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ACTIONS ON γ-MOTONEURONES ELICITED BY ELECTRICAL STIMULATION OF GROUP II MUSCLE AFFERENT FIBRES IN THE HIND LIMB OF THE CAT

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SUMMARY

1. The reflex effects elicited by electrical stimulation of group II muscle afferent fibres were recorded with micro-electrodes in ninety-eight hind-limb γ-motoneurones of cats anaesthetized with chloralose.

2. Eighty-one of the γ-cells were classified as either static or dynamic by means of stimulation in the mesencephalic area for dynamic control known to influence dynamic γ-motoneurones selectively.

3. A high responsiveness to activity in group II muscle fibres was found for the whole sample of γ-cells.

4. Group II muscle action on dynamic γ-motoneurones was found to be more frequent than that on static ones.

5. Excitation from group II fibres outweighed inhibition. This was clear cut for flexor γ-motoneurones. In extensor γ-cells, excitation prevailed by a small margin only. However, for both static and dynamic extensor γ-cells, excitation prevailed from both posterior biceps–semitendinosus and the gastrocnemius–soleus nerves, whereas inhibition was more frequent from the deep peroneal and quadriceps nerves.

6. All the reflex effects studied were likely to be mediated via oligosynaptic pathways. The shortest latencies of excitatory effects were compatible with a disynaptic coupling. The fastest inhibitions were presumably trisynaptic.

7. The present findings, supported by a parallel study of reflexes evoked by group III muscle afferents, strongly suggest that the reflexes on γ-motoneurones are not organized in accordance with the concept of flexion reflex afferents as conceived for α-motoneurones.

8. The interpretation of the results suggests a particularly independent position for dynamic γ-cells in relation to α- and static γ-motoneurones. Hence, the results also furnish an argument against the concept of α–γ linkage.

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INTRODUCTION

The controversy surrounding discussions of the function of secondary spindle afferent fibres has been a source of continuing embarassment for neurophysiologists. For a long time very little was known about their central actions, apart from their contribution to stereotyped flexor reflexes, as subsumed under the f.r.a. (flexion reflex afferents) concept (Eccles & Lundberg, 1959; Holmqvist & Lundberg, 1961; cf. Appelberg, Hulliger, Johansson & Sojka, 1983*b*). However, it has always been difficult for some investigators to accept this as the sole function of secondary spindle afferents. This opinion gained weight when Matthews (1969) suggested, on indirect evidence, that secondary muscle spindle afferents contributed to the stretch-evoked excitation of extensor α-motorneurones. This was based on a number of observations, such as the finding that 'reflex response to simple stretch was often rather stronger than the reflex response of the same muscle to high frequency vibration'. The proposal seemed only proper given the well known stretch-receptor properties of the secondary endings. It soon received support when it was shown, using the spike-triggered averaging technique, that secondary afferents could indeed evoke short-latency and even monosynaptic e.p.s.p.s in α-motoneurones (Kirkwood & Sears, 1975; Stauffer, Watt, Taylor, Reinking & Stuart, 1976).

Our findings provide another example of a specific central action exerted by group II afferents, presumably of spindle origin, in the reflex control of motoneurones. The present results clearly show that secondary but not primary (Appelberg, Hulliger, Johansson & Sojka, 1983*a*) spindle afferent units contribute considerably to the excitatory and inhibitory control of γ-motoneurones.

A preliminary report of this study has been published (Appelberg, Johansson & Kalistratov, 1977).

METHODS

Intra- or extracellular micro-electrode recordings were obtained from hind-limb γ-motoneurones of cats anaesthetized with chloralose. A number of hind-limb nerves (supplying various muscles, skin areas and the knee joint) were dissected for electrical stimulation. Each cell was first identified as a γ-motoneurone on the basis of its conduction velocity (below 55 m/sec). In the majority of cases the γ-cells were also classified further as either static or dynamic using the indirect method of mesencephalic stimulation (Appelberg, 1981). The γ-motoneurones were then tested by widely ranging graded electrical stimulation of a number of input nerves.

A detailed account of the methods used for the present series of experiments has been given in the accompanying paper (Appelberg, Hulliger, Johansson & Sojka, 1983*a*). The only addendum required concerns the criteria used for the differentiation between reflex effects mediated by group II and group III muscle afferent fibres.

Often an electrical activation of group II fibres gave rise to the same type of effect (inhibition or excitation) as did the activation of the group III afferents running in the same muscle nerve. Furthermore, the possibility that the stimulus strength necessary for recruitment of all group II fibres also activated group III fibres could not be ruled out (Boyd & Kalu, 1979). Thus, not surprisingly, a careful analysis of the results revealed that the reflex effects elicited in γ-motoneurones by graded electrical stimulation of muscle nerves could not always be attributed unambiguously to selective activity in only either group II or group III fibres. Even when a range of tests with finely graded electrical stimulation was available many responses turned out to be real mixtures of group II and group III effects. On the other hand, when only a limited number of tests (within the group II and/or group III range) were available, it could not always be determined whether

a proven group II effect was restricted to group II action or whether it also included group III action. Given this predicament, a strategy of classification was adopted to identify group II actions and to separate them from group III ones.

Group II effects were thus allocated to one of four categories (see below). The reflex effects were classified among these categories on the basis of (*a*) the effective stimulation intensity, (*b*) the latency of the response, (*c*) the relationship between the effect and the components of the incoming volley and (*d*) the following definitions of the nature of group II and group III responses.

Group II effects. A response or part of a response was judged to be of group II nature if the threshold of the effect was reached between 2 and 7–10 times Ia stimulation threshold and/or if the effect took place below the minimal group III latency but above the minimal group II latency (See Appelberg *et al.* 1983*a*, Methods, *Response latencies*).

Group III effects. A response or part of a response was judged to be of group III nature if it appeared only above 7 times threshold *and* at a latency longer than the minimal group III latency (see above).

Given the differences in peripheral conduction distance from the stimulating electrodes on different muscle nerves, the minimal group III latency (as measured from the initial positivity of the group I component of the incoming dorsal root volley) was obviously different for different muscle nerves. The values relied upon for the present analysis were calculated for each nerve from the mean value of measured conduction distances, a maximum group III conduction velocity of 24 m/sec (Hunt, 1954; Hursh, 1939; Lloyd & Chang, 1948) and a central delay for group III fibres of 1 msec. The figures so obtained were 4·3 msec (p.b.s.t.), 7·0 msec (d.p.), 7·0 msec (s.d.p.), 4·3 msec (a.b.s.m.), 6·0 msec (g.s.), 2·5 msec (quadriceps) and 7·0 msec (f.d.l.). If anything, these estimates of minimal group III latencies are too small since they are based on the assumption of a rather short central delay (1 msec) and on a conduction velocity figure for small myelinated fibres which might be too high (Coppin & Jack, 1972).

Considered in isolation, the magnitude of the group II effects did not influence the classification, since the sole basis of the employed criteria of classification was the extent of group III admixture with the group II component.

The categories of group II effects and the criteria of classification

Examples of each category are illustrated in Fig. 1.

