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29 **Abstract**

30 The goal of this study was to establish how increased ventilation modifies postural
31 stability, as characterized by body sway and leg muscle activities. Twelve healthy
32 subjects had to perform six 30-second postural tests: one pre-exercise test while
33 breathing gently and then one test every minute for the five minutes immediately
34 following a maximum-intensity, incremental cycling exercise test. Subjects were asked
35 to maintain an upright stance on a force plate for 30 s, with their eyes open. Movement
36 of the centre of pressure in the sagittal plane was monitored in the time and spectral
37 domains. Myoelectric activities of the soleus and tibialis anterior muscles were recorded
38 using surface electromyography. Ventilatory parameters were measured with a portable,
39 telemetric device. Postural changes related to respiratory variations were quantified by
40 coherence analysis. The results showed that hyperventilation induced by exercise was
41 accompanied by a significant increase in postural parameters, indicating a reduction in
42 postural stability following a change in ventilatory drive. Coherence analysis confirmed
43 the ventilatory origin of the postural oscillations. The results suggest that ventilation
44 may be an important factor in postural disturbance during physical activity. The
45 observed increases in leg muscle activities were most likely related to musculo-articular
46 stiffening.

47 **Introduction**

48 The ability to maintain balance plays a fundamental role in motor programming. In
49 many sporting activities, this ability is considered as a key factor for supporting
50 voluntary movement (3). In addition to gravity and other external forces, internal forces
51 (related to voluntary movement and cardiorespiratory activities) may also disturb
52 postural stability. In an upright stance at rest, it was well known that quiet breathing
53 perturbs body balance in the sagittal and frontal planes and that the effects of
54 compensatory motions of the trunk and lower limbs are more pronounced in the sagittal
55 plane (2, 4, 10, 11). During high-intensity physical activity, however, the tremendously
56 increased oxygen need translates into greater minute ventilation and deeper, faster rib
57 cage movements. Hence, in principal, body mass distributions and maintenance of
58 posture should be markedly perturbed during physical activity. Postural perturbation
59 related to physical exercise has been demonstrated (7, 18). However, these studies are
60 mainly focused on the effects of central and/or peripheral fatigue and have minimised
61 internal perturbation linked to a deep and paced breathing. Thus, the aim of the present
62 study was to investigate the extent to which an increase in ventilation might decrease
63 postural stability. We therefore studied postural sway and leg muscle electromyograms
64 (known to reflect postural strategies (12)) before and after a maximum-intensity,
65 incremental cycling exercise test.

66

67 **Materials and Methods**

68 *Subjects*

69 Twelve healthy university students participated in this non-invasive study (mean age:
70 20.6 ± 1.8 yrs; mean weight: 69.2 ± 9 kg; mean height: 1.7 ± 0.1 m). All subjects had

71 been fully informed of the experiment's aims and procedures (in accordance with the
72 Declaration of Helsinki) and gave their written consent. None of them presented any
73 known diseases or injuries in their medical history that might have impaired balance or
74 respiration. In order to evaluate the subjects' pulmonary capacity, routine spirometric
75 tests by using a Fleisch pneumotachograph (Masterlab, Jaeger, France) were always
76 performed by the same experimenter. The normality of parameters such as tidal volume
77 (V_T) or vital capacity (VC) was judged according to the reference values proposed by
78 the European Respiratory Society (9).

79 *Force plate data*

80 The ground reaction forces and their moments in the three planes (used to determine the
81 coordinates of the centre of pressure, COP) were recorded using a rectangular (60 x 40
82 cm), piezoelectric force plate (Kistler type 9281 B11, Kistler AG, Winterthur,
83 Switzerland) linked to a charge amplifier (Kistler type 9851, Kistler AG, Winterthur,
84 Switzerland). Since respiratory movements mainly concern the sagittal plane (11), we
85 only considered the COP's anteroposterior displacement (COP_x) recorded with a
86 sensitivity of 50 mm/V.

