1986

Control Of Human Limb Movement Trajectories

Warren Garfield Darling

Follow this and additional works at: https://ir.lib.uwo.ca/digitizedtheses

Recommended Citation
https://ir.lib.uwo.ca/digitizedtheses/1490

This Dissertation is brought to you for free and open access by the Digitized Special Collections at Scholarship@Western. It has been accepted for inclusion in Digitized Theses by an authorized administrator of Scholarship@Western. For more information, please contact tadam@uwo.ca, wlsadmin@uwo.ca.
NOTICE
The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct-print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c C-30.

THIS DISSERTATION HAS BEEN MICROFILMED EXACTLY AS RECEIVED.

AVIS
La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopy de qualité inférieure.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c C-30.

LA THÈSE A ÉTÉ MICROFILMÉE TELLE QUE NOUS L'AVONS RÉCU.
CONTROL OF HUMAN LIMB MOVEMENT TRAJECTORIES

by

Warren G. Darling

Department of Physiology

Submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

Faculty of Graduate Studies
The University of Western Ontario
London, Ontario
Permission has been granted to the National Library of Canada to microfilm this thesis and to lend or sell copies of the film.

The author (copyright owner) has reserved other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without his/her written permission.

L'autorisation a été accordée à la Bibliothèque nationale du Canada de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur (titulaire du droit d'auteur) se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation écrite.

Abstract

It is common experience that movements we make do not end at the same position during repeated attempts even if they begin from the same starting point. Movements made at higher speeds are generally associated with greater endpoint variability than slower ones. It is clear, however, that variability at endpoint must develop during movements, not just at their endpoint. Whether variability increases progressively throughout movements, or whether there are periods when decreases in variability occur, provide an indication of the mechanisms used in control of such movements. Recent studies have shown that muscular responses to perturbations applied during movement act to return the limb to a prelearned movement trajectory and not simply to movement endpoint. This is suggestive of an ongoing control of movement trajectory as opposed to ballistic or open-loop control. Experiments were carried out to further examine this hypothesis. The effects of practice and of movement speed on variability in the trajectories of human arm movements were studied. Variability in the neural activity associated with such movements was studied in the electromyographic (EMG) activities of movement-related muscles.

Variability in both movements and the EMGs of involved muscles depended on both movement speed and practice. Increases in movement speed produced greater variability in movement trajectories and muscle EMG activities. With practice, variability in the trajectories was reduced. The effects of practice on variability in the EMGs of related muscles depended on changes in movement speed during practice. If movement speed was kept constant during practice, EMG variability
decreased in association with decreased trajectory variability. In contrast, increases in movement speed during practice resulted in greater EMG variability in spite of decreased movement variability.

Detailed study of the time-course of development of variability throughout movements indicated that variability increased rapidly during acceleration. The rate of rise of variability was positively related to movement speed. Forces producing limb acceleration were, therefore, highly variable and their variability depended on movement speed. Near the end of acceleration and during deceleration movement variability decreased. Such decreases in variability are indicative of corrective mechanisms acting to compensate for the variable accelerative forces.

The variations in movement related phasic EMGs were not random. Deviations from intended movement trajectories produced by variations in the first agonist burst which initiates movement were accompanied by variations in antagonist muscle activity which returned the limb toward the intended trajectory. Reduced variability in movements in spite of greater variability in the agonist-antagonist EMG patterns was produced by this mechanism.

One mechanism which might underlie control of antagonist EMG patterns to compensate for agonist variability is the use of afferent feedback from sensory receptors in the moving limb. This was studied by applying vibration to tendons of muscles involved in movement to interfere with the sensations produced by muscle receptors. Vibration during acceleration strongly influenced muscle activity and movements. This suggested that sensory feedback received early in movements was used in predictive control of later phases of movements.

A new model for limb movement control was developed. This model
incorporated properties of models for final position control and movement variability. Two important features of the models were: (1) separation of phasic and tonic muscle torques and (2) an interdependence or linkage of the phasic torques produced by agonist and antagonist muscles. This linkage of agonist and antagonist torques was shown to be important in reducing variability of fast movements which are associated with large variabilities in individual muscle torque patterns.

It was concluded that the entire movement trajectory, rather than final position only, is controlled by the nervous system. The control is complex, however, because of the inherent variability in the motor system. During practice refinement of the linkage of agonist and antagonist muscle activation patterns is therefore critical in reducing variability in movements as speed increases.
Acknowledgements

I wish to express sincere thanks to my supervisor, Dr. David Cooke, for his guidance and suggestions. Thanks also to my co-supervisor, Dr. Keith Hayes, for helpful discussions.

I also wish to extend thanks to other faculty and graduate students for their support and friendship.

Finally I wish to express thanks to my wife, Patricia, and to my children, Bradley and Nicole, to whom this thesis is dedicated.
# TABLE OF CONTENTS

CERTIFICATE OF EXAMINATION .................................................. ii
ABSTRACT ........................................................................ iii
ACKNOWLEDGEMENTS .............................................................. vi
TABLE OF CONTENTS .............................................................. vii
LIST OF TABLES ................................................................. ix
LIST OF FIGURES .............................................................. xi

INTRODUCTION ...................................................................... 1

HISTORICAL REVIEW ........................................................... 8

I. Classification of movements ............................................. 8
II. Mechanical properties of muscles .................................. 14
III. Control of muscle variables related to limb movement .... 19
IV. Models of movement generation and control ................... 26
V. Effects of practice on movements .................................... 36
VI. EMG patterns associated with movements ...................... 39
VII. Summary ....................................................................... 45

METHODS ........................................................................ 48

I. Subjects ....................................................................... 48
II. Experimental paradigms ................................................. 48
III. Data recording .............................................................. 52
IV. Data analysis ................................................................. 53
V. Digital model of the limb .................................................... 61

RESULTS ........................................................................... 65

I. Effects of practice on movements .................................... 65
II. Effects of practice on variability of movements .............. 75
III. Effects of movement speed on trajectory variability ...... 92
IV. Point-to-point trajectory variability ............................... 95
V. Muscle EMG patterns ...................................................... 111
VI. Variability in muscle EMG activity .............................. 134
VII. Antagonist compensation for agonist variability in practiced movements .............................................. 146
VIII. Effects of vibration on movements ............................... 152
IX. Digital model of the limb ............................................... 179

DISCUSSION .................................................................. 207

SUMMARY ...................................................................... 237
List of Tables

Table

1. Mean durations of acceleratory, constant velocity and deceleratory phases of movements 1-10 and 51-60 performed under no-load and load conditions .............................. 73

2. Mean durations of acceleratory, constant velocity and deceleratory phases of movements 1-10 and 61-70 made from different elbow angles ........................................... 74

3. Mean rate of change of variability associated with initial movements (1-10) and practiced movements (51-60) ...................... 103

4. Correlations of mean rates of change of variability of the acceleratory and deceleratory phases for movements 1-10 to 51-60 ................................................................. 107

5. Correlations between mean rates of change of variability of the acceleratory and deceleratory phases for prolonged practice ................................................................. 108

6. Mean onset times of the antagonist burst for movements 1-10 and 51-60 and percentage changes in amplitude of the agonist and antagonist bursts from movements 1-10 to 51-60 ................................................................. 120

7. Mean onset times of antagonist bursts and percentage changes in agonist and antagonist burst amplitudes from movements 1-10 to 61-70 for flexion movements from flexed, extended and mid-range elbow angles .......... 122

8. Mean onset times of antagonists and percentage changes in burst amplitudes of agonists and antagonists from movements 1-10 to 61-70 for extension movements from flexed, mid-range and extended elbow angles ......................... 123

9. Mean correlations among the EMG activities of lateral, long and medial heads of triceps for flexion movements beginning from flexed, mid-range and extended elbow angles .. 124

10. Mean correlations among the phasic EMG bursts of lateral, long and medial heads of triceps for extension movements made from flexed, mid-range and extended elbow positions ................................................. 130

11. Results from multiple and single linear regression of percentage changes in EMG variability on percentage changes in peak velocity and trajectory variability during practice ........................................... 140
12. Correlations of EMG variability with peak velocity and trajectory variability of equally practiced movements ........................................ 143

13. Effects of vibration during acceleration and deceleration on the amplitude of 100° movements for each subject .......... 162

14. Effects of vibration during acceleration and deceleration on the amplitude of 300° movements for each subject .......... 163

15. Role of variability linkage in reduction of trajectory variability for practice simulations ................................. 206

16. Variability of acceleration duration and deceleration duration for movements 1-10 and 51-60 performed with a constant torque load .................................................. 253
List of Figures

Figure

1. Splitting of movements into acceleratory, constant velocity and deceleratory periods .......................... 55
2. Determination of onset times of phasic EMG activity and of the premovement reduction in antagonist EMG activity .... 59
3. Unpracticed movements by a subject who had never previously made movements of the laboratory apparatus .................. 67
4. Effects of practice on the time course of movement position and velocity ........................................... 71
5. Effects of practice on movement trajectories and their variability ......................................................... 78
6. Changes in trajectory variability of movements performed under the no-load condition with practice .................. 81
7. Changes in trajectory variability of movements performed under the load condition with practice .................. 83
8. Trajectory variability of unpracticed and practiced movements made from flexed, mid-range and extended elbow positions ......................................................... 86
9. Changes in trajectory variabilities of the acceleratory and deceleratory phases of 100 movements made from flexed, mid-range and extended elbow angles ......................................................... 88
10. Changes in trajectory variability with prolonged practice ...... 91
11. Changes in trajectory variability with practice up to 1000 movements ......................................................... 94
12. The relationship between trajectory variability and peak velocity for equally practiced movements .................. 97
13. Point-to-point trajectory variability of 100 movements ..... 99
14. Point-to-point trajectory variability of 300 movements ... 101
15. Velocity records from fast and slow movements .................. 105
16. Effects of movement speed on mean rate of change of variability during acceleration and deceleration ............ 110
17. EMG patterns of unpracticed movements .......................... 113
18. Different EMG patterns from unpracticed movements .......... 116
<table>
<thead>
<tr>
<th>Number</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>19</td>
<td>Effects of practice on muscle activity patterns</td>
<td>118</td>
</tr>
<tr>
<td>20</td>
<td>Agonist synergist muscle activity associated with practiced movements</td>
<td>125</td>
</tr>
<tr>
<td>21</td>
<td>Antagonist synergist muscle activity associated with practiced movements</td>
<td>127</td>
</tr>
<tr>
<td>22</td>
<td>Changes in variability of EMGs and movements with practice in which movement velocity increased</td>
<td>133</td>
</tr>
<tr>
<td>23</td>
<td>Changes in EMG and trajectory variability with practice in which there was no change in mean peak velocity</td>
<td>136</td>
</tr>
<tr>
<td>24</td>
<td>Changes in EMG variability with practice of movements from different start positions</td>
<td>139</td>
</tr>
<tr>
<td>25</td>
<td>Effects of movement velocity on variabilities of agonist and antagonist EMGs</td>
<td>145</td>
</tr>
<tr>
<td>26</td>
<td>Trajectory compensations in practiced 100 movements</td>
<td>149</td>
</tr>
<tr>
<td>27</td>
<td>Trajectory compensations in practiced 300 movements</td>
<td>151</td>
</tr>
<tr>
<td>28</td>
<td>Effects of antagonist vibration on 100 movements and related EMGs</td>
<td>155</td>
</tr>
<tr>
<td>29</td>
<td>Effects of antagonist vibration on 300 movements and related EMGs</td>
<td>157</td>
</tr>
<tr>
<td>30</td>
<td>Antagonist vibration effects on movement kinematics and durations</td>
<td>160</td>
</tr>
<tr>
<td>31</td>
<td>Agonist vibration effects on 100 movements and related EMGs</td>
<td>165</td>
</tr>
<tr>
<td>32</td>
<td>Effects of agonist vibration on kinematics and EMGs 300 movements</td>
<td>167</td>
</tr>
<tr>
<td>33</td>
<td>Effects of agonist vibration on movement kinematics and durations</td>
<td>170</td>
</tr>
<tr>
<td>34</td>
<td>Effects of vibration on EMGs of 100 movements</td>
<td>173</td>
</tr>
<tr>
<td>35</td>
<td>Effects of vibration on EMGs of 300 movements</td>
<td>175</td>
</tr>
<tr>
<td>36</td>
<td>EMG activity following vibration during acceleration</td>
<td>178</td>
</tr>
<tr>
<td>37</td>
<td>Results of control experiments on the effects of vibration applied during acceleration</td>
<td>181</td>
</tr>
</tbody>
</table>
38. Examples of 300 movements produced by the model and by a human subject ........................................................................ 184

39. Effects of movement start position, phasic torques and perturbations during movement on attainment of final limb position in movements generated by the model .................. 187

40. Agonist torque variability and movement variability relationships .................................................................................. 190

41. Point-to-point trajectory variability of movements produced by the model under no-antagonist, independent antagonist and dependent antagonist conditions ........................................... 194

42. Relationships between amplitude variability and average velocity during simulation of the rapid aiming paradigm ... 198

43. Relationships between amplitude variability and movement amplitude during simulation of the rapid timing paradigm ................................................................. 201

44. Practice simulations in a digital model of the limb .............. 204

45. Variability in durations of acceleration and deceleration throughout practice of 100 and 300 movements performed under the no-load condition ............ 250

46. Variability in durations of the acceleratory and deceleratory phases of 100 movements performed from flexed, mid-range and extended elbow angles ........... 252
Introduction

Movements may be generally classified on the basis of complexity (Holmes, 1939). Compound movements involve two or more joints and take place in two or three dimensions in space. Little is known about the control of such movements due to the large number of degrees of freedom associated with movement direction and extent as well as the numbers of joints and muscles involved. Movements about a single joint in one plane are usually described as simple limb movements (e.g. Holmes, 1939; Bouisset and Lestienne, 1973). The control mechanisms associated with such movements have been extensively studied because of the ease of description of these movements and their associated neuromuscular activity.

In spite of the term simple, the mechanisms by which such movements are controlled have yet to be fully explained. Much of the early work focussed on description and classification of the movements. Stetson and Bouman (1935) described 3 phases of movement: "(1) the sudden impulse ... due to 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 and (3) the arresting phase, during which an outside obstacle, or the contraction of the antagonists stops the movement...". The movements studied in this thesis were of the type which were stopped by contraction of the antagonist muscles. Thus mechanisms of control over voluntary limb movements directed at targets were studied.

Fundamental questions in motor control research are: what variables related to movement are controlled by the nervous system and in what way
are these variables controlled? The role of afferent feedback in
movement control has been the focus of a great deal of study. Important
variables of movement may be sensed and, through afferent feedback to the
CNS, provide the basis for servo control over these variables.
Sherrington investigated reflexive movements produced by spinal animals
and observed that much complex motor behaviour can be controlled at the
spinal level. This indicated a primary role for feedback from joint,
muscle and cutaneous receptors in movement control. More recently the
theory of servo control over voluntary movements was advanced (Merton,
1955). According to this theory muscle length is controlled through
feedback from muscle receptors sensitive to muscle length (muscle
spindles). Setting of desired muscle length through the fusimotor system
provides the reference signal to drive the muscle contraction and
movement. Although there were a number of problems with this concept
(see, for example, Stein, 1974) it raised the question of what muscle
variables are controlled during voluntary movement.

A popular approach among physiologists studying this question has
been to examine the function of reflexes evoked by altering the position
of limbs or the length of muscles in static situations or during
movements. Muscles contain receptors sensitive to length, velocity and
tension of the muscle. Thus the response of the nervous system to
alterations in muscle length, velocity and tension provides an indication
of how such information is used in control of these muscle parameters.
It has been suggested, for example, that muscle stiffness, equilibrium
length, force, velocity or length could all be considered as movement
variables controlled by the CNS (Stein, 1982). Indeed the control of any
one movement variable may depend on the purpose of the ongoing movement.
Another approach to the question of what movement variables are controlled has been to study the activity of cortical cells related to limb movement. Evarts (1968) studied the discharge rates of pyramidal tract neurons which were activated prior to wrist flexion and extension movements. It was observed that discharge rates varied in proportion to the load which was to be moved. Thus when greater muscle force was required higher firing rates were observed in the cortical neurons activated prior to and during the movements. Cortical control over muscle force was therefore suggested. Other studies of the activity of motor cortex neurons have also indicated that the force of muscular contraction is controlled by neurons in motor cortex (Evarts et al., 1983; Cheney and Fetz, 1980). Recently it has been suggested that motor cortex neurons are involved in controlling the kinematic features of simple movements (Bedingham and Tatton 1985). Recordings of the activity of motor cortex neurons in response to imposed limb movements showed that the firing probability of these neurons depended on limb position and its higher order time derivatives (i.e., velocity, acceleration and jerk). Limb position during movement could therefore be controlled in a predictive manner by motor cortex neurons responding to the time derivatives of limb position. Thus although the force of muscle contraction may be the regulated output of motor cortex neurons (Cheney and Fetz, 1980; Evarts et al., 1983) it is probably the motion of the limb that is controlled through predictive feedback of the derivatives of motion.

Although control of movement through sensory feedback has been the subject of considerable research many recent studies have focussed on central nervous mechanisms in movement control. In particular, the
concept of central nervous control over muscle mechanical properties has
been the focus of much investigation. Muscle stiffness, the slope of the
muscle force-length relation, has been the subject of a great deal of
study by muscle physiologists. It has been shown, for example, that limb
positions could be specified centrally through control over stiffness or
equilibrium lengths (length at which muscle force equals zero) of
opposing muscles about joints (Crossman and Goodeve, 1963; Feldman,
1966a,b). This led to the mass-spring hypothesis of final position
control that movement endpoint is specified centrally by control over the
spring-like characteristics of opposing muscles about joints. Movements
could be controlled centrally by altering the stiffness or equilibrium
length of the muscles around joints. The finding that deafferented
primates were able to reach the usual movement endpoint even when loads
were applied during movement provided strong support for this hypothesis
(Bizzi et al., 1976; Polit and Bizzi, 1979).

Central control over the elastic properties of muscle is the
presumed mechanism for movement control within the mass-spring model. An
important question within the mass-spring models is: what is the time
course of changes in muscle/limb properties during movement? Movement
trajectory, the time course of the change in movement position, will depend
on the time course of changes in muscle properties. Initially it was
thought that step changes in, for example, muscle stiffness or
equilibrium length (length at which isometric tension equals zero)
produced movements (cf. Cooke, 1979; 1980). Alternatively more complex
time-dependent changes in muscle properties may occur. In a recent study
it was shown that the time course of changes in muscle properties is
ramp-like rather than step-like for slow movements at least (Bizzi et
al., 1984). For faster movements more rapid, but not step-like, changes in limb properties may be used for movement generation (Hogan, 1984).

A common method for studying control over movement variables has been to study the effects of perturbations which alter the intended trajectory of limb movement. The results of studies by Marsden and colleagues indicated that muscle responses were part of a servo mechanism which opposed perturbations and aided attainment of final limb position (Marsden et al., 1972; 1976). Studies by Cooke (1979, 1980) showed that muscle responses to external disturbances act to return the limb to a prelearned (intended) phase plane trajectory of the movement (plot of velocity versus position during movement). This indicated that the relation between joint angular velocity and position throughout movement were of importance to the CNS.

Deviations from an intended movement trajectory may also occur naturally due to variations in the movements themselves. Much of the study of variability in movements has been limited to variability in movement endpoint or distance in association with the classic speed-accuracy tradeoff (Woodworth, 1899; Fitts, 1954). One set of models developed to explain this tradeoff are termed impulse-variability models (Schmidt et al., 1979; Meyer et al., 1982). In these models the variability of the impulses which produce movement are assumed to increase with increasing impulse magnitude. The variability in muscle force production is assumed to arise from variability in the neural signals which initiate muscular contraction. However studies to date have been limited to investigation of variability at movement endpoint only. Clearly endpoint variability must result from deviations from the movement trajectory which develop throughout movements. Control of
ongoing movements (cf. Eccles, 1968) may be involved in correcting for deviations from intended movement trajectories early in movements in order to reduce variability at later stages. This can be studied only if variability throughout movements is investigated rather than at movement endpoint only.

The impulse-variability and mass-spring models both address different, important concepts related to movement control. Mass-spring models have focussed on maintenance of movement end-position through the tonic activation of opposing muscles about joints. Only recently has the time course of equilibrium or virtual (cf. Hogan, 1984) position of the limb during movement been studied. Variability in movements has been largely ignored in mass-spring models. Impulse-variability models, in contrast, have ignored both the movement trajectory and how movement endpoint is maintained through tonic muscle activity. Rather, their focus has been on variability in the impulses which initiate and brake movements and how these influence variability in movement endpoint. These contrasting viewpoints emphasize the phasic (pulse-like) and tonic (step-like) components of movement control. Phasic impulses are involved in movement trajectory control but not in maintenance of movement endpoint. Tonic or step-like forces are involved in control of limb posture and final position. Indeed pulse-step models of movement control have been advanced for both eye (Robinson, 1968) and limb (Ghez, 1979) movements.

The hypothesis which directed the experiments presented in this thesis was that the entire trajectory of limb movements is controlled by the nervous system through control of phasic and tonic activity of opposing muscles about joints. Thus variability throughout the entire
trajectory of movements was examined in relation to the variability in the electromyographic (EMG) activity of movement related muscles. Since movement speed is known to affect variability at movement endpoint the effects of movement speed on variability in trajectories and in related muscle EMG activities were also studied. The objectives of these studies were to determine relationships between variability in movement trajectories and in the EMGs of related muscles as a function of practice and movement speed.
Historical Review

This review focuses on important concepts underlying the neuromuscular control of movement. The roles of muscle mechanical properties and of central commands in the control of movement and maintenance of limb postures are discussed in relation to current models of movement control. Studies of neuromuscular activity associated with movements are also reviewed. The results of previous investigations of the effects of practice on movements and muscle EMG activity are also discussed.

I. Classification of Movements

Movements have been classified in many different ways on the basis of complexity, speed and presumed mode of control (i.e., open-loop vs. feedback controlled). Holmes (1939) classified movements as either compound or simple. Compound movements are movements involving more than one joint which take place in 2 or 3 dimensions in space. Simple movements, in contrast, are those which take place at only one joint. Both simple and compound movements are also typically classified as either fast or slow. Fast movements are those which are step-like or ballistic, that is once they have been initiated they cannot be voluntarily modified (Richer, 1895). Slow movements are described as ramp-like and are thought to be controlled throughout their duration by visual and peripheral feedback. The speed of movements is, however, a continuous variable. Thus many movements often cannot be simply classified as either fast or slow (cf. Brown and Cooke, 1981).
A. Simple movements

The study of simple movements has been a major focus for motor control researchers. The reason for this lies in the ease of the analysis of these movements, both in terms of movements themselves and the associated neural/muscular activities. However, simple movements involving rotation at only one joint rarely occur outside the laboratory where there are no constraints placed on limb movements. Additionally, in the study of simple limb movements in the horizontal plane the need for postural support due to the effects of gravity are removed by providing support for the limb. Simple movements are studied because it is thought that neural mechanisms used in control of these movements may also apply in the case of more complex movements. That is, the rules of motor control developed for single joint movements may also apply to more complex movements. For example, recent studies have shown that simple elbow movements and more complex tongue movements associated with speech have similar characteristics (Ostry and Cooke, 1985).

The study of single joint movements in physiology has concentrated on the controlled parameters of these movements. The underlying question is how are the movements planned/programmed. Many possible control mechanisms have been advanced. The major hypotheses focus on the use of feedback versus open-loop control of either joint (e.g., joint angle, angular velocity) or muscle (e.g., length, velocity, force) parameters. An important theory in relation to control over muscle variables has been that final position of movements is controlled through the elastic forces developed in opposing muscle about joints (Crossman and Goodeve, 1963; Feldman, 1966a,b; Bizzi et al., 1976; Cooke, 1980; Kelso and Holt, 1980).
The control of muscle variables related to limb movement is the subject of a subsequent section in this review.

The importance of control over joint variables has been emphasized in several recent studies. For example Cooke (1979; 1980) proposed that the phase plane trajectory (plot of joint angular velocity versus position) may be controlled during simple movements. This was based on the finding that muscle responses to external perturbations imposed during learned movements resulted in return of the limb to a prelearned trajectory. This could not occur simply through control of either movement velocity or position. Hogan (1984) recently suggested that simple movements may be controlled in such a way as to minimize jerk, the time derivative of joint angular acceleration. Importantly, in the model advanced by Hogan it was hypothesized that the position trajectory of movements were controlled throughout their duration rather than by simple step changes in joint mechanical properties. These theories are discussed further in a later section on models of movement control.

B. Compound movements

The detailed study of compound movements in physiology has begun only recently as techniques developed for their recording and analysis. There has been a great deal of study of physiological mechanisms involved in gait control but relatively little study of voluntary movements of the upper limb. Recent studies of multi-joint movements of the upper limb have focussed on invariant features of the end-segment (e.g. hand) trajectory through space to different targets and the associated joint kinematics. An important question in such studies is whether movements
are planned in terms of the spatial trajectory of the end-segment (i.e., hand) or in terms of joint kinematics. It has consistently been observed that the hand follows a straight line trajectory to the target and that hand speed profiles are bell-shaped (Abend et al., 1981; Georgopolous et al., 1981; Hollerbach and Flash, 1982; Morasso, 1981; Soechting and Lacquaniti, 1981). Additionally, the hand speed profile appears to be scaled for movements of different speeds and distances and with varying loads. This scaling presumably simplifies the task of the nervous system in computing the joint position trajectories necessary to generate straight line trajectories of the terminal segment (cf. Hollerbach and Flash, 1982). Reaching movements therefore appear to be planned in terms of the spatial trajectory of the terminal segment (hand). Joint (shoulder and elbow) kinematic patterns would be specified subordinate to the spatial trajectory of the hand. Planning of movements in terms of joint kinematics has been suggested on the basis of studies finding a constant relationship between angular velocities of the shoulder and elbow during movement deceleration (Soechting and Lacquaniti, 1981). It has been shown, however, that for the particular type of movement studied by Soechting and Lacquaniti (1981) the ratio of shoulder to elbow angular velocity always approaches a constant during deceleration (Hollerbach and Atkeson, 1980). Multi-joint trajectories therefore appear to be planned in the spatial coordinates of the end-segment (i.e., hand) rather than of individual joints.

C. Fast movements.

Fast movements have been the subject of considerable study because
it is assumed that these movements must be wholly planned in advance of the movement. That is, once the movement is initiated there is insufficient time for visual or proprioceptive feedback to influence the movement. The study of fast movements therefore presumably allows one to examine open-loop, as opposed to closed-loop, control mechanisms in movement planning.

The underlying neural control mechanisms for fast movements have been inferred from the muscle activity associated with movement. An initial contraction of the agonist muscle, followed by its relaxation, initiates fast movements (Richer, 1895; Stetson and Bouman, 1935). Movement continues due to the limb's momentum until there is contact with an external object or contraction of the antagonist muscles to provide a braking force. An additional classification, called fast movements under tension, was described by Stetson and Bouman (1935). These fast tension movements were initiated from a postural state in which cocontraction of agonist and antagonist muscle existed. A sudden increase in contraction of the agonist initiated fast movement in spite of continued antagonist contraction, producing a fast movement under tension. Both types of fast movements were considered preprogrammed in that movements could be modified only through changes in the initial "set" of the motor system prior to movement.

D. Slow movements

The neural mechanisms underlying control of slow movements differ from those for fast movements because there is sufficient time for feedback to alter the course of the movement. As such it is generally
assumed that one can investigate feedback control mechanisms in movement control by studying slow movements.

Slow, tension movements have been divided into two types (Stetson and Bouman, 1935): (1) "movements of fixation" or "holding still" which were characterized by cocontraction of agonist and antagonist muscles which prevented movement and (2) "moving fixations" during which slow movement was accompanied by cocontraction of opposing muscles about the joint. Since these movements could be altered voluntarily throughout their execution they were assumed to be continuously controlled, probably through peripheral feedback mechanisms. Indeed the greater accuracy associated with slow, as opposed to fast, movements has been attributed to the use of visual feedback (Woodworth, 1899; Keele, 1968). Eccles (1968) proposed that slow movements were controlled by the cerebellum through its processing of peripheral feedback from the slowly moving limb.

E. Summary

Classifying movements implies that any given movement must fit into a certain type of class. While movements are easily grouped in the simple/compound classification it is generally less easy to classify movements as fast or slow. An exception may be the system for eye movement control. Saccades are examples of fast movements which apparently cannot be modified once initiated. However, recent studies have indicated that even saccades can be modified by sensory information after the beginning of the ocular muscle contractions on the basis of associated head movements (Bizzi et al., 1971). Smooth pursuit
movements, in contrast to saccades, require continuous utilization of visual information concerning direction and velocity of the moving target. During tracking of visual targets saccades are used for rapid acquisition of the target (Robinson, 1968). Smooth pursuit movements follow the saccades for accurate tracking of the target. Similarly limb movement control may consist of an initial ballistic impulse to move the limb rapidly toward the target and a current control phase during which feedback controls target acquisition (Woodworth, 1899). Thus many limb movements may not be easily classified as either fast or slow but may include components of both fast and slow movements.

