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

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SUMMARY

1. Effects on seventy-one single lumbar γ-motoneurones, evoked by graded electrical stimulation of fibres running in the posterior articular nerve of the ipsilateral knee joint (p.a.n.), were studied by micro-electrode recording in twenty-one cats anaesthetized with α-chloralose.

2. Sixty-seven of the γ-cells were classified indirectly as dynamic (thirty-seven) or static (thirty) using the method of mesencephalic stimulation (cf. Appelberg, Hulliger, Johansson & Sojka, 1982).

3. A high general responsiveness (i.e. number of cells with effect/number of cells tested) was found for the whole sample of γ-cells (91·9 % for dynamic and 93·3 % for static cells).

4. The thresholds for the effects were related to the stimulation intensity at which the early negative cord dorsum potential appeared (*T*). For all subpopulations of γ-cells (dynamic and static, flexor and extensor cells) excitatory as well as inhibitory effects were observed at 0·9–1·1 *T*, probably corresponding to 1·1–1·4 times the threshold for evoking a compound action potential in p.a.n. (cf. Discussion). In addition, a considerable number of high-threshold effects were found. Some cells were influenced only from low-threshold joint afferents, some only from high-threshold joint afferents and some cells were influenced from both low- and high-threshold joint afferents. No statistically significant differences in thresholds were found between dynamic and static cells.

5. Among flexor γ-cells excitatory effects were found to predominate, while for extensor γ-cells excitation and inhibition occurred with about equal frequency.

6. The shortest latencies for excitatory effects on dynamic γ-motoneurones were compatible with a trisynaptic pathway, while the routes for excitation of static units and for inhibition of both types of γ-cells seemed to be longer.

7. The possible functional significance of the findings is discussed. The findings seem to support the idea, as suggested by Freeman & Wyke (1967*b*), that the joint receptors may contribute to the 'co-ordination of muscle tone in posture and

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movement' via the γ-loop. It is furthermore suggested that the latter mechanism may serve to regulate joint stiffness and joint stability.

INTRODUCTION

The characteristics of joint receptors, and their role for position sense, movement sense and motor control, have been extensively studied and discussed over the years (for reviews, see e.g. Goodwin, 1976; McCloskey, 1978; Matthews, 1982). However, it is still a matter of debate whether joint receptors are of any importance at all in normal motor acts.

Early studies on the effects from joint afferents on α-motoneurones seemed to indicate that the skeletomotoneurones were influenced by activity in high-threshold joint afferents, but not by activity in low-threshold joint afferents (cf. Eccles & Lundberg, 1959; Holmqvist, 1961; Holmqvist & Lundberg, 1961). However, later on, Hongo, Jankowska & Lundberg (1969) observed that rubral facilitation could disclose excitatory as well as inhibitory effects from low-threshold joint afferents on both flexor and extensor α-motoneurones. A more recent study (Lundberg, Malmgren & Schomburg, 1978) then revealed that, in low spinal cats, stimulation of low-threshold ($< 2{\cdot}0$ times the nerve threshold) fibres in the posterior articular nerve of the knee joint (p.a.n.) evoked synaptic potentials in hind-limb α-motoneurones in one-third of the animals. In addition, it has been found that joint afferents can influence α-motoneurones through various different pathways, such as via the flexion reflex afferent paths (cf. Eccles & Lundberg, 1959; Holmqvist, 1961; Holmqvist & Lundberg, 1961), via the Ia interneurones (Fedina & Hultborn, 1972) and via the Ib pathways (Lundberg *et al.* 1978).

Ekholm, Eklund & Skoglund (1960) and Freeman & Wyke (1967*b*) were first to suggest that joint afferents participate in the reflex control of γ-motoneurones. So far, however, only a few investigations have been made of the influence of joint afferents on fusimotor neurones. Grillner, Hongo & Lund (1969) made micro-electrode recordings from lumbar γ-cells and observed that these cells could be excited as well as inhibited from p.a.n. and that 'the reflex effects from the knee joint nerve conforms to the reflex pattern evoked from the other peripheral nerves'. McIntyre, Proske & Tracey (1978*b*), in an investigation with electrical stimulation of p.a.n. and recording from primary muscle spindle afferents, concluded that joint receptor afferents have effects (almost exclusively excitatory) mainly on static fusimotor neurones, that the effects are similar to those seen in response to stimulation of the sural nerve, and that the responses are largely mediated by the high-threshold joint afferents. Furthermore, it has been shown that contralateral knee joint receptors, activated by pressure on the knee joint capsule, can influence dynamic fusimotor neurones projecting to the ipsilateral triceps surae muscle (Appelberg, Hulliger, Johansson & Sojka, 1979, 1981).

On account of the sparsity of information concerning the action of joint afferents on γ-motoneurones, we deemed it important to examine the effects of graded electrical stimulation of the p.a.n. on a population of static and dynamic γ-motoneurones in which the responses to stimulation of other afferents (muscle and skin afferents) were also investigated. Thus, the results presented in this article were obtained from a subsample ($n = 71$) of a larger sample ($n = 120$) of γ-cells which were

studied with intra- or extracellular recordings during graded electrical stimulation of central structures (nucleus ruber and the mesencephalic area for dynamic control), of muscle and skin nerves and/or of ventral roots (Johansson, 1981; Appelberg, Hulliger, Johansson & Sojka, 1982, 1983*a*, *b*, *c*, *d*; Johansson & Sojka, 1985; Sojka, 1985).