87 *Electromyography*

88 Electromyographic (EMG), bipolar recordings of soleus (SO) and tibialis anterior (TA)
89 muscle activities were made on the dominant leg by using Ag/AgCl surface electrodes
90 (Beckmann, 8 mm in diameter). Before placement of the latter, the skin at the electrode
91 sites was prepared so as to achieve an inter-electrode impedance of less than 5 k Ω . Pairs
92 of electrodes (centre-to-centre spacing: 20 mm) were respectively placed about 2 cm
93 below the insertion of the gastrocnemius on the Achilles tendon for the SO and on the
94 belly of the TA, parallel to the muscle fibres. A ground electrode was placed on the

95 medial surface of the tibia. EMG signals were differentially amplified (Gould™ 6600,
96 Cleveland, USA) with a gain set to between 1000 and 5000 and filtered using a
97 bandwidth of 10-1000 Hz. EMG and COP signals were simultaneously sampled at 1000
98 Hz by using a 12-bit analogue-to-digital converter (National Instrument Daq Card™-
99 AI-16E-4). The data were collected on a laptop computer interfaced with DataSet Pro
100 software (SETRI, France) and then stored for off-line processing.

101 *Ventilatory measurements*

102 Respiratory gas exchanges were monitored telemetrically using a portable gas analyser
103 (Cosmed™ K₂, Vacumetrics Inc., Ventura, CA, USA). The validity and reliability of
104 measurements with the Cosmed K₂ have been demonstrated by a number of detailed
105 studies (16). The Cosmed K₂ was carefully calibrated prior to each experiment. The
106 subject wore the transmitting unit, battery and face mask (represented a total mass of
107 850 g). The face mask sealed tightly over the subject's nose and mouth and was
108 connected to a turbine flow meter. The latter did not add any significant, additional air
109 flow resistance. Minute ventilation (\dot{V}_E) was measured, along with its tidal volume
110 (V_T) and breathing frequency (f_R) components. The values were averaged every 5
111 seconds, stored and then transferred onto the laptop computer for further analysis. For
112 each subject, the V_T to V_C ratio was used as an index of ventilatory change. The
113 ventilatory flow signal was obtained via the Cosmed K₂ by measuring variations in
114 timing breathing. When the subject expires through the face mask, the revolutions of the
115 turbine produce a voltage signal. Conversely, when the subject inspires, the turbine
116 stops and entails the cancellation of the voltage signal. The output signal is transmitted
117 (simultaneously with COP_x and EMG signals) to the 12-bit analogue-to-digital
118 converter acquisition system.

119 *Experimental procedure*

120 Subjects had to perform a total of 6 postural tests, each corresponding to maintenance of
121 a bipedal stance for 30 s (eyes open). A reference test (pre-effort postural test) was
122 carried out in quiet conditions prior to a maximal incremental cycling exercise (ICE)
123 test lasting about 10 minutes, i.e. enough to generate hyperventilation (HV). Five post-
124 effort postural tests (PEPTs) were then carried out - immediately after the ICE (PEPT₀)
125 and then during the first 30 seconds of each of the next 4 minutes (PEPT₁ to PEPT₄).

126 All postural tests were performed in a quiet room without sound and light
127 interference. Subjects were asked to stand barefoot on the force plate, relax with their
128 arms hanging alongside the trunk and focus on a target placed 2 m in front of the subject
129 at eye level. The foot position was standardized by placing foot prints on the force plate,
130 in agreement with the recommendations of the French Posturology Association (1). The
131 foot prints (adjusted to the subject's foot length) were drawn so that the respective
132 longitudinal axes formed a 30° angle and intersected the force plate's anteroposterior
133 axis, with the heels 2 cm apart.

134 ICE on a cycle ergometer (Monark™ 824&, Sweden) was performed with
135 cardiac monitoring until exhaustion, as judged by each subject's maximum oxygen
136 uptake ($\dot{V}O_2\text{max}$). This parameter was predicted by using the gender-adjusted
137 formulae for cycle ergometer exercises reported by Wasserman et al. (20). Resting
138 oxygen uptake ($\dot{V}O_2\text{rest}$) was measured in a relaxed, upright stance using the Cosmed
139 K₂. After subtraction of $\dot{V}O_2\text{rest}$ and by considering that each watt required 10.3
140 ml/min of O₂ (19), the maximum workload (W_{max}) was calculated as follows:
141 $W_{\text{max}} = (\dot{V}O_2\text{max} - \dot{V}O_2\text{rest})/10.3$. Subjects performed a 3-min warm-up (with a
142 workload corresponding to 20% of W_{max}) and then a 10-min period of incremental

143 exercise from 20 to 100% of Wmax, with an increased workload stage every minute.
 144 For each stage, the added workload was $(W_{max}-20\%W_{max})/10$. The pedalling rate
 145 was held at 60 revolutions per minute.

