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# DIFFERENTIAL ACTIVATION OF MOTOR UNITS IN THE WRIST EXTENSOR MUSCLES DURING THE TONIC VIBRATION REFLEX IN MAN

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SUMMARY

1. Single motor unit activity was recorded in the extensor carpi radialis longus and extensor carpi radialis brevis muscles of five healthy human subjects, using metal microelectrodes.

2. Motor units were characterized on the basis of their twitch contraction times and their force recruitment thresholds during voluntary imposed-ramp contractions.

3. The discharge patterns of forty-three motor units were studied during tonic vibration reflex elicited by prolonged (150 s) trains of vibration (30 Hz) applied to the distal tendons of the muscles. The temporal relationships between the individual small tendon taps of the vibratory stimulus and the motor unit impulses were analysed on dot raster displays and post-stimulus time histograms.

4. After tendon taps, the impulses of motor units with long twitch contraction times (mean ± S.D., 47·2 ± 10·7 ms) and low recruitment thresholds (0·88 ± 0·6 N) formed a single narrow peak (P1) with a latency (22·7 ± 1·4 ms) which was comparable to that of the tendon jerk in the extensor carpi radialis muscles. These motor units were named 'P1 units'. On the other hand, the response of motor units with shorter twitch contraction times (31·1 ± 3·3 ms) and higher recruitment thresholds (3·21 ± 1·3 N) showed two peaks: a short latency (23·4 ± 1·3 ms) P1 peak similar to the previous one and a P2 peak occurring 9·4 ± 1·2 ms later. These motor units were named 'P1–P2 units'.

5. When the reflex contraction increased slowly, the P1 peaks of 'P1–P2 units' were clearly predominant at the beginning of the contraction, during the rising phase of the motor unit discharge frequency, while the P2 peaks became predominant when the units had reached their maximal discharge frequency.

6. Increasing the tendon vibration frequency (35, 55, 75, 95 Hz) did not modify the 'P1 unit' discharge pattern. Due to interference between vibration period and

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peak latencies, increasing the vibration frequency caused the P1 and P2 peaks of 'P1–P2 units' to overlap.

7. Superficial cutaneous stimulation of the dorsal side of the forearm during tendon vibration noticeably decreased the P1 peaks in both types of motor units. In the P2 peaks it could result in either a decrease or an increase but the average effect was a slight increase.

8. When applied 10 s before tendon vibration, cutaneous stimulation considerably suppressed the tonic vibration reflex.

9. The nature of the pathways mediating the P1 and P2 peaks and the effects of cutaneous stimulation are discussed in relation to data obtained from both animal and human experiments. The hypothesis is put forward that during the tonic vibration reflex, the slow and fast motor units are differentially activated on the basis of either a monosynaptic reflex process or mono- and polysynaptic processes, respectively. It is suggested that the monosynaptic pathway plays a major role in tonic vibration reflex initiation at the onset of the tendon vibration and plays a significant role during a maintained reflex contraction. This is contrary to what was previously thought to be the case.

## INTRODUCTION

It was first established in 1966 that reflex muscle contraction can be evoked using mechanical vibration both in animals (Matthews, 1966) and in man (Hagbarth & Eklund, 1966). In man, this reflex, which was named the tonic vibration reflex (Hagbarth & Eklund, 1966), can be elicited by vibrating the muscle belly, but it is more commonly induced by applying prolonged vibration percutaneously to the distal tendon of muscles.

Experimental data concerning the sensitivity of muscle afferent fibres to vibration in animals (Granit & Henatch, 1956; Bianconi & Van der Meulen, 1963; Matthews, 1967; Brown, Engberg & Matthews, 1967) and humans (Hagbarth & Vallbo, 1968; Burke, Hagbarth, Löfstedt & Wallin, 1976*a*, *b*; Roll, Vedel & Ribot, 1989) have shown that the tonic vibration reflex is mainly attributable to muscle spindle Ia fibres which are able to respond one-to-one to vibration frequencies of up to 200 Hz.

In man, it has been reported that tendon vibration of the pretibial muscles with a peak-to-peak amplitude of 0·2–0·5 mm, selectively activating the muscle spindle Ia afferents (Roll *et al.* 1989), induced a tonic vibration reflex as efficiently as the larger amplitude vibration (1·5 mm; Burke *et al.* 1976*a*, *b*) which causes muscle spindle primary and secondary endings in the same muscles to fire in the harmonic mode with the vibration frequency up to 100 Hz.

It has not yet been clearly established whether the pathways involved in the tonic vibration reflex are monosynaptic or polysynaptic. In 1966, the tonic vibration reflex was thought by De Gail, Lance & Neilson to be mediated through polysynaptic pathways involving the cerebellum, whereas the present tendency is to attribute this tonic reflex to both monosynaptic and polysynaptic loops as first suggested by Matthews (1966). This idea was first supported by the detailed analysis of the firing patterns of single motor units activated by vibration in animals (Homma, Kanda & Watanabe, 1972), and then confirmed in humans, by analysing the cross-correlations between vibratory stimuli and the motor unit impulses elicited, which showed that

unitary electromyogram (EMG) reflex discharge is composed of impulses some which are locked and others unlocked to the vibration cycle (Hirayama, Homma, Mizote, Nakajima & Watanabe, 1974; Hirayama, Homma, Kanda, Nakajima & Watanabe, 1976). Since the unlocked motor unit impulses are more sensitive to the depressant effect of barbiturates than the relatively insensitive locked impulses, and since barbiturates abolished the tonic vibration reflex without modifying the tendon jerk (De Gail *et al.* 1966), Hori, Hiraga & Watanabe (1989) concluded that the unlocked impulses were of polysynaptic origin.

Concerning the respective roles of the monosynaptic and polysynaptic loops in tonic vibration reflex genesis, Hirayama and co-workers (1974) have suggested on the basis of animal data (Homma & Kanda, 1973) that the muscle contraction elicited might result from a slowly developing excitatory state caused by polysynaptic actions on which might be superimposed monosynaptic excitatory postsynaptic potentials locked to the vibration, leading to motoneurone firing when the total depolarization exceeded the firing threshold.

In the same way, Hagbarth, Hellsing & Löfstedt (1976) as well as Burke & Schiller (1976) came to the conclusion that during the tonic vibration reflex, the role of the monosynaptic pathway merely consisted of organizing the temporal patterning of the motor outflow of the tonic vibration reflex, which mainly involved polysynaptic mechanisms.

In the present study, reflex effects induced by low-frequency tendon vibration were studied in the motor units of the human wrist extensor muscles. Motor units were systematically characterized in terms of their twitch contraction times and their force recruitment thresholds with a view to investigating whether there exist any differences in the modalities involved in the activation of slow and fast motor units during the tonic vibration reflex.

The effects of forearm cutaneous stimulation on the motor unit discharge patterns during the tonic vibration reflex and on the tonic vibration reflex genesis were also studied, since cutaneous afferents are known to modify the excitability of slow and fast motor units differentially (Kanda, Burke & Walmsley, 1977; Garnett & Stephens, 1981) and to modulate the activity of the proprioceptive reflex loops (Lundberg, Malmgren & Schomburg, 1977*a*; Rudomin, 1990).

This study has been previously presented in abstract form (Romaiguère, Vedel & Pagni, 1989*a*).

## METHODS

Experiments were performed on five healthy human subjects aged 20–30 with the approval of the local Ethical Committee. All the subjects gave informed consent to the experimental procedure as required by the Helsinki declaration (1964).

The subjects were seated in an armchair, the position of which was adjustable. Their left forearm was placed in a cushioned groove to ensure that a stereotyped position was maintained from one experiment to another. The distal end of the forearm was immobilized in a device leaving the wrist joint free and maintaining the hand in a semi-prone position, and flexed at an angle of 10 deg. The hand was placed in a U-shaped device with an adjustable width, which maintained the back of the hand in contact with a rod holding an isometric force transducer.

The subjects were seated in front of two oscilloscopes on which the motor performances could be monitored by watching the vertical displacement of the spots driven by the force transducer. A third oscilloscope displaying motor unit activity together with the EMG of the wrist extensor and

flexor muscles was used to provide visual feedback when the subjects were asked to completely relax the muscles of their forearm during the resting periods.

*Instructions to subjects*

Subjects were first trained to perform selective contraction of the wrist extensor muscles by pushing on the force transducer device with the back of their hand. They quickly learned to keep finger, arm and shoulder muscles relaxed during this task. Subjects were also trained to relate wrist extensor muscle contraction to the displacement of the oscilloscope spots driven by the force transducer in order to become able to either maintain the force contraction at a given level or perform stereotyped ramp contractions.

