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Correlation of surface respiratory electromyography with esophageal diaphragm electromyography

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ABSTRACT

Objectives: To assess the correlation between surface respiratory electromyography(sEMG) and esophageal diaphragm electromyography(EMGdi) at different levels of neural respiratory drive (NRD).
Design: Randomised parallel design controlled trial.
Setting: The First Affiliated Hospital of Guangzhou Medical University.
Participants: 15 healthy subjects and 1 severe to very severe stable COPD patients were studied.
Interventions: 15 healthy subjects performed incremental inspiratory threshold loading (ILT) and 15 stable COPD patients underwent noninvasive positive pressure ventilation (NPPV).The correlation between EMGdi and sEMG at different NRD levels was analyzed. EMGdi was performed with a multi-pair esophageal electrode catheter; sEMG were was performed by surface diaphragm EMG(located in right anterior axillary line and left anterior axillary line respectively expressed as sEMGdi(r) andsEMGdi(l))、surface parasternal EMG(sEMGpara),and surface sternocleidomastoid EMG(sEMGsc).Signals were normalized using the peak EMG expressed as EMG% max.
Primary and secondary outcome measures: The mean ± standard deviation resting EMGdi%max was higher in patients with COPD than in healthy subjects (57.26% ± 15.45% vs13.64% ± 4.96%, respectively; $p < 0.001$).During ILT and NPPV, EMGdi was correlated with sEMGdi (r), sEMGdi (1), sEMGpara and sEMGsc ($r = 0.90, 0.87, 0.90, 0.90$ and $r = 0.92, 0.83, 0.92$ and 0.71, respectively; all $P < 0.001$).
Conclusion: A strong relationship is present between NRD measured by EMGdi%max and NRD measured by sEMG%max. sEMG%max serves as a non-invasive marker of NRD.

1. Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by persistent, irreversible airway obstruction that leads to increased neural respiratory drive (NRD). In healthy people, NRD increased slightly with age (Jolley et al., 2009). Some studies have suggested that NRD testing can be used in patients with COPD to evaluate the efficacy of inhaled bronchodilators (Li et al., 2016) and predict the risk of exacerbation (Murphy et al., 2011; Suh et al., 2015). NRD can be measured with minute ventilation, inspiratory pressure change, mean inspiratory flow and electromyography(EMG) of the inspiratory muscles(except in patients with neuromuscular diseases such as ALS).However, the first three methods are influenced by airway resistance and compliance of respiratory system (Jensen et al., 2011; Whitelaw and Derenne, 1993). Therefore, EMG is the most reliable technique for evaluation of NRD in humans, particularly in COPD patients (Luo et al., 2008).

There are two main methods for detecting EMG, namely, esophageal diaphragm EMG (EMGdi) and surface respiratory EMG (sEMG). EMGdi is measured by an esophageal multi-pair electrode catheter placed in the crural of the diaphragm (Luo et al., 2008). sEMG is recorded with surface electrodes placed in the skin of the respiratory muscles and includes surface diaphragm EMG(sEMGdi), [8] surface parasternal EMG(sEMGpara)and surface sternocleidomastoid EMG(sEMGsc) (Duiverman et al., 2004; Maarsingh et al., 2000). Previous studies have shown that in patients with cystic fibrosis (Reilly et al., 2011) and healthy individuals (Laghi et al., 2014) the sEMGpara provides a measure of NRD similar to that provided by EMGdi at rest and during incremental exercise. However, no relevant study involving patients with COPD has been performed in the past decade, and no data are available for the other various sEMG techniques.

The aim of this study was to examine and compare the relationship between NRD measured by EMGdi and that measured by sEMG at different levels. We hypothesized that a high correlation is present especially between EMGdi and sEMGdi.

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2. Methods

2.1. Ethical approval

This study has been reviewed and published at ClinicalTrials.gov (identifier: NCT03268616, date of registration: 30 August, 2017). The study was approved by The First Affiliated Hospital of Guangzhou Medical University Ethics Committee, and all participants provided written informed consent.The study conformed to the provisions of the Declaration of Helsinki.

2.2. Participants

15 healthy subjects and 15 severe to very severe stable COPD patients were studied. Initial patient management was performed according to standard local guidelines with oral corticosteroids, antibiotics,or a combination. None of the participants had undergone systemic application of corticosteroids for nearly 4 weeks, and none had neurological or cardiovascular disease.

2.3. EMG measurements

2.3.1. EMGdi

EMGdi measurements were recorded from the crural diaphragm using a multi-pair esophageal electrode catheter (Guangzhou Yinghui Medical Equipment Co. Ltd, Guangzhou, China). The electrode catheter comprised nine consecutive recording electrode coils, numbered from 1 to 9 (proximal to distal). Each coil was 10 mm in length, with a 0.5 mm space between adjacent recording electrodes. The external diameter of the catheter was 2 mm. Five electrodes pairs were formed, with an interelectrode distance of 3.2 cm between each recording pair as follows: 1 and 5 (1), 2 and 6(2), 3 and 7 (3), 4 and 8 (4), and 5 and 9 (5). A tenth electrode was positioned 2 cm proximal to electrode coil number 1, which was the reference electrode.[1] Prior to placement of the esophageal electrode catheter, the nasal mucosa and laryngopharynx were anaesthetized using a topical anesthetic drug. The optimal position of the electrode catheter was characterized by high EMG activity from pairs 1 and 5 and low EMG activity from pair 3 during breathing at rest.[7] The electrode catheter was then securely taped at the nose.

2.3.2. sEMGdi

Bipolar sEMGdi recordings were made from electrodes placed bilaterally.The recording electrodes were selected at the anterior axillary line and the 6th-8th intercostals junction areas, and the reference electrodes were placed in the bottom and away from the recording electrodes. (Wu et al. (2017))

2.3.3. sEMGpara

Electrodes were selected in the second intercostal spaces (one electrode left and one right), 3 cm away from the sternum, and the reference electrode was placed at the sternal angle (Duiverman et al. (2004); Maarsingh et al. (2000).