146 *Signal processing*

147 The off-line processing of COPx, EMG and respiratory signals was performed using a
 148 custom processing routine written with Matlab 6.5 (The Mathworks, Inc., Natick, MA,
 149 USA). The respiratory and COPx signals were passed through a 4th-order low-pass
 150 Butterworth filter with a cut-off frequency of 15 Hz. Over the 30 s of each postural test,
 151 the mean amplitude (Ax) and the sway path (SPx) were calculated using the method
 152 described by Hufschmidt et al. (13). The frequency content of the COPx displacements
 153 was computed by Fourier analysis in order to determine the mean power frequency
 154 (MPF). The power spectral densities of the respiratory and COPx signals were
 155 calculated by periodogram method. Coherence analysis was used for measuring changes
 156 in COPx related to respiratory variations. The magnitude squared coherence $C_{xy}(f)$ of
 157 the signals x and y was estimated at each frequency f as:

$$158 \quad C_{xy}(f) = \frac{|P_{xy}(f)|^2}{P_{xx}(f) \cdot P_{yy}(f)},$$

159 where $P_{xx}(f)$ and $P_{yy}(f)$ represent the power spectral densities of x and y respectively and
 160 $P_{xy}(f)$ the cross power spectral density of x and y . The spectral densities can be obtained
 161 by average Welch's periodogram. Coherence provides a normalised spectral measure
 162 (ranging between 0 and 1) of the linear association between two signals as a function of
 163 frequency, where 0 indicates that there is no association and 1 indicates a perfect
 164 association. During the postural tests, muscle activation levels were quantified from the
 165 raw EMG signals by computing the root mean square value (RMS) over 300 ms with a

166 sliding step of 20 ms. The RMS values were then averaged and expressed as
167 percentages of each subject's baseline values acquired during the pre-effort postural test.
168 Muscular fatigue was assessed through power spectral analysis of the EMG signals by
169 calculating the MPF value.

170 *Statistical analyses*

171 A simple repeated-measures analysis of variance was performed to assess the effects
172 caused by ICE on ventilatory, electromyographic and postural sway parameters
173 (Statview, SAS Institute Inc, Cary, NC, USA). Post hoc testing (the Student Newman-
174 Keuls test) was used to identify local differences. Using the data from all subjects and
175 all postural tests, Bravais-Pearson correlation coefficients (r) were calculated in order to
176 evaluate potentially linked parameters. For all statistical tests, differences were
177 considered significant when $p < 0.05$.

178

179 **Results**

180 The mean (\pm *SD*) power output produced by the subjects at the end of ICE was $249.6 \pm$
181 49.9 W. As shown in Table 1, measurements taken at PEPT₀ immediately following
182 ICE prompted a significant increase in $\dot{V}O_2$ (852%), \dot{V}_E (703%), V_T (260%), V_T/VC
183 (255%) and f_R (127%), ($p < 0.01$). Significant increases in both Ax (54%, $p < 0.05$) and
184 SPx (168%, $p < 0.01$) were observed. All ventilatory and postural parameters remained
185 significantly higher ($p < 0.05$) 4 minutes after ICE, with the exception of Ax, which
186 returned to pre-effort values one minute after ICE. Linear regression analysis showed a
187 number of significant correlations between ventilatory and postural sway parameters -
188 particularly between \dot{V}_E and SPx and between f_R and SPx (Figure 1). The correlations
189 between ventilatory parameters and Ax (ranging between 0.24 and 0.42) were always

190 lower than those found for ventilatory parameters and SPx (ranging between 0.51 and
191 0.82).

