Fluctuations in plantar flexion force are reduced after prolonged tendon vibration

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The purpose of the study was to examine the effect of prolonged vibration on the force fluctuations during a force-matching task performed at low-force levels. Fourteen young healthy men performed a submaximal force-matching task of isometric plantar flexion before and after Achilles tendon vibration (n = 8, vibration subjects) or lying without vibration (n = 6, control subjects) for 30 min. The target forces were 2.5–10% of the previbration maximal voluntary contraction force. The standard deviation of force decreased by a mean of 29 ± 20% across target forces after vibration, whereas it did not decrease significantly in control subjects (−5 ± 12%). This change was significantly greater compared with control subjects (P < 0.01 for both). Power spectral density of the force was predominantly composed of signals of low-frequency bandwidth (≤5 Hz) with few higher frequency components. In vibration subjects, there was a significant decrease in power in the frequency range ≤2 Hz after vibration. The decrease in power at this frequency range was linearly related to the decrease in the frequency range ≤2 Hz after vibration. The results indicate that prolonged Achilles tendon vibration reduces the fluctuations in plantar flexion force in the frequency range ≤2 Hz during low-level contractions. It suggests that Ia afferent inputs contribute to the low-frequency force fluctuations in plantar flexion.

IN HUMAN MOVEMENT, MOTOR OUTPUT always involves variability about the intended movement. For example, when an individual tries to match one’s output force to a submaximal target force as steadily as possible, the exerted force fluctuates about the target. It has been shown that the force fluctuations are influenced by a number of factors, including contraction type, muscle used for the task, age of the subjects, and activities of daily life (10). In particular, the force fluctuations are often greater in older adults compared with young adults (12, 30), and they are exacerbated by the reduced habitual activity during a prolonged bed rest in young adults (31).

The experimental and simulation studies on the final physiological cascades for the motor output of a single muscle, namely motor unit activation strategy, have revealed that the force fluctuations are influenced by multiple features of motor unit activity, including the strength of low-frequency (1 Hz) oscillation of motor unit discharge rate about a mean discharge rate and amount of variability of discharge rate (22, 34). Because motor unit activity is controlled by the neural inputs to the α-motoneuron pool in the spinal cord, the potential effect of afferent input to the force fluctuations has been postulated. Laidlaw et al. (22) speculated a possible involvement of altered stretch reflex function in the age-related increase in the fluctuations in motor output. The intrafusal muscle spindle senses small length changes in the muscle fiber. Information from the intrafusal muscle spindle is forwarded via Ia afferents to the spinal cord, which in turn gives excitatory inputs to the α-motoneuron pools of the homonymous muscle, thus facilitating the activation of motor units. Involvement of this pathway in fine motor control has been demonstrated by direct measurements of Ia afferent discharges (20, 39, 40), but its functional significance in fine motor control is controversial. Wessberg and Vallbo (37, 38), for example, cast their doubts on the basis of the lack of a close temporal relation between Ia afferent discharges and 8- to 10-Hz oscillations in the acceleration or muscle activity during slow finger movements. More recently, however, Cresswell and Lösch (4) found a reduction in the fatigue-induced increase in the force fluctuations in the frequency range of 5–30 Hz (tremor) for the plantar flexor muscles after prolonged vibration that is known to depress Ia circuit functions (9, 17, 28, 29).

The study by Cresswell and Lösch (4) demonstrated an effect of Ia circuit function on the force fluctuations only in the frequency range of 5–30 Hz that develops during fatiguing contractions. However, the major frequency range for the force fluctuations is <4–5 Hz during force-matching tasks performed at low force level in the nonfatigued state (3, 6–8, 33, 34, 36). Although the potential contribution of Ia circuit function to the higher frequency fluctuations in motor output (5–30 Hz) has been tested for a number of years (11, 14, 15, 23, 24, 26), there is no study that has examined the effect of Ia circuit function on the lower frequency fluctuations in force (<4–5 Hz).

The purpose of the study was to determine the effect of prolonged tendon vibration on force fluctuations during a force-matching task performed in the nonfatigued state. We expect to find that prolonged vibration reduces low-frequency fluctuations in force during a brief contraction because 1) prolonged vibration has been suggested to depress Ia afferent inputs, 2) a reduction in the Ia afferent inputs would increase the relative contribution of cortical projections to the motor

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units, and 3) low-frequency fluctuations in force (<4–5 Hz) are prominent during force-matching tasks at low force level.