METHODS

This paper belongs to a series of reports concerned with central, recurrent and reflex control of hind-limb γ-motoneurones of α-chloralose anaesthetized cats (see also Johansson, 1981; Appelberg *et al.* 1982, 1983*a*, *b*, *c*, *d*; Johansson & Sojka, 1985; Sojka, 1985). In all experiments, a number of ipsilateral hind-limb nerves (supplying different skin areas, muscles and the knee joint) were prepared for electrical stimulation. During graded electrical stimulation of the nerves, microelectrode single-unit recordings (intra-, juxta- or extracellular) were made from lumbar γ-motoneurones. Each cell was identified as a γ-motoneurone when antidromic all-or-nothing potentials, with fixed latency, could be elicited by stimulation of one particular muscle nerve, and when the conduction velocity of the cell axon was below 55 m/s (for a detailed account of the conduction velocities of the γ-cells in this investigation see Appelberg *et al.* 1982). Most of the γ-cells were also classified indirectly as either static or dynamic using the method of mesencephalic stimulation (see review by Appelberg, 1981; cf. also Jeneskog & Johansson, 1977; Appelberg *et al.* 1982). The cell responses and the incoming volleys, at the dorsal root entry zone, were recorded in parallel and stored on magnetic tape for analysis later on. When the p.a.n. was stimulated, the cord dorsum potential was recorded instead of the incoming volley (cf. below).

A full account of the general methods used in the present series of experiments has been given in a previous paper (Appelberg *et al.* 1983*a*). Therefore this section is confined to specific aspects related to the preparation and electrical stimulation of the p.a.n. nerve (twenty-one animals) and to the estimation of the latencies and the thresholds of the evoked reflex effects.

Preparation of the p.a.n. The p.a.n. nerve was dissected free over a sufficient length and cut distally, so that its central end was accessible for electrical stimulation (cf. Skoglund, 1956).

Stimulation. As a standard routine, single-shock stimulation at 1–2/s was employed. The intensities of the stimulations were always graded in multiples of the stimulation strength (1·0 *T* in this investigation) at which an early negative cord dorsum potential (cf. Carpenter, Engberg, Funkenstein & Lundberg, 1963; see also Holmqvist, 1961; Lundberg *et al.* 1978), recorded by a monopolar silver ball electrode on the dorsal root entry zone, appeared. The threshold intensity for the nerve was frequently checked and, if needed, readjusted. The cells were tested with a number (1–10) of different stimulation intensities. For determination of the thresholds for the responses the intensities were increased by steps of 0·05–0·10 *T*. On completion of two of the experiments, the stimulation strength threshold for the appearance of the negative cord dorsum potential (i.e. 1·0 *T*) was correlated with the stimulation intensity needed to activate the most excitable fibres in p.a.n., determined with recording from the sciatic nerve about 5 cm from the stimulating electrode.

Response latencies. The data processing and analysis was performed off-line by playing back the stored cell responses and cord dorsum potentials from magnetic tape, superimposing them on a cathode ray oscilloscope display and plotting their averages with the aid of an averaging computer, a desk-top calculator and an *X*–*Y* plotter (for details see Appelberg *et al.* 1983*a*). The occurrence of excitatory and inhibitory effects was assessed on the basis of conventional criteria. All measurements were made on the averaged records, but they were always checked by comparison with a superimposed cathode ray oscilloscope display of the responses played back from tape. Thus, the latencies were measured from a series of responses (averaged) at each stimulation strength (records with well developed responses were always used). First the latency from the stimulus artifact was measured. From this value the peripheral conduction delay (from the stimulating electrode to the recording electrode at the dorsal root entry zone), for the fastest conducting joint nerve afferents, was then subtracted (assumed conduction velocity for these fibres 90 m/s, cf. Lundberg *et al.* 1978; see also Boyd & Davey, 1968; Burgess & Clark, 1969; Ferrell, 1980). Thus each latency given in this paper represents the time from the presumed arrival of the activity in

the fastest joint afferent fibres at the site of the dorsal root entry zone electrode to the start of the actual response. Latencies could not be reliably estimated for all observed effects (e.g. for extracellularly recorded excitations in cells with high spontaneous activity, for extracellularly recorded inhibitions or for post-synaptic potential (p.s.p.) recordings of unsatisfactory quality).

Abbreviations used in the text. A.b.s.m., anterior biceps and semimembranosus (nerves or muscles); d.p., deep peroneal nerve (pure muscle nerve supplying extensor digitorum longus and tibialis anterior); g.s., gastrocnemius-soleus (triceps) (nerve or muscle); p.b.s.t., posterior biceps and semitendinosus (nerves or muscles); *T*, threshold; tib., tibial nerve.

RESULTS

The results presented here were obtained during a series of experiments in which a total sample of 120 γ-motoneurones was investigated by intra-, juxta- and extracellular recordings during electrical stimulation of central structures, of peripheral muscle, skin and joint nerves and/or of ventral roots (Johansson, 1981; Appelberg *et al.* 1982, 1983*a*, *b*, *c*, *d*; Johansson & Sojka, 1985; Sojka, 1985). A subsample of seventy-one cells were investigated for reflex effects elicited by graded electrical stimulation of the p.a.n. Thirty-one of these were studied with intracellular, eighteen with juxtacellular and twenty-two with extracellular recording. The γ-cells, which were tested with mesencephalic stimulation, were thus indirectly classified as dynamic or static, depending upon whether they were influenced or not. Only four of the cells could not be classified. The γ-cells of the sample tested with stimulation of p.a.n. projected to the p.b.s.t. (thirteen dynamic, ten static and one non-classified), g.s. (twenty dynamic, twelve static and one non-classified), a.b.s.m. (one dynamic, six static and one non-classified), tib. (one dynamic and one static) and d.p. (two dynamic, one static and one non-classified) muscles. Except for the fact that the shortest latencies were found only among the dynamic units, virtually no statistically significant differences (i.e. with respect to types of effect, stimulation strength thresholds for the effects) were found between the populations of static and dynamic γ-cells of this sample.

