Long-Latency Component of the Stretch Reflex in Human Muscle Is Not Mediated by Intramuscular Stretch Receptors

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Corden, D. M., O.C.J. Lippold, Katie Buchanan, and Caryll Norrington. Long-latency component of the stretch reflex in human muscle is not mediated by intramuscular stretch receptors. J Neurophysiol 84: 184–188, 2000. Reflex responses to mechanical stimulation of muscle (brief imposed movement) were investigated. Reflexes were elicited in the forefinger, recording from the first dorsal interosseous (FDI), and in the foot, recording from soleus. These responses typically consisted of a short-latency component (M1) and a long-latency component (M2) at 33 ms and 53 ms, respectively, after the stimulus in the case of FDI, and 37 ms and 68 ms, respectively, in soleus upon stimulation of the sole of the foot. Normally, when a muscle is stretched by a mechanical stimulus (either naturally or by an experimentally imposed movement), both skin receptors and muscle stretch receptors are activated. It is possible, however, to devise stimulation parameters where this is not the case. Fixating the finger with plasticine enables the effects of skin stimulation to be studied without stretching the FDI muscle. On the other hand, tapping a long tendon allows muscle stretch receptors to be activated without involving skin or subcutaneous structures. Component M1 was always abolished by finger fixation in 40 trials on 10 subjects, with M2 being essentially unchanged in latency, duration, or amplitude. Reflex responses were obtained in soleus muscle in nine experiments by prodding the sole of the foot (thereby stimulating both skin and muscle stretch receptors). Alternatively, the tendon achilles was prodded (which solely activates stretch receptors in the muscle). In the former, M1 and M2 were generated. In the latter, only M1 was produced. It is concluded that the long-latency component of the stretch reflex, M2, originates in skin and/or subcutaneous nerve terminals and that no part of M2 originates in muscle stretch receptors.

INTRODUCTION

When a limb is subjected to an external force, a complex reflex response occurs in the muscles that are stretched. In particular, if the muscle is voluntarily contracting at a small proportion of its maximum, a series of bursts of electromyographic activity (EMG) is elicited. The first component of this response is generally agreed to be the spinal stretch reflex, since its latency is compatible with the delay involved in the monosynaptic activation by Group Ia spindle afferents.

The second component, first found by Hammond (1955), is still the subject of considerable controversy. Its origin is often ascribed to a long-loop reflex that traverses the cerebral cortex because its latency is long enough to allow for this. Hammond et al. (1956), Phillips (1969), Evarts (1973), Marsden et al. (1972, 1976), Thilmann et al. (1991), and Day et al. (1991) espoused this view. Lee and Tatton (1975) found that the reflex response to stretch in wrist muscle is fractionated into two major components, which they called M1 and M2. According to them, the wave M2 is a transcortical reflex initiated by primary muscle spindle endings.

The supraspinal mechanism of the second peak in the EMG response (M2) has been questioned by Houk (1978, 1979) and Bawa and Tatton (1979). Matthews (1985, 1989) stated that the wave M2 is caused by Group II muscle spindle afferents, the lower conduction velocity of which accounts for the longer latency. Hagbarth et al. (1981) recorded from Ia spindle afferents in humans and found bursts of action potentials that they categorized as oscillations caused by a damped mechanical tremor in the stretched muscle. Capaday et al. (1991) published evidence for a contribution by the motor cortex to the long-latency stretch reflex in the thumb. This was based on the finding that stretching one thumb led to a long-latency EMG response in the corresponding muscle on the contralateral side. Peterson et al. (1998) found a cortical pathway involved in a component of the stretch reflex in the leg, but it is the third burst, M3, at a latency of 95 ms, and not M2.

All the explanations for the origin of M2 given above rely on the assumption that the primary event eliciting both M1 and M2 is the excitation of muscle spindles by the imposed stretch. However, Darton et al. (1985) suggested that M2 is the result of stimulation of afferent terminals in the skin and/or subcutaneous tissues by the mechanical device producing the muscle stretch.

To settle this controversy, it is first necessary to see whether both M1 and M2 originate in the excitation of muscle spindles or whether M2 has a different origin. It should be possible to devise stimulus parameters that will activate either skin and muscle together (msk), skin alone (sk), or muscle alone (m). If the reflex responses to stimulus (msk) differ from those to (sk) or to (m), this would provide very strong evidence for the origin of the long-latency component of the stretch reflex not being in muscle spindles.

