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ORIGINAL ARTICLE

Postural control and ventilatory drive during voluntary hyperventilation and carbon dioxide rebreathing

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Abstract 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 5 min of hypercapnic–hyperoxic rebreathing (CO_2–R); and, (2) a voluntary hyperventilation (VH) of 3 min imposed by a metronome set at 25 cycles per min. Recordings were obtained with eyes open, with the subjects standing on a force plate over 20-s periods. Ventilatory response, displacements in the centre of pressure in both the frontal and sagittal planes and fluctuations in the three planes of the ground reaction force were monitored in the time and frequency domains. Postural changes related to respiratory variations were quantified by coherence analysis. Myoelectric activities of the calf muscles were recorded using surface electromyography. Force plate measurements revealed a reduction in postural stability during both CO_2–R and VH conditions, mainly in the sagittal plane. Coherence analysis provided evidence of a ventilatory origin in the vertical ground reaction force fluctuations during VH. Electromyographic analyses showed different leg muscles strategies, assuming the existence of links between the control of respiration and the control of posture. Our results suggest that the greater disturbing effects caused by voluntary hyperventilation on body balance are more compensated when respiration is under automatic control. These findings may have implications for understanding the organisation of postural and respiratory activities and suggest that stability of the body may be compromised in situations in which respiratory demand increases and requires voluntary control.

Keywords Electromyography · Postural control · Rebreathing · Hyperventilation

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Introduction

The ability to maintain balance plays a fundamental role in motor programming (Bouisset and Do 2008). In many sport activities, this ability is considered a key factor to perform complex motor skills. In order to stabilise posture and maintain balance, the central nervous system involves anticipatory postural adjustments prior to initiating intentional movement (Bouisset and Zattara 1981; Oddsson and Thorstensson 1986) or postural reactions to compensate perturbations due to external forces (Nashner 1983; Horak and Nashner 1986).

In addition to these perturbations, internal forces related to cardiorespiratory activities may also disturb postural stability (Stürm et al. 1980). In an upright stance at rest, it is well known that quiet breathing (QB) perturbs body balance in the sagittal and frontal planes, and that the

effects of compensatory motions of the trunk and lower limbs are more pronounced in the sagittal plane (Gurfinkel et al. 1971; Bouisset and Duchêne 1994; Hodges et al. 2002; Caron et al. 2004). Moreover, Bouisset and Duchêne (1994) have reported that posture was more perturbed by respiration during QB in a seated posture than in a standing one, showing the dominating role of lower limb muscle compensatory activities during a standing stance. Furthermore, a series of studies (Hamaoui et al. 2002; Grimstone and Hodges 2003; Brumagne et al. 2004) described impairment in postural control in low back pain subjects, suggesting that the trunk plays also an important role regulating standing posture.

During voluntary hyperventilation (VH), including deep breathing with increased tidal volume for each breath (Hunter and Kearney 1981; Jeong 1991; Sakellari et al. 1997; Kantor et al. 2001), postural perturbations and compensatory counteractions may increase markedly. Otherwise, several studies have demonstrated that chest wall expansion increases in parallel with an increase in minute ventilation during VH and rebreathing of a hypercapnic–hyperoxic gas mixture (CO_2–R), but with a different pattern in thoracoabdominal wall muscle activities (Sackner et al. 1984; Romagnoli et al. 2004). These results suggest that different hyperventilation mechanisms could impair standing posture in different ways and, as a consequence, could induce different postural control responses.

Until now, the effect of metabolic hyperventilation has been examined mainly after an exercise test on a treadmill (Nardone et al. 1998; Bove et al. 2007) or a cycle ergometer (Nardone et al. 1997; Gauchard et al. 2002; Vuillerme and Hintzy 2007; David et al. 2010). In their study, David et al. (2010) reported an increase in leg muscle electromyographic activities during hyperventilation following the end of exercise, which was related enhancement of musculo-articular stiffness of the ankle joint to compensate for the respiratory disturbance. However, running or cycling activities can lead to leg muscle fatigue, which is known to specifically interfere with postural control (Lepers et al. 1997; Caron 2003; Corbeil et al. 2003). Hence, it is difficult to distinguish between what is due to compensatory fatigue mechanisms, and what is due to a specific postural correction pattern.

CO_2–R is an interesting metabolic hyperventilation model, avoiding any lower limb muscle fatigue phenomenon. During CO_2–R, increasing hypercapnia leads to an automatic increase in minute ventilation (Cherniack and Widdicombe 1986; Sebert et al. 1990; Jensen et al. 2005) which parallels modifications in thoracic and abdominal wall movement amplitude (Aliverti et al. 1997; Filippelli et al. 2002; Romagnoli et al. 2004). Therefore, this type of metabolic hyperventilation would also provide a way to examine how the compensatory postural regulation would be stressed during automatically raised ventilation in response to hypercapnia. Moreover, we could also examine how these two types of ventilatory perturbations and their respective postural compensatory strategies would differ, and to better understand how the central nervous system coordinates postural and respiratory movements.

Methods

Subjects

This non-invasive study involved two experimental sessions, each taking place on separate days: (1) a validation of the hyperventilation model; and, (2) an assessment of the effects of hyperventilation on standing balance. Eight healthy university students, two women and six men, participated in both experiments. Their average age, body mass and height were 26.4 ± 3 years, 74.1 ± 8.2 kg, and 176.4 ± 9.8 m, respectively. All subjects had been fully informed of the experiment's aims and procedures in accordance with the guidelines for the use of human subjects as stipulated by the 1964 Declaration of Helsinki, and gave their written consent. None of them presented with any known neurological or respiratory diseases or injuries that might have impaired balance or respiration.

