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# COMPLEX SPIKES IN PURKINJE CELLS IN THE LATERAL VERMIS (b ZONE) OF THE CAT CEREBELLUM DURING LOCOMOTION

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SUMMARY

1. Complex spikes (c.s.s) due to climbing fibre input were recorded from forty-one Purkinje cells in the lateral part of the vermis (i.e. the b zone) of lobule V of the cerebellum in cats walking on a moving belt or a horizontal ladder. Most cells were near the tips of the folia making up the lobule and some were shown by antidromic invasion to project to the ipsilateral lateral vestibular nucleus. In all cells c.s.s could be evoked through mechanical stimuli delivered manually to the neck and/or trunk and/or the limb girdles and/or the proximal parts of the limbs.

2. During walking c.s.s occurred at rates which ranged in different cells from 0·8 to 2·55/s (i.e. *ca.* 0·8 to 2/step). When activity was averaged across many successive steps the probability of c.s. occurrence was never completely constant throughout the step cycle, but no tendency was detected for c.s.s to recur at any precisely fixed time during the cycle.

3. When ladder locomotion was perturbed because a rung underwent an unexpected 2 cm descent when stepped on, some cells generated a c.s. at short latency in a proportion of trials. Such responses were well time-locked to the onset of rung movement but not to its cessation (which they often preceded).

4. For perturbations of either forelimb the earliest displacement-related c.s. occurred in different cells between 40 and 64 ms after the onset of rung movement. In different cells c.s.s occurred in from one out of five to three out of four perturbed steps (mean *ca.* two out of five steps). Eight out of seventeen cells responded to perturbation of the forelimb ipsilateral to the cell and five out of ten responded to contralateral perturbations.

5. Perturbation of the ipsilateral hind limb was accompanied by c.s.s in four out of nine cells and latency was usually longer (by *ca.* 30–40 ms). One cell showed a *decrease* in the probability of c.s. occurrence. Insufficient data were obtained for a systematic study of responsiveness to perturbation of the contralateral hind limb.

6. Cells showed different patterns of limb specificity, responding to perturbation of one, two or all of the three limbs studied. In total, c.s.s accompanied perturbation of at least one limb in thirteen out of twenty cells studied (65%).

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7. The findings are discussed in relation to the hypothesis (Oscarsson, 1980) that climbing fibres arising from inferior olivary neurones which are relays on spino-olivo-cerebellar pathways may function as detectors of motor control errors, including mismatches between intended and achieved movements. It is concluded that the results are compatible with the hypothesis, though they do not prove its correctness.

## INTRODUCTION

The cerebellar climbing fibres (c.f.s) arise from the inferior olivary neurones and each makes extensive synaptic contact with a single Purkinje (P) cell. Impulses in the c.f.s produce very large excitatory post-synaptic potentials in the P cells and as a result induce the occurrence of highly characteristic complex spikes (c.s.s). Many olive cells receive inputs from the spinal cord and therefore function as relay cells on a number of spino-olivo-cerebellar paths (s.o.c.p.s; see for reviews Oscarsson, 1973, 1980). These s.o.c.p.s are capable of mediating peripheral inputs because the olive cells concerned can be discharged by electrical stimulation of limb nerves and frequently by very modest mechanical stimuli delivered to the skin and/or deeper tissues (see for example, Oscarsson, 1968, 1969; Eccles, Sabah, Schmidt & Taborikova, 1972; Ishikawa, Kawaguchi & Rowe, 1972; Rushmer, Roberts & Augter, 1976; Armstrong & Rawson, 1979; Rushmer, Woollacott, Robertson & Laxer, 1980; Kolb & Rubia, 1980; Robertson, Laxer & Rushmer, 1982; Gellman, Houk & Gibson, 1983; Armstrong & Edgley, 1984*b*). Given this responsiveness it might be expected that during active movements by an awake animal olive cells would be excited by peripheral stimuli generated as a result of movement and would therefore discharge in fixed temporal relationship to some part of a repeated movement. Indeed, during discrete volitional movements of the forelimbs in monkeys movement-related c.s.s have been recorded (e.g. Thach, 1970; Harvey, Porter & Rawson, 1977; Gilbert & Thach, 1977) though their timing usually varied considerably from trial-to-trial and they were not present in every trial.

In the context of locomotor movements in the cat the evidence regarding the occurrence of time-locked (i.e. step-cycle-related) c.s.s is conflicting. In a study by Boylls (1980) of eighteen cells in the caudal part of the medial accessory olive (which supplies c.f.s to the cerebellar cortical 'a' zone in the medial part of the vermis; e.g. Groenewegen & Voogd, 1977) in decerebrate cats stepping on a moving belt, none exhibited any consistent step-related modulation of their discharge frequency and certainly there were no cases of discharges recurring at the same fixed time during successive step cycles.

Likewise, in the $c_1$ and $c_2$ zones of the paravermal part of lobule V of the cerebellar anterior lobe (which receive c.f.s from the dorsal and medial accessory olives) Armstrong, Campbell, Edgley, Schild & Trott (1982) failed in awake cats walking on a moving belt to demonstrate any precise time-locking of c.s.s to the step cycle in any of fifteen P cells. Subsequent study of many additional cells has confirmed this negative finding (Edgley, 1983; S. A. Edgley, M. Lidierth & D. M. Armstrong, in preparation). Recordings from cells further lateral in the paravermal cortex (in the $c_2$ zone and also in the $c_3$ zone which receives c.f.s from the rostral part of the dorsal accessory olive; Groenewegen, Voogd & Freedman, 1979) have also shown that the

timing of c.s.s during walking is essentially random with respect to the phases of the step cycle (S. A. Edgley, M. Lidierth & D. M. Armstrong, in preparation).

The only study in which any striking step-related modulations of c.s. frequency were found is by Udo, Matsukawa, Kamei, Minoda & Oda (1981) who found among ten forelimb-related P cells in the lateral part of the vermis of lobule V (i.e. in the cerebellar cortical 'b' zone which projects to Deiters' nucleus and receives c.f.s from the caudal part of the dorsal accessory olive; see Groenwegen & Voogd, 1977; Voogd & Bigaré, 1980) that during decerebrate locomotion the probability of c.s occurrence was much higher during the $E_1$ phase of the step cycle (when the forelimb is extended forwards just prior to footfall) than at other times. However, even then the probability averaged only one c.s per two steps.

One possible explanation for the apparent scarcity of locomotor-related c.s.s emerges from the hypothesis (see Miller & Oscarsson, 1970; Oscarsson, 1980) that the inferior olive may function to detect motor control errors such as the occurrence of mismatches between intended and achieved movements. This 'error detector' hypothesis is prompted partly by evidence regarding the connectivity patterns of olivary and spino-olivary neurones and partly by evidence that olive cells projecting to the cerebellar flocculus are activated by slip of the retinal image and may therefore signal errors in the control of eye movements (Simpson & Alley, 1974; Barmack & Hess, 1980; and see for detailed discussions Ito, 1984).

In the present study we have extended the work of Udo *et al.* (1981) by studying the c.s.s discharged during locomotion by b zone P cells in lobule V of the anterior lobe. However, the recordings were made in awake cats trained to walk for brief periods on a moving belt or to walk on a horizontal ladder. We have also carried out a partial test of the error detector hypothesis of s.o.c.p. function by incorporating in the ladder some rungs which could be allowed unexpectedly to 'give' slightly when stepped upon, thus displacing the limb downwards and producing a mismatch between the intended and the achieved stepping movement. Preliminary communication has been made of some of the results (Andersson & Armstrong, 1985).

## METHODS

The experiments were carried out on six purpose-bred young adult cats selected for confidence and tractability in the laboratory.

### *Training schedule*

The animals were housed in a large enclosure and fed *ad libitum*. They were brought daily to the laboratory for 2–4 weeks until they were accustomed to carrying out one of two locomotor tasks and to sitting on the experimenter's lap while natural mechanical stimuli (taps, pats, stroking the fur and movement of joints) were delivered manually to the body surface to determine receptive fields for evoking c.s.s in P cells. No aversive techniques were ever used during the training. Three animals were trained to walk steadily for short periods to maintain constant position on a motor-driven exercise belt. Most observations were made at a speed of 0·5 m/s which corresponds to comfortable walking in the adult cat. At this speed step cycle duration is usually *ca.* 850 ms (cf. Armstrong & Drew, 1984). The other three animals were trained to walk quietly to and fro on a horizontal ladder comprising seventeen rungs with a non-slip surface and circular cross-section (diameter 3 cm). The rungs were equally spaced at 19 cm intervals. This interval was chosen because 38 cm is a comfortable stride length. The animals walked confidently; each forefoot was placed on every second rung and immediately that foot was lifted the ipsilateral hind foot was placed on the

same rung. Adequate performance was encouraged (and readily achieved) by offering small morsels of codfish at each end of the ladder. However, the animals appeared to enjoy the task and rewards were often unnecessary.

The central portion of the ladder incorporated three rungs which were hinged at one end and held in position either by a latch or, if the latch was opened, by a weak spring. When one forefoot stepped onto such a rung when the latch was open, the walking was perturbed because the rung descended at one end until halted by a mechanical stop, which was reached when the centre of the rung had descended by 2 cm. When the forefoot was lifted the rung promptly returned, only to be depressed again when hind foot contact was made. The rungs involved were the seventh, ninth and twelfth in the sequence or the sixth, ninth and eleventh depending on the direction of the animal's approach. Latching and unlatching were carried out without providing visual or other cues to the animal and for any one pass along the ladder only one rung was unlatched, chosen in pseudo-random order. In addition, passes with a rung unlatched were interspersed with others without. As a result of these manoeuvres the animals were unable to predict which step, if any, would be perturbed, though they were of course accustomed to the fact that a rung might descend and with the nature of the consequent effect on their locomotion.

The presence of the movable rungs in no way inhibited the animals from undertaking the task though they did behave as if rung descent was regarded as a locomotor perturbation. Thus, although they often 'flowed' along the ladder without pause, in some trials they hesitated with one forefoot (or sometimes the hind foot) on the depressed rung. This behaviour occurred most often initially but was not infrequent even after many trials. In addition, one animal eventually developed a strategy of swerving in the central part of the ladder so as to walk close to the side at which the movable rungs were hinged, presumably to minimize any disturbance of its stepping. In another animal, when the forefoot underwent descent, a long pace was sometimes made with the ipsilateral hind limb so as to carry the foot over the movable rung and place it on the next (immovable) rung.