2.3.4. sEMGsc

Electrodes were selected at the intersection between the upper one-third and the lower one-third of the sternocleidomastoid at an interval of 3–5 cm,and the reference electrode was placed in the sternal fossa. (Duiverman et al. (2004).

2.4. EMG recording

The EMG signals were amplified and bandpass filtered between 20-Hz and 1k-Hz, and at a magnification of 1000 times (RA-8R biomedical amplifier; Yinghui Medical Tech Ltd., Guangzhou, China).The data were acquired and displayed on a computer running LabChart software (Chart version 7.5; ADInstruments, Colorado Springs, CO, USA).With analog-to-digital sampling of 10 kHz, raw EMG signals were converted to the root mean square (RMS) using LabChart software. The maximal EMG per breath was then manually determined by selecting EMG signals falling between QRS complexes of the ECG artifact.

The respiratory muscle EMGs data were normalized to maximal EMGdi,[3] which obtained during the following 3 measurements: 1) maximal inspiration to total lung capacity (TLC); 2) maximal static inspiratory effort at functional residual capacity (FRC) against a closed

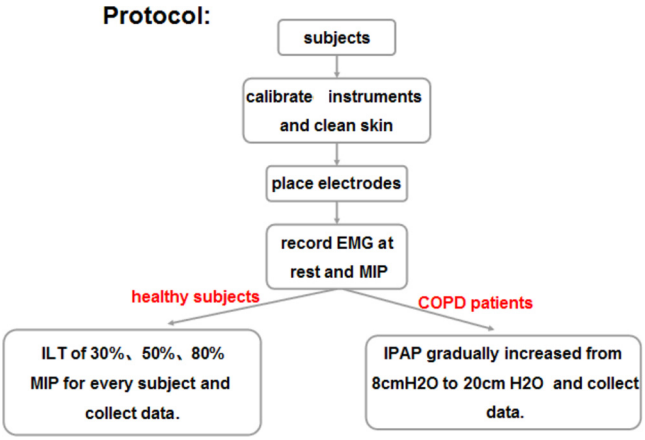

Fig. 1. Study protocol.

Table 1
Anthropometric characteristics and pulmonary function variables for healthy subjects and COPD patients.

	healthy subject	COPD patient	P Value
Subjects(n)	15	15	
Age(years)	29.32 ± 3.04	59.29 ± 7.09	〈0.001
Weight (kg)	72.34 ± 5.42	66.38 ± 3.42	0 . 44
Height (cm)	175.56 ± 12.36	169.54 ± 4.44	0 . 11
BMI(kg/m2)	23.57 ± 3.06	22.72 ± 1.66	0 . 55
FEV1(L)	2.64 ± 0.73	0.94 ± 0.18	< 0.001
FEV1%pred(%)	100.61 ± 1.43	28.46 ± 10.43	< 0.001
FVC(L)	3.02 ± 0.71	2.62 ± 0.64	< 0.001
FVC%pred(%)	87.07 ± 5.09	72.57 ± 18.09	< 0.001
FEV1/FVC(%)	83.41 ± 2.35	40.84 ± 7.19	< 0.001
MIP(cmH20)	79.22 ± 7.44	23.34 + 9.38	< 0.001
MIP%pred(%)	95.2 ± 7.22	30.12 ± 13.44	< 0.001

Data are expressed as mean ± standard deviation.
Abbreviations: COPD: chronic obstructive pulmonary disease; BMI: body mass index; FEV1: forced expiratory volume in 1 s; FVC: forced vital capacity; MIP: maximal inspiratory pressure.

Table 2
Resting levels of NRD in healthy subjects and COPD patients.

	healthy subject	COPD patient	P Value
EMGdi max(%)	13.64 ± 4.96	57.26 ± 15.45	〈0.001
sEMGdi(r)max(%)	8.81 ± 2.67	49.33 ± 15.33	〈0.001
sEMGdi(l)max(%)	7.20 ± 1.41	51.33 ± 13.91	〈0.001
sEMGpara max(%)	8.17 ± 1.15	48.45 ± 10.48	〈0.001
sEMGsc max(%)	7.06 ± 5.32	45.62 ± 16.48	〈0.001

Data expressed as mean ± standard deviation.
Abbreviations: COPD: chronic obstructive pulmonary disease; EMGdimax (%):diaphragmatic EMG activity normalized to maximal evoked EMG during maximal inspiratory pressure (MIP); sEMGdi(r)max(%):surface diaphragmatic EMG(right) activity normalized to maximal evoked EMG during MIP; sEMGdi(l) max(%):surface diaphragmatic EMG(left) activity normalized to maximal evoked EMG during MIP; sEMGpara max(%):surface parasternal EMG activity normalized to maximal evoked EMG during MIP; sEMGsc max(%):surface sternocleidomastoid EMG activity normalized to maximal evoked EMG during MIP.