II. Mechanical properties of Muscle

Movements are produced when muscles are activated by the nervous system so that force is generated and muscle length changes. Activation of muscle and force production is associated with both shortening (concentric) and lengthening (eccentric) contractions. The forces developed in muscle depend on its length and the velocity of contraction. The importance of muscle mechanical properties (stiffness and viscosity) in the control of voluntary movements is therefore obvious. Activation of agonist and antagonist muscles to produce appropriate accelerative and decelerative forces is necessary in order to produce movements which accurately reflect the intent of the performer.

The mechanical properties of muscle have received a great deal of study since the 1800's (for a more complete review see Partridge and Benton, 1981). These properties arise from muscle's structure. The contractile element of muscle is the sarcomere containing the contractile
proteins. Surrounding the sarcomere is connective tissue which forms the passive element of muscle. Both elements contribute to the observed mechanical properties of muscle—the dependence of muscle tension on muscle length and contraction velocity.

A. Force-length relationships

The effects of changes in length on the force produced by the contractile element are thought to be due to changes in the structure of the myofibril at the level of the sarcomere (Gordon et al., 1966). At resting muscle length stimulation of the contractile element results in maximum force production. Increases in length of the sarcomere cause decreased overlap of the actin and myosin proteins within the contractile element and, therefore, decreased crossbridge attachment between these proteins. Since force production is proportional to the number of active crossbridges, increases in muscle length result in decreased force production (Gordon et al., 1966). When the muscle is shortened to lengths less than resting there is overlapping of the the crossbridges and, thus, fewer active crossbridges resulting in decreased force production. Thus the contractile element force-length curve is the shape of an inverted-U with maximal force production at resting length.

The connective tissue surrounding the contractile element develops tension as it lengthens much like an elastic band or a spring. At resting muscle length or at shorter lengths this connective tissue is slack and thus produces no tension. Lengthening of the muscle past the resting length results in tension development in this component of muscle. In simple springs, the relationship between tension and length
is linear (Hooke's Law) and independent of the amount of stretch up to the elastic limit. The slope of the relation between force and length is spring stiffness and the length at which force is zero is the threshold or equilibrium length of the spring. The relationship between passive muscle tension and length is non-linear, indicating that the non-contracting muscle acts as a non-linear spring.

The force-length curve for the complete muscle is the addition of the curves for the passive and active components. Usually these curves are calculated based on maximal stimulation of the muscle (i.e., maximum muscular contraction). Under normal situations the amount of active tension produced by the contractile element is under voluntary control. The overall relationship between muscle force and length is therefore controlled by the level of motor unit activation of the muscle. Thus when muscles are activated to produce movements their stiffness is altered, thereby influencing force production as muscle length changes during movement.

The preceding discussion of muscle force-length characteristics is based on results of experiments in which the muscle was maintained isometric. The curves relating force and length under these conditions thus represent the static stiffness of muscle. The relationship between muscle force and length during changes in length depends on muscle's dynamic stiffness. For simple springs static and dynamic stiffness are identical, that is the ratio of muscle force to length is the same during stretch as in static situations. However muscle, due to its complex structure, has a different force-length relationship during imposed length changes. The stiffness of muscle is initially very high to small length changes (short-range stiffness). Once stretch exceeds a critical
length change force falls rapidly due to breakage of actin-myosin bonds. This is called yielding. After yielding, force once again rises with increasing muscle length but the stiffness (slope of this relation) is much less than the short-range stiffness. This dynamic stiffness is proportional to the initial force of the muscle, whether this is developed by recruitment or rate modulation of motor units (Houk and Rymer, 1981).

B. Force-velocity relationships

Muscle force is also dependent on the rate of muscle shortening or lengthening (Wyman, 1926; Levin and Wyman, 1927). During concentric (shortening) muscular contraction, muscle tension decreases as it shortens under load. With increased velocity of shortening there are greater decreases in muscle tension. This produces the well-known force-velocity curve of muscle. Equations which describe the shape of this curve have been developed by Fenn and Marsh (1935) and Hill (1938). The mathematical relationship described by Hill (1938) was related to the heat produced by the muscle while shortening. The force-velocity relationship has been attributed to: (1) decrease in tension as the cross-bridges in the contractile element break and reform as the muscle shortens and (2) fluid viscosity in both the contractile element and the connective tissue (Winter, 1979). The fluid viscosity must be overcome by the active force of the contractile element, resulting in less force transmitted to the tendon.

During eccentric (lengthening) contraction, muscle force increases as the velocity of lengthening increases (Wyman, 1926; Levin and Wyman,
1927). The curve is an extension of the force-velocity curve for concentric contractions. Unfortunately there has been little research on this property of muscle in spite of the fact that in intact humans or animals much of the work done by muscles is in eccentric contraction. For example the work of the antagonist muscles in braking the limb during simple movements is performed with the antagonist lengthening. Force increases as a function of the velocity of lengthening because the force required to break cross-bridge links is greater than that required to maintain it at its isometric length. In addition the viscous friction in the connective tissue affects lengthening as it does shortening of the muscle. However the viscous forces must be overcome during lengthening, resulting in higher tendon forces.

C. Summary

How do these relationships between muscle force, velocity and length influence limb movements and maintenance of limb postures? Maintenance of joint angle or limb position depends only on the static force-length curves of the opposing muscles about joints because muscle length is constant. Thus the limb will be at rest when the elastic forces produced by opposing muscles exactly balance. These can be controlled voluntarily through the level of motor unit activation. Increasing the level of muscular contraction results in development of greater length dependent forces in the muscles. Thus the equilibrium position of the limb can be altered by activation of muscles, resulting in movement. During discrete movements there is shortening of agonist muscles and lengthening of antagonist muscles. The forces developed by these muscles
will therefore depend on their length, velocity of shortening or lengthening and on the amount of motor unit activation (Winter, 1979; Partridge and Benton, 1981). The nervous system must take into account the muscle mechanical properties when generating neural activity which activates muscle in order to produce appropriate accelerative and decelerative forces for movement.

III. Control of muscle variables related to limb movement.

In order to control movements the nervous system must control muscles which produce the forces necessary to initiate and brake limb motion. As detailed in the previous section muscle has complex non-linear viscoelastic characteristics which are controlled by the level of neural activation of the muscle. Movements are associated with changes in length and velocity of muscle which also influence the force developed by the muscle as a consequence of neural activation. Thus neural control over muscles in a manner appropriate to produce purposeful voluntary movements is indeed a complex task. Methods by which this task may be simplified have been the focus of much neurophysiological research. Three popular methodologies exist for simplification of this problem. One is the use of feedback from muscle/limb receptors to aid in control over particular variables (e.g. muscle force, length, velocity). Negative feedback provides a powerful control mechanism as attested to by its widespread use in physiology and engineering. A second method is linearization of the non-linear force-length-velocity characteristics of muscle through, for example, feedback. Finally the possibility that the nervous system limits its control to only one variable (e.g. muscle
stiffness or length) may also simplify the nervous system's task in motor control.

A. Feedback control of muscle force and length

Muscles contain sensory receptors which respond to muscle contraction and limb movement. Primary receptors in muscle include the muscle spindles which detect both absolute muscle length and rate of change of length and the Golgi tendon organs which respond to changes in muscle tendon tension. Since both muscle length and its derivatives and muscle tension are measured by sensory receptors it is natural to assume that these parameters are controlled.

The distribution of afferent fibers from sensory receptors in muscles to the spinal cord suggest the possibility that muscle length and force may be controlled through feedback. The afferent fibers of muscle spindles (group Ia and II) are arranged such that they excite homonymous motor neurons and inhibit antagonist motor neurons. Thus, lengthening of the muscle is opposed by concentric force production in that muscle and inhibition of the opposing shortened muscle as a result of spindle excitation. Thus length feedback from the muscle spindles may be used to oppose changes in muscle length. Muscle spindle afferents are also known to project to the primary sensory and motor areas of cerebral cortex (Phillips and Porter, 1977) and thereby form a possible trans-cortical spindle reflex loop. Muscle spindle feedback has also been shown to contribute to conscious perception of joint position (Goodwin et al., 1972). Feedback from muscle spindles may therefore be used at a number of levels in the motor system for movement control.
Afferent fibers from GTOs are distributed such that they inhibit homonymous muscle contraction. Thus production of muscle force greater than some threshold value will result in inhibition of that muscle's contraction and decreased force production. Servo control over muscle force is, therefore, also possible.

Based on the presence of the fusimotor system to control muscle spindle sensitivity Merton (1953) proposed a servo theory of movement control. The specification of a desired muscle length through the fusimotor system would lead to movement by activation of alpha motor neurons through feedback pathways. Activation of the fusimotor neurons centrally will excite the muscle spindles which, through afferent feedback pathways, will excite that muscle and its antagonist until the specified length was reached. Although this theory was attractive there were a number of problems with control of movement in this manner relating to gain, delays and instability in the length feedback system (for a review see Stein, 1972; 1982). These problems led to the development of servo assistance (Matthews, 1972; Stein, 1974) and conditional feedback (Houk, 1972) theories.

The servo assistance theories were based on the use of alpha-gamma coactivation in the control of movement. Movements were thought to be initiated through the alpha system and the muscle spindles were "programmed" through the gamma and beta (skeletal-fusimotor) systems to detect differences between actual and intended muscle length and velocity throughout movement (cf. Matthews, 1972; 1981). Muscle spindle feedback would then assist in movement performance only if movements did not proceed as planned (i.e., conditional feedback). Evidence for alpha-gamma coactivation has been provided from experiments in animals (e.g.
Prochazka et al., 1979) and in humans (Hagbarth and Vallbo, 1968; Vallbo, 1970; 1971). Thus spindles in the shortening agonist muscle may provide servo assistance in movements. However, during fast movements in which velocities of muscle shortening exceed about 0.2 resting lengths (of muscle)/sec it is probable that fusimotor action is unable to maintain spindle sensitivity. Thus servo assistance may be limited to relatively slow movements.

Servo assistance theories have focused on control of the agonist muscle which shortens during movement. Alternatively, changes in muscle length during joint movement could be measured through the length of the antagonist, lengthening muscle. This would have the distinct advantage that muscle spindles are very sensitive to lengthening of the muscle. This concept has received support from studies employing vibration of muscle tendons during voluntary movement (Capaday and Cooke, 1981; 1983). Vibration is known to activate muscle spindles which are sensitive to small changes in muscle length (Burke et al., 1976a). Capaday and Cooke showed that vibration of the antagonist muscle during movement resulted in undershoot of the usual end position. This was consistent with excitation of antagonist muscle spindles by vibration since this would create the perception that the antagonist muscle was too long and/or it was lengthening too quickly. Movement undershoot would be expected since the perception would be that antagonist muscle length was longer than it actually was. Vibration of the agonist (shortening) muscle, in contrast, had no influence on movement end position. If servo assistance were active in the agonist one would expect movement overshoot since muscle spindle activation would indicate that the agonist muscle was too long or lengthening too quickly. Thus servo assistance in joint
movements may be provided through monitoring of muscle spindle afferent activity of the lengthening (antagonist) muscle rather than of the shortening (agonist) muscle.

Control over muscle force may also be possible through afferent feedback from GTO receptors. Individual GTOs provide an accurate signal of muscle force and could, therefore, be used in a force control system (Crago et al., 1982). However the gain of GTO feedback is very low at least in decerebrate cat preparations (Houk et al., 1970). GTO reflex gain may be higher in conscious man as indicated by studies of intercostal muscle reflexes during breathing movements (Newsom Davis et al., 1970; Sears, 1973). Indeed it was observed that during unexpected loading of the intercostal muscles during expiration an initial decrease in EMG activity was consistently observed which was attributed to GTO feedback (Newsom Davis et al., 1970). This finding has not, however, been replicated in studies of limb movements. Thus high GTO gain during active movement may be a peculiar characteristic of the intercostal muscles active in expiration during breathing. In recent studies it has been shown that GTOs may not be responsible for high-threshold force monitoring as illustrated in recent studies of force sensitive spinal interneurons (Cleland et al., 1982). Thus the potential use of GTO feedback in a force control system may be limited by its low gain (at least in association with limb movements) and limited range. This is important in view of the many factors affecting muscle force during movement (i.e., muscle length and velocity). The effects of fatigue (decreased muscle force for a constant neural input) and exercise (muscle hypertrophy and biochemical changes) make force control exceedingly complex (see Stein, 1982). Although human subjects can generate desired
force levels and maintain them under isometric conditions the role of GTOs in even this limited function is under question. Recordings from spindle afferents during isometric contractions and during slow movements indicate that their firing rate is modulated according to muscle force (Hulliger and Vallbo, 1979). Coactivation of the fusimotor system under isometric conditions results in activity in spindle afferents which varies according to muscle force. This may occur because central activation of greater numbers of alpha motor neurons at higher rates is probably associated with increased activation of gamma and beta (skeletofusimotor) motor neurons also. Since muscle length is constant there will be greater activity in muscle spindle afferents proportional to the exerted force/alpha motor neuron excitation.

Investigations of feedback control over muscle variables during movement have consistently shown that the feedback is not powerful enough to provide accurate control over these variables. The concept of servo assistance or conditional feedback is, however, a useful one since it allows for use of feedback to modify ongoing movements.

B. Control of muscle stiffness

The nervous system may control muscle variables singly, as discussed above, or in terms of relationships between these variables. Since both muscle force and length can be measured by muscle receptors their ratio, stiffness, could also be controlled by the nervous system. Muscle with intact reflex responses to both shortening and lengthening behave much more like a spring with constant stiffness than does a deafferented muscle (Nichols and Houk, 1976). Autogenic reflex responses to stretch
provide compensation for the yielding associated with breakage of actin-myosin bonds such that muscle stiffness remains relatively constant during stretch. Likewise, during muscle shortening (release) the autogenic reflex responses enhance the mechanical force decrease due to muscle's elasticity (i.e., decreased difference between actual and equilibrium length of the muscle). The relation between muscle force and length during imposed stretching or shortening is relatively constant if reflexes are present. In the absence of reflexes (i.e., afferent nerve supply to the muscle is cut) the relationship between muscle force and length during stretch and shortening are quite different. Thus with intact reflexes muscle behaviour resembles that of a simple spring (Nichols and Houk, 1976; Hoffer and Andreassen, 1981a,b). However stiffness is not constant over the complete range of muscle forces and indeed is proportional to muscle force at low levels (Hoffer and Andreassen, 1981a,b).

The preceding experiments were all carried out under static, postural situations in which the muscle was stretched or shortened in ramp fashion. Investigations of reflex function and stiffness during movements do not support the idea that muscle stiffness is regulated. For example stretch reflexes elicited in hindlimb muscles of walking cats are modulated in such a way that variations in muscle stiffness are enhanced, rather than decreased (Aldridge et al., 1981). Muscle stiffness was increased at points in the step cycle when this was advantageous to the animal (i.e., extensor reflexes during weightbearing). Similar variations in reflex responses during cyclic arm movements performed by normal humans have been observed (Mackay et al., 1980). Reflex responses tend to vary in parallel with both EMG and
muscle force, thereby enhancing rather than reducing stiffness variations. Thus there is little evidence for regulation of muscle stiffness during movement.

Theoretical arguments suggesting that maintaining muscle stiffness constant would simplify the brain's task in movement control have been advanced (Houk and Rymer, 1981). Constant stiffness would mean that the length and tension of muscle would be linked throughout movement. Movements could then be controlled by the brain based on a simple model of the relationships among neural activity and muscle force and length. As outlined by Stein (1982) there are a number of problems with this simplified control system. For example in some movement muscle length control is of obvious importance (e.g. surgery) while in others varying the muscle force-length relationship may be desirable (e.g. skiing, walking). Also cocontraction, typical of many movements, would pose theoretical complications to a system based on a regulated stiffness.

In conclusion control of muscle parameters during movement has been the subject of much study but with little success regarding what variables are actually controlled. The difficulties lie with the broad range of movements which human subjects and animals are able to perform. It is doubtful that any one variable is controlled. Rather, as Stein (1982) suggests, controlled parameters may be determined by the goals of the movements to be performed.

IV. Models of movement generation and control.

The development of models which explain certain aspects of movement control has been the focus of a major effort by motor control
researchers. As described in previous sections, the use of feedback to control or assist in the control of certain variables related to limb movement are the subject of much research. Other models have been developed in an attempt to explain behaviour in terms of centrally generated neural signals for movement. Two important concepts directing model development include the speed-accuracy tradeoff (cf. Woodworth, 1899; Fitts, 1954) and control over muscle properties. These will be the focus of the following discussion.

A. Models of the speed-accuracy tradeoff

Woodworth (1899) first studied the relationship between movement speed and accuracy in an attempt to develop a theory for movement control. Woodworth described aimed movements as consisting of two phases: (1) an initial impulse phase (similar to that of Stetson and Bouman, 1935) and (2) a current-control phase. During the initial impulse the limb was moved rapidly toward the target. This rapid movement was presumed to be preprogrammed and ballistic; that is it proceeded regardless of peripheral events occurring early in the movement. The current control phase occurred later and was presumed to correct any deviations from the intended path of the movement. The current-control phase depended on visual feedback for corrections to the movement since performance in the absence of vision deteriorated greatly. Both initial impulse and current control phases contributed to the speed-accuracy tradeoff since, in the absence of vision, movements with larger initial impulses were faster and less accurate than ones with small initial impulses.
Fitts (1954) further studied the speed-accuracy tradeoff using a reciprocal tapping task in which subjects moved a stylus alternately back and forth between two separate target regions. Fitts found a logarithmic relation between average movement time and the ratio of the distance between the targets divided by the targets' widths. This relationship became known as Fitts' Law. This law also holds for discrete movements (Fitts and Peterson, 1964). Fitts attributed the speed-accuracy tradeoff to noise in neuromotor channels that generate movements to targets. When the nervous system attempts to repetitively generate the same movement the force, direction and amplitude of all movements are continuous variables with a certain amount of variability. Performance of faster movements which require greater muscle forces is apparently associated with more variability or noise, presumably due to greater variability in the controlling neural signals.

Other models of the speed-accuracy tradeoff focussed on the role of feedback in movement control. In the iterative corrections model (Crossman and Goodeve, 1963; Keele, 1968) it was assumed that movement to a target consists of a series of discrete sub-movements. Each sub-movement is assumed to take a constant time and covers a constant proportion of the remaining distance from the center of the target. The movement stops when the remaining distance from the target is less than half the target width. Using these assumptions it has been shown mathematically that this model can account for Fitts' Law (Keele, 1968).

Evidence for the idea that movements are composed of a number of submovements has come from a number of studies. Discrete submovements are often observed near the end of movements, being associated with final acquisition of the target. These sub-movements were thought to be based
on visual feedback (cf. Woodworth, 1899). Crossman and Goodeve (1963) observed the presence of discrete submovements in velocity traces of slow wrist rotations. Others have also observed discrete submovements during slow movements (Stetson and Bouman, 1935; Brooks et al., 1973) and during learning (Brooks et al., 1983).

The concept of sub-movements therefore has a strong basis in many studies of movement control. However, the iterative corrections model cannot account for the finding by Woodworth (1899) that there is a speed-accuracy tradeoff in the initial impulse for movement (see Meyer et al., 1982). Since the initial sub-movement in the iterative-corrections model is assumed to be a constant proportion of the distance between the targets, there should be no speed-accuracy tradeoff (variability as a function of movement speed) in the initial impulse (i.e., the first sub-movement should travel a constant distance). Thus, the iterative corrections model may explain control during the current-control phase of the movement (cf. Woodworth, 1899) but not during the initial impulse of the movement. In an attempt to resolve this problem with iterative corrections models Meyer et al. (1982) proposed a model in which overlapping accelerative and decelerative impulses produced submovements which were variable in proportion to their size. In this way the initial movement toward the target would also show a speed-accuracy tradeoff as observed by Woodworth. This model was shown mathematically to also account for a speed-accuracy tradeoff.

More recent models have focused on central control over force/torque production by muscles. These recent models, termed impulse-variability models (Schmidt et al., 1979; Meyer et al., 1982), are based in the idea that the impulses (integrals of muscle torques exerted over time) which
produce movement acceleration and deceleration are variable. The variability in these impulses is assumed to be directly related to the mean size of the impulses such that the greater the magnitude of the impulses, the greater their variability. Movements of higher speed require larger impulses for both acceleration and deceleration. Such movements would, therefore, be associated with greater variability in the accelerative and decelerative impulses. Impulses vary in terms of both magnitude of the torque profile and its duration. Since the distance travelled in a discrete movement depends on the speed and duration of movement, impulse variability will produce variability in the movement amplitude and thereby influence movement accuracy in relation to a target.

Empirical support for these impulse-variability models has come from work by Schmidt and colleagues studying variability in impulse duration and peak exerted torques. Variability in impulse duration, as measured from torque profiles associated with alternating movements, was directly proportional to mean impulse duration (Schmidt et al., 1979) and therefore supported the model. Variability in peak isometric torques was also found to be directly proportional to mean peak torque up to about 65-70% of maximum torque, after which variability decreases (Schmidt and Sherwood, 1980). Thus for movements requiring peak torques less than 65-70% of maximum these data support the impulse-variability models. The original formulation of the impulse variability model considered only the impulse for acceleration of the limb (Schmidt et al., 1979). This led to a number of problems with the model. In the first instance the model was limited to movements with no active voluntary deceleration of the limb. Secondly, as shown by Meyer et al. (1982), this asymmetric impulse-
variability model did not actually predict a linear speed-accuracy tradeoff as was observed for rapid aiming movements (Schmidt et al., 1979). As a result of these shortcomings Meyer et al. (1982) proposed a symmetric impulse-variability model in which impulses for acceleration and deceleration were exactly equal and opposite. Mathematical proof that such a model could produce linear speed-accuracy tradeoffs was provided (Meyer et al., 1982).

Impulse variability models provide adequate explanations for variability in movement endpoint as a function of movement speed. A major experimental finding for which impulse-variability models cannot account is the effects of external disturbances on movements. External perturbations do not greatly influence attainment of final limb position in simple arm or head movements (Bizzi et al., 1976; Polit and Bizzi, 1979; Cooke, 1979; Schmidt and McGown, 1980; Kelso and Holt, 1980). In impulse variability models movements are assumed to be controlled through preprogrammed torque pulses which explicitly control movement acceleratory and deceleratory phases. External torques will, therefore, strongly influence both movement duration and endpoint. These problems have arisen partially because of incorporation of only inertial properties of the limb in the model and phasic impulses in movement production. As will be discussed in the following section the limb also has elastic properties, necessitating inclusion of a tonic (step) component in motor control schemes for final position control.

B. Mass-spring models for final position control

The mass-spring hypothesis for control of limb postures was
formulated by Feldman (1966a,b) and extended by Bizzi and colleagues (Bizzi et al., 1976; Polit and Bizzi 1979). Crossman and Goodeve (1963) first advanced the idea that joint angle could be controlled by the relative stiffnesses of opposing muscles about joints and that movements could be controlled by setting the stiffnesses of opposing muscles. When the limb is at rest all of the forces/torques acting about the joint (i.e., muscular torques and external loads) sum to zero. In the absence of external forces the joint torques produced by one set of muscles must be exactly balanced by the torques produced by the opposing muscles. Since muscle tension varies with muscle length, the muscles can be viewed as springs which provide opposing forces/torques about joints. The position of the limb at rest will therefore depend on the tension in the opposing springs. The tension in a spring, or the force opposing length change, varies with muscle length. Thus specification of the opposing muscle stiffnesses or equilibrium lengths through neural activity would specify the joint angle. As discussed previously the stiffness and/or equilibrium length of opposing muscles may be specified by central commands. Thus movements may be produced by changes in muscle stiffness, muscle equilibrium length or both. Movement occurs when a new equilibrium point is specified by the active states of opposing muscle about the joint.

Evidence in support of this model of movement control has come from experiments in human subjects and animal models. In experiments in human subjects instructed not to voluntarily correct for perturbations it was shown that rapid establishment of a certain joint angle depended only on the final steady states of the opposing muscles (Feldman, 1966b). There were no corrections for spring loads applied unexpectedly during
movement, indicating that in the absence of voluntary responses to the load no corrections in the final joint angle occurred. Also initial conditions (i.e., joint angle) prior to movement did not influence attainment of the final position. Bizzi and colleagues have shown that deafferented monkeys can make movements to a target even when a pulse (short duration torque) perturbation is applied during the movement (Bizzi et al., 1976; Polit and Bizzi 1979). That is, although the perturbation influences movement time course the intended end position of the movement is still reached in the absence of feedback from the moving limb. Experiments in human subjects have also provided support for such a model (e.g. Schmidt and McGown, 1980; Kelso and Holt, 1980; Kelso, 1977). Thus the motor apparatus exhibits the properties of a mass-spring system, specifically final position is independent of initial conditions and movement dynamics.

Other studies in human subjects have provided evidence against a mass-spring model. Day and Marsden (1982) studied the effects of unexpected changes in viscous load during flexion movements of the interphalangeal joint of the thumb. Small but significant alterations in movement endpoint as a result of these alterations in viscous load were observed when cutaneous afferents of the thumb were anaesthetized. Since the torques produced by viscous properties are equal to zero when movement ends, changes viscous loading should not influence endpoint of movement in the mass-spring model. When the thumb was not anaesthetized, compensatory changes in muscle activity were observed which provided compensation for the unexpected viscous loads. These EMG responses to viscous loads were attributed to the long latency stretch reflex machinery providing servo assistance to the movements. In studies of
perturbation evoked alterations in movements Sanes and Evarts (1983) observed that the magnitude of the induced movement disruptions depended on the size of movements. Small movements were affected more than larger movements in relative terms (i.e., as a percentage of movement amplitude). Both trajectory errors and final position errors (measured 500 msec after the perturbation ended) were greater in smaller amplitude movements. This result cannot be explained by mass-spring models which would predict no effect of these perturbations on final position control. However 500 msec may not be sufficient time for the muscle mechanical properties to establish the final position. While the results of these experiments cast some doubt on the mass-spring hypothesis it should be noted that the errors resulting from unexpected perturbations were very small (10-40). In the absence of visual feedback movement endpoint is, however, only specified within a rather wide range of about 8° for elbow movements (Sakitt et al., 1983). Thus these experiments indicate that sensory feedback does play some role in the fine accuracy of movements. However control over the spring-like properties of muscle is probably also important in determination of movement end position at least within a fairly wide target range.

In mass-spring models movement is considered to be a transition from one position of static equilibrium to another. The time course of the change from one equilibrium position to another will therefore be an important feature of movement control. Movement could, for example, be produced by a step change to a new equilibrium position or by a gradual shift toward a new equilibrium position. The use of a step change in limb stiffness to produce movements has been studied in a linear second order model of the limb (Cooke, 1979; 1980b). Movements produced in this way
were qualitatively similar to movements produced by human subjects. It can be shown that step changes in limb equilibrium position would produce similar movements. Thus step changes in limb properties could be used to initiate movements.

More recently it has been suggested that there is a gradual change in the limb's equilibrium position during movement. Studies by Bizzi et al. (1984) in monkeys and in a model of the limb (Hogan, 1984) have suggested a more gradual change in limb equilibrium position at least in relatively slow movements (about 700 msec duration). In experiments on deafferented monkeys the limb was moved to and held at the intended final position for a flexion movement prior to movement initiation. At the onset of movement-related EMG activity the limb was released and initial movement toward the start position occurred (Bizzi et al., 1984). Thus extensor motion occurred in the presence of predominantly flexor muscle activity associated with flexion movement initiation. This implied that the equilibrium (or virtual - cf. Hogan, 1984) position is gradually changed from start to final position. Studies in a model of the limb indicated that for faster movements the change in virtual position may be much faster and actually overshoot the intended position, however the change is still not a step. Rather, the initial overshoot in the equilibrium position is followed by a return toward movement start position and finally development of the intended final equilibrium position in a quasi-sinusoidal manner (Hogan, 1984). Alternatively, limb properties (stiffness) could be altered and the form of virtual position trajectory could be unchanged (Hogan, 1984). Both of these possibilities for control of the virtual trajectory of fast movements were suggested on the basis of simulations in a simple model of the forearm.
The issue of variability in movements has not been addressed in experiments conducted to test the mass-spring model. This has occurred in spite of the fact that the variability in endposition of movements made by monkeys (deafferented and normal) was very high since targets permitted movement termination within a 100°-150° target zone (Polis and Bizzi, 1979). Studies in human subjects indicated that variable error (variability in movement endpoint) increased when subjects were functionally deafferented (Kelso and Holt, 1980).

V. Effects of practice on movements

There are at least two reasons for studying the effects of practice on motor performance. One reason is to determine how performance of the task changes as a result of practice/learning. Are movements performed more smoothly, with less variability and error? Second, and more important, is the question of how the underlying control mechanisms for the movements change. A common assumption is that control changes from a feedback to a feedforward mode during practice. During learning sensory feedback may be used extensively in movement control. Once the movement is "learned", however, central commands may dominate in controlling the movements (feedforward) and there may be little emphasis on feedback control. An alternative hypothesis is that sensory information may be used more effectively to control practiced movements in order to permit better movement performance. Answers to these questions would provide significant information regarding how movements are controlled.

