Influence of prolonged bed-rest on spectral and temporal electromyographic motor control characteristics of the superficial lumbo-pelvic musculature

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Abstract

Little is known about the motor control of the lumbo-pelvic musculature in microgravity and its simulation (bed-rest). Analysis of spectral and temporal electromyographic variables can provide information on motor control relevant for normal function. This study examined the effect of 56-days of bed-rest with 1-year follow-up in 10 male subjects on the median frequency and the activation timing in surface electromyographic recordings from five superficial lumbo-pelvic muscles during a repetitive knee movement task. Trunk fat mass (from whole body-composition measurements) and movement accuracy as possible explanatory factors were included. Increased median frequency was observed in the lumbar erector spinae starting late in bed-rest, but this was not seen in its synergist, the thoracic erector spinae (p < .0001). These changes persisted up to 1-year after bed-rest and were independent of changes in body-composition or movement accuracy. Analysis suggested decreases of median frequency (p < .0001) in the abdominal and gluteal muscles to result from increased (p < .01) trunk fat levels during and after bed-rest. No changes in lumbo-pelvic muscle activation timing were seen. The results suggest that bed-rest particularly affects the shorter lumbar erector spinae and that the temporal sequencing of superficial lumbo-pelvic muscle activation is relatively robust.

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either an absence of anticipatory muscle activity (Layne and Spoor, 1990, 1994) or a reduction of its level (Mouchinho et al., 1996) in the microgravity phases of parabolic flight. Longer-term studies in spaceflight on the leg musculature (Clément et al., 1984, 1985), whilst showing the timing of anticipatory muscle activity to be relatively stable, found EMG amplitudes greatly reduced with continued exposure to microgravity. Other works (Layne et al., 1997, 1998, 2001), whilst finding more variable patterns of leg muscle activation in astronauts after spaceflight, also failed to find a consistent trend for changes in the activation timing of the leg musculature. In the current study, a first goal was to examine the influence of microgravity simulation (prolonged bed-rest) on the temporal activation of the LP musculature.

Some unique studies in astronauts that have also considered spectral analyses of leg muscle activity have observed frequency shifts after spaceflight during non-fatiguing leg extensor muscle contractions (Antonutto et al., 1998; Bodem et al., 1998; La Fevers et al., 1975, 1976). Whilst the works by Antonutto and Bodem did not directly consider the direction of the frequency shift, the works by La Fevers and colleagues (1975, 1976) found shifts to higher frequencies of activation in the gastrocnemius muscle. This suggests that spectral analyses could also provide valuable insight into motor control adaptation in spaceflight and simulation. As a second goal we therefore decided to analyze EMG frequency shifts in the LP musculature during and after microgravity simulation (prolonged bed-rest).

The aim of this experiment was to investigate changes in temporal (activation levels and timing) and spectral (frequency shifts in non-fatiguing muscle activation) analysis of the LP musculature using surface EMG during and after prolonged bed-rest. Due to the strong influence of subcutaneous fat levels on spectral variables and amplitudes in studies of surface EMG (Farina et al., 2004), to control for any parallel changes in fat levels, data from a parallel body-composition experiment were included.

2. Methods

2.1. Bed-rest protocol

The “Berlin Bed-Rest Study” was implemented by the Centre of Muscle and Bone Research at the Charité Benjamin Franklin Hospital in Berlin, Germany, from February 2003 to May 2005. Ten male subjects underwent 8-weeks of bed-rest with 1-year follow-up. The bed-rest protocol, as well as inclusion and exclusion criteria, is discussed in detail elsewhere (Rittweger et al., 2006). However, in brief, subjects were required to remain in bed at all times and were required to restrict activity in bed to the minimal required for hygiene and other necessary daily tasks. Adherence to this protocol was monitored by continuous video recordings and force transducers in the frame of the bed. The institutional ethics committee approved this study and subjects gave their informed consent. After bed-rest, all subjects were offered a generalised (cardiovascular fitness and strength) rehabilitation programme at a local physiotherapy practice and returned to their normal work and leisure activities.

