CONTRIBUTIONS OF CENTRAL PROGRAMS TO RAPID LIMB MOVEMENT IN THE CAT

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INTRODUCTION

SHERRINGTON considered that integration in the nervous system represented "interaction for a purpose" (GRANIT, 1977). In the case of voluntary responses directed towards a target, these interactions allow the translation of information about stimulus topography into an appropriate pattern of contraction of particular muscles. To be appropriate, however, the magnitude of the underlying neural output must be accurately scaled to particular stimulus variables. Voluntary responses are neither limited by "local sign" nor by the receptor surface transducing the stimulus. Their direction and magnitude is dependent only on the purpose to be achieved. Because of the large array of potential stimulus-response relations, the operation of presetting mechanisms has generally been postulated to reduce the time required for the selection of a particular response (WELFORD, 1976). Evidence exists that such presetting mechanisms may, in fact, determine the direction of intended responses to kinesthetic perturbations (EVARTS and TANJI, 1976). Since quick and accurate responses may be fully completed within less than a single reaction time from their onset, it is also generally recognized that critical parameters of the entire response must be determined by "central programs". There is little information however, concerning the output variables controlled by such central programs or about the control policy governing the generation of responses of different magnitudes. An analysis of these problems is complicated by the fact that, once a motor output is initiated, interactions between the ongoing central commands and afferent activity resulting from the response itself may occur at multiple sites in the nervous system. The function of presetting mechanisms, the response parameters specified by central programs and the constraints imposed by reflex interactions are all critical to an understanding of the physiology of sensory-motor integration in voluntary movement. To obtain a general framework within which these problems could be approached, we have studied tracking performance in the cat. The tracking task we have developed requires rapid and precise movement of the limb or, alternatively, a change in force applied to a lever in accord with sensory information provided by a display. First, we will consider the isometric adjustments in force exerted against a stationary lever. Such isometric conditions provide a simpler circumstance to analyze the commands which control motor responses, since the effects of limb inertia and the viscoelastic properties of limb and muscle may, to a first approximation, be neglected. In addition, variations in active tension and reflex interactions resulting from changes in muscle length are also minimized. Then, from a consideration of the configuration and latency of these responses, we will suggest a function for presetting

mechanisms in governing the input-output relations which characterize these responses. From a parametric analysis of output variables, a general control policy will be proposed which governs rapid force adjustments of different magnitudes (GHEZ and VICARIO, 1978c). Finally, from preliminary data, we will show the applicability of this model to the control of limb position and point out certain constraints which appear to be imposed by segmental mechanisms.

METHODS

The experimental procedure is more fully described elsewhere (GHEZ and VICARIO, 1978b). Figure 1 illustrates its major features. The animal was restrained snugly in a sleeve, its head and left humerus rigidly fixed to an external frame. The animal's forearm was strapped in a splint attached to the lever of a torque motor controlled manipulandum. Transducers indicated its angular position, velocity and the force applied to the lever.

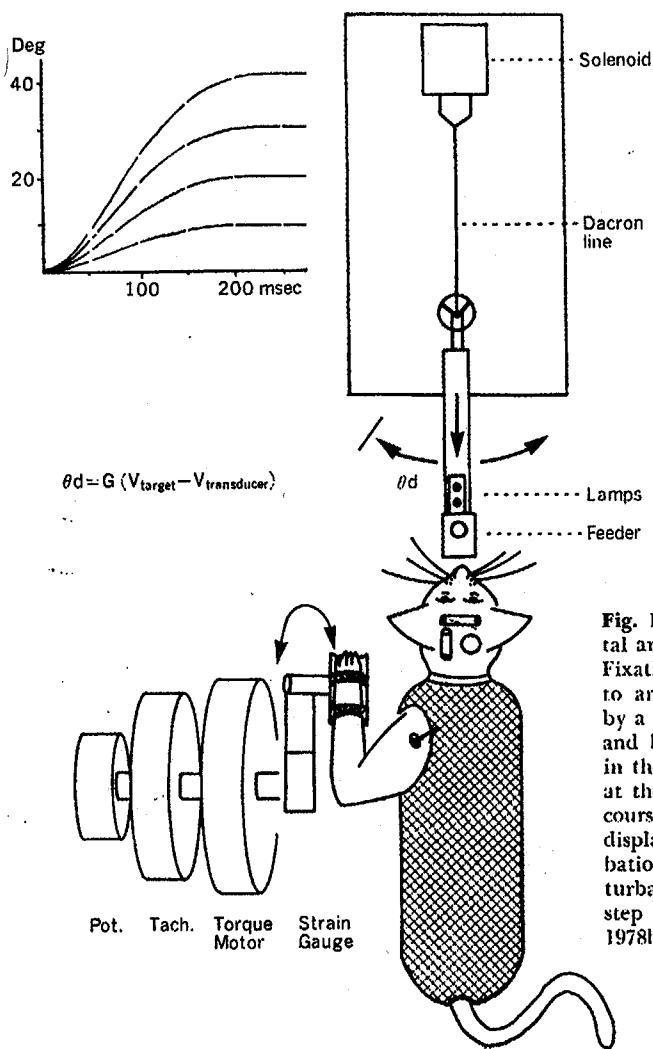$$\theta d = G\left(V_{target} - V_{transducer}\right)$$

Fig. 1 Schematic diagram of experimental arrangement. See text for explanation. Fixation of the head and the left humerus to an external frame were accomplished by a coupling device attached to the skull and by an intramedullary pin implanted in the humerus, protruding from the skin at the shoulds. The inset shows the time course of the changes in position of the display assembly produced by step perturbations in target voltage. The four perturbations are multiples of the smallest step change. (from GHEZ and VICARIO, 1978b)