Practice of a task is generally associated with improvements in some measure of task performance such as accuracy or speed in completing the
task (Schmidt, 1975). Indeed improvement as a consequence of practice may continue even after a large amount of practice. Crossman (1959) showed that factory workers using a small jig to make cigars continued to decrease the time required to make the cigars over 7 years of practice. Similarly Snoddy (1926) showed that subjects continued to improve in performance over 100 days of practice in a task in which they drew figures while seeing only the mirror image of their hand. Thus there is considerable evidence that practice improves task performance and that learning continues throughout even long periods of practice. However the important question is how is performance on the task improved during practice? It is believed that practice, in association with knowledge of results (i.e. how good the performance was) enhances development of motor memory or the "schema" of the task (Schmidt, 1975; Marteniuk, 1976). The schema defines the general characteristics of the desired movement. The difference between the schema and actual movements define an error in movement execution. On the basis of this error corrections to the motor commands for subsequent movements can be made (cf. Pew, 1974).

Most investigations of the effects of practice or learning have been of extremely complex tasks such as those mentioned above. In such complex tasks only gross measures of performance (i.e., duration, accuracy, etc.) could be analyzed. Changes in performance of very simple tasks (e.g. linear positioning) during practice have also been studied (e.g. Adams and Goetz, 1971). In such studies it has been found that practice is much less important than sensory feedback cues in determining accuracy in positioning. In linear positioning tasks such as those studied by Adams and Goetz (1971) the movement kinematics are not measured and, in general, are not constrained in any way (i.e., movements
are very slow). Under such conditions practice effects on accuracy may be minimal. Relatively few studies have been concerned with the effects of practice on characteristics of entire movement trajectories. Exceptions to this include studies in primates in which simple movements about one joint (e.g., Brooks et al., 1983) or pointing movements (e.g., Georgopolous et al., 1981) in which the trajectory of the end segment (i.e., finger) was measured. Brooks et al. (1983) reported that monkeys learn to make simple movements smoothly into a visual target only after months of practice. Initial movements were made very slowly as a series of submovements which produced so-called discontinuities (cf. Brooks et al., 1973) in the velocity profiles of the movements. Brooks and colleagues considered the emergence of smooth, continuous movements as evidence of movement programming. That is programmed (continuous) movements were made as a single movement into the target while non-programmed (discontinuous) movements consisted of a series of submovements towards the target. The sub-movements were considered to be controlled through sensory feedback (visual or proprioceptive) and reflected a lack of behavioural understanding of the task requirements.

Variability of two-dimensional trajectories of arm movements made by monkeys to different targets were studied by Georgopolous et al. (1981). Variability throughout these spatial trajectories was reduced during learning. This indicated that the processes generating these aimed movements became less variable during practice. The authors postulated that decreased trajectory variability resulted from improved motor coordination and better estimation of the appropriate aimed trajectory. Given the appropriate trajectory, shoulder and elbow movements would be consistent from trial-to-trial in order to maintain the desired
trajectory relatively constant (low variability). Practiced movements would therefore be performed as a whole and there would be high correlations between actions of muscle of the involved joints.

In summary it is apparent that motor performance improves during practice in relation to task goals. This is indicated particularly in the studies of complex movements such as drawing (Snoddy, 1926). Study of movement trajectories has shown that practiced movements are smoother, less variable and are performed as a single movement rather than as a series of submovements. This may indicate a shift from feedback control to central control over movements as subjects learn the task.

VI. EMG patterns associated with movement

The most common measure of neural activity associated with movement is muscle electrical activity. The motor unit, consisting of one alpha motor-neuron and the muscle fibers it innervates, is the functional unit of nervous control over muscle. Recording of the electrical activity of movement related muscles therefore provides an indication of the underlying neural control over movement.

Measurement of muscle EMG activity has also been used as an indicator of the force produced by muscles. A number of investigators have shown that there is a strong relation between EMG amplitude and isometric force production (e.g. Lippold, 1952; Inman et al., 1952; Bouisset and Goubel, 1968). The relationship is linear or curvilinear depending on the range of forces studied. The relation between EMG activity and movement kinetics is less well understood. Relations between EMGs of agonist and antagonist muscles to the work performed
during movement have been suggested (Bouisset, 1973). Strong relations between the amplitudes of agonist and antagonist bursts and kinematic parameters (i.e., peak velocity of movement) have also been reported (e.g. Brown and Cooke, 1981; Marsden et al., 1983; Flament et al., 1984). These will be discussed in more detail in the section on phasic EMG patterns.

Movement control has been described as consisting of "hold" and "move" programs (Brooks, 1984). Hold programs are those associated with maintaining a steady posture through maintained activity in opposing muscles. Programs for movement involve changes in the maintained or tonic muscle activity and/or phasic activation of muscle. Changes in tonic muscle activity are typically associated with slow movements (cf. Wacholder and Altenburger, 1926; Stetson and Bouman, 1935; Hallett et al., 1975). Phasic muscle activation is associated with more rapid movements in which the limb is accelerated and decelerated at higher rates.

A. Tonic EMG patterns

A role for tonic muscle activity in movement control was first put forth by Wacholder and Altenburger (1925) and was described further by Stetson and Bouman (1935). Maintenance of fixed postures by cocontraction of opposing muscles about joints and movements under tension in which muscle contraction occurs throughout movements were described by Stetson and Bouman (1935). Slow movements under tension were made with continuous activity in opposing muscle groups and thus were called "moving fixations". The presence of cocontraction and tonic
changes in muscle activity during slow movements have been observed by other investigators (e.g. Wacholder and Altenburger 1925; Tilney and Pike 1925; Lestienne et al., 1981; Sakitt et al., 1983). Tonic muscle activity in the antagonist muscle is often also associated with fast movements (fast movements under tension) (Stetson and Bouman, 1935).

The role of tonic muscle activity has been studied in more detail recently because of the focus on models for final position control. In a recent investigation Lestienne et al. (1981) observed that the ratio of opposing muscle activities varied monotonically with joint angle in a nonlinear fashion. Additionally, Cooke (unpublished observations) observed that the specification of limb position through this mechanism was independent of the level of cocontraction between the opposing muscles. That is with increased activity in opposing muscles the same joint angle was specified by the same ratio of these muscle activities. Thus specification of joint angle through central control over tonic muscle activity was possible. However in normal subjects tonic muscle activity may be maintained through afferent feedback from muscle receptors. In a related study Sakitt et al. (1983) found that the information transmitted at the final position of arm movements by the ratio of opposing muscle EMGs was greater than that transmitted by either of the opposing muscles alone. This indicated that the motor program for final position is probably based on relative innervations of opposing muscles rather than control over single muscle groups.

B. Phasic patterns of EMG activity associated with movement

A triphasic pattern of muscle activity associated with fast limb
movements was first described by Wacholder and Altenberger (1926). This pattern consisted of an initial burst of agonist activity followed by a period of reduced agonist activity which was terminated with a second burst of agonist activity. During the period of agonist inactivity a burst of antagonist muscle activity occurred. This pattern of activity in fast movements has since been confirmed in a number of studies in human subjects and animals (e.g. Angel et al., 1965; Terzuolo and Viviani, 1973; Hallett et al., 1975; Hallett and Marsden, 1979; Ghez, 1979; Lestienne, 1979; Brown and Cooke, 1981; Marsden et al., 1983; Flament et al., 1984). Brown and Cooke (1981) observed that the triphasic pattern was characteristic of a wide range of movement amplitudes and speeds.

The characteristics of this EMG pattern in relation to movement velocity and amplitude have received much experimental study. Early work indicated that the duration of the first agonist burst was invariant and modification of only its amplitude occurred (Hallett et al., 1975; Freund and Budingden, 1978; Ghez, 1979; Hallett and Marsden, 1979; Brown and Cooke, 1981). Thus increases in amplitude or velocity of movement were associated with increased amplitude of the first agonist burst. Similarly in rapid isometric contractions control over only agonist burst amplitude and not duration was observed (Freund and Budingden, 1978; Ghez, 1979). However it has recently been shown that the duration of this burst is modified on the basis of movement amplitude (Wadman et al., 1979; Brown and Cooke, 1984). Thus control over the first agonist burst, which sets the limb in motion, includes control over both its duration and amplitude. Central control over amplitude and duration of the first agonist burst has been indicated by studies in a deafferented patient
(Cooke et al., 1985).

The antagonist burst has received less study than the agonist burst but its amplitude and time of onset are influenced by movement speed and amplitude. Increases in movement speed are associated with earlier onset and larger amplitude of this burst (Lestienne, 1979; Brown and Cooke 1981; Flament et al., 1984; Marsden et al., 1983). Decreased movement amplitude is also associated with earlier onset of this burst. The timing and amplitude of the antagonist burst therefore depend on movement amplitude and velocity.

The role of the second agonist burst in movement control is presently controversial. Brown and Cooke (1981) studied the amplitude and duration of this burst in relation to movement kinematics. They observed that alterations in magnitude of this burst occurred in parallel with alterations in the first agonist burst. They thus concluded that the two bursts went through a common gain control mechanism. These findings indicated that fast movements and movements of larger amplitude were associated with larger second agonist bursts.

The question of how the triphasic pattern of muscle activity is generated has received much study, particularly the importance of peripheral feedback. The initial agonist burst is relatively immune to the effects of peripheral feedback. For example, passive shortening of the agonist prior to movement onset did not influence the magnitude or onset of the first burst (Garland and Angel, 1971). More recently Brown and Cooke (1982) reported that the magnitude, but not the duration, of the first agonist burst was influenced by perturbations prior to movement onset. Thus the duration, but not amplitude, of the agonist burst appears to be centrally programmed prior to movement.
Studies of deafferented human subjects and animals have produced conflicting results regarding central control over the triphasic pattern. Terzuolo et al. (1974) reported that deafferentation abolished phasic muscle activity associated with rapid limb movements in monkeys. However Polit and Bizzi (1979) observed phasic agonist activity in deafferented monkeys making rapid limb movements. Studies in human subjects have all found that the triphasic pattern is preserved in spite of deafferentation produced by disease (Rothwell et al., 1983, Hallet et al., 1975; Cooke et al., 1985) or occlusion of blood vessels (Sanes and Jennings, 1984). Thus generation of the triphasic pattern does not require afferent feedback. Feedback may, however, take part in fine control over the amplitude and timing (onset and duration) of the individual EMG bursts.

C. Effects of practice on EMG patterns

The effects of practice on patterns of muscle EMG activity has received remarkably little study. Person (1958) studied the EMG activity associated with learning a chiseling task. Early movements were associated with cocontraction of agonist and antagonist muscles. After a week of practice however the EMG pattern changed to one of reciprocal activation of opposing muscles. That is alternating bursts of activity in agonist and antagonist muscles were observed. Other studies have also shown a reduction in cocontraction of opposing muscles or a decrease in total amount of muscle activity during learning/practice of complex motor skills (e.g. Kamon and Gormley, 1968). Thus activation of opposing muscles in alternating bursts has been taken as evidence for learned movements which may be centrally generated as opposed to feedback
controlled.

D. Summary of EMG-movement studies

The studies of EMG patterns associated with movement have demonstrated that there are two components of muscle EMG activity associated with movement production. The phasic components (agonist and antagonist bursts) are involved in initiation and braking of movement. However they are not involved in maintaining movement end position. This occurs through tonic activity in both agonist and antagonist muscles and is specified by the ratio of these activities. Thus in both agonist and antagonist muscles a pulse-step form of control exists. The pulses are important in controlling the limb trajectory and the steps in control of limb posture. It is hypothesized that the pulse and step components are separately controlled at the motor program level (Bizzi et al., 1976; Polit and Bizzi, 1979). Within the mass-spring models it is the tonic component which is responsible for final position control, the phasic component in specification of the time course of the (virtual) trajectory of the equilibrium position of the limb (cf. Hogan, 1984). Within the impulse-variability models the phasic agonist and antagonist bursts would produce the accelerative and decelerative impulses for movement.

VII. Summary

The preceding review indicates that much remains to be learned about the mechanisms underlying movement control. Although much research has been directed at determining methods of simplifying the nervous system's task in movement control by emphasizing control over a single
variability it is possible that this approach underestimates the capacities of the CNS in movement control.

In the experiments undertaken as part of this thesis the major underlying hypothesis was that the entire trajectory of limb movements is controlled by the nervous system. Trajectory in this case refers to the phase plane trajectory of single joint limb movements (plot of joint angular velocity versus angular position). The basis for this hypothesis came initially from studies by Cooke (1979, 1980). In these studies Cooke showed that muscular responses elicited by perturbing well-learned movements resulted in return of the limb to the prelearned trajectory of the movements. This suggested that the relationship between limb velocity and position throughout movement may be planned and that deviations from the intended trajectory are corrected in such a way that the limb is returned to this intended path. Control of a single variable such as muscle stiffness could not account for this finding. Thus in the experiments conducted here the effects of practice on variability throughout limb trajectories was studied.

An important experiment was to study the effects of practice under the instruction to increase movement speed while maintaining accuracy. Increases in movement speed are typically associated with reductions in accuracy of movements (Woodworth, 1899; Fitts, 1954). Therefore the above instruction required subjects to alter the speed-accuracy tradeoff during practice. Relationships between variability in movements and in the controlling EMGs were studied in order to determine possible mechanisms underlying control of limb trajectories. Possible roles of afferent feedback in trajectory control were studied through the use of vibration applied to muscle tendons to activate movement-sensitive
receptors in muscles. Study of movements of the same amplitude made at different limb positions permitted examination of the effects of changes required in the "hold" and "move" programs on speed and variability of subsequent movements. Finally a new model for limb movement control was formulated which incorporated characteristics of mass-spring and impulse-variability models. Simulations of movements with this model permitted testing of its predictions regarding movement control.
Methods

I. Subjects

Twenty-five normal, healthy individuals (15 male, 10 female) served as subjects for the different experiments in this thesis. The subjects ranged in age from 21 to 60 years. At the time of experimentation no subjects reported any history of motor dysfunction.

II. Experimental Paradigms

A. General

Subjects were seated comfortably in a chair and grasped a vertical rod attached to a manipulandum handle which pivoted at the subject's elbow. The forearm was supported just in front of the elbow. The manipulandum permitted movement in the horizontal plane over a 120° range. The manipulandum could also be moved by a torque motor which provided an inertial and viscous load for movements made by the subjects. The viscous load was compensated with velocity feedback so that the resultant load was mainly inertial.

Subjects tracked a visual target with the manipulandum. The visual target was displayed as a vertical bar along with manipulandum position on an oscilloscope positioned .5-1 m in front of the subject. Target width (30-50 of handle position) was indicated by the width of the bar. Manipulandum position, derived from a precision potentiometer, was displayed as a narrow vertical line on the oscilloscope. The targets
were not mechanically detectable and were not bounded by stops. Movements were, therefore, self-terminated. The target moved (stepped) between 2 positions at a regular rate, usually 4 seconds between target switching. The amplitudes of target movement were 100 or 300.

B. Instructions to subjects

Subjects were instructed to make smooth, accurate movements with little terminal oscillation and to attempt to increase movement speed with practice. No other instructions as to how to perform the movements were given. Prior to data recording subjects were permitted to move the manipulandum without following the targets in order to accustom them to the manipulandum load and the visual display.

C. Specific Experiments

(i) Effects of practice on movements and related EMGs

Twelve subjects performed 60 flexion and 60 extension movements 100 or 300 in amplitude under the above instructions. A similar set of experiments, involving 10 subjects, was conducted in which a small constant torque load was placed on the manipulandum through the use of a torque motor. The constant torque load resisted elbow extension and assisted elbow flexion. It required constant contraction of the elbow extensor muscles (i.e., triceps brachii) to maintain the limb in target prior to and after movements. The purpose of applying this load was to examine the effects of practice on the premovement silent period in the antagonist triceps prior to flexion movements.
(ii) Effects of prolonged practice on movement variability.

The effects of an extended practice session were studied in 4 subjects who made 300 to 1000 flexion and extension movements. Two subjects performed 100 and 300 movements. The other two performed only one of the movement amplitudes.

(iii) Effects of muscle tendon vibration on movements and EMGs.

The role of afferent feedback from muscle receptors was studied by application of vibration to the triceps muscle tendon during movements. Vibration is known to stimulate muscle spindles and Golgi tendon organs (GTOs) (Burke et al., 1976a,b). Vibration (100 Hz) was applied to the triceps brachii muscle tendon during the acceleratory and deceleratory phases of movements. The vibrator (a small D.C. motor with an eccentric head enclosed in a metallic casing) was attached firmly over the triceps brachii muscle tendon using Velcro straps. The vibrator was triggered from the acceleration signal of the movement so that vibration could be applied during only the acceleratory or deceleratory phases of both flexion and extension movements. Vibration was applied randomly during either the acceleratory or deceleratory phases of about 1/2 of the movements. Thus the subjects did not know whether vibration would be applied or whether it would be applied early or late in any movement. In order to accustom subjects to the manipulandum, the visual display and the vibration, subjects performed movements with only the manipulandum position displayed on the oscilloscope (i.e. no targets displayed) prior to starting the experiment. During these movements vibration was applied randomly during the acceleratory and deceleratory phases.

The first 50 flexion and 50 extension movements made by each subject
were recorded (flexion and extension movements were made alternately). These movements were considered unpracticed movements. Following this, the subject continued to practice for about 100 movements which were not recorded. Vibration was also applied randomly during these movements. A second set of 50 flexion and 50 extension movements, with vibration randomly applied, were then recorded and these were considered practiced movements.

In order to determine whether the applied vibration startled subjects or whether there were any effects of vibration applied to the skin, control experiments were carried out in two subjects. In these experiments the subjects performed 50 flexion and 50 extension movements and vibration was applied randomly during acceleration and deceleration. The vibrator was placed in 3 positions for these experiments: (1) not touching but in close proximity to the subject, (2) less firmly over the triceps tendon than for actual experiments and (3) loosely on the forearm just below the elbow. When the vibrator was placed on the forearm care was taken to ensure the vibrator was not over the brachioradialis muscle.

(iv) Effects of elbow posture on movements and EMGs.

Changes in elbow posture (joint angle) cause changes in the lengths of muscles surrounding the joint and in the moment arms of these muscles (An et al., 1981). Thus the individual muscular forces used to hold the forearm in target prior to and after movement are a function of the elbow posture. Due to the effects of elbow angle on muscle moment arms the joint torques produced by individual muscles are a function of elbow posture. Thus by studying initial movements made following practiced movements at a different elbow posture the adaptations to these altered
Six subjects participated in these experiments. EMG activities of each of the three heads of triceps were studied in order to examine the role of synergist muscles during practice. Each subject performed 70 flexion and 70 extension movements of 100° amplitude at 3 different start positions each for flexions and extensions. These were midrange (elbow angle about 90°), flexed (elbow angle about 120°) and extended (elbow angle about 60°) positions. Thus flexion movements were initiated from elbow angles of about 550, 850 and 1150. Extension movements were initiated from elbow angles of about 650, 950 and 1250. Note that elbow angles are measured from full extension. The order in which movements were made from the different start positions was fixed such that each subject received a different order (i.e. there are 6 possible combinations for order of presentation of the 3 different start positions). Only movements 1-10, 31-40 and 61-70 for flexions and extensions were recorded and analysed.

III. Data recording

The primary data recorded were angular position and velocity of the manipulandum and the EMG activity of the biceps brachii and triceps brachii (lateral head) muscles. In one set of experiments the EMGs of the 3 heads of triceps brachii were recorded in order to examine the control of synergist muscles. Forearm position/elbow angle was recorded from a precision potentiometer attached to the pivot point of the manipulandum. Angular velocity was obtained from the back emf induced in a small linear d.c. torque motor by the handle movement.

EMGs were recorded using surface electrodes .9 cm in diameter placed
about 3 cm apart over the bellies of the biceps and triceps brachii (lateral head) muscles. In experiments in which the EMGs of the 3 heads of triceps were recorded, .4 cm diameter electrodes were used in order to reduce the possibility of crosstalk between the EMGs recorded from different muscles. EMGs were filtered (bandwidth 20 - 1000 Hz.) and full-wave rectified prior to recording. All data were digitized on-line with a sampling rate of 500 Hz. The data were stored on disk or magnetic tape for later analysis.

IV. Data Analysis

A. Movements

Each movement was split into acceleratory and deceleratory periods. A criterion value of 1200/sec² was used to determine the onset and termination of the acceleratory and deceleratory phases of the movements (Fig. 1). In this way the durations of these periods of the movements were equally defined for all movements analysed. The period between the end of acceleration and beginning of deceleration was labelled the constant velocity period. Although velocity is not absolutely constant during this time interval only very small changes in velocity occurred. With this method of analysis terminal corrections and tremor were removed and only the dynamic phases of movement acceleration and deceleration were studied.

Variability of movement phase plane trajectories (plots of velocity versus position during movement) was examined by defining ellipses representing variability with radii equal to 1 S.D. in position and
Splitting of movements into acceleratory, constant velocity and deceleratory periods. Position, velocity and acceleration records for a single movement are shown. Acceleration was determined by digital differentiation of the velocity signal after filtering (20 Hz. cutoff, 0 phase shift) with a differentiation interval of 12 msec. Timing points (vertical dashed lines) were determined using a criterion of 120°/sec² in acceleration.
velocity. As a measure of trajectory variability the area of these ellipses was calculated at 10 msec intervals throughout the movements. Trajectory variability of the acceleratory and deceleratory phases was calculated as the average area of these ellipses during these movement phases. Average area was used instead of total area in order to normalize for different movement durations (i.e., longer duration movements would have more variability ellipses). Single movement records were aligned to movement onset (as determined from acceleration) and to a common start position prior to averaging. Movements were aligned to a common start position because I was interested primarily in movement dynamics. Variability in movement start position was, in any case, very low (S.D. .10 to .30) and there was no evidence that movement start position was used as a cue for the movements. That is the variability of movement amplitude and movement end position were well correlated and almost identical in most movement averages.

The time course of trajectory variability during movement was studied by plotting area of the variability ellipses as a function of time throughout the movements. This was labelled point-to-point variability. From these plots it was possible to determine whether trajectory variability increased throughout movement or whether decreases indicative of movement corrections occurred.

The effects of muscle tendon vibration on the kinematic properties of movements were also studied. This was necessary because in these experiments movements were visually controlled. Thus, in contrast to previous experiments in which movements were proprioceptively guided (Capaday and Cooke, 1981; 1983), the intended final position of the movements was always attained. Movement amplitude was therefore measured
at the end of deceleration (first instance in which deceleration was greater than -1200/sec²). In some movements subjects made small movements back toward the movement start position as part of a movement correction (i.e., for overshoot) or due to terminal oscillation. The amplitude of limb movement back toward the start point after the end of deceleration (return movement) was measured for control (not vibrated) movements and for movements when vibration was applied during deceleration. When vibration was applied during acceleration there were usually undershoots of the intended final position, thus there were no return movements.

B. EMGs

Onset times of phasic agonist and antagonist EMG burst activity and reductions in antagonist EMG activity prior to movement were determined using statistical criteria. EMGs were rectified and low pass filtered (10 Hz. cutoff, 0 phase shift - 4th order Butterworth digital filter). The mean value and variability of premovement agonist and antagonist activities was calculated for each movement. The onset of the agonist and antagonist phasic EMG bursts were determined using a criterion of 2, 3 or 4 S.D.s above the mean of premovement muscle activity. That is the criterion value for burst onset was typically equal to the mean + 2 (or 3 or 4) S.D.s of pre-burst EMG amplitude. The actual criterion chosen for each subject was based on visual inspection of EMG records with the criterion superimposed. For any one subject the criterion used was maintained constant for all movements so that burst onsets were determined equally across all movements. Duration of the bursts were
assumed to be either 80 or 160 msec for calculation of burst amplitudes based on inspection of single and averaged EMG records. The onset of the premovement reduction in antagonist EMG activity was determined using a criterion of 1 S.D. below the mean of the premovement EMG level. This period of decreased EMG activity was considered complete when activity had returned to the mean level. Fig. 2 shows an example of the determination of EMG timing parameters for a single movement using these methods.

Variability in EMG patterns throughout movements was analysed in a way similar to that for movement trajectories. Single movement EMG records were aligned to movement onset, as determined from acceleration records, and averaged. Prior to averaging the rectified EMGs were smoothed by low pass filtering (10 Hz cutoff, 0 phase shift - digital filter) to produce a linear envelope EMG signal. The area of ellipses with radii equal to 1 standard deviation in amplitude of agonist and antagonist muscle activity were calculated at 10 msec intervals from 100 msec prior to movement onset to movement termination. EMG variability was defined as the average area of these ellipses over this time. In the calculation of EMG variability there was thus a multiplication of the standard deviations of the agonist and antagonist EMG amplitudes.

The effects of vibration on movement related muscle EMG activity were analysed by comparing the EMGs associated with control (not vibrated) and vibrated movements during the period of applied vibration. The mean activity of agonist and antagonist muscles during the vibration interval of vibration movements and during the same time interval of the control (not vibrated) movements was calculated. The vibration interval was the time during which the vibrator was on in an "average" vibrated movement
Determination of onset times of phasic EMG activity and of the premovement reduction in antagonist EMG activity. Agonist (biceps) and antagonist (triceps) EMG records for a single movement are shown. The EMGs were rectified and filtered (10 Hz. cutoff, 0 phase shift). Timing point 1 is the onset of the first agonist burst. Timing points 2 and 3 are the onset and termination of the premovement reduction in antagonist EMG activity. Timing point 4 marks the onset of the antagonist burst.
relative to movement onset (i.e., the mean start and end times of applied vibration were calculated and these defined the start and end of the vibration interval). Thus control and vibration movements could be compared in terms of muscle activity during this interval. The ratio of agonist and antagonist muscle activity during the vibration interval provided a measure of the relative change in muscle EMG activity (cf. Capaday and Cooke, 1983).

V. Digital model of the limb

The results of experiments carried out as part of this thesis and of previous investigations prompted me to develop a new model for limb movement control. This model has some of the characteristics of both impulse-variability models and mass-spring models.

The limb was modeled as a linear, time invariant second order system according to the following equation:

\[ T = I \ddot{\theta} + \beta \dot{\theta} + k\theta \]  

(1)

\( T \) - torque, \( I \) - inertia, \( \beta \) - damping coefficient, \( k \) - stiffness, \( \theta \) - joint angle

Linear models of the limb have been widely used in previous work (e.g. Cooke, 1979; 1980; Hogan, 1984). The input torque \( T \) which drives the movement was modeled as separate agonist and antagonist impulses plus a tonic postural agonist torque which maintained final limb position. The net joint torque driving the movement was the difference between agonist and antagonist torques. The postural torque which maintained final limb position was a step torque of the amplitude required to hold the final position based on the limb stiffness.
Variability in impulse duration was modeled as a function of the mean duration of the applied torques (cf. Schmidt et al., 1989). Mean durations of the sinusoidal agonist and antagonist torques were chosen with regard to known EMG burst durations. The duration of the first agonist burst has been shown to be amplitude dependent (Brown and Cooke, 1984) with a mean duration of 70 msec for movement amplitudes below about 30° and 140 msec for movement amplitudes above 30°. The duration of muscular forces/torques produced by EMG bursts would be greater than the EMG burst durations due to muscle's low pass characteristics (cf. Partridge, 1965). Thus mean torque durations of 150 and 250 msec were used. 250 msec was chosen as the mean impulse duration associated with the 140 msec agonist burst because this was approximately the duration in the torque-time curves for rapid isometric contractions performed by human subjects (cf. Freund and Budingden, 1978). The mean impulse duration associated with a 70 msec EMG burst was assumed to be 150 msec. The durations of impulses produced by agonist and antagonist phasic bursts were assumed to be equal for any given movement, but varied randomly for different movements.

The agonist and antagonist impulses were modeled as sinusoids of variable amplitude and duration. The sinusoidal shape was chosen as it closely resembles the shape of isometric torque-time curves observed in previous work in human subjects (eg. Freund and Budingden, 1978) and in cats (Ghez, 1979) producing rapid isometric contractions. The agonist sinusoidal torque ended in a step torque which would maintain final limb position according to the limb stiffness.

Variability in the tonic postural torque was not included in the model. In fast movements the tonic postural torque would have little
effect on movement dynamics because it is much smaller than the phasic agonist and antagonist torques; it is only required to maintain final position. Thus for simulations involving fast movements variability in the postural torque was set at zero for simplicity. In slower movements, variability in this postural torque would influence movement dynamics as well as end position.