1. *Pure group II action.* ((*a*) *and* (*b*), *below*)

(*a*) The effect was of group II nature (see above).

(*b*) The effect had its maximal magnitude between 2 and 7–10*T* (Fig. 1, 1*A–D*) and/or fell almost in its entirety below the minimal group III latency. This means that, when tested within the group III range of stimulation intensities, the size of the reflex effect should either not increase further (Fig. 1, 1*A–D*), or any enhancement of the effect should still occur below the minimal group III latency.

2. *Predominant group II action.* ((*a*) *and* (*b*), *below*)

(*a*) The effect had a group II component (see above).

(*b*) The major part of the effect grew up between 2 and 7–10*T* and/or the main part of the effect took place below the minimal group III latency (Fig. 1, 2*A–D*; see also Fig. 1*A–D* in Appelberg *et al.* 1983*a*).

3. *Undecided group II/III dominance.* ((*a*) *and* (*b*) *or* (*c*), *below*)

(*a*) The effect had a group II component (see above).

(*b*) The effect increased above 7–10*T*, and the enhancement had a latency which was longer than the minimal group III value and could not clearly be separated from, or was of approximately the same magnitude as, the group II component (Fig. 1, 3*A–D*).

(*c*) A group III component was established, but the cell was inadequately tested within the group II or group III stimulation ranges.

4. *Predominant group III action.* ((*a*) *and* (*b*), *below*)

(*a*) The effect had a group II component (see above).

(*b*) A group III contribution, clearly larger than the group II component, was established (Fig. 1, 4*A–D*).

When a cell from a muscle nerve was studied carefully within the group II intensity range (2 to 7–10*T*) and when a group II effect was found thereby, whilst tests in the lower group III intensity range gave no further increase of the effect, this response was classified as a pure group II effect.

When a cell was studied within the group III stimulation intensity range only and an effect, with a latency longer than the minimal group III value, was found, this response was classified as a pure group III effect.

Generally, the strategy of classification was conservative, with dubious borderline cases being allocated to the category with the higher group III contamination. In particular, the borderline between groups II and III was drawn by relying on the *minimal* group III latencies (see above).

Thus, if anything, the prevalence of pure or predominant group II effects was underestimated. In fact some of the category 2 effects might have been category 1 effects, and some of the category 3 effects might conceivably have been category 2 effects. However, given the criteria of classification that were employed it is highly unlikely that category 3 effects (undecided group II/III dominance) were falsely allocated to the category of group III dominance and vice versa.

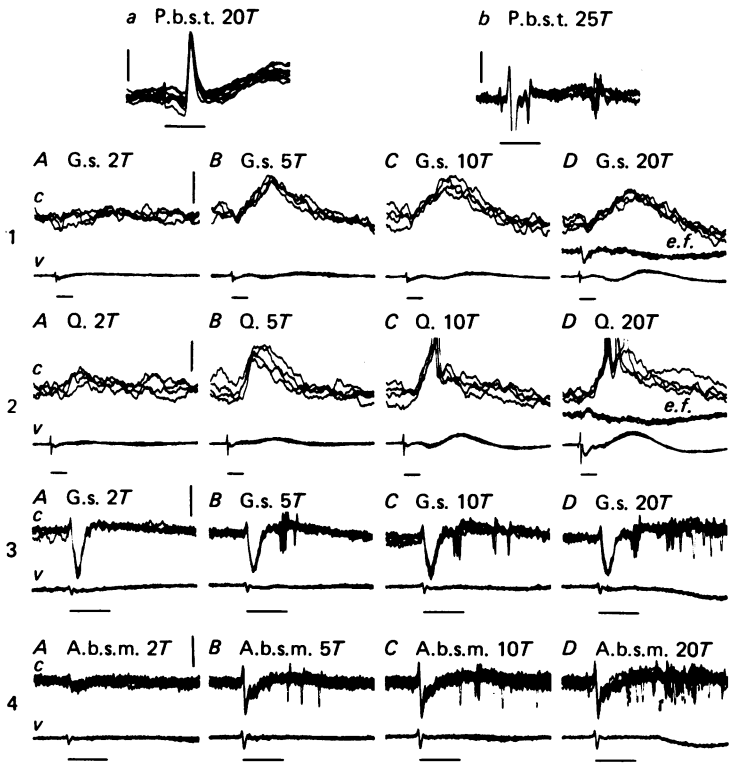
A perusal of the criteria of classification might surprise the reader, since they imply that some of the effects that occurred only with stimulation well within the conventional group III range (Eccles & Lundberg, 1959; Boyd & Kalu, 1979), should still be considered as pure or predominant group II effects. However, it should be stressed that such an assessment was made only when the latency of the effect was clearly below the minimal group III latency (see above). Given the single-shock stimulation regime at low repetition rate (1–2/sec; Appelberg *et al.* 1983*a*), the most likely explanation for such a discrepancy between the required intensities and the observed latencies seems to lie in the non-uniformity of the conditions of muscle nerve stimulation: given the relatively large diameter of the nerves and the possibility of short-circuiting of the nerve by tissue fluid, it is quite probable that the group II fibres furthest away from the electrode were only activated above the group II/III dividing line (Eccles & Lundberg, 1959; Boyd & Kalu, 1979). Moreover, when only high-threshold effects were investigated the threshold intensity of Ia fibres was not as frequently checked as it was with tests within the low-threshold range, so that drifts in threshold, leading to errors of up to 10%, could not always be excluded.

There was no such ambiguity about contributions to any of the responses from group IV fibres, not even with the highest intensities of stimulation used (50*T*). All the responses described in the following occurred at relatively short latencies, well below the minimal group IV latency (for details see Appelberg *et al.* 1983*b*).

Abbreviations used in the text

A.b.s.m., anterior biceps and semimembranosus (nerves or muscles); D, dynamic γ-cell; d.p., deep peroneal nerve (pure muscle nerve supplying extensor digitorum longus and tibialis anterior); s.d.p., deep peroneal nerve with small cutaneous admixture (comprising d.p., the branches to peroneus brevis, to peroneus tertius, to extensor digitorum brevis and the deep peroneal cutaneous branch); f.d.l., flexor digitorum et hallucis longus (nerve or muscle); f.r.a., flexion reflex afferents; g.s., gastrocnemius and soleus (triceps) (nerve or muscle); N, non-classified γ-cell; p.b.s.t., posterior biceps and semitendinosus (nerves or muscles); Q., quadriceps (nerve or muscle); S, static γ-cell; *T*, threshold; Tib., tibial nerve or muscle.

