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EVIDENCE FOR A CONTRIBUTION OF THE MOTOR CORTEX TO THE LONG-LATENCY STRETCH REFLEX OF THE HUMAN THUMB

BY C. CAPADAY, R. FORGET*, R. FRASER AND Y. LAMARRE†

*From the Centre de Recherche en Sciences Neurologiques and the *Ecole de Readaptation and Hôpital Hotel-Dieu de Montréal, Faculté de Médecine, Université de Montréal, C. P. 6128, Succursale A, Montréal, Québec, Canada H3C 3J7*

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SUMMARY

1. In normal subjects, transcranial magnetic stimulation of the hand region of the motor cortex evokes motor responses only in contralateral hand muscles at a latency of about 19–24 ms. In contrast, stimulation of the motor cortex of three mirror movement subjects evoked, nearly simultaneously, motor responses in hand muscles on both sides of the body at latencies similar to those of normal subjects. In these subjects no other neuroanatomical pathways appear to be abnormally directed across the mid-line. Thus, their mirror movements are probably due to a bilateral projection of the corticospinal tract to homologous motoneurone pools on each side of the body.

2. We reasoned that if the motor cortex contributes to the generation of long-latency stretch reflex responses then in these mirror movement subjects stretching a muscle on one side of the body should produce long-latency reflex responses in the ipsilateral and the homologous contralateral muscle.

3. To test this idea experiments were done on normal human subjects and on the subjects with mirror movements. The electromyographic (EMG) activity of the flexor pollicis longus muscle (FPL) on each side of the body was recorded. Stretch of the distal phalanx of the thumb of one hand produced a series of distinct reflex EMG responses in the ipsilateral FPL. The earliest response, when present, began at 25 ms (S.D. = 3·5 ms) and was followed by responses at 40 (S.D. = 3·9 ms) and 56 ms (S.D. = 4·3 ms). There was no difference, either in timing or intensity, between the ipsilateral FPL EMG responses of normal subjects and those of the mirror movement subjects.

4. No response of any kind was observed in the contralateral (unstretched) FPL of normal subjects. In contrast, we observed in all three mirror movement subjects EMG responses in the contralateral (unstretched) FPL beginning at 45–50 ms. The latency of this response is considerably shorter than the fastest voluntary kinaesthetic reaction time, which was on average 130 ms (S.D. = 11 ms). The contralateral long-latency EMG response observed in the mirror movement subjects was on average 30% (range 5–60%) of that on the ipsilateral side. No short-latency response (25 ms) was ever observed in the contralateral FPL of these subjects.

† To whom reprint requests should be sent.

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5. These observations are quite consistent with the idea that the long-latency stretch reflex responses of hand and finger muscles are produced, at least in part, by the motor cortex.

INTRODUCTION

Phillips (1969) was the first to suggest that in muscles of the primate hand the stretch reflex response may include a contribution from the motor cortex. Soon afterwards Marsden, Merton & Morton (1972, 1976) identified a stretch reflex response in the flexor pollicis longus muscle (FPL) whose latency ($\sim$ 40 ms) was intermediate between that of the monosynaptic response and the fastest kinaesthetic reaction. They suggested that this response, now commonly referred to as the long-latency stretch reflex, is mediated by the motor cortex. Lee & Tatton (1975) found a similar response (M2) in wrist muscles which they also suggested was a transcortical reflex.

Three alternative mechanisms have been proposed to explain how long-latency stretch reflex responses are generated. Matthews, until recently, suggested that the response was produced by group II muscle spindle afferents via a spinal cord circuit (Matthews, 1985; cf. Matthews, 1989). Hagbarth, Young, Hagglund & Wallin (1980) suggested that muscle stretch produces distinct bursts of activity in the Ia spindle afferents which in turn, via the monosynaptic pathways, produce the various bursts of EMG (electromyogram) activity. It has also been proposed that the long-latency responses are not mediated by muscle spindle afferents but rather by large-diameter cutaneous afferents (Darton, Lippold, Shahani & Shahani, 1985). Even the clear demonstration that, in monkeys, cells of the motor cortex which produce post-spike facilitation of EMG activity in wrist muscles also respond to stretch of their target muscle (Cheney & Fetz, 1984) has not settled the issue. The pathway may not have sufficient gain to directly discharge motoneurones and may only facilitate them subliminally.

