Journal of Physiology (1990), **426**, *pp.* 369–380
With 6 figures
Printed in Great Britain

CUTANEOUS EFFECTS ON PRESYNAPTIC INHIBITION OF FLEXOR Ia AFFERENTS IN THE HUMAN FOREARM

BY K. NAKASHIMA*, J. C. ROTHWELL, B. L. DAY, P. D. THOMPSON AND C. D. MARSDEN

From the MRC Human Movement and Balance Unit, Institute of Neurology, Queen Square, London WC1N 3BG

(*Received 23 October 1989*)

SUMMARY

1. In six subjects, H reflexes obtained in the flexor muscles in the forearm were inhibited by single motor threshold shocks to the radial nerve in the spiral groove. The first two phases of the inhibitory time course were studied: with intervals between the radial and median nerve shocks of −1 to +1 ms, and +5 to +30 ms. These two phases are thought to be due respectively to disynaptic inhibition between radial Ia afferents and flexor α-motoneurones, and to presynaptic inhibition of flexor Ia afferents.

2. Single or short trains (10 ms, 400 Hz) of cutaneous stimuli to the dorsal or palmar aspect of the proximal phalanx of the index finger or to the superficial radial nerve at the wrist, reduced the amount of presynaptic inhibition by 10–20%, but had no effect on the earlier disynaptic inhibition. Single stimuli to either side of the index finger or trains of stimuli to the ventral side, had no effect on the size of control H reflexes elicited alone.

3. Effects of cutaneous nerve shocks on presynaptic inhibition could be seen with stimuli as small as 1·5 × perceptual threshold.

4. Anaesthesia of the hand in one subject reversibly increased the amount of presynaptic inhibition and decreased the amount of disynaptic inhibition.

5. We conclude that, as in the cat, cutaneous input can modulate transmission in presynaptic inhibitory pathways in man.

INTRODUCTION

A single motor threshold conditioning stimulus to the radial nerve in the spiral groove can inhibit test H reflexes elicited in flexor muscles of the forearm following stimulation of the median nerve in the cubital fossa (Day, Marsden, Obeso & Rothwell, 1984). The time course of this effect consists of three phases. The first phase (with conditioning–test intervals of −1 to +1 ms) is probably caused by disynaptic Ia inhibition of flexor motoneurones by impulses in extensor muscle afferent fibres. The second phase (with peak inhibition at conditioning–test intervals of 10–30 ms) is thought to be caused by presynaptic inhibition of the flexor Ia afferent fibres (Berardelli, Day, Marsden & Rothwell, 1987). Mechanisms responsible

* Present address: Division of Neurology, Institute of Neurological Sciences, Tottori University School of Medicine, Yonago, Japan.