Fig. 1. Classification of group II muscle effects on γ-motoneurones. Examples of group II excitation, with various amounts of group III admixture, elicited from different muscle nerve inputs in two p.b.s.t. γ-cells. Rows 1 and 2 show intracellularly recorded responses of a dynamic p.b.s.t. γ-cell which was antidromically identified with the electrode in juxtacellular position (*a*, top row; cell no. 28; conduction velocity 39 m/sec; membrane potential > 20 mV). In 1, *pure group II action*. Stimulation of the g.s. nerve gave rise to an e.p.s.p. which was quite pronounced at 5*T* (1*B*) and which did not grow above 10*T* (division line between group II and group III stimulation strengths) (compare 1*C* with 1*D*). In 2, *predominant group II action*. Stimulation of the quadriceps (q.) nerve provoked an e.p.s.p. within the group II stimulation range (2*A* and 2*B*). When the stimulation strength was raised to 10*T*, action potentials were added to the early component of the



e.p.s.p. (2*C*). The further slight enhancement of the effect between 10*T* and 20*T* (occasional occurrence of a second action potential in 2*D*) could not safely be ascribed to activation of fibres within the group II range only, and a contribution of group III fibres could not be excluded. Rows 3 and 4 show extracellularly recorded responses of a static p.b.s.t. γ-cell which was antridromically identified (*b*, top row; cell no. 141; conduction velocity 31 m/sec). In 3, *undecided group II/III dominance.* An excitation (discharge of action potentials) was evoked by stimulation of the g.s. nerve within the group II range (3*B*). This first burst fell in its entirety within 2·1–3·0 msec (measured from the initial positivity of the group I component of the incoming dorsal root volley) which is shorter than the minimal group III latency (6·0 msec). A second burst of action potentials appeared at 10*T*. It fell above the minimal group III latency and increased in magnitude between 10*T* and 20*T*. This latter burst was of the same size as the first. In 4, *predominant group III action.* Stimulation of the a.b.s.m. nerve within the group II range provoked an impulse discharge (at 5*T*; 4*B*). The much more pronounced second burst of impulses, which (arrow in 4*D*) appeared between 10*T* and 20*T*, must be ascribed to the activation of group III fibres. Superimposed responses (four to ten sweeps) to single-shock stimulation: *c*, intra- or extracellular recordings from γ-cells; e.f., extracellularly recorded field potential responses to stimulation at the same intensity as for the cell recordings (see Appelberg *et al.* 1981 Methods); *v*, incoming dorsal root volley. The nerves stimulated and the intensity of stimulation are separately indicated for each diagram, the latter as relative intensities in multiples of the Ia threshold (*T*) (separately determined for each individual nerve, always relying on the incoming volley). Horizontal calibrations: 5 msec. Vertical calibrations: 2 mV.

RESULTS

Categories of effects

Electrical stimulation of hind-limb muscle nerves above the group II threshold intensity elicited a wide range of effects, as regards both the mixture of excitation and inhibition and the contribution from group II and higher threshold afferent fibres.

As is detailed in the Methods, it was deemed impossible to classify unambiguously all effects observed with activation of afferent fibres within the conventional group II or group III ranges of stimulation intensities as either pure group II or pure group III effects. Rather, the reflex effects, for which at least some contribution from group II fibres was established (see Methods), could exhibit different degrees of contamination by group III action. Group II reflex effects were therefore allocated to one out of four categories, for each of which an example is illustrated by the four sets of excitatory responses in Fig. 1: pure group II action, for which there was no evidence of group III contamination (Fig. 1, 1*A–D*); predominant group II action, with relatively weak group III contamination (Fig. 1, 2*A–D*); undecided group II/III dominance, when group II and group III contributions were comparable or when it was not feasible to settle the relative contribution from group II and group III fibres respectively (Fig. 1, 3*A–D*); predominant group III action, with a group III contribution that was clearly larger than the group II-mediated effect (Fig. 1, 4*A–D*). It should be stressed that for all four categories the occurrence of a group II contribution to an observed reflex effect was clearly established, and that it was only its relative size, compared with any concomitant group III action, that varied.

Over-all trends of group II reflex action

Of the total sample of 120 γ-motoneurones of this series of reports (Appelberg *et al.* 1983*a*, *b*; see also Johansson, 1981) most cells (ninety-eight) were investigated for their responses to stimulation of muscle group II afferent fibres. These cells are presented in this paper. Out of the ninety-eight cells, 15 dynamic (D), 16 static (S) and 6 non-classified (N) cells projected to p.b.s.t.; 24 D, 13 S and 7 N to g.s.; 2 D, 6 S and 2 N to a.b.s.m. 2 D, 1 S and 2 N to d.p. and 1 D and 1 S to the tibial muscle.

Fig. 2 shows the general features of the reflex actions exerted by group II muscle afferent fibres in the two largest populations of the present sample of γ-motoneurones, namely those projecting to the p.b.s.t. and g.s. muscles. The frequency histograms show, separately for dynamic and static γ-cells, in the upper part, the incidence of excitation (filled triangles) and of inhibition (open circles) and, in the lower part, the frequency of tests in which no group II action could be observed. The observations from all the muscle nerves studied were combined, irrespective of the category of group II action they belonged to. The results so obtained are displayed as percent of the total number of observations made for each of the four groups of γ-cells. The term 'observation' is used to describe the final assessment of the effects on a single γ-cell from one particular nerve tested within the range of group II fibre action. Thus in Fig. 2 a single cell may be represented by up to seven observations (see Figs. 4 and 5) and each observation may be based on anything up to fifteen tests with graded electrical stimulation.

It may be seen from Fig. 2 that group II muscle fibres acted very potently on

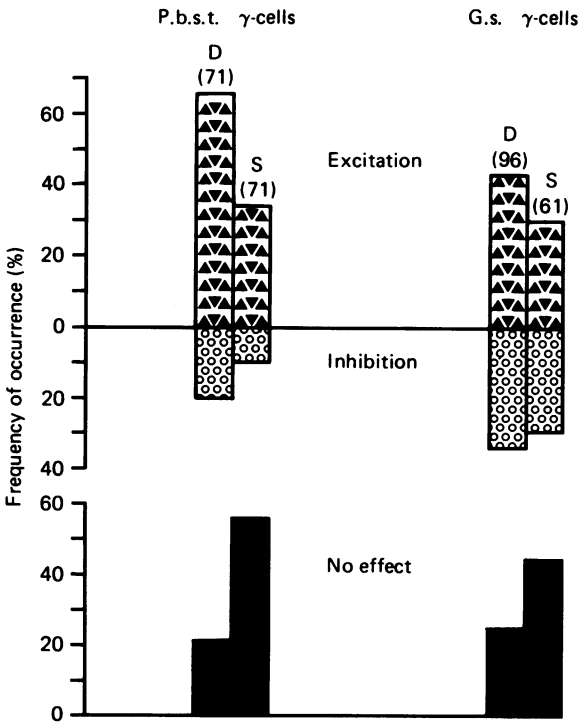


Fig. 2. Type and incidence of group II muscle reflex action elicited in p.b.s.t. and g.s. γ-motoneurones by electrical single-shock stimulation of hind-limb muscle nerves. The frequency of occurrence, expressed as percent of the total number of observations within each group of γ-cells, is plotted separately, in the upper part for excitation (upwards, triangle pattern) and inhibition (downwards, circle pattern) and in the lower part for absence of group II reflex action (black columns). For both excitation and inhibition individual effects (see text) could belong to any one of the four categories of group II reflex action described in the Methods (see also Figs. 1, 4 and 5). Pooled data from thirty-one p.b.s.t. γ-motoneurones (fifteen dynamic (D) and sixteen static (S) cells) and from thirty-seven g.s. γ-cells (twenty-four dynamic and thirteen static). Individual cells may be represented by between one and seven observations, each from different muscle nerves (see Figs. 4 and 5). Note that individual observations (i.e. one cell tested with one nerve within the group II range) could contain both excitatory and inhibitory effects (see e.g. Fig. 4*A*, cells 73, 29, 106 and 127). For each group of γ-cells the percentage figures displayed may therefore add up to more than 100%. The numbers of observations within each of the four populations of γ-cells illustrated are given above each column.

