Neuroscience Vol. 4, pp. 1493 to 1500
Pergamon Press Ltd 1979. Printed in Great Britain

REFLEX MOTOR OUTPUT TO TORQUE PULSES IN MAN: IDENTIFICATION OF SHORT- AND LONG-LATENCY LOOPS WITH INDIVIDUAL FEEDBACK PARAMETERS

J. R. DUFRESNE, J. F. SOECHTING and C. A. TERZUOLO
Laboratory of Neurophysiology, Department of Physiology, University of Minnesota Medical School, 315 Millard Hall, Minneapolis, MN 55455, U.S.A.

Abstract—Pseudo-random torque disturbances were applied to the human forearm to characterize the relations between the reflex motor output and the kinematic variables using a linear model. The best least-squares fit to the model relating average electromyographic activity to forearm angular position, velocity and acceleration was obtained by assuming separate time delays for each kinematic variable. Best estimates for the time delays were 86 ms for position, 25 ms for velocity and 45 ms for acceleration.

The central structures which could be primarily involved in mediating the velocity and acceleration feedbacks are discussed.

THE PROBLEM of the existence and functional role of long-latency reflexes mediated by the motor areas of the cortex has received much attention in recent years, following the suggestion of PHILLIPS (1969). He saw such transcortical reflexes as providing a focusing action for muscle spindle feedback.

Among the data which are consistent with the participation in motor control by a transcortical loop is the finding that the motor output response to a sharp pulse of torque is fractionated into distinct peaks, which have become known as M_1 and M_2 (EVARTS & TANJI, 1974; HAMMOND, 1960; LEE & TATTON, 1975; MARSDEN, MERTON & MORTON, 1972). While the latency of M_1 is consistent with a segmental conduction delay, the timing of M_2 provides ample time for its mediation by the cortex. Furthermore, it was found that precentral units in the cortex respond in a manner and with a latency consistent with a transcortical reflex action (CONRAD, MEYER-LOHMANN, MATSUNAMI & BROOKS, 1975; EVARTS, 1973). Finally LEE & TATTON (1975) showed that M_2 is abolished following a lesion of the postcentral cortex.

Alternative explanations of these findings have, however, been suggested. Indeed, the fractionation of the motor output response to a pulse of torque may well reflect receptor and motor neuron non-linearities brought into play by the initially high acceleration produced by the perturbation (GHEZ & SHINODA, 1978). It has also been shown that the M_2 peak requires sensory inputs with delays compatible with segmental reflexes, since this peak can be abolished or greatly reduced by a second pulse of torque in the opposite direction (VILIS & COOKE, 1976). More importantly, GHEZ & SHINODA (1978) have reported that M_2 can still be seen in spinalized and decerebrate cats. Also, the fact that M_2 is abolished by lesions of the postcentral areas of the cortex could be attributed to a removal of tonic facilitation upon segmental reflex mechanisms (TATTON, discussion of LEE & TATTON, 1975).

From a functional point of view, the role and the advantages for motor control of transcortical reflexes remain unclear. First, the longer latency of such reflexes could well be destabilizing (OGUZTORELI & STEIN, 1976). Second, the necessity for a focusing action, as suggested by PHILLIPS (1969), needs to be established. In fact, it would seem that coactivation of different muscles during movements and posture (cf. ENGBERG & LUNDBERG, 1969; THACH, 1970) is not uncommon. Finally, the modulation of reflex gain as a function of the task, suggested by EVARTS & TANJI (1974), could equally well be accomplished by segmental mechanisms.

Recently, we have studied adaptive features of the reflex control of forearm position and movement by attempting to quantitate changes in the gain and dynamics of the input–output relation between motor activity and angular position of the forearm as a function of such factors as the instruction given the subject and other variables (DUFRESNE, SOECHTING & TERZUOLO, 1978). In these experiments, changes in angular position of the forearm were produced by random sequences of torque pulses. The gain and dynamics of the reflex motor output were then quantitated by means of a linear model relating this output to position and its first two derivatives, using a single time delay.

The rationale for the basic assumption that electromyographic (EMG) activity depends on angular position and its derivatives is schematically illustrated in Fig. 1. Simply stated, the model assumes that a component of the motor output is generated by reflex

Abbreviations: EMG, electromyographic; M_1, M_2, distinct peaks in the motor output response to a sharp torque.

