INTRODUCTION

The origin of physiological tremor remains somewhat contentious. Neural explanations include central oscillatory activity (Conway et al. 1995), motor unit firing properties (Dietz et al. 1976; Elble and Randall 1976), and reflex loops (Hagbarth and Young 1979; Lippold 1970) (see McAuley and Marsden 2000 for review). However, there is strong evidence that mechanical resonance of the joints plays an important role in tremor genesis. Adding mass to the unsupported limb to increase its inertia causes a shift of the tremor power spectrum to lower frequencies (Elble and Randall 1978; Homberg et al. 1984; Joyce and Rack 1974; Stiles and Randall 1967). This suggests that any change in the resonant frequency of a joint will induce a concomitant change in tremor frequency. Resonant frequency can also be altered by manipulating joint stiffness, as well as inertia. Because muscle tissue contributes to joint stiffness, any changes in the mechanical properties of the muscle may therefore affect tremor frequency and amplitude (Lakie et al. 1986b).

It has been established both in vitro and in vivo that muscle stiffness is influenced by movement history. For example, the resistance of isolated muscle fibers to stretching is reduced by a prior stretch (Lakie and Robson 1990; Warner and Wiegner 2001). If the passive muscle is left still for some time, it gradually recovers its stiffness. This property has been termed muscle thixotropy and may be caused by gradual spontaneous reattachment of actin-myosin crossbridges (Campbell and Lakie 1998). The same effect has been shown in humans. Active or passive joint movement causes a temporary reduction in limb stiffness, whether measured directly or inferred from the resonant frequency (Hufschmidt and Schwaller 1987; Lakie and Robson 1988; Lakie et al. 1984). This suggests that prior movement may reduce the frequency and increase the amplitude of physiological tremor, by transiently reducing joint stiffness. This hypothesis is confirmed here by monitoring changes in tremor frequency and amplitude after voluntary movement.

METHODS

Subject recruitment

Eight right-handed subjects (24–55 yr; 7 male) gave informed consent to participate in this study. Ethical approval was received from The School of Sport and Exercise Sciences at The University of Birmingham, and the experiment was conducted in accordance with the Declaration of Helsinki.

Protocol and apparatus

Subjects sat with their forearm supported, with the outstretched prone hand unsupported (Fig. 1A). Hand tremor was recorded using an accelerometer placed at the end of the middle finger. The fingers were adducted so the hand and fingers moved as a single unit. A retroreflective laser range finder (Wenglor Sensoric) was pointed at the proximal end of the hand, and this was used to provide visual feedback of wrist angle on a large screen oscilloscope placed 1.5 m in front of the subject. To calibrate this signal, the wrist was positioned at known angles (20° up and down) using an inclinometer. EMG was recorded from the extensor digitorum communis muscle (Bagnoli, Delsys). All signals were digitized at 1 kHz using a USB-6229 data acquisition module (National Instruments).

The voluntary wrist movement consisted of a series of three consecutive extension-flexion actions, followed by a return to the original hand position (Fig. 1B). An acoustic stimulus was used to guide the movement, consisting of a series of high- and low-pitch
beeps lasting 1.8 s. This was preceded by a lower-pitch warning tone lasting 1 s. The mean range and peak velocity of the voluntary movement was 84°/s and 419°/s (SD), respectively. Subjects were instructed to arrest hand movement precisely at the time of the last beep and to hold the hand still until the following movement was cued. Practice trials were given to ensure that subjects could time their movement appropriately. Sounds were presented every 20 s. Visual feedback of hand position on the oscilloscope encouraged them to remain still during the postmovement phase. Each subject experienced four blocks of 12 movements with interleaved rest periods, providing a total of 48 trials.