Force plate data

The ground reaction forces (GRF) and their moments in the three orthogonal directions used to determine the coordinates of the centre of pressure (CoP) were recorded using a rectangular (60 × 40 cm), piezoelectric force plate (Kistler, 9281 B11, Switzerland) linked to a charge amplifier (Kistler, 9851, Switzerland). The force plate measured three components of GRF at a precision of 2,000 N/V for F_z (vertical GRF) and 200 N/V for F_x (antero-posterior GRF) and F_y (medio-lateral GRF). Horizontal displacements of CoP in the antero-posterior ($CoP_{A\text{-}P}$) and medio-lateral ($CoP_{M\text{-}L}$) directions were recorded with a sensitivity of 50 mm/V.

Ventilatory measurements

The CO_2 fraction of exhaled gas (F_{Eco_2}) was determined with a sampling capnograph (Anesthetic Gas Monitor type 1304, Brüel & Kjaer, Denmark), fixed at the mouthpiece, and provided a value every 30 s. A mechanical three-way respiratory valve (Hans Rudolf, Inc, MO, USA) allowed the subject to breathe either normally connected to open air, or into a Douglas bag (50-L, CH-Lyon, France). This valve allowed for rapid switching from QB to CO_2–R. The Douglas bag was also connected to a medical oxygen

supply to compensate for the subject's oxygen uptake and to prevent hypoxia within the bag with an initial volume set at 10 L. The whole apparatus was fixed to a device that allowed free breathing movements of the subject without disturbing the standing balance. A mono-axial piezoelectric accelerometer (DJB Instruments, UK), placed on the lower part of the sternum, was used to estimate the subject's rib cage movement and breathing frequency from the antero-posterior variations in the rib cage.

Electromyography

Electromyographic (EMG), bipolar recordings of the soleus (SO) and tibialis anterior (TA) muscle activities were made on the dominant leg using Ag/AgCl surface electrodes 8 mm in diameter (Beckman). Before their placement, the skin at the electrode sites was prepared to achieve an interelectrode impedance of less than 5 kΩ. The electrodes (centre-to-centre spacing, 20 mm) were, respectively, placed about 2 cm below the insertion of the gastrocnemius on the Achilles tendon for the SO, and on the proximal one-third for the TA, parallel to the muscle fibres. The reference electrode was placed on the medial surface of the tibia. The EMG signals were differentially amplified (Gould 6600, Gould Electronics, Germany) with a gain set between 1,000 and 5,000, and filtered using a bandwidth of 10–500 Hz. Data from the force plate, the accelerometer and EMG were simultaneously sampled at 1,000 Hz using a 12-bit analogue-to-digital converter (National Instrument Daq Card-AI-16E-4). The data were collected on a laptop computer interfaced with Matlab data acquisition toolbox (The Mathworks, Inc, Natick MA, USA) and then stored for off-line processing.

Procedure

Subjects had to perform three postural tests, each corresponding to the maintenance of a bipedal stance with eyes open. The recordings lasted 20-s. After the first postural recording (reference test), two types of ventilatory perturbations were applied: a metabolic hyperventilation induced by 5 min of CO_2 rebreathing, and a voluntary hyperventilation of 3 min imposed by a metronome set at 25 min^{-1}. The order of ventilatory perturbation testing was randomly assigned to each subject to avoid an order effect. To evaluate any possible bias due to perturbation influence of the respiratory apparatus, 3-min force plate recordings were made with and without apparatus, and did not show any significant difference. After each ventilatory perturbation, the subject rested for 5 min in a sitting position. All postural tests were performed in a quiet room without sound and light interference. Subjects were asked to stand barefoot on the force plate, relax with their arms hanging alongside the trunk and focus on a target placed 2 m in front of them. They were connected to a mouthpiece and respiratory apparatus. A nose-clip was worn to prevent nasal breathing. The foot position was standardized by placing foot prints on the force plate. The foot prints (adjusted to the subject's foot length) were drawn so that the respective longitudinal axes formed a 30° angle and intersected the force plate's anteroposterior axis, with the heels 2 cm apart.

