

## C14

**High frequency force tremor components are changed by local heating or cooling in man**

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Force tremor analysed using Fourier techniques has revealed low frequency volitional fluctuations, with a peak around 1 Hz. Furthermore, the major part of spectral power is contained below 4 Hz (Vaillancourt *et al.* 2002). Higher frequency components also exist within the data, but due to their small magnitude and aperiodic nature are difficult to assess using the fast Fourier transform (FFT) method and have therefore received little attention. These 'noise' components presumably represent unfused events during contractions and thus provide indirect information regarding muscle state during force maintenance tasks. Assuming this is the case, then altering the contractile properties of the muscle should modulate their amplitude.

We tested this by recording isometric force tremor during abduction of the index finger, in eight subjects at 20% of maximal voluntary force, before and after 10 min of hand cooling (10°C) or warming (44°C) by water immersion. This has previously been shown to rapidly change the contractile properties of the first dorsal interosseous muscle (Ranatunga *et al.* 1987). The high frequency 'noise' was decoupled from the volitional component and a time-domain interval technique was used to analyse the data.

The results revealed that, in all subjects, muscle cooling significantly reduced the amplitude of the noise by ~70%; conversely heating significantly increased the amplitude by ~50%. In addition, some subjects were tested with eyes open or closed and this was found to have no effect on the amplitude of the noise ( $P < 0.05$  in all cases, repeated measures analysis). Changes produced by cooling lasted longer than those produced by heating. These results support the suggestion by Lakie *et al.* (1994) that temperature related changes in isotonically recorded postural hand tremor were due to altered muscle properties.

We suggest that applying a simple interval technique to look at the instantaneous changes in the high frequency 'noise' component of force tremor can assist in understanding of the mechanisms by which tremor size can be altered and allow them to be differentiated.

Lakie M *et al.* (1994). *J Neurol Neurosurg Psychiatr* 57, 35–42.Ranatunga KW *et al.* (1987). *J Physiol* 390, 383–395.Vaillancourt DE *et al.* (2002). *Clin Neurophysiol* 113, 1325–1338.

*All procedures accord with current local guidelines and the Declaration of Helsinki.*

## C15

**The relationship between muscle force fluctuation and a  $\beta_2$  agonist in man – the role of extracellular  $K^+$** 

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Adrenergic agonists increase tremor size. The effect has been attributed to changes in the contractile properties of muscle rather than changes in the neural control system (Marsden *et al.* 1967). Here we use an isometric method of recording tremor to show that an adrenergic drug reduces stability and that this is associated with a clear change in the contractile characteristics of the muscle. Also, stability appears to correlate directly with the plasma potassium concentration.

We recorded tremor in an isometric force maintenance task. Subjects used the left first dorsal interosseus muscle to maintain a static force equal to 20% of the maximal voluntary force. Tremor was recorded under control conditions and following infusion of the  $\beta_2$  agonist drug, terbutaline (Bricanyl) at  $8 \mu\text{g kg}^{-1} \text{h}^{-1}$  by a cannula inserted into the left ante-cubital vein for a period of 45 min. A cannula was inserted in to the right ante-cubital fossa for venous blood sampling. The subject relaxed for a period of 45 min during which three control measurements of tremor were made and venous blood samples were taken.

Blood samples were processed and stored at  $-80^\circ\text{C}$  and were subsequently analysed for plasma  $K^+$  (indirect ion-specific electrode). In these experiments there were a number of infusions of different drugs and placebos with each subject being infused on five occasions. The subject was blind to the agent being infused.

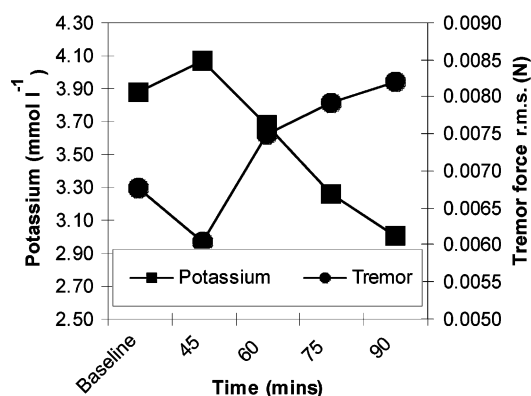


Figure 1. The variation of tremor size and potassium with time

Figure 1 shows the relationship between tremor size (r.m.s force fluctuation) and plasma concentration of  $K^+$ . Repeated measures analysis showed that terbutaline infusion produced a significant decrease in postural stability ( $P < 0.05$ ). Tremor size and plasma  $K^+$  vary reciprocally. This result is similar to that of Fowler & Lipworth (2001) who suggested that tremor measurement or plasma  $K^+$  could equivalently be used as a surrogate for the pharmacokinetic profile of inhaled salbutamol.

