ABSTRACT: Force fluctuations in leg muscles increase after bedrest, perhaps due to modulation of the neural strategy that is specific to a muscle or common to agonist muscles. The purpose of this study was to examine the modulation of muscle activity and force fluctuations during steady contractions with variable involvement of plantarflexor muscles after bedrest at knee-flexed (FLX) and extended (EXT) positions. Before and after 20-day bedrest, plantarflexion force and surface electromyogram (EMG) in the medial gastrocnemius (MG), lateral gastrocnemius, and soleus muscles were measured during steady isometric contractions in five young men. In EXT, power ≤10 Hz in the rectified EMG of MG increased significantly after bedrest. This low-frequency modulation of muscle activity in MG accompanied a 29% increase in the standard deviation of force. There was no change in EMG in other muscles. In FLX, there was no adjustment in EMG or force fluctuations. These results suggest that low-frequency modulation of MG plays a role in increasing force fluctuations during steady plantarflexion in EXT after bedrest. The findings indicate task/muscle specificity in the modulation of neural strategy during steady contractions after bedrest and underscore the importance of designing a specific training regimen targeted to particular tasks/muscles with regard to force fluctuations in multiple-agonist systems.

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MODULATION OF MUSCLE ACTIVITY AND FORCE FLUCTUATIONS IN THE PLANTARFLEXORS AFTER BEDREST DEPENDS ON KNEE POSITION

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The ability to control precise motor output is important in daily activities as well as in occupations that involve manipulation of objects. Compared with young adults, old adults often exhibit greater fluctuations in motor output,[9,11,18,30,32–34] which probably contribute to impaired control of precise motor output in old adults. However, fluctuations in motor output in old adults can be reduced by training,[15,17,19] indicating the role of habitual activity on fluctuations in motor output. In a single-agonist model, a reduction in the discharge-rate variability of motor units after training can partially explain a reduction in force fluctuations,[17] although other unknown factors may be involved. In contrast, the physiological mechanisms responsible for the adaptations in fluctuations in motor output are unknown for contractions that involve multiple-agonist muscles.

In leg muscles, force fluctuations increase due to reduced habitual activity during 20-day bedrest in healthy young adults.[29] In plantarflexor muscles, an increase in force fluctuations accompanies a greater increase in the average amplitude of surface electromyogram (EMG) in the medial gastrocnemius (MG) muscle than in the soleus (SOL).[29] This implies that activity of a specific muscle may contribute to the adaptation of force fluctuations in multiple-agonist systems. When the involvement of the specific mus-

Abbreviations: aEMG, root-mean-square amplitude of electromyographic activity; ANOVA, analysis of variance; CV, coefficient of variation; EMG, electromyography; EXT, knee-extended position; FLX, knee-flexed position; LG, lateral gastrocnemius muscle; MG, medial gastrocnemius muscle; MVC, maximal voluntary contraction; SOL, soleus muscle

Key words: bedrest; muscle length; plantarflexion; steadiness

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cle is minimized, therefore, modulation of the neural activation strategy across agonist muscles may not be substantial. Hence, we hypothesized that fluctuations in net force do not increase after bedrest when the involvement of the MG is minimized by shortening its muscle length (first hypothesis).

Mechanisms other than the altered distribution of muscle activity may contribute to the adaptation of force fluctuations in multiple-agonist systems, but further insight cannot be obtained if based solely on the analysis of average EMG amplitude. Since fluctuations in net force result from the temporal summation of force fluctuations from individual muscles, it is possible that alterations in temporal characteristics of individual muscle forces modify interactions of individual muscle forces and thereby fluctuations in net force. This possibility cannot be examined directly in humans because individual muscle forces cannot be measured. Hence, to approach this potential mechanism, other indirect means need to be utilized that are associated with alterations in the temporal characteristics of individual muscle force.

Since the temporal characteristics of individual muscle force can be altered by frequency modulation of neural activity to a pool of motor units, determination of the frequency content of the rectified EMG[5,22,30,33] is a useful approach that assesses the global frequency modulation of neural activity at the whole-muscle level. For example, an adaptation in neural activity with age has been demonstrated by an increase in the frequency content around 5–15 Hz in the rectified EMG that was associated with an increase in force fluctuations in a hand muscle.[33] Therefore, we hypothesized that an alteration in the temporal characteristics of individual forces attributable to frequency modulation of neural activity may be involved in the adaptation of fluctuations in net force after bedrest (second hypothesis).

This study was performed to test whether adaptations in force fluctuations in multiple-agonist systems are due to alterations in the neural activation strategy in a specific muscle with regard to magnitude and frequency. To test the first hypothesis, subjects performed steady contractions of plantarflexor muscles before and after bedrest with distinct knee positions to vary the contribution of the biarticular gastrocnemius muscles to the plantarflexion force. To test the second hypothesis, alterations in frequency content of the rectified EMG after bedrest were examined. In addition, the data in a previous experiment on bedrest with and without strength training[29] were examined for comparison.

Prolonged bedrest has been used not only as an experimental model of inactivity, but also as an experimental model of spaceflight in humans.[8] Hence, the findings from this study may contribute to the understanding of impaired motor output after spaceflight. Furthermore, our findings may shed light on physiological mechanisms for adaptations in force fluctuations in multiple-agonist models in general.

METHODS

Five healthy men voluntarily participated in this study. The subjects did not have any history or physical signs of neuromuscular disorder. Their mean (± SD) age, height, and body mass were 21.6 ± 1.3 years, 174.6 ± 9.6 cm, and 64.6 ± 5.3 kg, respectively. They gave informed consent to participate and the procedures were approved by the ethics committee of the University of Tokyo (Japan).

Experimental Protocol. To examine the effect of activation of the gastrocnemius muscle on force fluctuations before and after bedrest for 20 days, subjects performed a maximal voluntary contraction (MVC) task and submaximal steady contraction task of isometric plantarflexion at two knee joint angles. The procedure was the same as previously employed by our group.[28,29,37]

Plantarflexion force by the dominant (right) leg was measured with a custom-made force-measurement device. A strain-gauge transducer (LTZ-200KA; Kyowa, Tokyo, Japan; 0.013 V/N) was fixed to the device between a base metal plate and a foot lever plate near the distal part of the foot. The exact position of the foot and device was carefully adjusted so that the ankle joint was flexed at 90° and the axes of the ankle and footplate were aligned in each subject. The bottom end of the foot lever plate had a semicircular attachment that surrounded and secured the heel. Plantarflexion force was amplified, and low-pass filtered (<100 Hz) by a DC amplifier (DPM 700; Kyowa, Tokyo, Japan).

To alter the muscle length of the gastrocnemius muscle, the knee joint angle was manipulated. In the knee-extended position (EXT), the subject lay in a prone position on a padded bed with the knee fully extended. In the knee-flexed position (FLX), the subject rested the anterior trunk and arms on a box with the knee and hip joint angles at 90°.[16,28] The muscle length of the gastrocnemius muscles was thus shortened in FLX compared with EXT.

MVC Task. Subjects performed an MVC task before and after bedrest with the knee in FLX and

EXT positions. The MVC task involved a gradual increase in plantarflexion force exerted by the triceps surae muscles from baseline to maximum in 3–4 s, with the contraction then sustained at maximum for 2 s. The plantarflexion force was displayed in real time on an oscilloscope. The timing of each trial was based on a verbal count given at 1-s intervals, with vigorous encouragement from the investigators when the force began to plateau. Each subject performed a minimum of three MVC trials, with subsequent trials performed if the differences in the peak force of two MVCs were >5%. The interval between MVC trials was at least 3 min. Subjects were allowed to reject any effort that they did not regard as maximal. The trial with the highest peak force was selected for analysis. The order of the knee joint angles was randomized across subjects.

Steady Contraction Task. Before and after the bedrest period, subjects performed a steady contraction task in two knee positions: FLX and EXT. Subjects were asked to contract the plantarflexor muscles and to maintain plantarflexion force about the target level as steady as possible for 20 s with visual feedback. The target and exerted forces were represented as horizontal lines on the oscilloscope. The target forces were 2.5%, 5.0%, and 10.0% of MVC force that was measured before bedrest in the corresponding knee position. Hence, the target forces were identical before and after bedrest as in our previous study on bedrest,[29] with which the data were compared. These low target forces were chosen according to our previous studies on force fluctuations in plantarflexor muscles.[28,29,37] Practice for the steady contraction task was performed for several seconds at each target force. The data were collected for one trial at each target, and the order of the target force was randomized across subjects.

Bedrest. Subjects participated in a bedrest intervention for 20 days according to our previous study.[29]

The subject lay on the bed and remained in a 0.1-rad (6°) head-down position to simulate a microgravity environment.[8] Subjects were allowed to leave the bed only when being transported to the bathroom via a transporting bed. In addition, subjects were not allowed to exert unnecessary forces by the lower limbs. Hence, muscle activity in the lower limbs was minimized. Subjects were permitted to use their arms for such activities as reading, writing, and eating, but no vigorous activity was allowed in their upper limbs. The nursing staff supervised all subjects for 24 hours throughout the bedrest period.

Measurements and Analysis. EMG was recorded from the MG, lateral gastrocnemius muscle (LG), and SOL with bipolar Ag–AgCl electrodes (diameter, 8 mm; interelectrode distance, 20 mm). The electrodes were connected to a preamplifier (×20) and a differential amplifier (×50) having a bandwidth of 5 Hz to 1 kHz (1253A; NEC Medical Systems, Tokyo, Japan). Electrodes were attached over the muscle belly along the direction of fascicles of each muscle by ultrasound B-mode images to minimize potential cross-talk. The electrodes were attached at the same location before and after bedrest by using permanent-ink marks.

The force and EMG signals were collected at a sampling rate of 2 kHz by a 16-bit analog-to-digital converter (Power-Lab/16sp; ADInstruments, Sydney, Australia) and stored on a personal computer. After we confirmed that there was no power beyond 500 Hz in force and raw EMG signals, the data were resampled at 1 kHz to accommodate the limitation in the data analysis software (TransEra HTBasic; i²net, Tokyo, Japan).