Clearly, the neural mechanisms by which the long-latency responses to muscle stretch are produced in humans remains to be elucidated. In this paper we present results obtained from three subjects with congenital mirror movements that are pertinent to this issue. When subjects with congenital mirror movements are asked, for example, to flex the distal phalanx of the right thumb they also involuntarily and inevitably flex the homologous joint on the left side. We found, as a preliminary to the present study, that in the mirror movement subjects, stimulation of the hand area of the motor cortex of one hemisphere produces, nearly simultaneously, motor responses in homologous muscles on both sides of the body. This is, as well as could be determined, the only neurological abnormality of these subjects. (Mirror movement subjects without developmental or other neurological abnormalities are rare (Forget, Boghen, Attig & Lamarre, 1986).) They thus provide a unique opportunity for a nearly direct test of the transcortical stretch reflex hypothesis.

If the motor cortex contributes to the long-latency stretch reflex responses then, in these subjects, stretching a muscle on one side of the body should produce long-latency EMG responses in that muscle and in its contralateral homologue. This is indeed what we observed in these subjects, but never in normal subjects. A brief account of the present observations has been published (Capaday, Fraser, Forget &

Lamarre, 1989). Very recently, observations similar to our own were reported based on the study of one patient with the Klippel–Feil syndrome and mirror movements (Matthews, Farmer & Ingram, 1990). Our study complements and extends that of Matthews *et al.* (1990) in several respects. In particular, our subjects do not have the Klippel–Feil syndrome, and thus other potential neurological abnormalities, in addition to mirror movements (see for example, Forget *et al.* 1986). For example, the present subjects do not have contralateral long-latency cutaneous (E2) reflex responses. Therefore, the contralateral long-latency stretch responses in these subjects are uncontaminated by a contribution from cutaneous afferents. Taken together, the observations of Matthews *et al.* (1990) and the present ones are strong evidence for the functional operation of a transcortical stretch reflex during voluntary motor activity of the hand.

METHODS

Subjects

We used a total of eleven subjects, three of whom had congenital mirror movements. All subjects gave their consent after being informed of the nature of the experimental procedures. The experiments were carried out in accordance with the guidelines of the Declaration of Helsinki (as reprinted in the *British Medical Journal*, 1964) regarding the use of human subjects in experimental studies.

The case report of one of the subjects with mirror movements has been published in a recent paper (Forget *et al.* 1986). This subject (R.M.) is a left-handed male, aged 28 years who had mirror movements of the fingers and, to a much lesser extent, the wrist and elbow (biceps brachii). The mirror movements occur on either side but are more pronounced on the right side. The second subject (S.D.) was a right-handed woman aged 50 years. She has pronounced mirror movements of all the fingers, the thumb being particularly affected. She also has some slight mirror movements during pronation/supination movements of the forearm and flexion/extension of the elbow. She is affected almost equally on both sides of the body. The third mirror movement subject (M.M.) in our study was her son aged 28 years, left handed, and affected at the fingers and slightly at the wrist. In this subject also, the thumb was particularly affected. In summary, the mirror movements in these subjects are most pronounced in the hand and finger muscles. All three subjects had strong mirror movements of the distal phalanx of the thumb, which is moved by one pair of antagonistic muscles, the flexor and extensor pollicis longus. These subjects were chosen because aside from the mirror movements they had, as well as could be determined, no other neurological or physical problem such as Klippel–Feil syndrome or a myelomeningocele. Eight normal (two female and six male) subjects were used as controls.

Experimental apparatus

Subjects were seated with both forearms at a level about midway between the shoulders and the waist and supported in the prone position. The palm of each hand rested on a horizontal cylindrical stop with the four fingers curled around it. The hand was tied securely to the stop with linen bandages. The distal phalanx of the thumb of each hand was placed into a mould made of a hard elastomere material (Reprosil). The mould was placed inside a metallic holder connected to the shaft of a servo-controlled DC printed-circuit motor (PMI). The proximal phalanx of the thumb was braced by a plate and fixed to it by an adjustable metal ring. The axis of rotation of the distal phalanx of the thumb was aligned with that of the shaft of the motor. The movement about the axis of rotation was in the horizontal plane. The FPL muscle was stretched by applying a displacement of 10–12 deg to the distal phalanx of the thumb, at a constant velocity of 150 or 200 deg/s. The stiffness of the position servo-system was 1·9 N m/deg and had a maximum torque of 3·5 N m. The torque generated by the distal phalanx of each thumb was measured by strain gauges mounted on the shaft of the respective manipulandum. Each of these two signals was displayed on a large analog metre calibrated so that the full-scale deflection corresponded to the

subject's maximum torque. The two analog displays were placed about 1 m directly in front of the subject and provided a visual feedback of the relative torque produced by the distal phalanx of each thumb.