A display assembly in front of the animal consisted of a retractable feeder operated by a solenoid, and a pair of lamps. This assembly could move from side to side by means of another, servo operated, torque motor. The angular position of the assembly, θd, was a function of an error signal representing the voltage difference between the output of one of the transducers in the manipulandum and a 'target' level. Position or force adjustments could be elicited in the same animal by either clamping or releasing a lock on the manipulandum and changing the feedback conditions governing the display. Accordingly, if the transducer voltage was derived from the output of the strain gauge, the display assembly moved from side to side with fluctuations in the force exerted by the animal. If the potentiometer voltage was used, the display reflected changes in the lever position. In addition, the display assembly moved whenever the target voltage was stepped in one direction or another.

The animals were trained to adjust the force they applied to the lever, or its angular position, so as to align the display assembly with their midsagittal axis, in order to obtain a food reward. This was delivered by releasing the feeder which then came close to their mouths. Rapid changes in the position of the lever or the force exerted upon it were elicited by stepping the target voltage level at random times when the animal was steadily aligning the display. This perturbation moved the display assembly and required a corrective adjustment in force or position on the part of the animal. The food reward was given after a second period of stable alignment following target reacquisition.

The amount of displacement of the display system corresponding to a perturbation in target level or to a change in transducer voltage was under experimental control. The magnitude of display motion requiring a given response could be varied over an eight fold range by changing the gain, G, of the error signal controlling the display, as indicated, in Figure 1. Because of inertia and friction in the display device, approximately 200 msec were required for it to fully respond to a step perturbation. Responses of the display to step inputs of increasing size are illustrated as a function of time in Figure 1 (inset, upper left). The peaks of both the first and the second derivatives of its motion were approximately linear functions of the target perturbation. Thus, although the response of the display was slow, the full display trajectory was predictable from the early values of the derivatives of its motion. The changes in display position and its derivatives could be detected by the animal in either of two ways: by the deflection of its vibrissae as the display moved away from the midline, and by vision.

The behavior required of the animal in this experimental arrangement can be considered as an input-output transformation: the direction and magnitude of arbitrary stimulus variables must be converted by the animal into a response of a given direction and magnitude. Alterations in the gain or polarity of the signals controlling the display require the animal, in its turn, to change the gain and polarity of any internal transfer function it may have established.

RESULTS AND DISCUSSION

Isometric control of force

The distinctive features of rapid isometric adjustments in force are seen for two superimposed trials in Figure 2. About 60 msec after the perturbation in target level, a burst of activity in the agonist EMG occurs, associated with the rising phase of the first derivative of force, dF/dt. The force registered by the strain gauge increases to a maximum (with some overshoot) and is ultimately realigned with the new target level. Since the change in angular position of the display (which provides the actual stimulus for the animal) is
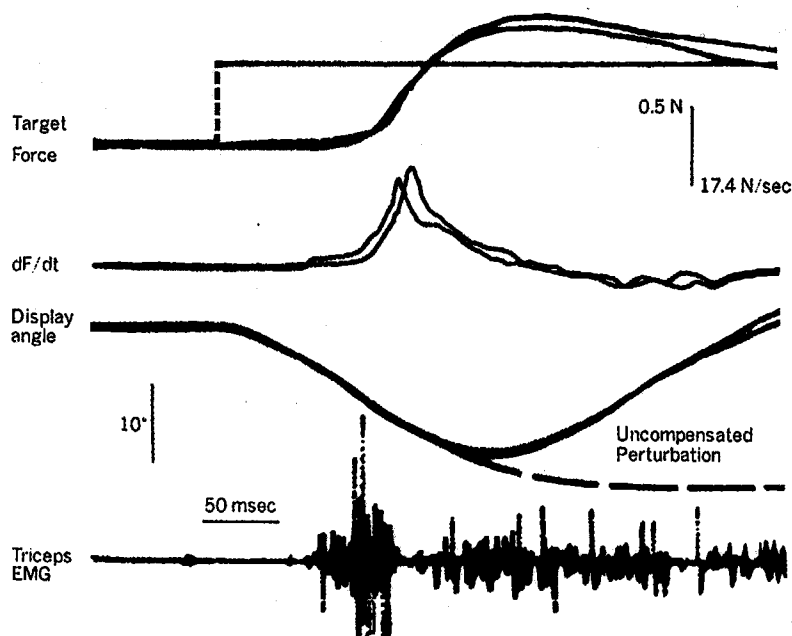
Fig. 2 Isometric responses to two equal step perturbations in target level. See text for explanation. (from GHEZ and VICARIO, 1978c)

controlled by a force error, its initial deflection is produced by the step in target voltage while the terminal portion of the trace (returning the display towards its initial position) results from the change in force generated by the animal.   The dashed line indicates the trajectory of the display to the same perturbation when the animal was prevented from exerting force on the lever.   In this and in virtually all cases, both the burst of agonist activity and the peak dF/dt occur before the display has fully responded to the perturbation. Moreover, the sluggishness of the display is such that these early response events are over before the corresponding change in force exerted by the animal can modify its trajectory.

1) Response scaling.   When the perturbation in target level was randomly varied from trial to trial, the peak force, the peak dF/dt and the integrated value of the agonist EMG remained scaled to the final force required (GHEZ and VICARIO, 1978c).   Thus, derivatives of display motion must provide the animal with sufficient information to both initiate a response and scale its magnitude. To use this early information appropriately, the animal must draw on its previous experience with the device to extrapolate the full extent of the display trajectory from the early sensory information.

