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INCREASE IN Ib INHIBITION BY ANTAGONISTIC VOLUNTARY CONTRACTION IN MAN

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SUMMARY

1. Ib inhibition from gastrocnemius medialis (GM) muscle to soleus (Sol) muscle was studied at rest and at the onset of phasic voluntary contraction of antagonistic pretibial muscles in seventeen normal subjects.

2. In twelve out of seventeen subjects there was inhibition of Sol H reflex by GM conditioning stimulation at rest with a latency of 1·5–3·0 ms and a threshold of 0·85–1·00 times the motor threshold (MT). The amount of inhibition at 0·95–1·05 × MT, which was calculated by subtracting the size of the conditioned reflex from that of the unconditioned one, ranged from 0·8 to 5·6% of the maximal M-response or 2·9–18·3% of control H reflex. This inhibition was ascribed to Ib inhibition, taking into account its latency and threshold.

3. On weak pretibial contraction the inhibition either increased in amount or newly appeared in all the subjects. When the strength of voluntary contraction was graded from 1 to 20% of the maximum, the increment in the amount of inhibition decreased or almost disappeared at strengths of more than several per cent. These facts imply that at least some of the Ib interneurones are facilitated to fire by descending commands alone without peripheral Ib impulses. Contralateral ankle dorsiflexion did not modify the inhibition.

4. Soleus muscle H reflex was not modulated at all by cutaneous stimulation instead of GM stimulation at rest, nor was it affected by cutaneous stimulation on ipsilateral antagonistic contraction.

5. It is concluded that activity in the Ib inhibitory pathway is facilitated at the onset of antagonistic voluntary contraction. This suggests that control of the Ib inhibitory pathway may be utilized in ordinary voluntary movement, and is presumably beneficial for smooth execution of movement.

INTRODUCTION

Ib afferents, which originate from Golgi tendon organs, have been reported to inhibit agonist and synergistic motoneurones and at the same time to facilitate antagonistic motoneurones (Lapport & Lloyd, 1952). Knowledge of functional significance of these reflex actions in voluntary movements, however, has been limited by a lack of information regarding the supraspinal and segmental inputs onto

the interposed interneurones, and by a lack of information regarding the behaviour of these reflex pathways during voluntary movements.

With regard to the Ib inhibitory interneurones, they have been demonstrated to have a wide congervence from supraspinal and segmental inputs (Eccles & Lundberg, 1959; Hongo, Jankowska & Lundberg, 1969; Brink, Harrison, Jankowska, McCrea & Skoog, 1983*a*; Brink, Jankowska, McCrea & Skoog, 1983*b*; Harrison & Jankowska, 1985; see also Baldissera, Hultborn & Illert, 1981). The supraspinal motor centres would hence be expected to control the gain and the direction of the action of Ib effects, depending on motor strategies. In this connection Fournier, Katz & Pierrot-Deseilligny (1983) have demonstrated in man that Ib inhibition decreased in amount during voluntary contraction of the homonymous muscle. It is therefore of interest to know how Ib inhibition is modulated by contraction of other muscles, especially of the antagonists. The purpose of the present study has been to investigate this problem and it will be shown that Ib inhibition is facilitated by antagonist voluntary contraction.

METHODS

Seventeen healthy volunteers including the authors aged between 20 and 54 years took part in the study, and gave their informed consent. In four subjects the same experiments were repeated more than three times to test the reproducibility of the results.

A subject was seated in a reclining chair with knee and ankle joints semi-flexed (160–170 deg and 100–110 deg, respectively). The foot was generally fixed to an immobile foot-plate, but in some experiments it was free from any contact with the plate or the straps in order to avoid excitation of cutaneous afferents from the foot, which have been demonstrated to modulate the activity of the Ib pathway both at rest and on contraction (Pierrot-Deseilligny, Bergego, Katz & Morin, 1981*a*; Pierrot-Deseilligny, Bergego & Katz, 1982). The H reflex was elicited in soleus (Sol) muscle by stimulating monopolarly the tibial nerve (TN) in the popliteal fossa with a rectangular pulse of 1·0 ms duration every 3–4 s, and was recorded through a pair of surface electrodes 3–4 cm apart. Its size was measured as an area after full-wave rectification and integration, and was expressed as a percentage of the maximal direct M-response (M_{max}). Preceding each test stimulus, a single conditioning stimulus (duration 1·0 ms) was delivered to gastrocnemius medialis (GM) nerve 6–11 cm distally and 4–5 cm medially from the point of the test stimulus. The intensity of conditioning stimuli was expressed as multiples of the threshold for the direct M-response ($\times$ MT). Since it was sometimes difficult to determine the real threshold for the M-response because of direct stimulation of the gastrocnemius muscle itself, the intensity was taken as the threshold where the response began to grow sharply enough. Conditioning intensities were kept below or equal to 1·0 $\times$ MT, except for the intensity curve (see below) and some experiments concerning grading of voluntary contraction, in order to exclude activities of other reflex pathways such as recurrent inhibition or group II effects. Special care was taken to check current spread from the conditioning stimulus to TN, and the position of the conditioning electrodes was set so as to produce neither H reflex nor M-response in Sol on conditioning stimuli to GM with intensities more than 2·5 $\times$ MT even during a weak tonic voluntary plantar flexion.