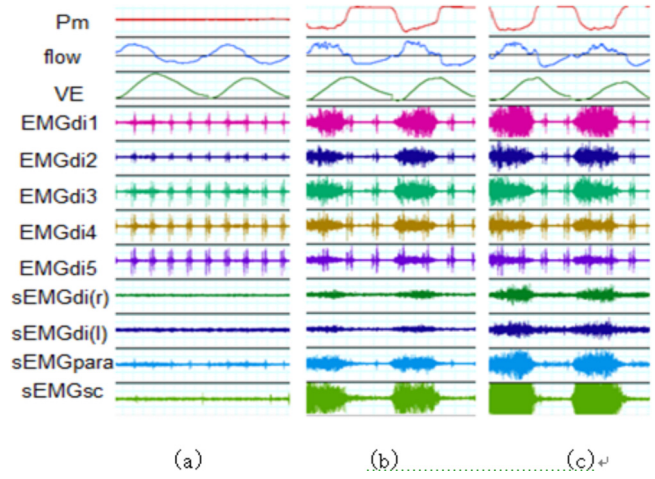

Fig. 2. Electromyogram of healthy subjects at resting (a),with ILT at 30% MIP (b),and with ILT at 80% MIP(c).

valve ;3) maximal sniff from FRC. As in a previous study,[1] maximum voluntary ventilation was also performed to obtain maximal EMGdi signals, since it is not generally recommended for patients with known or suspected respiratory muscle weakness but may be helpful in the assessment and monitoring of patients with extrapyramidal disorders, we did not use this measurement in this trial. The maneuvre was repeated at least five times, until three reproducible efforts < 10% variance were obtained (ATS/ERS, 2002).

2.5. Pulmonary ventilation function test

Indicators mainly included the forced vital capacity (FVC), FVC percentage of expected(FVC%), forced expiratory volume in 1 s (FEV1),FEV1 percentage of expected(FEV1%),and one second rate (FEV1/FVC%)(pulmonary function test: Cosmed, Rome, Italy).

2.6. Respiratory flow and capacity (volume) recordings

Pressure changes were measured using a laminar flowmeter (MLT300 L, ADInstruments–Australia, Bella Vista, New South Wales, Australia) and a pressure sensor (ML141; ADInstruments-Australia).The respiratory volume was measured by integrating the flow rate with respect to time.

2.7. Inspiratory threshold load (ILT)

For healthy subjects, ILT (CH et al., 2003) was performed using a water-sealed ILT respiratory muscle exerciser (Shanghai Qianshan Medical Technology Co., Ltd., Shanghai, China).

2.8. Non-invasive positive-pressure ventilation (NPPV)

For patients with COPD, the BiPAP Vision Ventilator (Philips Respironics, Murrysville, PA, USA) was used to reduce NRD (Nava et al., 1993). With S / T mode, and the expiratory positive airway pressure was set at 4 cm H_2O and remained constant. The inspiratory positive airway pressure (IPAP) gradually increased from 8 to 20 cm H_2O (corresponding to a gradual increase in the pressure support ventilation level from 4 to 16 cm H_2O).

2.9. Protocol (Fig.1)

Before the start of the experiment, the instruments for pressure and flow rate measurement were calibrated, and the skin was thoroughly cleaned with alcohol to optimize skin impedance. The electrode was then introduced through the nostril into the nasopharynx and swallowed down the esophagus, and the surface electrodes were placed as described above. EMG signals were recorded at rest and maximal inspiratory pressure (MIP).

For each healthy subjects, after a short period of unloaded breathing, ITLs of 30%、50% and 80% predetermined MIP for every subject were imposed for 3 min, with a 5 min rest between each load. For each patient with COPD, IPAP gradually increased from 8 to 20 cm H2O (2 cmH2O).Before the EMG measurement was made, each pressure support level was ventilated for 20 min.The data were collected in a breath-by-breath manner.

2.10. Data analysis

Data are expressed as mean ± standard deviation. Variables measured during ILT and NPPV were compared with repeated-measures analysis of variance, and the least significant difference test was used for multiple comparisons. Pearson's correlation analysis and Bland-Altman analysis were used to evaluate the relationship between the EMGdi and sEMG at various levels.All statistical analyses were performed using SPSS 21.0 statistical software (IBM Corp., Armonk, NY, USA).A P value of < 0.05 was considered statistically significant.

3. Result

In total, 30 subjects were studied.15 were healthy subjects (mean age, 29.3 ± 3.04 years) and 15 were stable COPD patients (mean age 59.3 ± 7.09 years).All subjects were male. The subjects'characteristics information, lung function and resting state NRD are summarized in Tables 1 and 2.

3.1. ILT

Mean minute ventilation increased from 9.30 ± 0.45 to 10.70 ± 3.23 L with an increase in ILT (P = 0.89). The tidal volume increased from 0.60 ± 0.09 L to 0.62 ± 0.14 L(P = 0.64), and the respiratory rate decreased from 17.50 ± 2.36 to 17.05 ± 2.00 breaths

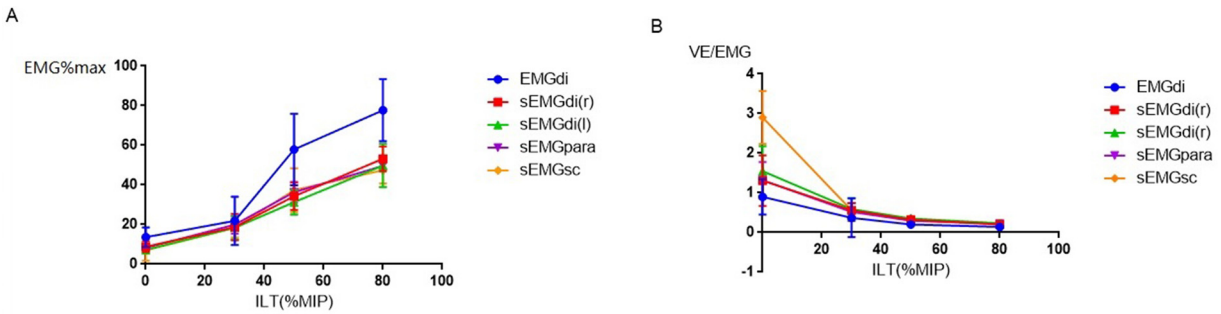

Fig. 3. (a) NRD and (b) neuroventilatory efficiency during gradually increasing ILT in healthy subjects.(Data are expressed as mean ± standard deviation.).

Table 3
Normalized RMS with gradually increasing ILT in healthy subjects.