During the experiments, the task instructions were clearly explained before the starting signal was given. Ten minute rest periods were introduced periodically so that the subjects could completely relax their forearm muscles. The experiments never lasted for more than 3 h.

*Muscle force recording*

Wrist extensor muscle contraction induced a slight displacement of the rod in contact with the back of the hand (0·2 mm/10 N at the level of the contact with the back of the hand). This displacement was converted into an isometric muscle contraction–force curve calibrated in Newtons, using a high-sensitivity Wheatstone bridge (150 mV/10 mN).

Since voluntary wrist extension involved three muscles (extensor carpi radialis longus, extensor carpi radialis brevis, extensor carpi ulnaris), it was not possible to express the motor unit firing properties in terms of the percentage of the maximal voluntary contraction force of their bearing muscles (extensor carpi radialis muscles), as is usually done. Motor unit reflex discharge was studied with isometric forces ranging from 0 to 8 N and mainly produced, in this case, by the contraction of the extensor carpi radialis muscles induced by the selective vibration of their tendons. For the sake of comparison, maximal voluntary isometric contraction of the three wrist extensor muscles can produce a force of 40–60 N, depending on the subject.

*Muscle activity recording*

Motor unit reflex activation was studied in wrist extensor muscles, the extensor carpi radialis longus and extensor carpi radialis brevis. The global EMGs of these muscles, and those of the extensor carpi ulnaris and the wrist flexor muscles, were recorded simultaneously by means of paired surface electrodes 1·5 cm apart. The EMG activities were amplified and quantitatively estimated using analog integrators.

Single motor unit activities were recorded by means of one or two metal microelectrodes insulated except on the tip (impedance 4–12 MΩ, tested at 1000 Hz, Frederick Haer & Co., USA). They were sterilized in a box saturated with formaldehyde vapour and washed with absolute alcohol before use. The microelectrodes were inserted transcutaneously and then moved in minute steps until a stable recording of a well-identifiable single muscle fibre activity was obtained. The microelectrodes were systematically destroyed after use.

Surface electrodes and microelectrodes were connected to P5 Grass amplifiers through probes (HIP511G high-impedance probes for microelectrodes) with an isolated ground for optimum subject protection (leakage current less than 3 μA). Experimental data were recorded on tape and analysed off-line.

*Checking the recording of single motor unit activity*

Three criteria were used to make sure that single motor unit activity was recorded:

(1) The stability of the muscle fibre potential amplitude was checked by the one-to-one driving of a Schmidt trigger, the triggering level of which was adjusted to 10% below the maximal amplitude of the action potential. The systematic occurrence of Schmidt trigger pulses after each potential was checked on an oscilloscope. These pulses were used subsequently to drive all the analytical devices (computer, instantaneous frequency meter, etc.).

(2) On the same oscilloscope, the motor unit potential was used to trigger the sweep, so that the consistency of the potential shape could be periodically checked from the motor unit impulses superimposed in the storage device.

(3) By computing the distribution histogram of impulse intervals on-line during the muscle

contraction tests, it was possible to detect any firing of another motor unit, which would give rise to abnormally short inter-impulse intervals leading to the occurrence of potentials in the refractory period of the impulses of the motor unit investigated.

The amplified and filtered (band pass = 300–3000 Hz) motor unit activity was continuously monitored on oscilloscopes and a loudspeaker. Each motor unit recording was analysed off-line on an electrostatic recorder (Gould ES 1000) using an instantaneous frequency meter, and was correlated with the muscle contraction parameters.

*Motor unit twitch recording*

During voluntary activation of the motor units investigated, their selective twitches were educed from the whole-muscle force recording (band-pass filtered at 0·1 Hz–1 kHz) using the spike-triggered averaging method. Although the spike-triggered averaging program included a 100 ms refractory process which made it possible to trigger muscle force analysis only when the motor unit discharge frequency was lower than 10 impulses $s^{-1}$, twitches were probably potentially distorted by partial fusion and by the contraction of other simultaneously firing motor units. With the twitch contraction time measurements, it was nevertheless possible to compare the contractile properties of the motor units tested, and thus to tentatively classify the motor units as fast and slow units (Romaiguère, Vedel, Pagni & Zenatti, 1989*c*). This classification was checked by extensively analysing the distribution of the twitch contraction times (17–80 ms) in 400 motor units of the extensor carpi radialis muscles tested in forty-three subjects (Romaiguère, 1990).

*Force recruitment threshold of the motor units*

Since the slow and fast motor units started to fire at different force levels, the motor units were also characterized in terms of their force recruitment thresholds measured during voluntary imposed-ramp contractions (force increase: 0·25 N $s^{-1}$). To perform this motor task, the subject had to follow a target line on the screen of the storage oscilloscope by causing the spot driven by the force transducer to move vertically. In view of the variability of the motor unit recruitment threshold during imposed-ramp tasks (Romaiguère, Vedel & Pagni, 1989*b*), a mean value was calculated from several voluntary imposed-ramp contractions.

*Reflex activation of the motor units by tendon vibration*

Tonic reflex contraction of the extensor carpi radialis muscles was induced by applying mechanical vibration to their distal tendons, while the muscles were completely relaxed. Vibrations were delivered using an electromagnetic vibrator driven by a square-pulse generator coupled to a power amplifier. The tip of the vibrating rod was machined so that muscle tendons could be selectively stimulated with a minimal contact surface. Since the tendons of both extensor carpi radialis longus and extensor carpi radialis brevis are parallel at the level of the wrist joint, they were vibrated simultaneously. The vibration amplitude was monitored using an emitting–detecting infra-red photocell to measure the displacement of the vibrating rod. The vibration amplitude was adjusted to between 0·2 and 0·5 mm peak-to-peak.

Tendon vibration was applied over a period of 150 s, each period being preceded by a rest period of 3–5 min.

*Cutaneous stimulation*

Cutaneous stimulation consisting of continuous light sweeping of the skin was applied using a soft paint brush. Broad proximo-distal and disto-proximal brush sweeps were made alternately on the skin on the dorsal side of the forearm except for the area covering the extensor carpi radialis muscles where the surface electrodes and the microelectrode were located. The cutaneous stimulation was applied over periods of 30–40 s, each period being separated by 20 s. The vibration was maintained throughout the whole session, i.e. until the sum of the cutaneous stimulation periods was 150 s.

*Data analysis*

The study was performed on five subjects chosen for their propensity to show a large amplitude tonic vibration reflex in their extensor carpi radialis muscles in response to a low-frequency stimulus (30 Hz). This vibration frequency was chosen because the corresponding stimulus period

