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RESEARCH ARTICLE

Gwyn N. Lewis · Eric J. Perreault
Colum D. MacKinnon

The influence of perturbation duration and velocity on the long-latency response to stretch in the biceps muscle

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Abstract Different neural pathways are proposed to mediate the long-latency stretch reflex response (M2) in muscles spanning distal and proximal joints of the upper limb. The M2 at the wrist joint is present only if the duration of the perturbation exceeds a critical time. Lee and Tatton put forward a converging input hypothesis, requiring an interaction of excitatory volleys at the spinal cord, to account for this feature. The goal of the present study was to examine the influence of the duration of perturbation on M2 responses elicited in a muscle spanning the elbow joint. Reflex responses were induced in the biceps brachii muscle by applying ramp-and-hold position displacements to the elbow. It was found that the M2 was strongly dependent on the duration of the perturbation. On average, responses were not elicited following perturbations of less than 35 ± 5 ms. Using a novel double-movement paradigm, we were unable to provide support for the converging input hypothesis. The effect of the duration of perturbation on the M2 may account for the conflicting characteristics of the M2 that have been provided by previous studies applying mechanical or electrical perturbations of varying time durations.

G. N. Lewis (✉) · E. J. Perreault
Sensory Motor Performance Program,
Rehabilitation Institute of Chicago,
345 E Superior St, Chicago, IL 60611, USA
E-mail: g-lewis3@northwestern.edu
Tel.: +1-312-2381235
Fax: +1-312-2382208

E. J. Perreault
Department of Physical Medicine and Rehabilitation,
Northwestern University, Chicago, IL 60611, USA

E. J. Perreault
Department of Biomedical Engineering,
Northwestern University, Evanston, IL 60208, USA

C. D. MacKinnon
Department of Physical Therapy
and Human Movement Sciences, Northwestern University,
Chicago, IL 60611, USA

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Introduction

In the muscles of the upper limb, the reflex response to a rapidly imposed joint displacement can normally be separated into two distinct components (Hammond 1955). The short-latency response (M1) almost certainly involves a monosynaptic pathway from Ia afferents to the motoneuron. The sensory receptors and pathway mediating the long-latency component (M2) are less understood. Muscle spindle group Ia (Lee and Tatton 1975; Matthews 1989), group II (Matthews 1984; although he subsequently promoted Ia afferents), and cutaneous afferents (Darton et al. 1985; Corden et al. 2000) have all been implicated. Two potential explanations for the controversy regarding the origin of the M2 are the vast array of experimental paradigms that have been implemented, and the different neural pathways proposed for the M2 between proximal and distal muscles of the upper limb.

Rapid joint torque pulses (Calancie and Bawa 1985; Capaday et al. 1994), electrical nerve stimulation (Deuschl and Eisen 1999), mechanical pulses (Darton et al. 1985; Corden et al. 2000), joint positional displacements (Wallace and Miles 2001; Lewis et al. 2004), and vibration (Matthews 1984) have all been employed to elicit muscular reflex responses. The use of these different paradigms may markedly alter the characteristics and mechanisms of the induced reflex response. One significant factor in the generation of an M2 in distal muscles appears to be the time duration of the applied perturbation. In a study involving the wrist joint, Lee and Tatton (1982) reported that an M2 could not be elicited unless the time duration of the perturbation was longer than a certain critical value. The time duration of the mechanical perturbation is noticeably different between the paradigms mentioned above, and it is likely that the reflex response recorded following brief stimuli may have an origin distinct from those induced by pertur-

bations of a longer duration. To account for the influence of the duration of the perturbation on the M2, Lee and Tatton (1982) proposed a hypothesis requiring converging excitatory input at the motoneuron. According to this hypothesis, an interaction at the spinal cord between the afferent input from stretch receptors and the volley mediating the M2 response is required for the propagation of the M2 to the muscle. If the perturbation is terminated prior to the arrival of the M2 volley at the spinal cord, then an interaction cannot occur and an M2 response is not elicited.

To our knowledge, the influence of the duration of perturbation on the M2 has yet to be examined in other joints of the upper limb. The mechanisms that give rise to the M2 response almost certainly differ between joints of proximal and distal location within the upper limb. For example, the M2 in the thenar and finger muscles has been shown to incorporate a pathway that traverses the motor cortex (Marsden et al. 1973, 1977; Capaday et al. 1991; Day et al. 1991; Palmer and Ashby 1992; Tsuji and Rothwell 2002); however, there has been no evidence to date for such a transcortical loop in more proximal upper limb muscles (Lenz et al. 1983; Cohen et al. 1991; Thilmann et al. 1991; Fellows et al. 1996).