In the MVC task, root-mean-square amplitude of EMG (aEMG) was determined over a 300-ms window centered with the time at which peak force was attained. In the steady-contraction task, the middle 16 s of the contraction was used for further analysis because there was no systematic change in fluctuations within trials. Then, the mean, standard deviation (SD), and coefficient of variation (CV; SD divided by the mean) of force were calculated. The SD and CV of force were calculated after removing a continuous linear trend that was detected by a least-square method. The aEMG during the steady-contraction task was normalized to the maximal aEMG across both knee positions and across before and after bedrest.

To quantify the frequency content of force, power spectral density of the force signal was calculated by the fast Fourier transformation (FFT) method (16,384 points, Hamming window, 0.061 Hz/bin) after eliminating the linear trend.[28,37] It is known that force fluctuations during a steady contraction of <20% MVC are composed predominantly of a signal frequency content of ≤15 Hz.[31,34,37] We have also confirmed in a pilot study that a power of >15 Hz was negligible (<0.2% of the total power) for the plantarflexion force ≤10% MVC. The mean power across the 1-Hz window (16 or 17 bins) was further calculated up to 15 Hz for statistical comparisons.

In addition, the power spectral density of the rectified EMG was calculated by the FFT method (16,384 points, Hamming window, 0.061 Hz/bin) for each muscle. The mean power across 5-Hz windows (81 bins) was further calculated up to 50 Hz for

statistical comparisons. Rectification of the EMG signal is a strategy that has been used to represent the temporal pattern of grouped motor-unit discharges.[10,23] The low-frequency content of the rectified EMG was examined because a global temporal modulation in the neural activation of a muscle is primarily reflected in the modulation of the EMG envelope, and the envelope of a signal is reflected in the low-frequency content of its rectified signal.

Analysis of the Previous EMG Data. EMG data from the previous bedrest study[29] were used for further analysis. In this study, 12 young healthy adults underwent a 20-day bedrest and were divided into non-training (n = 6) and strength-training (n = 6) groups. Steady contractions of the plantarflexor muscles (2.5–10% MVC) were performed before and after bedrest only in the knee-extended position. After bedrest, the increase in force fluctuations was significantly less in the training group (40% increase) than non-training group (88% increase) during plantarflexion in EXT. Surface EMG was recorded by the same method as in the current experiment, but only from the SOL and MG. The frequency content of the rectified EMG was obtained using the current method.

Statistical Analysis. MVC force was tested using a two-way analysis of variance (ANOVA; 2 angles × 2 times) with repeated measures. The aEMG during the MVC task was tested using a three-way ANOVA (3 muscles × 2 angles × 2 times) with repeated measures. The aEMG during the steady contraction task was tested using a four-way ANOVA (3 muscles × 2 angles × 3 intensities × 2 times) with repeated measures. The SD of force and the CV of force were compared with a three-factor ANOVA (3 intensities × 2 angles × 2 times) with repeated measures. The power spectral density of force and rectified EMG was compared with a three-way ANOVA (3 or 4 intensities × 2 times × 15 frequency bins) with repeated measures. An alpha level of 0.05 was used for all statistical comparisons, with post hoc comparisons (t-tests with Bonferroni correction) when appropriate. Significance was accepted at $P < 0.05$ or $P < 0.01$. Unless stated otherwise, all values are expressed as mean ± SE in the figures and as means ± SD in the text.

RESULTS

Peak Force and aEMG during MVC. When the main effect of knee positions was examined across periods, MVC force was greater ($P < 0.01$) in EXT than FLX (Fig. 1). The greater MVC force in EXT accompanied greater aEMG in both heads of the gastrocnemius muscle ($P < 0.05$) in EXT than FLX. After bedrest, MVC force decreased in EXT, but not in FLX, for all subjects with significant differences in group data between before and after bedrest (by 7%; $P < 0.05$). As a result, the difference between knee positions was 27% and 21% before and after bedrest. The decrease in MVC force in EXT was accompanied by a 10% decrease in aEMG in the MG ($P < 0.05$) and a 14% decrease in the LG ($P < 0.05$) after bedrest. In FLX, no change occurred in aEMG. The difference in aEMG between knee positions was 45% and 40% before and after bedrest for the MG ($P < 0.01$), and 39% and 30% before and after bedrest for the LG ($P < 0.01$), respectively. There was no difference between knee positions or change after bedrest for the SOL.

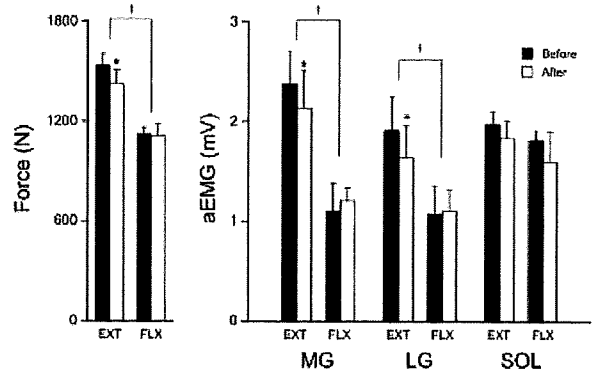

FIGURE 1. Peak force and the amplitude of electromyogram (aEMG) during a maximal voluntary contraction (MVC) at different knee joint angles before (solid bars) and after (open bars) bedrest. The knee joint angles were full extension (EXT) and flexed at 90° (FLX). MG, medial gastrocnemius; LG, lateral gastrocnemius; SOL, soleus. $^{*}P < 0.05$ before and after bedrest; $^{\dagger}P < 0.01$ between knee positions in the corresponding period.

Force Fluctuations and aEMG During Steady Contractions. The aEMG during the steady contraction task increased with force in both knee positions (Fig. 2). When collapsed across force and time, aEMGs were less in FLX for the MG (1.37 ± 0.52%) and LG (0.79 ± 0.35%) than the SOL (5.26 ± 3.84%; $P < 0.01$ for both). Compared with FLX, aEMGs in the MG (9.21 ± 7.57%) and LG (2.73 ± 1.65%) were greater in EXT ($P < 0.05$ for both), whereas there was no difference in the SOL (7.02 ± 4.13%). After bedrest, aEMG in the MG increased by 114% in EXT. When collapsed across target force, aEMG in the MG was 5.87 ± 7.52% before bedrest and 12.55 ± 11.25% thereafter ($P < 0.05$). This increase

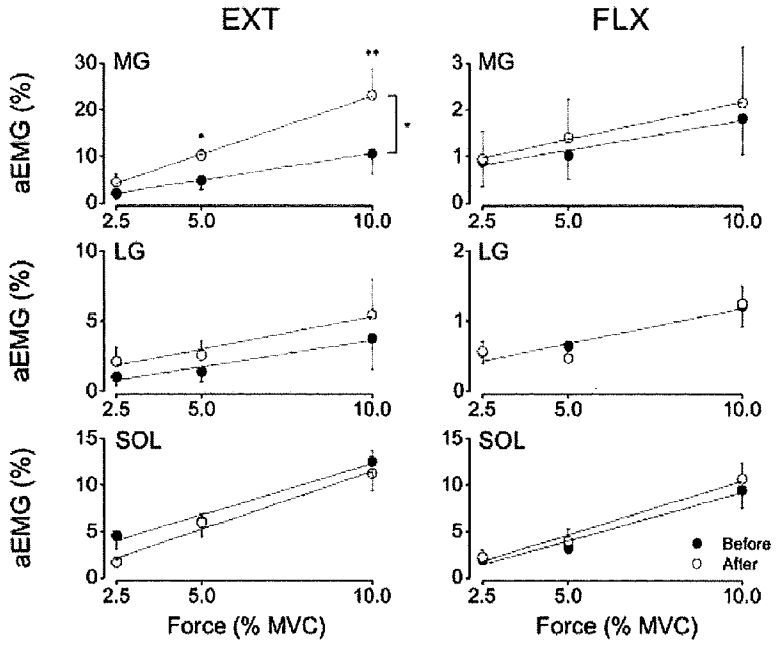

FIGURE 2. aEMG during the steady contraction task in different knee positions before (filled circles) and after (open circles) bedrest. Data are normalized to the maximal value across knee positions attained during the MVC task before bedrest. $^{*}P < 0.05$, $^{**}P < 0.01$ before and after bedrest.

was mainly due to the significant increases at 5% ($P < 0.05$) and 10% MVC ($P < 0.01$). The aEMG in the LG tended to increase by 65%, on average, but this increase was not statistically significant. There was no change in the SOL in either position.

The exerted force during the steady contraction task fluctuated about an average value (Fig. 3, top). In the grouped data, the SD of force (absolute fluctuations) increased with target force, whereas the coefficient of variation of force (relative fluctuations) decreased with force. When collapsed before and after bedrest, the coefficient of variation of force was smaller in EXT (1.62 ± 0.52%) than FLX (2.15 ± 0.90%, $P < 0.05$).

Similar to the adjustments in aEMG after bedrest, significant changes in force fluctuations were found only in EXT (Fig. 4). When collapsed across target forces, the SD of force in EXT increased by 29% ($P < 0.05$) from 1.29 ± 0.63 N before bedrest to 1.66 ± 0.86 N thereafter. Similarly, the CV of force in EXT increased significantly ($P < 0.05$) from 1.62 ± 0.52% to 2.10 ± 0.78%. These changes were not influenced by target force.

Frequency Content of Force and Rectified EMG during Steady Contractions. The major frequency for the force fluctuations during the steady contraction task was <5 Hz, and there was no significant difference in power distribution between knee positions before bedrest (Fig. 5). After bedrest, the power of force in EXT increased by 39% at <1-Hz bin ($P < 0.05$), by 43% at 1–2-Hz bin ($P < 0.05$), and by 167% at 2–3-Hz bin ($P < 0.05$), when collapsed across target forces. Although the power increased at higher frequencies around 3–10 Hz on average, the increase in this higher frequency range did not reach statistical significance. In FLX, there was no change across the frequency range.