The animal's reliance on experience and the role of learning can be demonstrated by unexpectedly changing the gain (G) of the display while the cat is holding the initial alignment and awaiting the perturbation.   Figure 3 illustrates the results of experiments where the gain of the display was either increased or decreased by a factor of two.   The perturbation in target level was maintained unchanged.   The animal was thus required to generate the same response, but the magnitude of the sensory input was different.   In Figure 3A the gain was increased by a factor of two and the first responses following the change in gain were approximately twice the size of control responses made earlier.   Over several trials the magnitude of this initial force response decreased progressively.   A decrease in gain (Fig. 3B) produced the converse effect.   The initial responses are approximately one half of those obtained during the control period.   These observations indicate
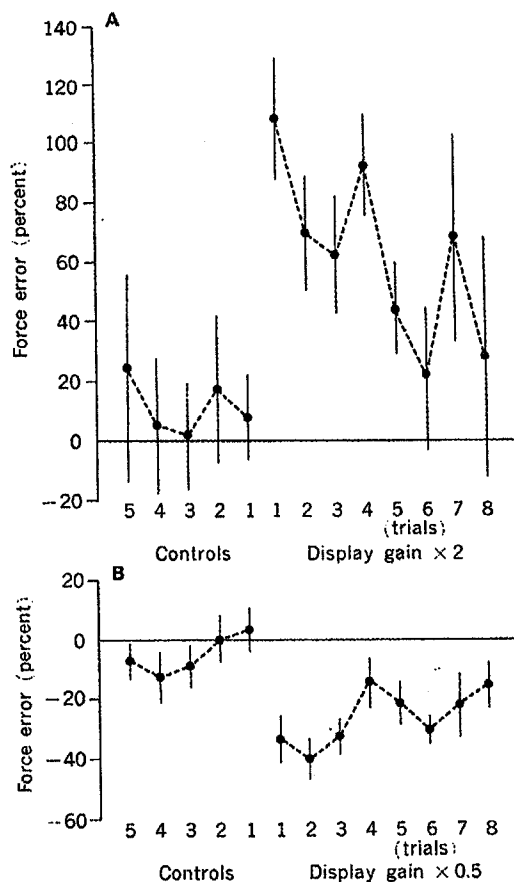
Fig. 3 Rescaling of input-output relations following changes in display gain. The display gain was either increased (A) or decreased (B) by a factor of two. The change was made during the initial alignment prior to the perturbation. The numbers on the abscissa represent the trial number before (controls) and after the change in display gain. The points and bars show the average of the error in peak force in four runs and the standard error of the mean. (from Ghez and Vicario, 1978c)

that the metrics of the transformation relating input and output magnitudes must be set prior to the stimulus itself.

2) Response latency. The extremely short latency of the responses supports the hypothesis of a preset transfer function. In the cases illustrated in Figure 2, the time from the perturbation to the burst of agonist EMG was about 60 msec. There, the animal relied on information provided by the deflection of vibrissae following the sudden movement of the display. When the animals relied on visual information alone, their response latencies were increased by about 20 msec over those with vibrissae alone (Ghez and Vicario, 1978b). This difference corresponds approximately to the retinal transfer time determined in Y cells (Shapley and Kaplan, personal communication), which provide velocity information to the central nervous system (Ikeda and Wright, 1972).

The response latency using either modality was dependent on two separate factors: the display motion produced by the perturbation, and the peak rate of force change generated by the animal. With perturbations of increasing size, both factors contribute to a progressive decrease in latency towards an asymptote. To dissociate the two factors,
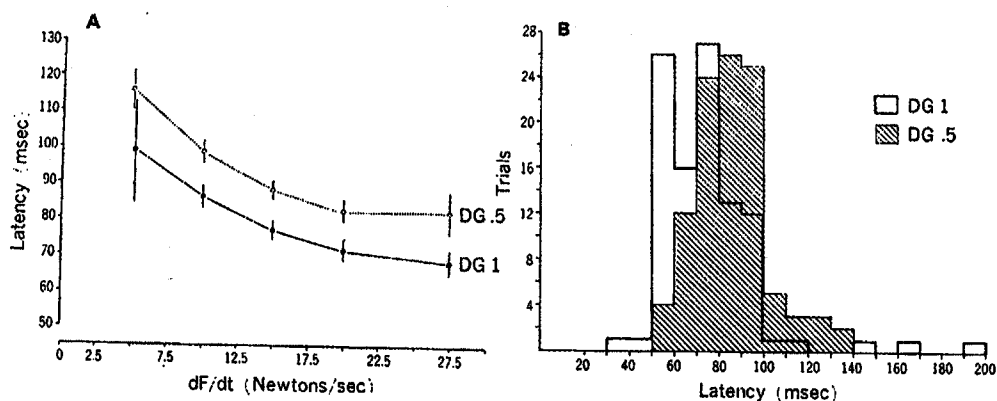
Fig. 4 Effects of changes in display gain on response latency. *A*: Changes in response latency as a function of dF/dt. Perturbations were of constant amplitude and required the same response magnitude, but the display gain was varied between two different values (DG 1, filled circles; DG 0.5, open circles). The points show the mean values and standard errors of the latencies of responses occurring in the intervals on the abscissa. Mean latencies are plotted above the midpoint of the range of the dF/dt. *B*: Latency histograms of responses elicited under the two display gain conditions. (from GHEZ and VICARIO, 1978b)