Electromyographic recording

The EMG activity of the FPL muscle was recorded by a pair of Ag–AgCl surface electrodes 8 mm in diameter. The electrodes were placed 1–2 cm apart over the distal portion of the FPL muscle in the forearm. The signals were amplified, high pass filtered at 10 Hz, rectified, and low-pass filtered at 100 Hz. They were digitized along with the shaft position signal of the torque motor at a rate of 1000 samples/s. Before every experiment we tested that the electrodes were appropriately positioned to record the EMG activity of the FPL muscle on each side of the body. We relied on obtaining reflex responses to stretch as indicative of appropriate electrode location. Thus, if stretching the distal phalanx of the thumb produced reflex EMG responses on that side then we could be sure that the electrodes were appropriately positioned.

Experimental protocol

Once proper location of the recording electrodes on each side of the body was determined the following experimental protocol was used. Subjects were instructed to maintain with each thumb a background effort of between 5 and 20% of the maximum. At each tonic level sixteen stretches were applied to the FPL. The stretches were applied at random intervals ranging from 3 to 7 s. Since the amplitude of reflex responses depends on the background level of activity in the motor pool, the amount of contralateral effort was varied in order to reveal any subliminal effects due to stretch of the ipsilateral side. Responses were obtained in up to sixteen combinations of ipsilateral and contralateral tonic effects.

Transcranial stimulation

A transcranial magnetic stimulator (Dantec) was used to stimulate the hand area of the motor cortex of normal as well as mirror movement subjects. Single stimuli of no more than 75% of the stimulator's maximum output (2·2 T) were used. The interstimulus interval was between 10 and 20 s. The landmark references C3 and C4 of the 10/20 system of cranial co-ordinates (Jasper, 1958) were measured and marked on the cranium of each subject. They were used to locate the hand area of the motor cortex of each hemisphere. In all subjects the motor cortex of each hemisphere was stimulated in turn. Simultaneous EMG recordings were made from a variety of hand and finger muscles such as the FPL, the thenar muscles, the wrist and finger flexors. All evoked motor responses were obtained with the subjects relaxed (i.e. no background EMG activity). Four normal and the three mirror movement subjects were used in the transcranial stimulation studies.

RESULTS

Mirror movement

In Fig. 1*A* two examples of a voluntary movement and its accompanying mirror movement are shown. The subject (M.M.) was asked to rhythmically abduct the thumb at his own pace. It can be seen that the abductor pollicis brevis muscle (APB) of the contralateral hand is activated nearly simultaneously with the APB muscle of the voluntary side. The reader should also note how closely the EMG activity of the mirror side resembles that of the voluntary side. For example, the small initial burst of EMG activity in the left (voluntary) APB is also clearly present in the right (mirror) APB. A detailed analysis comparing the EMG activity of the voluntary and mirror sides can be found in the paper by Forget *et al.* (1986). In the following section we provide evidence that the mirror movements of the present three subjects are due to a bilateral projection of the corticospinal tract to homologous motoneurone pools on each side of the body.

