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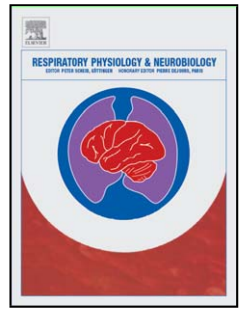

Title: Increases in inspiratory neural drive in response to rapid oscillating airflow braking forces (vibration)

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Increases in inspiratory neural drive in response to rapid oscillating airflow braking forces (vibration).

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Abstract

Objective: To investigate whether 10 breaths against a vibration stimulus elicits increments of spontaneous and maximal inspiratory mouth pressure (maxMP) and tidal mean inspiratory flow (iV_T/T_I) upon stimulus removal.
Methods: Twelve healthy subjects (8 female, 4 male; 22-50 years old), recruited from the University student body, completed 3 maximal inspirations before (PRE) and after (POST) 10 inspirations against resistive loading with a vibration-type stimulus (VIB; youbreathe, Exoscience Ltd., London, UK), pressure-matched resistive loading (RES) or resting breathing (CON; no load). The trials were presented in a random order. maxMP and involuntary tidal breathing were compared PRE and POST conditioning.
Results: Inspiratory neural drive increased only after VIB as evidenced by increased tidal and maxMP and mean inspiratory flow (iV_T/T_I; $p<0.05$). There was no effect of either resistance or control breathing on maximal maxMP or tidal responses.
Conclusions: 10 conditioning breaths of VIB lead to increased maximal inspiratory mouth pressure and spontaneous mouth pressure and mean inspiratory flow possibly through a common mechanism of increased descending respiratory drive.
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1. Introduction

Skeletal muscle activity and strength performance is enhanced during (Issurin & Tenenbaum, 1999; Bosco et al., 1999) and post acute vibration stimulation (Mileva et al., 2006). Vibration stimulation has also been applied to the respiratory musculature with demonstrable increases in respiratory motoneurone activity in rabbits during the stimulation (Jammes et al., 2000), reduced breathlessness at rest in healthy humans in the absence of any changes in motor output (Edo et al., 1998), and reduced breathlessness during exercise in chronic obstructive pulmonary disease patients (Fujie et al., 2002).

We therefore investigated whether a rapid oscillating braking force (vibration) applied through air as it passes into the airways, which uses similar stimulus principles to that of Mileva et al. (2006), elicits increments of inspiratory neural drive evidenced as increases in maximal inspiratory mouth pressure (InspMP) and tidal mouth pressure and mean inspiratory flow (iV_T/T_I) upon stimulus removal.

2. Materials and Methods

2.1. Subjects

Twelve healthy subjects (8 female, 4 male; 22-50 years old) with no history of respiratory disease consented to participate in the study, which had been approved by the local University Ethics Committee.

2.2. Methods

On a familiarisation visit, each subject performed 10 forced maximal dynamic inspirations (maxMP) after maximally emptying the lungs; subsequent maximal inspirations were initiated by the subject once expired carbon dioxide fractions had returned to pre-test levels. The maximal inspirations used here are not the isometric maximal inspirations seen in many studies (P_{Imax}). The subject sat in a comfortable chair and breathed through a respiratory mouth piece using a nose clip. The pressure developed by the subject at the mouth was measured with a bespoke pressure transducer (Range ± 50 cmH_20) and expired end-tidal carbon dioxide percentage ($P_{ET}CO_2$) was measured with a fast response Capnograph (Morgan Medical, Kent, UK). Induced airflow was measured with a heated pneumotachograph from which breath-by-breath parameters were calculated.

Each subject randomly completed all interventions at the same time of day, with at least 1 day between testing sessions. On each day of the study, the subject breathed through the respiratory apparatus for 10 breaths to familiarise with the equipment. They then performed 3 maximal inspirations (PRE) followed by 10 inspirations against either: resistive loading with a vibration-type stimulus (VIB; 21 $cmH_20\cdot(l\cdot s^{-1})^{-1}$youbreathe, Exoscience Ltd., London, UK), resistive loading (RES; 19 $cmH_20\cdot(l\cdot s^{-1})^{-1}$) or resting breathing (CON; no load). Finally, 3 forced inspirations were performed (POST), initiated within 5 breaths of the end of stimulation. Mouth pressure, airflow and end-tidal CO_2 signals were sampled at 200Hz with a computerised data acquisition system (CED 1401, Cambridge, UK). The youbreathe respiratory vibration training device embodied a rotating valve that completely resisted airflow for brief periods and operated at a frequency of 25 Hz with peak-to-peak amplitude of 1 cmH_2O. The pressure response to the RES stimulus was matched to the VIB stimulus in a pilot experiment using fixed bore tubing (Length 100mm; internal diameter 5mm; RES - 19 $cmH_20\cdot(l\cdot s^{-1})^{-1}$ s vs VIB - 21 $cmH_20\cdot(l\cdot s^{-1})^{-1}$).

2.3. Data Analysis

Voluntary maximal PRE and POST mouth pressure data were averaged across 12 subjects and analysed with a 2-factor repeated measures ANOVA. Statistically significant results were subjected to further analysis with Holm-Sidak corrected paired *t*-tests. Involuntary (spontaneous tidal) responses for mouth pressure and mean inspiratory flow (iV_T/T_I) were compared PRE (average of 5 resting breaths) to POST (first 2 breaths immediately post stimulation) with a 2-factor repeated measures ANOVA, again Holm-Sidak corrected paired *t*-tests were used to analyse significant findings. The 1^{st} and 10^{th} inspiration of the conditioning breaths were analysed with a 2-factor repeated measures ANOVA for changes in mouth pressure and $P_{ET}CO_2$. All data are presented as Mean±SEM.