192 In comparison with reference values, the spectral analysis of the COPx
193 displacements revealed a significant increase in MPF (34%, $p < 0.01$) which remained
194 significantly higher 4 minutes after ICE ($p < 0.01$). The results of coherence analysis
195 demonstrated a high level of correlation (0.70 ± 0.11) between respiratory and COPx
196 signals at PEPT₀ immediately after ICE over precise frequency range (0.5-0.7 Hz).
197 Examples of spectral and coherence analysis are illustrated in Figure 2.

198 As shown in Figure 3, ICE also led to a significant increase in SO (190%, $p <$
199 0.01) and TA (87%, $p < 0.01$) RMS values in all PEPTs. After normalizing RMS values
200 with respect to Ax, comparisons with pre-effort values remained significantly high for
201 both muscles ($p < 0.05$). Electromyogram MPF mean ($\pm SD$) values obtained prior and
202 just after ICE were 133.6 ± 8.7 Hz vs 128.5 ± 11.5 Hz for SO ($p > 0.05$) and 97.2 ± 10.2
203 Hz vs 88.2 ± 17 Hz for TA ($p > 0.05$).

204

205 **Discussion**

206 Hence, we observed that cycling exercise up to maximum oxygen consumption led to
207 major adaptations in ventilation: notably, oxygen debt had a lasting impact on the
208 postural control system. Indeed, the value of V_T/VC (revealing greater excursion of the
209 rib cage motion) remained significantly higher than at rest throughout the 5-min
210 recovery, providing a relevant model for post-exercise (metabolic) hyperventilation.

211 In agreement with other work (2, 11, 14, 15), increasing respiratory output
212 reduced postural stability (as measured by SPx and Ax changes). Because of higher
213 post-exercise ventilation, the body sway range quantified here was much higher than

214 those reported in previous studies (2, 15). Here, the postural parameters progressively
215 returned to their initial values following the kinetic of the ventilatory recovery (as
216 attested by correlations between \dot{V}_E or f_R and SPx), suggesting a ventilatory origin of
217 postural instability.

218 Nevertheless, the reduction in postural stability following exercise could also
219 correspond to leg muscle fatigue after cycling. Taking into account treadmill running
220 and cycling exercise at 125 Watts, Nardone et al. (18) have only observed postural
221 perturbation after running. They have attributed these differences to a lower oxygen
222 uptake in cycling (65.2% $\dot{V}O_{2max}$) compared to running (84.6% $\dot{V}O_{2max}$) and to a
223 lower peripheral fatigue of leg muscle in cycling. However, postural disturbance has
224 been demonstrated after a prolonged cycling exercise (17) and the incremental cycling
225 exercise used in the present study has been brought until ~250 Watts and 100% of the
226 maximal oxygen uptake. Fatigue is known to alter the mechanisms involved in postural
227 control (17). Studies of the effects of fatigue on the upright stance report an increase in
228 postural sway (5, 6). For example, Caron (5) reported that isometric contraction of the
229 plantar flexor until task failure increases the mean velocity of COP displacements by
230 31%. The mean velocity values reported in our study at PEPT₀ showed an increase by
231 170%. Thus, the effects of fatigue of the lower limbs induced by cycling exercise
232 cannot alone explain such modifications. Furthermore, several studies related to
233 voluntary hyperventilation (2, 15) - which should not induce leg muscle fatigue - have
234 reported perturbed balance, suggesting a ventilatory origin. Hence, in the present study,
235 muscle fatigue does not appear to be the main factor in postural disturbance. The high
236 level of coherence found in the current study between respiratory and COPx signals
237 over the low frequencies (0.5-0.7 Hz) clearly confirmed the ventilatory origin of the

238 COPx oscillations. Thus, our results suggest that ventilation may be an important factor
239 in postural disturbance during physical activity.

