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RELATION BETWEEN RED NUCLEUS DISCHARGE AND MOVEMENT PARAMETERS IN TRAINED MACAQUE MONKEYS

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SUMMARY

1. Correlation and regression analyses were performed on thirty-three of the magnocellular red nucleus cells described in the previous paper. We sought to test for reliable relations between the parameters of individual tracking movements and corresponding bursts of neural discharge.

2. High correlations were found between the following burst and movement parameters: (i) burst latency *versus* movement latency; (ii) burst duration *versus* movement duration; (iii) burst frequency *versus* movement velocity and (iv) number of spikes in the burst *versus* movement amplitude. Cells were ranked according to the average of the duration, velocity and amplitude correlation coefficients. The top twenty cells had average correlation coefficients ranging from 0·69 to 0·88 for their preferred movement. These cases were judged most likely to reveal the control functions of the red nucleus, and the following points refer to this sample.

3. Burst onset led movement onset by 118 ± 23 ms, and burst offset led movement offset by 50 ± 38 ms. Burst duration increased as the duration of the movement increased ($r = 0{\cdot}87 \pm 0{\cdot}11$). The duration of the burst was approximately equal to movement duration (slope of $0{\cdot}99 \pm 0{\cdot}16$) plus a constant ($72 \pm 34$ ms) throughout a broad range.

4. Average discharge rate during the burst increased with average movement velocity ($r = 0{\cdot}69 \pm 0{\cdot}15$). The slope of the relation was $0{\cdot}36 \pm 0{\cdot}21$ (pulses/s)/(deg/s) of joint rotation. The regression lines had consistent upward offsets (56 ± 15 pulses/s) that exceeded the spontaneous discharge rate (17 ± 10 pulses/s).

5. The number of spikes in the burst increased with movement amplitude independent of velocity ($r = 0{\cdot}72 \pm 0{\cdot}11$). The slope of the relation was 0·62 spikes/deg and the offset was 13 ± 4 spikes.

6. The preferred movement was co-ordinated hand in fifteen cases, digit in three, elbow in one and shoulder in one. When these cells were tested with an alternate movement, the failure rate (cases in which a burst did not accompany a movement) increased from 1·4 to 20%, and the correlation coefficients generally were low and lacked significance.

7. Cells in the top twenty had directionally specific responses, low variance in lead time, large depths of modulation (41–118 pulses/s) and low failure rates. Cells that failed to show strong parametric correlations often had one or more of the former

attributes. It appears that high parametric correlations with individual movements are particularly restrictive criteria of relatedness.

8. The introduction of a spring load (tested for nine cells) did not affect discharge rate.

9. The results are consistent with the hypothesis that single cells in the magnocellular red nucleus control the onset, velocity and duration of specific movements. Within the upper-limb zone, hand and finger movements are particularly well represented. The signals transmitted by these cells are probably generated by endogenous C.N.S. mechanisms rather than by continuous feed-back from the periphery.

INTRODUCTION

The previous paper dealt with the question of which types of movement the magnocellular red nucleus (r.n.m.) is likely to control. We found that single cells discharge in bursts, and that the frequency of action potentials in a burst varies with the type of movement. Modulations in discharge rate in excess of 50 pulses/s were not uncommon when the monkey subject made a particular type of movement, designated the preferred movement for that cell. For upper extremity cells the preferred movement for eliciting high frequency activity usually involved the hand and finger, but for some cells it involved more proximal joints. Cells were often weakly active or inactive for alternate movements. For some cells we apparently failed to define a preferred movement since the depths of modulation were low on all of the tested tracking devices even though high frequency discharge occurred during free-form-tests.

The purpose of the present paper is to describe the dependence of r.n.m. discharge on parameters of movement such as speed, amplitude and duration. Our working hypothesis is that this information would be most meaningful if it were obtained for cells that are well related to the particular movements that we studied. Correspondingly, we have restricted our sample to thirty-three of the most promising cells for which quantitative data was available, during tracking performance on a preferred device. We have extensively analysed this sample to seek out reliable relations to parameters of individual tracking movements, and we have compared performance on preferred and non-preferred devices for some of these cells.

The relation between single unit discharge and movement parameters has not been studied extensively for r.n.m. neurones. In the cat, temporal correlations between discharge rate and velocity were reported by Burton & Onoda (1978) and between discharge rate and rate of change of force by Ghez & Vicario (1978). In the monkey, two groups have found discharge to be more strongly correlated with the dynamic than static component of movement (Cheney, 1980; Fromm, Evarts, Kroller & Shinoda, 1981), but no attempt was made to describe parametric relations.

Our results indicate that a subset of r.n.m. neurones show very strong correlations with detailed features of movement. Cells discharge in bursts that precede each movement, burst duration correlates with movement duration, discharge rate correlates with movement velocity, and the number of spikes in the burst correlates with the amplitude of movement. These findings suggest that single cells in the r.n.m. code movement velocity.

METHODS

The procedures for training monkeys to operate several manipulanda and for simultaneous single cell recording in the r.n.m. are described in the previous paper (Gibson, Houk & Kohlerman, 1985). In addition to the step tracking task described there, we trained the monkeys to make slow, continuous movements by rewarding them for staying within the target window while it was moved at a low velocity. These ramp tracking movements usually consisted of several constant velocity segments that could be analysed individually. Similarly, the animals moved at different speeds in a high velocity range on successive step tracking trials, and they made small secondary movements when the primary movements failed to fall within the target zone. These sources of variation in the time course of movement facilitated our analysis of correlations between bursts of r.n.m. discharge and movement parameters.

Fig. 1 shows an example of the measurements performed for a simple case of step tracking in which the animal acquired the target with a single primary movement (lower traces in each of the panels). Panel *A* shows a plot of instantaneous frequency, and panels *B–D* show various treatments of the corresponding integrated spike plot. The latter was derived from an interspike interval record collected during an individual trial. A computer algorithm counted the number of discharges in successive 10 ms bins and plotted the cumulative spike count as a function of time (Gibson *et al.* 1985). The cusum (Ellaway, 1977) in panel *D* is simply an integrated plot from which initial discharge rate was subtracted. Panel *B* shows how the time of onset and offset was measured for both the movement and the burst of r.n.m. discharge. Panel *C* shows how the slope of the integrated spike trace and the slope of the movement trace were used to estimate average discharge rate and average velocity for a movement–response pair. Panel *C* also illustrates how the number of spikes in a burst was measured for comparison with movement amplitude.

Fig. 2 shows some more complex examples of movement–response pairs. In general, we were able to record the burst parameters for each 'on'-direction movement segment for all the trials in a data file. However, segments with slowly accelerating onsets or offsets or ones with very small amplitudes or durations presented measurement problems and were excluded on this basis. Also, we excluded trials with erratic behaviour that was either noted at the time of data collection or was recognized later by unsteady records in the pre-stimulus intervals or movement patterns suggesting the animal had abandoned the tracking task. Trials in which movements were not accompanied by a burst in discharge were designated failures and listed separately in Tables 1 and 2.

Scatter plots relating neural and movement parameters were constructed for each cell. Typically, five comparisons were made: burst onset *versus* movement onset (reaction time); burst offset *versus* movement offset; duration of the burst *versus* duration of the movement; average discharge rate during the burst *versus* average movement velocity, and the total number of spikes in a burst *versus* movement amplitude. Values of the Pearson product moment correlation coefficient (r) were calculated for each of the five relations.