responses elicited by perturbations displayed at different gains were compared (GHEZ and VICARIO, 1978b). The results of such an experiment are illustrated in Figure 4. In A, the mean latencies of the first change in dF/dt are plotted as a function of the peak value of this parameter under two conditions of display gain. There is a gradual decrease in latency with increasing dF/dt, and the latencies of the responses elicited at the higher display gain (where the actual neural input may be assumed to be greater), are shorter at all values of dF/dt. This uniform shift suggests that two independent processes underlie the changes in latency. The histograms in Figure 4B show the latencies of response pairs matched for peak dF/dt; the two are statistically different (Mann-Whitney U test). The dependence of latency on input and output magnitudes is undoubtedly the result of spatial and temporal summation occurring in both afferent and efferent pathways. Neurons responding to movement of a visual target as well as those responding to cutaneous stimuli have shorter latencies and more brisk responses with increasing stimulus velocities. Similarly, increasing rates of force change are produced by an increase in the rate of recruitment of motoneurons (TANJI and KATO, 1973). When the predictability of the time of onset of the perturbation and the predictability of either the magnitude or direction of the required response were varied systematically, no effect on the response latency was found.

On the basis of their short latency, these tracking responses must be mediated by relatively simple pathways in which only a small number of serial relays are likely to be interposed between afferent and efferent stations. Decision processes underlying the selection of response topography and metrics are not likely to take place during the reaction time interval itself. Rather these processes are likely to be determined by gating and biasing mechanisms operating prior to the stimulus. Since, on the one hand, the force adjustments were scaled from their onset to demands set by the initial sensory events, and since rescaling of the motor responses required 10–20 trials when the properties of the display were altered, it is likely that these presetting mechanisms operate to adjust the gain of a preset or "resident" transfer function.

3) "Pulse-step" control. To determine how different output parameters are controlled, it is necessary to consider the configuration of the responses themselves in greater detail. The cardinal features of isometric force adjustments of different magnitudes
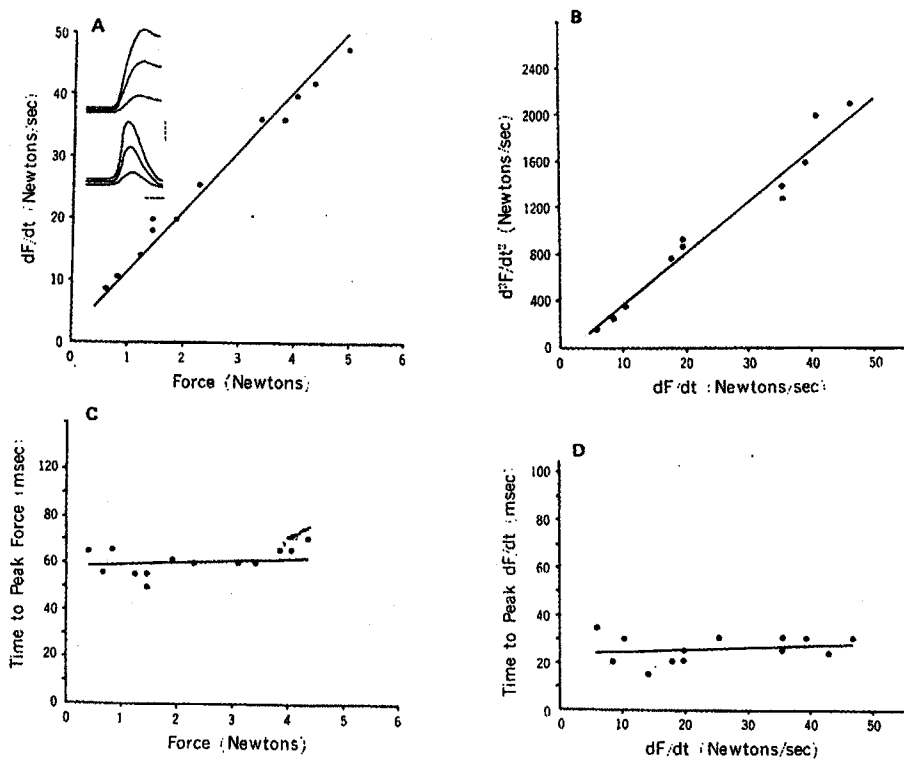
Fig. 5 Relations between output parameters under isometric conditions. *A*: Relation between peak force and peak dF/dt. The inset shows sample records of force and dF/dt for movements of different sizes. Horizontal calibration: 60 msec. Vertical calibration: 0.5 N, 12.5 N/sec. *B*: Relation between dF/dt and peak d²F/dt². *C*: Relation between peak force and the time between the first change in dF/dt and the peak force. *D*: Relation between peak dF/dt and time from onset to peak. (modified from GHEZ and VICARIO, 1978c)

are shown in Figure 5 which is representative of cases where the peak dF/dt increases mototonically to its peak and then decreases smoothly. Examples are illustrated in the inset in the upper left hand corner which shows the force above and dF/dt below. Increases in peak force were accompanied by increases in the peak of its first derivative and two parameters were linearly related, as shown in A. Similarly, a linear relationship bound the peak dF/dt and the peak of the second derivative of force (d²F/dt²) (B). By contrast, the time from onset to peak value of either force (C) or its first derivative (D) did not vary with increasing magnitudes of these parameters. Thus higher peak forces and dF/dt's were achieved without increases in time.