ILT	EMGdi max(%)	sEMGdi(r) max(%)	sEMGdi(l) max(%)	sEMGpara max(%)	sEMGsc max(%)
SB	13.64 ± 4.96	8.81 ± 2.67	7.20 ± 1.41	8.17 ± 1.15	7.06 ± 5.32
30%MIP	21.86 ± 12.1[a]	18.70 ± 6.67[a]	18.38 ± 5.85[a]	19.95 ± 4.52[a]	18.36 ± 4.86[a]
50%MIP	57.89 ± 18.11[a]	34.31 ± 6.96[a]	31.27 ± 6.26[a]	36.28 ± 5.25[a]	37.37 ± 11.0[a]
80%MIP	77.69 ± 15.71[a]	53.18 ± 6.25[a]	49.78 ± 10.83[a]	49.63 ± 1.83[a]	47.43 ± 6.87[a]
F Value	21.84	75.63	48.61	176.37	42.23
P Value	〈0.001	〈0.001	〈0.001	〈0.001	〈0.001

Data expressed as mean ± standard deviation.
Abbreviations: RMS: root mean square;SB: spontaneous breathing; MIP: maximal inspiratory pressure; EMGdimax(%):diaphragmatic EMG activity normalized to maximal evoked EMG during MIP; sEMGdi(r)max(%):surface diaphragmatic EMG(right) activity normalized to maximal evoked EMG during MIP; sEMGdi(l)max (%):surface diaphragmatic EMG(left) activity normalized to maximal evoked EMG during MIP; sEMGpara max(%):surface parasternal EMG activity normalized to maximal evoked EMG during MIP; sEMGsc max(%):surface sternocleidomastoid EMG activity normalized to maximal evoked EMG during MIP.
[a] denotes a significant difference between MIP and SB,P < 0.05.

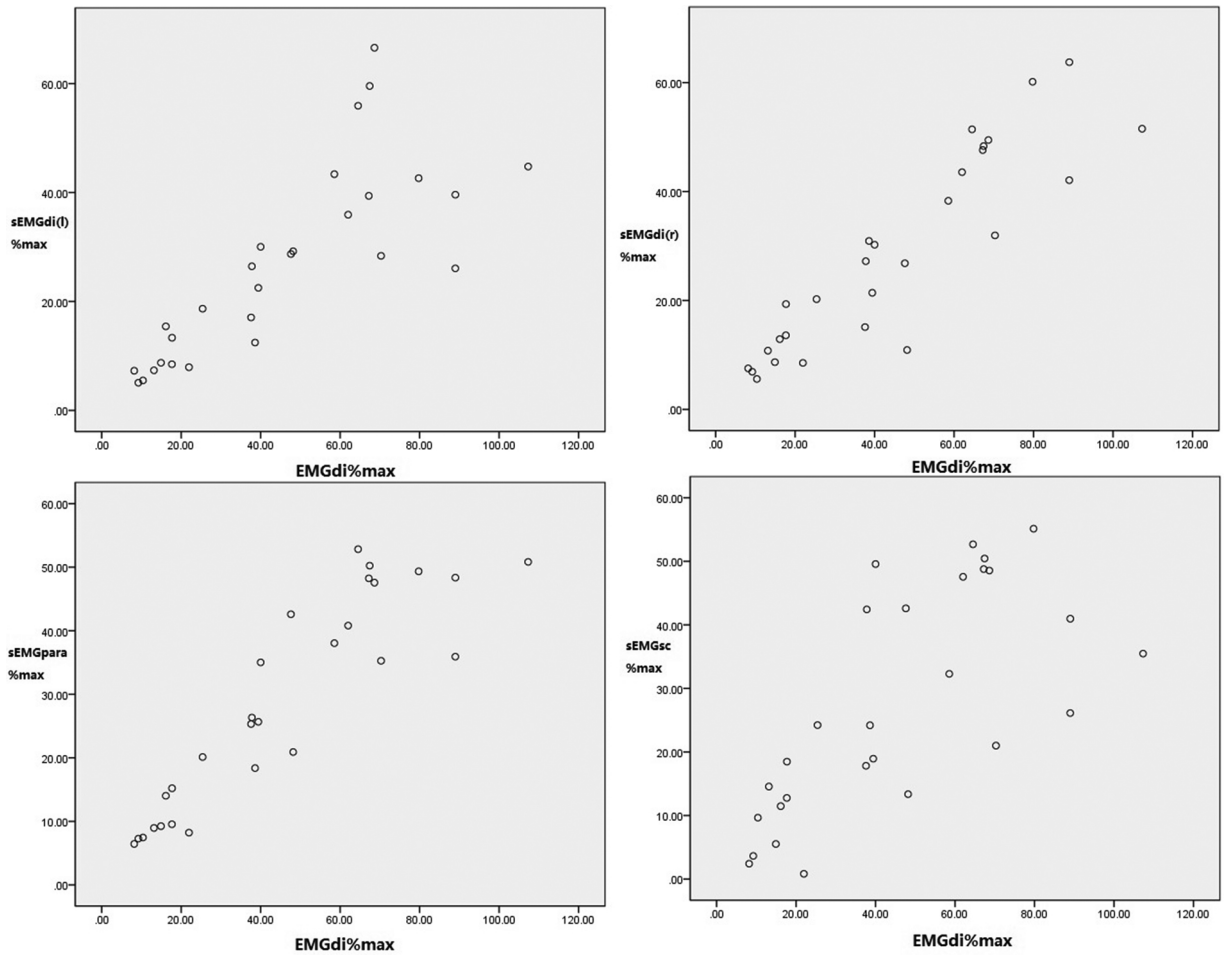

Fig. 4. Scatter diagram of EMGdi%max and sEMG%max during gradually increasing ILT in all healthy subjects.

per minute (bpm) (P = 0.69), and no significant difference were observed compared with spontaneous respiration. The NRD of all muscles was increased (P < 0.05) (Figs. 2 and 3; Table 3).