The following equations describe the agonist and antagonist torque-time curves.

\[ T_{ag} = T_{ag}^* \sin(\omega t) \quad 0 \leq t \leq D_{ag} \tag{2} \]
\[ T_{ag} = T_{ton} \quad D_{ag}^* \leq t \leq - \]
\[ T_{ant} = T_{ant}^* \sin(\omega t + \phi) \quad \phi \leq t \leq D_{ag} + \phi \tag{4} \]

\[ T_{ag} \quad \text{agonist torque} \]
\[ T_{ag}^* \quad \text{peak agonist torque} \]
\[ T_{ton} \quad \text{tonic agonist torque (k0)} \]
\[ T_{ant} \quad \text{antagonist torque} \]
\[ T_{ant}^* \quad \text{peak antagonist torque} \]
\[ D_{ag} \quad \text{duration of agonist torque (phasic)} \]
\[ D_{ag}^* \quad \text{duration of agonist torque until } T_{ag} \leq k0 \]
\[ \omega = 2\pi/(2 \cdot D_{ag}) \]
\[ \phi \quad \text{delay between agonist and antagonist torque onsets (sec.)} \]

Variability in peak agonist torque amplitude was assumed to be directly related to the mean peak amplitude (cf. Schmidt et al., 1979, Schmidt and Sherwood, 1980). This is apparently true up to only 65% of maximum torque (Schmidt and Sherwood, 1980), however most movements are performed at well below 65% of maximum. Torques greater than about 65% of maximum are associated with less variability, resulting in an inverted U shaped relation between torque variability and peak torque. No attempt was made to model the inverted U shape of the torque variability-peak torque
curve. Thus the model applies only to movements made with peak torques below about 65% of maximum. Practice was assumed to decrease peak agonist torque variability. The following equations describe the agonist impulse and its variability:

\[ \sigma_{\tau^*_{ag}} \propto \tau^*_{ag} \]  
\[ \sigma_{\Delta \tau_{ag}} \propto \Delta \tau_{ag} \]  
\[ \sigma_{\tau^*_{ag}} \propto \frac{1}{\text{practice}} \]  

The amplitude of the antagonist torque and the time of its onset were assumed to be dependent on the agonist torque amplitude. Based on studies of muscle EMG patterns associated with movements of different amplitude and speed (e.g. Lestienne, 1979; Brown and Cooke, 1981; Marsden et al., 1983; Flament et al., 1984) peak antagonist torque was modeled as directly related to the peak agonist torque and its time of onset as inversely related to the peak agonist torque. This is set out in the following equations:

\[ \tau^*_{\Delta t} \propto \tau^*_{ag} \]  
\[ \cdot \propto \frac{1}{\tau^*_{ag}} \]  

The equations describing the movement kinematics as a result of the applied torques are as follows:

\[ \ddot{\theta}(t) = (\tau_{ag}(t) - \tau_{\Delta t}(t) - B\dot{\theta}(t) - k\theta(t))/I \]  
\[ \dot{\theta}(t) = \int \ddot{\theta}(t) dt \]  
\[ \theta(t) = \int \dot{\theta}(t) dt \]
Results

In this chapter findings from studies of practice and its influence on movements and related EMGs are presented first. Relationships between variability in movement trajectories and the EMGs as a function of practice and movement speed serve as the important focus of this section. Results of studies using muscle tendon vibration are presented next. Possible role of afferent feedback from muscle receptors in control of the limb's trajectory and related muscle activity are examined. The chapter concludes with an examination of the features of movements simulated in a model of the limb. The important question in relation to this model was whether it could adequately explain the results of the present work and of previous studies of movement control.

I. Effects of practice on movements

A. Performance of initial movements

In most cases subjects made smooth movements into the targets within the first few movements. Fig. 3 shows position and velocity records for the first 3 flexion and extension movements made by one subject who had never previously participated in experiments in this laboratory. The first movements (Fig. 3A,B) were not made smoothly between the targets. These would be termed discontinuous because there was more than one peak in the velocity profile of the movement (Brooks et al., 1973). However the 2nd and 3rd flexions and 3rd extension movements were made smoothly into the target. All subjects made smooth, continuous movements into the
Unpracticed movements by a subject who had never previously made movements on the laboratory apparatus. Records from the first 3 flexion and extension movements made by subject M.M. are shown. Position and velocity records aligned to movement onset (dashed line) are shown. Movements were 30° in amplitude.
target within the first few movements. Similar results were obtained when movements were made with small constant torque loads resisting elbow extension and for movements made from different start positions. Thus human subjects are capable of performing smooth, continuous movements under a variety of conditions without practice. These data therefore clearly differ from the results of experiments in which monkeys learning a similar motor task performed discontinuous movements over a long period of time prior to learning the task (Brooks 1983). Probably a lack of understanding of the task by the monkeys explains these differences.

B. Performance of initial movements following a change in elbow posture

Experiments were also carried out in which subjects made movements beginning from 3 different elbow angles as detailed in the Methods. Thus unpracticed movements at a new elbow posture could be studied immediately following practiced movements beginning from a different angle. Alterations in elbow angle change the lengths of agonist and antagonist muscles and the moment arms of elbow joint muscles (An et al., 1981). Thus neural control over the movements must be changed to accommodate the new forearm position.

After a change in elbow angle the speed of initial movements was typically less than that of the practiced movements at the previous start position. Mean movement peak velocity of the first 10 movements following a change in start position was decreased by an average of 13.6% (s.e. = 3.6%, n = 24, includes flexions and extensions) when compared to the practiced movements (61-70) at the previous start position. Within
individual subjects mean peak velocity of movements was sometimes maintained almost constant or slightly increased following a change in start position. However these movements were typically more variable than the practiced movements at the previous start position. This will be discussed further in a later section on variability in movements. Thus changes in elbow posture usually resulted in decreased movement speed in the first 10 movements.

C. Changes in movement speed with practice

Most subjects moved slowly at first and increased movement velocity throughout practice while maintaining accuracy. In Fig. 4 the 60 movements made by each subject were divided into sequential groups of 10 movements. Each record is the average of one set of 10 movements. Thus, the top trace is the average of movements 1-10, the next trace of movements 11-20 and so on. For the flexion movements shown in Fig. 4 (left hand traces) each of the first 10 movements landed within the target at the defined movement endpoint (acceleration greater than 120°/sec²). With practice, the velocity of movements increased and accuracy was maintained. This pattern was seen in most subjects. However, a few subjects started out making fast, relatively inaccurate movements (Fig. 4 - extensions). In this example only 7 of the first 10 movements made by the subject landed within the target at the defined movement endpoint. There were also terminal oscillations as indicated by the large s.d.s (vertical bars superimposed on average records) in the position and velocity records near the end of these movements. These initially fast extension movements were followed by slower movements (Fig. 4 - movements
Effects of practice on the time course of movement position and velocity. Shown from top to bottom are averaged position and velocity records taken sequentially throughout practice. Data were filtered (20 Hz. cutoff, 0 phase shift - digital filter) and aligned to a common start time prior to averaging. Data for subject S.B. are shown on the left and for D.F. on the right.
11-20) and the subject thereafter progressively increased speed without decreasing accuracy. Similar strategies were observed for subjects performing movements with a constant torque load opposing extension and for movements made from different start positions.

The effects of practice on movement duration was examined for durations of each of the 3 movement phases - acceleratory phase, constant velocity phase and deceleratory phase. Table 1 contains the mean durations of the movement phases for movements 1-10 and 51-60 for 10° and 30° movements performed with and without loads. Table 2 contains the mean durations for these phases of movements performed from different start positions. Duration of deceleration was consistently longer than the acceleratory phase. The constant velocity phase of the movements was the shortest in duration. With practice (compare movements 1-10 to 51-60 (Table 1) or 61-70 (Table 2) duration of the acceleratory and deceleratory phases were little changed. An exception to this was in the 30° load condition in which practice resulted in decreased duration of both of these movement phases. The duration of the constant velocity phase of the movements was consistently reduced with practice (Tables 1, 2). This indicated that movement deceleration was initiated soon after movement acceleration ended in practiced movements. Since subjects were instructed to increase movement velocity during practice one would expect decreased movement duration in the practiced movements. The reductions in duration observed were relatively small because, as described previously, some subjects performed fast movements within the first 10 movements, subsequently slowed their movements and then increased velocity during practice to about the same level as in the first 10 movements. Also only dynamic phases of the movements were measured, thus
Table 1

Mean durations of acceleratory, constant velocity and deceleratory phases of movement 1-10 and 51-60 for 10° and 30° movements performed under no-load and load conditions.

<table>
<thead>
<tr>
<th></th>
<th>Acceleration duration (msec)</th>
<th>Constant Vel. duration (msec)</th>
<th>Deceleration duration (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 - 10 51 - 60</td>
<td>1 - 10 51 - 60</td>
<td>1 - 10 51 - 60</td>
</tr>
<tr>
<td>Flexions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10°WL_a</td>
<td>132.0 129.3</td>
<td>56.9 32.8</td>
<td>151.7 159.2</td>
</tr>
<tr>
<td>10°Lc</td>
<td>138.1 145.5</td>
<td>82.5 40.0</td>
<td>154.1 162.4</td>
</tr>
<tr>
<td>30°NL</td>
<td>195.4 177.8</td>
<td>30.2 20.6</td>
<td>222.5 219.3</td>
</tr>
<tr>
<td>30°L</td>
<td>179.9 138.2</td>
<td>23.2 5.0</td>
<td>209.4 191.7</td>
</tr>
<tr>
<td>Extensions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10°WL</td>
<td>134.1 131.2</td>
<td>57.4 33.3</td>
<td>147.0 162.7</td>
</tr>
<tr>
<td>10°L</td>
<td>149.8 147.5</td>
<td>70.5 32.2</td>
<td>173.5 179.2</td>
</tr>
<tr>
<td>30°NL</td>
<td>187.6 172.4</td>
<td>43.0 21.8</td>
<td>218.3 223.9</td>
</tr>
<tr>
<td>30°L</td>
<td>185.1 143.0</td>
<td>16.6 5.0</td>
<td>230.4 197.2</td>
</tr>
</tbody>
</table>

- a no-load movements
- b standard deviation
- c movements with constant torque load
<table>
<thead>
<tr>
<th>Movement Type</th>
<th>14.3</th>
<th>17.7</th>
<th>13.2</th>
<th>14.1</th>
<th>18.8</th>
<th>11.7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>169.8</td>
<td></td>
<td>14.7</td>
<td>13.7</td>
<td>11.8</td>
<td>13.8</td>
</tr>
<tr>
<td></td>
<td>17.0</td>
<td>17.7</td>
<td>13.2</td>
<td>14.1</td>
<td>18.8</td>
<td>11.7</td>
</tr>
<tr>
<td></td>
<td>150.6</td>
<td>9.4</td>
<td>40.7</td>
<td>16.5</td>
<td>12.2</td>
<td>36.1</td>
</tr>
<tr>
<td></td>
<td>151.6</td>
<td>9.2</td>
<td>44.9</td>
<td>44.3</td>
<td>16.3</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td>151.6</td>
<td>7.2</td>
<td>7.2</td>
<td>44.9</td>
<td>44.3</td>
<td>16.3</td>
</tr>
<tr>
<td></td>
<td>151.6</td>
<td>10.2</td>
<td>10.2</td>
<td>12.2</td>
<td>36.1</td>
<td>11.8</td>
</tr>
<tr>
<td></td>
<td>151.6</td>
<td>33.1</td>
<td>33.1</td>
<td>36.1</td>
<td>11.8</td>
<td>11.8</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>18.6</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td>14.1</td>
<td>15.2</td>
<td>15.2</td>
<td>15.2</td>
<td>15.2</td>
<td>15.2</td>
</tr>
<tr>
<td></td>
<td>18.6</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>18.6</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td></td>
<td>129.9</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
<td>116.4</td>
</tr>
<tr>
<td>Mean Durations of Acceleration, Constant Velocity, and Deceleration Phases of Movements</td>
<td>1.10 - 61.70</td>
<td>1.10 - 61.70</td>
<td>1.10 - 61.70</td>
<td>1.10 - 61.70</td>
<td>1.10 - 61.70</td>
<td>1.10 - 61.70</td>
</tr>
</tbody>
</table>

Table 2
terminal corrections and oscillations which may produce longer movement
durations in unpracticed movements were not included in this analysis.

II. Effects of practice on variability of movements

A. Variability in duration of movements

The control of movement duration is an important part of some
theories of movement control (cf. Schmidt, 1980). Thus the effects of
practice on variability in duration of the acceleratory and deceleratory
phases are of some interest. However control of movement trajectories is
the focus of this thesis, thus duration variability results are shown in
Appendix A rather than in the body of the thesis. Appendix A contains
plots of the changes in variability of the duration of these phases as a
function of practice. Data from movements performed under the no-load
condition are shown in Fig. 46 and for movements performed from different
start positions in Fig. 47. Duration variability data for movements
performed under the load condition are shown in tabular form for
movements 1-10 and 51-60. The data in Appendix A show that variability
in duration of acceleration was much less than variability in
deceleration duration. Also with practice acceleration duration
variability clearly decreased while deceleration duration variability
remained 2-3 times greater. Reductions in deceleration duration
variability were, however, also observed. These data indicated that
variability in movement duration can probably be attributed largely to
the deceleratory phase of the movement, since acceleration duration
variability is relatively low in both unpracticed and practiced
movements.
B. Variability in movement trajectories

Trajectories of simple movements are described here using the phase plane in which movement velocity is plotted as a function of movement position. Previous research had shown that muscle responses to perturbations during movement resulted in return of the limb to a prelearned trajectory, indicating that the entire movement trajectory may be controlled by the nervous system (Cooke, 1979; 1980). If this is true, variability throughout these trajectories should decrease as a function of practice.

Average movement phase plane trajectories and their variability throughout movements are shown in Fig. 5. Variability is indicated by the size of ellipses with radii equal to 1 S.D. in angular position and velocity of the elbow. Thus ellipse area depends on both velocity and position at a certain point in the trajectory. These ellipses are plotted at 10 msec intervals throughout the movements. Each trajectory is the mean from 10 movements grouped as in previous figures. Trajectory variability did not change in a consistent manner throughout practice.

Depending on the subject's initial strategy of speed versus accuracy variation in the early movements was relatively low (slow accurate movements) or high (fast movements). In Fig. 5 the initial 30° extensions made by subject J.S. were relatively slow whereas the initial 10° flexions were relatively fast. As practice progressed variability in the trajectories increased and decreased as the subjects attempted to increase movement velocity and maintain accuracy. For example, the subject making 30° extensions increased movement velocity in movements
Effects of practice on movement trajectories and their variability. From top to bottom the records are averages of sequential groups of 10 movements. Superimposed on the averaged trajectories are ellipses with radii equal to 1 S.D. in position (horizontal axis) and velocity (vertical axis) to show the variability in these trajectories. On the left are shown data for subject C.B. and on the right for subject J.S. Note the different scaling for 100 and 300 movements.
11-20. At the same time trajectory variability increased over that of the initial movements. Similarly the subject making 100 flexions decreased velocity in movements 11-20 with an accompanying decrease in trajectory variability.

Trajectory variability during the acceleratory and deceleratory phases was calculated as the average area of the variability ellipses during each of these phases for movements averaged in groups of 10. Average ellipse area was calculated rather than total area of all of the ellipses in order to normalize for movement time (i.e., longer duration movements would have more ellipses, and therefore a greater total area). In this way a measure of the mean deviation from the average trajectory was obtained. Fig. 6 shows the trajectory variability of both phases throughout practice. The data in Fig. 6 is for all subjects performing in the no-load condition. Data from subjects performing movements with a constant torque load opposing extension of the forearm are shown in Fig. 7. Trajectory variability of 300 movements was considerably greater than that of 100 movements. Also the deceleratory phase of movement was more variable than the acceleratory phase. It is also evident that there is considerable variation between subjects at any particular level of practice (i.e., number of movements performed). This variability between subjects results from different strategies used to increase velocity and maintain accuracy during practice (e.g., Fig. 5). The varied abilities of subjects to perform these movements in a reproducible manner also contributed to the between-subject variability. As a result trajectory variability was not always lowest at the end of 60 movements of practice.

The effects of practice on variability of 100 movements made from different start positions were also studied. Data from one subject are
Changes in trajectory variability of movements performed under the no-load condition with practice. The plotted points are the mean trajectory variabilities of the acceleratory (circles, solid lines) and deceleratory phases (rectangles, dashed lines) for movements 1-10 through 51-60 for all subjects. The vertical bars represent 1 S.E. of the mean for 6 subjects. 300 movements data are shown in A and B and 100 movements data in C and D. Flexions are the solid symbols, extensions are the open symbols.
Changes in trajectory variability of load movements with practice. Plotted points are the mean trajectory variabilities of the acceleratory (circles, solid lines) and deceleratory phases (rectangles, dashed lines) for movements 1-10 through 51-60 for all subjects. Movements were performed with a constant torque load resisting elbow extension. The vertical bars represent 1 S.E. of the mean for 5 subjects. In A and B are data for 300 movements and in C and D for 100 movements. Flexions are the solid symbols, extensions are the open symbols.
shown in Fig. 8 for movements 1-10 and 61-70 from each of the three start positions. With practice at each start position (flexed, mid-range and extended) movement peak velocity increased and trajectory variability decreased in this subject's movements. Trajectory variability data for all subjects performing movements from different start positions are shown in Fig. 9. Practice was usually associated with decreased variability in the acceleratory and deceleratory phases of movements. However as discussed previously practice of only 60-70 movements was not associated with consistent decreases in trajectory variability throughout practice. Thus trajectory variability was not always lowest at the end of 60-70 movements of practice.

C. Effects of changes in elbow posture on variability in movements

When subjects made movements starting from 3 different elbow angles (flexed, mid-range and extended) it was possible to study unpracticed movements at a new start position immediately following practiced movements at a previous start position. Thus the ability of subjects to adapt to new requirements required in movement performance were studied. Changes in movement start position were associated with decreased peak velocity (as already discussed) and increased variability of the movements. This is very clear in the data from one subject shown in Fig. 8. Each change in elbow posture resulted in decreased mean peak velocity of movements and increased trajectory variability. For all subjects trajectory variability increased by an average of 55.3% (s.e. 23.4%; n=24, flexions and extensions) in the 10 movements following a change in start position. For most subjects decreased speed and increased
Figure 8

Trajectory variability of unpracticed and practiced movements made from flexed, mid-range and extended elbow positions. Movement trajectories with superimposed variability ellipses for movements 1-10 and 61-70 made at each start position. For this subject (D.A.) the order of start positions was extended, flexed, mid-range (shown above trajectories). Thus numbering of the movements is from the first 10 made at the extended elbow position.
Changes in trajectory variabilities of the acceleratory and deceleratory phases of 100 movements made from flexed, mid-range and extended elbow angles during practice. The plotted points are the mean trajectory variability of the acceleratory (circles, solid lines) and deceleratory (squares, dashed lines) phases of 100 movements (6 subjects). The error bars represent 1 S.E. of the mean. Closed symbols are for flexion movements, open symbols are for extension movements.
trajectory variability occurred following changes in the elbow angle for movement start position. However if movement speed decreased greatly a smaller rise or even a decrease in trajectory variability was occurred. Conversely, if average speed of movement was maintained constant or increased following a change in start position, trajectory variability almost always increased. Thus changes in movement start position, for movements of the same amplitude, had a strong effect on movement performance.

D. Effects of prolonged practice on trajectory variability

As described in the previous section trajectory variability tended to decrease during practice, however this decrease was obscured by variations between subjects over practice of 60-70 movements. The effects of an extended practice period (300-1000 movements) was therefore studied in order to more clearly determine the effects of practice on the variability of movement trajectories. Trajectory variability during the deceleratory phase was also shown to be considerably greater than during acceleration for practice of 60-70 movements (Figs. 6, 7, 9). Thus the question of whether extended practice would result in about equal variability during the two phases of movement was also studied.

Trajectory variabilities of the acceleratory and deceleratory phases of movements averaged in groups of 50 are shown in Fig. 10 for 100° and 300° movements. These data are the average for 4 subjects with flexion and extension data combined; two subjects performed both 100° and 300° movements on different days. Trajectory variability of both the acceleratory and deceleratory phases of movements decreased with
Changes in trajectory variability with prolonged practice. The mean trajectory variabilities of the acceleratory (circles) and deceleratory (squares) phases for flexion and extension movements for sequential groups of 50 movements for all subjects are plotted. Data for $10^0$ movements are on the left and for $30^0$ movements on the right. The vertical bars represent 1 S.E. of the mean. Note the different scales for $10^0$ and $30^0$ movements.
practice. However variability of the acceleratory phase always remained less than that of the deceleratory phase. Also the variability present in 300 movements was about 3 times greater than that in 100 movement even after 300 movements of practice.

Shown in Fig. 11 are similar data for 1000 movements of practice for 1 subject performing 100 and 300 movements. The data were collected over a 2 day period at each movement amplitude as indicated by the vertical dashed lines. Trajectory variability decreased with practice but variability of the deceleratory phase remained greater than that of the acceleratory phase throughout. Also the variability associated with 300 movements remained greater than that associated with 100 movements by a factor of about 3 as observed in the group data.

III. Effects of movement speed on trajectory variability

In the studies of the effects of practice on variability in movements subjects were instructed to attempt to increase movement speed, while maintaining accuracy during practice. Thus the effects of practice and movement speed on trajectory variability were not independently examined. Previous investigations (e.g. Woodworth, 1899; Fitts, 1954) showed that increases in movement speed produced greater variability in movement endpoint or reduced accuracy. However the effects of movement speed on trajectory variability has not been studied previously. The dependence of trajectory variability on movement speed was studied in an experiment in which 4 subjects performed 300 movements of 4 different durations (about 200, 400, 600, 800 msec) with equal amounts of practice at each duration (40 flexions and extensions). Trajectory variability
Changes in trajectory variability with practice up to 1000 movements. Each point shows the trajectory variability the acceleratory (circles, solid lines) and deceleratory (rectangles, dashed lines) phases of groups of 100 consecutive movements throughout 1000 movements of practice for 1 subject (W.D.). Data for 100 movements are shown in A (flexions) and B (extensions) and for 300 movements in C (flexions) and D (extensions). Data were collected over a 2-day period as indicated by the dashed line on each graph which shows the break between days. Note the different scales for 100 and 300 movements.
increased with movement peak velocity under equal practice conditions for both flexion and extensions (Fig. 12). The relationship between trajectory variability and mean peak velocity was linear over this range of movement velocities ($r = .99$ for flexions and extensions). Thus these results confirm that increases in movement speed produce increases in variability of movement.

IV. Point-to-point trajectory variability

A. Effects of practice

Analysis of variability throughout the duration of the movements was carried out in order to determine whether variability increased throughout or decreases in variability were evident. Point-to-point trajectory variability was defined as the plot of the area of variability ellipses (radii equal to 1 S.D. in position and velocity) as a function of time throughout the averaged movements. Examples of these are shown in Figs. 13 and 14 for 100° and 300° movements respectively. On the left side of these figures are shown average movement trajectories with superimposed variability ellipses for movements 1-10 (top), 31-40 (middle) and 51-60 (bottom). On the right side are shown the associated point-to-point trajectory variability curves. The dashed lines indicate end of acceleration and start of deceleration (if only one line then the 2 occur almost simultaneously). Variability rose exponentially during the acceleratory phase of all these movements. However near the end of acceleration, particularly in more practiced movements (31-40 and 51-60), the slopes of the curves become negative for a period of time. After
The relationship between trajectory variability and peak velocity for equally practiced movements. Each point is the mean from 4 subjects. The lines of best fit based on least squares linear regression are shown for flexion and extension movements. Correlations are 0.99 for both flexions and extensions.
Figure 13

Point-to-point trajectory variability of 10 degrees movements. On the left are movement trajectories with superimposed variability ellipses from averages of 10 movements as indicated. On the right are the associated point-to-point variability curves which are plots of ellipse area versus time throughout averaged movements. The vertical dashed lines indicate the end of acceleration and start of deceleration in the averaged movements. Data are for 10 degrees extension movements performed by subject K.P.
Figure 14

Point-to-point trajectory variability plots for 30° movements. Average movement trajectories with superimposed variability ellipses are shown on the left and the associated point-to-point variability records on the right. Data are for subject S.B.
this point variability either began to rise again or continued to decrease. This reduction in slope of the curve near the peak velocity point was largely due to decreased velocity variability at this point. This is evident in the data presented in Fig. 3 in which position and velocity variability are shown separately and in Figs. 13 and 14 by examining the vertical axes of the variability ellipses. The cause of the reduced velocity variability at this point is shown in Fig. 15 which contains superimposed velocity-time records of single movements. In fast movements (higher peak velocity) deceleration is initiated earlier. This produces a decreased difference between the velocity-time curves shortly after the time of peak velocity and, thus, reduced variability in velocity at this point in time. Reduction in variability toward the end of movement occurred due to decreases in both position and velocity variability (Figs. 13 and 14).

Point-to-point variability curves were studied quantitatively by calculating the mean rate of change of variability during acceleration and deceleration (i.e., mean slope of the point-to-point variability curves during these phases). Table 3 contains the average rates of change of variability during acceleration and deceleration for movements 1-10 and 51-60. These data are for all subjects performing in the load and no-load conditions. Variability rises much faster during acceleration in 30° movements than in 10° movements. During deceleration variability rises at a much slower rate, or actually decreases. With practice variability rises at slower rates during acceleration in the case of 30° but not 10° movements. The mean rate of change of variability during deceleration was not changed in any consistent manner as a result of practice.
Table 3

Mean rate of change of variability associated with initial movements (1-10) and practiced movements (51-60).

<table>
<thead>
<tr>
<th></th>
<th>Acceleration</th>
<th></th>
<th>Deceleration</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 - 10</td>
<td>51 - 60</td>
<td>1 - 10</td>
<td>51 - 60</td>
</tr>
<tr>
<td>10°NL</td>
<td>1.06</td>
<td>1.36</td>
<td>2.36</td>
<td>0.66</td>
</tr>
<tr>
<td></td>
<td>(.17)</td>
<td>(.13)</td>
<td>(.87)</td>
<td>(.37)</td>
</tr>
<tr>
<td>10°L</td>
<td>1.98</td>
<td>2.50</td>
<td>1.6</td>
<td>0.74</td>
</tr>
<tr>
<td></td>
<td>(.9)</td>
<td>(1.2)</td>
<td>(.53)</td>
<td>(.48)</td>
</tr>
<tr>
<td>30°NL</td>
<td>8.47</td>
<td>5.42</td>
<td>-1.17</td>
<td>-1.55</td>
</tr>
<tr>
<td></td>
<td>(1.7)</td>
<td>(.94)</td>
<td>(1.74)</td>
<td>(.73)</td>
</tr>
<tr>
<td>30°L</td>
<td>11.4</td>
<td>10.0</td>
<td>-1.42</td>
<td>1.06</td>
</tr>
<tr>
<td></td>
<td>(3.3)</td>
<td>(2.7)</td>
<td>(2.8)</td>
<td>(.51)</td>
</tr>
<tr>
<td>10°NL</td>
<td>1.45</td>
<td>1.29</td>
<td>1.76</td>
<td>1.24</td>
</tr>
<tr>
<td></td>
<td>(.49)</td>
<td>(.38)</td>
<td>(.79)</td>
<td>(.29)</td>
</tr>
<tr>
<td>10°L</td>
<td>2.52</td>
<td>2.39</td>
<td>0.57</td>
<td>1.60</td>
</tr>
<tr>
<td></td>
<td>(1.1)</td>
<td>(.82)</td>
<td>(.45)</td>
<td>(.50)</td>
</tr>
<tr>
<td>30°NL</td>
<td>12.28</td>
<td>6.7</td>
<td>-1.61</td>
<td>-0.55</td>
</tr>
<tr>
<td></td>
<td>(3.95)</td>
<td>(2.36)</td>
<td>(1.1)</td>
<td>(.93)</td>
</tr>
<tr>
<td>30°L</td>
<td>16.1</td>
<td>10.3</td>
<td>-5.1</td>
<td>-0.73</td>
</tr>
<tr>
<td></td>
<td>(6.9)</td>
<td>(2.3)</td>
<td>(4.2)</td>
<td>(1.8)</td>
</tr>
</tbody>
</table>

\*a movements performed under no-load condition
\*b standard error of mean for 6 subjects (NL condition)
\*c movements performed under load condition
\*d standard error of mean for 5 subjects (L condition)
Figure 15

Velocity records from fast and slow movements. Single movement velocity records from within movements 1-10 (A) and 51-60 (B) of practice are superimposed. The records were aligned to movement onset. Data are for 300 movements performed by subject S.B.
In order to determine the relation between the mean rate of change of variability in each of these movement phases linear correlations were carried out for movements 1-10 through 51-60 for each subject. These correlations are shown in Table 4 for data from no-load and load movements conditions. They were usually negative, indicating that the greater the rate of rise in variability during acceleration the smaller the rate of change of variability during deceleration. This indicates that movements which rise in variability quickly during acceleration either rise in variability at a very low rate during deceleration or decrease in variability during deceleration. Conversely, movements which increase in variability at a very low rate during acceleration continue to rise in variability during deceleration. However the correlations were much higher for 30° than for 10° movements (Table 4). Correlations carried out for prolonged practiced (up to 300 movements) produced similar results (Table 5). Thus, for large amplitude movements subjects were consistently able to decrease the rate of change of variability during deceleration if the rate was high during acceleration.

B. Effects of movement speed

The effects of movement speed, independent of practice, on the rate of change of trajectory variability during acceleration and deceleration were also investigated. These data are shown in Fig. 16 for flexion and extension movements. Increases in movement speed produced increased rate of rise of variability during acceleration. The rate of change of variability during deceleration was inversely related to movement speed, especially for flexion movements. The fastest extension movements were
Table 4
Correlations between mean rates of change of variability of the acceleratory and deceleratory phases for movements 1-10 to 51-60.

