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Respiratory motor output during an inspiratory capacity maneuver is preserved despite submaximal exercise

Dong Zhang[a], Haihong Gong[a], Gan Lu[b], Hongxi Guo[a], Ruifa Li[a], Nanshan Zhong[a], M.I. Polkey[c], Yuanming Luo[a,*]

[a] Guangzhou Medical College, State Key Laboratory of Respiratory Disease, Guangzhou 510120, China
[b] Jiangsu Province Official Hospital, Nanjing 210024, China
[c] NIHR Respiratory Biomedical Research Unit at the Royal Brompton and Harefield NHS Foundation Trust and Imperial College, Royal Brompton Hospital, London SW3 6NP, UK

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ABSTRACT

It is unknown whether respiratory motor output is constrained during exhaustive exercise in healthy adults. We hypothesised that neural inhibition did occur; to test this hypothesis we measured diaphragm EMG from a maximal inspiratory capacity maneuver (EMG_{di}-IC) at rest and during exercise. EMG_{di}-IC was measured before and after the amplitude of the diaphragm EMG entered a plateau phase in eleven healthy adults undertaking exercise at 60% and 80% of maximal workload achieved from incremental exercise. The mean EMG_{di}-IC at rest was 65 ± 16% of the maximum that could be obtained from a battery of inspiratory tasks. Before and after the plateau phase of diaphragm EMG, EMG_{di}-IC was 68 ± 13% and 72 ± 12% ($p > 0.05$) during 60% of the maximum workload, and was 70 ± 13% and 78 ± 13% ($p > 0.05$) during 80% of the maximum workload achieved on an incremental test. A further sub-study in which 5 participants exercised at 90% of the maximum workload also showed that EMG_{di}-IC was not diminished during exercise. Our data show that exercise condition does not reduce the magnitude of EMG_{di}-IC. This argues against neural inhibition as feature of submaximal exercise in healthy adults.

1. Introduction

When patients with chronic obstructive pulmonary disease (COPD) perform exercise both we (Qin et al., 2010; Luo et al., 2011) and others (Sinderby et al., 2001) have observed that while respiratory motor output ('neural drive') rises during exercise, however maximal respiratory motor output is seldom reached with typical values at end-exercise being approximately 80% of maximal. Moreover when normal subjects performed constant exercise at 80% of maximal load, there was a plateau in diaphragm electromyography (EMG) (Qin et al., 2010; Luo et al., 2011), which leads to a hypothesis that there is neural inhibition during exercise. Indeed, McClaran et al. (1999) also highlighted that there was possibility of neural inhibition in normal subjects during heavy exercise based on ventilation and esophageal pressure response to chemical stimuli induced by added dead space although respiratory rate increased continually. Taken together these data raise the hypothesis that there is neural inhibition during exercise.

Inferring the presence of inhibition from something that is absent is intuitively unsatisfactory. If respiratory motor output is assessed during exercise then a submaximal output may simply be an appropriate response to the demand placed upon it, which we term submaximal activation rather than central inhibition. In animal studies (Ferguson, 1995; Sassoon et al., 1996) the concept of neural inhibition has also arisen because submaximal neural drive is observed even during experimental models in which the inspiratory load is so great that death ensues. This approach is of course not feasible or ethical in man, and indeed may not be very insightful outside the context of intensive care.

Conceptually one way of determining whether neural inhibition (rather than submaximal activation) is present would be to ask participants during exercise to perform a maximal inspiratory capacity (IC) maneuver. The reasoning underlying this conjecture is that true neural inhibition should be associated with a reduced EMG_{di} elicited by such a maneuver whether the inhibition were occurring at a cortical or brainstem level. We therefore used this method to seek the presence of neural inhibition during exercise at 80% of maximal work rate. In addition, as a control condition, we added a constant rate exercise run at 60% of maximal, reasoning that, since 80% of maximal was achievable, then neural inhibition should not be present during the lower intensity run. We further reasoned that if this novel approach was feasible in normal subjects

* Corresponding author at: State Key Laboratory of Respiratory Disease, 151 Yanjiang Road, Guangzhou 510120, China. Tel.: +86 20 3429 4087; fax: +86 20 3428 4122.
E-mail address: yuanmingluo9431@yahoo.co.uk (Y. Luo).

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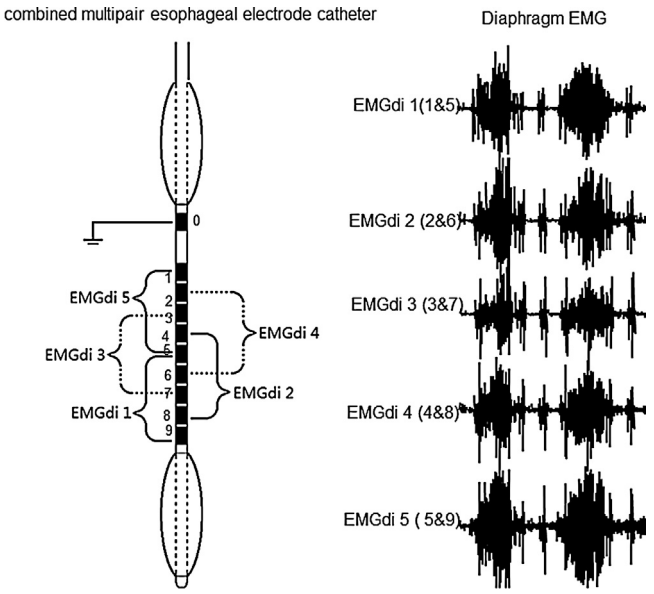

Fig. 1. A combined multipair esophageal electrode catheter with gastric and esophageal balloons. Electrode 5 was positioned close to the diaphragm based on the amplitude of signals during tidal breathing, which was characterized by a large EMG signal from pairs 1 and 5 and the smallest EMG signal from pair 3.

that a subsequent study could be undertaken in patients with COPD.