Regression analysis was used to assess the dependence of the neural response on the parameters of the movement. The interpretation of the regression model is compromised by the fact that variations in both the neural response and the movement were dependent on the behaviour of the animal. Thus, there was no independent variable that was rigidly controlled by the experimenter. Nevertheless, we considered it worthwhile to relate the neural response to the onset, duration, velocity and amplitude of movement. The movement variable was designated as independent since the movement produced generally matched the target movement which was controlled. The relation was estimated by the value of the slope of the regression equation and the ordinate intercept.

RESULTS

Some of the cells reported on in the previous paper showed very close relations between discharge rate and the detailed time course of individual movements. Fig. 2 shows examples of variations in the time course of movement and corresponding variations in the pattern of r.n.m. discharge for three cells. Brief bursts of activity preceded rapid movements whereas longer bursts preceded slower movements (compare discharge rate and position traces in Fig. 2*A* and *B*). Sometimes the

primary movement fell short of the target, and the monkey made one or more secondary corrective movements. Each movement in the 'on' direction, whether primary or secondary, was preceded by a burst of activity, and the strength of the burst appeared to correspond to the amplitude of the movement (Fig. 2*C* and *D*).

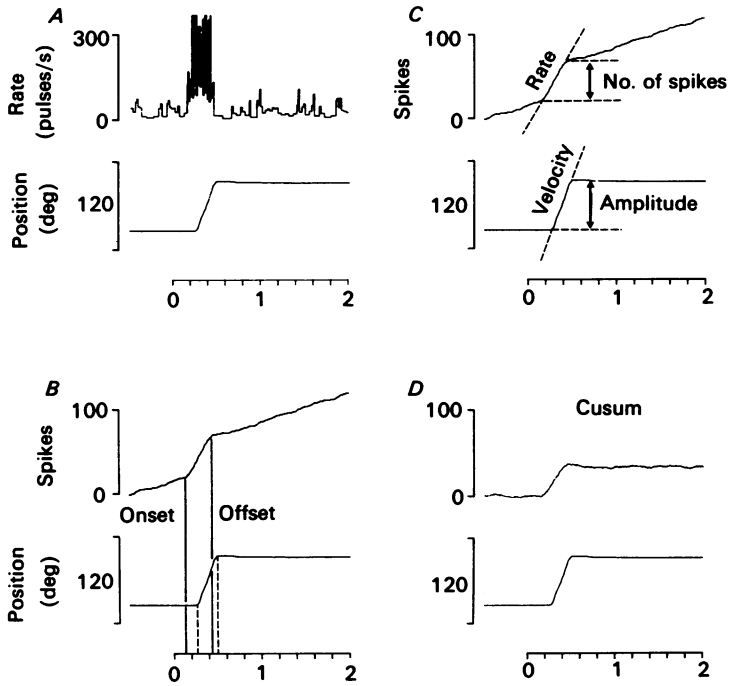

Fig. 1. Comparative measures of the parameters of movement and of r.n.m. discharge. Panels *A–D* show different treatments of an individual movement–response pair for cell H63. In *A*, an instantaneous frequency plot shows a burst of discharge preceding anticlockwise twister rotation. In traces *B–D* the instantaneous frequency plot is replaced by the integrated spike plot for the same trial. In *B* the onset and offset of the burst in neural discharge are indicated by inflexions in the integrated spike record (continuous lines) and the onset and offset of the movement are indicated by inflexions in the movement trace (dashed lines). In *C* discharge rate and movement velocity are indicated respectively by the slopes of the integrated spike and movement records, and number of spikes in the burst and movement amplitude are given by ordinate measures. In *D* the integrated spike record is replaced with a cusum calculated by subtracting initial discharge rate.

In order to encourage movements of lower velocity and longer duration, we trained the animals to track ramp targets (see Methods). Ramp tracking was frequently fragmented into several constant velocity segments, and transitions from one velocity to another were then preceded by corresponding changes in discharge rate (Fig. 2*F*).

Correlation analysis was performed on a sample of thirty-three cells with properties similar to those just described. These were selected from a group of eighty-one neurones that were studied during tracking (Gibson *et al.* 1985). Cells included in the analysed sample all had depths of modulation greater than 40 pulses/s. As a further

screening procedure, we scanned the computer records collected during tracking performance and eliminated cells that had high failure rates or frequent bursts of discharge not accompanied by movements and otherwise not showing high consistency between bursting and movement. The initial purpose of the correlation analysis was to find the best related cells, and we consider it unlikely that our screening process excluded any highly correlated cases.

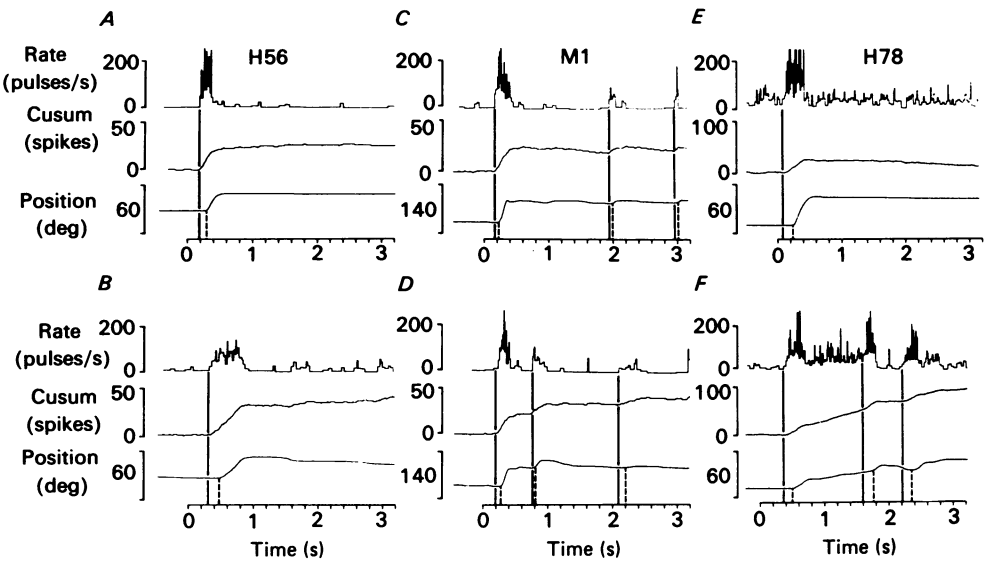

Fig. 2. Examples of movement–response pairs for three cells. In each panel, records of instantaneous frequency (top), cusum (middle) and position (bottom) can be compared. *A* and *B* compare the responses of cell H56 during clockwise twister rotation at two velocities. Note that burst frequency is lower and burst duration is longer for the slower movement shown in panel *B*. *C* and *D* illustrate that bursts which are appropriately graded in intensity precede the onsets of both primary target acquisition and secondary corrective movements for cell M1 during metacarpal extension. *E* and *F* compare the brief burst preceding a step movement with an extended burst accompanying a ramp movement for cell H78 during clockwise rotation of the twister. Note the correspondences between the cusum plot and the corresponding movement traces.