MS 8033

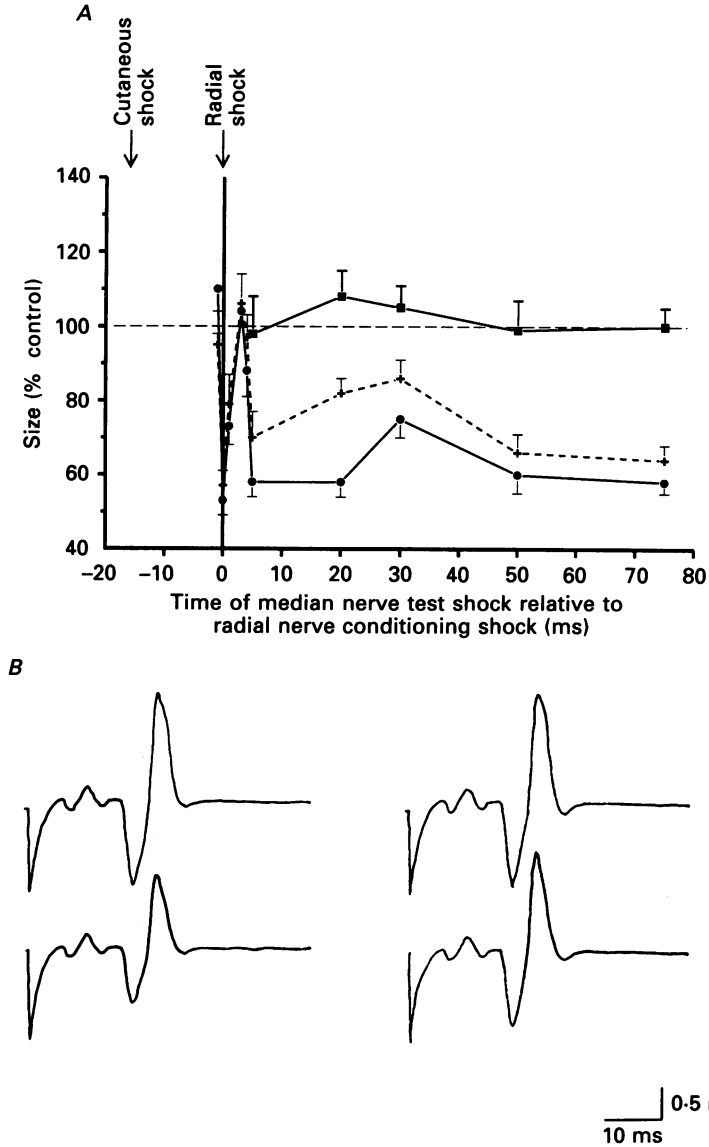

Fig. 1. Cutaneous effects on reciprocal inhibition in a single subject. *A*, time course of effects on the size of H reflexes elicited in forearm flexor muscles. ●, time course of reciprocal inhibition evoked by a single motor threshold conditioning stimulation of the radial nerve in the spiral groove given at $t = 0$ ms. The first (-1 to $+3$ ms) and second phases (maximal at 5–20 ms) of inhibition are clearly visible. +, time course of reciprocal inhibition of forearm flexor H reflexes by radial nerve shock, evaluated following a single cutaneous stimulation of the superficial radial nerve at the wrist given at $t-17$ ms. The depth of the first phase of inhibition is unaffected by the cutaneous shock, whereas there is a reduction in the amount of inhibition during the second phase. ■, time course of effect of a single cutaneous stimulation given to the superficial radial nerve at the wrist on the size of H reflexes given alone. The sizes of H reflexes conditioned by a radial nerve (spiral groove) or cutaneous shock given alone are expressed relative to the size of control H reflexes elicited in the absence of any conditioning stimulus. The size of H reflex conditioned by radial (spiral groove) plus cutaneous stimuli is expressed relative to the size of H reflexes conditioned by cutaneous stimuli given alone. Each point is the

for the third phase of inhibition, which can last for up to 500 ms or longer, have yet to be investigated.

Since animal experiments have shown that presynaptic inhibition can be modulated by cutaneous input (Lund, Lundberg & Vyklicky, 1965; Rudomin, Jimenez, Solodkin & Duenas, 1983; Rudomin, Solodkin & Jimenez, 1986), we sought to test whether cutaneous inputs could affect the Ia presynaptic inhibition described in man. A preliminary report of these findings has been presented to the Physiological Society (Day, Marsden, Nakashima & Rothwell, 1987).

METHODS

Experiments were performed on six normal consenting subjects (aged 28–36 years), including three of the authors, with the approval of the local ethical committee. Reciprocal inhibition from extensor afferents onto forearm flexor muscles was investigated as described in previous papers (Day *et al.* 1984; Berardelli *et al.* 1987). Briefly, subjects were seated comfortably in a reclining chair, with their forearm relaxed whilst H reflexes (approximately half-maximal) were elicited in the flexor muscles of the forearm by giving single electric stimuli to the median nerve in the cubital fossa every 5–6 s. At various times before or after the median nerve shock, a motor threshold conditioning stimulus was given to the radial nerve in the spiral groove in order to produce reciprocal inhibition of the flexor H reflex.

Cutaneous effects were examined by giving a single electrical stimulus, or a train of stimuli (400 Hz for 10 ms), to the superficial (cutaneous) branch of the radial nerve at the wrist, or by applying stimuli through 9 mm diameter electrodes on the dorsal or palmar side of the index finger. Stimuli were adjusted to be about twice perceptual threshold. The timing of the cutaneous stimulus varied in different experiments. For example, in initial experiments, the cutaneous stimulus was given a constant 17 ms before the radial nerve shock, whilst the interval between radial and median shocks was changed. In other experiments, the interval between radial and median shocks was fixed (at 0 ms if the first phase of reciprocal inhibition was being studied; at 20 ms if the presynaptic phase was being studied), and the timing of the cutaneous shock varied. In all experiments, conditions were randomized between (i) median shock alone, (ii) median plus radial, (iii) median plus cutaneous, and (iv) median plus radial plus cutaneous. At each time interval, 16–24 H reflexes were elicited under the four conditions. The size of EMG responses was measured peak-to-peak for each single trial. In order to collate results from different individuals, the size of the conditioned H reflexes in (ii) and (iii) above was expressed relative to the control H reflex in (i). H reflexes in (iv) were expressed relative to the size of H reflexes conditioned by a cutaneous shock given alone in (iii).

In order to study tonic cutaneous effects on presynaptic inhibition, the hand of one of the authors (K.N.) was anaesthetized using intravenous regional anaesthesia (Wallace, Guardini & Ellis, 1982). A tourniquet cuff around the forearm was inflated to 50 mmHg while a butterfly needle was inserted into the vein on the dorsum of the hand. The cuff was deflated for a few minutes, then reinflated to about 200 mmHg and 20 ml 1 % lignocaine injected slowly through the needle. Fifteen to twenty minutes later the hand was almost completely anaesthetized. Studies of reciprocal inhibition were preformed before, during and after recovery from the anaesthetic.

