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The resonant component of human physiological hand tremor is altered by slow voluntary movements

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Key points

- Postural physiological hand tremor has a prominent component at ∼8 Hz unlike the associated EMG. Consequently, the gain between EMG and tremor is sharply peaked at ∼8 Hz.
- Deduction and a simple model using pre-recorded EMG or random noise as an input show that the ∼8 Hz peak is a consequence of resonance.
- During voluntary movement the gain peak enlarges and shifts to a lower frequency but the EMG spectrum shows no corresponding changes. This reflects muscle thixotropy. Adjustment of the muscle properties of the model reproduces the effect.
- These findings suggest that the rhythm of hand tremor in posture and movement is related to muscle and limb mechanics rather than a neural oscillator.
- The discovery that the gain relating EMG to acceleration is very different when static and moving has implications for the control of movement in health and disease.

Abstract Limb resonance imparts a characteristic spectrum to hand tremor. Movement will alter the resonance. We have examined the consequences of this change. Rectified forearm extensor muscle EMG and physiological hand tremor were recorded. In postural conditions the EMG spectrum is relatively flat whereas the acceleration spectrum is sharply peaked. Consequently, the gain between EMG and acceleration is maximal at the frequency where the tremor is largest (∼8 Hz). The shape of the gain curve implies mechanical resonance. Substantial alterations in posture do not significantly change the characteristics of the tremor or the shape or size of the gain curve. By contrast, slow or moderately paced voluntary wrist flexion–extension movements dramatically increase the hand tremor size and lower its peak frequency. These changes in size and frequency of the tremor cannot be attributed to changes in the EMG. Instead they reflect a very large change in the size and shape of the gain curve relating EMG to acceleration. The gain becomes larger and the peak moves to a lower frequency (∼6 Hz). We suggest that a movement-related (thixotropic) alteration in resonant properties of the wrist provides a simple explanation for these changes. The mechanism is illustrated by a model. Our new findings confirm that resonance plays a major role in wrist tremor. We also demonstrate that muscles operate very differently under postural and dynamic conditions. The different coupling between EMG and movement in posture and when moving must pose a considerable challenge for neural predictive control of skeletal muscles.

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Introduction

When any limb is maintained in a postural configuration it is not perfectly static. The small movements that it exhibits occur at a range of frequencies. When it is recorded, most of the fluctuation in position is seen at low frequencies and probably represents the small slow adjustments which are the best efforts of the nervous system to maintain a posture. If, instead, velocity or acceleration are recorded the higher frequency involuntary components are greatly emphasized. This behaviour is clearly demonstrated by the hand when held in an outstretched position. There is a prominent frequency peak in the acceleration spectrum of the hand. Tremor is commonly recorded as an acceleration signal and most investigators agree that the central frequency of this peak lies somewhere between 7 and 11 Hz (Stiles & Randall, 1967; Marsden *et al.* 1969; Elble & Randall, 1978; Hömberg *et al.* 1987; Lakie, 1992; Raethjen *et al.* 2000) and it is commonly described as '10 Hz tremor' although in fact a figure of 8 Hz would better describe the mean frequency for the hand. Although people tend to have similar hand tremor frequencies there is a very wide range of tremor size in different subjects (Lakie, 1992).

Considerable attention has focused on 10 Hz tremor since it was first clearly described in the thumb by Schäfer (1886). Schäfer believed this rhythm to be associated with what would now be known as sub-tetanic motor unit firing and therefore to be ubiquitous in skeletal muscle. However, extensive subsequent work has shown that there are causative factors other than just the rate of motor unit firing. It has been held to result from oscillation in a reflex loop (Lippold, 1970; Durbaba *et al.* 2005) or to result from a central oscillatory process (Elble, 1996). In addition to central oscillators or feedback oscillation, one definite component of tremor is mechanical. Force produced by the motor units is filtered by the muscle itself and by the visco-elastic and inertial load of the tendons and limbs. Marshall & Walsh (1957) first suggested that tremor might be a consequence of the imperfect low pass filtering properties of muscles which allowed significant sub-tetanic ripple in force output. Subsequently, Lakie *et al.* (1986) showed that the ripple was combined with the mechanical properties of the hand which behaved as a resonator. The combination of the inertia and stiffness of the limb produced a resonance and the frequency of the tremor could be altered systematically by artificially changing the inertia of the hand. Resonance was also found to be the dominating mechanism in shaping the tremor of the hand by Raethjen *et al.* (2000). Also, recent work (Reynolds & Lakie, 2010) showed changes in the frequency of hand tremor which they attributed to alteration in the resonant frequency which was related to the history of movement. Hand tremor frequency increases as muscles progressively stiffen following voluntary movement and this rise in tremor frequency is not associated with a rise in frequency of the EMG responsible. It is not presently known what happens to hand tremor during slow movements when a large thixotropic reduction in stiffness occurs. Interestingly, large, tremor-like oscillations have been commonly observed during finger movements (for example Vallbo & Wessberg, 1993). These oscillations have been attributed to a central organization of motor output during movement and the same claim has been made for hand movements (Kakuda *et al.* 1999). Daneault *et al.* (2011) have very recently shown that finger tremor size greatly increases during slow movement. However, none of these papers considered the possibility that muscle properties will be greatly altered during movement and that this mechanical change, rather than a change in the pattern of neural firing, may be responsible for the large tremors that occur in dynamic conditions. In addition, there has been no systematic study of the relationship between tremor recorded posturally and in slow movement, although they have been said to be different (Vallbo & Wessberg, 1993; Kakuda *et al.* 1999).

In the present study hand acceleration (tremor) was examined under three postural conditions and four conditions of voluntary movement at different velocities. The gain that relates EMG to acceleration of the hand was calculated. The results were clear. The EMG spectrum is relatively flat and not greatly different in postural and moving conditions. The acceleration spectrum is sharply peaked (at ∼8 Hz in static, and ∼6 Hz in slow movement). The fact that acceleration is particularly large at a certain frequency in the absence of any corresponding large EMG activity is a clear indication that resonance is occurring. We found that the resonance was very different in postural and moving conditions and we suggest that thixotropic changes in muscle stiffness are responsible. To illustrate this we describe a simple model. We used previously determined values for hand inertia and wrist stiffness and existing estimates of the way in which stiffness is partitioned between muscle and tendon. In order to demonstrate the effect of movement we made two suppositions based on experimental evidence obtained from *in vitro* preparations and other limb–muscle combinations.

Other limbs have tremor spectra which can show a peak around 10 Hz. However, they may also have additional peaks. For example, tremor of the finger usually shows two components. One is at a broadly similar frequency to hand tremor but the other is at a higher frequency (20–25 Hz; Stiles & Randall, 1967; Lakie, 1992). The presence of an additional peak is clearly a complication and for this reason the present paper is restricted to hand tremor. A paper investigating the two components of finger tremor is under preparation. Some of these findings have been briefly reported (Lakie *et al.* 2010).

