THE ORGANIZATION OF SIMPLE, SKILLED MOVEMENTS

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A simple model of the limb is proposed in which the limb is visualized as a single damped spring with mass. 'Movements' in the model are produced by step changes in spring stiffness. Comparisons are made between the behaviour of the model and step-tracking movements made by human subjects. The model provides an adequate qualitative description of these simple limb movements and provides an explanation for such observations as the linear relation between movement amplitude and peak velocity seen in humans. Changes in movement performance produced by the instruction given to the subject and in some pathological states can be explained on the basis of the model.

From a comparison of the responses to sudden perturbations it is suggested that the stretch reflexes act to return the limb as quickly as possible to its non-perturbed or learned trajectory.

How do we move our limbs from one position to another? In recent years attention has been particularly directed at the role of the central nervous system in the generation and control of movement. The advent of single unit recording in the behaving animal has led to detailed studies of such areas as sensory and motor cortices (10,11,12,19,20,52), cerebellum (42,46,47), basal ganglia (16,17) and parietal cortex (15). Although studies of this type have deepened our understanding of the central motor apparatus, it must be acknowledged that we are still not much closer to being able to describe how the central systems operate or interact in the generation of simple movements.

One area which has been somewhat neglected by students of motor control is muscle, the final effector of the system. As Partridge (40) recently reminded us, 'muscle is the final filter through which all motor output must pass'. Partridge also suggested that, in some situations, muscle may act as something more than a final filter. Rather it may be that the central nervous system utilizes the mechanical properties of muscles to more easily accomplish desired movements.

Some experimental evidence for this point of view has come from studies by Feldman (22,23) and by Bizzi and co-workers (2,3,41) on the maintenance of static limb position. Feldman suggested that when the limb is in a static position a state of equilibrium exists between the forces acting about the joint (Fig. 1A). Since, if a static position is being maintained, muscle velocity is zero, the force exerted by the opposing muscles is determined by their length-tension curves (Fig. 1B). Additionally he suggested that movement to a new position would be accompanied by a shift in the relevant length-tension curves, by, for example a change in slope. Such a change in slope is equivalent to a change in the stiffness in the
Fig. 1: Representation of a simple model of the human arm. As indicated in A, movement of the forearm is considered to be under the control of two springs representing the opposing muscles acting about the elbow joint. In B are shown diagrammatically the length-tension curves of the opposing muscles. The slope of these lines indicated muscle stiffness. Note that movement to a new limb position (change in muscle lengths) can be produced by altering the stiffness of the muscles.

Thus as indicated in Fig. 1A, the muscles might be visualized as simple springs whose stiffnesses could be altered.

One of the most striking but least recognized characteristics of simple arm movements made by humans or by well trained primates is their reproducibility or degree of stereotyping. This is illustrated in Fig. 2 in which average records of movements made during a visual step-tracking task are shown. One is immediately struck by the low variance in these average records. This high degree of repeatability of the limb trajectory from movement to movement is also pointed out by examination of phase plane plots of the individual movements (Fig. 28). These representations of the movement trajectory are obtained by plotting limb velocity (ordinate) as a function of limb position (abscissa) and are relatively sensitive to minor variations in limb trajectory. As is seen in Fig. 28 these plots indicate little variation in limb trajectory in a series of movements.

Although the limb trajectories during performance of well practised movements show little variability, there is considerable variation in the EMG activity associated with these movements (Fig. 3A). The movements shown in this figure were associated with a typical pattern of EMG activity first described by Nachhold and Altenburger (48) and more recently by Hallett et al. (28). The EMG pattern consists of an initial burst of activity in the agonist (biceps) termed B1 by Hallett et al. Following this initial
Fig. 3: In A are shown average records from step-tracking movements by a normal subject. Each trace is the average of 20-25 movements. An upward deflection of the position trace indicates flexion of the forearm. Standard deviations are shown on the position trace and on the EMG records. The EMG bursts associated with the flexion movements are identified using the nomenclature of Hallet et al. (28). In B are shown the interrelation between the integrated EMG signal during bursts associated with flexion movements in A. The lines through the points are the best-fit linear regression lines and have the equations

\[
B2 = 270 + 0.90(B1) \quad r = 0.48
\]

\[
T1 = 307 + 0.01(B1) \quad r = 0.05
\]

\[
B2 = 544 + 3.84(T1) \quad r = 0.37
\]