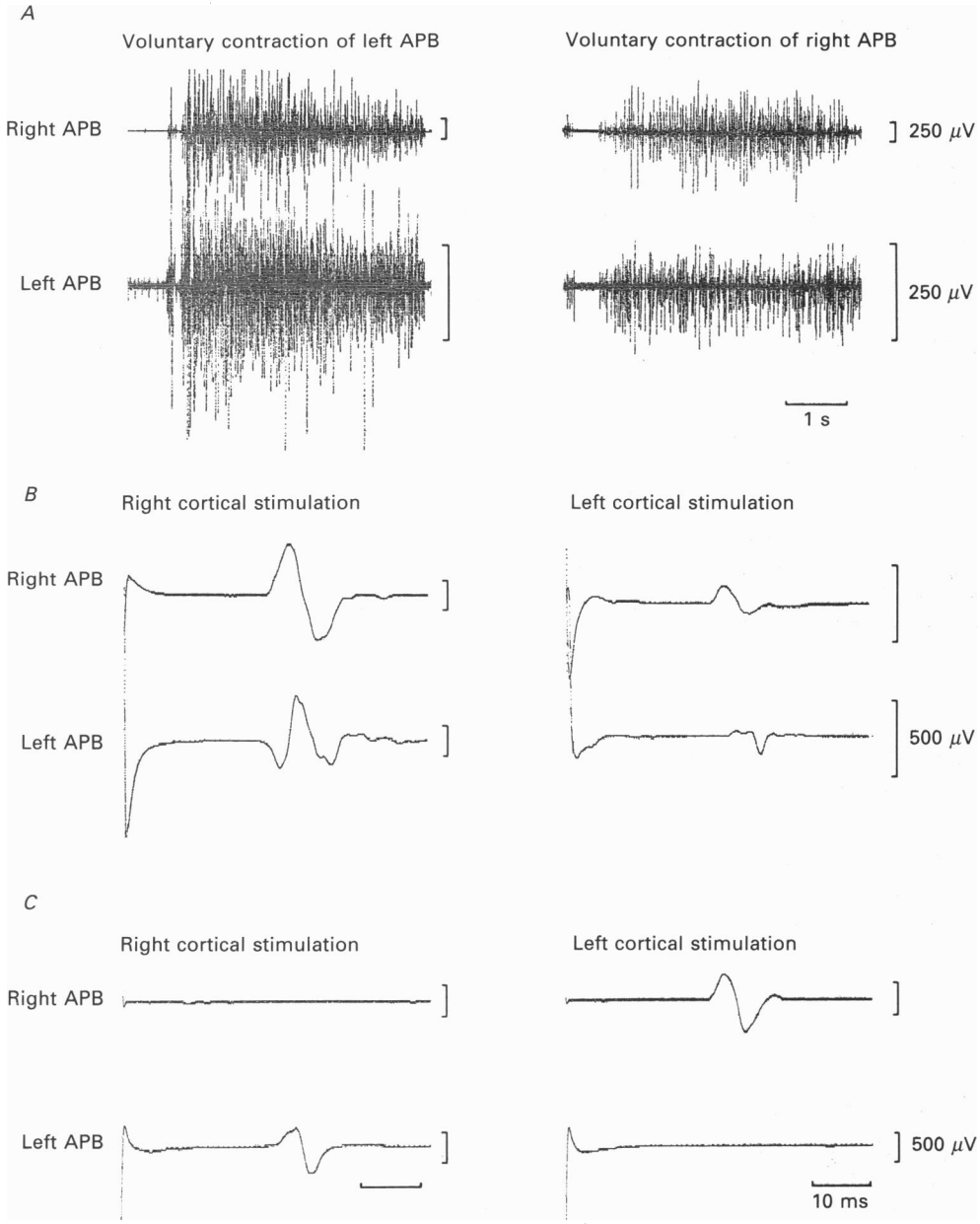

Fig. 1. *A*, example of EMG activity in the left and right APB when the mirror movement subject (M.M.) voluntarily abducted either the left or right thumb. *B*, in this subject transcranial stimulation of the hand area of the motor cortex of either hemisphere evoked EMG responses in hand muscles on both sides of the body. *C*, in normal subjects (R.F.) motor responses could only be evoked in hand muscles contralateral to the hemisphere stimulated.

Transcranial magnetic stimulation

In the four normal subjects used in this study stimulation of the hand area of the motor cortex on one side produced an evoked motor response only in contralateral hand muscles (Fig. 1*C*). The latency of the evoked EMG responses was between 18 and 24 ms.

When the hand area of the motor cortex of the mirror movement subjects was stimulated, motor responses were evoked in hand muscles on both sides of the body. In all subjects, including the normal ones, the APB was more easily activated by transcranial magnetic stimulation than the FPL and was thus chosen as a representative example. However, on those occasions when the PFL was activated by the transcranial stimulus the results were the same as with the APB. In the examples shown in Fig. 1*B* the motor cortex of each hemisphere was stimulated in turn and the EMG activity recorded from the right and left APB. The subject used was the same as in Fig. 1*A*. When the right motor cortex was stimulated a response of the left (contralateral) APB was evoked (latency 22·5 ms) as well as a clear response of the right (ipsilateral) APB muscle at nearly the same latency (23·5 ms). Stimulation of the left motor cortex also produced clear motor responses in the contralateral and ipsilateral APB (Fig. 1*B*). The latency of the ipsilateral evoked motor response was on average 2·4 ms (S.D. = 1·3 ms) longer than that of the contralateral motor response; the range was between 1 and 4 ms.