The primary aim of the present study was to investigate the time duration sensitivity of the M2 response in the biceps brachii muscle. The influence of the duration, velocity, and amplitude of perturbation on the area and latency of the M1 and M2 were characterized. The converging excitatory input hypothesis put forward by Lee and Tatton (1982) was also investigated by applying a novel double-movement protocol in which two short duration perturbations were delivered within a short period of time. We report results showing a clear effect of the time duration of the perturbation on the M2 that is not present for the M1.

Material and methods

Subjects

Ten right-handed individuals (age 31.1 ± 12.7 years, range 20–57) participated in the study. All subjects were required to be neurologically intact, to have no muscular or orthopedic limitations of the upper limb, and to demonstrate a clear M2 response in the biceps muscle following a rapid extension of the elbow joint. Ethical approval for the study was received from the Northwestern University Institutional Review Board and informed consent was obtained prior to testing.

Equipment

Manipulandum

Subjects were seated comfortably with the trunk secured to an adjustable chair (Biodex, Shirley, NY) using padded straps. The subject's right arm was positioned in the horizontal plane with the shoulder at 45° flexion and 90° abduction, the elbow joint at 90°, and the forearm fully pronated (Fig. 1a). The upper arm was placed in a height-adjustable trough support to ensure a constant position of the shoulder joint. A fitted fiberglass cast extending from the fingers to the middle of the forearm was used to maintain the wrist joint in a neutral position and to attach the forearm to a linear actuator (Copley ThrustTube TB3806; Copley Controls, Canton, MA). A 10-cm steel plate located on the underside of the cast, centered at the wrist joint, was secured to the top surface of the actuator via a precision bearing that allowed rotation in the horizontal plane. The actuator was mounted at shoulder height on an adjustable aluminum frame and was oriented 45° from the midline such that perturbations were applied in the horizontal plane in a direction orthogonal to forearm orientation. This resulted in flexion/extension motions primarily at the elbow joint. For the perturbations used in this study,

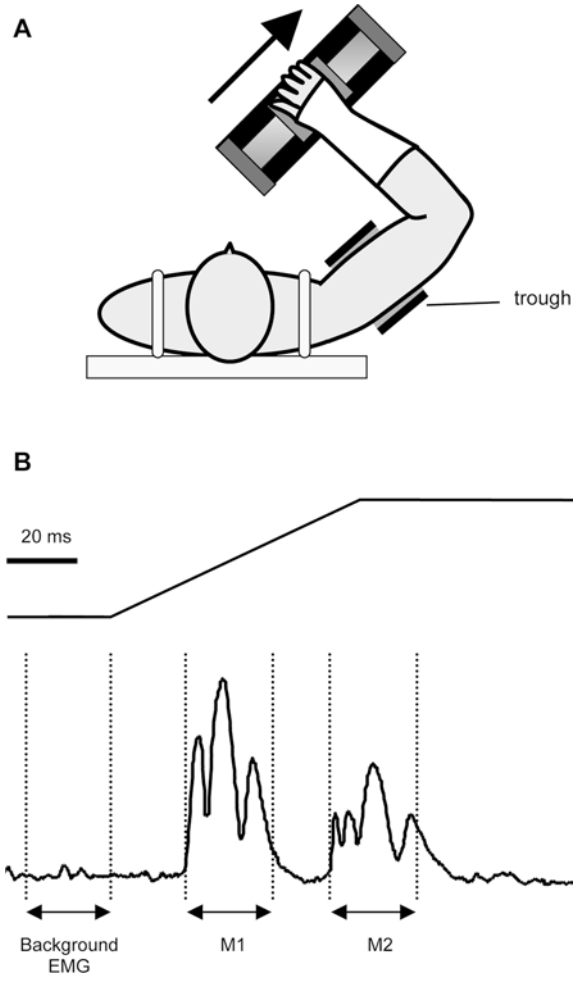

Fig. 1 **a** Task set-up. Perturbations were delivered in the direction of the *arrow*. **b** Example displacement (*top*) and electromyographic recordings (*bottom*) demonstrating the calculation of M1 and M2 response size. Response area (25-ms window) was expressed relative to an equivalent window of muscle activation prior to the perturbation

the largest shoulder joint motions were less than 1/25th of the corresponding elbow motions. The actuator was instrumented with a linear encoder (RGH24; Renishaw, Gloucestershire, UK) to provide position information (resolution 1 μm).

The manipulandum was controlled by custom software developed using Matlab xPC (The Mathworks Co, Natick, MA). The applied perturbations resulted in extension movements of the elbow joint, such that the elbow flexor muscles were lengthened by the movement. Physical stops limited actuator motion to ±60 mm from the central position, and software limits were implemented to cut power to the motor 10 mm before these limits were reached.