Methods

Subjects

These experiments were carried out on 10 subjects (9 male). The age of the subjects ranged from 19 to 55 years, mean 23 years. All subjects were, as far as they knew, free from neuromuscular disorder. Subjects were asked to abstain from alcohol for 12 h before the experiments. Ethical permission was obtained from the ethics committee of the School of Sport and Exercise Sciences, University of Birmingham, and experiments were performed in accordance with the *Declaration of Helsinki* and with the subjects' written, informed consent.

Apparatus

Tremor was recorded using a purpose-built apparatus. Subjects sat in a comfortable adjustable chair with a headrest. The forearm was supported in a foam moulding at waist height and a padded support surrounded the pronated wrist. The hand was free to move in the vertical plane. A miniature single axis accelerometer (ICS 3021, EuroSensor UK) was attached to the dorsum of the hand. A retro-reflective laser rangefinder (YP11MGV80, Wenglor Sensoric, Germany) was positioned so that it reflected from a white label attached to the upper surface of the accelerometer. Thus vertical displacement and acceleration of the hand could be simultaneously recorded. Hand position was displayed to the subjects as a spot on a screen 1.5 m in front of them. Computer-generated targets were presented to the subject as a short bar on the screen. The subject was instructed to match the spot and bar as accurately as possible by tracking the bar. The static positions were 'up, middle and down'. 'Middle' was adjusted so that the central screen position suited the subject who held the hand in a neutral position, somewhat below horizontal. 'Up' was a 15 deg upward (extension) and 'down' was 15 deg downwards (flexion). These were postural changes of a substantial size without approaching the limits of joint rotation. The compatible vertical shifts on the screen were 7.5 cm. In the dynamic conditions the target alternated from the predetermined 'bottom' position to the 'top'. The target moved at one of four preselected constant velocities which were triangular waveforms at 0.1, 0.2, 0.5 and 1.0 Hz. The corresponding maximal angular velocities of the wrist were approximately 6, 12, 30 and 60 deg s^{-1}. Subjectively, these hand movements were of moderate size and ranged from very slow (0.1 Hz) to moderate (1.0 Hz). Faster movements were not studied because they are difficult to pace successfully and it was desired to maintain a wide gap between the tremor frequency and the movement frequency. All recording sessions lasted 60 s and the order was randomized. Subjects were told which condition to expect and a short period of practice was allowed before each recording was started. Each task was very easy to perform and was not physically or mentally demanding but rest periods were allowed between each recording. Surface EMG was recorded by a 2-channel Bagnoli system (Delsys Inc., USA) with electrodes positioned above the muscle belly (determined by palpation) of the extensor digitorum communis muscle.

Analysis

Tremor was recorded as a filtered acceleration signal. The signal was band-pass filtered between 0.05 and 45 Hz to reduce DC offsets caused by tilt and high frequency noise. It was amplified so that 1 V = 0.05 g (50 cm s^{-2}). The position signal was recorded at a sensitivity of 1 V = 1 cm. The frequency response of the position sensor was flat up to 100 Hz. EMG was amplified by a factor of 1000 with a pass band of 20–300 Hz. The resulting waveform was rectified to obtain an amplitude-modulated signal. All signals were sampled at 1000 Hz using a MC 6043 PCI card and PC. Analysis was carried out offline on recorded signals using Matlab software (MathWorks Matlab 2011a, Natick, MA, USA). Frequency domain calculations were performed on each measured acceleration signal and rectified EMG signal using NeuroSpec-software for Matlab (NeuroSpec, Version 2.0, 2008. For a theoretical framework see Halliday *et al.* 1995). In addition, the cross-spectral gain response between the rectified EMG and acceleration was obtained with NeuroSpec. The denominator in the gain calculation was the auto spectrum of the input and this approach has been commonly used for tremor analysis (e.g. Halliday *et al.* 1995). The frequency spectra were all calculated with a resolution of 0.06 Hz. The mean frequency spectra per condition were computed and smoothed using a running average of 0.003 s. Mean peak frequencies of acceleration and gain spectra were determined by establishing the maximum amplitude of heavily low-pass-filtered individual spectra (4th order Butterworth filter, cut-off frequency: 20 Hz). Differences in the peak frequencies of all conditions were studied using a repeated measures ANOVA for both acceleration and gain with each of the seven conditions as the within subjects factor. A Bonferroni correction was used to adjust the main effects to compensate for multiple observations. In addition, to compare the peak frequencies between acceleration and gain, we used paired samples *t* tests between each of the conditions. To compare all static conditions with all dynamic conditions each subject's mean (of the three static conditions and of the four dynamic conditions) was compared by a paired samples *t* test. Gain ratios were additionally calculated by dividing the gain of separate dynamic conditions by the mean static gain.

Modelling

The limb was modelled using Simulink (MathWorks, USA). The model was a second order damped torsional oscillator (Model 1). The hand was regarded as a moment of inertia (J) connected to a muscle–tendon complex. The angular stiffness of the complex is k. The damping is c. The input was either white noise or a pre-recorded extensor EMG signal. Both were put through a low pass filter which simulated muscle force generation. The output of the model is acceleration, but we also recorded force.k is composed of the series combination of muscle stiffness (k_m) and tendon stiffness (k_t).

$$\text{Accordingly,} \quad \frac{1}{k} = \frac{1}{k_t} + \frac{1}{k_m}$$

In the model we used a single set of values for k_m, k_t, c and J to represent postural conditions. When moving, muscle stiffness and damping are known to decrease (Lakie *et al.* 1984; Bennett *et al.* 1992). Therefore to model the dynamic conditions we used a different value for k_m and c. We did not change the values for the other parameters. For computational convenience we used the transfer function form where (s) is the complex Laplace variable. For the muscle (low-pass filter):

$$G(s) = \frac{1}{(1 + s\tau)^2}$$

And for the damped oscillator:

$$G(s) = \frac{1}{Js^2 + cs + k}$$

Our intention in developing this model was not to produce a perfect fit to the data that we recorded. We wanted to show that it was possible to produce a realistic tremor acceleration spectrum with a very simple model involving nothing more than a spring, mass and damper. We further wanted to show that by making a plausible alteration to two values (muscle stiffness and damping) we could mimic the changes that occur in the acceleration spectrum during movement. We used recorded EMG as an input but we also used white noise in order to show that there is nothing 'special' about the EMG waveform.