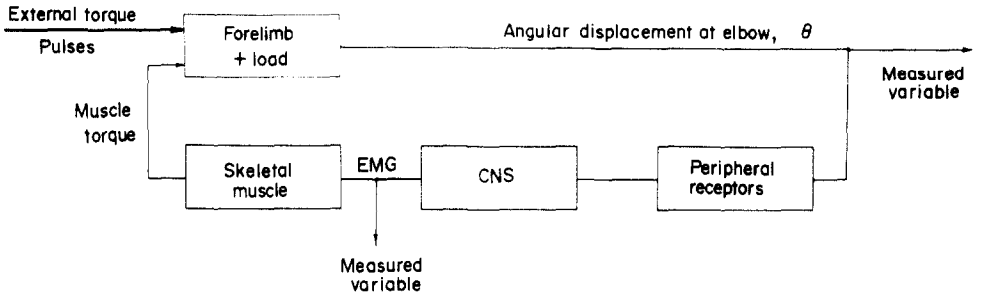

FIG. 1. Block diagram of the experimental system. This diagram schematically presents the assumptions which underly the model relating EMG activity to the variables of forearm motion.

activities originating from muscle receptors. Activities of these receptors are related either directly (in the case of muscle spindles) or indirectly via the viscoelastic properties of the muscle (in the case of tendon organs) to muscle length and its derivatives (cf. ROSENTHAL, MCKEAN, ROBERTS & TERZUOLO, 1970). While the dependence of motor output on the parameters of a movement is therefore to be expected, the exact form of the model chosen is in a sense arbitrary.

Several considerations dictated the form of model chosen. It was the simplest possible linear model (i.e. having the fewest terms) consistent with a Fourier analysis of the input–output relation between angular position and biceps/triceps EMG activity (DUFRESNE *et al.*, 1978). Simple non-linear terms, that is, those in the square and cube of velocity and acceleration, were found to be small. (Such non-linear extensions of the model will be considered in a subsequent paper.) Furthermore, angular position is directly measurable, whereas muscle force can only be indirectly estimated from the externally applied torque and the kinematic variables.

Using such a model with a single time delay, the optimal value for this delay was found to be approx 45 ms. This value is clearly in excess of segmental conduction delays but shorter than those for long loops as reported for man (cf. MARSDEN *et al.*, 1972). This consideration suggested to us that the time delay found might represent an average of short- and long-latency reflex actions. Therefore, we have re-examined the input–output relationship on the assumption that the reflex output to each of the kinematic variables (position, velocity and acceleration) may be processed largely at different levels in the central nervous system, with the result that each of these feedbacks would have different time delays.

The results to be presented will indicate that while the time delay of the velocity feedback is compatible with segmental conduction delays, those for both position and acceleration feedback have much longer time delays. In the discussion, we will then consider the central structures which could be primarily involved in mediating the changes in motor output related to each of these parameters. Particular emphasis will be placed on the possibility that the acceleration feedback may involve the participation of a transcortical loop.

A preliminary report was presented at the Pisa conference on the 'Reflex Control of Posture and Movements', 1978 (TERZUOLO, DUFRESNE & SOECHTING, *in press*).

EXPERIMENTAL PROCEDURES

Experimental set-up

The experimental set-up was fully described previously (DUFRESNE *et al.*, 1978). Briefly, the right forearm of human subjects is strapped to a rigid mould attached to the shaft of a torque motor, the elbow joint being aligned with its axis. Either single pulses of torque or a series of pulses, occurring at random times, were used. The latter are fully described in DUFRESNE *et al.* (1978).

EMG activity from biceps and triceps muscles was sampled either intramuscularly or with surface electrodes. Angular position and acceleration of the forearm were measured by means of a potentiometer and accelerometer. A measure of the torque was obtained by recording the current supplied to the motor.

Data analysis

Data acquisition was by a digital computer. Biceps and triceps EMG activity, angular position and acceleration, and the motor current were recorded. Ten or more individual trials were averaged after full-wave rectification of the EMG signal.

In one series of experiments, a single torque pulse tending to flex or extend the forearm was applied. The pulse width ranged from 10 to 80 ms and its amplitude from 1.2 to 6.0 N-m. In a second series, a pseudo-random train of pulses lasting from 5 to 8 s was applied (DUFRESNE *et al.*, 1978). The subjects were instructed to resist the perturbations.