Responsiveness and types of effects

The stimulation of p.a.n. was very effective in eliciting reflex responses in the γ-motoneurones of the present sample. Thus, out of the seventy-one cells tested with graded stimulation up to at least 10 *T* only seven (9·9 %) failed to respond. The over-all responsiveness figures (number of cells with effect/number of cells tested) were 91·9 % for the dynamic and 93·3 % for the static cells. Among the four non-classified cells, two were unresponsive to p.a.n. stimulation.

All types of reflex responses were observed, i.e. excitatory and inhibitory as well as blends of excitation and inhibition. Whenever mixed effects were encountered, the excitation preceded the inhibition. Examples of p.a.n.-mediated reflex responses obtained with intra- and extracellular recordings from different types of γ-motoneurones are shown in Fig. 1. In this Figure the cell types are given to the left of the diagrams showing the antidromic identification of the cells (*Aa*, *Ba*, *Ca* and *Da*). In the remaining diagrams the cell responses to single-shock stimulation of p.a.n. at different intensities (upper traces, marked c) and the cord dorsum potentials, recorded from the L7 dorsal root entry (lower traces, marked v), are displayed as

superimposed sweeps. The uppermost set of traces (Fig. 1 *Ab* and *Ac*) shows inhibitory action on a dynamic p.b.s.t. γ-cell (identified in *Aa*) observed in an extracellular recording. Stimulation of p.a.n. with 5 *T* gave a silent period in the resting discharge of the cell (Fig. 1 *Ac*: twenty superimposed sweeps; cf. also Fig. 1 *Ab*: twenty superimposed sweeps in absence of any peripheral nerve stimulation). Fig. 1 *Bb* and *Bc* show intracellularly recorded excitation of a dynamic p.b.s.t. γ-motoneurone (identified in *Ba*). A just-liminal excitation post-synaptic potential (e.p.s.p.) appeared at 2 *T* (see *Bb*). This e.p.s.p. increased as the stimulation intensity was raised to 5 *T* (see *Bc*). Also in extensor γ-motoneurones excitatory as well as inhibitory responses were observed, with stimulation of p.a.n. afferents. Examples of this are given for two static a.b.s.m. γ-cells (Fig. 1 *Ca–d* and *Da–h*). In the intracellularly recorded static a.b.s.m. γ-cell, illustrated in *Ca–d* (identified in *Ca*), a just-liminal e.p.s.p. was evoked at 1·4 *T*, and the e.p.s.p. grew as the stimulation intensity was raised to 10 *T*. With the lower-most cell of Fig. 1 (*Da–h*) a small low-threshold inhibitory post-synaptic potential (i.p.s.p.) was detected at 1·0 *T*. This did not grow much up to 1·3 *T* (see *Db–d*). However, when the stimulation intensity was raised to 1·5–1·6 *T* a second, much bigger, i.p.s.p. component was added (see *De–h*).

Stimulation strength thresholds for the effects

The stimulation strengths needed for eliciting effects in the γ-cells from p.a.n. were settled for forty units. Very low effect thresholds were found for all subpopulations of cells (dynamic and static, flexor and extensor cells), and for inhibitions as well as for excitations. However, the number of inhibitory effects on static flexor cells was small, and therefore no reliable thresholds could be settled for these effects. Thus, except for inhibition of static γ-cells, the lowest thresholds (in multiples of the stimulation intensity needed to evoke an early negative cord dorsum potential) observed were 0·9–1·1 *T*. Moreover, low-threshold effects (i.e. effects elicited at intensities below 1·6 *T*) were observed in 70 % of the cells and in 80 % of the animals of the present investigation.

The mean stimulation strengths needed for provoking excitatory (mean values for all cells: 1·4 $T \pm 0{\cdot}3$ S.D., $n = 29$; range: 0·9–2·0 *T*) and inhibitory (mean values for all cells: 1·4 $T \pm 0{\cdot}6$ S.D., $n = 11$; range: 0·9–2·5 *T*) responses were very similar, and no statistically significant threshold differences were found between excitation of dynamic cells and excitation of static cells. However, a Mann–Whitney analysis revealed that the thresholds for inhibition of static cells were significantly lower than the corresponding thresholds for dynamic cells ($P = 0{\cdot}036$, two-tailed test). It should be remarked, though, that when the a.b.s.m. cells were excluded, no such statistical difference could be established between the thresholds for inhibitory effects in dynamic and static cells. The sample of a.b.s.m. cells introduced a considerable bias into the material since it consisted of six intracellularly recorded static units and one single (extracellularly recorded) dynamic unit. Furthermore, the static a.b.s.m. cells are remarkable in that they seem to be recipients preferentially of inhibitory effects from all input sources (cf. Appelberg *et al.* 1983*c*; Johansson & Sojka, 1985).

The way in which the responses grew or changed character with increasing stimulation strengths varied a lot between the individual cells. However, on