Signal processing

Off-line processing of signals was performed using a custom processing routine written with Matlab 6.5 (The Mathworks, Inc.). Data from the accelerometer and the force plate were passed through a 4th-order low-pass Butterworth filter with a cut-off frequency of 15 Hz. The acceleration signal exhibited periodic patterns associated with durations of respiratory ribcage movement. Over the 20 s of each postural test recording, the breathing frequency was identified as the largest peak in the power spectrum of the accelerometric signal. Onset and end of tidal breathing inspiratory and expiratory phases were extracted from the accelerometer data using a peak detection function. The average magnitude of rib cage acceleration in the sagittal plane during each breathing condition was assessed by calculating the root mean square value (RMS). The mean amplitude (Δ) and the sway path (SP) of the CoP displacements in the two planes were calculated using the method described by Hufschmidt et al. (1980). The mean amplitude of F_x, F_y and F_z were also calculated. The frequency content of the GRF and the CoP displacements were computed by Fourier analysis to determine the mean power frequency (MPF). The power spectral densities of all the signals were calculated using the periodogram method. A coherence analysis was purposely used for estimating the correlation in the frequency domain between the chest wall accelerometric signal and F_z. The magnitude squared coherence $C_{xy}(f)$ of the signals x and y was estimated at each frequency f as: $C_{xy} = \frac{|P_{xy}(f)|^2}{P_{xx}(f)\cdot P_{yy}(f)}$, where $P_{xx}(f)$ and $P_{yy}(f)$ represent the power spectral densities of x and y, respectively, and $P_{xy}(f)$ is the cross power spectral density of x and y. The spectral densities can be obtained by averaging Welch's periodogram. Coherence provides a normalised spectral measure (ranging between 0 and 1) of the linear association between two signals as a function of frequency, where 0 indicates that there is no association, and 1 indicates a perfect association. During the postural tests, muscle activation levels were quantified from the raw EMG signals by computing RMS value over a 300 ms window with a sliding step of 20 ms. The RMS values were then averaged and expressed as percentages of each

subject's baseline values acquired during the reference test (i.e. during quiet breathing).

Statistical analysis

Data distributions were inspected statistically for normality using Kolmogorov–Smirnov's test. A logarithmic transformation was applied for non-normally distributed data. To validate the hyperventilation model, a two-way (order × breathing condition) repeated-measures analysis of variance (ANOVA) was used to analyse the overall changes in F_{Eco_2} and F_{Ico_2} measures. To assess the effects of hyperventilation on standing balance, a separate one-way repeated-measure ANOVA, with breathing conditions (i.e. QB, CO_2–R and VH) as a within-subject factor, was used to analyse overall changes in the outcome measurements. Tukey's HSD post hoc procedure was performed to assess pairwise comparisons of any significant main effects. In order to evaluate the influence of breathing conditions upon RMS from SO and TA, one-way analyses of covariance (ANCOVA) were performed taking F_x and F_z as covariates. The α level was set at 0.05 to identify the statistically significant difference between conditions. All data in the text are presented as mean + one standard deviation, unless indicated otherwise. All data analyses were conducted with Statview version 5.0 (SAS Institute, Inc, Cary, NC, USA).

Results

Breathing pattern

The first session was devoted to establishing the validity of the hyperventilation model. A significant main effect of breathing condition for F_{Eco_2} was observed ($F_{4,7} = 50.50$, $P < 0.001$). As illustrated in Fig. 1a, post hoc analyses revealed that the mean F_{Eco_2} values measured during the two tests were higher during CO_2–R than during VH ($P < 0.001$). Compared with reference mean values measured during QB, CO_2–R led to a significant increase ($P < 0.001$) in F_{Eco_2}, whereas VH led to a significant decrease ($P < 0.001$). No significant change in F_{Eco_2} was observed after each recovery period (R_1 and R_2). Similarly, a significant main effect of breathing condition for F_{Ico_2} was observed ($F_{4,7} = 168.21$, $P < 0.001$). As illustrated in Fig. 1b, post hoc analyses revealed that CO_2–R led to a significant increase in F_{Ico_2} ($P < 0.001$). The order in which the breathing conditions were applied was inverted (test 2 vs. test 1) to check that no influence existed between these ventilatory perturbations. The ANOVA did not show any statistically significant order effects for F_{Eco_2} ($F_{1,4} = 0.23$, $P > 0.05$), or F_{Ico_2} ($F_{1,4} = 1.99$, $P > 0.05$).

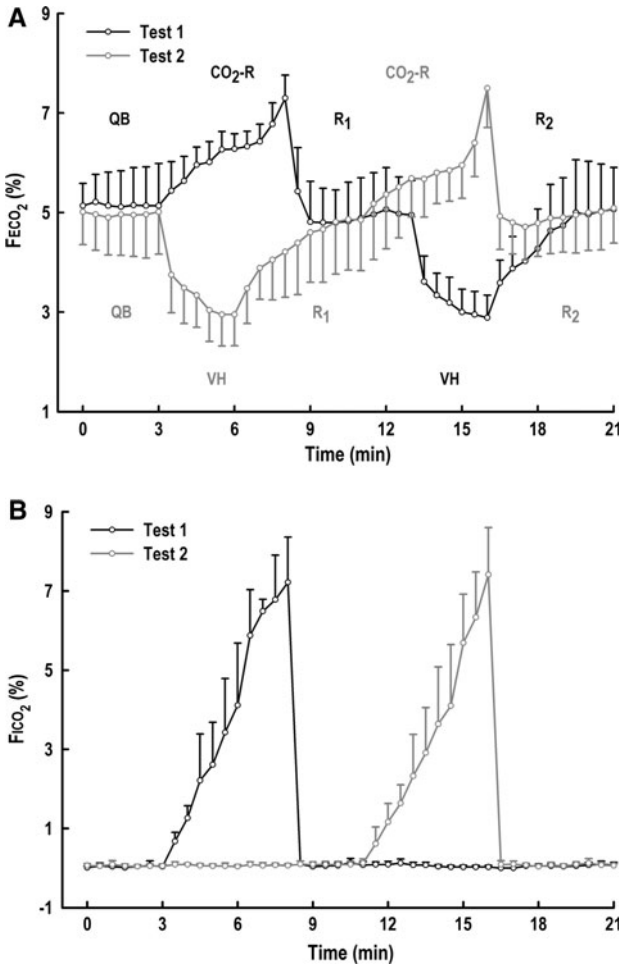

Fig. 1 Experimental conditions for measuring postural stability. Time courses of changes in **a** the carbon dioxide fraction of exhaled gas (F_{Eco_2}% ± one standard deviation), and **b** the carbon dioxide fraction of inhaled gas (F_{Ico_2}% + one standard deviation) over the different ventilatory conditions. Mean values are shown for all subjects ($n = 8$). Test 1: QB–CO_2–R–R_1–VH–R_2; Test 2: QB–VH–R_1–CO_2–R–R_2