A linear model was fitted to the experimental data by a least-squares method. This model, to be considered under Results, contains position, velocity and acceleration terms with individual delay times. To obtain the value of velocity, the position signal was numerically differentiated. The time delays were permitted to vary between 10 and 150 ms, usually in 10 ms increments. For each combination of time delays, the coefficient for the contribution by each of the parameters was optimized so as to obtain the least-squared error for the model. The combination for which the error was smallest was taken as the best estimate of model parameters and time delays for each set of data.

RESULTS

Can long-latency reflexes be inferred from the motor output to single pulses of torque?

The extent to which the EMG activity of biceps and triceps muscles depends on the amplitude and duration of single extension and flexion pulses was investigated to ascertain if such data could provide an indication of the contribution to the overall response by long-latency reflexes. Four torque-pulse amplitudes (2.4, 3.6, 4.8 and 6.0 N-m) and four pulse widths (20, 40, 60 and 80 ms) were used. Ten trials for each combination were obtained from two subjects. These trials were averaged after full wave rectification of the EMG activity.

Figure 2 shows ensemble averages of biceps EMG activity and of forearm angular position and velocity following torque pulses lasting from 20 ms (Fig. 2A) to 80 ms (Fig. 2D). The amplitude of the pulses was 6.0 N-m. The mean level of EMG activity for 250 ms prior to the pulse is also given. It will be noted that the EMG activity begins to deviate from its background level about 20–30 ms after the onset of the pulse and that the transient increase in activity is fractionated into two bursts, peaking at about 35 and 65 ms after the onset of the torque pulse. These bursts have already been described by other authors (cf. Conrad *et al.*, 1975; Lee & Tatton, 1975; Evarts & Tanji, 1974; Vilis & Cooke, 1976). While their timing is independent of pulse duration, other features depend on this parameter. For instance, the silent period which terminates the increased activity is progressively delayed as the pulse duration is increased. When the pulse width is 20 ms, the minimum of biceps activity occurs at 80 ms. It is delayed to 130 ms using a pulse width of 80 ms. More generally, the decrease in activity below background level taking place at about 250–300 ms overlaps the angular velocity at which the muscle is being unloaded (Fig. 2C and D).

As for the two bursts of activity, their amplitude rather than their timing depends on the values of the velocity and acceleration, as a function of the pulse parameters. This is shown in Fig. 3. The traces in Fig. 3(A) are the average biceps activity in response to extension torque pulses of two different amplitudes. The solid line under each trace indicates pulse duration; this ranged from 20 to 80 ms. The amplitude of the first burst is clearly related to pulse amplitude which in turn determines initial velocity and acceleration. Its timing (lower line in Fig. 3C), instead, does not appear to depend on either pulse amplitude or duration. This is also true for the second burst (upper trace in Fig. 3C). It is this fact and the coincidence of this latency with the delay time appropriate