Now, if movements are generated or controlled primarily by this phasic EMG activity one would have expected considerable variation in movement trajectories even though the limb could end up at the same final position. One possible explanation could be that changes in the activity of the agonist (for example) are compensated for by changed antagonist activity. For example, if the magnitude of the initial agonist burst (B1) were decreased, one might expect to see a concomitant decrease in the T1 amplitude. That such compensatory changes do not occur is indicated in Fig. 3B which shows correlograms of the integrated EMG components from a large series of movements. The possibility that the movement trajectory is not primarily determined by the associated phasic EMG activity must therefore be considered.

(It must, however, be pointed out that it is slightly naive to consider only biceps and triceps muscles; other muscles undoubtedly contribute to these movements. In addition it is possible that varying EMGs in the prime movers of the forearm may be compensated for or be in response to alterations in shoulder positioning, etc. An alternate explanation will be described below).

The suggestion I would like to put forward is an extension of the idea formulated by Falmam. This is that the trajectory of limb movement is determined to a large extent by simple changes in the limb mechanical properties (13). I would suggest that an adequate model for the generation of the kinds of movements I have been describing is provided by considering the limb as a simple second order system. That is, one in which the limb behaves as if it were a damped spring having mass. Such a model for muscle has been used for at least half a century, for example by Gasser and Hill in 1914 (37). The usefulness of the model is attested to by its continued use (1,37,38,43).

In order to test this hypothesis an analog model of a damped spring with mass was utilized (13). That is the following equation was modelled

\[
m \ddot{x} + n \dot{x} + kx = F_0\]

Mass (m), viscosity (n) and stiffness (k) were independently variable as was the 'externally' applied force to the system (F₀). The circuit utilized is shown in Fig. 4C. Movements in the model were produced by a step change in the slopes of the length-tension curves of the relevant muscles (Fig. 1A). Note that the model was a lumped model; the two antagonistic muscles were not modelled separately. In Fig. 4B are shown records of 'movements' obtained from the model which can be compared with those from a normal human subject in Fig. 4A. Casual inspection reveals little difference between them. Shown in Fig. 4D are phase plane plots of movements of different amplitudes performed by a human subject and by the model. The different amplitude movements in the model were obtained by step changes to different stiffness values from the same initial stiffness.

Recall that movements in the model were generated by step changes in resting stiffness. How dependent is the movement trajectory on variations in the final stiffness? That is, we are asking the same question as we asked about the variability in the EMG records: how much effect on the actual limb trajectory will random variations in the final stiffness value have? By considering how springs behave it is obvious that for isotonic movements the final position (or spring length) will vary with the stiffness. For an ideal spring, \( F = -kx \). Length, \( x \), is thus linearly dependent on \( k \) if the net force \( F \) is constant. One would thus expect some
shown that this relationship holds for step-tracking movement when the subject is required to vary his motor output over a wide range. In Fig. 5A are shown velocity-amplitude relationships from experiments in which subjects performed step-tracking movements. Trials were made with the subject being given the following instructions: a) move as accurately as possible, b) move as rapidly as possible and c) move in whatever manner you wish. For this latter instruction no visual target was provided, the subject being free to choose his own movement amplitude. Subjects were cued to start by an auditory tone. A linear relation between movement peak velocity and amplitude is seen in each case. Correlation coefficients of the individual regression lines ranged from 0.85 to 0.98. The effect of the different instructions, which could produce a doubling of the peak velocity, is to alter the slope of the relationship: the basic linear character is unchanged. In such a linear interrelationship between the parameters of the movements indicative of the 'organisation' of the movements or of the method by which the movements are generated? If so, one would suggest that all the different movements for Fig. 5A were generated utilizing some common organization or program. The differences in the movement output required to the different instructions might have been generated by altering variables in this common motor program.