The linearity of the relationship between the initial force change and the peak value of its first derivative suggests that the underlying response pattern is stereotyped in its general configuration and that adjustments of different magnitudes may be related to one another by different scaling factors. To interpret the time course of the changes in dF/dt it is necessary to recall that skeletal muscles have profound low-pass characteristics. As shown by PARTRIDGE (1964), the muscle tension produced by a frequency modulated neural input decreases markedly with increasing frequency of modulation and dramatic time lags are introduced by muscle properties. As a result, for the time to peak force and the time from onset to peak dF/dt to remain essentially constant as is characteristically the case, it is necessary that the initial phase of the adjustment be governed by the transient recruitment of additional motor units and that they initially fire at higher frequency than
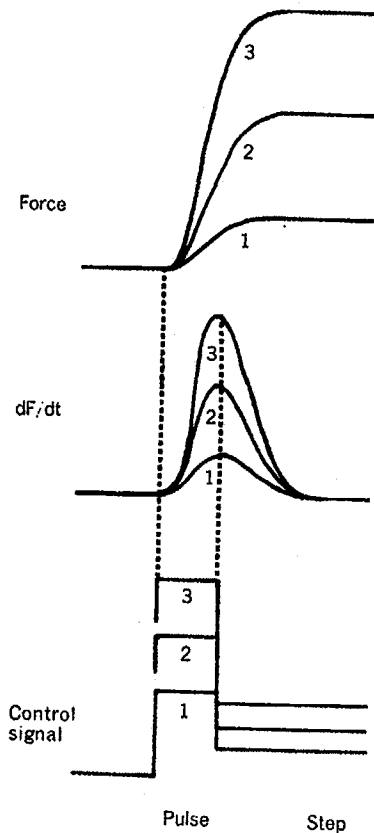
Fig. 6   Pulse-step model of rapid force adjustments.   See text for explanation.

during the application of the terminal steady state force.   In a first approximation the motor outputs observed here may thus be described by a pulse-step model in which the dynamic phase of the adjustment is controlled by the height or amplitude of an initial pulsatile output.   This is illustrated schematically in Figure 6.   The duration of the proposed initial control pulse approximates that of the rising phase of dF/dt and remains essentially constant.   Its amplitude governs the rate of increase of dF/dt and thereby the peak force as well.   The terminal steady state force can be assumed to be governed by a step increase in neural output.   The suggestion that these force adjustments are determined by an initial pulsatile output, modulated in amplitude rather than in duration, is supported by our finding that the rising phase of dF/dt is accompanied by a burst of EMG activity in the agonist muscle (as seen in Fig. 2) whose integrated value is proportional to the peak dF/dt but whose duration does not vary with this parameter (GHEZ and VICARIO, 1978c). Similar observations have recently been made in the human by FREUND and BÜDINGEN (1978).

Pulse-step models have also been proposed to account for the control of eye position during ocular saccades (ROBINSON, 1964, 1970; BAHILL et al., 1975).   In that system however, the magnitude of a saccade is governed primarily by the duration rather than the amplitude (or spike frequency) of an initial burst of activity of oculomotor neurons (ROBINSON, 1970).   In the present context the pulse-step model must be understood as the final result of phasic and tonic descending commands controlling the force output and its derivatives.   Thus, in limb muscles, this final output must also reflect the particular constraints imposed by regulatory properties of motoneurons (BURKE and RUDOMIN, 1977)

and feedback actions following the activation of peripheral receptors once the motor output is initiated.

These observations suggested the possibility that in rapid voluntary muscle contraction, descending commands may specifically control the derivatives of force. In parallel with these behavioral investigations, we have studied the relationship of the activity of single neurons in the red nuclei of these cats during isometric tracking (GHEZ and VICARIO, 1978a). These neurons were sampled in areas where microstimulation produced localized contraction of forelimb muscles and often responded to peripheral stimuli applied to the forelimb. The activity of cells was modulated when the animals exerted isometric force in either flexion or extension, and this change in activity occurred prior to the contraction of the agonist muscles. The vast majority of neurons in the red nucleus exhibited a pattern of discharge which paralleled the first derivative of force with a phase lead. Over a large range the magnitude of the peak dF/dt was a linear function of the preceding peak firing of these units. Neurons showing more tonic characteristics were rarely observed in the red nucleus. By contrast, from published accounts it would seem that output neurons of the motor cortex of monkeys do show a predominant tonic pattern of activity correlating with the steady state force (SMITH et al., 1975; FETZ, personal communication), and as shown recently by HEPP-REYMOND and WYSS (1978), individual units may have a threshold force level at which their activity is recruited. Although such data suggest that different neural structures may be responsible for the phasic and tonic control of output parameters, it is necessary to reserve final judgement on this matter since differences in species and in task conditions could also have contributed to the different properties observed.

From the present parametric studies of the force output and its derivatives we conclude that the initial sensory input, derived here from parameters of display motion, ultimately control the amplitude of both an initial pulsatile event and a terminal steady state output according to a preset transfer function. To implement this control policy, the subject must have prior information not only about the full target trajectory, as described earlier, but also of the peak force likely to result from the brief initial burst of neural output. Moreover, while the time to peak force and the time to peak dF/dt remained stable within any given day, two to three fold differences in these times occurred from day to day. This suggests that the duration of the initial pulsatile command is regulated by a higher order strategy rather than by the initial sensory events.

Control of rapid limb displacement

Recently, we have focussed our attention on the kinematics of limb displacements to determine whether a pulse-step control policy, similar to that noted under isometric conditions, also determined rapid movements of the limb and whether the input-output transformations underlying tracking performance could be considered to reflect the presetting of a relation between stimulus variables and the forces required to displace the limb. Given the wide variation in loads which may be encountered in the course of limb movements, an alternative hypothesis might have suggested that central programs preset a particular angular displacement in relation to the target variables. In this case length servo mechanisms would compensate for variations in load.