γ-motoneurones. In about two-thirds of all observations made, either excitatory or inhibitory effects were encountered. Excitation was more common than inhibition, particularly for p.b.s.t. γ-cells. Moreover, dynamic cells were more readily influenced by group II fibres than static cells, as regards the occurrence of both excitation and inhibition. This was true for the p.b.s.t. cells as well as for the g.s. cells. The main difference between these two populations lies in the balance between excitation and inhibition from group II fibres. The pooled histograms of Fig. 2 suggest that for g.s. cells excitation outweighed inhibition only by a small margin, whereas for p.b.s.t. cells there was a clear predominance of excitation. However, when the over-all reflex

actions elicited from *individual nerves* are considered (Fig. 3) it is apparent that the impression conveyed by Fig. 2 does not, as a generalization, apply to g.s. γ-motoneurones.

The frequency histograms of Fig. 3 show, in a way similar to the display of Fig. 2, the combined group II actions on the dynamic and static γ-cells of the p.b.s.t. and g.s. sample. Fig. 3 was obtained by counting separately the excitations, inhibitions

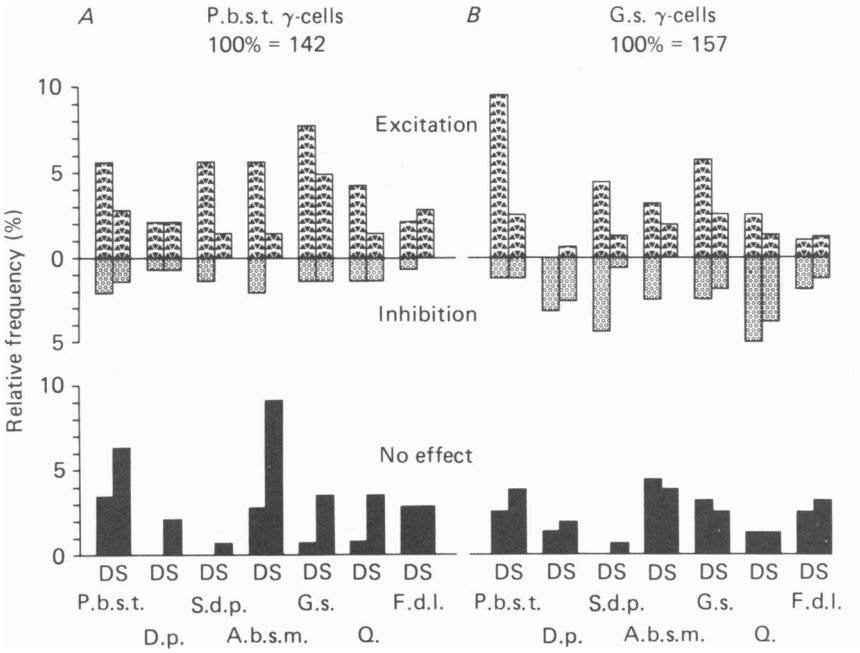


Fig. 3. Relative frequency of group II reflex actions from various muscle nerves on p.b.s.t. γ-cells (*A*) and g.s. γ-cells (*B*). The relative frequencies are expressed as percent of the total number of observations (see text) within each population (142 for all p.b.s.t. cells in *A*; 157 for all g.s. cells in *B*), in order to ease comparison between effects in dynamic and static γ-cells in this Fig. and between it and Fig. 4 of the accompanying paper on reflex actions from high-threshold muscle afferents (Appelberg *et al.* 1983*b*). Upper part, excitation (triangle pattern) plotted upwards, and inhibition (circle pattern) plotted downwards. Lower part, absence of group II reflex action (black columns). For each nerve investigated (see inputs, indicated along the abscissa) the effects on dynamic (D, left-hand columns) and static (S, right-hand columns) are plotted separately. Same cells as in Figs. 2, 4 and 5. For further details see legend of Fig. 2.

and negative observations for the various muscle nerve inputs in the group II range (each individually) within the different populations of γ-motoneurones (p.b.s.t. dynamic, p.b.s.t. static, g.s. dynamic and g.s. static). These sums were then expressed as percent of the total number of observations for all p.b.s.t. cells (Fig. 4*A* and *B*) and for all g.s. cells (Fig. 5*A* and *B*) respectively. As can be seen (Fig. 3*B*) there was a clear predominance of excitation also in g.s. γ-cells from some nerves, particularly in the dynamic neurones. The predominance of excitation was especially outstanding from the group II fibres of the flexor muscle nerves p.b.s.t. On the other hand, inhibition predominated from d.p. as well as from quadriceps (i.e. both from

antagonistic and functionally synergistic muscles), in both dynamic and static g.s. γ-cells (see Fig. 3*B*).

Reflex patterns of individual γ-cells

Whilst the pooled data of Figs. 2 and 3 portray the general trends of group II reflex action on the largest populations of γ-motoneurones studied, they ignore the considerable variability in effects from one and the same nerve among cells belonging to the same population. Therefore the reflex patterns found for individual γ-cells are displayed in the chequer-board diagrams of Figs. 4 and 5. In the diagrams each cell is represented by a horizontal row of squares and each input nerve by a vertical column of squares. A black or symbol-containing square (see legend in Fig. 4) denotes that the γ-cell at issue has been investigated for group II reflex action from the nerve indicated above the column. An open square shows that the cell has not been tested with the nerve in question. The cells are numbered for cross-reference to the other papers dealing with the present series of experiments, yet in each diagram they are arranged so as to focus attention onto the main features. The two columns at the far right of every chequer-board diagram characterize the over-all responsiveness of each cell to stimulation of skin and joint receptor afferent fibres, irrespective of the receptor or threshold categories that were responsible for the reflex effects.

In Figs. 4 (p.b.s.t. γ-cells) and 5 (g.s. γ-cells) it can be seen how the general features were distributed among the individual cells. These features were also encountered in the smaller cell populations studied (cells to other muscles and non-classified cells). The receptive territories of individual γ-cells were generally very wide. The reflex effects were not only elicited from a range of muscle nerves, but also from skin and joint afferent fibres (see columns at far right in Figs. 4 and 5 in which the symbols represent the combined effects covering the whole range of afferent fibres; triangles represent excitations and circles inhibitions). For muscle nerves, reflex action from autogenetic, synergistic and even antagonistic muscle nerves was frequent. Neither autogenetic, nor antagonistic reflex effects were dominant. Moreover, stimulation of group II muscle afferents could elicit both excitatory and inhibitory effects in one and the same cell, yet the proportion of these effects varied considerably, both between cells of the same population (see below) and between populations (see above, *Over-all trends of group II reflex action*). Not infrequently, individual cells were exclusive recipients of either excitation or inhibition, but no single nerve was an exclusive source of one or the other.