Data analysis

Wrist angle was differentiated to obtain velocity, and movement cessation was defined as 0.5 s after wrist velocity fell below 5°/s. The data were split into movement and postmovement sections lasting 3.5 and 14 s, respectively. Recently it has been suggested that EMG rectification may impair identification of oscillatory muscle activity (Neto and Christou 2010), although some authors suggest the opposite (Halliday and Farmer 2010; Myers et al. 2003). Therefore we examined the effect of rectification on our data. We found that, if EMG was not rectified, the wavelet analysis was unable to reliably identify oscillatory activity at the movement frequency (~2 Hz; Fig. 2B). Furthermore, the 12 Hz peak during the postmovement period was apparent only on rectified EMG (Fig. 3B). These differences were confirmed by calculating power spectra with conventional Fourier methods. Therefore we chose to rectify EMG data. The rectified EMG and acceleration signals were low-pass filtered (30 Hz, 4th order, 0-phase butterworth) and down-sampled to 100 Hz. EMG was normalized so that the total root mean square value of each subjects’ concatenated time series amounted to 1. Acceleration signals were mean-removed.

The resulting time series were converted into time-frequency representations using the continuous wavelet transform (CWT). The CWT is a method for analyzing nonstationary time series and has previously been used to analyze neurophysiological signals including tremor (Gilbertson et al. 2005; Samar et al. 1999). The CWT is the sum over all time of the signal multiplied by scaled shifted versions of the mother wavelet function (ψ). The wavelet coefficient of a signal s at scale a and position b is defined by

\[ C_{ab} = \int s(t) \frac{1}{a} \psi^\dagger(\frac{t - b}{a}) dt \]

where * denotes the complex conjugation. The chosen mother wavelet was a complex morlet, with a bandwidth parameter (fb) value of 1 and central frequency (fc) of 1.5

\[ \psi(\chi) = \frac{1}{\sqrt{\pi/b}} e^{2i\chi^2/\sigma^2} e^{-\chi^2/2b^2} \]

where \( i = \sqrt{-1} \). The mother wavelet was shifted in scale to correspond to frequencies of 1–25 Hz for the movement period and 5–25 Hz for the postmovement period. Wavelet power was averaged across trials. To quantify changes in signal amplitude and frequency over time, wavelet power was averaged into 0.5 and 1 s time bins during the movement and postmovement periods, respectively. Peak frequency and power were calculated for each time bin.

To determine time-dependent changes during the postmovement period, the first and last time bins were compared with repeated-measures t-test. \( P < 0.05 \) was considered significant. Data were fitted.
with exponential fits to determine time constants of postmovement changes. All errors reported in the text are SD, whereas those in figures are SE.

RESULTS

Voluntary movement

The CWTs showed distinct peaks of power during the voluntary wrist movement (Fig. 2, A and B). These peaks correspond to frequencies of 1.97 ± 0.17 and 1.91 ± 0.17 Hz for acceleration and EMG, respectively. Peak acceleration power lagged EMG by 118 ± 57 ms. However, the evolution of power and frequency throughout the movement is strikingly similar for acceleration and EMG (Fig. 2, C and D).

Postmovement period

CWTs for the postmovement phase are shown in Fig. 3, A and B. Compared with the movement phase, wavelet power is ~4 and 2 orders of magnitude smaller for acceleration and EMG, respectively. Clear peaks in frequency were identifiable on both signals. At 14 s, peak acceleration frequency was 8.0 ± 0.80 Hz, whereas EMG frequency was significantly higher at 11.9 ± 2.5 Hz (t = 3.8, P < 0.001). The 12 Hz EMG peak is likely to reflect motor unit firing rates (Elble and Randall 1976; Halliday et al. 1999). The key question is whether there are time-dependent changes in tremor after the movement. The dashed trace in Fig. 3C shows that there is an increase in acceleration peak frequency over the 14 s period postmovement, from 7.2 ± 0.64 to 8.0 ± 0.80 Hz (comparison of 1st and last time point: t = 6.9, P < 0.001). This is paralleled by a reduction in acceleration power from 0.076 ± 0.032 to 0.025 ± 0.020 a.u. (solid trace; t = 4.2, P = 0.004). Fitting each subjects’ data with exponential curves provided time constant estimates of 3.56 ± 2.0 and 2.03 ± 0.88 s for changes in frequency and power, respectively.

In contrast to acceleration, EMG showed no significant changes in either frequency or power (Fig. 3D; t < 1.1, P > 0.3).