Latency analysis

The onsets of bursts of neural discharge were found to correlate closely with the onsets of corresponding movements and movement segments throughout a wide range of reaction times, as illustrated for cell H78 in Fig. 3*A*. The open symbols represent primary movements and closed symbols secondary movements taken from both step and ramp tracking trials. Regression lines fitted through points such as these always had slopes close to 1·0 and fell below the line of simultaneity, shown dashed in Fig. 3*A*. This indicated that burst onset led movement onset by a relatively constant lead time. The lead time was the same when the animal's movement represented an error in tracking as it was for a correct response.

There was also a close correlation between burst offset and movement offset, or

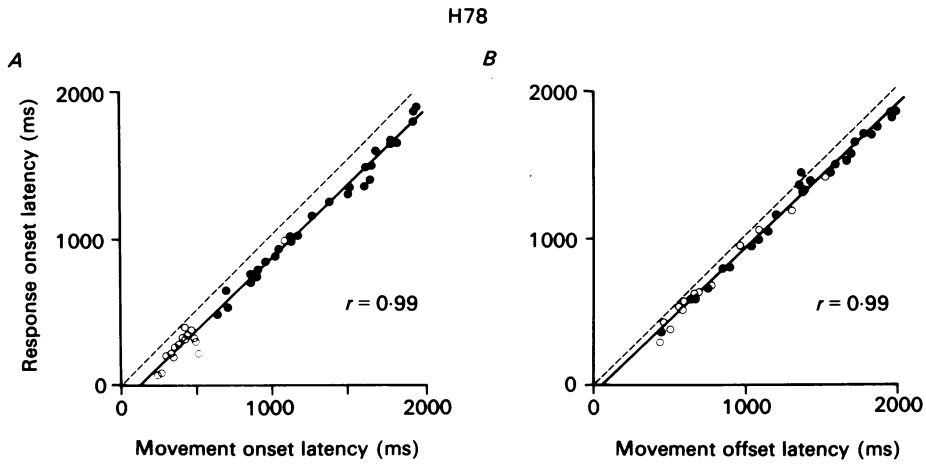

Fig. 3. Burst latency is plotted as a function of movement latency for cell H78 studied during clockwise twister rotation. *A*, each point relates burst onset to movement onset for either primary (open circles) or secondary (closed circles) movements. *B*, the timing of burst termination (offset) is plotted as a function of movement offset for the same burst–movement pairs represented in panel *A*. All but one of the points fall below the line of simultaneity (dashed diagonal line) which indicates that burst onset and offset lead movement onset and offset respectively.

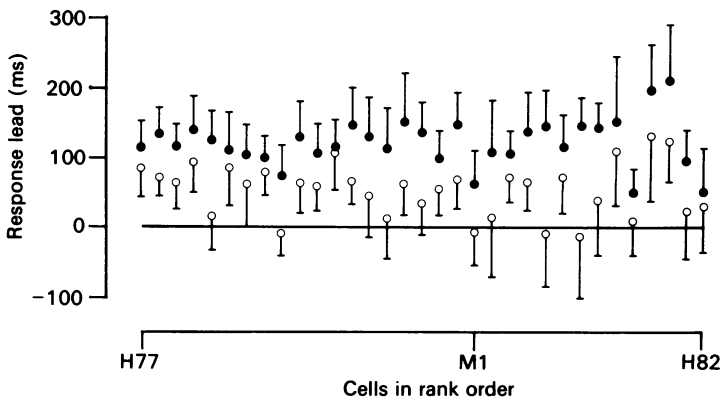

Fig. 4. Mean lead times and standard deviations for thirty-three cells. The cells are arranged along the abscissa in rank order according to the value of the average correlation coefficient (see Table 1). Filled points indicate mean lead times for burst onset with respect to movement onset and open circles indicate lead times for burst offset with respect to movement offset. Error bars represent one standard deviation.

the end of a movement segment, as shown for the same cell in Fig. 3*B*. Usually the response offset led movement offset, but for some units there were an appreciable number of trials in which response offset lagged.

Fig. 4 compares the mean lead times of burst onset and offset for the sample of thirty-three neurones selected for quantitative analysis. The cells are arranged in rank

order based on their average correlations with movement parameters (Table 1), a criterion of relatedness that emerges from the data presented later. Units with the better parametric relations (on the left) showed less variability in lead times both individually (standard deviations) and as a group (left to right trend in Fig. 4). None

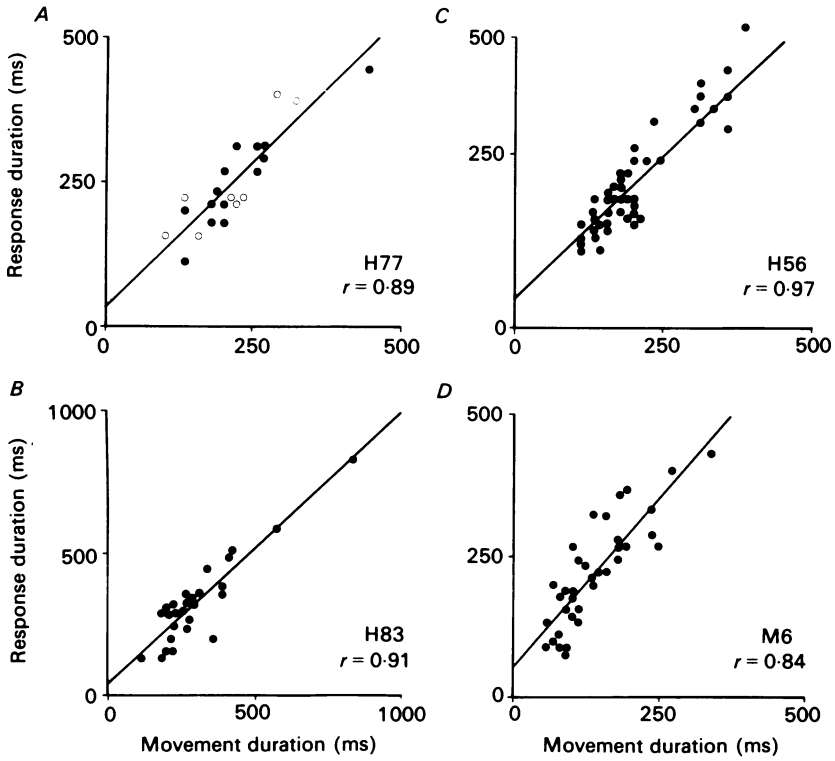

Fig. 5. Scatter plots showing the change in burst duration as a function of movement duration for four well-related r.n.m. cells. Cell numbers and correlation coefficients (r) are as indicated. In A the slope of the regression line was 0·99 and the ordinate offset was 34 ms; open circles represent cases where the movement opposed an elastic load. In B, slope is 0·95 and offset is 44 ms; note time scale is different for this panel. In C, slope is 0·99 and offset is 51 ms; one point (1110, 1180) is off scale. In D, slope is 1·2 and offset is 53 ms.

of the cells had mean onset lags, and only a few had offset lags. The population statistics for the top twenty cells (H77–M1 in Table 1) revealed a 118 ± 23 ms onset lead and a 50 ± 38 ms offset lead.