P.b.s.t. γ-motoneurones. The arrangement of *dynamic* p.b.s.t. γ-cells (Fig. 4*A*) draws attention, first to the effectiveness of the group II muscle afferents (note scarcity of black squares representing absence of effects: fifteen out of seventy-one observations) and, secondly, to the high proportion of pure or predominant group II effects (fifty-one out of sixty-one observations) compared to effects with group III or undecided group II/III dominance (ten out of sixty-one). Very distinct features are also the clear prevalence of excitations (occurring in twelve out of fifteen cells) over inhibitions (in six out of fifteen cells) and the very widespread distribution of inhibitory as well as excitatory inputs. In spite of the dominance of excitation, both types of effects could be elicited from all the nerves tested. Yet the majority of cells were exclusive recipients of excitation or of inhibition (ten out of fourteen responsive cells). Only

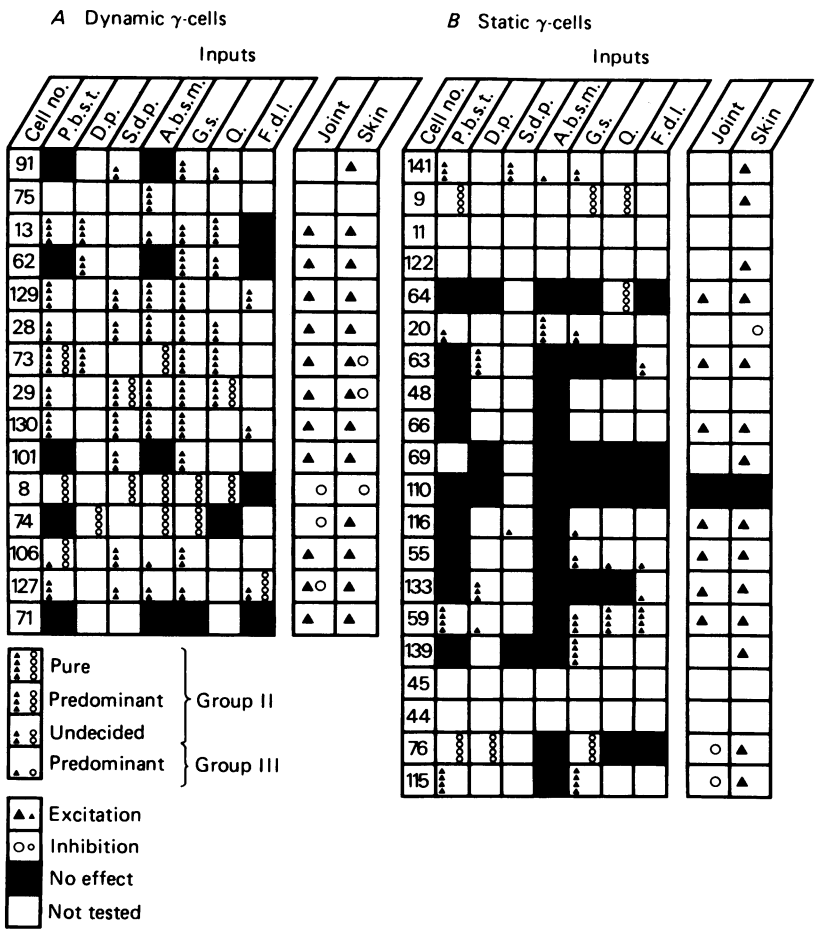


Fig. 4. The reflex actions of muscle group II, skin and joint receptor afferent fibres on p.b.s.t. γ-motoneurones. For details concerning the four categories of group II effects, see Methods and Fig. 1; for further explanations see text (*Reflex patterns of individual* γ-cells). The reflex actions from group III muscle afferent fibres on the same cells are described by Appelberg *et al.* (1983*b*). Note that cells 73 and 74 (in *A*, dynamic) and cells 9, 11, 64 and 76 (in *B*, static) were influenced also by group I muscle afferent fibres (Appelberg *et al.* 1983*a*, Figs. 3 and 4). In *B*, two cells (nos. 11, 44 and 45) are displayed although they were not tested with any of the nine inputs listed. They were nevertheless included to facilitate cross-reference with Appelberg *et al.* (1983*a*, *b*).

four cells received both types of reflex effects, interestingly often as mixed effects from one and the same nerve (see e.g. cells no. 73, 29, 106 and 127 in Fig. 4*A*).

The most efficacious source of group II reflex action appears to be the g.s. nerve, followed by s.d.p., d.p. and quadriceps, since in all these nerves black squares (absence of effects) are particularly rare, but no nerve stands out as particularly ineffective.

The pattern of responses observed with *static* p.b.s.t. γ-cells (Fig. 4*B*) differs strikingly from that of dynamic cells (Fig. 4*A*). Most salient in the chequer-board diagram is the high incidence of black squares (forty out of seventy-one observations),