Experimentally, fixating the limb but still applying the mechanical stimulus will satisfy (sk). For (m), muscles with long tendons can be stretched without simultaneously stimulating their muscles’ associated skin areas. The tendon can be depressed by a force applied at right angles to its long axis, which

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will generate a large enough displacement of the muscle fibers to give a reflex response, thus satisfying (m).

An example of the latter is soleus. This muscle can be stretched by prodding the sole of the foot, which necessarily involves activating the skin and subcutaneous receptors as well as stretching the muscle [experiment (msk) above]. It can also be stimulated by prodding the tendon achilles; stretching alone is produced and the relevant skin area, at least 20 cm distant from (m), is not involved.

**METHODS**

**Subjects**

With the prior approval of the Local Ethical Committee, the first dorsal interosseous (FDI) muscles of 10 subjects, five male and five female aged 18 to 65, were investigated. In general, the methods employed followed those described by Darton et al. (1985). The soleus muscles of an additional group of five subjects were also studied in nine experiments.

**Electrical recording from FDI**

Surface recording electrodes were rectangular pure silver plates (1.0 × 1.5 cm) placed as shown in Fig. 1A. Inter-electrode resistance was kept below 2 kΩ.

**Signal processing**

An electromyogram (EMG) was obtained by amplifying the signal via a low frequency filter with its 3 dB point at 80 Hz and a high frequency filter at 30 kHz. The full-wave rectified EMG was fed to a hard-wired averager whose sweep was triggered by the mechanical stimulus. Hard copy was obtained from an X-Y plotter. Sweep time was either 160 or 250 ms, cycle time was 677 or 731 ms, and either 128 or 256 sweeps were averaged.

Averaging a series of signals is a process fraught both with obvious and with obscure difficulties and hazards. The presence of movement artifacts caused by mechanical stimulation leading to relative movement between skin and electrodes and/or microphony in the electrode leads can be checked. If the rectifier is shorted out, the averaged response should be essentially flat. Any waveforms (especially of a time-course or latency similar to the genuine reflex responses) that do remain will reverse in phase in the averaged record when the electrodes are transposed. Examination of individual single sweeps also often shows the component muscle action potentials comprising the final average. For checking this, it is useful to use simultaneous display of the primary signal from the muscle, its rectified equivalent, and the accruing average. Other controls are given in Darton et al. (1985).

**Stimulation**

A crystal-controlled oscillator timed the events. The initial pulse started the averager sweep and, after a delay, a mechanical pulse (lasting 5 ms) was delivered to the skin over the end of the distal phalanx of the index finger. The direction of the prod was in the plane of the palm of the hand. Care was taken to ensure that precisely the same skin area was stimulated throughout each experiment. The displacement of the prod producing the mechanical stimulus varied between 0 mm and 2 mm. Peak force, measured with a strain gauge, was up to 1.9 N. The cross-sectional area of the tip of the prod was 0.9 mm². The parameters of the mechanical stimulus, after preliminary adjustment, were kept identical throughout each experiment.
traction strength of approximately 10% of a maximum voluntary contraction (MVC) was used. It was monitored isometrically with a strain gauge. A meter provided visual feedback of tension to the subject. In a few experiments the required background activation was achieved by the subject monitoring the integrated EMG as displayed on a meter. The EMG associated with this background activation was monitored once per sweep by a shorting switch that grounded the input electrodes at certain times during the averaging process. Its level was the same throughout procedures (msk), (sk), and (m).

Fixation of leg, ankle, and foot

The apparatus described by Lippold (1952) was used (Fig. 1B). In addition, the footplate had the back half of a shoe screwed on, to assist precise (and reproducible) location of the foot. The axis of rotation of the footplate was through the ankle joint. This enabled the contraction of soleus to be essentially isometric while the weight of the leg and foot itself did not contribute to the force that was developed. Force was measured by a pressure gauge connected by pressure tubing to a cylinder and piston. For most experiments, the required background voluntary contraction of approximately 10% MVC was maintained by the subject using this pressure gauge. It was necessary to ensure that the knee joint and ankle joint were kept at 90° of flexion in all experiments.