Experiments were typically done in the forms of 'time course' where conditioning–test stimulus intervals (C–T intervals) were changed with a fixed conditioning intensity, and of 'intensity curve' where intensities of conditioning stimuli were varied with a fixed C–T interval. C–T intervals in the former case and intensities of conditioning stimuli in the latter were changed systematically and randomly during one session of experiments. Triggering of conditioning and/or test stimulations, setting C–T intervals and intensities of conditioning stimuli, measurement of the size of H reflexes, and on-line analysis of data were all done with a 16-bit microcomputer (NEC PC-9801). Data were also stored on diskettes for later off-line analysis.

The experiments were performed both at rest and at the onset of voluntary contraction of antagonistic pretibial muscles, and the conditioning effects of GM stimulation were compared in these two situations. In some experiments the effect of voluntary contraction of the contralateral pretibial muscles was also studied. Before such experiments a preliminary time course and intensity

curve were studied in all the subjects to determine the appropriate C–T interval and conditioning intensity. Instructions were given to the subject before each session as to whether he should dorsiflex or not, and which ankle he should dorsiflex. Sessions at rest and on contraction were regularly alternated to avoid fluctuation in the activity of the reflex pathway during experiments.

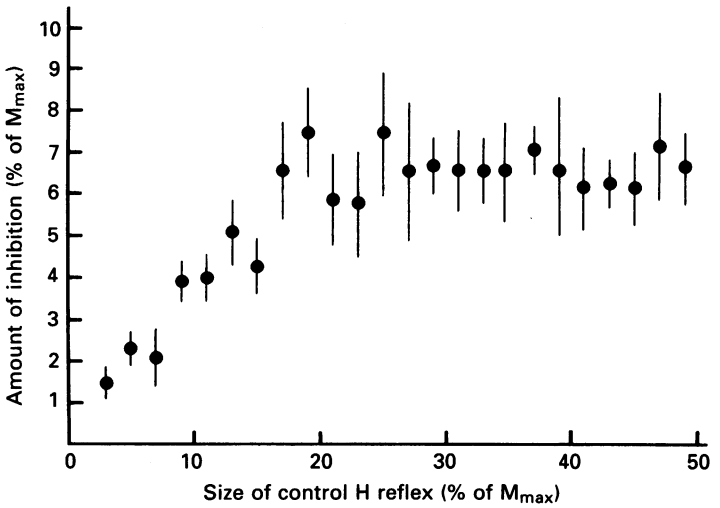

Fig. 1. Relationship between the size of control H reflex and the amount of inhibition by GM conditioning stimulation at rest in one subject. The amount of inhibition was calculated by subtracting the size of the conditioned reflex from that of the unconditioned one. Both the size of control reflex (abscissa) and the amount of inhibition (ordinate) were expressed as a percentage of M_{max}. When the control reflex was small, the amount of inhibition was also small. The latter increased gradaully as the control size was increased by augmenting the strength of the test stimulus, and then remained constant with a control size above 20% of M_{max}. ● and bars are the means ± S.D. Each symbol represents 20–80 measurements.

In the contraction session, subjects were required to perform a brief and phasic, but not ballistic, dorsiflexion for about 0·5 s on hearing a beep, which was delivered by the microcomputer. Contraction was almost isometric. Its strength was graded from 1 to 20% of the maximal voluntary ankle dorsiflexion by monitoring the rectified and integrated electromyogram (EMG) of pretibial muscles. In some subjects the effect of grading the contraction on Ib inhibition was studied with two different intensities of conditioning stimulation, both weak and strong, in order to clarify the mode of modulation of the Ib inhibitory pathway. The microcomputer and hence the stimulator were driven at the very onset of the EMG of pretibial muscles. EMGs of Sol and GM were also monitored on an oscilloscope to check co-contraction.