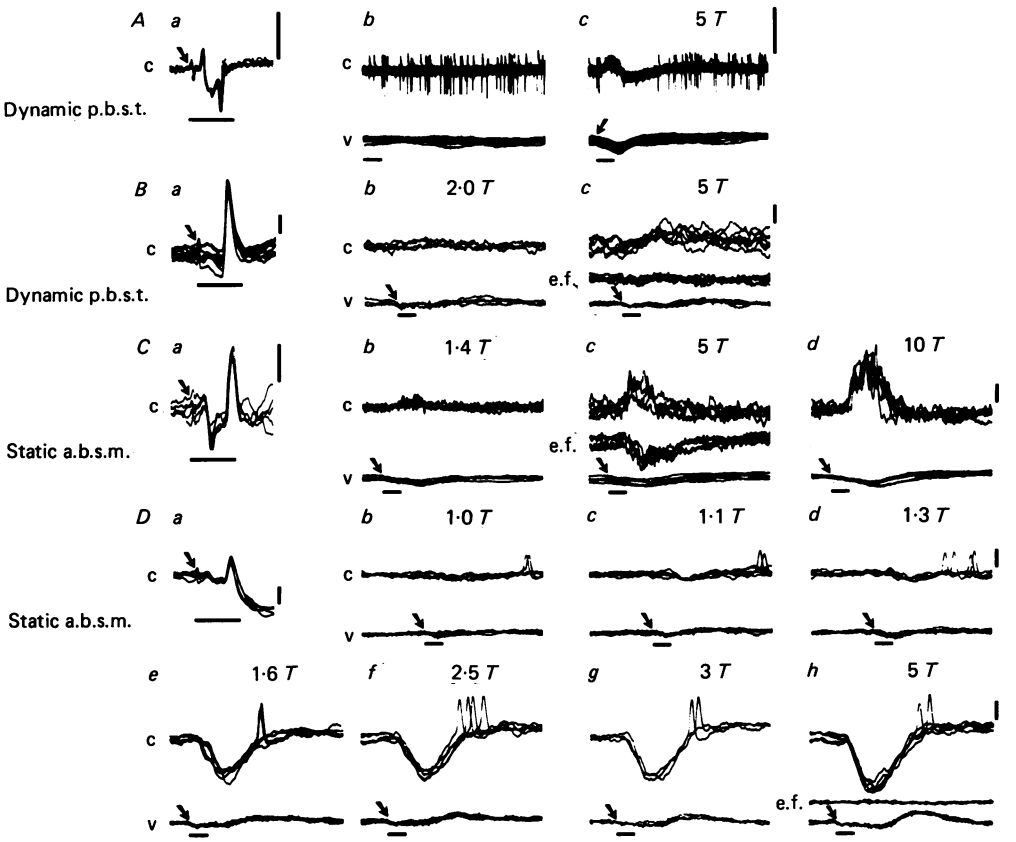

Fig. 1. Intra- and extracellularly recorded responses of static and dynamic flexor and extensor γ-motoneurones to electrical stimulation of the posterior articular nerve of the knee joint (p.a.n.). The stimulation intensities are given above each diagram (expressed as multiples of the stimulation intensity at which an early negative cord dorsum potential appeared, *T*). In each diagram, a number (three to seven, with the exception of *Ab* and *Ac*, in which twenty superimposed traces are shown) of consecutive responses to single-shock stimulations are displayed as superimposed sweeps. Upper traces marked c are cell recordings, lower traces marked v cord dorsum potentials. Extracellular field potentials (e.f.) recorded after withdrawal of the micro-electrode are shown only once for each intracellularly recorded cell (*Bc*, *Cc* and *Dh* respectively) as they are small and thereby clearly indicate that the intracellularly recorded potentials are not their reflexions. The cell types are given in the lower left-hand quadrant of the diagrams showing the antidromic identification of the cells (*Aa*, *Ba*, *Ca* and *Da*). The arrows in these diagrams point at the stimulus artifacts, while the arrows in the other diagrams mark the presumed arrival of the activity in the fastest conducting joint afferents (90 m/s) at the site of the cord dorsum electrode (on the dorsal root entry zone). *Aa–c*, extracellularly recorded responses of a dynamic p.b.s.t. γ-cell, antidromically identified in *Aa* (conduction velocity 36 m/s). In *Ac*, decrease in (inhibition of) spontaneous activity following electrical stimulation of p.a.n. with 5 *T*. For comparison the resting discharge (absence of any peripheral stimulation) of the cell is shown in *Ab*. *Ba–c*, intracellularly recorded responses of a dynamic p.b.s.t. γ-cell (membrane potential > 20 mV). *Ba*, antidromic identification (conduction velocity 39 m/s). A just-liminal e.p.s.p. appeared at 2 *T* (*Bb*), and this e.p.s.p. increased when the stimulation was raised to 5 *T* (*Bc*). Segmental latency: 5·9 ms. *Ca–d*,

occasions, when responses to p.a.n. stimulation developed between low (0·9–1·5 *T*) and high (1·6–5 *T*) stimulation intensities, more than one component could be discerned. As with the cell of Fig. 1 (*Da–h*), a low-threshold component was often detected (Fig. 1 *Db–d*), and sometimes also a high-threshold component (cf. Fig. 1 *De–h*) could be observed. For mixed effects (blends of excitation and inhibition), the excitation always appeared at lower stimulation intensities (and with shorter latencies) than the inhibitory effect.

In two experiments, the stimulation intensity needed to evoke the early negative cord dorsum potential was compared to the stimulation strength threshold for the compound action potential, recorded from the sciatic nerve. It was found that the cord dorsum potential appeared at 1·2 and 1·3 times the threshold for the most excitable fibres respectively. This is in agreement with the observations of Carpenter *et al.* (1963) (cf. also Lundberg *et al.* 1978).

Excitatory and inhibitory actions on flexor and extensor γ-cells

Fig. 2 shows the general features of the reflex actions exerted by activity in p.a.n. fibres in the two largest populations of classified γ-motoneurones, namely those projecting to the p.b.s.t. (thirteen dynamic, ten static) and g.s. (twenty dynamic, twelve static) muscles. The frequency histograms illustrate, separately for dynamic (left columns, marked D) and static (right columns, marked S) cells, the incidence of excitation (triangle pattern, plotted upwards), of inhibition (circle pattern, plotted downwards) and of tests in which stimulation of p.a.n. gave no effect (filled columns, plotted upwards in the lower part of the diagram). Mixed effects (blends of excitation and inhibition, one triangle and one circle) are presented in the mid-part of the diagram. In the Figure intra-, juxta- and extracellular observations are lumped together. The occurrencies of excitations, inhibitions, mixed effects and negative observations are expressed as percentage of the total number of observations for all classified p.b.s.t. cells and for all classified g.s. cells respectively. (The term 'observation' means, in this context, the definite assessment of all tests made on particular γ-cell with graded stimulation of p.a.n.) The total numbers of observations within the two populations of cells (i.e. 100%) are given above the two pairs of columns.