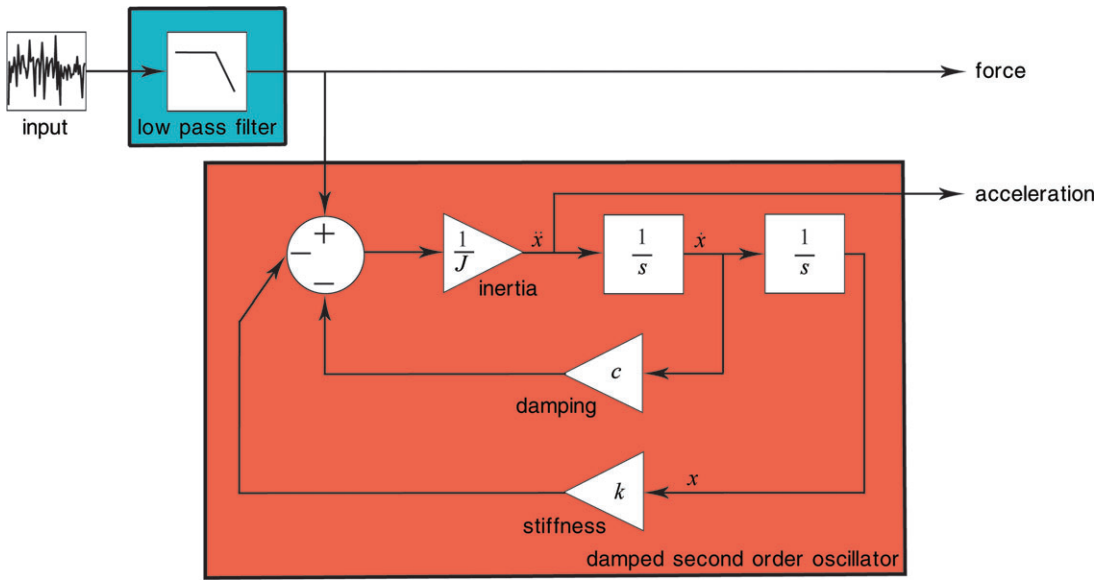
Choice of values. We used angular units for the model as these are common in the literature because they are easier to measure directly.

Moment of inertia of the hand (J). Estimates range between 0.001 and 0.004 kg m^2 (Lakie *et al.* 1986; Lehman & Calhoun, 1990; Milner & Cloutier, 1998). We used 0.0025 kg m^2.

Angular stiffness of the wrist (k). Estimates are 6–7.5 N m rad^{-1} (Lakie *et al.* 1984), 5–13 N m rad^{-1} (Milner & Cloutier, 1993) and an average value of 6.3 N m rad^{-1} (Grey, 1997). We used 7.5 N m rad^{-1}.

Damping of the wrist (c). The natural viscosity of the relaxed wrist was estimated to be 0.02–0.03 N m s rad^{-1} (Gielen & Houk, 1984) and 0.03 N m s rad^{-1} (Grey, 1997). We used 0.03 N m s rad^{-1}.

We made two assumptions about the change from static to moving conditions. First, when muscle moves there is



Model 1

a considerable change in the ratio of muscle stiffness to tendon stiffness. This results from a thixotropic decrease in muscle stiffness. We used values from Loram *et al.* (2007) obtained from the calf muscles. In this work it was shown that under static conditions the muscle was ∼15 times as stiff as the tendon. (15:1 ratio). When ankle rotation exceeded ∼0.5 deg this ratio changed rather abruptly to 1:1 because of a reduction in muscle stiffness. Accordingly, when we modelled dynamic conditions we reduced muscle stiffness by a factor of 15. Second, when muscle moves there is a decrease in limb damping. Halaki *et al.* (2006) found a reduction of 73% in larger movements. We reduced damping by a factor of three.

The input to the model was either recorded extensor EMG or white noise of a matched root mean square size. The filter which simulated the muscle had a time constant of 120 ms.

Results

All subjects found the experiments easy to perform. Results from a representative subject are shown in Fig. 1 (left) and the mean from all subjects in Fig. 1 (right).

In all conditions the rectified extensor EMG spectra (top row) are fairly flat. Most subjects had a small peak somewhere between 10 and 20 Hz. This is visible in the case of the representative subject who has a peak at ∼15 Hz in the postural conditions. When all subjects are averaged, the peak becomes very indistinct. The dynamic recordings additionally show a specific peak at the frequency of movement (0.1, 0.2, 0.5 and 1.0 Hz) as would be expected (inset). Because the waveform being tracked was triangular, low frequency harmonics of the movement frequency are also visible in the EMG spectrum. These are particularly visible in the 1.0 Hz record.

The inset shows the clear peaks in rectified EMG at the frequency of voluntary movement in the dynamic conditions. These can also be seen in the dynamic acceleration spectra. The vertical axis is logarithmic except for coherence.

In all conditions there is a clear peak in the acceleration spectrum (second row). In the representative subject it is impossible to distinguish clearly between the three static conditions. However, there is an obvious difference between the static conditions and the dynamic conditions. Under dynamic conditions the size of the peak was considerably bigger and the frequency of the peak was lower than under static conditions. The mean results from all subjects show these differences more clearly. The three static acceleration spectra remain almost indistinguishable. However, the four acceleration spectra under dynamic conditions are clearly different from the static conditions and from each other. With movement at the lowest speed (0.1 Hz) the acceleration spectra become considerably larger and the peak of the spectrum shifts to a lower frequency. There is a further increase in size and decrease in frequency as the movement is made faster. There are small peaks visible in the dynamic spectrum at the low frequencies of movement that were used in tracking which are a consequence of the tilt or acceleration at the frequency of the tracking task. The gain between rectified extensor EMG and acceleration is shown in Fig. 1 (third row). As may be anticipated from the shape of the EMG and acceleration spectra, these results show a gain which is sharply peaked at the tremor frequency. Results for all subjects show that, during movements of increasing speed, the mean gain has increased by a factor that ranges from approximately two to five and the frequency at which the gain is maximal has dropped from ∼8 Hz to 6 Hz. The cross-coherence between rectified EMG and acceleration is shown (bottom row). This shows that there is moderate coherence between EMG and acceleration at most frequencies. In all conditions this is enhanced where the acceleration is largest (between approximately 7 and 13 Hz). In the moving conditions there is also very high coherence at the frequency of the movement.

The consequence of the changes in EMG and acceleration is a very clear increase in gain under dynamic conditions. As the gain in the three static conditions is very similar it seemed sensible to combine these into a single set of representative values for postural conditions. Using these values, the ratio between the mean static gain and dynamic gain has been calculated for each of the dynamic conditions. These results are shown in Fig. 2.

This figure shows that the gain is higher at every frequency in the dynamic state compared to postural tremor although the difference becomes small around the frequency of tremor (approximately 8 Hz). This figure also shows that the gain ratio becomes greater as speed of movement increases. However, each increase represents an approximate doubling of speed and the last increase (from 0.5 Hz to 1.0 Hz) produces a relatively modest increase in gain ratio which suggests a saturating response.

For the gain measurements to be meaningful it is necessary to show that the EMG is being recorded from an appropriate muscle, that is, one that is involved in the postural and dynamic task. Figure 3 shows the mean level of rectified EMG from the extensor digitorum communis muscle in all of the static tasks and in one of the dynamic tasks. It can be seen that the level of EMG correlates with the demand of maintaining the different postures. Under dynamic conditions the depth of EMG modulation is revealed showing that the EMG variation slightly more than encompasses the range of bottom to top static EMG levels. The muscle from which the EMG is recorded is intimately concerned with the postural and dynamic regulation of hand position, a fact which was also shown by observation and palpation of the forearm.