Each movable rung was equipped with two photosensors, one of which provided a brief marker pulse whenever the rung began to descend and also when the original horizontal position was regained; the other sensor generated a (larger) pulse when the rung descended to the mechanical stop. Across the large number of trial perturbations studied the over-all mean delay which elapsed between onset and completion of rung descent was 115 ms, but because the speed of locomotion varied between trials the delay was sometimes over 300 ms and sometimes as short as 32 ms. For some P cells studied the rungs were completely depressed whenever they were encountered but for others there were some trials in which depression was incomplete (see Results). In such cases the proportion of complete descents ranged from 67 to 96 % but usually exceeded 85 %. Where sufficient trials were accumulated to compare P cell responsiveness to perturbations of the ipsilateral and the contralateral forelimb (see Results) there was no significant difference between the two sets of trials in respect of either the proportion of complete descents or the time for rung descent, suggesting that equivalent perturbations were imparted to the two limbs.

*Operative procedure*

After training each animal was deeply anaesthetized with sodium pentobarbitone (Sagatal, B.D.H.; initial dose 40 mg/kg I.P.; maintainance doses as required to maintain deep anaesthesia) and an operation was carried out with benefit of broad-spectrum antibiotic cover (Depopen P; Berk Pharmaceuticals Ltd) and full aseptic precautions. Operative details are given in an earlier paper (Armstrong & Edgley, 1984*a*) but, in brief, a small craniotomy was made over the lateral part of the vermis and medial part of the paravermis in lobule V b/c on the left side of the cerebellum. A small lightweight titanium chamber was cemented over the exposed dura which was then oversealed with a layer of cold-curing medical grade elastomer (Dow Corning 382) deposited in the bottom of the chamber. The chamber was capped except during recording sessions when it was used to mount a small micromanipulator with which a glass-insulated tungsten micro-electrode could be advanced into the cerebellum to record extracellularly from individual neurones.

A burr hole allowed the stereotaxic implantation in four animals of a bipolar stimulating electrode near the dorsal border of the left lateral vestibular nucleus. This electrode was constructed from two lengths of stainless-steel wire, Teflon-insulated except at the tips and glued together (with elastomer) with the tips staggered by 1 mm. Stereotaxic coordinates of the tip of the longer wire were P 7·5; H–3; L 4 (atlas of Snider & Niemer, 1961). Finally, paired electromyographic electrodes

were implanted subcutaneously into selected muscles of the limbs including triceps and biceps brachii muscles in both forelimbs and gastrocnemius muscle in the left hind limb. Recovery from the operation was rapid and uncomplicated in each case.

*Recording procedure*

Extracellular recordings were made from individual P cells during moving belt locomotion (three animals) or ladder locomotion (three animals) and whenever possible attempts were made while the animals rested to evoke c.s.s by manually applying mechanical stimuli (pats, taps, joint movements) to the body and limbs and to define the locations from which such responses were evokeable. In some cases the response of the neurone to electrical stimulation of the lateral vestibular nucleus was determined. The usual stimulus was a single 0·2 ms rectangular pulse of maximum current amplitude 1 mA delivered at a rate of 1 per 2 s from an isolated stimulator. When the presence of an antidromic response was suspected (see Results) the latency and electrical threshold were determined and two or three pulses at twice threshold were delivered at a frequency of 500 Hz or above. The stimuli produced no behavioural effects.

Neuronal discharges were amplified and recorded on one channel of a multichannel tape recorder (Racal Thermionic Store 7D). Other channels were used to store electromyographic (e.m.g.) signals, a time-code, a voice log and in the case of ladder locomotion the pulses generated by movement of the displaceable rungs.

*Data processing*

Most cells discharged both simple and complex spikes and it was necessary to discriminate between the two. This could sometimes be achieved by passing the neural signals through a time–voltage window discriminator but was often achieved more readily by visual inspection of filmed records prepared by photographing an oscilloscope tube face (Tektronix 565) using a Grass C4N Kymograph Camera. Measurements from the films were made manually. Step cycle duration was measured as the interval between successive onsets of e.m.g. activity in the left triceps brachii muscle, an elbow extensor which generates one burst of e.m.g. per step, beginning 15–30 ms before the forefoot touches down. The times at which c.s.s occurred were also measured relative to the time of onset of the triceps e.m.g. Successive step cycles were normalized with respect to duration, each cycle was divided into ten (sometimes twenty) equal time bins and step histograms were prepared which averaged the c.s. activity during as many successive steps as possible and showed the number of c.s.s occurring per bin per average step.

C.s activity occurring in association with movement of the movable rungs was also subjected to an averaging analysis. For each pass across a movable rung, the times of all c.s.s were measured (to an accuracy of $\pm$ 4 ms) relative to the pulse marking the onset of rung movement. All c.s.s from 2 s before until 2 s after the pulse were included. The measurements were used to construct perturbation histograms which averaged the activity during as many successive passes as possible. Bin widths of 80, 40 and 8 ms were used. In the Figures bin width is usually 40 ms and the histograms extend only from 1 s before until 1 s after the onset of rung movement.

*Statistical treatment of data*

In the perturbation histograms for some cells some bins shortly after the onset of rung movement showed an increase in bin count suggestive of a transient (*ca.* 80 ms) increase in the probability of c.s. occurrence. This increase was more marked in some cells than in others and a question inevitably arises as to what should be considered to be a response and what a merely chance increase in the base-line level of c.s. activity. A response has been taken to be present if the probability exceeded the mean for all 'base-line' bins by an amount greater than two standard deviations about that mean. The base-line or background period was taken as the 1 s preceding the onset of rung movement.

*Histology*

At the end of each experiment the animal was humanely killed with an overdose of barbiturate anaesthetic and the positions of the e.m.g. electrodes were verified. In addition the cerebellum and brain stem were removed into 10% neutral buffered formalin and fixed for several days. The tissue was then frozen, sectioned sagittally at 50 $\mu$m and stained with cresyl violet. The location of the electrode in the lateral vestibular nucleus was verified microscopically and the cerebellum was also

scrutinized. No gross damage was detected but micro-electrode tracks could be seen with some difficulty, marked by thin lines of gliosis. Such track remnants were always confined to lobule V b/c of Larsell (1953).

## RESULTS

### *General*

Recordings were made from a total of 235 P cells identifiable as such because they discharged c.s.s which were usually interspersed among a much larger number of simple spikes, though some units discharged only c.s.s for part or all of the recording period. In most of the cells which discharged simple spikes there were substantial modulations of the discharge frequency which were time-locked to the step cycle; however, description of the simple spike discharge patterns is deferred to a later paper (G. Andersson & D. M. Armstrong, in preparation). All cells were in lobule V of the anterior lobe and the vast majority were in micro-electrode tracks which entered the lateral part of the vermis, just medial to the paravermal vein. A small proportion of cells were further lateral, in the medialmost part of the paravermis (see below).

C.s.s occurred at low frequency both in the resting animal and during locomotion (see below) and to collect enough data it was therefore necessary to make good-quality recordings in which c.s.s were reliably discriminable from simple spikes throughout long sequences of steps. This was achieved in forty-five cells, of which nineteen were studied for locomotion on the moving belt and twenty-six for ladder locomotion. The number of steps made during the sampling period ranged from fifty-six to over 300. Most recordings were extracellular throughout but in a few cases the cell was soon injured by the micro-electrode and a prolonged period ensued during which climbing fibre excitatory post-synaptic potentials (c.f.-e.p.s.p.s) were recorded (cf. Armstrong & Rawson, 1979). These occurred sometimes singly and sometimes in brief, high-frequency bursts (of up to five e.p.s.p.s. at *ca.* 500 Hz) depending on whether the parent olive cell discharged one action potential or a brief burst. C.s., single c.f.-e.p.s.p.s and e.p.s.p. bursts are all treated below as unitary events. It should be noted that one advantage of sampling olive cell activity indirectly by studying c.s.s or c.f.-e.p.s.p.s is that there is no danger that the temporal pattern of activity is being artifactually modified, as might occur if recordings were made directly from the inferior olive, when relative movement between cell and micro-electrode tip might generate abnormal patterns of discharge.

Most of the forty-five cells were located fairly superficially in lobule V. 60 % were recorded when the electrode tip was less than 2 mm below the depth at which the first signs of neuronal discharge were detected and a further 30 % were between 2 and 4 mm below this depth. The remaining neurones were between 4 and 6 mm below it.

Four cells were in tracks which began lateral to the paravermal vein but the remaining forty-one were all in tracks beginning medial to the vein. It is *a priori* highly probable that this latter group was in the cerebellar cortical b zone because this zone occupies the lateral half of the vermis. However, confirmatory evidence can be obtained by antidromic identification because b zone P cells project to the ipsilateral lateral vestibular nucleus (see Voogd & Bigaré, 1980) and their axons can be excited via a stimulating electrode positioned stereotaxically at the dorsal margin of the nucleus (see Methods). In fact, because of the need to study the cells during

sufficiently long periods of locomotion, antidromic identification was attempted in only nine of the forty-one vermal cells. In two cases no response was obtainable using a 1 mA search stimulus. However, in the other seven cases a single fixed-latency action potential was evoked. Such responses were judged to be antidromically generated because they followed stimulus frequencies in excess of 500 Hz and collision cancellations were observed when their expected time of occurrence was immediately after a spontaneous discharge. Antidromic latencies ranged from 0·75 to 1·2 ms and threshold stimulus currents from 60$\mu$A to 0·4 mA. The remaining (untested) vermal cells were encountered either in, or very close to, micro-electrode tracks where antidromic responses were readily produced in substantial numbers of other P cells. Among the forty-one vermal cells there were thirty-seven in which the responsiveness to mechanical stimuli delivered to the body and limbs could be studied. C.s.s could be evoked in all these cells but the areas from which they were obtained were large and diffuse and substantial pats, taps or joint movements were needed. In five cases the head and/or neck, in sixteen the shoulder and/or the proximal part of one or both forelimbs (and sometimes also the neck) and in thirteen one or both hindquarters were involved. In the remaining three cells c.s.s were obtainable from a very wide area which included the shoulders, the hindquarters and the length of the back. These characteristics suggest that the neurones were distributed across the width of the b zone so that cells were sampled from several of the sagittal afferent microzones into which the zone is divisible (see Andersson & Oscarsson, 1978*b*; Andersson & Eriksson, 1981). The four paravermal cells differed markedly from the others: in each a c.s. was very readily evoked by a light tactile stimulus localized to within the ipsilateral forefoot. Such response properties are commonly found in the cortical $c_1$ zone which lies immediately lateral to the paravermal vein (cf. for example Rushmer *et al.* 1980).