By comparing parts *A* and *B* of Fig. 1 it can be seen that the results of the transcranial stimulation studies are fully consistent with the clinical profile of this subject. That is, mirror movements occur on both sides of the body and stimulation of the motor cortex of either hemisphere produces contralateral as well as ipsilateral evoked motor responses. This was also true for the two other mirror movement subjects used in this study.

Stretch reflex responses in normal subjects

Stretch of the distal phalanx of the thumb produced in the FPL muscle a series of distinct EMG bursts (Fig. 2) that have been previously described (see for example, Marsden, Rothwell & Day, 1983; Matthews, 1984). In some subjects a burst of EMG activity was observed at a latency of about 25 ms (mean = 25 ms, S.D. = 3·5 ms). In most subjects, however, when the muscle is stretched at moderate velocities (200–300 deg/s) such a short-latency response is not observed. The example shown in Fig. 2 is typical, with the first response occurring at about 40 ms (mean = 40·5 ms, S.D. = 3·9 ms) and a second one at 53 ms (mean = 56 ms, S.D. = 4·3 ms). The latter response was the largest of the three reflex responses in all subjects investigated. As the rate of stretch increases the amplitude of the response at 40 ms increases and becomes comparable to that of the response at 55 ms (Marsden *et al.* 1983). Often, however, these two responses are not clearly separated and only a single peak is evident (see, for example, Akazawa, Milner & Stein, 1983).

In the contralateral FPL muscle of normal subjects no response of any kind was observed, even when the subject exerted a tonic effort of as much as 30–50% of maximum. An example is shown in the averaged EMG record of Fig. 2. The cumulative sum (Davey, Ellaway & Stein, 1986), which was calculated in all cases

but is not shown here, confirmed the lack of response in the contralateral FPL EMG of normal subjects. The contralateral FPL EMG activity was further analysed by a one-way analysis of variance (ANOVA). The average EMG record was divided into five 15 ms segments, one segment of which just preceded the stretch and four others

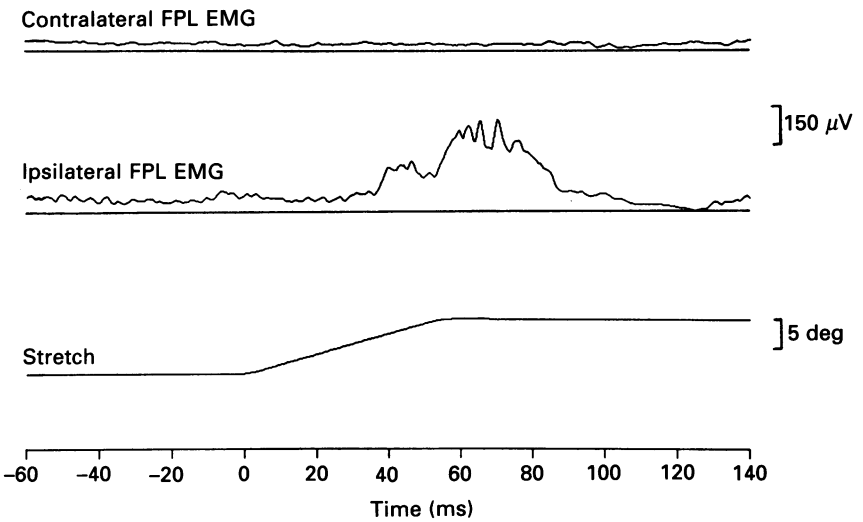

Fig. 2. Stretching the FPL in a normal subject (K.A.) produced long-latency stretch reflex responses beginning at about 40 ms. No response occurs in the contralateral (unstretched) FPL in normal subjects. In this example the subject exerted a tonic contraction of the distal phalanx of the thumb of approximately 20% of maximum on each side of the body. Each record is the average of sixteen responses. The EMG baseline level is indicated by the line segment underneath each record.

beginning 20 ms after the onset of stretch. The integrated level of activity in each of these five time bins was calculated and used in the ANOVA. No significant difference was found between the five time segments ($F = 0{\cdot}51$, $P > 0{\cdot}05$) of the contralateral FPL EMG of normal subjects.