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intracellularly recorded responses of a static a.b.s.m. γ-cell (membrane potential = 20 mV). *Ca*, antidromic identification (conduction velocity 28 m/s; damaged spike mechanism). In *Cb*, threshold for the e.p.s.p. at 1·4 *T*. *Cc* and *Cd*, increase of the e.p.s.p. up to 10 *T*. Note that, due to the fact that the cell had a relatively low membrane potential, there is reason to believe that the observed effect threshold is too high. Segmental latency: 4·3 ms. *Da–h*, intracellularly recorded responses of a static a.b.s.m. γ-cell (membrane potential = 35 mV). In *Db–d*, an i.p.s.p. was discernible at 1·0 *T* (*Db*) which grew very little up to 1·3 *T* (*Dd*). In *De–h*, at 1·5–1·6 *T* (*De*) a second large component was added, which increased further (possibly a third component) between 3 and 5 *T* (compare *Dg* and *Dh*). Segmental latency: 3·5 ms. Horizontal calibrations (given below each diagram): bars in *Aa* and *Ba–Dh* = 5 ms; bars in *Ab* and *Ac* = 10 ms. Vertical calibrations (bars applicable to the antidromic identifications are given to the right of these diagrams, bars applicable to the other recordings are given to the right of each row): bars in *Aa–Ca* = 1 mV; bars in *Cb–Dh* = 2 mV.

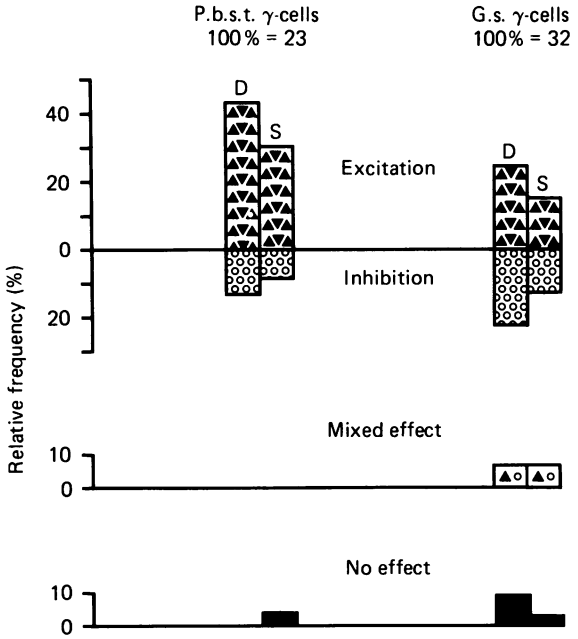

Fig. 2. Relative frequency histograms of reflex actions from the posterior articular nerve of the knee joint on dynamic and static flexor (p.b.s.t. γ-cells) and extensor (g.s. γ-cells) γ-motoneurones. The effects on dynamic (D, left-hand columns) and static (S, right-hand columns) cells are plotted separately. Intra-, juxta- and extracellularly recorded responses are lumped together. The relative frequencies are expressed as percentage of the total number of cells (effects) within the p.b.s.t. population (the two columns to the left: 100 % = 23) and the g.s. population (the two columns to the right: 100 % = 32) respectively (for further details, see text).

P.b.s.t. and g.s. γ-motoneurones. It may be seen from Fig. 2 (based on recordings from twenty-three p.b.s.t. and thirty-two g.s. cells) that reflex actions evoked by activity in p.a.n. afferents were very frequent in both p.b.s.t. and g.s. γ-motoneurones. Effects of the mixed type were seen only in four cells (two dynamic and two static g.s. cells). It should be pointed out that hyperpolarization of the cell membranes was not systematically performed in order to elucidate if there were admixtures of i.p.s.p.s to e.p.s.p.s or vice versa. Thus, admixtures of such small effects to what we have called 'pure excitations' and 'pure inhibitions' cannot be ruled out.

Another conspicuous feature was the clear difference between the effects on flexor and extensor cells. Among flexor (p.b.s.t.) γ-cells, irrespective of whether they were dynamic or static, excitation was by far predominant. Pure excitation occurred in seventeen out of twenty-three p.b.s.t. γ-cells, while only five were inhibited (one cell was uninfluenced). Among the extensor (g.s.) γ-cells, both dynamic and static, excitation outweighed inhibition only by a small margin. Thus, thirteen out of thirty-two g.s. γ-cells received pure excitation, eleven pure inhibition and four received mixed effects (four cells were uninfluenced). No significant difference, in the balance between excitatory and inhibitory responses, of dynamic and static cells was found in this sample of cells.

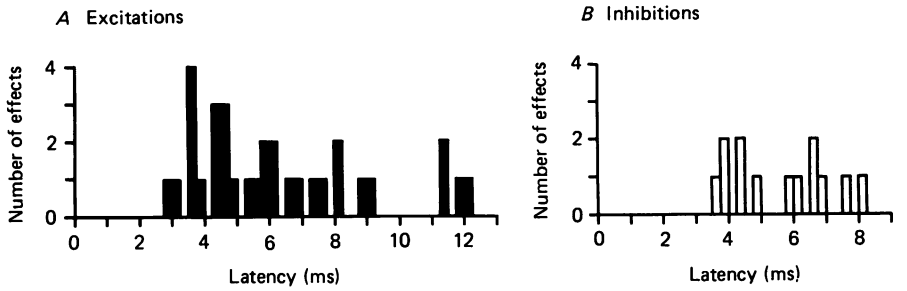

Fig. 3. Latency distributions of excitatory (*A*) and inhibitory (*B*) responses of dynamic, static and non-classified γ-motoneurones, evoked by electrical stimulation of the posterior articular nerve of the knee joint. Each value corresponds to the shortest latency of i.p.s.p.s, e.p.s.p.s or stimulus-locked spike discharges found for an individual cell with a series of tests (up to fifteen) with graded stimulation of the nerve. Only reliably estimated latencies were included. Effects on dynamic, static and non-classified cells are lumped together (for further details, see text). In *A*, latencies for thirty-two excitatory effects. The mean values of excitation latencies were 6·2 ms ± 2·7 S.D. (dynamic cells: 6·1 ms ± 2·9 S.D., $n = 20$; static cells: 6·6 ms ± 2·3 S.D., $n = 11$). In *B*, latencies for thirteen inhibitory effects. The mean values of inhibition latencies were 5·6 ms ± 1·5 S.D. (dynamic cells: 6·4 ms ± 1·2 S.D., $n = 5$; static cells: 4·5 ms ± 1·5 S.D., $n = 7$).