*C.s.s during steady stepping*

There were nineteen cells for which c.s.s could be recorded while the animal executed at least fifty-six steady steps keeping its position constant on a belt moving at a speed of 0·5 m/s. The rates with which c.s.s occurred in these cells were expressed per second and also per step for periods of locomotion which ranged in duration from 50 to 200 s (corresponding to from 56 to 238 steps). Rates ranged from 1·01 to 2·52 c.s.s per second (over-all mean $1{\cdot}90 \pm 0{\cdot}41$/s; S.D.) and from 0·8 to 2·02 c.s.s per step (over-all mean $1{\cdot}46 \pm 0{\cdot}35$/step; S.D.). Although in all but three cells the rate averaged more than 1 c.s. per step, it was found for every cell that there were some steps in which no c.s. was discharged. This is evident for one cell in Fig. 1 where the upper traces show the c.s. discharged during several successive paces. Step cycle timing is evident from the lower traces which show e.m.g. activity recorded from the ipsilateral triceps brachii muscle. Stance begins *ca.* 15–30 ms after triceps e.m.g. onset and continues until *ca.* two-thirds through the step cycle (cf. Armstrong & Drew, 1984).

In individual cells the c.s. rate during locomotion did not differ much from the rate in the resting animal. Direct comparison was possible in seven cases and the rate during locomotion was equal to that during rest in one case, lower in three cases and higher in the remaining three. The largest difference was in a cell which discharged at 1·9/s during rest and increased to 2·5/s during walking.

Fewer cells were studied during steady locomotion along a horizontal ladder because the stepping was usually subjected to intermittent perturbation as described in Methods. However, in four cases a large number of unperturbed paces were made and the over-all mean rates for c.s.s among these cells were 1·72/s and 1·3/step, which are very similar to the corresponding values for locomotion on the moving belt. For

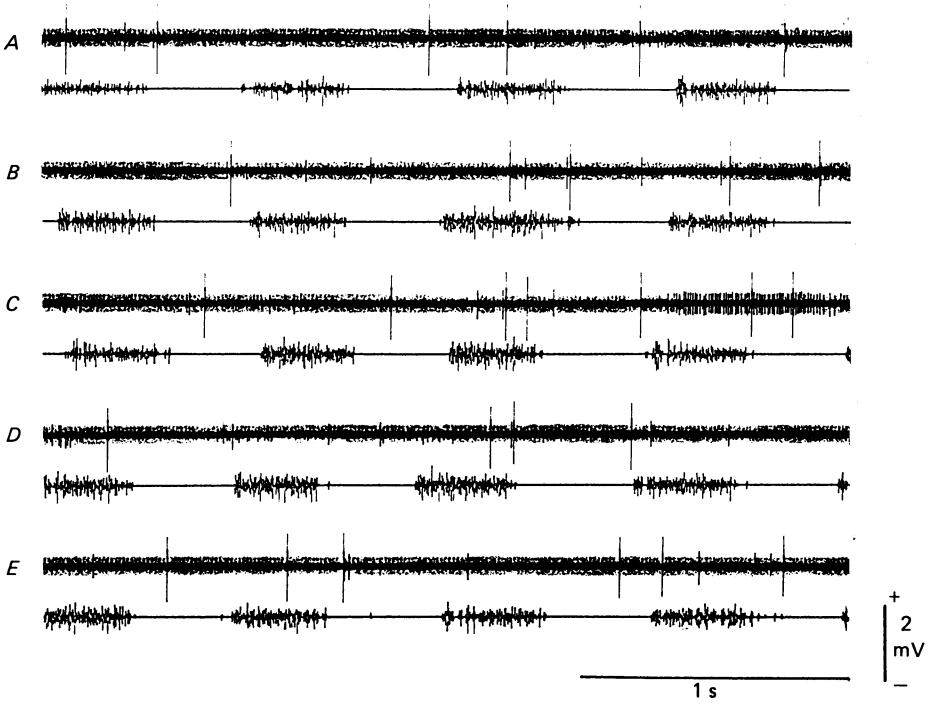


Fig. 1. C.s.s discharged by a P cell in the cerebellar cortical b zone during steady locomotion at 0·5 m/s on a moving belt. *A–E* are consecutive sections of record covering twenty successive paces. In each case upper trace shows unit discharge and lower trace shows e.m.g. recorded simultaneously from triceps brachii muscle in the ipsilateral forelimb. Voltage calibration applies to both traces.

a further eighteen vermal cells unperturbed steps on the ladder were studied in smaller numbers and in these cases the c.s. rates ranged from 0·8 to 2·55/s. For all twenty-two cells the average c.s. rate during ladder locomotion was $1{\cdot}63 \pm 0{\cdot}31$/s. This is just significantly lower (at the 5 % level; Student's *t* test) than the group mean for moving-belt locomotion so that the shift to a presumably more 'skilled' form of walking was apparently accompanied by no increase in the over-all rate of c.s. occurrence.

In addition, four cells in the $c_1$ cortical zone (see above) were studied during ladder locomotion and in these the c.s. rate ranged from 1·18 to 2·55/s.

*Temporal relationship between c.s. and the unperturbed step cycle*

Visual inspection of c.s.s recorded during steady stepping on the belt provided no immediate evidence that they recurred in any cell at any fixed time or times during

successive step cycles (c.f. Fig. 1). However, evidence for a statistical tendency for c.s.s to occur at preferred times was nevertheless sought by constructing step histograms for each cell to show the c.s. probability at different times during an 'average step' (see Methods). Typical examples are shown for three different cells in Fig. 2*A*, *B* and *D*.

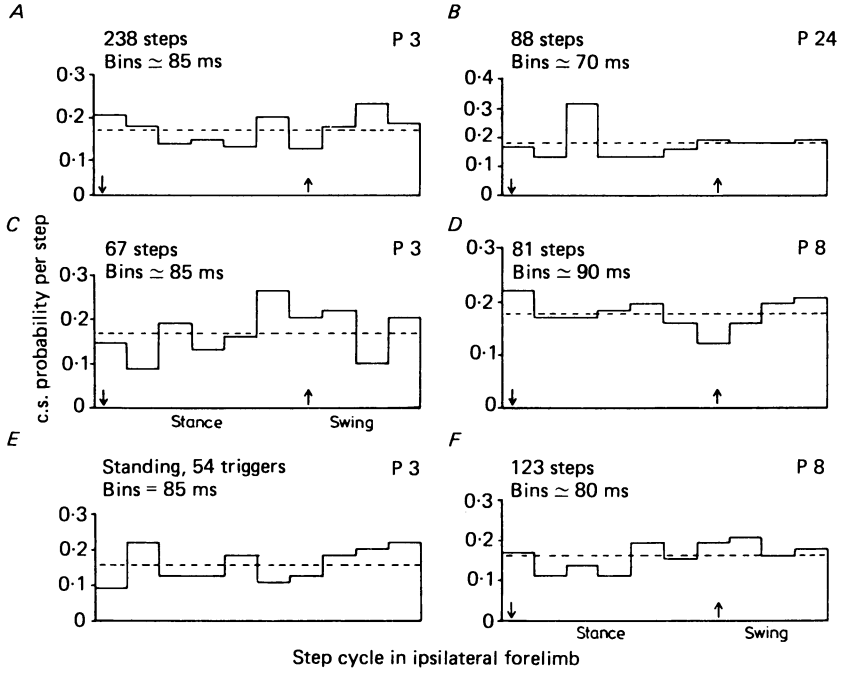


Fig. 2. Step histograms showing the average probability per step for the occurrence of c.s.s in each tenth of the step cycle during walking sequences involving many successive steps on a belt moving at constant speed. Speed of locomotion was 0·5 m/s in *A–D* and 0·7 m/s in *F*. *E* is a quasi-step histogram (for the same cell as in *A* and *C*) for a period of 46 s when the animal stood quietly; 54 regular pulses 850 ms apart were used to trigger the averaging. In the other histograms step cycle onset was taken as coincident with the onset of e.m.g. in the ipsilateral triceps brachii muscle. Individual steps varied somewhat in duration and as a result bin width varied slightly; the values stated represent mean bin width for the whole sequence of steps. Approximate times of footfall and foot-lift in the ipsilateral forelimb are shown by downward and upward arrows respectively. Horizontal interrupted lines indicate c.s. probability averaged across all ten bins.

Several features of these histograms (and of those prepared for the other sixteen cells) are worth noting. First, in no case was c.s. probability completely constant throughout the step cycle. Secondly, the highest probability encountered in any cell is represented by the third bin in the histogram which makes up Fig. 2*B*; in this case the value was 0·32 which is equivalent to the occurrence, during the third tenth of the step cycle, of one c.s. in (on average) a little less than one in three steps. Thirdly, considering the different cells, the histogram peaks (and troughs) were widely distributed throughout the step cycle so there was no one tenth of the step in which a particularly large number of cells reached peak (or minimum) probability for a c.s.

Fourthly, several observations cast doubt on the functional importance of the histogram fluctuations. Thus, in those nine cells sampled for fewer than 100 steps the over-all probability fluctuation (as indicated by the difference between their maximum and minimum probabilities during the step) exceeded 0·13 in seven cases whilst, by contrast, all of the five cells which were studied for more than 150 steps had fluctuations of less than 0·13 This suggests that at least a proportion of the

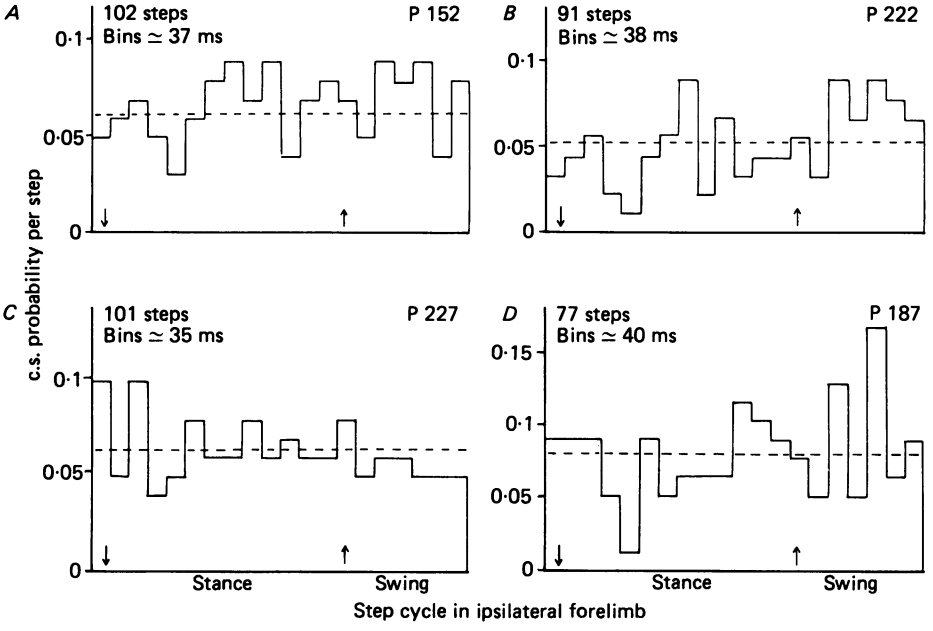


Fig. 3. Step histograms showing the average probability per step for the occurrence of c.s.s in four different P cells in each twentieth of the step cycle during the steps taken in the course of several (unperturbed) walks along a horizontal ladder. Conventions as in Fig. 2.

fluctuation may have arisen by chance (because of the limited size of the sample populations of steps). Another relevant observation is that in one cell studied at two different walking speeds the pattern of fluctuation was different in the two histograms (compare Fig. 2*D* and *F*). Somewhat similarly, histograms were made (for another cell) for two separate walks at the same speed and again they differed in their pattern of fluctuation (compare Fig. 2*A* and *C*). Finally, in two cells quasi-step histograms were made for periods when the animal was at rest, by using as start points for the averaging pulses regularly recurring at intervals similar to the mean duration of the step cycles during walking. In these displays (one of which is shown in Fig. 2*E*) the bin count showed (presumably random) variations similar to those seen in genuine step histograms.