<table>
<thead>
<tr>
<th>Flexions</th>
<th>Extensions</th>
<th>Correlations</th>
<th>Flexions</th>
<th>Extensions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100°NLa</td>
<td></td>
<td>300°NLc</td>
<td></td>
</tr>
<tr>
<td>Subj.</td>
<td></td>
<td></td>
<td>Subj.</td>
<td></td>
</tr>
<tr>
<td>K.P.</td>
<td>-.9b</td>
<td>-.06</td>
<td>P.G.</td>
<td>-.84b</td>
</tr>
<tr>
<td>B.B.</td>
<td>-.22</td>
<td>-.73b</td>
<td>C.D.</td>
<td>-.93b</td>
</tr>
<tr>
<td>J.S.</td>
<td>.38</td>
<td>-.02</td>
<td>S.B.</td>
<td>-.95b</td>
</tr>
<tr>
<td>M.H.</td>
<td>-.70</td>
<td>-.13</td>
<td>D.F.</td>
<td>-.98b</td>
</tr>
<tr>
<td>C.B.</td>
<td>.13</td>
<td>-.17</td>
<td>J.H.</td>
<td>.49</td>
</tr>
<tr>
<td>M.R.</td>
<td>-.76b</td>
<td>.31</td>
<td>M.M.</td>
<td>-.97b</td>
</tr>
<tr>
<td></td>
<td>100°Lc</td>
<td></td>
<td></td>
<td>300°Lc</td>
</tr>
<tr>
<td>S.T.</td>
<td>-.02</td>
<td>-.48</td>
<td>S.F.</td>
<td>-.19</td>
</tr>
<tr>
<td>J.J.</td>
<td>-.57</td>
<td>-.15</td>
<td>A.N.</td>
<td>-.95b</td>
</tr>
<tr>
<td>P.G.</td>
<td>-.88b</td>
<td>-.84b</td>
<td>B.B.</td>
<td>-.63</td>
</tr>
<tr>
<td>D.A.</td>
<td>-.89b</td>
<td>-.61</td>
<td>D.F.</td>
<td>-.90b</td>
</tr>
<tr>
<td>P.D.</td>
<td>-.68</td>
<td>.28</td>
<td>S.B.</td>
<td>-.91b</td>
</tr>
</tbody>
</table>

a movements performed under no-load condition
b correlation value differs significantly from zero (p < .05, df = 4)
c movements performed under load condition
Table 5

Correlations between mean rates of change of variability of the acceleratory and deceleratory phases for prolonged practice.

<table>
<thead>
<tr>
<th>Subj.</th>
<th>10° Flexions</th>
<th>10° Extensions</th>
<th>30° Flexions</th>
<th>30° Extensions</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.A.</td>
<td>-.99&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.09</td>
<td>D.A.</td>
<td>-.99&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>W.D.</td>
<td>-.22</td>
<td>-.08</td>
<td>W.D.</td>
<td>-.97&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>R.B.</td>
<td>-.72&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.87&lt;sup&gt;a&lt;/sup&gt;</td>
<td>C.T.</td>
<td>-.51</td>
</tr>
</tbody>
</table>

<sup>a</sup> correlation differs significantly from zero (p<.05)
Effects of movement speed on mean rate of change of variability during acceleration and deceleration. Data for the deceleratory phase are in the top graph and for the acceleratory phase in the bottom graph. Each point is the mean from 4 subjects. Correlations are .99 for each line in the bottom graph.
accompanied by continuing increases in variability during deceleration in two of the 4 subjects. Thus an inverse relation between rate of change of variability during acceleration and deceleration was not observed.

V. Muscle EMG patterns

In previous sections the effects of practice on features of movement phase plane trajectories was examined. In order to determine relationships between neural activity and movements the EMG patterns associated with movements were studied. EMG activities of movement related muscles associated with individual movements and the variability in these EMG patterns was studied.

A. Muscle activity patterns in unpracticed movements

In previous studies of more complex movements a common feature of movement-related EMG patterns has been continuous activity in all involved muscles during initial movements. After some practice subjects use phasic patterns of muscle activity in which EMG bursts in one muscle occur during relative inactivity in opposing antagonist muscles (Person, 1956). Thus a pattern of reciprocal activation of opposing muscles has been associated with practiced or programmed movements.

Movement velocity profiles and EMGs from the first 2 flexion (A,C) and extension (B,D) movements performed by one subject are shown in Fig. 17. This subject had never previously performed movements on this apparatus. These "unlearned" movements were made using phasic bursts of activity in both agonist and antagonist muscles. In both flexions and
Figure 17

EMG patterns of unpracticed movements. Shown are the velocity, agonist and antagonist EMG records for the first two flexion (A and C) and extension (B and D) movements made by one subject (M.M.) who had never previously participated in experiments in this laboratory. Note the phasic burst activity in both agonist and antagonist muscles in all movements. Also in the first flexion note the premovement reduction in antagonist EMG activity. The movements were 30° in amplitude.
extensions the muscles were reciprocally activated; one muscle was relatively inactive as the other contracted. Note also that premovement antagonist EMG activity was reduced prior to movement in these initial movements. Similar phasic EMG patterns were observed in unpracticed movements performed with constant torque loads opposing elbow extension and for unpracticed movements made from different elbow angles. Thus, in a simple task such as this human subjects made movements with reciprocal phasic muscle activity even without the benefit of practice.

In these unpracticed movements subjects could use different patterns of muscle activity. In Figure 18 are shown 3 of the first 10 movements made by another subject performing 100° flexions. The first movement (A) was made with a long duration (160-180 msec) first agonist burst followed by a burst in the antagonist muscle. The fourth movement (B) was made using a short duration (80-90 msec) agonist burst. There was also a decrease in antagonist activity before movement onset. The 7th movement (C) was performed with no obvious burst in the agonist biceps. In this movement there was a long duration reduction in antagonist muscle activity which began before the movement. Agonist activity was maintained more or less constant throughout the movement. Thus movements could be made using either phasic or tonic changes in muscle activity.

B. Effects of practice on EMG patterns

In Fig. 19 are shown records of average EMGs from 100 extension movements performed by one subject during practice. EMGs were averaged in sequential groups of 10 movements. For this subject mean movement peak velocity increased during practice from 420/sec. (movements 1-10) to
Different EMG patterns from unpracticed movements. The velocity, biceps and triceps EMG records for the first (A), fourth (B) and seventh (C) 10° flexion movements made by subject K.P. Note the different durations of the initial biceps burst in A and B and lack of a biceps burst in C. The vertical dashed line indicates movement onset. The calibration bar represents 50° sec in velocity.
Effects of practice on muscle activity patterns. From top to bottom are shown averaged records from 10 movements of agonist (left) and antagonist EMG activities during practice of 10° extension movements. The dashed lines indicate movement onset. The rectified EMG records were filtered (10 Hz. cutoff, 0 phase shift) and aligned to movement onset prior to averaging. Data are for movements performed by subject K.P.
600/sec (movements 51-60). As peak velocity increased so too did the amplitude of both the agonist and the antagonist bursts. In addition antagonist burst activity began earlier in the faster, practiced movement. Mean changes in onset time of the antagonist burst (relative to movement onset) and of percentage changes in agonist and antagonist burst amplitude from movements 1-10 to 51-60 are shown in Table 6 for movements performed under the load and no-load conditions. These data confirm that amplitudes of the agonist and antagonist burst increased during practice and that the onset of antagonist activity was earlier in practiced movements. Since movement speed increased during practice these results were anticipated on the basis of previous investigations of phasic EMG activity in relation to movement velocity (eg. Brown and Cooke, 1981; Marsden et al., 1983; Flament et al., 1984).

C. Effects of practice on synergist muscle activity

Muscles of a synergist group are generally thought to be activated together such that each would contribute in similar proportions to the forces need for movement. Indeed this is one way in which the many degrees of freedom in motor control may be reduced (cf. Bernstein, 1967). That is it would be simpler to activate synergist muscles with a single neural command as opposed to control over each muscle in the synergy. It is possible that control over synergies with a single command may be developed during practice in order to simplify control of well-learned movements. This hypothesis was studied in the triceps brachii muscle group during practice of 100 movements at 3 different elbow postures.

The EMG activities of synergist muscles was studied by recording
<table>
<thead>
<tr>
<th>Antagonist onset (msec)</th>
<th>Agonist amplitude (% change)</th>
<th>Antagonist amplitude (% change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - 10 51 - 60</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Flexions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$10^\circ\text{NL}^a$</td>
<td>38.8</td>
<td>18.2</td>
</tr>
<tr>
<td>$10^\circ\text{L}^b$</td>
<td>114.6</td>
<td>69.5</td>
</tr>
<tr>
<td>$30^\circ\text{NL}$</td>
<td>75.7</td>
<td>37.5</td>
</tr>
<tr>
<td>$30^\circ\text{L}$</td>
<td>55.7</td>
<td>14.1</td>
</tr>
<tr>
<td><strong>Extensions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$10^\circ\text{NL}$</td>
<td>69.9</td>
<td>40.3</td>
</tr>
<tr>
<td>$10^\circ\text{L}$</td>
<td>63.5</td>
<td>30.3</td>
</tr>
<tr>
<td>$30^\circ\text{NL}$</td>
<td>67.6</td>
<td>45.6</td>
</tr>
<tr>
<td>$30^\circ\text{L}$</td>
<td>39.4</td>
<td>3.2</td>
</tr>
</tbody>
</table>

*a* no-load movements  

*b* movements with constant torque load
from each of the three heads of triceps (lateral, long and medial heads) during elbow flexion and extension movements. Thus use of the triceps as both movement agonists and antagonists was examined. Tables 7 and 8 contain data showing changes in antagonist burst onset times and percentage changes in agonist and antagonist burst amplitudes during practice. Data for flexions is in Table 7 and for extensions in Table 8. With practice similar, but not identical, percentage increases in agonist and antagonist burst amplitudes for each of the 3 heads of triceps occurred. When the triceps acted as antagonists (flexions) earlier onset times for the antagonist bursts in all 3 heads of triceps were observed in practiced movements as compared to the first 10 movements. The onset times were, however, not identical for all 3 muscles. These results indicate that the muscles were indeed acting as synergists in that similar changes in burst amplitude and onset times occurred during practice.

The use of averaged data obscures possible variations in the EMG activities of these synergists which may occur in single trials. Figs. 20 and 21 show EMG activities of the 3 heads of triceps associated with single movements. These data are from one subject performing practiced movements (61-70) in a flexed elbow position. Fig. 20 shows data from extension movements (triceps as agonists) and Fig. 21 shows data from flexions movements (triceps as antagonists). Independent control over the activities of the 3 heads of triceps is indicated in these figures. For example in Fig. 20 A, B, D note that the lateral head of triceps had a much larger 2nd agonist burst, relative to the first agonist burst, than the medial and long heads. Comparing A and B in Fig. 20 note that in B the first agonist burst of the lateral head decreased but of the long
Table 7

<table>
<thead>
<tr>
<th>Ext</th>
<th>62.6</th>
<th>68.5</th>
<th>94.4</th>
<th>110.6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid</td>
<td>24.9</td>
<td>19.2</td>
<td>8.7</td>
<td>3.7</td>
</tr>
<tr>
<td>Fix</td>
<td>48.5</td>
<td>168.4</td>
<td>24.8</td>
<td>15.8</td>
</tr>
</tbody>
</table>

Biceps Lateral (msec) Mean onset times of antagonists bursts and %a mean changes in agonists and antagonist burst amplitudes from movements 1-10 to 61-70 flexion movements from flexed, mid-range and extended elbow angles.
<table>
<thead>
<tr>
<th>Burst Onset Relative to Movement Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>1.4 8.6 7.9 14.9 30.2</td>
</tr>
<tr>
<td>7.2 7.3 28.7 5 23.6 151.9 121.7</td>
</tr>
<tr>
<td>0.4 7 8.8 6.4 26.8 18.6 4.1</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>1-10 Biceps</td>
</tr>
<tr>
<td>Long Median</td>
</tr>
<tr>
<td>Burst Amplitude (in mmHg)</td>
</tr>
</tbody>
</table>

and extended elbow positions.

Antagonists from movements 1-10 to 61-70 for extension movements from flexed, extended range.

Mean onset times of antagonists and percentage changes in burst amplitudes of antagonists.

Table 8
Figure 20

Agonist synergist muscle activity associated with practiced movements. Movement velocity and EMGs of the lateral, long and medial heads of triceps for 4 movements made by subject S.T. are shown. These are 100 practiced movements (from within movements 61-70) made from a flexed start position. The rectified EMGs were filtered (10 Hz. cutoff, 0 phase shift).
Antagonist synergist muscle activity associated with practiced movements. The plotted records are velocity and the lateral, long and medial heads of triceps are from 100 movements made from a flexed start position by subject S.T. The rectified EMGs were filtered (10 Hz. cutoff, 0 phase shift).
head increased. Thus the decrease in lateral head activity was compensated by increased long head activity so that peak velocity of movement was not reduced. This was also observed on comparison of C and D (i.e., reduced lateral head activity was compensated by increased long head activity). In this case there was also reduced medial head activity. Similar compensations among the 3 heads of triceps can be observed when the muscles act as antagonists (Fig. 21). Also note that the pattern of activity in the 3 heads was not necessarily identical (i.e., medial head in B) and that onset times of the antagonist bursts are not identical. These data indicated that the 3 heads of triceps were not controlled together as a synergist group. Rather there appeared to be independent control over the activities of each of the 3 synergist muscles.

In order to examine the control of synergist muscle activity quantitatively, correlations between phasic burst amplitudes and onsets (antagonists) were carried out across single trials under unpracticed (movements 1-10) and practiced (movements 61-70) conditions. In this way it would be possible to determine whether correlations improved during practice, indicating a stronger link between the activities of these synergist muscles. Also, if these muscles were being controlled by a single command, one theory for control over synergist muscles (Bernstein, 1967), one would expect high correlations between the phasic burst activities of the 3 synergist muscles. Mean correlations are shown in Table 9 for flexion movements (triceps as antagonists) and in Table 10 for extension movements (triceps as agonists). For the data in Table 10, the amplitude of the first 80 msec of the first agonist burst was used for correlation purposes since this was the usual duration of the first
Table 9

Mean correlations among the EMG activities of lateral, long and medial heads of triceps for flexion movements 1-10 and 61-70 from flexed, mid-range and extended elbow angles.

<table>
<thead>
<tr>
<th></th>
<th>Burst Onset&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Burst amplitude</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lateral - Long 1-10 61-70</td>
<td>Lateral - Long 1-10 61-70</td>
<td>Lateral - Long 1-10 61-70</td>
</tr>
<tr>
<td></td>
<td>Lateral - Medial 1-10 61-70</td>
<td>Lateral - Medial 1-10 61-70</td>
<td>Lateral - Medial 1-10 61-70</td>
</tr>
<tr>
<td></td>
<td>Long - Medial 1-10 61-70</td>
<td>Long - Medial 1-10 61-70</td>
<td>Long - Medial 1-10 61-70</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flx</td>
<td>.55 b</td>
<td>.13 b</td>
<td>.09</td>
</tr>
<tr>
<td></td>
<td>.34</td>
<td>.84</td>
<td>.36</td>
</tr>
<tr>
<td></td>
<td>.73</td>
<td>.47</td>
<td>.72</td>
</tr>
<tr>
<td></td>
<td>.59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td>.31 .41</td>
<td>.30 .09 -.20</td>
<td>.52</td>
</tr>
<tr>
<td></td>
<td>.71 .51 b</td>
<td>.31 b</td>
<td>.31</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ext</td>
<td>.54 .25</td>
<td>.52 .48 .53</td>
<td>.38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>b</td>
<td>.39 .25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.37 .55 .45</td>
</tr>
</tbody>
</table>

<sup>a</sup> burst onset time relative to movement onset

<sup>b</sup> Chi² > .05 for Fisher transformations, mean correlation could not be computed
Table 10

<table>
<thead>
<tr>
<th></th>
<th>0°</th>
<th>4°</th>
<th>14°</th>
<th>28°</th>
<th>38°</th>
<th>49°</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ext</td>
<td>1.45</td>
<td>0.72</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Mid</td>
<td>0.49</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
</tr>
<tr>
<td>Flex</td>
<td>0.42</td>
<td>0.30</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Ch² for Fisher's transformations: 0.05 mean correlation value could not be calculated.
agonist burst in these movements. Also this portion of the burst is generally considered to be preprogrammed (cf. Wadman et al. 1979). The correlations were surprisingly low in most cases (mean r ranged from .1 to .6) and increased correlations were not observed with practice. This suggests that the phasic activities of these synergist muscles are controlled independently, rather than together as a synergist group. Also practice did not improve the linkage between these synergists.

E. Changes in duration of the premovement antagonist silence with practice.

In some experiments, tonic muscle activity prior to movement was accentuated by applying a low-level constant torque load (load condition) which, if unopposed, would flex the limb. Thus tonic triceps muscle activity was required to hold the limb in the target prior to movement. Under these conditions (e.g. Fig. 22) or if there was antagonist EMG activity prior to movement due to cocontraction (e.g. Figs. 17, 18) antagonist muscle activity decreased prior to movement onset. The duration of this reduced activity ranged from 30 to 300 msec. in the movements performed in these experiments. With practice the duration of this period of reduced activity usually decreased (Figs. 17, 22—compare for movements 1-10 and 51-60). Overall the average change in duration from movements 1-10 to 51-60 was -22.3 msec. (s.e. 5.4 msec). In percentage terms this represented an average decrease of 13.6% (s.e. 5.8%). Thus the period of reduced activity was decreased for faster practiced movements. This was primarily due to earlier onset of the antagonist burst in faster, practiced movements as shown earlier (Tables
Changes in variability of EMGs and movements with practice in which movement velocity increased. Shown are averaged (from 10 movements) movement trajectories (left) and associated EMGs (right) and their variabilities for 30° flexion movements with a constant torque load assisting the movements (subject S.F.). The vertical bars superimposed on the averaged EMG records are equal to 2 S.D. in length. The rectified EMGs were filtered (10 Hz. cutoff, 0 phase shift) and aligned to movement onset (time 0) prior to averaging.
The magnitude of the muscle activity during this period was usually unchanged during practice.

VI. Variability in muscle EMG activity

A. Effects of practice

Changes in variability of agonist-antagonist EMGs with practice was studied in relation to variability in movement trajectories. In Fig. 22 the ellipses superimposed on the average movement trajectories represent the variability of velocity and position throughout the trajectory. With practice the variability, as indicated by ellipse area, decreased (compare movements 1-10 with movements 31-40 and 51-60). Also shown in Fig. 22 are average agonist and antagonist EMGs from these same movements with S.D. bars superimposed at 20 msec intervals. Although variability in the movements decreased, agonist (biceps) EMG activity actually became more variable. Antagonist (triceps) EMG activity appeared somewhat less variable in movements 51-60 than in movements 1-10. This pattern of increased EMG variability during practice was observed in most subjects.

In 5 of the 20 subjects performing movements under the no-load and load conditions EMG variability did decrease during practice. In contrast to other subjects these subjects made movements of about the same average peak velocity at the end as at the start of practice. They made relatively fast, variable movements initially followed by slower movements and then increased movement speed as practice progressed. Shown in Fig. 23 are average movement trajectories and EMGs for movements 1-10 and 51-60 for one such subject performing 300 extension movements. Decreases in trajectory variability and in variability of the EMGs are
Changes in EMG and trajectory variability with practice in which there was no change in mean peak velocity. Averaged movement trajectories (left) and associated EMGs (right) are shown for 1 subject (D.F.) performing 30° extension movements. In A data for movements 1-10 are shown, in B are data for movements 51-60. Movement start is at time 0. The single movement records were aligned to movement onset prior to averaging. The rectified EMGs were filtered (10 Hz. cutoff, O phase shift) prior to averaging.
evident in movements 51-60 in comparison to movements 1-10. Mean peak velocity, however, was almost the same at the start and end of practice. Thus with practice EMG variability decreased only under conditions of maintained movement velocity.

The effects of practice on EMG variability therefore appeared to depend on changes in both movement speed and trajectory variability during practice. This was confirmed in multiple and single linear regressions of %change in EMG variability on %changes in peak velocity and trajectory variability during practice (Table 11). These results indicated that changes in EMG variability during practice depended on changes in movement speed or on changes in both movement speed and trajectory variability (Table 11). The regression results varied between conditions because of different strategies used by subjects during practice. That is changes in EMG variability depended mainly on peak velocity if most subjects increased movement velocity during practice in that condition (e.g. Table 11 - no-load flexions). However, for conditions in which some subjects maintained peak velocity almost constant during practice, changes in EMG variability depended on both speed and trajectory variability changes (e.g. Table 11 - load extensions).

In studies of 100 movements made from different elbow postures the effects of practice on EMG variability were also observed to depend on movement speed. In Fig. 24 are shown average EMGs of the biceps and each of the 3 heads of the triceps muscles with superimposed s.d. bars. The EMG data in Fig. 24 are for movements 1-10 and 61-70 at each elbow position (flexed, mid-range and extended) for the movements performed by subject D.A. shown in Fig. 8. With practice variability in the muscle
Changes in EMG variability with practice of movements from different start positions. Averaged EMGs of the biceps and lateral, long and medial heads of triceps from movements 1-10 (top) and 61-70 (bottom) at extended, flexed and mid-range start positions are shown. These EMGs are for the same movements shown in Fig. 10 for subject D.A. The rectified EMGs were filtered (10 Hz. cutoff, 0 phase shift) and aligned to movement onset prior to averaging.
Table 11
Results from multiple and single linear regression of percentage changes in EMG variability on percentage changes in peak velocity and trajectory variability during practice

<table>
<thead>
<tr>
<th></th>
<th>EMG var. vs. peak vel., traj. var.</th>
<th>EMG var. vs. peak vel.</th>
<th>EMG var. vs. traj. var.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexions (NL(^b))</td>
<td>.698</td>
<td>.698</td>
<td>.029</td>
</tr>
<tr>
<td>Extensions (NL)</td>
<td>.708</td>
<td>.471</td>
<td>.454</td>
</tr>
<tr>
<td>Flexions (LC(^c))</td>
<td>.561</td>
<td>.282</td>
<td>.284</td>
</tr>
<tr>
<td>Extensions (L)</td>
<td>.535</td>
<td>.520</td>
<td>.042</td>
</tr>
</tbody>
</table>

---

\(^a\) coefficients of determination from linear regressions

\(^b\) data from 100° and 300° movements with no load (df = 10)

\(^c\) data from 100° and 300° movements with load (df = 8)
activity increased in association with decreased variability in the movement trajectories. This was particularly evident at the flexed elbow position (middle records) in which the increase in movement velocity was greatest (Fig. 8 - middle records). At the extended position (left hand side records) increased variability in the biceps EMG pattern and in the EMGs of the lateral and long heads of triceps were evident but there was decreased variability in the activity of the medial head of triceps. Movements made from the mid-range position (right hand side) were associated with little change in EMG variability. There was a relatively small increase in mean peak velocity of these movements and trajectory variability was greatly decreased (Fig. 8 - right side records). Thus, as discussed previously, EMG variability appeared to depend on changes in mean peak velocity of movements. When subjects used the strategy of making fast movements initially and maintaining movement velocity during practice EMG variability decreased as was observed for 100 and 300 movements made under no-load and load conditions. Mean percentage change for all subjects in variability of biceps and lateral head of triceps EMG activity was 140.7% (s.e. 42.3%, n=36, flexions and extensions, movements beginning from all 3 elbow positions). The possibility that interactions between the 3 heads of triceps compensate for some of this variability was examined by studying variability of biceps EMG in combination with the summed activity of the 3 heads of triceps. If interactions took place which reduced variability one would expect smaller changes in combined agonist-antagonist EMG variability with practice. Mean percentage change in EMG variability for biceps and the summed activity of the 3 heads of triceps was 145.8% (s.e. 46.2%), indicating that any such interactions did not reduce overall EMG variability.
B. Effects of movement speed on EMG variability

The effects of practice on EMG variability were confounded by the apparent influence of movement speed. A second experiment was therefore carried out in order to determine the effects of movement speed on EMG variability independent of practice. Additionally the relationship between movement velocity, trajectory variability and EMG variability under constant practice conditions was studied.

Increases in movement speed produced increases in both trajectory variability (as already shown) and EMG variability. EMG variability was found to increase in logarithmic fashion with both trajectory variability and movement speed. This is shown in Table 12 which contains correlation coefficients from the logarithmic relations of EMG variability with trajectory variability and movement peak velocity. Since trajectory variability and movement speed were strongly related (Fig. 12) it was important to determine whether variability in the EMGs was independently related to movement velocity as was observed in the practice experiments. Movements of different speed in which trajectory variabilities were equal or nearly equal were therefore examined. In some subjects movements of 400 and 600 msec duration were associated with nearly identical trajectory variabilities. Fig. 25 shows average movement trajectories and EMGs for one such subject performing 30° extension movements of about 600 msec (A) and 400 msec (B) duration. Trajectory variabilities of these different velocity movements were nearly identical in terms of average ellipse size over the entire movement. Variabilities in both the agonist and antagonist EMGs were, however, much greater for the faster
Table 12

Correlations of EMG variability with peak velocity and trajectory variability of equally practiced movements.

<table>
<thead>
<tr>
<th>Subject</th>
<th>EMG var.-Pk. vel.</th>
<th>EMG var.-Traj. var.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Flexions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.F.</td>
<td>.97a</td>
<td>.89&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>S.B.</td>
<td>.98a</td>
<td>.96a</td>
</tr>
<tr>
<td>D.A.</td>
<td>.99a</td>
<td>.90&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>M.M.</td>
<td>.93a</td>
<td>.99a</td>
</tr>
</tbody>
</table>

| **Extensions** |                  |                     |
| D.F.    | .99a             | .97<sup>a</sup>     |
| S.B.    | .99a             | .97a                |
| D.A.    | .98a             | .97<sup>a</sup>     |
| M.M.    | .98a             | .99a                |

<sup>a</sup> Correlation differs significantly from zero (p<.05, df=2)
Effects of movement velocity on variabilities of agonist and antagonist EMGs. On the left are shown averaged movement trajectories with superimposed variability ellipses for 30° extension movements of about 600 msec (A) and 400 msec (B) duration. Shown on the right are the associated EMGs with superimposed S.D. bars. Note the greater variability in EMGs of fast (B) versus slow (A) movements. Data are for subject D.F.
movements. This indicated that EMG variability depended strongly on movement speed under these conditions.

VII. Antagonist compensation for agonist variability in practiced movements.

In spite of the above observations on the effects of movement speed on EMG variability an explanation of reduced movement variability in the presence of increased EMG variability was still lacking. An attractive hypothesis was that variations in agonist EMG activity were compensated by variations in antagonist EMG activity to maintain movement trajectory relatively constant. That is, a large agonist burst could be associated with a large and/or early antagonist burst to prevent overshoot and maintain the desired movement trajectory. Previous work by Cooke (1979, 1980) indicated that deviations from an intended movement trajectory due to imposed torque perturbations were corrected so that the limb was returned to a prelearned trajectory. That is, the muscle responses to the perturbations acted to return the limb to the intended trajectory—a form of trajectory compensation. In a similar way movements which deviate from an intended movement trajectory as a result of agonist EMG variations may be returned to the intended trajectory by modification of antagonist EMG activity. This would be a form of trajectory compensation for naturally occurring variations in the movements due to variations in movement-related muscle activity. Indeed, the possibility that trajectory compensations may occur in these movements was indicated by the study of point-to-point variability throughout movement trajectories. Rapid increases in variability during acceleration of the limb did not continue
during deceleration when decreases in variability could occur.

In order to study trajectory compensations the assumption was made that the average trajectory of practiced movements (i.e., movements 51-60) is representative of the intended trajectory of all of those movements. This is a valid assumption because the movements have been practiced and are usually of low variability. The concept of an intended trajectory is important because it suggests a planned (programmed) path of the limb.

On the left hand side in Figs. 26 and 27 are shown plots of average (intended) trajectories for practiced movements with single trials from within these same movements superimposed (dots). On the right side are the associated EMGs of movement related muscles (averages - solid lines, single movements - dots). The data are for 10° extension movements (Fig. 26) and 30° flexion movements with an assisting load (Fig. 27). The single movements chosen were among the fastest and slowest movements within the averages.

Examination of the movement trajectory data indicated that in some movements trajectory compensations occurred (Fig. 26B,D and Fig. 27A,B). In these movements initial deviations from the intended trajectory were corrected and the limb returned toward the intended trajectory. In other movements initial deviations from movement intent were not corrected and the limb was not returned toward the intended path.

The differences between these corrected and uncorrected movements can be observed in the controlling EMGs. In Fig. 26B a large first agonist burst was associated with a larger and earlier antagonist burst which apparently returned the limb to the intended trajectory. A larger second agonist burst was evident in this movement which may also have
Trajectory compensations in practiced 10° movements. On the left are shown averaged movement trajectories (solid lines) from movements 51-60 performed by subject C.B. Superimposed on the average records are trajectories of single movements (dots) from within movements 51-60. The associated EMGs for averaged (solid lines) and single movements (dots) are shown on the right. The dashed vertical line indicates movement onset. These were 10° extension movements. The rectified EMGs were filtered (10 Hz. cutoff, 0 phase shift).
Figure 27

Trajectory compensations in practiced 30° movements. Averaged trajectories (solid lines) of practiced 30° flexion movements (51-60) performed under the load condition are shown on the left. Superimposed on these averages are trajectories of single movements (dots) from within these averages. On the right are shown the associated averaged (solid lines) and single movements (dots) EMG patterns. The EMGs were rectified and filtered (10 Hz. cutoff, 0 phase shift).
contributed to the observed correction. Similarly in Fig. 27B a large first agonist burst which caused a high peak velocity was compensated by a large antagonist burst and large second agonist burst. In Fig. 26D a smaller than average first agonist burst was followed by a small antagonist burst which provided trajectory compensation. Similarly in Fig. 27A a small first agonist burst was followed by a late antagonist burst (20 msec delay in comparison to average). Thus trajectory compensations as a result of modification in antagonist EMGs and the second agonist burst as a function of the first agonist burst were evident.