Electrical recording from soleus

The same electrodes were used as for FDI and were placed over the lateral border of soleus, which is subcutaneous (see McMinn and Hutchings 1988), through the lower one-third of the calf.

Mechanical stimulation of soleus

The prod to the sole of the foot (msk) or the tendo achilles (m) was given by a printed motor driven by the same power supply, etc., as was used for FDI. Force was up to 2.5 N and displacement was up to 2.0 mm in each case. The pulse width of the power supplied to the printed motor was 5 ms.

Terminology

There are commonly three components in the reflex EMG response to stretch. They are usually termed M1, M2, and M3. In the present study, the components were defined in terms of their latency. The onset of M1 is 30–35 ms in FDI and 35–40 ms in soleus. The latency of M2 is 50–60 ms in FDI and 60–80 ms in soleus, whereas M3, when present, has its onset at least 100 ms after M1. These latencies differ by margins large enough to render confusion between M1 and M2 or M2 and M3 unlikely. Comparison of our results with those of previous authors must be made in terms of the latencies and durations of the waveforms M1, M2, and M3. For the stimulus parameters and muscles we used, our results are typical.

RESULTS

Effect of finger fixation on reflex responses of FDI, experiments (msk) and (sk)

Figure 2 shows how fixing the finger affects the components of the reflex response to mechanical stimulation. Line (msk) shows the typical segmented complex in the rectified and averaged EMG, the forefinger being free to move. At 50 ms, the 5 ms mechanical pulse was given. With a latency of 33 ms, the first component, M1, begins. At a latency of 53 ms, M2 follows. The amplitude of these two components is 25 μV and 28 μV, respectively. In all 10 subjects, a total of 40 responses of this nature were obtained.

Line (sk) shows the response when the forefinger is immobilized with plastocine. Component M1 is now absent and the only burst of EMG commences at 54 ms and is of the same form and duration as M2 in line (msk). Therefore it is very likely to be the expression of M2 under these circumstances. In all 10 subjects, M1 was abolished by immobilizing the forefinger. This elimination of M1 by fixing the finger contains its own internal control (a lack of M1) for minimizing muscle stretch.

It can be inferred from these results that, when the FDI muscle is stretched by a mechanical prod to the forefinger, both muscle stretch receptors and skin and subcutaneous tissue beneath the prod are activated. This gives M1 and M2, respectively, in the reflex response. When the finger is firmly fixed, however, the prod excites skin and/or cutaneous receptors (as before) but not muscle spindles, since the muscle is prevented from moving, and M1 does not arise. M2 is present as usual. Thus M2 cannot arise in muscle stretch receptors.

In most experiments a pronounced inhibition was seen in the records that were obtained when the forefinger was immobilized and only the skin was stimulated (sk). The latency of onset of the inhibition was 45–50 ms and its presence might affect the precise start of the wave M2, although not by a margin sufficient to confuse the identification of this component as M2. This inhibition is present in the control records (msk) but is partly or completely cancelled out by the excitatory M1.

The origin of this inhibition cannot be in 1b afferent from activation because the absence of M1 clearly shows that the muscle and its tendon were not being stretched. Cacchia et al. (1973) and Garnett and Stephens (1980) have shown conclusively that cutaneous stimulation, both electrical and mechanical, gives rise to an inhibition commencing after approximately 45–50 ms and lasting approximately 25–30 ms. Both of these figures correspond with the parameters found in our experiments, so we deduced that the inhibition is likely to result from the activation of cutaneous afferents.

Reflexes when soleus is stretched via skin (msk) and via tendon (m)

In nine experiments, when the the sole of the foot was prodded so that the foot was dorsiflexed, the usual configura-
ably caused by a mechanical artifact. In most experiments an
essarily conform with that usually found for M2. It was prob-
sometimes be seen in the record but the latency did not nec-
gave rise to waveform M1. Wave M2 was absent.

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ning reflex responses to mechanical stimulation, can differenti-

Controversy still surrounds the origin of the long-latency
component [named M2 by Lee and Tatton (1975)] in the
human stretch reflex. Burne et al. (1984) showed that the
stretch reflex component in physiological tremor can be abol-
ished by stopping the mechanical movement in a trembling
limb. The same technique of fixing the forefinger, when study-
ing reflex responses to mechanical stimulation, can differentiate between a skin and a muscle spindle origin for any component. Thus if M1 is abolished when the finger is fixed, it must arise as a reflex response to muscle stretch. Conversely, the fact that M2 is still present, as large as before, when the finger is fixed indicates that M2 cannot be caused by muscle spindle activation in muscle stretch.