The purpose of the second experimental session was to assess the effects of hyperventilation on standing balance. In this second session, mean F_{Eco_2} values measured during the last 30-s period of QB, CO_2–R and VH were 5.3 ± 0.7, 7.6 ± 0.6 and 2.8 ± 0.4%, respectively. The corresponding mean F_{Ico_2} value measured during CO_2–R was 7.3 ± 0.4%.

Significant main effects of breathing condition were observed for parameters obtained from the off-line processing of the accelerometric signal in the sagittal plane (Table 1). Post hoc analyses revealed a significant increase in the RMS acceleration values ($P < 0.05$) during CO_2–R compared with QB, but no significant change in BF was observed ($P > 0.05$). Inspiratory and expiratory times (T_I and T_E) and inspiratory time over the total breath duration (T_I/T_{TOT}) were not significantly modified during

Table 1 Parameters computed from the accelerometer signal collected in the antero-posterior direction during the last 20 s of each ventilatory condition (QB, CO_2–R, VH)

Parameters	QB	CO_2–R	VH	$F(2,7)$	P
BF (breaths/min)	19 ± 3	22 ± 5	26 ± 2	7.16	<0.01
T_E (ms)	2,355 ± 556	2,004 ± 1,097	1,107 ± 211	4.94	<0.05
T_I (ms)	1,234 ± 250	959 ± 350	1,172 ± 211	1.82	NS
T_{TOT} (ms)	3,589 ± 737	2,963 ± 1,058	2,280 ± 117	4.85	<0.05
T_I/T_{TOT}	0.35 ± 0.05	0.35 ± 0.17	0.51 ± 0.09	5.33	<0.05
RMS (m/s^2)	0.023 ± 0.012	0.071 ± 0.034	0.067 ± 0.036	7.02	<0.01

From top to bottom: breathing frequency (BF); expiratory phase duration (T_E); inspiratory phase duration (T_I); total breathing cycle duration (T_{TOT}); duty cycle (T_I/T_{TOT}) root mean square acceleration (RMS). Means, standard deviations and main effects of one-way Repeated Measure ANOVA are reported in the three breathing conditions, F values, degrees of freedom and significativity threshold are also shown

CO_2–R ($P > 0.05$). Compared to that observed for QB, the results showed a significant increase in the RMS acceleration values ($P < 0.05$) during VH, and a significant increase in BF ($P < 0.01$). In contrast with the CO_2–R condition, T_I/T_{TOT} was significantly prolonged during VH compared with QB ($P < 0.05$). No significant difference was observed between inspiratory and expiratory time ($P > 0.05$).

Force plate data

Significant main effects of breathing condition were observed for posturographic parameters (Table 2). Under the CO_2–R condition, Fig. 2a shows a significant increase in ΔF_x (31%, $P < 0.05$), and ΔF_z (27%, $P < 0.01$). The sway path (Fig. 2c) along the antero-posterior axis was also modified (47%, $P < 0.05$). In contrast, post hoc analyses indicated no change in the mean amplitude of the CoP displacements (Fig. 2d). Similarly, the frequency analysis of the GRF (Fig. 2b) and the CoP displacements (0.46 ± 0.07 vs. 0.50 ± 0.06 Hz for $CoP_{A\text{-}P}$ and 0.47 ± 0.05 vs. 0.44 ± 0.10 Hz for $CoP_{M\text{-}L}$, not shown) revealed no significant variation in signal power.

Under the VH condition, post hoc analyses showed a strong increase in ΔF_x (146%, $P < 0.01$), ΔF_y (139%, $P < 0.05$), and ΔF_z (167%, $P < 0.01$). These variations were significantly higher ($P < 0.05$) than those measured during CO_2–R (Fig. 2a). An ANOVA did not show any statistically significant difference ($F_{2,7} = 0.41$, $P > 0.05$) for the force fluctuation ratios ($\Delta F_z/\Delta F_x$) calculated during QB (1.7 ± 0.1), VH (1.9 ± 0.5) and CO_2–R (1.7 ± 0.5). As illustrated in Fig. 2c, a significant increase (90%, $P < 0.01$) was found in the sway path parameter, reflected exclusively along the antero-posterior axis. In contrast with the CO_2–R condition (Fig. 2d), VH caused a significant increase in the mean amplitude of the CoP displacements along the anterior (180%, $P < 0.01$) and medio-lateral (143%, $P < 0.05$) axes. Again, no change was observed in the signal power estimated from CoP displacements in both directions (0.46 ± 0.07 vs. 0.48 ± 0.07 Hz for $CoP_{A\text{-}P}$ and 0.47 ± 0.05 vs. 0.44 ± 0.05 Hz for $CoP_{M\text{-}L}$, not shown). However, VH had a significant effect on F_x and F_z power spectral densities, as shown by the mean MPF values, which were significantly lower ($P < 0.01$) than the reference values (Fig. 2b). Furthermore, the mean MPF values obtained for F_x and F_z were significantly lower than those measured during CO_2–R ($P < 0.01$).