In some movements the first agonist burst was not accompanied by appropriate antagonist or second agonist bursts. In these movements trajectory compensation was not observed. For example in Fig. 26A the antagonist burst was too large for the average sized agonist burst, resulting in low peak velocity and decreased movement amplitude. In Fig. 27C an early antagonist burst produced a reduction in peak velocity and undershoot in spite of the large first agonist burst. In Figs. 26C and 27D delayed antagonist bursts resulted in high peak velocities and overshoot of the usual movement endpoint.

These results indicated that modifications in the antagonist and second agonist bursts occur which can compensate for variations in the first agonist burst. As a result limb trajectory during the deceleratory phase is maintained relatively constant in spite of variations in the first agonist burst. However appropriate control over the antagonist and second agonist bursts does not always occur, at least after 50-60 practice movements.
VIII. Effects of vibration on movements

Possible mechanisms for the observed trajectory compensations include the use of afferent feedback from the moving limb to control muscle activity subsequent to the first agonist burst. Sensory feedback from muscle spindles or joint receptors can provide information about the position and velocity of the limb. As suggested by Matthews (1972) a model of the limb movement may be set up in muscle spindles through the fusimotor system so that the spindles can act as error detectors when limb trajectory deviates from movement intent. Deviations from intent produced by variations in the first agonist burst may be sensed by the muscle spindles and corrections may be initiated to return the limb toward the intended movement trajectory. In order to study the role of muscle spindles in these movements vibration was applied to the triceps muscle tendon. Vibration is known to activate muscle spindles and thereby produce an inaccurate signal of the limb's trajectory to the CNS (Burke, 1976a,b). Thus the effects of muscle tendon vibration applied during acceleration and deceleration on movements and EMG activity were investigated in order to determine whether they may play a role in trajectory compensations. As described in the Methods these movements were visually guided and performed under the same instructions as previous experiments.

A. Effects of antagonist muscle tendon vibration on movements

Vibration of the antagonist muscle altered both the end position (at the end of deceleration) and the time course of movements. Figs. 28 and
Figure 28

Effects of antagonist vibration on 10° movements and related EMGs. Averaged records of movement position, velocity, agonist and antagonist EMGs are plotted. The solid lines are averages from control movements in which no vibration was applied. Averages from movements in which vibration was applied are plotted as dots superimposed on the control movements. Data from unpracticed movements are shown in A and B and for practiced movements in C and D. The vertical dashed lines indicate movement onset. The bars below the antagonist EMG records show the when vibration was applied during the movements. Data are for subject N.F.
Effects of antagonist vibration on 30° movements and related EMGs. The records shown are averages of position, velocity, agonist and antagonist EMGs. Data from control movements (no vibration) are plotted as the solid lines. Superimposed on the solid lines are data from vibration movements (dots). The bars below the antagonist EMG records indicate when vibration was applied during the movements. Records from unpracticed movements are in A and B and from practiced movements in C and D. Data are from subject S.S.
29 show the effects of vibration of the antagonist muscle during performance of 100° and 300° movements respectively. In most cases vibration applied during acceleration produced an initial undershoot of the usual end position of movement. This hypometric movement was associated with a decrease in peak velocity and was followed by a corrective movement which took the arm into the target zone. Both practiced and unpracticed movements were similarly affected by vibration during acceleration (Fig. 28A,C and 29A,C). In some cases there was no clear undershoot of the intended target. However these movements were characterized by a long deceleration period during which the limb moved slowly into the target.

Antagonist vibration during deceleration produced strikingly different effects on movements. Movement position and velocity were usually unaffected until after the target position had been reached. At this point there was a small return movement toward the original starting position (Fig. 28B,D and 29D). This was usually followed by a corrective movement back into the target (eg. Fig. 28B,D).

Figure 30 summarizes the effects of antagonist muscle vibration on movement peak velocity, amplitude and duration of the acceleratory and deceleratory phases. The histograms show the difference between the means from vibrated and control movements (vibrated - control). Thus reductions in, for example, deceleration duration (Fig. 30C) as a result of vibration are indicated by negative numbers on the ordinate. Both movement peak velocity and acceleration duration decreased as a result of antagonist muscle tendon vibration during acceleration (Fig. 30A,B). The changes were similar for unpracticed and practiced movements. The duration of the deceleratory phase was greatly reduced by vibration.
Antagonist vibration effects on movement kinematics and durations. The plotted histograms are the difference between the means of control and vibrated movements (control-vibration) in peak velocity (A), acceleration duration (B), deceleration duration (C) and amplitude (D) as measured at the end of deceleration. For A and B data for vibration during acceleration only are shown. In C and D the left set of histograms depict data for vibration during acceleration, the right set show data from vibration during deceleration. Within each set of histograms the left pair are for 100 movements and the right pair are for 300 movements. The open bars are for unpracticed movements and the stippled bars are for practiced movements. In D histograms for the deceleratory phase which are negative indicate differences in movement amplitude, positive histograms indicate differences in amplitude of return movements. The error bars represent 1 S.E.M. for 6 subjects.
during acceleration for 100 movements and for unpracticed 300 movements (Fig. 30C). The deceleration duration of practiced 300 movements was unaffected by vibration during the acceleration (Fig. 30C). Vibration applied during deceleration, in contrast, resulted in decreased deceleration duration of 100 but not 300 movements (Fig. 30C). Average movement amplitude as measured at the end of deceleration was decreased for all conditions (Fig. 30D). Tables 13 and 14 contain the differences in mean amplitudes (as measured at the end of deceleration) between control and vibration movements. The results of t-tests of these amplitude differences for unpracticed and practiced movements for each subject are also shown. Vibration during acceleration produced significant reductions in movement amplitude for unpracticed and practiced movements for nearly all subjects. In contrast vibration during deceleration produced small effects on movement amplitude which were usually not significant.

Vibration during deceleration resulted in return movements back towards the movement start position. These return movements were generally greater than in control movements (Fig. 30D). However these differences were very small, on the order of 10.

B. Agonist vibration effects on movements

The effects of agonist muscle tendon vibration on movements are illustrated in Figs. 31 and 32. When vibration was applied during acceleration, movement amplitude decreased and a corrective movement followed. This was especially evident for 100 movements (Fig. 31A,C) and occurred in all 6 subjects tested. For 300 movements the effect was not
Table 13
Effects of vibration during acceleration and deceleration on the amplitude of 100 unpracticed (1-50) and practiced (101-150) movements for each subject.

<table>
<thead>
<tr>
<th>Subj.</th>
<th>Acceleration Vibration</th>
<th>Deceleration Vibration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>tb</td>
<td>101 - 150</td>
</tr>
<tr>
<td>D.A.</td>
<td>-2.7</td>
<td>-6.12d</td>
</tr>
<tr>
<td>N.F.</td>
<td>-3.1</td>
<td>-6.55d</td>
</tr>
<tr>
<td>D.J.</td>
<td>-2.5</td>
<td>-4.92d</td>
</tr>
<tr>
<td>A.H.</td>
<td>-4.1</td>
<td>-9.44d</td>
</tr>
<tr>
<td>R.B.</td>
<td>-3.4</td>
<td>-6.10d</td>
</tr>
<tr>
<td>S.S.</td>
<td>-2.8</td>
<td>-4.98d</td>
</tr>
</tbody>
</table>

Antagonist Vibration

<table>
<thead>
<tr>
<th>Subj.</th>
<th>Acceleration Vibration</th>
<th>Deceleration Vibration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>tb</td>
<td>101 - 150</td>
</tr>
<tr>
<td>D.A.</td>
<td>-2.7</td>
<td>-9.99d</td>
</tr>
<tr>
<td>N.F.</td>
<td>-3.1</td>
<td>-6.60d</td>
</tr>
<tr>
<td>D.J.</td>
<td>-3.5</td>
<td>-5.41d</td>
</tr>
<tr>
<td>A.H.</td>
<td>-4.1</td>
<td>-2.80d</td>
</tr>
<tr>
<td>R.B.</td>
<td>-3.3</td>
<td>-1.30</td>
</tr>
<tr>
<td>S.S.</td>
<td>-2.8</td>
<td>-3.96d</td>
</tr>
</tbody>
</table>

Agonist Vibration

---

a difference in movement amplitude between control and vibrated movements measured at the end of deceleration

b t value for amplitudes differences for movements 1 - 50
c t value for amplitudes differences for movements 101 - 150
d p < .05 for t value
Table 14
Effects of vibration during acceleration and deceleration on the amplitude of 30° unpracticed (1-50) and practiced (101-150) movements for each subject.

Amplitude Difference\(^a\)

<table>
<thead>
<tr>
<th>Subj.</th>
<th>1 - 50</th>
<th>tb</th>
<th>101 - 150</th>
<th>tc</th>
<th>1 - 50</th>
<th>t</th>
<th>101 - 150</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antagonist Vibration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.A.</td>
<td>-2.9</td>
<td>-3.97(^d)</td>
<td>-2.7</td>
<td>-4.64(^d)</td>
<td>-0.6</td>
<td>-1.01</td>
<td>-1.0</td>
<td>-1.51</td>
</tr>
<tr>
<td>D.S.</td>
<td>-8.7</td>
<td>-10.80(^d)</td>
<td>-3.3</td>
<td>-2.41d</td>
<td>-2.5</td>
<td>-4.54(^d)</td>
<td>-1.7</td>
<td>-3.9d</td>
</tr>
<tr>
<td>S.S.</td>
<td>-7.1</td>
<td>-9.43d</td>
<td>-1.6</td>
<td>-2.61d</td>
<td>-1.4</td>
<td>-2.46d</td>
<td>-0.5</td>
<td>&lt; 1(^c)</td>
</tr>
<tr>
<td>J.S.</td>
<td>-5.3</td>
<td>-5.10d</td>
<td>-5.1</td>
<td>-5.40d</td>
<td>-2.7</td>
<td>-1.62</td>
<td>-2.8</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>S.T.</td>
<td>-0.7</td>
<td>-1.15</td>
<td>0.1</td>
<td>&lt; 1</td>
<td>-2.2</td>
<td>-5.40d</td>
<td>-1.8</td>
<td>-4.41d</td>
</tr>
<tr>
<td>P.R.</td>
<td>-2.3</td>
<td>-2.30d</td>
<td>-1.7</td>
<td>-2.36d</td>
<td>0.0</td>
<td>0.0</td>
<td>-0.7</td>
<td>&lt; 1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Agonist Vibration</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.A.</td>
</tr>
<tr>
<td>D.S.</td>
</tr>
<tr>
<td>S.S.</td>
</tr>
<tr>
<td>J.S.</td>
</tr>
<tr>
<td>S.T.</td>
</tr>
<tr>
<td>P.R.</td>
</tr>
</tbody>
</table>

\(a\) difference in movement amplitude between control and vibrated movements

\(b\) t value for difference in amplitudes for movements 1 - 50

\(c\) t value for difference in amplitudes for movements 101 - 150

\(d\) p < .05 for t value
Figure 31

Agonist vibration effects on 10° movements and related EMGs. The plotted records are averages of position, velocity, agonist and antagonist EMGs for control (solid lines) and vibrated (dots) trials. The bars below the agonist EMG records show when vibration was applied. In A and B are data from unpracticed movements and in C and D for practiced movements. Data are for subject N.F.
Effects of agonist vibration on kinematics and EMGs of 30° movements. Averaged records of position, velocity, agonist and antagonist EMGs are shown. The solid lines are data for control (no vibration) movements. The superimposed dots are for vibrated movements. The solid bars beneath the agonist EMG records indicate the time during which vibration was applied. A and B contain data from unpracticed movements. In C and D are data for practiced movements. Data are for subject S.S.
as strong and was seen clearly in 4 of the 6 subjects. For the subject
whose data is shown in Fig. 32 undershoot was only evident in the case of
practiced movements (Fig. 32C). The unpracticed vibrated movements (Fig.
32A) were, by random occurrence, faster than the control non-vibrated
movements and vibration during acceleration did not clearly influence
movement time course. However there was a strong deceleration of these
faster movements which probably prevented overshoot of the usual movement
endpoint. The effects of agonist muscle tendon vibration during
acceleration were, therefore, remarkably similar to the effects of
antagonist vibration during acceleration.

Vibration of the agonist during deceleration also produced effects
similar to those of antagonist vibration applied during this phase.
There were small return movements toward the start point after completion
of the deceleratory phase of movement (Fig. 31B,D and 37B,D). These
effects were, however, usually less than for antagonist vibration during
this phase (compare Figs. 31 and 32 with Figs. 28 and 29 for effects of
vibration during deceleration).

Fig. 33 summarizes the effects of agonist muscle tendon vibration on
movement kinematics and durations of movement phases for all subjects.
For 10° movements the effects of agonist vibration during acceleration
were similar to, but somewhat less than those for antagonist vibration
during this phase of the movement. Durations of the acceleratory and
deceleratory phases were reduced (Fig. 33B,C). Peak velocity of 10°
movements and practiced 300° movements was also reduced (Fig. 33A). In
the case of 300° unpracticed movements peak velocity was higher for
vibrated movements (Fig. 33A). This sometimes appeared to be an effect
of vibration. At other times, however, this appeared to be due to random
Effects of agonist vibration on movement kinematics and duration. The plotted histograms are the differences between the means of vibrated and control movements (vibrated-control) for peak velocity (A), acceleration duration (B), deceleration duration (C) and movement amplitude (D) as measured at the end of deceleration. In A and B data for the effects of vibration during acceleration only are shown. In C and D the left histograms are for vibration applied during acceleration and the right histograms are for vibration applied during deceleration. Within each set of histograms the left pair are for 10⁰ movement and the right pair are for 30⁰ movements. The open bars are for unpracticed movements, the closed bars for practiced movements. The error bars represent 1 S.E.M. for 6 subjects.
occurrence of higher velocity vibrated movements produced by a larger than normal first agonist burst prior to the beginning of agonist vibration. Movement amplitude, as measured at the end of deceleration, was decreased for movements in which vibration was applied during acceleration (Fig. 33D). This was confirmed by t-tests in individual subjects (Tables 13 and 14). Vibration during deceleration had no consistent effects on movement kinematics or durations of 100 or 300 movements. The amplitudes of return movements were generally greater than for controls when vibration was applied during deceleration (Fig. 33D).

C. Effects of antagonist vibration on EMGs

As just described, vibration applied during movement, particularly during acceleration, altered the time course of visually guided movements. Movement related muscle EMG activity was therefore studied to determine the causes of these changes in the movements. Antagonist vibration during acceleration produced increased activity in the antagonist muscle during the vibration (Figs. 28A,C and 29A,C). After vibration ended agonist muscle activity was increased over control levels for 100-150 msec. Vibration applied during deceleration usually resulted in increased tonic activity of both agonist and antagonist muscles (Figs. 28B,D and 29B,D). The increase in agonist activity usually began later than that in the antagonist.

These data are summarized in Figures 34 and 35 which show the changes in agonist and antagonist muscle activity during vibration for 100 and 300 movements respectively. Vibration during deceleration (solid bars)
Figure 34

Effects of vibration on EMGs of 10\(^\circ\) movements. The plotted histograms are the percentage difference in EMG activity between vibrated and control movements during the period of vibration (i.e., (vibration-control)/100%/control). The open bars show data for vibration during acceleration and the closed bars for vibration during deceleration. Within each set of histograms the left pair refers to unpracticed movements, the right pair to practiced movements. The error bars represent 1 S.E.M for 4 subjects.
Figure 35

Effects of vibration on the EMGs of 30° movements. Each histogram is the percentage difference in EMG activity during the period of applied vibration between vibrated and control movements (i.e., (vibrated-control)/100%/control). The open bars show data for vibration during acceleration and the closed bars for vibration during deceleration. Within each set of histograms the left pair are for unpracticed movements and the right pair are for practiced movements. The error bars represent 1 S.E.M. for 4 subjects.
consistently resulted in greater activity of both agonist and antagonist muscles (Figs. 34 and 35). Increased antagonist muscle activity was always produced by vibration during acceleration (Figs. 34A and 35A - open bars). For practiced movements vibration during acceleration also produced increased agonist muscle activity (Figs. 34B and 35B). Relative muscle activity was usually decreased by vibration because of the larger increases in antagonist muscle activity (Figs. 34C and 35C).

Changes in agonist and antagonist EMG activity in the 150 msec period following vibration of the antagonist during acceleration are shown on the left side of Figure 36. Agonist muscle activity was typically enhanced during this period while changes in antagonist activity were generally smaller and more variable (Fig. 36A,B). Relative muscle activity (agonist/antagonist) was always enhanced following vibration (Fig. 36C).

D. Effects of agonist vibration on EMGs

Vibration of the agonist muscle during movement also altered movement time course and muscle activity. Vibration during acceleration resulted in greater antagonist muscle activity during vibration. This was true for movements of 100° (Fig. 31A,C) and 300° (Fig. 32A,C) amplitude. Following vibration agonist muscle activity was enhanced for about 150 msec. While antagonist activity was unchanged or decreased. Thus vibration of the agonist during acceleration had effects similar to those for antagonist vibration during acceleration. Vibration during deceleration had variable effects on the EMGs although in most cases activity of both agonist and antagonist muscles were enhanced. Again
Figure 36

**EMG activity following vibration during acceleration.** The plotted histograms are the percentage differences in EMG activity during the 150 msec following vibration during acceleration between vibrated and control movements (i.e., \((\text{vibrated-control})/100\%\)/control). The open bars are for unpracticed movements and the closed bars are for practiced movements. Within each set of histograms the left pair show data for 10° movements and the right pair for 30° movements. The error bars represent 1 S.E.M. for 4 subjects.
these results are remarkably similar to those for vibration of the antagonist muscle. Figs. 34 and 35 summarize the effects of agonist muscle vibration on EMG activity during vibration. Changes in muscle activity during the 150 msec following vibration are shown in Fig. 36 (right side). In terms of relative changes in muscle activity (agonist/antagonist) the results for agonist vibration were similar to those for antagonist vibration.

E. Results of vibration control experiments

The effects of vibration applied during acceleration in the control experiments are shown in Fig. 37 (or 1 subject). Vibration during deceleration had no effects on movements in the control experiments. Fig. 37A shows the usual effects of vibration during acceleration when the vibrator was placed firmly over the triceps tendon. When the vibrator was not touching the subject there was no effect of the sound of the vibrator on the movements (Fig. 37B). Placement of the vibrator less firmly over the triceps tendon (Fig. 37C) produced small changes in the movements which were similar to those seen when the vibrator was attached tightly. With the vibrator placed loosely over the skin below the elbow (Fig. 37D) extension movements were decreased in amplitude while flexion movements were relatively unaffected by the vibration.

IX. Digital model of the limb

The results of the experiments presented so far suggested properties for a new model of limb movement control based on a linkage between
Results of control experiments on the effects of vibration applied during acceleration. Averaged velocity and position records are plotted. The solid lines are for control (no vibration) movements and the dots are for vibrated movements. The data in A show the already described effects of vibration applied to the triceps tendon during acceleration on movement kinematic records. In B are shown records from movements in which the vibrator was in close proximity to, but not touching, the subject. In C the vibrator was placed loosely over the triceps tendon and in D the vibrator was strapped loosely over the forearm just below the elbow.
phasic agonist and antagonist muscle activities. In this model the concepts of variability in the first agonist burst and associated variations in the antagonist which compensate agonist variations were incorporated. Phasic bursts of EMG activity were assumed to produce impulses (exerted torque over time) to accelerate and decelerate the limb. As detailed in the methods variability in the agonist (accelerative) impulse depends on movement speed and the amount of practice. Also the linkage (i.e., correlation) between agonist and antagonist is thought to be improved with practice. The limb was modeled as a linear second order system in order to simulate known properties of the limb (cf. Cooke, 1979; 1980; Hogan, 1984). As a result the concept that tonic muscle drive is used to maintain limb postures is incorporated. Phasic muscle drive provides control over the trajectory of movement between different limb postures. Thus this model includes properties of impulse-variability models (cf. Schmidt et al., 1979; Meyer et al., 1982) and of mass-spring models for final position control (cf. Feldman 1966 a,b; Bizzi et al., 1976; Polit and Bizzi, 1979). The ability of the model to simulate experimental findings supporting these models and of this thesis are presented.

A. Kinematic features of simulated movements

An example of kinematic records of an individual movement produced by the model and by a human subject moving a manipulandum are shown in Fig. 38. The movements produced by the model and by the human subject are qualitatively similar. The agonist, antagonist and resultant (agonist-antagonist) torques used to produce movements in the model are
Examples of 30° movements produced by the model and by a human subject. On the left are shown position and velocity records from a movement produced by the model. Also shown are net, agonist and antagonist torque records which produced the movement. On the right are shown position, velocity and torque records from a movement made by subject MM along with rectified filtered (10 Hz. cutoff, 0 phase shift) agonist and antagonist EMGs.
also shown along with agonist and antagonist EMGs and resultant torque exerted on the manipulandum by the human subject making the movement. The resultant torque measured from movements produced by human subjects represents the torque exerted on a manipulandum which will depend on the load presented by the manipulandum. The resultant torque curves are qualitatively similar as are the filtered agonist and antagonist EMGs and the corresponding torques in the model. One obvious difference between the movement of the model and that by the human subject is in the mechanism for maintenance of final limb position. In the model this is done with an agonist torque only. In the case of the movement by the human subject there is cocontraction of agonist and antagonist muscles during maintenance of final limb position. In the mass-spring models, final position is maintained by equal torques produced by the opposing muscles through their elastic characteristics. In the model a limb or joint stiffness has been specified as opposed to separate muscle stiffnesses (i.e., this is a lumped model). Thus the possible role of cocontraction in maintenance of final limb position, particularly in fast movements, was not considered in this model.

The model includes viscoelastic characteristics and a final postural agonist torque to drive the limb to final position. Thus absolute final position of the movements (i.e., when limb movement actually ends) will be independent of the agonist and antagonist phasic torques and of any short duration perturbations imposed during movement. Fig. 39A, for example, shows three movements generated with different phasic torques and starting from different initial positions. Since the tonic torque is the same in all cases, the movements end at the same position. Fig. 39B shows records of movements in which a brief perturbation was applied at
Effects of movement start position, phasic torques and perturbations during movement on attainment of final limb position in movements generated by the model. In A the position records of 3 movements with different start positions are shown along with the agonist and antagonist torques. Final position of the 3 movements was independent of start position and the different phasic torques used in movement generation. In B perturbations are superimposed on 3 movements with the same agonist and antagonist torques. The net torque records (agonist-antagonist-perturbation torques) of the 3 movements are also shown. The perturbations were 3 Nm in amplitude and 100 msec duration with onset times of 20, 180 and 340 msec in relation to movement onset. The same final position is attained in all 3 movements independent of the perturbations.
different times following movement onset. Although the perturbations affected the course of the movement, they did not alter movement endpoint. The model thus simulates the experimental results which support mass-spring models of movement control (cf. Bizzi et al. 1976; Polit and Bizzi 1979).

B. Effects of variations in peak agonist torque amplitude

Although, as just shown, movement endpoint is the same for all conditions in which the static torque is the same, measurement of movement endpoint at the end of deceleration would produce endpoint variability. Position at this point was strongly dependent on the phasic torques which drive the movements. This definition of movement endpoint means that only the initial impulse for the movement is being considered (cf. Woodworth 1899). This technique would be similar to the case of rapid aiming paradigms employed by Schmidt et al. (1979) and others in which movements are arrested by striking a surface.

Variability in movement endpoint and trajectories as a function of peak agonist torque variability are shown in Fig. 40. The model was tested under three conditions - no antagonist torque, independent antagonist torque and dependent antagonist torque. As pointed out previously, EMG studies have indicated that the magnitude and timing of the antagonist EMG burst depends in some way on the magnitude of the first agonist burst (Marsden et al., 1983; Flament et al., 1984). Thus the timing (onset) and amplitude of torques produced by antagonist muscles would depend on the agonist torque amplitude. Such a model was called the "dependent antagonist" model (inverted triangles in Fig. 40). For
Figure 40

**Agonist torque variability and movement variability relationships.**

Effects of agonist torque variability on movement amplitude variability (A) and trajectory variability (B) are shown for 3 conditions of antagonist torque (no antagonist, independent antagonist and dependent antagonist).
comparison data obtained with no antagonist torque (circles) and an independent antagonist torque (squares) are also shown.

In the independent antagonist condition variability in antagonist amplitude and onset time were independent of the variations in agonist torque amplitude. That is there was a near zero correlation between peak agonist and antagonist torque amplitude and between peak agonist torque amplitude and onset of the antagonist. The mean amplitude of the agonist torque was held constant at 3 Nm in these simulations so that mean peak velocity of the movements would be similar. In this way the effects of only agonist torque variability, independent of its mean amplitude, were examined. This would be the situation if movements of similar speed were studied after varying amounts of practice. Linear relationships between peak agonist torque variability and amplitude variability (as measured at the end of deceleration) were observed. In all cases, the variability in movement amplitude increased with the variability of the agonist torque initiating the movement. With a dependent antagonist (triangles) the movement end-point variability changed little in the lower range of torque variability (s.d. < .28 Nm). Most importantly the sensitivity of movement endpoint to variability in agonist torques differed for the three cases. The most sensitive case was the independent antagonist condition and the least sensitive was the dependent antagonist condition. This indicated that an independent antagonist would contribute to increased variability in movement endpoint while a dependent antagonist would reduce variability in movement endpoint.

The effects of peak agonist torque variability on trajectory variability of the simulated movements was also studied. The relationship between trajectory variability and peak agonist torque variability was
exponential (Fig. 40B). As in the case of amplitude variability the greatest trajectory variabilities were associated with the independent antagonist condition. The lowest trajectory variabilities resulted from movements produced with antagonist onset and amplitude dependent on agonist amplitude. Thus these data also indicate that antagonist torques reduce movement variability only if they are appropriately correlated with agonist torques.

The effects of antagonist torques on the point-to-point trajectory variability plot were also investigated (Fig. 41). In normal humans trajectory variability was shown to rise exponentially during the acceleratory phase of movements. Near the time of peak velocity the rate of increase in trajectory variability decreased. Average trajectories (left side), point-to-point trajectory variability curves (middle) and associated torques (right) from simulated movements are shown for the 3 conditions: no antagonist (top), independent antagonist (middle) and dependent antagonist (bottom) (mean peak agonist torque = 3 Nm with s.d. of .22 Nm for all 3 conditions). The amplitude (at the end of deceleration) and duration of movements in the no antagonist condition were greater than in the other conditions due to overshoot of the final resting position in the absence of the antagonist torque (Fig. 41). Had the movements been allowed to continue, end position would be the same in all conditions. For the no antagonist and independent antagonist conditions trajectory variability rose exponentially until well past the time of peak velocity. Indeed for both these conditions variability increased until the end of the phasic agonist torque (second arrow on point-to-point variability curves). After this time variability decreased due to passive (no antagonist) and active (independent
Point-to-point trajectory variability of movements produced by the model under no-antagonist, independent antagonist and dependent antagonist conditions. Shown on the left are average movement trajectories with superimposed variability ellipses. In the middle are point-to-point variability curves in which ellipse area is plotted as a function of time throughout the movements. On the right are shown the averaged agonist and antagonist torque records and their variability (vertical bars are 2 S.D. in length). Data in A are for movements with no antagonist, in B are movements with an independent antagonist and in C are movements with a dependent antagonist.
antagonist) braking torques. Note that in the independent antagonist condition the antagonist torque contributed to the exponential rise in variability during acceleration since it began prior to the end of the agonist torque. In the dependent antagonist condition the rate of increase in trajectory variability was reduced near the time of peak velocity as was observed for movements by human subjects and at the end of the agonist torque. In Fig. 41 the point-to-point trajectory variability curves for the independent and dependent antagonist conditions (bottom curves) were superimposed in order to emphasize the effects of the reduction in the slope of the point-to-point trajectory variability curves. The dashed vertical lines indicate the end of acceleration and the end of the phasic agonist torque. Note that the reduction in slope, although small here, had a large effect on the ensuing movement variability.

C. Rapid aiming paradigm

Schmidt et al. (1978; 1979) reported a linear relationship between movement endpoint (distance) variability and average speed (amplitude/movement duration) for fast accurate movements which had been practiced. Simulations of these movement were carried out in the model for movements of 5 different amplitudes (100–500) and 4 speeds at each amplitude. The correlation between agonist and antagonist torques (timing and amplitude) was examined under two conditions: (1) constant correlation of .99 between peak agonist and antagonist torques and (2) agonist-antagonist correlation proportional to the size of the antagonist burst. According to the first condition the agonist-antagonist linkage
is constant. The basis for the second condition is that variability in the agonist-antagonist torque relationship may depend upon the size of the required antagonist torque. Increased variability in this relationship would produce decreased correlation. Thus it was assumed that faster and larger amplitude movements may have lower correlations between the agonist and antagonist. This may be true in experiments on human subjects if the same number of practice trials are permitted at each movement amplitude/speed combination.

The results for simulations in the model are shown in Fig. 42. When the correlation between agonist and antagonist was varied according to the size of the antagonist a linear relationship between amplitude variability and average movement speed was observed (Fig. 42A). When the agonist-antagonist correlation was maintained constant over the amplitude/speed conditions a linear relationship was observed but at each movement amplitude variability was constant (Fig. 42B). Thus amplitude variability was a function of movement amplitude rather than of average speed.