In all of the experiments, M1 was completely abolished by the fixing process. M2 was not decreased in amplitude by the procedure and, therefore, it is very unlikely that any part of the
M2 component is generated by the stretching of muscle (or stimulation of joint receptors). All of M2 must arise from skin and/or subcutaneous tissue stimulation by the mechanical prod.

Differing origins for M1 and M2

Other less conclusive evidence has been published that indicates differing origins for M1 and M2. Corden et al. (1988) have shown that the ratio of areas of M1 and M2 varies with age. In subjects less than 30 years old, this ratio is between 0.5 and 2. Above the age of 30, it declines to well below 0.5. In elderly persons 60 years old and more, M1 is often absent (under the conditions of these experiments). This also argues for a separate origin for M1 and M2.

Components M1 and M2 are differentially affected by fatigue. The short-latency reflex response is abolished by a brief maximal voluntary contraction in a small intrinsic hand muscle whereas the long-latency component is not (Corden and Lippold 1989). This also points to a mechanism responsible for M1 that is different from that of M2.

Dawson et al. (1987) and Milne et al. (1985) showed a dichotomy in the responses of M1 and M2 produced by external factors. They found that a contralateral intramuscular electrical stimulus altered the M1/M2 ratio during and after its application. During continuous electrical stimulation of a contralateral arm muscle, these authors found a diminution of the M1/M2 ratio from 0.85 to 0.40 in the pooled results of 17 subjects.

Abolition of M2

The complementary experiment is to demonstrate a situation in which M1 is generated in the absence of M2. Darton et al. (1985) showed that M2 is abolished by skin anesthesia. This can also be achieved if the muscle can be stretched without involving skin and subcutaneous receptors. Muscles with long tendons, such as gastrocnemius/soleus in the leg, allow this to be done. Stimulating the sole of the foot with a prod stretches the soleus via its functional skin field. We found that M1 and M2 were generated [Fig. 3, (msk)].

Stimulating the tendon (i.e., eliciting the ‘ankle-jerk’) never produced M2, although M1 was always present. The skin over the tendon is not involved in normal reflex action in soleus [Fig. 3, (m)].

Muscle stretch receptors involved

It is self-evident that Group Ia muscle spindles cannot be responsible for component M2. No matter which pathways are involved, or what their reflex latencies might be, fixing the finger completely abolishes M1, which must mean that these receptors are not then being activated by the stimulus. M2, however, remains at its original size under these conditions and must therefore originate elsewhere.

Group Ib receptors are primarily inhibitory in function and presumably are not involved in generating M2. In addition, the above considerations (in previous paragraph) also apply and Ib endings are unlikely to be excited by any residual vibration, etc., if Ia endings are silent.

Group II fibers originate in secondary muscle spindles, fascial endings, and other nonspecific sensory systems, and have a conduction velocity that is compatible with reflex production.
of M2. They are less sensitive and have a higher threshold to dynamic stretch than primary spindles (for review see Hasen and Stuart 1984) and are therefore very unlikely to originate M2 at an extremely low level of extraneous movement when finger fixation might be incomplete.

Strong evidence for M2 not originating in any stretch mechanism within muscle is provided by the fact that the size of M2 remains essentially constant when M1 is abolished by fixation. If any intramuscular origin for M2 is involved, the amplitude of the M2 waveform would be expected to be reduced, at least in part, by the fixation. It was not. Corden and Lippold (1996) showed the relation between the force of a mechanical prod to FDI and the voltage-time integral of the rectified and averaged EMG responses. As force is experimentally decreased, both M1 and M2 decrease in size with similar regression slopes.

These experiments show that the long-latency component is very likely to be generated in skin and/or subcutaneous tissues. However, they cast no light on the reflex path itself. In particular, the problem of whether or not a cortical loop is involved is not addressed.

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REFERENCES


CACCHIA MR, MCCOMAS AJ, UPTON ARM, AND BLOGG T. Cutaneous reflexes and M2 decrease in size with similar regression slopes.