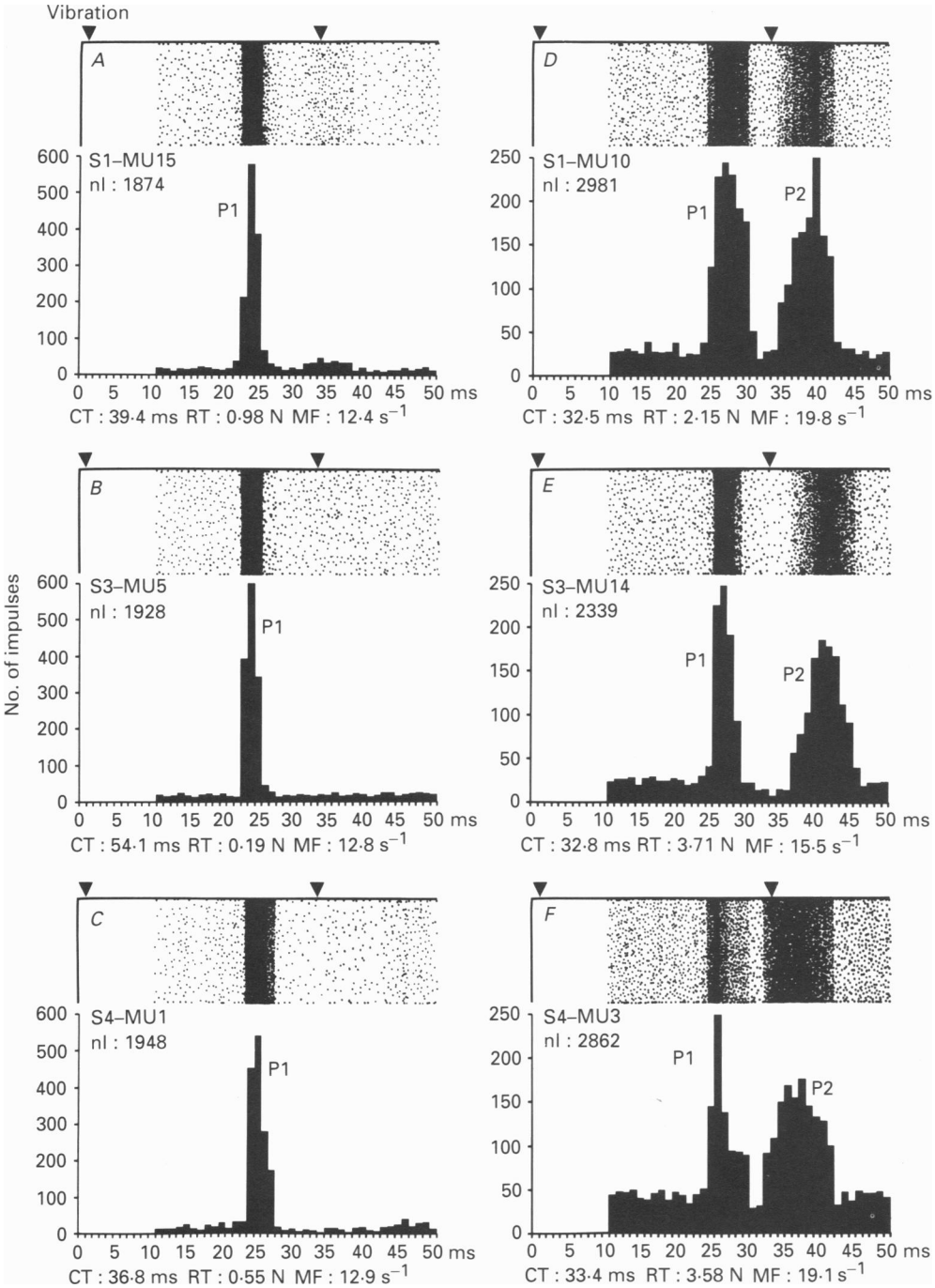


Fig. 1. Reflex patterning of motor unit discharge during the tonic vibration reflex. Analysis by dot raster display and post-stimulus time histogram of the phase relationships between vibratory tendon taps (▼) and motor unit impulses recorded in the extensor carpi radialis muscles of three human subjects (S1 : *A* and *D*; S2 : *B* and *E*; S3 : *C* and *F*). Analysis was performed during tonic vibration reflex induced by tendon vibrations imposed during 150 s at a frequency of 30 Hz. *A*, *B* and *C*, most of the impulses of motor

(33 ms) is long enough for monosynaptic and polysynaptic events occurring after tendon taps to be observed.

The temporal relationships between vibratory tendon taps and motor unit impulses were analysed using dot raster display and post-stimulus time histogram methods, within a 33 ms time window in order to avoid any visual confusion with the motor unit responses to successive tendon taps. Since the tendon jerk latency is around 22 ms in the wrist extensor muscles, the analysis was performed on the time window between 17 and 50 ms after each tendon tap.

A baseline mean and its standard deviation were calculated on the bins between 10 and 20 ms after the tendon taps. Any bin with a count which reached more than twice the standard deviation from the baseline mean was taken to reflect the presence of a peak in the histogram.

After the motor unit recruitment, this analysis was carried out on 150 s tendon vibration, both with and without cutaneous stimulation. The tendon jerk latency in the extensor carpi radialis muscles was measured in each subject.

## RESULTS

### *Analysis of motor unit reflex discharge*

#### *Temporal distribution of motor unit impulses in relation to the vibration cycle*

The reflex discharge patterns were compared with the contractile properties and with the recruitment thresholds of the motor units as illustrated in Fig. 1 and shown in Table 1.

The impulses of motor units with the longest contraction times and the lowest recruitment thresholds formed one peak (P1) with a latency of $22{\cdot}7 \pm 1{\cdot}4$ ms (mean $\pm$ S.D.) after the tendon taps and with a mean duration of $4{\cdot}9 \pm 1{\cdot}1$ ms, as illustrated in *A*, *B* and *C* in Fig. 1. The impulses of motor units with shorter contraction times and higher recruitment thresholds occurred in two peaks, as illustrated in *D*, *E* and *F* in Fig. 1. The first of these two peaks had a similar latency to that of the previous motor unit type ($23{\cdot}4 \pm 1{\cdot}3$ ms, *t* test: $t = 1{\cdot}7$; $P < 0{\cdot}08$) and was therefore named P1. Its duration was $6{\cdot}1 \pm 1{\cdot}1$ ms. The second peak (P2) had a mean latency of $32{\cdot}8 \pm 1{\cdot}7$ ms and its duration was $9{\cdot}3 \pm 1{\cdot}3$ ms. The duration of the P1 peaks differed significantly between the two types of motor units (*t* test: $t = 3{\cdot}5$, $P < 0{\cdot}01$).

Forty-three motor units were studied, and the contraction times and recruitment thresholds were compared (Table 1). All but one of the nineteen motor units with twitch contraction times which were longer than 35 ms showed a reflex discharge pattern with a P1 peak alone ('P1 units'); whereas all but one of the twenty-four motor units with twitch contraction times which were less than 35 ms showed a reflex discharge pattern with both P1 and P2 peaks ('P1–P2 units'). Six 'P1 units' showed a slight increase in the number of impulses counted in some bins after the P1 peak (Fig. 1*A*), with a latency similar to the P2 peak; this increase as compared with the

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units characterized by long twitch contraction times (CT) and low force recruitment thresholds (RT, measured during voluntary imposed-ramp contractions) occurred in one peak (P1 peak), the latency of which, in relation to the tendon taps triggering the analysis, was similar to that of the tendon jerk measured in the extensor carpi radialis muscles. *D*, *E* and *F*, impulses of motor units characterized by shorter contraction times and higher recruitment thresholds occurred in two peaks, a short latency one (P1 peak) and a longer latency one (P2 peak). nI, total number of motor unit impulses counted in the time window between 17 and 50 ms; MF, motor unit mean discharge frequency during the analysis.

TABLE 1. Relationships between motor unit characteristics (CT, twitch contraction time; RT, voluntary force recruitment threshold) and reflex patterning (P1 peak and P1–P2 peaks) during the tonic vibration reflex induced in the extensor carpi radialis muscles

| MU ($n = 43$) | CT (ms) | RT (N) | P1 | P2 |
|---|---|---|---|---|
| 1 | 71·5 | 0·29 | P1 | |
| 2 | 68·3 | 0·38 | P1 | * |
| 3 | 62·1 | 0·22 | P1 | |
| 4 | 58·2 | 0·32 | P1 | * |
| 5 | 54·1 | 0·19 | P1 | |
| 6 | 48·5 | 1·13 | P1 | |
| 7 | 48·2 | 0·88 | P1 | |
| 8 | 45·4 | 0·39 | P1 | |
| 9 | 43·5 | 2·35 | P1 | |
| 10 | 43·2 | 1·62 | P1 | |
| 11 | 42·2 | 1·66 | P1 | |
| 12 | 42·2 | 0·99 | P1 | |
| 13 | 41·8 | 0·95 | P1 | * |
| 14 | 39·7 | 0·18 | P1 | * |
| 15 | 39·4 | 0·98 | P1 | * |
| 16 | 39·3 | 1·58 | P1 | |
| 17 | 38·4 | 1·92 | P1 | P2 |
| 18 | 38·2 | 0·98 | P1 | |
| 19 | 36·8 | 0·55 | P1 | |
| 20 | 34·6 | 2·13 | P1 | P2 |
| 21 | 34·3 | 1·96 | P1 | P2 |
| 22 | 34·2 | 3·21 | P1 | P2 |
| 23 | 33·8 | 1·17 | P1 | * |
| 24 | 33·4 | 3·56 | P1 | P2 |
| 25 | 33·2 | 5·29 | P1 | P2 |
| 26 | 33·2 | 1·96 | P1 | P2 |
| 27 | 33·1 | 1·47 | P1 | P2 |
| 28 | 32·8 | 3·71 | P1 | P2 |
| 29 | 32·5 | 2·15 | P1 | P2 |
| 30 | 32·5 | 3·25 | P1 | P2 |
| 31 | 32·5 | 4·92 | P1 | P2 |
| 32 | 31·3 | 5·48 | P1 | P2 |
| 33 | 31·2 | 1·42 | P1 | P2 |
| 34 | 31·2 | 5·32 | P1 | P2 |
| 35 | 30·5 | 2·66 | P1 | P2 |
| 36 | 29·5 | 2·54 | P1 | P2 |
| 37 | 29·5 | 2·15 | P1 | P2 |
| 38 | 28·5 | 2·31 | P1 | P2 |
| 39 | 27·3 | 5·58 | P1 | P2 |
| 40 | 27·3 | 4·53 | P1 | P2 |
| 41 | 26·7 | 3·35 | P1 | P2 |
| 42 | 25·1 | 2·67 | P1 | P2 |
| 43 | 24·5 | 3·52 | P1 | P2 |

Asterisks indicate in the case of some 'P1 units' the presence of a non-significant increase in the number of impulses counted in some bins after the P1 peak. The sequence from top to bottom is in order of decreasing contraction time.

baseline mean was not significant, however. These 'P1 units' are indicated by an asterisk in Table 1.