240 Correlation analyses indicate that the sway path was better correlated with f_R
241 than with V_T , which is in good agreement with Jeong (14). This finding suggests that f_R
242 is the main factor disturbing balance in the upright stance. In comparison with SPx
243 values, correlations between ventilation parameters and Ax were lower: at the start of
244 the recovery period, Ax values did not significantly differ from their baseline values.
245 One could suppose that the nervous system sets up earlier compensatory mechanisms to
246 limit the amplitude (rather than the frequency) of the COPx oscillations. One possible
247 mechanism is co-contraction of antagonistic leg muscles, since we observed a
248 significant increase in the activation levels of both SO and TA. The increase in SO and
249 TA electromyography activity is known to enhance musculo-articular stiffness of the
250 ankle joint (21). Insofar as COP motion reflects the activity of the neuromuscular
251 system, this could also explain the increase in spectral content of COPx motion towards
252 high frequencies (as measured by MPF changes). However, the increase in leg muscle
253 activities observed in our study after cycling could also reflect a recruitment of
254 additional motor units and/or an increase in motor unit firing rates in response to muscle
255 fatigue (5) but the spectral analysis of SO and TA electromyography activity -
256 conducted before and immediately after ICE - revealed no significant change in the
257 mean power frequency, an index used to detect muscle fatigue (8). That reinforces the
258 hypothesis of a compensatory mechanism to decrease sway amplitude, rather than leg
259 muscle fatigue enhancing postural instability.

260 In summary, we demonstrated that post-exercise model of metabolic
261 hyperventilation leads to major postural disturbances, as notably reflected by increased

262 body oscillation in the sagittal plane. Coherence analysis proved to be a subtle method
263 to show the close relationship between breathing and postural stability. It is likely that
264 the strategy used by the nervous system to limit amplitude of body oscillation
265 corresponds to recruitment of leg muscle motor units and a resultant increase in
266 musculo-articular stiffness.

267

268 **References**

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324 **Figure captions**

325

326 Fig. 1. - Relationships between the sway path (SP_x) of the COP_x displacements and the
327 minute ventilation (\dot{V}_E in panel A) or the breathing frequency (f_R in panel B) in twelve
328 subjects. For each subject, the data were generated in a single pre-effort postural test
329 and then five postural tests following incremental cycling exercise.

330

331 Fig. 2. - Example of spectral and coherence analysis applied on both respiratory and
332 COP_x signals at PEPT₀ for one subject. In panel A, the frequency distribution of the two
333 signals (represented in grey for the ventilation and in black for the centre of pressure)
334 revealed a synchronisation in spectral densities around 0.6 Hz. In panel B, illustration of
335 coherence spectrum measurement between the respiratory and COP_x signals obtained
336 for the same subject. Its magnitude of 0.81 over the low frequencies (0.6 Hz) indicated a
337 high level of correlation between these two signals.

338

339 Fig. 3. - Soleus (black bars) and tibialis anterior (grey bars) EMG activities measured in
340 postural tests following incremental cycling exercise. EMGs were quantified by their
341 RMSs and expressed as percentages of the baseline values obtained in the pre-effort
342 postural test (dotted line). Means plus one standard deviation are represented. * and **
343 denote significant differences between the pre-effort postural test and the post-effort
344 postural tests at $p < 0.05$ and $p < 0.01$ respectively.

345 **Tables**

346 *Table 1 - Ventilatory ($\dot{V}O_2$, \dot{V}_E , V_T , V_T/VC , f_R) and postural sway (SPx, Ax, MPF)*
 347 *parameters measured in postural tests before (pre-effort test), just after (PEPT₀) and*
 348 *over the next 4 minutes following (PEPT₁ to PEPT₄) an incremental cycling exercise*
 349 *(mean \pm standard deviation). * and ** denote significant differences between the pre-*
 350 *effort postural test and the post-effort postural tests at $p < 0.05$ and $p < 0.01$*
 351 *respectively.*

| | Pre-effort test | PEPT ₀ | PEPT ₁ | PEPT ₄ |
|--|------------------|---------------------|---------------------|-------------------|
| $\dot{V}O_2$ (L _{STPD} .min ⁻¹) | 0.36 \pm 0.05 | 3.43 \pm 0.70** | 2.35 \pm 0.41** | 0.66 \pm 0.16** |
| \dot{V}_E (L _{BTPS} .min ⁻¹) | 12.8 \pm 1.4 | 102.8 \pm 19.1** | 71.0 \pm 11.2** | 28.4 \pm 5.5** |
| V_T (L _{BTPS}) | 0.75 \pm 0.10 | 2.70 \pm 0.65** | 2.40 \pm 0.61** | 1.28 \pm 0.30** |
| V_T/VC (%) | 14.5 \pm 1.9 | 51.5 \pm 9.0** | 45.6 \pm 8.4** | 24.3 \pm 4.4** |
| f_R (Hz) | 0.29 \pm 0.05 | 0.66 \pm 0.19** | 0.52 \pm 0.15** | 0.38 \pm 0.08** |
| SPx (mm) | 221.3 \pm 32.0 | 594.5 \pm 101.2** | 339.5 \pm 120.4** | 268.6 \pm 45.2* |
| Ax (mm) | 7.2 \pm 2.4 | 11.1 \pm 2.6* | 6.7 \pm 1.7 | 6.2 \pm 2.0 |
| MPF (Hz) | 0.46 \pm 0.06 | 0.62 \pm 0.13** | 0.56 \pm 0.16** | 0.59 \pm 0.09** |