3. Results

MaxMP ($p<0.05$) was significantly increased after 10 breaths of vibrated resistance (POST VIB) when compared to PRE VIB (-11.43±3.95* vs. -10.99±3.60 vs. -10.64±3.67; cmH_2O; VIB vs. CON vs. RES; $p<0.05$). There was no effect of either resistance or control breathing on maxMP (Figure 1). Involuntary mouth pressure responses were consistent with maximal voluntary responses showing a significant increase in POST breath 1 after vibration but not after the RES or CON conditions. POST breath 1 was also significantly greater after VIB than after RES (-0.92±0.41* vs. -0.55±0.16 vs. -0.52±0.15; cmH2O; VIB vs. CON vs. RES; * $p<0.05$). Mean inspiratory flow (iV_T/T_I) showed the same pattern of change as the mouth pressure responses, with only VIB having any effect on the involuntary tidal breathing responses (0.51±0.15* vs. 0.36±0.11 vs. 0.41±0.20 L. s-1; POST VIB vs. POST CON vs. POST RES; POST-breath 1; * $p<0.05$). ***Figure here***

Analysis of the conditioning breaths demonstrates that there was a significant difference between the conditions ($p<0.05$, main effect of condition). Post-hoc analysis revealed that mouth pressure was significantly greater during VIB and RES conditions than during CON condition (3.10±0.58 vs. 2.20±0.38 vs. 0.63±0.10; VIB vs. RES vs. CON; 1^{st} Breath). There was a tendency for mouth pressure to increase over time in the VIB trial (30±23%; 10^{th} breath – 1^{st} breath) but there was no significant time/condition interaction.

4. Discussion

10 breaths of vibrated breathing VIB lead to increased neural inspiratory drive suggesting that applying a rapid oscillating air braking force (vibration stimulus) increases the voluntary force generating capacity of the inspiratory muscles, in a similar manner observed when vibration is applied to other skeletal muscles (Mileva et al., 2006).

Upon stimulus removal involuntary (tidal) breathing responses also demonstrated an increase after VIB suggesting that the augmented mouth pressure seen during maximal voluntary and tidal inspirations share a common neural mechanism. Thus, the logical locus of action would be below the point of integration of the voluntary and involuntary pathways such as the respiratory motonuerons (Butler, 2007).

The mechanisms underlying the changes in mouth pressure require further study, however shifts in neuromuscular recruitment via increases in stretch reflex sensitivity may play a role (Cardinale, 2003). This would enhance recruitment of higher-threshold motor units and the activation of previously inactive motor units. Chest wall vibration is currently thought to only affect intercostal inspiratory activity and not diaphragmatic inspiratory activity due to the low concentration of muscle spindles in the diaphragm (Leduc & De Troyer, 2002). We could therefore predict that the results seen here are caused by augmentation of external intercostal activity with no effect on diaphragmatic function. Confirmation of the mechanisms involved would require the acquisition of respiratory muscle EMG, transcranial and peripheral nerve magnetic stimulation.

In summary, vibration via a rapid oscillating air braking force leads to an increased voluntary maxMP and involuntary spontaneous breathing suggesting there is an increase in neural inspiratory drive possibly via upregulation of the respiratory motoneurones.

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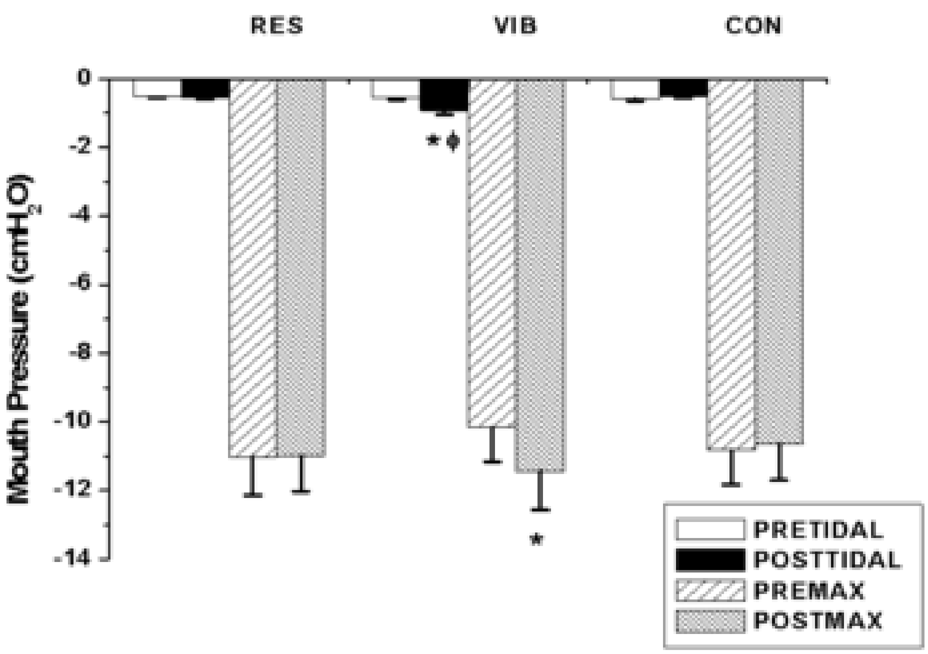
Figure 1. Tidal and maximal inspiratory mouth pressure before and after 10 breaths of RES, VIB or CON. * indicates significant difference from PRE; ϕ indicates significant difference from POST RES (p<0.05).


RES
VIB
CON
0
-2
-4
-6
-8
-10
-12
-14
Mouth Pressure (cmH2O)
*ϕ
*
PRETIDAL
POSTTIDAL
PREMAX
POSTMAX