The contraction times of 'P1 units' ($47{\cdot}2 \pm 10{\cdot}7$ ms, mean $\pm$ S.D.) were significantly

TABLE 2. Statistical data (mean ± S.D.) on the characteristics of P1 and P2 peaks

| | P1 MU ($n = 19$) | P1–P2 MU ($n = 24$) |
|---|---|---|
| CT (ms) | 47·2 ± 10·7 | 31·1 ± 3·3 |
| RT (N) | 0·88 ± 0·6 | 3·21 ± 1·3 |
| MU discharge frequency ($s^{-1}$) | 11·9 ± 2·3 | 13·1 ± 3·8 |
| P1 latency (ms) | 22·7 ± 1·4 | 23·4 ± 1·3 |
| P2 latency (ms) | — | 32·8 ± 1·7 |
| P1–P2 interval (ms) | — | 9·4 ± 1·2 |
| P1 width (ms) | 4·9 ± 1·1 | 6·1 ± 1·1 |
| P2 width (ms) | — | 9·3 ± 1·3 |
| nP1/nP2 ratio | — | 0·76 ± 0·3 |
| nP1/nI (%) | 68·4 ± 13·7 | 33·9 ± 7·6 |
| nP2/nI (%) | — | 47·5 ± 8·5 |
| nP1/nT ratio | 0·27 ± 0·06 | 0·15 ± 0·07 |
| nP2/nT ratio | — | 0·20 ± 0·06 |

nP1 and nP2, number of impulses counted in P1 and P2 peaks; nI: number of impulses counted in the histograms in the time window between 17 and 50 ms; nT, number of vibratory tendon taps triggering the post-stimulus time histogram analysis.

TABLE 3. Statistical data (mean ± S.D.) on the increase or decrease (± %) in P1 and P2 peaks resulting from superficial cutaneous stimulation (SCS)

| | P1 MU ($n = 10$) | P1–P2 MU ($n = 11$) |
|---|---|---|
| SCS/P1 (± %) | −61 ± 14·1 | −53 ± 9·2 |
| SCS/P2 (± %) | | +7 ± 46·1 |

longer than those of 'P1–P2 units' (31·1 ± 3·3 ms; *t* test: $t = 7·0$, $P < 0·01$), while the recruitment thresholds of 'P1 units' (0·88 ± 0·6 N) were significantly lower than those of 'P1–P2 units' (3·21 ± 1·3 N; *t* test: $t = 7·28$, $P < 0·01$).

It is noteworthy that during the tonic vibration reflex the motor unit recruitment order remained unchanged, since the low-threshold units were those which were recruited first during voluntary imposed-ramp contractions.

During 150 s train vibration, the mean discharge frequencies of 'P1 units' and 'P1–P2 units' were 11·95 ± 2·3 and 13·14 ± 3·8 impulses $s^{-1}$, respectively (Table 2), which is not significantly different (*t* test: $t = 1·16$, $P > 0·2$).

The characteristics of the P1 and P2 peaks of the forty-three recorded motor units are given in Table 2. In the case of the 'P1 units', the mean ratio of the impulses in the P1 peak to the total number of impulses counted in a 33 ms time window was 68·4 ± 13·7 % (nP1/nI in Table 2), and in that of the 'P1–P2 units', the ratios of the impulses in P1 and P2 peaks to the total number of impulses were 33·9 ± 7·6 and 47·7 ± 8·5 %, respectively (nP1/nI and nP2/nI in Table 2).

On the basis of microneurography results (Roll *et al.* 1989), which showed that low-amplitude tendon vibration at 30 Hz is likely to activate almost all the muscle spindle Ia afferents of the pretibial muscles in the harmonic mode (1/1), the ratios between the number of impulses in P1 and P2 peaks and the number of vibratory

tendon taps were calculated in order to determine the efficiency of the vibratory stimulus as a means of activating the motoneurones. These data require some reservations, however, since the arm muscle receptors might conceivably differ in their sensitivity to tendon vibration, and some afferents might fire twice in response to a single tendon tap.

With the 'P1 units', this ratio (nP1/nT = 0·27 ± 0·06, Table 2) showed that only one tendon tap out of four was followed by an impulse in the P1 peak.

With the 'P1–P2 units', these ratios were nP1/nT = 0·15 ± 0·07 and nP2/nT = 0·20 ± 0·06, respectively (Table 2). This means that one tendon tap out of 6·5 gave rise to an impulse in the P1 peak, and one tendon tap out of five to an impulse in the P2 peak.

Using vertically enlarged dot raster displays, the modalities of occurrence of the motor unit impulses after each tendon tap were analysed during 'P1–P2 unit' reflex discharge. No motor units were ever found to fire twice in response to a single tendon tap, since each efficient tap gave rise to only a P1 impulse or a P2 impulse.

*Time course of P1 and P2 peaks during the vibratory train*

In six cases, the rising tonic reflex contraction rate was slow enough to induce a very progressive increase in the motor unit discharge frequency. It was therefore possible from post-stimulus time histograms to analyse the discharge of the motor units at various activity levels, especially just after their recruitment. Figure 2 illustrates the results obtained under these conditions. Diagram *A* shows the post-stimulus time histogram of the discharge during the whole vibratory train (150 s), where the unit studied has been called a 'P1–P2 unit'. Diagram *B* shows the post-stimulus time histogram built up during the first 30 s of the motor unit discharge, the mean frequency of which was then 12·6 impulses $s^{-1}$ (see diagram *D*). Diagram *C* shows the post-stimulus time histogram built up over the same period, when the motor unit discharge had reached its maximal discharge frequency (15·6 impulses $s^{-1}$; see diagram *D*). By comparing the three post-stimulus time histograms, it can be seen that the P1 peak was predominant during the initial phase of the motor unit discharge (*B*, nP1/nP2 = 3·57), while the P2 peak was predominant in the period when the motor unit discharge had reached its maximal frequency (*C*, nP1/nP2 = 0·82).

Using the same method of analysis, no P2 peak occurrence was ever observed in the 'P1 unit' reflex discharge during slow rising muscle contraction induced by tendon vibration.

*Effects of various tendon vibration frequencies on the organization of motor unit reflex patterns*

Post-stimulus time histograms of motor unit discharges were analysed at vibration frequencies of about 35, 55, 75 and 95 Hz applied during 150 s. The results are shown in Fig. 3.

The diagrams on the left show that the 'P1 unit' reflex discharge pattern was constant whatever the vibration frequency. Nevertheless, it is noticeable that the number of motor unit impulses which were unlocked to the vibration cycle increased with the vibration frequency.