352

Figures

Figure 1

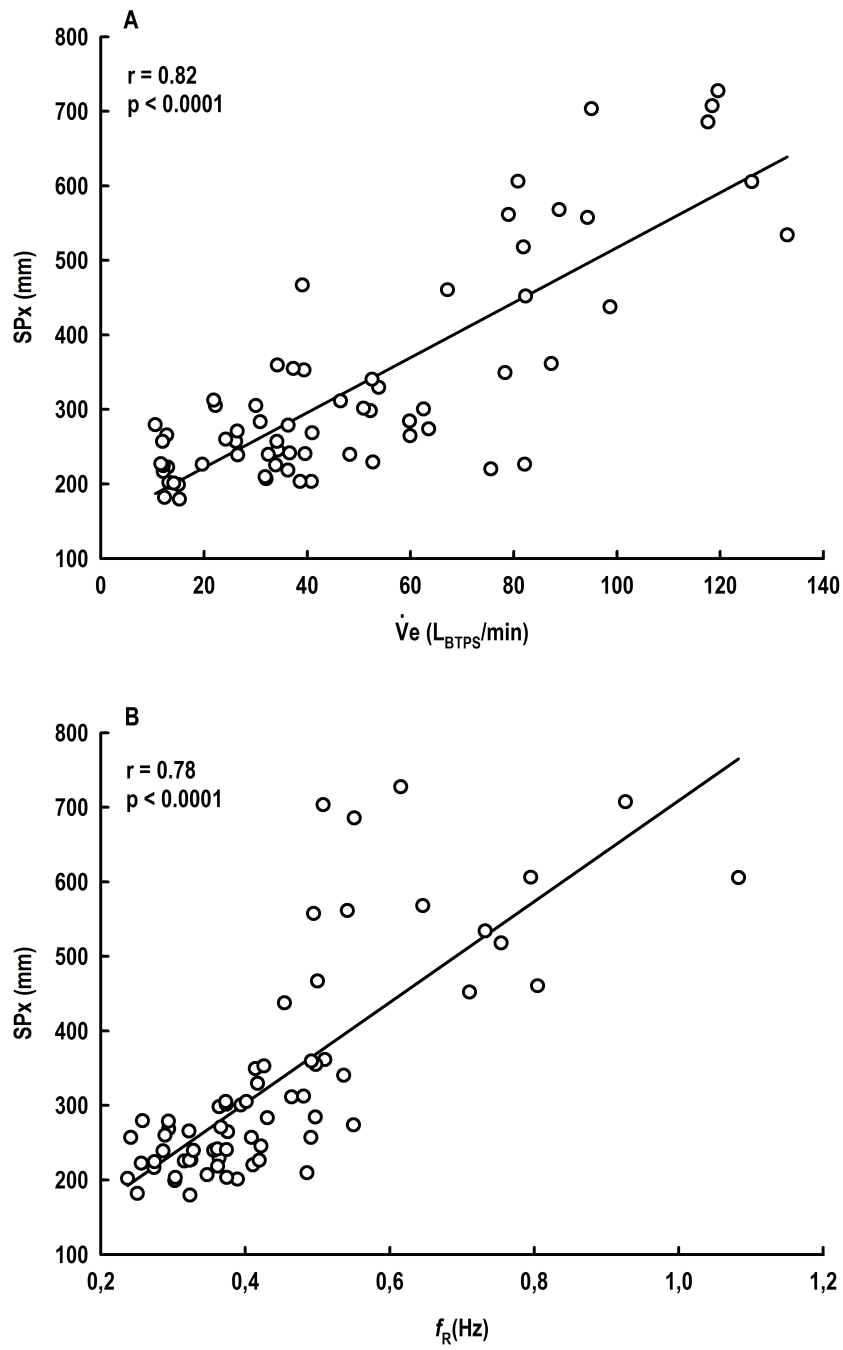