It should be noted that such relations between movement amplitude and peak velocity do not imply that movement time is independent of movement amplitude. Preliminary experiments indicate that movement time is directly dependent on movement amplitude as had been shown for arm movements in monkeys (5,6,9). The movements described here do not, however, obey Fitts' Law which describes interrelationships between movement time and movement amplitude and accuracy. This is presumably due to the lack of a mechanical end-point in these movements in contrast to the movements studied by Fitts (24,25).

In Fig. 5B are shown similar relations obtained from the model of the limb. The peak velocity-amplitude relationship is well approximated as a linear function. The model thus predicts the relationship observed in human subjects. On what is the slope of this relation dependent? The answer to this comes from considering that movements in the model were generated by a step change in spring stiffness from some resting or initial value (k€) to some final value (k€). As has been described (Fig. 4C) the amplitude or final end-point of the movement will depend on the value of k€. What of the initial stiffness? As indicated in Fig. 5A, changing the initial stiffness changes the slope of the peak velocity-amplitude curve for movements in the model: an increased slope is produced by increasing the resting stiffness. This observation accords with the common experience of tensing or co-contracting in the expectation of performing a very rapid movement.

Another observation explainable in terms of this model is the decrease in movement velocity when external masses are added to the limb. As shown in Fig. 5C, increasing the mass in the model produces a decrease in the slope of the peak velocity-amplitude curve. That is, for a given movement amplitude, velocity decreases with increasing mass.

This model of movements generated by relatively simple changes in the limb mechanical properties may bear on some of the alterations in movement performance seen in pathological states. For example, the hypotonia observed

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**Fig. 4:** Movements made by a normal human and by an analog model of the limb. Shown in C is a simplified circuit diagram of the analog model of the limb (see text). In A are shown position and velocity records of movements made by a normal subject during performance of a visually guided step-tracking task. The target center separation was 65 deg and the total duration of the trace is 15 sec. In B are shown analogous traces obtained from the model, in which 'movements' were produced by a step change in the resting spring constant. In D are shown phase plane plots of movements of different amplitudes obtained from a normal subject (upper set) and from the model (lower set). Each phase plane from the subject was derived from the average of 10-15 movements. Movement amplitudes ranged from 16-80 deg. Different amplitude movements in the model were produced by step changes to different final stiffnesses from the same initial stiffness (DC250479, DC900379).

The variation in the limb's trajectory associated with the changed movement amplitude. This indeed can be seen in the phase planes from the human subjects shown in Fig. 2. It should be noted however that the model is a lumped model; no attempt was made to separately model the antagonistic muscles. If one considers an ideal spring system, proportional changes in the stiffness of two springs acting in opposition to each other would result in no change in length of either of the springs. Such changes in stiffness would tend to counteract each other.

The behaviour of this mechanical model of movement is consistent with another heretofore puzzling aspect of simple movements. It has been found that there is a linear relationship between peak velocity of movement and movement amplitude (4,15). Recent experiments in our laboratory (15) have
adequate. Any such model must, however, be able to describe not only normal operation of the modelled system but must also respond to external disturbances in the same way as does the modelled system. Comparisons were therefore made of the behaviour of the model and of normal human subjects in response to brief torque perturbations applied during movement (13,14). In particular, attention has been paid to the effect of perturbations on the movement trajectory. As has been described (13) and is illustrated in Fig. 6A, one of the most striking observations with the human subject was that the limb trajectory following perturbation was very similar to the trajectory of the non-perturbed, control movements. This however did not occur in the model and it thus appeared that the model was inadequate.