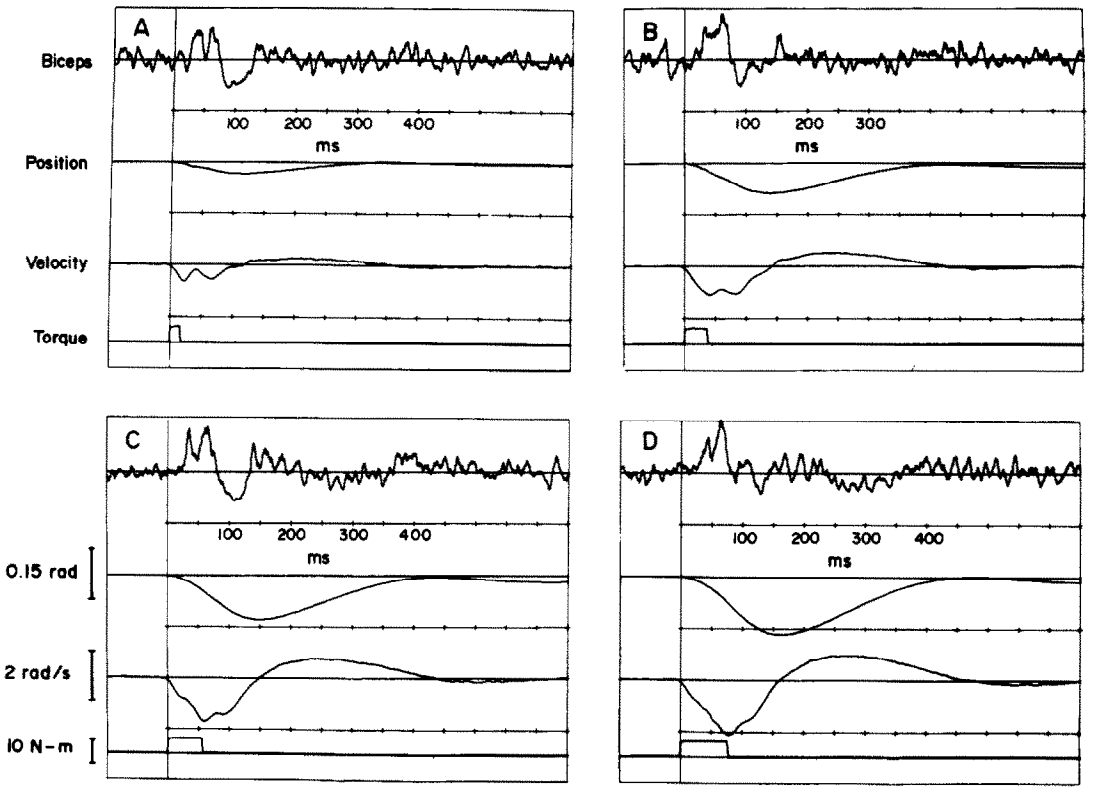

FIG. 2. Response to extension torque pulses. Six N-m torque pulses with durations of 20–80 ms were applied to the human forearm. Biceps EMG activity, forearm angular position and velocity, and applied torque were averaged over ten trials for a given pulse duration. The mean level of background EMG activity for the 250 ms preceding the pulse was subtracted from the entire EMG record.

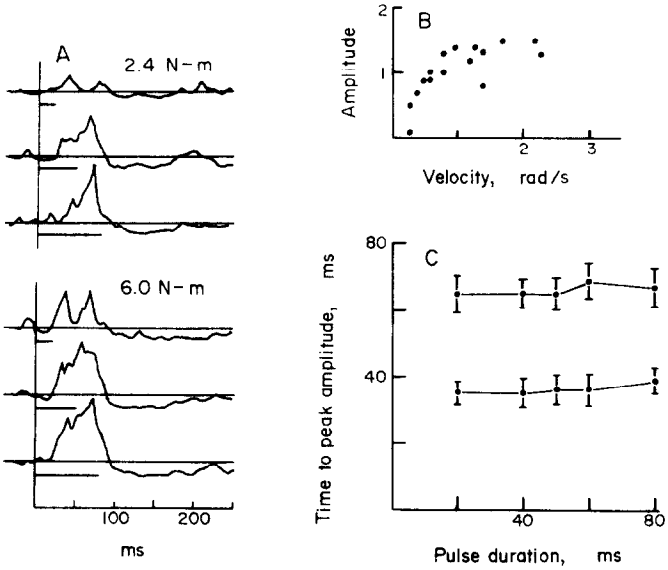

FIG. 3. Dependence of the biceps electromyographic response to extension torque pulses on kinematic and pulse parameters. Part (A) demonstrates the dependence of the ensemble-averaged EMG response on torque pulse duration (20, 50 and 80 ms pulses are indicated by the horizontal bars) at two different pulse amplitudes. The mean background EMG level prior to the pulse is given by the horizontal lines. Each trace is the average of ten trials. Part (B) shows the dependence of the peak amplitude of the second burst in EMG activity on the forearm velocity measured at the onset of this burst. Each dot represents the data of ten trials from one subject. Part (C) shows the absence of a dependence of the timing of the first (lower trace) and second bursts (upper trace) of the EMG response on pulse duration. The means (dots) and standard deviations (vertical bars) for data from two subjects are presented.

for a transcortical loop which, among other factors, have led some authors to suggest that the second burst is mediated by the cortex (cf. LEE & TATTON, 1975; VILIS & COOKE, 1976).