The range of movement speeds examined at each movement amplitude were chosen in order to produce some overlap between average speeds at different amplitudes. Ideally one would like to compare movements of the same average speed at each amplitude. However this is not always possible since very short duration, small amplitude movements and long duration, large amplitude movements would be required. In the case of human subjects performing these types of movements the longer duration large amplitude movements would probably be more accurate/less variable since more time for corrections would be allowed. Woodworth (1899) observed this same problem and examined movements of different amplitude
Relationships between amplitude variability and average velocity during simulation of the rapid aiming paradigm. In A are data generated under conditions in which the correlation between agonist and antagonist torques was indirectly related to the size of the antagonist. In B are data in which the correlation between agonist and antagonist torques was constant.
with equal durations as opposed to equal mean speeds. In his experiments increases in movement amplitude, with movement time held constant, produced increases in endpoint variability. Thus movement variability was observed to depend on movement amplitude, although variability increased at a slower rate than movement amplitude (Woodworth 1899).

D. Rapid timing paradigms

Schmidt et al. (1979) also studied movement variability in rapid timing paradigms. In these paradigms subjects attempted to move the limb through a certain movement amplitude in a specified time with no spatial accuracy required. Thus subjects could accelerate through the movement target. Movements of greater amplitude are made with larger and more variable torques according to impulse variability models. However shorter movement times reduce the variability in impulse duration. Schmidt et al. (1979) showed that amplitude variability at the end of the required movement time in rapid timing paradigms was a linear function of the required movement amplitude. Movement time did not influence this relationship in rapid timing paradigms. In the simulations carried out here only an agonist torque was employed since no active braking was required. Variability in movement amplitude at the end of 100 and 200 msec was examined. An identical result to that observed by Schmidt et al. (1979) was found (Fig. 43). That is amplitude variability at the end of 100 or 200 ms was linearly dependent on movement amplitude only.

E. Practice simulations

The tests of the model thus far described have shown how movement
Figure 43

Relationship between amplitude variability and movement amplitude during simulation of the rapid timing paradigm. The plotted points are the variability in distance travelled after 100 or 200 msec as a function of the mean distance/amplitude travelled.
variability depends on the variabilities in both the agonist and antagonist torques. Of most interest, the model has indicated that movement variability can best be reduced by linking the agonist and antagonist torques. One situation in which movement variability is known to decrease is during practice. During practice not only does movement end-point becomes less variable but so does the path or trajectory taken during the movement. It was therefore hypothesized that practice of a movement results in an improved linkage between the agonist and antagonist EMGs and resultant torques. This hypothesis of progressively altered linkage with practice was applied to determine what changes would be predicted in the variability of movement trajectories with practice.

The effects of practice were simulated under two conditions related to the strategies observed for human subjects practicing these movements. In one simulation, movement velocity was maintained about constant and the variability in agonist and antagonist torques was decreased. In the other simulation movement velocity was increased with progressively larger and more variable agonist torques. In both simulations the linkage between agonist and antagonist were improved during "practice".

The results of the two practice simulations are shown in Fig. 44. Movement trajectories with superimposed variability ellipses and associated agonist and antagonist torques with superimposed S.D. bars are shown. The top records are simulations of "initial" movements and the bottom records of "practiced" movements. When movement velocity (and peak agonist torque) was maintained constant, one strategy observed during practice, clear reductions in trajectory variability were observed (Fig. 44A). These were associated with decreased agonist/antagonist
Practice simulations in a digital model of the limb. Shown are averaged movement trajectories and associated agonist and antagonist torques. In A mean agonist torque amplitude was constant and its variability decreased during simulated practice. In B mean agonist torque amplitude and its variability increased during simulated practice. Correlations between agonist and antagonist torques increased during simulated practice.
torque variability and increased agonist/antagonist correlation. The decrease in trajectory variability was greater than what would be observed solely due to decreased variations in the agonist and antagonist torques (i.e., without improved agonist-antagonist linkage) (Table 15). When movement velocity and agonist/antagonist torque variability increased during "practice" a smaller reduction in trajectory variability was observed (Fig. 44B). This was due to the improved correlation between agonist and antagonist impulses. Without improved linkage between agonist and antagonist torques trajectory variability would have increased under these conditions (Table 15). Thus movements with decreased variability were produced in spite of increased variations in the agonist and antagonist torques.
Table 15

Role of variability linkage in reduction of trajectory variability for practice simulations in the model.

<table>
<thead>
<tr>
<th>% change in trajectory variability</th>
<th>Constant linkage\textsuperscript{a}</th>
<th>Improved linkage\textsuperscript{b}</th>
</tr>
</thead>
<tbody>
<tr>
<td>maintain velocity during practice</td>
<td>-5.9</td>
<td>-84.6</td>
</tr>
<tr>
<td>increase velocity during practice</td>
<td>12.4</td>
<td>-65.6</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Correlations between agonist and antagonist torques was unchanged

\textsuperscript{b} Correlations between agonist and antagonist improved during simulated practice
Discussion

These experiments were undertaken in an attempt to increase understanding of voluntary control over limb movements. Studies of the effects of movement speed and practice on variability in movement trajectories and in EMG activities of related muscles were carried out. For voluntary movements the intent refers to the desired speed and accuracy of the movements (cf. Brooks, 1979a). With practice, an improved relation between movement intent and the actual course of the movement is assumed to occur. In the experiments described here subjects were instructed to perform accurate movements to a target and to attempt to increase velocity of the movements, while maintaining accuracy, during practice. However it is well known that there is a speed-accuracy tradeoff in movements (Woodworth, 1899; Fitts, 1954). Increases in speed of movements are associated with increases in error (missing the target - Fitts, 1954) or increases in endpoint variability (Woodworth, 1899; Schmidt et al., 1979). Thus subjects attempted to alter the speed-accuracy tradeoff during practice such that movements of higher speed would be made with no decrease in accuracy.

The focus in this discussion is on mechanisms underlying variability in movement trajectories. In this way the present work differs from most previous investigations of movement variability which have been concerned with movement endpoint variability (accuracy) only. In this investigation the study of variability was limited to the dynamic phases of the movement trajectories. Variability in postural phases of the movements (i.e., during maintenance of final position) was not studied. Thus variability in postural control mechanisms was not studied directly.

207
As such this discussion will focus on so-called "move" motor programs as opposed to the "hold" programs (cf. Brooks, 1984).

In the study of variability in movements it is important to realize that endpoint variability must develop during movement and that the way in which this variability develops is critical in terms of understanding the neural mechanisms underlying movement control. Variability may increase progressively throughout movements or initial increases in movement variability may be followed by decreased variability due to corrective mechanisms which attempt to maintain movement variability low. This is an important distinction in motor control terms because it reflects two opposing views prevalent in motor control research. These are the ideas of open-loop feedforward movement control versus error detection-correction (feedback) control modes. Of course these two control mechanisms may both be used during most movements (i.e., other than "ballistic" movements). Thus movements may be initiated and their initial trajectory controlled through open-loop feedforward mechanisms. Feedback control may be used during later phases of the movements. It is important to recognize the possible predictive nature of feedback control in this view. That is feedback received early in movements may be used to control later stages of the same movements (cf. Abbs et al., 1984; Bedingham and Tatton, 1985).

The discussion is organized so that variability in movement trajectories as a function of movement speed and practice are discussed first. Variability in the EMG activities of related muscles as it relates to trajectory variability is examined next. Possible feedback control mechanisms involved in trajectory control are examined in relation to the effects of muscle tendon vibration applied during
different movement phases. Finally, the model developed as part of the present work and its implications for motor control models are discussed.

Initial experiments examined the effects of practice on movements of different amplitude with the instruction to increase movement speed during practice. Human subjects made smooth movements even in the initial stages of practice. Only the first 2 or 3 movements made had obvious discontinuities (cf. Brooks et al., 1973). In contrast, monkeys make discontinuous movements over a period of several months of learning (Brooks et al., 1983). This probably reflects a lack of understanding of the task by the monkeys. Human subjects also used phasic, alternating patterns of EMG activity in the first movements made between targets. Thus even the initial movements made by human subjects had the characteristics of so-called programmed movements (i.e., continuous movements, phasic muscle activity) (cf. Brooks et al., 1983; Person, 1956).

Changes in elbow posture (angle from which movements were initiated) had striking effects on performance of practiced movements of the same amplitude. Usually movement velocity decreased and trajectory variability increased following a change in start position for the movements. This indicated that the change in elbow posture necessitated reprogramming of the motor commands for movement presumably because of the changes in muscle (length-tension) and joint (muscle moment arms) properties with changing elbow angle. With practice subjects increased movement speed and reduced movement trajectory variability regardless of the elbow angles from which movements were made. This was accomplished with modifications in the phasic and tonic muscle activities associated with the "move" and "hold" components of the motor program (cf. Brooks,
Motor commands are, therefore, posture dependent and are not immediately modified when changes in posture occur.

Practice resulted in decreased variability throughout movements of $10^\circ$ and $30^\circ$ amplitude. This was indicated by decreased variation in the acceleratory and deceleratory phases of phase plane trajectories of the movements. The effects of speed of movement on variability of the movements was also investigated because subjects were instructed to increase movement speed during practice. Movements of greater speed were associated with greater variability in the associated movement trajectories when equal numbers of practice movements were performed at each of the different movement speeds. Similarly, others have observed that increases in movement speed resulted in decreased movement accuracy and increased endpoint variability of movements (Woodworth, 1899; Fitts, 1954; Schmidt et al., 1979). Thus, during practice of a simple movement task, subjects can improve performance in that movements can both be performed faster and with less variability.

An important question relates to how trajectory variability develops during movements. Whether trajectory variability increased progressively or decreased during movement was an important question. If, at any points during movement, decreases in trajectory variability occurred this would indicate corrective mechanisms acting to reduce variability in movements. It was found that trajectory variability rises rapidly during acceleration of the limb. This suggests that there is a great deal of variability associated with production of forces used to accelerate the limb. The rate of increase of variability during acceleration varied with both movement amplitude and speed. Larger amplitude movements ($30^\circ$ versus $10^\circ$) and faster movements (with equal
amounts of practice) were associated with a greater rate of rise of variability during acceleration. Thus, as the agonist muscles exert more force at faster rates the variability in this force output increases, producing greater variability in movement kinematics during acceleration. These observations are in support of impulse-variability models of movement control which postulate that motor output variability is directly proportional to the mean size of the motor output (cf. Schmidt et al., 1979; Meyer et al., 1982). Increase in the size of accelerative impulses for larger amplitude or faster movements were associated with greater variability in these impulses.

Near the end of acceleration, the rate of change of variability decreased or became negative (decrease in variability). These decreases in variability which began near the end of acceleration and often continued throughout the deceleratory phase are indicative of corrective processes acting to reduce variability in movement trajectories. This suggests that forces acting to decelerate movement of the limb may compensate for variations in the accelerative forces. Impulse variability theories have assumed either no influence of decelerative impulses on movement variability (Schmidt et al., 1979) or that decelerative impulses are a mirror image of accelerative impulses (Meyer et al., 1982). In these models the possibility that decelerative impulses could be organized to correct for accelerative impulse variations has not been considered. In the present study it appeared that variations in decelerative impulses were compensating for variations in the accelerative impulses. In this way continued increases in variability were prevented and reductions in variability could occur during deceleration.
The reduction in point-to-point variability occurring near the end of acceleration resulted primarily from decreased variability in velocity profiles at this point in the movements. Deceleration of faster movements was initiated earlier than in slower movements in order to prevent overshoot of the intended target of the movements. This could occur only if a larger decelerative torque was initiated earlier in fast movements. This larger, earlier decelerative torque would therefore contribute to reductions in variability of movements during the deceleratory phase.

The finding that trajectory variability of the deceleratory phase of movements was greater than in the acceleratory phase can also be explained on the basis of the point-to-point variability curves. Variability in later stages of movements must depend on the variations accumulated in earlier stages of the movements. Due to the fast rise in variability during acceleration, the variability is high at the beginning of deceleration. Thus, in spite of decreases in variability usually occurring during deceleration, the variability of this phase remains greater than in acceleration, even after a prolonged practice session (300-1000 movements). The motor control system must therefore either maintain a low rate of rise of variability during acceleration or compensate for accelerative variations appropriately in order to produce movements with low variability. Indeed the strategy of compensation for accelerative impulse variations may represent a "good enough" solution to the problem of producing fast movements of low variability. That is, it may be easier for the nervous system to compensate for variations in accelerative forces than to produce such forces with low variability.

Trajectory variability of 300 movements was about 3 times that of
10° movements even after extended practice (up to 1000 movements). This occurred because of the greater rate of rise of variability during acceleration in movements of greater amplitude as discussed above. It is known that movement peak velocity increases linearly with movement amplitude, with the slope of the peak velocity-amplitude curve being dependent on instruction to the subject (Cooke, 1979; 1980b). The higher peak velocities of 300, in comparison to 10°, movements require larger accelerative impulses, than smaller movements. Therefore, as predicted by impulse-variability models, there would be greater variability in the accelerative impulses and greater variability in limb acceleration. The greater variability of the acceleratory phase is also reflected in the deceleratory phase because variability in later phases of movements depends on the variability accumulated in earlier phases as already discussed.

How is variability in movements reduced during practice? As discussed previously there are two possible strategies the nervous system could use. One would be to reduce variability in accelerative and decelerative force profiles and thereby decrease variability throughout the practiced movements. Alternatively, the nervous system could control the decelerative forces in such a way as to correct for movement variations induced by accelerative force variations. These possibilities were examined by studying the EMG activity in movement related muscles (agonists and antagonists). Under isometric conditions EMG amplitude is strongly related to muscle tension (Lippold, 1952). During movements the timing and amplitude of phasic EMG bursts in agonist and antagonist muscles are strongly related to movement peak velocity (Brown and Cooke, 1981; Marsden et al., 1983; Flament et al., 1984). Movements of higher
peak velocity require larger accelerative and decelerative forces and are associated with larger agonist and antagonist EMG bursts. Thus the EMGs of movement related muscles are related to the forces produced by these muscles, although this relation may be complex (cf. Stein, 1982).

Study of variability in movement related muscle EMG activity showed that reductions in trajectory variability during practice could be associated with increased or decreased variability in the associated EMGs. Whether increased or decreased EMG variability was observed depended on the changes in movement speed during practice. If movement peak velocity was maintained relatively constant during practice, reductions in variability of agonist and antagonist EMGs and of the movement trajectories were observed. However most subjects increased movement peak velocity, as instructed, during practice. Under these conditions, variability in the EMGs of related muscles increased in spite of decreased variability in the associated movements. The dependence of EMG variability on movement speed was confirmed in a study of movements of different controlled speeds.

There are two questions which arise from the results of studies of relations between variability in movement related muscle activity and practice and movement speed. First, why does EMG variability increase in faster movements? Second, how can variability in movements be reduced in spite of greater variability in the EMGs of agonist and antagonist muscles?

There are a number of possible explanations for the increase in muscle EMG variability as a result of increased movement speed. One is that the recruitment and rate modulation of motor units may be a variable process in which the variability depends on the required
contraction forces. Larger contraction forces require increased recruitment and higher firing frequencies of motor units. In a recent study of muscle activity during ramp and ballistic isometric contractions it was observed that variability of the surface EMG was much greater in the case of ballistic contractions (Yoneda et al., 1983). Ballistic contractions were defined as those in which the time to peak force was less than 150 msec. The authors suggested that different recruitment properties of motor units during fast and slow contractions were responsible for the differences in variability of the surface EMGs. Although in dynamic contractions there is no comparable measure to the time to peak force in isometric contractions it is clear that higher velocity movements must be associated with an increase in contraction speed. Thus the greater variability observed for high velocity movements may be a function of different recruitment properties of fast twitch motor units. Neural commands to muscles may not be more variable in faster movements, but the actual recruitment and rate modulation of motor units may cause greater variability.

The finding of increased EMG variability in ballistic, isometric contractions and the findings of the present investigations would support the impulse variability models of movement control (Schmidt et al., 1979; Meyer et al., 1982). In these models the explanation for the classical speed-accuracy tradeoff (Woodworth, 1899; Fitts, 1954) is based on the idea that variability in the impulses causing movement acceleration and deceleration depends on impulse size. Fast movements require larger impulses and are, therefore, more variable according to this theory. The increased variability associated with larger impulses has been attributed to greater variability in the neural output. The findings of the present
studies of EMG variability confirm that there is greater motor output variability, as measured in surface EMGs, in movements of greater speed.

An important question is whether greater variability in muscle activity reflects greater variability in descending commands to the motor neurons or in recruitment properties of motor units. When descending neural drive to motor neurons is increased in order to excite high threshold, fast twitch motor units, the actual numbers of units recruited and their firing rate may become more variable. This increased variability could result from, for example, variable excitability of motor neurons. That is, the number of motor units activated by supraspinal commands would depend on the excitability of the motor neuron pool. Alternatively activation of greater numbers of motor cortical neurons may also be more variable, resulting in greater variability in the descending motor commands to the agonist and antagonist muscles.

Whether variability in neural activity arises at spinal or supraspinal levels is an interesting question which cannot be answered by the present experiments. However a more important question is how does the nervous system overcome this greater variability in order to produce fast movements of low variability. One possibility is that other muscles synergist to those recorded in these experiments may contribute in such a way as to decrease the variability in total muscle torque output. That is, although variability in activity of each muscle in a synergist group may increase with increased movement speed, the variability in torque output of the muscle group may be less variable due to compensatory interactions. Studies of muscle activity of each of the 3 heads of the triceps brachii muscle group in the present work indicated that such interactions in synergist muscle activity do indeed occur. Practiced
elbow flexion or extension movements of similar speeds and amplitudes were associated with opposing variations in the phasic EMG bursts of the 3 muscles. The possibility that synergist muscles may contribute differentially to the total muscle torque has also been suggested in previous studies associated with muscle fatigue and EMG/torque relationships (Hof and Van Den Berg, 1975; Viitasalo, 1981; Darling and Hayes, 1983). However studies of synergist muscle activity performed as part of this thesis did not provide complete support for this view. The compensatory interactions among individual muscles of a synergist group were not consistent from movement to movement so that overall synergist EMG variability was reduced.

It is also possible that the nervous system does not control the activity of individual muscles but rather some relationship between opposing muscles. It has been shown that the ratio of opposing muscle activities defines static limb position (Lestienne et al., 1981). It has been suggested that movements could be made by gradual changes in limb equilibrium position (Bizzi et al., 1984). If this is the case then the ratio of opposing muscle EMGs taken during movement may provide an indication of the time-course of changes in the limb equilibrium position during movement. Experiments performed on deafferented monkeys revealed that at least during slow movements there is probably a gradual change in limb equilibrium position (Bizzi et al., 1984). Thus the ratio of opposing muscle activities may be controlled by the nervous system during movement. One might therefore examine variability in the ratio of opposing muscle activities during movement as a function of speed and practice. However during slow movements there are relatively gradual changes in the EMG activities of the movement related muscles. Faster
movements are associated with phasic bursts of muscle activity. The concept of limb equilibrium positions is dependent on a maintained level of muscle activity. The question of whether EMG ratios during phasic muscle activity is representative of limb equilibrium position has not yet been addressed empirically.

Another possibility, suggested by the studies of point-to-point variability, is that variations in agonist muscle activity which initiate movement could be compensated by appropriate linked variations in antagonist muscle activity. Thus large or small agonist EMG bursts could be compensated by large and small antagonist bursts. The timing of antagonist muscle EMG activity could also be important in the compensation for agonist EMG burst variations. This idea of compensation for variability has also been suggested in studies of speech (Hughes and Abbs, 1976), hand (Cole and Abbs, 1985) and gait motor control (Winter, 1984). In such studies it has been observed that in compound movements there can be considerable variability in the kinematics and joint torques of individual body parts but relatively low variability in relation to the system goals for that movement. For example, in speech movements variations in jaw movements during lip closure for consonant production are compensated by appropriate linked variations in lower lip movements such that the goal of lip closure is always met (Hughes and Abbs, 1976).

The motor equivalence concept (cf. Hebb, 1949) has been invoked to explain the results of studies showing low variability in some movement parameters in spite of high variability in contributing movement variables. Motor equivalence was suggested as a means for reducing the many degrees of freedom associated with movement by control over only certain variables. For example in the speech movements studied by Hughes
and Abbs (1976) the lip and jaw movements can be considered redundant degrees of freedom since variations in movements of one structure could be compensated by linked variations in the other structure. The goal of lip closure could be attained with many combinations of jaw and lip positions.

In the simple movements studied as part of this thesis, motor equivalence may also be employed. Although there is only one degree of freedom in the direction of these movements there are many muscles involved in their control. Theoretically it would be possible to control discrete, single joint movements with a single agonist muscle. Joint movements in one direction could be initiated with an agonist EMG burst and final joint position controlled by the elastic properties (stiffness and equilibrium length) of the agonist. Under these conditions movement variations would be a direct function of variations in agonist muscle activity. However the use of antagonist muscles provides a method for compensating for variations in agonist muscle activity. Thus, if one viewed the phase plane trajectory as the overall system goal in these movements the intended trajectory could be accomplished with highly variable agonist and antagonist muscle activities if the variations were linked appropriately. For example a large agonist burst would result in a higher than intended peak velocity and, probably, overshoot of the intended movement endpoint. A large and/or early antagonist burst would compensate for the deviations from an intended trajectory induced by a large agonist burst so that peak velocity would be closer to intended and there would be little or no overshoot of the movement endpoint. It is important to note that this refers to control over the movement trajectory (velocity-position relation) as opposed to control over only
movement endpoint. If only movement endpoint were important the elastic muscle forces would drive the limb to final position in the presence of large or small agonist EMG bursts.

Study of the point-to-point changes in trajectory variability during movements did indicate that decelerative forces compensate for variability in accelerative forces. In this way variability could be decreased during deceleration of the movements and the limb braked along a path of low, decreasing variability toward the intended target of the movements. Neural activity generating acceleration of the limb is reflected in the first agonist burst which initiates limb movements. Thus variability in movement kinematics during acceleration would depend on the size and variability in the first agonist burst. Absolute variability in the first agonist burst, although not measured alone in these experiments, appeared to increase as a function of burst amplitude. Thus larger amplitude agonist bursts, associated with faster movements, were typically more variable. Greater variability in the size of this phasic burst of activity would produce greater variations in the accelerative torques imparted to the limb as indicated by studies of the relations between EMG and isometric forces.

Decelerative forces, or forces resisting movement, are provided by the viscoelastic characteristics of muscle, ligaments, etc. and by active force generation provided by the antagonist. Muscular contraction also increases muscle and joint stiffness and viscosity, thereby-increasing the magnitude of viscoelastic forces resisting movement. These viscoelastic forces can provide some compensation for variability in accelerative forces. For example a large accelerative force, resulting in high movement velocity, will be opposed by viscous forces of greater
magnitude. Such forces will tend to reduce kinematic variability in response to variable agonist EMG bursts since their magnitude is appropriately linked to the size of the agonist burst. However active force generation by antagonist contraction provides the major braking mechanism for fast movements in which viscoelastic forces are unable to brake the limb quickly (cf. Lestienne, 1979). Thus in fast movements of the type studied here one would expect to see variations in antagonist muscle activity providing compensation for variations in the first agonist burst. This was indeed the case as it was observed that variations in the first agonist burst were accompanied by variations in timing and amplitude of the antagonist burst. These antagonist burst variations usually provided appropriate compensation for agonist burst variations such that movement variability was reduced. Additional compensatory alterations in the second agonist burst were observed which apparently aided in reduction of movement variability during deceleration.

On the basis of the preceding discussion one would expect good correlations between the sizes of the agonist and antagonist bursts in well practiced simple limb movements. This would be necessary in order to produce the observed compensations. Previous work has indicated good relationships between the amplitudes of each of these bursts and onset time of the antagonist burst with kinematic variables such as peak velocity of movement (Brown and Cooke, 1981, Marsden et al., 1983, Flament et al., 1984). However relationships between amplitudes of the agonist and antagonist bursts were not presented in these studies. Cooke (1979) showed that there was not a strong positive relationship between these burst amplitudes in well practiced movements. This would argue
against the idea that variations in the agonist burst are compensated by variations in the antagonist burst in such a way as to maintain the movement trajectory constant. However the timing of the antagonist burst must also come into this relationship. That is a large agonist burst could be compensated by a large or an earlier antagonist burst. It has been shown that increases in movement speed for a given movement amplitude are associated with larger and earlier antagonist bursts (Brown and Cooke, 1981, Marsden et al., 1983, Flament et al., 1984). Thus there may be a complex relationship governing control of the timing and amplitude of antagonist burst activity as a function of agonist burst activity in relation to the intended movement trajectory. The presence of the second or late agonist burst and its role in trajectory compensation may make this relationship more complex. It is important to remember that it is the output torques of the agonist and antagonist muscles which must be well correlated in order to produce the trajectory compensations. As discussed earlier, synergist muscles may contribute differentially to the total torque and this contribution may vary from movement to movement resulting in low correlations between agonist-antagonist EMGs when only 2 muscles are considered.

What mechanisms might underly control of the timing and amplitude of the antagonist and late agonist bursts in such a way as to produce the observed trajectory compensations? One possible mechanism would be the use of efference copy of the first agonist burst to control antagonist and second agonist burst timing and amplitude. Efference copy is hypothesized to be a neural copy of a motor output signal and, perhaps, its expected sensory consequences. For eye movement control it has been hypothesized that efference copy of the oculomotor command signal is used
to maintain the visual world stable during movement. In a recent study Cooke and Diggles (1984) suggested that central monitoring of efferent commands for fast movements was necessary to explain the rapid error corrections they observed in a choice reaction time task.

Hore and Vilis (1984) have also suggested an efference copy mechanism for control over antagonist muscle activity associated with return movements to a target following perturbations. This suggestion was based on experiments in primates trained to return the limb to a target zone following step or pulse perturbations. Depending on the type of perturbation applied a small, late antagonist burst (step perturbation) or large, early antagonist burst (pulse perturbation) was required to return the limb accurately to the target. Responses in the stretched (agonist) muscle to step perturbations were of longer duration than for pulses because of the need to overcome the continued torque of the step perturbation. Based on experiments in which the "set" (i.e., type of perturbation expected) and the actual perturbation received were varied it was suggested that the antagonist burst was controlled in a predictive manner on the basis of agonist EMG activity. This suggestion was based on the observation that antagonist burst activity preceded, rather than followed, antagonist stretch during the return movement. Thus, such antagonist muscle activity was not stretch dependent and could not be attributed to a stretch reflex. This predictive control over antagonist muscle EMG activity was abolished by cooling of the cerebellum or was incorrect if the perturbation received was not the one expected (i.e., set for pulse, get step). It was suggested that predictive components associated with skilled movements by the monkeys were based on efference copy of the agonist command through the cerebellum which
apparently mediated the predictive modifications in antagonist muscle activity necessary for good performance. Hore and Vilis suggested that the appropriate use of efference copy depends both on set and training (practice).

Afferent feedback control over the antagonist and late agonist bursts would provide another possible mechanism for trajectory compensation. That is feedback from muscle, joint and cutaneous receptors in the moving limb may provide a signal to modify these EMG bursts appropriately. Support for this possibility comes from experiments in which the effects of muscle tendon vibration on movements and EMGs were studied. Vibration of the antagonist muscle during proprioceptively guided movement causes an increased antagonist burst and resulting undershoot of the intended target of the movement (Capaday and Cooke, 1981; 1983). In the experiments presented here, vibration applied to the antagonist during only the acceleration phase had a similar effect on visually guided movements. Increased antagonist muscle activity during the vibration resulted in a decreased duration of the deceleratory phase of movement and, usually, an undershoot of the usual endpoint of the movements. Since activation of antagonist muscles spindles during acceleration would indicate movement speed was higher than expected (cf. Sittig et al., 1985), the increased phasic antagonist activity would provide a greater braking force as would be needed in faster movements. Naturally occurring variations in the first agonist burst would produce alterations in movement speed during the acceleratory phase which could be sensed by muscle spindles. The resulting afferent signal could be used to appropriately control the antagonist EMG burst.

In previous experiments it was shown that vibration of the agonist
muscle had no influence on movements (Capaday and Cooke, 1981, 1983). This was explained by the fact that shortening of the agonist probably unloaded the spindles so that they were not activated by the vibration (Capaday and Cooke, 1981;1983). In the present study, however, vibration of the agonist muscle tendon during acceleration influenced movement time course in a manner similar to that for antagonist tendon vibration. It is possible that vibration of the agonist muscle was conducted through bone and soft tissue to affect the activity of antagonist muscle spindles. However, since such vibration would be greatly attenuated by conduction through the body tissues one would not expect agonist tendon vibration to strongly influence the firing of antagonist muscle spindles. The observed differences may therefore be due to the different experimental paradigms. In the present investigation vibration was randomly applied for short periods of time (80-200 msec) during visually guided movements to small targets. Capaday and Cooke (1981, 1983) applied vibration throughout proprioceptively guided movements. Unloading of agonist muscle spindles would occur during movements in either experimental paradigm (Goodwin and Luschei, 1975). However the presence of vision and the accuracy required in the present experiments may have favoured the use of other sensory afferents in movement control (e.g., Golgi Tendon Organs).