Similarly, a significant alteration in the frequency content of rectified EMG was observed only in EXT (Fig. 6). After bedrest, the power in the MG in EXT increased by 659% from 0.004 to 0.033 mV^2 at <5 Hz ($P < 0.05$) and by 151% from 0.080 to 0.201 mV^2 at 5–10 Hz ($P < 0.01$). There was no change in these variables in the LG or SOL in EXT, or in any muscles in FLX.

Additional analyses of the EMG data in our previous bedrest experiment[20] yielded qualitatively similar results to the current EMG data. In that previous experiment, steady plantarflexions at low levels (2.5%–10% MVC) were performed only in EXT, and the increase in force fluctuations was less in the training group (bedrest plus strength training) than the non-training (bedrest only) group. Similar to the

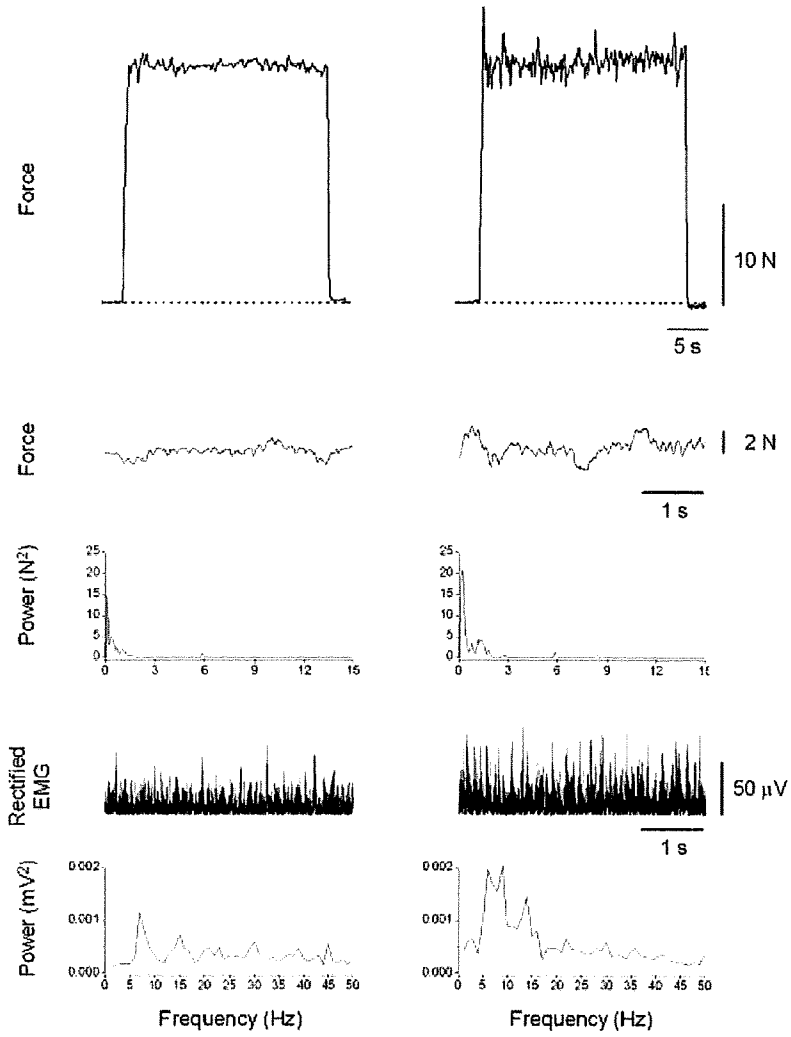

FIGURE 3. Representative recordings from a subject performing the steady contraction task in EXT before (left) and after (right) bedrest. Force (top), force fluctuations about the mean (second), power spectral density of force (third), rectified EMG in the MG (fourth), and power spectral density of the rectified EMG (bottom) are presented.

current experiment, power of the rectified EMG in the non-training group increased in the low-frequency range (≤15 Hz) only in the MG (Fig. 7). Consistent with the attenuated increase in force fluctuations, this modulation in the rectified EMG in the MG was attenuated significantly ($P < 0.05$) in the strength-training group.

DISCUSSION

We found that force fluctuations did not increase in the knee-flexed position, in which the involvement of the medial gastrocnemius muscle was minimized, thereby supporting our first hypothesis. In support of the second hypothesis, low-frequency modulation of EMG was observed in the medial gastrocnemius muscle only in the knee-extended position, in which force fluctuations increased.

Muscle Activity and Force Fluctuations in Different Knee Positions. The greater aEMG in the MG in EXT than FLX confirmed that involvement of MG in EXT was greater than FLX in the present experimental design. This difference is due to reduced fascicle length, increased pennation angle, and reduced motor neuron excitability in the MG in FLX.[12,16] In association with greater involvement of the MG, reduced force fluctuations in EXT were evidenced by the smaller SD and CV of force in EXT than FLX. This finding accords with our previous study,[28] where it was suggested that muscle force

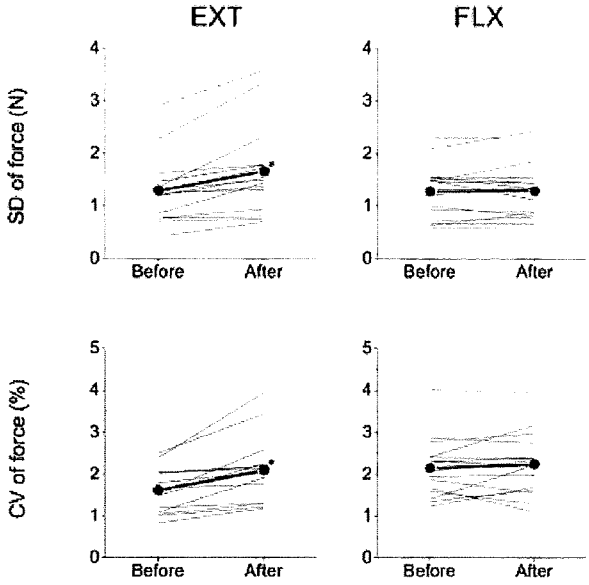

FIGURE 4. Force fluctuations during steady contractions at three target forces in two knee positions before and after bedrest. The standard deviation (SD, upper) and coefficient of variation (CV, lower) of force in each subject are plotted. The filled circles indicate mean values. $^*P < 0.05$ before and after bedrest.

transmitted from the MG fluctuates in a compensatory manner to other plantarflexor muscles under normal conditions. Hence, muscle activity and force fluctuations in different knee positions are consistent with previous reports.

Adjustment of Muscle Activity after Bedrest. We found that force fluctuations were unaltered in FLX after bedrest (Figs. 4 and 5). Most of the force fluctuations in FLX are likely attributable to the SOL because of minimal levels and changes in aEMG and disadvantageous anatomical characteristics of the gastrocnemius muscles.[12,13] The absence of adjustments in the EMG activity of the SOL in both time (Fig. 2) and frequency (Figs. 6 and 7) domains and unaltered force fluctuations in FLX thus indicate that activity of the SOL was not modulated with respect to force fluctuations by bedrest. Hence, bedrest does not seem to influence the activity of the SOL to such an extent as to modulate force fluctuations.

In contrast, consistent with our previous experiment on bedrest,[29] both the SD and CV of force increased and accompanied an increased aEMG in the MG in EXT after bedrest (Figs. 2 and 4). We found that modulation of MG activity after bedrest was observed not only in the average amplitude of EMG but also in the fluctuations (power) of the rectified EMG (Figs. 6 and 7), although the difference in the amount of changes between experiments may indicate variable responses depending on subjects and experimental conditions.

Potential Mechanisms for Increased Force Fluctuations after Bedrest. Increased fluctuations in net force need to be considered with respect to the magnitude

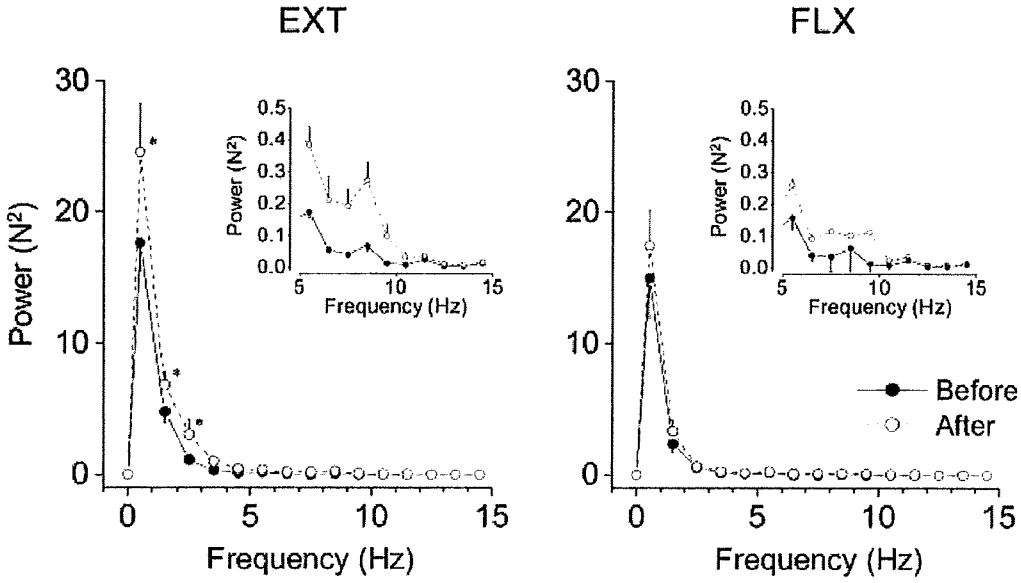

FIGURE 5. Power spectral density of force in different knee positions before (filled circles) and after (open circles) bedrest. Power spectral density collapsed across target forces is presented every 1-Hz bin. (*Inset*) Power spectral density of force ≥5 Hz in a magnified y-axis scale for visual purpose. $^*P < 0.05$ before and after bedrest.