Electromyography

Electromyographic (EMG) activity was recorded from the biceps brachii muscle of the right arm. Standard skin preparation techniques were applied prior to the application of disposable dual electrodes (Noraxon USA Inc, AZ). Surface EMGs were amplified and conditioned using a Bortec AMT-8 (Bortec Biomedical Ltd, Canada), with high- and low-pass cut-off frequencies of 10 and 1,000 Hz, respectively. The resulting signals were anti-aliased filtered using 4th order Bessel filters with a cut-off frequency of 500 Hz and then sampled at 5 kHz for subsequent analysis. The delay introduced by the anti-aliasing filters was accounted for when computing reflex latencies.

Protocols

Prior to the experiment, a maximum voluntary contraction (MVC) of the biceps muscle was recorded while the subject was attached to the manipulandum. The target background EMG level during testing was set to 5 ± 2% MVC. During all trials, visual feedback of EMG activity was provided to assist subjects in maintaining the correct level of activation. Perturbations were only delivered when EMG activity had been maintained at the target 5 ± 2% for at least 1 s. In each condition, 20 reflex responses were recorded following perturbations delivered at a variable interval of 3–5 s.

Effect of the duration of perturbation

The critical time duration for eliciting an M2 response was examined by applying perturbations of a constant velocity (250 mm/s; approximately 40–50°/s) while varying the duration of elbow extension. Stretch reflex responses were recorded in all individuals following perturbations with time durations of 20 and 60 ms (5, 15 mm; approximately 1°, 2.5–3°). The duration of the perturbation was then systematically varied between these two values until the minimum duration eliciting a clear M2 response was established. The critical value for the duration of perturbation was determined to the nearest 2 ms (0.5 mm).

Effect of the velocity of perturbation

Two time values were calculated for each individual that represented approximately 80 and 120% of their critical time duration for eliciting an M2 response (SHORT and LONG, respectively). To examine the effect of the velocity of perturbation on M1 and M2, responses to stretch were recorded following perturbations of SHORT and LONG duration at velocities of 100, 250, 400 and 600 mm/s. This enabled us to compare responses at different velocities in which either the time duration or the amplitude of the perturbation were matched.

Double-movement paradigm

To investigate the converging input hypothesis put forward by Lee and Tatton (1982), a double-movement protocol was implemented in which two perturbations of short duration were delivered within a variable time interval. The first perturbation was applied at a velocity of 250 mm/s for a distance of 2 mm (duration 8 ms). According to converging input hypothesis, this should elicit a M1 ($M1_1$) and a volley that would mediate an M2 ($M2_1$); however, the $M2_1$ will not be present as the perturbation is stopped prior to the arrival of this volley at the motoneuron. A second perturbation was then applied after a short time interval. The onset of this second perturbation was timed so that the onset of the corresponding M1 ($M1_2$) would occur approximately 2 ms prior to when the $M2_1$ volley *would have* appeared in the EMG recordings. Thus, afferent information from the second perturbation would be arriving at the spinal cord at a time period that should enable interaction with an excitatory volley mediating $M2_1$. If this did occur, a facilitation of EMG activity representing a combined $M1_2 + M2_1$ response should be evident. This composite response thus provided a measure of the net excitability of the motoneuronal pool in the M2 time period following a perturbation of short duration. The second perturbation was also applied at 250 mm/s; however, the duration was increased to 20 ms to lengthen the time for an interaction at the spinal cord to occur.

Three further perturbation conditions were included as controls for the double-movement protocol. In the first of these, the time interval between the first and second perturbations was increased by 10 ms so that an interaction between the two inputs would not occur. The remaining two conditions were single-movement perturbations; one in which a perturbation of 8-ms duration (2 mm) was applied at 250 mm/s, replicating the first of the double-movement perturbations above; the second was a single perturbation of a time duration equivalent to the total time of the double-movement protocol.

Data processing and analysis

The EMGs from each condition were rectified and averaged before further processing. The onset of the M1 response was determined as the first point at which the EMG activity exceeded 3 SD of the background muscle activation (see Fig. 1b). The onset of the M2 response was determined visually as EMG activity often did not return to baseline following the M1. M1 and M2 response size were defined as the area of integrated EMG in a 25-ms window following response onset. A further 25-ms window of EMG activity was evaluated immediately prior to M1 onset to quantify background muscle activation.