The EMGdi and sEMGdi (r), sEMGdi (l), sEMGpara, sEMGsc were strongly correlated during the ITL protocol (r = 0.90、0.87、0.90、0.70, P = 0.00, Figs. 4 and 5).

3.2. NPPV

As IPAP increased, mean minute ventilation increased from 6.33 ± 1.38 L to 12.59 ± 2.60 L (P < 0.001);this increase was principally due to an increased tidal volume (from 0.42 ± 0.06 to 0.81 ± 0.14 L, P < 0.001) with minimal change in the respiratory rate (decreased from 15.77 ± 3.83 to 15.50 ± 1.96 breaths per minute, P = 0.90).The NRD of all muscles showed a gradual decrease, then increased at the highest IPAP (P < 0.05)(Figs. 6 and 7; Table 4).

The EMGdi and sEMGdi (r), sEMGdi (l), sEMGpara,and sEMGsc were strongly correlated during the NPPV protocol (r = 0.92,0.83,0.92 and 0.71, P < 0.001) (Figs. 8 and 9).

4. Discussion

The main finding in this study is the strong correlations between EMGdi%max and sEMG%max, in both healthy subjects and stable

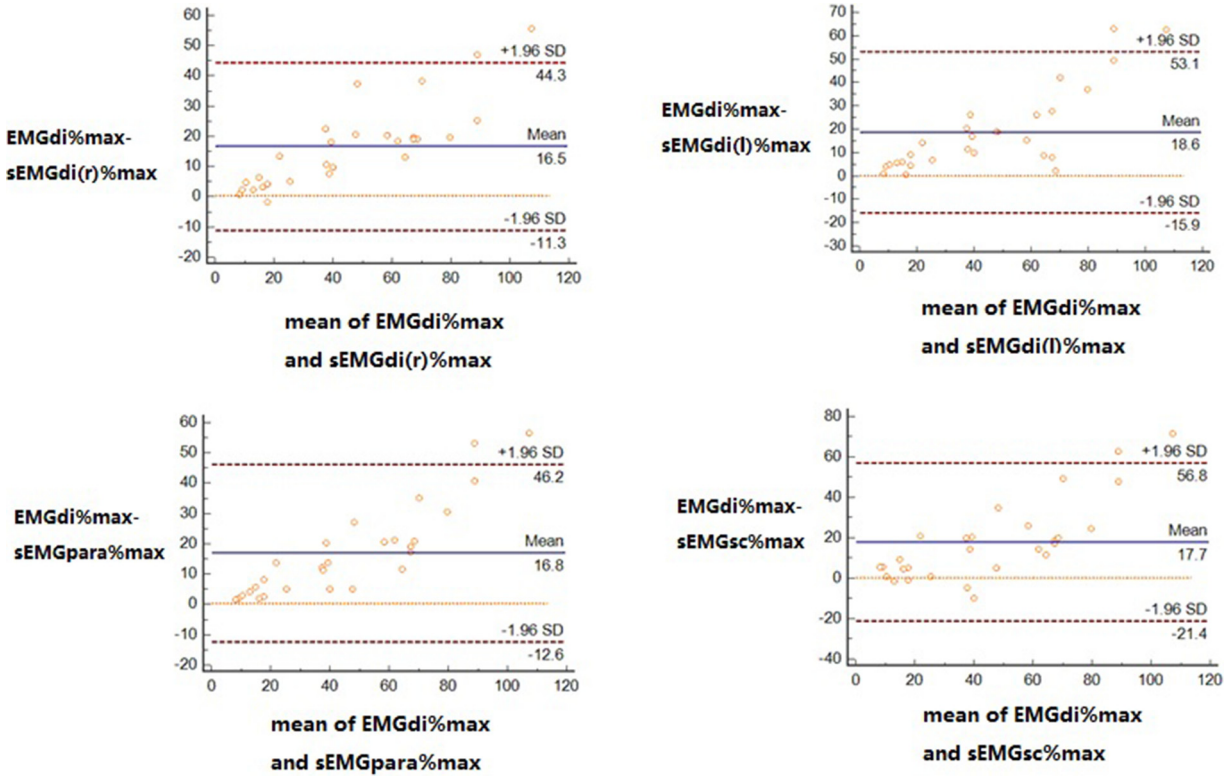

Fig. 5. Bland–Altman analysis of the agreement between EMGdi% max and sEMG% max during gradually increasing ILT in healthy subjects.

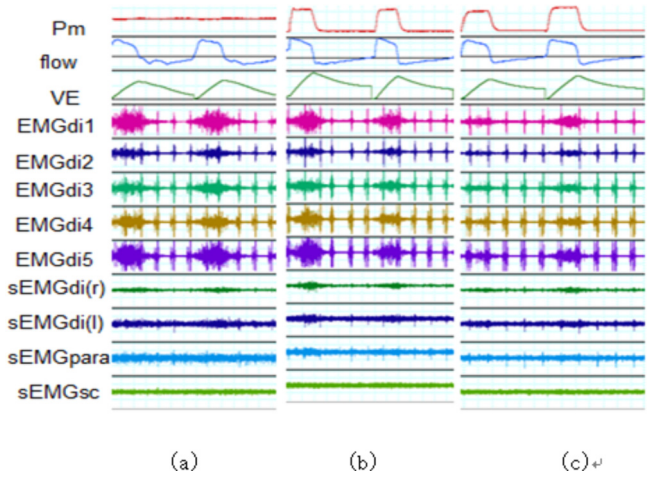

Fig. 6. Electromyogram of COPD patients at resting (a),with IPAP of 10cmH2O (b),and with IPAP of 16 cmH2O (c).