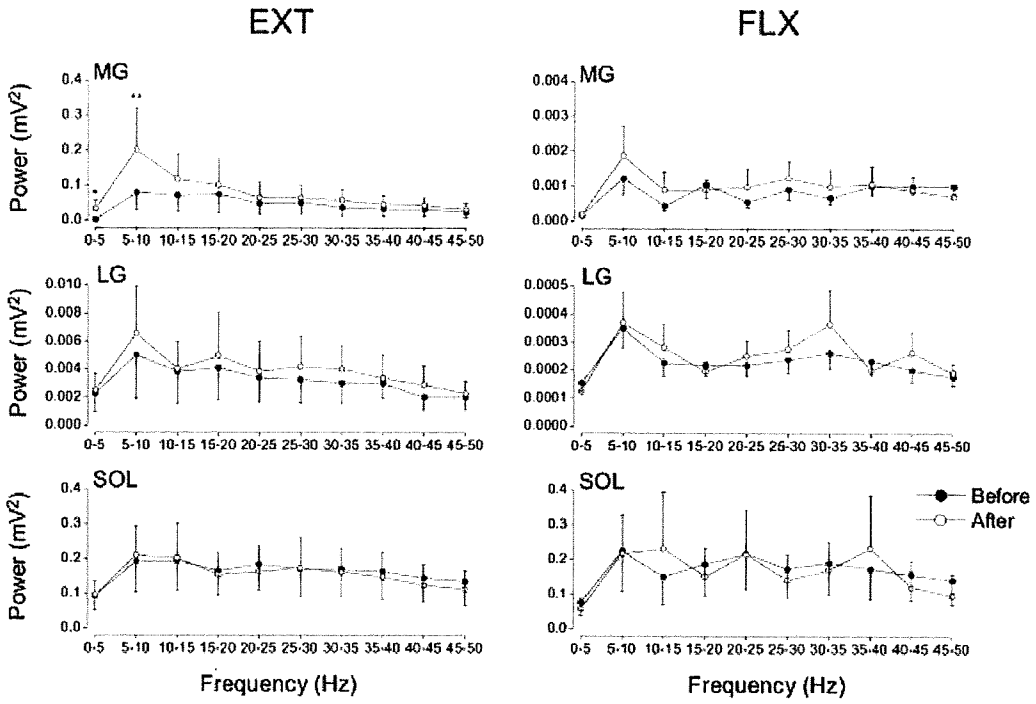

FIGURE 6. Power spectral density of the rectified EMG during the steady contraction task in different knee positions before (filled circles) and after (open circles) bedrest. Power spectral density collapsed across target forces is presented for every 5-Hz bin. $^{*}P < 0.05$, $^{**}P < 0.01$ before and after bedrest.

of fluctuations in MG activity and interactions of force fluctuations between the MG and other agonist muscles. Force fluctuations in plantarflexor muscles are not solely dependent on the magnitude of the MG activity because greater force fluctuations were associated with decreased MG activity due to a reduction in knee position[28] but with increased MG activity after bedrest.[29]

It is conceivable that the activation strategy of the MG is modified in such a manner that it attenuated the compensation for force fluctuations across agonist muscles after bedrest. In other words, force fluctuations produced by the MG may have changed not only in their magnitude, but also in their temporal relation to other agonist muscles. In the present study, frequency modulation of rectified EMG in the MG was found in EXT after bedrest, indicating that the temporal characteristics of force fluctuations in the MG were altered. Although the most prominent change in power was at the lower frequency range in force (<3 Hz) compared with the rectified EMG (≤10–15 Hz), this discrepancy is not surprising. Due to the slower mechanical kinetics of twitch force compared with the neural input (electrical activity) to the muscle, changes in the higher frequency domain in the neural input are dampened when compared with the lower frequency domain in the muscle force.[24,36,38]

Unique Modulation of MG Activity after Bedrest. The mechanisms underlying the low-frequency modulation of rectified EMG after these interventions are unknown. One of the potential confounding factors that could have a strong influence on EMG characteristics after bedrest is a reduction in conduction velocity in the axon terminals and muscle fibers.[6,7,27] However, there is no reason to expect that a reduction in conduction velocity after bedrest would be limited to a specific agonist muscle in a specific condition. In addition, a reduction in the conduction velocity would contribute to a reduction in the mean (or median) power frequency of the nonrectified EMG,[4,20] and this effect occurs at a much higher frequency range than the present low-frequency range (≤10–15 Hz). Therefore, the increase in the low-frequency content of the rectified EMG indicates an increased temporal modulation of the neural activity in the MG and not a change in conduction velocity.

The adaptation in the frequency content of the rectified EMG after bedrest is consistent with adaptation occuring with aging. Vaillancourt et al.[33]

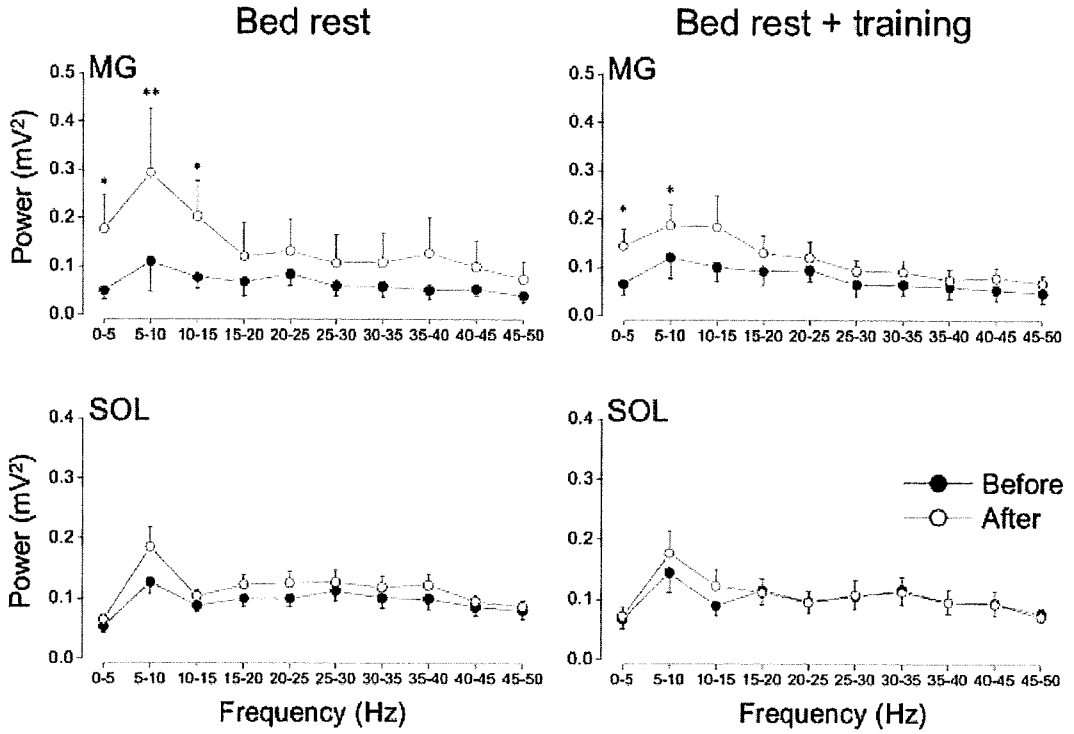

FIGURE 7. Power spectral density of the rectified EMG during the steady contraction task in the knee-extended position before (filled circles) and after (open circles) bedrest in the non-training (left) and training (right) groups. The EMG data from Shinohara et al. (2003)[29] was analyzed. Power spectral density collapsed across target forces is presented for every 5-Hz bin. $^{*}P < 0.05$, $^{**}P < 0.01$ before and after bedrest.

found an increase in the relative power of 5–15 Hz in rectified EMG with aging that accompanied a decrease in relative power at higher frequencies (~40 Hz) during steady contractions of the first dorsal interosseous muscle. Similar trends may have been obtained in the MG in our study because of an increase of <15 Hz and no change at higher frequencies in power in absolute units. Hence, it appears that reduced habitual activity, either due to aging or bedrest, induces a low-frequency (≤10–15 Hz) modulation of muscle activity that is associated with increased force fluctuations.

Since the frequency content of the rectified EMG represents the global activity of the motor unit pool, the increased power ≤10–15 Hz implies an increased oscillation in the discharge rate of motor units at this frequency range. According to Wessberg and Vallbo,[35] the low-frequency modulation of EMG around 8–10 Hz is not dependent on the stretch reflex, but is generated by central mechanisms. It is speculated, therefore, that oscillations ≤10–15 Hz may be increased in the descending command from higher centers and subsequent motor unit activity due to unknown mechanisms triggered by reduced habitual activity.

Our findings suggest a unique modulation of muscle activity in MG after bedrest, but not in other plantarflexor muscles including SOL. The fiber type composition is distinct between MG and SOL (more type I fibers in SOL). Hence, greater atrophy in MG compared with SOL may have played a role. However, this is unlikely because no difference was reported in atrophy between MG and SOL during 20-day bedrest in several other studies.[1–3,14] The increased aEMG specific to the MG after bedrest is consistent with preferential recruitment of the gastrocnemius muscle during a foot-pedal task and locomotion in rhesus monkeys after spaceflight.[25,26] Since the MG shows intermittent activities whereas the SOL shows tonic activity during quiet standing,[21] it is speculated that the muscle in favor of intermittent activity may be more susceptible to modulation due to a change in habitual activity. An individual with greater modulation in the MG due to reduced habitual activity or aging may have greater difficulty in controlling posture. This kind of modulation in muscle activity would be more critical in a microgravity environment (e.g., space) where small fluctuations in muscle force lead to large errors in movement (e.g., foot-pedal control, posture control).

Further investigations into task/muscle specific modulations of neural activity at the level of motor unit and higher centers are necessary.

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Effects of 20-day bed rest with and without strength training on postural sway during quiet standing

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Abstract

Aim: To examine the effect of unweighting as a possible contributory factor to a reduced calf muscle volume on postural sway during quiet standing, changes in postural sway following bed rest with or without strength training were investigated.

Methods: Twelve young men participated in a 20-day bed-rest study. Subjects were divided into a non-training group (BR-Con) and a strength training group (BR-Tr). For the BR-Tr group, training was comprised of dynamic calf-raise and leg-press exercises to maintain the muscle volume of the plantar flexors. Before and after bed rest, subjects maintained quiet standing in a barefoot position on a force platform with their eyes open or closed. During the quiet stance, foot centre-of-pressure (CoP) and the mean velocity of CoP was calculated. Muscle volume of the plantar flexors was computed using axial magnetic resonance images of the leg.