RESULTS

Figure 1 illustrates the effect of a single shock to the superficial radial nerve at the wrist on the time course of reciprocal inhibition in a representative subject. The time course of reciprocal inhibition was studied over conditioning–test intervals of −1 to

---

mean ± 1 S.E.M. from twenty-four trials at each interval. *B*, an example of the raw data which contributes to the graph in *A*. Top left, unconditioned H reflex; bottom left, H reflex conditioned by a radial nerve (spiral groove) shock given 20 ms earlier; top right, H reflex conditioned by a cutaneous shock given 37 ms earlier; bottom right, H reflex conditioned by combined cutaneous and radial nerve (spiral groove) stimuli.

75 ms (●). The first (disynaptic) phase is visible as a sharp peak at around 0 ms; the second (presynaptic) phase lasts from 5 to 30 ms; the start of the third phase of inhibition occurs at 50 ms. In this subject, the cutaneous shock was given on random trials at a constant 17 ms before the radial nerve shock in the spiral groove. Although

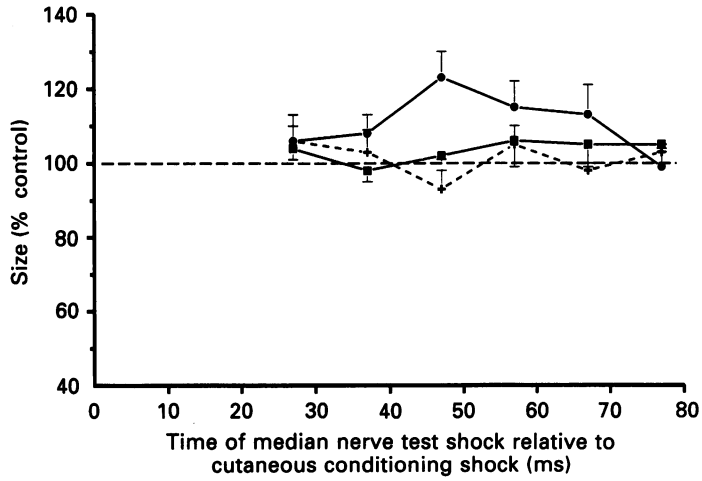

Fig. 2. Cutaneous facilitation of the forearm flexor H reflex in five subjects. Single cutaneous stimuli at twice perceptual threshold were given to three different sites at $t = 0$ ms: the superficial radial nerve at the wrist (●), the dorsal side of the index finger (+), and the ventral surface of the index finger (■). Each point is the mean ± 1 S.E.M. As in the subject of Fig. 1, single stimuli to the superficial radial nerve at the wrist produced facilitation of flexor H reflexes given 47, 57 or 67 ms afterwards. Single shocks to the dorsal or ventral side of the index finger had no effect.

the first phase of reciprocal inhibition was unchanged, the second and third phases were reduced by the cutaneous input (+). The largest effect occurred when the interval between the radial and median nerve shocks was 20 ms.

Cutaneous stimulation alone had only a small effect on the size of control flexor H reflexes. The third, almost horizontal, line in Fig. 1 shows its time course (■). In this subject cutaneous stimulation of the superficial radial nerve caused slight facilitation of the H reflex alone at 20 and 30 ms which may have contributed to the reduction in the amount of the second phase of reciprocal inhibition at these times. However, at 5 ms, the H reflex itself was unaffected by cutaneous input whilst presynaptic inhibition was significantly reduced.

The data from the subject in Fig. 1 were typical of all subjects tested. Details of the group data are given below.

Cutaneous effects on control H reflexes

In preliminary experiments we investigated the time course of cutaneous effects on control flexor H reflexes in five subjects. The results are shown in Fig. 2. Three types of cutaneous stimulation were used: stimulation of the superficial radial nerve at the wrist (●), stimulation of the dorsum of the index finger (+), and stimulation of the

ventral side of the index finger (■). Stimuli were single shocks at twice sensory threshold. As in the subject of Fig. 1, stimulation of the superficial radial nerve at the wrist produced a small facilitation of the flexor H reflex when given 47, 57 or 67 ms before the median nerve shocks (paired t test with control values: $P < 0{\cdot}05$). Stimuli to either the dorsal or ventral side of the finger produced no consistent effects on the flexor H reflex.

In some subjects, trains of stimuli (400 Hz for 10 ms) were given at the same sites (data not shown). The effects were only marginally larger than those seen after a single shock. Trains to the superficial radial nerve at the wrist produced an average 10–26% facilitation when the median nerve shock was given between 37 and 77 ms after the cutaneous stimulus; trains to the dorsum of the index finger evoked an average 10–15% facilitation at 57 and 77 ms; trains to the ventral side of the index finger produced no effect on the flexor H reflex.