Duration correlations

The durations of the individual bursts of discharge were found to be highly correlated with the durations of the corresponding movements. Scatter plots of burst *versus* movement duration for four cells are shown in Fig. 5, and the correlation coefficients obtained for the entire quantitative sample are listed in Table 1. Cases

TABLE 1. Parametric correlations for thirty-three r.n.m. cells studied on the preferred device

Cell	Dev.	Dir.	n	Failures	r(dur.)	r(vel.)	r(amp.)	r(avg.)	D.o.m.	D.s.
H77	Tw.	C	22	0	0·89	0·90	0·86	0·88	83	0·92
H80	Tw.	AC	10	1	0·78	0·97	0·86	0·87	80	0·94
H81	Tw.	AC	34	0	0·82	0·76	0·84	0·81	118	0·72
H63	Tw.	AC	14	0	0·74	0·90	0·76	0·80	101	1·18
M17	Tw.	C	24	0	0·90	0·71	0·78	0·80	41	0·82
H83	El.	F	28	0	0·91	0·78	0·71	0·80	97	0·59
H54	Tw.	C	28	3	0·97	0·76	0·61	0·78	98	0·96
H69	Tw.	AC	14	0	0·95	(0·48)	0·88	0·77	90	0·76
M6	Met.	E	37	0	0·84	0·60	0·84	0·76	74	0·63
H78	Tw.	C	47	0	0·95	0·71	0·63	0·76	118	1·00
H56	Tw.	C	49	0	0·97	0·70	0·58	0·75	81	0·74
H62	Sh.	F	10	0	0·88	0·64	0·67	0·73	83	0·57
H120	Tw.	AC	14	0	0·90	0·57	0·68	0·72	91	1·08
M18	Tw.	C	16	0	0·73	0·79	0·64	0·72	64	0·56
M16	Tw.	C	30	0	0·98	0·67	0·51	0·72	57	1·00
H50	Tw.	C	38	0	0·92	0·56	0·66	0·71	76	1·00
H104	Tw.	C	16	0	0·91	0·52	0·66	0·70	94	1·12
M37	Ph.	E	11	1	0·92	(0·47)	0·71	0·70	57	0·46
H60	Tw.	AC	22	0	0·86	0·47	0·75	0·69	96	1·15
M1	Met.	E	32	2	0·50	0·77	0·81	0·69	112	0·56
H91	Tw.	C	17	0	0·89	0·80	(0·37)	0·69	93	0·77
H82	Fi.	F	19	0	0·79	0·66	0·52	0·66	56	0·50
H92	Tw.	C	23	0	0·69	0·84	0·43	0·65	82	1·00
H90	Tw.	AC	20	0	0·75	0·60	0·55	0·63	77	0·82
M35	Tw.	C	38	4	0·89	0·38	0·59	0·62	96	0·56
H116	Tw.	C	32	2	0·92	(0·28)	0·67	0·62	89	0·61
H84	Fi.	F	20	1	0·78	(0·22)	0·76	0·59	87	0·27
H48	Tw.	C	24	0	0·97	(0·13)	0·63	0·58	72	0·88
M14	Met.	E	51	12	0·80	0·31	0·60	0·57	57	0·98
M31	Ph.	F	33	1	0·64	0·61	0·46	0·57	54	0·75
H73	Tw.	C	13	0	0·62	0·61	(0·39)	0·54	41	0·39
H64	Tw.	AC	15	6	0·78	(0·19)	(0·51)	0·49	118	0·88
H106	Met.	E	78	67	(0·02)	0·61	0·48	0·37	97	0·83

Columns: cells, listed in rank order based on r (avg.); device (Dev.) on which the cell was studied, namely twister (Tw.), elbow (El.), metacarpal (Met.), shoulder (Sh.), phalangeal (Ph.) or finger (Fi.); preferred direction (dir.) for high correlation, clockwise (C), anticlockwise (AC), flexion (F) or extension (E); number of movement–response pairs analysed (n); number of failures (no responses though movement occurred); correlation coefficients (r) for duration, velocity, amplitude and the average of the three, with parentheses designating cases in which the correlation was not significant at the $P < 0·05$ level; depth of modulation (d.o.m.) in pulses/s; index of directional selectivity (d.s.) where 0 is a bidirectional response, 1·0 is a unidirectional response and larger values indicate reciprocal responses (cf. Gibson *et al.* 1985).

in which there was no burst of discharge in association with a movement, designated 'failures', were not included in this analysis. Table 1 shows that there were very few failures for the top units. Generally speaking, the correlation coefficients are high; and the average value for the top twenty cells is $0·87 \pm 0·11$. All but the single case (not in the top twenty cells) shown in parentheses in Table 1 are significant at the $P < 0·05$ level.

While burst duration correlated closely with movement duration, the two were not

equal. For all thirty-three cells, regression lines fitted to the data had upward offsets, though the slopes were usually close to unity. For the top twenty cells, the average offset was 72 ± 34 ms and the average slope was $0{\cdot}99 \pm 0{\cdot}16$. Thus, burst duration averaged 72 ms longer than movement duration throughout the range studied.

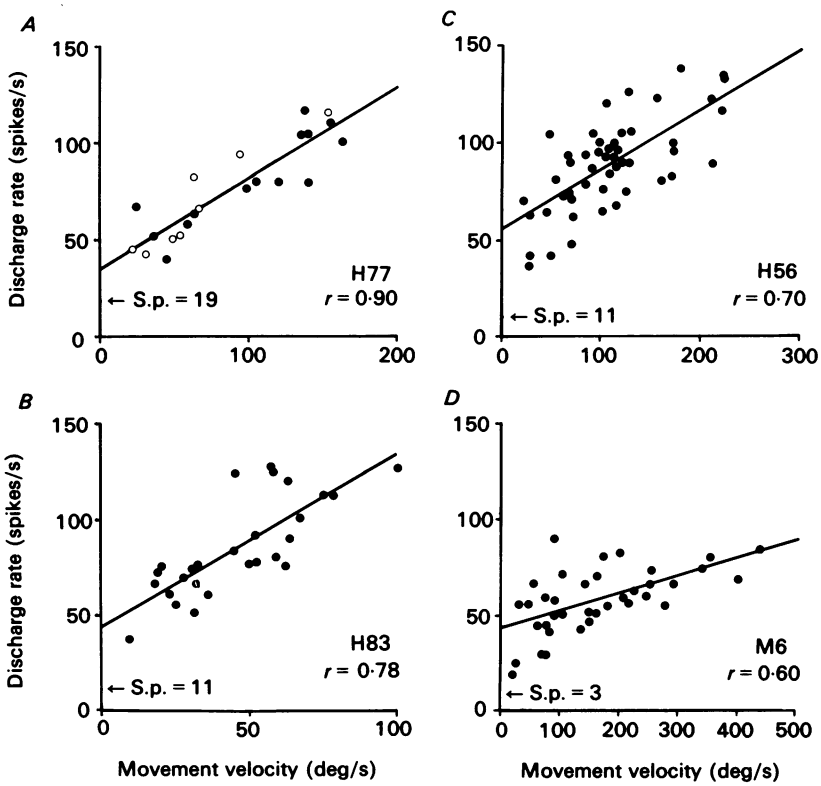

Fig. 6. Scatter plots showing average discharge rate as a function of average movement velocity for the four cells whose duration plots were shown in Fig. 5. In *A*, the slope of the regression line is 0·47 (pulses/s)/(deg/s) and the ordinate offset is 35 pulses/s; s.p. indicates spontaneous rate; open circles are spring-loaded trials. In *B*, slope is 0·90 (pulses/s)/(deg/s) and offset is 44 pulses/s. In *C*, slope is 0·31 (pulses/s)/(deg/s) and offset is 55 pulses/s. In *D*, slope is 0·09 (pulses/s)/(deg/s) and offset is 44 pulses/s.

