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COORDINATION MECHANISMS IN FAST HUMAN MOVEMENT -EXPERIMENTAL AND MODELLING STUDIES

ANNUAL SUMMARY REPORT

WALTER KROLL WILLIAM L. KILMER

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Introduction

" The present study is investigating the basic neuromotor coordination mechanisms involved in a rapid elbow flexion movement, and in developing mathematical models to explain the interaction of these basic neuromotor coordinations with the biomechanical parameters of movement speed. Speed of movement is being assessed in a biomechanics mode via displacement, velocity, acceleration, point of inflection between acceleration and deceleration, and the total time of an elbow flexion movement. Electromyographic analysis techniques are used to monitor the sequential timing of agonist and antagonist muscle activity. The mathematical modelling effort incorporates the biomechanical parameters into an interface with the neurophysiological parameters involving the central and peripheral nervous systems, and then extends the interface to include viscoelastic properties of the muscle, activation delays, and neuronal pools.

The experimental approach includes consideration of the neuromotor coordination mechanisms in both loaded and unloaded elbow flexion movements; changes in control mechanisms due to practice and learning effects; changes in control mechanisms due to local muscular fatigue induced by isometric exercise in the agonist and in the antagonist muscle groups; feasible training and practice regimens involving artificial means of enhancing beneficial changes in control mechanisms; and development of suitable mathematical models to explain in functional terms the ways in which the adaptive mechanisms can account for changes in basic coordination skill and the breakdown of skilled movement patterns due to local muscular fatigue. The planned series of studies incorporates research protocols from at least three usually distinct and isolated areas of research: neurophysiology, biomechanics, and computer science and mathematical modelling.

Summary of Part I:

The first formal investigation was completed December, 1980 and data are presently being analyzed. The first study was designed to secure criterion measure estimates essential for input into preliminary mathematical modelling attempts for an unloaded fast elbow flexion movement. As the early mathematical models developed, it was necessary to revise experimental methodologies and to collect additional criterion measures as dictated by modelling needs. The first stage of mathematical modelling has been completed and is described in Part II of this report.

Beginning in January, 1981 the second formal investigation was begun. Again guided by the insights and requirements generated by the mathematical modelling, the second study is assessing basic neuromotor mechanisms involved in fast elbow flexion: (a) in loaded and unloaded conditions, (b) under conditions of rested and induced fatigue of the agonist <u>or</u> the antagonist muscle groups, (c) a unique condition in which agonist <u>and</u> antagonist muscle groups are, simultaneously fatigued, and (d) in male and female subjects.

Part I-A Completed Work

Both Principal Investigators devoted full time during the summer to the grant by (a) identifying, reviewing, and evaluating recent research publications relevant to theoretical aspects of the grant project, (b) planning the revised research protocols in detailed methodology, and (c) identifying, reviewing and evaluating recent research publications relevant to data analysis and computer programming. Two research assistants were employed during the month of August to expedite these undertakings. Preliminary orders for necessary expendable supplies were sent out.

In September additional graduate research assistants (RA's) were recruited. In Dr. Kilmer's case the RA continued on with computer programming for the mathematical modelling aspects of the project. In Dr. Kroll's case new RA's were indoctrinated into research data collection protocols. The indoctrination phase included practice in apparatus usage, maintenance, and calibration techniques as well as pilot testing research measurement schedules to insure reliability of data collection. The indoctrination phase lasted six weeks resulting in highly proficient research assistants capable of collecting reliable data.

Beginning the end of October, data collection for the first project began. The first study was designed to secure essential criterion measure necessary for input into preliminary mathematical models. These criterion measures include:

...maximum isometric strength of elbow flexion and extension

... rate of tension development

- ...integrated electromyographic activity at maximal and sub-maximal tensions and joint angles
- ... agonist-antagonist contraction patterns during unloaded speedof-movement elbow flexion

... offects of local muscular fatigue upon neuromotor coordination

Preliminary mathematical modelling attempts quickly showed that additional criterion measures would be necessary. As a result of the modelling requirements, upper limb volumes and biceps and triceps reflex time and rate of muscle tension development were collected. Limb volumes are being estimated by anthropometric and water displacement techniques. The research assistant has been trained in these techniques by Professor Frank Katch of the Department of Exercise Science who is a recognized researcher in the body composition area. A MEDLARS literature search revealed only a few scattered articles dealing with upper limb reflex times with none of the data appropriate for modelling requirments. Suitable apparatus was designed and built for upper human limb reflex testing. Limb volumes and reflex times have been collected on all subjects tested to date. At this time 12 subjects have fully completed the first phase of data collection and the results of being analyzed.

In this first study of Phase I, each of the 12 subjects was tested over ten experimental sessions with each session taking 90-120 minutes. Eight of these sessions involved speed of movement testing while the other two sessions involved upper limb reflex and volume assessments. Each of the first four experimental days included 50 trials of rapid elbow flexion speed of movement to a designated target. Previous research has shown that this amount of practice insures that all subjects become well-practiced and exhibit consistent, stabilized performance both in the speed of movement as well as in the neuromotor coordination mechanisms. Following the establishment of well-practiced performance, local muscular fatigue was induced by two different effort/rest exercise regimens. A 5-second maximal isometric contraction

with a 5-second intertrial rest period--designated the 5:5 condition--for a total of 30 serial trials constituted the more intense fatigue series. A 5-second maximal isometric contraction with a 10-second intertrial rest period--designated the 5:10 condition--for a total of 30 serial trials constituted the less intense exercise regimen.

These two exercise regimens--the 5:5 and the 5:10 conditions--were designated to produce different levels of fatigue in the involved muscle group. The 5:5 and the 5:10 exercise regimens were administered on separate occasions to the agonist (elbow flexors) and to the antagonist (elbow extensors) muscle groups using a balanced order of presentation over subjects and across test sessions to minimize contaminating test order effects.

One of the purposes of inducing high and low intensity local muscular fatigue in the agonist or antagonist muscle groups was to ascertain the role of changes in the peripheral muscle state and in peripheral muscle afferent feedback to higher nervous system centers. Specifically, the question of whether or not programmed central commands for a fast ballistic movement can be altered by the presence of different levels of muscular fatigue.

Currently data are being collected in Phase II of the grant proposal (see pages 10-11 of original proposal). In this investigation the same testing schedule outlined for the Phase I study is being replicated for elbow flexion speed of movement. However, to further elucidate the basic neuromotor coordination mechanisms a load is imposed upon the limb movement task. The imposition of a load, of course, greatly affects the muscle activation time and sequential firing of involved muscle groups and constitutes a more complex movement task with distinctly more complex nervous system control system involvement.

In order to insure identical loading for subjects with different limb lengths, an inertial loading technique is being used. The natural moment of inertia can be altered by placement of light weights at long distances and heavy weights at short distances from the fulcrum point. The protocol being employed utilizes a fixed weight for all subjects but varies the distance from the fulcrum point to comply with the condition of imposed moment of inertia. One load condition being used is equal to two times the natural moment of inertial while the second load condition is equal to five times the natural moment of inertia.

In addition to the two different exercise regimens of 5:5 and 5:10 administered to the agonist and to the antagonist muscle groups on separate occasions, another set of exercise regimens are also being administered. Based upon mathematical modelling considerations, it is of interest to ascertain the effect of local muscular fatigue in <u>both</u> the agonist <u>and</u> antagonist muscle groups simultaneously upon basic neuromotor coordination mechanisms. To accomplish such a goal of producing fatigue in both agonist and antagonist muscle groups, a flexion-to-extension contraction sequence was designed.

By using a 5-second contraction of the elbow flexors followed immediately by a 5-second contraction of the elbow extensors with no intertrial rest period, fatigue effects equivalent to 5-second contraction and 5-second rest periods can be produced in both the agonist and antagonist muscle groups. Similarly, if the effort sequence is a 5-second contraction of the elbow flexors followed by a 5-second contraction of the elbow extensors followed by a 5-second rest period, the equivalent fatigue effects of a 5-second contraction, 10-second rest period exercise regimen can be produced in both the agonist and antagonist muscle groups simultaneously.

These two new exercise regimens, designated the 5/5:0 and the 5/5:5, will allow assessment of fatigue effects in the agonist <u>and</u> the antagonist muscle groups upon basic neuromotor coordination mechanisms and provide a stringent test of the predictive power of the mathematical model being developed. In actuality the two new exercise regimens will produce slightly more fatigue than the 5:5 and the 5:10 regimens upon which they were based because some degree of co-contraction of agonist and antagonist muscle groups occurs in maximal isometric contractions. Such co-contraction effects, however, are desirable since they will heighten the local muscular fatigue produced.

The Phase II study described above will involve 12 male and 12 female subjects with each subject attending 12 test sessions of 90-120 minutes each. Ten of these sessions involve speed of movement and exercise conditions while the other two sessions are for upper limb reflex and volume testing. One of the research assistants (Julma C. Garcia) will be using some of the data for her Ph.D. dissertation in exercise science under the chairmanship of PI Kroll. It is anticipated that data collection will be completed by May, 1981 with subsequent reduction and analysis of data taking place over the summer months. A Master's thesis is currently being completed which is a pilot study effort dealing with the effects of vibration upon speed of movement and neuromotor coordination mechanisms. This graduate student, Marilyn Teves, was not a research assistant but her work was supported in part by purchase of essential expendable supplies.