Time course of cutaneous effects on the presynaptic phase of reciprocal inhibition

We next tested the time course of cutaneous effects on the second (presynaptic) phase of reciprocal inhibition evoked by a radial nerve (spiral groove) shock. The average results from five subjects are shown in Fig. 3*A*. The cutaneous stimulus was a single shock given to the dorsum of the index finger and in these experiments, the interval between the radial nerve conditioning shock and the median nerve shock was fixed at 20 ms. As shown in the preliminary experiments, cutaneous stimulation to the dorsum of the index finger alone had no consistent effect on the size of control H reflexes at any of the timings used in the study (●). Despite this, it still was capable of reducing the amount of presynaptic inhibition evoked by a radial nerve shock (+). Significant effects (paired t test with control values: $P < 0{\cdot}05$) on presynaptic inhibition were seen when the cutaneous shock was given 17–47 ms before the radial nerve shock. There was no effect at other intervals. A detailed study of the onset latency of these effects was carried out in two subjects, one of whom is illustrated in the data of Fig. 3*B*. The interval between cutaneous and radial shocks was varied in 1 ms intervals between 7 and 17 ms. A consistent decrease in presynaptic inhibition was seen with intervals > 13 ms in both subjects.

The effectiveness of the site of stimulation on presynaptic inhibition was investigated in five subjects. As above, the median–radial interval was fixed at 20 ms to coincide with the maximum period of presynaptic inhibition. Cutaneous stimuli were given either to the superficial radial nerve at the wrist, to the dorsum of the index finger, or to the ventral side of the index finger, at six different intervals: 7, 17, 27, 37, 47 or 57 ms before the radial nerve (spiral groove) conditioning shock. As in Fig. 3*A*, maximum effects on presynaptic inhibition occurred when the cutaneous shock was given 17, 27, 37 and 47 ms before the radial nerve shock. Figure 4 compares the average decrease in presynaptic inhibition over these optimum intervals in five subjects when single or trains of cutaneous shocks were given at different sites.

Stimuli to the dorsal or ventral surface of the index finger decreased the amount of presynaptic inhibition by an average of 10–15%. The effect was the same whether the dorsal or ventral side of the finger was stimulated, or whether trains or single