Recently, in comparing the conditioning effects in two different situations such as at rest and on contraction, the necessity to adjust control H reflexes to the same size in two situations has been strongly emphasized (Crone, Hultborn & Jespersen, 1985; Fournier, Meunier, Pierrot-Deseilligny & Shindo, 1986; Crone, Hultborn, Jespersen & Nielsen, 1987), because the conditioning effect can be modulated not only by the intensity of the conditioning stimulus but also by the size of the test H reflex itself (Kuno, 1959; Crone, Hultborn, Mazières, Morin, Nielsen & Pierrot-Deseilligny, 1990). In fact, on voluntary dorsiflexion control H reflexes were usually smaller than those at rest. In the present study, however, adjustment was not made, and the same intensity of test stimulation was employed both at rest and on contraction. Instead, control experiments were done in order to reveal the relationship between the size of the control reflex and the amount of the conditioning effect of GM stimulation (Fig. 1). The amount of conditioning effect was calculated by subtracting the size of the conditioned reflex from that of the unconditioned one, and was expressed as a percentage of M_{max}, which would correspond to a percentage of the whole Sol motoneurone pool. As the size of the control H reflex increased from its minimum, the amount of inhibition

increased gradually, and then remained constant when the size of the control reflex was above 20% of M_{max}. In this situation, i.e. when both control reflexes are above 20% of M_{max}, we can compare two conditioning effects with each other even if the sizes of the two control reflexes are not identical. In our experiments, therefore, we adjusted the test-stimulus intensity to obtain control H reflexes larger than 20% of M_{max} both at rest and on voluntary dorsiflexion whenever possible. To fulfil this requirement the size of control reflexes at rest was between 20 and 50% of M_{max}, while the maximal H reflex in each subject ranged from 40 to 85% of M_{max}. According to this method, if test reflexes become smaller than 20% of M_{max}, the amount of inhibition should follow the curve shown in Fig. 1, when the activity of Ib reflex pathway is not changed, divergence from this curve thus indicating central and/or segmental modulation of the excitability in the Ib inhibitory pathway. In fact in a few subjects it was impossible to elicit large enough reflexes on contraction. However, the results obtained were qualitatively identical to those with larger reflexes: the inhibition increased, although the control reflexes were decreased in size on antagonist contraction.

Data were analysed statistically by Student's t test and by analysis of variance (ANOVA).

RESULTS

Figure 2 shows a time course of Ib inhibition from one subject. The conditioning intensity of GM nerve stimulation was $1{\cdot}00 \times MT$, at which the H reflex was often observed in GM muscle (but not in Sol) at rest. There was no apparent facilitation, but a small depression in the size of the H reflex on conditioning GM stimulation at rest.

At the onset of phasic antagonistic voluntary contraction, the size of the control reflex was reduced from 26·0 to 20·9% of M_{max}. With GM conditioning added, the reflex was further depressed. The latency of the inhibition was 2·0 ms, and the amount of inhibition increased afterwards, reaching the maximal amount of 2·9% of M_{max} at 3·0 ms. The reflex returned to the level of the unconditioned control size at 7·0 ms. The increase in inhibition between rest and contraction was statistically significant ($P < 0{\cdot}05$, ANOVA). Since the subject felt a tactile or tingling sensation on the skin under the conditioning electrodes, the effect of cutaneous conditioning on the test reflex was studied in the same way as in GM stimulation. Cutaneous stimuli were applied to the skin a little anterior to the GM electrodes, where the intensity was adjusted to give a similar sensation. However, such cutaneous stimulation produced no effect on the test reflex either at rest or on contraction (Fig. 2, open and filled triangles). Such a full time course of GM conditioning effect was studied with conditioning intensities between 0·95 and $1{\cdot}00 \times MT$ in eleven subjects, and similar results were also obtained in the other subjects. The maximal amount of inhibition at rest was between 0·8 and 5·6% of M_{max} with a mean value of 3·3% in eight subjects in whom the inhibition was observed. Expressed in percentages of the control reflex for comparison with previously published data (Pierrot-Deseilligny, Katz & Morin, 1979; Pierrot-Deseilligny, Morin, Bergego & Tankov, 1981*b*), it was between 2·9 and 18·4% with a mean value of 11·5% of the control. In the other three subjects there was no apparent inhibition at rest. At the onset of contraction, inhibition either increased in amount or newly appeared in all the subjects.