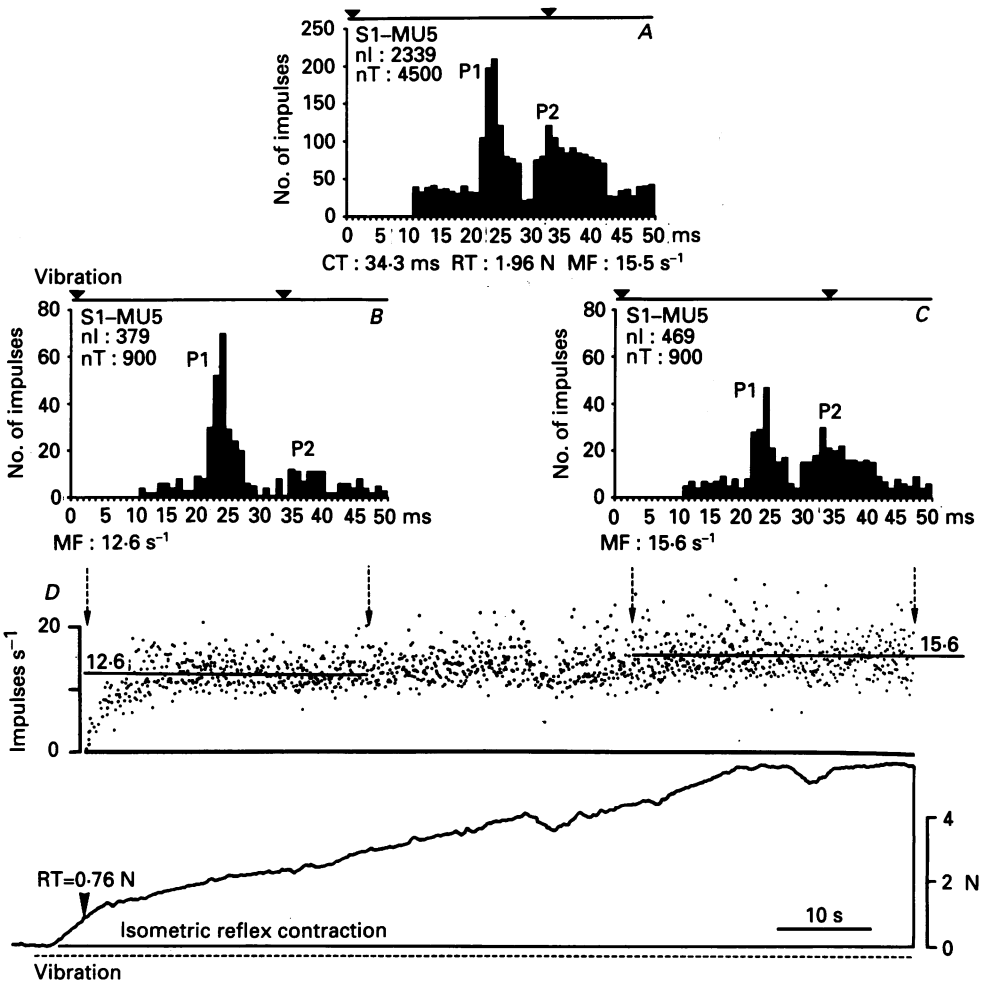


Fig. 2. Time course of the reflex patterning of a motor unit discharge during a slowly increasing tonic vibration reflex induced by mechanical vibration applied to the tendon of the extensor carpi radialis muscles. *A*, identification of the motor unit as a 'P1–P2 unit' by analysing (post-stimulus time histogram) its reflex discharge during a 150 s vibration train at the frequency of 30 Hz. *B* and *C*, characteristics of P1 and P2 peaks during the first 30 s of the motor unit reflex discharge (*B*, mean discharge frequency: 12·6 impulses $s^{-1}$) and during 30 s of motor unit discharge at maximal frequency (*C*, mean discharge frequency: 15·6 impulses $s^{-1}$). Diagram *D* shows the characteristics of the motor unit instantaneous discharge frequency and those of the force–contraction curve during the analysis periods corresponding to diagrams *B* and *C*. CT, motor unit twitch contraction time; RT, voluntary (*A*) and reflex (*D*) force recruitment thresholds of the motor unit; MF, motor unit mean discharge frequency during the analysis; vibration, vibratory tendon taps (*A*, *B* and *C*) or vibration train (*D*); nI, total number of motor unit impulses counted in the time window between 17 and 50 ms; nT, number of vibratory tendon taps triggering the post-stimulus time histogram analysis.

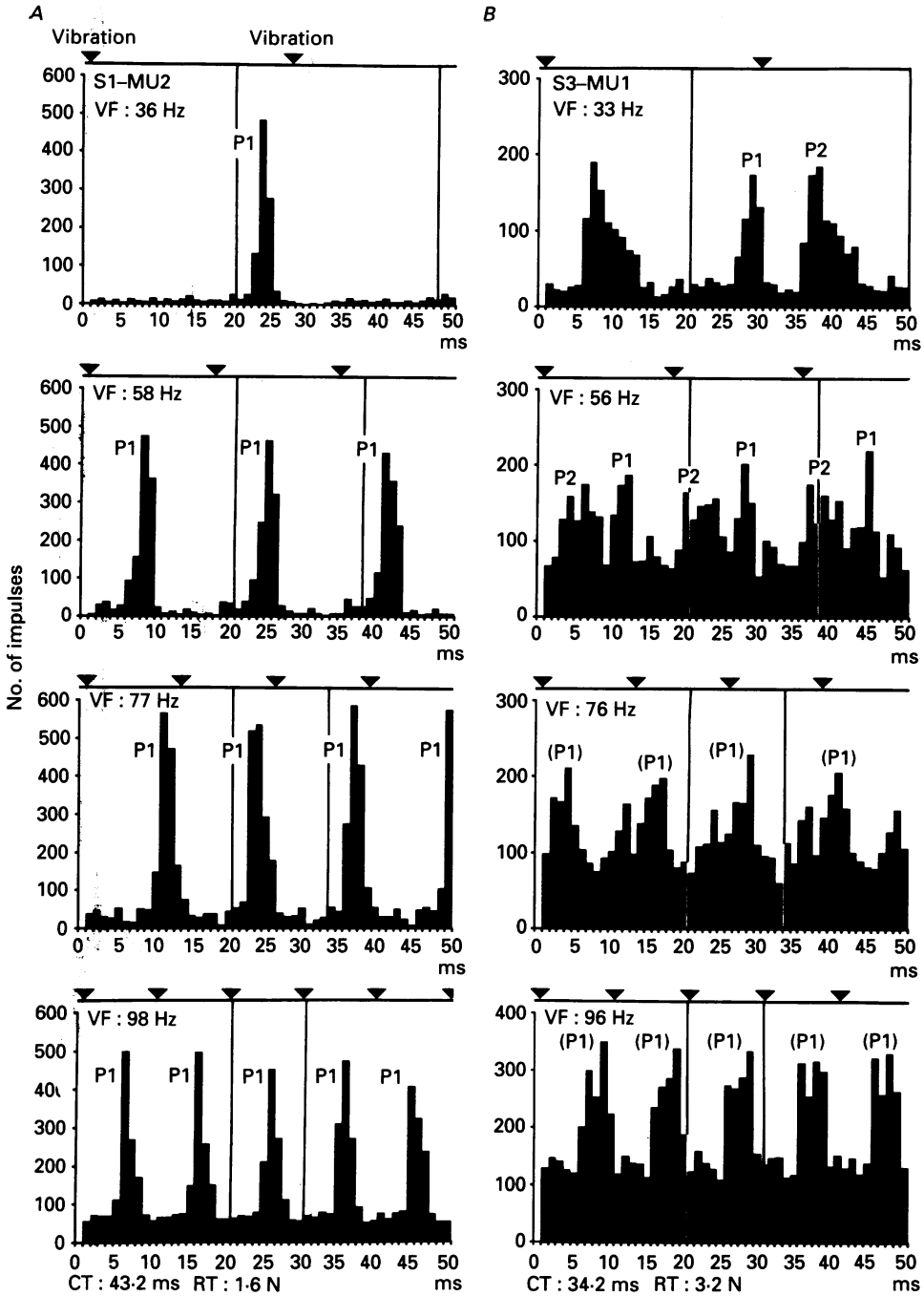


Fig. 3. Effects of increasing the tendon vibration frequency on the reflex patterns of post-stimulus time histograms of a 'P1' (*A*) and a 'P1–P2' motor unit (*B*) during tonic vibration reflex induced in the extensor carpi radialis muscles (vibration train duration: 150 s; VF, vibration frequencies: 35, 55, 75, 95 Hz). In each histogram, the vertical lines delimit the analysis window, the duration of which was equal to the vibratory stimulus period. The motor unit impulses are shown as a function of the tendon taps (▼) triggering the histogram analysis. Diagram *A* illustrates in the case of the 'P1 unit' the consistency

The diagrams on the right show that vibratory frequencies higher than 30 Hz increasingly caused the P1 and P2 peaks of 'P1–P2 units' to overlap due to the interference between the period of the vibratory stimulus, the latency of the P1 peak, and the time elapsing between the P1 and P2 peaks.

Increasing the vibration frequency did not modify the mean discharge frequency of the motor units linearly. For example, in Fig. 3 during vibration trains at 35, 55, 75, 95 Hz, the mean firing rates were 8·4, 9·5, 12·5 and 10·2 impulses $s^{-1}$ with the 'P1 unit' and 12·8, 14·1, 11·1 and 14·1 impulses $s^{-1}$ with the 'P1–P2 unit'.

*Effects of superficial cutaneous stimulation on the motor unit discharge pattern during the tonic vibration reflex*

The effects of cutaneous stimulation were tested by comparing the discharges of motor units during the tonic vibration reflex with and without superficial cutaneous stimulation applied during tendon vibration.