Correlation between discharge rate and velocity

The bursts of discharge that preceded rapid movements appeared to be more intense than the bursts that preceded slow movements, as illustrated for cell H56 in Fig. 2*A* and *B*. Similarly, when the animal was engaged in ramp tracking, transitions from one velocity to another were preceded by corresponding transitions in discharge rate (Fig. 2*F*). These observations suggested that discharge rate might correlate closely with movement velocity if tested on a trial-by-trial basis.

Burst frequency was difficult to estimate from the instantaneous frequency records due to variability, but reliable estimates were obtained by fitting slopes to integrated

spike records, as described in the Methods. Movement velocity was estimated in an analogous manner by fitting slopes to the movement traces. Fig. 6 shows examples of the rate–velocity scatter plots obtained from these measures. The four cells represented here are the same ones represented in the duration plots of Fig. 5 for convenience of comparison. The correlation coefficients obtained for these cells and the other twenty-nine in our sample are listed in Table 1. The values are generally high, and they are significant at the $P < 0{\cdot}05$ level in all but six cases. The average velocity correlation for the top twenty cells is $0{\cdot}69 \pm 0{\cdot}15$.

Although discharge rate correlated well with velocity, the relation between these variables was not strictly proportional. Regression lines fitted to the data had consistent upward offsets that exceeded the spontaneous discharge rate of the cell in every case. Mean values based on the top twenty cells indicate that the average offset was 56 ± 14 pulses/s as compared with an average spontaneous rate of 17 ± 10 pulses/s. The average slope of the regression line was $0{\cdot}36 \pm 0{\cdot}21$ (pulses/s)/(deg/s).

Correlation between spike count and movement amplitude

The findings already presented indicate that bursts of r.n.m. discharge (i) have onsets that precede movement; (ii) have durations that increase with movement duration and (iii) have discharge rates that increase with movement velocity. This suggested that movements of a given amplitude might be preceded by a specific number of spike discharges independent of movement velocity. For example, a rapid 20 deg movement might require a brief, high frequency burst whereas a slow movement of the same amplitude might require a long burst at a low frequency, but the number of spikes in the two bursts might be the same. Comparison of the movement and cusum traces in Fig. 2 reveals such a relation.

The cusum traces were constructed from interspike interval records by summing the number of spikes minus a constant rate representing spontaneous discharge in the pre-stimulus interval (see Methods). Thus, they count up the number of extra spikes above spontaneous which result from bursting. The cusum in panel *A* of Fig. 2 shows that twenty-three extra spikes occurred in the burst that preceded a rapid 22 deg movement. In panel *B* the same neurone discharged thirty-three extra spikes in a burst that accompanied a slower movement with a 29 deg amplitude. If one divides the number of extra spikes by the amplitude of movement, the number of spikes per degree of movement is found to be nearly constant (1·05 spikes/deg in *A versus* 1·14 spikes/deg in *B*). Comparing cusums in panels *C* and *D* indicates that a single movement of 63 deg was accompanied by twenty-four extra spikes whereas a 76 deg movement that took place in two steps was accompanied by thirty-two extra spikes. In spite of the rather different time course, the number of extra spikes per degree was again nearly constant (0·38 in *C* and 0·42 in *D*).

In order to test more thoroughly for an amplitude relation, scatter plots of the number of spikes in a burst *versus* movement amplitude were constructed and correlation coefficients were calculated. Fig. 7 shows the plots for the four cells represented earlier in duration and velocity plots, and the correlation coefficients for the sample of thirty-three cells are listed in Table 1. Pilot analyses indicated that correlations were no different when total number of spikes in the burst was used rather

than the number of extra spikes, so the former measure was selected because it was a direct rather than a derived value. The correlations are similar to those obtained from rate–velocity data. For the top twenty units the average correlation coefficient was $0{\cdot}72 \pm 0{\cdot}11$. Regression lines were also fitted to the data, and, for the top twenty cells, the average slope was $0{\cdot}62 \pm 0{\cdot}35$ spikes/deg and the average offset was 13 ± 4 spikes.

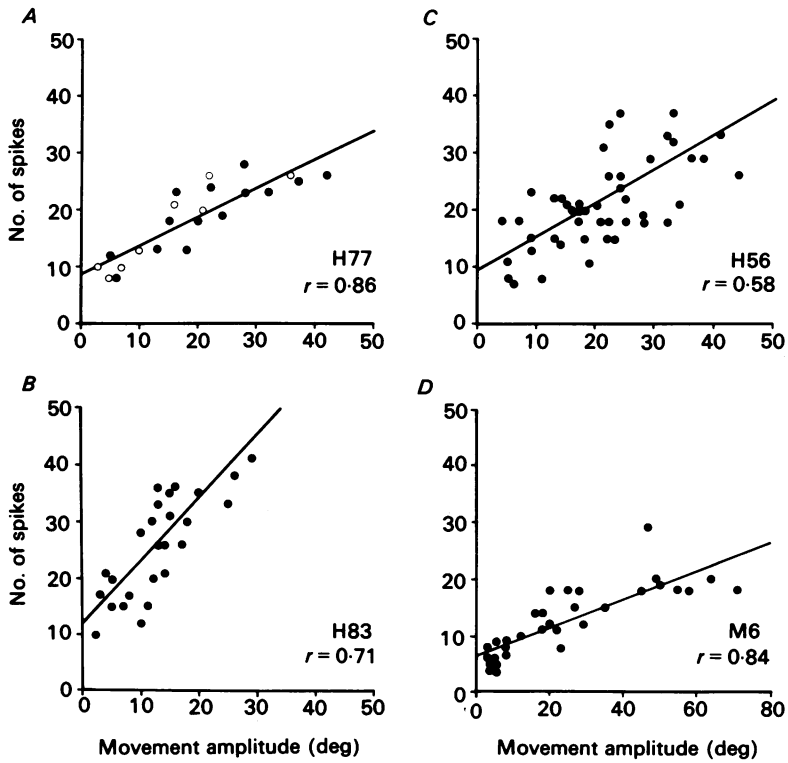

Fig. 7. Scatter plots showing the number of spikes in a burst as a function of movement amplitude for the same four cells presented in Figs. 5 and 6. In *A*, the slope of the regression line is 0·50 spikes/deg and the ordinate offset is 8·7 spikes; open circles are spring-loaded trials. In *B*, slope is 1·1 spikes/deg and offset is 12 spikes; one point (15, 56) was off the scale. In *C*, slope is 0·60 and offset is 9·6 spikes; one point (31, 70) is off the scale. In *D*, slope is 0·25 spikes/deg and offset is 6·5 spikes.

Rank order based on correlations

The cells in Table 1 are listed in rank order based on the average of the three correlation coefficients described in the preceding sections. While there is no compelling reason for this particular ranking system, cells with discharge patterns that correlate highly with three parameters of movement (velocity, duration and amplitude) seem more likely candidates for performing a causal role in controlling the movement under study than are cells showing poor correlations with one or more of these parameters. Correlation coefficients for response onset (Fig. 3) were not

included in this average because they were all close to 1·0 and therefore provided little basis for a critical ranking of cells. Onset correlations were also high for many cells that were judged poorly related to the movement based on more restrictive criteria. For the top twenty cells based on the chosen ranking, there are only two cases in which the correlation coefficients are not significant at the $P < 0{\cdot}05$ level. Also noteworthy is the low frequency of failures (cases in which the cell did not fire in association with a movement). Out of a total of 503 movement–response pairs, there were only seven failures (1·4 %).