Results: After the bed-rest period, the muscle volume decreased in the BR-Con group but not in the BR-Tr group. The mean velocity of CoP as an assessment of postural sway, however, increased in both groups. These results indicate that the strength training during bed rest cannot counteract the increase in postural sway.

Conclusion: We concluded that postural sway increases following 20 days of bed rest despite maintenance of the muscle volume of plantar flexors as the main working muscles for the human postural standing.

Keywords centre-of-pressure, inactivity, muscle volume, postural control.

The bipedal upright stance is inherently unstable because in this stance a large body mass with a high elevation centre is kept in an erect posture over a relatively small base of support. Based on the dynamics of the human quiet stance, ankle joint torque plays a significant role stabilizing the body during quiet standing (Morasso & Schieppati 1999, Masani *et al.* 2003). Plantar flexor activity during quiet stance is a major determinant for ankle joint torque because the centre of mass is in front of the ankle joint (Smith 1957), and the tibialis anterior muscle as antagonists maintain a strict silence (Panzer *et al.* 1995, Gatev *et al.* 1999, Masani *et al.* 2003). Additionally, the activities of the plantar flexors have been found to be coherent with both spontaneous body sway (Gatev *et al.* 1999, Masani *et al.* 2003) and mechanically induced body sway (Fitzpatrick *et al.* 1996). These previous findings suggest that the plantar flexors control postural sway via neural regulation.

Lower limb muscle atrophy caused by inactivity such as bed rest (Akima *et al.* 2000b, 2001) and spaceflight (Edgerton *et al.* 1995, Akima *et al.* 2000a) is well

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documented. It has been reported that such an absence of gravitational loading decreases exercise-induced elevations in growth hormone in humans (McCall *et al.* 1997, 1999). Furthermore, it has been suggested that growth hormone responses during bed rest or spaceflight can be attributed to the chronic alternations in proprioceptive inputs (Edgerton & Roy 1996). The adaptation of muscle mass caused by the absence of gravitational loading could be different between individual muscles around the ankle joint because of the divergent response of proprioceptive inputs between the plantar and dorsi flexors (McCall *et al.* 2000). This may lead to changes in postural sway because of imbalance between agonistic and antagonistic muscle activity levels. However, adaptability of the postural sway during gravitational unloading remains unclear from the point of view of muscle mass around the ankle joint. Several studies reported the significant contribution of pressure receptors in the plantar soles, which are known to transmit information contributing to postural control (Horak *et al.* 1990, Kavounoudias *et al.* 2001, Maurer *et al.* 2006). The influence of unweighting based on the previous reports has led to the hypothesis that the lack of ground reaction forces of the plantar soles and their spatial distribution affects postural sway because of bed rest.

Recently, additional strength training during 20 days of bed rest has been shown to prevent a decrease of muscle size and function in the lower limbs (Akima *et al.* 2000b, 2003). By using additional intervention, an experimental model for bed rest with or without strength training has made it possible to examine the influence of gravitational unloading related to muscle mass on postural sway. Therefore, the purpose of the present study was to compare postural sway in young subjects standing quietly before and after bed rest. The subjects were divided into a group that performed strength training during bed rest and a group that did not. The results obtained from the present study may enhance our understanding of the postural ataxia associated with inactivity as well as the roles of the plantar flexor muscle in human body balance.

Methods

Subjects

Twelve healthy young subjects participated in the study. To minimize the potential influence of postural sway and training effects, all subjects were males. The subjects had no significant medical history or signs of neurological disorders, and had not participated in any programmes of regular exercise. They were randomly assigned to one of two groups: a strength training during bed-rest group (BR-Tr, $n = 6$) and a non-training during bed-rest group (BR-Con, $n = 6$). The age, height and body mass of the subjects (mean ± SD) were 22.7 ± 2.9 years, 169.6 ± 7.8 cm and 67.3 ± 13.7 kg for the BR-Tr group and 23.3 ± 4.9 years, 169.8 ± 6.4 cm and 65.5 ± 17.1 kg for the BR-Con group respectively. There were no significant differences in physical characteristics between the two groups. All subjects gave their written informed consent for the study after receiving a detailed explanation of the purposes, potential benefits and risks associated with participation. All procedures used in this study were in accordance with the Declaration of Helsinki and were approved by the ethical standards of the Committee for Human Experimentation at the Faculty of Medicine, The University of Tokyo. The general aspects of this bed-rest study have been previously reported elsewhere (Akima *et al.* 2003).

Bed rest and strength training procedures

The bed rest and strength training procedures used in the present investigation have been described in our previous study (Akima *et al.* 2003). Briefly, the subjects remained in 0.1 rad (6°) head-down tilted bed rest for 20 days to simulate a microgravity environment (Convertino *et al.* 1981), except during the strength training exercise. The subjects were prohibited from any weight-bearing posture, and all physical activities were restricted except for the strength training exercise. All subjects completed the 20-day programme of bed rest.

The subjects in the BR-Tr group performed strength training for 16 days of the bed-rest programme with four non-training days. The strength training consisted of one morning session and one afternoon session per day. The morning and afternoon sessions consisted of a dynamic bilateral leg-press exercise and a dynamic bilateral calf-raise exercise, respectively, performed using a modified leg-press training device (VR-4100, Cybex, NY, USA). The leg-press exercise was designed to train the knee extensor as well as the plantar flexor muscles (Fig. 1a). The angles of the hip, knee and ankle joints at the initial position were 1.92, 1.57 and 1.40 rad (110°, 90° and 80°) respectively (Fig. 1a, upper panel). The training required the subjects' hip, knee and ankle joints to be fully extended against the load (Fig. 1a, bottom panel). The calf-raise exercise was designed to train the plantar flexor muscles (Fig. 1b). The training was done with a range of motion from the fully ankle-flexed position (Fig. 1b, upper panel) to the ankle extended position (Fig. 1b, bottom panel) with extended knee joints. Each exercise was performed in 3 s cycles with 1 s for the shortening phase (Fig. 1, from upper to bottom panels) and 2 s for the lengthening phase (Fig. 1, from bottom to upper panels). To determine the maximal force of each training exercise,

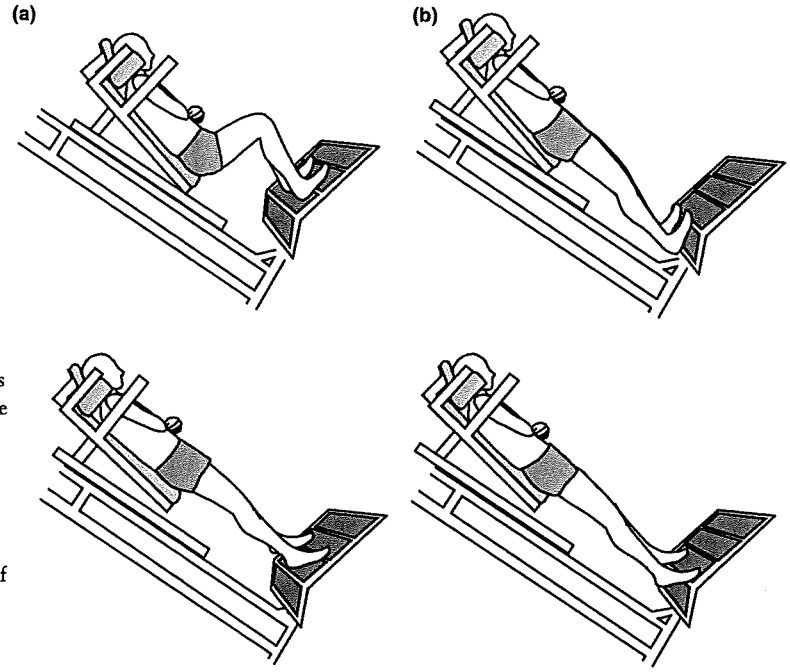

Figure 1 Schematic illustration of the strength training consisting of leg-presses and calf-raises. The initial position of the subjects during leg-presses (a) and calf-raises (b) as strength training (upper panels). The leg-press exercise required that the subjects' hip, knee and ankle joints be fully extended (bottom panel). The calf-raises were done with a range of motion from the fully ankle-flexed position (upper panel) to -extended (bottom panel). See text for details.

the subjects were first required to exert maximal isometric voluntary contraction force (MVC) for each exercise task. The MVC measurements of the static bilateral leg-press exercise and the bilateral calf-raise exercise were performed at the initial position of each training stance (Fig. 1, upper panels). MVCs were measured on the first, sixth, 13th and 20th days of the bed-rest period, and the training intensity corresponded to 70% of MVC. In both training sessions, the subjects performed five sets of 10 repetitions with a rest period of 60 s between the sets. If the subjects had difficulty in accomplishing the prescribed exercise load, they continued lifting with maximal effort even though the range of motion diminished.

Magnetic resonance imaging

To examine the quantitative features of the calf muscles, magnetic resonance images of the muscles were collected before and after bed rest. The subjects were imaged in a prone position at an ankle joint angle of 120°, with 180° being the full extension of the joint. Transverse scans were carried out at a slice thickness of 10 mm and an inter-slice gap of 7 mm. In a series of cross-sectional images, outlines of the soleus (SOL), medial gastrocnemius (MG), lateral gastrocnemius (LG) and tibialis anterior (TA) were traced, and anatomical cross-sectional areas (ACSA) of the individual muscles were determined. The volume of the plantar flexor muscles (MG, LG and SOL) and TA was determined by summing the ACSA of each image times the thickness and inter-slice gap of each section. The measurement and calculation procedures have been described in detail by Akima *et al.* (2003). To examine the balance between the muscle volume of the antagonist and that of the agonist, the ratio of muscle volume of TA to that of the plantar flexor muscles was calculated. Instead of using the physiological cross-sectional area (PCSA) we used the muscle volume as the quantitative feature of the muscles because muscle volume is closely correlated with joint torque (Fukunaga *et al.* 2001). The foot centre-of-pressure (CoP) position has been shown to be a linear approximation of ankle torque, with the single-joint inverted pendulum that rotates about the ankle joint of the human body during quiet stance approximated as a single segment (Morasso & Schieppati 1999).

