Abstract Changes in presynaptic inhibition of Ia terminals directed to flexor carpi radialis (FCR) motoneurones (MNs) were investigated in normal human subjects at rest and during voluntary wrist flexion and extension. To that end, two independent methods were used: (1) the radial-induced D1 inhibition of the FCR H reflex, which assesses the excitability of PAD (primary afferent depolarisation) interneurones controlling presynaptic inhibition of Ia terminals mediating the afferent volley of the FCR H reflex; and (2) the heteronymous monosynaptic Ia facilitation induced in the FCR H reflex by intrinsic muscle Ia afferent stimulation, which assesses the ongoing presynaptic inhibition of Ia terminals. With respect to results at rest, it was found that at the onset of (and during tonic) voluntary wrist flexion, D1 inhibition was reduced and heteronymous monosynaptic Ia facilitation was increased. This suggests that, as in the lower limb, presynaptic inhibition is decreased on Ia terminals projecting to MNs involved in the voluntary contraction. In contrast with results observed in the lower limb, presynaptic inhibition of Ia terminals projecting to MNs involved in the voluntary contraction was increased. This suggests that, as in the lower limb, presynaptic inhibition of Ia terminals projecting to MNs involved in the voluntary contraction was reduced. In contrast with results observed in the lower limb, presynaptic inhibition of Ia terminals projecting to MNs involved in the voluntary contraction was reduced.

Keywords Presynaptic Ia inhibition · Voluntary movement · Man · Upper limb

Presynaptic inhibition of Ia terminals, accompanied by primary afferent depolarization (PAD) and mediated by interneurones referred to as “PAD interneurones” below, contributes to the control of the gain of the monosynaptic stretch reflex during movement. Accordingly, in the human lower limb, at the onset of voluntary contraction, there is a decrease, probably descending in origin (Nielsen and Kagamihara 1993), in presynaptic inhibition of Ia terminals of the contracting muscle (Hultborn et al. 1987b; Iles and Roberts 1987). The question then arises whether this result may be transposed to the upper limb, since the descending control of presynaptic inhibition of Ia terminals in the cervical and lumbar enlargement seems to be different, as shown by the effects of cortical stimulation in normal subjects (Meunier and Pierrot-Deseilligny 1998; and see below) and the different changes in presynaptic inhibition of Ia terminals observed in the upper and lower limb of patients after stroke (Aymard et al. 2000).

The present investigation was undertaken to investigate changes in presynaptic inhibition of Ia terminals projecting to flexor carpi radialis (FCR) MNs during voluntary contraction of wrist muscles. Experiments were performed in healthy subjects who gave informed consent to the experimental procedure, which had been approved by the local Ethics Committee. Changes in presynaptic inhibition were investigated using two methods: the D1 inhibition of the FCR H reflex (Berardelli et al. 1987) and the heteronymous facilitation (as described in the leg by Hultborn et al. 1987a) of the FCR H reflex. Experiments were performed at rest (control situation), at the onset of voluntary wrist flexion and extension (conditioning and test stimuli being triggered by the first rectified ongoing EMG potential of the contracting muscle) and during tonic wrist flexion of 10–15% of the maximal force lasting 3 min. To ensure the selectivity of the contraction, the EMG of the non-contracting muscle was simultaneously recorded (Fig. 1A–D).

The FCR H reflex was obtained by stimulating (1-ms duration shocks) the median nerve in the cubital fossa, and 20 conditioned and unconditioned H reflexes were
randomly alternated in the same sequence. The amplitude of the unconditioned test reflex was adjusted to be the same in all the situations and above 5% of Mmax, i.e., within the range where its sensitivity no longer increases with the size of the unconditioned reflex (Malmgren and Pierrot-Deseilligny 1988).

Changes in the D1 inhibition of the FCR H reflex

Figure 1E shows the time course of the variations in the FCR H reflex induced by an electrical stimulation of the radial nerve at motor threshold (1 × MT) in the spiral groove. After the initial disynaptic reciprocal Ia inhibition, there is a second depression occurring at 5- to 50-ms interstimulus intervals (ISI), which may be attributed to presynaptic inhibition of Ia terminals, since, at 10–20 ms