To examine these problems, the display was made to reflect an error in position of the lever. Under these conditions perturbations in target level, which shifted the display, elicited rapid adjustments of the angular position of the lever by the cats. The extent and direction of the initial lever displacement was invariably correlated with the amplitude and direction of randomly varied perturbations and response latencies (measured again to the first change in dF/dt) were similar to those observed under isometric conditions.
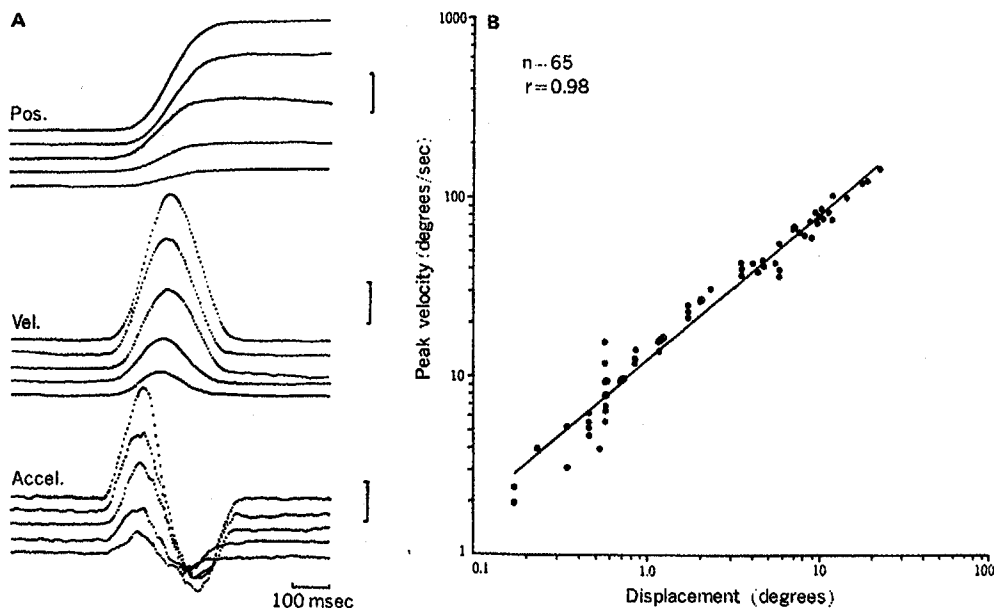
Fig. 7 Relations between parameters of movement. *A*: Displacement, velocity and accelerations of responses to perturbations of five different sizes. The traces are the average of eight trials for each perturbation. Calibrations: position 12°, velocity 67°/sec, acceleration 1000°/sec². *B*: Relationship between displacement and peak velocity for 65 separate movements (different experiment from Fig. 7A).

The peak velocity achieved during these adjustments was characteristically a quasi linear function of the displacement over an extensive range, as can be seen in Figure 7B. These increasing velocities were accomplished by corresponding increases in the peak acceleration (Fig. 7A). While the duration of movement often increased somewhat as the movement became larger, the time from onset to peak acceleration and velocity did not.

The peak velocity is, of course, determined by the magnitude and duration of the acceleration resulting from the sudden change in the contraction of agonist and antagonist muscles. It was not surprising therefore, that EMG recordings of agonist muscles showed a conspicuous burst of activity of approximately constant duration. As also noted by many investigators (MERTON, 1951; ANGEL et al., 1965; LESTIENNE, 1974; SOECHTING et al., 1976), a silent period interrupted the agonist activity just before the peak acceleration. After the silent period, EMG activity resumed at a level dependent upon the opposing forces.

If, for a moment, we neglect the factors controlling deceleration and the silent period in agonist muscles, these observations suggest that rapid limb displacements are also governed by an initial pulsatile output, modulated in amplitude and of approximately constant duration, and a terminal step-like output which determines the final position of the limb. The amplitude of this pulsatile output, which controls the peak angular acceleration, also determines the peak velocity and displacement corresponding to the change in intended position. This phasic initial output is needed to overcome the inertia of the limb and the viscoelastic properties of muscles and joints as well as the low-pass properties of muscles. A terminal step is also necessary, since gravitational and elastic forces acting on the limb vary with its angular position. Even when the mass of the forearm is considered against a constant gravitational field (and no antagonist activity is present), the central nervous system must provide additional input to the agonist in the
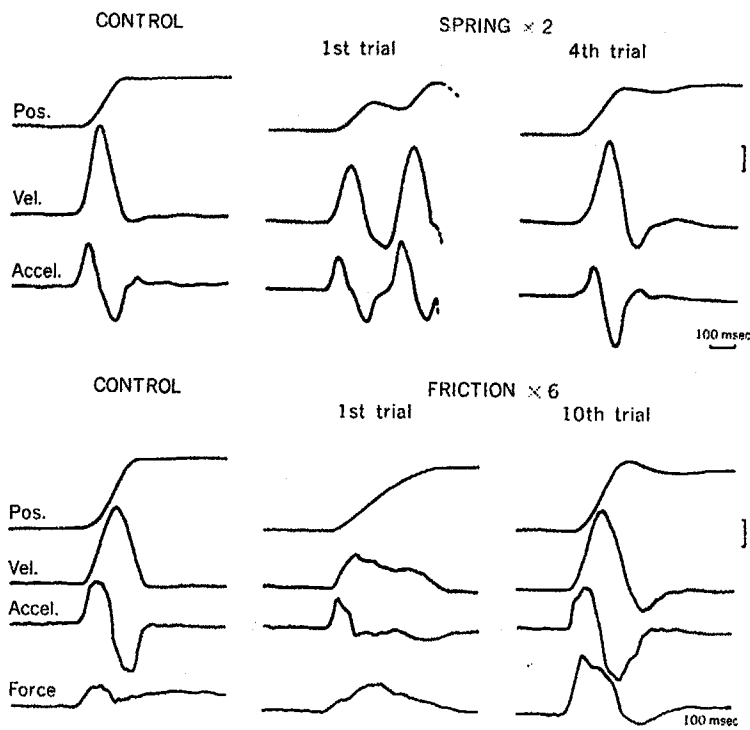
CONTROL SPRING × 2