2.2. Repetitive knee movement model and testing protocol

To allow the examination of both timing of muscular activity as well as median frequency during non-fatiguing isometric contraction, a model using repetitive knee movement to stimulate cyclic modulation of isometric LP muscular activity was implemented in conjunction with new signal processing approaches (Belavy et al., 2006). The movement paradigm was implemented using repetitive right knee movement in prone lying (Fig. 1). Straps were placed over the subject’s buttocks and distal thigh to reduce movement at these points and expedite isometric LP muscle action. Movement was conducted with the right leg and a spring was attached to the right ankle. Straps were positioned over the buttocks and lower thigh. For further details, see text.

Fig. 1. The repetitive knee movement model. The subject was positioned in prone lying and with a monitor placed under the apparatus support for feedback purposes. A goniometer was placed at the right knee to monitor knee position and a spring was attached to the right ankle. Straps were positioned over the buttocks and lower thigh. For further details, see text.

2.3. Lumbo-pelvic muscle EMG and signal acquisition

Five superficial LP muscles were monitored. To examine different functional parts of the erector spinae with surface EMG, electrodes were placed over the lumbar erector spinae with multifidus (LES; at the level of the 5th lumbar vertebrae between the spinous process and a line drawn from the posterior superior iliac spine (PSIS) to the interspace between the 1st and 2nd lumbar vertebrae (de Foa et al., 1989; Ng et al., 2001)) and thoracic erector spinae (TES; at the level of the 2nd and 3rd lumbar interspace, 1 cm medial to a line drawn from PSIS to the lateral border of the erector spinae at the 12th rib (de Foa et al., 1989; Ng et al., 2001)). Surface EMG using these placements has been shown to correlate highly with intra-muscular electrodes in the underlying muscles during a range of manoeuvres (Arokoski et al., 1999) and electrodes placed at least 3 cm apart over different parts of the erector spinae, as in the current work, can be regarded as giving sufficiently specific signals (Vink et al., 1989). The abdominal muscles were monitored
with electrodes placed over the internal oblique (IO; the superior electrode placed 1 cm medial to the ASIS, the inferior electrode placed parallel to the inguinal ligament at the standard inter-electrode distance (Ng et al., 1998, 2001)) and external oblique (EO, at the most inferior point of the costal margin orientated along a line from that point to the contralateral pubic tubercle (Ng et al., 1998, 2001)). The inferior gluteus maximus muscle (IGM) was monitored with electrodes placed inferior and medial to a line drawn between the PSIS and posterior greater trochanter (Lyons et al., 1983)). Bipolar Ag/AgCl surface electrodes were placed at an inter-electrode distance of 35 mm. Using the placements utilised in the current work, prior work has been able to distinguish functional differences in muscle activation in the different parts of the erector spinae and abdominal obliques (Lyons et al., 1983; Ng et al., 2001, 2002). Electrodes were also placed over the right biceps femoris muscle to monitor leg muscle activity at rest. A ground electrode was placed at the right elbow. Standardised skin preparation was performed involving washing the skin, shaving and the application of an abrasive-conductive gel.

EMG and goniometer data were sampled simultaneously at 2000 Hz using a Powerlab system running Chart version 4.2 software (AD Instruments, Sydney, Australia) with a 16 bit A/D converter, band-pass filtered from 15 to 500 Hz and were stored for offline processing. During testing, subjects were given real-time visual feedback on movement speed and position. A second computer also sampled the goniometer signal and implemented custom written software in the Labview environment (version 6.1, National Instruments, Texas) to provide this feedback.

2.4. Goniometer signal processing and movement accuracy

Prior to processing EMG data, the goniometer signal, which was sampled simultaneously with the EMG signals, was first processed to select data regions which fulfilled the following criteria: beginning at a minimum nearest 0°, three consecutive movement cycles during which each movement cycle’s speed was within ±5 cyc/min of the target speed, and the maxima (near 45°) and minima (near 0°) were within ±4° of their respective targets. This process was conducted to limit the effect of extremes of performance on the observed motor control patterns and further standardise the experiment. This processing also provided information on movement accuracy: mean-squared-error (MSE) of movement speed (MSEspeed), maxima positions (MSEA5°) and minima positions (MSEA5°). These MSE values were calculated for each data region.