The motor unit discharge was first analysed during vibration applied at 30 Hz and lasting 150 s. Superficial cutaneous stimulation was then applied during vibration, after the motor unit had reached its maximal discharge frequency. The analysis was performed during 150 s (resulting from the addition of several periods of 30–40 s) of concomitant application of vibratory and cutaneous stimulation.

Superficial cutaneous stimulation applied to the skin covering the dorsal side of the forearm (except for the proximal part of the extensor carpi radialis muscles) produced a strong decrease in the P1 peaks of both types of motor units, while the P2 peaks either increased (in seven cases out of eleven) or decreased (in four cases out of eleven).

These results are illustrated in Fig. 4 in the case of a 'P1 unit' (*A* and *B*) and a 'P1–P2 unit (*C* and *D*). In 'P1 units' the cutaneous stimulation could completely suppress the discharge for several seconds. In some cases, this arrest lasted until the cutaneous stimulation ceased, while in other cases the discharge resumed before the stimulation ceased. When cutaneous stimulation was applied for longer periods (more than 1 min), a cessation of discharge was usually followed by a reversal of activity. This cessation of motor unit discharge was always accompanied by a variable decrease in the global muscle contraction force. In 'P1–P2 units' the cutaneous stimulation mainly led to a decrease in activity, and in two cases the silent period was shorter than with the 'P1 units'. Contrary to the effects observed in 'P1 units', the decrease in 'P1–P2 unit' activity was not systematically accompanied by a decrease in the global muscle force.

Table 3 sums up the results obtained on twenty-one units. P1 peaks involving ten 'P1 units' decreased by $61 \pm 14\%$ (mean $\pm$ S.D., SCS/P1 in Table 3). P1 peaks involving eleven 'P1–P2 units' decreased by $53 \pm 9\%$ (SCS/P1), and on average the P2 peaks increased slightly, by $7 \pm 46\%$ (SCS/P2).

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of the P1 peak pattern whatever the vibration frequency. Diagram *B* shows in the case of the 'P1–P2 unit' that, due to interference between vibration period and peak latency, the vibration frequency increase progressively caused the P1 and P2 peaks to overlap. In the lower diagrams, (P1) indicates the occurrence of P1 peaks in terms of their estimated latency from the successive tendon taps. CT, motor unit twitch contraction time; RT, voluntary force recruitment threshold of the motor unit.

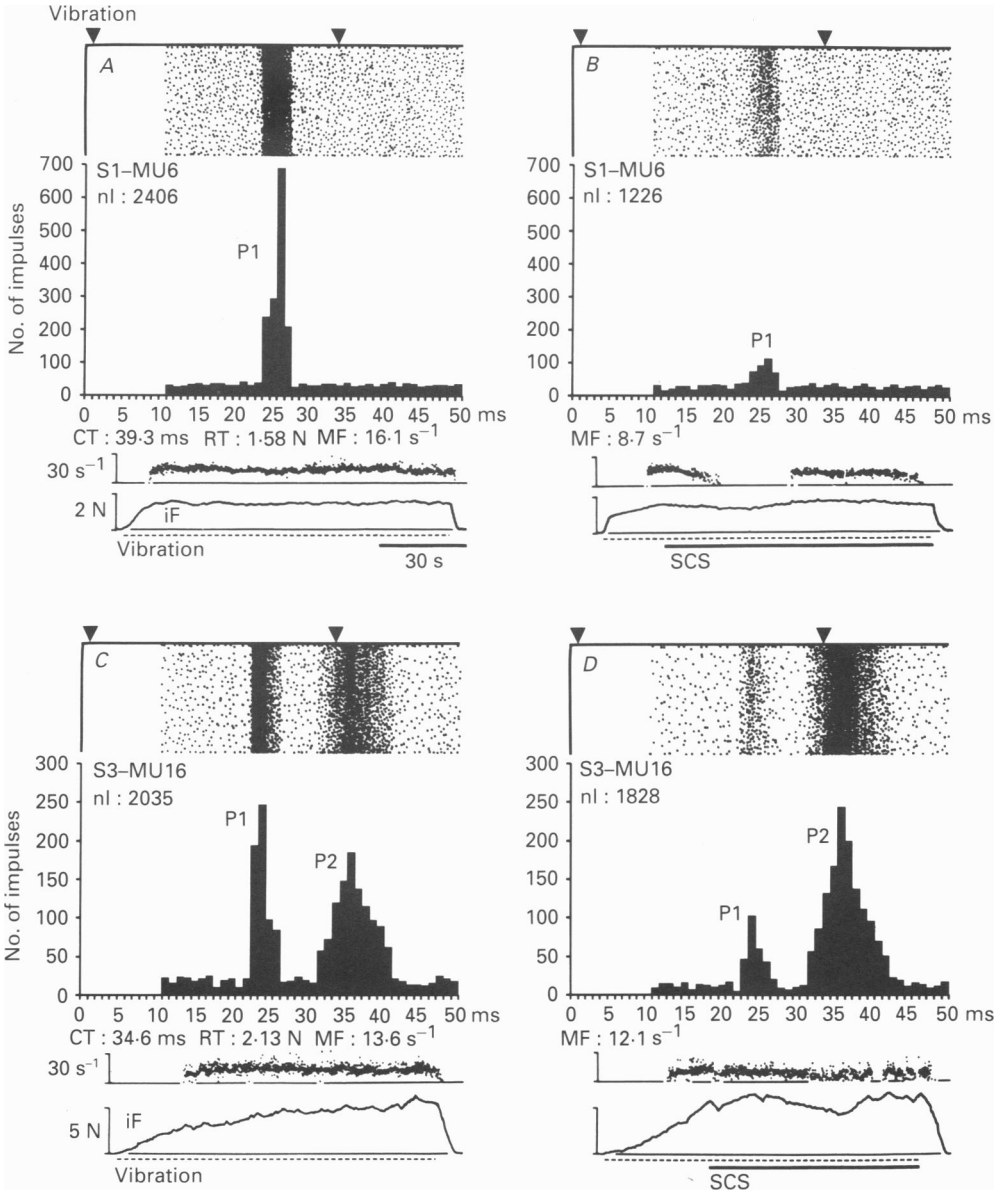


Fig. 4. Effects of superficial cutaneous stimulation (SCS) applied to the dorsal side of the forearm, on the reflex patterning of two motor unit discharges recorded in the extensor carpi radialis muscles during the tonic vibration reflex. *A* and *C*, characterization of the motor units as 'P1 unit' (*A*) and 'P1–P2 unit' (*C*), by post-stimulus time histogram analysis of their reflex discharge induced by tendon vibration applied during 150 s at a frequency of 30 Hz. *B* and *D*, effects on P1 and P2 peaks of superficial cutaneous stimulation superimposed on tendon vibration during 150 s. Cutaneous stimulation was applied over periods of 40 s, successive periods being separated by 20 s. The mean frequency (MF) noted under histograms *B* and *D* was the mean firing rate of the motor units during the whole period of analysis, including the silent periods due to the cutaneous stimulations. Comparisons between the recordings presented under each histogram illustrate the effects of long cutaneous stimulations (*B*, 95 s; *D*, 70 s) on the motor unit instantaneous discharge frequency and on the force curve of the reflex muscle contraction.

*Effects of cutaneous stimulation on the induction of the tonic vibration reflex*

The above data suggested investigating the effects of superficial cutaneous stimulation on the induction of the tonic vibration reflex in four subjects. Firstly, five control tonic reflex contractions (each preceded by a 3 min rest period) were evoked by applying 50 Hz vibration lasting 40 s. Secondly, superficial cutaneous stimulation was applied for 30 s while vibration (50 Hz) was initiated 10 s later. This sequence was also repeated five times. Thirdly, after a 5 min rest period, five control reflex contractions were again induced.

When applied 10 s before tendon vibration, cutaneous stimulation profoundly suppressed the tonic vibration reflex as long as it was applied, as shown in Fig. 5*B*. A few seconds after the end of cutaneous stimulation, the reflex contraction could develop, but at a much slower rate than in the control situation (Fig. 5*A*). After a rest period of 5 min, the mean rate of the reflex force increase was recovered during tendon vibration (Fig. 5*C*).

## DISCUSSION

*Origin of the two components of the motor unit reflex discharge*

Analysis of the post-stimulus time histograms of motor unit discharges during tonic vibration reflex showed that in the case of motor units with long twitch contraction times and low force recruitment thresholds ('P1 units'), the impulses occurred in one peak (P1), while in the case of motor units with shorter contraction times and higher recruitment thresholds ('P1–P2 units'), the impulses occurred in two peaks (P1 and P2). Given their contractile properties and their recruitment thresholds, and in the light of previous data (Romaiguère *et al.* 1989*c*), we suggest that 'P1 units' can be taken to be slow motor units and 'P1–P2 units', fast motor units.