Postural sway measurement

Postural sway was examined before and immediately after the bed-rest period. The basic set-up and measurement of the postural sway during the quiet standing procedure has been described in our previous study (Masani *et al.* 2003). The subjects were required to maintain a quiet stance barefoot on a force platform (Type 9281B, Kistler, Zürich, Switzerland) with eyes open (EO condition) or closed (EC condition) for approximately 40 s. The subjects held their arms by their sides and their feet parallel, with a distance of 15 cm between the heels. Five trials were conducted for each condition with sufficient rest between trials.

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The CoP position was obtained from the vertical components of the force platform. In the present study, we focused on anteroposterior CoP sway to examine the relation between changes in postural sway and muscle volume, because plantar flexor muscles contribute mainly to the stabilization of body sway in this direction (Masani *et al.* 2003). Surface electromyogram (EMG) was recorded from the right SOL, MG, LG and TA using Ag/AgCl electrodes with a diameter of 5 mm and an inter-electrode distance (centre-to-centre) of 20 mm. The bipolar electrodes were attached over the muscle belly along the direction of the fascicles of each muscle using ultrasound B-mode images (SSD-2000, Aloka, Tokyo, Japan). After careful abrasion of the skin, the electrodes were placed at the same locations before and after the bed-rest period with the use of permanent-ink spot marks. The reference electrode was placed over the lateral portion of the knee. The electrodes were connected to a pre-amplifier and a differential amplifier with a bandwidth of 5-Hz to 1 kHz (1253A, NEC Medical Systems, Tokyo, Japan). All electric signals were stored with a sampling frequency of 1 kHz on the hard disk of a personal computer using a 16-bit analogue-to-digital converter (PowerLab/16SP, ADInstruments, Sydney, Australia).

To assess postural sway during quiet stance, the mean velocity of the CoP in the anteroposterior direction was calculated (Panzer *et al.* 1995), because this parameter has higher reproducibility than other parameters, i.e. amplitude, area and standard deviation. Data for a 30-s period in the middle portion of the collected data (~40 s) were selected for analysis of individual trials. Before the calculation of the mean velocity of the CoP, a time series of the CoP was passed through a low-pass filter of 15 Hz using a fourth-order Butterworth filter. When the filtered CoP series are written as x_i, the mean velocity of the CoP (V) was calculated as

$$V = \sum_{i=2}^{n} |x_i - x_{i-1}|/T \quad x_i\, i = 1, 2, \ldots, n$$

where T is the duration (30 s) of analysis. Consequently, the mean velocity of the CoP was computed from the CoP total path length per calculated time. The CoP-based measures of postural sway have been evaluated by a variety of time and frequency domain measures of postural sway (Prieto *et al.* 1996). The best measure for postural control assessment as related to regulatory balancing activity (Maki *et al.* 1990) was the mean velocity of the CoP anteroposterior direction (Prieto *et al.* 1996). We therefore computed the mean velocity of CoP as an assessment of ability in postural balance, as this parameter has been reported by previous studies, which indicted that the mean velocity of CoP sway in the anteroposterior direction is related to the amount of regulatory balancing activity associated with this level of stability (Maki *et al.* 1990, Prieto *et al.* 1996).

In addition, structural post-urographic parameters were computed using a sway density plot approach (Baratto *et al.* 2002). The sway density curve (SDC) was calculated by counting the number of consecutive samples during which the postural sway in the anteroposterior and mediolateral axes remained inside a 2.5-mm radius. The computed SDC was low-pass filtered (cut-off frequency = 2.5 Hz) by a fourth-order Butterworth filter to perform a better peak extraction of SDC trajectory. The mean value of amplitude of all peaks (MP), the mean time interval between one peak and another (MT), and the distance between one peak and another (MD) were extracted from the SDC to document the possible physiological processes underlying the control of an upright stance (Baratto *et al.* 2002, Corbeil *et al.* 2004).

Postural control is integrated by information including information from the vestibular, visual and somatosensory systems, and individuals have an inherent cycle of body sway (Nashner 1981). To examine the contribution of this to our measurements, we analysed the power spectrum of the CoP sway. The 30-s data were first divided into seven segments that were 2^{13} points long (8.192 s). Almost half of the selected 8.192 s segments overlapped with the adjacent segments (Bloomfield 2000). A 13-bit fast-Fourier transform algorithm was then applied to these segments to yield the segments' power spectrum (cm^2). Consequently, the frequency resolution of the power spectrum was 0.122 Hz. An ensemble-averaged power spectrum across these segments was calculated as a power spectrum of the CoP sway. The power spectra of CoP sways greater than DC and less than 1 Hz were integrated and defined as the low-frequency component (LF) of the CoP sway. This is because body sway ≤1 Hz has been shown to reflect the sensitivity of vestibular (Kapteyn & de Wit 1972, Mauritz & Dietz 1980) and visual information (Diener *et al.* 1982). In contrast, CoP sway >1 Hz has been reported to depend on somatosensory inputs including proprioception (Diener *et al.* 1986, Nakagawa *et al.* 1993). In addition, the motor units in SOL showed that those units synchronized at an interval of approximately 100 ms (10 Hz) during quiet standing (Mori 1973). This rhythmic activity is referred to as 'physiological tremor' (Freund 1983), which is oscillation in the stretch reflex arc originating from Ia afferents. Therefore, the power spectrum of CoP sway from 1 to 10 Hz was integrated and defined as the high-frequency component (HF) of the CoP sway. Furthermore, the rectified EMG of each muscle was averaged over the period (30 s) to yield the average amplitude of EMG (AEMG). The AEMG in absolute units (μV) was further expressed in normalized

values as a percentage of the corresponding value (%MVC) during the MVC measurements of isometric plantar flexion and dorsiflexion.

Supplementary test for daily muscle activity during bed rest

A supplementary test was conducted to examine the muscle activity level during bed rest. The EMG of the SOL muscle was recorded with and without bed rest from 11:00 to 17:00 hours for 6 h in five subjects (age: 24.2 ± 2.1 years, height: 172.0 ± 4.6 cm and body mass: 69.0 ± 4.7 kg). To remove the influence of higher muscle activities during bed rest, the supplementary test was employed in subjects who did not participate in the present bed-rest study, because subjects must execute the MVC several times per day to normalize the EMG values. The procedures were exactly the same as in the bed-rest study, and the daily activities such as meals, toilet, bath and so on, during bed rest were strictly controlled in a way similar to the present bed-rest study. The procedures were exactly the same as in the bed-rest study, and daily activities such as having meals, going to the toilet and bathing during the bed-rest period were strictly controlled in a way similar to that in the present bed-rest study. The young Japanese males who participated in the supplementary test had similar physical characteristics to the subjects who participated in the present bed-rest study. A recording was obtained for each subject during regular daily activities without restraints (in other words, normal daily activity). For these EMG recordings, Ag/AgCl electrodes were placed (20 mm apart) over the muscle belly of the SOL. The EMGs were attached to a portable EMG device (ME-3000P8, Mega Electronics, Kuopio, Finland). For non-restrained daily activity, a portable EMG device was placed in a small hip bag and worn around the waist by the subject. EMG signals were amplified (bandwidth 8–500 Hz) and stored for later analyses. The recorded EMG was rectified and averaged over 0.1 s (AEMG), and then expressed relative to the EMG value observed during MVC of plantar flexion. The procedures used for long-term EMG measurements were those reported previously by Kern *et al.* (2001).

Statistical analyses

A two-way ANOVA with repeated measures and a Tukey's *post hoc* test was used to compare the mean velocity of CoP, LF and HF of CoP sway and MP, MT and MD extracted from SDC in each condition (EO and EC) and muscle volume before and after bed rest in the two subject groups (BR-Con and BR-Tr) as factors. To identify the significant difference of the ratio of muscle volume of TA to that of the plantar flexor muscles before and after bed rest in both subject groups, a two-way ANOVA with repeated measures and a Tukey's *post hoc* test was used. To compare the AEMGs before and after bed rest, a three-way ANOVA with repeated measures and a Tukey's *post hoc* test was used in the two subject groups (BR-Con and BR-Tr), four muscles (SOL, MG, LG and TA), and two time periods (before and after bed rest) as factors. The level of significance for all comparisons was set at $P = 0.05$ and corrected for multiple comparisons. Relative changes in the mean velocity of the CoP, LF and HF components in response to bed rest were calculated for each individual as the difference between the before- and after-bed-rest values divided by the before-bed-rest value. A linear regression analysis was performed between relative change in the LF and HF components or in muscle volume and that in the mean velocity of the CoP. Values are given as mean ± SE in the text, table and figures.

Results

In all calculated values before bed rest, i.e. muscle volume, mean velocity of CoP, each frequency component, structural post-urographic parameters and the AEMGs, no significant differences were seen between BR-Con and BR-Tr.

Daily EMG activity during bed rest

The long-term (6-h) AEMGs obtained from the SOL in different daily activities in five individual subjects are shown in Figure 2. It is obvious that the EMG amplitude and bursts are lower in bed rest than in unrestricted activity. The mean AEMG of the SOL over 6 h corresponded to 3.65 ± 1.71% of the MVC (range: 2.21–5.69%MVC) in the unrestricted activity. In contrast, during bed rest, the mean AEMG of the SOL was below 0.1% of the MVC in all subjects (0.078 ± 0.020%MVC, range: 0.042–0.093%MVC), and significantly lower than that when not restricted ($P < 0.05$). These results, therefore, demonstrated that bed rest employed in the present study succeeded in inducing substantially less activity of the lower leg than that of normal daily activity.