PART II

Modelling

Summary of Part II:

We describe a two-compartment model of the neuromuscular system involved in the voluntary fast arm movement to a target discussed by Kroll in Part I. The first concartment accepts averaged biceps and triceps ENG signals as inputs, and models the arm's musculo-skeletal response by producing elbow angular position $\phi(t)$ and velocity $\dot{\phi}(t)$ over the corresponding movement time. This is called our $E/Q/\dot{\phi}(t)$ model, and its defining equation is of the form

 $\dot{\phi}(t) = (extensor torque = Q_{E}) - (flexor torque = Q_{F}).$

A detailed version of this equation is being computer-simulated with the PASCAL programme listed in Appendix A.

Our second compartment, called our $\underline{com/cont/E \mod l}$, accepts volitional commands as inputs, and models the nervous system's response by producing smoothed flexor and extensor ENG signals to feed into the E/Q/2 model. Fig. II-4 gives a schematic representation of the com/cont/E model, which is explained in detail in the report.

The purpose of our two models is to separate the various control functions in the ballistic arm-movement system well enough to understand where adaptation occurs as speed, precision, and coordination of movement improve with practice.

This is the first publication that has arisen from work on this contract.

II-A Completed Work

Since last June we nave completed an initial specification of two models that will help us to better understand and guide the direction of Kroll's experiments. The <u>first model</u> accepts smoothed EMG inputs to flexor and extensor muscles and calculates arm angle dynamics for the fast arm movement described above by Kroll. This model is denoted the E/O/s(t) model (short for EMG/elbow torque = Q/elbow angular velocity dQ/dt = $\frac{1}{2}$). The <u>second model</u> feeds flexor and extensor smoothed EMG outputs into the first model, and receives its input commands from a volitional fast arm movement center in the brain, presumably via the pyramidal tract from the cerebellum to the brainstem and spinal cord (cf. Miles and Evarts, 1979). This model is denoted the <u>Com/Cont/E model</u> (short for command/neural control/EMG). The appropriate connection of our two models provides a complete overall model for the fast arm movement as griven by volitional commands.

Below we describe the $E/Q/\Rightarrow$ model first and the com/cont/E model second. Section II-B then proposes further studies for next year.

1) The $E/Q/\phi$ model

This model arose out of a desire to formulate a quantitative account of the E/Q/s part of the arm movement to target described by Kroll. After reviewing the relevant literature, we rejected possible model formulations at the molecular biophysical level as too complex (cf. Hatze, 1978, 1980, and Dijkatra et al., 1973a, 1973b). In addition, for our movement, they would be too difficult to specify quantitatively. We also rejected model formulations at the spring-mass postural level (cf. Sakitt, 1980) because they would be too steady-state oriented to allow an analysis of our large transient arm motion. Other control engineering models (cf., e.g., McRuer) seemed too general or too coarse-grained for our purpose.

Figure II-1 gives a schematic representation of the framework for the $E/Q/\phi$ model we chose. Its corresponding <u>top-level analysis formula</u> is

$$d\mathbf{\ddot{s}} = \mathbf{Q}_{\mathsf{E}} - \mathbf{Q}_{\mathsf{F}} , \qquad (1)$$

where each dot over \Rightarrow indicates a time derivative, and Q_E and Q_F are the respective torques about the elbow as exerted by the extensor and flexor groups of muscles (all the muscles in each group operate in approximate temporal and tensional unison throughout our movement (Lagasse, 1975)). J is the arm's moment of inertia for rotations about the fixed elbow joint as shown in Fig. II-1 (over a \Rightarrow change from 60° to about -30°).

At the second level of analytical refinement, we let

$$Q_{\rm E} = \Psi_{\rm VE} + \Psi_{\rm IE} + \zeta_{\rm E} \tag{2}$$

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where $\varsigma_{\rm E}$ is the torque due to viscoelastic braking of the movement (Lestienne, 1979), and $\psi_{\rm IE}$ is the maximum (over -90° \leq Q (90°) isometric torque produceable by the extensor, given the smoothed extensor EVG envelope.



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Figure II-1. Schematic for the framework of E/Q/5 model. See text for explanation.

 EMG_E , recorded over the last second (250 ms, or several seconds would serve as well, but we need a standard). To find Ψ_{IE} , we accept the claim of Messier, et al., 1971, that under static loads, "the averaged EMG is directly proportional to muscle tension and the constant of proportionality is independent of muscle length." Admittedly, the force properties of muscles undergoing rapid active or passive stretch or compression are very different than when these muscles are being actively or passively subjected to static loads. But from what little is known, the best we can do to account for this is to include the Ψ_{VE} factor appearing in (2). Ψ_{VE} is a fraction between 0 and 1 that inserts the force-velocity relationship suggested by Thorstensson et al., 1976, and Perrine and Edgerton, 1978, for the human knee, and by Lagasse's 1975 observations on forearm accelerations during maximally fast movements (quite like our experimental ones only untargeted).

Our
$$\Psi_{VE}$$
 function is

$$\Psi_{VE}(\dot{\phi}) = \begin{bmatrix} 1 & -\frac{\dot{\phi}}{1000} \end{bmatrix}$$
(3)

where ***** is in degrees/sec, and the double-cusped brackets mean

$$\begin{bmatrix} x \end{bmatrix} = \begin{cases} x & \text{if } x \ge 0 \\ 0 & \text{otherwise} \end{cases}$$

For the flexor side we let

$$Q_{\rm F} = \Psi_{\rm VF} + \Psi_{\rm IF} \tag{4}$$

where ψ_{IF} is similar to ψ_{IE} and $\psi_{VF} = \psi_{VE}$. Equating ψ_{VE} to ψ_{VF} for our movement is inaccurate to some degree because the flexor undergoes active contraction and/or passive compression whereas the extensor undergoes passive or active stretch (actually, the joint must be stabilized so there are no purely passive muscle states, only approximate ones).

Our method, then, reduces to choosing functions in (2) that best fit the averaged data before, during, and after enough trials by each subject (hundreds!) to enable him to achieve maximum speed. The resulting functions should tell us some valuable quantitative things about the muscle properties involved.

At the last level of analytical refinement, we let

$$\Psi_{IF}(t) = A_{F}\cos\varphi(t) \int_{t-\mu-\xi}^{t-\mu} k_{F}\overline{EMG}_{F}(\tau)h_{F}(t-\tau) d\tau, \qquad (5)$$

where $A_F \cos \phi(t)$ converts flexor force to torque according to the geometry of Fig. II-1; $\overline{EMG}_F(\tau)$ is the smoothed EMG_F envelope at time τ ; k_F is a constant of proportionality that we assume converts $EMG_F(\tau)$ into flexor force at τ (Messier, et al., 1971); and $h_F(t-\tau)$ is the normalized flexor twitch response at time t to a unit $k_F \overline{EMG}_F$ impulse that arrived at time τ .

Our $h_F(t-\tau)$ is defined in Fig. II-2, where we equate $h_F(t-\tau)$ with its extensor counterpart (for want of knowledge to do otherwise. This could be incorrect by perhaps 10% or so, especially after many practice trials). The shape of our h function is a ξ -truncation (for economy in computing) of Dijkstra et al.'s 1973a) twitch response function for biceps brachii muscle. But whereas their $\beta_1 = .015$ and $\beta_2 = .15$, we began with $\beta_1 = .05$ and $\beta_2 = .3$. As we continue to fit the incoming data, our 3's will doubtless change in the direction of Dijkstra et al.'s.

Equation (5) assumes linearity in the accumulation of twitch responses. This is probably somewhat incorrect for our movement. Yet Sakitt, 1980, makes the same linearity assumption for an arm movement similar to ours and



Figure II-2. Definition of $n_{f}(t-\tau) = h_{f}(t-\tau)$ in equation (5). See text for explanation.

obtains good results. In the future, however, we plan to try letting $h(t-\tau)$ vary as a function of

$$\int_{t-\tau-v}^{t-\tau} \frac{EMG(\sigma)e^{-\alpha(t-\tau-\sigma)}}{d\sigma} d\sigma, \qquad (6)$$

the exponentially-weighted $\overline{\text{EMG}}$ input from $t-\tau$ back to $t-\tau-\nu$ in time. This seems like the most expeditious way to introduce an appropriate nonlinearity for the accumulation of twitch responses into equation (5).

Letting $A_F k_F = C_F$ in (5) we get

$$\Psi_{IF}(t) = C_F \cos\phi(t) \int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_F(\tau)h_F(t-\tau)d\tau$$
(7)

Defining ψ_{IE} similarly and neglecting c_E in (2) (as does Sakitt, 1981. cf. also Lestienne, 1979), our final refined version of equation (1) becomes

$$\ddot{\phi}(t) = \psi_{V} \cos \phi(t) \left[\int_{-\frac{1}{2}}^{\frac{C_{E}}{2}} \int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_{E}(\tau)h_{E}(t-\tau)d\tau - \int_{t-\mu-\xi}^{\frac{C_{F}}{2}} \int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_{F}(\tau)h_{F}(t-\tau)d\tau \right]$$
(8)

with the constraining assumptions given above. To a first approximation we can obtain C_F and C_F in (8) by replacing J_{ϕ}^{ϕ} by the isometric torque found

first in the flexor and then in the extensor directions for each subject. There are various good ways of measuring each subject's J (e.g., Hatze, 1980, refers to one he has developed), but we have not yet selected one.