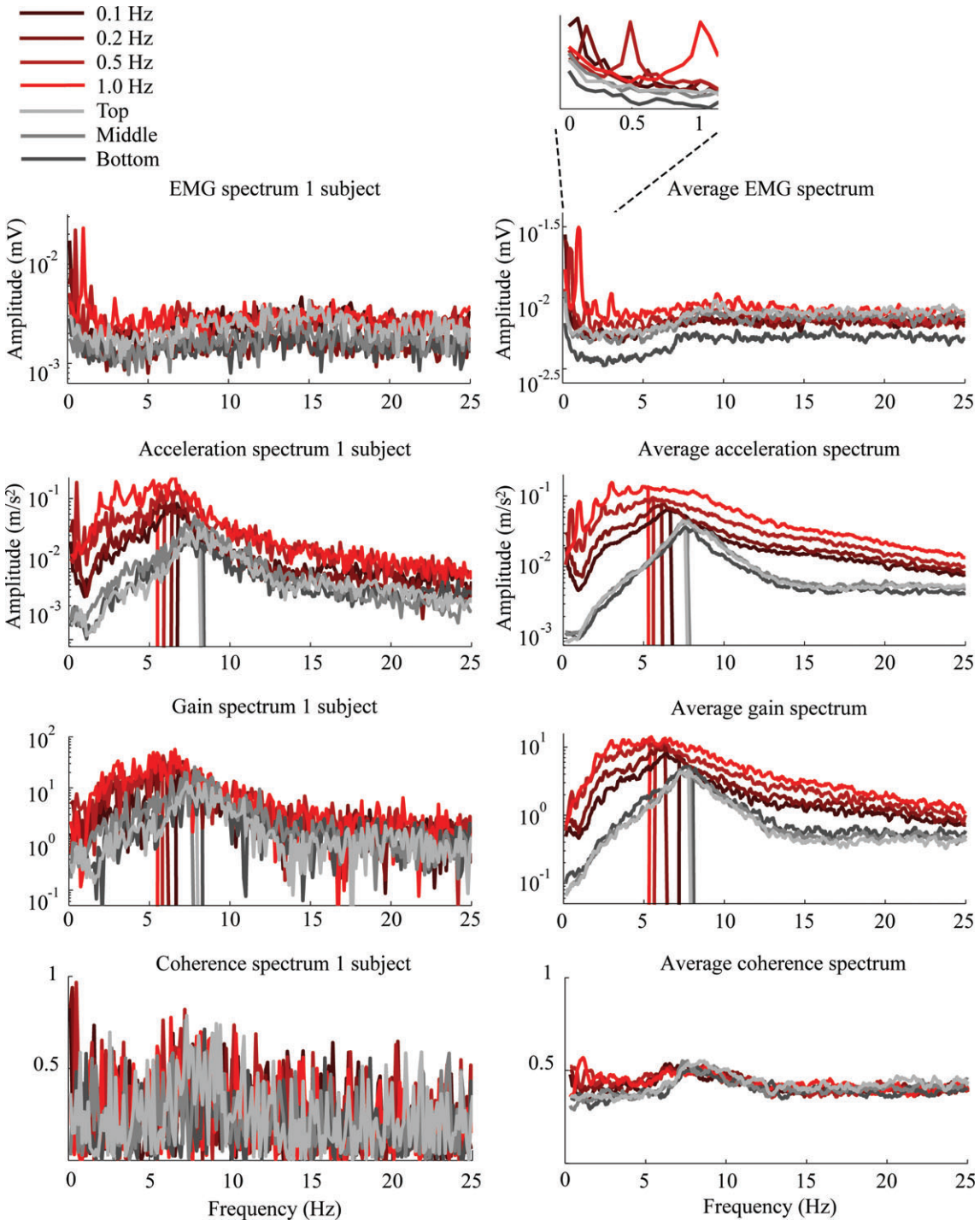


Figure 1. Spectral analysis from a representative subject (left) and all subjects (right)
Grey, postural trials; red, dynamic trials. Top row shows the rectified EMG spectrum of the extensor muscle in the seven different conditions. Second row shows the acceleration spectrum of the hand in the different conditions. Third row shows the gain between rectified EMG and acceleration in the different conditions. The cross-coherence (bottom row) between EMG and acceleration is generally moderate with very high coherence only at the frequency of the voluntary movement in the dynamic conditions. There is slightly elevated coherence in all conditions in a frequency range of approximately 7–13 Hz.

It was clear from Fig. 2 that the peak tremor frequency was different in static and dynamic conditions and this is explored more fully in Fig. 4. This figure shows the frequency at which the acceleration spectrum was maximal (± SD) and the frequency at which the gain was maximal (± SD) in every condition. In effect these values describe the frequency of the tremor. The principal findings of the statistical tests are as follows. (1) The peak frequencies between acceleration and gain in all conditions except for the 1.0 Hz and static bottom condition are not significantly different ($t = -1.000$ to 0.181; $P = 0.331$–0.932). For 1.0 Hz and static bottom condition, acceleration and gain peaks were slightly but significantly different (1.0 Hz, $t = -2.413$; $P = 0.027$: static bottom, $t = -2.650$; $P = 0.017$). These two conditions were considered the least constant since the movement speed was rather high and variable (1.0 Hz) or there was very little EMG present (static bottom). (2) The average static peak frequency of both acceleration and gain was significantly different from the average dynamic peak frequency ($t = -9.856$, $P < 0.001$; $t = -8.713$, $P < 0.001$, respectively). (3) None of the static peak frequencies was significantly different from any of the other static peak frequencies (all $P > 0.99$). (4) The acceleration and gain peaks for the dynamic conditions ranged from statistically different ($P < 0.001$ for acceleration peaks between 0.1 Hz and 1.0 Hz) to not significant (acceleration and gain peaks between 0.5 Hz and 1.0 Hz). This, in combination with Fig. 4, shows that tremor frequency is reduced by movement, reducing a little more as movement speed increases.