Stretch reflex responses in subjects with mirror movements

An example of a stretch reflex response in the FPL muscle of a subject (R.M.) with congenital mirror movements is shown in Fig. 3. In this example the left FPL was stretched. The first response in the left FPL occurred at 25 ms and was followed by a large second response beginning at 43 ms. In marked contrast to the lack of response in the contralateral FPL muscle of normal subjects, contralateral FPL long-latency responses were observed in all three mirror movement subjects. These contralateral responses could be elicited by stretching either side. In the example shown in Fig. 3 the response in the contralateral (right) FPL began at a latency of about 45 ms. Of particular interest was that in this subject stimulation of the motor cortex of either hemisphere evoked motor responses in the FPL muscle on both sides of the body. Contralateral background activity was not usually required to observe long-latency EMG responses on that side.

An ANOVA, as previously described, of the contralateral EMG activity of the mirror movement subjects was done. We found, as is clear from the average record in Fig. 3, that the EMG activity in the five time segments is significantly different ($F = 27{\cdot}5$, $P < 0{\cdot}001$). To determine the onset of the first significant increase in the

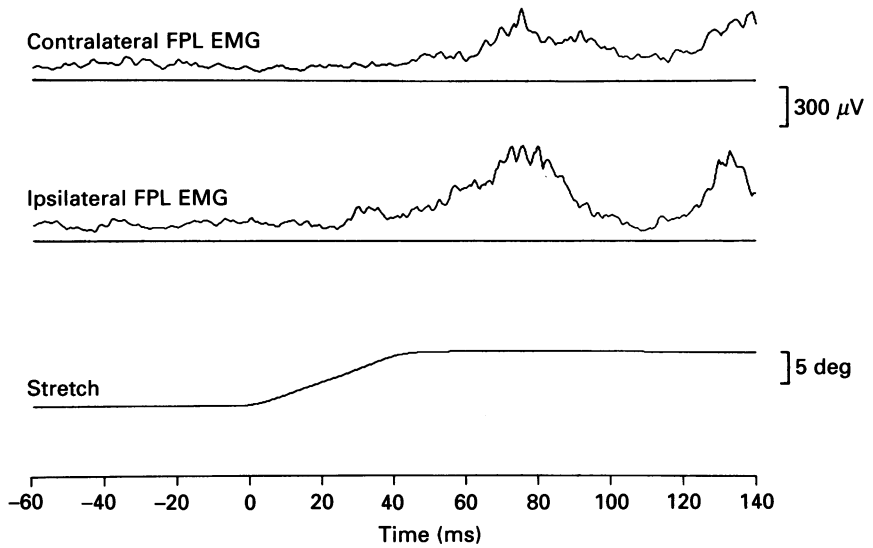

Fig. 3. In the mirror movement subject we studied (R.M.), stretching the FPL on one side of the body produced long-latency reflex responses in the FPL on both sides of the body. In this example the left (ipsilateral) FPL was stretched while the subject exerted a tonic effort of about 10% of maximum on each side. The subject was instructed to oppose the perturbation as quickly as possible. The voluntary activity can be seen to occur much later ($\sim$ 117 ms) than the long-latency reflex responses beginning at 45 ms. Each record is the average of sixteen responses. The EMG baseline level is indicated by the line segment underneath each record.

contralateral FPL EMG, an ANOVA using 5 ms time segments was done. The results confirmed the difference in EMG activity between the time segments ($F = 8{\cdot}4$, $P < 0{\cdot}001$) and a *post hoc* analysis revealed that the first significant response began between 45 and 50 ms.

When quantified by measuring the time integral of the response, the contralateral long-latency reflex was on average 30% (range 5–60%) of that on the ipsilateral side. The latency and duration of the contralateral long-latency responses were well within the normal range of the ipsilateral responses.

Displayed in Fig. 4 are the individual unrectified EMG responses of the contralateral FPL that constitute the average shown in Fig. 3. A 'mirror reflex' response beginning at about 45–50 ms occurs on every stretch of the contralateral FPL. These responses are therefore robust and reminiscent of the inevitable mirror movements accompanying voluntary motor activity in these subjects. Furthermore, the magnitude of the contralateral 'mirror reflex' response was correlated with that of the ipsilateral response (Fig. 5).