Muscle volume

In the BR-Con group, the muscle volume of the plantar flexor muscles after bed rest was significantly ($P < 0.05$) less than that before bed rest (Table 1). The decrease in muscle volume because of bed rest was 12.7%, from 786.4 ± 63.2 cm^3 before bed rest to 702.5 ± 71.8 cm^3 after bed rest. In contrast, muscle volume did not differ

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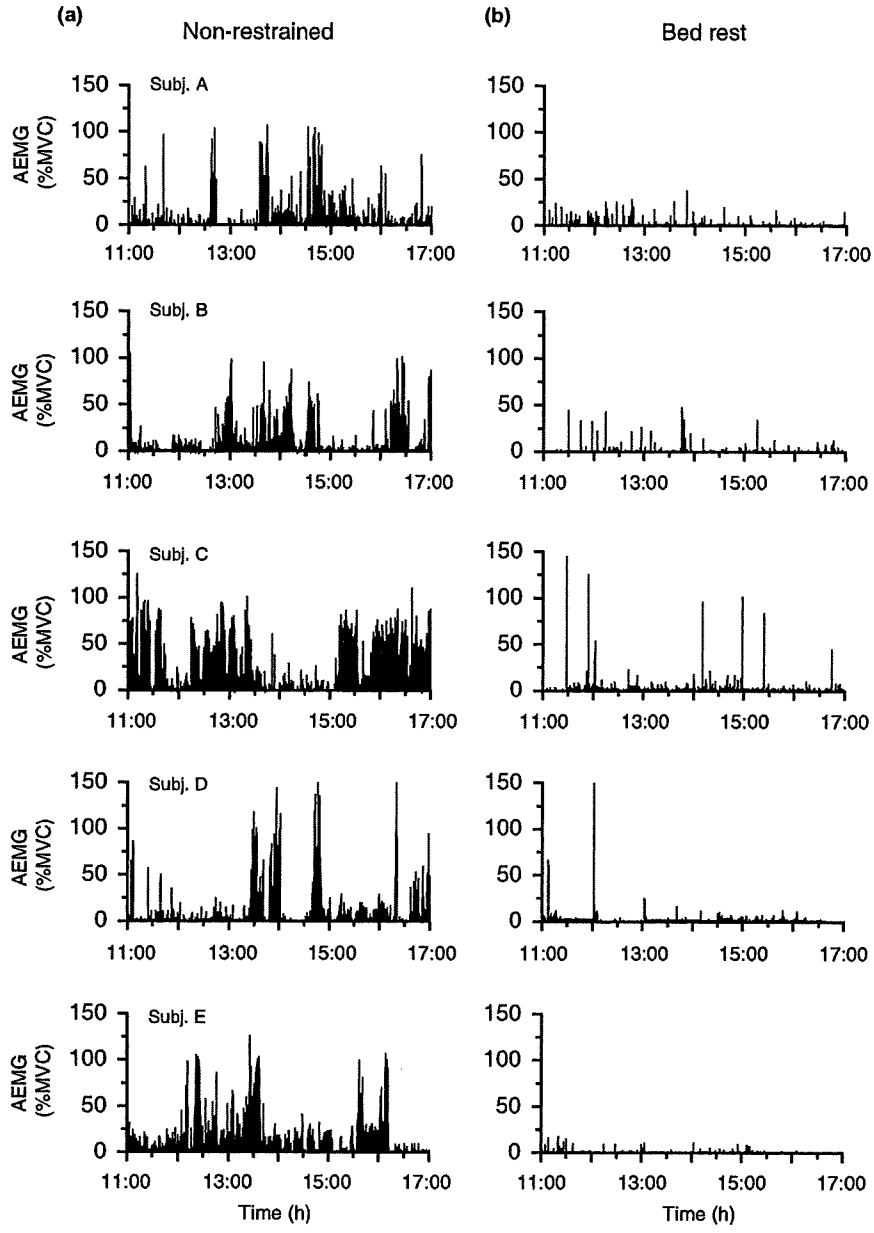

Figure 2 Electromyogram (EMG) activity in the soleus muscle during non-restrained daily activity and bed rest in five subjects (subjects A–E). (a) Non-restrained refers to the EMG recordings obtained while the subject was not restrained in the cast. (b) Bed rest refers to the EMG recordings obtained during the bed-rest period. Values are rectified and averaged EMG (AEMG) expressed relative to the average (0.1 s) EMG observed during a maximal voluntary contraction. EMG was recorded continuously from 11:00 to 17:00 hours for 6 h.

before and after bed rest with training in the BR-Tr group (Table 1). Changes in muscle volume in the present study were similar to the MVC and PCSA for plantar flexors reported by our previous study; i.e. MVC associated with PCSA significantly decreased because of bed rest in the BR-Con group, but not in the BR-Tr group (Akima *et al.* 2003). In contrast to plantar flexors as the main working muscles for the quiet stance, there was no change in the muscle volume of TA by bed rest in both the BR-Con and BR-Tr groups (Table 1). As a result, the ratio of TA to plantar flexor muscles (SOL, MG and LG) as a muscle volume balance between antagonist and agonist significantly increased in BR-Con ($P < 0.05$), but not in BR-Tr (Fig. 3).

Table 1 Muscle volume (cm^3) of plantar flexor muscles and tibialis anterior muscle before- and after- bed rest

	Before	After
BR-Con		
PF	786.4 ± 63.1	702.5 ± 71.8*
TA	122.9 ± 13.2	119.0 ± 12.4
BR-Tr		
PF	667.1 ± 51.5	646.2 ± 63.4
TA	108.3 ± 7.7	105.5 ± 9.9

Data are mean ± SE for six subjects. BR-Con and BR-Tr indicate non-training group during bed rest and strength training group during bed rest respectively. PF, plantar flexor muscles; TA, tibialis anterior muscle. Significant difference from before bed-rest measurement (*$P < 0.05$).

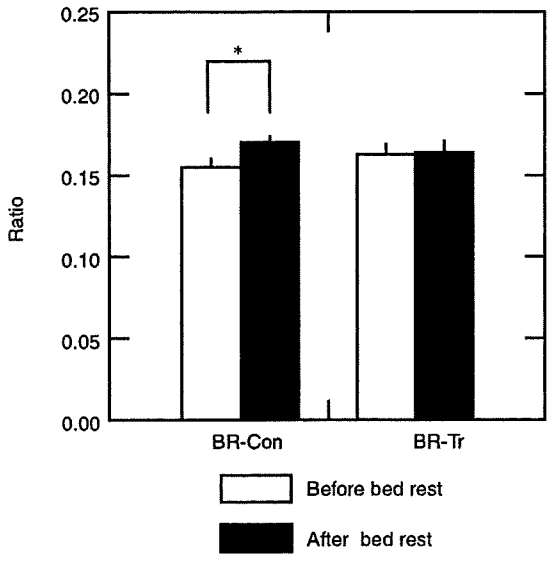

Figure 3 Ratio of muscle volume of tibialis anterior muscle to that of plantar flexor muscles before and after bed rest. Average (±SE) values before (open bar) and after bed rest (filled bar). *$P < 0.05$ was the significant difference between the before- and after-bed-rest values.

Postural sway and EMGs during quiet standing

When the subjects performed even quiet standing, the CoP fluctuated. Clearly, the fluctuations in the CoP were greater after bed rest than before bed rest in both groups (Fig. 4a). The mean velocity of the CoP before and after bed rest averaged in both the BR-Con and BR-Tr groups across subjects is presented in Figure 4b. The mean velocity of the CoP significantly ($P < 0.05$) increased by bed rest in the BR-Con group and the BR-Tr group in EO (upper) and EC (bottom). Relative changes in CoP velocity by bed rest were not significantly different between BR-Con (EO: 38.6 ± 14.9%; EC: 29.2 ± 9.3%) and BR-Tr (EO: 46.7 ± 10.7%; EC: 40.7 ± 14.6%). No significant correlation between relative changes in the mean velocity of the CoP and those in muscle volume was found (EO: $r = -0.302$, $P = 0.339$; EC: $r = 0.159$, $P = 0.628$).

A representative example of SDC is shown before and after bed rest in Figure 5a. Clearly, the amplitude of SDC was higher before bed rest compared with after bed rest. MP, MT and MD calculated from SDC before and after bed rest averaged in both the BR-Con and the BR-Tr groups across subjects is presented in Figure 5b. The MP significantly ($P < 0.05$) decreased by bed rest in the BR-Con group and the BR-Tr group in EO (upper) and EC (bottom). The MD significantly ($P < 0.05$) increased by bed rest in the BR-Con group and the BR-Tr group in EO and EC. In contrast, there were no significant changes in MT by bed rest in either group for both EO and EC.

The power spectrum of the CoP was calculated to examine how alterations in the frequency content of the CoP could be associated with increases in the CoP sway after bed rest. Most of the power was ≤1 Hz without peak, and increased after bed rest in both the BR-Con and BR-Tr groups. The high frequency content above 1 Hz also had no peak, and the power was constant before- and after-bed rest (Fig. 6). To quantify the frequency range that contributed to CoP sway, LF (≤1 Hz) and HF (1–10 Hz) were calculated (Fig. 7). There were no significant differences between the before- and after-bed-rest HF values in either group for both EO and EC (Fig. 7b). In contrast, LF in both BR-Con and BR-Tr significantly ($P < 0.05$) increased by bed rest for both the EO and EC conditions (Fig. 7a). To further examine the relation between CoP sway and each frequency component because of bed rest with and without strength training, relative changes in LF and HF were plotted against the relative change in the mean velocity of CoP after bed rest (Fig. 8). A significant correlation was found between the relative change in LF and that in the mean velocity of the CoP (EO: $r = 0.684$, $P < 0.05$; EC: $r = 0.688$, $P < 0.05$). In contrast, there was no significant correlation for the HF. These results indicate that the increase in the mean velocity of the CoP after bed rest is associated with the enhancement in the LF (≤1 Hz) of the CoP sway.

The AEMG values of individual muscles of the ankle extensors during quiet standing before and after bed rest in both groups are summarized in Table 2. The AEMG of TA as the antagonist muscle for ankle extension was substantially smaller than that of the ankle extensor muscles, and there was no influence because of bed rest in either the BR-Con or BR-Tr groups. There was no systematic change in AEMG across the muscles in either group despite the increase in CoP by bed rest.