Figure 3 shows the intensity curve in the same subject as in Fig. 2. The C–T interval was fixed at 3·5 ms and the stimulus intensity was altered from 0·70 to $1{\cdot}05 \times MT$ systematically. At rest, the threshold for the inhibition was $0{\cdot}90 \times MT$, and the inhibition augmented almost linearly up to $1{\cdot}00 \times MT$, when it reached a plateau. At the onset of phasic voluntary contraction of antagonistic pretibial muscles, the control H reflex itself decreased in size from 29·4 to 24·9% of M_{max}, but the inhibition

by GM stimulation increased in amount from the very beginning of the intensity curve. Furthermore, the threshold for the inhibition was lowered from 0·90 to 0·80 × MT by contraction, and the plateau level of inhibition was observed at 0·95 × MT. Such intensity-curve experiments were carried out in six subjects

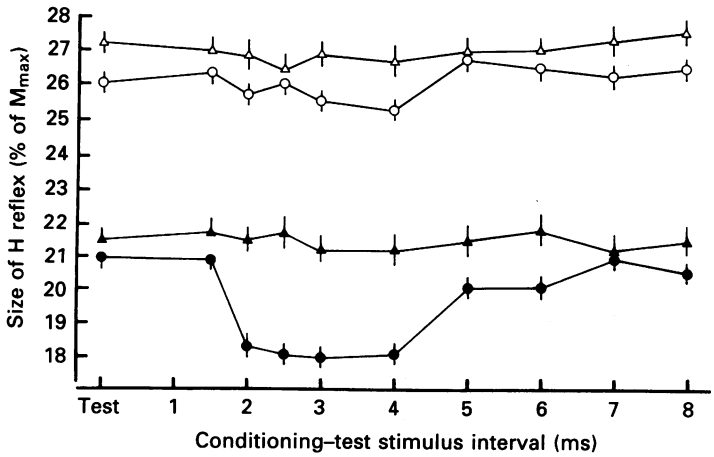

Fig. 2. Time course of the conditioning effect from GM to Sol both at rest (○) and at the onset of pretibial muscle contraction (●) in one subject. The strength of contraction was 2% of the maximal contraction. The size of H reflex (ordinate) was plotted against C–T intervals (abscissa). GM conditioning intensity was 1·00 × MT. At rest, there was no apparent facilitation, but a small depression in the size of the H reflex by GM stimulation (○). On contraction, control reflex decreased from 26 to 20·9% of M_{max} (●), but at the same time the amount of inhibition was increased by GM conditioning stimuli from the very onset of this time course. Cutaneous conditioning, which was applied near GM electrodes and produced a similar cutaneous sensation, did not reveal any effect on test reflex either at rest (△) or on contraction (▲). Each symbol represents mean ± S.E.M. from 60 data.

including the one presented in Fig. 3. The threshold for the inhibition at rest ranged from 0·85 to 1·00 × MT, and the amount of inhibition reached a plateau at 1·00–1·25 × MT. In all six subjects the amount of inhibition increased around the threshold on contraction, the strength of which was between 2 and 5% of the maximum. Plateau levels of inhibition, in other words the maximal amount of inhibition, of more than 1·00–1·25 × MT on contraction were greater in three subjects, almost equal in two, and less in the remaining subject than those at rest. In four of them, including the one whose plateau level on contraction was smaller than at rest, not only did the amount of inhibition increase around the threshold (below 1·00 × MT), but also the threshold itself was lowered when compared with that at rest (Fig. 3). Among all the subjects so examined, either the threshold was lowered on contraction, or the plateau level of inhibition was larger with the threshold unchanged. In the latter case the plateau level was never smaller on contraction. In no subjects was the threshold increased on contraction.