The depths of modulation (d.o.m. in Table 1) for the top twenty cells exceed the 50 pulses/s criterion for relatedness used in the previous paper in all but one case; the mean value is 86 ± 20 pulses/s. However, not all cells with large modulations have high correlation coefficients when analysed parametrically, as is clear by examining the entries ranked below 20 (H91–H106 in Table 1). Many of the top twenty cells have indices of directional selectivity (d.s. in Table 1) close to unity, representing a unidirectional response. The mean value is $0{\cdot}84 \pm 0{\cdot}23$, and only one of the values is below 0·5.

Correlations with non-preferred movements

Sixty-five cells were studied while the animal operated more than a single device (Gibson *et al.* 1985). Of these, nineteen failed to respond during performance on any device even though all discharged vigorously when the monkey took food from the experimenter. Another seven responded only to one device. Observations in both categories are consistent with a high degree of specificity in movement relations.

Device specificity was assessed in detail for fourteen responsive cells. These cells were selected since each was well related to performance on at least one device, based on its ranking within the top twenty cells in Table 1. While Table 1 gives the results for performance on the preferred device, in the preferred direction, as judged by the average correlation coefficient, Table 2 presents the results obtained when the same cells were tested on alternate (non-preferred) devices. Table 2 lists a total of twenty-three non-preferred cases.

Most of the cases in Table 2 document a much lower degree of relatedness between cell discharge and the movements performed on an alternate device as contrasted with relations to performance on the preferred device (Table 1). One indication of this is an 11-fold higher failure rate (20 % as contrasted with 1·4 %). Another is the generally lower values of correlation coefficients and the absence, except in two cases, of statistical significance for the full complement of tests (duration, velocity and amplitude). We did not calculate correlation coefficients for ten cases in which fewer than ten trials were available unless the depth of modulation was high (50 pulses/s criterion level). However, these ten cases were characterized by a high failure rate and/or a low depth of modulation, indicating that discharge was not well related to the movement under study.

One can summarize the data in Table 2 as follows. There are seven cases (out of twenty-three tested) involving five cells (out of fourteen tested) in which activity was reasonably well related to performance on an alternate device. Here the main criterion of relatedness is an average correlation coefficient greater than 0·5. Overlap in movement relations occurred between the twister and a digit device for 5 (M17,

H54, M6, H78 and M37) of the six cells. One cell showed overlap between shoulder and elbow (H83) and another between finger, twister and elbow (H78). For the majority of cases and cells, discharge was poorly related to performance on an alternate device.

TABLE 2. Parametric correlations for top twenty cells studied on an alternate device

Cell	Dev.	Dir.	*n*	Failures	*r*(dur.)	*r*(vel.)	*r*(amp.)	*r*(avg.)	D.o.m.	D.s.
H77	Fi.	E	15	1	(0·22)	(0·09)	(0·20)	0·04	80	0·36
	El.	E	1	4	—	—	—	—	5	—
	Sh.	E, F	0	9	—	—	—	—	0	—
H80	Fi.	E	15	4	(0·21)	(0·22)	0·63	0·21	35	0·08
	El.	E	5	0	—	—	—	—	22	0·87
	Sh.	E	4	0	—	—	—	—	39	0·70
M17	Met.	E	22	0	0·90	0·80	0·44	0·71	47	0·47
H83	Tw.	AC	2	3	—	—	—	—	13	0·45
	Fi.	F	4	2	—	—	—	—	15	0·81
	Sh.	F	7	0	0·96	(0·63)	0·85	0·81	75	0·65
H69	Fi.	F	24	0	0·86	(0·09)	(0·29)	0·41	93	0·22
		E	16	2	0·57	0·54	(0·27)	0·28	73	—
H54	Kn.	C	7	3	0·97	0·95	(0·44)	0·79	102	0·72
H56	P.p.	Push	1	3	—	—	—	—	—	—
M6	Tw.	C	29	0	0·86	(0·23)	0·56	0·55	44	0·23
		AC	21	0	0·71	(0·05)	0·53	0·43	34	—
H78	Fi.	E	22	1	0·66	0·49	0·48	0·54	65	0·52
	El.	F	12	0	0·86	0·86	(0·45)	0·72	72	0·82
H62	Tw.	C	5	1	—	—	—	—	49	0·59
M18	Met.	E	8	2	—	—	—	—	41	1·00
H104	Wr.	E	3	4	—	—	—	—	9	—
M37	Tw.	C	21	1	0·81	(0·19)	0·77	0·59	124	0·81
M16	Met.	E	15	8	0·85	(0·06)	(0·45)	0·45	47	1·00

See legend to Table 1 for definitions. Devices not defined there are the knob (Kn.), push–pull (P.p.) and wrist (Wr.) which are described in the Methods section of Gibson *et al.* (1985).

Movements against loads

The twister and shoulder/elbow devices described in the previous paper included a provision for engaging a mechanical spring. This permitted a given neurone to be studied both in the presence and absence of an elastic load. E.m.g. control recordings indicated that spring loading of the twister resulted in an appreciable enhancement of forearm muscle activity, including a well-developed tonic component in the holding phase. In contrast, for the nine neurones (five of these were in the top twenty) in which loading was tested there was little if any effect on either the phasic or tonic features of neural response.

Fig. 8*A* shows an example in which a loaded and an unloaded trial are superimposed for comparison. The two movements are very similar in amplitude, velocity and duration, although the loaded case required an extra holding torque of 0·03 N m. There are no signs of changes in unit discharge, as indicated by the close similarity of the superimposed instantaneous frequency and integrated spike records. For this cell and two others for which a sufficient number of trials were available, parametric analyses were performed with and without loading. The open points shown in the

duration (Fig. 5*A*), velocity (Fig. 6*A*) and amplitude (Fig. 7*A*) scatter plots for cell H77 represent spring-loaded movements. These points fall within the range of the filled points which represent unloaded movements. For the three cases thus analysed, loading did not appreciably alter the correlation coefficients or the parameters of regression fits to the data.