For the four (b zone) cells studied during many successive ladder steps the findings were essentially similar, as shown by the step histograms in Fig. 3*A*, *B*, *C* and *D* (note that in these cases the step cycle has been subdivided into twenty bins).

*C.s.s occurring in relation to unexpected limb displacements during stepping*

In twenty-four cells (twenty in the b zone, four in the $c_1$ zone) it was possible to record c.s.s during ladder locomotion for so long that there were at least seven passes in which one of the movable rungs was allowed unexpectedly to swivel downwards when stepped on by the same limb (for example the forelimb ipsilateral to the recorded cell). In the best case there was an over-all total of thirty-five passes involving a locomotor perturbation and the average total was sixteen.

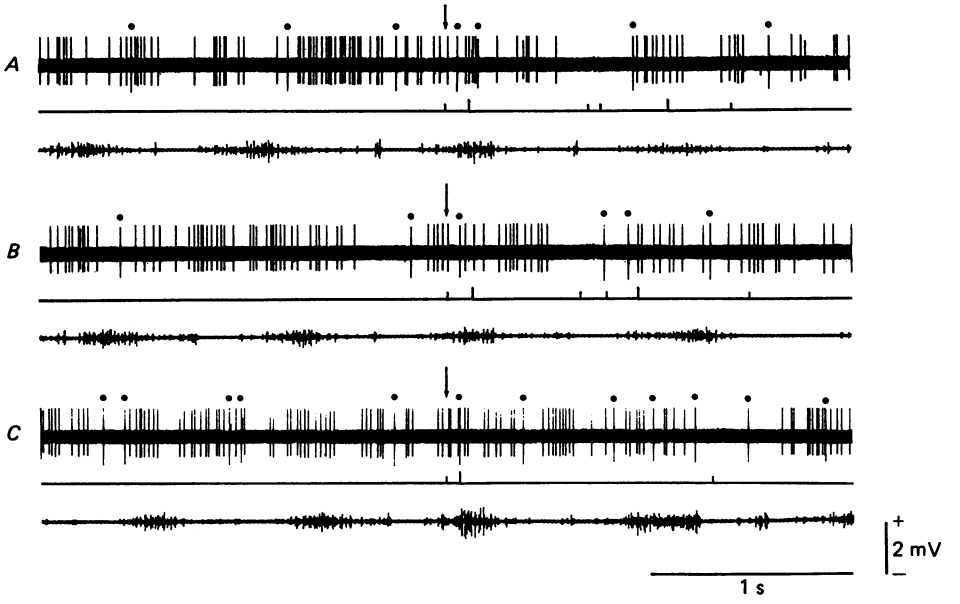


Fig. 4. Discharges of a P cell (cell 187) during ladder locomotion in three trials (*A*,*B* and *C*) in which one ladder rung descended through 2 cm when stepped on by the ipsilateral limbs. Cell discharge (upper trace) consists of a mixture of numerous simple spikes intermixed with sporadic c.s.s (marked by dots). Middle trace shows rung position pulses signalling departure from and return to the horizontal position (small pulses) and contact with a stop which limited the downward movement (larger pulses). Bottom trace shows e.m.g. recorded from the ipsilateral triceps brachii muscle. Note that the trials have been aligned on the moment when the movable rung began to descend as it was stepped on by the ipsilateral forelimb. Voltage calibration applies to both the P cell and the e.m.g. traces.

Visual inspection revealed for some b zone cells that, in at least some trials, a c.s. occurred very shortly after the moment when a foot was placed on the movable rung and the rung began to descend. This is illustrated for one cell in Fig. 4 where *A*, *B* and *C* show records obtained in three different trials in which the animal stepped on a movable rung using the ipsilateral forefoot (followed by the hind foot). The upper trace shows c.s.s (marked by dots) interspersed among a larger number of simple spikes and the bottom trace shows the bursts of e.m.g. which occurred (normally once per step) in the triceps brachii muscle in the forelimb which contacted the movable rung. The middle trace shows the pulses which signalled rung position and the first

small pulse (arrowed) shows the time of onset of rung descent while the next, larger pulse shows when the rung reached the stop limiting its downward movement. The second small pulse in each trial marks return of the rung to the horizontal position and in Fig. 4*C* this event is delayed because the animal paused with the forefoot on the depressed rung. In Fig. 4*A* and *B* there is a second similar sequence of three pulses which signal movement of the rung by the ipsilateral hind foot but in Fig. 4*C* such a sequence is absent because when the forefoot was transferred to the next rung the animal paused again so that the hind foot was not placed on the movable rung until after the end of the record.

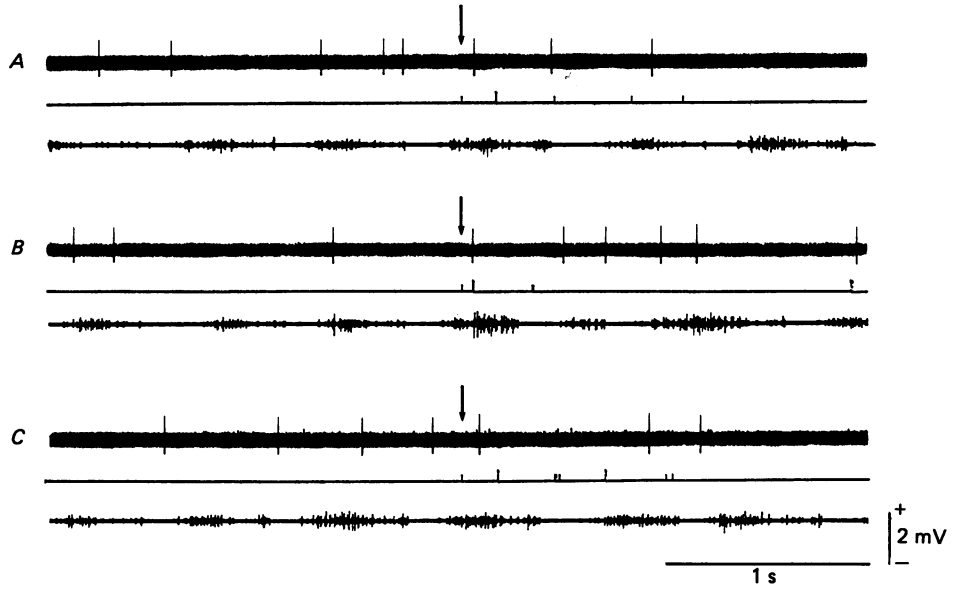


Fig. 5. Discharges of a P cell (cell 219) during ladder locomotion in three trials (*A*,*B* and *C*) in which one ladder rung descended when stepped on by the ipsilateral limbs. Cell discharge (upper trace) consists of c.s.s with no admixture of simple spikes. Other conventions as in Fig. 4.

Fig. 5*A*, *B* and *C* shows similar discharges of displacement-related c.s.s in another cell in each of three ipsilateral trials during a period when the discharge consisted only of c.s.s (though both before and after these records were made the cell generated simple spikes as well as c.s.s). Note that in Fig. 5*A* the rung was not completely depressed by the hind limb and in *B* the onset of rung depression by the hind limb is apparently not signalled. However, records at higher time resolution showed that the second small pulse in Fig. 5*B* is in fact two pulses only 6 ms apart, indicating that the hind foot was placed almost the instant that lift of the forefoot allowed the rung to regain the horizontal position. Note, however, that the hind foot subsequently took over 1·5 s fully to depress the rung.

Displacement-related c.s.s also occurred in other b zone cells and the remainder of Results reports in detail on the probability of occurrence of these responses, their latency and the extent to which their presence depended on which limb contacted the movable rung.

*C.s.s occurring in relation to forelimb displacements*

The displacement-related c.s.s were studied by averaging over as many trial walks as were available. Successive trials were aligned using the pulse which marked the onset of rung movement and the results were displayed in the form of event histograms (see Methods). Such a perturbation histogram for one neurone is shown in Fig. 6*A* where only those twenty trials have been included in which contact with the rung was made by the forefoot ipsilateral to the recorded cell. It is evident that c.s. probability is greatly elevated during the second bin after the pulse used to initiate the averaging (i.e. during the period 40–80 ms after the onset of rung

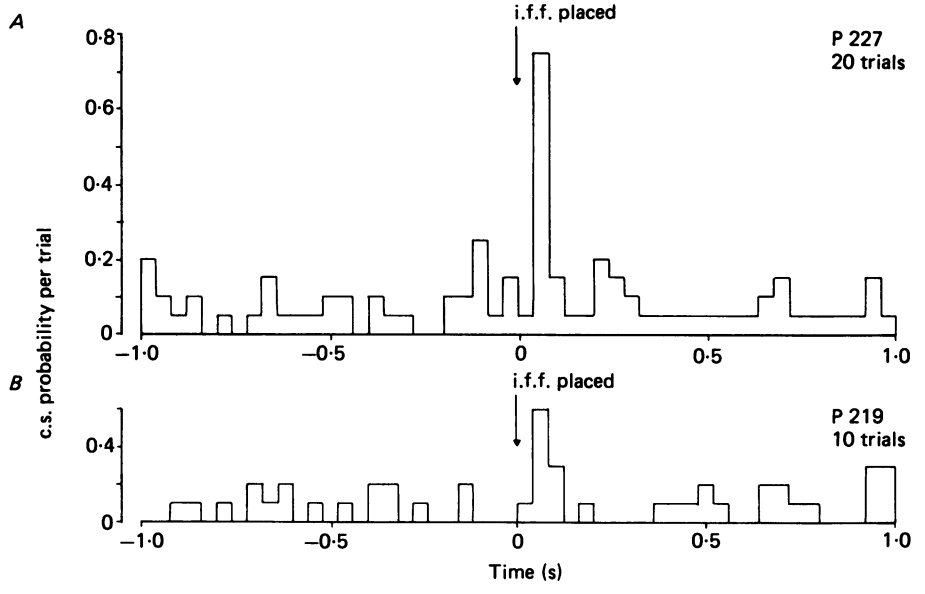


Fig. 6. Perturbation histograms which average the c.s. activity of two different P cells during the stated numbers of trials in which a movable rung was stepped on with the ipsilateral forefoot (i.f.f.). Note that averaging was initiated using the pulse which signalled the onset of downward movement of the rung and was carried out for 1 s before and after that event. Bin width 40 ms in both *A* and *B*.

movement). Inspection of the individual trials showed that in any one trial in which a response occurred it consisted of a single c.s. and this was almost invariably the case in other reponsive cells: only extremely rarely was there a trial in which two c.s.s appeared in quick succession.