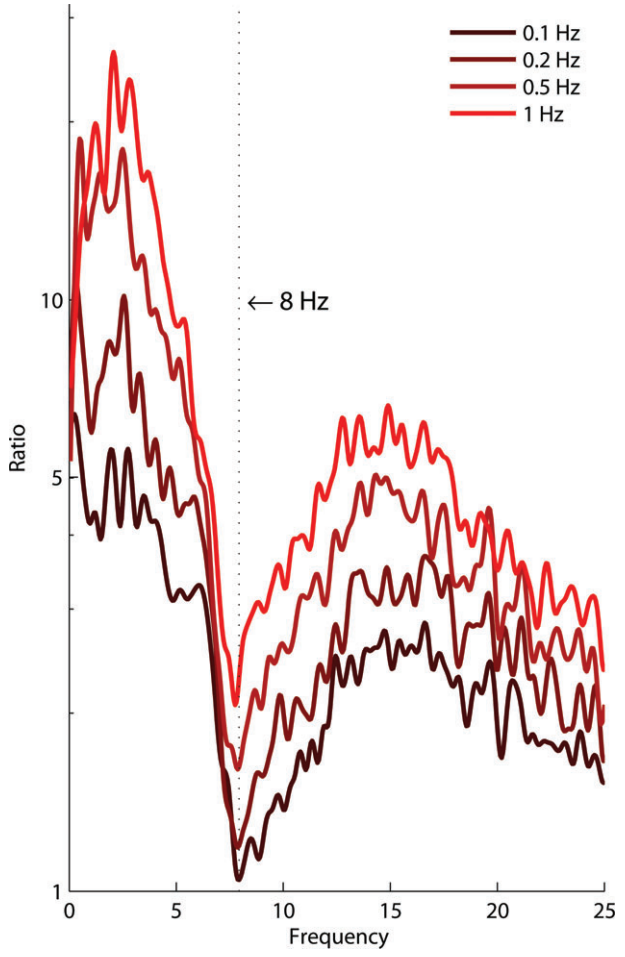


Figure 2. Gain ratios, dynamic *vs*. average static condition
The gain for each of the four dynamic conditions is compared with the mean gain of the three static conditions. The gain ratio is always greater than unity and is highest with the fastest movements. The gain ratios show a local minimum at approximately 7–8 Hz.

Is there a systematic relationship between the tremor during posture and the tremor during movement? This question is addressed in Fig. 5. This figure compares the frequency of tremor in postural and dynamic conditions. For simplicity the values plotted for each subject are the mean of their three postural measurements and the mean of their four dynamic measurements. There is a moderate correlation ($r^2 = 0.38$) between postural and dynamic frequencies. The equation of the best fit linear regression suggests that on average the frequency of dynamic tremor is 2 Hz lower than that of postural tremor.

We aimed to produce a simple model which would recreate the acceleration spectra from the EMG that we recorded. This model and its results are shown in Fig. 6. The top left panel shows the recorded EMG under a postural ('static middle') and dynamic condition (0.2 Hz) for a representative subject. The corresponding recorded tremor acceleration spectra are shown in the right middle panel. It was these acceleration spectra that we aimed to recreate. First, the recorded EMG was used as an input to the model. The acceleration spectra that it produced is shown in the top right panel – the resemblance to the real data is obvious. Second, a simpler input was used. This took the form of white noise, with a flat spectrum. Fed into the same model, it too produced a good approximation of the experimentally recorded acceleration spectrum (bottom right panel). The force spectra show (top) the force that results from passing the recorded EMG through a filter which resembles a muscle and (bottom) the force resulting from similarly filtering a white noise input. The model used published estimates for limb stiffness and inertia. Because it represents an under-damped second order oscillator its resonant frequency is proportional to the square root of these parameters. Therefore the effect of changing either of these values is relatively slight and a broad range of realistic inertia and stiffness values will give a satisfactory match for a representative subject. The effect of changing the damping is almost exclusively an alteration of the height of the resonant peak. The most striking feature of the model was that it successfully produced a realistic small reduction in resonant frequency with

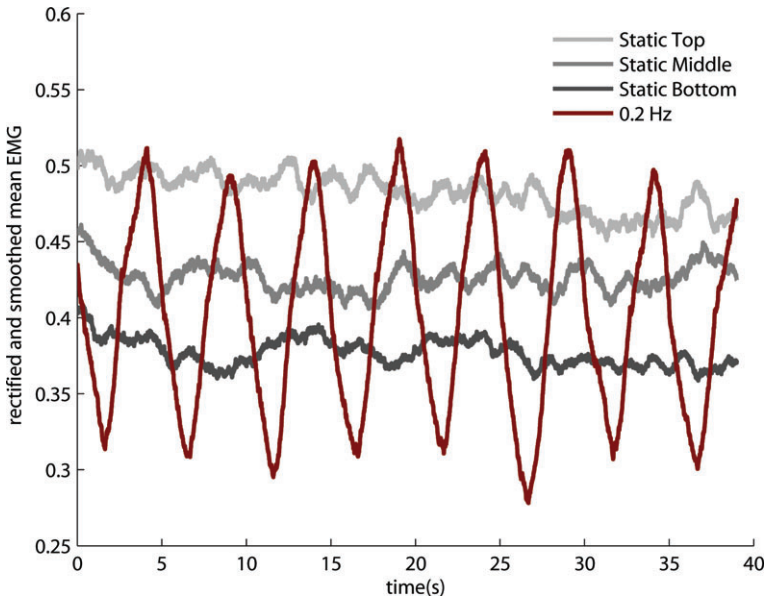


Figure 3. The mean rectified EMG (all subjects) of the extensor muscle in all 3 static conditions and in one of the dynamic conditions (0.2 Hz)
The other 3 dynamic EMG results show a similar range of modulation but are omitted for clarity. EMG has been smoothed by 50 point moving averaging.

movement. The stiffness of skeletal muscle is known to change very considerably with movement and this might have been expected to produce a much larger reduction in resonant frequency. The reason that this does not occur is because of the relatively low and constant stiffness of the series coupled tendon. We employed results from experiments on the ankle. It may be that in the wrist the ratios are different. Accordingly we carried out a form of sensitivity analysis to examine this question (Fig. 7). This figure shows the resonant frequency of the model at different values of stiffness ratio. It shows clearly that a reduction of approximately 2 Hz results from a change in stiffness ratio of 15:1 to 1:1. The upper level of this ratio is not very critical; a completely rigid muscle would produce only a slight elevation in resonant frequency. Furthermore, the muscle stiffness would have to decrease to extremely low values to produce a larger reduction in resonant frequency.