In each subject, the latency of the tendon jerk in extensor carpi radialis longus and extensor carpi radialis brevis muscles was comparable to that of the P1 peak, which may therefore be attributable to the monosynaptic activation of the motoneurones by muscle spindle Ia afferents.

The origin of the P2 peak is much less obvious and needs to be discussed in relation to various explanations which have been advanced for the long-latency wave observed during the stretch reflex. The stretch reflex is composed of three waves, generally named M1, M2 and M3 (Tatton, Forner, Gerstein, Chambers & Liu, 1975). The M1 wave corresponds to the monosynaptic activation of the motoneurones by Ia afferents (Tatton *et al.* 1975; Tatton, Bawa, Bruce & Lee, 1978), which is consistent with the P1 peak latency. The M2 wave is generally thought to result from the activation by Ia afferents of a transcortical reflex loop (Tatton *et al.* 1975, 1978; Chan, Melvill-Jones, Kearney & Watt, 1979*b*; Chan, Melvill-Jones & Cutchlove, 1979*a*). Other explanations have been proposed, however. In particular, Eklund, Hagbarth, Hägglund & Wallin (1982*a*, *b*) have suggested that the M2 wave might result from the reactivation of Ia afferents by intramuscular mechanical resonance

CT, motor unit twitch contraction time; RT, voluntary force recruitment threshold of the motor unit; nI, total number of motor unit impulses counted in the time window between 17 and 50 ms; MF, motor unit mean discharge frequency during the whole period of the analysis; Vibration, vibratory tendon taps (*A*, *B*, *C*, *D*); iF, isometric contraction force.

after muscle stretching, while Matthews (1984) interpreted the long-latency wave as resulting from the activation of the motoneurones by the muscle spindle group II afferents.

The latency generally measured in the M2 wave in wrist muscles in man is about 50 ms (Tatton *et al.* 1978; Bonnet & Requin, 1982; Eklund *et al.* 1982*a*, *b*), which is

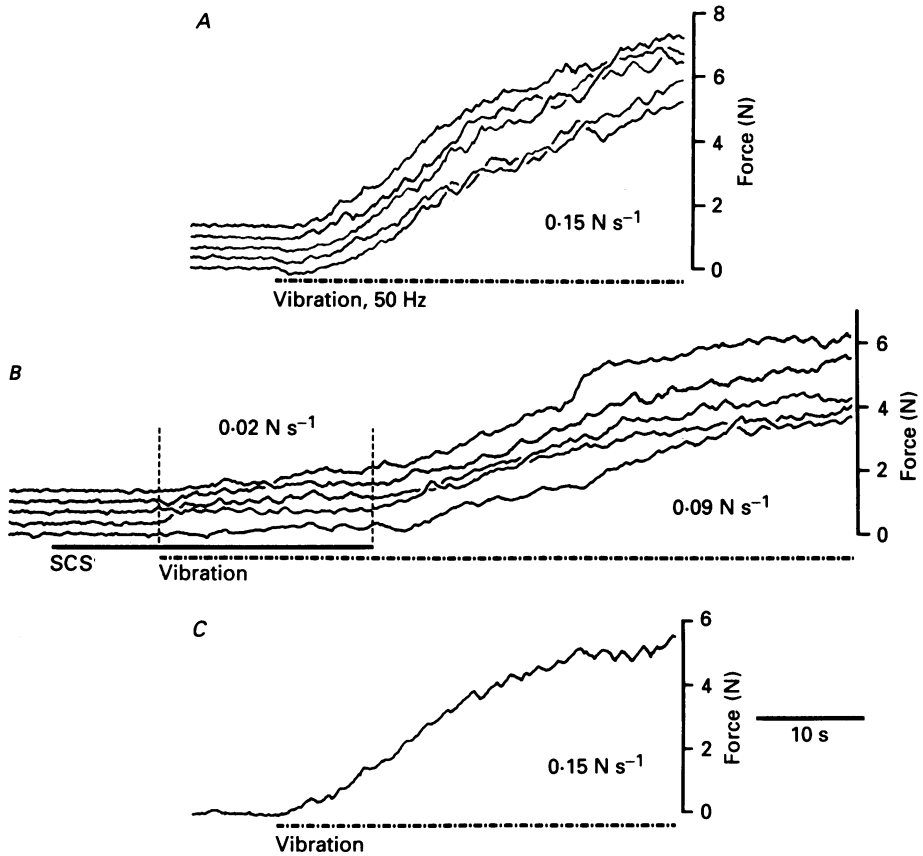


Fig. 5. Effects of superficial cutaneous stimulation applied on the dorsal side of the forearm (SCS) on the genesis of the tonic vibration reflex induced in extensor carpi radialis muscles by tendon vibration. *A*: control situation; force–contraction curves of five tonic vibration reflexes induced by 40 s tendon vibration trains (vibration frequency: 50 Hz), each preceded by a forearm muscle relaxation of 3 min. *B*: effects on the tonic vibration reflex genesis of superficial cutaneous stimulation (30 s duration) occurring 10 s before the beginning of the tendon vibration (50 Hz). Each of the five sequences was preceded by a forearm muscle relaxation of 3 min. *C*: control situation; force–contraction curve of a tonic vibration reflex induced by 40 s tendon vibration at 50 Hz after a 5 min relaxation period. The mean velocity of the force increase (N $s^{-1}$) is indicated for each situation.

not comparable with the P2 peak latency. It thus turns out that no relationships exist between these two reflex phenomena.

The mechanical resonance demonstrated by Eklund and co-workers (1982*a*, *b*) during brisk wrist extensions consisted of intramuscular oscillations with a frequency

of between 30 and 50 Hz. This would imply that any reactivation of the Ia afferents would take place between 20 and 30 ms after their previous activation. This interval is much longer than that measured between the P1 and P2 peaks ($9{\cdot}4 \pm 1{\cdot}2$ ms). Moreover, large amplitude vibration (1·5 mm) applied to the back of the hand and to the distal ends of the radius and cubitus, which induced large mechanical oscillations in the extensor carpi radialis muscles, did not lead to any activation of motor units under our experimental conditions. On the other hand, the small amplitude vibration used in our study was probably less likely to trigger intramuscular oscillations than a large amplitude stretch imposed on a voluntarily contracting muscle. Finally, mechanical resonance can be expected to affect all motor units alike, which is in disagreement with the occurrence of the P2 peak being restricted to the fast motor unit population.

Matthews' data (1984) differed from previous data in that the long-latency reflex activity he investigated occurred only 10–15 ms after the monosynaptic muscle activation by Ia afferents. The muscle studied by Matthews was the flexor pollicis longus located in the distal part of the forearm, and the interval measured here between the short- and long-latency reflex muscle activities seems to be comparable to that measured in the present study between the P1 and P2 peaks in the extensor carpi radialis muscles.

Muscle spindle group II fibres are generally included in the flexor reflex afferents, and may consequently have facilitatory effects on the flexor muscles and inhibitory effects on the extensor muscles (Laporte & Lloyd, 1952). Matthews' hypothesis that the extensor and flexor muscles may be autogenetically activated by their muscle spindle group II fibres is supported, however, by the finding that these afferents evoke excitatory postsynaptic potentials in the motoneurones of their own muscle via a monosynaptic pathway (Kirkwood & Sears, 1975). The existence of these monosynaptic excitatory projections has been confirmed in the case of both the flexor and extensor muscles (Lundberg, Malmgren & Schomburg, 1977*b*). Yet the efficiency of these monosynaptic connections seems to be weak in comparison with those involving primary spindle afferents (cf. Baldissera, Hultborn & Illert, 1981). On the other hand, it has been shown that secondary spindle afferents probably have disynaptic excitatory effects on extensor muscle motoneurones (Lundberg, Malmgren & Schomburg, 1975).

In view of the above arguments, the P2 peak might possibly be due to the activation of the motoneurones of extensor carpi radialis muscles by secondary spindle afferents, via either monosynaptic or polysynaptic pathways. One should remember, however, that various studies have shown that the secondary spindle afferents are poorly activated by tendon vibration (Brown *et al.* 1967; Burke *et al.* 1967*a*; Roll *et al.* 1989), and that their sensitivity to this stimulus tends to decrease during the tonic vibration reflex (Burke *et al.* 1976*b*). Nevertheless, these data were obtained during short-duration vibratory trains, and no data have been published up to now on the time course of primary and secondary muscle spindle afferent discharges during exposure to vibration for several minutes, as in our experimental situation. It is possible that the activation of $\beta$-skeleto-fusimotor neurones via monosynaptic pathways, and possibly also that of $\gamma$-fusimotor neurones via polysynaptic pathways (Ellaway & Trott, 1978) during a long-lasting tonic vibration

reflex, may progressively reinforce the vibration sensitivity of the muscle spindle sensory endings, especially the secondary ones. This may be consistent with the fact that the P2 peak does not occur at the beginning of the reflex muscle contraction, but only after several seconds of vibration (Fig. 2).