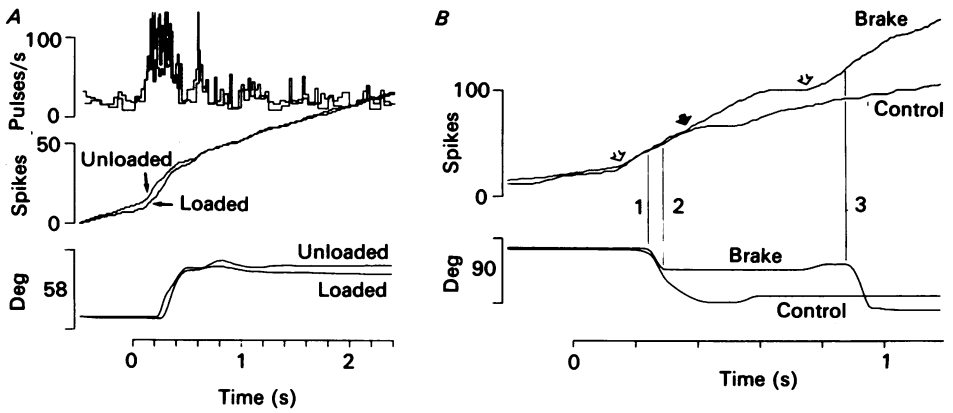

Fig. 8. Effect of loading and interrupting movements. In *A*, the two trials involve similar velocities and amplitudes of clockwise twister rotation (bottom traces) studied for cell H77. The superimposed instantaneous frequency plots (top) and integrated spike plots (middle) illustrate that r.n.m. activity was similar for loaded and unloaded trials. In *B*, a trial in which the movement was interrupted by a brake is compared with a control trial for cell H111 studied during anticlockwise twister rotation. The onsets of primary and secondary movements, indicated by vertical lines 1 and 3 respectively, were preceded by corresponding bursts in discharge (inflexions in integrated spike plots marked by open arrows). The interruption of movement by the brake (vertical line 2) elicited no change in discharge rate (slope of integrated spike plot is unaltered) but did result in a prolongation of the burst that began after a latency of about 100 ms. The parametric correlations for this cell were high and significant (r(dur.) = 0·85; r(vel.) = 0·57; r(amp.) = 0·70) and the depth of modulation was 87 pulses/s.

We tested the effect of interrupting tracking movements with a brake for three cells, and in none of the brake trials was there any evidence for a short latency modification in discharge rate. Fig. 8*B* compares a brake trial with a control trial for a cell that was strongly correlated with performance on the twister. This cell was not included in Table 1 since the data file was filled with randomly inserted brake trials. However, selective analysis on non-brake trials did yield high parametric correlations that fell within the range of our top twenty cells. The cell had a lead time of 137 ± 61 ms as illustrated by the inflexions (open arrows) in the integrated spike records that preceded primary (line 1) and secondary (line 3) movements in the on direction. In the brake trial the primary movement was interrupted at the time marked by line 2, and comparison of the integrated spike records illustrates the absence of any upward modulation in discharge rate. While the brake did not produce alterations in discharge rate, it resulted in a prolongation of burst duration on some trials. For the example shown in Fig. 8*B*, the prolongation became evident (filled arrow) 100 ms

after braking (line 2); longer and shorter intervals were also observed. Burst prolongation on selected trials without any effect on discharge rate was also seen for the other cells tested for responses to braking.

DISCUSSION

The present results suggest that the forelimb area of magnocellular red nucleus (r.n.m.) is preferentially involved in the control of hand movements, and that the discharge rates of single cells code movement velocity. Before discussing these functional issues, it is important to evaluate the suitability of the approach used here for the assessment of r.n.m. control functions.

Criteria for the assessment of r.n.m. control functions

Anatomical studies have demonstrated that r.n.m. neurones in the primate project to laterally placed interneurones in the spinal cord (Kuypers, Fleming & Farinholt, 1962). Furthermore, electrical stimulation of the r.n.m. evokes mono- and polysynaptic excitatory post-synaptic potentials (e.p.s.p.s) in motor neurones (Shapovalov, Karamjan, Kurchavyi & Repina, 1971) and discrete contractions of limb muscles (Larsen & Yumiya, 1980). Cheney (1980) reported that two out of thirty-four r.n.m. cells showed significant spike-triggered averages with e.m.g. activity recorded from wrist flexor and extensor muscles. Such observations demonstrate that the rubrospinal tract is available as a pathway for transmitting motor control signals, but the conditions under which this pathway is used are not revealed by these methods. Lesion studies provide useful information on the latter issue. When most of the large fibre descending tracts are interrupted while sparing the rubrospinal tract, monkeys are able to execute arm and hand movements after a period of 'functional recovery' (Lawrence & Kuypers, 1968). However, a lack of understanding of the processes that constitute functional recovery weakens this evidence in the assessment of normal control functions.

A more direct approach to the assessment of motor control functions is to record the activity of r.n.m. neurones while the monkey executes movements, which is the method used here and in several previous studies (Otero, 1976; Cheney, 1980; Fromm *et al.* 1981). The disadvantage of this signal-analysis method is that neurones at many brain sites show some modulation in discharge in association with a variety of limb movements, and it has been difficult to determine whether or not the observed modulations serve specific control functions. The innovation pursued in the previous paper was that of switching between tasks in order to vary the type of movement while recording from a single cell. While this method worked well, the extent to which a given neurone was related to a particular movement was judged solely on the basis of the depth of modulation of discharge. In the present paper we have used additional criteria to make this judgement. Our working hypotheses have been that (1) the activity of a single cell should show detailed correlations with the temporal and parametric features of the movements it controls and (2) these correlations should become weaker or disappear when other movements are performed. The first hypothesis is strongly supported by the results in Table 1, and the second is supported, with minor qualification, by the results in Table 2.

The choice of parameters for correlation analysis was guided by simple inspection of time plots of discharge rate and manipulanda position. It was apparent that bursts of discharge preceded each movement segment for all cells that showed any consistent temporal relation to the movement. It was then quite natural to seek correlations between burst duration and movement duration, burst frequency and velocity, and the number of spikes in the burst and movement amplitude. There is some redundancy here since any two of the former relations imply the presence of the third. For example, if the number of spikes relates to movement amplitude, and discharge rate relates to the velocity, it follows that burst duration should be related to movement duration. In practice, however, the range of data available in each of these domains varied from cell to cell. Thus, for some cells the data were better for assessing velocity and duration whereas for others they were better for assessing amplitude and duration. A data base suitable for comparing all cells was provided by running the three correlations on all cells and using the average of the correlation coefficients as a single measure of relatedness.

After cells had been ranked according to the average value of correlation coefficient, it became clear that the cells with high parametric correlations also had other distinguishing properties. One, is that the values of lead time were less variable for the top twenty cells than for the remainder of the sample (Fig. 3). This was true for the variance surrounding the mean lead times of individual neurones, and it was also true for the variance from the mean of the cell sample. The clustering of lead times for the top twenty cells around the mean value of 118 ms is nicely contrasted with the larger variance of our entire sample in Fig. 7 of the previous paper (Gibson *et al.* 1985). The filled bins in the histogram illustrate the observed values for the top twenty cells. Note that all cells in this select group have lead times equal to or greater than the lead time of forearm e.m.g. activity (arrow).

We also found that the values of directional selectivity were consistently high for the top twenty cells. These values are compared with those of the entire sample in Fig. 6 of the preceding paper. The filled bins again represent the top twenty observations. The comparison suggests that a directional firing pattern may be a prerequisite for strong parametric correlations, as suggested on the basis of functional arguments in the preceding paper.

Fig. 5 in the preceding paper compares the depths of modulation of the top twenty cells (filled bins in the histogram) with the values seen for the entire sample. It is clear that a large depth of modulation does not guarantee a high parametric correlation. However, the two measures are not unrelated. Of the fifty cases showing depths of modulation in excess of 70 pulses/s, 17 (34%) represent cells in the top twenty. In contrast, only three of fifty cases (6%) with depths of modulation in the range 40–70 pulses/s represent cells in the top twenty.

These various observations suggest that strong correlations with movement parameters are highly restrictive criteria of relatedness. The high values of correlation observed for the top twenty cells increase the likelihood that these neurones participated in the control of the movements under study.

Types of movement controlled by the r.n.m.