We now discuss how the $\overline{\text{EMG}}$ signals in (8) are obtained. Figure II-3 shows an example test record. The EMG_{av} signals are rectified and filtered raw EMG signals. EMG_{av} signals are derived in an on-line computer which "continuously" leaks off a fraction of the present integral of the rectified EMG signal. Denoting the latter EMG_{r} , $\widehat{\text{EMG}}$ in Figure II-3, to a good approximation, is given by

$$\widehat{EMG}(t) = \int_{0}^{\infty} EMG_{r}(\tau)e^{-\alpha(t-\tau)}d\tau \qquad (9)$$

Thus if $EMG_r(\tau) = K$, a constant, then

$$\widehat{EMG}(t) = \frac{K}{\alpha} \left[1 - e^{-\alpha t}\right] + \frac{K}{\alpha}$$
(10)

as t gets large. Also, if $EMG_r(\tau) = C\tau$, C a constant, then

$$\widehat{EMG}(t) = \frac{C}{\alpha}t - \frac{C}{\alpha^2}[1 - e^{-\alpha t}] + \frac{K}{\alpha}t$$
(11)

for small t. Finally, it is easily shown that if $EMG_r = K$ as in (10) for a long time and then EMG_r becomes 0 at t,

$$\frac{d \ \widehat{EMG}(t+\varepsilon)}{d\varepsilon} = - \ Ke^{-\alpha\varepsilon} + -K$$
(12)

for small ϵ . Comparing (10) and (12) gives us a way to determine α from appropriate data samples.

Equations (10) - (12) enable us to find easily the $\overline{EMG}(t)$ quantities in (8) from $\widehat{EMG}(t)$ records such as shown in Fig. II-3. For example, given the



Figure II-3. An example test record. The EMG $_{\rm av}$ signals are marked to obtain $\overline{\rm CMG}$ signals as explained in the test.

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 \widehat{EMG}_F signal in Fig. II-3, we form a straight-line-segmental approximation to it by connecting a to b, b to c, etc. If in this approximation, a segment is essentially constant at K, we let \widehat{EMG} there equal Ka. If the segment rises with slope C > 0, we let \widehat{EMG} rise correspondingly with slope Ca. If the segment falls from K with slope -K, we let \widehat{EMG} there equal 0. Thus our $\widehat{EMG}(t)$ equals a times the straight-line-segmental approximation to \widehat{EMG} except where that approximation falls off. The falloff intervals are easy to handle because the \widehat{EMG} signals for our movement are either on at high intensity or nearly off over most of every trial.

There is, however, one adjustment to the $\overline{\mbox{MG}}(t)$ signal derived above that we need to make. In order to compensate for $\overline{\mbox{EMG}}$ inaccuracies during EMG falloff phases and intrinsic EMG distortions of nervous excitation to muscle at low signal levels, we shall augment our $\overline{\mbox{EMG}}$ signal with a $\mbox{(t)}$ postural feedback term. This is justified by assumption 4 in the next section.

To make the adjustment, we denote the previous \overline{EMG} by \overline{EMG}^* , and add a feedback term denoted \overline{EMG}^{**} . This gives us

$$\overline{EMG}(t) = \overline{EMG}^{*}(t) + \overline{EMG}^{**}(t)$$
(13)

on both the flexor and extensor sides. We define:

$$\overline{EMG}_{F}^{**}(t) = P_{F1} \left[\phi(t-20) + P_{F2} \phi(t-20) + P_{F3} \phi(t-20) \right] G(t)$$
(14)

where the double-cusped brackets mean the same as in (4); where the quantity inside these brackets is $\phi(t)$ as predicted from ϕ , $\dot{\phi}$, and $\ddot{\phi}$ 20 ms earlier (20 ms being the total delay around the loop from γ receptor to a stimulation of muscle); where G(t) = 0 until ϕ first reaches 1 degrees, and G(t) = 1 thereafter; and where the P_{Fi} are constants (now set at P_{F1} = .25, P_{F2} = 10, P_{F3} = 100 -- recall that $\dot{\phi}$ is in degrees/ms and $\ddot{\phi}$ is in degrees/ms²). Similarly,

$$\overline{EMG}_{E}^{**}(t) = P_{E1} \left[-\left(\phi(t-20) + P_{E2} \phi(t-20) + P_{E3} \phi(t-20) \right) \right] G(t)$$
(15)

where $P_{Ei} = P_{Fi}$.

<u>Finally</u>, to keep (14) and (15) from causing everlasting ϕ oscillations, we change (1) to

$$J\ddot{\phi}(t) = Q_{E}(t) - Q_{F}(t) - C_{c}\dot{\phi}(t)$$
 (16)

where the C_{ζ} viscoelastic damping factor (cf. Lestienne, 1979) is set "near" the level that just prevents a second \Rightarrow overshoot of the O degree target position.

We now consider adaptation.

As our subjects accumulate more experience, we know from past results (Lagasse, 1975, e.g.) that their movement times will decrease. If the adaptations enabling this occur partly in the muscles, this should require us to modify the h functions in (8). Since in previous pilot experiments our flexor movement adapted several times faster and further than the opposite extensor movement, it could happen that only h_F in (8) adapts with practice. It also might develop that all adaptation occurs in the nervous system (cf. the com/cont/E model, next Section).

<u>The crucial feature of our $E/Q/\phi$ model</u> is that eventually it should locate and characterize the adaptive nonlinear effects involved when speed of movement is increased with practice. <u>This should give us a mechanical</u> <u>perspective on how to train for speed, precision, grace and skill--in</u> <u>short, for well coordinated fitness</u>.

We have included as Appendix A a listing of the PASCAL programme written by Valerie Congdon to compute $\dot{\phi}(t)$ and $\phi(t)$, as outlined above, using a file containing the $\overline{\text{EMG}}_{\text{F}}(t)$ and $\overline{\text{EMG}}_{\text{E}}(t)$ data for one movement. The program is well documented, hierarchically organized into procedural and functional levels identical to the analysis levels in this report, and specified so as to be easy to change. Ms. Congdon, who will leave UMass in January, 1981, has done an exemplary job.

Personnel Change (for Kilmer)

By the end of February 1981 the modelling project's RA will change from Ms. V. Congdon, who is leaving school to accept a job, to R. Pelosi, a Ph.D. student in the IE/Operations Research Department. Ms. Congdon will complete the EMG-torque-movement simulation programme before leaving. Her work has been of the highest professional caliber (cf. Appendix A, a listing of our PASCAL simulation programme). Mr. Pelosi will need much of the spring term after March 1 to train for the continuation of Congdon's work.

2. The Com/Cont/E Model

This model concerns the functional organization of the neural control system for the $E/Q/\phi$ model described above. It is an attempt to make precise only those conclusions that have been formulated by several experimenters as necessary consequences of their tests on fast arm movements involving the elbow and sometimes one other joint. The com/cont/E model is amenable to few significant neuroanatomical interpretations. Eventually we hope to obtain a good structural model that does not suffer from this defect, but that may be a couple of years away.

Our specification of the com/cont/E model arose out of the following Assumptions

- For about the first 100 ms at least, our fast arm movement is entirely feedforward (is independent of any feedback), and thus is unaffected by stretch reflex or tendon afferents (cf. Wadman, et al., 1979; Desmedt and Godaux, 1978).
- 2. Selecting the levels L_F and L_E of flexor and extensor EMG activity require the first volitional decisions. (As noted by Kroll above, all of the flexor muscles act approximately in temporal and tensional unison with the biceps during our arm movement. Likewise for the triceps and the extensor muscles.) These levels are selected to set the speed and amplitude, M, of the movement with allowances for expected inertial loading of the limb and estimated fatigue of the muscles being activated (cf. Lestienne, et al., 1979; Lestienne, 1979; Wadman, et al., 1979; Wadman, et al., 1980).
- 3. The next movement decision is to set the relative timing of the first flexor and extensor EMG bursts. This determines the movement's combina-

tion of magnitude and speed, again allowing for the expected loading and muscle fatigue (cf. references in 2 above). Force-velocity and force-length effects are probably also included in this reckoning (Thorstensson, et al., 1976; Perrine and Edgerton, 1978), as well as viscoelastic braking (Lestienne, 1979; Maughan and Godt, 1979).

- 4. The flexor and extensor muscle tension levels at the end of the movement epoch when the postural maintenance phase is entered are variable, but the ratio R between these two levels, (as reflected by their leaky-integrated EMG levels) is a fixed postural command, and is not affected by the starting point, direction, amplitude, or speed of movement (Lestienne, et al., 1979; Sakitt, 1980). Presumably the EMG levels themselves at the end of the movement epoch are quickly adjusted if need be (how fast?) to obtain the desired postural stretch reflex stiffness (Houk, 1976). Since the biceps tendon reflex loop delay is about 35 ms, the stretch reflex loop delay is about 50 ms (Marsden, et al., 1976), and our actual arm movement duration is about 300 ms, the presently unknown speed with which the set point of the stretch reflex is reset and the reflex subsequently engaged will be imortant to discover.
- 5. In males at least, continued practice of a fast arm movement reduces execution time and alters the movement's EMG signature (Lagasse, 1975).