The power spectra for all subjects and for each ventilatory condition are shown in Fig. 3a–c. The results indicated a close relationship between breathing and vertical GRF signals with peaks over a frequency range of 0.30–0.36 Hz for QB, 0.28–0.44 Hz for CO_2–R and 0.40–0.45 Hz for VH. A significant effect of breathing condition for coherence values was found for these peak

Table 2 Results from the one-way repeated measure ANOVA of force plate and EMG data

Parameters	Breathing conditions	
	$F(2,7)$	P
$\Delta CoP_{A\text{-}P}$	7.54	<0.01
$\Delta CoP_{M\text{-}L}$	5.12	<0.05
SPx	7.55	<0.01
SPy	2.16	NS
ΔF_x	19.02	<0.001
ΔF_y	5.30	<0.05
ΔF_z	12.23	<0.01
MPF $CoP_{A\text{-}P}$	0.68	NS
MPF $CoP_{M\text{-}L}$	0.46	NS
MPF F_x	8.76	<0.01
MPF F_y	1.60	NS
MPF F_z	37.22	<0.001

Main effects of the within-subjects factor breathing conditions (QB, CO_2–R, VH) on the posturographic parameters in the time domain ($\Delta CoP_{A\text{-}P}$, $\Delta CoP_{M\text{-}L}$, SPx, Spy, ΔF_x, ΔF_y, ΔF_z,) and in the frequency domain (MPF $CoP_{A\text{-}P}$, MPF $CoP_{M\text{-}L}$, MPF F_x, MPF F_y MPF F_z). NS: no significant

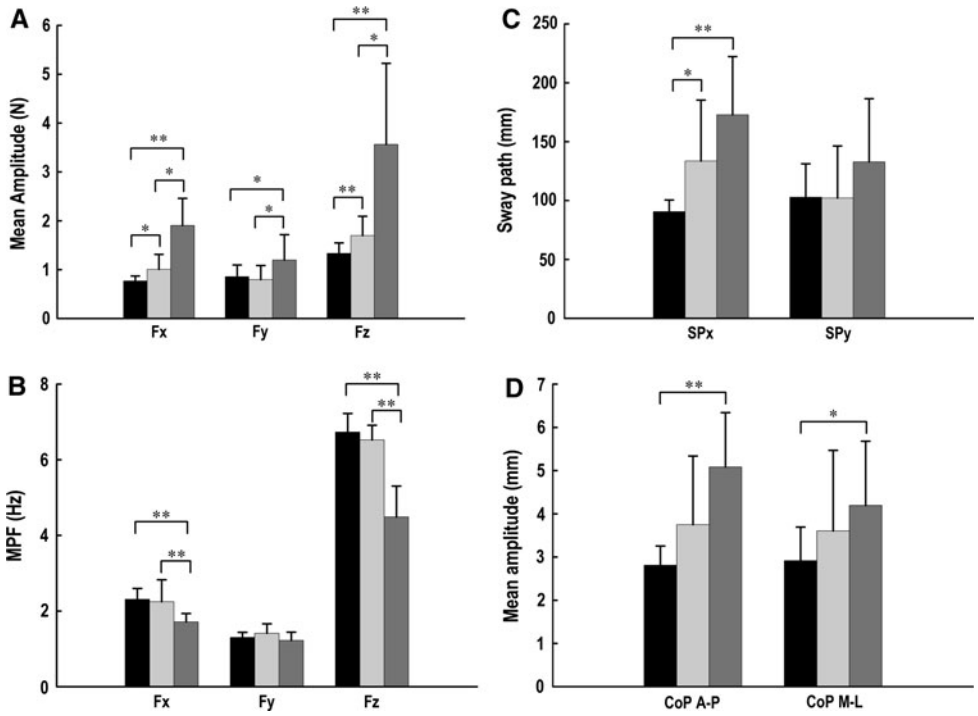

Fig. 2 Posturographic parameters measured in postural tests during the last 20 s of each ventilatory condition: quiet breathing (*black bars*), rebreathing (*light grey bars*) and voluntary hyperventilation (*dark grey bars*). **a** The mean amplitude of the ground reaction force in the sagittal (F_x), frontal (F_y) and transverse (F_z) planes. **b** The mean power frequency (MPF) of the ground reaction force in the (F_x), (F_y) and (F_z) planes. **c** The sway path along the antero-posterior (SPx) and medio-lateral (SPy) axes. **d** The mean amplitude of the CoP displacements in the antero-posterior ($CoP_{A\text{-}P}$) and medio-lateral ($CoP_{M\text{-}L}$) axes, mean values + one standard deviation are presented for all these parameters. * and ** indicate significant differences at $P < 0.05$ and $P < 0.01$, respectively

intervals ($F_{2,7} = 26.6$, $P < 0.001$). As illustrated in Fig. 3d, post hoc analyses showed a significant increase ($P < 0.01$) in the coherence value calculated during VH compared with QB and CO_2–R.