Discussion

The results show the following main features. (1) The rectified EMG spectrum is broad-band and has no distinguishable peak at the tremor frequency. (2) The gain between rectified extensor EMG and acceleration of the hand shows a pronounced peak which coincides with the frequency of the tremor. (3) When posture is replaced by slow movement there are two very clear changes. First, tremor size is greatly increased and this is attributable to an increase in gain, not to an increase in EMG, at the tremor frequency. Second, the peak frequency of hand

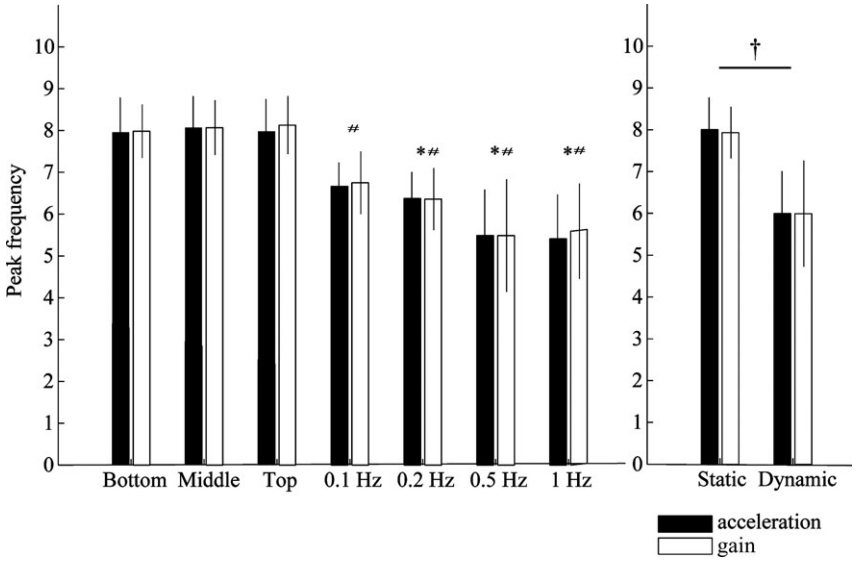


Figure 4. The frequency at which the acceleration spectrum and the gain spectrum are maximal in size ('tremor frequency')
Left, results are pooled from all subjects in each condition. The mean and SD are shown for each condition. Right, the mean (±SD) of all static and all dynamic conditions is also displayed. *Significantly different from 0.1 Hz. #Significantly different from 0.5 Hz. †Significant group difference.

tremor is reduced 2 Hz by movement. These features are reproduced by a simple model. Finally, (4) the relationship of these results to central oscillators is considered and (5) the implications are discussed.

The rectified EMG spectrum is broad-band and has no distinguishable peak at the tremor frequency

In all conditions the average rectified extensor EMG spectra (Fig. 1) are fairly flat. This average does mask a degree of inter-individual variation with some subjects having a broad peak between 10 and 20 Hz. Using an unsophisticated frequency analyser, we previously estimated this mean peak frequency at 14 Hz (Lakie *et al.* 1986) and more recently using wavelet analysis at 12 Hz (Reynolds & Lakie, 2010). Others report very similar wrist tremor EMG spectra; for example, Timmer *et al.* (1998) show a slight peak at ∼15 Hz. Raethjen *et al.* (2000) describe a similar spectrum with an EMG peak normally distributed within a frequency range between 8 and 18 Hz for the hand. These values are generally higher than that of hand tremor. It is probable that the EMG peak represents the firing of individual motor units at these frequencies which are strongly picked up by the surface EMG as it coincides with the likely rate of motor neuronal discharge in these minor postural tasks (Erimaki & Christakos, 2008). Kakuda *et al.* (1999) report firing rates of ∼8–16 Hz. If hand tremor resulted largely from modulated EMG activity there would be a clear peak in the EMG at the frequency of the tremor. For the hand tremor that we have studied this makes it very unlikely that any substantial part of the tremor results from a neural oscillator of central or peripheral (reflex or Renshaw cell) origin. These results do not exclude the possibility that some part of the EMG at the tremor frequency comes from an oscillator of central or other origin. Interestingly, if a neural oscillator were to start operating at an appropriate frequency (perhaps due to a tremor pathology) its mechanical consequences would be magnified by the gain at that frequency.

The inference of the present experiments seems robust. For all of our subjects the large acceleration of tremor is produced by broad-band forcing with no particularly large amount of neural drive at the characteristic tremor frequency. A number of studies have shown that there is a degree of synchronization between tremor and EMG at frequencies around the tremor frequency. However, these studies have employed cross-coherence or cross-correlation of EMG and tremor (discussed in Gantert *et al.* 1992; Timmer *et al.* 1998). The cross-coherence that we show in Fig. 1 is moderate at all frequencies. As the EMG 'drives' the acceleration the result is inevitable. The slight elevation in cross-coherence that occurs around 7–13 Hz in all conditions and at the frequency of movement in the dynamic conditions is almost certainly a feature of the improved signal to noise ratio at these frequencies. While cross-coherence will measure the linear relationship between EMG and mechanical output it cannot express the effect of this correlation in terms of explaining how much of the tremor is due to EMG activity at different frequencies.

The gain between rectified extensor EMG and acceleration of the hand shows a pronounced peak which coincides with the frequency of the tremor.

The gain between EMG and isometric force is well established (Bawa & Stein, 1976) It conforms to that of a second order, low-pass filter so that as excitation frequency increases, the size of the force fluctuations decreases until full fusion of the response occurs and the muscle cannot produce any pulsatile output. However, the gain between rectified EMG and acceleration does not appear to have been previously described. From Newton's second law, acceleration is proportional to force and it might therefore be expected that hand acceleration would behave in exactly the same way as muscle force. The present results show that that is not the case (Fig. 1 third

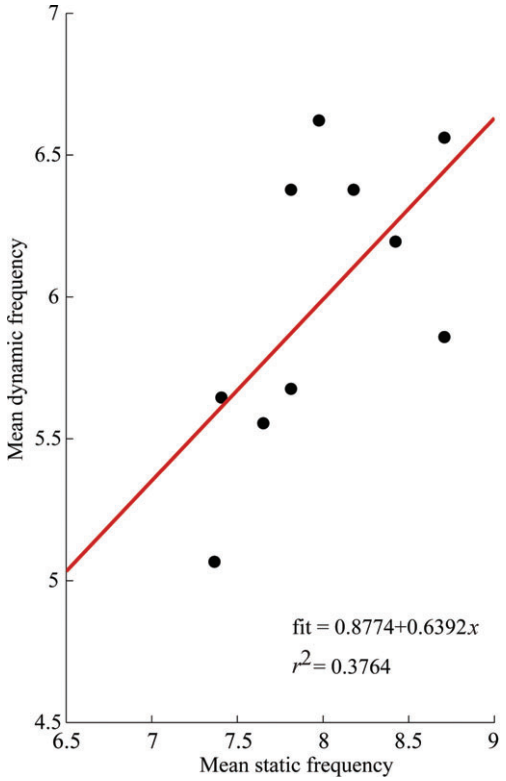


Figure 5. Frequency of the tremor peak under static and dynamic conditions for 10 subjects
The static values are the mean of the three static measurements and the dynamic measurements are the mean of the four dynamic measurements.