In the previous paper we suggested that the majority of r.n.m. cells control hand and foot movements. This conclusion was based on the intensity of discharge in free-form tests and on the depth of modulation observed during tracking movements. One of the most successful tracking devices for eliciting large modulations in discharge rate was the twister. Various finger devices were also quite successful. The parametric tests used in the present paper yielded similar results. The majority of the top twenty cells (75 %) showed their best relations during twister operation, and another 15 % were best related to finger tasks. However, two cells (10 %) were best correlated with movement about proximal joints. These data suggest that the r.n.m. may participate in the control of both proximal and distal movements, but with a heavy bias towards the most distal joints.

Parametric analysis of a cell's discharge during performance on more than one device suggested a high degree of specificity in movement relations for the majority of cells, but these results also provided evidence that some cells may participate in the control of neighbouring joints (Table 2). Among the six cells that were reasonably well correlated with performance on more than a single device, five showed an overlap between the twister and a digit device. This fits with the suggestion advanced in the previous paper that the success of the twister derives from an involvement of finger movements in its operation. However, another five cells tested on both the twister and a finger device did not show an overlap in relatedness. Clearly units relating to twister performance do not represent a single cell type. A shoulder–elbow overlap occurred for one cell and an elbow–twister–finger overlap occurred with another. These latter cases illustrate most clearly that individual r.n.m. neurones may participate in the control of neighbouring joints.

Velocity signals

Our results suggest that the signals transmitted by r.n.m. discharge code movement velocity. This hypothesis is supported first of all by the high correlation coefficients found between average discharge rate during a burst and movement velocity throughout a 5–10-fold range. Since the movement continues at this velocity for a specific duration, one would further expect, and we have found, a high correlation between burst and movement durations. An additional test of the velocity signal hypothesis is provided by the cusum traces shown in Fig. 2. These traces represent the time integral of the incremental neural signal. Thus, if the neural signal codes velocity on an instantaneous basis, the cusum should resemble the time integral of velocity which is position. Comparison of the cusum traces with the position records shows that there can be a remarkable correspondence on a moment-to-moment basis (Fig. 2). The only major discrepancies occur in instances where the movement is in the 'off' direction for the cell, which is to be expected because of the strong directional nature of the response.

Our results in the monkey agree well with the reported correlations between discharge rate and movement velocity for red nucleus cells in the cat (Burton & Onoda, 1978; Soechting, Burton & Onoda, 1978). These authors based their conclusions on temporal correspondence and on cross-correlation functions calculated from

recordings during self-paced elbow movements. The range of velocities over which this relation held was not considered. Another study in the cat (Ghez & Vicario, 1978) reported that discharge rate is related to the rate of force change in an isometric step tracking task. These authors did not attempt to vary rate of force change to test the range over which the correlation might apply. Thus, our data are the first to demonstrate parametric relations that hold for both fast and slow contractions.

There probably is no major conflict between the data suggesting a velocity signal and that suggesting a relation to rate of force change (cf. Ghez & Vicario, 1978). Under the isometric conditions of the latter study, the velocity of internal muscle shortening (due to extension of muscle series elasticity) should have been proportional to the observed rate of force change. We would propose, however, that velocity is a better descriptor of the r.n.m. signal, at least in the monkey, since we observed discharge to continue at a rate proportional to velocity during the performance of slow, long duration ramp movements. Strain gauges on the tracking manipulandum indicated that force was constant, and hence rate of force change was zero, during these long duration movements.

Burton and collaborators (Burton & Onoda, 1978; Soechting *et al.* 1978) suggested that the velocity signal in the red nucleus originates as a consequence of feed-back from the periphery, and other authors have advanced the potentially related suggestion that this nucleus is selectively involved in controlling movement termination rather than initiation (Otero, 1976; Fromm *et al.* 1981). If feed-back were an important mechanism shaping r.n.m. discharge, one would expect that velocity correlated neural signals would be delayed by the conduction time through afferent pathways. Instead, we have found that the signals precede movement by approximately 120 ms (Figs. 2, 3 and 4). These results cannot be explained by a feed-back mechanism. The results also rule out a special role for the r.n.m. in the control of movement termination. Instead, our results favour a role in the control of the entire movement from onset through termination.

The results of brake trials in which movements were abruptly halted also argue against a feed-back mechanism (Fig. 8*B*). If feed-back were prominent, there should have been short latency changes in discharge rate in response to the forces and sudden deceleration produced by the brake, but no changes occurred. This result is in agreement with the reported absence of short latency responses to passive limb displacements in monkey red nucleus (Fromm *et al.* 1981). Similar findings have also been reported for cells that provide input to the r.n.m., namely cerebellar interpositus neurones (Harvey, Porter & Rawson, 1979) and a subpopulation of motor cortical neurones (Fromm *et al.* 1981). However, in some brake trials we noted a change that can be attributed to delayed feed-back, namely a compensatory prolongation of burst duration (Fig. 8*B*). The nature of the modification (prolongation of the burst rather than alteration in discharge rate) and the relatively long latencies at which these effects were manifested suggest a complex process quite unlike conventional continuous linear feed-back.

If the velocity signals recorded in r.n.m. are not shaped by feed-back, as seems likely, they must be attributed to some endogenous C.N.S. mechanisms with properties analogous to those of a function generator. Since the main input to r.n.m. is from the interpositus nucleus in the cerebellum (Humphrey & Rietz, 1976), and since

interpositus neurones also appear to be relatively insensitive to sensory feed-back (Harvey *et al.* 1979, but see Strick, 1983, for an alternative view), these velocity signals may be generated by cerebellar circuitry.

Velocity commands

Since r.n.m. discharge rate is a velocity signal that precedes movement, it becomes a prime candidate for being a velocity command. Based on our current knowledge of limb muscle properties and spinal reflex mechanisms, changes in limb position are likely to be controlled by tonic shifts in the thresholds of stretch reflexes (cf. Houk, 1979). R.n.m. velocity signals would have to be converted to position signals in order to produce tonic shifts in reflex threshold. The situation is similar to that faced by eye-movement physiologists several years ago when Robinson (1975) postulated a brain-stem integrator to convert the eye velocity signals recorded in premotor neurones into eye position commands.

An alternative interpretation of our data can be advanced if one assumes that the segmental motor apparatus is inactive before the start of a movement and is again inactive at the end of the movement. Then, no tonic position command would be needed. The observations we made under conditions of spring loading were intended to test this alternative possibility. E.m.g. recordings indicated that the spring loads resulted in tonic activity of forearm muscles that move the wrist and fingers. In spite of this apparent evidence for the presence of tonic motor commands, we did not observe the appearance of a tonic r.n.m. signal. However, it remains possible that our loading conditions did not elicit tonic activity in the appropriate combination of muscles, and this issue needs to be addressed in future studies with a larger sample of neurones.

A third interpretation that can be contemplated is that the rubrospinal pathway is specialized for the control of phasic components of movement, namely velocity. Tonic control might then be achieved via other descending pathways such as the corticospinal tract.

In conclusion, the present results document the existence of strong correlations between specific types of movement and r.n.m. discharge. Relations to hand and finger movements are particularly well represented in the population. The results further demonstrate that single cells are capable of transmitting a precise velocity signal. The latter is not a product of sensory feed-back but instead appears to be generated by endogenous C.N.S. mechanisms, perhaps within the cerebellum. The velocity signals transmitted by the r.n.m. precede movement and may serve as velocity commands.

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