Pulsatile central nervous control of human movement

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The findings of Wessberg & Kakuda reported in the current issue of *The Journal of Physiology* represent another important piece in the emerging picture that constant velocity (4–62 degree s^{-1}) finger movements are produced by pulsatile discontinuities in the descending motor command. The findings provide further evidence in man that the firing of motor units is modulated by centrally derived rhythms which are of physiological significance.

For a number of years the Göteborg group have been studying the discontinuities that accompany slow finger movements in man. The initial observations of this phenomenon were made over a century ago when it was noted that small discontinuities appear in the finger position trace despite the fact that subjects try their hardest to produce a completely smooth slow movement. Until the landmark paper of Valbo & Wessberg (1993) it was generally assumed that these ∼8–10 Hz discontinuities represent physiological tremor imposed on movement. Valbo & Wessberg (1993) argued that the mechanical characteristics of movement related discontinuities were different from those of tremor. Rather than there being a small sinusoidal oscillation about a neutral point, the peaks of acceleration and deceleration are asymmetric, of large amplitude and interspersed, during very slow movements, by periods of movement standstill.

Analysis of the EMG during movement has revealed bursts at ∼8–10 Hz intervals occurring close to the peak acceleration when the muscle acts as agonist and close to peak deceleration when the muscle acts as antagonist. Furthermore, an alternating pattern of EMG suggests that the minute movement steps are achieved through pulsatile agonist–antagonist activity. Subsequent experiments by the group have demonstrated that the firing of group Ia, Ib and II reflex afferents is modulated by the ∼8–10 Hz discontinuities, suggesting that the spinal reflex circuitry resonates with the signal (Wessberg & Valbo, 1995). However, the temporal relationships between EMG, acceleration and afferent firing do not support a primary role for spinal and supraspinal stretch reflexes in actually generating the discontinuities (Wessberg & Valbo, 1996).

Whilst the various lines of evidence have been converging in support of a centrally imposed ∼8–10 Hz signal, the possibility has remained that discontinuities during movement are the result of sub-tetanic motor unit firing at ∼8–10 Hz. This hypothesis has been examined directly by Wessberg & Kakuda (1999) through calculation of the coherence between simultaneous recordings of single motor unit activity and the acceleration signal during different velocities of movement. Their results show ∼8–10 Hz coherence between motor unit firing and the acceleration, indicating that the two signals are co-modulated. The frequency of the coherence is the same as that of the movement discontinuities but, importantly, it is quite different from the motor unit firing rate. Thus it is the temporal modulation pattern rather than the motor unit firing rate that is instrumental in the production of movement discontinuity. Whilst to date pairs of motor units have not been studied with this protocol, it seems likely that during movement an ∼8–10 Hz signal is imposed on the motoneurone pool producing ∼8–10 Hz co-modulation of individual motor units that may be firing at different rates. This co-modulation is then expressed as movement discontinuity.

In the context of the previous data describing out-of-phase co-modulation of agonist and antagonist motoneurones, an interesting model emerges in which slow movements are produced through a discontinuous (∼8–10 Hz) command generating small pulses of acceleration and braking. Increasing movement velocity is associated with larger amplitude discontinuities, suggesting that the amplitude of the pulses sets the velocity of movement. As previously highlighted by the group there is an interesting parallel with ballistic movements in which the triphasic agonist–antagonist–agonist EMG bursts arrive in a similar temporal pattern to that detected for 'constant' velocity finger movements.

The idea that motor unit firing may be modulated by different frequencies above, below or at the mean motor unit firing rate has many interesting implications for motor control. During isometric contraction motor units and acceleration may be modulated by frequencies in the ranges 1–12 and 16–32 Hz; the higher frequency range is coherent with signals recorded from over the primary motor cortex. During a maintained contraction ∼20 Hz supraspinal oscillations co-modulate motor unit activity both within hand muscles and between closely related agonist muscles. During movement the ∼8–10 Hz signal dominates (see Farmer, 1998 for review). Little is known about the anatomical substrates of these signals although it is tempting, in the light of evidence from animal experiments, to postulate that ∼20 Hz oscillations are derived from synchronous activity in the primary motor cortex, whereas ∼8–10 Hz signals are imposed on the descending motor command by cerebellar and sensory pathways (Farmer, 1998).

Although discontinuities have been described in animals and man during reaching and other more proximal movements, it is not yet known if movement related ∼8–10 Hz co-modulation of motor units and acceleration is a general phenomenon. Furthermore, the relationships and interactions between ∼8–10 Hz movement related frequencies, 8–12 Hz physiological tremor and ∼20 Hz frequencies associated with position holding remain to be elucidated. However, the results of Wessberg & Kakuda (1999) and other recent findings suggest that ∼8–10 Hz, ∼20 Hz and other rhythms form an important part of the syntax of movement and posture. We may anticipate that the phenomenon of ∼8–10 Hz movement discontinuity will find its place in a general framework that views temporal pattern coding, synchronization and rhythmicity as an integral part of central nervous system information processing.

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