Our conclusion is that terbutaline increases tremor size by changing the contractile characteristics of muscle. The effect is traditionally attributed to  $\beta_2$  adrenoreceptors in skeletal muscle.

We alternatively suggest that the mechanism may be linked to interstitial  $K^+$  concentration, which directly affects the activation of skeletal muscle by decreasing the conductivity of the T tubules. We tentatively suggest that other factors, which influence tremor size, may involve this link.

Marsden CD *et al.* (1967). *Clin Sci* **33**, 53–65.

Fowler SJ & Lipworth BJ (2001). *Br J Clin Pharmacol* **51**, 359–362.

*All procedures accord with current local guidelines and the Declaration of Helsinki.*

We conclude that the inclination of the body in the gravitational field has a substantial influence on the magnitude of postural aftercontractions.

Brice T & McDonagh M (2001). *J Physiol* **536.P**, 51P.

Forbes A *et al.* (1926). *Am J Physiol* **781**, 81–103.

*All procedures accord with current local guidelines and the Declaration of Helsinki.*

## PC46

### The strength of postural aftercontractions varies with the inclination of the body in the gravitational field

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Postural after contractions are involuntary contractions that occur predominantly in proximal muscles following a prolonged, strong, voluntary, isometric contraction of the same muscle (Forbes *et al.* 1926). Aftercontractions can be easily elicited in m. deltoid when the subject is standing vertically (Brice & McDonagh, 2001). The mechanism underlying these contractions is unknown. One hypothesis is that aftercontractions are driven by a control system that seeks to maintain the arm position against the gravitational torque experienced at that position. An alternative view is that gravitational influences are irrelevant. We reasoned that if the latter were true, aftercontractions should be of the same magnitude with the subject standing or horizontal.

Nine subjects, five of whom were female, took part in the experiment which had local ethical committee approval and the written consent of the subjects (means  $\pm$  s.d.: age  $21 \pm 0.9$  years; height  $173.4 \pm 8.2$  cm; weight  $73.5 \pm 10.2$  kg). Subjects were asked to produce a 1 min long isometric contraction of m. deltoid equal to 60 % of their maximal voluntary contraction (MVC) against a force transducer. They could see the force they were producing on one channel of a monitor screen and were asked to match this to a target equal to 60 % of their MVC that was also shown on the screen. When necessary the subjects wore prism glasses so that they could still view the screen. At the end of the voluntary contraction the subjects closed their eyes. Then a pneumatic ram withdrew the force transducer, and involuntary abduction of the arm ensued. The subjects lay supine on a tilt table that allowed experiments to be carried out with the subjects body at inclinations of 0, 15, 30, 45, 60, 75 and 90 (head uppermost) deg to the horizontal. At inclinations less than 90 deg the subjects' arms were supported by slings such that arm abduction was free to move only in the coronal plane of the body.

The results showed that aftercontractions were almost four times stronger with the subject standing than they were with the subject horizontal. There was an approximately linear relationship between aftercontraction EMG amplitude and the angle at which the body was inclined. To normalise the data across subjects, the maximum rectified surface EMG amplitudes from m. deltoid during the aftercontraction were expressed as a ratio of the EMG amplitude found during the preceding voluntary contraction. The mean EMG data for the group of nine subjects at each body inclination angle were: means  $\pm$  s.d.: 0 deg  $12.7 \pm 23.6\%$ ; 15 deg  $12.1 \pm 2.4\%^*$ ; 30 deg  $23.3 \pm 9.7\%^*$ ; 45 deg  $29.5 \pm 21.9\%^*$ ; 60 deg  $30.3 \pm 21.0\%^*$ ; 75 deg  $39.1 \pm 22.7\%^*$ ; 90 deg  $46.6 \pm 24.5\%^*$ . Values significantly greater than the value at zero degrees of body inclination are shown as  $*P < 0.05$ ,  $**P < 0.01$  (repeated measures ANOVA with LSD *post hoc*).