The pooling of intra-, juxta- and extracellular responses in Fig. 2 was undertaken in order to get reasonably large samples. The samples of intracellularly recorded units were too small (six dynamic p.b.s.t., five static p.b.s.t., six dynamic g.s. and seven static g.s. units) to safely draw general conclusions from. However, roughly the same balance between excitatory and inhibitory effects as described above (Fig. 2) could be discerned in these populations.

Other populations of γ-cells. Among six intracellularly recorded static a.b.s.m. γ-motoneurones, excitatory responses were recorded in two cells (cf. Fig. 1*Ca–d*), while four received pure inhibition (cf. Fig. 1*Da–h*). Three of the latter four cells were also inhibited from nearly all effective group II and group III muscle nerve inputs and from all tested skin nerves (cf. Appelberg *et al.* 1983*b*, *c*; Johansson & Sojka, 1985), and in addition two of these (an unexpectedly high proportion) were inhibited by muscle group I stimulation (cf. Appelberg *et al.* 1983*a*). The single dynamic a.b.s.m. γ-cell recorded from was excited from p.a.n. Two tib. γ-cells were tested with p.a.n. stimulation, and they were both massively excited. These two tib. cells were excited from all effective muscle group II, muscle group III and skin inputs as well (cf. Appelberg *et al.* 1983*b*, *c*). Among the three d.p. γ-cells, one dynamic unit was excited and another was inhibited by stimulation of p.a.n. The single static d.p. γ-cell recorded from received inhibition via p.a.n. afferents.

Segmental delay of excitatory and inhibitory effects

Fig. 3 displays the distribution of segmental latencies of excitations (Fig. 3*A*, 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 all (dynamic, static and non-classified) γ-motoneurones projecting to the p.b.s.t., g.s., a.b.s.m., tib. and d.p. muscles. The latencies were always estimated as the time from the presumed arrival

of the activity, in the most rapidly conducting p.a.n. fibres, at the dorsal root entry zone, to the start of the cell response. Thus, the latency from the stimulus artifact was first measured. The segmental latency was then obtained by subtracting from this figure the peripheral conduction delay (from the stimulating electrode to the dorsal root entry zone) for the fastest conducting joint afferents (assumed conduction velocity for these fibres 90 m/s). For a certain effect on a particular γ-cell, a number of latencies were always measured, at different stimulation intensities. Among these, the shortest reliable one was selected and regarded as the true latency for the effect in question. For extracellularly recorded responses it was sometimes impossible to determine a reliable latency value (see Methods).

In total, thirty-two reliable latency values for excitatory effects and thirteen values for inhibitory effects could be determined. Comparisons between dynamic and static γ-cells, flexor and extensor γ-cells or, for that matter, other subpopulations of cells (such as dynamic and static cells projecting to particular muscles) revealed no statistically significant differences, as regards excitatory effects. However, the shortest latencies of excitatory effects were found for the dynamic γ-cells (four values below 4 ms; $n = 20$; range: 2·8–12·0 ms). Only one value below 4 ms was detected for static γ-cells ($n = 11$; range: 3·7–11·4 ms). Thus, the fastest route from p.a.n. afferents to dynamic γ-motoneurones seems to be shorter than the one to static cells, although the sample of latencies was too small to settle the issue. It should be remarked that the shortest latencies frequently were observed at stimulation intensities above the thresholds for the effects and hence also above the threshold for the negative cord dorsum potential.

As can be seen from the distribution, in Fig. 3, of all reliably estimated latencies of responses caused by activity in p.a.n. afferents, the fastest excitatory pathways (2·8 ms; cf. Fig. 3*A*) to γ-motoneurones seem to be shorter than the fastest inhibitory (3·5 ms; cf. Fig 3*B*) pathways. However, no statistically significant difference could be established between the total samples of excitatory and inhibitory effects elicited from p.a.n.

A Mann–Whitney test (two-tailed test) revealed a significant ($P = 0{\cdot}037$) difference between the latencies of inhibitory effects in dynamic (mean: 6·4 ms $\pm$ 1·2 S.D., $n = 5$; range: 4·8–8·0 ms) and static (mean: 4·9 ms $\pm$ 1·5 S.D., $n = 7$; range: 3·5–7·6 ms) cells respectively. However, three of the shortest latencies of inhibitory effects were obtained from static a.b.s.m. cells. When the a.b.s.m. cells were excluded, no such difference could be established between the latencies of inhibitory effects in dynamic and static γ-cells.

Comparison with effects evoked from muscle and skin nerves

As with effects elicited from muscle and skin nerves, there were considerable differences, with respect to thresholds and latencies, between the individial γ-motoneurones within a single subpopulation of cells (dynamic and static, flexor and extensor cells). Also, for any individual unit, the type (excitation and/or inhibition), the threshold or the latency of the effect elicited from p.a.n. could not be predicted either from the allocation of the unit to a certain population or from the character of the effects evoked from any other input source (i.e. different classes of afferents in any one of the muscle or skin nerves used as inputs).