COPD patients during the performance of ILT to increase NRD or NPPV to reduce NRD. Additionally NRD of patients with COPD was significantly higher than that of healthy subjects.

The EMGdi was recorded from the crural diaphragm using a multipair esophageal electrode catheter (Luo et al., 2008) because this site is far away from the chest wall and close to the diaphragm, reducing contamination from the EMG of other respiratory muscles. However, catheter placement in the esophagus is necessary for this measurement, which limits its use in daily practice. Additionally, the sEMG signal is the superposition of motor unit action potential trains show in the skin when the muscle is excited, and the signal exhibits weak non-stationarity. Some researchers (Luo et al., 2008; Boccia et al., 2015) have considered that its inaccuracy is related to subcutaneous fat thickness, electrode placement, peripheral interference, and other factors. During the data acquisition process,the normalized root mean square can be used to exclude the influence of fat thickness; the electrode should be placed parallel to the muscle, which can produce the largest EMG signal,and avoiding metal electromagnetic waves interference can also improve the accuracy of sEMG. This technique is noninvasive and convenient,and its dynamics make it more practical in the clinical

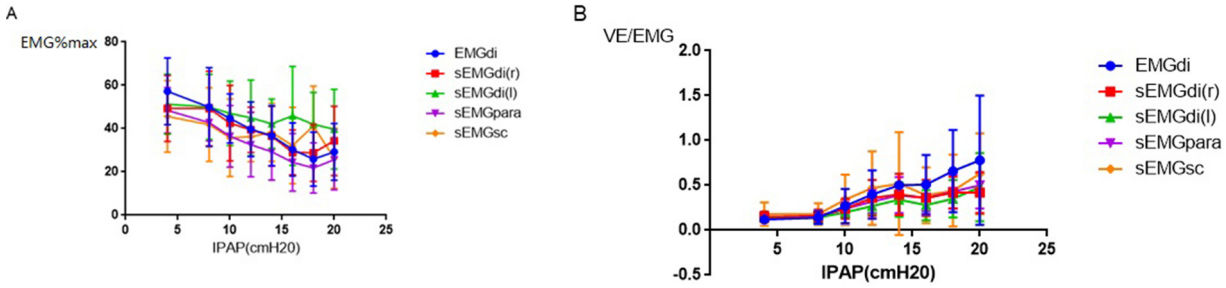

Fig. 7. (a) NRD and (b) neuroventilatory efficiency during gradually increasing NPPV in COPD patients.(Data are expressed as means ± standard deviation.).

Table 4
Normalized RMS with gradually increasing NPPV in COPD patients.

IPAP(cmH2O)	EMGdi max(%)	sEMGdi(r) max(%)	sEMGdi(l) max(%)	sEMGpara max(%)	sEMGsc max(%)
SB	57.26 ± 15.45	49.33 ± 15.33	51.33 ± 13.91	48.45 ± 10.48	45.62 ± 16.48
8	49.88 ± 18.24	49.33 ± 15.33	49.99 ± 15.06	42.74 ± 8.42	41.85 ± 17.00
10	44.67 ± 11.37	42.52 ± 17.39	47.03 ± 14.88	36.40 ± 14.26	35.74 ± 17.87
12	39.69 ± 12.62	39.46 ± 10.59	44.95 ± 17.49	32.59 ± 14.88	36.25 ± 11.47
14	36.67 ± 13.88	36.47 ± 13.83	42.24 ± 11.41	29.32 ± 13.11[a]	38.25 ± 13.38
16	30.34 ± 12.30[*]	28.95 ± 10.33	45.93 ± 22.93	24.45 ± 13.31[a]	32.14 ± 17.71
18	25.89 ± 12.49[a]	28.84 ± 13.24	41.97 ± 14.74	21.81 ± 11.57[a]	41.22 ± 18.39
20	29.26 ± 13.09[a]	34.35 ± 16.02	39.70 ± 18.40	25.66 ± 13.88[a]	26.31 ± 13.89
F Value	2.06	0.99	0.30	2.65	0.35
P Value	0.06	0.45	0.95	0.02	0.93

Data expressed as mean ± standard deviation.
Abbreviations: RMS: root mean square; SB: Spontaneous breathing; IPAP: inspiratory positive airway pressure; EMGdimax(%):diaphragmatic EMG activity normalized to maximal evoked EMG during MIP; sEMGdi(r)max(%):surface diaphragmatic EMG(right) activity normalized to maximal evoked EMG during MIP; sEMGdi (l)max(%):surface diaphragmatic EMG(left) activity normalized to maximal evoked EMG during MIP; sEMGpara max(%):surface parasternal EMG activity normalized to maximal evoked EMG during MIP; sEMGsc max(%):surface sternocleidomastoid EMG activity normalized to maximal evoked EMG during MIP.
[a] denotes a significant difference between IPAP and SB,$P < 0.05$.