1st trial 4th trial

Pos.

Vel.

Accel.

100 msec

CONTROL FRICTION × 6

1st trial 10th trial

Pos.

Vel.

Accel.

Force

100 msec

Fig. 8 Movement parameters following unexpected changes in opposing forces. See text for explanation. Calibrations: upper traces: position 6°, velocity 33°/sec, acceleration 500°/sec²; lower traces: position 12°, velocity 47°/sec, acceleration 250°/sec², force 1 Newton.

terminal position to compensate for its loss of tension due to shortening and the increased tension of the antagonist which is stretched.

To investigate the possibility that the initial pulsatile component and the later tonic events can be controlled independently, we have determined, in preliminary studies, the changes in the time course and extent of movement when lever displacement was unexpectedly opposed by loads of different configurations. These loads were generated by changing the feedback conditions controlling the torque of the manipulandum. Figure 8 illustrates the changes in response variables following an unexpected increase in opposing forces. The upper left hand traces (control) show the displacement, velocity and acceleration when the animal adjusted the lever position against a light spring load. Just prior to the perturbation eliciting the response shown in the center (first trial) the spring constant was doubled without changing the force required for the initial alignment. The increase in terminal steady state force required for alignment was 25 g. The response to the first perturbation at the new spring constant (first trial) fell short of the intended position and a second movement was made after a brief interval. Again, however, the tonic forces generated were inadequate to maintain postural stability. Nevertheless, after a few trials the animal learned the new conditions and produced movements which were similar to controls in their displacement, velocity and acceleration.

The lower frames show the alterations in movement trajectory when, again unexpectedly, the frictional force opposing movement was increased by a factor of six. The first response in the new condition differs from that seen when the spring constant was increased: while the final position following the load change was similar to the control, the peak

velocity was markedly lower. Nevertheless, again following an adaptation period, the animal readjusted its output so that the peak velocity and acceleration approximated those of the controls. The bottom traces document that this adaptation was achieved by a marked increase (here about 200 g) in the forces generated during the initial phase of movement.

These observations show that when the task requires the control of limb position (rather than force), the input-output transformations also relate derivatives of display motion to an intended force output. This is evident from the fact that the increase in spring constant opposing both phasic and tonic components of the force output resulted in an undershoot of the final limb position. Such undershoots were still seen when only 10 g opposed the final position. While the frictional load, which is a function of angular velocity, prevented the initial phasic events from resulting in a normal velocity of movement, it did not interfere with the achievement of a correct final position. Thus, as in the case of the head movements studied by BIZZI et al. (1976), a normal final position is achieved as a result of the terminal muscle tension in agonist and antagonist muscles. The different modes of compensation, which occur when the animal has learned the particular nature of the load to be expected, indicate that the phasic and tonic components of the motor program may be separately adjusted. Just as a change in display gain required the animal to rescale the transfer function relating a given sensory input to the amplitudes of an initial pulsatile output and a terminal step, a change in the anticipated load results in a comparable resetting. When loads oppose primarily the dynamic phase of movement, the initial pulsatile output is reset. When the load opposes both the dynamic and static phases, both the early and late components of the output are readjusted. Finally, while load-compensating position servos may be brought into action as a result of differences between actual and intended muscle shortening, they appear to have limited impact on the trajectory of the limb. These observations strongly suggest that rapid limb movements are primarily governed by central programs specifying the force output as a function, not only of input variables, but also of the magnitude and configuration of anticipated loads.

In the previous comments the mechanisms underlying deceleration of the limb were disregarded. However, although our data on this matter are incomplete, some consideration of this problem is in order. Indeed, once the acceleration of the limb crosses zero, the momentum will tend to passively propel the limb to a new position which depends on the balance of active and passive forces. It has long been known that concommitant with the increased EMG activity in agonist muscles, the antagonists become silent. In addition, a burst of EMG activity in antagonists commonly starts at about the time of the peak acceleration and reaches its maximum at the peak velocity during the silent period in the agonist. In human and monkey subjects, this burst of activity varies in magnitude with the peak velocity (LESTIENNE, 1974; SOECHTING et al., 1974). While the silent period in the agonist is primarily due to disfacilitation produced by the segmental actions of muscle receptors following their unloading (ANGEL et al., 1965) and is also seen in the cat, the burst of activity in the antagonist has been thought to reflect a transient excitatory descending command forming part of the motor program (HALLET et al., 1975). This interpretation seems unlikely since the timed antagonist burst is not observed following deafferentation (TERZUOLO et al., 1974). Moreover, in the cat, this activity in the antagonist is only present under anisometric conditions where limb displacement stretches the antagonist muscle; it does not occur under isometric conditions (GHEZ and VICARIO, in preparation). Additionally, provided the 'set' of the animal is identical, this antagonist activity is consistently greater when the limb is passively (and unexpectedly) displaced than when the same velocity and acceleration are actively achieved by the animal (GHEZ and