DISCUSSION

This paper is based on a series of experiments which was undertaken in order to assess the relative contribution from activity in muscle, skin and joint afferents to the segmental control of γ-motoneurones. A number of reports from our laboratory on the effects, in a large sample of intra- and extracellularly recorded lumbar γ-motoneurones, mediated via muscle, skin and ventral root fibres have already been published (Johansson, 1981; Appelberg *et al.* 1982, 1983*a*, *b*, *c*, *d*; Johansson, 1985; Johansson & Sojka, 1985; Sojka, 1985). The present paper deals with the responses elicited by electrical stimulation of the p.a.n. on the same population of cells.

Reflexes from knee joint afferents (p.a.n.)

Responsiveness to stimulation of the p.a.n. The vast majority of the γ-cells of the present study were readily influenced by electrical stimulation of the p.a.n. (only 9·9 % negative observations), and static and dynamic cells seemed to be equally responsive. Thus, the general responsiveness to joint nerve stimulation was higher than that observed for stimulation of group II and III muscle afferents (14·1 % negative observations; cf. Appelberg *et al.* 1983*c*; Johansson & Sojka, 1985) but slightly lower than the responsiveness to skin nerve stimulation (5·2 % negative observations; cf. Johansson & Sojka, 1985).

Thresholds for the effects. In this investigation the thresholds for the effects were related to the stimulation intensity at which the early negative cord dorsum potential appeared (cf. Holmqvist, 1961; Carpenter *et al.* 1963; Lundberg *et al.* 1978). Carpenter *et al.* (1963) found that this early negative component was discernible at 1·21 times the threshold for the most excitable fibres in p.a.n. (cf. also Lundberg *et al.* 1978). During the present series of experiments, the stimulation strength, at which the early negative component of the cord dorsum potential appeared, was compared to the threshold for the compound action potential in the sciatic nerve on two occasions. The two figures obtained were 1·2 and 1·3 times the nerve threshold. Hence it may be assumed that, in order to obtain the stimulation strengths expressed in multiples of the threshold for the most excitable p.a.n. afferents, a stimulation intensity of XT in the present investigation should be multiplied with a factor of 1·2–1·3.

If the last assumption is correct, then the majority of the cells of the present investigation were influenced at low stimulation intensities. For all subpopulations of cells (dynamic and static, flexor and extensor cells) excitatory as well as inhibitory effects were observed at stimulation strengths of 0·9–1·1 T, which most likely corresponds to 1·1–1·4 times the threshold for evoking of a compound action potential in p.a.n. Accordingly, the mean thresholds for excitatory and inhibitory effects found in the present investigation probably are equivalent to about 1·70 times the nerve threshold. The finding of such low effect thresholds clearly is at variance with the results presented by McIntyre *et al.* (1978*b*). These authors, in a study with electrical stimulation of p.a.n. and indirect evaluation of the fusimotor activity (by recording from primary muscle spindle afferents), found no effects with stimuli below twice the threshold for the nerve volley.

Interestingly, no significant difference in stimulation strengths needed for eliciting responses (excitatory or inhibitory) in dynamic and static γ-cells was found in this

sample of cells. This is in contrast to the study on effects from cutaneous afferents (Johansson & Sojka, 1985), in which the dynamic cells were found to be excited at lower thresholds than the static units.

In addition to the low-threshold effects, a considerable number of responses were found which were elicited at stimulation intensities above 1·6 times the threshold for the most excitable p.a.n. afferents (cf. Clark, Landgren & Silfvenius, 1973). Furthermore, a second component was often added to the low-threshold effects at higher stimulation intensities. It thus seems likely that all subpopulations of static and dynamic γ-motoneurones (projecting to different hind-limb muscles) contain some cells which are influenced only from low-threshold p.a.n. afferents, some cells which are influenced only from high-threshold p.a.n. afferents and also some cells which are influenced from both low- and high-threshold afferents. Does this reflect selective activation by different receptor categories? Although there is a considerable overlap between afferents from different receptor types, studies in which fibre diameters and fibre conduction velocities have been related to the different receptor categories (see e.g. Skoglund, 1956; Freeman & Wyke, 1967*a*; Wyke, 1967; Boyd & Davey, 1968; Burgess & Clark, 1969; Ferrell, 1980; cf. also Clark *et al.* 1973), seem to indicate that the afferents with lowest thresholds are fibres from Golgi-type endings and Paciniform corpuscles and that the fibres from Ruffini endings and free nerve endings have higher thresholds. However, it should be pointed out that in about half of the animals (McIntyre, Proske & Tracey, 1978*a*) some popliteus muscle afferents have their course in the p.a.n. (cf. also Skoglund, 1956; Boyd & Davey, 1968; Burgess & Clark, 1969; Lindström & Takata, 1972).

In α-motoneurones, Eccles & Lundberg (1959) found no low-threshold effects from p.a.n., but in a more recent investigation Lundberg *et al.* (1978) state that weak stimulation of p.a.n. (below twice the nerve threshold) evoked p.s.p.s in α-motoneurones in one-third of the cats used in their investigation. In the present study, low-threshold effects (effects elicited at intensities below 1·6 times the threshold for evoking of the early negative cord dorsum potential, roughly corresponding to twice the nerve threshold) were seen in 80% of the animals and in 70% of the neurones in which thresholds could be settled. Thus we tentatively suggest that low-threshold joint afferents act more potently on γ- than on α-motoneurones.

General pattern of effects. Grillner *et al.* (1969) gave examples of both excitatory and inhibitory effects in γ-motoneurones, caused by electrical stimulation of p.a.n. On the other hand McIntyre *et al.* (1978*b*) observed no inhibitory effects at all on the fusimotor neurones studied in their investigation (supplying the medial gastrocnemius, soleus, semitendinosus, tibialis anterior or flexor digitorum longus muscles). Moreover, they also concluded, on indirect evidence, that the effects seen in their study were mainly on static fusimotor neurones. In contrast, the present study clearly demonstrates that stimulation of p.a.n. afferents can elicit excitatory as well as inhibitory responses in both dynamic and static flexor and extensor γ-motoneurones.