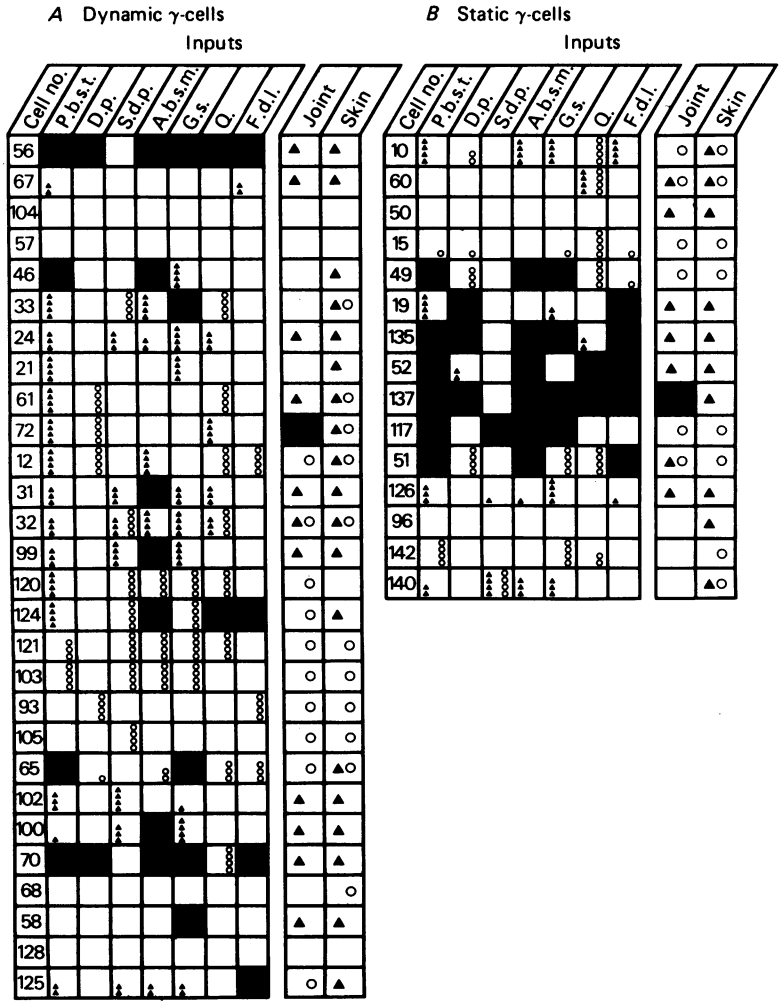


Fig. 5. Reflex effects elicited in g.s. γ-motoneurones by muscle group II, skin and joint receptor afferent fibres. For account of symbols see Fig. 4. In cells 58, 61, 65, 102, 121 (in *A*, dynamic γ-cells) and in cell 60 (in *B*, static γ-cells) reflex effects were elicited also by stimulation of muscle afferent fibres in the group I range (see Appelberg *et al.* 1983*a*, Figs. 3 and 4). The cells, which were not tested with the inputs listed in the present diagrams (cells 104, 57 and 128 in *A*), were included for cross-reference with Appelberg *et al.* (1983*a*, *b*).

i.e. the frequency with which group II muscle afferent fibres failed to act on static p.b.s.t. γ-motoneurones. The static cells also show a proportionally much higher incidence of undecided II/III or group III dominated reflex effects (twelve out of thirty-one compared with ten out of sixty-one for dynamic cells).

As for the dynamic neurones, on the other hand, the proportion of unsuccessful tests was highest with stimulation of afferents of the antagonist a.b.s.m. and with the autogenetic input. This stands out markedly in the summarizing histogram of Fig. 3*A* (right-hand columns, labelled S, for each nerve). The most consistent source of group II reflex inputs again seemed to be the g.s. nerve and,

moreover, excitation prevailed over inhibition. All static p.b.s.t. cells were exclusive recipients of either excitation or inhibition, but most nerves were non-selective sources of both excitation and inhibition.

G.s. γ-motoneurones. In Fig. 5 the response patterns of g.s. γ-cells for muscle group II, joint and skin inputs, are displayed in the same way as those for p.b.s.t. γ-cells (Fig. 4). Some of the features encountered with p.b.s.t. γ-cells were equally manifest in the samples of dynamic (Fig. 5*A*) and static (Fig. 5*B*) g.s. γ-cells. Thus (see also Fig. 2), as was also the case with p.b.s.t. cells, the over-all responsiveness to group II stimulation of the dynamic cells (Fig. 5*A*; seventy-two out of ninety-six observations) was considerably larger than that for static cells (Fig. 5*B*; thirty-four out of sixty-one observations).

The distribution of the effective inputs was very widespread and the autogenetic inputs did not assume any preferential position (see also Fig. 3*B*). The main difference to p.b.s.t., which applied to both the dynamic and static populations, was the more equal division between excitation and inhibition. Also, while for p.b.s.t. cells excitatory group II effects dominated over inhibitory ones from all muscle nerves, with the g.s. cells there was a tendency for some muscle nerves to be preferential sources either of excitation (p.b.s.t. and g.s.) or inhibition (d.p. and quadriceps). Moreover, it was observed that the proportion of γ-cells which were non-selective recipients of both excitation and inhibition was somewhat higher than in the p.b.s.t. population (seven out of twenty-two group II responsive dynamic g.s. γ-cells and three out of eleven group II responsive static g.s. γ-cells).

Other populations of γ-cells. An intricate and non-uniform reflex pattern was also found in the smaller populations of static and dynamic d.p. and tibial γ-cells, and in the non-classified populations. The *static a.b.s.m. γ-motoneurones* were remarkable in that they seemed to be recipients preferentially of inhibitory inputs from widely distributed sources. Interestingly, the same cells were also targets of muscle group I inhibition (see Appelberg *et al.* 1983*a*, Fig. 4*A*), and from high-threshold muscle (Appelberg *et al.* 1983*b*), skin and joint afferents inhibitory effects prevailed as well.

State of preparation

As is pointed out above, the occurrence of both excitation and inhibition is a feature common to muscle group II reflex pattern in all populations of γ-motoneurones. Still, one of the salient aspects of the chequer-board diagrams in this account is that individual cells were frequently exclusive recipients of either excitation or inhibition. The question immediately arising is whether this is a fixed property of the reflex pathways converging onto individual γ-motoneurones or whether it reflects specific states of the preparations, favouring either excitation or inhibition for the whole pool of γ-cells. Pertinent information concerning this point could be obtained from experiments in which relatively large numbers of γ-cells were studied. Thus in the experiment that yielded most results ten γ-motoneurones (nos. 24–33) were studied in considerable detail (see Fig. 4*A*: nos. 28 and 29; Fig. 5*A*: nos. 24 and 31–33). Three of these showed excitation, two showed inhibition, and in five excitation and inhibition was blended. In seventeen experiments more than one γ-motoneurone could be tested in detail. Both types of effects were observed in fourteen of these experiments, and in eight experiments cells receiving a blend of excitation and inhibition were found. In fact, cells with mixed effects were regularly found in experiments which also yielded cells which were selective recipients of either

excitation or inhibition. A predominance of either excitation or inhibition was seen only in five experiments. Thus it seems unlikely that the state of preparation should favour one or the other type of effect.

Synaptic linkage of group II reflex effects

Fig. 6 shows the distribution of latencies of excitations (e.p.s.p.s or stimulus-locked discharges of action potentials) and inhibitions (i.p.s.p.s or stimulus-locked decreases in resting discharge) in dynamic and static γ-cells projecting to the p.b.s.t. and g.s. muscles. Each response latency displayed represents the time from the presumed arrival of the activity in the fastest group II fibres at the site of the volley-electrode, at the dorsal root entry zone, to the start of the actual cell response.

The latency from the earliest positive peak of the group I incoming volley was measured. The group II latency was then obtained by subtracting from this figure the delay between the arrival of the activity in the fastest group I fibres (assumed conduction velocity 120 m/sec) and in the fastest group II fibres (assumed conduction velocity 72 m/sec). The two latter figures were calculated separately for each input nerve, so that we obtained samples of response latencies which were comparable, irrespective of the differing lengths of the muscle nerves.

Each of the latencies shown in the histogram corresponds to one observation (i.e. a square containing one symbol in Figs. 4 and 5) and thus to a number (up to fifteen) of tests with graded electrical stimulation. This implies that every latency in Fig. 6 stands for a number of latencies obtained during the tests. Out of these latency figures the shortest reliable one was regarded as the true latency of the effect in question and is hence represented in Fig. 6. However, reliable latencies could not be defined for all of the effects demonstrated. It was, for instance, impossible to determine the latency for an extracellularly recorded excitation when the spike response was recorded in a cell with high spontaneous activity. For extracellularly recorded inhibitions it was also impossible to establish reliable latencies, nor could it be achieved when repetitive stimulation was used. Finally, for p.s.p. responses the quality of the recording determined whether the latencies found should be relied upon or not.