Figure 4 represents the results from all seventeen subjects, comparing quantitatively the amount of inhibition at rest and on antagonistic contraction. C–T intervals between 3·0 ms and 4·0 ms were fixed in each subject, being within 1·0 ms after the latency in the preliminary time-course. The conditioning intensity was fixed

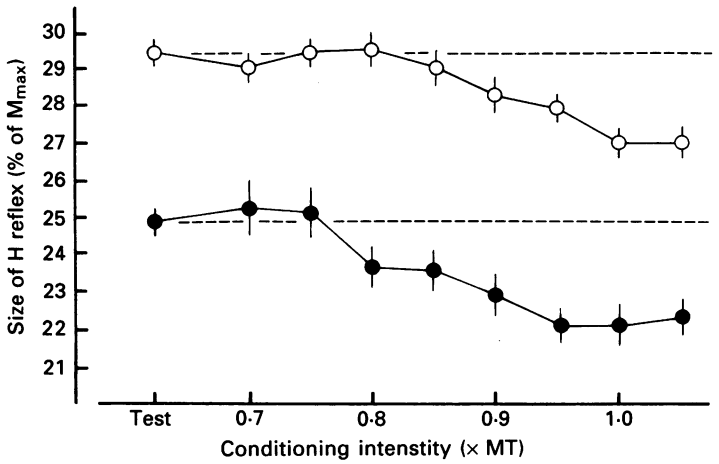

Fig. 3. Intensity curve of GM conditioning stimulation both at rest (○) and at the onset of pretibial muscle contraction (●) in the same subject as in Fig. 2. The ordinate represents the size of the reflex. Intensities, which are shown on the abscissa, were randomly distributed in one experimental session, and sessions at rest and with contraction were regularly alternated. At rest, H reflex was inhibited by GM conditioning stimuli, and the threshold was 0·90 × MT. Inhibition increased in amount as the conditioning intensity was strengthened up to around 1·00 × MT, above which it remained constant. On contraction, the reflex began to be inhibited from 0·80 × MT, and the inhibition was augmented as the conditioning intensity was strengthened, reaching a plateau level at 0·95 × MT. At every intensity except above 1·00 × MT, the amount of inhibition was larger on contraction than at rest. Symbols and bars are means ± S.E.M. Each symbol represents 60 measurements.

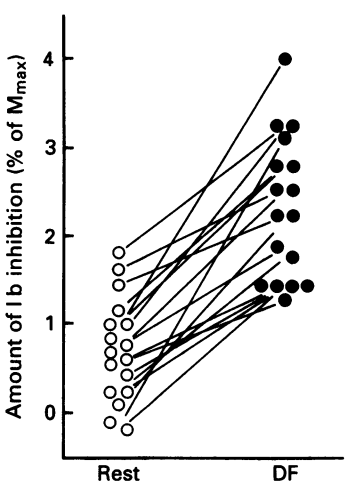

Fig. 4. Amounts of inhibition at rest and at the onset of phasic dorsiflexion (DF) in all the subjects. C–T intervals and conditioning intensities were fixed in each subject, being within 1·0 ms after the latency of the inhibition (3·5–4·0 ms) and 0·95–1·00 × MT, respectively. The strength of contraction was 2–5% of the maximum. The mean value was 0·71 and 2·29% at rest and on contraction respectively, the difference being significant ($P < 0·001$).

in each subject between 0·95 and 1·00 × MT so as to obtain only a small inhibition at rest. The strength of voluntary contraction was between 1 and 5% of the maximal contraction. A considerable increase in inhibition was obtained at the onset of antagonist contraction. The mean value and the standard deviation (S.D.) of Ib

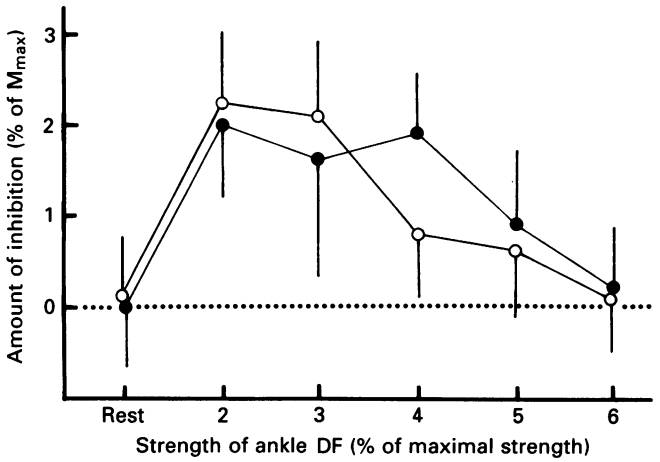

Fig. 5. Effect of strengths of voluntary contraction of pretibial muscles on Ib inhibition in one subject. The C–T interval was fixed at 3·5 ms, and the conditioning intensity at 1·05 × MT (○) and 0·8 × MT (●). Data with both intensities were obtained within the same experimental session. The amount of inhibition (ordinate) was plotted against strengths of voluntary dorsiflexion (abscissa). With 1·05 × MT the inhibition increased at 2 and 3% of the maximal contraction, and began to be suppressed at 4%, decreasing progressively at further strengths of contraction (5 and 6%). With 0·8 × MT the increase in inhibition was observed as the contraction strength increased up to 4% of the maximum, and began to be suppressed at 5%. When the amount of contraction was compared between 0·8 and 1·05 × MT at 4% of the maximal contraction, it was smaller with 1·05 × MT ($P < 0·001$).