Statistical analysis

Four durations of perturbation were selected that were tested in all subjects at the 250 mm/s velocity (8 ms, 20 ms, SHORT, LONG). The effect of the duration of perturbation on response area was then analyzed using one-way repeated measures (RM) ANOVA. Separate analyses were conducted for the M1 and M2 responses. The interaction between the velocity and duration of perturbation was analyzed using a two-way duration (SHORT, LONG) and velocity (100, 250, 400, 600 mm/s) RM ANOVA.

To examine the results of the double-movement paradigm, a one-way ANOVA was used to compare the size of second component of the reflex response following the double-movement protocol (combined $M1_2 + M2_1$) to the M1 following the short duration control, the M2 response following the long duration control, and the combined $M1_2 + M2_1$ of the second double-movement protocol where the inter-perturbation interval was increased. Bonferroni-corrected t-tests were used to investigate significant main effects and interactions. The level of significance was set at 0.05. Results are reported as mean ± standard deviation.

Results

Effect of the duration of perturbation

Figure 2 displays typical reflex responses in the biceps muscle from an individual subject as the time duration of the applied perturbation is manipulated. The M2 is absent in this individual following perturbations of short duration, but becomes apparent as the time duration is extended. The critical time duration for each individual was defined as the shortest duration of perturbation that gave rise to a measurable averaged M2 response greater than the level of background EMG activity prior to the stimulus. The average critical duration was 35 ± 5 ms (range 29–41 ms, see Table 1). To examine the transition to the appearance of an M2 more clearly, we inspected the individual trials collected at each duration of perturbation. Figure 3a shows a plot of the probability of eliciting an M2 response in an individual trial (total 20 trials in each condition) against the duration of perturbation for each subject. There was a sharp increase in the probability of eliciting an M2 once the critical duration had been surpassed.

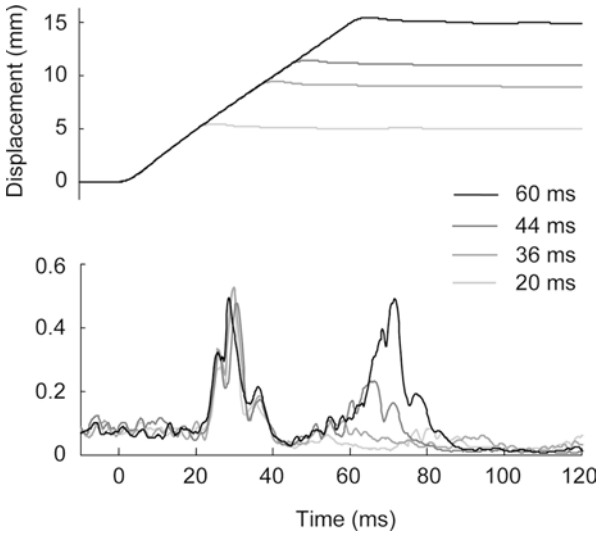

Fig. 2 Electromyographic (EMG) recordings from an individual subject showing the effect of perturbation duration on the reflex responses in the target muscles. Perturbations were delivered at 250 mm/s. Positive limb displacements reflect elbow joint extension. The M2 response in the biceps muscle is clearly dependent on perturbation duration, while the M1 is unaffected. EMG traces are the average of 20 trials

Based on each individual's critical duration, the average time durations selected for the SHORT and LONG duration perturbations were 29 ± 4 ms and 40 ± 5 ms, respectively. Group results of M1 and M2 area at the four selected durations of perturbation are shown in Fig. 3b. Clearly, the size of the M1 was not influenced by the duration of the applied perturbation ($F_{(1,8)} = 0.6$, $P = 0.4$), whereas the effect of duration was significant for the M2 ($F_{(1,8)} = 64.0$, $P < 0.001$). Following the 8-ms perturbation, EMG activity during the M2 time period was significantly reduced compared with the

Table 1 M1 onset latency, M2 onset latency, and the critical time duration for eliciting an M2 for each subject (velocity 250 mm/s). The critical duration was determined to the nearest 2 ms

Subject	M1 latency (ms)	M2 latency (ms)	Critical duration (ms)
1	22	62	33
2	23	58	41
3	23	59	31
4	22	52	31
5	20	58	41
6	21	48	29
7	23	59	41
8	24	58	29
9	22	55	39
Avg	22 ± 0.1	57 ± 4.2	36 ± 5