row). Gain is low at low frequencies and high frequencies, and has a pronounced peak at approximately 8 Hz in postural conditions. This occurs because the hand to which the accelerometer is fastened acts as a resonant load. The load is susceptible to forcing. Small fluctuations in force close to the resonant frequency will produce exaggerated excursions of the limb at that frequency. The acceleration is the second derivative of that excursion and because the acceleration signal increases with the square of the frequency, the acceleration will be very large at resonance. Thus the shape of the gain spectrum reflects the resonant properties of the hand, muscle and tendon. It is possible to recreate this behaviour using an under-damped second order oscillator model (Fig. 6). The model consists of an inertial load which represents the hand and a spring which reflects the series combination of muscle and tendon. The model produces a realistic 'tremor' spectrum when its input is either extensor muscle EMG or pure white noise. The gain spectra are not statistically distinguishable under bottom, middle or top postural conditions, all of which show a peak at approximately 8 Hz. This value is similar to those typically reported for hand tremor (Marsden *et al.* 1969; Lakie, 1992; Raethjen *et al.* 2000; Reynolds & Lakie 2010). Changes in static posture are not sufficient to alter tremor frequency. The inertia reflects the mass of the moving parts and is unlikely to change. The stiffness represents primarily the elasticity of the muscles, tendons and ligaments which cross the joint because the stiffness of the joint itself is trivial (Johns & Wright, 1962). The main cause of the stiffness lies in the series combination of the muscles and tendons (Loram *et al.* 2007). For the differences in degree of activation of the muscle required to maintain a 'top', 'middle' or 'bottom' posture (Fig. 4) by counteracting gravitational forces the stiffness differences are negligible and the tremor frequency does not change.

Slow movement systematically alters tremor

The relationship between postural tremor and tremor during movement has not been very extensively studied. Examining the discontinuities that occurred during

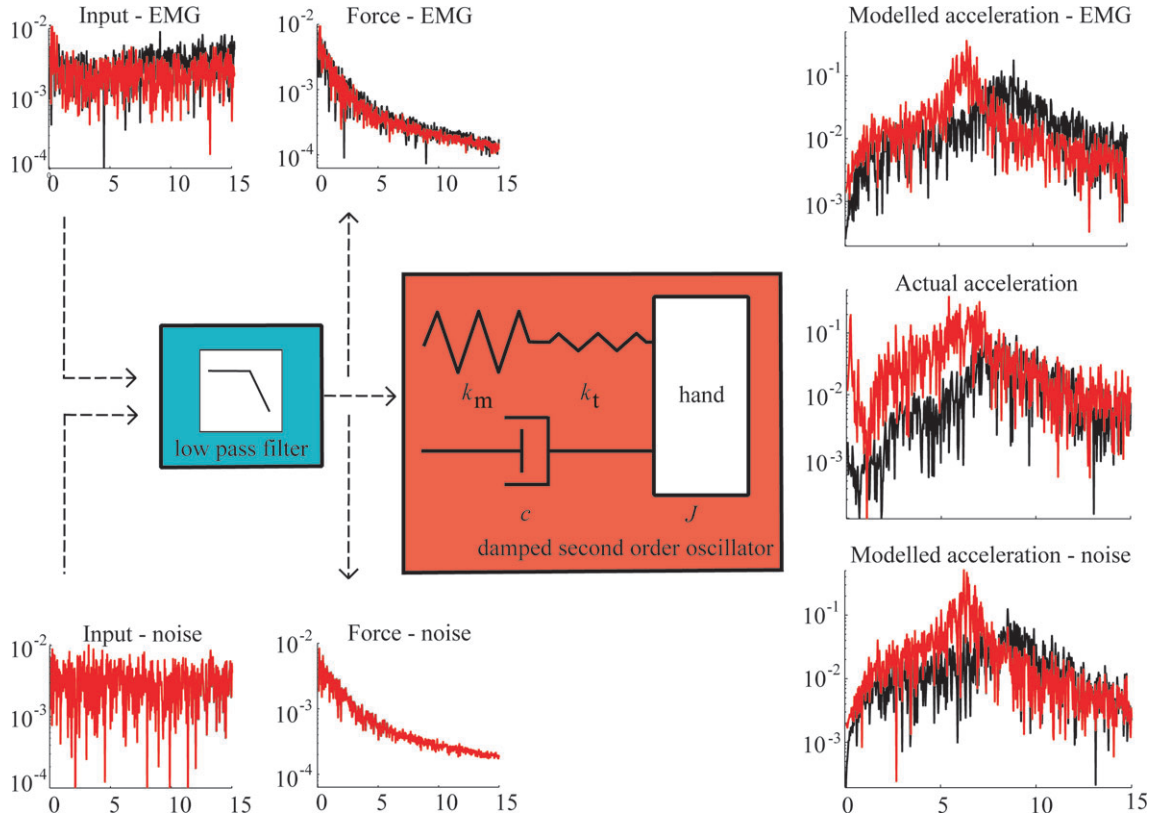


Figure 6. Model results
Black, static conditions; red, dynamic conditions. Left panels show the input signals; real EMG (top: black, static; middle: red, 0.2 Hz), or white noise (bottom). Middle panels show the force spectra that are produced by the model 'muscle'. Right panels show the acceleration spectra produced by the oscillator model (top and bottom). These are compared with the actual tremor acceleration spectrum (centre). Reducing muscle stiffness by a factor of 15 and damping by a factor of 3 has produced a resonance (acceleration peak) that is ~2 Hz lower and ~10 times bigger. The model does not recreate the low frequency (0.2 Hz) peak in the acceleration spectrum. This peak results mainly from accelerometer tilt in the real data and is accordingly not reproduced by the model.

slow movements of the finger, Vallbo & Wessberg (1993) concluded that although there was a resemblance to tremor the discontinuities represented something different. However, these authors did not carry out a direct comparison of the postural tremor and dynamic tremor of their subjects. Kakuda *et al.* (1999) observed that the size of hand tremor was considerably larger during movement than when stationary and they predicted that the frequency would be lower. Recently, Daneault *et al.* (2011) showed that during slow random tracking movements finger tremor amplitude increased by a factor of 4. They also showed that the median power frequency of the tremor reduced from 9.70 Hz in the static condition to 6.45 in the tracking condition.

Here we confirm that tremor size is greatly increased during slow movements. As the movement velocity increases up to a certain limit, the tremor size increases further. We studied slow movements; 0.1 Hz corresponds to a target velocity of only 6 deg s^{-1}. Naturally, in performing the task, the subject's hand velocity will sometimes exceed this value and sometimes lag behind it. Figure 1 strongly suggests that there is a critical zone in which gain increases as movement speed increases. In maintaining a posture a limb is never perfectly still so the velocity is only transiently zero. We suggest that as the mean speed increases there is a rapid and progressive increase in gain coupling EMG to acceleration until a velocity of approximately 30 deg s^{-1} is attained (0.5 Hz) when no further large increase in gain occurs.