In Fig. 6*A* the size of the histogram peak is such as to leave no doubt that a response occurred, though note that peak probability was 0·75, corresponding to the occurrence of a c.s. in only fifteen out of the twenty trials. There were other cases in which a response was very prominent and a second example is shown in Fig. 6*B* where again c.s. probability is raised in the second bin after the onset of rung movement (and perhaps also in the third bin). By contrast, in other cases, such as that illustrated in Fig. 7*A*, it was equally clear that no response occurred. However, there were some

cells in which it seemed possible that a response was present but peak probability was smaller than in Fig. 6*A* and *B*. One such cell is illustrated in Fig. 7*B*. In view of the existence of a range of response sizes it was judged important to quantify the likelihood that any particular response was real. As explained in Methods, the approach adopted was to express the amplitude of the histogram peak in terms of the number of standard deviations by which it exceeded the mean 'background' level. It should be noted that, throughout the cell population, responses to forelimb displacements appeared to be confined to the period 40–120 ms after the onset of rung

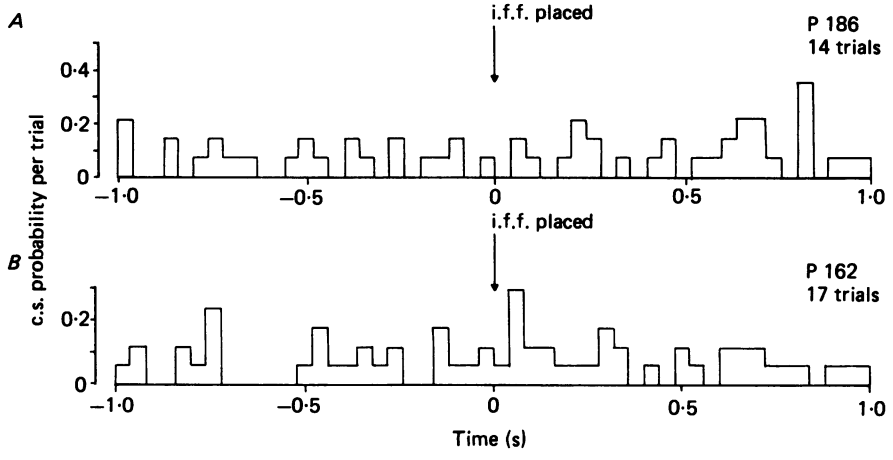


Fig. 7. Perturbation histograms which average the c.s. activity of two different P cells during the stated numbers of trials in which a movable rung was stepped on with the ipsilateral forefoot. Conventions as in Fig. 6.

movement. This was therefore taken as the 'response period' and the mean background probability and its standard deviation were therefore determined using 80 ms bins. Note also that the number of c.s.s taken as representing a possible response was the number remaining after subtracting (from the total counts in the response period) the mean number of c.s.s expected to occur as a result whatever process produced the 'background' c.s.s. The results of this treatment are shown in Table 1A for all seventeen cells for which there were at least 7 trials (range 7–22) in which the movable rung was depressed by the ipsilateral forefoot. It is evident from the response column that there was considerable variation between the different cells. In those eight cells in which the response exceeded the background by more than two S.D.s (marked by asterisks in the $P_R$ column) response probability ranged from 0·2 to 0·75. This corresponds to a range from one 'extra' c.s. in one out of every five paces to one extra c.s. in three out of every four paces. The over-all mean response probability was 0·43 ($\pm$0·20; S.D.; $n = 8$)

As regards responses when the rung moved unexpectedly under the *contralateral* forefoot, the number of available trials was usually fewer but there were ten cells in which 7 or more trials (range 7–15) were accumulated and in five cases there was a significant increase in c.s. probability during the period 40–120 ms after the onset

of rung movement. Data relating to these cells are shown in Table 1B and again the response probability varied, the range being from 0·33 to 0·57 (over-all mean $0{\cdot}47 \pm 0{\cdot}10$; S.D.; $n = 5$).

No responses to unexpected descents of the forelimbs (or hind limbs) were present in any of the four cells located in the paravermal $c_1$ zone of the cortex.

TABLE 1. Probability of c.s. occurrence per trial in the background period ($P_B$) and in the response period ($P_R$) for unexpected displacements of the forelimb during ladder locomotion. Seventeen cells in A; ten in B

| Cell | No. of trials | $P_B$ | S.D.$_B$ | $P_R$ | $P_R$/S.D.$_B$ |
|---|---|---|---|---|---|
| | (A) Displacements of ipsilateral forelimb | | | | |
| 106 | 7 | 0·12 | 0·12 | 0·45* | 3·7 |
| 152 | 14 | 0·16 | 0·05 | 0·20* | 3·7 |
| 159 | 8 | 0·12 | 0·15 | −0·12 | −0·8 |
| 161 | 22 | 0·15 | 0·06 | 0·00 | 0·0 |
| 162 | 17 | 0·13 | 0·10 | 0·28* | 2·8 |
| 179 | 7 | 0·08 | 0·09 | 0·06 | 0·6 |
| 186 | 14 | 0·13 | 0·08 | 0·08 | 1·1 |
| 187 | 21 | 0·14 | 0·10 | 0·43* | 4·3 |
| 194 | 7 | 0·11 | 0·14 | −0·11 | −0·8 |
| 197 | 20 | 0·15 | 0·06 | −0·05 | −0·6 |
| 210 | 7 | 0·11 | 0·14 | 0·03 | 0·2 |
| 216 | 8 | 0·09 | 0·09 | 0·16 | 1·7 |
| 219 | 10 | 0·15 | 0·12 | 0·75* | 6·4 |
| 222 | 17 | 0·14 | 0·05 | 0·45* | 8·6 |
| 227 | 20 | 0·14 | 0·08 | 0·61* | 7·4 |
| 230 | 8 | 0·10 | 0·07 | 0·28* | 3·7 |
| 232 | 13 | 0·17 | 0·10 | 0·06 | 0·6 |
| | (B) Displacements of contralateral forelimb | | | | |
| 146 | 10 | 0·17 | 0·13 | 0·33* | 2.6 |
| 160 | 7 | 0·10 | 0·09 | 0·47* | 5·2 |
| 161 | 13 | 0·11 | 0·09 | −0·03 | −0·3 |
| 187 | 8 | 0·18 | 0·15 | 0·20 | 1·3 |
| 197 | 15 | 0·12 | 0·07 | 0·08 | 1·1 |
| 210 | 9 | 0·12 | 0·10 | 0·10 | 1·0 |
| 216 | 7 | 0·02 | 0·05 | 0·55* | 9·8 |
| 222 | 9 | 0·13 | 0·12 | 0·43* | 3.5 |
| 230 | 9 | 0·10 | 0·09 | 0·12 | 1·3 |
| 235 | 7 | 0·14 | 0·14 | 0·57* | 4·1 |

Background probability ($P_B$) is the mean value for all 80 ms periods during the 960 ms preceding the onset of rung movement. S.D.$_B$ is the standard deviation about the background mean. Response probability ($P_R$) is the probability above or below (minus values) background for the 80 ms period beginning 40 ms after onset of rung movement. $P_R$/S.D.$_B$ indicates response magnitude expressed in terms of the number of S.D.s by which it exceeded the background mean. Zero and negative values indicate $P_R = P_B$ and $P_R < P_B$ respectively. Response probabilities for which $P_R$/S.D.$_B > 2{\cdot}0$ are asterisked.

*Responses to hind-limb displacement*

Considerably fewer trials were available for study in which the hind limbs encountered a movable rung and several factors contributed to this. First, there were some trials in which the hind limb was placed on the rung before lifting of the ipsilateral forelimb had allowed the rung fully to regain its original position. No signal

was then available to indicate the time at which the rung began to descend under the weight of the hind limb. In addition, there were some trials in which descent of the forelimb induced the animal to pause and on some of these occasions the limb was flexed and extended rhythmically so that the rung oscillated over part or all of its range of movement and a confusing sequence of e.m.g. and position signals was generated.

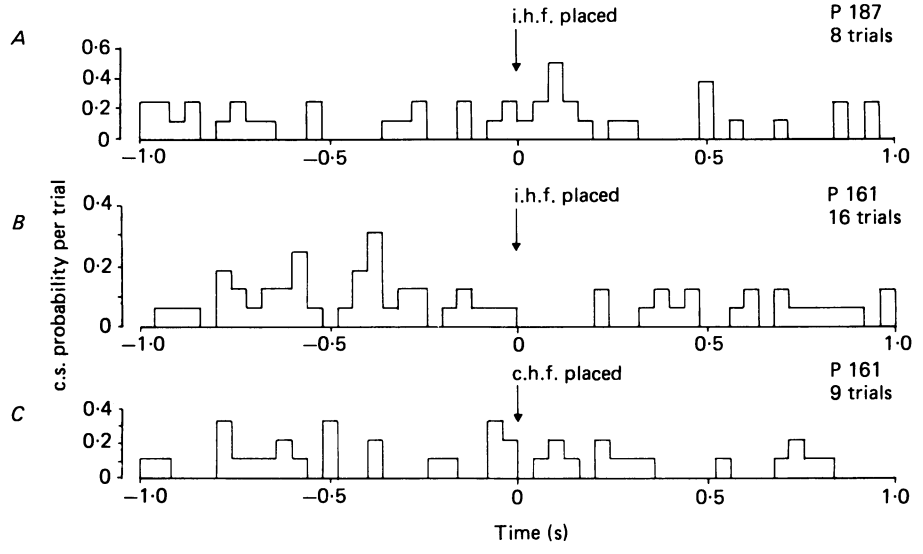


Fig. 8. Perturbation histograms which average the c.s. activity of two different P cells during the stated numbers of trials in which a movable rung was stepped on with a hind limb (downward arrows). In *A* averaging has been initiated at the moment when the rung began to move under the ipsilateral hindfoot (i.h.f.). *B* is a similar histogram for another cell while *C* is for the same cell as in *B* but shows trials involving the contralateral hindfoot (c.h.f.).