No stretch reflex response at monosynaptic latency (25 ms) was ever recorded in the contralateral FPL muscle in these subjects, as can be seen for example in Fig. 3. These long-latency 'mirror reflex' responses are not generalized responses which occur in a variety of hand and finger muscles. If the electrodes are moved away from

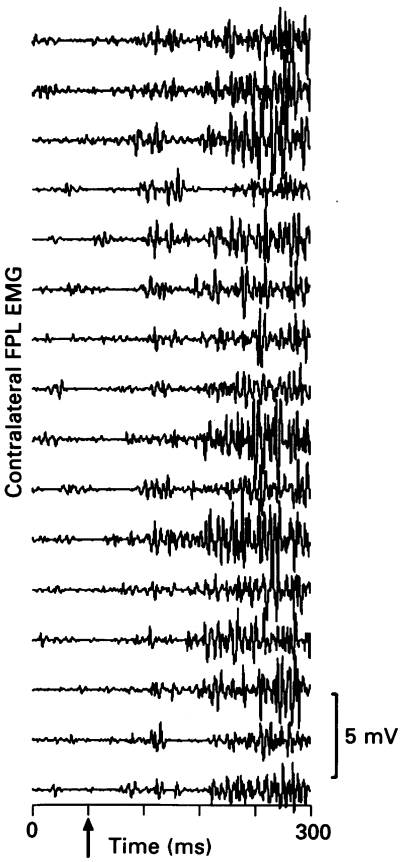

Fig. 4. In this figure the individual EMG responses (unrectified) that constitute the average contralateral EMG activity shown in Fig. 3 are displayed sequentially from top to bottom for mirror movement subject R.M. The arrow indicates the onset of stretch. A clear 'mirror' long-latency response, in the interval between 40 and 80 ms, occurs on every trial as it does in the ipsilateral FPL.

the optimal site for recording FPL EMG activity and thus closer to other finger flexor and wrist flexor muscles no response of any kind was observed. These responses thus appear to be genuine 'mirror' long-latency reflex responses.

Ancillary observations

H reflexes, elicited by stimulation of the median nerve in the cubital fossa, were recorded from wrist and finger flexor muscles of the mirror movement patients. Clear responses were obtained on the stimulated side but never on the contralateral side. Cutaneous reflexes, elicited by electrical stimulation of the index finger (Jenner &

Stephens, 1982), were evoked in the first dorsal interosseus (FDI) on the stimulated side but never on the contralateral side. Lastly, none of the present mirror movement subjects have sensations that are referred to the side opposite to the one stimulated. The primary somatosensory evoked potential of these subjects was

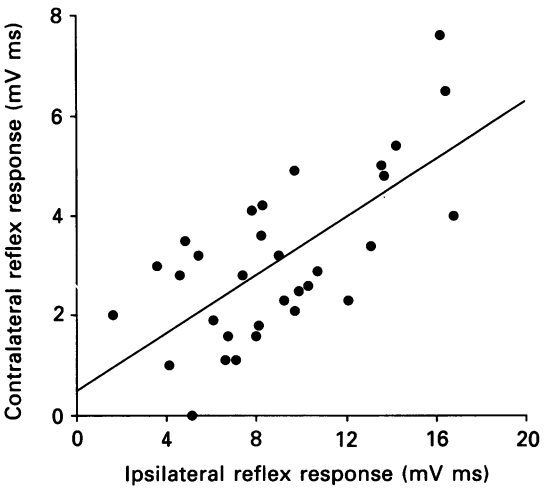

Fig. 5. The magnitude of the FPL long-latency 'mirror reflex' response is correlated with that of the homologous response on the stretched side. The subject (R.M.) was asked to exert a tonic effort of 20% of maximum on the left (stretched) side and 10% on the right (unstretched) side. The EMG was numerically integrated between 45 and 95 ms following the onset of stretch. The correlation coefficient of the least mean squares fitted line is $r = 0{\cdot}69$ and is significant at $P < 0{\cdot}0001$. The slope is 0·29.

recorded only over the sensory cortex contralateral to the stimulated side (see also, Cohen, Dubinsky, Hallett & Jabbari, 1988).