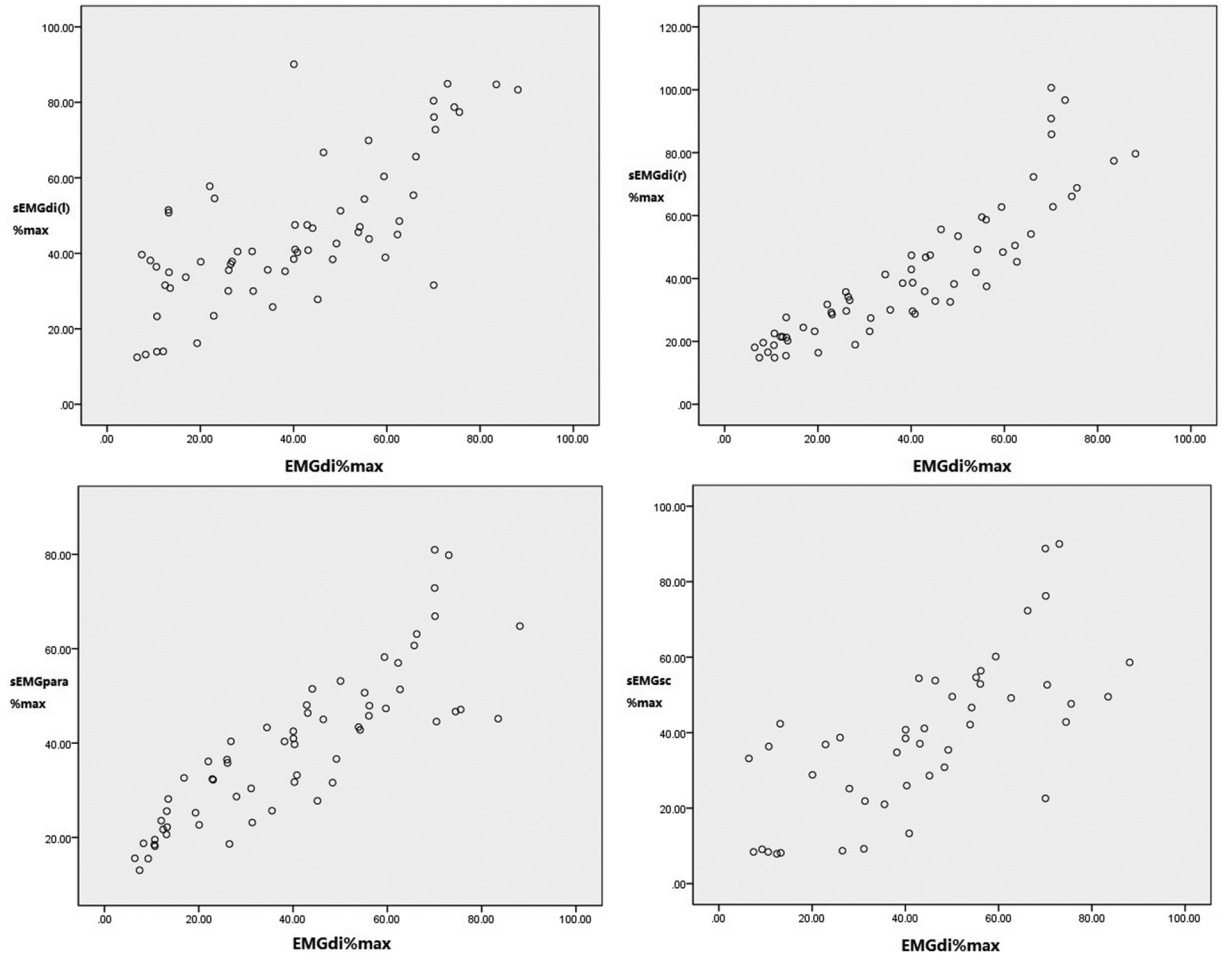

Fig. 8. Scatter diagram of EMGdi%max and sEMG%max during gradually increasing NPPV in COPD patients.

setting.The NRD level is low in healthy subjects, and the EMG signal is weak;this signal does not appear in the spontaneous breathing state. ILD (Laghi et al., 2014) or CO2 rebreathing (Wanke et al., 1992; Reilly et al., 2013) are experimental model commonly used to represent obstructive lung disease. In COPD, mechanical abnormalities including airflow obstruction, static and dynamic hyperinflation and intrinsic positive end-expiratory pressure increase the load on the respiratory muscles.[1] The translation of inspiratory muscle contraction into negative intrathoracic pressure and of pressure changes to ventilation is impaired as a consequence of muscle shortening, increased velocity of contraction, alterations in geometry, and reduced compliance of the respiratory system (Ju and Chen, 2014). This results in high NRD in COPD patients, and the EMG signal is obvious during spontaneous breathing. The present study showed a strong correlation between NRD measured by EMGdi%max and sEMG%max, especially with sEMGpara, and the lowest correlation with sEMGsc. Another study (De Troyer,