As can be seen from the distribution, in Fig. 6, of all reliably estimated group II latencies from all muscle nerve inputs, the fastest excitatory pathways to γ-motoneurones seem to be at least one synapse shorter (see below) than the fastest inhibitory pathways. Furthermore, the excitation diagrams show an accumulation of latency values around 2 msec, and for the *dynamic cells* (Fig. 6*A*) there is a second concentration of latencies between 2·5 and 4·5 msec. This latter accumulation of excitatory effects is paralleled by a similar grouping of inhibitions (between 3 and 4 msec, lower diagram in *A*). Also the histogram depicting latencies of excitatory effects on *static cells* (*B*) shows a second concentration of effects (between 4–5 msec). No obvious latency difference could be seen between inhibitory effects in dynamic and static cells respectively. However, the size of the sample was rather small.

The calculation of the minimal synaptic coupling was based on the assumption of a central delay (i.e. the delay within the central part of the dorsal root and the intraspinal conduction time) of 0·9 msec (Fu & Schomburg, 1974) and a delay for a single synapse of 0·3–0·4 msec (Stauffer *et al.* 1976). The shortest route for group II

excitation was found to be disynaptic for both dynamic and static γ-cells. There were differences between the muscle nerve inputs as well as between different categories of cells. Disynaptic excitatory effects on dynamic cells (both p.b.s.t. and g.s.) were found from half the muscle nerve inputs. For static g.s. γ-cells, a disynaptic excitatory linkage was found, while excitatory effects on p.b.s.t. static γ-cells were mediated via three (or more) synapses. It was, however, not possible to find any consistent pattern in the differences between various input nerves.

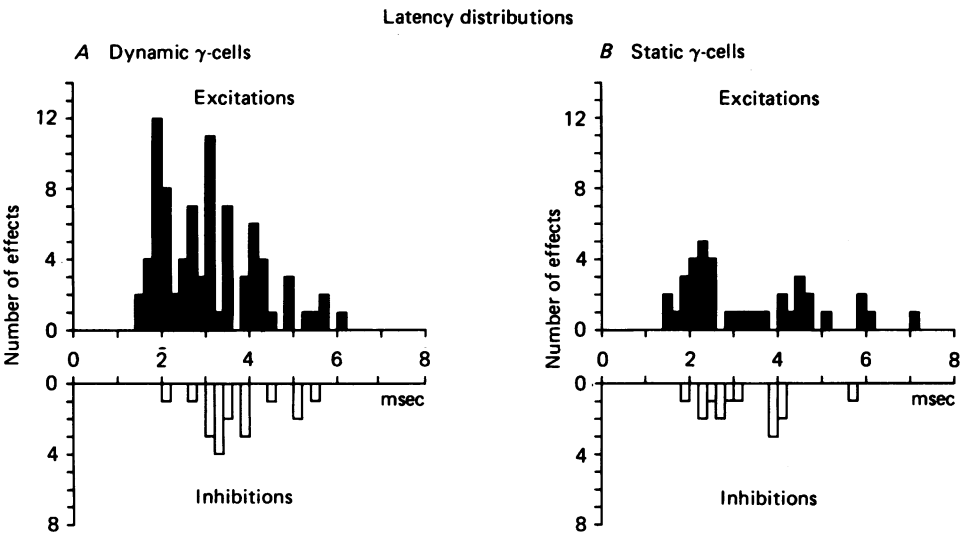


Fig. 6. Latency distributions of group II muscle effects in the p.b.s.t. and g.s. populations of dynamic and static γ-cells. Each latency corresponds to one observation and thus to the most reliable figure obtained with a number of tests (see text). Reliable latencies could not be determined for all effects. Note that only effects with reliably estimated latencies are presented. Upper diagram: excitations (e.p.s.p.s or stimulus-locked discharges of action potentials). Lower diagram: inhibitions (i.p.s.p.s or stimulus-locked decreases in resting discharge). *A*, latencies of effects in dynamic p.b.s.t and g.s. γ-motoneurones. Eighty-three latencies of excitatory effects are given, out of a total number of eighty-eight observations. Of the inhibitions only eighteen (out of forty-seven) are illustrated. Three properly measured inhibition latencies (of 9·7, 11·6 and 14·0 msec) are omitted, given the size of the scales. *B*, latencies of effects in static p.b.s.t. and g.s. γ-motoneurones. Thirty-seven (out of forty-two) excitations and fourteen (out of twenty-five) inhibitions, could be estimated reliably.

DISCUSSION

In the present series of investigations the reflex actions of muscle, skin and joint afferent fibres on γ-motoneurones were studied, using electrical stimulation of peripheral nerves and micro-electode recording from single dynamic or static γ-cells in the spinal cord (Appelberg *et al.* 1983*a*, *b*).

Compatibility with the f.r.a. concept. The main finding of this study is that group II muscle afferent fibres acted potently on γ-motoneurones, preferentially on the dynamic type. The majority of the cells studied belonged to the p.b.s.t. and g.s. populations. In p.b.s.t. γ-cells (i.e. flexor motoneurones) there was a clear prevalence

of excitation over inhibition, irrespective of the nerves from which the reflex was elicited. Also with the g.s. cells (i.e. extensor motoneurones) the over-all incidence of excitation was greater than that of inhibition, although the difference was smaller. However, when the effects from individual nerves were considered, cases of clear predominance of excitation over inhibition were found also for the g.s. population. This was true for group II afferents in both flexor (p.b.s.t.) and extensor (g.s.) muscle nerves. For both dynamic and static γ-cells, in the p.b.s.t. and g.s. populations, group II excitation could be seen even from muscle nerves which gave group III inhibition (see Appelberg *et al.* 1983*b*).

Static γ-cells, in contrast to dynamic cells, were much less frequently influenced by group II muscle afferent fibres, no matter whether flexor or extensor cells were studied. Thus there are clear differences between the two functional classes of fusimotor neurones as regards the reflex pattern from muscle afferent fibres. Dynamic fusimotor neurones are strongly influenced by group II muscle afferents, whereas static fusimotor neurones are mainly operated by group III afferent fibres (Appelberg *et al.* 1983*b*).

In an earlier investigation of the reflex control of γ-cells it was suggested (Grillner, Hongo & Lund, 1969), that the organization of reflexes to γ-cells from high-threshold muscle afferents was the same as described for α-motoneurones by the f.r.a. concept. The present results clearly contradict this inerpretation. For a more detailed discussion the reader is referred to the accompanying paper on the reflex action of group III muscle afferents on γ-motoneurones (Appelberg *et al.* 1983*b*).