However, the relationship between the amplitude of this burst and the pulse parameters, to be described below, strongly implies a large contribution by reflex actions with short, segmental latencies. Its amplitude was not uniquely related to either one of the pulse parameters (amplitude or duration), but instead varied with both parameters as may be seen in Fig. 3(A). Also, this burst could be absent when large amplitude (6 N-m) and short duration (10 ms) pulses were used. (Note that the value of the velocity at short time intervals after the onset of the pulse is also not simply related to either of the pulse parameters, but depends on both.) In fact, the best correlation we found is with the value of angular velocity at 20–30 ms prior to the onset of the burst. This correlation is shown in Fig. 3(C) and suggests that the amplitude of M_2 depends on the parameters of the motion (velocity) with a latency similar to that for segmental conduction delays. This is in agreement with the finding of DUFRESNE (1977) and VILIS & COOKE (1976), that the second burst can be significantly reduced when a brief torque pulse is followed immediately by a second pulse of torque in the opposite direction. The second pulse acted to reduce the value of the velocity of the perturbation resulting from the first pulse at a time appropriate for M_2.

Thus, the following conclusion should be drawn: while the data are not inconsistent with the proposition of a loop with a long time delay, no assessment of its contribution relative to segmental reflex actions can be made using single torque pulses. The main difficulty is that the output response to this type of input is highly non-linear. Actually, one reason for the nonlinearity (over and above those brought about in muscle receptors and motoneurons by the fast transitions in muscle lengths (HASAN & HOUK, 1975) caused by single pulses, may be the summation of long- and short-latency responses.

However, it was recently shown (DUFRESNE *et al.*, 1978) that difficulties inherent to non-linear behaviour can be minimized by using pseudo-random sequences of torque pulses. In this approach, a pulse of torque may occur sometimes when the muscle is being stretched and sometimes when it is shortening, i.e. at varying background levels of motor activity. Fourier analysis has shown that when this type of input is used, reflex changes in motor output can be adequately characterized by a linear dependence on position, velocity and acceleration (DUFRESNE *et al.*, 1978) Thus, the approach can be used for attempting to sort out the contribution by long- and short-latency loops, if the assumption is made that each loop can be identified with one of the kinematic variables.

Evidence from modelling about short- and long-latency feedbacks

The linear parametric model used, which incorpor-

ates the above stated relationsh ps, is:

$$\mathrm{EMG}(t) = A\theta(t - \tau_a) + B\dot{\theta}(t - \tau_b) + C\ddot{\theta}(t - \tau_c) \quad (1)$$

where EMG(t) represents the average rectified EMG activity, and τ_a, τ_b and τ_c are the time delays for the position (θ), velocity ($\dot{\theta}$) and acceleration ($\ddot{\theta}$) variables, respectively. A, B and C are coefficients expressing the amount by which the reflex motor output depends on each variable. For any given set of time delays, the best fit of the model to the data was obtained by estimating the mean squared error (see Methods). Figure 4 shows for one set of data how this error (expressed as a fraction of the variance of the motor output) varies with the time delays. The left side of Fig. 4 shows the dependence of the error on velocity (τ_b) and acceleration (τ_c) time delays, the position time delay (τ_a) being fixed at 80 ms. The plots on the right side describe the variation of the error of each of the three time delays when the other two are fixed. It is evident that the error is very sensitive to velocity and acceleration delays, thus permitting a reliable estimation of these parameters. Instead, the error is rather insensitive to the time delay of the position parameter.

The best estimates for the time delay of the velocity and acceleration feedbacks in Fig. 4 are not the same, being 30 ms for velocity and 50 ms for acceleration. These values are typical for both biceps and triceps muscles. Table 1 shows average values for each parameter, obtained from eight experiments on five subjects. Note that the standard deviations for the velocity and acceleration time delays are rather small and that their average values differ significantly (25 and 45 ms, respectively). Obviously, the velocity delay is consistent with segmental conduction delays, while that for acceleration clearly is not. Since the error is rather insensitive to position delay, little can be said about this parameter except that its delay (86 ms) is also in excess of that for segmental mechanisms.

TABLE 1. TIME DELAYS FOR INDIVIDUAL FEEDBACK PARAMETERS

	Position delay (ms)	Velocity delay (ms)	Acceleration delay (ms)	N
Biceps	100	29	49	(12)
	133	21	36	(9)
	90	30	40	(3)
	57	24	48	(4)
	80	30	47	(3)
	90	24	45	(2)
	68	36	48	(3)
	63	28	44	(6)
Triceps	102	15	48	(12)
	130	26	57	(6)
	68	22	36	(3)
	115	18	53	(4)
	90	23	40	(3)
	42	30	48	(2)
	66	21	43	(6)
	86 ± 26	25 ± 5	45 ± 6	

N represents the number of ensemble averages, each from ten independent trials, which provided the data base for a given experiment.