Fig. 6: Effect of sudden perturbations during 'fast' and 'slow' movements. Shown in A-D are records of limb position and velocity and of rectified surface biceps and triceps EMGs during performance of step-tracking movements. Each record is the average of 10-15 movements. A and B were obtained from 'fast' movements and C and D from 'slow' movements. The vertical dashed lines in E and F indicate the time of onset of a 50 msec perturbation opposing the flexion movements. The corresponding phase planes are shown in E (slow movements) and F (fast movements). Note that following the perturbation in E the limb trajectory closely resembles the control trajectory whereas in F the control trajectory was not regained following the perturbation. In G the difference between the biceps EMG during the perturbed and non-perturbed movements is plotted. EMG traces were aligned at the onset of the EMG activity preceding movement and integration performed over discrete bins preceding and following onset of the perturbation. The solid bars show data from the 'slow' movements and the open bars from the 'fast' movements. The thin vertical bars indicate movement onset and the vertical dashed line the perturbation onset. Note that 'reflex' activity is present in the period 30-80 msec following the perturbation in the 'slow' but not in the 'fast' movements (A-D VNI40379, E-G RJOG0379).
One difference between this simple model and real limbs is that the model does not generate reflex responses to sudden perturbations. As seen in the ENG records in Fig. 6B such responses were indeed evoked by the perturbation in the human subjects. The possibility that reflex responses to sudden limb displacement could be involved with the return to the control trajectory was tested by incorporating a 'pseudo-reflex' into the model. This reflex consisted of a force following the perturbation and in a direction opposite to the perturbation. It was found that the trajectory of the perturbed movements in the model could be made to match the control trajectory as closely as desired by appropriate manipulation of the magnitude of the pseudo-reflex (13,14). This then suggested that the degree of matching of limb trajectories following perturbations depends on the gain of the stretch reflexes.

Little or no reflex response occurs following a perturbation applied during movements where the subject is required to move as rapidly as possible (14,18) (Fig. 6A, D). One would therefore predict that a perturbation applied during such movements would produce a more marked disturbance of movement trajectory which would not return to the control trajectory. Such an effect has indeed been seen and an example is shown in Fig. 6E, F. For the experiment in E, the subject was requested to move 'as accurately as possible' between the target zones. As was described, the trajectory following the perturbation closely matched the control trajectory. For part F of the figure the subject was asked to make rapid movements as indicated by the increased peak velocity of movement in the phase planes. In this case, the movement trajectory following the perturbation did not return to the control trajectory. In G of this figure are shown the differences between the integrated ENGs of the perturbed and non-perturbed movements. In this period from 30-100 msec following the perturbation, activity is seen in the slow movements which is not present in the fast movements. It is in this time period that one would expect any reflex responses to the perturbation to be present (31,32).

As has been clearly stated by Marsden (33) "It is generally accepted that the muscle spindle machinery possesses the properties of a length servosystem and generates a constant muscle length". Recent activity in response to sudden perturbations has thus been seen as a load compensating system attempting to restore muscle length and thereby restore limb position. This view has recently been questioned by Houk (30,37) who has suggested that the stretch reflex acts primarily to linearize the length-tension properties of muscle. The value of this action is that "... the central nervous system probably has access to a model of the system that is controls and ... the model is bound to be simpler than the neuromuscular system behaves in a linear manner" (37). The data presented here and elsewhere (13,14) also suggest that the reflex responses to sudden stretch are not simply involved in length regulation. In agreement with Houk, I would suggest that the CNS has a model of these simple, well learned movements and that on the basis of learning the CNS can predict limb velocities at any limb position during a given movement. The action of the stretch reflex is to return the limb to some point on this known trajectory so that the CNS can predict the future course of the movement. It must be remembered that in the experiments which have been described the perturbation which was applied was always of the same magnitude but was applied randomly throughout a series of movements. Initial observations indicate that limb trajectory does not return to the control trajectory following the initial application of the perturbation. Thus, in this action of returning the limb to a known state the stretch reflex may be adaptive as has been indicated in other situations (36).