inhibition were 0·71 and 0·58% of M_{max} at rest, and 2·29 and 0·81% at the onset of pretibial muscle contraction respectively, and their difference was statistically significant overall ($P < 0·001$, t test). Antagonist contraction sometimes reduced the size of the control H reflex below 20% of M_{max}, but even in these cases the amount of Ib inhibition increased.

In order to evaluate the relationship between the strength of voluntary contraction and the degree of Ib inhibition, the strength of voluntary contraction was graded into several steps from 1 to 20% of the maximum in eight subjects. The C–T interval was set at 3·5 to 4·0 ms, and experiments were done with two conditioning intensities of 0·75–0·85 × MT and 0·95–1·05 × MT. Data with 'weak' and 'strong' intensities were obtained simultaneously during the same experimental session, the contraction being exactly the same for both intensities. Figure 5 shows the result of such an experiment from one of the subjects. With a conditioning intensity of 1·05 × MT, the inhibition increased in amount at 2 and 3% of maximal contraction, began to be suppressed at 4%, and continued to decrease at further strengths of contraction (5 and 6%). On the other hand, at 0·8 × MT the inhibition increased as the contraction

strength increased up to 4% of the maximum, and the increase began to be suppressed at 5%. When the amount of inhibition was compared between that at 0·8 × MT and that at 1·05 × MT at 4% of maximal contraction, it was found to be smaller at 1·05 × MT ($P < 0·001$). In all subjects with a conditioning intensity around

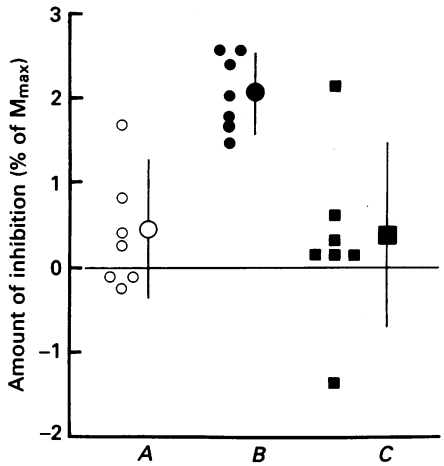

Fig. 6. Comparison between the effects of ipsilateral and contralateral pretibial contraction on the amount of Ib inhibition. The C–T interval, the intensity of GM conditioning and the strength of contraction were fixed at 3·0–4·0 ms, 0·95–1·00 × MT and 2–5% of the maximum, respectively. Ib inhibition increased significantly in amount on ipsilateral contraction (*B*; $P < 0·001$), while on contralateral contraction (*C*) it did not differ from that at rest (*A*). Small symbols indicate amounts of inhibition in individual subjects, consisting of 40–80 pairs of data. A large symbol and bar are the mean ± S.D. of a group.

1·0 × MT the inhibition was augmented on very weak contraction (usually 1–4% of the maximum), whereas it decreased or disappeared on stronger contraction (usually above 4%). When the conditioning intensity was decreased, however, the increase in inhibition began to be suppressed on a slightly stronger contraction. Furthermore, it is worth noting that the amount of inhibition at more than several percent of contraction was less than that at rest in three subjects.

Since the excitability of the Ib inhibitory pathway was modulated on very weak contraction, modification of the excitability of Ib interneurones might possibly have occurred in a non-specific way on voluntary contraction of any muscles including the antagonists. To examine this hypothesis, changes in the conditioning effect were compared at the onset of voluntary contraction of contralateral and ipsilateral pretibial muscles. The C–T interval and the conditioning intensity were fixed at 3·5 or 4·0 ms and 0·95–1·00 × MT respectively, which could easily manifest augmentation of Ib inhibition with contraction of the ipsilateral muscle. As shown in Fig. 6, in all seven subjects so examined there was a specific increase in Ib inhibition only with ipsilateral contraction ($P < 0·001$), and no significant change with contralateral.