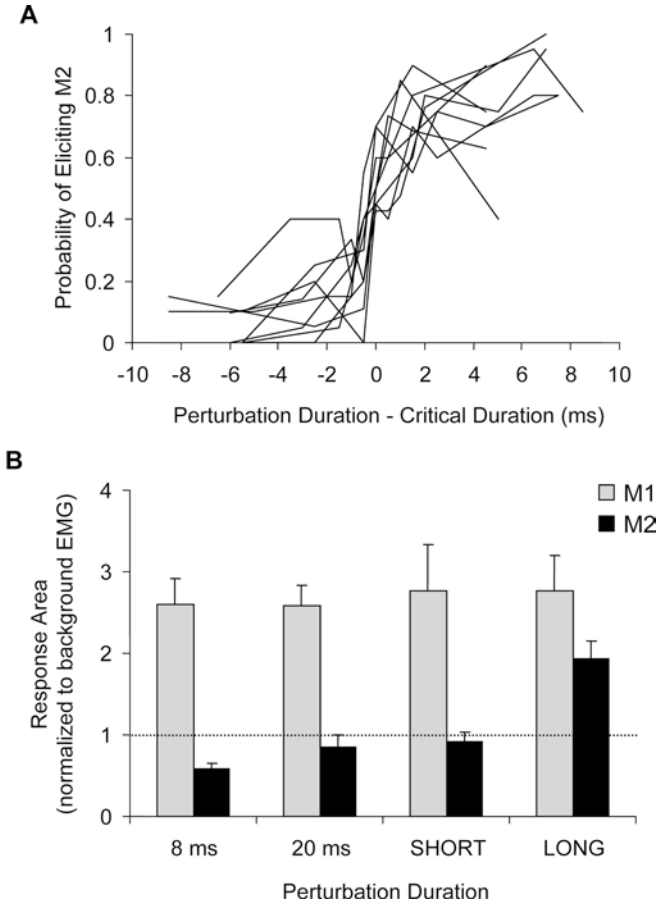

Fig. 3 **a** The probability of eliciting an M2 in an individual trial as a function of the duration of the perturbation. Each line represents an individual subject. The *x* axis is expressed as the duration of the perturbation less the individual critical duration (milliseconds), where negative values indicate a perturbation shorter than the critical duration. Twenty responses were collected at each duration. **b** Group averages of biceps M1 and M2 response area at four durations of perturbation. The SHORT and LONG duration perturbations were approximately 80 and 120% of each individual's critical duration. While the M1 is not influenced by the duration of the perturbations, an M2 response is only elicited following the LONG duration. Error bars indicate 1 SEM

background EMG prior to the perturbation ($P < 0.001$). When the duration of the perturbation was lengthened to 20 ms, EMG activity was not significantly different from background levels; the same was true for the SHORT perturbation (both $P > 0.1$). Following the LONG perturbation, a clear M2 response was evident in the averaged EMG recordings.

It is possible that the relative reduction in EMG activity in the M2 time period following the shorter duration perturbations may result from the abrupt termination of the applied movement, or to the slight positional overshoot and correction that occurs at high velocities. To investigate this, in one subject we applied a series of perturbations in which the movement was initiated in a linear manner but was then subject to a constant deceleration until the final amplitude was reached. This resulted in a less abrupt termination of the movement. The reflex responses obtained are displayed in Fig. 4. The M2 remained duration-dependent with this movement trajectory, and a relative reduction in EMG activity was also apparent following the short duration perturbation. This reduced activation cannot be attributed to the rapid termination of movement that occurs in the ramp-and-hold displacements.

Effect of the velocity of perturbation

The duration of the perturbation, not amplitude, was the critical factor for determining whether or not an M2 was present. The main effects of velocity ($F_{(1,8)} = 10.0$, $P = 0.1$) and duration ($F_{(1,8)} = 30.5$, $P = 0.001$) as well as their interaction ($F_{(1,8)} = 13.9$, $P = 0.006$) were significant for the M2 area (Fig. 5). A discernable M2 was not detected following any of the SHORT duration perturbations. The distance that the actuator traveled at the fastest velocity (600 mm/s) at the SHORT duration was equivalent to or greater than the distance of the perturbation at the lower velocities in the LONG duration conditions.

For the M1 response, the two-way (velocity × duration) ANOVA revealed a main effect of velocity ($F_{(1,8)} = 21.8$, $P = 0.002$), reflecting the almost linear increase in M1 area. The size of the M1 at the different velocities of perturbation did not differ between the SHORT and LONG duration stimuli ($F_{(1,8)} = 0.004$, $P = 0.9$).