DISCUSSION

These results show postmovement changes in tremor that cannot be explained by altered muscle activity. After a voluntary wrist movement, hand acceleration displayed a transient increase in amplitude and a decrease in frequency, both of which recovered progressively during the rest period. Wrist extensor EMG was entirely different from acceleration, showing no significant changes in power or frequency during the rest period. Furthermore, the overall peak frequencies of EMG and acceleration were quite different (~12 and 8 Hz, respectively), in contrast with the movement period itself. Therefore the observed changes in hand tremor after movement cannot be attributed to alterations in neural output.

Physiological tremor has been attributed to central neural oscillations (Conway et al. 1995), reflex oscillations (Hagbarth and Young 1979; Lippold 1970), and firing properties of motor units (Dietz et al. 1976; Elble and Randall 1976) (see McAuley and Marsden 2000 for review). These explanations cannot account for the postmovement changes in tremor observed here, because there were no concomitant changes in EMG amplitude or frequency. In the absence of neural explanations, changes in mechanical factors must be responsible for the observed effect. The main evidence for a mechanical contribution to tremor comes from experiments in which limb inertia is artificially increased by addition of mass, producing a reduction in tremor frequency (Elble and Randall 1978; Homberg et al. 1987; Joyce and Rack 1974; Stiles and Randall 1967). This suggests that the resonant frequency of a joint determines tremor frequency (Lakie et al. 1986b). Anything that can influence the stiffness of a joint, including muscle tissue, will also affect its resonant frequency.

Converging evidence from animal and human experiments shows that muscle stiffness is affected by prior movement. Isolated animal muscle tissue exhibits less resistance to a stretch when it is preceded by a prior stretch (Lakie and Robson 1990; Warner and Wiegener 1990; Whitehead et al. 2001). Thixotropic effects have also been shown in the human finger, wrist, and ankle joints (Hufschmidt and Schwaller 1987; Lakie and Robson 1988; Lakie et al. 1986a). This has known consequences for motor control (Axelson and Hagbarth 2001) and muscle proprioception (Hagbarth et al. 1987), requiring the nervous system to adapt its output to compensate for reduced stiffness. Our results extend these findings to show that thixotropic effects are also observable in human subjects.

FIG. 3. Time-frequency representation of postmovement period. Mean continuous wavelet transforms are shown for hand acceleration (A) and extensor EMG (B) after voluntary wrist movement. The frequency (dashed) and magnitude (solid) of peak wavelet power is shown for 1 s successive time bins during the postmovement period in C and D.
otropy also has consequences for physiological tremor. The postmovement changes in tremor occurred with a time constant of 2–4 s. This is comparable to previously reported time constants of thixotropic changes following movement, which generally range between 2 and 10 s (Campbell and Moss 2000; Hufschmidt and Schwallier 1987; Whitehead et al. 2001).

A constant level of wrist extensor activity was required to maintain the hand position against gravity, but this was presumably a small percentage of a maximum voluntary contraction. Therefore the muscle consisted of a mixture of active and relaxed fibers during the postmovement phase. What implications does this have for the effects on tremor? Thixotropy has been shown in both active and relaxed muscle (Campbell and Moss 2000). However, isolated muscle experiments suggest that the time course of stiffness recovery may be faster in contracting muscle tissue (Campbell and Moss 2002). Whether this is true in vivo remains to be seen.

Thixotropic properties of muscle tissue may be ideal for promoting stability during static postures while allowing for movement when necessary. Our results show that tremor gradually reduces when the hand is kept still. This may have consequences for pathological tremors, such as occurs in Parkinson’s disease. The ongoing large limb movements may prevent muscles achieving increased stiffness. This may result in a “double-whammy” effect—a primary tremor of neurological origin exacerbated by increased tremor caused by reduced muscle stiffness.

In summary, we showed that voluntary movement causes a transient reduction in the frequency of hand tremor, accompanied by an increase in amplitude. This effect cannot be explained by changes in neural output. The most likely cause is a temporary reduction in joint stiffness caused by muscle thixotropy, reducing the resonant frequency of the hand.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

REFERENCES