METHODS

Subjects. Fourteen young healthy men volunteered to participate in this study. The subjects had no medical history or physical signs of neurological disorder. Only male subjects with relatively small subcutaneous fat thickness were recruited to ensure clear electrical recordings during low-force contractions. The age, height, and body mass of the subjects (means ± SD) were 27.1 ± 4.8 yr, 172.4 ± 6.0 cm, and 68.6 ± 10.9 kg, respectively. Eight subjects (vibration subjects) received vibration to the Achilles tendon, and the effects of vibration on maximal voluntary contraction (MVC) force and force fluctuations were examined. Six subjects (control subjects) performed the same tasks as the experimental subjects, however, without vibration. Reproducibility of MVC force and the force fluctuations were confirmed from the data in control subjects. There were no significant differences in physical characteristics between the groups. Subjects gave informed consent, and the experiments were approved by the Ethics committee of the Department of Life Sciences, The University of Tokyo, Japan.

Experimental protocol. Subjects performed the MVC and submaximal force-matching tasks of isometric plantar flexion with the dominant (right) leg before and after prolonged Achilles tendon vibration (vibration subjects) or lying for the same time period without vibrations (control subjects). The MVC preceded the force-matching task before vibration or lying, whereas it followed the force-matching task after vibration or lying.

Vibration. Prolonged vibration was applied to the Achilles tendon for 30 min by a specially designed mechanical stimulator (model DPS-285, Dia Medical System, Tokyo, Japan). The mechanical stimulator consisted of a direct-current motor with the shaft embedded in a plastic tube. The vibrator shaft was attached to the Achilles tendon at the ankle joint level with a force of 10–15 N. Vibration was applied with a frequency of 100 Hz and displacement of ±0.75 mm.

MVC. The MVC task involved a gradual increase in plantar flexion force exerted by the triceps surae muscle from baseline to maximum in 3–4 s, which was then sustained at maximum for 2 s. The plantar flexion force was displayed in real time on the oscilloscope. The timing of the task was based on a verbal count given at 1-s intervals, with vigorous encouragement from the investigator when the force began to plateau. Each subject performed at least three MVC trials, with subsequent trials performed if the differences in the peak force of two MVCs were >5%. Subjects were allowed to reject any effort that they did not regard as “maximal.” The trial with the highest peak force was chosen for analysis.

Force-matching task. The procedure for the force-matching task was the same as previously employed by our group (31). Subjects were asked to contract their plantar flexor muscles and to maintain a plantar flexion force as steady as possible about the target displayed on an oscilloscope with visual feedback for 30 s. The gain of the display on the oscilloscope was adjusted so that displacement between the two MVCs was 20%. Subjects were asked to contract their plantar flexor muscles and to maintain a plantar flexion force from baseline to maximum for 2 s. The plantar flexion force was displayed in real time on the oscilloscope. The timing of the task was based on a verbal count given at 1-s intervals, with vigorous encouragement from the investigator when the force began to plateau. Each subject performed at least three MVC trials, with subsequent trials performed if the differences in the peak force of two MVCs were >5%. Subjects were allowed to reject any effort that they did not regard as “maximal.” The trial with the highest peak force was chosen for analysis.

Mechanical recordings. Subjects lay in a prone position on a padded bed with the thigh secured to the bed by a strap. Force was measured with a strain gauge transducer positioned between a metal baseplate and a foot lever plate. The bottom end of the foot lever plate had a half-round-shaped attachment that surrounded and secured the heel. The heel was secured with a strap at the bottom end of the foot lever plate. The strain gauge transducer was aligned between the two plates near the distal part of the foot. The exact position of the entire device was carefully adjusted so the knee and hip was fully extended with the ankle joint angle at 90°. A low-sensitivity force transducer (model LTZ-200KA, Kyowa, Tokyo, Japan; 0.013 V/N) was used during the MVC task, and a more sensitive transducer (model LUR-A-100NSA1, Kyowa; 0.18 V/N) was used during the force-matching task. The force was amplified and low-pass filtered (<100 Hz) by a direct-current amplifier with a filter (model DPM 700, Kyowa). Electrical recordings. Surface electromyogram (EMG) was recorded from the medial gastrocnemius (MG), lateral gastrocnemius (LG), and soleus (Sol) with bipolar Ag-AgCl electrodes (diameter: 8 mm, interelectrode distance: 20 mm). The electrodes were connected to a preamplifier and a differential amplifier (>1,000) having a bandwidth of 5 Hz to 1 kHz (model 1253A, NEC Medical Systems, Tokyo, Japan).