2.5. EMG signal processing

In each selected data region the corresponding EMG signal was extracted and the root-mean-square (RMS) activation level calculated. The median frequency (fM) of the same signal subset was computed (Basmajian and De Luca, 1985). To calculate the timing of LP muscle activation, an algorithm, described in a prior study (Belavy et al., 2006), to quantify the temporal displacement of the peaks and troughs of muscle activity in relation to the movement cycle (phase-lead/lag) was used. This was implemented using frequency domain analyses according to the following algorithm (Fig. 2): (a) a “linear-envelope” from the entire 11 second EMG signal (high pass filtering at 20 Hz using a 10th order digital Butterworth filter, full-wave rectification, and then low-pass filtering at 10 Hz with a 10th order digital Bessel filter) is extracted; (b) this signal is then truncated (along with the goniometer signal) to the data region of interest; (c) the amplitude spectrum of the goniometer signal is calculated and the peak positive value found (movement frequency); (d) the phase spectra (in radians) of the linear-envelope and goniometer signals are calculated; (e) the phase-value (in radians) at the movement frequency of the linear-envelope spectrum is subtracted from the corresponding value of the goniometer phase spectrum; and (f) this value is then coerced between 180° and −180°, giving the timing variable (phase-lead/lag, PHZ) for further analysis (see Belavy et al., 2006 for further details). Using this algorithm, a positive phase-lead/lag indicates that the cyclic bursts (peaks) of muscle activity during repetitive movement lead the goniometer signal. A negative phase-lead/lag indicates the cyclic bursts (peaks) of EMG activity during cyclic movement trail the peaks of the goniometer signal.

The presence of modulation (peaks and troughs) of muscle activity during repetitive movement is a prerequisite for the detection of a reliable phase-lead/lag. Prior work determined which levels of signal modulation are needed to ensure that the detected phase-lead/lag is reliable. A reliable phase-lead/lag can be detected when the peak linear-envelope value is 1.920, 2.011, 2.129 or 2.326 (at the 50, 75, 100 or 125 cyc/min movement speeds) times the trough value of the linear-envelope (Belavy et al., 2006).

2.6. Body composition data

As changes in subcutaneous fat levels may very well influence surface EMG signals (Farina et al., 2004) and it could be anticipated that body fat levels may change during strict inactivity, we also sought to control for any potential influence of changes in subcutaneous fat on the surface EMG signals collected. We chose to use data from parallel measurements of whole body composition (trunk sub-region), rather skin-fold thickness or body-mass index as the former is not considered useful for evaluating variations in EMG signals (Nordander et al., 2003) and the later (BMI), although being useful in EMG signal analysis (Nordander et al., 2003), would also in the current study be affected by changes in lean and fat mass in other body regions. A Delphi W (Hologic, Waltham, MA) system was used to perform total body scans according to the standard Hologic Operator’s Manual 3-days prior to bed-rest (BDC-3), on day BR2, BR17, BR31, BR45 and BR55 of the bed-rest phase and on R+14, R+28, R+90, R+180 and R+360 during recovery phase. Fat mass (in grams) of the trunk (from pelvis to shoulders) were derived from the whole body scan. All scanning and analyses were performed by the same operator to ensure consistency and standard quality control procedures were followed.

2.7. Statistical analysis

For each of the EMG variables (fM, RMS and PHZ), linear-mixed effects models (Pinheiro and Bates, 2000) were used to fit fixed effects for muscle, study-date, movement speed and all interactions up to a three-way interaction between these variables. To assess the relationship between movement accuracy and motor control, each of the movement accuracy variables (MSEspeed, MSEA5° and MSE5°) were included as linear co-variates, as well as in interaction with muscle. To assess the influence of trunk fat levels on the RMS and fM variables, trunk fat mass was also included as linear co-variates in the statistical models for these variables and in interaction with muscle. Where needed to ensure adherence to assumptions of normality, allowances for heterogeneity of variance such as due to muscle and/or movement-speed were permitted. Random effects were modelled for subject, muscle within subject, movement-speed within muscle, study-date within movement-speed and repetition within study-date. A natural-log transformation of the RMS data was applied to approximate normality. As sufficient statistical models do not currently exist to examine changes in “circular variables” (such the phase-lead/lag variable [PHZ] of the current study) across repeated measurements (study-date in the current work), the variable was first converted into its y- and x-components (yPHZ and...
and \( xPHZ \) via basic trigonometric transformation prior to analysis. Subsequent analysis of variance (ANOVA) then evaluated the significance of each of the fixed-effects variables.