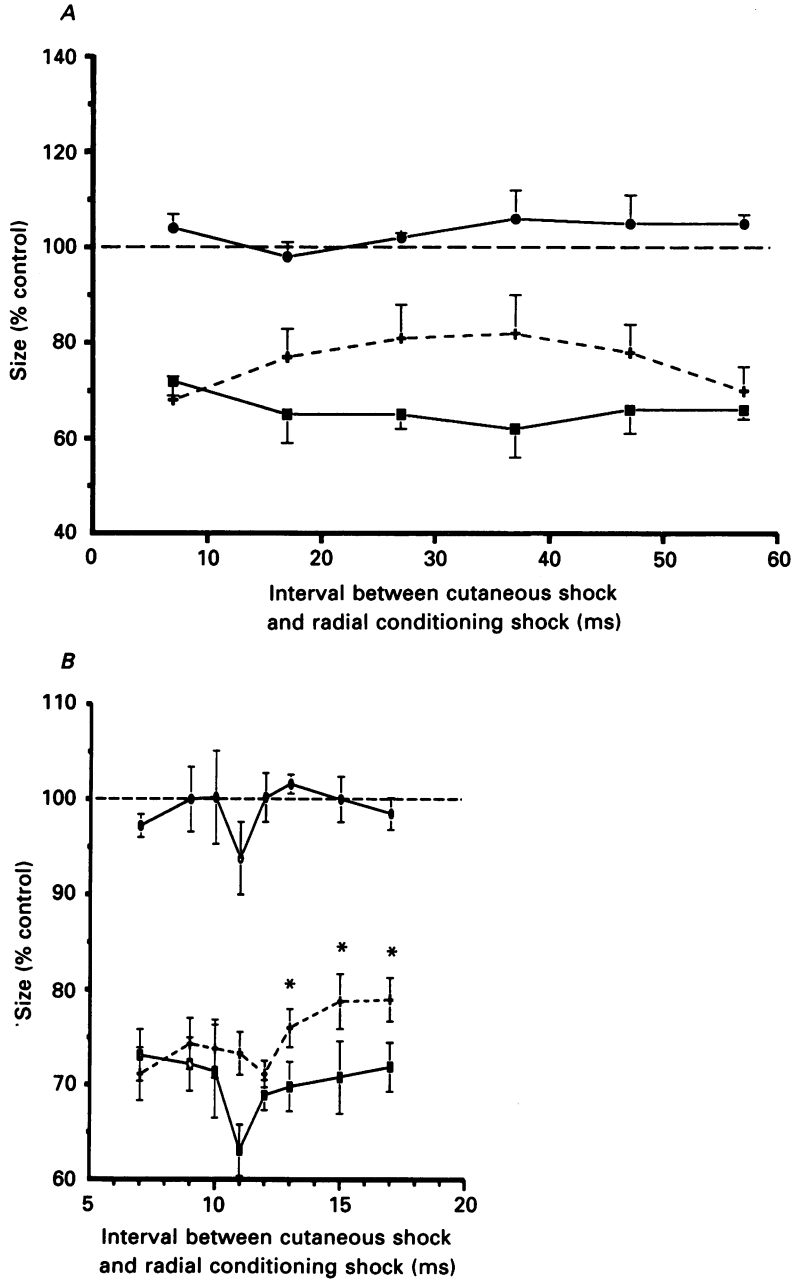

Fig. 3. *A*, mean (±1 S.E.M.) time course of reduction in presynaptic inhibition in five subjects produced by a single cutaneous stimulus given to the dorsum of the index finger. Presynaptic inhibition was produced by using a constant interval of 20 ms between a conditioning shock to the radial nerve in the spiral groove and a test shock to the median nerve in the cubital fossa. Cutaneous effects on this inhibition were then investigated by varying the timing of a cutaneous stimulus given prior to the radial nerve shock. ■ shows how the level of control reciprocal inhibition remained constant throughout the experiment at around 60–70 %. ● shows virtual absence of any effect of cutaneous input

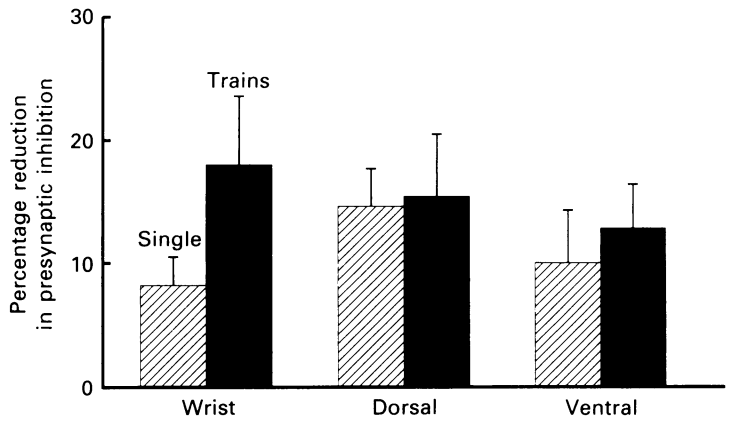

Fig. 4. Mean (±1 S.E.M.) data from five subjects illustrating how cutaneous stimulation at different sites reduced the amount of presynaptic inhibition of flexor H reflexes. Presynaptic inhibition was evaluated by giving a motor threshold conditioning shock to the radial nerve in the spiral groove 20 ms prior to the H reflex test shock in the cubital fossa. Single or short trains (400 Hz for 10 ms) of shocks were given to cutaneous nerves, either to the superficial radial nerve, at the wrist, or the dorsal or ventral side of the index finger 17, 27, 37 or 47 ms prior to the radial nerve (spiral groove) shock. The bars show the average reduction in control levels of presynaptic inhibition produced by such stimuli over these intervals. The only significant difference was between single and trains of shocks given at the wrist ($P < 0{\cdot}05$). The site of stimulation had no significant effect on the reduction of presynaptic inhibition produced by single or trains of cutaneous shocks.

shocks were given (*t* test on mean data: $P > 0{\cdot}05$). Trains of shocks at the wrist produced the most effective decrease in presynaptic inhibition, which was significantly greater than that observed when single shocks were given at the same site ($P < 0{\cdot}05$).

Cutaneous effects on disynaptic Ia reciprocal inhibition

As shown in Fig. 1 and as found previously by Cavallari, Fournier, Katz, Malmgren, Pierrot-Deseilligny & Shindo (1985), cutaneous stimulation had no effect on the amount of disynaptic Ia reciprocal inhibition. This was confirmed in the present experiment in five subjects. Trains of stimuli were applied to the dorsum of the index finger 27 ms prior to the conditioning radial nerve (spiral groove) shock. An analysis of variance for conditioning–test intervals between radial and median nerve shocks of -1, $-0{\cdot}5$, 0, $+0{\cdot}5$ and $+1{\cdot}0$ ms ($f = 0{\cdot}16$; d.f. = 1, 4; $P > 0{\cdot}05$) showed no

---

on H reflexes given alone. + shows the effect of cutaneous stimulation on the level of presynaptic inhibition. When the radial nerve shock was given 17–47 ms after the cutaneous shock the amount of inhibition was reduced. *B*, detailed time course of the onset of cutaneous effects on presynaptic inhibition in one subject. Intervals between cutaneous and radial shocks were studied every millisecond between 7 and 17 ms. Same symbols as in Fig. 3*A*. Each point is the mean of ten measurements ±1 S.E.M.. Asterisks indicate a significant ($P < 0{\cdot}05$) decrease of presynaptic inhibition following the cutaneous shock at 13, 15 and 17 ms. At 11 ms, the control level of presynaptic inhibition increased by 10%. Since this increase was not sustained we regard this point as being aberrant.

difference in the intensity of disynaptic Ia reciprocal inhibition with and without the cutaneous stimulus.