Data analysis. The force and EMG signals were collected at a sampling frequency of 2 kHz by a 16-bit analog-to-digital converter (PowerLab/16sp, ADInstruments, Toyko, Japan) and stored on a personal computer. The middle 16 s of the contraction were used for further analysis in the force-matching task. The mean value and the SD of force, and root mean square amplitude of EMG (EMGrms) across 16 s were calculated (bin = 0.5 ms) by the standard methods. The relation between the SD and mean force (normalized to MVCpre or corresponding MVC) was evaluated with the slope and intercept obtained by a linear regression analysis with the least squares error method.

Power spectrum density of the force signal was obtained by the fast Fourier transformation method (16,384 points, Hamming window, 0.061 Hz/bin) after elimination of the direct-current component and resampling at 1 kHz. It is known that the force fluctuations during steady contractions are predominantly in the range of less than ~12 Hz when the level of force is <20% MVC (34, 36). We have confirmed in a pilot study that the frequency component above 12 Hz is very small (<0.3% of the total power) for plantar flexion forces <10% MVC. For the purpose of statistical comparison, the mean power across 1-Hz windows (16 or 17 bins) was further calculated up to 12 Hz. In addition, linear regression analyses were performed between the relative change in the SD and the relative change in the low- (<5 Hz) or high-frequency power (>5 Hz). The frequency ranges that could explain the alterations in the SD of force after vibration were identified by these analyses. In MVC tasks, EMGrms was calculated over a 1-s window centered with the time at which peak force was attained.

Statistical analysis. For the control subjects, the intraclass correlation coefficient (ICC) was calculated for MVC force and the SD of force before and after 30 min of lying. This was calculated under the assumption of a one-way random effects model (32), and linear correlation coefficient (Bravais-Pearson’s r) between the data. Relative changes in MVC force and the slope and y-intercept of SD-force relation were compared between two subject groups with unpaired Student’s t-test. Relative change in the SD of force between two subject groups was compared with two-way ANOVA (2 subject groups × 4 intensities) with repeated measures. In vibration subjects, the power spectral density of force in each 1-Hz frequency bin was compared with a three-factor ANOVA (4 intensities × 2 times × 15 frequencies) with repeated measures. Linear correlation coefficient (Bravais-Pearson’s r) was obtained between the relative change in the SD of force and the relative change in the low- or high-frequency power of the force signal. EMGrms during MVC task was tested by using a two-way ANOVA (3 muscles × 2 times) with repeated measures. EMG rms during force-matching task was tested by using a three-way ANOVA (3 muscles × 2 times × 4 intensities) with repeated measures. An α level of 0.05 was chosen for all statistical
analyses with post hoc comparisons (Newman-Keuls test) when appropriate. All values are expressed as means ± SE in the figures and means ± SD in the text and Table 1 unless stated otherwise.

RESULTS

In control subjects, the ICCs for MVC force before and after 30 min of lying was 0.98, and the linear correlation coefficient (Bravais-Pearson’s $r$) in MVC force before and after 30 min of lying was 0.99. Consequently, there was no significant change in MVC force (0.06 ± 14%). In contrast, MVC force in vibration subjects decreased by 19 ± 10% after vibration (Table 1). The relative change in MVC force in vibration subjects was significantly greater ($P < 0.05$) compared with control subjects (see Fig. 3). The reduction in MVC force accompanied decreases in EMG$_{rms}$ in vibration subjects (Table 1). The average decrease in EMG$_{rms}$ was 32 ± 25% ($P < 0.01$), 12 ± 19% ($P < 0.05$), and 12 ± 16% ($P = 0.053$) for MG, LG, and Sol, respectively.

When the subject matched the force to the target, the exerted force fluctuated about an average value (Fig. 1). The amount of force fluctuations (SD of force) was examined from the middle 16 s of data. In Fig. 2 (top), the SD of force was plotted against the mean force normalized to the MVC$_{pre}$. The SD of force increased linearly as the level of target force increased. In control subjects, the SD of force did not change after 30 min of lying (Fig. 2, left). The ICC for the SD of force before and after 30 min of lying was 0.96. In addition, a high linear correlation coefficient (Bravais-Pearson’s $r = 0.98$) was found between the SD of force before and after 30 min of lying. These analyses confirmed the test-to-retest reproducibility of the measurements in the present design. It was also confirmed that the SD of force before lying or vibration was not statistically different between the control and vibration groups ($P > 0.05$). In vibration subjects, the force fluctuations appeared to decrease after vibration (Fig. 1). On average, the SD of force decreased across target forces after vibration (Fig. 2, right), and the relative change in the SD was significantly greater in vibration subjects (−29 ± 20%) compared with control subjects (−5 ± 12%) across target forces ($P < 0.01$; Fig. 3).