EMG recordings

No significant main effect for the RMS values normalised with respect to baseline RMS values acquired during QB was observed for SO ($F_{2,7} = 2.58$, $P > 0.05$), and TA ($F_{2,7} = 1.26$, $P > 0.05$). To establish comparisons between ankle muscle electromyographic activities without the influence of ventilatory range reported earlier, one-way analyses of covariance (ANCOVA) were used to test for differences between the three breathing conditions; adjusting either for F_x or F_z considered as covariates. In doing so, there was a significant main effect in RMS SO values after adjusting for F_x ($F_{2,1} = 4.56$, $P < 0.05$) and F_z ($F_{2,1} = 4.66$, $P < 0.05$). Compared with the baseline values obtained during QB, RMS SO values were significantly lower ($P < 0.05$) under the VH condition (Fig. 4). ANCOVA did not reveal any significant main effect for RMS TA values when F_x ($F_{2,1} = 0.15$, $P > 0.05$) and F_z ($F_{2,1} = 0.10$, $P > 0.05$) were statistically controlled.

Discussion

Our experimental hyperventilation model including both VH and CO_2–R induced significant changes in posturographic parameters, showing that postural stability was reduced during those two experimental conditions, and confirming the findings of previous studies dealing with VH (Hunter and Kearney 1981; Jeong 1991; Sakellari et al. 1997; Kantor et al. 2001; Hodges et al. 2002), according to whom hyperventilation represents a significant input for the postural control system. However, the novelty of our study was that these different hyperventilation modalities, resulting from either cortical motor control or automatic chemical bulbar regulation, may lead to different postural control strategies. In our study herein, VH induces a wide range of posturographic perturbations with less postural control, whereas automatic chemoreflex increasing ventilation in response to hypercapnia seems to be less perturbing with a more favourable postural control. These results suggest that a functional link may exist between ventilation and posture control centres.

During VH, the breathing frequency was fixed by a metronome, and therefore appeared as the only controlled component of the minute ventilation. Nevertheless, it is likely that tidal volume increased enough to produce

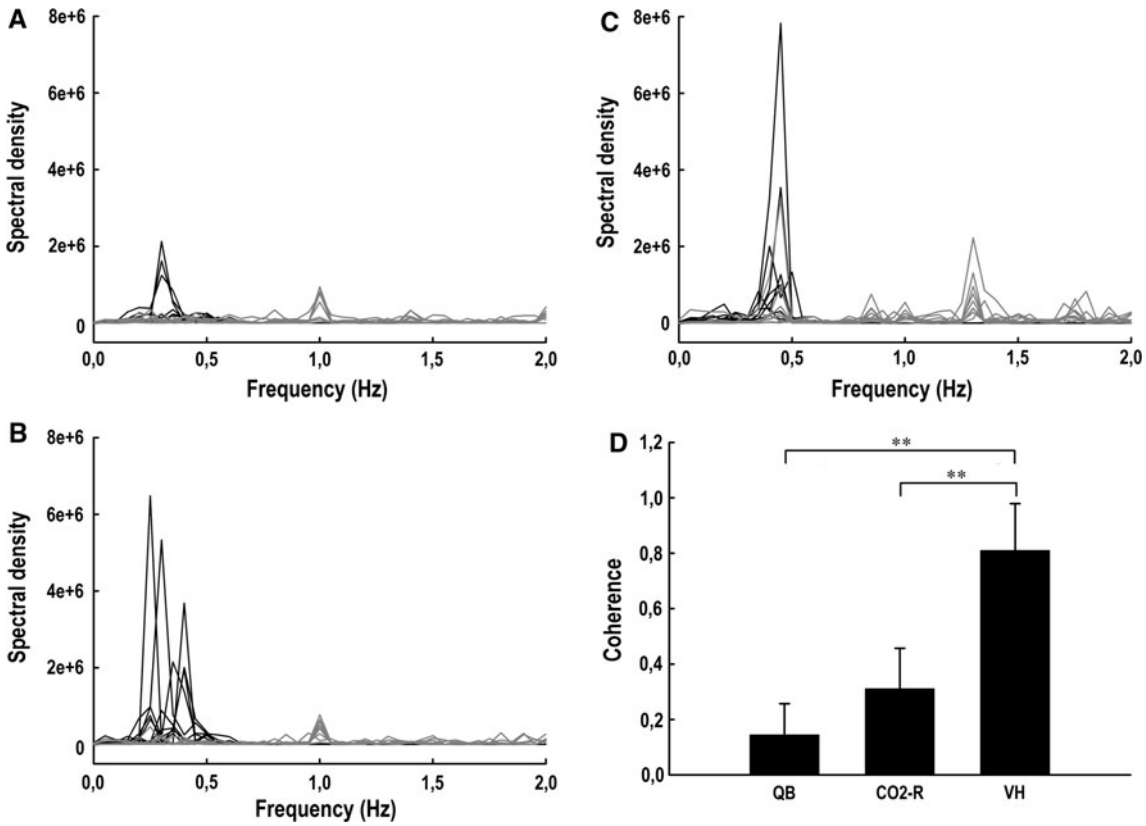

Fig. 3 Power spectrum and coherence analysis applied to both breathing and transverse (F_z) plane signals during the last 20 s of each ventilatory condition for all subjects ($n = 8$). Frequency distributions of the two signals (represented in *black* for chest wall accelerometric signal and in *dark grey* for F_z) during **a** quiet breathing, **b** rebreathing, and **c** voluntary hyperventilation. **d** Coherence, means + one standard deviation for each breathing condition. ** indicates significant differences at $P < 0.01$