Changes in trunk body fat in grams were also assessed with ANOVA using linear-mixed effects models. The BDC-3 and BR2 measurements were used as a “double baseline” to improve assessment of starting fat levels (as detectable changes in body-fat are unlikely to occur on this timeframe). Changes over study-date were assessed.

The “R” statistical environment (version 2.0.1, www.r-project.org) was used to implement analyses. Where necessary, allowances were made for heterogeneity of variance across different grouping levels (such as movement speed or muscle). An \( \alpha \) of 0.05 was taken for statistical significance. As multiple measurement sessions were undertaken on the same subjects, we examined for consistent significant differences across testing days. To examine changes over study-date in the \( PHZ \) variable, where significant changes of the separate \( y \)- and \( x \)-components (with \( F \)-statistics and \( p \)-values reported as \( F_y \) or \( F_x \), and \( p_y \) or \( p_x \), respectively) were observed in ANOVA, the mean change in the \( PHZ \) variable over time and its 95% confidence interval were calculated via trigonometric transformation to permit better statistical inference.

3. Results

Due to subject absence or technical difficulties, not all data from each subject on every scheduled testing day were available. The numbers of subjects able to be included in statistical analysis on each testing day are given in Table 1.

3.1. Changes in trunk fat levels

Mean (SD) baseline trunk fat levels were 7151(2833) g. Significant changes of trunk fat mass (\( F = 4.17, p = .00002 \)) occurred over the study period (Fig. 3). Whilst trunk fat levels are greater in magnitude by the 31st day of bed-rest, strong statistical evidence for these increases is not apparent until the 55th day of bed-rest. Trunk fat levels appear to decrease beyond the 28th day of post-bed-rest recovery (R+28), but remain greater than at baseline up to 1-year after bed-rest (R+360).

3.2. Median frequency

Strong effects existed for differences between muscles and movement speeds for the \( f_m \) variable (muscle: \( F = 25.15, \)
Table 1

Number of subjects available for analysis on each study-date for the lumbo-pelvic motor control and body-composition (DXA) measurements.

<table>
<thead>
<tr>
<th>Study control</th>
<th>Number of subjects</th>
<th>Body composition (DXA)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Study-date</td>
<td></td>
</tr>
<tr>
<td>BR1</td>
<td>8</td>
<td>BDC-3</td>
</tr>
<tr>
<td>BR4</td>
<td>6</td>
<td>BR2</td>
</tr>
<tr>
<td>BR3</td>
<td>8</td>
<td>BR17</td>
</tr>
<tr>
<td>BR27</td>
<td>8</td>
<td>BR31</td>
</tr>
<tr>
<td>BR41</td>
<td>8</td>
<td>BR45</td>
</tr>
<tr>
<td>BR53</td>
<td>10</td>
<td>BR55</td>
</tr>
<tr>
<td>R+14</td>
<td>9</td>
<td>R+14</td>
</tr>
<tr>
<td>R+28</td>
<td>9</td>
<td>R+28</td>
</tr>
<tr>
<td>R+90</td>
<td>8</td>
<td>R+90</td>
</tr>
<tr>
<td>R+180</td>
<td>6</td>
<td>R+180</td>
</tr>
<tr>
<td>R+360</td>
<td>4</td>
<td>R+360</td>
</tr>
</tbody>
</table>

p < .0001; speed: F = 41.64, p < .0001; muscle × speed: F = 2.08, p = .022. Table 2 presents the baseline (BR1) f_m values for each muscle and speed. The movement accuracy MSE variables bore little statistical relation to the f_m variable (MSE_speed: F = 25.68, MSE_speed × muscle: F = 4.97, p = .0001 and t = .211, respectively). For both EO (t = -.143, p = .886) and IGM (t = -1.01, p = .314) a non-significant negative relationship between trunk fat levels and f_m existed. For TES the relationship was positive but non-significant (t = 1.25, p = .211). Overall, these results suggest that a decrease in f_m can be explained by increased trunk fat mass, but that any increase in f_m over study-date cannot.