DISCUSSION

Ib inhibition on antagonist contraction

Stimulating the GM nerve near motor threshold resulted in an inhibition of the soleus H reflex which occurred at a short central latency (about 1 ms) and had a short duration. Evidence has been presented that this inhibition is most probably Ib in origin (Pierrot-Deseilligny *et al.* 1979, 1981*b*). Higher threshold for the inhibition than those by the previous authors might be mainly due to methodological differences (Fournier, Katz & Pierrot-Deseilligny, 1984).

On antagonistic pretibial contraction, the amount of Ib inhibition increased as shown in Figs 2, 3 and 4. Although in a few subjects control H reflex decreased in size below 20% of M_{max} and it was impossible to keep it larger on contraction, it must be an underestimation of the change in the amount of inhibition, because without any modulation of the pathway it should have decreased when the control reflex dipped below 20% of M_{max} on contraction (Fig. 1; see Methods).

A remarkable finding was that Ib inhibition increased in amount when antagonist contraction was very weak, but that this increase began to be suppressed on gradual increase in the strength of contraction. Such a mode of change in the excitability of the Ib inhibitory pathway could be explained either by additional inhibition of Ib interneurones or by an occlusion (saturation) at the Ib interneuronal level when the contraction was intensified. In order to differentiate these two mechanisms the effect on Ib inhibition of grading contraction was studied with two different intensities of conditioning stimulation, weak and relatively strong; increase in Ib inhibition by antagonist contraction began to be suppressed at a stronger contraction when weaker conditioning was applied, and vice versa (Fig. 5). This finding favours the mechanism of occlusion. If Ib interneurones were inhibited, such inhibitory mechanisms would depend on the strength of contraction regardless of conditioning intensities, and Ib inhibition should have increased in amount when GM stimulation was intensified, since the intensity curve of GM stimulation revealed a progressive increase in inhibition (Fig. 3). But conversely, if they were occluded, the amount of increase in Ib inhibition should depend on the conditioning intensity, and stronger GM intensity would then be more likely to cause saturation in facilitating Ib interneurones, leading to earlier suppression of increase in Ib inhibition. An alternative explanation would be that this suppression is due to inhibitory interaction among Ib interneurones (Brink *et al.* 1983*b*). The latter two mechanisms could be further supported by the intensity curve, which demonstrated in a few subjects that the inhibition always increased in amount below or around its threshold on contraction, whereas it decreased with a stronger conditioning intensity even at the same strength of contraction. In any event Ib interneurones could not only be facilitated subliminally but also be fired by descending facilitation alone without peripheral Ib impulses.

Mechanism of increase in Ib inhibition on antagonist contraction

The next issue is what mechanisms are concerned in the increase in inhibition. Since the foot was fixed to the footplate with straps and contraction was performed almost isometrically, cutaneous afferents in the dorsum of the foot must have been

excited on pretibial contraction. Cutaneous afferents have been known to modulate activity in the Ib inhibitory pathway (Lundberg, Malmgren & Schomburg, 1977; Pierrot-Deseilligny *et al.* 1981*a*), and furthermore this modulation is selectively controlled in voluntary contraction depending on the connection from one muscle to another and on the muscle contracted, either homonymous or synergistic (Pierrot-Deseilligny *et al.* 1982). According to these authors, the effective skin regions where cutaneous afferents could modulate the activity of the Ib inhibitory pathway were restricted to the foot. In this study, however, even when the foot was not fixed to the plate, being free from contact with straps or the foot-plate, inhibition did increase. Cutaneous afferents, therefore, are unlikely to have modulated the amount of inhibition on pretibial contraction. Nevertheless, this notion does not exclude the possibility that the activity of the inhibitory pathway was controlled by descending pathways via interneurones mediating cutaneous effects onto Ib interneurones, which was postulated by Pierrot-Deseilligny *et al.* (1982).

On contraction, cutaneous afferents could be excited even without fixation of the foot by sliding of the tendon underneath the skin and skin movement associated with the voluntary movement etc. Therefore, the latency of the actual movement from the onset of pretibial EMG was measured on a storage oscilloscope using an accelerometer which was attached to the dorsum of the foot. It was revealed that foot movement occurred too late to infuence test reflexes.

Neuronal organization from descending pathway to Ib interneurones

The experiment comparing the effect of ipsilateral and contralateral contraction on Ib inhibition (Fig. 6) revealed that the increase in inhibition was specifically related to contraction of the direct antagonists of the tested muscle (Sol).