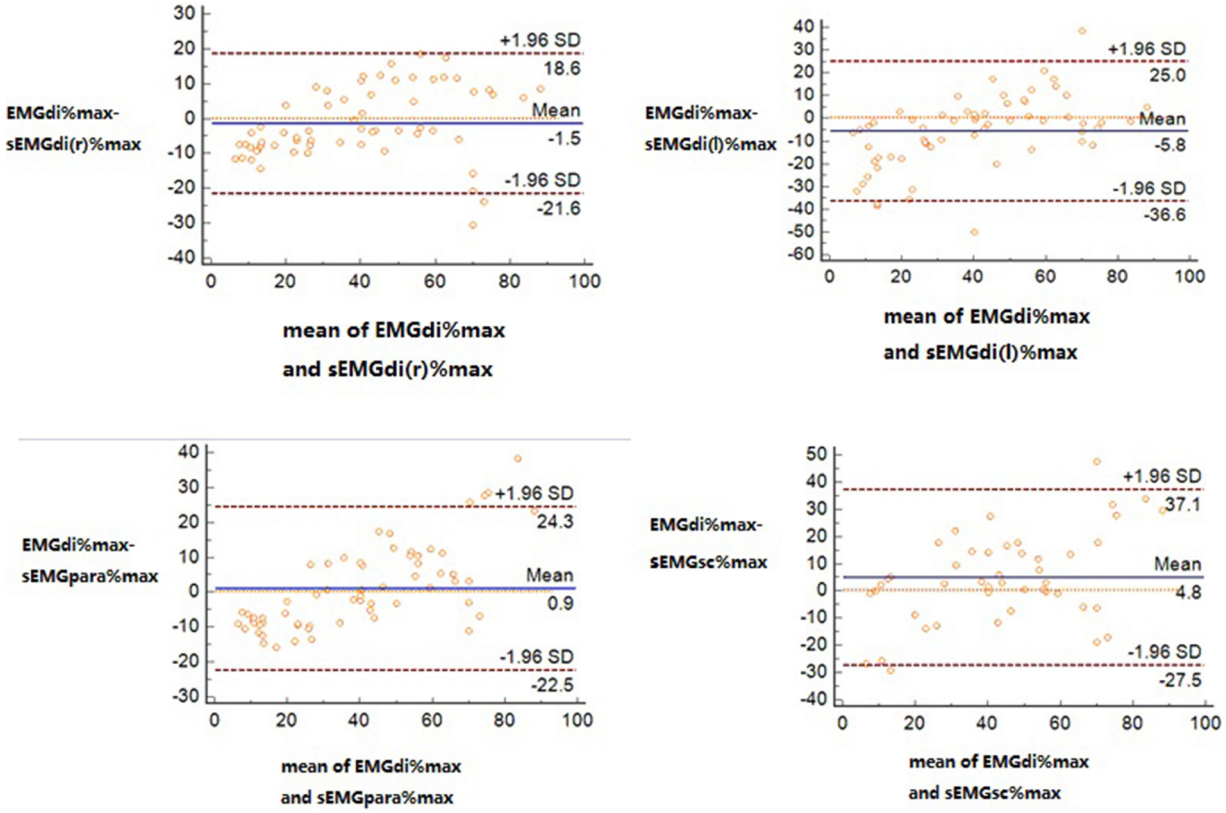

Fig. 9. Bland–Altman analysis of the agreement between EMGdi%max and sEMG%max during gradually increasing NPPV in COPD patients.

1991) suggested that activation of the parasternal intercostal muscles and diaphragm depends on a common neural pathway or, alternatively, that their activation depends on different parallel pathways that are normally activated together;thus,a high correlation was shown in the diaphragm, especially when using accessory breathing muscles during dyspnea(as in sever COPD patients).Additionally,a low correlation with sEMGsc is mainly related to the anatomical location and function of the muscle: (Cardoso et al., 2016) in the deep of platysma muscle where the neck muscles are asymmetrical and contamination from cross-talk[13] is obvious, and where main function is to maintain head movement, EMG may represent NRD only when the head is completely fixed,. Also in the present study, the correlation between EMGdi and sEMGdi(r) was much higher than that between EMGdi and sEMGdi (l), probably because the existent of electrocardiography, and sample error also couldn't be ruled out. Bland-Altman analysis shown that the biased values in COPD patients were much lower than that in healthy subjects, meaning that the agreement between EMGdi and sEMG was better in COPD patients. We consider that this is because COPD patients constantly activate the accessory breathing muscles. In COPD patients, we found the lowest biased values (-0.9%) was between EMGdi and sEMGpara and the highest biased values was between EMGdi and sEMGsc, as discussed above. Bland-Altman analysis also revealed thatpoints outside the 95% confidence intervals(CI) were acceptable in clinical practice, especially between EMGdi and sEMGpara indicating that there was good consistency.

In the present study, the average EMGdi%max in the healthy individuals and patients with COPD was slightly higher than the levels described previously by Jolley et al. (2009). The higher average EMGdi %max found in the present COPD patients was likely caused by the difference in disease severity: FEV1 was lower than the value in the above-mentioned patients, reflecting more severe airflow obstruction. The long-term clinical effect of NPPV for patients with respiratory failure who experience acute exacerbations of COPD has been acknowledged (VENTILA Group, 2008). NPPV can also reduce NRD, (Thys et al., 2002) but its function is ambiguous for stable patients. (Köhnlein et al., 2014; Casanova et al., 2000) High-intensity NPPV has been applied to hypercapnic COPD patients, (Dreher et al., 2010) but NRD was not detected in these studies. The highest IPAP was 20 cmH2O in the present study, and most patients could tolerate this level. The results showed that as the NPPV increased, the NRD of COPD patients decreased, indicating unload of the respiratory muscle and relief of dyspnea. Chinese scholars (Zhang et al., 2014) titrated the NPPV at the most comfortable pressure for every patient previously, and found that NRD decreased larger than that in the present study. This may because (1) the participants in the present study were stable COPD patients rather than AECOPD, the NRD of whom is easy to improved;(2) Moreover, none of COPD had previous experience with NPPV, so complete relaxation of the patients and clear instructions by the doctors were very important. However, no worldwide standard has yet been established regarding how much the NRD should be reduced during NPPV.

Although the NRD of COPD patients decreased during NPPV, rebound of NRD occurred at 20 cmH_2O in some patients. This means that higher NPPV is not necessarily better, especially in severe COPD patients. Because it may induce intrinsic positive end-expiratory pressure and an increasing in respiratory work, which resulting in “escape phenomenon” (Passam et al., 2003). This suggests that in clinical practice, NPPV parameters can be adjusted by detecting NRD, so that patients can experience increased ventilation and avoiding burdens on the respiratory muscles.

Naturally, our study had some limitations. The sample size was relatively small, and all subjects were male. A larger and more diverse sample would provide more convincing results. Additionally, we examined only short-term respiratory physiologic changes and did not apply them to long-term clinical effect; thus, further long-term studies are needed.

5. Conclusion

A strong relationship is present between NRD measured by EMGdi% max and NRD measured by sEMG%max, especially during an increase in NRD, as occurs in patients with COPD patients. These data support the use of sEMG%max, as a non-invasive method to quantify NRD. This technique can allow for real-time detection for adjustment of NPPV parameters in COPD patients, increasing the benefit of NPPV.

Contributor ship statement

L-L wrote the manuscript. G-LL,W-WL modified the manuscript content and submitted the paper. L-L,G-LL and W-WL were involved with Data management. C-RC reviewed and modified the manuscript content.

Competing interests

None declared.

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Data sharing statement

There are no additional unpublished data from the study.

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