Significant changes in f_m values occurred over study-date (F = 7.44, p < .0001) and the different muscles also responded differently over study-date (study-date × muscle: F = 4.59, p < .0001). Movement speed, however, did not influence the changes over study-date (study-date × speed: F = 1.3, p = .103; study-date × muscle × speed: F = .81, p = .933). Fig. 4a and b shows the changes in f_m for each muscle over study-date averaged across all movement speeds. The f_m of the IO and IGM muscles is decreased from early- to mid-bed-rest and in IO continues to be decreased up to 1-year afterwards. The EO and TES muscles show little change in f_m. Interestingly, and in contrast to TES and all other muscles, the LES muscle group shows a strong decrease of f_m during the first weeks of bed-rest, but at BR53 it is the only muscle with an increased f_m, which continues to be so up to 1-year after bed-rest.

Given the relationship between trunk fat mass and the f_m variable and the increases in trunk fat over time, the decreased f_m of the IO and IGM muscles, as well as the LES muscle during the initial phases of bed-rest, may be explained by changes in trunk fat. This, however, cannot explain the increase f_m of the LES muscle group beyond BR53.

3.3. Root mean square muscle activity levels

A similar pattern as for f_m was seen for RMS. As could be expected, strong statistical evidence existed for differences between muscles and movement speeds (muscle: F = 23.74, p < .0001; speed: F = 358.98, p < .0001; muscle × speed: F = 4.48, p < .0001). Table 2 shows the baseline (BR1) RMS values for each muscle and speed. Similar to the f_m variable, no evidence existed for a relationship between movement accuracy and the RMS variable (MSE_speed: F = 1.24, MSE_speed × muscle: F = 4.65, p = .0001 and t = .314) indicating any changes in movement accuracy did not influence RMS values. Trunk fat mass influenced RMS values (F = 25.68, p < .0001) and between between muscle (fat mass × muscle: F = 6.50, p < .0001). For both EO and IO, a significant negative relationship (decreasing RMS with increasing fat levels) existed (t = -4.65, p < .0001 and t = -4.97, p < .0001, respectively). For the remaining muscles (IGM, LES, TES), non-significant negative relationships existed (t all < -10, p all > .499) indicating that any decreases in RMS over study-date could potentially fully or in part result from increases in fat levels.

![Fig. 3. Percentage changes in trunk fat mass over the study period. Error bars represent standard error of the mean difference to baseline values. ⊕: p < .01; ⊕: p < .001. BR = bed-rest; R = recovery.](image-url)
Strong effects existed for changes in RMS values over study-date ($F = 6.43, p < .0001$) and also for different responses of the muscles over time (study-date/C2 muscle: $F = 4.63, p < .0001$). Although RMS values at each movement speed behaved differently over time (study-date/C2 speed: $F = 1.73, p = .0085$), this did not impact upon the effects on each muscle (study-date/C2 muscle/C2 speed: $F = 0.67, p = 0.997$).

Fig. 5a and b reports the changes in RMS values for each muscle over time. Strong decreases in the raw activity levels detected at the skin surface of the IO, EO and IGM muscles occur during bed-rest. These decreases persist up to 1-year after bed-rest. Marginal increases in the activation levels of the LES muscle group occurs over time and even less change in the TES muscle.

Table 2

<table>
<thead>
<tr>
<th>Movement Speed</th>
<th>Muscle</th>
<th>Median frequency ($f_m$, Hz)</th>
<th>Root-mean-square (RMS) activation level (mV)</th>
<th>Activation timing (phase-lead/lag; PHZ) relative to goniometer signal (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>IO</td>
<td>EO</td>
<td>LES</td>
</tr>
<tr>
<td>50</td>
<td></td>
<td>70.2(29.4)</td>
<td>36.9(8.8)</td>
<td>77.4(30.8)</td>
</tr>
<tr>
<td>75</td>
<td></td>
<td>71.1(26.3)</td>
<td>39.7(8.9)</td>
<td>91.6(32.3)</td>
</tr>
<tr>
<td>100</td>
<td></td>
<td>84.0(31.5)</td>
<td>44.9(7.5)</td>
<td>95.4(22.6)</td>
</tr>
<tr>
<td>125</td>
<td></td>
<td>91.7(34.3)</td>
<td>46.7(3.8)</td>
<td>102.7(20.8)</td>
</tr>
</tbody>
</table>

Table 2: Baseline (BR1) values of each lumbo-pelvic muscle electromyographic variable.