Effect of cutaneous stimulus intensity on presynaptic inhibition

The full range of stimulus intensities was explored only in one subject, although similar effects were observed with a smaller range of stimulus intensities in all the

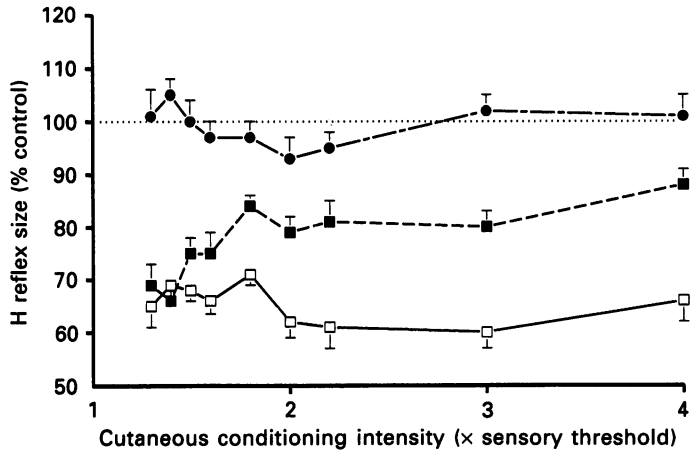

Fig. 5. The effect of stimulation intensity on reduction in the amount of presynaptic inhibition (evaluated at a constant median–radial nerve interval of 20 ms) in a single individual. The baseline level of presynaptic inhibition throughout the experiment remained approximately constant and is shown by □. The amount of presynaptic inhibition evaluated following a cutaneous stimulation is shown by ■, and the effect of cutaneous stimulation on size of H reflexes given alone is shown by ●. In all cases the cutaneous shock was given at a constant 17 ms prior to the radial nerve shock in the spiral groove. The depth of presynaptic inhibition was reduced by cutaneous stimuli as small as 1·5 × sensory threshold, and the effect appeared to begin to saturate at about twice sensory threshold.

other subjects. In the graph of Fig. 5, presynaptic inhibition was evaluated with an interval of 20 ms between the radial nerve conditioning shock and the median nerve H reflex. The cutaneous shock was a single stimulus given to the superficial branch of the radial nerve at a constant 17 ms before the radial nerve shock in the spiral groove. In this subject single shocks of all intensities to the superficial radial nerve had no consistent effect on the size of control H reflexes alone (●). However, they did reduce the amount of presynaptic inhibition (■). The threshold of this effect was about 1·5 × sensory threshold and became maximal at about twice sensory threshold. This subject also was explored with trains of superficial radial nerve shocks and with shocks given 27 ms before the radial nerve conditioning shock. The threshold for producing an effect on presynaptic inhibition was the same in both cases (i.e. about 1·5 × sensory threshold).

Single and trains of cutaneous stimulation to the dorsal or palmar side of the index finger shared similar results. The threshold for reducing presynaptic inhibition was 1·6 times perceptual threshold for stimuli to the dorsum of the finger and twice perceptual threshold for stimuli on the palmar side.

Effect of anaesthesia of the hand

Since cutaneous stimulation reduced the amount of presynaptic inhibition, we also attempted to test whether the converse, i.e. removal of cutaneous input, would produce the opposite effect. The hand of one of the authors was anaesthetized using