Nevertheless for nine cells there were at least 7 trials (range 7–16) in which responses to descent of the ipsilateral hind limb could be studied and one such cell is shown in Fig. 8*A*. Inspection of this histogram (and those for other cells) showed that, except in one case (cell 197), response latencies were somewhat longer than for forelimb displacements so that when the bin width was 40 ms the appropriate 'response' bins were the third and fourth after the onset of rung movement. The results for the nine cells are summarized in Table 2 where it may be seen that there were four in which c.s. probability was significantly elevated during the response period. In one cell probability was as low as 0·12 but in the other three the values were close to 0·6, corresponding to the appearance of an 'extra' c.s. in six out of every ten trials.

P cell 161 in Table 2 was exceptional in showing a significant *reduction* in c.s. probability during the response period. The perturbation histogram for ipsilateral hind-limb trials in this cell is presented in Fig. 8*B* where it is evident that the probability reduction in fact lasted until *ca.* 200 ms after the onset of rung movement. This was the only cell in which such an 'inhibitory' response was detected for either

the forelimbs or the hind limb. There were other cells (159, 194 and 197 in Table 1 A, 161 in Table 1 B, 179 and 227 in Table 2) which generated few or no c.s.s during the response period but in these cases the reduction in probability was not statistically significant.

Cell 161 was also the only cell for which there were sufficient trials involving the *contralateral* hind limb and the data from these trials are shown in Fig. 8*C* where it can be seen that probability was neither increased nor decreased during the response period.

TABLE 2. Probability of c.s. occurrence per trial in the background period ($P_B$) and in the response period ($P_R$) for unexpected displacements of the ipsilateral hind limb (nine cells)

| Cell | No. of trials | $P_B$ | S.D.$_B$ | $P_R$ | $P_R$/S.D.$_B$ |
|---|---|---|---|---|---|
| 152 | 9 | 0·16 | 0·05 | 0·62* | 13·5 |
| 161 | 16 | 0·15 | 0·06 | −0·15* | −2·5 |
| 162 | 10 | 0·13 | 0·10 | 0·16 | 1·6 |
| 179 | 7 | 0·09 | 0·08 | −0·09 | −1·1 |
| 187 | 8 | 0·14 | 0·10 | 0·61* | 6·1 |
| 197 | 11 | 0·15 | 0·07 | 0·59* | 8·3 |
| 216 | 7 | 0·09 | 0·09 | 0·05 | 0·6 |
| 222 | 8 | 0·13 | 0·05 | 0·12* | 2·4 |
| 277 | 11 | 0·14 | 0·08 | −0·05 | −0·6 |

Conventions as in Table 1, but the response period was the 80 ms beginning 80 ms after the onset of rung movement, except in cell 197 where it was the 80 ms beginning 40 ms after movement onset.

P cell 197 also was exceptional but in a different manner. It generated an unusually large number of c.s.s when there was descent of the ipsilateral hind limb. As may be seen from the histogram in Fig. 9*A* (bin width 80 ms) there was no immediate response to twenty contacts involving the ipsilateral forefoot but beginning at *ca.* 750 ms there was an extended period of elevated c.s. probability. In eleven of these trials the data could be realigned about the moment when the rung began to descend under the ipsilateral hind foot and the result is shown in Fig. 9*D* (bin width 40 ms) where no fewer than twenty-four c.s.s occurred in the period 40–400 ms after the onset of rung movement. This implies, of course, an average of *ca.* 2 c.s.s per trial. The individual trials used to construct Fig. 9*D* are shown in raster form in Fig. 9*E* where it can be seen that the number of c.s.s in fact varied from one to four per trial in the 40–400 ms period. In the bottom three trials (c.s.s shown by open circles) it was possible to unlatch the rung just after the forelimb ceased contact and just before hind-limb contact was established. In these trials, therefore, perturbation was confined to the hind limb and it is clear that responses persisted.

Another feature of this neurone which was of special interest was that, when the forelimb underwent displacement, the animal followed through with the hind limb in some trials but in others a long stride was made with the hind limb so that the movable rung was bypassed in favour of the next (fixed) rung. When equal numbers of such trials were compared it became evident (compare Fig. 9*B* and *C*; 80 ms bins) that displacement-related c.s.s occurred in the former cases (Fig. 9*C*) but not in the latter (see Fig. 9*B*).

Note that in Fig. 9*B* and *C* the histogram start point was the onset of forelimb displacement. The extra c.s.s which begin to occur 800 ms after this in Fig. 9*C* are those associated with displacement of the hind limb. It was necessary to use the forelimb signal to construct Fig. 9*B* and *C* because inevitably when the hind limb avoided the movable rung as in Fig. 9*B* it generated no position signals.

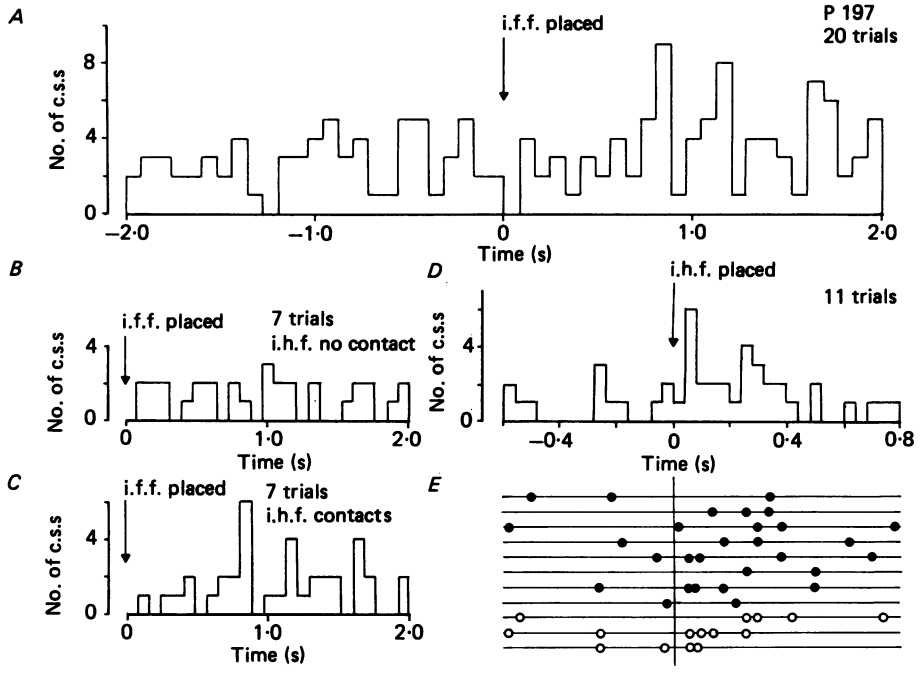


Fig. 9. Perturbation histograms which sum the c.s. activity in P cell 197 during trials in which a movable rung was stepped on with a particular limb (downward arrows). *A* shows activity in twenty trials summed for 2 s before and after contact of the ipsilateral forefoot (i.f.f.) with the movable rung. Note that bin width is 80 ms. *B* and *C* respectively are for trials (from among those in *A*) in which the hind foot either avoided or made contact with the movable rung (see text). Bin width 80 ms. *D* shows eleven of the same trials as in *A* but summing was initiated about the moment when the rung began to descend under the ipsilateral hind foot (i.h.f.). Note that bin width is 40 ms. *E* shows the data used to construct *D* displayed in raster form; time scale is as in *D*, each line is a separate trial and c.s.s are shown by circles; the open circles in the three lowest lines signify that in these trials the rung descended under the ipsilateral hind foot but was earlier prevented from descending under the forefoot (see text).

*Minimum latencies of perturbation-related c.s.s*

As already mentioned, responses to forelimb displacements were confined to the period 40–120 ms after onset of rung movement. However, in order to define more accurately the latency of the earliest responses, perturbation histograms were constructed to a higher time resolution (bin width 8 ms) than that adopted in Figs. 6, 7, 8 and 9. An example is shown in Fig. 10*A* where for ipsilateral displacements (open bins), the first bin in which c.s. probability was significantly above background, is that which begins 56 ms after the onset of rung movement. For contralateral

displacements (filled bins) the corresponding value is 72 ms (note that this cell, P 227, does not feature in Table 1B because only six contralateral forelimb trials were available). Considering all those cells in which displacements were accompanied by significant increases in c.s. probability there was in fact relatively little variation in the time of the first significant bin. For displacements of the ipsilateral forelimb the first increase in probability began at 56 ms in six of the eight cells concerned and at 40 ms and at 64 ms in the two remaining cells. For the contralateral forelimb the

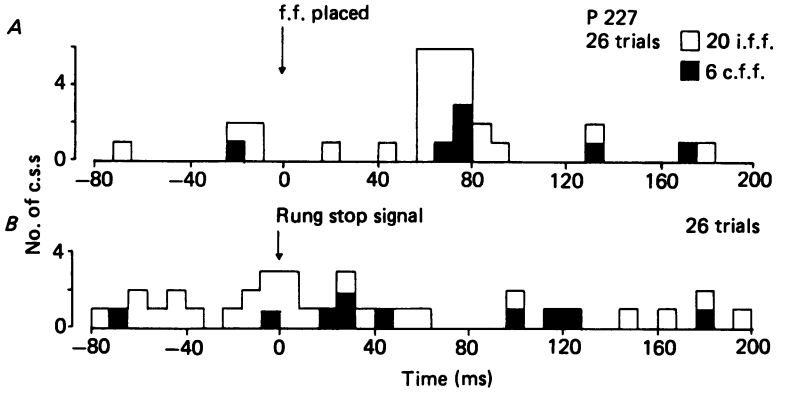


Fig. 10. Perturbation histograms which sum the c.s. activity of P cell 227 during trials in which a movable rung was stepped on. *A* sums the activity during trials in which either the ipsilateral (i.f.f.; open bins) or the contralateral (c.f.f.; filled bins) forefoot made contact with the rung. *B* utilizes the same trials as in *A* but activity is summed about the end rather than the beginning of rung descent. Note bin width is 8 ms.

corresponding timings in the five cells concerned were 40, 48, 56, 56 and 64 ms. There was only one cell (P 222) for which at least seven trials were available for each forelimb and which responded bilaterally and in this case the first significant bins were at 64 ms for ipsilateral and 56 ms for contralateral displacements.

For displacements of the ipsilateral hind limb the shortest latencies estimated on a similar basis for three cells (152, 187 and 222; see Table 3) were 72, 88 and 110 ms respectively. Values for the ipsilateral forelimb were also available for each of these cells and in each case the forelimb latency was shorter (by 32, 16 and 46 ms respectively). Cell 197, which responded only to hind-limb displacements, did so at both shorter and longer latencies than the other three cells yielding hind-limb responses. As pointed out above (see Fig. 9) it frequently gave more than one c.s. per trial, spread out over a considerable period. Its earliest significant response was in the 40–48 ms bin.