In the light of the above assumptions, we now give a point-by-point description of the com/cont/E model (Figure II-4):

 At the top of Figure II-4, R, L, M, ILF, and FF are commands issued from the volitional decision center for our fast arm movement (this "center" might be distributed over a large part of the brain). R is the ratio described in assumption 4 above; L times g_F, L.g_F, and L.g_c are the



levels L_F and L_E described in 2 above; M is defined in 2; ILF, the inertial loading factor, and FF, the fatigue factor, referred to in 2 and 3 are appropriately scaled for multiplication into the subject's moment of inertia, J, for the arm movement in question. Thus ILF ≥ 1 , with IFL = 1 when no external load is added; and $0 < FF \leq 1$, with FF = 0 interpreted as complete paralysis and FF = 1 as zero fatigue (defined in terms of force development capability).

- 2. N_F and N_E are loosely interpretable respectively as flexor and extensor motorneuronal pools that drive either the flexor and extensor arm musculature for our movement or just the biceps and triceps.
- 3. At t = 0 the flexor EMG is activated. t* is the time at the end of the first extensor EMG burst. The time courses of R and L with respect to t* are hypothesized as shown at the top of the figure.
- 4. At t = 0, L turns N_R on, which then turns N_F on (see 6 below), all with delay less than de. N_f 's output firing rate rises and falls somewhat sporadically, but peaks at the L.g_F level. The stretch reflex pathway, SP_F , into N_F is intially inactive as noted in assumption 1 above. z_F 's output equals the algebraic sum of its inputs. N_F keeps N_E shut off over the reciprocal inhibitory pathway.
- 5. At time D, a shot out of N_D turns N_F off to end the first flexor EMG burst. c and k in the formula for D are scaling constants. Since L turns N_D on, the value of c would be O if the flexor muscle M_F were perfectly linear in L and if no co-contraction of M_F and M_E were necessary to stabilize the elbow joint during movement. Since neither of these conditions holds, c > 0. After N_F shuts off, N_E is no longer inhibited and turns on. Then N_E keeps N_F shut off by reciprocal inhibition.

- 6. Backtracking a bit, only one of N_R 's outputs is on at a time. Initially, L turns N_R 's z_F output on, which is necessary to keep N_F on. Later N_D 's output shot switches N_R 's output over to z_E , which is necessary to keep N_F on.
- 7. N_R 's input T_F provides N_R with an estimate of M_F 's tension, as computed (or simulated) by Nh_F . The function h_F is Nh_F 's estimate of M_F 's twitch response function h_F (see Part II-A-1) on the E/Q/* model). In contrast with the nervous system, we would model Nh_F 's output at time t with

 $N_{F}(o)\hat{h}_{F}(t-\sigma)d\sigma$. By assumption 4 above, the flexor tendon organ

response, TOR_{F} , is effectively unavailable to Nh_{F} for at least 30-85 ms. Mutatis mutandis, N_{P} 's extensor circuits are the same as its flexor ones.

- 8. If our movement is entirely feedforward, some such centers as Nh_F and Nh_E are necessary to control N_F and N_E 's development of an appropriately stiff arm posture at the end of the movement (assumption 4 above). The possibility that there are Nh_F and Nh_E circuits in the carebellum seems to fit Eccles' concept of the cerebellum as a control reference in fast voluntary movements (Eccles, 1977). Lagasse, 1975's results showing that EMG temporal organizations for fast human limb movements don't change with fatigue support the idea that such movements are entirely feedforward.
- 9. When N_R 's \hat{T}_E input reaches the desired final level, N_R 's output switches from \hat{z}_E to \hat{z}_F in case \hat{T}_F is not up to the desired final level, and to \hat{z}_C in case $\hat{\tau}$ is up to its final level. An active \hat{z}_C signals the end of the movement and subsequent reinstatement of the normal postural mainte-

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nance mechanisms. The desired final flexor and extensor tensions are denoted \overline{T}_F and \overline{T}_E , and are assumed to satisfy $\overline{T}_E R = \overline{T}_F = L$ (assumption 4 above). Thus $(\overline{T}_F/\overline{T}_E) = R$, and the N_R component in Fig. II-4 has sufficient inputs to compute all of its outputs.

- 10. If we assume some background SP_F , SP_E , and other noise, the sequence of events in 9 will not always be as simple as described. The first N_F or N_E EMG burst might sometimes be cut off prematurely, forcing another one or even two small bursts later on (see the L time course, top right in Figure II-4). The famous third EMG burst (e.g., Wadman, et al., 1980) might be accounted for in this way -- an attractive hypothesis because Kroll's experiments to date with highly practiced subjects usually don't show a third EMG burst. The ability of the control components in Fig II-4 to learn (with lots of practice) the amount of viscoelastic braking that actually occurs, the force-velocity and force-length relationships of the muscles, and various nonlinearities of the system, offer other adaptation possibilities.
- The → symbol in Fig. II-4 points to likely adaptation sites as the same movement is practiced over successive trials and days.
- 12. N_D and the (N_D, Nh_F, Nh_E) complex could be combined instead of functionally partitioned as shown, but that would defeat the purpose of the com/cont/E model.

II-B Proposed Modelling Studies Next Year

We wish to proceed on three fronts:

 The E/Q/\$\phi\$ model will be adjusted, and perhaps revised or augmented, to fit Kroll's experimental results. His tests with different loads and fatigue levels should be very helpful to us during this phase.

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- 2. The Com/Cont/E model will be scrutinized. In particular, the N_D and N_h components will be checked for plausibility against Kroll's fatigue and load variational test data. We now believe that "efferent-copy" M_F simulators such as the N_h functions actually exist (perhaps at the highly integrative thalamic level atop the brain stem reticular formation, or in the cerebellum), but we shall seek more evidence to support this idea. We also plan pilot tests of various kinds to further elucidate the role of stretch reflexes at different times during and immediately following fast arm movements. To date, Wadman et al. (1979) have shown that when a subject's planned fast arm movement is unexpectedly blocked, the pattern of EMG activity over at least the first 100 ms is the same as before blocking. But we know of nothing else in the literature on the dynamics of rapidly changing the set points of stretch reflexes.
- 3. We shall develop computer programs in PASCAL for solving the nonlinear delay-differential equations that will arise when the E/Q/* model is augmented to include feedback stretch reflex information. Banks (1979, 1980) has recently developed the requisite mathematical methods, but as yet PASCAL programs to implement them do not exist.

In 1 to 3 above, we shall be especially watchful of adaptation parameters and characteristics. In year three of the contract, we shall apply the results of the first two years to a search for better ways to measure and train optimally coordinated fast human limb movements, with an emphasis on kinesthetic learning.

V Note on RA Budget for Kilmer

UMass now has no limits on what RAs can make. The going rate for a full RA to a grad student in the Engineering School is at least 6000 per year, and for Ph.D. students this will probably soar to over 10,000 per year within the next year (due to the new UMass-Industry Program headed by the Dean of Engineering). We will try to hold the line, however, at the originally proposed 5700 per year.

Personnel Change (for Kilmer)

By the end of February 1981 the modelling project's RA will change from Ms. V. Congdon, who is leaving school to accept a job, to R. Pelosi, a Ph.D. student in the IE/Operations Research Department. Ms. Congdon will complete the EMG-torque-movement simulation programme before leaving. Her work has been of the highest professional caliber (cf. Appendix A, a listing of our PASCAL simulation programme). Mr. Pelosi will need much of the spring term after March 1 to train for the continuation of Congdon's work.

APPENDIX A

Contents:

- An example test record of a subject whose responses were modelled.
- 2) Model output from the computer when the example test records EMG traces in 1) were used to derive the input to the model.
- 3) Computer plot of φ in 2) versus time. Drawn in for comparison is φ of 1).
- 4) Documentation for and listing of the PASCAL program that produced the output of 2).

NOTE BENE:

The text of the report was prepared before the computer run for this appendix was made. Some late changes in the PASCAL program are NOT incorporated into the report. They include the new values of the model parameters given in the header of 2), where P_1 , P_2 , and P_3 are the P parameters of equations (14) and (15). Also, C_5 was changed to equal C_{51} until ϕ 's first return to 0 degrees after its initial overshoot of the target position, and to equal C_{52} after that. This causes ϕ to be heavily damped upon its first return to target as required by the test data. Our computer studies showed that with the long delays in the stretch reflex loop, nonlinearities in the nerve-to-muscle pathways present or not, the required damping could only come from a large increase in viccelastic damping. Thus we are postulating a new mechanism on the basis of our model simulation results.