Another hypothesis, which is more in line with classical interpretations of the tonic vibration reflex, is that the P2 peak may result from the activation of the motoneurones by a polysynaptic projection from primary spindle afferents. Actually, since muscle spindle Ia afferents are the most sensitive to vibratory stimuli (Brown *et al.* 1967; Burke *et al.* 1976*a*, *b*; Roll *et al.* 1989), the tonic vibration reflex is usually thought to result mainly from the activation of a polysynaptic excitatory pathway by the muscle spindle Ia afferents (De Gail *et al.* 1966; Desmedt & Godaux, 1975; Godaux, Desmedt & Demaret, 1975; Burke & Schiller, 1976; Hagbarth *et al.* 1976).

A study by Malmgren & Pierrot-Deseilligny (1988*a*, *b*) has shown that in human wrist flexor muscles, excitation of Ia afferents by electrical nerve stimulation induces a motor unit response with a latency which is 3–6 ms longer than that of the monosynaptic activation. The authors suggested that this polysynaptic effect may be mediated via the cervical propriospinal system described by Lundberg (1979) in cats. Since tendon vibration may activate the muscle spindle Ia afferents less efficiently and more diffusely than electrical nerve stimulation, which is very likely to activate almost all the Ia afferents synchronously, it is possible that a P2 peak beginning on average $9{\cdot}4 \pm 1{\cdot}2$ ms after the monosynaptic P1 peak may also be mediated via cervical propriospinal interneurones. Nevertheless, the difference between the latencies of the two non-monosynaptic events measured in the two experimental situations can seem to be too great to be able to attribute the P2 peak to a cervical propriospinal mechanism, and therefore the possibility that a supraspinal (but sub-cortical) polysynaptic loop may be involved cannot be excluded.

To sum up, although the possible involvement of muscle spindle group II afferents has not been completely ruled out, it seems more likely that P2 peaks are produced by Ia afferents acting either via propriospinal interneurones at the cervical level, or via some supraspinal polysynaptic pathway. The present study therefore not only confirms the contribution of monosynaptic and polysynaptic reflex pathways to the tonic vibration reflex, but also demonstrates that the reflex patterning differs depending on the type of motor unit. Slow motor units are thus mostly activated monosynaptically (P1 peak), while fast motor units are activated both via monosynaptic (P1 peak) and polysynaptic (P2 peak) pathways.

Previous cross-correlation analyses of muscle activity induced by high vibration frequency have shown that the motor unit reflex discharge was composed of impulses which were either monosynaptically locked or unlocked to the vibration cycle (Hirayama *et al.* 1974, 1976; Hori *et al.* 1989). In the present study it was shown using low vibration frequency and post-stimulus time histogram analysis that, in fact, the phase-locked impulses are induced both monosynaptically and polysynaptically.

*Effects of superficial cutaneous stimulation on motor unit discharge patterns during the tonic vibration reflex*

The most noteworthy finding made in this study was the fact that superficial cutaneous stimulation had differential effects on P1 and P2 peaks: the P1 peaks

decreased systematically, while the P2 peaks either increased or decreased, averaging a slight increase.

First of all, it is noteworthy that the decrease in the monosynaptic P1 peak induced by cutaneous stimulation, which did not include the skin area covering the extensor carpi radialis muscles (where surface electrodes and microelectrode were located), links up with data published by Hagbarth in 1952. This author showed in the decerebrated cat that the monosynaptic reflex induced in limb extensor muscles was inhibited by cutaneous stimulation of the whole limb, except for the part exactly covering the muscle studied, stimulation of which had an excitatory effect.

One explanation for the selective decrease in the monosynaptic P1 peak might be that cutaneous stimulation enhances the presynaptic inhibition of Ia terminals acting monosynaptically on the motoneurones, by reinforcing, for example, the well-known Ia/Ia presynaptic inhibition. No such effect has ever been described so far, however, whereas it has been demonstrated in animals that cutaneous afferents inhibit Ia/Ia presynaptic inhibition (cf. Rudomin, 1990). It is nevertheless worth noting that in awake human subjects, Ia terminals may be presynaptically inhibited by cutaneous afferents, which is consistent with previous data on human H reflex by Delwaide, Crenna & Fleron (1981).

Another possibility is that several processes acting simultaneously may have produced the differential effects observed on the P1 and P2 peaks. Motoneuronal postsynaptic inhibition has been found to be indirectly induced by cutaneous stimulation which reinforces the inhibitory action of Ib afferents on their homonymous muscle (Lundberg *et al.* 1977*a*). To be able to explain the monosynaptic P1 peak decrease in terms of a mechanism of this kind, it would be necessary for the cutaneous afferents to simultaneously enhance the polysynaptic P2 peak strongly enough to counterbalance the motoneuronal postsynaptic inhibition. Several hypotheses may be advanced:

(1) The P2 peak originating from muscle spindle group II fibre activity might be increased due to the fact that cutaneous stimulation facilitates the disynaptic projections of these muscle spindle afferents onto motoneurones (cf. Baldissera *et al.* 1981).

(2) The P2 peak induced by Ia reflex action mediated via cervical propriospinal interneurones might be increased by the concomitant activation exerted by cutaneous afferents contributing to the non-monosynaptic propriospinal response described by Malmgren & Pierrot-Deseilligny (1988*a*).

(3) The P2 peak resulting from the activation of a Ia polysynaptic supraspinal pathway might be increased by the convergence of muscle spindle and cutaneous afferents onto sub-cortical structures (Andersen, Etholm & Gordon, 1970; Hongo, Kitazawa, Ohki, Sasaki & Xi, 1989*a*; Hongo, Kitazawa, Ohki & Xi, 1989*b*).

*Effects of superficial cutaneous stimulation on the induction of the tonic vibration reflex*

Cutaneous stimulation of the dorsal side of the forearm occurring 10 s before the beginning of the tendon vibrations almost completely prevented the induction of the tonic vibration reflex in the extensor carpi radialis muscles. Since under the present conditions, cutaneous stimulation had inhibitory effects on the monosynaptic

component of the tonic vibration reflex, the blocking of this reflex might be attributable to the suppression of the activation of the slow motor units, which are normally monosynaptically activated at the onset of the reflex contraction. This would mean that the Ia monosynaptic reflex pathway plays a major role in the initiation of the tonic vibration reflex at the onset of the vibration train, in contrast with previous hypotheses suggesting that its role might be limited to the organization of the temporal patterning of the motor outflow (Burke & Schiller, 1976; Hagbarth *et al.* 1976).

During the maintained reflex contraction, the monosynaptic pathway may continue to play a significant part, and the role of the P2 pathway is then likely to be equally important, since it was still able to mediate the tonic muscle contraction when the monosynaptic motoneuronal activation was strongly depressed by the cutaneous stimulation.

In conclusion, it might be suggested that the muscle contraction induced by the tonic vibration is necessarily triggered by Ia monosynaptic activation of slow motor units, the role of which might be to ensure the transition from the relaxed to the contracting muscle state by adjusting the viscoelastic properties of the muscle tissue. This basic contracting state might be necessary for fast motor units to develop the force component of the reflex contraction. Their activation via both monosynaptic and polysynaptic processes would give much wider scope for the adjustment and the modulation of the muscle activity.

As a footnote, we are quite aware that the arguments we have put forward to account for the origin of the P2 peak and the effects of cutaneous stimulation are no doubt somewhat speculative. This can hardly be avoided when interpreting results obtained in awake human subjects on the basis of data mainly obtained from animal experiments. Here we fully agree with Rudomin (1990), when he concludes at the end of his exhaustive review on the presynaptic control processes: 'but we must recognize that defining connectivities between spinal interneurons in the anesthetized and decerebrated preparation gives only a restricted view of the complexity of the neuronal circuits associated with a given reflex pathway. Interneurons must be considered as nodal points of converging and diverging information. Their functional connectivities will depend on balance of excitatory and inhibitory influences received by the network at a given moment, and this in turn will depend on the specific motor and sensory task to be executed'.

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