There are 3 possible mechanisms by which agonist vibration might influence antagonist muscle activity. One is that activation of the vibrator and the resulting noise startled the subject, causing antagonist activation regardless of which muscle was vibrated. This was disproved by the control experiments which showed that the sound of the vibrator alone did not influence movements. Alternatively stimulation of
cutaneous afferents by vibration could activate the antagonist biceps and influence movement time course. The control experiments did indicate that stimulation of cutaneous receptors could affect the time course of extension movements in a manner consistent with this hypothesis. Thus feedback from cutaneous afferents may also be involved in control of these movements.

A third possibility is that agonist tendon vibration activated Golgi tendon organs (GTOs). Agonist GTO activation would indicate that the agonist muscle force was too great. This could be opposed by activating the antagonist muscle to prevent overshoot of the desired end point of the movement. Vibration of relaxed muscle does stimulate GTOs (Burke et al., 1976). Thus GTOs in the shortening agonist may be activated by the vibration resulting in antagonist muscle contraction and movement undershoot. In the case of antagonist vibration, the spindle response probably dominates the GTO response because the muscle is lengthening. In addition the CNS may selectively gate the spindle activity in the lengthening muscle for use in movement control. In the case of a shortening muscle the GTO stimulation may dominate because the spindles are unloaded while the GTOs are active due to tension in the tendon from contraction. Under normal conditions a large agonist force sensed by GTOs would also be associated with rapid lengthening of the antagonist muscle. Thus afferent feedback from both agonist GTOs and antagonist spindles during acceleration of the limb may be used in control of the antagonist burst and the movement trajectory.

When vibration was applied during deceleration to either the agonist or antagonist there was relatively little effect on the ongoing movement. Usually there was a movement of the limb back toward the movement start
point after the end of deceleration. Although these return movements were very small they do indicate effects similar to, but less than, the effects of vibration during acceleration. This was consistent with the observation that vibration during deceleration of either the agonist or antagonist results in greater activity in both muscles. Since there is mainly tonic muscle activity occurring during this phase of movement the increased muscle activity due to vibration would effectively increase the impedance of the joint to rotation (Hoffer and Andreassen, 1981a,b). The effects of unexpected afferent feedback during deceleration may therefore be to increase joint impedance and thereby decrease the effect of the apparent perturbation on the trajectory of the ongoing movement. Indeed effects of vibration applied during deceleration were not observed until after the usual movement endpoint had been reached.

The results of experiments in which muscle tendon vibration was applied clearly indicate a role for afferent feedback in control of fast accurate limb movements (peak velocities of 150-2000/sec for 300 movements and 60-900/sec for 100 movements). The movement phase during which altered feedback occurs is important. Vibration applied during acceleration has a greater influence on movement and related muscle activity, particularly of the antagonist, than vibration applied during deceleration. The deceleratory phase of movements could therefore be controlled in a predictive manner based on afferent feedback concerning the state of the limb during acceleration.

The results of the experiments discussed so far point to a number of new concepts in motor control. These findings indicate that the trajectory of fast movements is controlled in a rather complex manner. The variability in trajectories of fast movements does not depend on the
variability in agonist or antagonist muscle activities alone. Rather covariations between agonist and antagonist muscle activities are important in determining movement variability. This represents a significant departure from previous models of limb control attempting to explain variability in fast movements. In these previous models, termed impulse-variability models, the decelerative forces were either ignored (Schmidt et al., 1979 - asymmetric impulse-variability model) or were considered to be a mirror image of accelerative force profiles (Meyer et al., 1982 - symmetric impulse-variability model). Mirror image symmetry in accelerative and decelerative forces is indicative of perfect covariation between accelerative and decelerative force-time profiles. However in the context of the symmetric impulse-variability model this did not reflect internal muscle forces but rather reflected the external joint torques producing movement in an inertial system. This symmetry simply insured movement ended at the end of the decelerative impulse. It was not hypothesized that decelerative impulse variations compensated for accelerative impulse variations. The internal torques produced by agonist and antagonist muscles in fast movements overlap in time because of the twitch characteristics of muscle. Since net joint torque represents the difference between opposing muscle torques, alterations in the timing and amplitude of antagonist torques can influence the variability of the net joint torque pattern during acceleration. Thus consideration of internal muscle forces/torques is necessary in a new impulse-variability model.

In previous impulse-variability models the limb was considered a purely inertial system. It is well known that muscles and, therefore, joints exhibit viscoelastic properties (cf. Cooke, 1979; 1980b; Kelso and
Holt, 1980). Thus these properties of the limb should be included in a new impulse-variability model. Inclusion of elastic properties provides for control of limb posture on the basis of elastic properties of the limb as proposed in final position control models (cf. Polit and Bizzi, 1979). A new impulse-variability model is therefore proposed here which includes many of these characteristics. The formal description of the model is in the Methods section.

As described in the methods section it was assumed in the model that phasic agonist and antagonist bursts produce sinusoidal torque-time curves (impulses). A tonic agonist torque was assumed to maintain final end position according to the limb (joint) stiffness. Use of agonist and antagonist torque curves permitted incorporation of known relationships between phasic agonist and antagonist bursts as related to movement kinematics. Interdependence of agonist and anagonist torques was varied by control of the correlation between peak agonist and antagonist torque amplitude and the correlation between peak agonist torque and onset time of the antagonist torque. Antagonist torques were considered independent of agonist torques when these correlations were near zero. Movements were simulated in the model in order to determine whether the movement characteristics were similar to normal human limb movements and whether the model could account for experimental observations of the present and previous investigations of movement variability.

As the results of experimental simulations indicate, this model is able to explain the observations of a number of previous investigations. The model adequately accounts for a linear speed-accuracy tradeoff for rapid aiming and timing tasks as described by Schmidt et al. (1979). Also the effects of practice can be explained by increased correlation
and/or decreased variability of agonist and antagonist torque curves as observed in the present experiments. The inclusion of viscoelastic properties is also an improvement over previous impulse-variability models. Where the model is inadequate is in the lack of a phasic torque associated with the second agonist burst. Additional inadequacies are the lack of modeling of the effects of muscle contraction/torque production on muscle viscoelastic properties and the role of cocontraction in increasing limb impedance. The role of the second agonist burst is controversial and, thus, was left out of this model. The effects of muscular contraction on viscoelastic properties of muscle have been studied mainly in static situations with constant levels of motor unit activation. The effects of phasic muscle activity on viscoelastic properties has received little study and, therefore, could not be included in this model.

The function of the torque contributed by antagonist muscles was studied by removing it from the model and by manipulating its dependence on the agonist torque. It was shown that the antagonist torque was effective in reducing trajectory and endpoint variability only if a good correlation existed between agonist and antagonist torques. This indicated that one function of the antagonist may be to reduce movement variability by correcting for variations in movements that arise from variability in the agonist. As pointed out earlier in this discussion this would be required for reduction in trajectory variability in association with increased variability in movement-related muscle activity. That is the timing and amplitude of the antagonist burst may be manipulated to correct for deviations from movement intent produced by the variable agonist bursts.
Correction for deviations from movement intent by the antagonist burst are evident in plots of point-to-point trajectory variability versus time throughout movement. Although, as discussed earlier, point-to-point variability rises rapidly during acceleration there is a reduction in the rate of change of variability near the time of peak velocity which was attributed to decelerative forces providing compensation for accelerative force variability. This was also observed in the model for dependent antagonist torques only. Independent antagonist torques did not alter the slope of this relationship until after the time of peak velocity. Rather, under no-antagonist and independent antagonist conditions point-to-point variability continued to rise until the end of the phasic agonist torque. Variability then decreased due to passive viscoelastic and active antagonist torques.

Simulations of "initial" and "practiced" movements in the model showed that strengthening the linkage between agonist (accelerative) and antagonist (decelerative) impulses provides a mechanism for reducing movement variability. In particular these simulations showed that reduced variability in movements in the presence of greater variability in agonist and antagonist impulses could occur only if the linkage between these impulses improved with practice. Thus one would hypothesize that during practice one must set-up appropriate linking between the phasic agonist and antagonist EMG bursts in order to lower trajectory variability when movement speed (and impulse variability) increase.

In conclusion the model presented here provides a major improvement over previous models attempting to explain variability in movements. Separate modeling of the agonist and antagonist torques, instead of the
net torque, allows the incorporation of neurophysiological data regarding the relationships between agonist and antagonist EMG activity in movements of different speeds and amplitudes. The concept of linked muscular activation patterns is a powerful mechanism for reducing movement variability in spite of large variabilities in the associated muscle activities. This model provides a further basis to view movement control as consisting of phasic and tonic components (cf. Ghez, 1979). The phasic components control the trajectory of a movement while the tonic component determines the final movement endpoint. In this way the model is similar to the pulse-step model of movement control, especially for the agonist torque. In the case of the antagonist a pulse-step relationship may also hold. Cocontraction of agonists and antagonists is often observed at movement end in fast movements. This would increase the limb's impedance and prevent large oscillations in fast movements.

The results of the experiments presented in this thesis point to a number of conclusions regarding control of simple limb movements. First it appears that the entire movement trajectory is controlled, rather than simply movement endpoint, velocity or amplitude. Thus, as first postulated by Cooke (1979), the relationship between limb velocity and position throughout movements is of importance to the CNS. There are 3 lines of evidence for this statement: (1) muscular responses evoked by perturbations applied during movements act to return the limb to a prelearned trajectory, rather than simply aiding movement to the intended endpoint, (2) variability in movement trajectories is reduced during practice and (3) deviations from a learned trajectory as a result of naturally occurring variations in movement initiation are corrected in such a way as to return the limb toward a prelearned trajectory. In
Recent theories of movement control have suggested that the motor system may be divided between higher levels which plan ideal trajectories and lower level processes which translate trajectories into muscle forces (e.g., Hogan, 1985). On the basis of results presented here, it may be the phase plane trajectory of simple movements which is planned at higher levels.

The translation of the motor plan into neural commands to the involved muscles is apparently accompanied by variability in motor output which depends on the magnitude of muscle activity required. This results in variability in the EMG activities of muscles involved in the movements. However, the variability is not random in that variations in muscle activity involved in deceleration are linked to variations in muscle activity which initiates limb movement. This is probably necessary because as the nervous system attempts to increase the neural drive to muscles in order to increase the force and velocity of contraction (and the speed of movements) the actual numbers of motor units activated and their firing frequency becomes more variable. This may occur because of greater variability in descending commands or as a result of segmental mechanisms (e.g., excitability of motor neurons). Increased variability in the first agonist burst which accelerates the limb must therefore be compensated by appropriate modifications in subsequent agonist and antagonist muscle activity. Thus, for example, a large agonist burst may be compensated by a large and/or earlier antagonist burst and a large second agonist burst. The finding that a large second agonist burst provided compensation for a large first agonist burst was surprising since one would expect the opposite. However, the large second agonist burst may be necessary to stiffen the limb at the end of movement.
to prevent oscillation and to prevent undershoot because of a large antagonist burst.

At least two possible mechanisms underly control of the antagonist and late agonist bursts on the basis of the first agonist burst. These include efference copy and the use of afferent feedback from the moving limb. In this thesis evidence for use of afferent feedback from muscle receptors in agonist and antagonist muscles was provided. Efference copy may be involved preferentially in very fast movements when there is little time for afferent feedback. Neuroanatomical pathways certainly exist for the use of efference copy since, for example, corticospinal tract neurons send collaterals through the cerebellum. During practice the CNS may program the use of afferent feedback and efference copy pathways to reduce variability in movement trajectories. This could occur through control over the antagonist and second agonist bursts based on prediction of the movement trajectory which would result from the first agonist burst. This implies that a model of the limb exists in the CNS from which these predictions can be made.

Recently it has been suggested that motor cortex neurons are involved in control over movement kinematics in a predictive manner because the probability of firing of these neurons in response to imposed movements depended on movement position and its time derivatives (velocity, acceleration and jerk) (Bedingham and Tatton, 1985). Measurement of derivatives of position allows prediction of the subsequent motion of the limb. This therefore provides a mechanism for early corrections to neural activity controlling movement such that the movement may better reflect intent. As shown in this thesis afferent feedback received during acceleration may be involved in control over
deceleration. Thus predictions about the course of the movement based on afferent feedback received early in the movement may be an important mechanism in control of fast movements.

A second important conclusion regarding the use of afferent feedback in movement control is that feedback received during deceleration may not be used in control of the limb trajectory. Rather it appears that coactivation of agonists and antagonists occurs in response to afferent feedback from muscle receptors during this movement phase. This coactivation would effectively increase limb impedance and thereby reduce deviations from movement intent during this phase. This strategy may be used because there is probably insufficient time to initiate a phasic burst of muscle activity to appropriately modify the limb's trajectory in relation to movement intent.

Overall the findings of this thesis point to a motor programming view recently put forth by Abbs and colleagues for compound movements (Abbs et al., 1984). In this theory the hypothesized motor program consists of general activation patterns for the appropriate muscles followed by adaptive preadjustments in this pattern based on the state of the periphery. Appropriate mechanisms for sensory evaluation of the movement kinematics and the necessary motor responses for appropriate compensatory adjustments would also be set up by the motor program. This is a view similar to that of Brooks (1979b) and in sharp contrast to earlier motor programming theories which postulated a final set of motor commands present prior to movement initiation which would be relatively uninfluenced by sensory feedback (cf. Schmidt, 1975b). Variability in movements under this view would arise from the variability in general activation patterns of the involved muscles. The importance of adaptive
preadjustments in the muscle activation patterns based on the state of the periphery is indicated by the study of movements made from different elbow postures (start angles for movements) as part of this thesis. When movements are made from different elbow angles the motor program must be modified to account for the changes in muscle properties and joint biomechanics. The role of practice may be in the development of appropriate premovement adjustments in the muscle activation patterns and of the use of sensory feedback to control the deceleratory phase of movements. Predictive control over muscle activity involved in deceleration through the use of efference copy of neural commands to muscles which initiate movement may also be developed during practice.
Summary

1. Experiments were carried out to examine relationships between variability of human arm movement trajectories and of EMG activities of related muscles as a function of practice and movement speed. The hypothesis directing these experiments was that the CNS controlled the entire movement trajectory.

2. Variability in both movements and muscle EMGs depended on both movement speed and practice. Increases in movement speed resulted in greater variability in movement trajectories and the EMGs of related muscles. With practice variability in movement trajectories decreased. Changes in variability of muscle activity with practice depended on changes in movement speed. If speed was kept constant during practice variability in the EMGs decreased. However, increases in movement speed during practice were associated with greater variations in muscle EMG activity in spite of decreases in movement variability.

3. The importance of initial limb position in the control of movements of the same amplitude was indicated in studies of movements initiated from different joint angles. Changes in the joint angle from which movements were initiated during practice had striking effects on movement performance. Movement velocity decreased and trajectory variability increased following changes in the start position for movements. Motor commands are therefore posture dependent and reprogramming of such commands is necessary following a change in the joint angle from which
movements are initiated.

4. Study of the time course of the development of trajectory variability throughout movement showed that variability increased at a rapid rate throughout limb acceleration. The rate of rise of variability was positively related to movement speed, indicating that variability in accelerative forces depends on movement speed. Near the time of peak velocity and during deceleration the rate of change of variability decreased and, in many cases, became negative. This indicated that decelerative forces were acting to compensate for the variable accelerative forces.

5. Linked variations in agonist and antagonist muscle EMG patterns accounted for reduced movement trajectory variability in the presence of increased EMG variability during practice. Variations in the first agonist burst which initiates movement produced deviations from an intended trajectory. The agonist variations were accompanied by variations in the antagonist and late agonist burst which returned the limb towards the intended trajectory.

6. One mechanism which might underly control of the antagonist EMG burst involved in braking limb movements is afferent feedback from sensory receptors in the moving limb. This possibility was studied by applying vibration to tendons of muscles involved in the movements. Vibration applied during acceleration resulted in an increased antagonist EMG and subsequent undershoot of the usual endpoint of the movements. Sensory feedback received early in movements may, therefore, be used in
used in predictive control of later phases of movements.

7. A new model of limb movement control was developed in order to explain these results and those of previous investigations. The model included properties of impulse-variability models and of mass-spring model for final position control. Two important features of the model were: (1) separation of phasic and tonic muscle torques and (2) an interdependence or linkage of the phasic torques produced by agonist and antagonist muscles. The linkage of agonist and antagonist torques was shown to be important in reducing variability of fast movements. Refinements in this linkage during practice could explain decreased variability in movements in spite of increased variability in the torque patterns produced by opposing muscles.

8. It was concluded that the entire movement trajectory, not simply movement speed or end-position, is controlled in practiced movements. Implementation of a planned movement trajectory is a variable process, with the variability in activation of individual muscles being a function of desired movement speed. The variability is, however, not random in that there are linked variations in the activation patterns of opposing muscles. This linkage is important in maintaining variability in movements low in spite of large variability in the controlling muscle EMG patterns. Refinements of this linkage in muscular activation patterns occurs during practice and may be based on feedback from the moving limb or, perhaps, efference copy of the first agonist burst which initiates movements.
References


Appendix A

Variability in duration of acceleratory and deceleratory phases of movements during practice.
Variability in durations of acceleration and deceleration throughout practice of 10° and 30° movements performed under the no-load condition. The plotted points are means for 6 subjects of S.D. acceleration duration (circles) and deceleration duration (rectangles). Data for 30° movements are in A and B and for 10° movements in C and D. Closed symbols are for flexions and open symbols are for extensions. The error bars represent 1 S.E.M. for 6 subjects.
Variability in durations of the acceleratory and deceleratory phases of 100° movements performed from flexed, mid-range extended elbow angles. The plotted points are mean S.D.s of acceleration duration (circles) and deceleration duration (rectangles) for 6 subjects. In A are data for extension movements (open symbols) and in B for flexion movements (open symbols). The error bars are 1 S.E.M. for 6 subjects.
Table 16

Variability of acceleration duration and deceleration duration for movements 1-10 and 51-60 performed with a constant torque load.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>msec</td>
<td></td>
<td>msec</td>
</tr>
<tr>
<td>1-10</td>
<td>51-60</td>
<td>1-10</td>
<td>51-60</td>
</tr>
<tr>
<td>10°</td>
<td>19.1</td>
<td>11.5</td>
<td>36.7</td>
</tr>
<tr>
<td></td>
<td>537a</td>
<td>2.6</td>
<td>3.7</td>
</tr>
<tr>
<td>30°</td>
<td>17.2</td>
<td>7.2</td>
<td>29.7</td>
</tr>
<tr>
<td></td>
<td>3.5</td>
<td>0.9</td>
<td>4.4</td>
</tr>
</tbody>
</table>

**Extensions**

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>10°</td>
<td>16.9</td>
<td>10.0</td>
<td>38.7</td>
<td>21.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.1</td>
<td>2.6</td>
<td>8.2</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>30°</td>
<td>16.5</td>
<td>8.9</td>
<td>32.0</td>
<td>20.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4.6</td>
<td>2.4</td>
<td>7.1</td>
<td>2.8</td>
<td></td>
</tr>
</tbody>
</table>

*a standard error of mean for 5 subjects*
Appendix B

Fortran computer program of the digital model of the limb.
This program calculates the position and velocity outputs for agonist and antagonist torque inputs to a linear, time invariant second order system representing the forearm. Physical specifications of the system (stiffness, inertia, damping ratio) are specified by the user. Position and velocity of movements and the input torques are output to disk in getrec format for use with CP3 and MATRIX programs. The user specifies mean agonist and antagonist torque amplitudes and movement start and end positions. The relationship between peak agonist torque and peak antagonist torque and time of onset of the antagonist torque is controlled by the variables antcof and icoeff. Larger numbers specified for these variables result in lower correlations between agonist and antagonist torques. Note that antcof affects the relationships between agonist torque amplitude and antagonist torque timing and amplitude but icoeff affects only the relationship between agonist torque amplitude and onset of the antagonist.

Torque perturbations of any amplitude and timing (onset and duration) may be specified.

---

List of Variables

- **rotini** - rotational inertial forearm and hand about the elbow
- **stiff** - stiffness of the elbow joint
- **dfraco** - damping ratio of the elbow joint
- **dcoeff** - damping coefficient of the elbow joint
- **impos** - movement start position
- **iepos** - movement end position
- **vepos** - range of variability in tonic agonist torque to maintain end position

- **endtor** - tonic agonist torque required to maintain movement end position
- **agpl** - mean peak agonist torque
- **antpl** - mean peak antagonist torque
- **diffpl** - difference between peak agonist and antagonist torques
- **tantl** - mean onset time of antagonist torque
- **agcoeff** - range of variability in peak agonist torque
- **antcoeff** - range of variability in correlation between agonist and antagonist torques
- **ipdur** - mean duration of agonist and antagonist torques
- **locdf** - range of variability in duration of agonist and antagonist torques
- **peramp** - perturbation amplitude
- **perdur** - perturbation duration
- **pertat** - perturbation onset relative to movement onset
- **perfrq** - frequency of sinusoidal torque perturbation (i.e., duration is \(1/2\) period of sin wave)
- **pos** - elbow angle
vel - elbow angular velocity
acc - elbow angular acceleration
sinfrq - frequency of sinusoidal agonist and antagonist torques (i.e., duration is 1/2 period of sin wave)
agasin - amplitude of agonist torque at time i
antasin - amplitude of antagonist torque at time i
pertrq - amplitude of perturbation torque at time i

common /flonl/ flun, frec
integer*2) file(6), pcoeff, mvtamp, pdur, timant, tmpdur
1,sqrt,chan1(1024), chan2(1024), chan3(1024), flun, frec
2,pdrl, tanti, chan4(1024), chan5(1024)
logical*1 cont
data flun/2/

5 write(5,10)
10 format (' Enter damping ratio, stiff., inertia > ',$)
read (5,20) dratio, stiff, rotini
20 format (3(f10.5))

c... calculate damping coefficient

dcoeff = 2.0*dratio*rotini*sqrt(stiff/rotini)
write (5,30)
30 format (' Enter movement start and end positions (deg) > ',$)
read (5,40) ispos, iersos
40 format (2(i3))
write (5,41)
41 format (' Enter 1 to vary movement start positions > ',$)
read (5,42) isvpos
42 format (i3)
write (5,43)
43 format (' Enter var. coeff. for end position > ',$)

c... This allows the user to specify variability in the tonic agonist torque
c... which maintains final position

read (5,44) vepos
format(f6.3)

44 calculate tonic agonist torque required to maintain final position

vepos = float(iepos)/57.3
etor1 = stiff*vepos
freq = sqrt(stiff/rotini)
write (5,70)
70 format (' Enter ag., antag. mean sin amplitudes > ',$)
read (5,81) aspl, antpl
81 format (2(f5.2))

diffpl = apl-antpI
80 format(2f5.2)
    write(5,75)
75 format('Enter coeff. for ag, ant variation > ',$)
    read(5,80) agcoeff,antcoeff
    write(5,85)
85 format('Enter sin duration (msec),coeff(@) > ',$)
    read(5,86) ipdur, idcoeff
86 format(2f15)
    scoeff = float(idcoeff)/100.0
    write(5,90)
    read(5,86) tant1

write(5,107)
107 format('Enter time coefficient for variation > ',$)
    read(5,88) tcoeff
88 format(5i5)
    write(5,95)
95 format('Enter perturbation amp, dur, onset (sec) > ',$)
    read(5,96) peramp, perdur, pertst
96 format(3f5.2)
    perfreq = 6.28*$0.5/perdur
    perend = pertst + perdur
    iperan = int(perend*1000.0)
    ipertes = int(pertst*1000.0)
    write(5,90)
90 format('Enter desired filename > ',$)
    read(5,100) ifile
100 format(6a2)
105 format('Enter ant. pulse onset time (msec) > ',$)
    call fopen(ifile,errcode)
    if (errcode .ne. 0) go to 200

loop through 10 bursts

    call wrtdat(chan1,0,0)
    l = 0
    m = 0
    do 250 int = 1,20
        iflag = 0

Initial conditions

    if (ivpos .eq. 1) ispos=ispos-int/2
    spos = float(ispos)/57.3
    storg = stiff*spos
    edtor = etor1 + ran(l,m)*vepos
    pos = spos
    vel = 0.0
    acc = 0.0

... Calculate duration for phasic torques for this burst
pdur = ipdur-sincoeff*ipdur+2*sincoeff*ipdur*ran(1,m)

sinfreq = 6.28*(0.5/(.001*float(pdur))

imagkl = pdur/2

c... Calculate peak agonist torque for this burst

agp = agpl-agcoef+ran(1,m)*(2.0*agcoef)

xx = 4.0*(ran(1,m)-0.5)

c... Calculate peak antagonist torque for this burst based on agonist torque

c... and antcof

antcp = agp-diffpl+ran(1,m)*xx*antcof

c... Calculate onset time of antagonist based on difference between actual and
c... mean antagonist torques and icoeff.

tinant = tantl+ran(1,m)*icoeff*(antpl-antp)
timd = timant + pdur

c... vary perturbation onset times

iperts = iperts + 20
iperen = iperen + 20

c... pertst = pertst + 0.02
write (5,104) pdur,agp,antp,spos

104 format(1x,i5,i5,1x,f5.2,2(1x,f5.2),lx,i5,1x,f5.2)
do 110 i=0,2048,2

u = float(i)
j = 1+j/2

time = u/1000

atime = time - float(timant)/1000

timpert = time - pertst

agsin = agp*sin(sinfreq*time)+storq

if ((i.ge.imagkl).and.(agsin.le.endtor))

agsin = endtor

antsin = antp*sin(sinfreq*atime)

pertrq = peramp*sin(pertim*perfrq)
if (i.lt.timant) torque = agsin
if (i.ge.timant) torque = agsin-antsin
if ((i.ge.iperts).and.(i.le.iperen))

1 torque = torque + pertrq
if (i.le.pdur) go to 115
if (i.lt.timant) torque = endtor
if (i.ge.timant) torque = endtor-antsin
if ((i.ge.iperts).and.(i.le.iperen))

1 torque = torque + pertrq
if (i.le.timdur) go to 115

torque = endtor

if ((i.ge.iperts).and.(i.le.iperen))

1 torque = torque + pertrq

115 acc = (torque-dcoeff*vel-stiff*pos)/rotini

vel = vel + acc*.002
pos = pos + vel*.002

c assign position, velocity and torque to chnl, chan2 and chan3 with
c values converted from radians to getrec (integer data) with calibrations
c of 60 deg/volt (position), 400 deg/sec/volt (velocity) and 8 Nm/volt
c (torque)

    chnl(j) = nint(pos*57.3*34.13)
    chan2(j) = nint(vel*57.3*5.12)
    chan3(j) = nint(torque*256)
    if (i.le.pdur) chnl(j) = nint(asi*256)
    if (i.gt.pdur) chnl(j) = nint(endtor*256)
    if (i.lt.timant) chan5(j) = 0
    if ((i.ge.timant) and (i.le.timdur))
    chan5(j) = nint(-antsin*256)
    if (i.gt.timdur) chan5(j) = 0

110  continue

c... output data to disk

call wrtdat(chnl,int,1)
call wrtdat(chan2,int,2)
call wrtdat(chan3,int,3)
call wrtdat(chan4,int,4)
call wrtdat(chan5,int,5)
250  continue
    go to 300
140  format (2x,f8.1)
200  write (5,210)
210  format (' error on file open')
300  close(unit=2)
    write (5,220)
220  format (' Continue (y/n)? ',5)
    read (5,230) cont
230  format (al)
    if (cont .eq. 'y') go to 5
    stop
end

c subroutine to write data to disk file in online/getrec format

subroutine wrtdat(idat,int,icn)

common /flcnt1/ flun, frec
integer*2 idat(512),ibuff(256),label(256),flun,frec
if (int.ne. 0) go to 10
   label(19) = 500
   label(20) = 1
   label(36) = 1024
   label(37) = 4
   label(38) = 401
   label(44) = 5
   label(45) = 1
   label(52) = 20
write(/flun'/l, err=500) label
go to 95
10 npts = 1024
iw = 256
j = 1
frec = 29*(int-1) + 4*(icn-1) + 2
do 90 i=1,1024
   if ((iw.gt.0).and.(i.lt.npts)) goto 92
   if (i.gt.npts) goto 93
   write(/flun'/frec, err=500) ibuff
   iw = 256
   j = 1
92 ibuff(j) = idat(i)
goto 94
93 ibuff(j) = idat(npts)
94 iw = iw - 1
   j = j + 1
90 continue
95 return
500 call exit
end

subroutine fopen( fname, ercode )

This routine is called to open an output data file on
the user's default disk, with name and logical unit
specified by the variables in the file control 'flcntl'
common block

integer*2 ercode  
   name of current data file
integer*2 flun, frec  
   rsx-llm i/o error code
data file unit, record numbers

integer*2 fname(6)  
   common /flcntl/ flun, frec

   call asmlun( flun, 'my', 0 )
   call setext( fname, 'dat' )
   open( unit=flun, name=fname, type='new', access='direct',
        form='unformatted', recordsize=128,
        associatevariable = frec, err=10 )

   ercode = 0
   frec = 1
   goto 20

10 continue

set error code to 'file not found'
errcode = -16

20 continue

return
end

set error code to 'file not found'

errcode = -16
frec = 1
.goto 20

20 continue

return
end