*Response timing relative to rung movement*

One possible explanation for the occurrence of c.s.s in association with rung descent was that they were evoked by a jarring imparted to the limb when the descending rung was halted by the mechanical stop (see Methods). This possibility was investigated via histograms initiated using the pulse which marked the end of rung descent. The results are shown for one cell in Fig. 10*B* which shows the same trials as in Fig. 10*A*. In Fig. 10*A* time zero is taken as the onset of rung movement while

in Fig. 10*B* it is taken as the end of rung descent. Descent time varied considerably between trials and it is evident that although the responses are closely locked to the onset of rung movement they are not at all well locked to its cessation. Indeed in some trials the c.s.s occurred before the stop was reached (cf. Figs 4 and 5) and in others they occurred less than 40 ms afterward, which is probably too early for contact with the stop to have played a causative role in their appearance (see Discussion).

*Limb specificity of responses to rung descent*

*Specificity between forelimbs.* There were seven cells (compare Tables 1A and B) which could be compared in respect of their responsiveness to displacements of the left and right forelimb (i.e. the ipsilateral and the contralateral limb). Two of these cells (187 and 230) responded only to ipsilateral displacements, three (161, 197 and 210) responded to displacement of neither forelimb, one responded to either limb (222, ipsilateral and contralateral response probabilities were rather similar) and the remaining cell (216) responded only to contralateral displacements. Note, however, that in this cell there were only eight ipsilateral trials and $P_R/S.D._B$ was 1·7 so it is possible that had more trials been available a significant (albeit low probability) response might have been revealed. In any event, the variation between the cells implies that depending on which forelimb makes contact with the movable rung, c.s.s will occur in two different, though partially overlapping, populations of b zone P cells (see Discussion).

*Forelimb–hind-limb specificity.* There were nine cells which could be compared in respect of their responsiveness to displacements of one or both forelimbs and the ipsilateral hind limb. These cells are shown in Table 3 and among them there was clearly a very wide variation in limb specificity. For example, cell 187 responded to displacement of both ipsilateral limbs but not of the contralateral forelimb, cell 222 responded to displacement of either forelimb and the ipsilateral hind limb while cell 197 responded only to hind-limb displacement.

TABLE 3. Limb specificity for c.s.s evoked in nine different cells by unexpected displacements of the ipsilateral forelimb (i.f.l.), the contralateral forelimb (c.f.l.) and the ipsilateral hind limb (i.h.l.). Asterisks indicate significant responses; bars, no significant response; n.t., not tested; open circle (cell 161), c.s. probability significantly reduced in the response period. r.f., receptive field location

| Cell | i.f.l. | c.f.l. | i.h.l. | r.f. |
|---|---|---|---|---|
| 152 | * | n.t. | * | ?l.s. |
| 161 | — | — | 0 | l.s./h.l./t. |
| 162 | * | n.t. | — | n./sh. |
| 179 | — | n.t. | — | l.s./h.l. |
| 187 | * | — | * | sh./th. |
| 197 | — | — | * | ?l.s./h.l. |
| 216 | — | * | — | l.s./h.l. |
| 222 | * | * | * | ?l.s. |
| 227 | * | n.t. | — | n./sh./th. |

For receptive fields l.s. is lumbosacral area; h.l., proximal parts of hind limb; t., tail; n., neck; sh., shoulders; th., thoracic part of trunk. ? indicates that c.s.s were evoked from the r.f. with low probability.

*Limb specificity compared with responsiveness to mechanical stimuli delivered to the resting animal*

For each cell an area of the body was located from which c.s.s could be evoked by mechanical stimuli delivered to the resting animal and it was of interest to determine whether any correlation existed between this location and the limb specificity shown for the appearance of displacement-related c.s.s. In fact, as may be seen from the cells in Table 3, correlation was only partial. Some cells (e.g. 162 and 227) responded only to stimuli in the neck–shoulder region and responded only to forelimb displacement while another (197) responded only to stimuli in the lumbosacral area and the proximal parts of the hind limbs and to displacement only of the hind limb. However, other cells behaved differently: 152 and 187, for example, each responded to displacements of both ipsilateral limbs but c.s.s were driven from the lumbosacral area in the first and from around the shoulders in the second. In addition, cells 216 and 222 both received lumbosacral input but the first responded only to displacement of the contralateral forelimb while the second responded for all three limbs. Finally, it may be noted that there was only one case (cell 216) in which the area from which c.s.s were evokable in the resting animal did not include at least one of the limbs for which there was a displacement response.

## DISCUSSION

*Receptive fields for evoking c.s.s in the resting animal*

Whenever adequate testing could be carried out, c.s.s could be evoked in b zone P cells by natural mechanical stimuli to some part of the body and limbs and the variety of receptive field locations encountered suggested that cells were sampled from across the width of the b zone and therefore from its different afferent 'microzones' (Andersson & Oscarsson, 1978*b*; Andersson & Eriksson, 1981). In general, the stimuli required were such as to suggest it was necessary to activate deep rather than (or possibly as well as) cutaneous mechanoreceptors and it was difficult to precisely define the spatial extent of the receptive fields, though clearly many were rather large (see Results). Inputs from distal to the elbow were rarely effective and in this respect the b zone differs markedly from the intermediate or paravermal cortex of lobule V. In other experiments c.s.s were readily evoked in the $c_1$ and $c_2$ zones by tactile stimuli delivered to hairy or glabrous skin of the forelimbs; more often than not the paw and/or wrist were involved in the receptive fields and in a substantial number of cases the latter were considerably smaller than for any of the present neurones (Edgley, 1983; S. A. Edgley, M. Lidierth & D. M. Armstrong, in preparation). We have no doubt this difference is genuine because in the present experiments the few paravermal cells were all readily driven from the distal part of the ipsilateral forelimb.

Our results from the b zone came as a surprise because an area of cortex apparently including the b zone has been studied in barbiturate-anaesthetized cats by Robertson *et al.* (1982) who found that 95% of c.s. receptive fields involved light tactile stimuli, that many were small and that the distal parts of the limbs were well represented. The marked difference between the two studies might suggest that transmission of

cutaneous mechanoreceptor input to the b zone is subject, in the awake resting animal, to some central inhibitory action which is reduced or absent during barbiturate anaesthesia. However, according to Gellman *et al.* (1983), who recorded in barbiturate-anaesthetized (and in decerebrate) cats from neurones in the caudal part of the dorsal accessory olive (which provides the c.f.s to the b zone), most cells responded exclusively to deep inputs, such as squeezes to muscles or passive movements imposed on joints; only in the caudalmost tip of the dorsal accessory olive was there a small zone of cells receiving cutaneous as well as deep inputs. These characteristics accord well with our results, as does the finding that receptive fields were usually substantially larger in the caudal half of the dorsal accessory olive than in the rostral half (which provides c.f.s to the paravermal part of the cerebellum). The difference between our findings and those of Robertson *et al.* (1982) therefore remains, for the present, unexplained.

*C.s.s during steady stepping*

Both in the absence of movements and during unperturbed walking on a moving belt all nineteen P cells studied discharged c.s.s irregularly at a low rate, which ranged from 1·0 to 2·5/s. Interestingly enough, although ladder walking is a form of locomotion which demands considerable visuo-motor co-ordination, c.s. rates were very similar, ranging from 0·8 to 2·5/s (the population mean was in fact just significantly *lower*; see Results). These rates are similar to those encountered in numerous studies of c.s. rates in awake animals, whether at rest or engaged in active movement (cf. for example Thach, 1970; Harvey *et al.* 1977; Gilbert & Thach, 1977; Armstrong & Rawson, 1979).

Other studies have demonstrated similar levels of c.s. activity in P cells in the paravermal part of lobule V during walking at the same speed (Edgley, 1983; Armstrong *et al.* 1982; S. A. Edgley, M. Lidierth & D. M. Armstrong, in preparation) and similar rates were also found in decerebrate walking cats among olive cells projecting to the cerebellar cortical a zone which occupies the medial part of the vermis (Boylls, 1980). Broadly similar levels (0·36–3·5/s; 0·35–2·1/step) were also encountered in the b zone in decerebrate walking cats by Udo *et al.* (1981) who recorded c.s.s from ten P cells identified as receiving input from the forelimb and also as projecting to the lateral vestibular nucleus.

However, in one important respect our findings differ markedly from those of Udo *et al.* (1981) who found that in their cells the probability of c.s. occurrence was highest (0·56/step; ±0·58, S.D.) during the $E_1$ phase of the step cycle. The other phases of the step are the flexion (F) phase (which together with $E_1$ makes up the swing part of the cycle), the $E_2$ phase when the limb yields slightly under the body weight and the $E_3$ phase when the limb is extended to thrust the animal forwards ($E_2$ and $E_3$ together constitute stance) and during these phases c.s. probability was 0·12, 0·13 and 0·10 respectively. As might be expected from these values most individual cells achieved peak c.s. probability in the $E_1$ phase.

In the present experiments the cells all showed some fluctuation in c.s. probability during the cycle but averaging across all nineteen studied on the moving belt plus the four studied in comparable detail during ladder locomotion the probability for the $E_1$ phase (i.e. the period occupied by bins 9, 10 and 1 in our histograms with ten

bins) was lower (mean 0·43) and the variation between cells was less (S.D. ±0·13) than found by Udo *et al.* (1981) Moreover, in the other phases the probability was *higher* so that, for example, in the combined $E_2$ and $E_3$ phases the population mean was 0·74 (S.D. ±0·17, bins 2–6) as compared with their 0·23. Likewise for the F phase the present mean was 0·27 (±0·09) as compared with their 0·12. Our values were virtually unchanged when only those cells were considered which had forequarter receptive fields (seven cells).

When c.s. probabilities during the step were expressed per *bin* the means for the present cell population hardly varied both for the whole group and for the sub-group with forequarter receptive fields: for the whole population they were 0·15 per bin during the $E_1$ phase and 0·14 throughout the $E_2$, $E_3$ and F phases. Thus, over a sequence of steps, the population output of c.s.s from the b zone cells was virtually constant throughout the step cycle. However, this does not preclude the possibility that in particular steps a much more (or less) than average number of cells might discharge a c.s. in one particular phase. Whether such fluctuations occur (and how marked they might be) can only be determined by recording simultaneously from a number of cells, but they are not intrinsically unlikely: at least in anaesthetized animals the activity of neighbouring olive cells shows a significant degree of synchronization, perhaps because there is electrotonic coupling between them (see for example Llinás & Yarom, 1981; Bloedel & Ebner, 1984).

Finally, as mentioned in Results, some observations suggested that the probability fluctuations in the step histograms are not of major functional significance. In future it may be possible to record for longer and determine whether progressive increase in the number of steps sampled results in progressive diminution in the fluctuations, as might be expected if they are due entirely to random 'noise'.