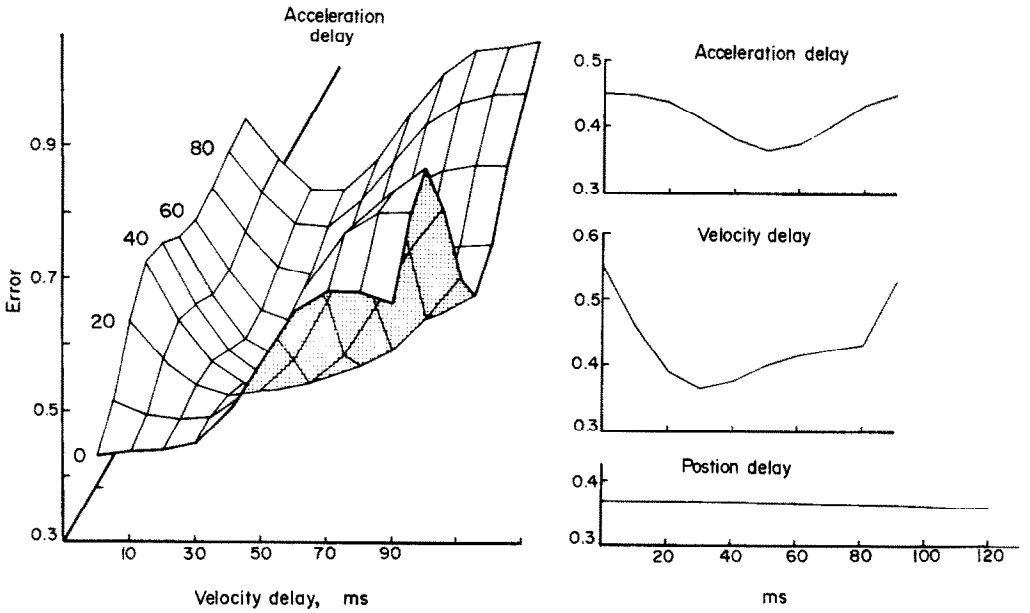

FIG. 4. Dependence of the least-squared error given by a linear model on the feedback time delays. On the left, this error (expressed as a fraction of the variance) is plotted as a function of the velocity and acceleration delays. The position delay is 80 ms. The right-hand plots demonstrate that the acceleration and velocity delays strongly influence the modelling error (when all other delays are fixed), while the position delay does not. (The fixed time delays used are: 80 ms for position, 30 ms for velocity and 50 ms for acceleration.)

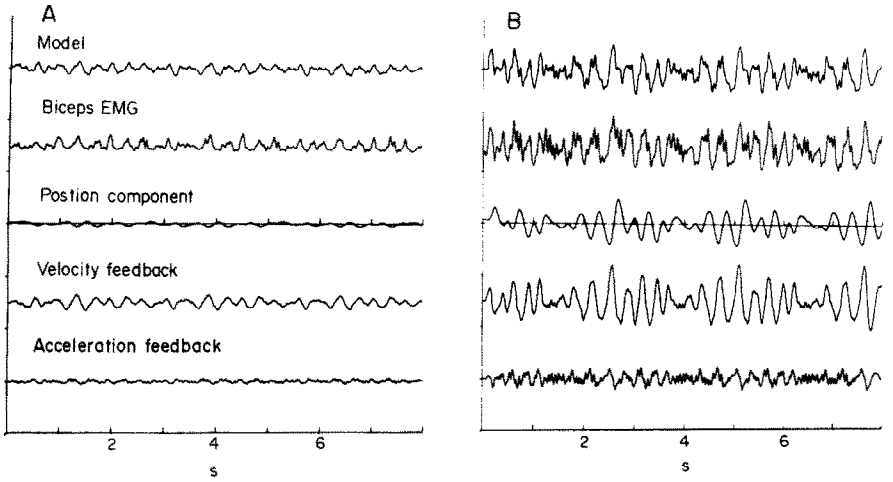

FIG. 5. Relative dependence of biceps electromyographic activity on each of the feedback parameters. The traces from top to bottom are: integrated EMG activity predicted by the model, the measured activity, and the contribution to the model by position, velocity and acceleration feedback. All traces are plotted to the same scale. In (A) the mean applied torque was zero, in (B) it was 16 N-m, tending to extend the forearm. Changes in position were produced by applying a pseudo-random sequence of torque pulses.