The comparison of velocity effects on the M1 and M2 may also provide information on the receptors involved

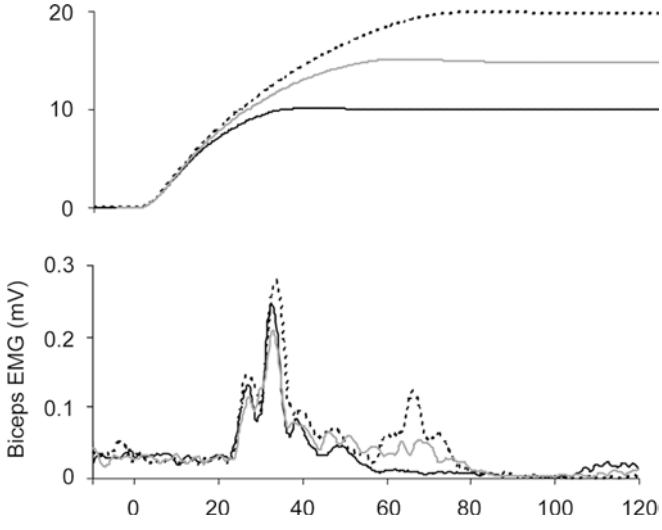

Fig. 4 Biceps electromyographic (EMG) recordings from an individual subject showing the responses following perturbations with a decelerated termination. Positive limb displacements reflect elbow joint extension. Responses are an average of 20 trials. The evoked M2 response remains duration-dependent with the less abrupt termination of the perturbation. There is also a relative inhibition of EMG activity following the shortest duration stimulus

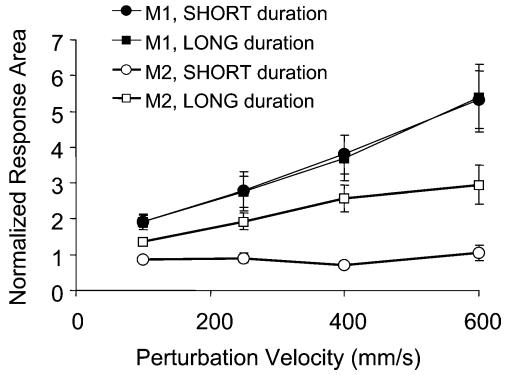

Fig. 5 Group averages of biceps M1 (*filled symbols*) and M2 (*open symbols*) response area following perturbation of SHORT (*circle*) and LONG (*square*) duration. Responses were normalized to background EMG. Error bars represent 1 SEM

in the long-latency component. The significant interaction between the duration and velocity of perturbation reflects the increase in M2 area that occurred as the velocity of perturbation increased. Initially, this was a linear relationship, but it appears that M2 area begins to plateau at the fastest velocity. A two-way (velocity × response) ANOVA was used to investigate the effect of velocity on the size of the two responses. There were main effects of velocity ($F_{(1,8)} = 27.4$, $P = 0.005$) and response ($F_{(1,8)} = 11.9$, $P = 0.001$), as well as their interaction ($F_{(3,24)} = 6.4$, $P = 0.011$). A linear fit of the initial component of the velocity-response area relationship (100–400 mm/s) showed no significant difference between the slopes for the two responses ($P = 0.6$), suggesting that the significant interaction between perturbation velocity and response type reflects the relative decrease in the M2 response at the fastest velocity.

Double-movement paradigm

In the double-movement paradigm, two short perturbations (less than the critical duration) were applied to generate a total time duration of perturbation that was greater than the critical duration. To substantiate the converging input hypothesis, a facilitated response to the second perturbation would be required. This facilitation would represent an interaction between the volley mediating the $M2_1$ and the $M1_2$ responses. The results of the double-movement paradigm, as well as the associated control conditions, are displayed in Fig. 6.

A second response at the $M2_1$ time period was present when the second component of the movement was applied. The size of this response was not significantly different from the responses recorded in any of the control conditions: the M2 response present when a single perturbation of the same total time duration was given; the M1 response to the initial stimulus; and the second response when the second perturbation was delayed by 10 ms to ensure that an interaction did not occur ($F_{(1,8)} = 2.5$, $P = 0.2$).