Ib interneurones have a wide convergence from supraspinal and segmental inputs (see Fig. 12 in Harrison & Jankowska, 1985). Pierrot-Deseilligny *et al.* (1982) demonstrated the change in the excitability of the Ib inhibitory pathway from GM to quadriceps motoneurones in voluntary contraction of triceps surae muscle. According to them Ib inhibition was not modulated on contraction. However, when GM stimuli were preceded by cutaneous stimulation, Ib inhibition decreased in amount at rest and increased on triceps surae contraction, whereas cutaneous stimulation itself exerted no effect on the test reflex either at rest or on contraction. From these results they concluded that modulation of excitability in the Ib inhibitory pathway was not effected directly on Ib inhibitory interneurones, but was achieved via facilitatory or inhibitory interneurones (tentatively referred to as cutaneous interneurones here) receiving input from cutaneous afferents and acting on Ib inhibitory interneurones. The increase in Ib inhibition from GM to Sol on pretibial contraction, which was presented in this study, is possibly mediated by such cutaneous interneurones. It may be noted, however, that Ib interneurones must have fired on contraction as weak as a few per cent of the maximal voluntary contraction (Fig. 5). If cutaneous interneurones are included in modulation of excitability in the Ib inhibitory pathway, it would mean that such cutaneous interneurones (here facilitatory) can be fired by pretibial contraction alone (i.e. without GM conditioning stimulation) to give excitatory postsynaptic potentials (EPSPs) in Ib interneurones and furthermore to make them fire. But the fact that cutaneous stimulation itself evoked no effect on test reflexes even on stronger

contraction indicates that no firing of such cutaneous interneurones took place on contraction.

On the other hand, it is known that the Ib inhibitory interneurones are facilitated by stimulation of the cortico- and rubrospinal tracts in the cat, and that the connection is monosynaptic (Harrison & Jankowska, 1985). Consequently, the increase in Ib inhibition revealed in this study might have been driven by descending pathways directly onto Ib inhibitory interneurones (and maybe to a lesser extent indirectly through cutaneous interneurones). This notion, of course, does not exclude the possibility that central mechanisms, which modulate the excitability of the Ib pathway, can be organized in different ways in other situations such as locomotion (Pierrot-Deseilligny *et al.* 1982) or exploratory movements (Lundberg *et al.* 1977; Cavallari, Fournier, Katz, Malmgren, Pierrot-Deseilligny & Shindo, 1985), because supraspinal centres seem able to choose one from the available patterns of Ib effects (Baldissera *et al.* 1981).

Functional significance

Increase in Ib inhibition onto antagonist motoneurones on voluntary contraction will be beneficial in suppressing these motoneurones and thus in assuring the smoothness of voluntary movement. This modulation occurred on such weak contraction that control of the Ib inhibitory pathway can be assumed to be very sensitive, implying that the Ib inhibitory pathway will actually function in the ordinary performance of movements.

In the present study it is worth noting that Ib interneurones on the antagonist side are considered to be excited to *fire* simultaneously as agonist motoneuronal discharge. In general the stretch reflex of the *antagonists* seems to be disadvantageous in voluntary movement: voluntary contraction causes passive stretch of the antagonists, and motoneurones innervating the stretched antagonists would be first facilitated by the fastest conducting Ia fibres from the muscle spindle which has the lowest threshold for stretch. The stretch reflex then evokes their contraction, counteracting the voluntary movement. However, if Ib interneurones fire prior to actual stretch of the antagonists, evoking an inhibitory postsynaptic potential in antagonist motoneurones, these motoneurones will have already been suppressed to prevent the stretch reflex, when Ia impulses reach them.

Although Ib inhibition is well known as an autogenetic inhibitory mechanism (see Baldissera *et al.* 1981), Ib inhibitory interneurones will also contribute to 'reciprocity', inhibiting actively antagonist motoneurones. Reciprocally inhibitory action to antagonist motoneurones is seemingly peculiar, since Ib effect on antagonist motoneurones is known as facilitatory (Laporte & Lloyd, 1952). On tonic contraction Ib discharge from active tendon organs is naturally considered to exert some effects on antagonist motoneurones through Ib *excitatory* interneurones. However, little is known whether and how these excitatory interneurones are modulated in such a situation, and supraspinal control on these excitatory interneurones remains to be clarified.

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