VICARIO, in preparation). These considerations strongly suggest that the burst of EMG activity in the antagonist during rapid limb movement represents a segmental stretch reflex upon which may be superimposed additional feedback actions of supraspinal centers receiving input from receptors sensitive to limb velocity (BURTON and ONODA, 1978; GHEZ and VICARIO, 1978a; EVARTS and TANJI, 1976). When the descending excitatory drive is conveyed to the agonist, a concommitant inhibitory control appears to be exerted upon the antagonist (see also LAMARRE et al., 1978), but which may be insufficient to prevent the reflex when the velocity of stretch is high. It is uncertain at present whether this inhibitory control includes both phasic and tonic components or whether the depth and duration of the inhibition can be modulated independently of the excitatory drive to the agonist. Such an independent control could be especially important in the presence of high moments of inertia to facilitate the arrest of movement.

CONCLUDING REMARKS

An examination of tracking performance in the cat suggests that the processes controlling rapid limb movement can be considered to involve several distinct levels of control which operate to overcome constraints imposed by lower order processes. In the first analysis, the animal's overall response can be considered as the expression of an input-output transformation whereby derivatives of display motion control the amplitude of both an initial pulsatile event and a terminal steady state. The initial pulsatile component is required to overcome the constraints imposed by mechanical properties of the muscle and limb. Since the initial events are scaled from their onset to the final force required by randomly varying perturbations, they must represent the expression of central programs. The gain of the underlying transfer function must be determined by higher order processes which enable the magnitude of the final output required to be estimated from the initial values of the derivatives of display motion, the properties of the peripheral plant and, in the case of limb displacement, the loads which will oppose movement. This "resident" transfer function can be rescaled when either the display gain or the loading conditions are altered, but whether by different neural mechanisms remains to be investigated. The establishment of such predictive behavior must be based upon information from the results of past responses and is an essential feature of "motor learning". It is critical for accurate movements in an "evolving situation" (WELFORD, 1976) because of the long time which inevitably elapses between stimulus and feedback from the response.

The factors governing the duration of the initial pulsatile command are uncertain. While it is attractive to consider that the control policy aims to minimize flight time while preserving accuracy, motivational factors, over which we do not, as yet, have good control, must also be important. BOUISSET and LESTIENNE (1974) have shown in humans that the kinematics of a movement of constant amplitude are critically dependent upon the instructions given to the subject. From their figures it appears that when the instructions specified a higher velocity, the time to peak acceleration was reduced; it is legitimate to suppose that the subjects then programmed a shorter pulse of agonist activity.

It must be emphasized that the pulsatile nature of the initial output need not necessarily reflect any intermittency in the sampling of peripheral input (NAVAS and STARK, 1968). Indeed, the output pulse can, in the cat, be updated at all times by new sensory information even when it arrives during the course of the reaction time or the response itself (VICARIO et al., 1978; see also MEGAW, 1974). The accuracy of such corrections is, however, limited by that of the transfer function in effect. Thus, corrections may be ineffective in the case of unexpected loads (Fig. 8) which require a rescaling of the transfer function between input

variables and the derivatives of the force output.

As regards the role of sensory events arising from afferents in the limb, consideration must be given to the fact that such inputs are necessary not only to provide information about the properties of the peripheral plant, but also about the initial position of the limb. While this may not be critical for the final position (Bizzi et al., 1976), it is critical for the accurate scaling of the early phasic events. This information could be incorporated in the motor program at a spinal level, perhaps using propriospinal neurons whose patterns of convergence have been elegantly described by Professor Lundberg (this volume) or at a higher level and/or after a reaction time. While the segmental actions of particular afferent systems may be important in the regulation of slow movements and in respiration (Sears, 1973), in the case of rapid limb movements, we are more struck by the constraints which they seem to provide. It is within these constraints that the descending actions of central programs must operate. Thus, afferent input may, to paraphrase Weiss (1941), play more a constructive than a regulatory role. This could favor, during the acquisition of skill, the development of a pulse-height control policy, since the initial burst of neural activity will, as its action is exerted, be automatically terminated by the silent period. Since afferent input provides the information required to reset the gain of the input-output relations of rapid targeted movements, its role must certainly be constructive since it ultimately enables the purpose of such movements to be achieved.

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DISCUSSION PERIOD

BROOKS: Does the animal's accuracy go down as the displacement and velocity go up, and is the standard deviation of the error greater for higher velocities?

GHEZ: The accuracy of responses made by the cats to perturbations of randomly varied amplitudes was not dependent on their velocity *per se*. Rather, as is also the case in human subjects performing a step tracking task with varied step sizes, accuracy is most heavily dependent on range effects, that is, the average value of preceding perturbations (POULTON, 1974) and boundary effects, that is, the difference between the maximal possible extent of display motion and that produced by the particular perturbation (BUCK, 1976). The latter effect results in a tendency towards undershooting for the largest perturbations. That both of these factors are important, underscores the role of predictive mechanisms in rapid movements.

Of course, movements of constant amplitude can be performed with different peak velocities and then, accuracy may decrease proportionally as velocity increases (FITTS, 1954). With varied amplitudes of movement, the error will then increase as a function of the ratio between velocity and displacement. Although this suggests an inverse relation between the duration of the initial pulsatile output and the error, we have not, as yet, studied this in the cat.