Figure 2

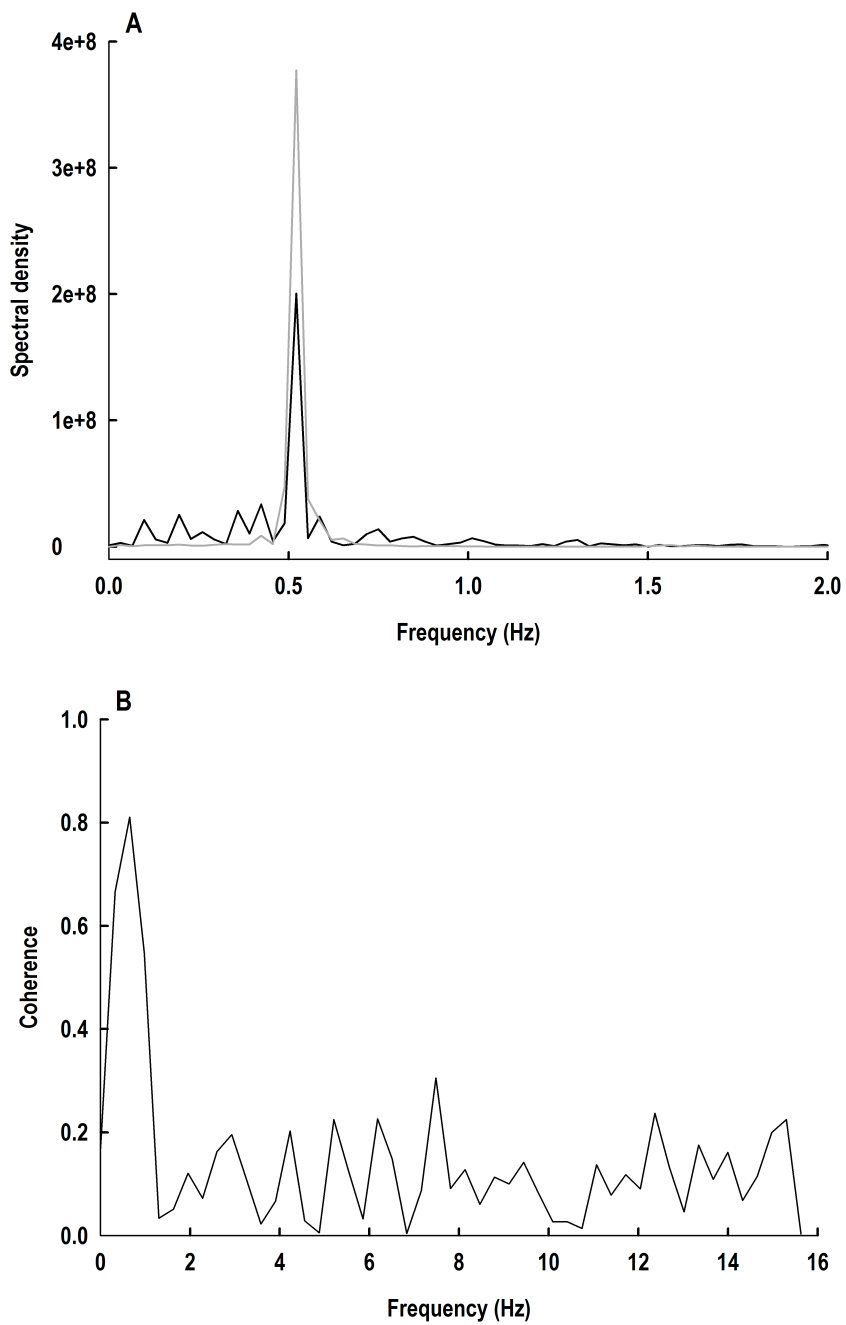


Figure 3