What conclusions can be drawn from the foregoing? First, it appears that many of the characteristics of simple step movements by normal human subjects are explainable if the movements are generated or programmed through changes in the limb mechanical properties. The studies with the model suggest that a step change in limb stiffness would suffice to produce movements similar to those actually observed. It must, however, be emphasized that there are indeed other factors involved in the performance of these simple movements. The question must be raised as to the function of the phasic ENG activity seen in association with movements (cf. Figs. 3A, 6A, C). Current views on the phasic ENG components undoubtedly owe, in great part, to the analysis of Stetson and Bouman (44) who suggested that "the excursion of the limb may be divided into three phases (1) the sudden impulse due to the momentary contraction of the unopposed driving muscles; (2) the free phase, during which the limb swings by its own momentum and has no dynamic connection with the pivot joint; (3) the arresting phase, during which an outside obstacle, or the contraction of the antagonists stops the movement". At least for 'fast' movements, it has been suggested that the three phase pattern of ENG activity is centrally programmed and only partially under the influence of peripheral events (25,28). Recent studies in our laboratory (8,15) have however indicated the following. First, a burst pattern of ENG activity is seen in 'fast' movements which varies widely in terms of movement amplitude and/or duration. Secondly, movement amplitude and velocity are poorly and inconsistently related to the magnitudes or durations of the various ENG bursts. Surprisingly, when one looks at these movements which are made relatively slowly by asking the subject to be 'accurate' in his movements (cf. Figs. 3A, 6C) movement amplitude and velocity are strongly related to the initial burst of agonist activity. Finally, in contrast to what was suggested by Hallett et al. (26) it was found that the initial burst of agonist activity could be strongly modified by a perturbation applied preceding the onset of the burst.

In view of the above I would suggest that the phasic drive to alpha motorneurons which is associated with these step movements may subserve different functions under different contexts in which the movements are made. In general their role is to augment or assist the underlying mechanical mechanism for movement generation. Contextual information has been found to be of importance in the generation of cortical or muscular responses to sudden limb perturbation (21). I would suggest that the subject's interpretation of the experimenter's requirements for such things as accuracy, speed, etc. lead to a variable modification of the underlying or base movement by the generation of the phasic input to the muscles.

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THE ORGANIZATION OF SIMPLE, SKILLED MOVEMENTS

ELECTROMYOGRAPHIC RESPONSES TO SUDDEN TORQUES ABOUT THE ANKLE JOINT IN MAN

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ABSTRACT

Measurements of the myotatic reflex in soleus and lateral gastrocnemius muscles show a latency of about 40 ms and an amplitude which is linearly and highly correlated with the rate of muscle stretch. The reflex gain is linearly proportional to the level of tonic voluntary activation and is proportionately reduced by the tonic contraction of the antagonist. The anterior tibial muscle behaves similarly except the myotatic reflex latency is on the order of 80 ms in relaxed foot and the latency decreases with tonic contraction. The EMG responses in these muscles in the interval beyond that of the myotatic reflex (> 100 ms) depend on the stimulus parameters, prior instructions, and subject expectation. These post-myotatic responses are voluntary, triggered reactions and unlike myotatic reflexes, they are relatively insensitive to prior tonic muscle activity. Vibration inhibits the myotatic reflex to a degree proportional to the vibration frequency. The post-myotatic component is not influenced by vibration. During phasic voluntary flexions of the ankle, prior to and at the movement's onset, myotatic responses are facilitated but post-myotatic reactions are not. Both responses are suppressed during the movement and for some time after its completion. We conclude that phasic movement is not assisted by effective load-compensating reflex mechanisms. If the planned movement is in error because of misjudgement or unexpected perturbation, effective correction requires a new and separate action.

INTRODUCTION

Over an extended period of time and a variety of experimental paradigms both in animal and human research, it has been recognized that the myotatic reflex, on its own, is not very effective in regulating limb position against changing loads. This has prompted many investigators, particularly in the past ten years, to deal with two resulting questions: What other functions might the myotatic reflex serve and by what mechanisms is load compensation finally achieved? Neither question has yet been satisfactorily answered.

The concept of servo-assistance to movement by spinal or supraspinal pathways has been extensively looked at. While the evidence for the existence of various closed-loop mechanisms is abundant, the evidence for the mechanical effectiveness of such loops is not.