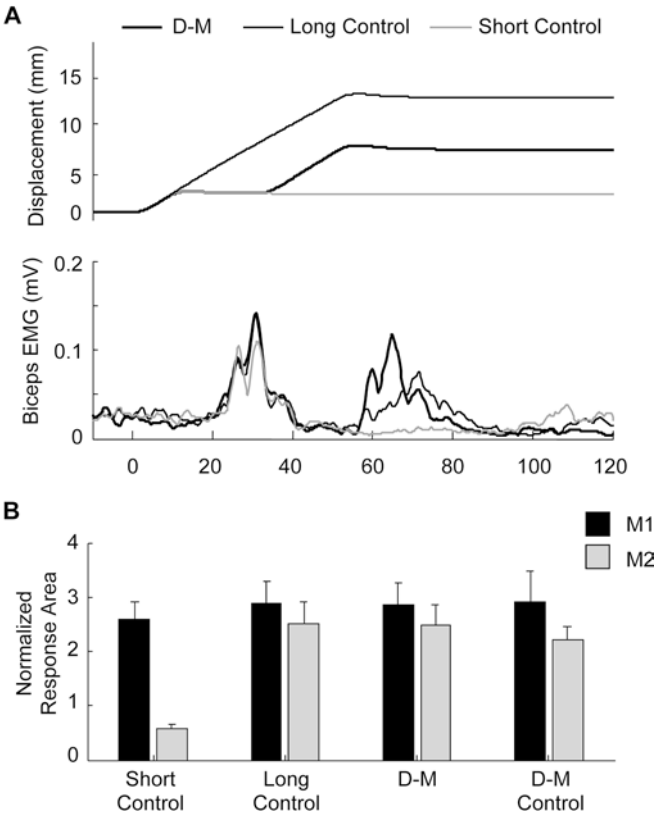

Fig. 6 **a** Biceps electromyographic (EMG) recordings from an individual subject showing the response to the double-movement protocol. Positive limb displacements reflect elbow joint extension. Responses are an average of 20 trials. The reflex response following the double movement (D-M) is shown by the *thick black line*. The second movement was timed so that the afferent volley would reach the spinal cord just prior to a volley mediating the M2. A control condition in which a single perturbation of the same time duration as the double movement is also shown (long control, *thin black line*), as well as a single perturbation comparable to the first component of the double movement (short control, *thin grey line*). **b** Group results of M1 (*dark bars*) and M2 (*light bars*) response size in the double-movement and control conditions. In double-movement control condition (D-M control) the second movement was delayed by a further 10 ms to ensure that an interaction between excitatory inputs did not occur. The time window over which the M2 response size was evaluated was also delayed by 10 ms in this condition. Overall, there was no difference in the size of the M2 response in the double-movement, long control, or double-movement control conditions. Error bars represent 1 SEM

Discussion

The results of our study are the first to demonstrate that the M2 response in the biceps brachii is dependent upon the time duration of the applied perturbation. These findings fit well with those noted at the wrist joint by Lee and Tatton (1982) and suggest that the influence of the duration of the perturbation on the M2 may be a common finding across distal and proximal joints of the upper limb. It also substantiates the possibility that the induced reflex responses arising from brief mechanical or electrical stimuli (e.g., Deuschl and Eisen 1999; Corden et al. 2000) are not equivalent to those induced by

longer duration joint displacements that give rise to a sustained period of muscle stretch.

Effect of the duration of perturbation on M2

The converging input hypothesis put forward by Lee and Tatton (1982) required simultaneous excitatory inputs for the motoneurons to generate a long-latency response. One of these inputs would represent a long-latency response to afferent activity generated at the onset of the perturbation. It was proposed that, during a long duration perturbation, the continued peripheral input from muscle spindle receptors would provide a second excitatory input. Our results did not support the proposal that appropriately timed converging input from a peripheral origin could elicit an M2 response. By initiating a second perturbation a short time after a brief initial stimulus, we ensured that afferent input mediating an M1 response ($M1_2$) would arrive at the motoneurons at time period coincident with a secondary input mediating the $M2_1$ volley. Thus, a facilitation of the $M1_2$ response would be expected if a secondary excitatory input were present. The response obtained in this condition was not different from a normal M1 at the equivalent velocity or an M2 elicited following a single, long duration perturbation. The fact that a larger M2 response could be elicited at this same time period when the velocity of perturbation was increased indicates that motoneuron output was not a limiting factor in the size of the response to the double-perturbation (Fig. 5).

Previous studies have indicated that the H-reflex response is depressed for up to several hundred milliseconds following prior activation of the monosynaptic reflex arc (Magladery et al. 1951; Curtis and Eccles 1960; Hultborn et al. 1996; Kagamihara et al. 1998; Rossi-Durand et al. 1999; Schindler-Ivens and Shields 2000). This effect has been attributed to presynaptic inhibition of the Ia terminals (Schieppati and Crenna 1984; Calancie et al. 1993) or to a change in presynaptic transmitter release (Hultborn et al. 1996; Kohn et al. 1997). This depression could potentially inhibit the response to a second joint perturbation delivered within the time periods used in our study, although no significant depression was observed. We offer three alternative possibilities to account for the dependence of the M2 on the duration of the imposed perturbation: 1) decreased motoneuron firing following the M1; 2) response characteristics of the muscle spindle receptors; and 3) reduced temporal summation along the reflex pathway.