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CUTPUT FROM PLOT : EMGE (t) and EMG (t)

MODEL : E / G / PHIDOT (EMG / TORQUE / ANGULAR VELOCITY)

E : EXTENSOR (TRICEPS) F : FLEXOR (BICEPS) 1 : INDICATES THE 1.0 LINE 0 : INDICATES THE 0.0 LINE



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FAST MOVEMENT PROJECT								3:		
UNIVERSITY OF MASSACHUSETTS/AMMERST PRINCIPLE INVESTIGATORS I DR. WILLIAM KILMER AND DR. WALTER KROLL Research Assistants I Valirie Concoon and Jean Boucher										
MODEL	: E/:	3 / PHID	DT (EM	G / TOPOUS	E / ANGULA	R VELCCI	тү)			
MODEL	PARAMETI TRAPEZO	ERS :	STEP SIZ	Ę_: <u>1</u> .2	DELAY :	20.00 000 22	: 0.000	P3 : 0.0	200	
EXTENSOR FLEXOR	A 0.10 (0.10 (X BETA 0.40 0.1 0.35 0.1	1 BETA2 0 0.15 0 0.15	XI MU 40.00 SO. 40.00 SO.	G(AITCH 0 0.31575 0 0.31575			. 0.050		
SUBJE	CT ID :	DATE 11/14/	SL3JE 80 1	ET AM/PM 0	SESSION 4	BLOCX	TRIAL 15			
TIME (MSEC)	EMG (BICEP)	ENG (TRICEP)	PHI (PC3)	PHI (VEL)	PHI (ACC)	PSIV	PSIIE	GE	PSIIF	GF
										0.0000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.00000 0.000000

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NOTE:

NOTE: Allowing for start-time uncertainty because of unknown initial background activity in the subject, and noting that the relative timing and snapes of the two PHI curves above can be matched almost exactly by introducing nonlinearities into the n(t) functions in the integrals of the PHIDDOT equation, the two PHI curves above compare very well. Note especially two things: PI=PC=FC=D, so the stretch reflex is not ensaged; also, the effect of the heavy viscoelastic damping first appears in both PHI curves upon their return to zero degrees after their initial cysthesis. In the subject's trace, this damping first appears about 100ms afterPHIDCT first turns positive. Cur model best fit the subject's trace when $M + 4 + \frac{2}{3}/2 \implies$ 100ms, so the obvious suggestion is that as soon as the spinal cord receives a PHIDOT=O feesback, it generates a nervous signal to greatly increase viscoelastic damping in the limp.

Appendix A : PASCAL Program for E/Q/PHI Model

Listing of PASCAL PROGRAM developed for EMG-Torque-Movement simulation model. Written and tested by Valerie Congdon in collaboration with Prof. W. Kilmer, Fall and Winter 1930, 1981.

Section Titles :

Notation Titles and Global Variables Procedures and Functions The Main Program Input and Output Exemplary Output Notation

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SYMEOL IN TEXT SYMBOL(S) IN PRCGRAM

φ, φ, φ	PHI, PHIDCT, PHIDDCT ; Position, Velocity, Acceleration
Q _E , Q _F	QUEE, QUEF ; Torque produced by triceps, biceps
ς, C _ζ	ZETA,CZETA ; viscoelastic damping term , constant
v ^u	<pre>PSIV(dot) : Force-Velocity Relation</pre>
ΨI	PSII() : Integral Factor of Torque
EMGĚ	ET , GETEMG(EMGTRICEPS): Smoothed EMG for triceps
EMGF	EB , GETEMG(EMGBICEPS): Smcothed EMG for biceps
EMG _E , EMG _F	EMGE,EMGF ; Corrected EMG variables;EXTENSOR,FLEXOR
$EMG = \overline{EMG} + \overline{EMG}$	EMG ; Function which returns Corrected EMG values.
G(_T)	GOFTAU ; equals 0.0 if PHI >= 0, 1.0 otherwise
h _E (t-τ)	AITCH(T-TAU, EXTENSCR); Response of EXTENSOR
h _F (t- .)	AITCH(T-TAU,FLEXOR) ; Response of FLEXOR
q	QUE(MUSCLE) ; q computed for AITCH function
۹ _E	QE ; q in AITCH Function for EXTENSOR
۹ _F	QF ; q in AITCH function for FLEXOR
^u , ^µ E, ^µ F	MU , MUE , MUF
5, 5E, 5E	XI , XIE , XIF
A, A _E , A _F	A , AE , AF
k, k _E , k _F	K, KE, KF
: , J, t*	TAU , SIGMA , TSTAR
t	T , ATTIME ; time variables
⁸ 1, ⁸ 1E, ⁸ 1F	BETA1 , BETA1E , BETA1F
² 2, ² 2E, ² 2F	BETA2 , BETA2E , BETA2F
e ^x , cos x	EXP(X) , CCS(XRADIANS)

P₁, P₂, P₃ P1, P2, P3

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Notation

OTHER SYMBOLS IN PROGRAM

TRAPEZCID ; Rule for integration HTRAP ; step size in trapezoid rule LASTCNE, LASTFUN, FUN; Hold the last integration value, the last value of t the function , and the current function value HRUNGE : step size in Runge-Kutta Rule(PSII) INITALL ; Initializing Procedure SAVEIT, ONTO = ARRAY[1..70,0..2] of REAL SAVEPHIS ; ARRAY to hold values of PHI, PHIDCT, PHIDDOT PUSH ; Procedure to save PHI values in ARRAY SAVEPHIS READEMGFILE ; Read Input Procedure EMGFILE ; Input file CUTFILE , TAPEC , TAPE1 ; Cutput Files MM , DD , YY ; Mounth , Day and Year for Subject ID. SUB , AP , SES ; Subject No., AM or FM , session BL , TR ; Block and Trial numbers WHICHCNE = (EXTENSOR , FLEXOR) MUSCLE is of type WHICHONE REAL2D = ARRAY[0..25,1..2] of REAL EMGA , EMGTRICEP , EMGBICEP are of type REAL2D VALU , TIME ; constants for array postion MAX , MAXEMGTIME ; hold value of maximum time to run

38 THE FASTMOV PROGRAM WAS WRITTEN, DESIGNED, AND IMPLEMENTED BY VALERIE CONGDON, M.S. COMPUTER AND INFORMATION SCIENCE, FOR THE RESEARCH SEING CONDUCTED BY DRS. WILLIAM KILMER OF THE ELECTRICAL AND COMPUTER ENGINEERING DEPARTMENT AND WALTER KROL OF THE ENERCISE SCIENCE DEPARTMENT FUNDED BY THE ARMY MEDICAL (# *) (* *) (* *) (* *) (* *) RESEARCH COMMAND . (4 *1 PROGRAM FASTMOV(INPUT/,OUTPUT/,EMGFILE , CUTFILE,TAPEO , TAPE1); CONST TABI1 = ' TABI2 = ' TABI3 = ' ; FAST MOVEMENT PROJECT '; DEPARTMENT OF EXERCISE SCIENCE '; UNIVERSITY OF MASSACHUSETTS/AMHERST'; PRINCIPLE INVESTIGATORS : DR. WILLIAM KILMER AND DR. WALTER KRCLL'; RESEARCH ASSISTANTS : VALERIE CONGDON AND JEAN BOUCHER'; MODEL : E / G / PHIDOT (EMG / TORQUE / ANGULAR VELOCITY) '; MODEL PARAMETERS ; '; TRAPEZOID RULE STEP SIZE : '; RUNGE-KUTTA RULE STEP SIZE : '; A K BETAI BETAZ XI MU G(AITCH) '; TABI4 = 'TABIS = 'TABIS = ' TABI7 = ' TABI8 = ' TABI11= ' TAB115= ' DATE SESSION BLOCK TRIAL ' ; SUBJECT AM/PM 13 TABI12= ' SUBJECT ID : TABI13= ' TABI14= ' PHI (ACC) / ; ENG ENG PHI (BICEP)(TRICEP) (POS) PHI TIME GF PSIV PSIIE GE PSIIF TABI15= ' (MSEC) (VEL) TYPE "HICHONE = (FLEXOR , EXTENSOR) ; REALID = ARRAY[0..25,1..2] OF REAL ; SAVEIT = ARRAY[1..70,0..2] OF REAL ; (* NOTE: 7) = MU + XI + DELAY(=20) *) VAR EMGBICEP : REAL2D ; EMGTRICEP: REAL2D ; SAVEPHIS : SAVEIT ; ET , EB , PSIVE , PSIIE , PSIIF , QUEE , QUEF , PHI , PHIDOT , PHIDDOT : REAL ; BETAIF , BETAIE , BETA2F , BETA2E , XIF , XIE , MUF , MUE , AF , AE , KF , KE , ZETA , CZETA1 , CZETA2 , CZETA , GF , GE : REAL ; LASTPHI , LASTPHIDOT , LASTPHIDDOT , P1 , P2 , P3 , GOFTAU , T , MAX , DELAY : REAL ; BACKINTIME , I,MM,DD,YY,SUB,AP,SES,2L,TR : INTEGER ; HTRAP , HRUNGE : REAL ; (* H FCR TRAPEZOID AND RUNGE-KUTTA RULES *) EMGFILE , GUTFILE , TAPEO , TAPEI : TEXT ; DAMPSET : BCCLEAN ;

TITLES AND GLOBAL VARIABLES

Procedures and Functions

PROCEDURE READEMOFILE : Reads in data from input file ENGFILE and places it in the appropriate array. See Input and Output section.

FUNCTION GETENG :

For a particular time this function calculates the Smootned EMG value for either the flexor or extensor.

