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Effects of two types of hyperventilation on standing balance

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The present study sought to establish links between hyperventilation and postural stability. Eight university students were asked to stand upright under two hyperventilation conditions applied randomly: (1) a metabolic hyperventilation (induced by CO₂ rebreathing), and (2) voluntary hyperventilation (set at 25 cycles per minute). Recordings were obtained with eyes open, standing on a force platform over 20-second periods. The ventilatory variations and displacements of the center of pressure in both frontal and sagittal planes were monitored in the time and spectral domains. Postural changes related to respiratory variations were quantified by coherence analysis. Myoelectric activities of the soleus and tibialis anterior muscles were recorded using surface electromyography. Results showed that characteristics of postural perturbation and mechanisms of adjustments markedly differ as the hyperventilation was metabolic or voluntary. These results suggest there is a link between control of respiration and control of posture, although their mechanisms are differently organised.

Keywords: Hyperventilation, Posture, Electromyography

INTRODUCTION

Tidal ventilation and hyperventilation are known to perturb standing balance (Hodges et al., 2002). However, hyperventilation can be performed either voluntarily or by the mean of a metabolic change like CO₂ rebreathing (Mercier et al., 1992; Sakellari and Bronstein, 1997). We hypothesized that ventilation itself was not the only factor influencing postural strategies. With regards to CO₂ level, voluntary hyperventilation leads to hypocapnia whereas CO₂ rebreathing goes with hypercapnia. Despite the fact that standing balance is perturbed during both, ventilatory control is different and motor control may also be different. The aim of the present study sought to examine the effects of two types of ventilatory perturbation on the maintenance of standing posture by analysing postural sway, characterising postural disturbance and leg muscle activities, reflecting postural control.

METHODS

Eight voluntary healthy students (mean age: 26.4 ± 3 yrs; mean weight: 74.1 ± 8.2 kg; mean height: 176.4 ± 9.8 cm) were examined. Postural tests correspond to the maintenance of a bipedal stance for 20-s with eyes open. The ground reaction forces and their moments in the three planes - used to determine the coordinates of the centre of pressure (COP) in both frontal and sagittal planes - were recorded using a piezoelectric force plate (Kistler, Switzerland). COP basal measures of postural sway (path, area and mean amplitude) were chosen as indexes of postural stability. A mono-axial accelerometer placed on the low part of the sternum estimated the subject's breathing frequency (BF). Surface electromyogram (EMG) from the *Soleus* (SO) and *Tibialis anterior* (TA) muscles were recorded on the dominant leg. Muscle activation levels were quantified from the raw signals by computing root mean square value (RMS).

After a first postural recording (reference test), two types of ventilatory perturbation were randomly applied: a metabolic hyperventilation (HM) induced by 5-min of CO₂ rebreathing using Douglas bag system with supplemental oxygen supply to avoid hypoxemia, and a voluntary hyperventilation (HV) of 3-min imposed by a metronome set at 25-min⁻¹. The CO₂ fraction of exhaled gas (F_{Eco2}) was determined with a capnograph (Radiometer, France) connected to mouth piece. The Douglas bag was fixed to a device allowing free breathing movements of the subject without disturbing the standing balance. All the parameters were monitored in time and spectral domains. Coherence analysis (coh) was purposely used for estimating correlation in frequency domain between postural disturbances and ventilation.

Mean values of each parameter obtained before and during each ventilatory condition were compared using a paired Student's *t*-test.

RESULTS

The mean values of $F_{E_{CO_2}}$ measured were significantly higher ($p < 0.001$) during HM (7.6 ± 0.6 %) than during HV (2.8 ± 0.4 %). The off-line processing of the accelerometric signal in the sagittal plane revealed a significant ($p < 0.05$) increase in the mean amplitude, suggesting that the ventilatory adaptation during HM is better related to an increase in the tidal volume than a change in BF. In contrast, HV prompted a significant increase ($p < 0.05$) in BF and postural sway parameters - principally along the antero-posterior axis - indicating a reduction in postural stability due to rib cage motions. Also, HV led to a significant increase ($p < 0.01$) in the fluctuations of the vertical (ΔF_z) and antero-posterior (ΔF_x) ground reaction forces. The results of coherence analysis showed a close relationship between breathing and ΔF_z ($coh = 0.80 \pm 0.14$), suggesting a ventilatory origin of these postural disturbances. No significant change in SO and TA raw EMG amplitudes was found during HV. However, normalizing RMS values with respect to ΔF_z and ΔF_x allowed comparisons between ankle muscle electromyographic activities without the influence of ventilation range obtained either during HV or HM. There was a significant decrease in both RMS SO/ ΔF_z and RMS SO/ ΔF_x ratios ($p < 0.05$ and $p < 0.01$ respectively). These results suggest that leg muscle strategies were different and directly linked to ventilatory control modes. Moreover, it is likely that postural control is more efficient during metabolic hyperventilation, HV inducing higher unpredictable postural instability than HM.

CONCLUSION

Our results provide evidence that mechanisms of postural adjustments markedly differ as the hyperventilation was metabolic or voluntary. Further investigations are needed to better understand how those mechanisms imply some links between ventilatory drive and postural control.

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