Values are mean (SD). EO: external oblique, IO: internal oblique, IGM: inferior gluteus maximus, TES: thoracic erector spinae, LES: lumbar erector spinae. Movement speed is in cycles of knee flexion-extension per minute. A negative phase-lead/lag indicates that the cyclic bursts (peaks) of EMG activity are trailing the cyclic movement signal. A positive phase-lead/lag indicates cyclic bursts (peaks) of muscle activity lead the cyclic movement signal. See text for further details.

Fig. 4. (a) and (b) Changes in electromyographic median frequency ($f_m$) of the lumbo-pelvic muscles over the study-course in Hz. Error bars represent standard error of the mean difference to baseline (BR1) values. Positive values indicate increased median frequency compared to baseline testing. : $p < 0.05$; †: $p < 0.01$; ‡: $p < 0.001$. BR = bed-rest, R+ = recovery. EO: external oblique, IO: internal oblique, IGM: inferior gluteus maximus, TES: thoracic erector spinae, LES: lumbar erector spinae. Movement speed did not influence changes of each muscle over study-date ($F = .81, p = .933$), therefore results are averaged across movement speeds.
increases in trunk fat over time, the decreased RMS of the EO, IO and IGM muscles may be explained by changes in trunk fat. Interestingly, the limited change, or even marginal increase, in RMS of the LES and TES muscles occurs independently of the increases in trunk fat mass.

3.4. Timing of muscle activity: phase-lead/lag

As could be expected, differences existed between muscles and movement speeds for activation timing (muscle: $F_Y = 21.19$, $p_x < .0001$, $F_x = 22.76$, $p_x < .0001$; speed: $F_Y = 1.84$, $p_x = .142$, $F_x = 1.30$, $p_x = .279$; muscle $\times$ speed: $F_Y = 9.36$, $p_x < .0001$, $F_x = 10.18$, $p_x < .0001$). Table 2 shows the baseline (BR1) PHZ values for each muscle and speed. Similar to the other EMG variables, no evidence existed for a relationship between muscle activation timing and any of the movement accuracy variables, including in interaction with muscle ($F_Y$ all $< 2.10$, $p_x$ all $> .078$; $F_x < 1.97$, $p_x$ all $> .161$). Strong effects existed, however, for changes in PHZ values occurring over study-date ($F_Y = 3.36$, $p_x < .0001$, $F_x = 3.85$, $p_x < .0001$) but interestingly, interactions with muscle and speed were non-significant ($F_Y$ all $< 1.19$, $p_x$ all $> .174$; $F_x < 1.10$, $p_x$ all $> .299$) indicating that any changes in activation timing were generalised across all muscles and movement speeds.

### Table 3

<table>
<thead>
<tr>
<th>Study-date</th>
<th>Difference in timing</th>
<th>95% Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean % Degrees ($)</td>
<td></td>
</tr>
<tr>
<td>BR4</td>
<td>-2.5</td>
<td>(-94.6$-$76.7$^\circ$)</td>
</tr>
<tr>
<td>BR13</td>
<td>-4.6</td>
<td>(-102.1$-$69.2$^\circ$)</td>
</tr>
<tr>
<td>BR27</td>
<td>-5.4</td>
<td>(-105.3$-$66.1$^\circ$)</td>
</tr>
<tr>
<td>BR41</td>
<td>-8.9</td>
<td>(-117.9$-$53.5$^\circ$)</td>
</tr>
<tr>
<td>BR53</td>
<td>-7.8</td>
<td>(-113.9$-$57.4$^\circ$)</td>
</tr>
<tr>
<td>R+14</td>
<td>-1.3</td>
<td>(-90.2$-$81.2$^\circ$)</td>
</tr>
<tr>
<td>R+28</td>
<td>-4.5</td>
<td>(-101.9$-$69.4$^\circ$)</td>
</tr>
<tr>
<td>R+90</td>
<td>-4.0</td>
<td>(-100.1$-$71.4$^\circ$)</td>
</tr>
<tr>
<td>R+180</td>
<td>2.3</td>
<td>(-77.4$-$94.1$^\circ$)</td>
</tr>
<tr>
<td>R+360</td>
<td>-2.0</td>
<td>(-92.8$-$78.8$^\circ$)</td>
</tr>
</tbody>
</table>

