG in these subjects has different characteristics in awake, sleep, apnea, and after apnea. The second was the detection of Pcrit. The gold standard for measuring upper airway collapsibility during sleep is the pharyngeal critical closing pressure (Pcrit). Passive Pcrit represents the structural load of UA, and active Pcrit adds the regulatory factors of neuromuscular to UA on the basis of passive Pcrit. The *D* value is different in subjects with different DISE phenotypes.

Pcrit

Passive Pcrit measured in prior studies, while participants were on nasal CPAP in asleep. Subjects were first given the most appropriate pressure relief for respiratory events, where neuromuscular activity was thought to be minimized. CPAP was reduced a brief differential pressure drop to provoke transient airflow limitation, the 'passive' upper airway collapses was simulated, the changes in the nasal airflow was recorded, and the closing pressure when the airflow was zero was calculated. Use of CPAP could relatively minimize pharyngeal dilator muscle's activity [13–16], and propofol alone could also reduce muscle's activity according to the above methods [16–19]. Eastwood PR [20] used propofol 2.5, 4.0, and6.0 μg/ml in random subjects, respectively. With the increase of drug concentration, Pcrit values ranged from -0.3 ± 3.5, 0.5 ± 3.7, to 1.4 ± 3.5 cm H_2O. Increasing depth of propofol anesthesia was associated with increased collapsibility of the upper airway. In that kind of method, because of breath-by-breath changes in UA muscles collapsibility, only the first breath can be studied. UA muscle activity progressive returned.

Table 3 Pcrit-related indicators among groups

Pcrit	Groups			*P**	*P***	*P****
	Group 1	Group 2	Group 3			
Active Pcrit	12.44 ± 2.01	10.56 ± 1.72	9.11 ± 1.42	0.24	0.11	0.64
Passive Pcrit	15.44 ± 2.05	19.33 ± 1.18	13.00 ± 1.21	0.02	0.13	0.00***
D value	3.00 ± 0.85	8.78 ± 2.10	3.89 ± 0.99	0.00*	0.36	0.01***

*Significant difference between group 1 and group 2

**Significant difference between group 1 and group 3

***Significant difference between group 2 and group 3

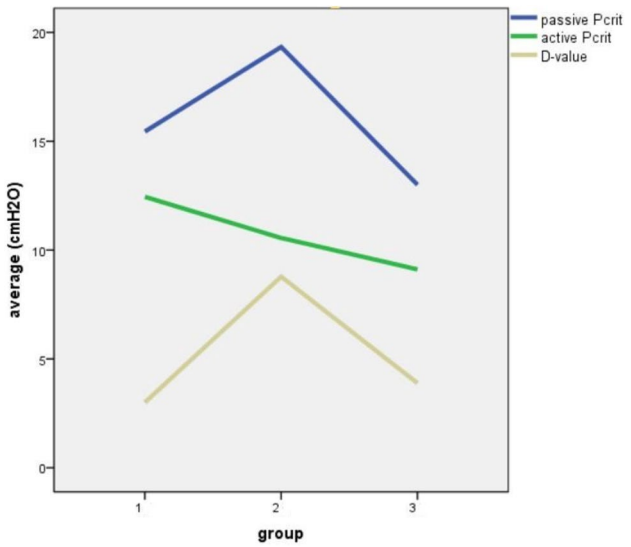

Fig.7 Comparison of groups active and passive Pcrit of OSA patients. There was statistical difference between group 2 and group 3 ($P=0.00$) in passive Pcrit; between group 1 and group 2($P=0.00$) and group 2 and group 3 ($P=0.01$) in D value (difference between passive Pcrit and active Pcrit)

We tried to completely block the autonomic respiration, remove the central drive and reflex input, and the effect of compensatory neuromuscular responses on passive Pcrit test by changing from sedation to anesthesia and intubation.

Catheter presence in the UA could influence UA collapsibility. On one side, it splinted the UA longitudinally, decreased its propensity to collapse; on the other side, a catheter could encroach upon the UA lumen, increase secretion and tube wall edema, and increase pharyngeal resistance, with the potential to increase propensity to collapse [19]. A small catheter (external diameter of 2.7 mm or less) that traverses the pharynx does not affect upper airway collapsibility [19, 21, 22]. Although we used both electronic laryngoscope and anesthesia intubation in the UA, the influencing factors were the same for the three groups of subjects. We focused on comparing the differences (D value) among the three groups.

TPEMG

Although TP does not directly raise or lower the soft palate or directly expand the airway, its tonic activity in inspiratory made UA less prone to deformation by negative pressure produced by diaphragm. Andrew J.M et al. found that rapid pulses of negative pressure could stimulate TP, which could change pharyngeal wall tension, thus affecting Pcrit and reducing the collapse of soft palate [23]. Eisele DW et al. and Schwartz AR et al. found that selective stimulation of TP could reduce Pcrit by 2.4 cm H_2O [24, 25].

When OSA patients have respiratory events, they are excited by chemical and pressure receptor, often accompanied by arousal. The change of state improves the excitability of TP, but different phenotypes of collapse pattern at velum level OSA patients had different myoelectric activity responses. It could be seen from this study that the subjects with concentric collapse had the highest UA collapsibility, the subjects with anteroposterior collapse had the largest structural load, but the neuromuscular response was also the largest.

Multiple factors can modulate upper airway muscle activity. These include sleep–wake state, central pattern generator input, and chemical and negative pressure reflex input and lung volume [26]. Excitatory TP has different motor controls from GG. It has unchanged activity in response to chemical and negative pressure reflex input. Its major input appears to be the wakefulness stimulus [12]. Therefore, the parameters related to respiratory effort, such as esophageal manometry, intercostal and diaphragm electromyogram, were not included in our experimental analysis.

Limitations of this study

We acknowledge a number of potential limitations in this study. First, the sample size of our study was limited which may affect the scientific power of the analyses of data. A multicenter study that can include a greater number of patients has been proposed to further test the hypothesis. Second, we just analyze the TPEMG values at simulative of N2 sleep stage under DISE, not at various sleep stages. This

is because the limitations of current research, the operation under the DISE cannot restore all the natural sleep stages. The response of TPEMG in other sleep stages of the different phenotypes OSA subjects needs further study. The anesthesia depth control under DISE needed to be more accurate, because different anesthesia depth, muscle activity change, and awakening response were different. Third, passive Pcrit detection although we completely blocked the neuromuscular regulation of UA through the use of anesthetized muscle relaxants, but anesthesia intubation and electronic laryngoscope have a certain impact on the collapse of UA, we need to further explore better detection methods.

Conclusions

As a result, our study results revealed that during different states of awake, sleep onset, apnea, and after apnea, the response force of tensor palatini muscle of OSA subjects with different phenotypes under DISE was different. EMG activity during awake and sleep onset, group 1 was the highest, during apnea and after apnea, the compensatory effect of group 2 was significantly high. The anatomic load (passive Pcrit) and the neuromuscular compensatory effect (*D* value) were the largest in group 2. Group 1 had the largest active Pcrit value and was most prone to collapse.

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