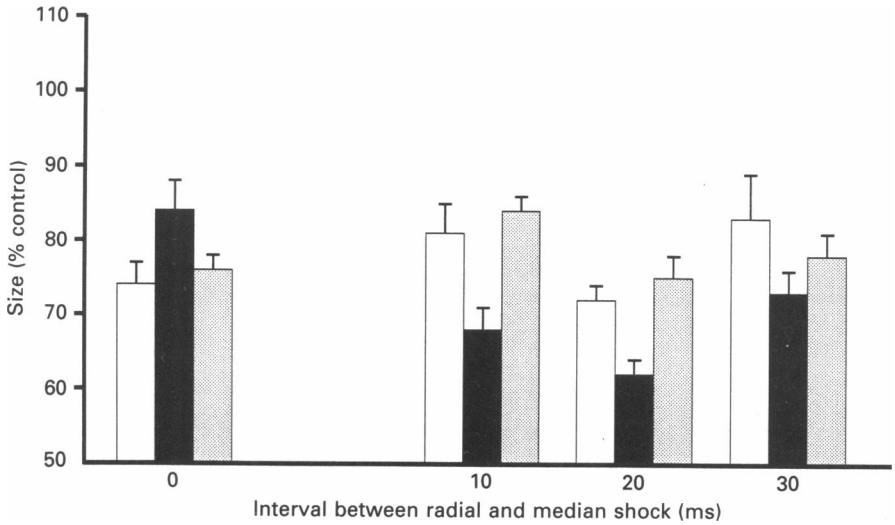

Fig. 6. The effect of anaesthesia of the hand on reciprocal inhibition of forearm flexor H reflexes by radial nerve (spiral groove) shocks given 0 (disynaptic inhibition) and 10, 20 and 30 ms (presynaptic inhibition) prior to the median nerve shock. Data from one subject: twenty-four control and twenty-four reflexes conditioned by a radial nerve shock were elicited at each timing. The three bars plotted at each timing show the amount of reciprocal inhibition before □, during ■ and after ▨ recovery from anaesthesia. Anaesthesia produced a small decrease in the depth of the first phase of inhibition (at 0 ms). In contrast the presynaptic phase of inhibition (at 10, 20 and 30 ms) was increased during anaesthesia. All comparisons of control data with those obtained during anaesthesia were significant at 0, 10 and 20 ms ($P < 0{\cdot}05$).

intravenous lignocaine, and the amount of disynaptic and presynaptic inhibition was tested before, during and after the block. Figure 6 illustrates the results. Presynaptic inhibition was increased during the period of anaesthesia ($P < 0{\cdot}05$ at 10 and 20 ms), whereas disynaptic inhibition was slightly reduced ($P < 0{\cdot}05$ at 0 ms).

DISCUSSION

A single motor threshold stimulus to the radial nerve in the spiral groove reduces the size of H reflexes in forearm flexor muscles produced by median nerve stimulation in the cubital fossa (Day *et al.* 1984). The first phase of inhibition occurs with an interval of -1 to $+1$ ms between the radial and median nerve stimuli. It is short lasting and probably represents activity in the classical disynaptic Ia reciprocal inhibitory pathway. The second phase of inhibition occurs with intervals of 10–30 ms between median and radial nerve shocks. Berardelli *et al.* (1987) have suggested that this is due to presynaptic inhibition of the terminals of flexor muscles Ia afferents.

The present results show that cutaneous stimulation can reduce the depth of the second phase of inhibition without affecting the first. The cutaneous afferents which produce this effect are of low threshold, since it occurs with stimuli of only 1·5–2 × sensory perception. The duration of these effects at 40 ms is long lasting, and probably indicative of activity in a polysynaptic pathway. A similar cutaneous effect has been noted in leg muscles by Iles & Roberts (1987). They reported that a short train of stimuli applied to the sural nerve (six shocks at 300 Hz) reduced presynaptic inhibition of Ia afferents in the leg muscles of three normal subjects.

What mechanisms could account for the cutaneous influence on the second phase of reciprocal inhibition? One possibility is that there is an excitatory effect on flexor motoneurones in the spinal cord, which overrides any on-going inhibition. This could be due to direct excitatory projections of cutaneous afferents onto flexor motoneurones, or by more indirect routes, such as disinhibition of Ib pathways from flexor muscle afferents. However, these possibilities seem unlikely to account for the present results. While shocks to the wrist facilitated H reflexes given alone, single shocks to the index finger had no direct effect on control H reflexes, yet produced a pronounced reduction in the second phase of reciprocal inhibition. Because of this we favour the explanation that cutaneous afferents can influence the inhibitory pathway itself. If the second phase of reciprocal inhibition is caused by presynaptic inhibition, this would involve cutaneous influence on a presynaptic pathway. Such an effect is probable on the basis of known animal data. In the cat, cutaneous afferents project to the spinal interneurones which mediate presynaptic reciprocal inhibition and inhibit the primary afferent depolarization generated by excitation of group I fibres (Lund *et al.* 1965; Rudomin *et al.* 1986). A similar mechanism may therefore account for the present results. In particular the timing of various stimuli used is appropriate. In two subjects the earliest effect occurred when cutaneous stimuli were given more than 13 ms before the radial nerve shock. Allowing 7 ms for the conduction time between the wrist and the spiral groove, cutaneous input must arrive about 6 ms before radial nerve input in order to affect the level of presynaptic inhibition.

Why then can cutaneous input influence presynaptic inhibitory pathways, yet fail to have any direct effect on the excitability of spinal α-motoneurones? Cutaneous inputs from the finger are well known to produce modulation of EMG activity in forearm muscles (Caccia, McComas, Upton & Blogg, 1973), although this is less powerful than that seen in intrinsic hand muscles (Jenner & Stephens, 1982). One possibility is that the effect on presynaptic inhibition is much more powerful than direct excitation of spinal motoneurones. Alternatively, it is known that cutaneous excitation of motoneurones is directed preferentially to large cells. These might not be recruited by the H reflexes used in the present experiments and hence the direct effect may have been missed.