FUNCTION EMG : Computes Augmented EMG values.

PROCEDURE PUSH : Saves PHI, PHIDOT, and PHIDDOT values back in time.

FUNCTION QUE : For each set of beta values this function calculates the 'q' used in the h(t-tau) function.(See text).

FUNCTION AITCH : The reflex and mechanical response of the flexor or extensor is returned from this function. (h(t-tAu))

FUNCTION TRAPEZOID : A numerical method for approximating the integral which determines the value of PHI from PHIDOT and PHIDOT from PHIDDOT.

FUNCTION PSIV : The Force-Velocity relation is returned when given PHIDOT as a parameter.

FUNCTION PSII : The Integral Factor of Torque is approximated using the Runge-Kutta method for integration.

PROCEDURE INITALL : Initialization of all constants , variables and arrays. In addition the headers of the output files are printed.

```
PROCEDURE READEMGFILE(VAR EMGBICEP : REAL2D ;
VAR EMGTRICEP: REAL2D ;
VAR MAXEMGTIME : REAL )
VAR INDEX,TIME,VALU : INTEGER ;
ENDFILE : BCCLEAN ;
        BEGIN
              RESET(EMGFILE) ;
            INDEX := 0 ;
TIME := 1 ;
VALU := 2 ;
ENDFILE := FALSE ;
            READ(EMOFILE, MM.DD, YY, SUB, AP, SES, BL, TR , MAXEMOTIME) ;
              WHILE NOT ENDFILE DO
                        BEGIN
                              READ (EMOFILE)
                                        EMGBICEPCINDEX,TIME], EMGBICEPCINDEX,VALUJ,
EMGTRICEPCINDEX,TIME],EMGTRICEPCINDEX,VALUJ) ;
                          IF EMGBICEPCINDEX.TIME1 >= MAXEMGTIME THEN BEGIN
                                          ENDFILE := TRUE ;
INDEX := INDEX - 1
                                    END
                                            ELSE
                              INDEX := INDEX + 1
                          END
    END; (* END OF PROCEDURE READEMOFILE *)
    FUNCTION GETEMG( ATTIME , MAXEMGTIME : REAL ; VAR EMGA : REAL2D ): REAL;
(* THIS FINCTION RETURNS THE VALUE OF THE EMG AT TIME = ATTIME
USING THE FOLLOWING FORMULA :
F(X) = ((F(B) - F(A)) / (B - A)) * (X - A) + F(A) *)
(* THE EMGA HERE IS THE ARRAY HOLDING THE INTERVAL VALUES E.G. EMGBICEP *)
            VAR PREVIOUSTIME , PREVIOUSVALU : REAL ;
TIME , VALU , INDEX : INTEGER ;
       BEGIN
IF ATTIME < 0.0
THEN GETENG := 0.0
ELSE BEGIN
                  INDEX := 1 ;
TIME := 1 ;
VALU := 2 ;
            (* INCREMENT INDEX UNTIL UPPER BOUND OF INTERVAL IS FOUND *)
WHILE (( AITIME > EMGALINDEX,TIME]) AND
( ATTIME <= MAXEMGTIME))
DO INDEX := INDEX + 1 ;
            (* GET LOWER BOUND OF INTERVAL *)
IF INDEX >= 1
THEN BEGIN
                                      PREVIOUSTIME := EMGALINDEX-1,TIME] ;
PREVIOUSVALU:= EMGALINDEX-1,VALU3
                                END;
                  ATTIME IS EQUAL TO UPPER BOUND OR WHOLE INTERVAL IS THE SAME VALUE *)
IF_((_ATTIME = EMGACINDEX,TIME] ) OR ( EMGACINDEX,VALU] = PREVIOUSVALU ))
            (#
                      THEN
                              GETEMG := EMGACINDEX, VALUI
                      ELSE
                       UPPER BOUND IS ZERO THEREFORE THE WHOLE INTERVAL IS ZEROES *)
IF EMGALINDEX,VALUJ = 0.0
<u>THEN</u> GETEMG := 0.0
                (#
                    (* ATTIME IS SOMEWHERE INSIDE THE INTERVAL *)
GETEMG := (( EMGALINDEX, VALUI- PREVIOUSVALU)
/( EMGALINDEX, TIME] - PREVIOUSTIME))
* (ATTIME - PREVIOUSTIME) + PREVIOUSVALU
            END
END;(* END OF FUNCTION GETEMG *)
```

```
FUNCTION EMG( T , TAU : REAL ; MUSCLE : WHICHONE ) : REAL ; (* RETURNS THE AUGMENTED EMG VALUE FOR EITHER THE FLEXOR OR EXTENSOR *)
     VAR BACK : INTEGER ;
EMGA : REAL2D ; ZERO , CNE , TWO , X : REAL ;
      BEGIN
         BACX := TRUNC( T - TAU + DELAY );
ZERD := SAVEPHISIBACX , C ];
ONE := SAVEPHISIBACX , 1 ];
TWO := SAVEPHISIBACX , 2 ];
          IF MUSCLE = FLEXCR
           THEN BEGIN
                     EMGA := EMGBICEP ;
X := ZERO + P2 * CNE + P3 * TWO
                  END
                  EMGA := EMGTRICEP ;
X := -1.0 * ZERO - P2 * CNE - P3 * TWO
END ;
           ELSE BEGIN
      IF (( ZERD >= 0.0 ) AND (GEFTAU = 0.0))
      THEN
         EMG := GETEMG(TAU,MAX,EMGA)
     ELSE BEGIN
GOFTAU := 1.0 7
                IF X > 0.0 (* CUSPED BRACKETS RULE *)
                      EMG := ( P1 * X * GOFTAU) + GETEMG(TAU, MAX, EMGA)
                  ELSE
                      EMG := GETEMG(TAU,MAX,EMGA)
            END
     END ; (* END OF FUNCTION EMG *)
```

```
PROCEDURE PUSH( VAR U0,V1,V2 : REAL ; VAR CNTD : SAVEIT) ;
(* PLACES THE VALUES V0,V1,V2 IN A STACK NAMED
DNTO WHICH SAVES UPTD 70(MU+XI+20) VALUES BACK IN TIME *)
VAR I : INTEGER ;
BEGIN
FCR I := 0 TO BACKINTIME - 2
D0 BEGIN
CNTO[BACKINTIME-I,0] := CNTO[BACKINTIME-I-1,0] ;
ONTO[BACKINTIME-I,1] := ONTO[BACKINTIME-I-1,1] ;
CNTO[BACKINTIME-I,2] := CNTO[BACKINTIME-I-1,2] ;
END ;
ONTO[1,0] := V0 ;
ONTO[1,2] := V2
END ;(* END CF PRCCEDURE PUSH *)
```

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FUNCTION QUE(MUSCLE ; WHICHONE) : REAL ; (* THIS FUNCTION FINDS THE VALUE OF QUE SUCH THAT THE AITCH FUNCTION CONDITIONS ARE SATISFIED IE. QUE TIMES THE INTEGRAL = 1.0 *) VAR SIGMA , B1 , B2 , XI : REAL ; BEGIN IF MUSCLE = FLEXCR THEN BEGIN 81 := BETA1F ; 82 := BETA2F ; XI := XIF ; ELSE BEGIN B1 := BETA1E ; B2 := BETA1E ; XI := XIE ; END ; SIGMA := XI : GUE := (1.0 / (((EXP(-1.0*82*SIGMA) - 1.0) / 82) + ((1.0 - EXP(-1.0*81*SIGMA)) / 81))) END ; (* END OF FUNCTION QUE *) FUNCTION AITCH(TIME : REAL ; MUSCLE : WHICHONE): REAL ; (* THE AITCH FUNCTION RETURNS THE REFLEX AND MECHANICAL RESPONSE VALUE FOR TIME = T - TAU *) VAR HH , Q , B1 , B2 , XI , MU , TSTAR : REAL ; BEGIN IF MUSCLE = FLEXCR THEN BEGIN G;= GF; B1:= BETA1F; B2:= BETA1F; XI:= SETA2F; XI:= XIF; MU:= MUF; END ELSE BEGIN BEGIN G := GE ; B1 := BETA1E ; B2 := BETA2E ; XI := XIE ; MU := MUE END ; IF ((TIME < MU) CR (TIME > (XI + MU))) THEN AITCH := 0.0 THEN ELSE BEGIN DIN TSTAR := TIME - MU ; HH :≈ B * (EXP(~1.0*B1*TSTAR) - EXP(-1.0*B2*TSTAR)) ; AITCH := HH ; [*WRITELN(' H(',TIME:4:0,') = ',HH:10:5) ;*) END END; (* END FUNCTION AITCH *) FUNCTION TRAFEZOID(HTRAP , LASTONE , LASTFUN , FUN : REAL) : REAL ;
(* WE ARE USING THE TRAPEZOID RULE TO GET PHI FROM PHIDDT FROM PHIDDT
E.G. FHI(N) = PHI(N-1) + .5*HTRAP*(PHIDDT(N-1) + PHIDDT(N))
LASTONE LASTFUN FUN #) BEGIN TRAPEZDID := LASTONE + (0.5* HTRAP * (LASTFUN + FUN)) END ; END OF FUNCTION TRAPEZOID *) (#