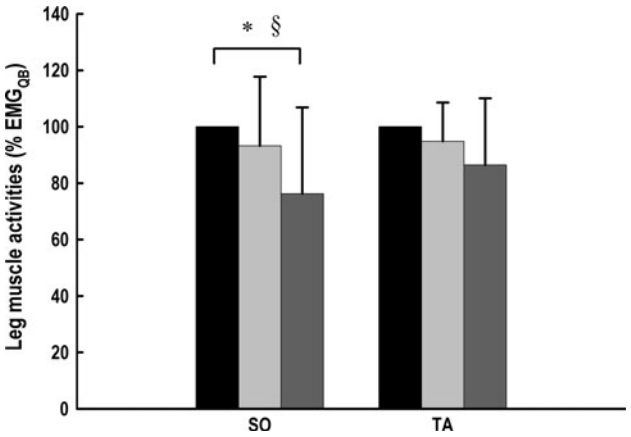

Fig. 4 Electromyographic (EMG) activities of the soleus (SO) and the tibialis anterior (TA) measured in postural tests during the last 20 s of each ventilatory condition: quiet breathing (*black bars*), rebreathing (*light grey bars*), and voluntary hyperventilation (*dark grey bars*). EMGs were quantified in terms of the root mean square and expressed as percentages of each subject's root mean square baseline values obtained during quiet breathing. Mean + one standard deviation are represented. * and § indicates significant difference at $P < 0.05$ when F_x and F_z, respectively, were included as covariates in the analysis

alveolar hyperventilation, as attested in our study by the significant decrease in $F_{\mathrm{Eco_2}}$ values. Conversely, CO_2–R provided hyperventilation with a significant increase in $F_{\mathrm{Eco_2}}$ and $F_{\mathrm{Ico_2}}$ values. However, breathing frequency measured during this period was more irregular than that measured during the QB and VH periods, suggesting greater inter-individual variability. Moreover, the parallel increase in tidal volume was probably large enough to induce a posturographic disturbance, and it may be hypothesized that an increase in tidal volume, as well as breathing frequency during hyperventilation, is much more likely to change mass repartition of the trunk and to disturb posture. In this view, the accelerometer signal RMS increasing values with VH and CO_2–R provided in this study showed a significant increase in the range of ribcage movements in the antero-posterior direction, which is in line with an increase in tidal volume. It is then likely that the marked posturographic perturbations observed during VH are due to a combination of an increase in both breathing frequency and tidal volume, whereas CO_2–R was less perturbing, because of significant increase in rib cage motion amplitude only, without any significant change in breathing frequency and T_I/T_{TOT} ratio.

In a different prospect, Sebert et al. (1990), and more recently, Jensen et al. (2005) reported that gender could interfere with the chemoreflex control of breathing. According to Jensen et al. (2005), using a similar experimental set up to ours (10 L-Rebreathing Douglas bag and oxygen supply), healthy women preferentially increase their breathing frequency during CO_2 rebreathing, whereas men increase tidal volume. Thus, it is likely that our subject sample, which included six men and two women, would exhibit an increase in tidal volume as a group rather than an increase in breathing frequency. According to previous studies (Sackner et al. 1984; Romagnoli et al. 2004), the metabolic variations measured in our study and related to VH and CO_2–R are sufficient to induce significant changes in the volume of the rib cage and abdomen. Previous investigations, using linearized magnetometers (Filippelli et al. 2002) or more recently with optoelectronic plethysmography (Aliverti et al. 1997; Romagnoli et al. 2004), have reported that CO_2–R corresponds to an equally distributed tidal volume between rib cage and abdominal wall motions. In these studies, both rib cage and abdominal muscles are recruited through a coordinated action to assist diaphragmatic function, and to limit rib cage distortion. To our knowledge, chest wall kinematics and respiratory muscle action during hypocapnia related to VH has not been previously investigated. Nevertheless, it is well known that abdominal muscles are important contributors to ventilation, notably during voluntary expiratory effort (Bai et al. 1984). Nevertheless, our results suggest that respiratory muscle recruitment cannot itself account for all of the different postural disturbances between VH and CO_2–R, as revealed by posturographic analysis. Data obtained from the force plate indicated that postural perturbations during VH in the transversal and sagittal planes ratio ($\Delta F_z/\Delta F_x$) were the same order of magnitude as those measured during CO_2–R. Insofar as the larger thoracic movements during VH did not correspond to any metabolic need, and that the breathing frequency was a controlled variable, the slight increase in CoP amplitude noticed in the lateral direction during VH, and not during CO_2–R, can be regarded as a consequence of either a more disturbed posture or a less efficient postural control. The postural instability observed during VH can be related to an increase in the proportion of rib cage contribution to tidal volume, as reported elsewhere (Sackner et al. 1984), and shown recently as more disturbing than the abdominal contribution (Hamaoui et al. 2010). During VH, data in the time and spectral domains from the vertical ground reaction force (F_z) revealed an increase in the mean amplitude and a decrease in MPF, respectively. This shift towards these frequencies, corresponding to the target breathing frequency during VH, suggests a better coupling between respiratory movement and postural sway in the transverse plane. Moreover, the finding that the coherence value was significantly higher for VH than for CO_2–R and QB reinforces this hypothesis, and shows a strong correlation between breathing frequency and force oscillations in the frequency domain. In comparison with CO_2–R, this did not induce such coupling, where the MPF value was in the same range as in QB, and the mean coherence value was close to that obtained in QB. Finally, these results suggest a specific alteration of postural control during VH.