Methods and sample bias. The cells of the present series of experiments were classified as static or dynamic fusimotor neurones, by the indirect method of mesencephalic stimulation (Appelberg, 1981; Johansson, 1981). Thus, the type of fusimotor action was not determined directly on muscle spindle afferent fibres. A high proportion of dynamic γ-cells was found. For g.s., the dynamic cells were twice as frequent as static cells. This is in contrast to previous studies in which static fibres prevailed over dynamic fibres by a factor of two to three, when they were classified directly on the basis of their action on spindle afferent units (Matthews, 1972). Although we cannot completely exclude the possibility that some cells were incorrectly classified, it is unlikely that this can account fully for the observed discrepancy. During the experiments it was noticed repeatedly that several dynamic cells were obtained within the same electrode track. It is likely, therefore, that dynamic γ-motoneurones occur in clusters within the motor nuclei while static cells are more scattered. Such an arrangement obviously could lead to a considerable sampling bias.

Since recurrent effects from γ-axon collaterals to γ-cells have been described (Appelberg, Hulliger, Johansson & Sojka, 1983*c*), it is conceivable that some of the effects classified as group II responses were in fact recurrent effects. If so, they must be few, since the recurrent effects found with stimulation of high-threshold muscle efferents (γ-axons) were very rare in preparations with generally high responsiveness of γ-cells. Also they were generally much weaker than the responses classified as group II responses. Yet a final and more quantitative answer to this question could only be arrived at in experiments involving either reversible blocking or even sectioning of the dorsal roots whilst still recording from one and the same γ-cell.

Latencies and synaptic coupling. The fastest group II muscle excitatory effects on

both dynamic and static p.b.s.t. and g.s. γ-cells were found to be disynaptically mediated for half of the muscle nerve inputs tested and trisynaptically for the other half. However, neither a comparison between agonists and antagonists nor between flexors and extensors permitted conclusive generalizations about the differences of minimal synaptic coupling between various input nerves. Rather, such a lack of consistent pattern suggests that the differences in minimal synaptic linkages found from different excitatory inputs are partly due to the limited size of the samples and partly to the fact that the latency figures were calculated on the basis of the conduction delays of the fastest group II fibres (72 m/sec) (see Results, *Synaptic linkage of group II reflexes*). This might have led to a general over-estimation of the number of synapses involved. Hence, more excitatory inputs were probably disynaptically linked to both dynamic and static cells. In particular, it is likely that most of the latencies concentrated around 2 msec (Fig. 6, upper diagrams) represent disynaptically mediated effects. It should be pointed out that for most excitatory inputs monosynaptic connexions cannot be ruled out completely, provided only that the effects were provoked by fibres with intermediate or slow group II conduction velocities.

For all input nerves, the shortest pathways for inhibitory effects were one or two synapses longer than the fastest excitatory pathways.

Although the group II actions on fusimotor neurones studied were oligosynaptic (see also Appelberg *et al.* 1983*a*), the participation of at least one interneurone permits these reflex pathways to be subjected to descending and reflex modulation at the pre-motoneuronal level.

Supporting evidence. The finding of a prevalence of excitation from group II muscle afferents in dynamic γ-motoneurones is also supported by other experimental evidence. Following our initial preliminary account on this aspect of γ-cell reflex organization (Appelberg *et al.* 1977), Noth & Thilmann (1980) and Noth (1981) have taken up the matter using a different technique. They confirmed the prevalence of excitation from p.b.s.t. group II fibres in non-classified g.s. γ-cells by recording from ventral root filaments. Their use of repetitive stimulation might explain the somewhat higher incidence of group II excitation compared with the present figures, since the group II reflex pathways to γ-motoneurones are at least disynaptic (see above) and thus temporal facilitation is likely to occur. Further, using natural stimulation of muscle afferent fibres from p.b.s.t., the predominance of excitation over inhibition, as well as the predominance for action on dynamic fusimotor neurones to triceps, has been confirmed (Appelberg, Hulliger, Johansson & Sojka, 1982).

Functional implications. It should be kept in mind that the reflex pattern from muscle spindle secondary afferents may be obscured by reflex effects from concomitantly activated non-spindle group II afferents. However, in p.b.s.t. and g.s. the majority of group II fibres are known to be secondary muscle spindle afferents, whereas in other nerves an unknown contribution of fibres of other origin cannot be excluded (Boyd & Davey, 1968). Therefore, given the high incidence of effects from g.s. and p.b.s.t., it seems clear that γ-motoneurones indeed *are* influenced by secondary muscle spindle afferents. Also, Appelberg *et al.* (1981, 1982) demonstrated dynamic and, to some extent, static fusimotor effects in g.s. spindles caused by natural stimulation of p.b.s.t. stretch-sensitive receptors. Some functional implica-

tions of this have been discussed previously (Appelberg *et al.* 1977; Appelberg *et al.* 1981, 1982) and a more comprehensive hypothesis concerning the general significance of the reflexes to γ-motoneurones is presented in an accompanying paper (Appelberg *et al.* 1983*b*).

Here we should like to draw attention to aspects of the present results that bear on the concept of α–γ linkage (Granit, 1955, 1970, 1979; see also Grillner *et al.* 1969; Grillner, 1969; Appelberg *et al.* 1983*a*; Hulliger, 1981). With the same regime of electrical stimulation, the action of group II muscle afferents on α-motoneurones has been found to be rather sterotyped and markedly different from the action on γ-motoneurones reported here. α-cells are generally influenced, so that flexor cells are excited and extensor cells are inhibited from wide receptive fields, i.e. from group II afferents from many different flexor as well as extensor muscles (Eccles & Lundberg, 1959; Lundberg, Malmgren & Schomburg, 1975, 1977), and excitatory effects were found in only 15 % of the extensor α-motoneurones (Lundberg *et al.* 1977).

The findings in the present report indicate a rather intricate and less stereotyped pattern of actions of group II afferents on γ-motoneurones (see Figs. 4 and 5). Furthermore, most cells, no matter whether they projected to flexors or extensors or whether they were dynamic or static, had at least a tendency to be pure recipients of either excitation or inhibition, whereas the nerves tended to be non-specific sources of both excitation and inhibition to all types of cells (Figs. 4 and 5). Also, excitatory effects dominated for both flexor and extensor cells. Among a total of forty-four triceps γ-cells, twenty-five (i.e. 57 %) received group II excitation from one or several muscle nerves. In conclusion, primary (Appelberg *et al.* 1983*a*) and secondary muscle spindle afferents as well as group III muscle afferents (Appelberg *et al.* 1983*b*) and recurrent motor axon collaterals, act very differently on α- and γ-motoneurones. This renders it highly unlikely that α and γ activity should, as a general principle, be rigidly linked in the sense that this activity would always be strictly parallel or very similar. In fact, even if a central command initiated a linked activity, this should break down as soon as muscle afferent reflexes come into play.

Given the clear prevalence of muscle group II excitatory action on dynamic and of group III excitation on static γ-motoneurones (Appelberg *et al.* 1983*b*), γ-motoneurones cannot even be considered as a homogenous group as regards their control by proprioceptive reflexes. In particular, dynamic fusimotor neurones stand out as more independent from α-motoneurones than static γ-cells (Appelberg *et al.* 1983*b*). Again, this is not compatible with the stereotyped concept of rigid linkage.

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