2. Methods

11 healthy young subjects (6 men, 5 women, age 27.9 ± 3.1 years) who did not undertake regular physical training were studied. All subjects were the laboratory staff and were free from the nervous system diseases and cardiopulmonary diseases. Our local ethics committee approved the study, and all subjects gave their informed consent.

EMG_{di} was recorded from a combined multipair esophageal electrode catheter with gastric and esophageal balloons mounted on it. The diameter of the catheter was 2.8 mm. The most proximal coil (coil 0) was connected to the ground and was 2 cm away from coil 1. Coils 1–9 were designed for recording and were 1 cm in length with a gap of 1 mm between adjacent coils. Five consecutive recording pairs were formed with an interelectrode distance of 3.4 cm within a pair. Two balloons, 7 cm in length and 6 mm in diameter, were mounted on the catheter 1 cm proximal to coil 0 and 2 cm distal to coil 9, and the distance between the two balloons was 15.8 cm. The proximal balloon was for measurement of esophageal pressure and the distal for measurement of gastric pressure. The combined electrode-balloon catheter was passed pernasally. Electrode 5 was positioned close to the diaphragm based on the amplitude of signals during tidal breathing, which was characterized by a large EMG signal from pairs 1 and 5 (Fig. 1) and the smallest EMG signal from pair 3, as described previously (Luo and Moxham, 2005; Luo et al., 2011). The EMG signals were filtered bandpass between 20 and 1000 Hz and amplified (Bioamplifier Model RA-8, Yinghui Guangzhou, China).

2.1. Esophageal pressure and gastric pressures

By design, when electrode coil 5 was located in the correct position, the two balloons were positioned at the esophagus and stomach, respectively. Satisfactory placement of the balloons was further confirmed by a sniff maneuver and cough maneuver. When the catheter was satisfactorily placed, the balloon-electrode catheter was fixed at the nose securely. The esophageal balloon was filled with 0.5 ml air and the gastric balloon filled with 1.0 ml air. Both esophageal pressure (Pes) and gastric pressure (Pga) were measured with a pressure transducer (DP15, Validyne Corp., Northrige, CA, USA). Respiratory pressures and the diaphragmatic EMG were record continuously using the Powerlab recording system (ADInstruments, Castle Hill, Australia) during both incremental and constant rate exercises. SpO_2 was measured by pulse oximetry (Capocheck Plus 9004-001, BCI, Waukesha, Wisc., USA). Metabolic data were recorded with an automated exercise testing system (AD instruments, Castle Hill, Australia).

2.2. Study protocol

Each subject visited the laboratory three times within a week on separate days. The purpose of the first visit was to perform lung function tests, to allow subjects to practice the inspiratory maneuvers described below, and perform incremental exercise to determine maximal power (in watts) achieved during exhaustive incremental cycle exercise. The inspiratory maneuvers used were maximum sniff efforts from functional residual capacity (FRC), maximal iso-volumetric contraction at FRC, and maximal inspiration from FRC to total lung capacity (TLC), i.e. an inspiratory capacity maneuver. Each maneuver was repeated until three reproducible values were obtained, and there was a minimum 1 min period of rest between maneuvers. The incremental exercise consisted of 3 min standing still, followed by an increase in the workload every 2 min by increasing the load by 20 W until intolerable dyspnea or leg fatigue occurred or exercise last for 60 min. Diaphragm EMG, esophageal pressure, gastric pressure, ventilation, oxygen and carbon dioxide consumption were measured during exercise. Maximal inspiratory capacity maneuvers were performed every 2 min during exercise to obtain maximal EMG during exercise and dyspnea and leg fatigue scores were asked every 1 min.

On the other two visits, a constant work rate load exercise, either 80% or 60% of the maximum load exercise test, was performed in random order. As before (Qin et al., 2010; Luo et al., 2011), maximal inspiratory capacity maneuvers were performed at rest and every minute during exercise to obtain maximal EMG during exercise, in exactly the same fashion as we had done during the incremental exercise.

2.2.1. Additional study

To determine whether exercise at high load could induce neural inhibition, we studied additional five young healthy subjects (three men and two women, aged 28 ± 2 years, BMI 19.4 ± 1.5 m^2/kg) by recording diaphragm EMG using maximal inspiratory capacity maneuvers during a constant cycle exercise at 90% maximal load derived from an incremental exercise test as described above. The maximal inspiratory capacity maneuvers were also performed at rest and every minute during exercise.

2.3. Data analysis and presentation

The diaphragmatic EMG reported was that from the electrode pair with the largest EMG amplitude for each breath. The largest value from different maneuvers including inspiratory capacity during exercise was taken as the maximal EMG and was used to standardize data during exercise. Data were presented as mean ± SD. Single factor analysis of variance with a Tukey's post hoc test was used to determine differences. Significance was accepted at $p < 0.05$.

3. Results

Demographic data from the subjects is shown in Table 1. The mean maximal root mean square of the diaphragmatic EMG at rest