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FUNCTION PSIV( DOT : REAL ) : REAL ;
(* PSIV , THE FORCE - VELOCITY RELATIONSHIP
(* PSIV = PSIVE = PSIVF
VAR X : REAL ;
                                                                                              +)
                                                                                               #)
          BEGIN
                 IN
X := 1.0 - ( DOT / 1000.0 ) ;
IF X >= 0.0
THEN PSIV := X
ELSE PSIV := 0.0
          END;
 (* END OF FUNCTION PSIV *)
  FUNCTION PSII( MUSCLE : WHICHONE ; T : REAL) : REAL ;
(* THE RUNGE-KUITA METHOD FOR INTEGRATION IS USED HERE
REF: NUMERICAL METHODS ; DAHLQUIST 2T.AL. , PRENTICE
HALL, 1974. PP 346-7 *
                                                                                                a 1
         VAR INTEGRAL , X , KONE , KTWO , KTHREE , A , H ,
XI , MU : REAL ;
I , NUMINTS : INTEGER ;
        FUNCTION F( TAU , T : REAL ; MUSCLE : WHICHENE ) : REAL ;
                 VAR EMGA : REAL2D ; K : REAL ;
               BEGIN
IF MUSCLE = FLEXOR
THEN K 1= KF
ELSE K 1= KE ;
               F := K + EMG(T, TAU, MUSCLE)
+ AITCH(T-TAU, MUSCLE)
               END ; (* END OF FUNCTION F IN PSII *)
          BEGIN (* PSII MAIN SECTION *)
                 IF MUSCLE = FLEXOR
                    THEN BEGIN
XI := XIF ;
MU := MUF ;
A := AF
                             END
                    ELSE BEGIN
                                   XI := XIE ;
MU := MUE ;
A := AE
                             A
END ;
                 H := HRUNGE ;
INTEGRAL := 0.0 ;
NUMINTS := TRUNC( (XI/H) - 1.0 ) ;
                 FOR I := 0 TO NUMINTS
                    DO BEGIN

X := T - MU - XI + (H * I) ; (* TAU IN TEXT *)

KONE := F( X , T , MUSCLE ) ;

KTWO := F( X + H/2.0 , T , MUSCLE ) ;

KTHREE := F( X + H , T , MUSCLE ) ;
                              INTEGRAL := INTEGRAL + ( (H/S.C) * ( KONE + 4.0 * KTWO + KTHREE ))
                          END :
               PSII := A * CDS(PHI + 0.0174532) + INTEGRAL
(* PHI IN RADIANS 2*PI / 360
                                                                                                  +)
          END ; (* END OF FUNCTION PSIL *)
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PROCEDURE INITALL ;

44 VAR : INTEGER ; T BEGIN DAMPSET := FALSE ; REWRITE(OUTFILE) ; REWRITE(TAPEO) ; REWRITE(TAPE1) CZETA1 := 0.01 ; CZETA2 := 0.09 ; BETA1E := 0.10 ; BETA2E := 0.15 ; BETA1F := 0.10 ; BETA2F := 0.15 ; XIE := 40.0 ; MLF := 50.0 ; MLF := 50.0 ; GE := GUE(EXTENSCR) ; GF := GUE(FLEXCR) ; HTRAP := 1.0 ; HRUNGE := 0.5 ; P1 := 0.00 ; P2 := 0.0 ; P3 := 00.0 ; GOFTAU := 0.0 ; KE := 0.40 ; KF := 0.65 ; AE := 0.10 ; AF := 0.1 ; PHI := 60.0 ; PHIDDT := 0.0 ; PHIDDDT := 0.0 ; LASTPHI := 60.0 ; LASTPHIDDT := 0.0 ; LASTPHIDDDT := 0.0 ; DELAY := 20.0 ; SACKINTIME := TRUNC(MUE + XIE + DELAY) ; FOR I := 1 TO BACKINTIME DO BEGIN SAVEPHISCI,01 := 0.0 (* PHI *) SAVEPHISCI,11 := 0.0 (* PHICOT *) SAVEPHISCI,21 := 0.0 (* PHICOT *) END READEMOFILE(EMGBICEP , EMGTRICEP , MAX) ;

WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI2) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI3) ; WRITELN(OUTFILE) ; WRITELN(OUTFILE) ;
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9) ;
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, HRUNGE:4:1, ' CZETA1 : ',CZETA1:S:3, ' CZET
WRITELN(OUTFILE, TABI9, ' ',AE:4:2,XE:S:2,ZETA1E:S:2, ' ',AE:4:2,XE: READEMOFILE(EMGBICEP , EMGTRICEP , MAX) ; CZETA1 : ',CZETA1:5:3,' CZETA2 : ',CZETA2:5:3) ; ',A2:1,' ',SES:1 , END ; (* END OF PROCEDURE INITALL *)

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_____ THE MAIN PROGRAM . - #) BEGIN INITALL ; T := 0.0 ; WHILE T <= MAX DO BEGIN ET := GETEMG(T, MAX, EMGTRICEP); EB := GETEMG(T, MAX, EMGBICEP); PSIVE := PSIV(PHIDDT); PSIIE := PSIV(PHIDDT); PSIIE := PSII(EXTENSOR ,T); PSIIF := PSII(FLEXOR ,T); GUEE := PSIVE * PSIIE ; GUEF := PSIVE * PSIIF ; IF ((PHI > 0.0) AND (PHIDDT > 0.0)) THEN DAMPSET := TRUE ; IF DAMPSET THEN CZETA := CZETA2 ELSE CZETA := CZETA1 ; PHIDDOT := QUEE - QUEF - CZETA*PHIDOT ; (* ZETA *) PHIDOT := TRAPEZOID(HTRAP , LASTPHIDOT , LASTPHIDDOT , PHIDDOT) ; PHI := TRAPEZOID(HTRAP , LASTPHI , LASTPHIDOT , PHIDOT) ; (* WRITE TO OUTPUT FILE*) PUT FILE*) IF (TRUNC(T) MOD 10) = 0 THEN BEGIN WRITELN(TAPEO-EB:8:4,ET:8:4) ; WRITELN(TAPE1,FHI:8:4,PHIDOT:8:4) ; WRITELN(OUTFILE,T:S:0, ',EB:7:3,ET:8:3,PHI:9:4, WRITELN(OUTFILE,T:S:0, ',EB:7:3,ET:8:3,PHI:9:4, PHIDOT:9:4,PHIDDOT:9:4,PSIVE:9:4,PSIIE:9:4,OUEE:9:4, PSIIF:9:4,QUEF:9:4) END ; PUSH(PHI, PHIDOT, PHIDDOT, SAVEPHIS) ; IF T = 555 THEN FOR I := 1 TO BACKINTIME DO WRITELN(I:4,SAVEPHIS[1,0]:8:3,SAVEPHIS[1,1]:9:3, SAVEPHIS[1,2]:9:3); LASTPHI := PHI ; LASTPHIDOT := PHIDOT ; LASTPHIDDOT := PHIDDOT ; T := T + 1.0;END ;

END.

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EMGFILE : Input file containg the Smoothed EMG data

OUTFILE : Output file showing values used/computed in table form : TIME, ET, EB, PHI, PHIDOT, PHIDDOT, PSIV PSIIE, QE, PSIIF, QF.

TAPEO : Output file holding values of ET and EB for PLOT Program

TAPE1 : Output file holding values of PHI and PHIDOT for PLOTPHI Program.

PLOT Program : Written in FORTRAN this Program plots the EMG values over time. TAPEO is its input file.

PLOTPHI Program : Written in FORTRAN this Program plots the Position and velocity values over time. TAPE1 is its input file.

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EMGFILE

Line number 1 contains the Subject Identification numbers and the maximum time of trial.

Lines 2 through (a possible)26 hold the following data :

Column 1 : The Biceps stop time of interval Column 2 : The Biceps value at end of interval Column 3 : The Triceps stop time of interval Column 4 : The Triceps value at end of interval

Note : If either the Biceps or Triceps has fewer intervals the table Must be padded so that each column has the same number of entries.

The SUBJECT ID. is made up of eight(8) variables :

Mounth, Day, Year, Subject number, AM or PM, Session, Block, and Trial numbers.

EXAMPLE :

On November 14,1980 subject #1 exercised in the AM for the fourth day first session, block one and 15th trial with the following results after 500 milliseconds :

11 14 80	1	041	15 500.	.0
0.0		0.0	0.0	0.0
64.0		0.65	166.0	0.07
96.0		0.65	136.0	0.58
323.0		0.0	228.0	0.95
324.0		0.09	300.0	0.95
350.0		0.13	350.0	0.0
500.0		0.0	500.0	0.0

Contains the parameters used and the values computed in PROGRAM FASTMCV.