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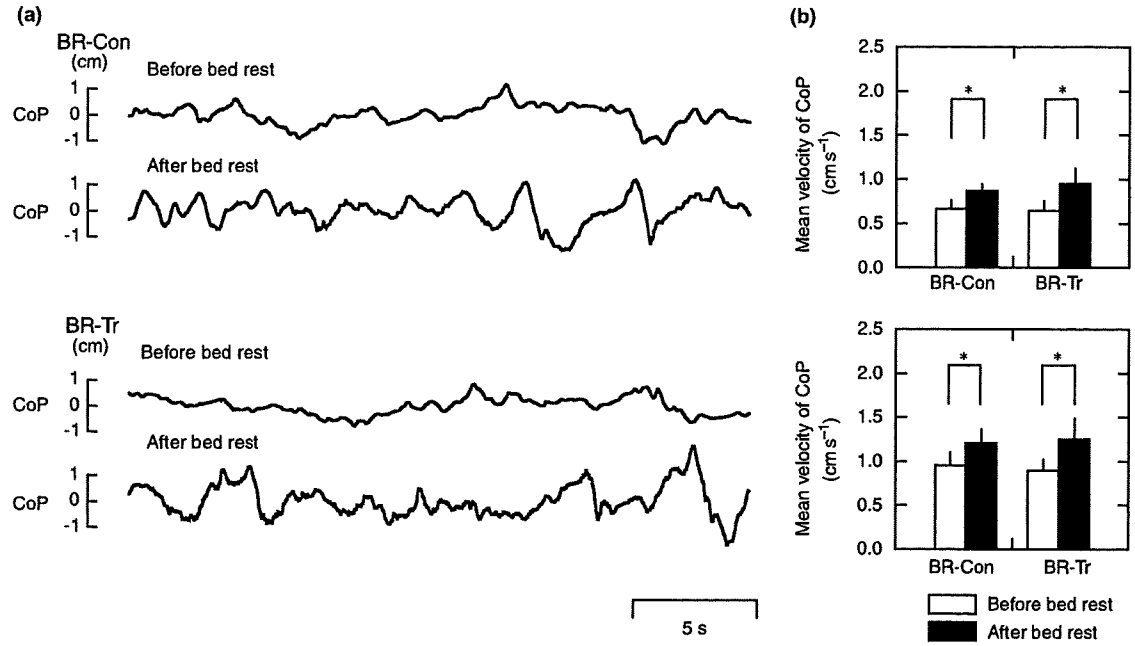

Figure 4 Foot centre-of-pressure (CoP) sway during the quiet stance before- and after-bed rest. (a) Typical example of CoP sway in the anteroposterior direction before and after bed rest without (BR-Con, upper panels) and with strength training (BR-Tr, bottom panels). (b) The group-averaged (±SE) mean velocity of CoP before (open bar) and after bed rest (filled bar) with both groups in eye open (EO, upper panel) and closed (EC, bottom panel) conditions. $*P < 0.05$ was the significant difference between the before- and after-bed-rest values.

Discussion

The aim of the present study was to compare the postural sway in response to bed rest of two groups of healthy young subjects, one that performed strength training during bed rest to maintain muscle volume and the other that did not. The main results were that the postural sway in response to bed rest with and without strength training was not influenced by changes in the muscle volume of the main working muscles involved in quiet standing. To our knowledge, this is the first paper examining the effects of the lack of gravitational loading with the intervention of strength training on postural sway based on the dynamics of the human quiet stance. Our novel finding was that postural sway increased because of bed rest despite the maintenance of muscle volume around the ankle joint by strength training.

Effects of bed rest and training on muscle volume

Human skeletal muscle atrophy induced by prolonged bed rest has been shown by many studies (e.g. LeBlanc *et al.* 1992), including our studies (Akima *et al.* 2000b, 2001, Akima *et al.* 2003). In the present study, the decline of muscle volume of the plantar flexors in the BR-Con group was 12%, similar to the results from our previous study (Akima *et al.* 2000b). To examine whether this decrease can be attributed to the inactivity of lower limbs because of bed rest, we compared the muscle activity level of the SOL during bed rest and normal daily living in one subject by means of surface EMG. The AEMG of the SOL during bed rest was below 0.1% of MVC in all subjects (0.078 ± 0.020%MVC, range: 0.042–0.093%MVC). The AEMG of the SOL during bed rest was significantly less than that during normal daily activity ($P < 0.05$). This result ensures that bed rest without strength training causes a significant reduction in the muscle activity of the plantar flexors, suggesting that muscle volume loss during bed rest derives from muscle inactivity. The present study focused on the muscle volume around the ankle joint following bed rest, because it has been reported that growth hormone responses during bed rest or spaceflight can be attributed to the chronic alternations in proprioceptive inputs (Edgerton & Roy 1996), which have different effects on the plantar and dorsi flexors (McCall *et al.* 2000). In the present study, the gravitational unloading by bed rest without strength training induced a decrease in the muscle volume of the plantar flexor muscles as agonists, not of the TA as the antagonist. As a result, the balance of muscle volume around the ankle joint changed because of bed rest.

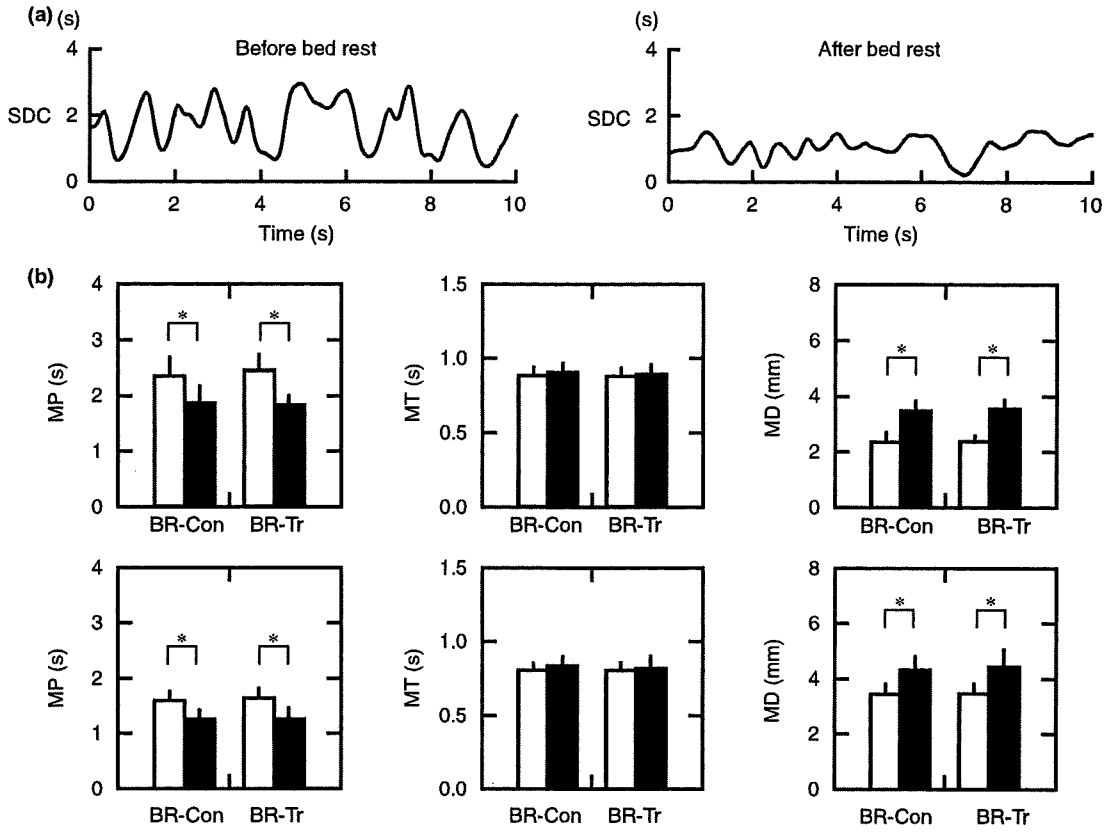

Figure 5 Sway density curve (SDC) during quiet stance before- and after-bed rest. (a) Typical example of SDC during quiet stance with open eyes before (left panel) and after bed rest (right panel). (b) Group-averaged (±SE) mean value of all peaks (MP), mean value of the time interval between successive peaks (MT) and mean value of all distance between the successive peaks (MD) extracted from the SDC before (open bar) and after bed rest (filled bar) with both groups in the eye open (EO, upper panel) and closed (EC, bottom panel) conditions. $*P < 0.05$ was the significant difference between the before- and after-bed-rest values. See text for details on the method of calculating SDC.

To prevent this muscle deconditioning, we designed strength training for use during bed rest. In our previous study (Akima *et al.* 2000b), it was demonstrated that isometric and concentric leg-press training during a 20-day bed period rest did not maintain the size of the plantar flexor muscle group, but did counteract the decline of the knee extensor muscle groups. In the present study, a calf-raise exercise was added to the previous training programme to prevent atrophy of the plantar flexor muscle group. Its muscle volume was maintained, and as a result, the balance of muscle volume around the ankle joint was maintained. Therefore, it became possible to compare the BR-Con and BR-Tr groups to examine the effects of muscle volume around the ankle joint on postural sway.

Effects of muscle volume on postural sway

In the human bipedal stance, the model of a single-joint inverted pendulum rotating around the ankle joint suggests the importance of ankle joint torque in stabilizing the body during quiet standing (Morasso & Schieppati 1999, Masani *et al.* 2003). As the dorsi flexor muscles during quiet standing maintain a strict silence (Panzer *et al.* 1995, Gatev *et al.* 1999, Masani *et al.* 2003) and the body centre of mass is located in front of the ankle joint (Smith 1957), the plantar flexor muscles are major components in ankle joint torque. Postural instability has been well documented in older adults (Maki *et al.* 1990, Panzer *et al.* 1995), who have significantly less muscle mass and decreased function of lower limb muscle groups than do the young (Janssen *et al.* 2000). As stated, the strength training employed in the present study during bed rest maintained the muscle volume of the plantar extensors, while bed rest without training was accompanied by a decrease in muscle volume. The result is that the balance of muscle volume between the agonists and antagonist for postural sway was maintained in the BR-Tr, not in the BR-Con. Nonetheless, the mean velocity of CoP