DISCUSSION

We have made two salient observations in the present experiments. Firstly, we demonstrated that in our mirror movement subjects positioning the coil so as to optimally stimulate the motor cortex of one hemisphere activates homologous motoneurone pools on each side of the body nearly simultaneously. Secondly, in these subjects, in marked contrast to normal subjects, long-latency stretch reflex responses can be recorded in the contralateral FPL muscle when only the ipsilateral FPL is stretched. We conclude that in these subjects the corticospinal tract projects bilaterally to homologous motoneurone pools on each side of the body. Since we found no evidence in these subjects of other neuroanatomical pathways abnormally directed across the mid-line, we conclude that the long-latency 'mirror reflex' response is most likely a transcortical reflex (Capaday *et al.* 1989; Matthews *et al.* 1990). It follows that the long-latency reflex response on the stretched side is also, at least in part, produced by the motor cortex. These conclusions and the related observations are discussed below.

Neuroanatomical basis of mirror movements

In a previous study from this laboratory it was found that during rapid voluntary thumb abductions of mirror movement subjects the timing, pattern, and amount of EMG activity on the mirror side were highly correlated with those on the voluntary side (Forget *et al.* 1986). It was suggested that in these subjects the same movement command from the motor cortex of one hemisphere reached homologous motoneurone pools on each side of the body nearly simultaneously. The present observation that transcranial stimulation of the hand area of the motor cortex (Fig. 1*B*) also leads to evoked motor responses at similar latencies on both sides is strong support for this idea. These two sets of observations virtually exclude the possibility that a slower (e.g. transcallosal, see Cracco, Amassian, Maccabee & Cracco, 1989) or more circuitous pathway (e.g. a subcortical relay) interposed between the motor cortex and the motoneurone pool is involved in producing the mirror movements. It is, therefore, almost certain that a bilateral projection of the corticospinal tract to homologous motoneurone pools on each side of the body is the neuroanatomical basis of the mirror movements in the present subjects (see also Cohen *et al.* 1988; Farmer, Ingram & Stephens, 1990).

This appears to be the only neurological abnormality of these subjects. We found no evidence of other neuroanatomical pathways abnormally directed across the midline. The somatic sensations and somatosensory evoked potentials of these subjects are quite normal. Short-latency stretch reflex responses and H reflexes occur unilaterally in these subjects, as do cutaneous reflex (E1, E2) responses. The latter observation seems contrary to the recent finding of Farmer *et al.* (1990) that a long-latency cutaneous reflex response (E2) occurs in the contralateral FDI of one Klippel–Feil syndrome patient. One important implication of this difference is that cutaneous afferents did not contribute to the long-latency 'mirror reflex' response in the present subjects. Therefore, the present 'mirror reflex' responses may be entirely due to muscle afferent input. The discrepancy between the finding of Farmer *et al.* (1990) and the present one may be attributed to differences in the aetiology of the Klippel–Feil syndrome and that of the present mirror movement subjects. Our mirror movement subjects are not Klippel–Feil syndrome patients and have no evidence of failure of closure of the neural tube.

Site of origin of the long-latency stretch reflex

The latency of the 'mirror reflex' response in the contralateral FPL ($\sim$ 45 ms) was similar to that of the long-latency response in the stretched FPL muscle. Thus, as with the mirror movements resulting from voluntary activity and the evoked motor responses, the long-latency 'mirror reflex' is closely timed to the homologous response on the ipsilateral side. The long-latency 'mirror reflex' response is also robust, occurring on every stretch of the homologous contralateral muscle (Fig. 4), reminiscent of the mirror movements. It is specifically directed to the FPL muscle of the contralateral side and is not a diffuse response which can be recorded from any contralateral finger flexor (see Methods and Results). Finally, it is correlated in amplitude with the long-latency response on the stretched side (Fig. 5). Clearly, the long-latency 'mirror reflex' response resembles in all main respects the mirror

movements. Furthermore, as already discussed, in these subjects no other neuroanatomical pathways appear to be abnormally directed across the mid-line. We conclude, therefore, that the 'mirror reflex' response is generated by the same pathway which produces mirror movements; the motor cortex via the corticospinal tract. It follows that the long-latency reflex response in the stretched muscle is also produced, at least in part, by the motor cortex as originally proposed by Phillips (1969).

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