Sample Header :

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FAST MOVEMENT PROJECT

DEPARTMENT OF EXERCISE SCIENCE UNIVERSITY OF MASSACHUSETTS/AMHERST PRINCIPLE INVESTIGATORS : DR. WILLIAM KILMER AND DR. WALTER KRCLL RESEARCH ASSISTANTS : VALERIE CONGDON AND JEAN BOUCHER

MODEL : E / G / PHIDOT (EMG / TORQUE / ANGULAR VELOCITY)

 MODEL
 PARAMETERS : TRAPEZCID RULE STEP SIZE : 1.0 RUNGE-KUTTA RULE STEP SIZE : 0.5
 DELAY : 20.00 P1 : 0.030 P2 : 6.000 P3 : 20.000 CZETA1 : 0.010 CZETA2 : 0.500

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 K
 BETA1 BETA2 XI 0.10 0.40 0.09 0.15 40.00 60.00 0.24054
 MU 0.10 0.65 0.09 0.15 40.00 60.00 0.24054

SUBJEC	T ID :	DATE 11/14/8	SUBJECT 0 1	AM/PM 0	SESSION 4	BLOCK 1	TRIAL 15			
TIME (MSEC)	EMG (BICEP)(EMG TRICEP)	PHI (PCS) (PĤI VEL)	PHI (ACC)	PSIV	PSIIE	0E	PSIIF	QF

TAPEO :

Column 1 holds the value of the Smoothed EMG (BICEPS). (XXX.XXXX) Column 2 holds the value of the Smoothed EMG (TRICEPS). (XXX.XXXX)

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TAPE1 :

Column 1 holds the value of PHI(t). (XXX.XXXX) Column 2 holds the value of PHIDOT(t). (XXX.XXXX)

PLOT

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PROGRAM PLOT(INPUT,CUTPUT,TAPEO) PLOTS TWO GRAPHS SIMULTANEQUSLY WITHIN THE RANGE -.5 TO 1.5 TOTAL OF 51 POINTS. DIMENSION DATA(51,3,3) DIMENSION DATA(51,3,3) PRINT 20 FORMAT(//," MCDEL : E / G / PHIDOT (EMG / TORQUE / ", "ANGULAR VELOCITY)",//," E : EXTENSOR (TRICEPS) ",/, F : FLEXOR (3ICEPS) ",/," 1 : INDICATES THE 1.0 LINE", /," 0 : INDICATES THE 0.0 LINE ",//) DO 10 I=1,51 READ(0,50) DAT1, DAT2 IF(EDF(0))99,57 IF(DAT1.LT.-.5) DAT1 = 1.5 IF(DAT2.GT.1.5) DAT2 = 1.5 IF(DAT2.LT.-.5) DAT2 = -.5 IALT = 10 20 + +* ÷ 57 56 IP (DA12.L1.-.5) DA12 = -.5 IALT = 10 DATA(I,1,1) = FLDAT(I*IALT) - 10.0 DATA(I,1,2) = DAT1 DATA(I,1,3) = 1HF DATA(I,2,2) = DAT2 DATA(I,2,3) = 1HE DATA(I,3,3) = 1H1 CONTINUE CONTINUE CALL PLOTIN(DATA:51,51,3,-.5,1.5,1H0,75,0) FORMAT(2F8.4) 10 50 99 END SUBROUTINE PLOTIN(DATA, NX, MAXX, NYSETS, YMIN, YMAX, AXIS, IPW, IRCL) DIMENSION DATA(MAXX,NYSETS,3),PLOT2(120) DU 9 L=1,120 9 PLOT2(L)=1H IF(YMAX.GE.YMIN)GOTO10 PRINT 2,YMIN,YMAX 2 FORMAT(* MIN Y IS GREATER THAN MAX Y*/* MIN Y= *,G17.10,* MAX Y= PLOT PLOT PLOT *PLOT PLOT X G17 10) RETURN PLOT 10 YINC=(YMAX-YMIN)/FLOAT(IPW-12) PLOT DG 100 JX=1,NX DG 20 JY=1,NYSETS Y=DATA(JX,JY,2) IF (Y.GE.YMIN.AND.Y.LE.YMAX) GOT030 PRINT 3,Y PLOT PLOT PLOT 3 FORMAT(* Y VALUE DUES NOT FIT WITHIN DEFINED Y RANGE*, G17.10) INDX=INT(.5+(Y-YMIN)/YINC) IF(INDX.LT.1)INDX=1 IF(INDX.GT.120)INDX=120 PLOT2(INDX)=DATA(JX,JY,3) CONTINUE RETURN 30 PLOT PLOT PLOT 20 С IF (AXIS.ED.0)GDTD40 IF (YMIN.GT.0)GDTD40 IXAXIS=(O-YMIN)/YINC IF(PL0T2(IXAXIS).E0.1H)PL0T2(IXAXIS)=AXIS LEN=LENGTH(PL0T2) PRINT 5,DATA(JX,1,1),(PL0T2(K),K=1,LEN) FORMAT(G11.5,IX,120A1) D0 50 L=1,120 PL0T2(L)=1H CONTINUE RETURN PLOT PLOT PLOT PLOT PLOT 40 PLOT 5 PLOT PLOT PLOT 50 100 PLOT RETURN END INTEGER FUNCTION LENGTH(IARRAY) RETURNS LENGTH OF NON-BLANK PART OF IARRAY. DIMENSION IARRAY(120) ε LENGTH=0 LEAGTH=0 DD 100 I=1,120 J=121-I IF(IARRAY(J).NE.1H) GOTO 200 CENTINUE LENGTH=J RETURN END 100 200

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PLOTPHI

PROGRAM PLOTPHI(INPUT, OUTPUT, TAPE1) PROBREM PLUIPHI(INPUT, DUTPUT, TAPE1) PLOTS TWO GRAPHS SIMULTANEOUSLY WITHIN THE RANGE -150. TO 150. TOTAL OF SO POINTS. DIMENSION DATA(51,3,3) PRINT 20 20 FORMAT(///" MODEL: E / G / PHIDDT (EMG / TORGUE FORMAT(//," MODEL : E / G / PHIDDT (EMG / 1 "ANGULAR VELOCITY)",//," P : POSITICN (PHI) " V : VELOCITY * 100 (PHIDDT * 100) ", 20 (EMG / TORGUE / ", ";/, . VELULIT * 100 (PHIDUT * 100) ", INDICATES SO DEGREE LINE ",/, 00000 : INDICATES O DEGREE LINE ",//) ٠ 1." + + DO 10 I=1,51 READ(1,50) DAT1 , DAT2 IF(EDF(1))99,57 DAT2 = DAT2 * 100.0 IF(DAT1.GT.150.) DAT1 = 150. IF(DAT1.LT.-150.) DAT1 = -150. IF(DAT2.GT.150.) DAT2 = 150. IF(DAT2.GT.150.) DAT2 = -150. IALT = 10 DATA(I,1.1) = FLOAT(I*IALT) - 10.0 DATA(I,1.2) = DAT1 DATA(I,2.2) = DAT2 DATA(I,2.3) = 1HV DATA(I,3.2) = 50. DATA(I,3.3) = 1H2 C 57 56 DATA(1,3,3) = 1H: CONTINUE CALL PLOTIN(DATA,51,51,3,-150.,150.,1H0,75,0) FORMAT(2F8.4) 10 50 99 END 9 END SUBROUTINE PLOTIN(DATA,NX,MAXX,NYSETS,YMIN,YMAX,AXIS,IPW,IRCL) DIMENSION DATA(MAXX,NYSETS,3),PLOT2(120) DO 9 L=1,120 9 PLOT2(L)=1H IF(YMAX.GE.YMIN)GOTO10 PRINT 2,YMIN,YMAX 2 FORMAT(* MIN Y IS GREATER THAN MAX Y*/* MIN Y= *,G17.10,* MAX Y= *PLOT Y 517 10) X G17.10) RETURN PLOT NEIURN NO YINC=(YMAX-YMIN)/FLOAT(IPW-12) DO JO JX=1,NX DO 20 JY=1,NYSETS Y=DATA(JX,JY.2) IF(Y.GE.YMIN.AND.Y.LE.YMAX)GOTO30 PRINT 3,Y 3 FORMAT(* Y VALUE DOES NOT FIT WITHIN DEFINED Y RANGE*,G17.10) PETURN PLOT 10 PLDT PLOT PLOT PLOT PLOT PLOT RETURN INDX=INT(.5+(Y-YMIN)/YINC) IF(INDX.LT.1)INDX=1 IF(INDX.GT.120)INDX=120 PLDT2(INDX)=DATA(JX,JY,3) 30 PLOT PLOT PLOT 20 CONTINUE C PLOT IF(AXIS.EG.0)GOT040 IF(YMIN.GT.))GOTO40 IXAXIS=(0-YMIN)/YINC IF(PLOT2(IXAXIS).EQ.1H)PLOT2(IXAXIS)=AXIS PLOT PLOT 1P (PLU(2(1XAX15).EU.1H)PLU(2(1XAX15)=A 40 LEN=LENGTH(PLU(2) PRINT 5.0ATA(JX,1,1),(PLU(2),X=1,LEN) 5 FORMAT(G11.5,1X,120A1) DU 50 L=1,120 50 PLU(2)=1H 100 CONTINUE RETURN END PLOT PLOT PLOT END DINTEGER FUNCTION LENGTH(IARRAY) RETURNS LENGTH OF NON-BLANK PART OF IARRAY. DIMENSION IARRAY(120) C LENGTH=0 DO 100 I=1,120 J=121-I IF(IARRAY(J).NE.1H) GDTD 200 CONTINUE 100 LENGTH=J RETURN 200 END

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