Why does this increase in gain occur? In making these slow and low force movements only a small number of motor units will be required. Figure 3 shows that the range of EMG is comparable in dynamic and static conditions. In order to move, muscle must overcome its own stiffness and the stiffness of the surrounding passive muscle fibres. In the case of the hand there are also numerous relaxed synergistic and antagonistic muscles to consider. During (and for some time after) movement, the resistance to movement of these muscles is considerably reduced, a phenomenon known as muscular thixotropy which has been demonstrated in a range of human limb muscles (Lakie *et al.* 1986; Hufschmidt & Schwaller, 1987; Lakie & Robson, 1988; Deserres & Milner, 1991; Proske *et al.* 1993; Axelson & Hagbarth, 2001). We suggest that, during movement, muscle stiffness decreases, producing both the considerable increase in size and the modest drop in frequency that is observed during movement. The modest drop in frequency is of particular interest because the stiffness decrease of muscle during movement is very large. This has been shown by numerous studies in a range of *in vitro* and *in vivo* preparations. In the model we used a reduction factor of 15, derived from ultrasound calf muscle measurements (Loram *et al.* 2007). Resonant frequency is proportional to the square root of stiffness so it might seem that this would produce a frequency reduction of close to fourfold rather than the ∼25% reduction which is observed. The reason for this is the unchanging and relatively low value of tendon stiffness which mitigates the overall stiffness decrease that occurs. The effect of altering the reduction factor is shown in Fig. 7. The increase in size and decrease in frequency of tremor during movement are similar to, but more pronounced than, the changes described immediately following movement (Reynolds & Lakie, 2010). This is because thixotropic muscle effects begin to reduce very rapidly as soon as movement ceases (Lakie & Robson, 1988).

We have correlated tremor frequencies in postural and moving conditions Fig. 5. The correlation suggests that there is a shift in frequency in each individual so that his or her tremor frequency drops by ∼2 Hz during movement. The imperfection of the correlation is no surprise. Small variations in the spectrum from trial to trial introduce a degree of variability in the frequency of maximal oscillation. However, the systematic shift resulting from movement strongly suggests that hand tremor arises from a single process which is systematically altered by movement. The model that we demonstrate in Fig. 6 produces rather sharp frequency peaks as would be expected for a lightly damped second order oscillator driven by a random input. Real tremor is less sharply tuned. The reason is straightforward: the model uses a single fixed value of stiffness for static conditions and a single fixed value of stiffness for moving conditions. The reality must be an ever-shifting value of stiffness which

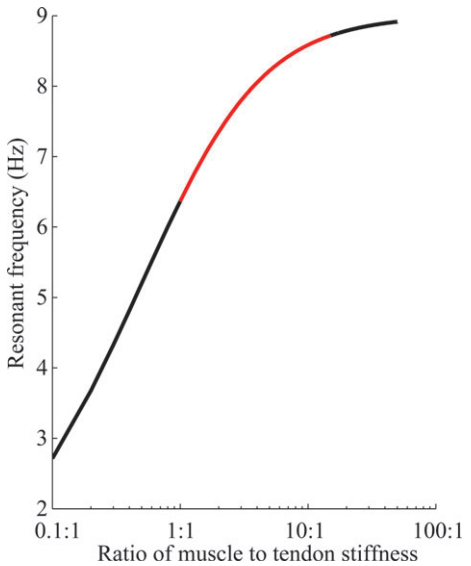


Figure 7. Stiffness ratio sensitivity analysis
This shows the effect on the resonant frequency of the model of adjusting the ratio muscle stiffness:tendon stiffness. In postural conditions we have estimated this ratio as 15:1 and in movement we estimate it as 1:1. These limits are indicated by the grey area (red online).

reflects a parameter such as the mean muscle speed. More sophisticated models would work on this basis and would create broader and more variable frequency peaks.

There is nothing in the EMG spectrum during movement that suggests the emergence of a neural oscillator acting at the frequency of the tremor. The EMG spectrum is very similar to the spectrum in postural conditions. As the EMG does not change much and the acceleration changes considerably the simplest explanation is an alteration in the way in which muscle transduces EMG into acceleration.

How do these results relate to central oscillators?

Could the large tremor during movement represent something different from postural tremor? Looking at finger movements, Vallbo and colleagues (Vallbo & Wessberg, 1993; Wessberg & Vallbo, 1996) have determined that the discontinuities represent purposive intermittent adjustments in the neural drive to finger muscles that occur during slow movement. Kakuda *et al.* (1999) have extended this idea to hand movement. They have shown that during hand movement, but not posture, there is increased coherence between simultaneously sampled motor units in the hand extensor muscles and also between individual motor units and acceleration.

There has been a tendency to attribute the coherence to common descending central drive of motor neurones and there is an ongoing search for central oscillators operating at the appropriate frequency(ies) during movement. Our results for the wrist suggest an alternative interpretation. The fact that, during movement, there is a large resonant, asynchronously driven tremor implies that some common input to the motor units of reflex origin is perhaps more likely, with synchronizing volleys of spindle activity perhaps inevitably produced at the tremor frequency during movement but not in posture. Thus any coherence close to the tremor frequency may be a consequence of the tremor oscillation rather than its cause. In this respect, arguments about the phase shift or loop delay of such feedback pathways are not very relevant. Driving of the EMG as a consequence of movement may be destabilizing or stabilizing, but so long as the reflex gain is low it will not sustain an oscillation on its own (for example as proposed by Lippold, 1970; Durbaba *et al.* 2005). It is possible that the slight coherence between motor neurones or between motor neurones and tremor is a general phenomenon produced by resonance and spindle drive rather than by a central oscillator. Christakos *et al.* (2006) have shown that coherence might result from very small fluctuations in spindle length even in pseudo-isometric conditions. Such coherence would naturally be enhanced during movement when, as seen here, the resonance becomes much larger.

What are the implications of these new findings?

Tremor size increases even during slow movement. This may provide an explanation for the wide range of tremor sizes observed in healthy subjects (Lakie, 1992). It is a testable hypothesis that larger physiological tremors are observed in subjects who are less able to keep still. The reduction of tremor frequency as tremor size increases is consistent with observations of normal physiological tremor where it is a general finding that as tremor size increases its frequency decreases (Lakie, 1992). There is also the possibility of a form of positive feedback where movement caused by tremor might generate further tremor thus giving rise to an enhanced or possibly essential tremor. The results also have more general implications for motor control. They show that even slow movements have a very large effect on the effectiveness of coupling EMG activity to output. It has been previously suggested that muscle thixotropy produces a situation where the nervous system is confronted by mechanical behaviour which is very difficult or perhaps impossible to predict because it depends on the history of movement and the presence or absence of other unassociated small limb movements (Lakie & Robson, 1988; Axelson & Hagbarth, 2001). A neural control system can compensate by using feedback. If feedback is too slow then predictive (internal model based) mechanisms can be used. The fickle nature of muscle must make such prediction very difficult and may favour a form of control such as 'bang-bang' or intermittent trial and error in order to overcome this severe non-linearity in response.

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Author contributions

All experiments were performed in the human neurophysiology laboratory in the School of Sport and Exercise Sciences, University of Birmingham. All authors contributed to the conception and design of the experiments, the collection, analysis and interpretation of data, and to drafting and revising the paper, and they affirm that they have approved the final version of the manuscript.

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