It is of note that the target forces for the force-matching task were the same in absolute units within a subject before and after vibration.

Table 1. Force and EMG amplitude during MVC and force-matching tasks before and after vibration

| Force, N | EMG$_{rms}$, mV |
|---|---|---|---|---|---|---|
| MVC, % | MG | LG | Sol |
| Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| 2.5 | 10.3±4.0 | 10.6±4.1 | 7.3±5.1 | 5.4±4.5 | 1.6±0.6 | 1.5±0.6 | 17.8±9.9 | 16.3±7.7 |
| 5.0 | 19.4±5.0 | 19.4±5.0 | 15.0±7.5 | 11.8±5.9 | 2.8±0.8 | 2.6±0.8 | 31.1±14.5 | 36.3±20.6 |
| 7.5 | 28.1±6.0 | 28.1±6.3 | 21.2±6.9 | 21.0±6.7 | 3.5±6.9 | 4.1±1.4 | 34.0±14.6 | 35.4±15.2 |
| 10 | 36.0±5.8 | 36.0±5.6 | 23.5±6.8 | 26.3±7.6 | 4.2±6.8 | 5.2±1.5 | 41.7±16.1 | 39.3±19.0 |
| 100 | 372.7±66.2 | 299.4±57.8† | 228.0±178.0 | 154.1±123.5† | 70.6±22.9 | 61.6±24.5* | 83.3±28.2 | 73.3±25.4 |

Values are group means ± SD. Pre, before vibration; Post, after vibration; EMG$_{rms}$, root mean square amplitude of surface electromyogram; MG, medial gastrocnemius muscle; LG, lateral gastrocnemius muscle; Sol, soleus muscle. *$P < 0.05$. †$P < 0.01$ between Pre and Post values.

Fig. 1. Representative data for force and electromyogram (EMG) during force-matching task before (Pre) and after (Post) vibration. Data are from 1 subject matching 10% maximal voluntary contraction (MVC). MG, medial gastrocnemius muscle; LG, lateral gastrocnemius muscle; Sol, soleus muscle. SD of force decreased from 0.79 to 0.49 N after vibration in this example.
after vibration (Table 1). Because of the reduction in MVC force in vibration subjects, one might argue that the decrease in the force fluctuations could be due to the difference in the levels of target relative to the corresponding MVC force. When the SD of force was plotted against the mean force normalized to the corresponding MVC force before and after vibration, the plots shifted to the right, but with very slight displacements (Fig. 2, bottom). To eliminate the effect of the decreased MVC force and to make a statistical comparison for the relation between the force fluctuations and the intensity normalized to the corresponding MVC force, the slope and y-intercept of the linear relation between the SD and mean force (normalized to the corresponding MVC force) before and after vibration were calculated for each subject. It was apparent that the y-intercept decreased in vibration subjects, and the statistical analysis confirmed that the relative change in vibration subjects (−23%) was significantly greater (P < 0.05) compared with control subjects (−7%) (Fig. 3). Relative change in the slope was small, and there was no difference between subject groups (−4% and −5%). These results indicate that the decrease in the force fluctuations in the vibration subjects was independent of the decrease in MVC force after vibration. There was no systematic change in EMGms across muscles despite the slight difference in the level of target force relative to the corresponding MVC force in vibration subjects (Table 1).

Power spectral density of the force was calculated to examine how alterations in the frequency content of the force signal could be associated with reductions in force fluctuations after vibration. Most of the power was ~5 Hz with a peak around 0.5 Hz before vibration, and the peak power was reduced after vibration (Fig. 4). In Fig. 5, the grouped data after vibration are overlaid with the data before vibration, indicating that the remaining filled portions are the amount of reductions in power after vibration. The greatest power in the force signal was observed ≤1 Hz across target forces, for which significant decline in power was found after vibration across all target forces (P < 0.01). The second greatest power was observed in 1–2 Hz, for which significant decline in power was found for the two of the four target forces (P < 0.01). On average, the power declined in other frequency ranges as well, but there was no significant change at any frequency band >2 Hz. Relative change in the power of the force signal for low-frequency (<5 Hz) and high-frequency (>5 Hz) range was further plotted against the relative change in the force fluctuations after vibration (Fig. 6). A highly significant correlation was found between the relative change in low-frequency power of force and the SD of force (r = 0.96, P < 0.001, Fig. 6A). There was no significant correlation for the high-frequency power (r = 0.25, P > 0.05; Fig. 6B). These results indicate that the
decrease in the force fluctuations after vibration is associated with the reductions in the low-frequency band (≤5 Hz) of the force signal.