Following short duration perturbations, EMG activity was reduced significantly in the M2 time period. This reduction may obscure excitatory inputs, including those mediating the M2 response. The retention of the reduced EMG activity following the constant deceleration perturbation (Fig. 4) suggests that the actuator positional overshoot and correction was not a factor in this effect. Other potential contributors include inhibition of the Ia-motoneuron reflex pathway (Renshaw 1941; Eccles et al. 1958; Burke and Rudomin 1977; Brooke et al. 1993), the withdrawal of facilitatory input from muscle spindles at the termination of the perturbation (motoneuron disfacilitation; Poliakov and Miles 1994), or motor unit synchronization (Turker and Powers 2001). Our results demonstrating that a second M1 ($M1_2$) response, equal in magnitude to the initial M1 ($M1_1$) response, could be generated in the M2 time period following a perturbation of short time duration argue against inhibition in the Ia-motoneuron pathway. Synchronous disfacilitation of the motoneurons is highly likely to contribute to the reduced EMG response and to the observed duration dependence, as has been demonstrated in the analogous M2 responses recorded in the human masseter (Poliakov and Miles 1994). This phenomenon arises through the withdrawal of excitatory afferent input to the motoneuron at the time period of the M2 response. However, if this disfacilitation were the sole cause of the duration dependence, we would have expected to see a facilitated $M1_2$ response following the double-movement perturbation as the timing of the second perturbation ensured that excitatory afferent input was restored at the M2 time period. This was not observed. The synchronized firing and subsequent afterhyperpolarization of motoneurons following the elbow perturbation also is likely to have contributed to the reduced EMG, but since this event is linked to the perturbation onset, it would not contribute to the duration dependence of the M2 response.

The firing characteristics of muscle spindle receptors also may contribute to the duration dependence of the M2 response. Muscle spindle primary afferents respond to ramp-and-hold perturbation with an initial burst of activity followed by a linearly increasing firing rate (Matthews 1972). It is possible that the M1, which is not influenced by the duration of the perturbation, is mediated by the dynamic component of the spindle response and that the M2 is mediated by the tonic component. If the perturbation was of sufficiently short duration that the tonic component was not elicited or was abbreviated in duration, an M2 would not be generated. However, recent intracellular recordings from motoneurons suggest that the initial burst does not contribute significantly to subsequent motoneuron firing patterns (Haftel et al. 2004). Alternatively, the M2 could be mediated by group II receptors, which are less insensitive to rapid, short perturbations, although we argue against this possibility below. Hence, there is currently little evidence supporting a direct role for muscle spindles in the observed duration dependence.

A third possibility is that temporal summation of an excitatory input is required at some component of the reflex loop for an M2 to be elicited. If the perturbation is halted prior to a sufficient level of temporal summation, the post-synaptic neuron would fail to reach threshold and the M2 would not be generated. It should also be acknowledged that the duration dependence feature of the M2 may arise through a combination of the mechanisms mentioned above.

Sensory receptors mediating the M2

Our results suggest that the biceps M2 response is mediated by group Ia afferents. The average critical duration for eliciting an M2 in our subjects was approximately 35 ms. This compares with 44 ms reported at the wrist joint (Lee and Tatton 1982). At the 250 mm/s velocity employed in our study, the M1 response was generated at an average latency of 22 ms. This represents a 7–12 ms difference from the M1 onset reported in the wrist flexor muscle (30–35 ms), which is similar to the difference in critical duration between the two joints. The average M2 onset latencies were almost identical in the biceps (48–62 ms) and wrist flexor muscles (55–65 ms). If the delayed latency of the M2 were due to peripheral factors such as a slower conduction velocity of the sensory fibers, it would be expected that the difference in M2 latency between the two joints would be more marked than the difference in M1 latency. Our results instead provide support for a central delay in response latency rather than a lower peripheral conduction velocity.

Although only a limited range of velocities was investigated, the finding that the initial component of the velocity-response area relationship was equivalent between the M1 and M2 also supports the contention that a similar, velocity-sensitive receptor mediates the two responses. At the fastest velocity of perturbation there was evidence that the M2 response size was beginning to plateau. The feature has been demonstrated previously, and it is suggested that it arises through motoneuron refractoriness following a preferential firing at the M1 time period following fast duration perturbations (Calancie and Bawa 1985).

Conclusions

In summary, we have demonstrated a clear influence of the time duration of the perturbation on the M2 response in the biceps muscle. The results of the double-movement protocol did not provide support for the hypothesis that the effect of duration is related to a requirement of converging excitatory input at the motoneuron. Alternatively, we propose that a reduction in ongoing EMG activity following the M1, firing characteristics of the sensory receptors, or reduced temporal summation may give rise to the dependence on the duration of the perturbation. In addition, our results suggest that the M2 response in the biceps muscle is mediated by velocity sensitive receptors and is therefore likely to involve a contribution from Ia receptors.

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