In contrast with the pronounced effects on the second phase of reciprocal inhibition, we failed to find any influence of cutaneous stimuli on the first phase of reciprocal inhibition from -1 to $+1$ ms. This is similar to the results of Cavallari *et al.* (1985), who found that cutaneous input from the dorsum of the finger produced no effect on Ia disynaptic inhibition, but could reduce the degree of Ib excitation which followed the onset of Ia inhibition by about 0·5 ms. In their study Cavallari

et al. (1985) noted that the Ib effect was not present if the palmar side of the finger was stimulated. This contrasts with the present results on presynaptic inhibition, which showed no significant difference between the effect of stimulation on the dorsal or ventral side of the finger. This suggests that the pathways mediating cutaneous effects on presynaptic inhibition are different from those mediating cutaneous effects on Ib facilitation.

Finally, the experiments in which the hand was anaesthetized suggest that there is a tonic cutaneous influence on the level of excitability in presynaptic inhibitory pathways. Removal of this cutaneous input during anaesthesia resulted in a slight reduction of early disynaptic inhibition, but facilitation of the presynaptic inhibitory pathways activated by the radial nerve shock. This may be of relevance to a clinical observation made by Sheehy, Rothwell & Marsden (1987) in a patient with idiopathic torsion dystonia. In patients with dystonia, the presynaptic phase of reciprocal inhibition is reduced or absent, whereas the earlier disynaptic phase of inhibition is unaffected. It has been suggested that this is due to an abnormality in the descending, supraspinal control of interneurones within the presynaptic inhibitory pathway (Nakashima, Rothwell, Day, Thompson, Shannon & Marsden, 1989). Writers' cramp is a form of focal dystonia. Sheehy *et al.* (1987) showed that the writing of a patient with this form of dystonia was improved following intravenous regional anaesthesia of the hand. Anaesthesia may have increased presynaptic inhibition to more normal levels, and compensated for deficits in the descending control of spinal interneurones.

We should like to thank Mr R. Bedlington for building and maintaining much of the specialized equipment used in these experiments. The work was funded by the Medical Research Council.

REFERENCES

BERARDELLI, A., DAY, B. L., MARSDEN, C. D. & ROTHWELL, J. C. (1987). Evidence favouring presynaptic inhibition between antagonist muscle afferents in the human forearm. *Journal of Physiology* **391**, 71–83.

CACCIA, M. R., MCCOMAS, A. J., UPTON, A. R. M. & BLOGG, T. (1973). Cutaneous reflexes in small muscles of the hand. *Journal of Neurology, Neurosurgery and Psychiatry* **36**, 960–977.

CAVALLARI, P., FOURNIER, E., KATZ, R., MALMGREN, K., PIERROT-DESEILLIGNY, E. & SHINDO, M. (1985). Cutaneous facilitation of transmission in Ib reflex pathways in the human upper limb. *Experimental Brain Research* **60**, 197–199.

DAY, B. L., MARSDEN, C. D., NAKASHIMA, K. & ROTHWELL, J. C. (1987). Cutaneous effects on presynaptic inhibition of flexor Ia afferents in the human forearm. *Journal of Physiology* **394**, 160*P*.

DAY, B. L., MARSDEN, C. D., OBESO, J. A. & ROTHWELL, J. C. (1984). Reciprocal inhibition between the muscles of the human forearm. *Journal of Physiology* **349**, 519–534.

ILES, J. F. & ROBERTS, R. C. (1987). Inhibition of monosynaptic reflexes in the human lower limb. *Journal of Physiology* **385**, 69–87.

JENNER, J. R. & STEPHENS, J. A. (1982). Cutaneous reflex responses and their central nervous pathways studied in man. *Journal of Physiology* **333**, 405–419.

LUND, S., LUNDBERG, A. & VYKLICKY, L. (1965). Inhibitory action from the flexor reflex afferents on transmission to Ia afferents. *Acta physiologica scandinavica* **64**, 345–355.

NAKASHIMA, K., ROTHWELL, J. C., DAY, B. L., THOMPSON, P. D., SHANNON, K. & MARSDEN, C. D. (1989). Reciprocal inhibition between forearm muscles in patients with Writer's cramp and other occupational cramps, symptomatic hemidystonia and hemiparesis due to stroke. *Brain* **112**, 681–697.

RUDOMIN, P., JIMENEZ, I., SOLODKIN, M. & DUENAS, S. (1983). Sites of action of segmental and descending control of transmission on pathways mediating PAD of Ia and Ib-afferent fibres in cat spinal cord. *Journal of Neurophysiology* **50**, 743–769.

RUDOMIN, P., SOLODKIN, M. & JIMENEZ, I. (1986). PAD and PAH response patterns of group Ia- and Ib-fibres to cutaneous and descending inputs in the cat spinal cord. *Journal of Neurophysiology* **56**, 987–1005.

SHEEHY, M. P., ROTHWELL, J. C. & MARSDEN, C. D. (1988). Writer's cramp. *Advances in Neurology* **50**, 457–472.

WALLACE, A., GUARDINI, R. & ELLIS, S. J. (1982). Standard intravenous regional analgesia. *British Medical Journal* **285**, 554–556.