DISCUSSION

The purpose of the study was to determine the effect of prolonged tendon vibration on force fluctuations during a force-matching task performed in the nonfatigued state and to examine the alterations in the frequency content of the force signals. The results demonstrated a reduction in the fluctuations in plantar flexion force after prolonged Achilles tendon vibration and identified the frequency range that was influenced by vibration.

Physiological mechanisms underlying the fluctuations in motor output are suggested to be dependent on the frequency range of the fluctuations (11, 27, 33, 34). According to McAuley and Marsden (27), the short-latency stretch reflex tends to create oscillations at 10 Hz, whereas the long-latency stretch reflex creates oscillations at 7 Hz, as well as central oscillations. Rhythmic oscillations in muscle activity around 20 Hz and 40–50 Hz observed in EMG may originate centrally. In particular, the force fluctuations during a fatiguing contraction...
fluctuations and that of low-frequency power (≤5 Hz; A) and in the high-frequency range (≤5 Hz; B). A significant linear regression line \( r = 0.96, P < 0.001 \) is superimposed in A. Correlation coefficient in B was 0.25 \( (P > 0.05) \), NS, not significant.

The frequency range in which significant reductions in power were observed after vibration was ≤2 Hz (Fig. 5), indicating the possibility of the close relation between Ia circuit function and low force of force. It has been suggested in a hand muscle that the force fluctuations in this frequency range could be attributable to the sensorimotor processing of the visual feedback information (33) and low-frequency modulation of motoneuron discharges (34). Slifkin et al. (33) provided evidence for reductions in power ≤2 Hz with a decrease in the time interval for the visual feedback. In the present study, visual feedback of the force signal was continuously provided, and it is unlikely that depression of the sensory feedback from muscle spindles affected the processing of the visual feedback information. Taylor et al. (34) showed in a simulation study that an addition of a low-frequency oscillation into the excitation of the motor unit population model was necessary to approximate the experimental finding of peak power at a frequency ≤2 Hz. Low-frequency modulation of motor unit discharges could be attributed to the rhythmicity in the spinal network or in the descending drive from supraspinal centers (1, 25). A recent study reported that elevated levels of physiological arousal with stress (electrical stimulation to the contralateral hand) increased the low-frequency power \( (1–2 \text{ Hz}) \) of force during a pinch-grip task (3). In the present study, there was no obvious stressor that could be changed before and after vibration. Hence, it is less likely that rhythmicity in the descending drive was depressed, and it is more likely that rhythmicity generated in the spinal network was altered, due to reduced afferent input to the spinal cord. It is of note that the altered frequency range in force is much lower than the frequency range of Ia afferent discharge \( (\text{greater than} \, -10 \text{ Hz}) \) (16). It indicates that the motor unit discharges are not directly modulated by the individual Ia discharges. This is also supported by the absence of a postspike positive peak in the Ia spike-triggered average in force during low-force isometric contractions (16). Hence, it is more likely that a reduction in Ia discharges modulated the rhythmicity in the spinal network, which is generated through multiple mechanisms, including the
activation of neurotransmitters and ion channels. In humans, there is no study that demonstrates the relation between afferent input and the strength of rhythmicity in the spinal network. However, in the neonatal rat, Marchetti et al. (25) observed that constant-frequency (2–10 Hz) stimulation of the dorsal root evoked low-frequency oscillatory patterns (≤2 Hz) in the ventral root of the isolated spinal cord. Although it is not clear how this finding can be extrapolated to the humans at this point, reduced spinal rhythmicity due to the reduced afferent inputs could be a potential mechanism underlying the depression of the low-frequency oscillation in force. Other possible mechanisms would include the potential influence of other afferents (e.g., cutaneous and pain), but there is not enough evidence to determine the potential effect of prolonged vibration on other afferents.

Changes in the distribution of muscle activity between the synergistic muscles have been suggested as one of the mechanisms that may influence the fluctuations in motor output in contractions of multiple muscles (13, 31) in addition to the motor unit activation strategy. Lack of changes in the distribution of EMG activity across triceps surae muscle implies that there was not a noticeable change in the global distribution of muscle activity, but it does not rule out the possible changes in the temporal muscle activity or motor unit activation strategy that could not be detected by surface EMG.

In conclusion, prolonged Achilles tendon vibration reduces the fluctuations in plantar flexion force during a low-level force-matching task. It seems that afferent input from Ia circuit contributes not only to the tremor component (8–12 Hz) during fatiguing contractions (4) but also to the force fluctuations of lower frequency range (<5 Hz) during a brief plantar flexion contraction performed in the nonfatigued state.

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