As presented, our results do not exclude that c.s.s might occur at a fixed time in the step cycle for a few steps and subsequently shift to coincide with another part of the cycle. However, such episodic time-locking was not detected when the data were examined on a step-by-step basis.

*C.s.s discharged in relation to perturbations of ladder locomotion*

Our most interesting finding was that some b zone P cells discharged a c.s. on some of the occasions when one ladder rung unexpectedly gave way. The responses were closely time-locked to the onset of rung movement but not to its cessation; indeed the responses often preceded the latter event. They cannot therefore have been generated by any excitation of mechanoreceptors brought about by jarring imparted to the limb as the rung ceased to move. Moreover, none of the cells exhibited any later c.s. attributable to such a stimulus. It is also unlikely, in view of the different patterns of limb specificity shown by the different cells, that the c.s.s were elicited by vestibular input arising from any head movements which may have resulted from rung descent.

It is not known precisely what mechanism was activated to give rise to the c.s.s but most probably they were due to impulses ascending from the spinal cord to the inferior olive (and thence to the cerebellum via the olivo-cerebellar projection). That being the case it is important that there were responses to perturbations of both the fore- and hind limb, sometimes in the same P cell. Three spino-olivo-cerebellar paths (s.o.c.p.s) terminate in the b zone (see Andersson & Eriksson, 1981), mediated

respectively via the dorsal, dorsolateral and ventral funiculi of the spinal white matter (d.f.-, d.l.f.- and v.f.-s.o.c.p., respectively). The first of these transmits inputs only from hind-limb nerves (in experiments involving electrical stimulation of peripheral nerves) so it is not likely to have made a major contribution to the responses. Similarly, the d.l.f.-s.o.c.p. conveys only ipsilateral input so, by exclusion, it is probable that traffic in the v.f.-s.o.c.p. was mainly responsible. When this bilateral path is activated by nerve stimulation in acute preparations the shortest latency c.s responses in the b zone have latencies of 16 and 19 ms for the ipsilateral and contralateral forelimbs respectively and 17 and 18 ms for the ipsilateral and contralateral hind limbs. Because some of the perturbation-related c.s.s had latencies as short as 40 ms and because descent of the movable rungs typically required over 100 ms for completion it is clear that quite small displacements must sometimes have elicited responses and therefore that the v.f.-s.o.c.p. is capable of reporting to the cerebellum the occurrence of quite small perturbations of the limb trajectory (and, by implication, quite small mismatches between intended and achieved movement). In future studies it would be worthwhile to vary the range of rung movement in order to determine the minimum disturbance needed to evoke responses.

The v.f.-s.o.c.p. latencies quoted above for nerve stimulation are obviously very similar for the fore- and hind-limbs (probably due to a slower conduction velocity in the spino-olivary axons mediating forelimb responses). It is therefore not clear why, in three cells which responded to hind-limb as well as forelimb displacements, the latency was substantially greater in the former case. It is possible that, when natural mechanical rather than electrical stimuli are involved, segmental delay is greater at the lumbosacral level. However, another factor may have been that perturbations were often smaller for the hind limb, as evidenced by the fact that the rung often descended more slowly and quite frequently failed to reach the mechanical stop (cf. Figs. 4*A* and 5).

In awake monkeys startled by sudden loud sounds c.s.s were evoked with high probability in almost 50% of P cells in lobule VI (lobulus simplex) and in the paramedian lobule and dorsal paraflocculus (Mortimer, 1975). Latencies ranged from 16 to 70 ms and the possibility must therefore be considered that the c.s.s we observed were a component of a generalized startle reaction. However, such reactions are accompanied by brief e.m.g. responses with latency 16–18 ms which are widely distributed among extensor and flexor muscles. In the present experiments, inspection of the e.m.g. traces revealed no such responses, nor did we observe behavioural signs of startling such as eye blinks or pinna twitches. In some trials the animal paused on the depressed rung and this might be indicative of a startle. However, c.s.s occurred not only in these trials but also in others in which there was no overt change in the over-all gait pattern. Moreover the c.s. usually showed limb specificity.

*Information carried by climbing fibre pathways*

There was no precise correlation between the limb specificity shown by individual cells and the location of their c.s. receptive fields. It is therefore likely that, although peripheral input evoked by rung movement triggers activity in the spino-olivary neurones of the v.f.-s.o.c.p., what these are doing is not directly reflecting peripheral input but rather some consequence it has for the pattern of activity existing within a spinal interneuronal network. This accords well with the suggestion that some s.o.c.p.s (including the v.f.-s.o.c.p.) seem designed not to transmit peripheral infor-

mation but to report on the excitability of particular motor mechanisms in the spinal cord, these latter being envisaged as comprising functional groups of spinal interneurones (e.g. Andersson & Sjölund, 1978; Sjölund, 1978).

In this connexion it is of great interest that Oscarsson has put forward the hypothesis (Miller & Oscarsson, 1970; Oscarsson, 1980) that olivary neurones which are relays on s.o.c.p.s may serve to inform the cerebellum that movement control errors have occurred. This hypothesis assumes that the olivary neurones monitor commands from higher motor centres (via the abundant descending connexions they receive), the activity these commands evoke in lower (i.e. spinal) motor circuits and the peripheral inputs resulting from the commanded movements. By somehow comparing these inputs 'the olive might detect perturbations of the commands introduced in the lower centre by reflex activity and perturbations of the evolving movement due to unexpected changes in load or resistance'. Inasmuch as our responses were associated with a developing unexpected mismatch between the intended and the achieved trajectory of a stepping limb they are in precise accord with this hypothesis, though obviously they do not prove its correctness. This interpretation perhaps receives additional support from the findings in relation to cell 197 (Fig. 9). In this case responses related to the ipsilateral hind limb occurred only when the limb was placed on the movable rung and not when prior perturbation of the forelimb induced the animal voluntarily to step over the movable rung and place the hind foot on the firm support provided by the next rung.

It might, of course, be objected, because no one P cell responded on every occasion that a particular limb was perturbed, that the individual c.f.s failed to provide a reliable mismatch or error signal to the cerebellum. However, there was some variation in walking speed (and doubtless also subtle variation in other locomotor parameters) both between different passes along the ladder and between individual paces. As a result it is highly probable that the nature and extent of the movement perturbation was not identical in every trial (vide the variations in Figs. 4 and 5 in the time for completion of rung descent). Viewed in this light it is not surprising that cells responded on some occasions but not on others. Equally, if different c.f.s signal different aspects of the movement error, then the differences in response probability between cells are again unsurprising.

That there were cells which responded to perturbation of the hind limb is of interest. An appreciable time lag (usually over 400 ms) existed between forelimb and hind-limb placement onto the same rung and it might have been expected the animals would learn that when a forelimb disturbance was experienced a similar disturbance to the hind limb would follow. Indeed in the animal from which cell 197 was recorded it is clear that the forelimb perturbation was sometimes used to cue an adjustment to the locomotor activity of the hind limb. All that can be said at present is that in the other two animals (and often in that animal) hind-limb avoidance strategies were not used and hind-limb-related responses were present. Perhaps the disturbance of the forelimb was not regarded by the animal as imperilling its progress sufficiently to warrant an anticipatory correction by the hind limb: a hind-limb perturbation was therefore allowed to occur (and was signalled to the cerebellum).

The present findings can be compared with those of Gellman, Gibson & Houk (1985) who recorded from olivary neurones in awake cats and found that, although

many cells were exquisitely sensitive to tactile stimuli delivered to the passive animal, they failed to respond to similar stimuli generated as a result of active movements. This parallels our finding that, in neurones with input from peripheral mechanoreceptors, steady locomotion was accompanied by no appreciable time-locking of c.s.s to the step cycle. Gellman *et al.* (1985) also found that nine out of ten cells with tactile receptive fields *did* respond with quite high probability to unexpected contact with obstacles during a volitional movement of the forepaw. They suggested that tactile-related olive cells may function as detectors of 'unexpected (i.e. externally imposed) somatic events'. Clearly, this suggestion has much in common with the view that some cells can function as detectors of unexpected perturbations of movements.

It is also worthwhile to compare the present results with two studies (Harvey *et al.* 1977; Gilbert & Thach, 1977) in which volitional movements of the monkey forelimb were perturbed by unexpected changes in the load on the contracting muscles. In one case (Harvey *et al.* 1977) brief (8 ms) increases in load were applied, sufficient to jerk the limb through 8–10 mm of displacement. This failed to evoke c.s.s in any of 120 P cells, most of which were in the paravermal part of lobule V. In the study by Gilbert & Thach (1977) a lever held by the monkey was repeatedly displaced by a change in load and active wrist movements were made to restore the lever to the original position. When the magnitude of the displacing force was unexpectedly altered, an increase in c.s. frequency occurred during subsequent trials in seventeen out of twenty-eight task-related P cells and these responses persisted for approximately the same number of trials as were needed for the monkey to adapt to the novel load. Many of these additional c.s.s occurred with latencies between 50 and 150 ms and (as in the present experiments) they were present in some trials and absent in others. Why c.s.s occurred in the experiments of Gilbert & Thach (1977) and not in those of Harvey *et al.* (1981) is unkown, but it would seem that externally imposed perturbations of ongoing movements can evoke c.s.s both during locomotion (the present study) and during volitional movements of the forelimb (Gilbert & Thach, 1977).

*Climbing fibres and movement control*

Our results imply that 40–120 ms after the onset of rung movement c.s.s will occur near-synchronously in a substantial population of b zone P cells. Taking as an example the responses to perturbation of the ipsilateral forelimb, 'extra' c.s.s occurred in eight out of seventeen P cells with an average probability of 0·43. It has been estimated (Andersson & Oscarsson, 1978*a*) that there are 36000 b zone cells so that in an average trial an extra c.s will occur in approximately 8800 P cells (mostly in the 50–100 ms period). Similar changes involving different (but overlapping) populations of cells will accompany perturbations of the other limbs. Uncertainty still exists as to whether a single c.s. results in one spike or a short burst of spikes propagating down the P cell axon (see Ito, 1984 for references) but, whichever is the case, it is likely that a powerful inhibitory action will be exerted on the *ca.* 1100 lateral vestibular nucleus neurones which are the targets of the b zone P cells. This will presumably be preceded by an excitation because the c.f.s supplying the b zone also provide excitatory collaterals to the Deiters' neurones (see Ito, 1984). These influences (together with any which reach the neurones via spino-vestibular fibres,

via collaterals of spino-cerebellar mossy fibres and via mossy fibre inputs to the b zone P cells), will presumably produce perturbation-related changes in the pattern of traffic in the lateral vestibulospinal tract which we hope to study in future experiments.

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