% implies percentage of a movement cycle, one movement cycle comprises 360°, 95% confidence interval of mean changes (in degrees) compared to baseline (BR1) values are reported. BR = bed-rest, R+ = recovery. No significant difference existed between muscles; therefore results represent pooled data for all muscles. A negative change in phase-lead/lag indicates that the cyclic bursts (peaks) of EMG activity trailed the temporal position of bursts/troughs of muscle activation during repetitive movement at baseline testing (i.e. shifted to left when viewed in time series). A positive value indicates that the bursts of EMG activity led the temporal position at baseline testing (i.e. shifted to right when viewed in time series). See text for further details.

Table 3 reports the mean change in activation timing over study-date (transformed into the original units of degrees and subsequently percentage of a movement cycle) with corresponding 95% confidence intervals. Although the analysis of the individual $y$- and $x$-components of the circular timing variable suggested a generalised change in LP muscle activation timing, the mean change, when considered in the original angular units, was small. Also, the 95% confidence intervals of the mean change over time (in degrees) crossed 0° at every time point. This suggests, in contrast to the analysis of the individual $y$- and $x$-components, which alone have little physiological meaning, that activation timing of the LP musculature was actually relatively stable over the course of the study.

### 4. Discussion

This study was the first to conduct a detailed analysis of the effect of prolonged bed-rest on superficial LP muscle motor control as investigated by spectral analysis and temporal activation. The most striking finding was the development of increased median frequency of the lumbar erector spinae late in bed-rest and the persistence of this change up to 1-year after bed-rest. This change cannot be explained by alterations of trunk body fat levels or by changes in motor skill. Contrastingly, the synergistic thoracic erector spinae did not show these changes. Strong effects for decreases in median frequency (and raw activation levels) were seen in the abdominal and gluteal musculature, but it cannot be excluded that this is due to increases in local fat levels, generating a low-pass filtering effect on the EMG signals (de Luca, 1997; Farina et al., 2004). Another obvious cause of a decreasing $f_{\text{NL}}$ during bed-rest is muscular atrophy, decreasing muscle fibre cross-section and thus action potential conduction velocity (Blijham et al., 2006), and consequently EMG median frequency of the EMG signal (Mulder et al., 2009). Another important observation was the absence of a change in the temporal activation patterns of the superficial LP muscle activation during or after bed-rest.

The stability of the temporal activation of the superficial LP musculature may be surprising considering the myriad of effects that microgravity and simulation has on motor control. The finding...
is, however, consistent with the findings of other studies. A number of studies on astronauts during and/or after spaceflight and also on parabolic flight subjects have found little evidence for either a shift to earlier activation, or of a delay of activation, in the leg musculature (Clément et al., 1984, 1985; Layne et al., 1997, 1998, 2001; Mouchonno et al., 1996). Coupled with the findings of the current study, the findings overall seem to suggest that the motor programme for the temporal sequencing of muscle activation is quite stable in microgravity and simulation, even though other motor control characteristics (activation level, even a part of the f_m changes) may be altered. One consideration, however, before drawing general conclusions on the temporal activation of all muscles in microgravity and simulation is that all studies to date have used surface EMG. Typically, the activation timing of muscles that are more intimately involved in fine joint movements, tend to require fine wire EMG, rather than muscles involved in gross postural adjustments which can be readily observed with surface EMG. The activation timing of these less accessible muscles can be more sensitive in “dysfunction” (see, for example, research on transversus abdominis in low back pain, experimental pain and stress; Hodges and Moseley, 2003). Further work, utilising fine-wire EMG would be an appropriate next step in evaluating the effects of microgravity and simulation on the temporal activation of the LP musculature.