In γ-motoneurones projecting to p.b.s.t. (flexor muscles), irrespective of whether they were dynamic or static, the stimulation of p.a.n. afferents mainly provoked excitatory effects, which clearly outweighed inhibitory actions. Among g.s. (extensor) γ-cells, also irrespective of whether they were dynamic or static, there was an about even balance between excitation and inhibition, with a slight predominance for

excitation. Thus the general balance between excitation and inhibition of flexor γ-motoneurones (dynamic as well as static) in our sample conforms with the pattern observed for flexor α-motoneurones in spinal preparations with stimulation of p.a.n. afferents (Lundberg *et al.* 1978). However, the clear-cut prevalence for inhibitory effects from p.a.n. on extensor α-motoneurones (cf. Lundberg *et al.* 1978) contrasts with the even blend of excitatory and inhibitory effects on extensor γ-cells.

Latencies and synaptic coupling. In the present investigation the shortest excitatory latencies (2·8 ms) were found for dynamic cells. The fastest excitatory responses in static cells were roughly 1 ms later. However, the samples of latencies were too small to reveal possible differences between the populations of latency values for dynamic and for static cells.

In our attempts to estimate the segmental delays we have assumed that the effects were transmitted via the most rapidly conducting p.a.n. fibres (assumed conduction velocity 90 m/s). However, two lines of evidence suggest that this leads to an over-estimation of the figures for the shortest segmental delays. First, few fibres in p.a.n. conduct at such high velocities (cf. Boyd & Davey, 1968; Burgess & Clark, 1969; Ferrell, 1980). Secondly, the shortest latencies were found for stimulus intensities above the threshold for the negative cord dorsum potential (and mostly also above the threshold for the effect; often at 2–5 *T*). Therefore we conclude that the earliest components of the effects with the shortest latencies were usually not caused by the fastest afferents. Thus, it seems likely that the fastest excitatory pathway to dynamic γ-motoneurones is trisynaptic (cf. Lundberg *et al.* 1978), or perhaps even disynaptic (cf. Harrison & Jankowska, 1985), while the shortest route to the static cells probably is longer.

The sample of reliable latencies of inhibitory effects was quite small (twelve values), but the smallest values obtained indicate a synaptic linkage comparable to the one inferred for the excitatory pathway to static γ-cells. Statistically, the population of inhibitory latencies on static units was found to differ significantly from the population of inhibitory latencies on dynamic cells, but this difference might be explained by a sample bias (over-representation of static a.b.s.m. cells).

Information concerning latencies of effects elicited via p.a.n. afferents in α-motoneurones is scarce, but, according to Lundberg *et al.* (1978), the excitatory effects are likely to be trisynaptically mediated. It is thus possible that, in this particular respect, the dynamic γ-cells are more similar to the α-motoneurones than are the static γ-cells.

Comparison with effects evoked from muscle and skin nerves

The analysis of the reflex effects on γ-motoneurones of muscle and skin nerve stimulation (Johansson, 1981; Appelberg *et al.* 1983*a*, *b*, *c*, *d*; Johansson, 1985; Johansson & Sojka, 1985; Sojka, 1985) revealed that, behind the general features of the reflex effects, there was concealed a great amount of variation in effects on the individual γ-cells. This was the case also for the effects from p.a.n. Furthermore, for any individual unit, the type and characteristics of the effect evoked from p.a.n. could be predicted neither from the allocation of the unit to a certain subpopulation of γ-cells nor from the effects elicited by any other input. This is in contrast to the findings of Grillner *et al.* (1969), who stated that the effects from p.a.n. 'conforms to the reflex

pattern evoked from the other peripheral nerves', and to the findings reported by McIntyre *et al.* (1978*b*) who reported that 'if stimulation of the sural nerve had an effect, then stimulation of p.a.n. or the interosseus nerve has a similar but weaker action'. The results of the present study seem further to strengthen the view that the receptive profiles of γ-motoneurones are different from and more individualized than those of α-motoneurones (Johansson, 1981; Appelberg *et al.* 1983*c*). It is of interest in this context that evidence recently has been presented for ultrastructural differences between synapses on α- and γ-motoneurones (Johnson, 1985).

Possible functional significance

The present study clearly demonstrates that activity in fibres running in the p.a.n. of the knee joint causes excitatory and/or inhibitory post-synaptic effects on static and dynamic γ-motoneurones projecting to flexor and extensor muscles. Accordingly, if primary muscle spindle afferents contribute to movement and position sense (cf. Goodwin, McCloskey & Matthews, 1972), then joint afferents are also likely to contribute (see Johansson, 1981). Moreover, there is also evidence that joint receptors in the contralateral hind limb can influence fusimotor neurones (Appelberg *et al.* 1979, 1981).

If the γ-motor system is of importance in co-ordination, or learning of co-ordination, between different muscles and between different parts of a single muscle (cf. Johansson, 1985; Johansson & Sojka, 1985; Appelberg, Johansson & Sojka, 1986), then the findings of this report may also lend some support to the idea that articular mechanoreceptor reflexes operating via the γ-motoneurone loop may significantly contribute to the 'co-ordination of muscle tone in posture and movement' (Freeman & Wyke, 1967*b*). Thereby they may also contribute to the regulation of joint stiffness and joint stability.

Finally, the potent and complex interplay between fusimotor reflexes from different receptor types in the ipsilateral and also in the contralateral limb (cf. Appelberg *et al.* 1979, 1981, 1983*c*, 1984, 1986; Johansson, 1985; Johansson & Sojka, 1985) must be of great interest also for interpretations of muscle spindle afferent responses in experiments with non-reduced preparations, aimed at studying movement and position sense.

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