Relative contribution of short- and long-latency feedbacks

We now come to consider the question of the relative contribution to the reflex changes in motor output by each feedback, as identified by modelling. Figure 5 illustrates the relative amplitude of position, velocity and acceleration feedback in one subject under two experimental conditions; namely, when the mean torque is zero (Fig. 5A) and when there is a mean torque tending to extend the forearm (Fig. 5B). In each case, the top trace represents the average rectified biceps EMG. The trace labelled 'model' represents the best fit of the data with the linear model (Eq. 1). The traces labelled 'position', 'velocity' and 'acceleration' represent the contribution to the overall output by each of these feedbacks. The scaling for all traces is the same. Both in (A) and (B), the velocity feedback is dominant. The acceleration component is approximately one-third of the velocity contribution in these examples, when peak-to-peak values are compared. In other experiments and subjects, this ratio ranged from one-fifth to two-fifths. The ratio of position to velocity feedback is approximately one-fifth in Fig. 5(A), and three-quarters in Fig. 5(B), where the mean torque tends to extend the biceps. Changes in the contribution by each feedback as a function of such variables as the mean level of force, mean elbow joint angle and instruction given the subject have been discussed previously (DUFRESNE *et al.*, 1978).

DISCUSSION

The first problem to be considered is the reliability of the estimates for the time delay of each of the model parameters. For the velocity and acceleration, the pertinent points are: (1) the standard deviations, both within and among subjects, are quite small (Table 1); and (2) the least-squared error was found to be quite sensitive to changes of either one of these delay times (Fig. 4). Thus, it seems safe to conclude that the difference between them is significant, given the chosen model. The delay of the position parameter, instead, could be varied between 10 and 150 ms without changing, in most cases, the estimates of the delay for the other two variables. This point is of some significance *vis-à-vis* the physical interpretation of the results of modelling. In this context, one should note that the velocity and acceleration variables have previously been reported to be represented in the reflex motor output under different experimental conditions (SOECHTING, 1973; TERZUOLO & VIVIANI, 1973). As for the position parameter, it will be considered later. At this time, we shall continue instead to discuss the estimates for the velocity and acceleration delays as they may be affected by the model chosen.

First of all, it could be argued that if nonlinear models were used—in which, for example, the changes in motor output were related to a power function of the velocity, as seems to be the case for the primary endings of the muscle spindles (CROWE & MATTHEWS, 1964)—different values could eventually be obtained. To answer this question, nonlinear models with squared and cubed velocity and acceleration terms were also used. The optimal delay times predicted by these models were virtually identical to those reported here. Moreover, only very modest improvements of the residual error were obtained using these non-linear models. This subject, which is outside our present scope, will be dealt with in a subsequent paper in which the question of non-linearities will be confronted directly.

Secondly, the possibility cannot be readily dismissed that each of the feedback delays may represent a weighted average of two or more distinct time delays. Indeed, available experimental evidence concerning the velocity feedback suggests that this may be the case.

Velocity feedback

The average value for the delay of this feedback (25 ms) is compatible with: (1) the known velocity sensitivity of the primary endings of muscle spindles; and (2) a mediation by segmental mechanisms. Indeed, the average values are very close to the latency of the tendon jerk in man (cf. HAMMOND, 1960). However, there is strong evidence for the participation of cerebellar activities in the velocity feedback since: (1) after cerebellar lesions, the modulation of the motor output related to velocity is either absent or greatly reduced (SOECHTING, RANISH, PALMINTERI & TERZUOLO, 1976; TERZUOLO & VIVIANI, 1973); and (2) velocity is represented in the activity of units in the interpositus nucleus (BURTON & ONODA, 1978). Since the problem of cerebellar involvement in the automatic control of movement has been discussed extensively elsewhere (ECCLES, 1973; ALLEN & TSUKAHARA, 1974; BURTON & ONODA, 1978; TERZUOLO & VIVIANI, 1973), it will not be further considered.