Because rib cage movements and the respiratory muscle pattern cannot themselves explain all of the differences observed between VH and CO_2–R on the maintenance of standing posture, we may suggest extra-thoraco-abdominal factors, such as sensory afferents or descending motor outputs. It is now well established that postural control requires the integration of a variety of sensory information including visual, vestibular and somatosensory systems (Diener and Dichgans 1988). Because all experiments were conducted with eyes open, visual afferences cannot specifically explain our results. Amongst other sensory inputs, large diameter myelinated afferents (group I and II) provide the primary source of lower limb proprioceptive information for maintaining a standing posture (Fitzpatrick et al. 1994; Nardone et al. 2006). However, small diameter unmyelinated afferents transmitting noxious stimuli (group IV) can also influence motor behaviour and impair postural control (Blouin et al. 2003), as demonstrated in low back pain patients compared with healthy subjects (Brumagne et al. 2004; Hamaoui et al. 2004).

Following this conceptual frame of multiple sensory afferences modulating postural control during hyperventilation, our EMG recordings should provide some insight about the integration of such disturbing and regulating influences. However, as suggested before, minute ventilation (as a combination of breathing frequency and rib cage motion) was probably larger during VH than CO_2–R, enhancing larger postural disturbance as recorded by the ground reaction force. This could introduce a bias in the interpretation of our EMG data, preventing us to distinguish what is due to differential ventilatory perturbation from specific change of the postural control, if any. Because we did not measure tidal volume or chest wall changes directly, and hyperventilation tests cannot be paired strictly, we normalised the EMG data by comparing it with that of F_x and F_z, the most respiratory-sensitive postural indexes. Then, a significant difference was revealed, suggesting that postural control is likely to be lower in response to voluntary hyperventilation known to bypass automatic centres at the level of the forebrain or the phrenic motoneuron pool (Aminoff and Sears 1971; Corfield et al. 1998). Moreover, the difference between VH and CO_2–R is retrieved when comparing EMG RMS values from SO. This particular result makes sense, because it

involves a strong calf antigravitational muscle, and upholds that different postural control strategies may be expressed in response to different breathing patterns. Nevertheless, we have to assume that our RMS values calculated over 20-s periods corresponded to several single breaths only. In this study, data collection did not allow us to examine breath-by-breath ankle muscle EMG bursts in response to ribcage motion or postural sway. Because our results suggest a difference in EMG pattern between CO_2–R and VH, further investigations are thus justified.

To explain this difference, proprioceptive afferents should be increased relative to a higher contribution of lower limbs and changes in pressure range at the level of the sole of the foot. In fact, our results reveal an increase in ground reaction force fluctuations during VH, especially for the vertical and sagittal components. The diaphragm pushes down on the abdominal viscera to increase thoracic cavity diameter and produce inspiratory airflow (De Troyer and Estenne 1988); thus, changing mass repartition in the body. It is then likely that mechanoreceptors would fire in response to this postural stimulus. Changes in somatosensory information should facilitate an increment of EMG activity in the leg muscles, known to enhance musculoarticular stiffness of the ankle joint (Winter et al. 1998) during VH preferably. In their study, Sakellari et al. (1997) attempted to establish links between voluntary hyperventilation and somatosensory information from the lower limbs by investigating peripheral sensory action potentials (SAP) through stimulating sural nerves. These authors reported a change in the peripheral SAP amplitude during VH, reflecting an increase in peripheral nerve excitability. In our study, we cannot rule out that increasing proprioceptive fibre excitability or ectopic somatosensory fibre firing during the VH condition impacted either spinal reflex loops or postural centres at higher input levels. However, we should have recorded larger leg muscle electromyographic responses during VH. Conversely, we should have achieved a marked reduction in postural control indices from the force plate during CO_2–R, but our results showed the opposite.

Amongst the sensory information involved in postural control during hyperventilation, vestibular activity as well as posturo-kinetic spine receptors is likely to increase with head movements induced by upper trunk motion. Thus, we may hypothesise that neck and head movements were larger during VH than during CO_2–R and that should impact postural control (Keshner et al. 1987). However, results from Sakellari et al. (1997) indicate that VH did not create unsteadiness by disrupting vestibulo-spinal activity. Moreover, patients with a total absence of vestibular function could develop hyperventilation unsteadiness as well as normal subjects (Sakellari and Bronstein 1997). So, this cannot explain the change in postural control that seemed different during these two different hyperventilation modalities.

In conclusion, our data indicates physiological vulnerability of the standing posture during voluntary hyperventilation compared with automatic ventilatory regulation. These results support our hypothesis that respiratory centres may modulate postural control directly. The present study focused on the ankle muscles (soleus and tibialis anterior) and we cannot rule out the role played by muscles acting at the hip or trunk levels. Further investigations are needed to examine how this inhibition could target different muscle groups (hip and trunk muscles) involved in postural control, and to better understand to what extent this kind of inhibition should act at a peripheral or a supraspinal level. Finally, our results suggest that stability of the body may be compromised in situations in which respiratory demand increases and requires voluntary control.

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