Perhaps the most interesting findings of the current work are the changes in median frequency observed in the erector spinae. An increased electromyographic median frequency was observed in the lumbar erector spinae during and up to one-year after bed-rest, but not in the thoracic erector spinae. It is interesting to note that magnetic resonance imaging findings from the same subjects found the greatest losses in cross-sectional area during bed-rest in the more medially (lumbar) components of the paravertebral muscles (Belavy et al., 2008; Hides et al., 2007). Functionally, the lumbar erector spinae has a predominant role in directly controlling the lumbar spine (Bergmark, 1989), controlling the lumbar lordosis (Kiefer et al., 1997; MacIntosh and Bogduk, 1986) and providing the overwhelming majority of lumbar spine stiffness (Kiefer et al., 1998; Wilke et al., 1995). In prolonged bed-rest, the necessity of this role is reduced and this may underlie the relatively greater susceptibility of the lumbar erector spinae in bed-rest. The persistence of these changes long-term after bed-rest may suggest a stable change in motor control due to bed-rest. The physiological or functional implications of the increased median frequency are, however, more difficult to elucidate. The increases in median frequency may be associated with changes in muscle fibre type (Farina et al., 2004; Gordon and Pattullo, 1993) or with the influence of changes in motor unit firing rates on the muscle fibre conduction velocity (Mihelin et al., 1991) and consequently on the median frequency. Further work is necessary to understand any potential link.

Changes in movement accuracy appear unlikely to be involved in the EMG changes observed in the erector spinæ. Whilst subjects’ overall performance of the movement task did improve (Belavy et al., 2007b) an algorithm was used to exclude sections of data with extremes of movement performance. The resulting accuracy of movement in the subset of data examined changed little over the course of the study (data published in Belavy et al., 2007a). Moreover, the results of the current study showed no association between movement accuracy and median frequency or EMG amplitude. Some authors have concluded that changes in EMG signal characteristics with motor learning are individual dependent (Carson and Riek, 2001), though others have noted skill acquisition to be associated with decreases in EMG amplitude (Gribble et al., 2003) and median frequency (Bernardi et al., 1996). In the context of the current study, improvements in movement skill could certainly not explain the increase in median frequency of the lumbar erector spinæ, after an initial decrease in the first few weeks of bed-rest, late in bed-rest and in recovery.

There are some further limitations of the current study. The DXA measurement provided information on the fat mass in the trunk. This data does not necessarily equate to the thickness of the fat layer between the electrodes and the muscle of interest. We were, however, primarily interested in the changes in the amount of subcutaneous fat and its potential influence on the EMG signals. Of skin-fold thickness, direct measurements muscle-electrode distance with ultrasound and BMI, BMI is surprisingly the best at explaining variation in EMG amplitude between individuals (Nordander et al., 2003). We argue that DXA would be a more appropriate measure than BMI in the context of the current study as DXA focuses on fat changes in the trunk itself, whereas BMI, in the current study, would be strongly influenced by changes in fat and lean mass in the legs. Another issue is that it was not possible to conduct fatiguing contractions of the trunk muscles during bed-rest, which could then have been used in further spectral analysis to provide more information on electromyographic fatigue. Such physical activity would, of course, have been inherently inappropriate during a bed-rest study.

In conclusion, this study was the first to examine the effect of prolonged bed-rest on the motor control of the superficial LP musculature in spectral and temporal domain analyses. The main finding was of an increased median frequency of activation in the lumbar erector spinæ which began late in bed-rest and persisted up to 1-year afterwards, but with no change in its synergist the thoracic erector spinæ. This may suggest incomplete recovery after bed-rest. The observed spectral and EMG amplitude changes in the abdominal and gluteal musculature were likely due to changes in body composition. Interestingly, no change was observed in the temporal activation of the superficial LP muscles examined suggesting stability of this aspect of the motor programme during bed-rest.

Conflict of interest statement

None declared.

Acknowledgments

The authors wish to thank Mr. Benny Elmann-Larsen of the European Space Agency, the subjects who participated in the study, and the staff of ward 18A in the Charité Campus Benjamin Franklin Hospital, Berlin, Germany. Björn Bühring is also thanked for his assistance. The Berlin Bed-Rest Study was also sponsored by the Humboldt Foundation.

References


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