Instead, we will address the question of the utility and adequacy of a velocity feedback. On this subject, the following points can be made: (1) although a velocity feedback will suffice to damp out any applied perturbation, it will be less useful for an accurate control of position. Since our experimental conditions did not necessitate such accurate control, little can be said on this point; (2) the characteristics of the relation between motoneuron output and muscle tension (cf. ROSENTHAL *et al.*, 1970) poses limits on the utilization of high frequencies in the control of muscle force (see below); and (3) even the fastest human movements are largely limited in their frequency bandwidth to below 5–8 Hz, and components of the power spectrum above this value decay rapidly with the frequency (cf. VIVIANI & TERZUOLO, 1973).

Acceleration feedback

Regarding the origin of the input signal for this feedback, the following should be noted: (1) HENATSCH (1971) has shown that the acceleration variable is represented in the output of the primary endings of muscle spindles; (2) the phase lead of this output, with respect to changes in muscle length, exceeds velocity at frequencies above 4 Hz; and (3) successful modelling of the behaviour of these receptors requires an acceleration term (POPPELE & BOWMAN, 1970).

Despite these facts, we suggest that a functional 'differentiation' of muscle spindle data provides the basis of the acceleration feedback. If this were not the case, the acceleration data present in the output of the primary afferents would affect the motor output at segmental latencies. As for the central structures involved, the only place in the central nervous system where it is presently known that acceleration is represented is the precentral cortex. More precisely, CONRAD *et al.* (1975) found that the activity of precentral units was related to acceleration, when pulses of torque were introduced while the monkeys were performing active movements. This observation, when taken together with our findings on the time delay for the acceleration feedback, suggests that this feedback could be mediated by the cortex. This does not exclude, however, contributions by other loops (including spinal ones), although their contribution would have to be quantitatively smaller, if the value found for this delay is viewed as a weighted average (see above).

The other finding in accord with an identification of the acceleration feedback with a transcortical loop is the change in the reflex motor output related to the acceleration variable during ballistically initiated movements when the inflow of sensory and cerebellar data to the cortex is removed by thalamic lesions (RANISH & SOECHTING, 1976). We have no suggestions to make, however, about the specific site at which the postulated functional 'differentiation' could occur.

We can now consider the utility and appropriateness of an acceleration feedback, mediated by a loop involving the cortex. We first wish to stress that potential instabilities (OGUZTORELI & STEIN, 1976) introduced by long time delays could be minimized. In fact, for frequency components of the input below 6 Hz, an acceleration feedback would still lead the velocity feedback at the time of convergence upon alpha motoneurons, even if the difference between the two time delays is as much as 40 ms. Thus, only for the sharpest transients, such as those produced by single pulses of torque, would the velocity feedback be dominant first.

As for the question whether or not an acceleration feedback could substantially improve the overall performance of reflex motor control, the following point should be considered. Under our experimental conditions, the contributions of the acceleration variable to the motor output was less than 40%. Because of the low-pass filter characteristics of the muscle contractile properties (ROSENTHAL *et al.*, 1970), the contribution by this feedback to the control of muscle force would be less (the power spectrum of the acceleration being shifted toward higher frequencies relative to velocity). Whether this situation can be generalized to other experimental conditions remains, however, to be established. More importantly, another possibility can be entertained, if indeed the feedback involves the motor cortex. This is as follows: it may be highly appropriate that the activity of a structure which generates command signals responsible (from a mechanistic viewpoint) for initiating movements, to be influenced by that parameter (acceleration) which ultimately determines both the *force* and *speed* of the movement. In other words, it is possible that the ac-

celeration feedback should be viewed less as a feedback acting on motoneuron output than as a feedback to central structures involved in the initiation of movement (in the sense stated above).

Position-related motor output

On this subject, it should be noted that an accurate control of position was not specified as a part of the task. Thus, little inference can be made about the utilization of this parameter. Also, the form of the position feedback as specified in our model may be inappropriate [see STARK (1968) for a discussion of this problem].

Acknowledgements—This work was supported by USPHS Grant NS-02567 and a grant from the American Parkinson's Disease Association. Computer facilities were made available in part by the Air Force Office of Scientific Research, AFSC (Grant AFOSR-1221).

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(*Accepted* 4 *May* 1979)